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Draft Screening Assessment

Certain Organic Flame Retardants Substance Grouping

Benzene, 1,1'-(1,2-ethanediyl)bis [2,3,4,5,6-pentabromo-

Decabromodiphenyl ethane (DBDPE)

Chemical Abstracts Service Registry Number

84852-53-9

Environment Canada

Health Canada

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Synopsis

Pursuant to section 68 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), the Ministers of the Environment and Health have conducted a screening assessment of benzene, 1,1'-(1,2-ethanediyl)bis[2,3,4,5,6-pentabromo-. This substance, commonly known as decabromodiphenyl ethane, or DBDPE, is identified by the Chemical Abstracts Service Registry Number (CAS RN) 84852-53-9. This substance is included in the Certain Organic Flame Retardants (OFR) Substance Grouping under Canada's Chemicals Management Plan, which includes ten organic substances having a similar function: the application to materials to slow the ignition and spread of fire. DBDPE was identified as priority for assessment as an evaluation of this substance done in response to notification under the New Substances provision of CEPA indicated ecological concerns. While this substance is not on the Domestic Substances List (DSL), it has been in commerce in Canada since the transitional period between the establishment of the DSL and the coming into force of the *New Substances Notification Regulations* (January 1, 1987 and July 1, 1994).

Based on a survey conducted under section 71 of CEPA 1999, as well as data from the New Substances program, DBDPE imports to Canada ranged from 1000 to 10 000 tonnes in 2011, including DBDPE in neat form, in formulations, and in consumer or commercial products. DBDPE is used in Canada as an additive flame retardant in many applications, such as plastic and rubber materials, electrical and electronic equipment, adhesives and sealants.

DBDPE does not occur naturally in the environment. Globally, sources of exposure to DBDPE are primarily from waste streams or effluents of manufacturing and processing plants using DBDPE as an additive flame retardant, but also from releases from consumer or commercial products in service. DBDPE has become commercially important since the early 1990s as a flame retardant in its own right, and more recently as an alternative for commercial decabromodiphenyl ether (DecaBDE).

Generally, DBDPE is characterized by very low water solubility, low vapour pressure, and a very high organic carbon-water partition coefficient and octanol-water partition coefficient. A close structural analogue, decaBDE, was considered for read-across of certain physical-chemical properties, as well as to predict substance behaviour in the environment. DBDPE has been measured in the Canadian environment, as well as internationally, with highest concentrations near urban and/or industrial areas. When released to the environment, DBDPE is expected to predominantly reside in soil and/or sediment. Particle-bound transport may contribute to long range transport and deposition in remote areas.

Experimental and modelled data indicate that aerobic and anaerobic biodegradation of DBDPE is limited and that DBDPE is expected to be persistent in water, soil, and sediment. Studies report that photodegradation of DBDPE may proceed quickly in solvents, but more slowly in other matrices/substrates, and modelled predictions for atmospheric degradation suggest DBDPE is persistent in air (gas phase half-life > 4

days). Although degradation of DBDPE is expected to be slow or limited, there is uncertainty with respect to ultimate transformation products in the environment. Potential DBDPE transformation products were evaluated based on predictions from photodegradation studies, biodegradation/metabolism modelling and considering analogue decaBDE. DBDPE debromination was expected to continue from nona and octaBDPEs through the formation of hepta-, hexa-, and pentaBDPEs (similar to decaBDE), or lead to a hydroxylated nonaBDPE pathway. As there are no experimental data, QSAR modelling was conducted to assess the characteristics of these potential DBDPE transformation products. Preliminary modelling indicates DBDPE transformation products can be considered analogues to lower brominated polybrominated diphenyl ethers (PBDEs), and would be persistent, would be bioaccumulative in some cases, and potentially highly toxic to aquatic organisms. The Ecological Screening Assessment on PBDEs (June 2006) concluded that lower brominated PBDEs, namely tetraBDE, pentaBDE and hexaBDE, satisfy the criteria outlined in the Persistence and Bioaccumulation Regulations of CEPA 1999.

Empirical data indicate that DBDPE may accumulate to some degree in the tissues of biota (low to moderate bioaccumulation potential); however, at present there is inadequate evidence indicating that the substance has the potential for high bioaccumulation.

Based on soil chronic toxicity testing, DBDPE has the potential to cause reproductive effects at high concentrations to earthworms as well as effects on plant survival and growth. No effects up to the highest tested dose (5000 mg/kg) were observed for sediment organisms in chronic toxicity tests. No water (pelagic) critical toxicity value (CTV) is determined for DBDPE in this assessment, based on uncertain aquatic test results. Information gaps on the toxicity of DBDPE to wildlife and effects on pelagic, sediment and terrestrial species from prolonged (e.g., lifetime and mutigenerational) exposure, as well as recent aquatic and sediment analogue decaBDE studies reporting effects at low concentrations, highlight the possibility that future DBDPE toxicity studies may determine similar effects at low concentrations. This uncertainty was considered in the application of precaution for the assessment.

It is expected that DBDPE may be released to the Canadian environment as a result of industrial processing activities. Additive use of DBDPE in products suggests diffuse emissions may occur from consumer or commercial products and, although there are uncertainties, the rate is assumed to be low in comparison to industrial point sources during incorporation of the substance into products. Industrial scenarios (which considered available site information), with DBDPE release to water and predicted partitioning to sediment and releases to soil, were used to estimate exposure. Risk quotient analyses, integrating conservative estimates of exposure with toxicity information, were performed for the sediment and terrestrial compartments (soil and wildlife). These analyses showed that current risks posed by the parent DBDPE are low.

A risk quotient analysis for DBDPE transformation products was not conducted given the lack of information on transformation product quantity in Canada. Transformation products are expected to represent a minor fraction relative to parent DBDPE; however, they are similar to predicted/measured fractions of analogue decaBDE debromination products, and if DBDPE levels in the environment continue to increase (e.g., due to its use as a replacement flame retardant), the pool of potential brominated transformation products could become important.

Considering the evidence presented in this draft screening assessment for DBDPE and the potential for persistence, bioaccumulation and inherent toxicity of some transformation products, there is risk of harm to organisms, but not to the broader integrity of the environment from DBDPE. It is proposed to conclude that DBDPE meets the criteria under paragraph 64(*a*) of CEPA 1999 as it is entering or may enter 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. However, it is proposed to conclude that DBDPE does not meet the criteria under paragraph 64(*b*) of CEPA 1999 as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger to the environment on which life depends.

No classifications of the health effects of DBDPE by national or international regulatory agencies were identified. No chronic or carcinogenicity studies using DBDPE were identified. On the basis of the available information regarding genotoxicity, DBDPE is not considered genotoxic. No adverse effects were observed in sub-chronic animal studies. In two separate developmental toxicity studies, no treatment-related maternal or developmental effects were observed in experimental animals exposed to DBDPE via the oral route. Limited biomonitoring data in humans is available.

The highest doses tested in experimental animal studies, with no treatment related effects, are seven orders of magnitude higher than the estimates of exposure to DBDPE from environmental media for the Canadian general population. This margin is considered adequate to account for uncertainties in the health effects and exposure databases. Based on this, it is proposed that DBDPE does not meet the criteria under paragraph 64(*c*) of CEPA 1999.

Overall Proposed Conclusion

It is proposed to conclude that DBDPE meets one or more of the criteria set out in section 64 of CEPA 1999. DBDPE is proposed to meet the persistence, but not bioaccumulation criteria as set out in the *Persistence and Bioaccumulation Regulations of CEPA 1999*. However, DBDPE may contribute to the formation of persistent, bioaccumulative, and inherently toxic transformation products, such as lower brominated BDPEs, in the environment.

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1. Introduction

Pursuant to sections 68 and 74 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999), the Minister of the Environment and the Minister of Health conduct screening assessments of substances to determine whether these substances present or may present a risk to the environment or to human health.

The Substance Groupings Initiative is a key element of the Government of Canada's Chemicals Management Plan (CMP). The Organic Flame Retardant (OFR) Substance Grouping consists of ten substances that were identified as priorities for assessment, as they met the categorization criteria under section 73 of CEPA 1999, and/or were considered as a priority based on ecological and/or human health concerns (Environment Canada and Health Canada 2007). All of these substances have a similar function: the application to materials to slow the ignition and spread of fire. Also, these substances are potential alternatives for other flame retardants which are presently subject to regulatory controls or phase-out in Canada and/or globally. This draft screening assessment focuses on the substance benzene, 1,1'-(1,2-ethanediyl)bis[2,3,4,5,6-pentabromo-, or decabromodiphenyl ethane (DBDPE) (CAS RN 84852-53-9).

As DBDPE is not present on the Domestic Substances List (DSL), it is subject to the *New Substances Notifications Regulations* (Chemicals and Polymers) pursuant to CEPA 1999 (Canada 2005). Following New Substances ecological and human health risk assessments, the evaluation indicated ecological concerns and this substance was suspected of being "Toxic." DBDPE has been in commerce in Canada since the transitional period between the establishment of the DSL and the coming into force of the *New Substance Notification Regulations* (between January 1, 1987 and July 1, 1994). Risk management measures (i.e., Ministerial Conditions) have been imposed on New Substance notifiers to mitigate potential risks to the environment.

Screening assessments focus on information critical to determining whether substances within a grouping meet the criteria as set out in section 64 of CEPA, by examining scientific information to develop conclusions by incorporating a weight-of-evidence approach and precaution.[1]

[1] A determination of whether one or more of the criteria of section 64 are met is based upon an assessment of potential risks to the environment and/or to human health associated with exposures in the general environment. For humans, this includes, but is not limited to, exposures from ambient and indoor air, drinking water, foodstuffs, and the use of consumer products. A conclusion under CEPA 1999 is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Hazardous Products Regulations* and the *Controlled Products Regulations* which are part of the regulatory framework for the Workplace Hazardous Materials Information System

This draft screening assessment includes consideration of information on chemical properties, environmental fate, hazards, uses and exposure, as well as additional information submitted by stakeholders. Relevant data were identified up to at least April 2014 for the ecological assessment and August 2014 for the human health assessment. However, a cursory search was conducted to include any salient literature up to June 2015. Empirical data from key studies, analogue data, as well as some results from models were used to reach proposed conclusions. When available and relevant, information presented in assessments from other jurisdictions was considered. The draft screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents the most critical studies and lines of evidence pertinent to the proposed conclusion.

This draft screening assessment was prepared by staff in the Existing Substances Programs at Health Canada and Environment Canada and incorporates input from other programs within these departments. The ecological and human health portions of this assessment have undergone external written peer review and/or consultation. Comments on the technical portions relevant to the environment were received from Jon Arnot (Arnot Research and Consulting), John Biesemier (Chemtura), Adrian Covaci (University of Antwerp), Miriam Diamond (University of Toronto), and Marcia Hardy (Albermarle). Comments on the technical portions relevant to human health were received from Michael Jayjock of the LifeLine group, Paul Rumsby of the U.S. National Centre for Environmental Toxicology, and Pam William of E Risk Sciences. While external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment Canada.

2. Substance Identity

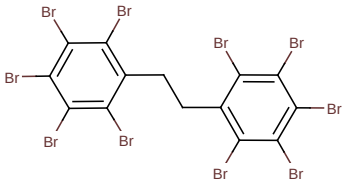
2.1 Substance Identity

Benzene, 1,1'-(1,2-ethanediyl)bis[2,3,4,5,6-pentabromo- or decabromodiphenyl ethane (DBDPE) is an organic flame retardant, grouped with the OFRs under the Substance Grouping Initiative of the CMP. The structural identity of this substance is presented in Table 2-1. Other names for the substance are presented in Appendix A (Table A-1). For this assessment, decabromodiphenyl ethane will be referred to as DBDPE.

Table 2-1. Substance identity for Decabromodiphenyl ethane

CAS RN	Chemical structure	Molecular mass	Chemical formula

for products intended for workplace use. Similarly, a conclusion based on the criteria contained in section 64 of CEPA 1999 does not preclude actions being taken under other sections of CEPA 1999 or other Acts.

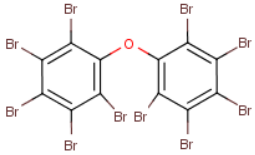
CAS RN	Chemical structure	Molecular mass	Chemical formula
84852-53-9		971.23 g/mol	$C_{14}H_4Br_{10}$

2.2 Selection of Analogues and Use of (Q)SAR Models

Guidance on the use of a read-across approach and Quantitative Structure-Activity Relationships or (Q)SAR models for filling data gaps has been prepared by various organizations such as the Organisation for Economic Co-operation and Development (OECD). These methods have been applied in various regulatory programs including the European Union's (EU) Existing Substances Programme. In this assessment, a read-across approach using data from analogues and the results of (Q)SAR models, where appropriate, have been used to inform the ecological and human health assessments. An analogue was selected that was structurally similar and/or functionally similar to DBDPE (e.g., based on physical-chemical properties, toxicokinetics), and that had relevant empirical data that could be used to read-across. The applicability of (Q)SAR models was determined on a case-by-case basis. Details of the read-across data and (Q)SAR models chosen to inform the ecological and human health assessments of DBDPE are further discussed in the relevant sections of this report.

The analogue used to inform the ecological assessment is presented in Table 2-2. Decabromodiphenyl ether (decaBDE) represents a close structural analogue, and is considered appropriate for certain physical-chemical properties (e.g., octanol-water partition coefficient (logKow), water solubility, vapour pressure). DecaBDE, is discussed throughout the assessment in comparisons of substance behaviour with DBDPE (e.g., degradation, long range transport, bioaccumulation potential, ecotoxicity etc.). However, it is noted that differences in molecular makeup, dimensions, and configurations exist between DBDPE and decaBDE that may affect the manner in which these molecules interact with their environment (2014 manufacturer communication to Environment Canada; unreferenced).

Table 2-2. Analogue identity

Substance CAS RN	Substance name	Molecular Weight (g/mol)	Empirical Structure/ Molecular Formula/
1163-19-5	decabromodiphenyl ether (decaBDE)	959.171	 C12-Br10-O

3. Physical and Chemical Properties

Physical and chemical properties determine the overall characteristics of a substance and are used to determine the suitability of different substances for different types of applications. Such properties also play a critical role in determining the environmental fate of substances (including their potential for long-range transport), as well as their toxicity to humans and non-human organisms. A summary of experimental, modelled, and key values for the physical and chemical properties of DBDPE that are relevant to its environmental fate and ecotoxicity can be found in Table 3-1. A detailed table of physical and chemical properties of DBDPE (empirical and modelled) and a summary of analogue physical and chemical properties can be found in Appendix B.

DBDPE was considered amenable to model prediction of physical-chemical properties using (Q)SARs, as it is within the model domain of applicability (i.e., structural and/or property parameter domains are represented in the training set used for the models).

Physical-chemical properties of DBDPE were checked for internal consistency according to the Least-Squares Adjustment Procedure (LSA) (Schenker et al. 2005). Geometric mean or arithmetic mean (for logarithmic variables) values of the most reliable and independent values found from empirical data, modelling, and an analogue were used to determine the inputs to the LSA (Appendix B, Table B-1; for all Physical-Chemical values see Table B-2). Subcooled adjusted values were input for water solubility, vapour pressure, and octanol solubility (Schenker et al. 2005). In determining internal consistency of the properties, the LSA model also produces predicted values. While experimental based estimates for log Kow, water solubility, and vapour pressure exist for DBDPE, there remains uncertainty with these values, in particular, with the experimental logKow value of 3.55 (e.g. Stieger 2014). For the purposes of this assessment, the logKow value 9.89, derived from the LSA method, was selected. To maintain internal consistency of physical-chemical values, the LSA method value for water solubility and vapour pressure were also considered. Final selected values are summarized in Table 3-1.

Generally, DBDPE is characterized by very low water solubility, low to very low vapour pressure, and a very high organic carbon-water partition coefficient and octanol-water partition coefficient.

Table 3-1. A summary of the physical and chemical properties for DBDPE

Property	Experimental	Modelled	Selected Value for Modelling[c]
Physical state	(off) white powder	N/A	N/A
Melting point (ºC)	345 - 355	259.7	345
Boiling point (ºC)	N/A- degrade before boiling	600.9	N/A
Density (kg/m^3)	868 – 3250 (packed)	N/A	N/A

Vapour pressure (Pa)	~1 x 10^{-6a} - <1 x 10^{-4}	2.85 x 10^{-16} - 5.59 x 10^{-10}	5.59 x 10^{-10} (liquid subcooled 8.21 x 10^{-7})
Henry's Law constant ($Pa \cdot m^3/mol$)	NA	2.59 x 10^{-4}- 6.71 x10^{-2}	6.51 x10^{-3} to 6.71 x10^{-2}
Log K_{ow} (dimensionless)	~3.55[b], 8.7[a]	7.86 -13.64	9.89
Log K_{oc} (dimensionless)	NA	6.38 – 8.58	8.58
Log K_{oa} (dimensionless)	NA	14.45– 19.22	14.45
Water solubility (mg/L)	<1 x 10^{-4a} – 7.2 x 10^{-4}	7.34 x 10^{-10} - 2.15 x 10^{-2}	8.10 x10^{-6} (liquid subcooled: 1.19 x10^{-2})
pKa	N/A	N/A	N/A

[a] Read-across value from analogue decaBDE.
[b] Experimentally estimated logKow value (3.55) evaluated to be highly uncertain and therefore not included in mean logKow for LSA.
[c] See Appendix B, Table B-2 for detailed physical-chemical property values and references.

4. Sources

There is no reference in the published literature for the natural occurrence of DBDPE in the environment. Sources of exposure to DBDPE are anthropogenic, primarily from waste streams or effluents of manufacturing and processing plants using DBDPE as an additive flame retardant, and release from consumer or commercial products to the environment.

DBDPE has become increasingly important commercially since the 1990s as a flame retardant in its own right and as a replacement for commercial Decabromodiphenyl Ether (DecaBDE) (Kierkegaard et al. 2004, Covaci et al. 2011; EFSA 2012). Manufacturers of decaBDE have voluntarily phased out the production, import, and sale of Deca-BDE by 2012 in cooperation with the US Environmental Protection Agency (EPA) and Environment Canada (BSEF c.2001-2015). There also exist governmental efforts to limit the manufacturing and use of decaBDE (Environment Canada c.2006-2013). In Canada, risk management of decaBDE is supported by concerns respecting the transformation of decaBDE to lower brominated polybrominated diphenyl ethers (PBDEs), which include tetra-, penta- and hexaBDEs, substances considered to be highly persistent and bioaccumlative (Environment Canada 2010, Canada c.2006-2013).

In Canada, as DBDPE is not present on the DSL, it is subject to the *New Substances Notifications Regulations (Chemicals and Polymers)* pursuant to CEPA. Based on a survey conducted under section 71 of CEPA, and considering data from New Substances notifications, the total quantity of DBDPE imported into Canada in 2011 was

in the range of 1000 – 10 000 tonnes, including DBDPE in some products. No DBDPE was identified as being manufactured in Canada. The total quantity of DBDPE exported out of Canada in 2011 was less than 100 tonnes (Canada 2005, ECCC 2013-2014).

Globally, manufacturing of DBDPE is known to occur in the United States (US) (Covaci et al. 2011, US EPA 2012). In the US, DBDPE is a chemical on the Toxic Substances Control Act (TSCA) Inventory, and the substance is subject to a Significant New Use Rule (SNUR), which indicates that certain measures should be taken in the absence of sufficient information to make a decision on the potential for the substance to cause an unreasonable risk of injury to human health or the environment. In 2012, the US production/import volume was between 22 720 to 45 450 metric tonnes (50 to 100 million pounds) (US EPA 2012). According to the US EPA 2012 Chemical Data Reporting (CDR), 5 companies were associated with DBDPE as producers or importers in the US.

No manufacturing of DBDPE has been reported to occur in Europe (Environment Agency 2007). Although the substance is listed as a Low Production Volume chemical (under 1000 tonnes/yr) on the European Chemical Substance Information Systems (ESIS) website (searched March 2014)(ESIS 1995-2012), other sources suggest use in Europe may be higher. For example, use in Europe has been estimated at 2500 tonnes/year with data trends suggesting increasing consumption (Environment Agency 2007), primarily in Germany (Covaci et al. 2011). A Substances in Preparations in Nordic Countries (SPIN) database (searched April 2014) indicated records of use for 2006, and 2008 through 2011 in Sweden, with substance use ranging between 5 and 39 tonnes/year (SPIN 2006). The substance is listed as a High Production Volume chemical on the OECD Existing Chemicals Website (searched March 2014).

Recent production (e.g., 2006) of DBDPE in China has been reported to range from 11 000 to 12 000 tonnes per year (Shi et al. 2009, Zhang et al. 2009). Shi et al. (2009) suggests that China has become a significant brominated flame retardant producer during the past decades.

5. Uses

DBDPE is made by the direct bromination of diphenylethane (Weil and Levchik 2009), and marketed globally under different trade names (see Appendix A, Table A-1). DBDPE is reported to be a relatively pure substance; the commercial grade is reported to be typically 96 to 98.5% (by weight) pure, with the remainder consisting largely of nonabromodiphenyl ethane congeners (<2% by weight), and octabromodiphenyl ethane congeners <1% by weight (Chemtura 2005, Environment Agency 2007, Albermarle 2008, Albermarle 2013).

In Canada, as DBDPE is not present on the DSL, it is subject to the *New Substances (NS) Notifications Regulations (Chemicals and Polymers)* pursuant to CEPA 1999 (Canada 2005). Recent risk management measures (i.e., Ministerial Conditions), based on New Substances ecological risk assessments, have limited the import of the

substance for use as a flame retardant component of wire and cable coatings, thermoplastic parts, thermoplastic coatings, thermoset parts and thermoset coatings, as well as placing some restrictions on its release and disposal (Canada 2004, 2011). According to submissions made under section 71 of CEPA 1999 (Canada 2013) and publications under the New Substance Notifications Regulations, (Canada 2004, 2011), DBDPE is used in Canada as a flame retardant in applications of: plastic and rubber materials such as thermoplastic or thermoset parts and coatings (for use in polymer resins and polymer plastics); electrical and electronics including appliances and wire and cable coatings for the telecommunications industry; automotive, aircraft, and transportation, adhesives and sealants, appliances; and basic organic chemical manufacturing. DBDPE was also reported to be used in the manufacture of airbag textile and generally in motor vehicles (ECCC 2013-2014). It is expected that typical polymer loading rates are similar to those of decaBDE, i.e., 10-15% by weight (Environment Agency 2007).

Globally, DBDPE is used as a substitute for decaBDE, and is therefore used in similar applications, such as the manufacture of plastics (including polyester and vinyl ester resins) and rubber products, and as an additive in textiles, such as cotton and polyester (Covaci et al 2011). The substance is also used in polymers used for electronic and electrical applications, as well as in adhesives and sealants (EFSA 2012). Manufacturer literature indicates DBDPE is suitable for use in systems where mechanical recycling is anticipated, due to very good thermal stability and low blooming characteristics (i.e., additives migrating to the surface of the material over time) in finished resins (Albermarle 2007).

Available information from Europe has indicated that the major use of DBDPE in Europe and the United Kingdom (accounting for at least 90% of the tonnage supplied) is as an additive flame retardant for polymers. The remaining consumption is expected to be largely for textiles (Environment Agency 2007). SPIN database records of use for 2006, and 2008 through 2011 for Sweden, indicate that all DBDPE use falls in the category of manufacture of rubber and plastic products (specifically flame retardants and extinguishing agents). For Finland, records note DBDPE use for the manufacture of other transport equipment (specifically adhesives and binding agents).

Currently, DBDPE is the second highest used additive brominated flame retardant (BFR) in China with production increasing at 80% per year (Covaci et al. 2011). This is likely related to the rapid growth of its electrical and electronic industries during the past decades (Shi et al. 2009). In Japan, the use of DBDPE is likely replacing the use of DecaBDE (Watanabe and Sakai 2003, Covaci et al. 2011). The use of DBDPE increased continuously in Japan from 1993 to 2000, whereas the consumption of DecaBDE decreased over the same time period (de Wit et al. 2011).

DBDPE is not listed as an approved food additive in the Lists of Permitted Food Additives as regulated under the *Food and Drugs Act*, nor has it been identified as being used/present in formulations of food packaging materials or incidental additives (Health Canada 2013, August 2013 email from Food Directorate, Health Canada, to

Risk Management Bureau, Health Canada; unreferenced). DBDPE is not listed in the Drug Products Database, the Therapeutic Products Directorate's internal Non-Medicinal Ingredient Database, the Natural Health Products Ingredients Database or the Licensed Natural Health Products Database as a medicinal or non-medicinal ingredient present in final pharmaceutical products, natural health products or veterinary drugs in Canada (DPD 2013; NHPID 2014; July 2013 email from the Therapeutic Products Directorate, Health Canada, to Risk Management Bureau, Health Canada; unreferenced). Based on notifications submitted under the *Cosmetic Regulations* to Health Canada, DBDPE is not anticipated to be used in cosmetic products in Canada (June 2013 email from the Consumer Product Safety Directorate, Health Canada to the Risk Management Bureau, Health Canada; unreferenced).

DBDPE is not in any registered products regulated under the *Pest Control Products Act* (May 2012 email from Pest Management Regulatory Agency, Health Canada to the Risk Management Bureau, Health Canada; unreferenced).

With increasing regulation and phasing-out of production of the polybrominated diphenyl ethers (PBDEs), it is expected that the production and usage of DBDPE is likely to increase (Ricklund et al. 2008).

6. Releases to the Environment

Anthropogenic releases to the environment depend upon various losses occurring during the manufacture, industrial use, consumer/commercial use, service life and disposal of a substance. Releases of DBDPE to the Canadian environment, due to the substance's use as an additive flame retardant, are expected to be from both point sources (e.g., from processing facilities, product manufacturing) as well as from diffuse sources. Releases may occur in both indoor and outdoor environments.

According to submissions made under section 71 of CEPA 1999 and publicly available technical literature, DBDPE is imported into Canada in neat form, as formulations, and consumer or commercial products (Canada 2013, CCC 2011).

DBDPE release to the environment is most likely to occur during the manufacturing, formulation and/or industrial use stages of these sectors. Releases to the environment are expected to occur primarily through wastewater, with some release to water directly from industrial sites. Canadian effluent and wastewater sludge data show that publicly owned wastewater treatment plants (WWTP) with higher proportional industrial inputs (e.g., >30%) have higher DBDPE concentrations (e.g., 10X) than those dominated by domestic (non-industrial) influent input (Kim et al. 2014, Melymuk et al. 2014). Release to the soil could occur through the application of wastewater biosolids to agricultural and pasture lands.

In terms of migration from consumer /commercial products, as an additive brominated flame retardant that is blended with the polymer product (rather than a reactive flame retardant chemical bonded to the polymer product), there is the possibility of some

release from consumer products to the environment (Guerra et al. 2011). DBDPE is proposed to be released to air or dust by volatilization or abrasion of product containing flame retardant substance (Melymuk et al. 2014), which could result in DBDPE deposition to soil, water, and/or release to publicly owned WWTPs.

Although DBDPE has low volatility, emissions to air (e.g., from airborne particles, dust or release from products) could result in atmospheric deposition to soil and water. For example, a pattern of increasing pond sediment DBDPE concentrations with proximity to chemical manufacturing facilities has been attributed largely to transport through the movement of air and airborne particles (Wei et al. 2012). When a substance is transferred to land, it may become bound to soil, be washed into the sewer or surface water or transferred by wind or rain to nearby soil.

Finally, landfills that do not collect and treat their leachate may potentially release substances to ground or surface water via leachate or, although unlikely, there is potential for releases of substances to the atmosphere through gas from landfills that do not collect and destroy their landfill gas.

This information and fate in the environment are used to further develop exposure characterization scenarios to estimate resulting environmental concentrations.

7. Measured Environmental Concentrations

There are challenges to measuring and analyzing very hydrophobic substances like DBDPE in environmental media, including very low solubility in water and organic solvents; tendency to adsorb to particulates and solids (e.g., organisms and/or chamber walls); degradation during clean-up and instrumental analysis; and generally less expertise in the analysis of the substance (Breitholtz et al. 2006, Kierkegaard et al. 2009). DBDPE is a difficult brominated flame retardant to analyze, and uncertainty can reach 40-60%, depending on the internal standard used for quantification (2014 communication from A. Covaci to Environment Canada; unreferenced). Most studies to date have analyzed DBDPE using gas chromatography/mass spectrometry (GC-MS) using analysis for the bromide ion.

DBDPE has been reported in the Canadian environment, as well as in other countries, generally at low levels. Highest concentrations of DBDPE tend to be found close to urban or industrial areas. For specific study results and details (including detection limits, sample size, etc.) see the Supporting Information document (Environment Canada 2015). For a simplified range of concentrations measured in Canada and globally, see Tables 7-1 and 7-2.

Few studies have reported the presence of DBDPE in air. Air and precipitation samples were collected every 12 days at five sites near the North American Great Lakes from 2005 to 2011 (inclusive) by the Integrated Atmospheric Deposition Network (IADN). Based on IADN data, Ma et al. (2013) reported overall DBDPE average atmospheric concentrations (vapour + particle) of 1.2 to 5.2 pg/m3 for the five sites (detection

frequency ranged from 8 to 54%), with the highest concentrations near urban areas (increasing as a function of population). Vernier and Hites (2008) evaluated earlier data from the same sites, and determined a maximum mean concentration of ~ 22 pg/m^3 near an urban area (Cleveland) but lower levels in a remote area (Eagle Harbour, Michigan,~ 1 pg/m^3, read from graph). Global air monitoring through the Global Atmospheric Passive Sampling (GAPS) network which measures more than 40 sites reported one detection of DBDPE (concentration not specified) in North America in 2005 (Lee et al. 2010).

In the Canadian Arctic (Devon Ice Cap, Nunavut), DBDPE was detectable in some horizons of snow pits; however, the concentration patterns did not show clear deposition time trends (Meyer et al. 2012), and the concentrations (Non Detected (ND) – 24 pg/L, detected only twice) were, on average, lower than those reported for the Norwegian Arctic. DBDPE has been measured in precipitation in the Great Lakes area, with mean concentrations of 256 to 1440 pg/L between 2003 and 2009 (Salmova and Hites 2010, Salmova and Hites 2011).

To date, only one study has detected DBDPE in Canadian surface water (Venier et al. 2014), reporting Great Lake basin wide DBDPE concentrations from an average of 0.25 ± 0.05 pg/L (Lake Huron) to 10.8 pg/L (Lake Ontario). In the same study, the DBDPE concentration observed in Lake Superior was 6.7 pg/L, influenced by a sampling station near the heavily industrialized urban centre of Thunder Bay, Ontario. Other studies of Canadian surface water have not detected DBDPE (Law et al. 2006, Muir et al. 2011).

While no soil measurements for DBDPE have been reported for Canada or North America, DBDPE has been measured and detected in soil in Asia (e.g., 1.13 (farmland) to 1612 (industrialized land of an e-waste site) ng/g dw for China (Lin et al. 2015).

DBDPE sediment concentrations have been reported for the Great Lakes (0.11 to ~200 ng/g dw) (Kolic et al. 2009, Yang et al. 2012). A recent sediment core study from the Great Lakes (Canada and the United States) reported DBDPE surface sediment concentrations ranging from 0.11 to 2.8 ng/g dw, with the highest concentrations in Lake Michigan (up to 2.5 ng/g dw) and Lake Huron (up to 2.8 ng/g dw) (Yang et al. 2012). DBDPE was the sixth most frequently detected (46% detection) of the 13 BFRs surveyed. While DBDPE surface sediment concentrations were approximately one order of magnitude lower than those of decaBDE (0.87 to 106 ng/g dw), DBDPE input is increasing rapidly, with sediment concentrations estimated to double every 3-5 years in Lake Michigan and approximately every 6 to 7 years in Lake Ontario (Yang et al. 2012). In another study, Kolic et al. (2009) presented DBDPE concentrations ranging from approximately 8 to 200 ng/g dw (read from graph) in surface sediment from Lake Ontario and its tributaries. However, DBDPE was not detected in the sediments of Lake Winnipeg during a 2003 sampling program (Law et al. 2006). In an Arctic marine sediment study, Cai et al. (2012) measured DBDPE in sediment from the Canada Basin, Chukchi Sea, and Bering Sea of the western Arctic Ocean (non-detect to 452.6 pg/g dw), with an average of 166.7 pg/g dw. These concentrations were on the same order of magnitude as decaBDE concentrations measured in the same study.

Elsewhere in North America, Wei et al. (2012) reported a pattern of increasing lake or pond sediment DBDPE concentrations with increasing proximity to chemical manufacturing facilities that produce DBDPE (and DecaBDE) in Arkansas, USA, including the highest concentration yet reported for DBDPE (up to 2394 ng/g dw). As there are no manufacturing water releases to surface waters (but WWTP water and sludge input occurred at on site from 1952 to1989), the dominant pathway for transport of DBDPE and decaBDE from the emission sources to the sampling sites was assumed to be through the movement of air and airborne particles.

DBDPE has been widely reported in wastewater effluent and sludge (a potential route to the surface water and soil environments). In Canada, a recent study of 20 WWTP by Kim et al. (2014) reported mean DBDPE concentrations in final effluent to range from ND to 7.1 ng/L, with 86% of samples having nondetectable levels of DBDPE. Another study of six WWTPs in Canada found DBDPE was detected in two of four samples, estimated at ~3 ng/L (value read from graph) (Zhou et al 2010a). DBDPE estimates for WWTP sludge vary greatly. Within Canada, measurements from Ontario range from 5.6 ng/g dw (wastewater sludge) (Konstantinov et al. 2006) to ~100 ng/g dw (Kolic et al 2009, value read from graph), although the level of treatment was not indicated. A study of 20 WWTPs in Canada reported treated biosolids concentrations ranged from non-detect to 220 ng/g dw (Kim et al. 2014).

In Canada, DBDPE has been sampled in the fish tissue of several freshwater species, and concentrations have generally ranged from non-detect to very low (i.e., mean concentration ≤ 1 ng/g lipid weight (lw) (or wet weight (ww)) (Law et al. 2006, Ismail et al. 2006, Kolic et al. 2009, Byer et al. 2010, Byer 2013, Zhou et al. 2010b, Muir et al. 2011, Environment Canada 2014). The exception is a study reporting liver concentrations in 1 of 11 northern pike from the St. Lawrence River area and tributaries of 26.7 ng/g lw (3.78 ng/g ww) (Houde et al. 2014). DBDPE was not detected in mussels and plankton in Lake Winnipeg (2000 to 2002) (Law et al 2006), nor in zooplankton sampled between 2006 and 2010 in the Great Lakes (Lake Ontario and Lake Erie), nor in a remote Ontario lake (Lake Opeongo) (Muir et al 2011).

Avian studies in Canada have occasionally detected DBDPE. DBDPE was not detected in eggs of four gull species (glaucous-winged (*Larus glaucescens*), California (*Larus californicus*), ring-billed (*Larus delawarensis*), and herring gulls (*Larus argentatus)* collected from 26 colonies across Canada (Atlantic to Pacific) (Chen et al. 2012). DBDPE was detected in 1 of 12 Peregrine Falcon eggs collected from the Great Lakes watershed, at 8.2 ng/g lw (Guerra et al. 2012), but DBDPE was not detected in Bald Eagle plasma collected from birds in the Great Lakes area, despite high levels of decaBDE in the same samples (Venier et al. 2010). Of the pools of eggs from seven Laurentian area Great Lakes colonies of herring gulls (*Larus argentatus*) collected between 1982 and 2006 (Gauthier et al. 2009), DBDPE was not detected prior to 1996, but was detected (mean concentration up to 11 ng/g ww) in 5 of 63 non-consecutive pools of eggs between 1996 and 2004. In 2005, DBDPE was detected in eggs from 3 of 7 colonies with concentrations up to 288 ng/g ww, and in 2006, eggs from 2 of 7 colonies had DBDPE concentrations up to 44 ng/g ww (Gauthier et al. 2009)

Ringed seal blubber samples from the Canadian Arctic in 2006 did not have measurable levels of DBDPE (de Wit et al 2011). Polar bear adipose tissue samples from the Canadian Arctic, Alaska, and Svalbard, collected between 2005 and 2008, detected DBDPE in less than 14% of samples (McKinney et al. 2011b).

Table 7-1. Environmental Concentration Range, Canada[a,b]

Media	Location(s)	Years (not continuous)	Concentration Range
Air (pg/m^3)	Nunavut, Great Lakes	2005 to 2008	ND – 22
Surface Water (pg/L)	Lake Winnipeg, Great Lakes, Ontario	2004 to 2012	ND-10.8
Sediment (ng/g dw)	Lake Winnipeg, Great Lakes, Ontario, Canada Basin (arctic marine)	2003 to 2008	ND – 200
Wastewater effluent (ng/L)	Ontario	NS	ND – 7.1
Biosolids, sludge (ng/g dw)	Ontario	2003 to 2010	ND – 220
Biota – aquatic ng/g lw	St. Lawrence, Ontario, Quebec, New Brunswick, Nova Scotia, Great Lakes, Lake Winnipeg	2000 to 2012	ND – 26.7
Biota –terrestrial and avian ng/g lw	Great Lakes, Canadian Arctic, Southern NWT	1982 to 2010	ND – 8.2[c]

Abbreviations: ND = not detected; NS = Not stated.
[a] See Supporting Information (Environment Canada 2015) for references and study details.
[b] Although wastewater system effluent and sludge/biosolids are not "environment," they represent a direct source to the environment and are included in this table.
[c] Note the DBDPE range reported for terrestrial and avian organisms in "wet weight" (ww) is ND to 288 ng/g.

Table 7-2. Environmental Concentration Range, Global[a]

Media	Location(s)	Years (not continuous)	Concentration Range
Air (pg/m^3)	North America, Europe, Africa	2003 to 2011	ND - 3578
Surface Water (pg/L)	North America, Europe, Asia	2003 to 2010	ND – 38
Soil (ng/g dw)	Asia	2006 to 2007	ND – 1612
Sediment (ng/g dw)	North America, Europe, Asia	2002 to 2009	ND - 2394
Wastewater, effluent (ng/L)	North America, Europe	2006 to 2009	ND – 7.1 (±5.6 SD)
Biosolids, sludge (ng/g dw)	North America, Australia, Africa, Asia,	1998 to 2010	ND – 4820
Biota – aquatic ng/g lw	North America, South America, Asia, Europe	1986 to 2010	ND - 352

Biota – terrestrial and avian ng/g lw	North America, Europe, Asia	1982 to 2010	ND – 863

[a] See Supporting Information (Environment Canada 2015) for references and study details.
[b] Although wastewater system effluent and sludge/biosolids are not "environment," they represent a direct source to the environment and are included in this table.

However, it is also important to consider how DBDPE levels in the environment may increase in future, for example, relative to the OFR it is proposed to replace, decaBDE. Ma et al. (2013) recently determined that DBDPE particle air concentrations in the Great Lakes area were similar to decaBDE air concentrations at most sampling locations, with the exception of Cleveland where manufacturing of decaBDE was expected to occur; the authors suggest the pattern indicates both substances are in use and share similar applications and sources (e.g., consumer products). Goosey et al. (2013) also determined DBDPE dust concentrations in Toronto homes to be similar to those of decaBDE (see Human health section), both likely related to electrical and electronic equipment.

A comparison of DBDPE to decaBDE measurement ratios for WWTP data can provide evidence of where DBDPE use is high and/or substitution for decaBDE has occurred (Ricklund et al. 2008). Kim et al. (2013; 2014) measured both DBDPE and decaBDE in the same influent and effluent samples from 20 WWTPs in Canada, representing populations of 1500 to >1 000 000. The median influent DBDPE concentration was: 3.7 ng/L (maximum= 130 ng/L), while the median decaBDE concentration was 74.8 (and maximum= 433 ng/L), resulting in a median [DBDPE]/[decaBDE] influent ratio of 0.05. Similarly, the median final effluent DBDPE concentration was 0.2 (and maximum=7.1 ng/L), while the median decaBDE concentration was 3.7 (and maximum=59.9 ng/L), resulting in a similar [DBDPE]/[decaBDE] ratio of 0.055. An international survey of DBDPE in wastewater sludge (Ricklund et al. 2008), showed ratios for Canadian samples of [DBDPE]/[decaBDE] in sludge that ranged from 0.01 to 0.078. These ratios in both wastewater and sludge/biosolids indicate that DBDPE release reaching WWTPs (including that from consumer or commercial products) is still lower than that of decaBDE.

8. Environmental Fate and Behaviour

8.1 Environmental Distribution

DBDPE is expected to be released to the environment primarily through wastewater, but may undergo some migration from products to the atmosphere as non-reactive brominated flame retardants have potential for some release from polymers (e.g., PBDEs)(Guerra et al. 2011). DBDPE is very likely highly removed by adsorption to sludges in wastewater treatment plants and can be applied to agricultural soils during biosolids amendment. Level III fugacity modelling (Table 8-1) using the updated EQC model (v 1.0, 2012), was applied to describe the fate for these expected modes of entry into the environment. Generally, the results of Level III fugacity modelling show that

DBDPE is expected to predominantly reside in soil and/or sediment, depending on the compartment of release.

Table 8-1. Results of the Level III fugacity modelling (New EQC 2012) for DBDPE

Substance released to:	Air (%)	Water (%)	Soil (%)	Sediment (%)
Air (100%)	0.5	0.4	82.7	16.4
Water (100%)	Negligible	2.5	Negligible	97.5
Soil (100%)	Negligible	Negligible	99.9	0.1

Very low water solubility (8.10×10^{-6} mg/L), low vapour pressure (5.59×10^{-10} Pa at 25°C), low air–water partition coefficient (log K_{aw} =-4.57), and very high partition coefficients (log K_{ow} of 9.89, estimated log K_{oc} of 8.58) suggest that DBDPE released into the environment will be less likely to partition into and/or remain in air and water, moving instead to the sediments and soil. If released to air, a small fraction (<1%) DBDPE is expected to remain in air, with most of the substance depositing to soil and water with further partitioning to sediment. However, based on predicted rates of degradation (>4 days) and predicted patterns of transport (see description below), the small mass of DBDPE that remains in air has the potential for dispersion.

The high partition coefficients indicate that DBDPE released into surface water from wastewater treatment systems is expected to adsorb to the organic fraction of suspended solids and sediments, with 2.5% remaining in water. Volatilization from surface water to air is not expected. However, as in the case with air, the small fraction remaining is likely persistent and has the potential for some transport (e.g., particle transport). Based on its high log K_{oc}, once in the sediment, DBDPE is not expected to be mobile, and may remain in this compartment with little degradation.

When DBDPE is released to soil as a function of biosolids application to agricultural lands, the majority of the mass fraction is expected to remain adsorbed to soil (99.9%) due to its very hydrophobic nature. Evaporation from soil into air is not expected due to an extremely low vapour pressure. If released to soil, DBDPE is expected to be immobile based on its high estimated log K_{oc}. In addition, low degradation is expected in soil; DBDPE is therefore likely to remain in this compartment, and the loss process in soil will mainly be driven by soil burial or surface runoff. The results of Level III fugacity modelling (Table 8-1) support the expectation that at steady state DBDPE predominantly distributes in soil or sediment, depending on the compartment of release (New EQC 2012).

8.1.1 Long-range Transport Potential

Predicted log K_{oa} (14.45) and log K_{aw} (-4.57) values for DBDPE suggest low potential to reach the Arctic (Wania 2006, Brown and Wania 2008). The substance is identified as highly sorptive, sorbing to particles in atmospheric and aqueous media, and therefore, particle settling is predicted to limit long range transport (Brown and Wania 2008). However, if particle bound transport is more efficient than expected, given low predicted

rates of degradation in air (>4 days in gas phase, longer with air particles), there is the possibility that DBDPE could persist and be transported to the Arctic.

DBDPE was measured in horizons of snow pits dug in 2005, 2006 and 2008 in the Canadian Arctic (Devon Ice Cap, Nunavut); however, concentration patterns did not show clear deposition time trends (Meyer et al. 2012). In the Norwegian Arctic, Hermanson et al. (2010) measured DBDPE inputs of approximately 3.6 pg/cm^2/yr (~1988) to 3.4 pg/cm^2/yr (2005) in the top 34 m of an ice core (representing 1953 to 2005) from the western-most ice sheet of Svalbard; this suggests that either particle phase transport could be more important than previously thought, and/or that the quantity of the flame retardant used in atmospheric source areas to the ice sheet affects observations (Hermanson et al. 2010). However, other monitoring data for remote areas suggests that DBDPE does not currently seem to be found widely in the Arctic (e.g., de Wit et al. 2010, McKinney et al. 2011b).

The OECD POPs Screening Model can be used to help identify chemicals with high persistence and long-range transport potential (Scheringer et al. 2006). The Characteristic Travel Distance (CTD) calculated for DBDPE using the OECD model is 2860 km indicating that DBDPE has a significant potential for transport in air (with 99.99% of mass in air partitioned to particles/aerosols), but this is below the boundary (5097 km, CTD of PCB 28) suggested for global pollutants by Klasmeier et al. (2006). The model also calculates an overall persistence (Pov) of 277 days, and the transfer efficiency (TE), which is the percentage of emission flux to air that is deposited to the surface (water and soil) in a remote region. The TE for DBDPE was calculated to be 12.7%, which is above the boundary of 2.248% (PCB-28) established based on the model's reference substances empirically known to be deposited from air to soil or water. The high TE means that DBDPE might be deposited to Earth's surface in remote regions.

In general, while DBDPE (based on physical chemical properties and some models) might not be expected to be a high concern for long-range transport, based on a high predicted transfer efficiency and some detection of DBDPE in remote areas, the role of particle bound transport allows long-range transport of DBDPE to be possible. As well, it is unknown how susceptible potential DBDPE degradation products (see next section) could be for long-range transport.

8.2 Environmental Persistence

Based on likely DBDPE releases and partitioning characteristics, environmental persistence is most relevant for the soil and sediment compartments where the majority of the substance is expected to be found. However, due to the potential particle transport of DBDPE in air and water, all media are considered in this section. Empirical and modelled data were considered in the weight-of-evidence for DBDPE persistence. Data were also compared to the analogue, decaBDE. Relevant transformation processes for DBDPE include photodegradation, biodegradation and biotransformation, as well as combustion/pyrolysis.

Generally, model predictions are consistent with experimental findings that aerobic and anaerobic biodegradation of DBDPE is limited and that DBDPE is expected to be persistent in water, soil, and sediment. Photodegradation of DBDPE in solvents (e.g., n-hexane, tetrahydro-furan (THF)) may be fast; however, photodegradation could take much longer in other matrices/substrates (e.g., >224 days in HIPS powder; Kajiwara et al. 2008). Modelled predictions for DBDPE in air suggest a half-life > 4 days (gas phase) and or an overall persistence (Pov) of 277 days (OECD POPs model). DBDPE testing under longer-term, environmentally relevant conditions to determine the degradation pathways and transformation products is lacking. Nevertheless, potential DBDPE transformation products were evaluated based on predictions from photodegradation studies and biodegradation modeling, and by considering analogue decaBDE transformation products.

Tables 8-2 and 8-3 present empirical and modelled degradation data for DBDPE. See Appendix D (Table D-1) for detailed description of studies.

8.2.1 Abiotic Degradation

Kajiwara et al. (2008) observed no degradation of DBDPE in spiked high-impact polystyrene (HIPS) powder exposed to sunlight for 224 days (half-life was estimated at >224 days), while the half-life of decaBDE in the same matrix was estimated at 51 days. Differences between DBDPE and decaBDE were attributed to structural differences in ether bond vs. ethane bond.

Nadjia et al. (2014) measured very rapid photolytic degradation of DBDPE in solvent under artificial UV-visible light: 63.18% degradation within 180s. The degradation process is reported as stepwise reductive debromination.

Wang et al. (2012) studied the photolytic degradation of DBDPE under UV light using a range of matrices and solvents, including methanol/water and humic acid/water (to simulate the aquatic environment) and silica gel (to simulate the soil/sediment environment). These latter matrices are the most relevant to environmental conditions. Photolytic degradation occurred in all solvents/matrices (and none in the dark control), with 33.7 to 99.6% of the DBDPE lost. Degradation rates depended on the solvent used (Table 8-2). All matrices showed debromination and formation of nonaBDPEs, with subsequent degradation to octa-, and heptaBDPE congeners, although the percentage of transformation product relative to parent was not reported in the study. The authors recognize the presence of nona-BDPEs in the original solutions may be a result of impurities of the technical products (purity not reported) and/or degradation during sample injection; however, the concentrations of nona-BDPEs increased continuously from 0 to 45 min, and octaBDPEs (from 4 mins on) and heptaBDPEs (from 30 mins on) increased continuously during the experiments. The formation of tetra- to hexaBDPEs with longer exposure times was proposed, but not monitored.

In a preliminary study, Kierkegaard et al. (2009) studied technical DBDPE in n-hexane exposed to a daylight-mimicking fluorescent lamp, and found DBDPE was degraded,

producing two nonabrominated congeners, as well as a number of peaks tentatively identified as octabrominated products. The authors also reported that DBDPE degrades to lower (mainly two nona-) brominated congeners during sample preparation/analysis, although it appeared to be less sensitive to thermal degradation than decaBDE (Kierkegaard et al. 2009).

The predicted half-life for atmospheric degradation of DBDPE due to reaction with the hydroxyl radical is 4.47 days for the fraction of chemical that is in the gas phase (12-hr day, AOPWIN 2010). The results of AEROWIN (2010) predict a high fraction of DBDPE absorption to airborne particles (Phi = 1), and therefore, that the rate of DBDPE photolysis is likely lower than predicted (i.e., half-life longer than predicted 4.47 days). This is consistent with the OECD Pops model which finds 99.9% of DBDPE in air is sorbed to aerosols, and an overall persistence of 277 days for the substance.

Based on abiotic degradation modelled data and empirical data for DBDPE, the substance is expected to be persistent in air (Table 8-2). However, fate modelling indicates only a very small proportion of DBDPE released into the environment is expected to partition into air, and atmospheric concentrations of DBDPE are expected to be low. The high K_{oc} of DBDPE suggests that DBDPE released directly into air will likely adsorb to particulates, with subsequent removal to soil or water by wet and dry deposition. Due to DBDPE's low water solubility, particle adsorption behaviour, as well as light attenuation by humic materials, photolysis in natural waters, soils, and sludge is expected to be limited (Environment Agency 2007).

DBDPE does not contain functional groups expected to undergo hydrolysis.

Table 8-2. Summary of key data for abiotic degradation of DBDPE

Medium	Fate process	Degradation value	Degradation endpoint / units	Methods	Reference
High-Impact Polystyrene (HIPS)	Photolysis	> 224 days	half-life/days	Published study	Kajiwara et al. 2008
tetrahydro-furan	Photolysis	1.89 min	half-life/min	Published study	Nadjia et al. 2014
n-hexane	Photolysis	16.6 min	half-life/min	Published study	Wang et al. 2012
tetrahydro-furan	Photolysis	6 min	half-life/min	Published study	Wang et al. 2012
methanol/ water	Photolysis	>240 min	half-life/min	Published study	Wang et al. 2012

humic acid/ water	Photolysis	30 – 60 min	half-life/min	Published study	Wang et al. 2012
silica gel	Photolysis	75.9 min	half-life/min	Published study	Wang et al. 2012
Air	Atmospheric oxidation	4.47 days[b]	half-life/days	Model	AOPWIN 2010[a]
Air	Ozone reaction	n/a[c]	n/a[c]	Model	AOPWIN 2010[a]
Water	Hydrolysis	n/a[c]	n/a[c]	Model	HYDROWIN 2010[a]

[a] EPIsuite (2010-2012).
[b] AEROWIN (2010) predicts high fraction of DBDPE absorption to airborne particles (Phi = 1), therefore rate of DBDPE photolysis likely lower than predicted (i.e., half-life longer than predicted).
[c] Model does not provide an estimate for this type of structure.

8.2.2 Biodegradation

Laboratory tests have shown DBDPE is not likely to biodegrade under aerobic conditions. A CITI (1991a) study measuring biodegradation by microorganisms found a range of 1–6 % biodegradation (mean of 2%) over 28 days in a ready-biodegradation test for DBDPE.

An inherent biodegradability study by Schaefer and Carpenter (2010) which followed the Concawe Test (OECD Draft Method 302D), found an average cumulative rate of 2.2% biodegradation after 90 days, indicating that DBDPE is not inherently biodegradable under aerobic conditions. The test media were also analyzed for parent and predicted degradation products (using radiolabeled test chambers), and found no transformation products over the 90 day period.

The four ultimate biodegradation submodels (BIOWIN (2010) and Catalogic (2012) predict that biodegradation is very slow or recalcitrant. In addition, a primary biodegradation model, BIOWIN Sub-model 4 (primary survey model), predicts that the substance is recalcitrant.

The existing data for anaerobic degradation of DBDPE suggests that if the substance degrades, it does so very slowly. An anaerobic biodegradation study that compared radiolabeled DBDPE in biotic and abiotic treatments of anaerobic digester sludge (initial dose of DBDPE 31 mg/L in THF), showed no mineralization or transformation over 63 days (Schaefer and Matthews 2011). Measurements of ^{14}C activity at days 0, 30 and 63, as well as a mass-balance of compartments (solid sludge, extractable, and volatized), found only the parent DBDPE. Earlier laboratory studies of analogue decaBDE (Gerecke et al. 2005, 2006) have also shown decaBDE to undergo slow anaerobically mediated reductive debromination, with a half-life of up to 700 days.

These aerobic and anaerobic biodegradation tests, as well as modelling results, indicate that the half-life in water is likely to be long, and that the substance is therefore likely to persist in water (Table 8-3). Using an extrapolation ratio of 1:1:4 for a water: soil: sediment biodegradation half-life (Boethling et al. 1995), DBDPE is expected to be persistent in soil and sediment, and thus is likely to present long-term exposures in these media.

Table 8-3. Summary of key data for biodegradation of DBDPE

Medium	Fate process	Degradation value	Degradation endpoint / units	Methods	Reference
Activated sludge	Bio-degradation	2%	28-day Biodegradation BOD/%	OECD 301C (Modified MITI I test)	CITI 1991a
Mixture of pre-exposed sludge and soil	Enhance aerobic Bio-degradation	0	90-day aerobic Biodegradation IC/TOC/14C-activity	OECD 302D (CONCAWE test)	Schaefer and Carpenter 2010
Anaerobic digester sludge	Biotic/ Abiotic Anaerobic minerali-zation	0 (biotic) 0 (abiotic)	63-day anaerobic mineralization /%	OECD 314C (Anaerobic digester sludge)	Schaefer and Matthews 2011
Water	Primary Bio-degradation (aerobic)	0.7743[a] "recalcitrant"	NA	QSAR Model	BIOWIN 2010[d]
Water	Bio-degradation (aerobic)	-0.4568[a] "recalcitrant"	NA	QSAR Model	BIOWIN 2010[e]
Water	Bio-degradation (aerobic)	-0.6209[b] "biodegrades slowly"	NA	QSAR Model	BIOWIN 2010[f]
Water	Bio-degradation (aerobic)	0.00[b] "biodegrades slowly"	NA	QSAR Model	BIOWIN 2010[g]
Water	Bio-degradation (aerobic)	% BOD = 0.41[c] "biodegrades very slowly"	, ultimate half-life > 10 years	QSAR Model	Catalogic 2012

[a] Output is a numerical score from 0 to 5. NA=not applicable.
[b] Output is a probability score.
[c] Some uncertainty associated with model predictions, as substance is <60% (42%) covered by structural domain of model.
[e] Sub-model 4: Expert Survey (qualitative results).
[f] Sub-model 3: Expert Survey (qualitative results).
[g] Sub-model 5: MITI linear probability.

[h] Sub-model 6: MITI non-linear probability.

DBDPE appears to be moderately well covered by the biodegradation models used to estimate degradation. The number of fragments and molecular size covered by the domain of the BIOWIN Submodels 5 and 6 (aerobic biodegradation, MITI) suggest high coverage for DBDPE. The domain covered by BIOWIN Submodels 3 and 4 (aerobic biodegradation, Expert Survey) includes substances with fewer aromatic bromide fragments and smaller molecular weights than DBDPE; however, the degradation predictions are consistent with other modeled results and empirical data. There is some uncertainty associated with the CATALOGIC (2012) model predictions in that there is only 42% coverage of the DBDPE by the structural domain of the Catalogic model (>60% structural coverage is recommended). However, given that the predicted results agree with other model predictions (BIOWIN submodels), and agree with the existing empirical data, it would suggest that the model is extrapolating correctly beyond interpolation space.

Modeling predictions and experimental data indicate very slow and/or limited biodegradation of DBDPE. Catalogic model (2012) predictions indicate that when biodegradation occurs, while most of the substance stays as parent, there is a low probability of limited debromination under aerobic and anaerobic conditions, leading to nonaBDPEs and octaBDPEs, hydroxylated nonaBDPEs, and a form of brominated phenyl acid (see the Supporting Information (Environment Canada 2015) for empirical studies of DBDPE transformation products).

Wei et al.(2012) measured two unknown brominated compounds in pond sediments near a DBDPE manufacturing plant in Arkansas, USA (a former WWTP sludge pond), which were further identified as two nonabromodiphenyl ethanes (nonaBDPEs) when matched to photolytic debromination peaks in hexane. The nonaBDPE concentration increased towards the surface, with a ratio of 1.3 nona/DBDPE compared to ~0.7 nona/DBDPE in standards or other pond sediments. The increased nonaBDPE presence was attributed to DBDPE debromination in the upper sediment of one of the ponds. This pond also demonstrated PBDE debromination, which was attributed to a complex process likely involving physical and biological influences (see the Supporting Information (Environment Canada 2015)).

Recently, slow decaBDE debromination has been measured in sediment microcosms in a remote lake on the Canadian Shield under natural conditions over 1 month (Orihel et al. 2016). Although only a small loss of ^{13}C-decaBDE was measured, octa- and nonaBDEs were formed from ^{13}C-decaBDE in littoral and profundal sediments, and trace amounts of di- to heptaBDEs were detected in some samples. Degree of debromination was determined not to be influenced by light or dark conditions, although more nona- and octaBDEs were formed under oxic conditions than under anoxic conditions**.**

8.2.3 Metabolic Biotransformation

Empirical metabolic biotransformation studies that discuss potential for DBDPE degradation pathways and transformation products are described in the Potential for Bioaccumulation section.

8.2.4 Combustion and Pyrolysis

Thermal degradation for DBDPE in fires is identified as a potential source of degradation products. DBDPE may be susceptible to the formation of bromotoluenes, via cleavage of the ethane bridge (Eljarrat and Barceló 2011). Jakab et al. (2003) reports that under pyrolysis conditions, DBDPE in high-impact polystyrene (HIPS) samples produces a relatively high yield of brominated toluenes, including the major product, pentabromotoluene (PBT) along with other lower brominated toluenes. This pathway differs from that of decaBDE, which can decompose to produce brominated dibenzofurans under pyrolysis conditions (Jakab et al. 2003); the formation of brominated dibenzofurans is not generally observed for DBDPE.

8.2.5 Persistence of Transformation Products

A summary of potential DBDPE transformation is found in the Supporting Information document (Environment Canada 2015). Laboratory photodegradation studies (largely conducted with solvents) described in the section above report DBDPE debromination (Nadjia et al. 2014) to nona-, octa- and heptaBDPEs (Wang et al. 2012). Sediment studies have measured nonaBDPEs in the environment (Wei et al. 2012, He et al. 2012). Catalogic model (2012) predictions indicate that biodegradation may lead to limited debromination under aerobic and anaerobic conditions, leading to nonaBDPEs and octaBDPEs, hydroxylated nonaBDPEs, and a form of brominated phenyl acid. These predicted transformation products are expected to represent a minor fraction relative to parent DBDPE; however, they are similar to predicted/measured fractions of analogue decaBDE debromination products. Within the CPOPs model, the B_{max} model (CPOPs 2012) uses rat metabolism data to predict DBDPE transformation to hydroxylated DBDPE and hydroxylated nonaBDPE. Considering DBDPE's close analogue decaBDE, a substance that debrominates to lower brominated PBDEs (i.e., nonaBDEs through tetraBDEs) under specific conditions (e.g., photodegradation, aerobic biodegradation and metabolism) (Environment Canada 2010), it is reasonable to expect that DBDPE may also debrominate to lower BDPE transformation products (e.g., nona, octa, hepta-, hexa, and pentaBDPEs) following the pathway of debromination established for decaBDE (Appendix C, Table C-1).

Due to the lack of experimental data on the transformation products, (Q)SAR modelling was conducted to assess their persistence, biodegradation and toxicity characteristics. See Appendix C for a description of transformation product (Q)SAR modeling and physical chemical properties (Table C-2).

To evaluate persistence of potential transformation products, a (Q)SAR-based degradation modelling approach was used. Results of BIOWIN (2010) sub-models

suggest that all potential transformation products (nona- through pentaBDPEs, hydroxylated DBDPE and nonaBDPE, and brominated phenyl acids) demonstrate biodegradation is very slow or recalcitrant in water (see Appendix C, Table C-3).

The predicted transformation products do not contain functional groups expected to undergo hydrolysis, with the exception of brominated phenyl acids, which are recognized to have acyl halides. HYDROWIN (2010) predicts acyl halides react readily with water to yield the parent acid and hydrogen halide, with a half-life less than 10 minutes (or faster) (Table C-3).

The predicted half-life for atmospheric degradation of nona- through pentaBDPEs, hydroxylated DBDPE and nonaBDPE due to their reaction with the hydroxyl radical ranges from ~2 days to > 4 days (12-hr day, AOPWIN 2010) (Table C-3). AOPWIN (2010) also identifies the brominated phenyl acids to undergo ozone reaction, and predicts a half-life of 79.3 days.

Therefore, considering all model results, there is evidence that the predicted degradation of the debrominated BDPEs (penta through nonaBDPEs) and hydroxylated nonaBDPE, is slow and that the substances are persistent in water and air. Using an extrapolation ratio of 1:1:4 for a water: soil: sediment biodegradation half-life (Boethling et al. 1995), it is expected that nona- through pentaBDPEs, hydroxylated DBDPE and nonaBDPE, and brominated phenyl acids are also very persistent in soil and sediment.

8.3 Potential for Bioaccumulation

Properties of the substance (i.e., log Kow, log Koa, molecular size and cross-sectional diameters) as well as empirical data (bioconcentration factor (BCF), biomagnification factor (BMF), trophic magnification factor (TMF) and bioaccumulation factor (BAF)) were considered for evaluation of DBDPE bioaccumulation potential.

Kelly et al. (2004) demonstrated that the absorption of ingested chemical in fish (and other wildlife) decreases with increasing log K_{ow} starting at ~ 7 – 7.5 because the diffusion of hydrophobic substances across an unstirred water layer to the luminal membrane (i.e., gastrointeslinal tract) of an organism is rate limiting for substances like DBDPE which have very high log K_{ow} and low water solubility. Although Arnot and Gobas (2003a, 2004, 2006) do state that the log K_{ow} domain of their model ranges from 1-9, there is considered to be insufficient empirical field evidence (i.e., BAFs) to support model estimates beyond log K_{ow}~ 8.2. Therefore, the log K_{ow} of 9.89 for DBDPE is considered out of the model domain for the mass-balance three trophic level BCFBAF model (Arnot and Gobas 2003a) and the (Q)SAR based Dimitrov et al. (2005) model. Importantly, lack of empirical BCF and BAF data for chemicals with log Kow greater than 8.3 does not allow for benchmarking of predicted results. Consequently, DBDPE was not modelled in this assessment. However, empirical analogue decaBDE was considered in the discussion, due to similar structure and physical-chemical properties.

There has been some debate in the literature as to the bioaccumulation potential of DBDPE (Law et al. 2006, Wang et al. 2010, and Hardy et al. 2012). While DBDPE appears to be bioavailable to some organisms, the available evidence is equivocal with respect to higher bioaccumulation. Based on its physical and chemical properties (e.g., moderately large maximum diameter, very low water solubility, high log K_{ow}), DBDPE is expected to have a low bioconcentration potential. Monitoring studies from many parts of the world have reported measurable DBDPE in aquatic and terrestrial organisms; however, there are also very high proportions on non-detects in biota studies (frequently >50%) (see Supporting Information document (Environment Canada 2015)). Data for field-based BMFs and BAFs are not consistent and have uncertainties to consider. Studies of DBDPE in rats and wildlife suggest that DBDPE may be bioavailable for uptake, and metabolism may occur in some species.

Therefore, while it appears that DBDPE may accumulate in the tissues of some organisms to some extent, at present there is not adequate evidence of potential for high bioaccumulation. Currently there are more lines of evidence to suggest that bioaccumulation potential of DBDPE is limited by low bioavailability and dietary assimilation efficiency (Kelly et al. 2004), steric uptake restriction and some metabolism.

With respect to possible DBDPE transformation products, this assessment provides preliminary modelling evidence to suggest that that hexaBDPE though nonaBDPEs may have potential for high bioaccumulation, and that the bioaccumulation potential will increase from nona to hexaBDPEs.

The evaluation of DBDPE bioaccumulation potential presented below considers empirical bioconcentration factor, biomagnification, trophic magnification, bioaccumulation factor, and metabolism data.

8.3.1 Bioconcentration Factor (BCF)

Experimental BCF data for DBDPE exist from one study (BCF<2.5 to 34 L/kg) (CITI 1991b). This study examined Japanese carp exposed for 8 weeks to DBDPE concentrations of 0.5 mg/L and 0.05 mg/L. While the study indicated that it met 1981 Good Laboratory Practice (GLP) standards, the exposure concentrations in the study greatly exceed DBDPE water solubility and dispersants were used and it is not certain that steady state conditions were reached in the test system.

Recent investigations relating fish BCF data and molecular size parameters (Dimitrov et al. 2005, Sakuratani et al. 2008) suggest that the probability of a molecule crossing gill cell membranes as a result of passive diffusion declines significantly with increasing maximum diameter (D_{max}). Based on the BCF_{max} Model with Mitigating Factors (Dimitrov et al. 2005), the maximum diameter of DBDPE ranges from 1.4 to 1.7 nm. This suggests that DBDPE is likely to experience a reduced rate of uptake from steric effects at the gill surface allowing elimination processes to mitigate accumulation.

At a log K_{ow} of 9.89 (See Table 3-1), the predicted bioavailable fraction of DBDPE in the water column (excluding loss from volatilization) according to mass-balance fish models is 0.15% (BCFBAF v. 1.5), which indicates the majority of the chemical in the water column is not taken up at the gill surface.

8.3.2 Bioaccumulation Factor (BAF)

Bioaccumulation factors are measured under field conditions as the ratio of the whole body of chemical concentrations taken up from all exposures to that of the ambient water concentrations. Measures of BAF are a preferred metric for assessing the bioaccumulation potential of substances because it incorporates all chemical exposures including the diet, which predominates for substances with log Kow > ~4.0 (Arnot and Gobas 2003a).

A study conducted in the Dongjiang River (tributary of Pearl River) of China, where previous studies reported levels of PBDEs and DBDPE in sediments among the highest in the world, (He et al. 2012) compared DBDPE in water, sediment and fish muscle samples for Mud carp (*Cirrhinus molitorella*), Tilapia (*Tilapia nilotica*), and Plecostomus (*Hypostomus plecostomus*) (collected 2009 and 2010). The measured DBDPE concentrations were approximately one order of magnitude higher than the decaBDE concentrations in fish but not in sediment in the same study. Concentrations of DBDPE in water (dissolved) and fish muscle were used to estimate logBAF values, resulting in DBDPE logBAFs of 6.1 to 7.0, suggesting high bioaccumulation (Table 8-5). There are, however, limitations/uncertainties associated with this study. For instance, total DBDPE concentration (including particulates which could be ingested) may better represent a water exposure since dietary exposure is not discussed but could be very important. Also, the study had small water sample sizes, and water/fish samples were collected at varying times, which suggests that “steady state” was not achieved (Arnot et al. 2006).

8.3.3 Biomagnification Factor (BMF)

BMF values describe the process in which the concentration of a chemical in an organism reaches a level that is higher than that in the organism’s diet, due to dietary absorption (Gobas and Morrison 2000). A BMF exceeding 1 indicates that biomagnification is potentially occurring, and may be considered an indicator of the potential for uptake and accumulation in biota. Table 8-4 presents empirical BMF data for DBDPE.

In a recent study in south China, DBDPE and decaBDE in common kingfishers (*Alcedo atthis*), a piscivorous bird, and their prey fish from an e-waste-recycling site were examined (Mo et al. 2012). BMFs were calculated as the ratios of lipid normalized concentrations in the muscle of kingfisher to mean lipid-based concentrations in prey fish. For DBDPE, BMFs ranged from 0.10 to 0.77, which suggests biomagnification was not occurring in the investigated feeding relationship. This contrasts with the study’s BMF values for decaBDE, which exceeded 1. In a similar study comparing DBDPE in

kingfishers between the Dinghushan Biosphere Reserve and a reference site in South China, Mo et al. (2013) also reported BMF <1 for DBDPE from prey fish to kingfisher.

Another study in South China (She et al. 2013) examined a small herbivorous food chain (paddy soils to rice plant to apple snails) and found that although DBDPE concentrations were measureable in the paddy soils (14.7 ng/g dw) and rice plants (3.59 ng/g dw), it was non-detectable in the snail. A plant to soil concentration ratio of 0.20 was determined for DBDPE.

Law et al. (2006) examined potential DBDPE bio and trophic magnification in Lake Winnipeg, Canada. Samples of fish muscle tissue (six species), plankton, mussels, sediment and water were collected between 2000 and 2004. DBDPE was not detected in water, sediment, zooplankton, mussels or whitefish (*Coregonus clupeaformis)*, but was detected in walleye (*Stizostedion vitreum*) (1.01 ng/g lw), emerald shiner (*Notropis atherinoides*) (0.30 ng/g lw), goldeye (*Hiodon alosoides*) (0.62 ng/g lw), white sucker (*Catostomus commersoni*) (0.08 ng/g lw), and burbot (*Lota lota*) (0.66 ng/g lw). Biomagnification factors (lipid weight corrected) for fish species with detectable concentrations of DBDPE in prey ranged from 0.2 to 9.2, with four of the five BMFs calculated as >1. However, this study also suffers from a number of limitations including that concentrations of DBDPE were low and near the detection limit (0.1 µg/g), sample sizes were small and samples were collected at varying times, meaning that steady state cannot be assumed. Also, lipid concentrations were very low for some species; therefore lipid normalized data may have resulted in an over-estimation of the BMF (see Environment Canada 2010).

Table 8-4. Empirical biomagnification factors (BMF) for DBDPE

Test organism	Steady-state, kinetic and lipid normalized values (/kg)	Reference
Common kingfishers (*Alcedo atthis*)/prey fish	<1	Mo et al. 2013
Common kingfishers (*Alcedo atthis*)/prey fish	0.10 to 0.77 (BMF<1)	Mo et al. 2012
Walleye/emerald shiner	3.0	Law et al. 2006
Walleye/white sucker	9.2	Law et al. 2006
Walleye/goldeye	1.6	Law et al. 2006
Burbot/emerald shiner	2.0	Law et al. 2006
Whitefish/ emerald shiner	0.2	Law et al. 2006

The available biomagnification data are limited, and most studies do not provide kinetic data (e.g., dietary assimilation efficiency, elimination rates). Reported dietary assimilation efficiencies in fish for analogue decaBDE are low, and range from 0.02% to 0.5% (Kierkegaard et al. 1999, Stapleton et al. 2004, Wan et al. 2013). In a study establishing benchmarks of absorbable and non-absorbable compounds, DBDPE was detected in feces but not in fish, confirming that it was a suitable choice as the nonabsorbable benchmark (Xiao et al 2013).

The combination of high log K_{ow} and log K_{oa} (9.89 and 14.45, respectively) suggests that DBDPE may have the potential to biomagnify in terrestrial food webs as suggested by Gobas et al. (2003) and Kelly et al. (2007). However, these partition coefficients do not account for physiological parameters such as metabolism. Kelly et al. (2004) discuss that diminished BMFs due to metabolic transformation are more common in birds and mammals compared to fish, because those organisms generally have a greater capacity to metabolize organic contaminants.

The available studies do not unequivocally suggest that the BMF of DBDPE exceeds 1. However, DBDPE dietary exposures still may contribute to individual body burdens, as well as DBDPE trophic transfer and accumulation in certain species and/or food webs.

8.3.4 Trophic Magnification Factor (TMF)

The TMF is a measure of the averaged biomagnification potential of a substance within a studied foodweb under field conditions, and is estimated by correlating the normalized substance concentrations in biota at different trophic levels.

Only one North American study was found that presented results for TMF of DBDPE in a pelagic foodweb (Law et al. 2006, see BMF section for study details). A TMF was determined for Lake Winnipeg as 2.7, suggesting trophic magnification was occurring. However, as described above, the uncertainties with this study are considered important and may result in an over-estimation of biomagnification and trophic magnification.

TMF estimates were reported by a Norwegian monitoring report (KLIF 2013) for both mainland and arctic areas. As TMFs were based on representative samples from food webs rather than a true “food chain” (i.e., not a feeding relationship), the values are presented in the reports as estimates. Furthermore, the authors advise that study uncertainties should be considered: for example, tissue samples for organisms were collected from liver, plasma, and egg samples (rather than traditional muscle or fat), and as this influences turnover rate (i.e., the shorter turnover rate of these tissues reflects a shorter exposure period), biomagnification potential in the environment may not be accurately reflected. Despite these caveats, the TMF estimate (log transformed lipid weight) was 3.9 (r^2=0.49) for DBDPE in the Arctic marine environment, from polar cod up to polar bear, and 14.5 (r^2=0.23) for the mainland marine samples (cod to harbour seal), suggesting high trophic magnification.

8.3.5 Other Bioaccumulation and Metabolic Transformation-related Studies

Two single dose pharmacokinetic studies in rats demonstrated negligible to limited uptake after oral administration. A pharmacokinetic study (Black 2012) observed DBDPE has limited uptake by rats after a single dose in corn oil (100 mg/kg unlabeled plus ^{14}C labeled DBDPE), and was not accumulated in tissues. Nearly all the administered radioactivity was recovered in feces (89.7%) and cage rinse (0.25%) after 168 h. In total tissues sampled contained less than 0.02% of dose, suggesting that

DBDPE was eliminated in feces rather than absorbed from the GI tract (see Human Health Assessment).

Wang et al. (2010) studied potential for DBDPE bioavailability to mammals (from 100 mg/kg bw/day of DBDPE (and decaBDE) in corn oil for 90 days) and investigated possible metabolites in DBDPE exposed rats compared to control rats. At least seven unknown bromine containing compounds were observed in the livers of DBDPE-exposed rats. However, none of these compounds matched the analytical retention time of DBDPE debromination products (hepta through nona BDPEs) identified during a concurrent photodegradation experiment (see the Supporting Information document (Environment Canada 2015)).The only two metabolic products with peaks strong enough to provide structural information were found mainly in liver and kidney, and were hypothesized to be a methyl sulfone ($MeSO_{2)}$-nona-BDPE and an ethyl sulfone ($EtSO_{2)}$-nona-BDPE. However, Banasik et al. (2010) argue the mass spectral data do not conform to the sulfone structures. Furthermore, the percentage of DBDPE uptake vs. transformation vs. elimination was not quantified in this study. See the Human Health section for further details on this study.

The B_{max} model of CPOPs (2012) uses rat metabolism data to predict DBDPE transformation products. Two transformation products were predicted: hydroxylated DBDPE and hydroxylated nonaBDPE.

McKinney et al. (2011a) compared oxidative and reductive debromination of DBDPE and other brominated flame retardants (including decaBDE) using an *in vitro* test based on liver microsomes from arctic marine feeding mammals (polar bear, beluga whale, ringed seal and laboratory rat). Biotransformation assays were performed by incubating hepatic microsomes with either individual OFRs or a mixture of OFRs, and compared to controls (i.e., no enzyme activity). Generally, the microsomes of all specimens substantially depleted DBDPE (44-74% in the single OFR assays, and 27-59% in the OFR mixture assays, more than any of the PBDEs). The authors concluded simple debromination was not the primary pathway (no clear evidence for the formation of any debrominated DBDPE metabolites). Two likely nonaBDPEs were detected in assays and controls (which had no enzyme activity), possibly resulting from contamination and some metabolic debromination. Two phenolic metabolites were detected in polar bear liver microsomes, but only accounted for $<1/5^{th}$ of DBDPE depleted. In general, metabolites were not detected that could account for the degree of DBDPE (or decaBDE) depletion. The authors suggest this could be due to low/non-detectable concentrations of metabolites, the possibility of non-extractable metabolites, etc. An industry review of this study suggests that adsorption, rather than metabolism, is responsible for the DBDPE depletion (Hardy 2012).

Although a single exposure, in a study of dietary efficiency of chemicals by fish, DBDPE was selected as a “nonabsorbable benchmark”, where no absorption into the gastrointestinal tract was expected (Xiao et al. 2013). Study fish were fed a single meal of contaminated feed, and then analyzed for chemical distribution after 5 days. For all tests, DBDPE was detected in feces but not fish tissue.

A recent study of close analogue decaBDE (high purity) dietary exposure to kestrals (21 days) found that there was substance uptake and accumulation as well as degradation (debromination) to lower brominated PBDEs *in vivo* (Letcher et al. 2014). At the end of the 25-day exposure period, decaBDE in liver and fat of exposed birds was significantly greater than in control birds. NonaBDEs and octaBDEs were found in kestrel plasma, while heptaBDEs were identified in liver and fat. *In vivo* elimination was also observed: by day 25 of the elimination period, decaBDE in plasma was depleted by 82%.

These fish, rodent and wildlife studies provide variable evidence of the degree of bioavailability and metabolism of DBDPE. Shorter-term and single-dose studies show DBDPE is excreted with only minimal uptake in fish and rodents. Other studies indicate DBDPE bioavailability (uptake) and potential metabolic transformation. Bioavailability and metabolic transformation potential for DBDPE may be species-specific, and vary with length of dietary exposure.

8.3.6 Predicted Bioaccumulation Potential of Transformation Products

As described in the persistence section, there is evidence to suggest DBDPE may debrominate to transformation products. As there are no experimental data characterizing the bioaccumulation potential for potential DBDPE transformation products, these are subjected to (Q)SAR modelling. For predictions with the (Q)SAR model, the log K_{ow} for predicted transformation products was estimated using the Experimental Value Adjustment (EVA) method based on DBDPE (see Appendix C, Table C-2). The resulting modelled log K_{ow} values of 4.83 to 9.0 suggest that some of these substances have a moderate to high potential to bioaccumulate in aquatic biota.

The detailed results of the BCF and BAF modelling are given in Appendix C (Table C-4). Results vary widely; BCFBAF (Episuite 2000-2012) predicts most transformation products will have low to moderate bioconcentration, with estimated BCFs based on total water concentrations ranging from 3.2 to 3803 ((linear regression method)). BCF and BAF estimates for a middle trophic level fish representative of Canadian waters, corrected for potential biotransformation (predicted k_M = 0.016 to 0.271 for 100 g fish at 15℃), were 38.9 to 3510 (low to moderate) and 738 to 176 400 (low to high), respectively (Episuite 2010-2012). This modelling suggests that the transformation products with greatest potential for bioaccumulation are the hexaBDPE though octaBDPEs.

Modelled metabolic biotransformation rates (k_M) for transformation products generally fall within the range of those predicted for parent DBDPE (BCFBAF (2010) k_M = 0.006 /day for 100 g fish at 15 °C middle trophic level fish representative of Canadian waters, CPOPS k_M= ~0.0209 /day), with the exception of the brominate phenyl acids. The k_M values for octa and nonaBDPEs are also comparable to those measured and/or predicted for their octa- and nonaBDE counterparts (0.03 /day and 0.02, respectively, for 184 g fish, 15℃) (Environment Canada 2010). The lower brominated BDPEs (penta-hepta) predicted k_M values are higher than those reported for lower brominated PBDEs (Environment Canada 2010).

These results suggest high bioaccumulation potential for some DBDPE lower brominated transformation products, which is supported by research on the transformation products of the analogue decaBDE. However, it must be considered that there is uncertainty with these estimates (see Uncertainties section). In particular, at log K_{ow} of >8.2, the bioaccumulation estimates for hydroxylated nonaBDPE and hydroxylated DBDPE transformation products are outside of the dataset of the BCFBAF model, and results based on this extrapolation are less certain. It is noted that the BCF model estimates bioconcentration from water exposure, which may not be the dominant exposure pathway for DBDPE transformation products; therefore BAF estimates may better represent bioaccumulation potential for these substances. In addition, consideration of both the log K_{ow} values (4.83 to 9.0) and log K_{oa} values (8.61 to 17.27) suggests that at least some of these substances will have the potential to biomagnify in terrestrial food webs as a result of dietary exposure and low loss via exhalation. Due to the lack of measured data for reliable input data, terrestrial biomagnification modelling was not undertaken in this assessment.

8.4 Summary of Environmental Fate

DBDPE is expected to be released to surface water from industrial sources primarily through wastewater. There may be a potential for migration of DBDPE from plastics to the atmosphere (dust and air) given that the substance is added to the polymer matrix and thus could leach to some extent. A strong tendency to sorb to the solid phase in various media (including suspended air particles) indicates that DBDPE will reside in biosolids, sediments, suspended air particles and be transferred to soil from dry deposition and application of biosolids to agricultural lands. Exposure to organisms directly via water is expected to be minimal. DBDPE's high intrinsic persistence suggests that long-term exposures can be expected in sediment and soil with a potential for significant build-up in near-field environments from continuous emissions. Removal process from the environment would include sediment and soil burial. DBDPE might be expected to undergo long-range transport in air and deposition to remote environments due to fine particle transport as has been evidenced with other hydrophobic flame retardants with high air particle sorption. Even with long-term exposure to DBDPE in terrestrial and aquatic environments, based on the limited available literature, this substance is not expected to be highly bioavailable and thus tissue residue levels in organisms and migration in foodwebs is not expected to be significant. However, future bioaccumulation studies should be monitored.

Almost no data are available for potential DBDPE transformation products. Predictions based on (Q)SAR modelling suggest that expected transformation products of DBDPE are likely to be found and behave in the environment in a similar fashion to DBDPE itself (residing in solid phase in various media, long-term exposures expected in sediment and soil, possible long range transport in air due to fine particle transport); however, some also have a high potential to bioaccumulate in organisms, similar to analogous PBDEs.

9. Potential to Cause Ecological Harm

9.1 Ecological Effects Assessment

Empirical data for DBDPE, as well as relevant comparative data for the structural analogue, decaBDE, were considered in the weight-of-evidence for assessing the ecological effects of DBDPE. DBDPE (Q)SAR aquatic toxicity modelling for DBDPE was considered unreliable due to exceedance of log K_{ow} cut-offs, or the substance was poorly covered by the toxicity training sets (See the Supporting Information (Environment Canada 2015)).

Results from most empirical aquatic toxicity studies have high uncertainty, questionable reliability and/or results that are difficult to translate to the environment (e.g., use of solvents, WAF studies). Since the vast majority of DBDPE settles in soil or sediment compartments (e.g., bioavailable fraction of DBDPE in water is 0.15% (BCFBAF v.1.5), water-based exposure is not considered an environmentally important pathway for exposure. For this reason, combined with the uncertainty of available aquatic toxicity data, no water (pelagic) critical toxicity value (CTV) or predicted no effects concentration (PNEC) are determined for DBDPE in this assessment. However, recent analogue decaBDE studies in aquatic environments suggest that molecular-level effects may occur in some species.

Based on the results of sediment and soil chronic toxicity testing, DBDPE appears to have the potential to cause effects at high exposure concentrations to reproduction of earthworms as well as plant survival and growth, while no effects were observed for sediment organisms. No overtly toxic effects were found in wildlife, although DBDPE may affect enzyme activity in some test species.

9.1.1 Empirical Studies in Water

Due to slow uptake kinetics of highly hydrophobic compounds, acute toxicity tests are expected to not allow enough time for a steady state to be established (Tolls et al. 2009; Mayer and Reichenberg 2006). For DBDPE, results from the available empirical aquatic toxicity studies have high uncertainty and/or have unclear results: for example, exceeding the water solubility or employing test methods difficult to translate to effects levels in the environment (e.g., WAF approach, solubilizer etc).

In one study, Nakari and Huhtala (2010) found DBDPE effects on aquatic organisms are possible at low concentrations (e.g., DBDPE= 0.0063 to 0.025 mg/L), although the dose greatly exceeded DBDPE's water solubility limit and solubilizers were used. In this study, DBDPE was shown to suppress hatching success of zebrafish eggs, while in rainbow trout and brown trout hepatocytes, DBDPE increased vitellogenin synthesis (a marker of estrogenic effects), inhibited CYP1-dependent monooxygenase activity, and increased the activity of UGT (Nakari and Huhtala 2010). Although there are some questions about the reliability of this study (e.g., concentration of solvent used, exceedance of water solubility), the results suggest aquatic effects may be possible in

environments where solubilizer-like substances co-occur with DBDPE in water (e.g., cosolvation in waste effluents; dissolved organic carbon interactions, surfactant interactions); or possibly where very long-term exposure allows steady state conditions to establish.

Analogue decaBDE also has limited water solubility and early hazard assessments suggested that significant acute or chronic toxic effects were not likely to occur in aquatic organisms at concentrations under water solubility (e.g., EC 2006, Environment Agency 2007). However, recent aquatic toxicity studies of the substance have reported effects on aquatic organisms, fish and amphibians, such as effects on growth, reproduction, development, thyroid hormone disruption, and neurobehavioural alterations after exposure to low level decaBDE (Kuo et al. 2010, He et al. 2011, Noyes et al. 2011, Qin et al. 2012, ECHA 2012). The lowest aquatic NOEC for exposure via water is reported below 0.001 mg/L for delayed metamorphosis in amphibians (Qin 2010, see also ECHA 2012a). DecaBDE exposure to fish via diet (feeding studies) with fathead minnows conducted at environmentally relevant concentrations showed that the substance may interfere with the thyroid hormone system in juvenile fish (Noyes 2011, Noyes 2013). Some questions regarding the above studies include methodological problems in quantifying decaBDE effects in the range of water solubility, non-GLP studies, etc. As well, it cannot be excluded that PBDE congeners other than decaBDE may have contributed to the effects reported (i.e., debromination products observed in these studies include nona-, octa-, hepta-, hexa- and pentaBDEs (ECHA 2012). Nonetheless, these studies raise concerns for potentially serious toxicological effects for decaBDE, and by analogy, DBDPE.

Within the OECD QSAR Toolbox (2012) profile, DBDPE is classified as base surface narcotic/neutral organic for aquatic toxicity by OASIS (v 1.2) and ECOSAR; however, aquatic classification by Verhaar (Modified) reports DBDPE as Class 4 (compounds and groups of compounds acting by a specific mechanism). As well, protein binding alerts are also identified (OASIS v.1.2, OECD).

Mayer and Reichenberg 2006 have shown evidence for a melting point “cut-off” of about 210 °C above which a chemical with only a baseline narcosis mode of action cannot exert acute lethal toxicity through aqueous exposures, i.e., LC50s. Given that observed sub-lethal effects require exposure levels well above chemical solubility limits, the weight of evidence suggests that DBDPE is probably a baseline toxicant and that aqueous-based exposures alone are not capable of exerting a lethal effect to organisms in aquatic environments. While exposure-based modelling using critical body residues (CBR) may provide insights into the potential for adverse effects (sub-lethal) as a result of combined environmental and dietary exposures, due to uncertainty in BAF estimates, a CBR analysis was not conducted.

See the Supporting Information (Environment Canada 2015) for a review of existing aquatic toxicity studies.

9.1.2 Empirical Studies in Sediment

Sediment organism toxicity tests have been performed with chironomids (*Chironomus riparius*) and oligochaetes (*Lumbriculus variegates*) (Krueger et al. 2003a, b; Hardy et al. 2012) (Table 9-1). Chironomids (midge) were exposed to DBDPE in sediment with overlying water over 28 days under static conditions. For oligochaete tests, 10 oligochaetes per test concentration were exposed to DBDPE for 28 days under flow-through conditions. In both studies, potential effects were noted, but endpoints did not show a significant effect. Therefore, EC_{50} values and NOECs for all measured endpoints were reported to be above the highest concentration level of >5000 mg/kg for both the chironomid and oligochaete studies. As the test sediment contained 1.8% organic carbon, the maximum 'solubility' (see Supporting Information (Environment 2015)) of DBDPE in sediment was 52.9 mg/kg dw. The sediment solubility limit, therefore, may have been exceeded under the conditions of the study, although no adverse effects were observed in the test organisms.

Table 9-1. Key sediment toxicity studies considered in choosing a DBDPE critical toxicity value for sediment

Test Organism	Test Type	Endpoint	Value (mg/kg dry weight (dw))	Reference
Midge (*Chironomus riparius*)	Prolonged sediment toxicity: survival, emergence and development	28d EC_{50} LOEC NOEC	>5000 >5000 5000	Krueger et al. 2003a, Hardy et al. 2012
Oligochaete (*Lumbriculus variegates*)	Prolonged sediment toxicity: survivorship and growth	28d EC_{50} LOEC NOEC	>5000 >5000 >5000	Krueger et al. 2003b, Hardy et al. 2012

EC: effective concentration
LOEC: lowest-observed effect concentration
NOEC: no-observed effect concentration

As with aquatic toxicity, a recent sediment toxicity study with analogue decaBDE reported effects on fish larvae in sediment (behaviour and neurological pathway expression) at lower sediment concentrations than previously determined (12.5 mg/kg) (Garcia Reyero 2014), suggesting toxicological effects from low levels of DBDPE are a possibility if a similar mode of action is determined.

A CTV of 5000 mg/kg (>5000 mg/kg) is selected for DBDPE in sediment, representing the only DBDPE toxicity endpoint available, although this value is unbounded with no effects observed at this concentration. Due to uncertainty in inter/intra species variation for a chronic test, an assessment factor of 10 is applied. The resulting PNEC is 500 mg/kg dw. When this value is adjusted from test organic carbon content (1.8%) to standard sediment organic carbon content (4%) (Webster et al. 2004), the PNEC for sediment organisms is 1111 mg/kg dw.

9.1.3 Empirical Studies in Soil

Terrestrial soil toxicity tests were undertaken with wastewater and soil bacteria, earthworms, and plants (Hardy et al. 2011) (Table 9-2). In the earthworm (*Eisenia fetida*), survival (28 day) and reproduction (56 day) were not impacted (NOEC and EC_{50} >3720 mg/kg), but reproduction by day 56 was affected at the highest test concentration (reproduction reduced 60%). DBDPE concentrations showed a substantial decrease in the soil in all test groups, ranging from a 17-40% decrease, exceeding the 20% typically accepted as a stable testing environment. However, given that effects are still noted, it is likely that effects would have remained the same or be more pronounced with stable concentrations.

The effects of DBDPE on terrestrial plant seedling emergence and growth were evaluated by Hardy et al. (2011) in a 21-day study. Corn (*Zea mays*), onion (*Allium cepa*) and ryegrass (*Lolium perenne*) represented monocotyledons, while cucumber (*Cucumis sativa*), soybean (*Glycine max*) and tomato (*Lycopersicon esculentum*) represented the dicotyledons. No adverse effects on any endpoint were reported for corn, ryegrass or soybean, resulting in EC_{25} values greater than 6250 mg/kg. Cucumber's group mean survival was reduced by 18% at the highest test concentration (LOEC =6250, NOEC=3125 mg/kg). Reductions in onion plant mean height of 22% and 24% and weight reductions of 32% and 30% respectively, were observed at the two highest concentrations (LOEC=3125, NOEC=1563 mg/kg). Effects on height and weight of tomato at the highest concentration of DBDPE resulted in reductions of 37% and 40% compared to the controls (LOEC=6250, NOEC=3125 mg/kg). An EC_{25} for onion was reported as 2440 mg/kg.

As the test soil for this latter study contained 2.7% OC, the maximum 'solubility' of DBDPE was 79.4 mg/kg dw (Supporting Information (Environment Canada 2015)). Therefore, the soil solubility limit may have been exceeded under the conditions of the study. This suggests that free DBDPE may have been present in the test system and may have contributed to the observed effects through factors such as the physical clogging of respiratory surfaces.

Table 9-2. Soil toxicity studies considered in choosing a DBDPE critical toxicity value for soil

Test Organism	Test Type	Endpoint	Value (mg/kg dw)	Reference
Earthworms (*Eisenia fetida*)	28-day Survival	LC_{50}	>3720	Aufderheide 2003
Earthworms (*Eisenia fetida*)	56-day Reproduction	EC_{10} EC_{50} LOEC NOEC	1860 3180 3720 (reproduction reduced 60%) 1910	Aufderheide 2003
Plants: Monocoty-ledons	21-day Survival / Reproduction	LOEC NOEC EC_{25}	3091 (3125 nominal) 1722 (1563 nominal) 2440	Porch and Krueger 2005

Onion (*Allium cepa*)			(22% and 24% height reduction at 3125, 32% and 30% weight reduction at 6250 respectively)	
Plants: Dicotyledons Tomato (*Lycopersicon esculentum*)	21-day Survival / Reproduction	LOEC NOEC EC_{25}	6076 (6250 nominal) 2677 (3125 nominal) 4990 (37% height reduction and 40% weight reduction at 6250)	Porch and Krueger 2005

LC: lethal concentration
EC: effective concentration
LOEC: lowest-observed effect concentration
NOEC: no-observed effect concentration

Based on endpoints from a range of soil toxicity studies (soil bacteria, earthworms, and six plant species), the lowest concentration at which a clear effect was determined is the EC_{10} value for earthworm reproduction of 1860 mg/kg DBDPE in soil and the EC_{25} for decreased onion weight of 2440 mg/kg (onion NOEC = 1563 mg/kg, but low EC values are preferred over NOEC values). The EC_{25} value is considered a clear, measurable effect (i.e., the EC10 value is close to a 'no-effect' value), therefore for the purposes of this assessment, the value of 2440 mg/kg is selected as the CTV. Based on inter/intra species variation for a chronic test and the uncertainty in exposure time needed for DBDPE, a total assessment factor of 10 is applied, and the resulting PNEC is 244 mg/kg dry soil. When this value is adjusted from test organic carbon content (0.027) to standard soil 2% organic carbon content (ECHA 2010), the PNEC for soil organisms is 180.7 mg/kg dry soil.

9.1.4 Empirical Studies in Wildlife

There are limited DBDPE studies relevant to wildlife. Standard mammalian (rodent) repeated dose toxicity studies conducted with DBDPE have generally shown No Observed Adverse Effect Levels (NOAEL) at the highest dose tested: 1000 mg/kg/day (90 day rat study), and 1250 mg/kg/day (rat and rabbit pre-natal) (Margitich 1992, Hardy et al. 2002, 2010, and 2011). Wang et al. (2010) reported a Lowest Observed Effect Level (LOEL) of 100 mg/kg bw/day based on significant increase in serum thyroid hormone triiodothyronine (T3) levels at this dose level in Male Sprague-Dawley rats (6 per treatment, orally administrated 100 mg/kg/day of DBDPE in corn oil for 90 days). See the Health Effects section for detailed analysis of rodent toxicity studies.

Egloff et al. (2011) conducted a study of combined *in vitro*/*in ovo* approach to determine concentration-dependent effects on overt toxicity and hepatic messenger RNA (mRNA) expression levels of 11 transcripts in primary cultures of chicken embryonic hepatocytes. DBDPE was added at the following concentrations: 0.001, 0.003, 0.01, 0.03, 0.1, 0.2 µM DBDPE (concentrations exceeding those reported in wildlife)(n= 3 replicates per treatment group) and incubated for 36 hours. Hepatocyte viability was not

affected by DBDPE (or any BFR). DBDPE induced CYP1A 4/5 by 29 and 59 fold at 0.2 uM.

The existing mammalian and avian studies suggest that although DBDPE may be bioavailable to wildlife, it is not overtly toxic (no effects at highest dose), and organisms may show indication of potential to metabolize DBDPE. However, the recent molecular-level studies (avian and fish) suggest DBDPE may affect enzyme activity levels in test species.

CTV in wildlife estimates of 754 and 458 mg/kg bw day were determined from a wildlife Toxicity Reference Value (TRV) approach (Sample et al. 1996), where effects in rats were normalized to a typical body weight of mink (*Mustela vison)* and river otter (*Lontra canadensis)* respectively, which are representative wildlife species (Environment Canada 2015), for detailed calculation and input values). Toxicity endpoints (NOAEL =1000 mg/kg day) from Margitich (1992) were selected from a range of laboratory rodents tests to determine a TRV, based upon a 90-day oral sub-chronic toxicity study with rats (see Health Assessment Section, Margitich (1992)). An assessment factor of 10 was applied to account for extrapolation from a subchronic no-effect value to a chronic no-effect value. The resulting TRV was 45.8 to 75.4 mg/kg food bw day for mink and river otter respectively.

9.1.5 Ecological Effects of Transformation Products

Modelled aquatic toxicity predictions were undertaken for possible DBDPE transformation products (Appendix C, Table C-5). Similar to DBDPE, the pelagic environment is unlikely to be the most important route of exposure for the very hydrophobic higher brominated BDPEs (e.g., octa and nonaBDPEs, and hydroxylated nona and DBDPEs), but currently represents the only toxicity data for potential transformation products and therefore serve as a coarse level of screening. ECOSAR (2012) classifies the penta- through nonaBDPEs as neutral organics, based on chemical structure. Two potential transformation products predicted by Catalogic (2012) include hydroxylated nonaBDPEs (classified as phenols) and the brominated phenyl acids (classified as halo acids, vinyl/allyl halides-acid, and neutral organics). The hydroxylated DBDPE transformation product predicted by the rat metabolism (CPOPS 2012) was classified as a benzyl alcohol in addition to neutral organic. In general, most groups of predicted transformation products were estimated to have toxic chronic effects at very low concentrations (<<1 mg/L) for at least some endpoints, representing a concern if the substances form in the environment. NonaBDPEs, octaBDPEs and hydroxylated nonaBDPEs were estimated to have no effects at saturation due to very low water solubility and high logK_{ow} (>8).

Considering that potential transformation products are expected to be more bioavailable and likely more bioaccumulative than parent DBDPE, the predicted aquatic toxicity at low concentrations should be considered, despite model uncertainty. With no empirical toxicity data to evaluate for lower brominated BDPEs, data from analogue decaBDE are

also considered, which support that potential transformation products could be inherently toxic (e.g., tetra through hexaBDEs) (EC 2006).

9.2 Ecological Exposure Assessment

While measured DBDPE concentrations in the environment have been presented, limited data concerning the concentrations of DBDPE in water in Canada have been identified. Therefore, environmental concentrations have been estimated from available Canadian information, including estimated substance quantities, estimated release rates, and characteristics of the receiving environment. Environmental concentrations have been estimated for industrial release scenarios, as described below.

9.2.1 Industrial Exposure Scenarios and Predicted Environmental Concentrations

Aquatic exposure to DBDPE is expected if the substance is released from industry (e.g., formulation) either directly to receiving surface water or to a wastewater treatment system that discharges its effluent to a receiving surface water body. The concentration of the substance in the receiving water near the discharge point of the wastewater system is used as the predicted environmental concentration (PEC) in evaluating the aquatic risk (e.g., to water and/or sediment) of the substance. It can be calculated using the equation:

$$PEC = \frac{1000 \times Q \times L \times (1 - R)}{N \times F \times D}$$

Where

PEC: aquatic concentration resulting from industrial releases, mg/L
Q: total substance quantity used annually at an industrial site, kg/yr
L: loss to wastewater, fraction
R: wastewater system removal rate, fraction
N: number of annual release days, d/yr
F: wastewater system effluent flow, m^3/d
D: receiving water dilution factor, dimensionless

As DBDPE is used by industrial facilities and is expected to be released to water, several conservative aquatic industrial release scenarios were developed to cover a range of different potential industrial activities in Canada. The scenarios include rubber compounders, plastic compounders, plastic injection molders, plastic extrusion facilities, textile backcoating facilities and other facilities using DBDPE for unspecified industrial activities (see Environment Canada (2015) for descriptions of industrial scenarios). Information from the different facilities considered was collected and scenarios reflected expected practices and conditions, including type of wastewater treatment, direct or indirect releases to the receiving media, and receiving environment.

Table 9-3 presents the range of inputs used to estimate resulting aquatic concentrations close to the industrial point of discharge. Based on these assumptions, these industrial scenarios yield total predicted environmental aquatic concentrations (PECs) of 3.0 x 10^{-7} to 2.1 x 10^{-3} mg/L. These PEC values represent the total DBDPE concentrations (particle associated and dissolved) in the receiving water near the point of the discharge at each site, and in cases these exceed the water solubility of DBDPE (i.e., dissolved DBDPE limit) by 1 to >2 orders of magnitude. The highest PECs result from industrial scenarios associated with high releases which are also uncertain (e.g., typically textile or unspecified industries), and therefore are considered more conservative.

Table 9-3. Summary of input values used for scenarios estimating aquatic concentrations resulting from industrial releases of DBDPE

Input	Value	Justification and reference
Quantity used per site (kg)	1000 to 1 000 000	Section 71 survey or New Substances Notification
Loss to wastewater (%)	0.001 to 1.0	OECD 2004, 2009
Wastewater system removal efficiency (%)	0, 59.7, or 94	Predicted for no treatment, primary treatment, secondary treatment
Number of annual release days (days)	250 or 350	EC standard assumption for continuous releases or NPRI data
Wastewater system effluent flow (m^3/d)	2900 to 400 000	Site specific wastewater system data
Dilution factor (–)	1 to 10	Site specific wastewater systemflow rate/ receiving environment flow rate. When a dilution factor was greater than 10, a maximum default value of 10 was used.

An equilibrium sediment-water partition approach was used to estimate the concentration of DBDPE in bottom sediment. This approach is based on a partitioning principle described by the European Chemicals Agency (ECHA 2010) and incorporates two additional calculation methods. The first method is to estimate the substance's concentration in the aqueous phase (dissolved) of the overlying water from its total concentration, according to studies by Gobas (2007 and 2010). The second method is to estimate a substance's concentration in bottom sediment from its concentration in the aqueous phase of the overlying water based on an equilibrium partitioning assumption between bottom sediment and overlying water described by the USEPA's National Center for Environmental Assessment (USEPA 2003). At equilibrium, the predicted environmental concentration (PEC) in bottom sediment can linearly correlate with the concentration in the aqueous phase of the overlying water. Sediment exposure scenarios were developed as an extension of the industrial aquatic release scenarios described above to determine equilibrium sediment PECs, standardized to 4% organic

carbon (typical organic carbon content in bottom sediment for rivers and lakes). However, where predicted aquatic PEC values exceeded water solubility (8.10×10^{-6} mg/L), the water solubility limit for DBDPE was used for the upper limit, and the resulting water PECs ranged from 3.0×10^{-7} mg/L to 8.1×10^{-6} mg/L. The resulting PEC values in bottom sediment ranged from 0.01 to 0.24 mg/kg dw.

An approach described by the ECHA (2010) was used to estimate predicted environmental concentrations in soil (soil PECs) resulting from the land application of wastewater biosolids. This approach employed the quantity of biosolids accumulated within the top 20-cm layer (ploughing depth) of soil over 10 consecutive years as the basis for soil PECs. One underlying assumption of the approach was that substances were subject to no loss due to degradation, volatilization, leaching and soil run-off upon their entry into soil via biosolids land application. This assumption, therefore, yielded conservative soil PECs. Soil exposure scenarios were developed as an extension of the aquatic release scenarios described above, using sludge concentration and production rates based on site specific waste water treatment plants. The estimated concentration in biosolids ranged from 0.1 mg/kg dw to 730 mg/kg dw, which is well above the current range of Canadian biosolid DBDPE concentrations reported (0.034 to 0.220 mg/kg dw). Soil PECs were standardized to 2% organic carbon (ECHA 2010), and for application over 10 years (20 cm), and the resulting PEC values ranged from 0.0047 to 25.3 mg/kg dw.

9.2.2 Consumer Product Exposure Scenario and Predicted Environmental Concentrations

In addition to industrial sources of DBDPE, consumer or commercial products represent a potential source of DBDPE to the environment, through both volatilization and particulates from abrasion (ECB 2004). The presence of DBDPE in dust samples (see Human Health section) and wastewater treatment plant media (influent, effluent, and wastewater sludge) (Ricklund et al. 2008, Kolic et al. 2009, Davis et al. 2012, Kim et al. 2014), support that the substance can be released from consumer products (Davis et al. 2012, Melymuk et al. 2014), although there is minimal literature quantifying such releases. While service life release rates from consumer or commercial products were not found for DBDPE, a study by Kemmlein et al. (2003) determined specific emission rates (SER) of 0.3 ng/m^2/h for decaBDE (from the Commercial OctaBDE mixture) during a 105-day test of television set housing (23 °C). OECD (2009) identifies potential volatility to atmosphere from service life for generic OFRs in plastics, estimated at 0.05% over lifetime for indoor or outdoor use; however, this generic value may be an overestimate for a very low volatility OFR like DBDPE. Environmental release of the substance from plastic polymers via leaching is considered possible, albeit low, as many products identified with DBDPE will not be in contact with water on a regular basis (e.g., electronics). The potential release of OFRs from plastics during service life to water is estimated at 0.05% over lifetime if the substance is for indoor use or 0.16% over service life for outdoor use (OECD 2009). The large majority of DBDPE containing products would be enclosed or used for indoor use; the release rate of 0.05% is

therefore most applicable and may likely be an overestimate since contact with water is not expected.

Current research on decaBDE release from products was examined to investigate likely DBDPE behaviour, due to the lack of data on DBDPE. Based on measurements in Toronto, Canada, Melymuk et al. (2014) have proposed a typical pathway of urban PBDE release as follows:

- release from consumer products to the indoor environment (e.g. by volatilization, abrasion of product containing flame retardant substance), then transportation (of dust) to the outdoor environment;
- deposition on surfaces; and
- wash-off to storm water (e.g., WWTPs and tributaries).

Examination of decaBDE loadings to Lake Ontario from Toronto determined that about 30% of total loading was via WWTP sources, approximately 60% via tributaries, and less than 8% via air deposition (wet and dry particle) (Melymuk et al. 2014). Assuming a similar pathway for DBDPE release from products, available measured concentrations in water, sediment and biosolids (Kim et al. 2014) are well below those estimated by the exposure scenarios from industrial uses, and thus, existing scenarios appear adequately protective of additional, potential releases from consumer and commercial products.

Various mechanisms for OFR transfer from consumer products to dust have been proposed (Toms et al. 2011). In one study, clothing, and the dust entrapped with it, has been proposed as an important source of additive flame retardants, including DBDPE to wastewater treatment systems via cleaning and laundering activities (Schreder and La Guardia 2014, Melmyuk et al. 2014).

Schreder and La Guardia (2014) measured the mean concentration of DBDPE in residential dust and laundry wastewater sampled from 20 homes in the northwestern United States between 2011 and 2012. The mean concentration of DBDPE in the laundry wastewater was measured as 24.5 ng/L. It is noted that the concentration of DBDPE in laundry wastewater is slightly above the modeled water solubility, but may reflect a total concentration. The authors also measured the influent and effluent concentrations of DBDPE at two wastewater treatment plants serving these homes. These plants receive over 80% of their input from households, with no known flame retardant discharges from the remaining industrial contribution. Using the proportion of influent expected from laundry wastewater and the proportion of influent expected from households, the authors determined that laundry wastewater may be a primary source of these flame retardants to the wastewater treatment plants (Schreder and La Guardia 2014).

Laundry wastewater data from the northwestern United States from the Schreder and La Guardia study (2014) is considered sufficiently representative to construct an exposure scenario relevant to Canada for laundry wastewater, as a route to the

environment for DBDPE released from consumer products. Environment Canada indicates that the average domestic water use is 343 L/day/Canadian, while 20% of the water is used for laundry (Environment Canada 2013). These values, multiplied by 365 days/year, 35,540,400 Canadians, and the mean concentrations of DBDPE in laundry wastewater reported above give an annual national release of 21.8 kg/year for DBDPE (Schreder and La Guardia 2014, Statistics Canada 2014).

Overall, releases from consumer products are expected to be geographically dispersed and spread out over the duration of the service life and end-of-life stages. The low release rate of 21.8 kg/year, dispersed across Canada, suggests the potential for exposure to DBDPE from laundry wastewaster is lower than potential for exposure from industrial sources. Therefore, the exposure scenarios from industrial uses presented above, appear adequately protective of potential releases from this laundry wastewater source.

While the laundry scenario presented above may address a major source of release to the environment during service life of consumer products, there is an absence of data to quantitatively address solid waste disposal of dust and end-of-life releases from all manufactured items, including non-residential sources. For example, seepage water from a metal recycling site in Norway reported a DBDPE average concentration of 80 ng/L, although DBDPE was not detected in seepage water from a municipal landfill in the same study (Nyholm et al. 2013).

9.2.3 Exposure of Degradation Products

With no empirical data for potential DBDPE transformation products, methods to estimate the potential exposure of such substances in the environment are limited. The quantity of transformation products is assumed to be less than the reported volumes for parent DBDPE. Using CPOPs (2012) generated molar ratios of predicted DBDPE transformation products relative to DBDPE, depending on the transformation pathway, ratios of initial transformation products produced could range from 3 to 6% of DBDPE (formation of nonaBDPE or hydroxylated nonaBDPE via aerobic biodegradation), to 31 to 56% of DBDPE (hydroxylated DBDPE and hydroxylated nona BDPE predicted for *in vivo* rat metabolism products). Latter molar ratios for *in vivo* rat metabolism suggest that a third to half of parent DBDPE bioavailable to organisms could be transformed, which could have implications for wildlife exposure. However, the evidence presented suggests that only a small fraction of DBDPE will be bioavailable (see bioaccumulation section), thereby limiting wildlife exposure to such biotransformation products. Molar ratios for aerobic biodegradation may be most relevant for DBDPE found in soil and possibly sediment or wastewater /sludge/biosolids. A small ratio (3 to 6%) is attributed to these transformation products relative to the parent DBDPE; however, similar to DBDPE, models suggest some transformation products could be persistent, and therefore, build-up in the environment over extended periods of time. Furthermore, this percentage of transformation is similar to measured decaBDE debromination in sediment microcosms over a one-month period ([^{13}C]decaBDE decreased by up to 3.5%, as a percent of total PBDEs) (Orihel et al. 2016), as well as predicted decaBDE

transformation using CPOPS (2012). The molar ratio of lower brominated BDPEs (i.e., octaBDPEs and lower) is predicted to be <1% relative to parent. Therefore, while it appears, based on predictive degradation pathway molar ratios, that transformation products are expected to represent a minor fraction relative to parent DBDPE, they are a similar fraction to those predicted/measured for analogue decaBDE. Furthermore, if DBDPE levels in the environment continue to increase, the pool of potential transformation products would also be expected to increase in importance.

9.3. Characterization of Ecological Risk

9.3.1 Risk Quotient Analysis

A risk quotient analysis, integrating conservative estimates of exposure with toxicity information, was performed for the sediment and soil media, as well as for wildlife, to determine whether there is potential for ecological harm in Canada. A risk quotient analysis was not conducted for the pelagic aquatic environment due to low relevance and unreliable toxicity data.

The site-specific industrial scenarios (considering the actual receiving water bodies) presented above yielded predicted environmental concentrations (PEC) from 3.0×10^{-7} mg/L to $.8.1 \times 10^{-6}$ mg/L (water solubility). These PEC values represent the level of exposure in the receiving water near the point of the discharge. Using aquatic PECs in water to determine equilibrium sediment PECs, standardized to 4% OC, the resulting sediment PEC values ranged from 0.01 to 0.24 mg/kg dw. A predicted no-effect concentration (PNEC) was derived from the chronic sediment organism toxicity values to give a value of 1111 mg/ kg dw (see Ecological Effects Assessment section). The resulting risk quotients (PEC/PNEC) = 8.0×10^{-6} to 2.2×10^{-4} . Therefore harm to sediment organisms is unlikely for these industrial scenarios, within greater than 1000-fold margin of error.

Predicted soil PECs resulting from the land application of wastewater biosolids to land (standardized to 2% OC) ranged from 0.0047 to 25.3 mg/kg dw. The PNEC for soil organisms is calculated as 180.7 mg/kg dw (See Ecological Effects Assessment section). The resulting risk quotients (PEC/PNEC) =2.6×10^{-5} to 0.14. Therefore harm to soil organisms is unlikely at these sites, within 10 fold margin of error.

A wildlife TDI was derived from a Total Daily Intake (TDI) for mink (*Mustela vison*) and river otter (*Lontra canadensis*) consuming fish following the approach of US EPA (1993). In calculating TDI, a northern pike liver tissue concentration (Ci) of 0.00378 mg/kw (ww) was selected and equated to whole body concentration as a conservative assumption. This value represented the highest published concentration of DBDPE in Canadian biota (Houde et al. 2014), resulting in a TDI of 0.0007 (otter) to 0.002 (mink) mg/kg bw day (see Supporting Information (Environment Canada 2015) for details of TDI model inputs). The derived TRV was 75.4 (mink) and 45.8 (otter) mg/kg bw day (see Ecological Effects Assessment section). The resulting risk quotient results (TDI/TRV) are 9.48×10^{-6} for mink and 4.07×10^{-5} for otter, indicating that even with

conservative assumptions, current DBDPE concentrations in Canadian biota are unlikely to exceed minimum effects levels, within > 1000-fold margin of error (Table 9-4).

Table 9-4. Risk quotients obtained for different media and exposure scenarios for DBDPE

Media	Scenario	PNEC/TRV	PEC/TDI	RQ
Sediment	Industrial release to water	1111 mg/kg dw	0.01 to 0.24 mg/kg dw	8.0×10^{-6} to 2.2×10^{-4}
Soil	Biosolids application to soil	180.7 mg/kg dw	0.0047 to 25.3 mg/kg dw	2.6×10^{-5} to 0.14
Wildlife	Piscivore TDI (mink/fish)	75.4 (mink) and 45.8 (otter) mg/kg bw /day	0.0007 (otter) to 0.002 (mink) mg/kg bw /day	9.48×10^{-6} (mink) 4.07×10^{-5} (otter)

9.3.2 Consideration of Lines of Evidence and Conclusion

DBDPE, as well as potential transformation products, are expected to be persistent in air, water, soil and sediment. It is difficult to conclude based on the limited bioaccumulation data for DBDPE; however, the weight of evidence to date suggests DBDPE itself is expected to have a low to moderate bioaccumulation potential. Yet, considering the analogue decaBDE, as well as preliminary DBDPE transformation product studies and modeling, there is concern for potential formation of bioaccumulative (and more hazardous) transformation products, which can be considered analogues to lower brominated polybrominated diphenyl ethers (PBDEs). The Ecological Screening Assessment on PBDEs (June 2006) concluded that lower brominated PBDEs, namely tetraBDE, pentaBDE and hexaBDE, satisfy the criteria outlined in the Persistence and Bioaccumulation Regulations of CEPA 1999.

The high importation volumes of DBDPE into Canada, along with information on its uses, indicate potential for widespread release into the Canadian environment. Once released into the environment, DBDPE will be found mainly in sediment and soil, where it may persist for long periods of time, resulting in DBDPE build-up, as seen by rapid doubling times in sediment in the Great Lakes. DBDPE, via sorption to particles, also has potential for long-range transport and deposition in remote areas, based on results of Long Range Transport modelling and some measurements in remote areas. Most early hazard studies suggest DBDPE itself demonstrates low potential for toxicity to aquatic sediment, terrestrial organisms, and wildlife; however, there is some uncertainty with respect to DBDPE mode of action. At least one set of DBDPE aquatic studies (although using solvent) suggest molecular-level effects at low concentrations; recent analogue decaBDE studies have also reported effects on aquatic organisms (water, sediment, and diet exposure) at low concentrations, warranting consideration of precaution. Modelled aquatic toxicity data for potential DBDPE debrominated transformation products suggest effects at low concentrations in the range of water solubility. The lack of empirical toxicity data (e.g., for pelagic, benthic and soil

organisms) for potential transformation products is an important source of uncertainty, warranting consideration of precaution. While it appears that, based on predictive degradation pathway molar ratios, transformation products are expected to represent a minor fraction relative to parent DBDPE, they are similar to predicted/measured fractions of analogue decaBDE debromination products, and if DBDPE levels in the environment continue to increase (as seen in Great Lakes sediment), the pool of potential brominated BDPE transformation products could become important.

This information indicates that DBDPE has the potential to cause ecological harm in Canada.

9.3.3 Uncertainties in Evaluation of Ecological Risk

One of the major areas of uncertainty affecting results in the assessment is 'analytical uncertainty'. DBDPE is a difficult brominated flame retardant to analyze, and uncertainty can reach 40-60%, depending on the internal standard used for quantification (A. Covaci pers. comm.). Inaccurate measurement of DBDPE in media could impact all areas of the assessment involving measured data, and importantly, the bioaccumulation assessment and hazard assessment.

There is high confidence that DBDPE is very stable in the environment with a long residence time.

Laboratory-based and measured data on bioaccumulation potential for DBDPE (i.e., BAFs and BMFs) are lacking. Generally, existing data on DBDPE bioaccumulation are limited and uncertain, and reliable quality laboratory and field measurements are not available. While the current weight of evidence suggests low to moderate bioaccumulation potential, consideration of log K_{ow} and log K_{oa} values suggests that DBDPE may have the potential to biomagnify in terrestrial food webs as a result of dietary exposure. An underestimation of bioavailability of DBDPE could result in underestimation of DBDPE exposure to organisms. There is a moderate level of confidence with the conclusion of low to moderate DBDPE bioaccumulation potential.

PNECs/TRVs for risk analysis were developed based on available CTVs from DBDPE toxicity studies for soil, sediment, and rodents, which reported low to no toxicity. However, there are information gaps on the toxicity of DBDPE to wildlife and effects on pelagic, sediment and terrestrial species resulting from prolonged (e.g., lifetime and mutigenerational) exposure. In addition, there are some recent aquatic and sediment analogue decaBDE studies reporting effects at lower concentrations (e.g., ~10x lower concentration in sediment than current PNEC); although there are uncertainties with these studies as well, there is the possibility future DBDPE toxicity studies may determine similar effects at low concentrations. This uncertainty was considered in the application of precaution and the proposed conclusion for the assessment. Due to unreliable aquatic toxicity results and assumptions of low relevance of the pelagic environment for DBDPE exposure, risk was not determined for the pelagic aquatic environment. This could represent a source of uncertainty for risk to aquatic organisms;

however, pelagic exposure to DBDPE is considered to be lower than exposures via the benthos and benthic-related food chains. Generally, confidence in DBDPE toxicity results is low to moderate.

Uncertainties are present due to the lack of information on the environmental concentrations in Canada, particularly in water, soils, sediments and biota. In spite of this limitation, exposure scenarios for use in risk analysis were developed based on the best available information and they are considered sufficiently conservative to characterize potential risks from releases of DBDPE to the Canadian environment. Even with conservative assumptions of large DBDPE quantities in use at industrial sites, risk quotients were much less than one, suggesting low risk.

The exposure assessment focuses on industrial point sources as being most relevant for DBDPE in the environment. From its use as an additive flame retardant, some DBDPE will migrate from products, as evidenced by concentrations in household dust (see Health Assessment section). There is also limited information characterizing potential releases from products in use and during disposal/recycling of at the end of their service life in this assessment. No CanadianDBDPE landfill leachate data have been reported to date, but such data could help interpret end-of-life releases. DBDPE quantity in products (considering all products imported and in use) could be high; however, based on available analogue decaBDE information, it assumed that major DBDPE pathways of release from products in service are covered under the current industrial release scenarios. Furthermore the dust-laundry wastewater exposure scenario based on measured DBDPE in USA suggests predicted DBDPE release via this pathway in Canada is also covered under the current industrial release scenarios. Generally, there is moderate confidence in the DBDPE exposure scenarios.

The greatest uncertainty in this assessment relates to the lack of data for the occurrence of DBDPE transformation products in the environment; it is uncertain what transformation products may form and in what quantities. There are very limited empirical data available for transformation products, and the few laboratory studies reporting DBDPE degradation are for photodegradation in solvents, which is not environmentally relevant. However, based on the analogous chemical, decaBDE, debromination of DBDPE in the environment is expected. While the only DBDPE debrominated products identified in empirical studies to date are nonaBDPEs (in sediment and solvents) through heptaBDPEs (solvents only), DBDPE debromination to hexa and pentaBDPEs is also expected based on the close analogue, decaBDE.

Therefore, while this assessment predicts that DBDPE transformation products have high overall persistence, bioaccumulation potential, and ecotoxicity, it must be considered that there is uncertainty with these estimates. There are no experimental studies evaluating physical chemical properties, persistence, bioaccumulation potential, or toxicity of any DBDPE debrominated products to verify the predicted values. Predicted transformation products are within the domain of the Persistence and Bioaccumulation models presented, but there is less certainty with aquatic toxicity predictions. It is possible aquatic toxicity is over-estimated for transformation products.

However, based on analogue decaBDE, it is expected that DBDPE transformation products are more bioavailable and hazardous than parent DBDPE.

10. Potential to Cause Harm to Human Health

10.1 Exposure Assessment

10.1.1 Environmental Media and Food

DBDPE is associated with negligible solubility in water and a very low volatility, and is expected to partition predominantly to dust and sediment when released to the environment. In residential environments, DBDPE is a common component of house dust. Based on Health Canada (1998) intake rates for air, water, food and dust/soil, the estimated total intake of DBDPE from environmental media and food for the Canadian population ranges from 0.001 for adults over 60 years old to 0.061 µg/kg bw/day, for infants aged 0 to 0.5 years. Dust is the main contributor to the total daily exposure from environmental media and food. These results are summarized in Appendix D.

Concentrations in environmental media selected to derive estimated intakes are described in the following sections.

10.1.1.1 Air

Ambient Air

Reported concentrations of DBDPE in ambient air are presented in Supporting Information document (Environment Canada 2015).

No reports of DBDPE in ambient air from Canadian environments were identified, although it has been detected in ice cores from Devon Island, Nunavut (Meyer et al. 2012). DBDPE has been detected in ambient air samples from other countries. The atmospheric concentration of DBDPE in the US is monitored as a part of the Integrated Atmospheric Deposition Network (IADN), with published data available from 2003 to 2011. The highest mean concentration (vapour plus particle phases) reported for an urban site was 22 pg/m^3 for Cleveland, Ohio, when monitored between 2005 and 2006 (Venier and Hites 2008). In the same time period (2005-2006), the lowest mean concentration reported was for a remote site in Eagle Harbor, Michigan, at 1.0 pg/m^3. Over the 2005-2011 time period, the highest mean concentration (particle phase only) for an urban site was 5.2 pg/m^3, also for Cleveland, Ohio, while the lowest mean concentration (particle phase only) reported for a remote site was 1.2 pg/m^3, again for Eagle Harbor, Michigan (Ma et al. 2013). The authors of this study stated that data were too sparse for DBDPE to determine if there was a temporal trend (no consistent trends amongst the five sampling sites in the Great Lakes). Mean concentrations of 7 or 10 substances measured, including DBDPE, were higher in Cleveland than in Chicago even though Cleveland's population is only about one third of Chicago's population, and the authors suggested that this may be related to the presence of one or more point or

industrial sources of flame retardants in or near Cleveland (Ma et al. 2103). DBDPE was also detected in atmospheric particles in Aspvreten, Sweden; a maximum concentration of 7.9 pg/m^3 was reported for this remote location (Egebäck et al. 2012). A Nordic screening project reported ambient air concentrations of DBDPE for locations in Denmark, Norway and Sweden (Nordic Co-operation 2011). Of all the locations monitored, the highest concentration was reported for atmospheric particles collected from an urban area in Oslo, Norway, at 44 pg/m^3.

The maximum DBDPE concentration in ambient air reported for China was 3578 pg/m^3 in a highly industrialized and urbanized area (Shi et al. 2009). Tian et al. (2011) also reported a deposition rate of 41 600 ng/m^2/year (approximately 114 ng/m^2/day) in the same highly industrialized and urbanized area (as well as 9780 and 850 ng/m^2/year in near an electronics waste recycling site and in a rural site, respectively) in southern China.

To derive the upper-bounding estimate of daily intake from outdoor air for the Canadian population, the highest reported mean concentration from Cleveland (Venier and Hites 2008) was used as it is considered representative of urban exposure in Canada, and as suggested by Ma et al. (2013), it may also account for point sources of DBDPE.

Indoor Air

No reports of DBDPE in indoor air in Canada were identified. Residential indoor air studies in Oslo, Norway and three different cities in Sweden reported concentrations ranging from not detected to 0.963 ng/m^3 (963 pg/m^3) (Karlsson et al. 2007; Cequier et al. 2014). A Nordic screening project reported a maximum concentration of 0.0056 ng/m^3 (56 pg/m^3) for DBDPE in atmospheric particles in an office building in Oslo, Norway (Nordic Co-operation 2011). DBBPE was not detected in the indoor air of three aircraft during flights in Sweden (LOD = 0.01 picomol; Strid et al. 2014), nor was it detected in the indoor air of a hotel in Japan (Takigami et al. 2009).

The maximum concentration of 0.963 ng/m^3 reported in the Norwegian residential study (Cequier et al. 2014) was used to derive the upper-bounding estimate of daily indoor air intake for the Canadian population.

Estimates of exposure via air (indoor and ambient), as shown in Appendix D. For indoor air, the upper bounding estimates ranged from 0.0002 to 0.0005 µg/kg bw/day. For outdoor air, the estimates are less than 0.0001 µg/kg bw/day which are considered negligible.

10.1.1.2 Dust

DBDPE is a common component of house dust and its presence has been reported in numerous house dust samples. Although the source of DBDPE is not typically identified, it may be electronic equipment that contains DBDPE as an additive flame retardant and/or it may occur in dust transported into homes from outside environments. In one

unpublished Canadian study, 5 homes in Toronto were sampled each year from 2010 to 2012 and the maximum dust concentration was 5600 ng/g. The authors of this study stated that DBDPE dust profiles for living rooms and bedrooms were higher, compared to kitchens. In 2011, DBDPE was not detected in additional window wipe samples from 5 homes in Toronto nor in the majority of hand wipe samples from occupants living in the sampled homes [8% detection frequency; number of participants not stated] (Diamond et al. 2013).The highest maximum concentration of DBDPE in US house dust is 11 070 ng/g, reported by Stapleton et al. (2008) in a study in which samples were collected from 20 homes in the Greater Boston Area. It was also measured in dust samples from 16 homes in San Francisco with the highest level reported at 2800 ng/g (Dodson et al. 2012).

In Europe, the highest maximum concentration of DBDPE reported for house dust was from Sweden at 24 000 ng/g, measured from one of 6 apartments; however, concentrations in the 5 other apartments and one home ranged from 470 to 2200 ng/g (Sahlström et al. 2012; Remberger et al. 2014). The maximum concentration reported in Romania (12 240 ng/g) was comparable to the maximum level reported in the US (Dirtu et al. 2012), and lower values were reported for maximum concentrations in house dust from Oslo, Norway (4460 ng/g; Cequier et al. 2014), Prague, the Czech Republic (3567 ng/g, Kalachova et al. 2012), West Midlands, UK (3400 ng/g, Harrad et al. 2008) and Belgium (2126 ng/g, Ali et al. 2011a), and Germany (1570 ng/g, Fromme et al. 2014). In Asia, the maximum reported concentration in urban house dust was 16 000 ng/g reported from China (Qi et al. 2014), but lower maximums were reported in other countries: 2175 ng/g in Kuwait City, Kuwait (Ali et al. 2013a) and 850 ng/g in Gujrat, Pakistan (Ali et al. 2012a). The maximum reported concentration of DBDPE in house dust from New Zealand was 1430 ng/g (Ali et al. 2012b). The differences in concentrations observed may be due to varying flammability standards in different jurisdictions, or the diversity of consumer products found in each home.

DBDPE has also been detected in non-residential indoor environments. Among three studies of dust from classrooms, offices and vehicles from West Midlands, UK (Ali et al. 2011a, Goosey et al. 2009, Harrad et al. 2008), vehicle dust was found to contain the highest maximum concentration of DBDPE at 2900 ng/g (Harrad et al. 2008). In Prague, the Czech Republic, the maximum reported concentration of DBDPE in vehicle dust was 3567 ng/g (Kalachova et al. 2012). Maximum concentrations in vehicle dust in Kuwait City, Kuwait (8200 ng/g) and Faisalabad, Pakistan (5420 ng/g) were generally higher than those reported for vehicles in Europe. However, one sample of dust in a new car in Sweden revealed a concentration of 92,000 ng/g (Remberger et al. 2014). During flights within Sweden, DBDPE levels in three aircraft ranged from < 1460 to 5730 ng/g (Strid et al. 2014). Reported maximum concentrations in dust in non-residential buildings in Germany and Sweden ranged from 140 (school) to 8100 ng/g (conference centre) DBDPE (Brommer et al. 2012; Remberger et al. 2014). . In a study of 81 urban and rural homes and buildings in China, higher concentrations were noted in urban buildings versus rural buildings, with maximum concentrations in ranging from 5300 (office) to 13 000 ng/g (mushroom factory) (Qi et al. 2014). The highest concentration reported in three offices in Beijing, China was 16,200 ng/g (Cao et al. 2014). In

electronic waste facilities in Thailand, DBDPE concentrations in dust ranged from 380 to 44 000 ng/g (mean = 8,630 ng/g); the six highest measures (12 500-44 000 ng/g) in all 21 samples were attributed to dust from rooms storing personal computers and printers (Ali et al. 2011b). Concentrations of 20 000 and 23 0000 ng/g were also reported in a waste recycling facility in Sweden (Remberger et al. 2014).

Results from North American studies are considered most representative of levels in Canadian dust. Since results from the Toronto study showed a lower maximum indoor dust concentration and were based on a smaller sample size (5 homes sampled in the Toronto study compared to 20 in Boston), the maximum value of 11 070 ng/g from the Boston study (Stapleton et al., 2008) was selected to derive an upper bound estimate of daily intake from dust for the Canadian general population. This level is considered upper bound and accounts for potential variability in dust levels from different indoor settings (e.g., offices, vehicles, aircraft).

For all age groups, exposure from ingesting dust contributed 76-100% of the estimated daily intake of DBDPE.

10.1.1.3 Soil and Sediment

No monitoring data on DBDPE in soil in Canada or the U.S. were identified. Although there were reported concentrations of DBDPE in surface soil from Indonesia and in farmland soil from China (Ilyas et al. 2011; Shi et al. 2009), it was considered that modeled estimates of soil concentration for Canada (see below) were more relevant to the estimates of intake based on soil.

A maximum DBDPE soil predicted environmental concentration (PEC) of 25.3 µg/g dw (25.3 mg/kg dw) was estimated for land application of biosolids on an agricultural field using conservative approaches (see section 9.2.1). As no relevant monitoring studies on DBDPE in soil were identified, the soil maximum PEC (59 000 µg/g dw) was selected for upper-bounding intakes from the ingestion of soil for the general population in Canada.

10.1.1.4 Water

Reported concentrations of DBDPE in surface water and precipitation are presented in Supporting Information (Environment Canada 2015). Measurements in groundwater or drinking water were not identified.

In a recent study, DBDPE was detected in all five Great Lakes at average concentrations of 0.25 ± 0.05 pg/L (L. Huron) to 6.7 ± 5.0 pg/L (L. Superior) [LOD not stated]. The highest concentration was from Lake Ontario at 10.8 pg/L, although there was only one sample from this lake, in which DBDPE was detected. The authors stated that the concentration measured in Lake Ontario was similar to the concentrations measured in Lake Superior (Venier et al. 2014).Other studies of Canadian surface water have not detected DBDPE (Law et al. 2006, Muir et al. 2011).

In Norway, DBDPE was detected in one of three inlet water sources to waste-water treatment plants in Norway at a mean concentration of 5.1 ng/L (in 2/3 of composite samples of incoming wastewater), but it was not detected in seepage water from a municipal landfill site in Drammen, Norway (Nyholm et al. 2013). DBDPE was not detected in sea, tidal inlet, and river water in Spain (LOD = 5.0 ng/L) (Valls-Cantenys et al. 2013).

DBDPE was not detected in a southern Chinese pond (Wu et al. 2010). Another study sampled surface water from the Dongjiang River in southern China and reported the presence of DBDPE in both the dissolved phase (i.e., filtered water from the sample) and the particulate phase (He et al. 2012). DBDPE was found at a maximum concentration of 38 pg/L in the dissolved phase and 110 ng/g dry weight in the particulate phase. The Dongjiang River runs through Dongguan City, an intensive electronic and telecommunication equipment manufacturing base, and thus, the neighbouring industrial activities are assumed to contribute to the loading of DBDPE in the river.

The highest concentration of DBDPE reported in North American surface water (10.8 pg/L measured in Lake Ontario) was used to derive the upper-bounding estimate of drinking water intake for the Canadian population. Note, however, that DBDPE was not detected in surface water from 3 of 5 studies including Canadian locations, and in the two studies with detections, only negligible quantities (<1 $x10^{-6}$ g/L), were measured.

As part of the Integrated Atmospheric Deposition Network(IADN), measurable amounts of DBDPE have also been reported in precipitation for multiple US locations (Salamova and Hites 2010, Salamova and Hites 2011; Ma et al. 2013). Because precipitation undergoes many transformations prior to becoming a part of drinking water, the applicability of precipitation data for estimating general population exposure to DBDPE through drinking water is uncertain.

10.1.1.5 Food

No reports of DBDPE in Canadian food were identified. In two studies of food from the UK and Ireland, over 100 types of food samples were analyzed (including meat, fish, milk, cheese, eggs and vegetables) and DBDPE was not detected in any of the food samples (Fernandes et al. 2010; Tlustos et al. 2010). However, DBDPE was detected in infant formula and baby foods in the US and China in a recent study. The maximum DBPDE concentrations, measured in samples purchased in 2013 in U.S. stores, were 28.6 pg/g fresh weight (fw) in infant formula and 48.8 pg/g fw in baby food cereal. Although DBDPE was not detected in baby food puree in the US, it was detected in Chinese samples (10.2 to 16.2 pg/g in meat, vegetable, and mixed puree types). In the same study, several other halogenated flame retardants were also detected in infant formula and baby foods, including declorane plus (DP). Based on analyses of both food and packaging for DP, the authors concluded that it was unlikely that the samples were contaminated from their packaging, but rather that raw foods were contaminated or that they became contaminated during processing (Liu et al. 2014). By extension, it is

assumed that DBDPE levels in baby food cereal products are due to contamination of raw foods or during processing.

As shown in section 7, DBDPE has been sampled in the fish tissue of several freshwater species in Canada, and concentrations have generally ranged from non-detect to very low (i.e., mean concentration ≤ 1 ng/g lipid weight [lw]) (Ismail et al. 2006, Law et al. 2006, Kolic et al. 2009, Byer et al. 2010, Zhou et al. 2010b, Muir et al. 2011). The highest concentration measured in fish muscle was 3.3 ng/g lw, from sampling of six different fish species in Lake Winnipeg (Law et al. 2006), whereas the highest liver concentration was 26.7 ng/g lw, sampled from two fish species in the St. Lawrence River (Houde et al. 2014). Daily intake estimates of DBDPE from the consumption of infant formula and cereal products for the general population were based on the maximum concentrations in formula and cereal samples, respectively (Liu et al. 2014). Daily intake estimates of DBDPE from the consumption of fish were based on the maximum concentration of DBDPE in fish muscle reported for Lake Winnipeg fish, specifically Whitefish (Law et al. 2006), This estimate assumes that all fish consumed contains DBDPE and is considered to take into consideration potential variability in dietary exposure due to varying eating habits, including increased fish consumption or potential consumption of fish liver by certain subpopulations.

10.1.1.6 Breast milk

DBDPE was not detected (LOD = 1.7 ng/g lipid weight [lw]) in 91% of breast milk samples collected from 105 adult nursing women in Sherbrooke, Quebec, in 2008-2009 (see Appendix D). However, the 95th percentile and maximum detected values were 3.3 and 25 ng/g lw, respectively (9% detection frequency) (Zhou et al. 2014). In New Zealand, DBDPE was not detected in the breast milk of 35 of 36 first-time mothers; one sample showed a value of 325.50 pg/g lw (Mannetje et al. 2013). An upper-bounding estimate of daily intake from breastmilk was derived based on the 95th percentile concentration of DBDPE reported in the Sherbrooke, Quebec study (Zhou et al. 2014).

10.1.2 Consumer Products

DBDPE is an additive flame retardant used in the manufacture of plastics and rubber products, as an additive in textiles and polymers for electrical applications (Covaci et al 2011; EFSA 2012) (see section 5). DBDPE are expected to have similar polymer loading rates as decaBDE, which it is marketed to replace (Environment Agency 2007). Appendix E summarizes the reported concentrations of DBDPE in consumer products.

Health Canada conducted product testing on 23 items (e.g., children's toys, nursing pillows, crib mattress and other plastic, foam or textile products) purchased in retail stores in Ottawa in 2014. DBDPE was detected above the limit of quantification (LOQ of 0.5%) in the textile of a children's play tent; it was not detected in any of the other 22 products (Health Canada 2014a, b). Another Canadian study tested for brominated flame retardant (BFR) concentrations in a number of manufactured items available to consumers from the Greater Toronto area in 2012. DBDPE was not detected in the

majority of these products, except for the casings of audiovisual, computer products, and TV monitors, with maximum concentrations of 0.1 to 0.5 mg/g. The authors noted that DBPDE and some other targeted BFRs were often detected in a single sample of each product type, and stated that “The sources of these flame retardants at such a low level were not clear.” (Mochungong et al. 2014).

Black polymeric food contact articles from different distributors within Europe were analyzed for bromine content and subsequent identification of the flame retardants within them. Of the 10 articles tested, three were found to contain DBDPE and tetrabromobisphenol A together (electric frying pan), plus decabromodiphenylether [decaBDE] (thermos-cup cover) or bis(2,4,6-tribromophenoxy)ethane (apple cutter) at bromine concentrations ranging from 279 to 5975 mg/kg. These concentrations were lower than expected to obtain flame retardancy and the authors suggested that there was a high probability that recycled plastic fractions containing flame retardants, were used in the production of these articles. Although concentration of the individual substances were not determined, the authors also stated, "The obtained data show that in some cases DBDPE as the newer replacement for decaBDE was found in the samples." (Puype et al. 2015).

In Asia, other studies have detected DBDPE in many manufactured items including electronic components (television casings, computer monitor displays and accessories), car interiors (plastic, seat polyurethane and textile coating), household items (curtains, wallpaper and building materials) and furniture (sofa, mattress, pillow and carpet padding). Across the studies, concentrations were highest in TV casings (ND – 268.23 µg/g). DBDPE was not detected in computer monitor casings nor in furniture, curtains, wallpaper or insulation boards (Chen et al. 2010b, Kajiwara et al. 2011). In a separate Japanese study, DBDPE blended with high-impact polystyrene plastic and DBDPE present in plastic casings of televisions did not degrade in natural sunlight over 244 days (experiment conducted in a temperature controlled glass room, in which tubes containing samples were rotated constantly over 14 hrs each day to achieve uniform light exposure, and sample tubes shaken once a week to ensure homogeneous sunlight exposure of the particles; Kajiwara et al. 2008). Exposure to DBDPE from direct contact with electronics is considered minimal. Any abrasion to the plastic surfaces during the service life of these items may result in DBDPE adhering to dust particles in the surrounding area which is accounted for through exposure estimates in dust (see section 10.1.1.3).

Although Chen et al. (2010b) reported DBDPE concentrations in textile coatings in car interiors in China, it was not detected in car seats nor in furniture upholstery in one recent study in Canada (Muchongong et al. 2014). DBDPE was reported for unspecified uses in motor vehicles, as well as in the manufacture of airbag textile in Canada (ECCC 2013-2014 Health Canada 2014a, b). Evidence based on limited studies in Europe and Asia indicate that time spent in a car may represent a source of exposure to DBDPE in dust. However, the upper bound daily intake of dust derived based on studies in residences is considered to account for potential variability in other indoor settings, such as vehicles (see section 10.1.1.2). Use of DBDPE in furniture textiles has not been

reported in North America. Current information indicates that the potential for exposure of the Canadian general population to DBDPE from furniture textile is low.

DBDPE was detected in different types of toys purchased in China (hard plastic, rubber/soft plastic, foam, textile and stuffed toys) with the highest concentrations measured in hard plastic toys. Concentrations are reported in Appendix E (Chen et al. 2009).[2] Since DBDPE use in Canada is in plastic, rubber and textile materials rather than foam (ECCC 2013-2014; Health Canada 2014a,b),the potential exposure to DBDPE from mouthing of hard plastic toys by 0.5- to 4-year old children was modelled.A migration rate of 0.00769 µg/cm^2 per minute, based on volunteers mouthing plastic toys or rubber (Chen et al. 2009), was used in an algorithm to estimate oral daily intake. The resulting upper-bounding estimate of exposure from the mouthing of hard plastic toys was 2.0×10^{-4} mg/kg body weight (kg-bw) per day (Appendix F). Although skin contact with toys or hand-to-mouth contact following play with toys may result in potential exposure to DBDPE, it is expected to be minimal based on DBDPE physico-chemical properties, and therefore it is not expected to contribute significantly to the overall exposure from toys.

Chen et al. (2009) conducted their own exposure estimates from mouthing of hard plastic toys using their own algorithms for the brominated flame retardants measured in the study. Their estimates for DBDPE (1.3×10^{-6} to 1.5×10^{-5} mg/kg-bw/day) are close to the estimate derived from the model used in this assessment.

10.1.3. Biomonitoring

Appendix G summarizes the reported concentrations of DBDPE in serum and plasma, and human hair.

DBDPE was not detected in the majority of serum samples collected from 102 adult nursing women in Sherbrooke, Quebec, during 2008-2009. DBDPE was detected in 6% of samples, with concentrations between 3.4 (LOD) and 123 ng/g lipid weight (lw) (Zhou et al. 2014). The authors of this study suggested that toxicokinetics and analytical methods are playing a role in the low detection frequencies of DBDPE in human samples. DBDPE was not detected in the serum of residents from North China (Zhu et al. 2009) nor in women from California, US (Petreas et al. 2012). In addition, DBDPE was not detected in the plasma or serum of residents from Sweden (Karlsson et al. 2007; Remberger et al. 2014).

[2] Concentrations of DBDPE and PBDEs were lowest in the textile and stuffed toys, and the authors stated, “It is likely that the BFRs in these toys were not additives but were a result of adsorption of the chemicals from the environment by the outer covering textiles or inner stuffing (PUF or floss) of the toys.”

The presence of DBDPE in human hair samples from South China was reported by Zheng et al. (2011), but is presumed to be from deposition of dust rather than an indication of sequestration in human hair. The authors showed that DBDPE concentrations in dust amongst these different areas followed the same spatial distribution as that in hair, and that there was a significant correlation of DBDPE concentrations ($r = 0.97$, $p = 0.03$) between dust samples and hair samples, indicating that dust was one of the primary exposure routes for DBDPE in hair (i.e., dust depositing in hair). In another study, lower concentrations of DBDPE were reported in the hair of pet dogs and cats in Pakistan (maximum of 17.6 ng/g), and DBDPE was not detected in the serum of these animals (Ali et al. 2013b)**.**

The low levels of DBDPE found in human serum is consistent with the expected low levels of exposure estimated using models. It is noted that different studies used variations in the gas chromatography-mass spectrometry (GC-MS) analytical methodology for detection of DBDPE in human serum/plasma and breast milk. For analysis of DBDPE in serum/plasma, Petreas et al. (2012) used GC-high resolution (RM) MS with the MS operating in electron impact ionization mode using multiple ion detection, Zhou et al. (2014) used GC-MS with the MS operating in electron capture negative chemical ionization (ECNI) mode using selective ion monitoring (SIM) [ion pairs m/z 890.5 and 892.6], whereas Zhu et al. (2009) and Karlsson et al. (2007) used GC-MS with ECNI using SIM [ion pairs m/z 79 and 81]. For analysis of breast milk (see Appendix G), Mannetje et al. (2013) used HRGC-HRMS with SIM using ion pairs (specific ions not stated in article), whereas Zhou et al. (2014) used GC-MS with ECNI using SIM and ion pairs. Zhou et al. (2014) and Mannetje et al. (2013) were the only studies that detected DBDPE in serum/breast milk. Due to the variations in analytical methodology amongst the available studies attempting to measure DBDPE in serum/plasma/breast milk, there is evidence to support the observation that biomonitoring results in low (or very low) detections of DBDPE in biological fluids.

10. 2 Health Effects Assessment[3]

A summary of the available health effects information for DBDPE is presented in Appendix H.

No classifications of the health effects of DBDPE by national or international regulatory agencies were identified.

No chronic or carcinogenicity studies on DBDPE were identified. Mutagenicity studies with DBDPE in *Salmonella typhimurium* (TA 98, TA 100, TA 1535, TA 1537, and TA 1538 strains) and *Escherichia coli* (WP2 uvrA strain) were negative when tested with or

without metabolic activation at concentrations ranging from 0 to 5000 µg/plate in DMSO (Stankowski 1988; San and Wagner 1991). No chromosomal aberrations were found in Chinese hamster lung (CHL) cells at concentrations from 78 to 625 µg/ml for 6 hours with or without metabolic activation (Putman and Morris 1992). An independent repeat assay conducted with DBDPE suspended in carboxymethyl cellulose at concentrations of 625 to 5000 µg/ml with or without metabolic activation were also negative (Putman and Morris 1992). No *in vivo* studies were identified. Therefore, based on the available information, DBDPE is not mutagenic *in vitro*.

In the absence of chronic or carcinogenicity study on DBDPE, Health Canada conducted an analysis of (Q)SAR model predictions for carcinogenicity. Results were inconclusive. However, the (Q)SAR model predictions supported the findings from *in vitro*assays that DBDPE is not genotoxic. More detailed information of (Q)SAR modelling on carcinogenicity and genotoxicity is presented in Appendix H.

The UK Environment Agency (EA) assessed DBDPE in 2007 (Environment Agency 2007) and indicated in their report that the absence of signs of carcinogenicity from repeated-dose studies, such as proliferative changes, and the lack of genotoxicity in the available data, would suggest that DBDPE is unlikely to be carcinogenic (Environment Agency 2007).

Several repeated dose studies have been identified. In a sub-chronic oral study, DBDPE was administered to male and female Sprague-Dawley rats (10 animals/sex/group) at dose levels of 0, 100, 320 and 1000 mg/kg/day by gavage in corn oil for 90 consecutive days. At the end of the 90 days, all animals were sacrificed except for 10 animals per sex from the control group and 10 animals per sex from the high-dose group which were kept alive for a recovery period of 28 days to determine the reversibility, persistence or delayed occurrence of toxic effects. Results of the study showed that no treatment-related clinical signs of systemic toxicity, ocular lesions, alterations in urinalysis, clinical chemistry or hematology values were observed in the treated or recovery groups. No biologically or toxicologically significant differences were observed in body weights, body weight gains, and food consumption. In the male exposed rats, there were statistically significant differences in mean absolute or relative liver weights at the 1000 mg/kg-bw/day dose level as well as low-grade liver changes, consisting of minimal to slight hepatocellular vacuolation (high-dose males) and minimal to slight centrilobular hepatocyte hypertrophy (high- and possibly mid-dose males). These liver weight and histomorphological changes were, however, resolved by the end of the 28-day recovery period. No treatment-related changes were found in the liver of female rats. No treatment-related histomorphological changes were present in any of the other tissues (40 organs were examined). Overall, a NOAEL of 1000 mg/kg-bw/day, the highest dose tested, was identified from this study (Margitich 1992; Hardy et al. 2002).

In a more recent study conducted by Wang et al. (2010), male SD rats were orally administered 100 mg/kg bw/day of DBDPE in corn oil for 90 days. No significant changes in body weight, liver and kidney weight were observed. However, DBDPE

induced changes in clinical parameters (suppression of aspartate aminotransferase (AST), alkaline phosphatase (ALP) and creatinine (Cr), an increase in total bile acids (TBA). The EFSA Panel on Contaminants in the Food Chain (CONTAM Panel) considered these effects to be toxicologically non-significant (EFSA 2012). The results of this study also indicated that DBDPE may induce pregnane X receptor (PXR)-dependent gene expression because CYP3A mRNA was slightly increased in the liver, but no arylhydrocarbon receptor (AhR)- nor constitutive androgen receptor (CAR)-dependent gene expression nor consequent possible adverse effects were observed (Wang et al. 2010; EFSA 2012). Banasik et al. (2011) commented on the findings of this study, and considered various changes to be natural adaptive responses.

One short-term oral study was identified where DBDPE was administered to SD rats (6 animals/sex/group) at dose levels of 0 to 1250 mg/kg/day by gavage in corn oil for 28 consecutive days. At 1250 mg/kg-bw/day, the highest dose tested, there were no effects on mortality, clinical signs, body weight, food consumption, body weight gain, hematology and serum chemistry values, urinalysis, ocular examinations, gross necropsy results, and light microscopy of selected tissues (adrenals, heart, kidneys, liver, mesenteric lymph node, parathyroids, spleen, and thyroid). A mild and reversible increase in relative liver weights in the high-dose females was observed without any histopathology. Also, no evidence of delayed or progressive effects was found in the 14-day recovery period (Margitich 1991).

The acute oral toxicity of DBDPE is considered to be low, based on the absence of mortality and any clinical signs of toxicity in SD rats administered 5000 mg/kg-bw DBDPE via gavage (Mallory 1988a). An acute dermal toxicity assay was performed using New Zealand White Rabbits where DBDPE was applied to the intact shaved dorsal skin at a dose of 2000 mg/kg-bw and covered for 24 hours. There were no mortalities and all animals gained weight (Mallory 1988b).

The developmental toxicity of DBDPE has been examined in rats and rabbits via the oral route. In a study conducted by Mercieca (1992a), groups of 25 mated female SD rats were administered 0, 125, 400 or 1250 mg/kg/day DBDPE suspended in corn oil by gavage on gestational day (GD) 6-15 and sacrificed on day 20. There were no indications of toxicity among the dams during gestation except for whitish coloured faeces; this is likely due to the large quantity of the test material in the faeces, observed in about half of the highest dose dams. All the animals survived until scheduled necropsy and the body weights and food consumption of the treated animals were comparable to those of the controls. There were no treatment-related effects on foetal weight, sex ratio and late resorption. There were no external or visceral abnormalities in offspring. However, a statistically significant increase in the number of litters with unossified hyoid and reduced ossification of the skull was observed at 400 mg/kg-bw/day. This observation was considered to be incidental by the author, as a similar increase was not seen at the top dose. A number of variations occurred to a similar extent in the control and treated animals. In this study, no developmental toxicity was observed in rats up to the highest dose tested of 1250 mg/kg/day (Mercieca, 1992a).

Similar examinations were conducted in rabbits. Groups of 20 previously artificially inseminated female New Zealand White Rabbits were administered 0, 125, 400 or 1,250 mg/kg-bw/day DBDPE suspended in 0.5% methylcellulose by gavage on GD 6-18. No treatment-related mortality or clinical signs of toxicity were seen in the dams. Abortion occurred in one animal in each of the groups treated with 125 and 400 mg/kg-bw/day and in two animals of the 1250 mg/kg-bw/day group. However, in view of the low incidence of this observation and given that rabbits are known to have a high spontaneous abortion rate, this finding is considered to be incidental. There were no treatment-related effects on foetal weight, sex ratio, and early or late resorption. No abnormalities related to treatment with DBDPE were seen. The only statistically significant difference was an increased number of litters with the 27th presacral vertebra at 1250 mg/kg-bw/day (9 litters compared to 4 in controls). However, given that this is a common finding in rabbits (the laboratory historical control range is 12.5-93.8%), it was not considered adverse. A low incidence of vascular abnormalities was observed in the mid- and high-dose groups. Enlarged aortic valve, bulbous aortic arch and poorly developed right ventricle were observed in one foetus from the 400 mg/kg-bw/day group. Malformation of the aortic arch and undeveloped right ventricle were seen in two foetuses from a single litter of the 1250 mg/kg-bw/day group. Based on these low incidences, the reported malformations were not considered to be indicative of a treatment-related effect. Overall, no maternal toxicity was observed in this study with any of the doses tested. There were no adverse developmental effects observed in rabbits treated with DBDPE at doses up to 1250 mg/kg-bw/day (Mercieca, 1992b).

Recently, Hardy et al. (2010) conducted a similar study both in rats and rabbits to evaluate potential embryotoxic and teratogenic effects of DBDPE. Animals were administered DBDPE via gavage at dosage levels of 0, 125, 400 or 1250 mg/kg-bw/day from GD 6 through 15 for rats and GD 6 through 18 for rabbits. All female rats and rabbits were sacrificed on day 20 or 29 respectively, and subjected to caesarean section. No treatment-related mortality, abortions or clinical signs of toxicity were observed during the study. Body weights, body weight gain and food consumption were not affected by treatment. No significant internal abnormalities were observed in either species on necropsy. Caesarean section parameters were comparable between control and treated group. No treatment-induced malformations or developmental variations occurred. These study results showed no evidence of maternal toxicity, developmental toxicity, or teratogenicity in rats or rabbits treated with DBDPE at dosage levels up to 1250 mg/kg-bw/day (Hardy et al. 2010).

No reproductive studies have been identified. However, there were no adverse effects on the reproductive organs (among the 40 organs examined) in the 90-day oral study in rats at doses up to 1000 mg/kg-bw/day (Margitich 1992; Hardy et al. 2002).

Information on the toxicokinetics of DBDPE indicated that DBDPE was poorly absorbed via the oral route. In a study cited in the UK assessment report, no radioactivity was detected in plasma, bile and urine of rats exposed to a single dose of 1000 mg/kg bw/day of ^{14}C-DBDPE suspended in corn oil at various intervals for up to 168 hours post-dosing (Environment Agency 2007).

In a recent study conducted by Black (2012), rats were administered DBDPE via gavage with a single dose of 100 mg/kg-bw/day of unlabeled and ^{14}C-labeled DBDPE in corn oil, and tissues, bile, feces and urine were assayed for radiochemical content. Up to 168 hours post dosing, 89% of radioactive content was recovered in the feces with none in urine. Radioactivity in tissues (adipose, kidney, liver, spleen and GI tract consisting of stomach, small and large intestine, and cecum (all with contents)) were generally below the limit of detection. No bile samples had increased levels of radioactivity compared to controls. In addition, levels of radioactive content in blood and plasma were below the limit of detection at all time points. The majority of radioactivity in the GI tract was found through 24 hours, with no radioactivity found by 72 hours post exposure. It was presumed to have been excreted in the feces (Black 2012).

10.3 Characterization of Risk to Human Health

No classifications of the health effects of DBDPE by national or international regulatory agencies were identified. No chronic or carcinogenicity studies using DBDPE were identified. On the basis of the available information regarding genotoxicity, DBDPE is not *genotoxic in vitro.*

No adverse effects were observed in rats exposed to DBDPE orally for 28 or 90 days, up to doses of 1250 or 1000 mg/kg bw/day, respectively. No reproductive studies were identified. In two separate developmental toxicity studies, no treatment related maternal effects were observed in rats and rabbits exposed to DBDPE via the oral route; and no malformations or developmental variations occurred in the offspring.

There were no adverse effects observed in experimental animals exposed to doses up to 1000 mg/kg bw/d in their diet in sub-chronic studies. There are seven orders of magnitude between this dose and the highest estimate of total daily intake of DBDPE from environmental media and food (0.000061 mg/kg-bw/day in infants aged 0-0.5 years).This margin of exposure is considered to be adequate to address uncertainties in the health effects and exposure databases. Limited biomonitoring data for DBDPE in adults in Canada (Zhou et al. 2014), the U.S. (Petreas et al. 2012), and other locations around the world (Remberger et al. 2014; Mannetje et al. 2013; Zhu et al. 2009; Karlsson et al. 2007) resulted in low detection frequency in serum, plasma or breast milk, which appears to support a low level of environmental exposure in the general population.

Consumer products, specifically children's toys, were identified as a potential source of exposure of young children to DBDPE. There is, however, seven orders of magnitude between the highest doses tested in short-term or chronic oral studies (1250 or 1000 mg/kg bw\day) associated with no adverse effects in experimental animals, and the estimate of exposure (2.0×10^{-5}mg/kg-bw) for children aged 0.5-4 years mouthing hard plastic toys. This margin of exposure is considered adequate to address uncertainties in the health effects and exposure databases.

10.4 Uncertainties in Evaluation of Risk to Human Health

There are uncertainties associated with the estimate of human exposure to DBDPE from environmental media, because it is a difficult brominated flame retardant to analyze, and uncertainty can reach 40-60%, depending on the internal standard used for quantification (2014 communication from A. Covaci to Environment Canada, unreferenced). Although studies selected to derive exposure estimates were considered to be relevant for Canada, most were non-Canadian and the data from Canadian locations was limited (e.g., surface water concentrations as a surrogate for drinking water concentrations; breast milk concentrations sampled from one urban population). However, the estimates of exposure from environmental media were based on conservative assumptions.

There are also uncertainties associated with the estimate of exposure to consumer products, including uncertainties associated with the assumptions used in the model for estimating exposure to DBDPE via mouthing. The experimental migration rate for transfer of DBDPE from toys to the mouth was based on the highest rate observed in 5 volunteers and not an average rate. However, there is confidence that exposure was not underestimated. In addition, although limited, the biomonitoring data appears to support the expected low levels of environmental exposure of the general population.

There is an uncertainty associated with the assessment of carcinogenic and reproductive effects of DBDPE because of the absence of chronic and carcinogenic studies and reproductive toxicity studies. However, the collective evidence indicates that this substance is unlikely to be mutagenic; there were no adverse effects observed at the highest dose tested in the available short-term and sub-chronic studies, and large margins of exposure were determined for non-cancer effects.

11. Conclusion

Considering all available lines of evidence presented in this draft screening assessment, there is risk of harm to organisms, but not to the broader integrity of the environment from DBDPE. It is proposed to conclude that DBDPE meets the criteria under paragraph 64(*a*) of CEPA 1999 as it is entering or may enter 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. However, it is proposed to conclude that DBDPE does not meet the criteria under paragraph 64(*b*) of CEPA 1999 as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger to the environment on which life depends.

DBDPE is proposed to meet the Persistence criteria but does not meet the Bioaccumulation criteria as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000). However, DBDPE may contribute to the formation of persistent,

bioaccumulative, and inherently toxic transformation products, such as lower brominated BDPEs, in the environment.

On the basis of the adequacy of the margin between estimates of exposure from environmental media or consumer products and the highest doses tested in experimental animals, with no treatment related effects for subchronic exposure, it is proposed that DBDPE does not meet the criteria under paragraph 64(*c*) of CEPA 1999 as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

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Appendices

Appendix A: Structural Identity

Table A-1. Other selected names for DBDPE

CAS RN	Other selected names[a]
84852-53-9	Benzene, 1,1'-(1,2-ethanediyl)bis[2,3,4,5,6-pentabromo- (TSCA, ASIA-PAC, NZIoC) 1,1'-(ethane-1,2-diyl)bis[pentabromobenzene] (EINECS) 1,2-Bis(2,3,4,5,6-pentabromophenyl)ethane;1,2-Bis(pentabromophenyl)ethane; Decabromodiphenylethane;Decabromodiphenylethylene Decadiphenyl 8010; Ethylenebis(pentabromobiphenyl); Ethylenebispentabromobenzene; 1,2,3,4,5-pentabromo-6-[2-(2,3,4,5,6-pentabromophenyl)ethyl]benzene; 2,2',3,3',4,4',5,5',6,6'-Decabromobibenzyl; Ethane 1,2-bis(pentabromophenyl); BDPE-209; DBDiPhEtb;DBDE; EBPE; DeBrPylE FCP 801; Firemaster 2100; Firemaster 2100C; Planelon BDE; RDT 3; S 8010; SAYTEX 8010; CG 801; PBB-209; SLFR-2; SAYTEX 4010 Flame Retardant; SAYTEX 4010 ZD; SAYTEX 402 Flame Retardant (no longer marketed); SAYTEX 8010 Flame Retardant; SAYTEX 8010 ZD; Netguard 8010; NNN® Br-971, Ecoflame B-971, YCFR-03, DBDPE/RDT-3, FR-1410

[a] Names acquired from the National Chemical Inventories (NCI 2009), ECHA (c. 2007-2013) (accessed March 6, 2014), Bergman et al. 2012, etc.

Selection of Analogues

Decabromodiphenyl ether (decaBDE) represents a close structural analogue, and is considered appropriate for certain physical-chemical properties. DecaBDE, is discussed throughout the assessment in comparisons of substance behaviour with DBDPE (e.g., degradation, long-range transport, bioaccumulation potential, ecotoxicity etc.). However, it is noted that differences in molecular makeup, dimensions, and configurations exist that may affect the manner in which molecules interact with their environment (2014 manufacturer communication to Environment Canada; unreferenced). For example, according to a chemical manufacturer, DecaBDE exists as a single 3-diminsional conformer, arranged in space such that the aromatic rings are orthogonal (approximately perpendicular) to one another with a 120° bend at the oxygen bridge, whereas DBDPEs ethane bridge creates enough separation between the two fully substituted aromatic rings that the molecule can assume several 3-diminsional configurations, each with its own molecular dimensions. DBDPE's most stable conformer is folded at an acute angle at the ethane bridge resulting in a shorter molecular length than DecaBDE. Nevertheless, DBDPE molecular

volume, surface area and cross sectional diameter are larger than DecaBDE (2014 manufacturer communication to Environment Canada; unreferenced).

Appendix B: Physical and Chemical Properties

Some modelled K_{ow} and water solubility values for DBDPE were determined using the experimental value adjustment (EVA) option in KOWWIN 2010. This approach estimates K_{ow} and/or water solubility for a queried chemical (i.e., DBDPE) by comparing its structure to that of an analogue chemical that has an empirical value (i.e., decaBDE). The empirical value for the analogue is quantitatively adjusted based on the influence that structural differences have on K_{ow} and/or water solubility when the two chemicals are compared. Direct read across of the empirical values of analogue decaDBE were also considered for water solubility, logK_{ow}, and vapour pressure. In comparing the two substances, DBDPE's ethane bridge between the aromatic rings (rather than the ether bridge in decaBDE) is expected to make it slightly more hydrophobic than of decaBDE, and is expected to introduce more conformational flexibility in the molecule (Covaci et al. 2011).

Physical and chemical properties of DBDPE were checked for internal consistency according to the Least-Squares Adjustment Procedure (LSA) (Schenker et al. 2005). To conduct this, the geometric mean or arithmetic mean of available values for each physical and chemical parameter (vapour pressure, water solubility, octanol solubility, log K_{ow}, log K_{oa}, log K_{aw}) was entered into the model. Sub-cooled values were used for vapour pressure, water solubility, and octanol solubility. The values used to determine the geo and aritmentic (for log partition coefficients) means represent the most reliable and independent values available from empirical data, modelling, and analogues (Table B-1; for all physical-chemical values see Table B-2). In determining internal consistency of the properties, the LSA model also produces predicted values. Generally, DBDPE is characterized by very low water solubility, low to very low vapour pressure, and very high K_{oc} and K_{ow} values. While experimental based estimates for log K_{ow}, water solubility, and vapour pressure exist for DBDPE, there remains uncertainty with these values (e.g. Stieger 2014). For the purposes of this assessment, the log K_{ow} value of 9.89, representing the geomentric mean value checked with least squares adjustment method, was selected. This value is slightly higher than that of close analogue, decaBDE (8.7) (Environment Canada 2010), but slightly lower than the value determined from the EVA method using analogue decaBDE (10.23). To maintain internal consistency of physical chemical values, the least squares adjustment method value for water solubility and vapour pressure were also considered. Based on the large percent (%) adjustment of the octanol solubility value after checking with LSA, it is likely that this original input value is greatly underestimated. Were no value included for this variable in the LSA, the resulting logK_{ow} would be >10. Final selected values are summarized in Table B-1.

Table B-1. Physical Chemical Value Inputs for Least Squares Adjustment Model

Data Source	Vapour Pressure (Pa)	Water Solubility (mol/m^3)	Octanol Solubility (mol/m^3)	Log K_{ow}	Log K_{aw}	Log K_{oa}
Experimental DBDPE	1.00×10^{-4}	7.41×10^{-7}	1.96×10^{-3}[i]	-	-	-
Experimental DBDPE	-	-	8.75×10^{-4}[i]	-	-	-
Read Across decaBDE	4.63×10^{-6}	1.04×10^{-7}	-	8.7	-	-
EVA decaBDE	-	4.23×10^{-9}	-	10.23	-	-
Episuite Model (no inputs)	2.11×10^{-12}	1.19×10^{-15}[a]	-	13.64	-5.58	19.22[c]
Episuite Model	-	1.0×10^{-9}[b]	-	-	-	14.28[d]
Episuite Model	-	-	-	-	-	15.81[e]
Episuite Model	-	-	-	-	-	13.44[f]
VCC/AGLOGS	-	2.21×10^{-5}	-	7.86	-	-
ACD/Percepta	-	5.0×10^{-9}	-	10.63	-	-
Geomean/ Mean[g] LSA Input Values (sub-cooled value)	**9.92×10^{-8}** (1.46×10^{-4})	**1.72×10^{-9}**[h] ($2.52E-6$)	**0.00131** (1.92)[i]	**10.21**	**-5.58**	**15.69**[h]
LSA Output Values (sub-cooled value)	**5.59×10^{-10}** (8.21×10^{-7})	**8.34×10^{-9}** (1.22×10^{-5})	**64.1** (9.41×10^{4})	**9.89**	**-4.57**	**14.45**
% value adjustment	-99%	385%	4.88×10^{6}%	53%	929%	-94%

[a] WSKOWWIN 2010.
[b] WATERNT 2010 (fragment method).
[c] KoaWin 2010 using $\log K_{ow}$ 13.64 from KOWWIN (no inputs).
[d] KoaWin 2010 using $\log K_{ow}$ 8.7.
[e] KoaWin 2010 using $\log K_{ow}$ 10.23 from EVA decaBDE).
[f] KoaWin 2010 using $\log K_{ow}$ 7.86.
[g] Calculated arithmetic mean for logged values is equivalent to geometric mean for antilog values (of partition coefficients). In LSA, all input values =default 1 for estimated variance, except octanol solubility=3, due to high uncertainty with octanol solubility data.
[h] In order to maximize independence of parameter estimates, model values reliant on user $\log k_{ow}$ value (e.g., water solubility from WSKOWWIN, other than default) were not included in the

calculation of water solubility geomean. However, KoaWin was run with all logK_{ow} values, as the model was the only source of Koa data (to avoid skewing K_{oa} towards a single logK_{ow} value).
[i] All data sources in Table B-2 (note unit conversions), other than octanol solubility [personal communication, Manufacturer unpublished report, presented to Ecological Assessment Division (Environment Canada), 2013; unreferenced]. Individual listed values are solid state values, however Water solubility, Vapour Pressure, and Octanol solubility inputs were "subcooled liquid values" (Shenker al. 2005) by dividing by fugacity ratio for DBDPE. The subcooled LSA Input and Output values are in brackets beneath the solid state values.

Table B-2. Detailed physical and chemical properties for DBDPE

Property	Type	Value[a]	Temperature (°C)	Reference
Physical state	Experimental	white/off-white powder	N/A	Albermarle 2008
Melting point (ºC)	Experimental	(>)345[§]	N/A	Chemtura 2005, Albermarle 2008
Melting point (ºC)	Experimental	351-355	N/A	Great Lakes 2003
Melting point (ºC)	Experimental	350	N/A	Albermarle 2001
Melting point (ºC)	Modelled	259.71	N/A	MPBPWIN 2010
Boiling point (ºC)	Experimental	Irrelevant; expected to degrade before boiling	N/A	Environment Agency 2007
Boiling point (ºC)	Modelled	600.86	N/A	MPBPWIN 2010
Density (kg/m^3)	Experimental	868 (0.868 g/mL)	25	Albemarle 2001
Density (kg/m^3)	Packed density	3250 (3.25 g/cm^3)	25	Albemarle 2001
Density (kg/m^3)	Aerated density	1760 (1.760 g/mL)	25	Albemarle 2001
Vapour pressure (Pa)	Experimental	<1 x 10^{-4} Pa	20	European Commission 2002, Van Hoven et al.

Property	Type	Value[a]	Temperature (°C)	Reference
				2002 (Spinning rotor gauge)
Vapour pressure (Pa)	Modelled	2.11×10^{-12} (1.58×10^{-14} mmHg)[c]	25	MPBPWIN 2010 (Modified Grain method)
Vapour pressure (Pa)	Modelled	1.35×10^{-11} (1.01×10^{-13} mmHg)	25	MPBPWIN 2010 (MacKay method)
Vapour pressure (Pa)	Modelled	2.85×10^{-16} (2.14×10^{-18} mmHg)[c]	25	MPBPWIN 2010 (Antoine method)
Vapour pressure (Pa)	Modelled	$\sim 1 \times 10^{-6}$	25	Environment Agency 2007
Vapour pressure (Pa)	Modelled	5.59×10^{-10} (subcooled liquid : 8.21×10^{-7})§	25	Least-Squares Adjustment Method (LSA) Schenker et al. 2005
Henry's Law constant ($Pa \cdot m^3/mol$)	Modelled	6.51×10^{-3}§ (6.42×10^{-8} atm m^3/mol) (log K_{aw}= -5.58)	25	HENRYWIN 2011 (Bond method)
Henry's Law constant ($Pa \cdot m^3/mol$)	Modelled	2.98×10^{-3} (2.94×10^{-8} atm m^3/mol)	25	HENRYWIN 2011 (Group Method)
Henry's Law constant ($Pa \cdot m^3/mol$)	Modelled	6.7×10^{-2} (6.16×10^{-9} atm m^3/mol), (log K_{aw}=-4.57)	25	(VP/WS[g]), LSA (Schenker et al. 2005)
Log K_{ow} (dimensionless)	Experimental/ Estimated	~3.55 (considered estimate based on uncertainty, not reliable)	25	Van Hoven et al. 1999b

Property	Type	Value[a]	Temperature (°C)	Reference
Log K_{ow} (dimensionless)	Modelled	13.64	25	KOWWIN 2010
Log K_{ow} (dimensionless)	Modelled	10.23[c]	25	KOWWIN 2010 Experimental Value Adjusted (EVA)
Log K_{ow} (dimensionless)	Modelled	7.86	25	ALOGPS 2.1 VCCLAB 2005
Log K_{ow} (dimensionless)	Modelled	10.63	25	ACD/Percepta 1997-2012
Log K_{ow} (dimensionless)	Modelled	9.89[g§]	25	LSA (Schenker et al. 2005)
Log K_{oc} (dimensionless)	Modelled	6.38 (MCI method)	25	KOCWIN 2010
Log K_{oc} (dimensionless)	Modelled	8.58 [d§] (K_{ow} method)	25	KOCWIN 2010
Log K_{oa} (dimensionless)	Modelled	19.22	25	KOAWIN 2010
Log K_{oa} (dimensionless)	Modelled	15.44[d§]	25	KOAWIN 2010
Log K_{oa} (dimensionless)	Modelled	14.45[g]	25	LSA (Schenker et al. 2005)
Water solubility (mg/L)	Experimental	~7.2 x 10^{-4} (~0.72 µg/L)	25	Van Hoven et al. 1999a (generator column)
Water solubility (mg/L)	Modelled	9.71 x 10^{-7} (fragment method)	25	WATERNT 2010
Water solubility (mg/L)	Modelled	2.16 x 10^{-8}[c]	25	WSKOWWIN 2010
Water solubility (mg/L)	Modelled	3.85 x 10^{-9}[d]	25	WSKOWWIN

Property	Type	Value[a]	Temperature (°C)	Reference
				2010
Water solubility (mg/L)	Modelled	7.34×10^{-10}[f]	25	WSKOWWIN 2010
Water solubility (mg/L)	Modelled	2.15×10^{-2}	25	ALOGPS 2.1 VCCLAB 2005
Water solubility (mg/L)	Modelled	4.11×10^{-6}[e]	25	WATERNT (EVA) 2010
Water solubility (mg/L)	Modelled	5.0×10^{-9}	25	ACD/Percepta 1997-2012
Water solubility (mg/L)	Modelled	8.10×10^{-6}[g§] (subcooled liquid: 1.19×10^{-2})	25	LSA (Schenker et al. 2005)

Abbreviations: log K_{ow} = octanol-water partition coefficient; log K_{oc} = organic carbon-water partition coefficient; log K_{oa} = octanol-air partition coefficient; pK_a = acid dissociation constant; N/A = not applicable.

[§] Indicates selected value for modelling.

[a] Values in parentheses represent the original ones as reported by the authors or as estimated by the models.

[b] Bulk Density.

[c] Used log K_{ow} = 8.7 (from read across decaBDE analogue).

[d] Used log K_{ow} = 9.89 (LSA).

[e] Used water solubility = 1.0×10^{-4} mg/L (max from decaBDE analogue (<1.0×10^{-4} mg/L)).

[f] Used log K_{ow} =10.23 (from EVA of decaBDE analogue log Kow).

[g] For Least Squares Method (LSA); see Table B-1 for list of input values used.

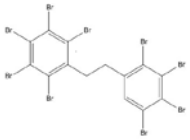
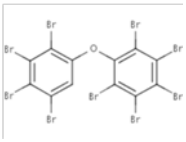
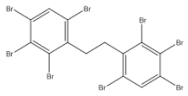
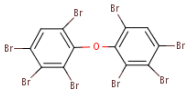
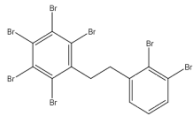
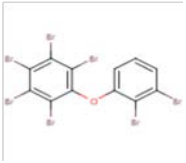
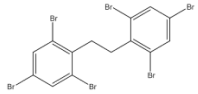
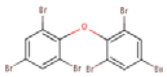
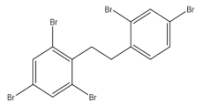
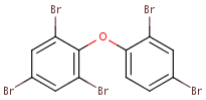
Table B-3. Summary of physical and chemical properties for DBDPE analogue: decaBDE

Property	Type	Value	Reference
Vapour pressure (Pa)	Experimental	4.63×10^{-6} (21 °C)	EC 2002
Vapour pressure (Pa)	Experimental	< 133.32(250 °C)	OECD 1994

Log K_{ow} dimensionless)	Experimental	8.7 (6.27–9.7	Environment Canada 2010, EC 2002 CMABFRIP 1997a
Log K_{ow} (dimensionless)	Experimental	6.265 (25 °C)	MacGregor and Nixon 1997
Water solubility (mg/L)	Experimental	0.02-0.03 (20-30 µg/L)<0.1 ug/L	OECD 1994

Appendix C: DBDPE Potential Transformation Products Modelling: Physical-Chemical Properties, Degradation, Bioaccumulation, and Aquatic Toxicity

Table C-1. Comparison of lower brominated diphenyl ethanes and lower brominated diphenyl ethers

BDPE Degradation Products[a]	MW (g)[b]	Log K_{ow}[b]	PBDEs[c]	MW (g)[d]	Log K_{ow}[e]
nonaBDPEs 	892.33	9.0	nonaBDEs 	880.4	7.8
octaBDPEs 	813.4	8.11	octaBDEs 	801.4	6.9
heptaBDPEs 	734.5	7.22	heptaBDEs 	722.3	6.0
hexaBPDEs 	655.6	6.33	hexaBDEs 	643.6	5.1
pentaBPDEs 	576.8	5.44	pentaBDEs 	564.7	4.3

[a] Smiles for nonaBDPEs from Catalogic (2012); remainder of BDPEs generated based on modifying PCBs congeners using: www.epa.gov/osw/hazard/tsd/pcbs/pubs/congenertable.pdf as structure source. Generated BDPE congener Smiles codes were verified in EPI drawing program.
[b] Estimated from EPISUITE models (2002-2012). Kow estimated by KOWWIN v 1.68 (2010) using the EVA method with a reference logKow of 9.89 for DBDPE.
[c] Selected from Chem ID Plus (accessed Dec 2014), to match BDPE congeners.
[d] Values from PBDE Screening Assessment (Environment Canada 2006).

[e] Values from Ecological State of the Science Decabromodipheny ether (Environment Canada 2010).

Table C-2. QSAR (EPISUITE 2000-2012) predicted physical-chemical properties for representative potential DBDPE transformation products considered in assessment

Degradation Product	MW (g/mol)	Log Kow[a]	Water solubility (mg/L)[b]	Smiles (representative)[c,d]
DBDPE	971.23	9.89	8.10×10^{-6}	c1(c(c(c(c(c1Br)CCc2c(c(c(c(c2Br)Br)Br)Br)Br)Br)Br)Br
nonaBDPEs	892.33	9.0	8.92×10^{-7}	Brc1cc(CCc2c(Br)c(Br)c(Br)c(Br)c2Br)c(Br)c(Br)c1Br
octaBDPEs	813.44	8.11	3.06×10^{-6}	BrC1=C(C(Br)=C(Br)C(Br)=C1)CCC2=C(Br)C=C(Br)C(Br)=C2Br
heptaBDPEs	734.54	7.22	1.63×10^{-5}	BrC1=C(C(Br)=C(Br)C(Br)=C1Br)CCC2=CC=CC(Br)=C2Br
hexaBPDEs	655.64	6.33	8.55×10^{-5}	BrC1=C(C(Br)=CC(Br)=C1)CCC2=C(Br)C=C(Br)C=C2Br
pentaBPDEs	576.75	5.44	4.4×10^{-4}	BrC1=C(C(Br)=CC(Br)=C1)CCC2=CC=C(Br)C=C2Br
Hydroxylated DBDPE	987.27	8.35	1.05×10^{-5}	OC(Cc1c(Br)c(Br)c(Br)c(Br)c1Br)c1c(Br)c(Br)c(Br)c(Br)c1Br
Hydroxylated nonaBPDEs	908.33	8.52	1.65×10^{-5}	Oc1c(Br)c(Br)c(CCc2c(Br)c(Br)c(Br)c(Br)c2Br)c(Br)c1Br
Brominated phenyl acids	940.33	4.83	2.33×10^{-4}	OC(=O)C(Br)=C(Br)C(Br)=C(CCc1c(Br)c(Br)c(Br)c(Br)c1Br)C(=O)Br

[a] EVA based on DBDPE log Kow value (KOWWIN 2010).
[b] Water solubility value from WATNT (2010) model, except DBDPE (see Table B-2)
[c] Smiles for nona and hydroxylated and brominated phenyl acids from Catalogic (2012); remainder of BDPEs generated based on modifying PCBs congeners using: www.epa.gov/osw/hazard/tsd/pcbs/pubs/congenertable.pdf as structure source. Generated BDPE congener Smiles codes were verified in EPI drawing program.
[d] Single smiles code selected for presentation purposes from list of several different Smiles per brominated species; however, QSAR modeling was run using representative smiles of an "extreme" configurations (unbalanced distribution of bromine atoms) and "balanced" configurations (balanced distribution of bromine atoms) for each congener. The only modelling differences reported among different bromine configurations for the same congener were for photolytic degradation (AOPWIN), but differences were minor.

Table C-3. Modelled degradation of potential transformation products.

Degradation Product	Log Kow[a]	Atmospheric oxidation (AOPWIN v. 1.92) predicted half-life	Biodegradation (Biowin v. 4.10 SubModels 3, 4, 5, 6) and extrapolated half-life	Ozone reaction (AOPWIN v.1.92) predicted half-life	Hydrolysis (HYDROWIN v. 2) predicted half-life
nonaBDPEs	9.0	4.27 days	'Does not biodegrade fast' to 'Recalcitrant'	n/a[b]	n/a[b]
octaBDPEs	8.11	4.093 days	Does not biodegrade fast' to 'Recalcitrant'	n/a[b]	n/a[b]
heptaBDPEs	7.22	3.925 days	Does not biodegrade fast' to 'Recalcitrant'	n/a[b]	n/a[b]
hexaBPDEs	6.33	3.071 days	Submodels, 3 5,6: Does not biodegrade fast' to 'Recalcitrant', submodel 4 (primary degradation): 'months'	n/a[b]	n/a[b]
pentaBPDEs	5.44	2.224 days[a]	Submodels 3 5,6: Does not biodegrade fast' to 'Recalcitrant', submodel 4 (primary degradation): 'months';	n/a[b]	n/a[b]
Hydroxylated DBDPE	8.35	1.098 days	Does not biodegrade fast' to 'Recalcitrant';	n/a[b]	n/a[b]
Hydroxylated nonaBPDEs	8.51	2.809 days	Does not biodegrade fast' to 'Recalcitrant';	n/a[b]	n/a[b]

Degradation Product	Log Kow[a]	Atmospheric oxidation (AOPWIN v. 1.92) predicted half-life	Biodegradation (Biowin v. 4.10 SubModels 3, 4, 5, 6) and extrapolated half-life	Ozone reaction (AOPWIN v.1.92) predicted half-life	Hydrolysis (HYDROWIN v. 2) predicted half-life
Brominated phenyl acids	4.83	2.955 days	Submodels, 3 5,6: Does not biodegrade fast' to 'Recalcitrant', submodel 4 (primary degradation): 'months';	79.323 days	Hydrolyzable Function detected: Acyl Halides; Acyl halides react readily (some violently) with water to yield the parent acid and hydrogen halide. Hydrolysis half-lives are less than 10 minutes (or faster).

[a] Depending on arrangement of Br atoms, pentaDBPE half-life in air ranges from <2 days (i.e. 1.5 days) to >2 days.
[b] Model does not provide an estimate for this type of structure.

Similar to parent DBDPE, it is expected the DBDPE transformation products are moderately well covered by the BIOWIN biodegradation models used to estimate degradation. However, there appears to be poor coverage of DBDPE transformation products by CPOPs (2012) models (most transformation products are <30% within structural domain). For this reason, biodegradation predictions from CPOPs/Catalogic are not included in the assessment for transformation products.

The BCF and BAF of potential DBDPE transformation products were estimated using both structure-based models and a three trophic level kinetic mass-balance model. All estimates of BCF and BAF, except sub-model 1 of the BCFBAF model in EPIWIN v4.1, were corrected for metabolism because it represents a fundamental elimination pathway for many chemicals. This correction was performed by deriving metabolism rate constants (k_M) using a structure-based QSAR method.

Most predicted DBDPE transformation products meet criteria to be considered "in domain" for the Arnot-Gobas mass-balance model; the substances meet the mechanistic and global parameter criteria of: being a neutral organic (except hydroxylated nonaBDPEs and brominated phenyl acids), having a molecular weight less than ~ 1200, as well as having a log K_{ow} <9 (except for nonBDPE).

There appears to be poor coverage of DBDPE transformation products by CPOPs models (most transformation products are <40% within structural domain). For this reason, bioaccumulation predictions from CPOPs/Catalogic are not included in the assessment.

Table C-4. Modelled bioconcentration factors (BCF) and bioaccumulation factors (BAF) for predicted transformation products of DBDPE

Degradation Product	MW (g)	Log K_{ow}[a]	Predicted BCF (no metabolism) (L/kg w-w)	Predicted BCF[b] Mid-trophic level fish (metabolism) (L/kg w-w)	Predicted BAF[b] Mid-trophic level fish (metabolism) (L/kg w-w)	Predicted kM[b] (1/day) (100 g fish, 15 degree C)
octaBDPEs	813.4	8.11	3803	466.8	165600	0.016
heptaBDPEs	734.5	7.22	108380	1790	17600	0.025
hexaBPDEs	655.6	6.33	6975	3510	44210	0.039
pentaBPDEs	576.8	5.44	1804	2756	5636	0.062
hydroxylated DBDPE	987.27	8.35	1610	98.31	16380	0.047
hydroxylated nonaBPDEs	908.3	8.52	2394	38.9	4042	0.083
brominated phenyl acids	940.3	4.83	3.16	693.9	738.5	0.271

[a] Estimated using KOWWIN v 1.68 (2010) using the EVA method with a reference log Kow of 9.89 for DBDPE. Note that nonaBDPE was not included since at log Kow=9, substance exceeds cut-off for training set chemicals in model.
[b] Estimated from BCFBAF v 3.01(2008).

Table C-5. Summary of modelled aquatic toxicity values for DBDPE transformation products in water[a]

Common name	Test organism	Endpoint	Value (mg/L)	Reference
nonaBDPEs	Fish, *Daphnia magna*, green algae, etc.	Acute and Chronic	NES	ECOSAR v. 1.11

octaBDPEs	Fish, *Daphnia magna*, green algae, etc.	Acute and Chronic	NES	ECOSAR v. 1.11
heptaBPDEs	*Daphnia magna*, Fish, Mysid saltwater	ChV	7.3×10^{-11} to 6.75×10^{-6} (NES for remaining tests)	ECOSAR v. 1.11
hexaBDPEs	*Daphnia magna*, Fish, Mysid saltwater, Green algae (acute and chronic)	ChV, 96 h EC50	9.72×10^{-10} to 0.00080 (NES for remaining tests)	ECOSAR v. 1.11
pentaBDPEs	Green Algae, *Daphnia magna*, Fish, Mysid saltwater	ChV, 96 h LC50	1.27×10^{-8} to 0.002 (NES for remaining tests)	ECOSAR v. 1.11
Hydroxylated DBDPE (Neutral Organic)	Fish, *Daphnia magna*, green algae	ChV	2.02×10^{-7} to 7.3×10^{-5} (NES for remaining tests)	ECOSAR v. 1.11
Hydroxylated DBDPE (Benzyl alcohols)	Fish, *Daphnia magna*	ChV	2.92×10^{-7} to 1.05×10^{-4} (NES for remaining tests)	ECOSAR v. 1.11
Hydroxylated nonaBDPEs (Neutral Organic /Phenols)	Fish, *Daphnia magna*, green algae, etc.	Acute and Chronic	NES	ECOSAR v. 1.11
Brominated phenyl acid (Halo Acids)	Fish	ChV	0.000186	ECOSAR v. 1.11

Brominated phenyl acid (Vinyl/Allyl Halides-acid)	Fish	ChV	$9.44x10^{-6}$	ECOSAR v. 1.11
Brominated phenyl acid (Neutral Organic)	Fish, *Daphnia magna*	ChV, 96 h LC50, 48 h LC_{50}	0.000191 to 0.000992	ECOSAR v. 1.11

[a] All values estimated from ECOSAR v 1.11 (2012) using physical-chemical properties in Table D-2.
[b] NES- No effects at saturation, ChV-Chronic value, LC- Lethal concentration.

Appendix D: Upper-bounding estimates of daily intake (ug/kg bw /d) of DBDPE by various age groups within the general population in Canada

Upper-bounding estimates of daily intake (ug/kg bw /d) of DBDPE by various age groups within the general population in Canada

Route of Exposure	0–6 mo[a](breast milk fed)[b]	0–6 mo[a] (formu-la fed)[c]	0–6 mo[a] (not formula fed)[d]	0.5–4 yr[e]	5–11 yr[f]	12–19 yr[g]	20–59 yr[h]	60+ yr[i]
Ambient air[j]	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Indoor air[k]	0.0002	0.0002	0.0002	0.0005	0.0004	0.0002	0.0002	0.0002
Drinking water[l]	N/A	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Food[m]	0.005	0.0004	0.0003	0.0007	0.0006	0.0003	0.0002	0.0002
Dust[n]	0.056	0.056	0.056	0.029	0.011	0.0004	0.0004	0.0004
Soil[o]	N/A	N/A	N/A	0.023	0.017	0.0006	0.0006	0.0005
Total intake	0.061	0.056	0.056	0.053	0.029	0.0015	0.0014	0.0013

Abbreviations: N/A = not applicable; mo = months; yr = years.

[a] Assumed to weigh 7.5 kg, to breathe 2.1 m^3 of air per day, to drink 0.8 L of water per day (formula fed) or 0.3 L/day (not formula fed, respectively (Health Canada 1998), and to ingest 38 and 0 mg of dust and soil per day, respectively (Wilson et al. 2013).

[b] Exclusively for breast milk-fed infants, assumed to consume 0.742 L of breast milk per day(Health Canada 1998), and breast milk is assumed to be the only dietary source. The concentration for whole (breast) milk of 0.049 ug/L was based on a reported 95th percentile of 0.0473 ng/g wet weight [ww] x 1.03 g/ml (density of breast milk) identified from 105 samples of human breast milk collected in 2008-2009 from adult nursing women in Sherbrooke, Quebec (Zhou et al. 2014; Zhou et al. 2014; personal communication from EHSRB, Health Canada, to ESRAB, Health Canada dated May 15, 2014).

[c] Exclusively for formula-fed infants, assumed to drink 0.8L of water per day (Health Canada 1998), where water is used to reconstitute formula. No monitoring data on DBDPE in formula were identified; therefore dietary intakes are only those from water. See footnote on water for details.

[d] Exclusively for not formula-fed infants, assumed to drink 0.7 L of water per day (Health Canada 1998), and approximately 50% of non-formula-fed infants are introduced to solid foods by 4 months of age, and 90% by 6 months of age (NHW 1990).

[e] Assumed to weigh 15.5 kg, to breathe 9.3 m^3 of air per day, to drink 0.7 L of water per day, to consume 54.7 g of fish per day, and 162.2 g of cereal products per day (Health Canada 1998), and to ingest 41 and 14 mg of dust and soil per day, respectively (Wilson et al. 2013).

[f] Assumed to weigh 31.0 kg, to breathe 14.5 m^3 of air per day, to drink 1.1 L of water per day, to consume 89.8 g of fish per day, and 290.1 g of cereal products per day (Health Canada 1998), and to ingest 31 and 21 mg of dust and soil per day, respectively (Wilson et al. 2013).

[g] Assumed to weigh 59.4 kg, to breathe 15.8 m^3 of air per day, to drink 1.2 L of water per day, to consume 97.3 g of fish per day, and 320.9 g of cereal products per day (Health Canada 1998), and to ingest 2.2 and 1.4 mg of dust and soil per day, respectively (Wilson et al. 2013).

[h] Assumed to weigh 70.9 kg, to breathe 16.2 m^3 of air per day, to drink 1.5 L of water per day, to consume 111.7 g of fish per day, and 248.4 g of cereal products per day (Health Canada 1998), and to ingest 2.5 and 1.6 mg of dust and soil per day, respectively (Wilson et al. 2013).

[l] Assumed to weigh 72.0 kg, to breathe 14.3 m^3 of air per day, to drink 1.6 L of water per day, to consume 72.9 g of fish per day, and 229.0 g of cereal products per day (Health Canada 1998), and to ingest 2.5 and 1.5 mg of dust and soil per day, respectively (Wilson et al. 2013).
[j] No monitoring data of ambient air in Canada were identified. The 95th percentile for Cleveland, Ohio (22 + 1.96 x 13 pg/m^3 = 47.5 pg/m^3) was used for deriving upper-bounding estimates of daily intake for ambient air exposure. Concentration of DBDPE from this urban location was considered to be representative for Canada. Canadians are assumed to spend 3 hours outdoors each day (Health Canada 1998).
[k] No monitoring data of indoor air in Canada were identified. The maximum concentration of DBDPE (0.963 ng/m^3) measured in indoor air samples from houses in Norway (Cequier et al. 2014), was selected for deriving upper-bounding estimates of daily intake for indoor air exposure. Canadians are assumed to spend 21 hours indoors each day (Health Canada 1998).
[l] No monitoring data of drinking water in Canada were identified. The highest mean concentrations of DBDPE (10.8 pg/L), measured in Lake Ontario (Venier et al. 2014), was selected for deriving upper-bounding estimates of daily intake for drinking water exposure.
[m] No monitoring data on marketed foods in Canada were identified; however, data on two baby food categories were identified for samples collected in the U.S (Liu et al. 2014). DBDPE maximum concentrations in formula (28.6 pg/g ww) and cereal (48.8 pg/g ww) from the U.S. were selected for deriving upper-bounding estimates of daily intake for exposure to infant formula and cereal products, respectively. Environmental fish data were also available. The highest concentration on a fresh weight basis of 0.44 ug/kg ("fresh" or "wet" weight) based on 1/2 MDL (MDL = 0.1 ng/g lw) X 8.78% lipid in Whitefish from Lake Winnipeg (Law et al. 2006) was selected for deriving upper-bounding estimates of daily intake for exposure to all fish-related food items in the fish food group. Amounts of foods consumed on a daily basis by each age group over 12 food groups were obtained from the 1970–1972 Nutrition Canada Survey (Health Canada 1998).
[n] The maximum concentration of DBDPE (11070 ng/g dust) from 20 homes in Boston, Massachusetts (Stapleton et al. 2008) was selected for deriving upper-bounding estimates of daily intake for dust exposure.
[o] No monitoring data of soil in North America were identified. Therefore, the maximum soil predicted environmental concentration (PEC) of 25.3 µg/g dw (25.3 mg/kg dw) was selected for deriving upper-bounding estimates of daily intake for soil exposure.

Appendix E: DBDPE Concentrations in Consumer Products.

Table E-1. DBDPE concentrations in consumer products

Product Category	Product Type	Sample Size	Limit of Detection	Concentration	Reference
Toys	Foam toy	7	0.3% wt. in sample	ND	Health Canada 2014a, b
Toys	Plastic Toy	2	0.3% wt. in sample	ND	Health Canada 2014a, b
Toys	Foam chair (children's)	1	0.3% wt. in sample	ND	Health Canada 2014a, b
Toys	Textile Playtent	2	0.3% wt. in sample	≥ 0.5% wt. in sample (≥ LOQ)	Health Canada 2014a, b
Furniture	Nursing pillow	2	0.3% wt. in sample	ND	Health Canada 2014a, b
Furniture	Crib mattress	1	0.3% wt. in sample	ND	Health Canada 2014a, b
Furniture	Polyurethane foam (PUF) upholstery–cars	18	Not determined (signal-to-noise ratio of 3 used instead)	ND	Mochungong et al. 2014
Furniture	PUF upholstery – home	9	Not determined (signal-to-noise ratio of 3 used instead)	ND	Mochungong et al. 2014
Furniture	Fabric upholstery – cars	12	Not determined (signal-to-noise ratio of 3 used instead)	ND	Mochungong et al. 2014
Furniture	Fabric upholstery – home	2	Not determined (signal-to-noise ratio of 3 used	ND	Mochungong et al. 2014

Product Category	Product Type	Sample Size	Limit of Detection	Concentration	Reference
			instead)		
Other Objects	Rubber/ plastic items	5	0.3% wt. in sample	ND	Health Canada 2014a, b
Other Objects	Purple textile	1	0.3% wt. in sample	ND	Health Canada 2014a, b
Other Objects	White foam	1	0.3% wt. in sample	ND	Health Canada 2014a, b
Other Objects	Unknown composition (picture)	1	0.3% wt. in sample	ND	Health Canada 2014a, b
Toys	Hard plastic toy	30 (80% detection frequency)	"...ranged from 0.00005 to 0.050 µg/g."	ND-117 µg/g; Mean = 9.17; Median = 5.54	Chen et al. 2009
Toys	Foam toy	18 (89% detection frequency)	"...ranged from 0.00005 to 0.050 µg/g."	ND-5.69 µg/g; Mean = 1.32; Median = 0.72	Chen et al. 2009
Toys	Rubber/Soft plastic toy	15 (40% detection frequency)	"...ranged from 0.00005 to 0.050 µg/g."	ND-7.55 µg/g; Mean = 1.14; Median = ND	Chen et al. 2009
Toys	Stuffed toy	6 (0% or 50% detection frequency - See NOTE)	"...ranged from 0.00005 to 0.050 µg/g."	ND (or ND-0.26 µg/g); See NOTE.	Chen et al. 2009 (NOTE: Supplementary tables for Chen et al. (2009) provided conflicting info: Table S2 shows a range of ND-0.26 µg/g, whereas Table S5 shows ND for

Product Category	Product Type	Sample Size	Limit of Detection	Concentration	Reference
					all 6 samples).
TVs	TV Casing	20	"…ranged from 0.5 to 25 ng/g" (0.0005 to 0.0025 µg/g)	ND-268.23 µg/g; Mean = 30.15 µg/g; (33.3% detection frequency)	Chen et al. 2010b
Computers	Computer display casing	4	"…ranged from 0.5 to 25 ng/g" (0.0005 to 0.0025 µg/g)	ND	Chen et al. 2010b
Computers	Computer component (computer printed circuit boards (n = 4) and computer accessories (key board and mouse, n = 4))	8	"…ranged from 0.5 to 25 ng/g" (0.0005 to 0.0025 µg/g)	ND-66.02 µg/g; Mean = 15.06 µg/g; (50% detection frequency	Chen et al. 2010b
Computers	Computer monitor casing	13	Not determined (signal-to-noise ratio of 3 used instead)	ND-0.1 mg/g	Mochungong et al. 2014
Computers	Computer accessories casing	11	Not determined (signal-to-noise ratio of 3 used instead)	ND	Mochungong et al. 2014
Cars	Car interior (plastic interiors and seat PUF and textile	5	"…ranged from 0.5 to 25 ng/g" (0.0005 to 0.0025	ND-1.41 µg/g; Mean = 0.28 µg/g; (20% detection frequency)	Chen et al. 2010b

Product Category	Product Type	Sample Size	Limit of Detection	Concentration	Reference
	coating)		µg/g)		
Furniture	Sofa, mattress, pillow, and carpet padding	7	"…ranged from 0.5 to 25 ng/g" (0.0005 to 0.0025 µg/g)	ND	Chen et al. 2010 b
LCD TV	Rear Plastic Cover	2	NS	NA, 130 µg/g	Kajiwara et al. 2011
LCD TV	Front Plastic Cover	2	NS	NA, 92 µg/g	Kajiwara et al. 2011
LCD TV	Power Board	1	NS	1.1 µg/g	Kajiwara et al. 2011
LCD TV	Printed Circuit (PC) board for fluorescent tube (and power supply)	2	NS	0.77, 2.4 µ/g	Kajiwara et al. 2011
LCD TV	Other PC boards	2	NS	0.036, 0.38 µg/g	Kajiwara et al. 2011
LCD TV	LCD Panel	2	NS	NA	Kajiwara et al. 2011
Laptop Computer	Chassis	1	NS	0.67 µg/g	Kajiwara et al. 2011
	Keyboard top	1	NS	NA	Kajiwara et al. 2011
	PC boards	1	NS	0.037 µg/g	Kajiwara et al. 2011
	Cooling fan and speaker	1	NS	19 µg/g	Kajiwara et al. 2011
	AC adapter	1	NS	NA	Kajiwara et al. 2011
	LCD Panel	1	NS	NA	Kajiwara et al. 2011
Textile	Curtain	2	NS	NA	Kajiwara et al. 2011
Electrical Outlet	Electrical Outlet	2	NS	0.32, 6.1 µg/g	Kajiwara et al. 2011
Insulation board	Extruded polystyrene	2	NS	NA	Kajiwara et al. 2011

Product Category	Product Type	Sample Size	Limit of Detection	Concentration	Reference
	insulation board				
Wallpaper	PVC Wallpaper	4	NS	NA	Kajiwara et al. 2011
TV Casing	TV Casing (Hard impact polystyrene [HIPS] plastic)	3 (apparently, each prepared from 50 used TV casings)	50 ng/g	Mean = 140 ± 5.7 µg/g	Kajiwara et al. 2008
TV Casing	TV Monitor casing	15	Not determined (signal-to-noise ratio of 3 used instead)	ND-0.5 mg/g; Mean = 0.0 ± 0.1 mg/g	Mochungong et al. 2014
Audio-visual Polymer Casings	"Audiovisual"	8	Not determined (signal-to-noise ratio of 3 used instead)	ND-0.5 mg/g; Mean = 0.0 ± 0.1 mg/g	Mochungong et al. 2014

Abbreviations: ND = not detected; NA = No data available; NS = Not stated

Appendix F: Consumer Product Exposure Estimates

$$\text{Intake} = \frac{SA \times M \times ED}{BW}$$

Table F-1. Oral mouthing of hard plastic toys by children aged 0.5-4 years

Symbol	Description	Value
SA[a]	Surface area of direct mouthing	10 cm^2
M[b]	Migration rate	0.00769 µg/cm^2 per min
ED[c]	Exposure duration	39 min/d
BW[d]	Body weight	15.5 kg (Toddler)
Intake	Intake	1.5 ×10^{-5} mg/kg bw/d = 1.93 × 10^{-4} mg/kg-bw/day

[a] The contact area assumed for mouthing a plastic doll in a 7.5 month old child (Bremmer and van Veen 2002).
[b] Migration rate was measured *in vivo* using 5 volunteers who mouthed small pieces from a rubber and hard plastic toy for 15 or 30 min and then collecting saliva samples from each volunteer every 5 min for processing and analysis of flame retardants (Chen et al. 2009).
[c] Upper bound of the mean for children aged 0.5-4 years mouthing toys (Norris and Smith 2002 cited in US EPA 2011).
[d] Health Canada (1998).

Appendix G: Levels of DBDPE in Human Tissue

Table G-1. Levels of DBDPE in human tissue

Tissue type and Location	Sampling Period	Number of Samples	Limit of Detection (ng/g)	Mean Concentration (ng/g)	Reference
Breast Milk Sherbrooke, Quebec	2008-2009	105 adult nursing women	1.7 ng/g lipid weight [lw]; 8.6% d.f.[a]	Median: ND[b] 95th percentile: 3.3 [ND – 25]	Zhou et al. 2014.
Breast Milk New Zealand, 2 urban areas plus 2 rural areas	2007-2010	36 first time mothers (mean age – 26.9)	227 pg/g	113.5 pg/g: below LOD [ND – 325.50]	Mannetje et al. 2013
Serum	2008-2009	102 adult nursing women	3.5 ng/g	Median: ND	Zhou et al. 2014.

Tissue type and Location	Sampling Period	Number of Samples	Limit of Detection (ng/g)	Mean Concentration (ng/g)	Reference
Sherbrooke, Quebec			lw; 5.9% d.f.	95th percentile: 3.40 [ND – 123]	
Serum Tianjin, China	2006	128 (30 office cleaners, 69 university students, 29 policemen)	15	ND	Zhu et al. 2009
Serum California, US	1996-98; 2008-09	1996-98: 50 samples from Laotian immigrant women to San Francisco; 2008-09: 30 samples from adult women and 25 samples from pregnant women in California.	NS[c]	ND or at levels below quantitation	Petreas et al. 2012
Plasma Multiple cities, Sweden	NS	5	1.03 ng/g lipid weight	ND	Karlsson et al. 2007
Hair Guangzhou City, China	NS	29	LOQ[d]: 2.01	Median: 17.8 [6.05 – 88.7]	Zheng et al. 2011

Tissue type and Location	Sampling Period	Number of Samples	Limit of Detection (ng/g)	Mean Concentration (ng/g)	Reference
Hair Yuantan Town, China	NS	32	LOQ: 2.01	Median: 9.57 [2.32 – 128]	Zheng et al. 2011

[a] d.f. = detection frequency
[b] ND = not detected
[c] NS = not specified
[d] LOQ = limit of quantitation

Appendix H: Summary of health effects information for Decabromodiphenyl ethane (DBDPE) CAS RN 84852-53-9

Table H-1: Summary of health effects information for Decabromodiphenyl ethane (DBDPE) CAS RN 84852-53-9

Endpoint	Lowest effect levels[1]/results
Animal Studies	
Acute toxicity	**Lowest oral LD_{50}(rat)**> 5000 mg/kg (Mallory 1988a cited in Environment Agency 2007). **Lowest dermal LD_{50} (rabbit, 24hrs)** >2000 mg/kg (Mallory 1988b cited in Environment Agency 2007). **LOAEC (inhalation, rats)** = 50 mg/L(no more detailed information were available)(Li et al., 2004 cited in Norwegian Pollution Control Authority (SFT) 2008)
Short-term repeated-dose toxicity	**NOAEL (oral, rat) = 1250 mg/kg bw/day** based on an absence of effects on mortality, clinical signs, body weight, food consumption, body weight gain, hematology and serum chemistry values, urinalysis, ocular examinations, gross necropsy results, organ weights, and light microscopy of selected tissues (adrenals, heart, kidneys, liver, mesenteric lymph node, parathyroids, spleen, and thyroid). DBDPE was administered to Sprague-Dawley (SD) rats (6 animals/sex/group) at dose levels of 0, 125, 400 and 1250 mg/kg/day by gavage in corn oil for 28 consecutive days. A mild and reversible increase in relative liver weights in the high-dose females was observed without any histopathology. Further, a 14-day recovery period after administration of 1250 mg/kg/day for 28 days found no evidence of delayed or progressive effects (Margitich 1991cited in Environment Agency 2007). No inhalation and dermal studies were identified.

Endpoint	Lowest effect levels[1]/results
Animal Studies	
Subchronic toxicity	**NOAEL (oral, rat) = 1000 mg/kg bw/day.** DBDPE was administered to male and female Sprague-Dawley rats (10 animals/sex/group) at dose levels of 0, 100, 320 and 1000 mg/kg/day by gavage in corn oil for 90 consecutive days. Randomly selected animals (10 animals/sex/group) from the vehicle control and high-dose recovery groups remained on test, untreated, for an additional 28 days to determine the reversibility, persistence, or delayed occurrence of toxic effects. DBDPE exposure produced no compound-related clinical signs of systemic toxicity, ocular lesions, or alterations in urinalysis, clinical chemistry, and hematology values in the treated or recovery groups (at 30 and 90 days). No biologically or toxicologically significant differences were observed in body weights, body weight gains, and food consumption. There were statistically significant differences in mean absolute or relative liver weights at 1000 mg/kg-bw/day. There were also low-grade liver changes consisting of minimal to slight hepatocellular vacuolation (high-dose males) and minimal to slight centrilobular hepatomegaly (high- and possibly mid-dose males) observed. But these effects were not seen after a 28-day recovery, which indicated an adaptive and reversible response. Authors, therefore, suggested a **NOAEL of 1000 mg/kg bw/day.** No treatment-related changes were found in the livers of female rats. No treatment-related histomorphological changes were present in any of the other tissues (40 organs total) examined in either sex, except for evidence of aspirated test article in individual rats (Margitich 1992 cited in Environment Agency 2007; Hardy et al. 2002). **<u>Other study:</u>** **LOEL (oral, rat) = 100 mg/kg bw/day** based on significant alterations in serum creatinine (Cr) and total bile acid (TBA) levels, as well as aspartate aminotransferase (AST) and alkaline phosphatase (ALP) at this dose level. Male Sprague-Dawley rats (12 per group) were administered DBDPE orally in corn oil for 90 days. Thyroid hormone T3 levels were significantly increased in the exposed rats, but T4 levels did not appear to be significantly altered. Biochemical parameters, including thyroid hormone levels, 13 clinical chemistry parameters, and the mRNA expression levels of certain enzymes were also monitored from 6 animals per group. Along with the above mentioned biochemical effects, CYP3A2 liver mRNA was also significantly up-regulated at this dose level, but CYP1A1, CYP2B1, and CYP2B2 mRNA were not affected. There were, however, no significant changes in body, liver, or kidney (relative and absolute) weights (Wang et al. 2010). The results of this study were challenged by other scientists in the public literature (Banasik et al. 2011). No inhalation and dermal studies were identified.
Chronic toxicity/ Carcinogenic	No empirical studies were available.

Endpoint	Lowest effect levels[1]/results
Animal Studies	
ity	**(Q)SAR models:** In the absence of chronic studies on DBDPE the information on its carcinogenicity potential was obtained from (Q)SAR models. For the majority of models such as CASE Ultra Tox, CAESAR and ACD Percepta the predictions obtained on DBDPE were inconclusive. One of the rodent carcinogenicity models, Leadscope Model Applier, which is built on a statistical algorithm, predicted absence of activity. No structural-alerts were flagged by the (Q)SAR model, Toxtree, for potential genotoxic and non-genotoxic carcinogenicity. On the other hand, DEREK Nexus, an Expert System, predicted positive carcinogenicity based on a non-genotoxic pathway. In order to confirm if any metabolites of DBDPE could exert carcinogenic activity it was subjected to rat *in vitro*liver metabolism simulator using the Toolbox. None of the metabolites were flagged for the presence of structural alerts for potential non-genotoxic carcinogenicity. Majority of rodent carcinogenicity (Q)SAR models except DEREK Nexus predicted lack of carcinogenic potential for the metabolites. The prediction by DEREK Nexus was again based on the non-genotoxic pathway typically found in polyhalogenated aromatics. Metabolites that were flagged for the presence of structural alert for positive genotoxicity were ruled out by (Q)SAR models for Ames mutagenicity (Leadscope Model Applier, ACD Percepta, Toxtree, hybrid model TIMES) as well as for *in vivo* genotoxicity (DEREK Nexus) including rodent *in vivo* Micronuclei (Leadscope Model Applier and TIMES). The TIMES *in vivo* Micronuclei model algorithm integrates the rat *in vivo* metabolic simulator. DBDPE was not found to metabolize in *in vitro* hepatic microsomal preparations from rat, and other higher mammals such as beluga whale and polar bear (McKinney et al. 2011). Given the fact that DBDPE has a large molecular weight and size in addition to its limited solubility in water and organic solvents, it is likely to result in limited uptake and systemic exposure.

Endpoint	Lowest effect levels[1]/results
Animal Studies	
Reproductive/ Developmental toxicity	**NOAEL (oral, rat) = 1250 mg/kg/day** based on the absence of effects in both dams and foetuses at this dose level. Female Crl:CD®BR VAF/Plus® rats were mated with Sprague-Dawley Crl:CD®BR VAF/Plus® male rats. DBDPE was administered via gavage in corn oil at dose levels of 0, 125, 400, or 1,250 mg/kg-bw/day to females (25 per group) from gestation day (GD) 6 through 15. Animals were observed daily for clinical signs of toxicity. Body weights and food consumption were also measured. All female rats were sacrificed on GD 20 and subjected to caesarean section. Foetuses were individually weighed, sexed, and examined for external, visceral and skeletal abnormalities. No treatment-related mortality, abortions, or clinical signs of toxicity were observed during the study. Body weights, body weight gain, and food consumption were not affected by treatment. No significant internal abnormalities (uterus) were observed in on necropsy. Caesarean section parameters were comparable between control and treated groups. No treatment-induced malformations or developmental variations occurred (there was a statistically significant increase in the number of litters with unossified hyoid bones and reduced ossification of the skull in the 400 mg/kg dose group, but not in the higher dose level) (Mercieca 1992a cited in Environment Agency 2007; Hardy et al. 2010). **NOAEL (oral, rabbit) = 1250 mg/kg/day** based on the absence of effects in both dams and foetuses at this dose level. Female New Zealand White Rabbits were artificially inseminated at 51/2 months of age. DBDPE was administered via gavage in corn oil at dose levels of 0, 125, 400, or 1,250 mg/kg-day to females (25 per group) from gestation day (GD) 6 through 18. Animals were observed daily for clinical signs of toxicity. Body weights and food consumption were also measured. All female rats were sacrificed on GD 29 and subjected to caesarean section. Foetuses were individually weighed, sexed, and examined for external, visceral and skeletal abnormalities. No treatment-related mortality, abortions, or clinical signs of toxicity were observed during the study. Body weights, body weight gain, and food consumption were not affected by treatment. No significant internal abnormalities (uterus) were observed on necropsy. Caesarean section parameters were comparable between control and treated groups. No treatment-induced malformations or developmental variations occurred (there was a statistically significant increase in the number of litters with 27 presacral vertebrae at the high-dose group, but well within historical range) (Mercieca 1992b cited in Environment Agency 2007; Hardy et al. 2010). No inhalation and dermal studies were identified.
Genotoxicity endpoints *in vivo*	No studies identified.
Genotoxicity and related	**Mutagenicity Assay** **Negative:** *Salmonella typhimurium* (TA 98, TA 100, TA 1535, TA 1537, and TA

Endpoint	Lowest effect levels[1]/results
Animal Studies	
endpoints *in vitro*	1538 strains) and *Escherichia coli* (WP2 uvrA strain) when tested with and without metabolic activation at concentrations ranging from 0 to 5000 µg/plate in DMSO (Stankowski 1988; San and Wagner 1991 - all cited in Environment Agency 2007). **Chromosome Aberrations** **Negative**: in Chinese hamster lung (CHL) cells at concentrations from 0 to 625 µg/ml for 6 hours with and without metabolic activation (Putman and Morris1992 cited in Environment Agency 2007). **Negative**: in an independent repeat assay conducted with DBDPE suspended in carboxymethylcellulose (selected to reduce precipitation) at concentrations of 625, 1,250, 2,500 and 5,000 µg/ml with and without metabolic activation. The exposure times were the same as those used in the first assay (Putman and Morris 1992cited in Environment Agency 2007).
Endocrine Disruption Activity	**Lowest LOEL = 100 mg/kg bw/day** based on significant increase in serum thyroid hormone triiodothyronine (T3) levels at this dose level. Male Sprague-Dawley (6 per treatment) rats were orally administrated 100 mg/kg/d of DBDPE in corn oil for 90 days. Thyroid hormone levels were monitored by radioimmunoassay from blood samples collected at the same time in the day. Thyroxine (T4) levels did not appear to be significantly altered. No weight measurements or histopathological survey of the thyroid were indicated in the study (Wang et al. 2010). Also, the results of this study were challenged by other scientists in the public literature (Banasik et al. 2011). **QSAR Model**: A QSAR model developed by Papa et al. (2010) predicted that DBDPE would have high binding affinity to the AhR receptor. The application of the prediction of the model did show that the binding activity to AhR of the PBDEs is at least 50-100 times lower than that of the reference toxicant TCDD. The model also predicted that DBDPE would have a moderate activity as a progesterone antagonist. Among the endpoints where the model predicted DBDPE to induce low activity were: EROD, DR agonist, ER agonist, T4, and E2 sulphonyl transferase (Papa et al. 2010). It should be noted that, in some cases, DBDPE fell out of the model domain for prediction and could be less reliable.
Irritation	**No skin irritation**: New Zealand White Rabbits (3 males; 3 females) were administered a dose of 500 mg of DBDPE with saline to the shaved skin under occlusive conditions for 4 hrs. Following exposure, the application site was rinsed off with water and observed for signs of irritation (erythema and oedema) with scores recorded at 1, 24, 48, and 72 hours post-treatment. No signs of skin irritation were observed; the mean 24-72 hour score for erythema and oedema

Endpoint	Lowest effect levels[1]/results
Animal Studies	
	was 0 (Mallory, 1988ccited in Environment Agency 2007). **Slight eye irritation**: DBDPE (100 mg) was instilled into the conjunctival sac of one eye of each of six albino New Zealand rabbits (3 males; 3 females) and the eyes were examined at 1, 24, 48 and 72 hours post-application. No iridial or corneal effects were noted at any of the time points. Conjunctival redness (score 1) was noted in all of the animals at 1 hour. This persisted until 48 hours in one male only. No effects were seen in any of the animals at 72 hours. The maximum average score at 1 hour was 3.0 (Mallory, 1988dcited in Environment Agency 2007).
Sensitization	**Inconclusive**: In a guinea pig maximization test, 40 guinea pigs (10 per sex/group) were injected intradermally with 5% DBDPE, vehicle only (0.5% methylcellulose) as a negative control, or 5% hexylcinnamic aldehyde (HCA) as a positive control. After one week, animals were administered patches of the vehicle, positive control (100% HCA) or DBDPE (100%) topically to the shaved test sites under occlusive wrappings for 48 hours. After 14 days, animals were challenged with 1% DBDPE, vehicle or 50% HCA for 24 hours and observed at 24 and 48 hours. A positive response (mild erythema) was observed at 24 hours in 90% of the animals in all of the groups. After 48 hrs, only 2/10 control and 4/20 of the treated animals showed a positive response. It is predicted that DBDPE would have low potential to cause skin sensitisation given that it is generally unreactive (Newton 2003; cited in Environment Agency 2007).
Human studies	
In vitro	HepG2 cells (human hepatocytes) were cultured in the presence of DBDPE (3.125-100.0 mg/L) for 24, 48, and 72 h respectively and the toxic effect of DBDPE was studied. DBDPE inhibited HepG2 viability in a time and dose-dependent manner within a range of 12.5 mg/L to 100 mg/L and for 48 h and 72 h. Induction of apoptosis was detected at 12.5-100 mg/L at 48 h and 72 h by propidium iodide staining, accompanied with overproduction of reactive oxygen species (ROS) (Sun et al. 2012). The authors stated that the mechanism of cytotoxicity of DBDPE is unclear and therefore requires further study.
Skin sensitization	No evidence of skin sensitization properties was observed on 200 professional workers from Wei-Dong Chemical Company from a repeated application of DBDPE in petrolatum for three weeks (Li et al. 2004; cited in Norwegian Pollution Control Authority (SFT) 2008).

[1] LD_{50}, median lethal dose; LOAEL = lowest-observed-adverse-effect level; NOAEL = no-observed-adverse-effect level.

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