Rotterdam Convention

Operation of the prior informed consent procedure for banned or severely restricted chemicals

Decision Guidance Document

Decabromodiphenyl ether

Secretariat of the Rotterdam Convention
on the Prior Informed Consent Procedure
for Certain Hazardous Chemicals and Pesticides
in International Trade

Food and Agriculture Organization of the United Nations

UNEP

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Introduction

The objective of the Rotterdam Convention is to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm and to contribute to their environmentally sound use, by facilitating information exchange about their characteristics, by providing for a national decision-making process on their import and export and by disseminating these decisions to Parties. The Secretariat of the Convention is provided jointly by the United Nations Environment Programme (UNEP) and the Food and Agriculture Organization of the United Nations (FAO).

Candidate chemicals[1] for inclusion in the prior informed consent (PIC) procedure under the Rotterdam Convention include those that have been banned or severely restricted by national regulatory actions in two or more Parties[2] in two different regions. Inclusion of a chemical in the PIC procedure is based on regulatory actions taken by Parties that have addressed the risks associated with the chemical by banning or severely restricting it. Other ways might be available to control or reduce such risks. Inclusion does not, however, imply that all Parties to the Convention have banned or severely restricted the chemical. For each chemical included in Annex III of the Rotterdam Convention and subject to the PIC procedure, Parties are requested to make an informed decision whether they consent or not to the future import of the chemical.

At the face-to-face segment of its tenth meeting, held in Geneva from 6 to 17 June 2022, the Conference of the Parties agreed to list decabromodiphenyl ether in Annex III of the Convention and adopted the decision-guidance document with the effect that these chemicals became subject to the PIC procedure.

The present decision-guidance document was communicated to designated national authorities on 21 October 2022, in accordance with Articles 7 and 10 of the Rotterdam Convention.

Purpose of the decision guidance document

For each chemical included in Annex III of the Rotterdam Convention, a decision-guidance document has been approved by the Conference of the Parties. Decision-guidance documents are sent to all Parties with a request that they make a decision regarding future import of the chemical listed in the relevant category(ies) in Annex III to the Convention. Further information on import response can be found on the website of the Rotterdam Convention.[3]

Decision-guidance documents are prepared by the Chemical Review Committee. The Committee is a group of government-designated experts established in line with Article 18 of the Convention, which evaluates candidate chemicals for possible inclusion in Annex III of the Convention. Decision-guidance documents reflect the information provided by two or more Parties in support of their national regulatory actions to ban or severely restrict the chemical. They are not intended as the only source of information on a chemical nor are they updated or revised following their adoption by the Conference of the Parties.

There may be additional Parties that have taken regulatory actions to ban or severely restrict the chemical and others that have not banned or severely restricted it. Risk evaluations or information on alternative risk mitigation measures submitted by such Parties may be found on the Rotterdam Convention website (www.pic.int).

Under Article 14 of the Convention, Parties can exchange scientific, technical, economic and legal information concerning the chemicals under the scope of the Convention including toxicological, ecotoxicological and safety information. This information may be provided directly to other Parties or through the Secretariat. Information provided to the Secretariat will be posted on the Rotterdam Convention website.

Information on the chemical may also be available from other sources.

[1] According to the Convention, the term “chemical” means a substance, whether by itself or in a mixture or preparation and whether manufactured or obtained from nature, but does not include any living organism. It consists of the following categories: pesticide (including severely hazardous pesticide formulations) and industrial.

[2] According to the Convention, the term “Party” means a State or regional economic integration organization that has consented to be bound by the Convention and for which the Convention is in force.

[3] http://www.pic.int/Procedures/ImportResponses/tabid/1162/language/en-US/Default.aspx.

Disclaimer

The use of trade names in the present document is primarily intended to facilitate the correct identification of the chemical. It is not intended to imply any approval or disapproval of any particular company. As it is not possible to include all trade names currently in use, only a certain number of commonly used and published trade names have been included in the document.

While the information provided is believed to be accurate according to data available at the time of preparation of the present decision-guidance document, FAO and UNEP disclaim any responsibility for omissions or any consequences that may arise there from. Neither FAO nor UNEP shall be liable for any injury, loss, damage or prejudice of any kind that may be suffered as a result of importing or prohibiting the import of this chemical.

The designations employed and the presentation of material in this publication do not imply the expression of any opinion whatsoever on the part of FAO or UNEP concerning the legal status of any country, territory, city or area or of its authorities or concerning the delimitation of its frontiers or boundaries.

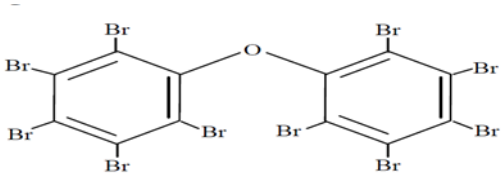
STANDARD CORE SET OF ABBREVIATIONS	
<	less than
≤	less than or equal to
>	greater than
≥	greater than or equal to
µg	microgram
µM	micromolar
BAF	bioaccumulation factor
BMF	biomagnification factor
bw	body weight
°C	degree Celsius (centigrade)
CAS	Chemical Abstracts Service
cm	centimetre
DNA	deoxyribose nucleic acid
dw	dry weight
ECHA	European Chemicals Agency
EFSA	European Food Safety Authority
EU	European Union
FAO	Food and Agriculture Organization of the United Nations
g	gram
h	hour
IARC	International Agency for Research on Cancer
IPCS	International Programme on Chemical Safety
IUPAC	International Union of Pure and Applied Chemistry
k	kilo- (x 1000)
kg	kilogram
Kow	octanol-water partition coefficient
L	litre
LC_{50}	median lethal concentration
LD_{50}	median lethal dose
LOAEL	lowest-observed-adverse-effect level
LOEL	lowest-observed-effect-level
lw	liquid weight
m	metre
mg	milligram
ng	nanogram
NOAEL	no-observed-adverse-effect level
NOEC	no-observed-effect concentration
OECD	Organisation for Economic Co-operation and Development
POPRC	Persistent Organic Pollutants Review Committee of the Stockholm Convention
RfD	reference dose (for chronic oral exposure; comparable to ADI)
TH	thyroid hormone
TMF	trophic magnification factor
TSH	thyroid-stimulating hormone
UNEP	United Nations Environment Programme
US EPA	United States Environmental Protection Agency
w/w	weight for weight
WHO	World Health Organization
wt	weight

Decision guidance document for a banned or severely restricted chemical

DECABROMODIPHENYL ETHER

Published:

1. Identification and uses (see Annex 1 for further details)

Common name	Decabromodiphenyl ether (decaBDE or BDE-209)
Chemical name and other names or synonyms	2,3,4,5,6-Pentabromo-1-(2,3,4,5,6-pentabromophenoxy)benzene, 1,1'-Oxybis(pentabromobenzene), Bis(pentabromophenyl) ether, Decabromodiphenyl oxide, Bis(pentabromophenyl) oxide, Decabromo biphenyl oxide, Decabromo phenoxybenzene, Benzene, 1,1'-oxybis[2,3,4,5,6-pentabromo-], Benzene, 1,1' oxybis-, decabromo derivative, DBBE, DBBO, DBDPO Present in: commercial decaBDE mixture, c-decaBDE, technical decaBDE, technical DeBDE
Molecular formula	$C_{12}Br_{10}O$
Chemical structure	
CAS-No.(s)	1163-19-5
Harmonized System Customs Code	29093038[4]
Other numbers	EINECS No: 214-604-9 RTECS No: KN3525000 MITI No: 3-2846
Category	Industrial
Regulated category	Industrial chemical
Use(s) in regulated category	**Canada:** The major end-use applications of PBDEs, including decaBDE, has been as flame retardants, mostly in consumer products such as furniture, televisions and computers. The automotive sector was using products that contain decaBDE and have since transitioned away to alternative products for new vehicles; however, decaBDE continues to be used in automotive replacement parts imported into Canada. The aerospace sector was using products that contain decaBDE for specialised applications, but has since completed their transition to alternate products that do not contain decaBDE. **Japan:** DecaBDE has been used as a flame retardant for resins and for textiles and is contained in consumer products such as electrical appliances, plastic products and car seats. **Norway:** DecaBDE has been used as a flame retardant in polymers and high impact polystyrene (HIPS) with end-uses in electrical and electronic equipment. It is also known to be used in the plastics and textile industries.

[4] This harmonized system customs code applies to Brominated derivatives of aromatic ethers (excluding pentabromodiphenyl ether, 1,2,4,5-tetrabromo-3,6-bis"pentabromophenoxy"benzene and 1,2-bis"2,4,6-tribromophenoxy"ethane for the manufacture of acrylonitrile-butadiene-styrene [ABS]).

Trade names	Trade names of commercial decaBDE (c-decaBDE) are : DE-83R, DE-83, Nonnen DP 10, Plasafety EBR 700, Saytex 102E, Tardex 100, Bromkal 82-0DE, Bromkal 70-5, FR1210, Flamecut 110R, FR-300-BA. *This is an indicative list of trade names. It is not intended to be exhaustive.*
Formulation types	Commercial decaBDE (c-decaBDE) consists predominantly of decaBDE (BDE-209) (≥97%), with low levels of nonaBDE (0.3-3%) (CAS No. 63936-56-1), and octaBDE (0-0.04%) (CAS No. 32536-52-0).
Uses in other categories	Not applicable
Basic manufacturers	Albemarle Corporation, Chemtura Corporation, ICL Industrial Products, Kalk, Dow, Ethyl/Saytech, Tosoh Corporation *This is an indicative list of known current and former manufacturers. It is not intended to be exhaustive.*

2. Reasons for inclusion in the PIC procedure

Decabromodiphenyl ether (CAS number 1163-19-5), subsequently referred to as decaBDE, is included in the PIC procedure in the industrial chemical category. DecaBDE is listed on the basis of final regulatory actions notified by Canada, Japan, and Norway that ban its use as an industrial chemical.

2.1 Final regulatory action (see Annex 2 for further details)

Canada:

The regulatory action notified by Canada relates to the industrial uses of polybrominated diphenyl ethers (PBDEs) that have the molecular formula $C_{12}H_{(10-n)}Br_nO$, in which n is between 4 and 10 inclusive. This group includes decaBDE (CAS No. 1163-19-5). This notification for polybrominated diphenyl ethers (PBDEs) replaces previously submitted notifications by Canada for pentabromodiphenyl ether commercial mixture (c-pentaBDE), and octabromodiphenyl ether commercial mixture (c-octaBDE), on 14 October 2010. The notification states that the manufacture, use, sale, offer for sale or import of PBDEs, including decaBDE, and all products that contain PBDEs, except for manufactured items, are prohibited. The substance is regulated under the Prohibition of Certain Toxic Substances Regulations, 2012, as amended in 2016, made under the Canadian Environmental Protection Act, 1999 (CEPA). (UNEP/FAO/RC/CRC.15/5, sections 1 and 2 of the Canadian notification).

Reason: Environment. The notification states that the screening assessment concluded that PBDEs, including decaBDE, were entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

Japan:

The regulatory action notified by Japan relates to the industrial uses of decaBDE (CAS number 1163-19-5). The notification states that it is prohibited to manufacture, import, and use this chemical substance. It also states that all uses are prohibited by the final regulatory action, and that no uses remain allowed. The substance has been designated as a Class 1 Specified Chemical Substance under the Chemical Substances Control Law of Japan and its Enforcement Order. (UNEP/FAO/RC/CRC.15/5, section 2 of the Japanese notification).

Reason: Human health and the environment. The notification states that this chemical is persistent, highly bioaccumulative and has long-term toxicity to humans. The notification also states that decaBDE exerts reproductive, developmental, endocrine and neurotoxic effects in aquatic organisms, mammals and birds.

Norway:

The regulatory action notified by Norway relates to the industrial uses of decaBDE (CAS number 1163-19-5). The notification states that the production, import, export, sale and use of decaBDE in pure form, in preparations, in products, and in parts of products containing greater than or equal to 0.1% by weight, are prohibited. The substance has been regulated under the 'Regulations relating to restrictions on the manufacture, import, export, sale and use of chemicals and other products hazardous to health and the environment (Product Regulations)', by the Ministry of the Environment, Act no. 922 of 1 June 2004. (UNEP/FAO/RC/CRC.15/5, section 2 of the Norwegian notification).

Reason: Human health and the environment. Norwegian authorities banned decaBDE based on its potential persistent, bioaccumulative, and toxic (PBT) properties and the general concern about the ubiquitous presence and increase of decaBDE in the environment including the Norwegian Arctic, and a concern for the presence of decaBDE in human matrices and human health.

2.2 Risk evaluation (see Annex 1 for further details)

Canada:

PBDEs, including decaBDE, were among the substances selected for a pilot project for screening assessments under the Canadian Environmental Protection Act, 1999 (CEPA), on the basis of their potential persistence and/or bioaccumulation in the environment and inherent toxicity to organisms. The Screening Assessment Report prepared by Environment Canada in 2006 addresses prevailing conditions within Canada and the findings have been evaluated against Canadian legislative criteria: namely, it is concluded that PBDEs, including decaBDE, were entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity. It was concluded that while all PBDEs, including decaBDE, met criteria for persistence, only tetra- to hexaBDE met the legislative criteria for bioaccumulation. However, the analysis also noted that decaBDE could accumulate to some degree in biota and debrominate to the bioaccumulative and persistent transformation products, the lower brominated diphenyl ether homologues.

Summarized in section 3.2.3 of the notification from Canada is evidence of the detection of PBDEs in all environmental media as well as sewage sludge, and there is evidence that their levels in the North American environment are increasing. Results were reported on biota in the Canadian Arctic and some temporal trends are noted such as the increase in PBDE levels in marine mammals, such as ringed seals and beluga whales.

In section 3.2.3 of the Canadian notification, it is stated that the analysis of risk quotients indicates that the greatest potential for risk from PBDEs in the Canadian environment is due to the secondary poisoning of wildlife from the consumption of prey containing elevated c-pentaBDE and c-octaBDE congener concentrations. Also, it indicated that elevated concentrations of components of c-pentaBDE in sediments may present risk to benthic organisms. The risks associated with these congeners may be due to debromination of highly brominated PBDEs, such as decaBDE.

Although overall, the available data does not show that decaBDE itself meets the numeric criteria for bioaccumulation, as defined in the Persistence and Bioaccumulation Regulations under CEPA, some studies have shown concentrations of decaBDE to be increasing steadily in some wildlife species. In some cases, such as in the tissues of kestrel, sparrowhawk, peregrine falcon, glaucous gull, red fox, shark, harbour porpoise, and whitebeaked dolphin, measured concentrations of decaBDE are interpreted as high.

The available information on persistence, bioaccumulation and toxicity, as well as the risk quotient analysis for pelagic, benthic, and soil organisms, and wildlife consumers, indicate that PBDEs, including decaBDE, have the potential to cause ecological harm in Canada. The widespread presence of decaBDE in the environment warrants concern in light of strong evidence that the substance is environmentally persistent and bioaccumulative, through debromination to lower brominated PBDEs. (summarised from section 3.2.3 of the Canadian notification, UNEP/FAO/RC/CRC.15/5).

The screening assessment also concluded that the presence of PBDEs in the environment results primarily from human activity (that is, releases from product manufacturing and processing, and throughout the product lifecycle).

The major end-use applications of PBDEs, including decaBDE, has been as flame retardants, mostly in consumer products such as furniture, televisions and computers. The automotive sector was using products that contain decaBDE and have since transitioned away to alternative products for new vehicles; however, decaBDE continues to be used in automotive replacement parts imported into Canada. In addition, the use of decaBDE in products which are not manufactured items (e.g. adhesives, sealants, caulking) has been phased out. Until recently, the aerospace sector was using products that contain decaBDE for specialised applications, but has since completed their transition to alternate products that do not contain decaBDE. (UNEP/FAO/RC/CRC.15/5 section 2.3.1 of the Canadian notification).

Japan:

The human health risk assessment is given in the Risk assessment for chemical substances contained in products, decabromodiphenyl ether, CAS No. 1163-19-5, prepared by the National Institute of Technology and Evaluation, Ministry of Economy, Trade and Industry, Ministry of Health, Labour and Welfare, Japan. An English language summary of this is given in the supporting information (UNEP/FAO/RC/CRC.15/INF/9).

It is noted that decaBDE is mainly used as flame retardant for resins and textiles, and is contained in consumer products such as electrical appliances, plastic products, and car seats. The National Institute of Technology and Evaluation (NITE) therefore conducted the risk assessment on the health effects for Japanese people who are exposed to decaBDE via those products indoors and in a car interior. Based on data from Japanese investigations, together with information from overseas risk assessments, furniture, car fabrics, and indoor and car-interior dust, were set as the exposure sources to be investigated. Adults and children under the age of 6, living in Japan, were set as the target groups of people in this risk assessment. The reason why the assessment included young children was due to their intake behaviours such as holding objects in their mouth and licking objects, or through dust, being different to the intake of adults.

Toxicity effect level and minimal risk level (MRL) were taken from the hazard assessment report on PBDEs from the US Agency for Toxic Substances and Disease Registry (ATSDR) of March 2017, and adjusted to reflect chronic-duration exposure (lifetime exposure). For the estimation of exposure amounts, eight exposure scenarios in total were set for environments inside houses and cars where the products to be investigated are used or exist. The estimated human exposure (EHE) per day was calculated by summing the exposure amounts estimated for each of the eight exposure scenarios. As the EHE values for adults and children were substantially different, they were averaged, using a 6-year span for children and a 64 year span for adults, to obtain an average lifetime EHE over 70 years.

The total estimated human exposure (EHE) of decaBDE in ng/kg/day was then compared with the toxicity effect value (hazard assessment value), derived from the ASTDR results, to obtain a Hazard Quotient (HQ). As the lifetime average exposure value was less than the hazard assessment value, a HQ of less than 1 (0.6) was obtained, indicating the risk is not at a level of concern.

The Japanese decaBDE risk assessment for the environment is given in the Environmental risk assessment of short-chain chlorinated paraffins and decabromodiphenyl ether, prepared by Ministry of Environment, Japan, 22 September 2017. A summary in Chapter 6 of the document was translated into English ((UNEP/FAO/RC/CRC.15/INF/9, page 6). This summary contained the following information:

An environmental risk assessment of decaBDE was carried out based on environmental monitoring data which was implemented and released by the Japanese government from 2003 to 2017. As a result, when comparing the D value for humans and high-level predators based on the predicted maximum exposure amount and the decaBDE toxicity data, at present, it has become clear that there are risk concerns. (UNEP/FAO/RC/CRC.15/INF/9, page 6). (D value is the Hazard Evaluation Value, which is LOAEL/Uncertainty Factors).

The future environmental risk was estimated based on the scenario that the production, import and use of decaBDE would be prohibited in the future. As a result, the environmental risk was reduced in the scenario of prohibition of production, import and use of decaBDE, and the predicted maximum exposure amount was predicted to be lower than the D value for humans and high-level predators based on decaBDE toxicity data. (UNEP/FAO/RC/CRC.15/INF/9, page 6).

Therefore, from the information obtained at the present time (2017), it was considered that there is no need to take additional measures such as collection of products to prevent progression of environmental pollution. However, it was considered necessary to continuously carry out environmental monitoring of decaBDE in the future and to take necessary measures according to the situation. (UNEP/FAO/RC/CRC.15/INF/9, page 6).

Norway:

Norwegian monitoring data show detectable levels of decaBDE in several environmental compartments, and high concentrations of decaBDE (BDE-209), the main component of c-decaBDE, is detected at some locations. BDE-209 has been detected in sediments, water, and in biota – moss, mussels, fish, and in moose and lynx, among other species. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.2 of the Norwegian notification).

Norwegian monitoring data shows that BDE-209 deposited to the Arctic environment is bioavailable to the organisms living there and that BDE-209 is widespread in Arctic food webs. Norwegian environmental monitoring studies investigating congener pattern and levels of PBDEs in eggs and plasma of glaucous gulls breeding at Bjørnøya in the Arctic revealed detectable levels of BDE-209 in bird plasma comparable to levels found in liver samples of birds located at more southern parts of Europe. Similar results were reported in liver samples from glaucous gulls from Svalbard. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.2 of the Norwegian notification).

In animal studies of amphibian, fish and rodents exposed to BDE-209 at vulnerable stages as the developmental phase, effects on hormonal axis as the thyroid and steroid is of concern. Although the toxicology data of BDE-209 is ambiguous, some studies indicate negative effects on neurological development at low doses. (UNEP/FAO/RC/CRC.15/5 sections 2.4.2.1 and 2.4.2.2 of the Norwegian notification).

The notification states that the evaluation of decaBDE gives rise to concern for long term effects in the environment. The general concern about the ubiquitous presence and increase of decaBDE in the environment, together with the risk for endocrine disrupting effects of the mix of PBDE congeners to organisms at vulnerable stages, led Norwegian authorities to ban further use of decaBDE. (UNEP/FAO/RC/CRC.15/5 sections 2.4.2.1 and 2.4.2.2 of the Norwegian notification).

In food samples analysed in Norway for BDE-209, high levels were found in eggs, vegetable oil, ice cream and biscuits, while the highest amounts were found in dairy products, which include milk, cheese, and butter. However, household dust and occupational exposure are thought to be the main sources for exposure to BDE-209 and other congeners present in c-decaBDE. Toddlers and infants have a higher daily intake of dust and dairy products than adults, and higher serum levels of BDE-209 have been found in children less than 5 years compared to their parents. Some professions are exposed to higher levels of c-decaBDE than the average population and other workers. Foam recycling workers, carpet installers and PC technicians are reported to have higher serum levels of BDE-209 than control groups. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.1 of the Norwegian notification).

High levels of BDE-209 (10 ng/g lipid) have been found in pooled serum samples from the Norwegian population. A similar study detected an average of 2.26 ng/g lipid in plasma from pregnant women from the Bodø region of Norway. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.1 of the Norwegian notification).

The widespread presence of decaBDE in the environment warrants concern in light of strong evidence that the substance is environmentally persistent and bioaccumulative, through debromination to lower brominated PBDEs. The potential PBT properties of decaBDE and a concern for its presence in human matrices and effects on human health, were also contributory reasons for the Norwegian ban on decaBDE.

DecaBDE has been used as a flame retardant in polymers and high impact polystyrene (HIPS) with end-uses in electrical and electronic equipment. It is also known to be used in the plastics and textile industries. (UNEP/FAO/RC/CRC.15/5 section 2.3.1 of the Norwegian notification).

3. Protective measures that have been applied concerning the chemical

3.1 Regulatory measures to reduce exposure

Canada: The notification stated that the manufacture, use, sale, offer for sale or import of PBDEs, including decaBDE, and all products that contain PBDEs, except for manufactured items, are prohibited with a limited number of exemptions. PBDEs, including decaBDE, are regulated under the Prohibition of Certain Toxic Substances Regulations, 2012, as amended in 2016, made under the Canadian Environmental Protection Act, 1999 (CEPA). The regulatory action came into force on 23 December 2016.

The final regulatory action provides for a limited number of exemptions to the prohibition of PBDEs:

- The manufacture, use, sale, offer for sale, or import of PBDEs or a product containing them, if PBDEs are incidentally present.
- The manufacture, use, sale, offer for sale, or import of manufactured items containing PBDEs.
- The use, sale or offer for sale of:
    - Products containing PBDEs that were manufactured or imported before the Regulations came into force;
    - PBDEs that were imported in accordance with a permit;
    - Products that contain PBDEs that were manufactured or imported in accordance with a permit.

In addition, the Prohibition of Certain Toxic Substances Regulations 2012 do not apply to any toxic substance, including PBDEs, that is:

- Contained in a hazardous waste, hazardous recyclable material or non-hazardous waste to which Division 8 of Part 7 of CEPA applies;
- Contained in a pest control product as defined in subsection 2(1) of the Pest Control Products Act;
- Present as a contaminant in a chemical feedstock that is used in a process from which there are no releases of the toxic substance and on the condition that the toxic substance is destroyed or completely converted in that process to a substance that is not a toxic substance set out in either Schedule 1 or 2 of the regulations; or
- To be used in a laboratory for analysis, in scientific research or as a laboratory analytical standard.

Information related to the use of toxic substance, or a product containing it for the purpose in the last bullet above must be submitted to the Minister of Environment in any calendar year as soon as feasible before the use of more than 10 g of the substance, by itself or in a product, in that calendar year.

(UNEP/FAO/RC/CRC.15/5 Section 2.3.2 of the Canadian notification).

Japan: The substance has been designated as a Class 1 Specified Chemical Substance under the Chemical Substances Control Law of Japan and its Enforcement Order. The regulatory action came into force on 1 April 2018 (UNEP/FAO/RC/CRC.15/5, section 2 of the Japanese notification).

The Japanese final regulatory action prohibits the manufacture, import, and use of decaBDE (UNEP/FAO/RC/CRC.15/5, section 2.2.1 of the Japanese notification). The notification also indicates that all uses are prohibited by the final regulatory action and that no uses remain allowed

(UNEP/FAO/RC/CRC.15/5, section 2.3.2 of the Japanese notification), and that the final regulatory action is a ban (UNEP/FAO/RC/CRC.15/5, section 2.1 of the Japanese notification).

Norway: DecaBDE has been regulated under the 'Regulations relating to restrictions on the manufacture, import, export, sale and use of chemicals and other products hazardous to health and the environment (Product Regulations)', by the Ministry of the Environment, Act no. 922 of 1 June 2004. The regulatory action came into force on 1 April 2008 and was amended 1 July 2013.

The Norwegian final regulatory action prohibits the manufacture, import, export, sale and use of substances or preparations that contain 0.1% by weight or more of decaBDE. It is also prohibited to manufacture, import, export and place on the market products or flame-retardant parts of products that contain 0.1% by weight or more of decaBDE. The prohibition in respect of products and parts of products also applies to electrical and electronic equipment (EEE). For some categories of EEE, the restrictions took effect over a period of time, from July 2014 until July 2019. (UNEP/FAO/RC/CRC.15/5 section 2.3.2 of the Norwegian notification).

There are some limited uses that remain allowed:

- Use in certain approved vehicles, registered aircraft, registered vessels, and rolling stock for use on railways, including tramways, underground railways, suburban lines and similar forms of rail transport.
- Time limited exemptions for certain categories of EEE.
- Reused spare parts, recovered from EEE placed on the EEA market before 1 July 2006 and used in equipment placed on the EEA market before 1 July 2016, provided that reuse takes place in auditable closed-loop business-to-business return systems, and that the reuse of parts is notified to the consumer. (UNEP/FAO/RC/CRC.15/5 section 2.3.2 of the Norwegian notification).

The Norwegian notification indicates this is a ban.

3.2 Other measures to reduce exposure

The information in this section was prepared based on the information on actions under international conventions and forums, and national and/or regional regulations, contained in document UNEP/POPS/POPRC.11/10/Add.1, Chapter 1.4 and 1.5 of the POPRC Risk Management Evaluation and the information provided during the sixteenth meeting of the Chemical Review Committee held from 8 to 11 September 2020. The information in the POPRC Risk Management Evaluation mainly relates to commercial decaBDE (c-decaBDE) the main component of which is decaBDE (CAS number 1163-19-5) which is the subject of this DGD.

In 1995, OECD Member countries agreed to oversee a voluntary industry commitment (VIC) by some of the global manufacturers of brominated flame retardants (BFRs), among them c-decaBDE, to take certain risk management actions. The VIC was implemented in the United States, Europe and Japan. In parallel to this work, OECD conducted an investigation of the waste management practices in member countries with respect to products containing BFRs. The results of this investigation are documented in the Report on the Incineration of Products Containing Brominated Flame Retardants (OECD 1998). A SIDS Initial Assessment Profile (SIAP) on decaBDE was prepared under the Environment, Health and Safety (EHS) Programme of the OECD and adopted by SIAM 16 and later endorsed by the OECD Joint Meeting in 2003. The Hazard/Risk Information Sheets for c-decaBDE and four other BFRs were updated in 2005, 2008 and 2009. PBDEs, including decaBDE, are listed as endocrine disrupting chemicals of concern in the WHO/UNEP "State of the science of endocrine disrupting chemicals" (UNEP/WHO 2013).

In Europe, with the Restriction of Hazardous Substances Directive (RoHS), the use of PBDEs, including c-decaBDE was banned in electronic and electrical equipment (EEE) in the EU at concentrations above 0.1% by weight of homogeneous material. Although this legislation came into force in February 2008, medical equipment was initially exempt. In June 2011, however, this exemption was removed and medical devices fell within the scope of RoHS with effect from 22 July 2014. In 2012, c-decaBDE was identified as a PBT/vPvB (persistent, bioaccumulative and toxic / very persistent, very bioaccumulative) substance in the EU and included in the Candidate List of substances of very high concern (SVHCs) under the Registration, Evaluation, Authorisation and Restriction of Chemicals Regulation (REACH). In 2017, decaBDE was included in Annex XVII (restriction) of the REACH regulation (Commission Regulation (EU) 2017/227 of 9 February 2017). This means decaBDE shall not be manufactured or placed on the market as a substance on its own after 2 March 2019, and shall not be used in the production of, or placed on the market in: another substance, as a constituent; a mixture; an article, or any part thereof, in a concentration equal to or greater than 0,1 % by weight, after 2 March 2019. The restriction includes several exemptions. In 2019, decaBDE was included in Annex I to the EU POP regulation 2019/1021 leading to a complete ban of the production, placing on the market and use of this substance on its own, contained in mixtures or in articles with some specific exemptions that are limited in time.

A voluntary phase out is also ongoing in the U.S. On December 17, 2009, as a result of negotiations with the United States Environment Protection Agency (U.S. EPA), the two U.S. producers of c-decaBDE and the largest U.S. importer announced commitments to voluntary phase out c-decaBDE in the U.S. The commitments consisted of reductions in the domestic manufacture, import, and sales of c-decaBDE starting in 2010. The U.S. EPA then encouraged other importers of c-decaBDE to join this initiative. As part of this encouragement, the U.S. EPA developed a Design for the Environment and Green Chemistry alternatives assessment for c-decaBDE to aid users in selecting suitable alternatives. In addition, the U.S. EPA proposed an update to the PBDE Significant New Use Rule (SNUR) and simultaneously proposed a Toxic Substances Control Act (TSCA) section 4 test rule for c-pentaBDE, c-octaBDE, and c-decaBDE. On June 21, 2019, U.S. EPA proposed a TSCA section 6 rule on five persistent, bioaccumulative and toxic (PBT) chemicals, meeting a statutory deadline under amended TSCA which required U.S. EPA to take expedited action on certain PBT chemicals by June 2019, c-decaBDE was one of those chemicals. U.S. EPA has a statutory deadline of December 22, 2020 to finalize the c-decaBDE rule. In addition, the U.S. EPA helped establish the Furniture Flame Retardancy Partnership (as part of the U.S. EPA's Design for the Environment Program). This is a joint venture between the Furniture Industry, Chemical Manufacturers, Environmental Groups, and the U.S. EPA to better understand fire safety options for the furniture industry. This type of group has helped the textile and foam industries to quickly transition away from BFRs. Additionally, in the U.S., several states have also imposed restrictions on the manufacture and/or use of c-decaBDE in certain applications, including in mattresses, mattress pads and other bedding products, seating, furniture and electronic products.

In Asia, restrictions have been adopted in China, India and Korea. In the revision of the Chinese RoHS legislation (Administrative Measure on the Control of Pollution Caused by Electronic Information Products) a restriction on the use of c-decaBDE in EEE was adopted. According to Annex F information from China, PBDEs are not allowed in EEE at concentrations above 0.1% by weight for environmental labelling of products. E-waste must be handled in accordance with the legislation on the Waste of Electrical and Electronic Equipment. Furthermore, it has been reported that in China e-waste containing PBDE FRs should be separated out. It should be disposed of as hazardous waste. Korea enforced the Act on Resource Circulation of Electrical and Electronic Equipment and Vehicles in 2008, which ensures that the concentration of PBDEs, including c-decaBDE, in electrical and electronic equipment is less than 0.1 % by weight. This is the same as the EU RoHS Directive. As decaBDE was listed in the Stockholm Convention, Korea ratified a relevant amendment and has prohibited any manufacture, import and export, or use of c-decaBDE under the Persistent Organic Pollutants Control Act from 20 February 2020 when Korea's enforcement process was completed. Korea's specific exemption on decaBDE is limited only to additives of electrical parts, and spare parts of legacy vehicles and aircraft that shall comply with the concentration limits specified under the Act on Resource Circulation of Electrical and Electronic Equipment and Vehicles, with the exceptions listed to the Stockholm Convention. In India, the e-waste (management and handling) Rules came into effect in May 2012. The chapter on the Restriction of hazardous substances under the e-waste rules restricts the use of PBDEs in EEE with a threshold limit of 0.1%. In Japan, under the Chemical Substances Control Law, the annual production or import volumes of decaBDE have to be reported along with shipment volumes.

In addition to the above measures taken by countries, initiatives to voluntarily phase-out c-decaBDE have been taken by industry. The member companies of the Bromine Science and Environmental Forum (BSEF) moreover agreed with the U.S. EPA and Canadian authorities and voluntarily phased out production, import and sales of c-decaBDE in the United States and Canada as of the end of 2013. Also, the automotive industry represented by ACEA has committed in its latest input into the public SEAC consultation on the EU REACH Restriction to completely phase out decaBDE globally, latest by mid of 2018 for current production and new developments. Phase out is also ongoing in North America and China. Many electronics firms have already eliminated or committed to eliminating c-decaBDE in accordance with the EU RoHS, including Philips, Electrolux, Sony, Dell, Intel, Sharp, Apple and Hewlett Packard. Other industrial stakeholders have also implemented/ launched voluntary initiatives. Furthermore, large global furniture producers, have phased-out the use PBDEs including c-decaBDE and several mattress producers globally now offer PBDE-free mattresses. In addition, there are voluntary initiatives to control and reduce potential emissions of commercial decaBDE into the environment. The European Flame Retardant Association (EFRA) together with the industry's global organisation, BSEF, has moreover launched a voluntary initiative whereby member companies aim to manage, monitor and minimize industrial emissions of high production volume BFRs, including decaBDE through partnership with the supply chain. This program, called the Voluntary Emissions Control Action Program (VECAP) started in Europe in 2004 but has later also been introduced in North America and in Japan.

Stockholm Convention on Persistent Organic Pollutants

At its eighth meeting, the Conference of the Parties of the Stockholm Convention, by its decision SC-8/10, decided to list decaBDE, present in commercial decabromodiphenyl ether, in Annex A of the Convention, with specific exemptions for the production and use of commercial decabromodiphenyl ether. The specific exemption was limited to the following uses:

- Parts for use in vehicles limited to the following:
  a) Parts for use in legacy vehicles (defined as vehicles that have ceased mass production) falling into one or more of the following categories:
    i. Powertrain and under-hood applications such as battery mass wires, battery interconnection wires, mobile air-conditioning (MAC) pipes, powertrains, exhaust manifold bushings, under-hood insulation, wiring and harness under hood (engine wiring, etc.), speed sensors, hoses, fan modules and knock sensors;
    ii. Fuel system applications such as fuel hoses, fuel tanks and fuel tanks under body;
    iii. Pyrotechnical devices and applications affected by pyrotechnical devices such as air bag ignition cables, seat covers/fabrics (only if airbag relevant) and airbags (front and side);
    iv. Suspension and interior applications such as trim components, acoustic material and seat belts;
  b) Parts in vehicles specified in a) i to iv above and those falling into one or more of the following categories:
    i. Reinforced plastics (instrument panels and interior trim);
    ii. Under the hood or dash (terminal/fuse blocks, higher-amperage wires and cable jacketing (spark plug wires));
    iii. Electric and electronic equipment (battery cases and battery trays, engine control electrical connectors, components of radio disks, navigation satellite systems, global positioning systems and computer systems);
    iv. Fabric such as rear decks, upholstery, headliners, automobile seats, head rests, sun visors, trim panels, carpets.
- Aircraft for which type approval has been applied for before December 2018 and has been received before December 2022 and spare parts for those aircraft;
- Textile products that require anti-flammable characteristics, excluding clothing and toys;
- Additives in plastic housings and parts used for heating home appliances, irons, fans, immersion heaters that contain or are in direct contact with electrical parts or are required to comply with fire retardancy standards, at concentrations lower than 10 per cent by weight of the part;
- Polyurethane foam for building insulation.

The specific exemptions for parts in vehicles expires at the end of the service life of vehicles or in 2036, whichever comes earlier.

The specific exemptions for spare parts for aircraft for which type approval has been applied for before December 2018 and has been received before December 2022 shall expire at the end of the service life of those aircraft.

As at 11 September 2020, six Parties to the Stockholm Convention have registered for the exemption for vehicle parts, one for the exemption for aircraft/parts, and one for the exemption for textile products that require anti-flammable characteristics.

3.3 Alternatives

It is essential that before a country considers substituting alternatives, it ensures that the use is relevant to its national needs, and the anticipated local conditions of use. The hazards of the substitute materials and the controls needed for safe use should also be evaluated.

Canada:

ALTERNATIVE CHEMICALS

Chemical alternatives to PBDEs are available for the vast majority of industrial and manufacturing applications, and these vary by application. However, several issues need to be addressed as some potential alternatives are:

- Currently under scrutiny themselves;
- New proprietary chemicals for which data on environmental and health effects are very limited;
- More costly; and
- Less effective, hence much higher levels are required and products may be less likely to meet flammability standards.

ALTERNATIVE TECHNIQUES

The need for PBDEs can be reduced through the use of alternative techniques such as:

- Use of materials that are less prone to fire hazard in electronics equipment (such as aluminum or "super-plastics" with very high oxygen requirements for combustion); or
- Use of barrier fabrics, wrappings or coatings for foams to replace chemical flame retardants.

Some of these alternative techniques present challenges, such as increased weight of final products and methods to collect, reuse and reassemble products with components containing PBDEs.

Many alternatives to decaBDE exist and are commercially available. While PBDEs have been used for a wide array of applications, a variety of different alternatives are being used as replacements such as other brominated flame retardants and halogen free flame retardants.

A move away from decaBDE towards alternative flame retardants and, in certain cases, flame retardant barriers in products, in lieu of chemicals, means that many of the applications no longer use decaBDE, especially in view of the phase-out in the US and the broad controls proposed in other jurisdictions.

Substances which are new to Canada, including new substitutes for decaBDE, are subject to the New Substances provisions of CEPA and the New Substances Notification Regulations (Chemicals and Polymers). Any individual or company intending to import or manufacture such a substance must submit a notification, with the substance undergoing an assessment by Environment and Climate Change Canada and Health Canada to determine whether it meets the definition of "toxic" set out in section 64 of CEPA.

UNEP/FAO/RC/CRC.15/5 section 2.5.3.2 of the Canadian notification.

Japan:

Information was not provided in the notification on this criterion, however, it was stated that alternative actions were completed before April 2018.

UNEP/FAO/RC/CRC.15/5 section 2.5.3.2 of the Japanese notification

Norway:

Alternatives to decaBDE in all applications are available including non-chemical alternatives (US EPA, 2006 and 2007). Furthermore, a number of alternative flame retardants (FR) are available as substitutes for decaBDE in EEE products (Danish EPA, 2006). Alternatives include other brominated FRs and other chemical FRs.

References:

DecaBDE Study: A Review of Available Scientific Research, US EPA, 2006

Report on Alternatives to the Flame Retardant DecaBDE: Evaluation of Toxicity, Availability, Affordability, and Fire Safety Issues, US EPA, 2007.

Deca-BDE and Alternatives in Electrical and Electronic Equipment, Danish EPA, 2006.

UNEP/FAO/RC/CRC.15/5 section 2.5.3.2 of the Norwegian notification.

General:

The U.S. EPA and the European Chemicals Agency (ECHA) have published comprehensive assessments of chemical alternatives to c-decaBDE (U.S. EPA, 2014; ECHA, 2014)

U.S. EPA, 2014: An alternatives assessment for the flame retardant decabromodiphenyl ether (decaBDE). https://www.epa.gov/sites/production/files/2014-05/documents/decabde_final.pdf

ECHA (2014). Annex XV Restriction report. Proposal for a restriction – Bis(pentabromophenyl) ether. https://echa.europa.eu/documents/10162/13641/annex_xv_dossier_decabde_en.pdf/

The U.S. EPA assessment provides detailed human health and ecological hazard information for 29 substances and mixtures that have been identified as potentially alternatives to c-decaBDE in a variety of applications, while the report published by ECHA identified 13 chemicals for further assessment and evaluation as alternatives to c-decaBDE.

Most assessments of alternatives to decaBDE have focused on the replacement of decaBDE with alternative chemicals (i.e. a chemical that have flame retardant properties that can be substituted directly for decaBDE in articles). However, alternative techniques to improve fire safety also exist and are also described in the assessments above.

For more information, see chapter 2.3 of the POPRC Risk Management Evaluation on decabromodiphenyl ether: UNEP/POPS/POPRC.11/10/Add.1.[5]

3.4 Socio-economic effects

Canada:

The Regulations were expected to have a low-cost impact on the industry. The substances were never manufactured in Canada and there are no known Canadian users or importers of c-decaBDE. In addition, the use of decaBDE in products, which are not manufactured items (e.g. adhesives, sealant, caulking), has been phased out.

The three main manufacturers of c-decaBDE operating in the United States made a commitment to the U.S. EPA to cease production and sales by the end of 2013 to comply with the Significant New Use Rule. In mid-2012, these same companies also voluntarily ceased their export of c-decaBDE to Canada.

UNEP/FAO/RC/CRC.15/5 section 2.5.3.1 of the Canadian notification.

Japan:

No information on socio-economic effects of the regulatory action was provided by Japan (UNEP/FAO/RC/CRC.15/5 section 2.5.3.1 of the Japanese notification).

Norway:

No information on socio-economic effects of the regulatory action was provided by Norway (UNEP/FAO/RC/CRC.15/5 section 2.5.3.1 of the Norwegian notification).

General:

According to the POPRC Risk Management Evaluation on decabromodiphenyl ether, based on information such as price, accessibility and availability of different alternatives as well as information on regulatory measures and use in different countries, the socioeconomic costs of implementing a ban and/ or restriction on the use of c-decaBDE are considered small and outweighed by the benefits of an elimination/ regulation. An important factor as discussed in the EU restriction proposal is that although c-decaBDE is currently less expensive than the alternatives assessed, the difference in cost might gradually change in response to the increasing demand for alternatives.

With further regard to social costs, a report prepared by the Nordic Council of Ministers and other scientific publications suggests that endocrine disrupting compounds (EDCs), like c-decaBDE, are a great economic burden to society.

For more information on economic aspects and social costs, see section 2.4.4 of the POPRC Risk Management Evaluation on decabromodiphenyl ether: UNEP/POPS/POPRC.11/10/Add.1.

[5] http://chm.pops.int/tabid/5985/Default.aspx.

4. Hazards and Risks to human health and the environment	
4.1 Hazard Classification	
WHO / IPCS	Not available.
IARC	Not classifiable as to its carcinogenicity to humans (Group 3) (1990, 1999).
European Union	No Harmonised Classification in Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation). According to the classifications provided by companies to ECHA in CLP inventory notifications, this substance is harmful if swallowed, is harmful in contact with skin, is harmful if inhaled, causes serious eye irritation, is suspected of causing genetic defects, may cause damage to organs through prolonged or repeated exposure, and may cause long lasting harmful effects to aquatic life. The following classifications have been notified: Acute Tox. 4: -H302 (Harmful if swallowed) -H312 (Harmful in contact with skin) -H332 (Harmful if inhaled) Eye Irrit. 2: -H319 (Causes serious eye irritation) Muta. 2: -H341 (suspected of causing genetic defects) STOT RE 2: -H373 (liver, kidney) (May cause damage to organs (liver, kidney) through prolonged or repeated exposure) Aquatic chronic 4: -H413 (May cause long lasting harmful effects to aquatic life) https://echa.europa.eu/pl/information-on-chemicals/cl-inventory-database/-/discli/details/131436 Officially recognised in the EU as Persistent, Bioaccumulative and Toxic (PBT), and very Persistent and very Bioaccumulative (vPvB) and included on the Candidate list for authorisation as a substance of very high concern (SVHC) under REACH Regulation No 1907/2006 in December 2012. https://echa.europa.eu/pl/substance-information/-/substanceinfo/100.013.277
US EPA	Evidence of carcinogenic potential is suggested for decaBDE (EPA Integrated Risk Information System (IRIS). 2008. “2,2',3,3',4,4',5,5',6,6' -Decabromodiphenyl ether (BDE-209) (CASRN 1163-19-5).” www.epa.gov/iris). Studies in rats and mice show that PBDEs cause neurotoxicity, developmental neurotoxicity, reproductive toxicity, thyroid toxicity, immunotoxicity, liver toxicity, pancreas effects (diabetes) and cancer (penta and decabromodiphenyl ether) (ATSDR. 2015. “Draft Toxicological Profile for Polybrominated Diphenyl Ethers.” www.atsdr.cdc.gov/toxprofiles/tp207.pdf).

4.2 Exposure limits

Dietary intake and dust (household, in car) are considered as the most important routes of human exposure to decaBDE.

US EPA has established a chronic oral reference dose (RfD) for decaBDE of 7µg/kg/day (EPA. 2017. Regional Screening Level (RSL) Summary Table. www.epa.gov/risk/regional-screening-levels-rsls-generic-tables-may-2016).

For decaBDE, US EPA has assigned an oral slope factor for carcinogenic risk of 7×10^{-4} $(mg/kg/day)^{-1}$ and a drinking water unit risk of 2.0×10^{-8} per µg/L. US EPA risk assessments indicate that the drinking water concentration representing a 1×10^{-6} cancer risk level for decaBDE is 50 µg/L (EPA Integrated Risk Information System (IRIS). 2008. “2,2',3,3',4,4',5,5',6,6' -Decabromodiphenyl ether (BDE-209) (CASRN 1163-19-5).” www.epa.gov/iris).

US EPA has calculated the following screening levels for decaBDE (EPA. 2017. Regional Screening Level (RSL) Summary Table. www.epa.gov/risk/regional-screening-levels-rsls-generic-tables-may-2016):

- Residential soil – 440 mg/kg
- Industrial soil – 3,300 mg/kg
- Tap water - 110 µg/L

4.3 Packaging and labelling	
Hazard Class and Packing Group:	Not applicable
International Maritime Dangerous Goods (IMDG) Code	Not applicable
Transport Emergency Card	Not applicable

4.4 First aid

NOTE: The following advice is based on information available from the World Health Organisation and was correct at the time of publication. This advice is provided for information only and is not intended to supersede any national first aid protocols.

The information below refers to decaBDE (CAS 1163-19-5).

	Prevention	First Aid
Inhalation	Use ventilation.	Fresh air, rest.
Skin	Protective gloves.	Rinse and then wash skin with water and soap.
Eyes	Wear safety spectacles.	Rinse with plenty of water (remove contact lenses if easily possible).
Ingestion	Do not eat, drink, or smoke during work.	Rinse mouth. Give one or two glasses of water to drink.

International Chemical Safety Card (ICSC) 1689:
http://www.inchem.org/documents/icsc/icsc/eics1689.htm

4.5 Waste management

DecaBDE is listed as a persistent organic pollutant in Annex A of the Stockholm Convention. Accordingly, waste management of decaBDE, including products and articles upon becoming wastes, must be in accordance with Article 6 of the Stockholm Convention. In conjunction with the Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and their Disposal, guidelines on the environmentally sound management of wastes containing decaBDE have been prepared:

'Technical guidelines on the environmentally sound management of wastes consisting of, containing or contaminated with hexabromodiphenyl ether and heptabromodiphenyl ether, or tetrabromodiphenyl ether and pentabromodiphenyl ether or decabromodiphenyl ether', UNEP/CHW.14/7/Add.3/Rev.1, 20 June 2019

http://www.basel.int/TheConvention/ConferenceoftheParties/Meetings/COP14/tabid/7520/Default.aspx

These are supported by general technical guidelines on POPs wastes: 'General technical guidelines on the environmentally sound management of wastes consisting of, containing or contaminated with persistent organic pollutants', UNEP/CHW.14/7/Add.1/Rev.1, 20 June 2019. Available at - http://www.basel.int/Implementation/TechnicalMatters/DevelopmentofTechnicalGuidelines/TechnicalGuidelines/tabid/8025/Default.aspx

Annexes

Annex 1 **Further information on the substance**

Annex 2 **Details on final regulatory action reported**

Annex 3 **Address of designated national authorities**

Annex 4 **References**

Annex 1 Further information on the substance

Introductory text to Annex I

The information presented in the present annex reflects the evaluations and conclusions of the three notifying Parties, namely Canada, Japan and Norway. Where possible, information provided by these three Parties on hazards has been presented together, while the risk assessments, which are specific to the conditions prevailing in the Parties, are presented separately. This information is taken from the documents referenced in the notifications in support of the final regulatory actions relating to decaBDE.

The notification from Canada was reported in PIC Circular XLVIII (48) of December 2018. The notification was on polybrominated diphenyl ethers ($C_{12}H_{(10-n)}Br_{(n)}O$, $4 \leq n \leq 10$), which included decabromodiphenyl ether (decaBDE), CAS No. 1163-19-5. This notification replaced two previous notifications for pentabromodiphenyl ether commercial mixture (c-pentaBDE), and octabromodiphenyl ether commercial mixture (c-octaBDE), which were issued on 14 October 2010.

The notification from Japan was reported in PIC Circular XLVIII (48) of December 2018. The notification was on decabromodiphenyl ether (main constituent BDE-209), CAS No, 1163-19-5.

The notification from Norway was reported in PIC Circular XXXIX (39) of June 2014. The notification was on decabromodiphenyl ether (decaBDE), CAS No, 1163-19-5.

DecaBDE has also been assessed by the Persistent Organic Pollutants Review Committee (POPRC) of the Stockholm Convention and listed in Annex A of that Convention. Information and conclusions contained in the Risk Profile on decabromodiphenyl ether, prepared by POPRC (UNEP/POPS/POPRC.10/10/Add.2, 25 November 2014) has also been used in this Annex.

The information below refers to decaBDE, CAS No. 1163-19-5.

Annex 1 – Further information on decaBDE

1	**Identity and physico-chemical properties**	
1.1	**Identity**	Common name: Decabromodiphenyl ether (decaBDE) CAS name: Benzene, 1,1'-oxybis[2,3,4,5,6-pentabromo- IUPAC name: 2,3,4,5,6-Pentabromo-1-(2,3,4,5,6-pentabromophenoxy)benzene CAS number: 1163-19-5 (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
1.2	**Formula**	$C_{12}Br_{10}O$ (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
1.3	**Colour and Texture**	Fine, white to off-white crystalline powder (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
1.4	**Decomposition temperature**	Decomposes on heating above 320°C (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
1.6	**Density (g/cm^3)**	Relative density: 3.0 (ICSC 1689), 2.63 (ECHA Brief Profile, accessed at: https://echa.europa.eu/brief-profile/-/briefprofile/100.013.277)
1.7	**Resistance to acids**	Not available.
1.8	**Resistance to alkalis**	Not available.
1.9	**Tensile strength (10^3 kg/cm^2)**	Not available.
2	**Toxicological properties**	
2.1	**General**	National and regional assessments conducted by the EU, the United Kingdom, Canada and US have evaluated the potential for decaBDE to induce adverse effects in wildlife and humans. In vertebrates, the liver, the thyroid hormone (TH) axis and the nervous system appear to be the main targets for decaBDE toxicity. In both wildlife and humans, early developmental stages appear more vulnerable to decaBDE exposure than adults. (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
2.1.1	**Mode of Action**	The toxicity of decaBDE to terrestrial mammals has mainly been investigated in rodents. Although several effects are reported including reproductive toxicity, data in particular point to neurodevelopmental toxicity and effects on the TH-system. Several mechanisms for developmental neurotox effects are proposed, for instance impaired thyroid homeostasis, direct toxicity to neuronal and stem cells, and disturbing neurotransmitter systems. (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
2.1.2	**Symptoms of poisoning**	Not available.
2.1.3	**Absorption, distribution, excretion and metabolism in mammals**	<u>Toxicokinetics, metabolism and distribution</u> In male rats it was found that the majority (>90 %) of detected radioactivity were detected in the faeces 72 h after oral exposure of rats to radiolabeled decaBDE. Results indicate after analysis of the faeces that 22%, 42% and 45% of the radioactivity present at day 1, 2 and 3 respectively was present as 8 phenolic metabolites. DecaBDE is metabolised via oxidative debromination, as deduced from the presence of debrominated dihydroxylated diphenyl oxides. The remaining radioactivity present in the faeces was identified as unchanged decaBDE. In mice exposed to radiolabelled decaBDE at postnatal day 3, 10 or 19, radioactivity was found in the brain, liver and heart 24 h after exposure. During 7 days of exposure levels in brain increased for the mice exposed at postnatal day 3. Only limited data on human toxicokinetics are available. Data from monitoring indicate that decaBDE can be absorbed into the body and is distributed to the blood,

adipose tissue and maternal milk. There are no data available on the rate of elimination or of bioaccumulation of decaBDE in human adipose tissue. Low levels of decaBDE are found both in breast milk, but serum levels of decaBDE and lower brominated BDE in the breast-fed child and a 5 year old child was found to be higher than the levels in the parents.

There is also evidence from the feeding study with cows that nonaBDE congeners may be more accumulative than decaBDE. This should be taken into account since commercial decaBDE products typically contain up to 3% nonabromodiphenyl ether congeners.

(UNEP/FAO/RC/CRC.15/5 section 3.2.2 of the Norway notification)

In rats, oral absorption is reported to range from 1-26%, inhalation absorption is estimated to be negligible, and in an in vitro experiment dermal absorption was less than 20%. Furthermore an in vitro assessment using a human gastrointestinal tract model showed that decaBDE was bioaccessible (14%) after exposure to indoor dust samples. In the rat and cow the majority of decaBDE administered is recovered in feces as the original compound.

Studies have shown that decaBDE preferentially sequesters to blood-rich tissues such as muscle, liver, intestine, gills (fish), and to lesser extent to adipose tissue. The sequestration to blood rich tissues may possibly be explained by decaBDE binding to proteins.

In a bioaccumulation study on harbor seals, average hepatic ΣPBDE (tri- to octa-BDE) concentrations were similar to those of the average seal blubber ΣPBDE (mono- to hexa-BDE). In contrast, decaBDE concentrations in liver were up to five times higher than those in blubber, which is consistent with observations that decaBDE migrates to perfused tissues such as the liver in biota. In rats, based on organ fresh weights, the highest concentrations were found in adrenals, kidney, heart, liver and ovaries. In lactating cows fed naturally contaminated silage decaBDE was the dominating congener in feed, organs, adipose tissue and feces, but not milk. In dietary exposure of American kestrels, higher levels were observed in fat than liver on wet weight basis at the end of the depuration period.

Human data demonstrate that decaBDE is absorbed and distributed to fat, blood, cord blood, placenta, fetuses and breast milk. Maternal transfer of decaBDE to eggs and offspring has also been reported in fish, frogs, birds, rats and reindeer.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

2.2 Toxicology studies

The toxicity of decaBDE has been evaluated by ECHA, US EPA, ATSDR, Environment Canada and Health Canada, the UK Environment Agency, and EFSA.

2.2.1 Acute toxicity

DecaBDE exhibits a low acute oral, dermal and inhalation toxicity.

(UNEP/FAO/RC/CRC.15/5 Norwegian notification)

DecaBDE is not acutely toxic by the oral, dermal or inhalation routes.

Rat Oral LD50 > 2000 mg/kg bw.

Rabbit Dermal LD50 > 8000 mg/kg bw

Rat Inhalation LC50 > 48.2 mg/L, 2 hr exposure or > 48200 mg/m3

(ECHA registration dossier, accessed at: https://echa.europa.eu/registration-dossier/-/registered-dossier/14217)

2.2.2 Short term toxicity

Repeated dose toxicity

The subchronic and chronic oral toxicity of decaBDE is low. NOAELs of 7,000 mg/kg/day (in male mice) and 2,800 mg/kg/day (in male rats) were obtained in subchronic studies (90 days). In chronic studies (2 years), in mice, LOAEL of 3,200 mg/kg/day (in male) was established and in rats, NOAEL (systemic toxicity) of 1,120 mg/kg/day (in male) and LOAEL (local effects) of 1,120 mg/kg/day were established with only moderate effects observed either at this dose when LOAEL was established or at the just above tested dose when NOAEL was determined. (UNEP/FAO/RC/CRC.15/5 Norwegian notification)

2.2.3	**Genotoxicity (including mutagenicity)**	Mutagenicity Studies using different bacterial tests as well as in vivo experiments provided negative results. DecaBDE does not exhibit any cytogenetic effects in vitro or in vivo. (UNEP/FAO/RC/CRC.15/5 Norwegian notification)
2.2.4	**Long term toxicity and carcinogenicity**	Carcinogenicity In male mice signs of carcinogenicity were shown by increased incidence of hepatocellular adenomas or carcinomas (combined) in the low dose group and marginally increased incidence of thyroid gland follicular cell adenomas or carcinomas (combined) in both dosed groups. Several non-neoplastic lesions were observed at increased incidence, the most notable being thyroid gland follicular cell hyperplasia. In male and female rats some evidence of carcinogenicity as shown by a dose-dependent increased incidence of neoplastic nodules of the liver. (UNEP/FAO/RC/CRC.15/5 Norwegian notification)
2.2.5	**Effects on reproduction**	Developmental and reproductive toxicity No adverse effects have been observed in mice and rats exposed in utero or during lactation, but some studies indicate possible adverse effects on the reproductive system in male offspring. Several studies indicate that decaBDE disrupts the steroid- and thyroid hormone system. In frog tadpole metamorphosis, which is regulated by both thyroid hormones and glucocorticoid, is disrupted. Based on this study an aquatic NOEC of around 0.001 mg/L (1 µg/L) for delayed metamorphosis in Xenopus laevis tadpoles was indicated (ECHA 2012). In fish thyroid and possibly steroid hormone system was affected after 28 days exposure to ~10 µg decaBDE/ g food followed by a depuration period. Both reduced circulating T3 and T4, and deiodinase activity was observed. In rats modulation of sex steroid hormone pathway has been reported as reduced adrenal CYP17 activity in females, increased seminal vesicle/coagulating gland weights and increased expression of hepatic CYP1A and CYP2B in males after 28 days oral exposure to BDE-209. In mice exposed to BDE-209 from gestation day 0-17, anogenital distance, sperm-head abnormalities, and testicular histopathology were significantly affected in mice exposed to 1500 mg/kg. Sperm DNA damage, H202 generation, and vacuolization of interstitial cells in the testis was observed in mice exposed to 10 mg/kg. Vacuolization of Leydig cells indicates accumulation of hormone precursors. (UNEP/FAO/RC/CRC.15/5, Norwegian notification)
2.2.6	**Neurotoxicity/ delayed neurotoxicity, Special studies where available**	Behavioral disturbances observed in the mature rats and mice exposed to decaBDE as neonatales raise concern about possible developmental neurotoxicity in children. (UNEP/FAO/RC/CRC.15/5, Norwegian notification) The observation that exposure takes place already during the early phases of human development i.e. in utero via placental transfer and postnatal via mothers milk, support the notion that the developmental neurotoxicity observed in mammalian models could have implications also for humans. The risk for implications to human health is further underpinned by epidemiological data. Although having a limited number of individuals, studies have shown an association between BDE-209 levels in cholostrum and lower mental development scores in children 12-18 months of age, and that human prenatal or postnatal exposure to BDE-209 delays cognitions and potentially affects neurological development. Furthermore, several epidemiological studies support that exposure to PBDEs may result in human neurodevelopmental toxicity. Some human studies also observed associations between TH/TSH levels and exposure to BDE-209 or other high congeners such as BDE≥183. (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
2.2.7	**Summary of mammalian toxicity and overall evaluation**	There is no internationally adopted hazard classification for decaBDE. In the EU, there is no Harmonised Classification in Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation). However, according to the classifications provided by companies to ECHA in CLP inventory notifications this substance is harmful if swallowed, is harmful in contact with skin, is harmful if inhaled, causes serious eye

irritation, suspected of causing genetic defects, may cause damage to organs (liver, kidney) through prolonged or repeated exposure, and may cause long lasting harmful effects to aquatic life.

(UNEP/FAO/RC/CRC.15/5 section 3.1 of the Canadian notification)

DecaBDE is officially recognised in the EU as Persistent, Bioaccumulative and Toxic (PBT), and very Persistent and very Bioaccumulative (vPvB) and included on the Candidate list for authorisation as a substance of very high concern (SVHC) under REACH Regulation No 1907/2006 in December 2012.

IARC has categorised decaBDE as Not classifiable as to its carcinogenicity to humans (Group 3) (1990, 1999).

The toxicity of decaBDE to terrestrial mammals has mainly been investigated in rodents. Although several effects are reported including reproductive toxicity, data in particular point to neurodevelopmental toxicity and effects on the thyroid hormone (TH) system. Several studies have indicated epidemiological association for cognitive developmental effects in humans with decaBDE exposure.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

3 Human exposure/Risk evaluation

3.1 Food

PBDE exposure through food and the resulting serum levels have been investigated in Norway. In food samples analyzed for decaBDE high levels were found in eggs, vegetable oil, ice cream and biscuits, while the highest amounts were found in dairy products, which include milk, cheese, and butter. The calculated exposure to decaBDE was 1.5 (mean) and 1.4 (median) ng/kg bw per day, which is higher than the exposure to ΣPBDEs of other PBDE. Results indicate that intake of decaBDE and ΣPBDEs have different dietary sources and that dairy products proved to be the most important dietary source of decaBDE exposure. Serum levels of decaBDE were not analyzed in these samples.

Thomsen et al. 2007 found high levels of decaBDE (10 ng / g lipid) in pooled serum samples from Norwegian humans. A similar study detected an average of 2.26 ng / g lipid in plasma from pregnant women from the Bodø region (TA-2303). The reason for this large difference in decaBDE levels is not known. Thomsen et al. have previously reported decaBDE as the dominant congener of PBDE congeners analyzed and this was also confirmed in the study of women from Bodø. These results are much higher than found in a similar study on blood plasma from Swedish men.

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

BDE-209 is widely present in food and is reported in concentrations ranging from ~2 to >50,000 pg/g ww. The highest concentrations of lower brominated PBDE were generally measured in fish, and shellfish, while BDE-209 was found in sausage and dairy products, but also food wrapping may contribute.

BDE-209 has been measured in placental samples in concentrations ranging from 0.05 to 8.4 ng/g lw in a Danish and Spanish study, the median were 1.14 and 1.0 ng/g lw, respectively. Both studies reported BDE-209 to be the dominating PBDE, representing around 50% of the total PBDEs. A similar congener pattern was observed in a recent study from China, where prenatal placental concentrations were in the range of 1.33 to 8.84 ng/g lw (median 2.64 ng/ g lw). Biomonitoring studies on cord blood showed BDE-209 median concentrations to be in the range <1.2 to 27.1 ng/g lw. BDE-209 was in general the largest contributor to the sum of the PBDEs. Exposures to BDE-209 continue in early infancy due to its presence in breast milk. An extensive review by Frederiksen covered studies published until 2007 and showed that BDE 209 was reported in the concentration range of 0.1 to 2.9 ng/g lw. More recent studies report similar median concentrations, while maximum values vary considerably within and between geographical regions. BDE-209 concentrations in serum or plasma of adult populations with no known occupational exposure were shown to range from 1 to 18.5 ng/g lw. More recent studies show similar levels, except from the strikingly high levels (mean 220 ng/g lw) reported from Laizhou in China, a previous production area of halogenated flame retardants. One study from Sweden has assessed the concentration of BDE-209 in serum from first time mothers living in Uppsala sampled from 1996 to 2010. The mean of the 36 serum pools was

		1.3 ng/g lw and no significant temporal trend was seen. This is in accordance with the lack of time trend seen for breast milk collected at the Faroe Islands in 1987, 1994-5 and 1999. In summary, the biomonitoring data show widespread and ongoing exposures to BDE-209 throughout the world, and confirm fetal exposure and absorption in adults. Breast milk concentrations measured in Europe, China/Taiwan, Ghana and India were recently used to estimate mean daily intakes for breastfed infants ≤3 months. The intakes were similar, ranging from 1.0 (LB) to 13.8 (UB) ng/kg bw/day. Health Canada estimated the total intake of breast fed infants up to 6 months to be between 50-187 ng/kg bw per day, with dust contributing 40 ng/kg bw per day. (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
3.2	**Air**	Household dust and occupational exposure is thought to be the main sources for exposure to BDE-209 and other congeners present in c- decaBDE. Toddlers and infants have a higher daily intake of dust and dairy products than adults, and higher serum levels of BDE-209 have been found in children less than 5 years compared to their parents. PBDE congener composition was also different in the children compared to their parents indicating possible debromination to more bioaccumulative and toxic congeners (US EPA 2010). (UNEP/FAO/RC/CRC.15/5, Norwegian notification) Dust, indoor air and to a lesser extent food, are considered to be the most important sources and pathways for human exposure to PBDEs (US EPA 2010). In this assessment household consumer products were identified as the main source for the PBDEs in house dust. On the contrary a Canadian assessment identified food and dust as main sources for exposure in adults. Detected BDE-209 concentrations in the indoor air range from <LOQ to 651 pg/m3 and from 63 to 10,000 ng/g in dust from Germany, Sweden and the UK. The BDE-209 concentrations in dust exceeded by far the sum of the lower brominated PBDEs that have been detected. The concentrations of BDE-209 in North American house dust were comparable to those in Europe (<500-2000 ng/g). Further, the occupancy in cars and airplanes may be a significant source to PBDE exposure. The median levels of BDE-209 in dust from cars were about 20 times higher than in house dust, although the levels varied substantially between the studies. This is in line with a recent German study where the mean BDE-209 concentration in car, house, and office dust samples were 940, 45 and 120 ng/g, respectively. A correlation between BDE-209 in house dust and mother's milk is also reported, suggesting that BDE-209 levels in indoor environments have an impact on the exposure of breastfeeding children. A study from New Zealand estimated that BDE-209 intakes for infants aged 3 to 6 months was 11.7 ng/kg bw/day, while that in 6 to 12 months old children was estimated to 8.2 ng/kg bw/day. Children 1 to 2 years old had the highest estimated intakes of BDE-209 with 13.2 ng/kg bw/day which likely reflects the high dust ingestion rate (60 mg/day) for this group. The daily intake of BDE-209 and other PBDEs from dust and breast milk measured in this study was below US EPA Reference Dose values (7 µg/kg bw d). (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
3.3	**Water**	Contributions from drinking water and outdoor air to indirect BDE-209 exposure are low compared to intakes from food and often considered negligible. (UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)
3.4	**Occupational exposure**	Some professions are exposed to higher c-decaBDE concentrations than the average population and other workers. Foam recycling workers, carpet installer and PC technicians are reported to have higher serum levels of BDE-209 than control groups. In a Swedish study employees at a recycling plant and rubber mixers had higher levels of BDE-209 in serum than control. Samples taken during and after 5 weeks of vacation revealed that BDE-209 and other highly brominated PBDE congeners had lower half-life than the lower brominated congeners. (US EPA 2010). (UNEP/FAO/RC/CRC.15/5, Norwegian notification) Studies on occupational exposure are mostly from Scandinavia and Asia, where high-exposure occupational groups like electronic dismantlers have been the main

focus. In Sweden, the median BDE 209 blood level in electronic dismantling workers and computer technicians was reported to be 4.8 and 1.53 ng/g lw, respectively, while a median of 35 ng/g lw were reported among rubber workers. The widespread recycling and dismantling of e-waste under primitive conditions in China has received increasing attention. Median BDE-209 concentrations in Guiyu were 50-200 times higher than previously reported in the occupationally exposed populations in Sweden. The highest concentration of BDE-209 in human serum ever reported, i.e. 3,436 ng/g lw, is about 3,000 times higher than usually observed in general populations. In contrast, a recent study did not find significant difference between the residents in an e-waste recycling area and the reference group.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

3.5 Medical data contributing to regulatory decision

Not available.

3.6 Public exposure

Analysis of serum and plasma samples demonstrate that decaBDE is detectable across the general population in Norway.

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

The estimated mean dietary intake of BDE-209 for average consumers in Europe ranged from 0.35 (minimum lower bound (LB)1 to 2.82 ng/kg bw (maximum upper bound (UB)) per day. Based on a daily intake of 50 mg dust and a bw of 70 kg, EFSA estimated the exposure of adults to be 0.045 to 7 ng/kg bw per day. A review of exposure to PBDEs in the US showed that for BDE-209, soil/dust ingestion with 104.8 ng/day made the largest contribution to the exposure, followed by soil/dust through dermal contact (25.2 ng/day). The total exposure was estimated to 147.9 ng/day of which food and drinking water contributed only 16.3 and 0.09 ng/day, respectively. The total exposure corresponds to 2.11 ng/kg bw per day given a body weight of 70 kg as used by EFSA. Health Canada estimated the upper-bound total daily intake of BDE-209 to be 9.3 ng/kg bw for Canadian adults (20-59 years). Food and indoor dust were the dominant sources of exposure, contributing 51 and 45% to the total intake, respectively.

Several studies show that toddlers and young children have higher levels of PBDEs than adults, which was also seen for BDE-209. Small children, as a result of their behavior receive considerable PBDE doses from house dust. Assuming a daily ingestion of 100 mg dust the exposure for 1-3 year old children in Europe were estimated to range from 0.53 to 83 ng/kg bw per day, which is higher than the corresponding calculated median dietary intake ranging between 2.59 and 6.4 ng/kg bw. Health Canada estimated the daily BDE-209 intake for the age group 0.5 to 4 years to be 89 ng/kg bw of which diet and dust contributed 24 and 64 ng/g kg bw, respectively. Children's toys, specifically hard plastic toys, have been identified as a potential source of exposure of young children to c-decaBDE. This exposure was modelled in the assessment of oral intake of BDE-209 of Canadian children for the 0.5- to 4-year age group. The upper-bound estimate was 120 ng/kg bw per day, which was twice the exposure estimate from soil (dust) for this age group. Congener-to-congener correlations within the mother or toddler cohorts in a Swedish study suggested diet as an important exposure pathway for tetra to nonaBDEs for mothers. For infants breastfeeding was the predominant exposure pathway for tetra-to hexaBDEs and dust the most important exposure pathway for octa- to decaBDEs for toddlers. Despite some geographic differences, all available intake estimates for BDE 209 point out the importance of dust exposure, particularly for small children.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

3.7 Summary-overall risk evaluation

Norwegian authorities banned decaBDE based on its potential PBT properties and the general concern about the ubiquitous presence and increase of decaBDE in the environment including the Norwegian Arctic and a concern for presence of decaBDE in human matrices and human health. The concern for increased levels of persistent PBDEs due to continuously debromination from the pool of decaBDE in the environment.

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

The Japanese government designates chemical substances that are persistent, highly bioaccumulative, and have long-term toxicity for humans as Class I Specified Chemical Substances to be banned under the Chemical Substances Control Law (CSCL). As a result of internal evaluation using the scientific data in Japan, Japanese authorities concluded that this chemical met the criteria to be designated as a Class I Specified Chemical Substance under the CSCL.

The Japanese notification also states that BDE-209, the main component of c-decaBDE, exerts reproductive, developmental, endocrine and neurotoxic effects in aquatic organisms, mammals and birds. Effects on growth, survival and mortality are also reported.

(UNEP/FAO/RC/CRC.15/5, Japanese notification).

A risk characterization and a hazard and dose-response assessment of BDE-209 suggested that the daily intake of BDE-209 in the USA and Canada was not likely to result in neurodevelopmental toxicity for infants. EFSA also concluded that current dietary exposure or the intake of BDE-209 by breast-fed infants does not constitute a health concern in the EU. Among the four PBDEs (BDE-47, BDE-99, BDE-153 and BDE-209) investigated by EFSA, a potential health concern with respect to current dietary exposure was only identified for BDE-99. A recent PBDE risk assessment based on oral, dermal, and inhalation exposure of infants 0-5 years of age, indicates no risk for adverse health effects in infants that are restrained in a car seat. However, these assessments do not consider the possibility that several PBDEs could act in concert, inducing additive or synergistic effects as suggested by the available in vitro data, or that there may be multiple sources of exposure.

While the mixture toxicity of BDE-209 and other PBDEs have not been studied experimentally to a large extent, a combination of BDE-47 and -99 was observed to induce synergistic cytotoxic effects in neuronal cells. Furthermore, a mixture of PBDE congeners (BDE-47, -99, -100 and -209) at levels detected in human blood had irreversible effect on hormone secretion in ovarian follicles. The results from this study suggest that combined effects of PBDEs may be much larger than indicated by the sum of the effects of the individual congeners. The presence of other POPs may also affect the toxicity of PBDEs. In an in vitro study with binary mixtures of PCBs and PBDEs (BDE-47, -153, -183, or 209), it was found that PCB-126 and PBDEs could mutually inhibit each other while PCB-153 and PBDEs jointly could exacerbate the observed biochemical alterations. PBDEs are considered to be potential endocrine disruptors which may act additively at low concentrations.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4 Environmental fate and effects

4.1 Fate

The environmental fate properties of BDE-209 have been assessed in various reports published by the EU, Canada and the United Kingdom. Fugacity modeling predicts that most of the BDE-209 (> 96%) in the environment partitions to sediment and soil. Less than 3.4% of BDE-209 is expected to be associated with bulk air or bulk water phases. Given its low water solubility and strong particle affinity, its mobility in soils is also likely to be low. Debromination of BDE-209 to lower brominated PBDEs in environmental matrices and biota has important implications for the risk from c-decaBDE imposed to the environment, due to the PBT, vPvB and POP properties of its metabolites.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.1.1 Soil

In Norway, high levels of BDE-209 were detected in sediments and BDE-209 represented up to 90% of ΣPBDEs (TA-2252). A study conducted in Lake Mjøsa in the Southern part of Norway revealed that BDE-209 was the dominant congener (50-90%) in sediments and waste water in many areas (TA-2104).

DecaBDE (BDE-209) dominates completely in all sediment samples, representing more than 97% of ΣPBDE at all sediment sites in Asefjorden and surrounding areas in the western part of Norway (TA-2146).

Sediment samples from outside a marina downstream (Muusøya, close of the city of Drammen, southern Norway) showed significantly elevated concentrations with a high percentage of BDE- 209. In all the fish samples from the inner Drammensfjord was BDE-209 detected. The concentrations were in general low (0.1-20% of ΣPBDE), but the results are in accordance with new knowledge about BDE-209 as a bioavailable substance (TA-2051).

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

PBDEs have been detected in sediment and soil samples collected in North America, and high concentrations have been measured in sewage sludge. PBDEs have been measured in sediments from Lake Ontario tributaries flowing to Lake Ontario. The total PBDEs (tri-, tetra, penta-, hexa-, hepta- and decaBDEs) measured in sediment samples taken from fourteen tributary sites (6 reported) ranged from approximately 12 to 430 µg/kg dw. Of the reported sediment results, concentrations of tetra- to hexaBDEs ranged from approximately 5 to 49 µg/kg dw. Concentrations of BDE-209 (decaBDE) ranged from 6.9 to 400 µg/kg dw. BDE 47, 99 and 209 were the predominant congeners measured in sediments. Measured PBDE concentrations (sum of 8 di- to pentaBDE congeners) ranged from 2.7 to 91 µg/kg organic carbon (OC) in 11 surficial sediments collected in 2001 from several sites along the Columbia River system in south eastern British Columbia.

(UNEP/FAO/RC/CRC.15/5, Canadian notification)

Most available data reporting BDE-209 levels in soil are from affected areas. Reported levels in soil worldwide range from non-detectible up to 8600 pg g^{-1} dw soil in polluted areas, but may possibly be even higher. BDE-209 was detected in soil at landfill sites in Arctic Canada, but PBDE soil levels outside the landfills were similar to levels measured in soil at background locations elsewhere in the Arctic, suggesting that emissions of BDE-209 and other PBDEs from these sources to the Arctic environment at present are small. Compared to remote sites, BDE-levels in urban and rural areas are significantly higher. In particular, the levels of BDE-209 in soil at e-waste sites such as recycling plants, dumping- and industrial sites in China are very high. Sewage sludge from several countries is reported to contain BDE-209 and when soil is amended with sludge BDE-209 is transferred to soil and biota. Levels of BDE-209 were 100-1000 fold higher at sites fertilized with sewage sludge compared to reference sites. In this study, BDE-209 was the dominant congener in soil and earthworms, with higher levels reported in the worms than in the soil.

Reported BDE-209 levels in sediments worldwide range from non-detectable to 16,000 ng/g dw i.e. slightly higher than in soil. High concentrations in sediment are typically found in the vicinity of industrial sites. Similar to findings in soil, BDE-209 is the predominant congener reported in sediments contributing almost 100% to the total PBDE measured in some studies. Levels of BDE-209 in soil and sediment in remote regions are low, but have been found to be elevated at a few sites affected by local contamination such as at landfills and in the vicinity waste water outfalls. The doubling time for BDE-209 in these sediments range between 5.3 and 8.4 years.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.1.2 Water

DecaDBE has very low solubility in water. Due to the lack of any functional groups that are readily susceptible to hydrolysis and a very low water solubility of BDE-209, < 0.1 µg/L at 25 °C, hydrolysis is unlikely to be a relevant degradation process in the environment.

Estimations of half-lives in water are generally complicated by the poor water solubility of BDE-209 and are highly dependent on the experimental conditions. Yet, when correcting for the use of solvents and taking into account natural light conditions, environmental half-lives ranging from a few hours up to 660 days in water have recently been suggested.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.1.3 Air

Measured total PBDEs (sum of 21 congeners) ranged from 10 to 1300 pg/m3 in air samples collected at a rural southern Ontario site in early spring of 2000. Total PBDEs (congeners not specified) up to 28 pg/m3 were detected in air samples from the Canadian Arctic collected over the period 1994-1995.

(UNEP/FAO/RC/CRC.15/5, Canadian notification)

Several studies have reported that BDE-209 is the predominant or one of the dominating PBDEs in Arctic air. The levels of BDE-209 in the Arctic atmosphere together with studies showing a significant deposition on Arctic ice and snow underlines the potential of BDE-209 to undergo long-range environmental transport to remote regions. For example, in a study assessing a total of 19 different BFRs in ice core samples from the Norwegian Arctic, BDE-209 was found to provide the second greatest share of the deposition of BFRs from air to the Arctic ice. The deposition rate for BDE-209 was found to be 320 pg cm^{-2} y^{-1} in the period 1995 2005, surpassed only by hexabromocyclododecane (HBCD), and substantially higher than for other PBDEs. The detection of BDE-209 in Antarctic air and deposition samples provide further evidence of the long-range transport of this compound over remarkable long distances.

BDE-209 is also found in air in remote areas of Asia on the Tibetan Plateau. Snow pack samples in the Tartra Mountains in Slovakia showed remarkably high levels of BDE-209. Systematic monitoring at open sea from ships has also proved the abundance of BDE-209 in air samples from the Arctic, Atlantic, Indian, and Pacific oceans. Both oceanic and atmospheric processes contribute to the environmental transport of BDE-209. Since BDE-209 has a very low vapor pressure, volatilization is unlikely to contribute significantly to the long-range environmental transport, rather the atmospheric long-range transport appears to be controlled by the atmospheric mobility of the particles to which it is attached. Finer particles (with a diameter around a few micrometres) might remain airborne for hours or days, provided that they are not removed by wet deposition. Furthermore, particles can protect the BDE 209 molecule from photolysis and lengthen its life-time in the air to >200 days. In the Arctic, the deposition of airborne particles is found to be higher during the Arctic haze season. In tropical Asia, long-range environmental transport of PBDEs including BDE-209 associated with gas and/or particles is assisted by the monsoon.

BDE-209 is detected in air in urban, rural and remote regions, as well as in precipitation. In urban and rural environments detected levels range between 4.1 and 60 pg m^{-3} while concentrations in Arctic air ranges from non-detectable to 41 pg m^{-3}. Levels in background locations outside the Arctic have been reported to range from non-detectable to 29 pg m^{-3}, i.e. higher than the levels found in the Arctic and lower than the levels found in urban and rural environments. Recently, however, particle-bound and gas-phase BDE-209 concentrations measured in tropical Atlantic Ocean air were as high as 43.89 and 260 pg m^{-3}, respectively. Based on these measurements, the total deposition of BDE-209 to the Atlantic Ocean from air was calculated to be approximately 27.5 tonnes annually, 20 and 7.5 tonnes each for the gas- and particle phase, respectively. The findings indicate that air-levels and deposition over the global oceans may be higher than previously thought.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.1.4 Bioconcentration and bioaccumulation

Bioaccumulation

BMFs >1 were found for the following predator/prey combinations: walleye/white suckers (BMF 2.0), walleye/white fish (BMF 6.8), emerald shiner/zooplankton (BMF 33), white suckers/zooplankton (BMF 9.9), burbot/emerald shiner (BMF 2.4), burbot/mussels (BMF 1.9), white fish/zooplankton (2.9) and goldeye/zooplankton (BMF 34). Furthermore, a BMF of 2.2 was found from polar cod to harbor seals.

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

Overall, available data do not show that the decaBDE itself meets the numeric criteria for bioaccumulation as defined in the Persistence and Bioaccumulation Regulations. With regard to bioaccumulation, potential factors such as low assimilation efficiency and/or metabolic transformation appear to be important determinants of accumulation in organisms. Nevertheless, some recent studies have shown concentrations of decaBDE to be increasing steadily in some wildlife species and there are a few

equivocal reports of BMFs exceeding 1. In some cases, such as in the tissues of kestrel, sparrowhawk, peregrine falcon, glaucous gull, red fox, shark, harbour porpoise and whitebeaked dolphin, measured concentrations of decaBDE in tissues are interpreted as high. While trophic magnification or bioaccumulation is a potential explanation for these high concentrations, it is also very possible that some biota are exposed to very high exposure concentrations of decaBDE by consuming contaminated refuse and/or inhabiting decaBDE hotspots close to industrialized areas.

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

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

(UNEP/FAO/RC/CRC.15/5, Canadian notification)

BDE-209 is found in elevated concentrations in top predators.

Log Kow ranges between 6.27 and 12.11.

BAF>5000 and BMFs>1 in aquatic organisms.

BMFs >1 in terrestrial organisms.

TMFs> 1 in Arctic aquatic organisms.

BDE-209 debrominates to lower brominated PBDEs that are known to bioaccumulate (i.e. with PBT/vPvB properties or are POPs).

Toxic effects are observed at low and/or environmentally relevant concentrations in birds, fish and frog.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.1.5 Persistence

Based on the information in the original risk assessment reports (EC 2002 and updates) decaBDE is likely to be highly persistent in the environment, and so is considered to meet the vP criterion. This conclusion is further supported by monitoring data, which show that the substance is widely distributed in the environment, and present in the Arctic. The slow rate of degradation could nevertheless still lead to the formation of other substances that are of concern (including some that are considered to meet the PBT/vPvB criteria).

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

There are a variety of data indicating that all PBDE congeners subject to the Canadian assessment are highly persistent and each satisfies the requirements for persistence as defined by CEPA Persistence and Bioaccumulation Regulations. Although uncertainty regarding the possible transformation products of decaBDE exists, there is sufficient evidence to conclude that some level of decaBDE phototransformation likely occurs in the environment and that lower brominated PBDEs are being formed during this process. These products are likely to be more bioaccumulative than the

parent compound and could be considered persistent and may be directly toxic to organisms. It is expected that decaBDE in the environment would mainly sequester into sediment or soil and this could limit the amount available for photodegradation, but it could make some amount available for transformation via other processes such as anaerobic biodegradation or reaction with reducing agents. Overall, it is very difficult to determine the extent to which the transformation of decaBDE in the environment may contribute to the potential accumulation of lower BDEs and other products. Nevertheless, it is reasonable to consider that various transformation processes could contribute to the formation of at least some amount of lower brominated PBDEs and polybrominated dibenzofurans (PBDFs).

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

(UNEP/FAO/RC/CRC.15/5, Canadian notification)

Dated sediment cores indicate no degradation of BDE-209 over a period of almost 30 years.

Reported BDE-209 half-life in sediment range between 6 and 50 years, with an average of around 14 years at 22°C and under dark conditions.

BDE-209 degradation half-life in sludge-amended soil under aerobic and anaerobic conditions >360 days.

No degradation of decaBDE after 180 days in soil samples spiked with BDE-209.

Temporally increasing concentrations is observed in some organisms and support the picture of BDE-209 as a persistent substance.

BDE-209 debrominates to lower brominated PBDEs that are known to be persistent (i.e. with PBT/vPvB properties or are POPs).

Based on hydroxyl radical reaction BDE-209 has an estimated atmospheric half-life of 94 days in air according to calculations from the chemical structure using the Syracuse Research Corporation AOP program and assuming a hydroxyl radical concentration of $5x10^5$ molecule cm^{-3} and a reaction rate of $1.7x10^{-13}$ cm^3 $molecule^{-1}$ s^{-1}. Other applications such as EPISuite 4.1 (AOPwin module) and PBTProfiler estimate a different reaction rate ($3.37x10^{-14}$ cm^3 $molecule^{-1}$ s^{-1}) and therefore predict even longer half-lives of 317 days (12 h day, 1.5×10^6 OH radicals cm^{-3}) and 470 days (24 h day, $5x10^5$ molecules cm^{-3}) respectively.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.2 Effects on non-target organisms

4.2.1 Terrestrial vertebrates

DecaBDE is present in many types of aquatic and terrestrial wildlife species also in vulnerable stages such as bird eggs. The debromination ability of species tested raise concern for production of more toxic and bioaccumulative congeners. Although the available ecotoxicity data from controlled laboratory study for the decaBDE indicate no adverse toxicological effects in terrestrial organisms tested, and it was considered unlikely that significant acute or chronic toxic effects would occur in aquatic organisms at concentrations up to the water solubility limit, some studies show effect

on behavioral of fish and birds that might reduce their ability to reproduce and survive in the environment.

Several studies indicate that decaBDE disrupts the steroid- and thyroid hormone system. In frog, tadpole metamorphosis, which is regulated by both thyroid hormones and glucocorticoid, is disrupted. Based on this study an aquatic NOEC of around 0.001 mg/L (1 µg/L) for delayed metamorphosis in Xenopus laevis tadpoles was indicated (ECHA 2012).

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

Toxicity in terrestrial mammals

The toxicity of c-decaBDE to terrestrial mammals has mainly been investigated in rodents. Although several effects are reported including reproductive toxicity, data in particular point to neurodevelopmental toxicity and effects on the TH-system. In addition, available scientific evidence suggests that BDE-209 either alone or in concert with other PBDEs could act as a developmental neurotoxicant in terrestrial mammals and humans.

Developmental neurotoxicity is the reported critical endpoint of several PBDEs. Several mechanisms for developmental neurotox effects are proposed, for instance impaired thyroid homeostasis, direct toxicity to neuronal and stem cells, and disturbing neurotransmitter systems. Developmental neurotoxicity has also been reported for BDE-209 in some studies but not by others. Neurodevelopmental effects of BDE-209 are reported in mice at dose levels relevant for pregnant women. Neurobehavioral effects of BDE-209 in rodents during juvenile development or adulthood have also been reported more recently. For example, long-lasting effects in spatial learning and memory were observed in transgenic mice after postnatal exposure to BDE-209 and reduction in anxiety levels and delayed learning in spatial memory tasks were found in wild type mice. In another study a single dose of BDE-209, administered orally at post natal day 10, was also observed to cause long-lasting effects on emotional learning and TH-levels in mice carrying two variants of apolipoprotein E, apoE2 and E3. Moreover, prenatal BDE-209 exposure in rats impaired learning acquisition in a dose dependent manner, and in vitro data suggested that this impairment in rat learning acquisition may be linked to effects on brain neurogenesis.

The majority of developmental studies with BDE-209 used oral administration, but only a few were designed according to the OECD 426 guideline “Developmental Neurotoxicity Studies”. In mice and rats administered a single dose of BDE-209 during the “brain growth spurt” period consistent and persistent alterations in behavior, habituation and memory were observed. Other researchers have noted limitations with the former studies, in particular for not using the litter as a basis for the statistical evaluation. Despite this, the US EPA used the studies from Eriksson and Viberg in their derivation of oral reference doses for BDE-209. A study did not show a consistent depression in motor activity over time in mice, however a follow-up study showed neurobehavioral long-lasting deficits when tested at 16 months. Similar to the findings of Viberg and Johansson, behavioral effects from developmental BDE-209 exposure appeared to get worse with age. Additional evidence for neurodevelopmental effects of BDE-209 come from several publications that indicate that PBDEs affect the cholinergic system in both mouse and rat brain which could lead to disturbed cognition (learning and memory). In further support of findings indicating that BDE-209 can act as a neurotoxicant in mammals, it has been shown that BDE-209 exposure resulted in reductions in the neural connections between the left and right brain hemispheres (the corpus callosum area) and that it caused irreversible white matter hypoplasia targeting oligodendrocytes in rats. This effect was accompanied by developmental hypothyroidism. In contrast, no clinical signs, or any neurobehavioral changes, effects on startle response, or learning behavior were reported at any dose level by Biesemeier, where motor activity and behavior of BDE-209 exposed rats was assessed at two, four, and six months of age. The Biesemeier study has, however, since been critically evaluated by Shibutani who noted the omission of measurement of thyroid-related effects, histopathological parameters on neuronal migration, oligodendroglial development, discussions of the significant decreases in the hemisphere height and decrease in the pons and cortex

vertical thicknesses. The Biesemeier study has also been discussed in the Health Canada report, where lower LOAEL and NOAEL values have been suggested instead of the value reported in the original study.

Other studies show that BDE-209 can exert direct toxic effects on neuronal cells and interfere with neuronal signalling, neuronal development and induce oxidative stress and apoptosis effects that may lead to neurotoxicity and interfere with learning and memory by affecting long-term potentiation. BDE-209 is further shown to cause changes in gene expression, intracellular protein levels, and disturbance of synaptogenesis and cell differentiation.

In addition to neurotoxic effects available data point to BDE-209 and lower brominated PBDEs as potential endocrine disruptors. PBDEs structurally resemble THs, and as indicated earlier, effects on the TH system (TH: T4 and T3), along with the above mentioned and more direct toxic effects to neuronal cells is suggested as underlying mechanisms of BDE-209 and PBDE neurotoxicity. In vitro and in vivo studies assessing TH/TSH effects due to BDE-209 administration show that BDE-209 and other PBDEs interfere with the TH-system, but the results on BDE 209 or c-decaBDE mixtures are not consistent in terms of what effects are observed. For instance, whereas most animal studies report decreased T3 levels following high BDE-209 exposures, also no change, and increase in T3 levels has been reported. For T4, animal studies report both decreases in T4 levels at high dose as well as no change in T4 level. For TSH, two animal studies performed with BDE-209 both report increased TSH levels at the highest BDE-209 exposures while no effects were reported in adult rats dosed with commercial c decaBDE mixture DE-83R at doses of 0.3-300 mg/kg/day for four days. Repeated dietary administration of BDE-209 (at a high dose) induced thyroid follicular cell hyperplasia in male mice but not in female mice or in either sex of rats. Studies reporting significant changes in TH/TSH levels in rats and mice have often administered BDE-209 at doses that are orders of magnitude higher than human exposures. However, studies on rodent offspring have indicated that low doses of BDE 209 may adversely affect the developing thyroid organ. The recent WHO/UNEP report (2013) concluded that endocrine disruptors can cause adverse effects at low environmental levels, may display non-monotonic dose-responses, and that the timing of exposure can be more critical than the level of exposure. Thus, the observed inconsistencies in reported TH/THS effects may possibly, at least in part, be explained by differences in the experimental conditions used in these studies.

Studies suggest that in utero exposure to BDE-209 at high parental doses may cause reproductive toxicity and lead to developmental abnormalities such as decreased anogenital distance and testicular histopathological changes, sperm-head abnormality, and sperm chromatin DNA damage. Effects on testicular development has also been reported following exposure at post natal days 1-5 at low doses. Reported low-dose effects (0.025 mg/kg, subcutaneously) included reduction in testicular weight, sperm count, elongated spermatid and sertoli cell numbers as well as changes in protein expression and phosphorylation status. Also, possible modulation of sex steroids in the male and female genital system cannot be entirely ruled out. In contrast, no reproductive toxicity was observed in Sprague-Dawley pregnant female rats exposed to BDE-209 from gestational day 0-19. Similarly, it was reported that a mixture composed of three commercial BDEs (52.1% DE-71, 0.4% DE-79, and 44.2% decaBDE) affected liver and thyroid physiology but not male reproductive parameters in exposed rats. Yet, in female mice adrenals, decreased activity in the dehydroepiandrosteron synthesis assay was observed indicating reduced CYP17 enzyme activity and potential effects on steroid hormone production. Further, BDE-209 can inhibit estradiol-sulfotransferase in vitro, which could implicate a (local) increase of endogenous estradiol in vivo. In another in vitro study, it was found that BDE-209 exposure led to increased testosterone-, progesterone- and estradiol secretion in porcupine ovary cells, a finding that suggests that BDE-209 can induce preterm luteinization in antral follicles followed by the disruption of ovulation.

Oxidative stress and impaired glucose homeostasis have been reported in rats exposed to BDE 209. Dose-related fasting hyperglycemia was observed in adult rats exposed to BDE-209 (0.05 mg/kg) for 8 weeks. Reduced insulin levels and increased levels of tumor necrosis factor- (TNF-alpha) were observed in plasma followed by reduction in the oxidative stress markers glutathione and superoxide dismutase. Dose-dependent

morphological changes such as blurring boundaries among pancreatic islet cells were observed. Insulitis was also observed in male rats in a 28-days exposure study however, no differences were observed between the exposure groups. Similar to the reported effects on the steroid and TH systems the observed effects on glucose homeostasis/ insulin levels are suggestive of the endocrine disruptive potential of BDE-209.

Immunotoxic effects of BDE-209 have been reported in some studies, although immunotoxicity is not regarded as a critical toxic endpoint of PBDEs in general. In the most recent of the studies showing that BDE-209 can act as an immunotoxicant, reduced qualitative and quantitative CD8 T-cell response was observed in mice after long-term BDE 209 exposure. In contrast, another study reported no immunotoxic effects on the T cells in rats.

Gene mutations are suggested not to occur after exposure to BDE-209 or other PBDEs, although recent studies have indicated that BDE-209 may cause DNA damage through the induction of oxidative stress in vitro. There is limited evidence for carcinogenicity of BDE-209 in experimental animals. According to the NTP report (1986) there is some evidence at high dose levels for an increase in liver adenoma in rats and liver adenoma and carcinoma in mice, but this may be related to a secondary mode of action.

Toxicity in birds

Birds exhibit some of the highest concentrations of BDE-209 reported in wildlife and may be at risk for experiencing adverse effects. However, a limited number of studies examining adverse effects of BDE-209 exposure to birds are available.

In a study on swallows nesting at a WWTP, a positive relationship between egg size and BDE 209 levels were found, however, no significant correlation was found for reproductive parameters. BDE-209 concentrations were not reported.

Sifleet (2009) observed a mortality of up to 98% in embryos of captive chicken injected with a single dose of 80 µg BDE-209 /egg and exposed for 20-days via the yolk sac. The reported LD50 from this study was 44 µg/egg (740 µg kg ww). An assessment undertaken by the EU, revealed that the BDE 209 concentrations typically found in wild bird eggs are around 2-10 times lower than the concentrations that according to Sifleet (2009) induce mortality. Reported concentrations in bird eggs typically range between 1-100 µg/kg ww, but up to 420 µg/kg ww have been reported. In spite of important study limitations, the EU risk assessment indicated that the margin between exposure levels in wild birds and observed effect levels is not high, especially considering that Sifleet (2009) did not take into account potential sub-lethal effects, and that additional BDE-209 would likely have been assimilated following hatching and desorption of the remaining yolk thereby further increasing exposure.

A reduction in body mass was observed in European starlings exposed to BDE-209 by silica implants.

Birds are reported to metabolize BDE-209 to lower brominated PBDEs, including some POP BDEs (BDE-183) and exposure to lower brominated PBDEs have been associated with immunomodulatory changes, developmental toxicity, altered reproductive behavior, reduced fertility and reproductive success. In a study on captive American kestrels exposed to DE-71, a commercial penta-PBDE mixture, at environmentally relevant levels in ovo, the low BDE-209 levels present (<2.5 %) was found to be associated with an increase in flight behavior of male kestrels both in the courtship period as well as during brood rearing later in life). The BDE-209 concentrations measured in this study were not reported. These findings suggest that BDE-209 like other PBDEs may affect behavior in birds and is consistent with research on laboratory rodents where some studies report that BDE-209 causes changes in spontaneous behaviour.

Available studies also suggest a risk to birds arising from exposure to a combination of different PBDEs and other environmental pollutants. In a field study concentrations of the hexa-, hepta-, octa- and BDE-209 congeners (BDE-154, -183, -201 and -209) in liver and BDE 209 in plasma of male ring-billed gulls breeding in the urbanized Montreal region were negatively correlated with trabecular and cortical

bone mineral density of the tarsus. The finding suggests that the PBDEs at the levels reported in these birds (i.e. liver BDE-209 2.74-283 ng /g ww and ΣPBDE 26.2 680 ng/g ww, plasma BDE-209 0.70-19.1 ng/g ww and ΣPBDE 3.55-89.2 ng/g ww) can negatively affect bone tissue structure and metabolism in birds. In another study, the combined effects of several organochlorine pesticides, PCBs and PBDEs including BDE-209 and several nonaBDEs were postulated to have contributed to the death of weakened individuals of glaucous gull found in the breeding seasons 2003-2005 on Bjørnøya in the Barents Sea. However, BDE-209 was only detected at very low concentrations in liver and brain (<MDL-2.6 and <MDL-0.01µg/g lipid, in liver and brain respectively), along with other PBDEs, POPs and mercury. In relation to the study that shows effects on bone tissue it is worth noting that common kestrels in urban areas in China were reported to have mean liver concentration of 2870± 1040 ng/g lw of BDE-209 and levels ranging from 4.46 to as high as 1710 ng/g lw have been reported in house sparrows from Helgeland, a remote/ rural site in Norway. However, as these studies report BDE-209 concentration on a ng/g lw basis they are not directly comparable to the findings that reported BDE-209 concentrations in ng/g ww.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.2.2 Aquatic species

In animal studies of amphibian, fish and rodents exposed to BDE-209 at vulnerable stages such as the developmental phase, effects on hormonal axis as the thyroid and steroid is of concern. Although the toxicology data of BDE-209 is ambiguous, some studies indicate negative effect on neurological development at low doses.

Recent laboratory work with fish shows that decaBDE can be metabolised in the liver to form lower PBDE congeners, with typically nona- to hepta- or hexabromodiphenyl ethers being found (some of which are considered to be vPvB substances). The extent of metabolism appears to vary amongst different fish species.

In frog, tadpole metamorphosis, which is regulated by both thyroid hormones and glucocorticoid, is disrupted. Based on this study an aquatic NOEC of around 0.001 mg/L (1 µg/L) for delayed metamorphosis in Xenopus laevis tadpoles was indicated (ECHA 2012). In fish thyroid and possibly steroid hormone system was affected after 28 days exposure to ~10 µg BDE-209/ g food followed by a depuration period. Both reduced circulating T3 and T4, and deiodinase activity was observed. The observed effects on fish thyroid hormone pathway and effects on tadpole metamorphosis are of concern.

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

DecaBDE has limited water solubility, and early hazard assessments suggested that significant acute or chronic toxic effects was not likely to occur in aquatic organisms at concentrations below water solubility. However, the most recent EU assessment of BDE-209 raised a concern for adverse effects also to aquatic organisms based on new studies documenting effects on important biological endpoints including reproduction, development, nervous system, endocrine system, growth and fitness.

Aquatic toxicity studies have revealed a number of effects on aquatic organisms, mostly fish and amphibians. Through their influence on the TH system, PBDEs including nonaBDE and BDE-209, was shown to have the potential to affect development and metamorphosis in amphibians. According to the available studies BDE-209 and BDE-206, which is one of the congeners present in c-decaBDE and a possible degradation product of BDE-209, can delay metamorphosis in African Clawed Frog tadpoles. A significantly reduced tail tip regression was observed following BDE-206 exposure of tails ex vivo. In a more recent in vivo study, a c-decaBDE (DE-83R) consisting of 98.5% w/w BDE-209 was reported to affect metamorphosis in African Clawed Frog tadpoles by delaying the time to forelimb emergence. The delayed forelimb emergence was accompanied by histological changes in the thyroid gland and reduced expression of the thyroid receptor in tail tissue. Based on this study an aquatic NOEC of around 0.001 mg/L (1 µg/L) for delayed metamorphosis in African Clawed Frog tadpoles was indicated. Studies have also demonstrated that BDE-209, following flow-through exposure to 0 ppb, 0.1 ppb, 10 ppb, and 100 ppb for 12 weeks, can alter the anatomy and function of the African Clawed Frog vocal system by affecting the laryngeal motor neurons when animals are exposed during the androgen sensitive critical period of vocal system development

and during adulthood when the tissues are utilizing androgens to vocalize. BDE-209 also inhibited male typical vocalization, a critical aspect of mating behavior by reducing the number of calls elicited as well as the average call amplitude. The data suggest that BDE-209 can alter anatomy and function, mediated through pathways that include blocking the androgens necessary for proper vocal system. These findings may be of concern given that wild frogs are exposed to BDE-209 already at the egg stage and that BDE-209 in frogs also is transferred to brain and testis.

In fish, controlled feeding studies with fathead minnows conducted at environmentally relevant concentrations have shown that BDE-209 either alone and/or in combination with its debromination products may interfere with the TH system in adult and juvenile fathead minnow. In the latter study, adult fish dietary exposed to a low dose of ~3 ng/g BDE-209 bw per day for 28 days showed a 53% and 46% decline in circulating total thyroxine (TT4) and 3,5,3'-triiodothyronine (TT3), respectively, compared to controls. In fish exposed to a high dose of 300 ng BDE-209/g bw, the levels of TT4 and TT3 were lowered to 62 and 59%, respectively. Both in high and low-dose exposed fish, TH levels remained supressed after a 14-day depuration period. Both doses also reduced brain deiodinase activity (T4-ORD) with 65% compared to control. BDE-209 also has the potential to cause adverse effects in zebrafish at early life stages with impacts on T3 and T4 concentrations. Changes in expression of TH-associated genes were observed in rare minnow larvae and adults following exposure to 0.01- 10 µg/L BDE-209 via water for 21 days. In contrast to these findings, other studies report no visible effect on thyroid function in exposed fish embryos. However, it should be noted that in one study, where embryos were exposed 48 hours post fertilization to 960 µg/L BDE-209 for three days, it was concluded that the assay used, a T4 immunofluoresence quantitative disruption test, was not suitable for detecting effects of chemical pollutants such as BDE-209 that indirectly disrupt thyroid gland function. Another study speculated that the absence of effects on the TH-system in the study may be explained by shorter exposure and/or lower doses than those used in other studies. Potential TH disruption in fish by several PBDEs was also investigated in vitro with negative results. In this study neither BDE-209 nor BDE-206 showed any binding to sea bream transthyretin (TTR), a TH binding protein in the blood. The result suggests that BDE-209 likely does not interfere with binding of TH to TTR.

Other effects, both chronic and acute, have also been observed in fish following exposure to BDE-209 including a significant increase in percent cumulative mortality, and a decline in gonadal-somatic index. Significant decreases in body weight and survival rate of zebrafish larvae exposed to 1.92 mg/L BDE-209 via water for 14-days were also observed. Significant changes were not observed at any of the lower exposure doses tested (0, 0.08, 0.38 mg/L).

Based on measurements of otolith increment widths in juvenile lake whitefish (~5 months old) fed BDE-209-spiked diets (control, 0.1, 1, and 2 µg/g-diet) there were indications that BDE-209 may affect growth rates in fish at environmentally relevant levels of BDE-209 found in sediment.

Effects on overall fitness, reproductive parameters and behavior as well as motor neuron and skeletal muscle development in a low dose chronic toxicity study with zebrafish were documented. Several of these effects were trans-generational i.e. they were observed in offspring of exposed parents and are according to the authors likely explained by maternal transfer of BDE-209. In male fish, indicators of sperm quality were significantly affected even at the lowest exposure dose (0.001 µM or 0.96 µg/L).

Potential reproductive toxicity of BDE-209 was also demonstrated in rare minnow. Reduction of spermatocytes and inhibition of spermatogenesis was demonstrated in adult rare minnow exposed to 10 µg BDE-209/L via water. Changes in the expression of TH and spermatogenesis associated genes in rare minnow larvae and adults were observed following exposure to 0.1-10 µg BDE 209/L. In addition, effects on body length and gonadosomatic index of adult females were observed at 10 µg/L, but no significant histological changes were found in the ovary at any of the concentrations tested. Furthermore, no change in mortality or body length of larvae and adult males was observed.

BDE-209 impacted expression of neurological pathways and altered the behavior of zebrafish larvae, although it had no visible effects on TH function or motor neuron and neuromast development. In this study fish were exposed to BDE-209 spiked sediment, at a concentration of 12.5 mg/kg. Concentrations in exposed larvae and solvent control measured after 8 days were 69.6±9.8 ng/g ww and 6.7±0.5 ng/ g ww, respectively.

Besides the other effect reported above, BDE-209 was shown to induce oxidative stress in the liver of goldfish. A reduction in glutathion level and in the activity of antioxidant enzymes, (glutathione peroxidase, superoxide and catalase) was observed from 7-30 days after a single intraperitoneal injection of 10 mg/kg.

In several of the above fish studies BDE-209 was reported to debrominate to lower brominated PBDEs, thus it is possible that other PBDE congeners besides BDE-209 contributed to the effects reported in these studies. Reported debromination products included nona-, octa-, hepta-, hexa- and pentaBDEs.

In summary, the lowest aquatic NOEC for exposure via water reported appears to be below 0.001 mg/L (1 µg/L) and was observed for delayed metamorphosis in amphibians. A LOEL of ~3 ng/g BDE-209 bw/day or 0.41 ng/g ww food can be derived for TH disruptive effects and mortality in fish. Overall the aquatic toxicity data suggest that BDE-209 can have adverse effects on critical endpoints such as survival, growth, fitness, reproduction, development, somatic maintenance, thyroid hormone homeostasis and neurological function. The data, moreover, add to the concern regarding the bioaccumulation potential of BDE-209 and debromination in organisms in the environment, since they show that the accumulation of BDE-209 can lead to adverse effects in vulnerable life stages of mammals, fish and amphibians. The levels used in some of the experiments were comparable to levels in more polluted areas.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.2.3 Honeybees and other arthropods

Not available.

4.2.4 Earthworms

Based on a plant toxicity study, and two 28- and 56-day toxicity studies with earthworms, no effects were seen on plants at concentrations up to 5,349 mg/kg dry weight and a NOEC ≥ 4,910 could be derived for earthworms. Based on these results and using an assessment factor of 50, PNEC values for soil of 98 mg/kg dry and 87 mg/kg wet weight were estimated.

A significant increase in hydroxyl radical levels in earthworms at 0.01 10 mg/kg of BDE-209 was observed, which is within the range of environmental levels reported in soil. The effect was paralleled by oxidative damage to protein and lipids and a reduction in antioxidant capacity. Oxidative stress and oxidative lipid damages were observed at concentrations as low as 0.01 mg/kg. In a more recent acute earthworm study, effects on behavior, survival, growth and reproductive parameters were investigated following exposure to 0.1-100 mg/kg BDE-209 for 48 hours and 28 days. Except for a significant decrease in the number of juveniles per hatched cocoon and non-significant changes in avoidance response at 1000 mg/kg BDE-209, no other effects were reported suggesting that adult earthworms have a strong tolerance for BDE-209 in soils, but that a potential toxicity exist for earthworm embryos or juveniles.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.2.5 Soil microorganisms

BDE-209 appears not to be acutely toxic to plants and soil organisms and adverse effects are generally observed at high doses. However, new data suggests that toxic effects of BDE-209 in some instances may occur at lower doses (0.01-1 mg BDE-209/kg) than previously shown.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

4.2.6 Terrestrial plants

In ryegrass seedlings exposed to 100 mg/kg BDE-209, a 35% inhibition of root growth and 30% decrease of the chlorophyll b and carotenoid contents of leaves was observed. No other visual signs of toxicity were observed, but BDE-209 exposure induced oxidative stress and damage, altered the activity of several antioxidant enzymes and reduced the non-enzymatic antioxidant capacity at concentrations

starting from 1 mg/kg. No effects were observed on nitrifying bacteria, red clover seedling emergence or survival and reproduction of soil invertebrates at concentrations up to 1,000 mg BDE-209/kg spiked soil and it was speculated that the absence of toxicity could be due to the low water solubility of BDE-209.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

5 Environmental Exposure/Risk Evaluation

5.1 Terrestrial vertebrates

Norwegian environmental monitoring studies investigating congener pattern and levels of PBDEs in eggs and plasma of glaucous gulls breeding at Bjørnøya in the Arctic revealed detectable levels of decaBDE in bird plasma comparable to levels found in liver samples of birds located at more southern parts of Europe (TA-2006). Similar results were reported in liver samples from glaucous gulls from Svalbard. The concerns about occurrence of decaBDE in the environment have now been further strengthened. These recent studies from the Arctic document the occurrence of decaBDE in birds from remote areas in the Arctic. DecaBDE was also present in moose and lynx.

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

Some recent studies have shown concentrations of decaBDE to be increasing steadily in some wildlife species and there are a few equivocal reports of BMFs exceeding 1. In some cases, such as in the tissues of kestrel, sparrowhawk, peregrine falcon, glaucous gull, red fox, shark, harbour porpoise and whitebeaked dolphin, measured concentrations of decaBDE in tissues are interpreted as high. While trophic magnification or bioaccumulation is a potential explanation for these high concentrations, it is also very possible that some biota are exposed to very high exposure concentrations of decaBDE by consuming contaminated refuse and/or inhabiting decaBDE hotspots close to industrialized areas.

(UNEP/FAO/RC/CRC.15/5, Canadian notification)

5.2 Aquatic species

In all the fish samples from the inner Drammensfjord, BDE-209 was detected. The concentrations were in general low (0.1-20% of ΣPBDE), but the results are in accordance with new knowledge about BDE-209 as a bioavailable substance (TA-2051). BDE-209 is also detected in aquatic biota such as mussels, fish and in leakage from landfills (TA-2006).

(UNEP/FAO/RC/CRC.15/5, Norwegian notification)

Average concentrations in the blubber of marine mammals from the Canadian Arctic were reported as 25.8 µg/kg lipid in female ringed seals (Phoca hispida), 50.0 µg/kg in the blubber of male ringed seals, 81.2 µg/kg lipid in female beluga (Delphinapterus leucus) and 160 µg/kg lipid in male beluga. In these samples, tetraBDE and pentaBDE congeners were predominant. PBDE concentrations in biota samples from the west coast and Northwest Territories of Canada were reported. The highest concentration of total PBDE residues, 2269 µg/kg lipid, was found in the blubber of a harbour porpoise from the Vancouver area. With a concentration of about 1200 µg/kg lipid, a tetraBDE congener accounted for slightly more than half of the total PBDE in the sample. Temporal trends in Arctic marine mammals were analysed by measuring PBDE levels in the blubber of Arctic male ringed seals over the period 1981-2000. Mean total PBDE concentrations increased exponentially from approximately 0.6 µg/kg lipid in 1981 to 6.0 µg/kg lipid in 2000, a greater than 8-fold increase. TetraBDE was again predominant, followed by pentaBDE. A marked increase in tissue PBDE levels was also evident in blubber samples collected from San Francisco Bay harbour seals over the period 1989-1998. Concentrations of total PBDEs (tetra-, penta- and hexaBDE) rose from 88 µg/kg lipid in 1989 to a maximum of 8325 µg/kg lipid in 1998, a period of only 10 years. PBDE levels in the blubber of male southeast Baffin beluga whales were examined over the period 1982-1997 and the levels of total PBDEs (tri- to hexaBDE) increased significantly. Mean total PBDE concentrations were about 2 µg/kg lipid in 1982 and reached a maximum value of about 15 µg/kg lipid in 1997. Total PBDE residues in the blubber of St. Lawrence estuary belugas sampled in 1997-1999 amounted to 466 (± 230) µg/kg wet weight (ww) blubber in adult males and 665 (± 457) µg/kg ww blubber in adult females.

These values were approximately 20 times higher than concentrations in beluga samples collected in 1988-1990.

(UNEP/FAO/RC/CRC.15/5, Canadian notification)

5.3	**Honey bees**	Not available.
5.4	**Earthworms**	Not available.
5.5	**Soil microorganisms**	Not available.
5.6	**Summary – overall risk evaluation**	The evaluation of decaBDE gives rise to concern for long term effects in the environment. In Norway, BDE-209 has been investigated and detected in a number of studies. Furthermore, BDE-209 has been found in various environmental compartments in the Arctic, including the Norwegian Arctic, and can undergo long-range environmental transport (Hermanson et al., 2010, Mariussen et al. 2008). Norwegian monitoring data shows that BDE-209 deposited to the Arctic environment is bioavailable to the organisms living there and that BDE-209 is widespread in Arctic food webs (de Wit et al 2006, 2010). The general concern about the ubiquitous presence and increase of decaBDE in the environment and the concern for increased levels of persistent PBDEs due to continuously debromination from the pool of decaBDE in the environment, together with the risk for endocrine disrupting effects of the mix of PBDE congeners to organisms at vulnerable stages, led Norwegian authorities to ban further use of decaBDE. (UNEP/FAO/RC/CRC.15/5, Norwegian notification) Risk quotient analyses, integrating known or potential exposures with known or potential adverse environmental effects, were performed for each of the commercial PBDE products subject to this assessment. An analysis of exposure pathways and subsequent identification of sensitive receptors were used to select ecological assessment endpoints (e.g., adverse reproductive effects on sensitive fish species in a community). For each endpoint, a conservative Estimated Exposure Value (EEV) was selected based on empirical data from monitoring studies. Where monitoring data were not available, the EEVs were based on simple calculation procedures taking into account some degree of local environmental conditions, but largely relying on generic environmental parameters. Chemical concentrations from the Canadian and North American environment were used preferentially for EEVs; however, data from other regions in the world were used in the absence of sufficient Canadian data of satisfactory quality or to provide a weight of evidence. EEVs usually represented worse-case scenarios, as an indication of the potential for these substances to reach concentrations of concern and to identify areas where those concerns would be most likely. An Estimated No-Effects Value (ENEV) was also determined by dividing a Critical Toxicity Value (CTV) by an application factor. CTVs typically represented the lowest ecotoxicity value from an available and acceptable data set. Preference was generally for chronic toxicity data, as long-term exposure was a concern. The risk quotient analysis indicates that the greatest potential for risk from PBDEs in the Canadian environment is due to the secondary poisoning of wildlife from the consumption of prey containing elevated c-pentaBDE and c-octaBDE congener concentrations. Risk associated with components of c-pentaBDE may be due to the use of c-octaBDE or debromination of highly brominated PBDEs, in addition to the use of c-pentaBDE itself. The risk analysis for soil organisms indicates that risk quotients were below 1 for c-pentaBDE, c-octaBDE and c-decaBDE; however, the lack of data characterizing PBDE concentrations in soil and sewage sludge applied to soil indicates the need for further research. C-pentaBDE, c-octaBDE and c-decaBDE would present low potential for risk as a result of direct toxicity to pelagic organisms due to their very low water solubility. Environment Canada's Ecological Screening Assessment Report concluded that PBDEs are entering the environment in a quantity or concentration, or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity. The review conducted in the Ecological State

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

The findings of the Ecological State of the Science Report provided justification for the development of additional regulatory controls for decaBDE.

(UNEP/FAO/RC/CRC.15/5, Canadian notification)

BDE-209 is persistent in the environment and bioaccumulates and biomagnifies in several species of fish, birds and mammals as well as in food webs. There is evidence for adverse effects to critical endpoints including reproduction, survival, nerve- and endocrine systems. BDE-209 is also degraded to lower brominated PBDEs, with known PBT/vPvB and POP properties. Lower brominated congeners contribute in the outcome of BDE-209 toxicity. Due to debromination and historical reservoirs of c-penta- and c-octaBDE congeners in the environment, organisms are exposed to a complex mixture of PBDEs that in combination pose a higher risk than BDE-209 alone. Measured BDE-209 levels in some species of biota, including higher trophic levels such as birds and mammals in source and remote regions are close to reported effect concentrations and indicate that BDE-209 together with other PBDEs pose a significant concern for human health and the environment.

(UNEP/POPS/POPRC.10/10/Add.2, POPRC Risk Profile)

Annex 2 – Details on final regulatory actions reported

Country Name: Canada

1	**Effective date(s) of entry into force of actions**	December 23, 2016.
	Reference to the regulatory document	*Prohibition of Certain Toxic Substances Regulations, 2012* (SOR/2012-285), as amended, 2016 (SOR/2016-252) under the *Canadian Environmental Protection Act, 1999* (CEPA). http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html
2	**Succinct details of the final regulatory action(s)**	The regulatory action notified by Canada relates to the industrial uses of polybrominated diphenyl ethers (PBDEs) that have the molecular formula $C_{12}H_{(10-n)}Br_nO$, in which n is between 4 and 10 inclusive. This group includes tetrabromodiphenyl ether, pentabromodiphenyl ether, hexabromodiphenyl ether, heptabromodiphenyl ether, octabromodiphenyl ether, nonabromodiphenyl ether, and decabromodiphenyl ether. The decabromodiphenyl ether commercial mixture (c-decaBDE) is described as being almost completely composed of decaBDE and a very small amount of nonaBDE, but may contain trace amounts of octaBDE. This notification for polybrominated diphenyl ethers (PBDEs) replaces previously submitted notifications by Canada for pentabromodiphenyl ether commercial mixture (c-pentaBDE), and octabromodiphenyl ether commercial mixture (c-octaBDE) on 14 October 2010. The Prohibition of Certain Toxic Substances Regulations, 2012 prohibit the manufacture, use, sale, offer for sale, or import of PBDEs, including decaBDE, and all products that contain PBDEs, except for manufactured items.
3	**Reasons for action**	The regulatory action was based on concerns related to the environment.
4	**Basis for inclusion into Annex III**	The regulatory action was taken to protect environment. The regulatory action was based on a risk evaluation taking into account the prevailing conditions in Canada. The screening assessments, on which the regulatory action was based and that was performed in Canada makes use of the extensive information on uses, releases and environmental levels of decaBDE in Canada, including the Canadian Arctic. Release of PBDEs, including decaBDE, into the environment may occur during manufacture, processing, throughout the service life of products and articles containing them, and during disposal of the substance or products containing the substance. Since the final regulatory action is intended to protect the Canadian environment from risks associated with PBDEs, including decaBDE, by prohibiting the manufacture, use, sale, offer for sale, or import, it would be expected that the final regulatory action would result in a significant reduction in risks to the environment in Canada.
4.1	**Risk evaluation**	The notification from Canada indicates that the regulatory action was based on a risk evaluation and that it was relevant to the environment. The notification specifically cites the Screening Assessment Report on PBDEs, prepared by Environment Canada, June 2006, and the Ecological State of the Science Report on Decabromodiphenyl Ether (decaBDE), prepared by Environment Canada, August 2010 (UNEP/FAO/RC/CRC.15/5, section 2.4 of the Canadian notification). Summarized in section 3.2.3 of the notification from Canada is evidence of the detection of PBDEs in all environmental media as well as sewage sludge, and there is evidence that their levels in the North American environment are increasing. Results were reported on biota in the Canadian Arctic and some temporal trends are noted such as the increase in PBDE levels in marine mammals, such as ringed seals and beluga whales.

The analysis of risk quotients indicates that the greatest potential for risk from PBDEs in the Canadian environment is due to the secondary poisoning of wildlife from the consumption of prey containing elevated c-pentaBDE and c-octaBDE congener concentrations. Also, it indicated that elevated concentrations of components of c-pentaBDE in sediments may present risk to benthic organisms. The risks associated with these congeners may be due to debromination of highly brominated PBDEs, such as decaBDE.

Although overall, the available data does not show that decaBDE itself meets the numeric criteria for bioaccumulation, as defined in the Persistence and Bioaccumulation Regulations under CEPA, some studies have shown concentrations of decaBDE to be increasing steadily in some wildlife species. In some cases, such as in the tissues of kestrel, sparrowhawk, peregrine falcon, glaucous gull, red fox, shark, harbour porpoise, and whitebeaked dolphin, measured concentrations of decaBDE are interpreted as high.

Given the information on hazardous properties, the detection of PBDEs, including decaBDE, (sometimes with increasing trends from temporal studies) in Canadian environmental monitoring, and ecological biomonitoring studies, it was concluded that PBDEs, including decaBDE, were entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity.

4.2 Criteria used

Risk to environment.

Relevance to other States and Region

Canada indicates that the Parties to the Stockholm Convention have agreed on the listing of decaBDE in Annex A with some specific exemptions for production and use (UNEP/FAO/RC/CRC.15/5 section 2.5.2 of the Canadian notification). Substances listed in Annex A of the Stockholm Convention are targeted for global elimination, through prohibition on manufacture, import and use. As a persistent organic pollutant, decaBDE has hazardous properties and is subject to long range transport.

Given the hazards and long-range transport of this substance as described in the screening assessment on which the regulatory action is based, any state or region in which exposure or release is possible may find the regulatory action relevant.

5 Alternatives

ALTERNATIVE CHEMICALS

Chemical alternatives to PBDEs are available for the vast majority of industrial and manufacturing applications, and these vary by application. However, several issues need to be addressed as some potential alternatives are:

- Currently under scrutiny themselves;
- New proprietary chemicals for which data on environmental and health effects are very limited;
- More costly; and
- Less effective, hence much higher levels are required and products may be less likely to meet flammability standards.

ALTERNATIVE TECHNIQUES

The need for PBDEs can be reduced through the use of alternative techniques such as:

- Use of materials that are less prone to fire hazard in electronics equipment (such as aluminum or "super-plastics" with very high oxygen requirements for combustion); or
- Use of barrier fabrics, wrappings or coatings for foams to replace chemical flame retardants.

Some of these alternative techniques present challenges, such as increased weight of final products and methods to collect, reuse and reassemble products with components containing PBDEs.

		Work was undertaken under the US EPA Design for the Environment programme to identify alternatives to decaBDE and assess risks they may pose. A final report was published in January 2014 (https://www.epa.gov/sites/production/files/2014-05/documents/decabde_final.pdf) A move away from decaBDE towards alternative flame retardants and, in certain cases, flame retardant barriers in products, in lieu of chemicals, means that many of the applications no longer use decaBDE, especially in view of the phase-out in the US and the broad controls proposed in other jurisdictions. (UNEP/FAO/RC/CRC.15/5, Canadian notification)
6	**Waste management**	The notifying Party did not provide information on waste management of decaBDE or articles containing it.
7	**Other**	None.

Country Name: Japan

1	**Effective date(s) of entry into force of actions**	1 April 2018
	Reference to the regulatory document	Chemical Substances Control Law of Japan and its Enforcement Order.
2	**Succinct details of the final regulatory action(s)**	The substance has been designated as a Class 1 Specified Chemical Substance under the Chemical Substances Control Law of Japan and its Enforcement Order. It is prohibited to manufacture, import, and use this chemical substance. All uses are prohibited by the final regulatory action, and that no uses remain allowed.
3	**Reasons for action**	The regulatory action was based on concerns related to human health and the environment.
4	**Basis for inclusion into Annex III**	Release of decaBDE into the environment may occur during manufacture, processing, throughout the service life of products and articles containing it, and at disposal of the substance or products containing the substance. Since the final regulatory action prohibits the manufacture, import, and use of decaBDE, it would be expected that the final regulatory action would lead to a reduction in the exposure of people and the environment to decaBDE as its use is phased out, resulting in a significant reduction in risks to human health and the environment in Japan.
4.1	**Risk evaluation**	Japanese authorities banned decaBDE as this chemical is persistent, highly bioaccumulative and has long-term toxicity to humans, based on not only the scientific evaluation by POPRC (under the Stockholm Convention) but also domestic risk evaluation in Japan. The Japanese government designates chemical substances that are persistent, highly bioaccumulative, and have long-term toxicity for humans as Class I Specified Chemical Substances to be banned under the Chemical Substances Control Law (CSCL). As a result of internal evaluation using the scientific data in Japan, Japanese authorities concluded that this chemical met the criteria to be designated as a Class I Specified Chemical Substance under the CSCL. The notification also states that BDE-209, the main component of c-decaBDE, exerts reproductive, developmental, endocrine and neurotoxic effects in aquatic organisms, mammals and birds. Effects on growth, survival and mortality are also reported. The final regulatory action is intended to lead to a reduction of exposure to humans and a reduction of exposure to the environment from decaBDE as its use is phased out. (UNEP/FAO/RC/CRC.15/5, sections 2.4.1 and 2.4.2 of the Japanese notification).
4.2	**Criteria used**	Risk to human health and the environment.
	Relevance to other States and Region	Information was not provided in the notification on this criterion, however, it is noted that decaBDE has been listed in Annex A of the Stockholm Convention with some specific exemptions for production and use. Substances listed in Annex A of the Stockholm Convention are targeted for global elimination, through prohibition on manufacture, import and use. As a persistent organic pollutant, decaBDE has hazardous properties and is subject to long range transport. Any state or region where exposure or release is possible may find the regulatory action relevant.
5	**Alternatives**	Information was not provided in the notification on this criterion, however, it was stated that alternative actions were completed before April 2018.
6	**Waste management**	The notifying Party did not provide information on waste management of decaBDE or articles containing it.
7	**Other**	None.

Country Name: Norway		
1	**Effective date(s) of entry into force of actions**	1 April 2008 amended 1 July 2013
	Reference to the regulatory document	DecaBDE has been regulated under the 'Regulations relating to restrictions on the manufacture, import, export, sale and use of chemicals and other products hazardous to health and the environment (Product Regulations)' (chapter 1 to 7), by the Ministry of the Environment, Act no. 922 of 1 June 2004. Following Norway's proposal to list decaBDE in Annex A to the Stockholm Convention on Persistent Organic Pollutants, the EU amended Annex XVII to REACH to include decaBDE (Commission Regulation (EU) 2017/227 of 9 February 2017). The REACH regulation is a part of the European Economic Area (EEA) agreement and, as such, the REACH regulation covers also Norway. The government in Norway decided in 2017 that as a result of the amendment of Annex XVII to REACH, no further action on decaBDE was needed in Norway since Norway already had prohibited the production, import, export, sale and use. The Commission Regulation (EU) 2017/227 of 9 February 2017 amending Annex XVII to Regulation (EC) No 1907/2006 of the European Parliament and of the Council concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) as regards bis(pentabromophenyl)ether (decaBDE) entered into force on March 2, 2019.
2	**Succinct details of the final regulatory action(s)**	It is prohibited to produce, import, export, sell and use decabromodiphenyl ether in pure form, in preparations, in products, and in parts of products containing greater than or equal to 0,1 % by weight of decabromodiphenyl ether. The prohibition in respect of products and parts of products also applies to electrical and electronic equipment (EEE). For some categories of EEE, the restrictions took effect over a period of time, from July 2014 until July 2019. (UNEP/FAO/RC/CRC.15/5 section 2.3.2 of the Norwegian notification).
3	**Reasons for action**	The regulatory action was based on concerns related to human health and the environment.
4	**Basis for inclusion into Annex III**	The final regulatory action is intended to protect the Norwegian people and environment from risks associated with the manufacture, import, sale, and use of decaBDE, and products containing decaBDE. The regulatory action was based on a risk evaluation taking into account the prevailing conditions in Norway.
4.1	**Risk evaluation**	Norwegian authorities banned decaBDE based on its potential persistent, bioaccumulative, and toxic (PBT) properties and the general concern about the ubiquitous presence and increase of decaBDE in the environment including the Norwegian Arctic, and a concern for the presence of decaBDE in human matrices and human health. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.1 of the Norwegian notification). Norwegian monitoring data show detectable levels of BDE-209 in several environmental compartments, and high concentrations of BDE-209, the main component of c-decaBDE, is detected at some locations. In food samples analysed for BDE-209, high levels were found in eggs, vegetable oil, ice cream and biscuits, while the highest amounts were found in dairy products, which include milk, cheese, and butter, thus indicating exposure of the human population to BDE-209 through food. High levels of BDE-209 (10 ng / g lipid) were found in pooled serum samples from Norwegians, and also in a separate study in plasma from pregnant women. Household dust and occupational exposure is thought to be the main sources for exposure to BDE-209 and other PBDE congeners present in c-decaBDE. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.1 of the Norwegian notification). The notification also states that the evaluation of decaBDE gives rise to concern for long term effects in the environment. In Norway, a number of studies on BDE-209 report its detection in a range of environmental samples. Norwegian monitoring data shows that BDE-209 deposited to the Arctic environment is bioavailable to the organisms living there and that BDE-209 is widespread in

		Arctic food webs. Norwegian environmental monitoring studies investigating congener pattern and levels of PBDEs in eggs and plasma of glaucous gulls breeding at Bjørnøya in the Arctic revealed detectable levels of BDE-209 in bird plasma comparable to levels found in liver samples of birds located at more southern parts of Europe. In Norway, high levels of BDE-209 were detected in sediments and BDE-209 represented up to 90% of ΣPBDEs, and similar results were found in other studies in Norway. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.2 of the Norwegian notification). In animal studies of amphibian, fish and rodents exposed to BDE-209 at vulnerable stages as the developmental phase, effects on hormonal axis as the thyroid and steroid is of concern. Although the toxicology data of BDE-209 is ambiguous, some studies indicate negative effect on neurological development at low doses. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.2 of the Norwegian notification). The general concern about the ubiquitous presence and increase of decaBDE in the environment and the concern for increased levels of persistent PBDEs due to continuously debromination from the pool of decaBDE in the environment, together with the risk for endocrine disrupting effects of the mix of PBDE congeners to organisms at vulnerable stages, led Norwegian authorities to ban further use of decaBDE. (UNEP/FAO/RC/CRC.15/5 section 2.4.2.2 of the Norwegian notification).
4.2	**Criteria used**	Risk to human health and the environment.
	Relevance to other States and Region	The notification states that concerns similar to those identified in Norway are likely to be encountered in other countries where the substance is used. In addition, it is noted that decaBDE has been listed in Annex A of the Stockholm Convention with some specific exemptions for production and use. Substances listed in Annex A of the Stockholm Convention are targeted for global elimination, through prohibition on manufacture, import and use. As a persistent organic pollutant, decaBDE has hazardous properties and is subject to long range transport. Any state or region where exposure or release is possible may find the regulatory action relevant.
5	**Alternatives**	Alternatives to decaBDE in all applications are available including non-chemical alternatives (US EPA, 2006 and 2007). Furthermore, a number of alternative flame retardant (FR) available as a substitute for decaBDE in EEE products (Danish EPA, 2006). Alternatives include other brominated FRs and other chemical FR. References: DecaBDE Study: A Review of Available Scientific Research, US EPA, 2006 Report on Alternatives to the Flame Retardant DecaBDE: Evaluation of Toxicity, Availability, Affordability, and Fire Safety Issues, US EPA, 2007. Deca-BDE and Alternatives in Electrical and Electronic Equipment, Danish EPA, 2006. (UNEP/FAO/RC/CRC.15/5 Norwegian notification)
6	**Waste management**	The notifying Party did not provide information on waste management of decaBDE or articles containing it.
7	**Other**	None.

Annex 3 – Addresses of designated national authorities	
Canada	
C	
From PIC website (September 2020): Ms. Nicole Folliet Director Chemical Production Division Environment and Climate Change Canada 351 St. Joseph Boulevard K1A 0H3 Gatineau Québec Canada Phone: +1 819 420 7708 Fax: +1 819 938 4218 Email: nicole.folliet@canada.ca; ec.substancedexportationcontrolee-exportcontrolledsubstance.ec@canada.ca	
Norway	
C	
From PIC website (September 2020): Ms. Christel Moræus Olsen Senior Advisor, Rotterdam Convention Designated National Authority for Industrial Chemicals and Biocides Department: Section for Biocides and Declaration of Chemicals/ Chemicals and Waste Department Institution: Norwegian Environment Agency Postal address: Grensesvingen 7 7485 Trondheim Norway Phone: +47 73 580 500 Fax: +47 73 580 501 Email: christel.moraeus.olsen@miljodir.no, pic@miljodir.no	Ms. Mitsuko Komada Senior Advisor, Rotterdam Convention Designated National Authority for Industrial Chemicals and Biocides Norwegian Environment Agency Torgarden P.O. Box 5672 7485 Trondheim Norway Phone: +47 73 580 500 Fax: +47 73 580 501 Email: mitsuko.komada@miljodir.no, pic@miljodir.no
Japan	
CP	
From PIC website (September 2020): Ms. Tomoe Kotani Deputy Director International Cooperation Bureau / Global Environment Division Ministry of Foreign Affairs 2-2-1, Kasumigaseki, Chiyoda-ku 100-8919 Tokyo Japan Phone: +81 3 5501 8245 Fax: +81 3 5501 8244 Email: tomoe.kotani@mofa.go.jp	

C Industrial chemicals
CP Pesticides and industrial chemicals

Annex 4 – References

Regulatory actions

Canada:

Prohibition of Certain Toxic Substances Regulations, 2012 (SOR/2012-285), as amended, 2016 (SOR/2016-252) under the Canadian Environmental Protection Act, 1999 (CEPA). http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html

Norway:

Chapter 1 to 7 of the *Regulations related to restrictions of the manufacture, import and placing on the market of chemicals and other products hazardous to the human health and the environment (Product Regulations)*, Ministry of the Environment, Act no. 922 of June 2004.

Japan:

Chemical Substances Control Law (CSCL) of Japan and its Enforcement Order, 1 April 2018.

Supporting documentation provided by Canada

Regulatory Impact Analysis Statement, *Regulations Amending the Prohibition of Certain Toxic Substances Regulations, 2012*, Environment Canada and Health Canada, October 2016. http://www.gazette.gc.ca/rp-pr/p2/2016/2016-10-05/html/sor-dors252-eng.html

Substance Prohibition Summary for Polybrominated Diphenyl Ethers, Environment and Climate Change Canada, July 2017. https://www.canada.ca/en/environment-climate-change/services/canadian-environmental-protection-act-registry/substance-prohibition-summary-polybrominated-diphenyl-ethers.html

Ecological Screening Assessment Report on Polybrominated Diphenyl Ethers (PBDEs), Environment Canada, June 2006. https://www.canada.ca/en/environment-climate-change/services/canadian-environmental-protection-act-registry/publications/ecological-screening-assessment-report-polybrominated.html

Ecological State of the Science Report on Decabromodiphenyl Ether (decaBDE), Environment Canada, August 2010. http://www.ec.gc.ca/lcpe-cepa/default.asp?lang=En&n=B901A9EB

An Alternatives Assessment For The Flame Retardant Decabromodiphenyl Ether (DecaBDE) Final Report (January 2014), United States Environmental Protection Agency. https://www.epa.gov/sites/production/files/2014-05/documents/decabde_final.pdf

Supporting documentation provided by Norway

EC 2002: Risk Assessment of bis(pentabromophenyl) ether (decabromodiphenyl ether), CAS Number: 1163-19-5 EINECS Number: 214-604-9, European Commission 2002 and updates: https://echa.europa.eu/documents/10162/da9bc4c4-8e5b-4562-964c-5b4cf59d2432

http://ec.europa.eu/health/ph_risk/committees/04_scher/docs/scher_o_012.pdf

ECHA 2012: Proposal for identification of a PBT/vPvB substance, Annex XV dossier, Health & Safety Executive, Redgrave Court, Bootle, Merseyside L20 7HS, United Kingdom (August 2012). https://echa.europa.eu/documents/10162/23665416/SVHC_AXVREP_pub_EC_214_604_9_decabromodiphenylether_11446_en.pdf/36c9d78c-d9dd-286b-8930-dad8c2247669

US EPA 2010.Exposure assessment: https://www.federalregister.gov/documents/2010/05/24/2010-12378/an-exposure-assessment-of-polybrominated-diphenyl-ethers-pbdes

DecaBDE Study: A Review of Available Scientific Research, US EPA, 2006: http://www.epa.state.il.us/reports/decabde-study/available-research-review.pdf

Report on Alternatives to the Flame Retardant DecaBDE: Evaluation of Toxicity, Availability, Affordability, and Fire Safety Issues, US EPA, 2007: http://www.epa.state.il.us/reports/decabde-study/decabde-alternatives.pdf

Deca-BDE and Alternatives in Electrical and Electronic Equipment, Danish EPA, 2006: http://www2.mst.dk/Udgiv/publications/2007/978-87-7052-349-3/pdf/978-87-7052-350-9.pdf

Hermanson et al., 2010: https://www.ncbi.nlm.nih.gov/pubmed/20839863

Knutsen et al. 2008: http://onlinelibrary.wiley.com/doi/10.1002/mnfr.200700096/epdf

Mariussen et al., 2008: https://www.ncbi.nlm.nih.gov/pubmed/18627904

de Wit et al., 2006: https://www.ncbi.nlm.nih.gov/pubmed/16458344

de Wit et al., 2010: https://www.ncbi.nlm.nih.gov/pubmed/19815253

Reports in Norwegian (with only a short summary in English):

TA-2051:
http://tema.miljodirektoratet.no/no/Publikasjoner/Publikasjoner/2004/Desember/Miljogifter_i_sedimenter_og_fisk_i_Mjosa_Drammensvassdraget_og_Drammensfjorden_oppfolgende_undersokelser_i_2004/

TA-2096: https://www.miljodirektoratet.no/publikasjoner/publikasjoner-fra-klif/2005/juli/kartlegging-av-utvalgte-nye-organiske-miljogifter-2004.-bromerte-flammehemmere-perfluoralkylstoffer-irgaroldiuron-bht-og-dicofol/

TA-2146: https://www.miljodirektoratet.no/publikasjoner/publikasjoner-fra-klif/2006/mars/kartlegging-av-utvalgte-miljogifter-i-asefjorden-ogomkringliggende-omrader.-bromerte-flammehemmereklororganiske-forbindelser-kvikksolv-og-tribromanisol/

TA-2006:
http://tema.miljodirektoratet.no/no/Publikasjoner/Publikasjoner/2004/Mars/Kartlegging_av_utvalgte_nye_organiske_miljogifter_bromerte_flammehemmere_klorerte_parafiner_bisfenol_A_og_triclosan/

Reports in Norwegian only:

TA-2104 : https://www.miljodirektoratet.no/publikasjoner/publikasjoner-fra-klif/2005/november/vurdering-av-bromerte-flammehemmere-til-mjosa-fra-deponier-kommunale-renseanlegg-og-elver/

TA-2252 : https://www.miljodirektoratet.no/publikasjoner/publikasjoner-fra-klif/2007/juni/kartlegging-av-bromerte-flammehemmere-klor--og-bromorganiske-forbindelser-kvikksolv-og-metylkvikksolv-i-fjorder-nar-alesund/

TA-2303 : https://www.miljodirektoratet.no/publikasjoner/publikasjoner-fra-klif/2007/oktober/bromerte-flammehemmere-i-blodprover-fra-gravide-kvinner-i-bodo/

Supporting documentation provided by Japan

Environmental risk assessment of short-chain chlorinated paraffins and decabromodiphenyl ether, Ministry of Environment, Japan (22 September 2017) (Chapter 6 of the document translated into English).

Environmental risk assessment of short-chain chlorinated paraffins and decabromodiphenyl ether, Ministry of Environment, Japan (22 September 2017) (Full report in Japanese).

Risk assessment for chemical substances contained in products, decabromodiphenyl ether, CAS No. 1163-19-5, National Institute of Technology and Evaluation, Ministry of Economy, Trade and Industry, Ministry of Health, Labour and Welfare, Japan (June 2018) (Summary in English).

Risk assessment for chemical substances contained in products, decabromodiphenyl ether, CAS No. 1163-19-5, National Institute of Technology and Evaluation, Ministry of Economy, Trade and Industry, Ministry of Health, Labour and Welfare, Japan (June 2018) (Full report in Japanese).

Introduction of risk assessment examples of chemicals contained in consumer products (decabromodiphenyl ether) (21 and 22 November 2018), Risk Assessment Division Chemical Management Center, NITE.

Other documents

UNEP/POPS/POPRC.9/2 (2013). Proposal to list decabromodiphenyl ether (commercial mixture, c-decaBDE) in Annexes A, B and/or C to the Stockholm Convention on Persistent Organic Pollutants.

UNEP/POPS/POPRC.10/10/Add.2 (2014). Report of the Persistent Organic Pollutants Review Committee on the work of its tenth meeting: Risk profile on decabromodiphenyl ether (commercial mixture, c-decaBDE).

UNEP/POPS/POPRC.11/10/Add.1 (2015). Report of the Persistent Organic Pollutants Review Committee on the work of its eleventh meeting: Risk management evaluation on decabromodiphenyl ether (commercial mixture, c-decaBDE).

UNEP/POPS/POPRC.12/11/Add.4 (2016). Report of the Persistent Organic Pollutants Review Committee on the work of its twelfth meeting: Assessment of additional information on decabromodiphenyl ether (commercial mixture, c-decaBDE) for the further defining of some critical spare parts in the automotive and aerospace industries and on its use in textiles in developing countries (summary, conclusion and recommendations).

Relevant guidelines and reference documents

IPCS INCHEM (2008): BIS(PENTABROMOPHENYL) ETHER. International Chemical Safety Card (ICSC) 1689. Available at: http://www.inchem.org/documents/icsc/icsc/eics1689.htm