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Persistent Organic Pollutants Review Committee
Tenth meeting
Rome, 27–30 October 2014
Item 4 (b) of the provisional agenda*

Technical work: consideration of a draft risk profile on decabromodiphenyl ether (commercial mixture, c-decaBDE)

Draft risk profile: decabromodiphenyl ether

Note by the Secretariat

I. Introduction

1. At its ninth meeting, the Persistent Organic Pollutants Review Committee adopted decision POPRC-9/4 on decabromodiphenyl ether (see UNEP/POPS/POPRC.9/13, annex I). By paragraph 2 of that decision, the Committee decided to establish an ad hoc working group to further review the proposal to list decabromodiphenyl ether in Annexes A, B and/or C to the Stockholm Convention on Persistent Organic Pollutants (UNEP/POPS/POPRC.9/2) and to prepare a draft risk profile in accordance with Annex E to the Convention.

2. In accordance with decision POPRC-9/4 and the workplan for the preparation of a draft risk profile adopted by the Committee (UNEP/POPS/POPRC.9/13, annex III), the intersessional working group has prepared the draft risk profile set out in the annex to the present note. It has not been formally edited. A supporting document and a compilation of comments and responses relating to the draft risk profile are set out in documents UNEP/POPS/POPRC.10/INF/5 and UNEP/POPS/POPRC.10/INF/6 respectively.

II. Proposed action

3. The Committee may wish:

(a) To adopt, with any amendments, the draft risk profile set out in the annex to the present note;

(b) To decide, in accordance with paragraph 7 of Article 8 of the Convention and on the basis of the risk profile, whether decabromodiphenyl ether is likely, as a result of long-range environmental transport, to lead to significant adverse human health and/or environmental effects, such that global action is warranted;

* UNEP/POPS/POPRC.10/1.

(c) To agree, depending on the decision taken under subparagraph (b) above:

(i) To invite all parties and observers to provide information pursuant to Annex F to the Convention, to establish an intersessional working group to develop a draft risk management evaluation and to agree on a workplan for completing that draft evaluation; or

(ii) To make the risk profile available to all parties and observers and set it aside.

Annex

DECABROMODIPHENYL ETHER

DRAFT RISK PROFILE

Draft prepared by the ad hoc working group on decabromodiphenyl ether
Persistent Organic Pollutants Review Committee

July 2014

Table of Contents

Executive summary

1. Commercially available decabromodiphenyl ether (c-decaBDE) is a polybrominated diphenyl ether (PBDE) formulation consisting of decabromodiphenyl ether (BDE-209, ≥90%), with small amounts of nonabromodiphenyl ether and octabromodiphenyl ether. C-decaBDE has been under investigation for its potential health and environmental impacts for more than a decade and has been subject to restrictions and voluntary risk management actions in some countries and regions.

2. C-decaBDE consumption peaked in the early 2000's, but c-decaBDE is still extensively used worldwide. C-decaBDE is used as an additive flame retardant, and has a variety of applications including in plastics/ polymers/composites, textiles, adhesives, sealants, coatings and inks. C-decaBDE containing plastics are used in housings of computers and TVs, wires and cables, pipes and carpets. It is used in commercial textiles, mainly for public buildings and transport, and in textiles for domestic furnitures. Emissions of c-decaBDE to the environment occur at all its life cycle stages, but are assumed to be highest during service-life and in the waste phase. Emissions from industrial point sources can also be significant. Use of c-decaBDE in the production of textiles and electronics lead to emissions to the environment and transboundary air pollution either directly from articles or during production and disposal.

3. BDE-209 has low water solubility (< 0.1 µg/L at 24 °C) and in the environment it adsorbs strongly to organic matter and readily partitions to sediment and soil. It is very persistent and reported environmental half-lives in these media typically exceed 180 days.

4. BDE-209 is widespread and one of the most prevalent PBDEs in the global environment. When BDE-209 is detected in the environment and biota, it is typically found along with other PBDEs. Monitoring data show high concentrations of BDE-209 in sediments and soil, but is also found in biota worldwide, with high levels reported in some species. Levels are generally highest in the urban regions, near waste water discharges and in areas around electronic waste and recycling plants. In air, BDE-209 binds to particles that protect the chemical from photolytic degradation and it can be transported over long distances. The estimated atmospheric half-life is 94 days, but can exceed 200 days. BDE-209 is also detected in environmental and biological samples from remote regions and is one of the predominant PBDEs in Arctic air and deposition samples. Temporal trend data of BDE-209 in Arctic air and some Arctic organisms for 2002-2005 showed increasing levels, but levels may now be stabilizing.

5. BDE-209 has limited bioavailability because of its large size that constrains its ability topass cell membranes via passive diffusion. However, biomonitoring data shows that BDE-209 is bioavailable and is taken up by humans and other organisms. BDE-209 has been found in a variety of different organisms and biological matrices including human blood serum, cord blood, placenta, fetus, breast milk and in milk of lactating cows. In some species reported levels are close to reported adverse effect concentrations. In rodents and birds, small amounts of BDE-209 are shown to cross the blood-brain barrier and enter the brain. There is also evidence of transfer of BDE-209 from adult stages to eggs in fish and birds and of effective transport through the placenta to foetus in mammals. For humans, the available intake estimates for BDE-209 also point out the importance of dust exposure, particularly for small children. Higher levels of PBDEs and BDE-209 are reported in toddlers and young children than in adults. In aquatic organisms intake via diet appears to be the most important exposure route.

6. The available bioaccumulation data for BDE-209 are equivocal, but several lines of evidence show that BDE-209 is bioaccumulative, at least in some species. The equivocation in the available bioaccumulation data largely reflects species and tissue differences in uptake, metabolism and elimination, as well as differences in exposure and also analytical challenges in measuring BDE-209.

7. Debromination of BDE-209 in environmental matrices and biota to more persistent, toxic and bioaccumulative PBDEs including those already listed under the Stockholm Convention (POP-BDEs) is considered to be of concern in a number of assessments. Several PBDE congeners that are not part of any commercial mixture have been identified mainly in biota but also in the environment and are considered to provide evidence for debromination of BDE-209. Due to debromination of c-decaBDE and past releases of commercial penta- and octabromodiphenyl ether organisms are often exposed to a multitude of PBDEs.

8. BDE-209 toxicity studies provide evidence for potential adverse effects to reproductive health and output in a number of species as well as developmental and neurotoxic effects. BDE-209 and/or its degradation products may also act as endocrine disruptors and affect thyroid hormone homeostasis. On the basis of common modes of action and common adverse outcomes, there is concern that BDE-209 and other PBDEs may act in combination, in an additive or synergistic manner and induce developmental neurotoxicity in both humans and wildlife at environmentally relevant concentrations. Observed effect concentrations for increased mortality in birds and developmental effects in frogs

derived from controlled laboratory studies raise concern that adverse effects may occur at environmentally realistic concentrations.

9. The high persistence of BDE-209 combined with the simultaneous exposure of organisms to a wide range of PBDEs and the fact that endocrine disruptors like BDE-209 and/or its degradation products may elicit adverse effects even at low environmental levels increase the likelihood for long-term adverse effects.

10. Based on the available evidence it is concluded that c-decaBDE is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted.

1. Introduction

11. On 13 May 2013, Norway as a Party to the Stockholm Convention, submitted a proposal to list decabromodiphenyl ether (commercial mixture, c-decaBDE) in Annexes A, B and/or C to the Convention. The proposal (UNEP/POPS/POPRC.9/2) was submitted in accordance with Article 8 of the Convention and was reviewed by the POPs Review Committee (POPRC) at its ninth meeting in October 2013.

12. In the present document the abbreviation c-decaBDE is used for technical or commercial decaBDE products. Decabromodiphenyl ether (BDE-209) refers to the single fully brominated PBDE, which elsewhere sometimes is also denoted as decaBDE.

1.1 Chemical identity of the proposed substance

13. The risk profile concerns c-decaBDE and its degradation products, in accordance with Annex E of the Convention. C-decaBDE is a commercial PBDE formulation that is widely used as an additive flame retardant in textiles and plastics, additional uses are in adhesives and in coatings and inks (ECHA 2013b). C-decaBDE consist predominantly of the congener BDE-209 (≥97%), with low levels of other brominated diphenyl ether congeners such as nonabromodiphenyl ether (0.3-3%) and octabromodiphenyl ether (0-0.04%). Chen (2007a) reported that the octaBDE and nonaBDE content of two c-decaBDE products from China was in the range 8.2 to 10.4% suggesting that a higher degree of impurities may be found in some commercial mixtures. Historically a range of 77.4-98% of decaBDE, and smaller amounts of the congeners of nonaBDE (0.3-21.8 %) and octaBDE (0-0.85%) has been reported (ECHA 2012a, US EPA 2008, RPA 2014, in press). Total tri-, tetra-, penta-, hexa- and heptaBDEs are typically present at concentrations below 0.0039% w/w (ECB 2002, ECHA 2012a). Trace amounts of other compounds, thought to be hydroxybrominated diphenyl compounds can also be present as impurities. In addition, polybrominated dibenzo-p-dioxins and polybrominated dibenzofurans (PBDD/Fs) have been reported as impurities in some c-decaBDE products (Ren 2011).

14. According to available information c-decaBDE is currently available from several producers and suppliers globally (Ren 2013a, RPA 2014, in press) and is being marketed under different tradenames (Table 1).

15. Chemical data on the main component of c-decaBDE, BDE-209, are presented in Figure 1 and in Tables 1 and 2 below (ECHA 2012a). Like other PBDEs, BDE-209 shares structural similarities with PCBs. Chemical data on octa- and nonaBDE, which are minor constituents of commercial c-decaBDE, are provided along with other supplementary information in a supporting document for the risk profile (UNEP/POPS/POPRC.10/INF5). Information on c-decaBDE degradation products are provided in chapter 2.2.2 and in UNEP/POPS/POPRC.10/INF5.

Figure 1. Structural formula

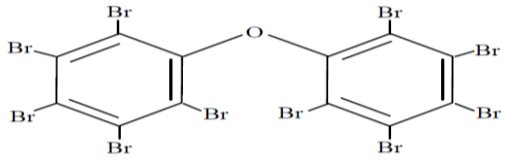

Table 1. Chemical identity of c-decaBDE and its main constituent BDE-209

CAS number:	1163-19-5[1]
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
EC number:	214-604-9
EC name:	Bis(pentabromophenyl) ether
Molecular formula:	$C_{12}Br_{10}O$
Molecular weight:	959.2 g/mole
Synonyms:	decabromodiphenyl ether, decabromodiphenyl oxide, bis(pentabromophenyl) oxide, decabromo biphenyl oxide, decabromo phenoxybenzene, benzene 1,1' oxybis-, decabromo derivative, decaBDE, DBDPE[2], DBBE, DBBO, DBDPO
Trade names	DE-83R, DE-83, Bromkal 82-ODE, Bromkal 70-5, Saytex 102 E, FR1210, Flamecut 110R. FR-300-BA, which was produced in the 1970s, is no longer commercially available (ECA, 2010).

[1]In the past CAS no. 109945-70-2, 145538-74-5 and 1201677-32-8 were also used. These CAS no. have now formally been deleted, but may still be in practical use by some suppliers and manufacturers.

[2]DBDPE is also used as an abbreviation for Decabromodiphenyl Ethane CAS no. 84852-53-9.

Table 2. Overview of relevant physicochemical properties of c-decaBDE and its main constituent BDE-209

Property	Value	Reference
Physical state at 20°C and 101.3 kPa	Fine, white to off-white crystalline powder	ECB (2002)
Melting/freezing point	300-310°C	Dead Sea Bromine Group (1993), cited in ECB (2002)
Boiling point	Decomposes at >320°C	Dead Sea Bromine Group (1993), cited in ECB (2002)
Vapour pressure	4.63×10^{-6} Pa at 21°C	Wildlife International Ltd (1997), cited in ECB (2002)
Water solubility	<0.1 µg/L at 25°C (column elution method)	Stenzel and Markley (1997), cited in ECB (2002)
n-Octanol/water partition coefficient, K_{ow} (log value)	6.27 (measured – generator column method) 9.97 (estimated using an HPLC method)	MacGregor and Nixon (1997), Watanabe and Tatsukawa (1990), respectively, cited in ECB (2002)
Octanol-air partition coefficient K_{oa} (log value)	13.1	Kelly (2007)

1.2 Conclusion of the Review Committee regarding Annex D information

16. The POPs Review Committee examined the proposal by Norway to list c-decaBDE under the Stockholm Convention on Persistent Organic Pollutants as well as additional scientific information provided by members and observers at its ninth meeting and concluded that decabromodiphenyl ether met the screening criteria specified in Annex D (decision POPRC-9/4).

1.3 Data sources

17. The risk profile is not an exhaustive review of all available data, but rather, it presents the most critical studies and lines of evidence with relevance to the criteria in Annex E and Annex D of the Convention. It centres on the main constituent of c-decaBDE, BDE-209, and its degradation products, in particular lower brominated PBDEs which are formed via abiotic and biotic degradation (described in 2.2.2). As several of the lower brominated PBDE degradation products are widely recognized as Persistent, Bioaccumulative and Toxic/very Persistent, very Bioaccumulative PBT/vPvB substances and/or POPs a re-assessment of the properties of these compounds were considered redundant (POPRC 2006, POPRC 2007, ECHA 2013a,b, ECA 2010, Table 3.2-3.4, UNEP/POPS/POPRC.10/INF5). However, the potential for mixture toxicity between BDE-209 and other PBDEs is discussed in chapter 2.4.6.

18. The risk profile was developed using the Annex D information submitted by Norway in 2013 and Annex E information submitted by parties and other stakeholders including non-governmental organizations as well as industry. The following parties and observers submitted information under the Annex E process: Argentina, Austria, Bulgaria, Canada, China, Croatia, Denmark, Germany, Japan, Mexico, Morocco, Nepal, Netherlands, New Zealand, Serbia, Sweden, USA, Bromine Science and Environmental Forum (BSEF) and International POPs Elimination Network (IPEN) together with Inuit Circumpolar Council. All Annex E submissions are available on the Convention website (www.pops.int).

19. Updated scientific literature obtained from scientific databases such as ISI Web of Science and PubMed was assessed as well as "grey" literature such as government reports, risk- and hazard assessments, industry fact sheets etc. To provide the best possible overview of the existing data/ literature which covers more than 984 reports and peer-reviewed scientific publications (Kortenkamp 2014), an emphasis was put on providing excerpts of existing risk assessments and reports when such information was available as well as more detailed descriptions of newer literature.

20. In the past, assessments of c-decaBDE and its main constituent BDE-209 were conducted and published by the EU, Canada, the United Kingdom and the United States (ECB 2002 2004, ECHA 2012a, ECA 2006, 2010, UK EA 2009, US EPA 2008). The EU risk assessment, which examines in depth the PBT/ vPvB properties of BDE-209, was conducted over a period of more than ten years (ECHA 2012b) and is the most up to date of these assessments.

1.4 Status of the chemical under other international conventions and forums

21. C-decaBDE has been under scrutiny for its potential health and environmental impacts for more than a decade. Steps to restrict the use of c-decaBDE have been taken in several countries and regions, as well as by some of the major electronic companies (for an overview: UNEP/POPS/POPRC.9/2, Ren 2011).

22. In 1992, c-decaBDE and other brominated flame retardants (BFRs) were given priority in the OSPAR action plan and in 1998 BDE-209 along with the other PBDEs was included in the list of "Chemicals for Priority Action" as well in the Joint Assessment and Monitoring Programme in OSPAR. OSPAR has promoted actions in the EU on risk-reduction strategies for c-decaBDE and electronic waste legislation.

23. In 1995, OECD Member countries agreed to oversee a voluntary industry commitment (VIC) by some of the global manufacturers of BFRs, among them c-decaBDE, to take certain risk management actions. The VIC was implemented in the United States, Europe and Japan. C-decaBDE production has since ceased in United States and Europe (see Section 2.1.1 below). In Japan, compliance with the VIC is on-going. 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 BDE-209 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 (OECD 2014). PBDEs, including BDE-209, are listed as chemicals of concern in the WHO/UNEP "State of the science of endocrine disrupting chemicals" (UNEP/WHO 2013). In the EU, c-decaBDE is included in the list of Substances of Very High Concern Annex XIV under the REACH Regulation (EC) 1907/2006 based on its PBT and vPvB properties, high volumes and wide dispersive uses.

2. Summary information relevant to the risk profile

2.1 Sources

2.1.1 Production, trade, stockpiles

24. Global industrial c-decaBDE consumption peaked in the early 2000's (Earnshaw 2013). Yet, due to limited regulatory restrictions, c-decaBDE is still extensively used worldwide (Table 2.1-2.3, UNEP/POPS/POPRC.10/INF5). Past, production data indicate that about 75% of all the world production of PBDEs was c-decaBDE (RPA 2014 in press). Total production of c-decaBDE in the period 1970-2005 was between 1.1-1.25 million tonnes, similar to the scale of production of PCBs (POPRC 2010c, Breivik 2002). Globally, the total market demand for c-decaBDE differs considerably between countries and continents (Table 2.2 and 2.3, UNEP/POPS/POPRC.10/INF5).

25. The overall scale of c-decaBDE production today is currently unknown, and data on production, trade and stockpiles is only available for some countries. There is also very little information on the tonnages that may be imported in mixtures (chemical formulations, also resins, polymers and other substrates) and articles (either in semi-finished articles, materials or components, or in finished products). Production facilities for BFRs exist in all global regions (e.g. ACAP 2007, RPA 2014 in press, Annex E IPEN). At present it is unknown how many of these produce c-decaBDE. Among the main BFR producing countries China and India are known to produce and export c-decaBDE (Xiang 2007, Chen 2007b, Xia 2005, Zou 2007, Annex E IPEN and China). Japan produces c-decaBDE, but consumes all domestically (Annex E Japan). Production of c-decaBDE no longer takes place in the EU, the United States and Canada (ECB 2002, ECHA 2012a,b, ECA 2008, US EPA 2012).

26. At present, China is the largest producer and supplier of c-decaBDE with an annual production of around 21,000 tonnes (Ni 2013). Around 20 different Chinese companies claim to be suppliers of c-decaBDE (Annex E IPEN). Japan produces an estimated 600 metric tonnes of c-decaBDE per year (Annex E Japan), and there are currently two Japanese producers (Annex E IPEN). In 2002, the demand for c-decaBDE in Japan was 2200 tonnes/year and the stock level was about 60,000 tonnes (Sakai 2006). In 2013, the c-decaBDE import to Japan was 1,000 tonnes adding to a total consumption of 1,600 assuming that no exports occurred. India has six manufacturers and or suppliers (Annex E IPEN), but total production is unknown. In Europe, production of c-decaBDE ceased in 1999, but c-decaBDE is still imported in considerable quantities (ECB 2002, ECHA 2012a, c, RPA 2014 (in press)). In the United States the main producers and importers have committed to end all uses by the end of 2013. In 2012 the national production volume which includes both domestic production and import was 8215 tonnes/year. In Canada manufacture of octa-, nona- and decaBDE was banned in 2008 and, the three main manufacturers have committed to voluntarily phase-out all exports to Canada by 2013 (ECA 2008, 2013).

27. Besides any stocks of pure c-decaBDE, substantial stocks of c-decaBDE are also present in treated articles in the technosphere (UK EA 2009, Sakai. 2006).

2.1.2 Uses

28. C-decaBDE is a general purpose additive flame retardant, that is physically combined with the material in which it is used to inhibit the ignition and slow the rate at which flames spread. It is compatible with a wide variety of materials. Applications include plastics/polymers/composites, textiles, adhesives, sealants, coatings and inks (e.g. ECHA 2012c, 2013a, RPA 2014 (in press), Sakai 2006, Table 2.4, UNEP/POPS/POPRC.10/INF5).

29. End uses in plastics/ polymers include housings of computers and TV sets, wires and cables, pipes and carpets (BSEF 2013, US EPA 2014, Table 2.5, UNEP/POPS/POPRC.10/INF5). Typically c-decaBDE is used in plastics/polymers at loadings of 10-15% by weight, though in some cases loadings as high as 20% have been reported (ECHA 2012c). In a Japanese study, c-decaBDE is reported to account for about 98% of the bromine content found in plastic parts of older TVs (Tasaki 2004). BDE-209 is also found in products made from recycled plastics, including food contact materials (Samsonek and Puype, 2013).

30. In the textile sector, c-decaBDE can be used to treat a wide range of synthetic, blended and natural fibres (ECHA 2013a). Main end uses are upholstery, window blinds, curtains, mattress textiles, tentage (e.g. military tents and textiles, also commercial marquees, tents and canvasses) and transportation (e.g. interior fabrics in cars, rail passenger rolling stock and aircraft). The most common method of applying flame retardants to textiles is back coating. The amount that is applied will usually be in the range 7.5-20%. Padding processes and printing processes may also be used to apply flame retardant treatments (ECHA 2012a,c).

31. Information from public consultations in Europe report that c-decaBDE can be used in adhesives in the aeronautic sector for civil and defense applications (ECHA 2012d). Norwegian Authorities also identified uses of c-decaBDE in the adhesive layer of reflective tapes on work wear which are used as fire fighter uniforms, by the staff at oil platforms, in the energy sector, etc (CPAN 2012a). The reflective tapes contained c-decaBDE in the range 1-5% (by weight of reflective material). Other uses can be coatings used in applications for the building and construction sector and in inks (RPA 2014 (in press)).

32. According to VECAP data, textiles and plastics account for 52% and 48% of the c-decaBDE volume sold in Europe, respectively (VECAP 2012). In Japan 60% of the c-decaBDE is used in vehicle seats, 19% in construction materials and 15% in textiles. The remaining 6% is used for other purposes. In Switzerland 45% of c-decaBDE consumed were in electrical and electronic (EE) products, 30% was in imported motor vehicles and 25% in construction material (Buser 2007b). The consumption of c-decaBDE in the United States could be broken down as follows (excluding import in articles): automotive and transportation 26%, building and construction 26%, textiles 26%, electrical and electronic equipment (EEE) 13% and others 9% (Levchick 2010).

2.1.3 Releases to the environment

33. As an additive flame retardant, c-decaBDE is not chemically bound to the product or the material in which it is used. It therefore has the potential to ‘leak’ to the surrounding environment. Emissions of c-decaBDE to the environment may occur at all its life cycle stages, e.g. during production, formulation and other first- and second-line uses at industrial/professional sites, as well as during service life of articles, their disposal as waste and during recycling operations (ECHA 2012c, Ren 2014, Gao 2013, VECAP 2010a,b, 2014). The release and distribution of c-decaBDE to the environment via these routes is confirmed by monitoring data (see Section 2.3.1-2.3.4), and are likely to occur over a long time frame.

34. As a general purpose flame retardant c-decaBDE is used and released to the surrounding environment at many industrial and professional sites (e.g. VECAP 2012, 2014, Li 2013, Gao 2011, Odabasi 2009). In the EU alone there are more than 100 sites of second-line use (compounders/formulators, master batchers, injection moulders and finishers) (ECHA 2012a). Globally there are additional point sources (Annex E IPEN) contributing to the emissions of c-decaBDE including production sites for c-decaBDE as well as other industrial sources such as second-line users, recycling facilities and steel-production plants as well as other metallurgical facilities (e.g. Odabasi 2009, Wang 2010d, Lin 2012, Ren 2014, Gao 2011, Tang 2014). Elevated BDE-209 levels have been measured in the vicinity of industrial facilities (e.g. Zhang 2013d, Wang 2011d), and although VECAP estimates suggests otherwise (VECAP 2010a,b, 2012, 2014), the releases from industrial point sources to the surrounding environment can be considerable (ACAP 2007). For example, in 2003 the production of c-decaBDE in the United States released as much as 31 tonnes of c-decaBDE to the atmosphere alone (ACAP 2007).

35. Emissions of c-decaBDE during the service life and upon disposal of products are considered to be substantial. An assessment by the UK Environment Agency indicated that the main source of emissions were from landfill and waste incineration, followed by releases of waste water and releases to air from articles during service-life. Polymers and textiles in articles and waste were the main contributors (UK EA 2009, OSPAR 2009). Similar findings are reported also by others (ECHA 2012c, ACAP 2007, Sakai 2006). In addition, recycling can also be an important source to environmental releases of BDE-209 (Yu 2008, Gao 2011, Tang 2014 and references therein).

36. Controlled product testing has indicated low or no emission of BDE-209 from synthetic, vulcanised rubber products (Kemmlein 2003). However, BDE-209 has been shown to be emitted to the surrounding environment from textiles and TV-casings (Kemmlein 2006, Kajiwara 2013a). Higher levels of BDE-209 are moreover typically reported in indoor environments with many c-decaBDE containing products such as office environments, airplane interiors etc. (Björklund 2012, Allen 2013). BDE-209 is also reported to be the most prevalent PBDE congener in house dust and indoor air (e.g. Harrad 2010, Fredriksen 2009a, Besis and Samara 2012, Fromme 2009, Coakley 2013, EFSA 2011). BDE-209 in indoor environments is also a significant source to BDE-209 pollution in urban outdoor air (Björklund 2012, Cousins 2014), and to human exposure (see Section 2.3.4). Based on measurements in sewage sludge the estimated releases of BDE-209 from the technosphere, in Europe, are 16±8.6 tonnes annually and 41±22 mg annually per person or 0.2% of annual c-decaBDE usage in Europe (Ricklund 2008). Hence, use of c-decaBDE in the production of textiles and electronics result in environmental releases of BDE-209 and other PBDEs, either during production or directly from articles or during disposal stage (RPA 2014 (in press), VECAP 2010) and thus contribute to environmental relases and transboundary air pollution. Physical abrasion, disintegration and weathering as well as photolysis, increased temperatures and termal stress are all factors that contribute to release of c-decaBDE and lower brominated PBDEs from products (Earnshaw 2013, Chen 2013, Kajiwara 2008, 2013 a, b).

37. PBDEs are not removed during waste-water treatment (Danon-Schaffer 2007, Kim 2013b) and a substantial amount of c-decaBDE emitted from products during service life and as waste ends up in waste-water treatment plants (WWTP) through the disposal of wash water from contaminated indoor dust, leachate from landfilled PBDE-containing products and discharge from industrial sites processing PBDE containing material, and ultimately biosolids (Kim 2013a,b). In line with this, elevated levels of BDE-209 is reported in sediment near the outflow of WWTP and sludge (biosoil) is found to be an important pathway for BDE-209 emissions to soil when applied as an agricultural fertilizer (Sellström 2005, de Wit 2005).

38. Emission estimates are available for some countries (e.g. ECB 2002, Morf 2003, 2007, 2008, Palm 2002, Sakai 2006 as cited in Earnshaw 2013, Buser 2007a). A comparison of available European estimates shows large differences in predicted environmental emissions of BDE-209 to all environmental compartments and for air in particular (three orders of magnitude, Earnshaw 2013). The dissimilarities likely reflect country specific differences in production, use and waste disposal as well as uncertainties/ differences in emission estimates and overall suggest that release estimates should be viewed in light of environmental monitoring data.

39. With regards to time-trends, emission estimates for the period 1970 to 2020 calculated by Earnshaw (2013) using a dynamic substance flow analysis model and available consumption data indicate that BDE-209 atmospheric emissions in Europe increased steadily from the 1970s and reached a peak in 2004 at 10 tonnes/ year. Emissions to soil and the hydrosphere are lower but follow a similar trend of increase from the 1970s, peaking in the late 2000s and declining thereafter. Emissions to soil peaked at 4 tonnes/year in 2000 whilst those to the hydrosphere peaked in the 2010s at 3.5 tonnes/ year. In Switzerland maximum emissions is estimated to have occurred in the 1990's (Buser 2007b, Morf 2007). 31 tonnes of BDE-209 were released to air in 2003 alone according to the US EPA Toxic Release Inventory (TRI) Public Data Release (ACAP 2007), in 2011 the release to air was down to 3.1 tonnes (http://www.epa.gov/tri/).

40. Further information on potential emission sources and environmental levels resulting from releases of c-decaBDE to the environment is provided in Section 2.3.1. In general, as indicated by measured environmental levels, releases to the environment are higher in industrialized and urban areas than in rural and agricultural areas where there are fewer sources (see Section 2.3.1). Environmental levels are typically lowest in remote regions, such as the Arctic.

2.2 Environmental fate

41. The environmental fate properties of BDE-209 have been assessed in various reports published by the EU, Canada and the United Kingdom (ECB 2002, 2004, ECHA 2012a, ECA 2006, 2010, UK EA 2009). Fugacity modeling predicts that most of the BDE-209 (> 96%) in the environment partitions to sediment and soil (ECA 2010, ECHA 2013a). Less than 3.4% of BDE-209 is expected to be associated with bulk air or bulk water phases. Due to its intrinsic properties i.e. an organic carbon-water partition coefficient (Koc) in the range 150,900 to 149,000,000L/kg, BDE-209 is known to adsorb strongly to organic matter in suspended particles, sewage sludge, sediment and soil (ECHA 2013a). Given its low water solubility and strong particle affinity, its mobility in soils is also likely to be low (ECHA 2013a). Consequently, transfer to other environmental compartments, by soil erosion and run-off, will depend on particle bounded transport. In the environment BDE-209 is persistent and elevated levels is found in soil and sediments.

42. BDE-209 is also found to be a major PBDE congener in snow and ice in the Arctic (Hermanson 2010, Meyer 2012), showing that the detected air levels at lower latitudes contributes to long-range transport and pollution in remote areas. BDE-209 is also found in biota, sometimes at high levels, where it along with other PBDEs bioaccumulates and biomagnifies via the food chain (see Sections 2.2.4, 2.3.1. and 2.3.2). As further discussed in chapters 2.2.2 and 2.4.6 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.

2.2.1 Persistence

43. Photodegradation and biodegradation are the main mechanisms for transformation of BDE-209 in the environment (ECA 2006, 2010). 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 (Stenzel and Markley 1997), hydrolysis is unlikely to be a relevant degradation process in the environment (ECHA 2012a). Photodegradation might, however, be a contributing factor for BDE-209 degradation in air and topsoil (see Section 2.2.2). Yet, in the atmospheric compartment, BDE-209 will almost exclusively be adsorbed to air-particles. As air-particles protect the BDE-209 molecule, degradation in air via photolysis is not substantial (see Section 2.2.2).

44. High persistency of BDE-209 in soil, sediment and air is demonstrated in several studies and appears to be dependent on slow biodegradation processes and the degree of exposure to light (ECHA 2012a, ECA 2010). The type of particle to which BDE-209 is bound may also influence the degradation rate. For example, studies of photolytic degradation on various solid matrices has revealed half-lifes of 36 and 44 days for BDE-209 adsorbed to montmorillonite or kaolinite, respectively, with much slower degradation occurring when sorbed on organic carbon-rich natural sediment (t1/2 =150 days) (Ahn 2006). Half-life in sand was found to be only 35-37 hours while the corresponding half-lives in sediment and soil were estimated to be 100 and 200 hours, respectively (Söderström 2004 and Tysklind 2001 as cited in ECHA 2012a). In natural waters the presence of other organic substances such as humic substances can limit photodegradation by absorbing light or by hydrophobic interactions with the BDE-209 molecule (Leal 2013). Similarly, it has been shown that sand particles coated with humic acid may decrease degradation rates of BDE-209 when irradiated with UV light (Hua 2003). In addition the nature of dissolved organic matter, the amount of suspended particulates, the adsorption of BDE-209 to solid surfaces and the depth are of importance (Leal 2013). Increased adsorption to the soil or sediment matrix with ageing is an additional factor can contribute to longer environmental half-lives under natural conditions (ECHA 2013a).

45. Under conditions (e.g. deep sea sediments) where light attenuation and matrix shielding would affect overall exposure to sunlight and potential for photodegradation, the persistency of BDE-209 appears to be high (ECHA 2012a and references therein). 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 (Kuivikko 2007 in Leal 2013). The longest environmental half-life is reported by Tokarz (2008), who by conducting a laboratory microcosm experiment over a period of 3.5 years at 22°C under dark conditions found the half-life of BDE-209 in sediment to range between 6 and 50 years, with an average of around 14 years. The long half-lives found by Tokarz are underpinned by monitoring in the field. Kohler (2008) investigated concentrations and temporal trends of BDE-209 in the sediments of a small lake located in an urban area in Switzerland. BDE-209 first appeared in sediment layers corresponding to the mid 1970s and the levels increased steadily to 7.4 ng/g dry weight (dw) in 2001 with a doubling time of about 9 years. No evidence for sediment-related long-term transformation processes was found in this study covering almost 30 years.

46. Further evidence for the persistency of BDE-209 is provided by studies on sludge and soil. Liu (2011a) observed no degradation of BDE-209 after 180 days in soil samples spiked with BDE-209 in darkness. In another study on sludge-amended soil, the extrapolated primary degradation half-life under both aerobic and anaerobic conditions was found to be >360 days assuming exponential decay (Nyholm 2010, 2011, as cited in ECHA 2012a). In a controlled laboratory experiment at 37 °C under dark anaerobic conditions using digested sewage sludge spiked with BDE-209, the concentration of BDE-209 decreased by only 30% during the incubation time of 238 days (Gerecke 2005). These results are further strengthened by field studies. Eljarrat (2008) examined the fate of PBDEs in sewage sludge from five municipal WWTPs after agricultural application of sludge to the topsoil at six sludge application sites and one reference site. According to the authors, BDE-209 concentrations in soil remained high (71.7 ng/g dw) even at one site that had not received sludge applications for four years, illustrating the persistency of BDE-209 in soils. Similarly, Sellström (2005) measured PBDE levels in agricultural soil from sites that had received past sewage sludge amendments and found that levels in a farm soil between 0.015 to 22,000 ng/g dw even though contaminated sewage sludge had not been applied to the soil for many years. The highest levels were detected at a farm site that had not received amendments for 20 years.

2.2.2 Degradation and debromination

47. In spite of BDE-209's persistence and long environmental half-lives in sediment, soil and air, there is considerable evidence that BDE-209 is debrominated to lower brominated PBDEs in the abiotic environment, as well as in biota (ECHA 2012a,c, 2013a,b UK EA 2009, ECA 2010, POPRC 2010c, 2013a, NCP 2013). Observed debromination products detected range from mono- to nonaBDEs, and include listed POPs such as tetra-to heptaBDE and bromophenols, as well as recognized PBT/ vPvB substances such as brominated dioxins and furans (PBDD/ PBDF) and hexabromobenzene (Cristiansson 2009, UK EA 2009, ECHA 2012a,c, ECA 2010, see Tables 3.1 to 3.4 UNEP/POPS/POPRC.10/INF5). The biotransformation of BDE-209 in biota, in particular, is considered to be of concern in a number of recent reports and published studies (ACHS 2010, ECHA 2012a,c, EFSA 2011, ECA 2010, POPRC 2010a,b,c, Ross 2009, McKinney 2011a).

48. Abiotic degradation studies have shown the formation of nona- to triBDEs (reviewed in ECHA 2012c). The most unequivocal evidence that photodebromination occur in soil, sediment, air and other matrices come from controlled laboratory studies with natural sunlight. While the identity of the degradation products is inconclusive in some studies (Örn 1997, Palm 2003, Gerecke 2006), other studies provide good evidence for the formation of hepta- and hexaBDE congeners in freshly spiked sediment, soil and sand following exposure to light under laboratory conditions (Sellström 1998a, Tysklind 2001, Söderström 2003, 2004, ECHA 2013a, Jafvert and Hua 2001a, Eriksson 2004). Ahn (2006) found that debromination of BDE-209 adsorbed to minerals was a stepwise reaction, initially forming nona-, then octa- and heptaBDE congeners after 14 days exposure to sunlight, but with increased exposure time, hexa- to triBDEs were also formed. A number of studies, while not necessarily being representative of environmental conditions, have shown that microorganisms can influence BDE-209 degradation in soil and sediments as they are capable of transforming deca-, nona- and octaBDEs to at least hepta- and hexaBDEs (Robrock 2008, Lee and He 2010, Deng 2011, Qiu 2012). Photodegradation and debromination of BDE-209 have also been studied in abiotic material such as dust, plastic and textile exposed to light, and degradation products have been identified from hexa- to nonaBDE (Stapleton and Dodder, 2008, Kajiwara 2008, 2013a,b). Other degradation products such as PBDD/ PBDF, pentabromophenol and hexabromobenzene can moreover be formed from BDE-209 during processing (recycling), plastics production, photolysis, food preparation (cooking of fish) and waste disposal (Vetter 2012, Kajiwara 2008, 2013a,b, Hamm 2001, Ebert and Bahadir 2003, Weber and Kuch 2003, POPRC 2010b, Thoma and Hutzinger 1987, Christiansson 2009). Formation is strongly dependent on conditions like temperature and purity of the flame retardant.

49. Monitoring data provide supporting evidence that degradation of BDE-209 occurs under environmental conditions (ECHA 2012c, Hermanson 2010, Xiao 2012). Results provide evidence of the formation of small amounts of nona- and octaBDEs over a period of 30 days in lake sediments (Orihel 2014 in press, see also ECHA 2012c). A few studies demonstrate degradation of BDE-209 (mainly to nona- and octaBDEs) in sewage sludge (Stiborova 2008, Gerecke 2006, ECHA 2012c), as well as in precipitation (Arinaitwe 2014). Change in the congener ratio in sludge compared to commercial formulations has also been observed (Knoth 2007). Although minimal debromination during WWT was reported in the past (Kim 2013a, Zennegg 2013), findings support the contention that BDE-209 in sewage sludge can be dehalogenated to less brominated congeners (Hale 2012). In soil, debromination of BDE-209 is assisted by the presence of plants (Du 2013, Huang 2010a, 2013, Lu 2013, Wang 2011a, 2014). The distribution pattern of lower brominated PBDEs in plant tissues was different from the soil spiked with BDE-209 suggesting that debromination of BDE-209 in the soil occurred and that further debromination within the plants may occur (Du 2013, Wang 2011a, 2014).

An overview of degradation products in abiotic matrices is given in UNEP/POPS/POPRC.10/INF5 Tables 3.3 and 3.4.

50. Debromination is also shown in studies with higher vertebrates, including birds, fish and rodents (ECHA 2012a, c, UK EA 2009, ECA 2010, POPRC 2013a). While most vertebrates appear to be able to degrade BDE-209 to lower brominated PBDEs, different species may have different ability to debrominate BDE-209, with debromination occurring more rapidly and to a greater extent in some species than in others (McKinney 2011a).

51. Several laboratory experiments and field studies on fish have shown debromination of BDE-209 after dietary- or water exposure, or after injection of BDE-209 (Kierkegaard 1999, Stapleton 2004, 2006, Kuo 2010, Munschy 2011, Vigano 2011, Noyes 2011, 2013, Zeng 2012, Wan 2013, Feng 2010, 2012, Luo 2013, Bhavsar 2008, Orihel 2014 in press or as reviewed in ECHA 2012c). A number of apparent degradation products have been detected in terms of lower brominated PBDEs ranging from mono- to octaBDE. In several studies congeners (BDE-49, BDE-126, BDE-179, BDE-188, BDE-202) not present in any technical PBDE products have been detected and reported as evidence for biotransformation of BDE-209 (Munschy 2011, Wan 2013, Vigano 2011). The concentrations of BDE-209 and its degradation products varied between the different fish species which might be explained by species-specific differences in bioaccumulation capacity and metabolism between the fish species (Stapleton 2006, Luo 2013, Roberts 2011). Formation of hydroxy- and methoxyBDE degradation products have also been reported (Feng 2010, 2012, Zeng 2012).

52. A number of studies have also revealed debromination of BDE-209 in birds or bird eggs (reviewed by Chen and Hale 2010, Park 2009, Van den Steen 2007, Letcher 2014, Holden 2009, Munoz-Arnanz 2011, Mo 2012, Crosse 2012). In American kestrels exposed to BDE-209 via diet the half-life of BDE-209 was estimated to 14 days based on plasma concentrations measured during the uptake and elimination periods (Letcher 2014). In addition, debromination products from nona- to heptaBDE were observed. As observed in fish, BDE-202 along with other unidentified congeners not present in c-decaBDE was detected in bird eggs and seen as evidence for debromination (Park 2009, Holden 2009, Mo 2012). Furthermore, the ratio between nonaBDE/BDE-209 congeners in eggs or prey fish was higher than the ratio seen in the commercial mixture indicating biotransformation of BDE-209 in birds/bird eggs (Holden 2009, Mo 2013). The bird egg congener profiles differ markedly from what have been reported in marine and aquatic biota, where lower-brominated (tetra- and penta-BDE) congeners predominate. These differences in congener profiles may be due to lower bioavailability to BDE-209 compared to lower-brominated congeners, debromination- and depletion of BDE-209 in marine and aquatic biota (McKinney 2011a, Huwe 2008). Indication of BDE-209 biotransformation in the terrestrial environment was also shown by the high amounts of BDE-208 in earthworms following exposure to BDE-209 (Sellstrøm 2005, Klosterhaus and Baker 2010). An overview of degradation products in biota are reported in UNEP/POPS/POPRC.10/INF5 Tables 3.1 and 3.2.

53. Mammalian data indicate that debromination (nona - hepta-BDE) is the first step in biotransformation of BDE-209, followed by hydroxylation to phenols and catechols (Riu 2008, Wang 2010a, Huwe 2007), and that debromination either occurs in the intestine via metabolism by intestinal microflora or by first-pass metabolism by cytochrome P450 enzymes in the intestinal wall following uptake (Mörck 2003, Sandholm 2003).

54. The toxicity of lower brominated congeners is well known, thus debromination of BDE-209 to lower brominated congeners contributes to the outcome of c-decaBDE toxicity (Kodavanti 2011). Environmental degradation and/or biotransformation of BDE-209 showing degradation/transformation to POP BDEs (BDE-47, 99, 153, 154 and 183) has been reported (Wan 2013, Letcher 2014, She 2013, Zhang 2014, Munschy 2011, Stapleton 2004, Feng 2010, Luo 2013, Lu 2013, Huang 2013, see UNEP/POPS/POPRC.10/INF5 Tables 3.1-3.4). Due to the wide distribution and high persistency organisms are continuously exposed, through their lifetime, to a complex mixture of BDE-209, lower brominated BDEs and other BDE-209 degradation products (ECHA 2012), increasing the likelihood for adverse effects (Ross 2009, McKinney 2011a, Kortenkamp 2014). A study by He (2011) showed that long-term chronic exposure to low doses of BDE-209 not only affects F0 growth and reproduction, but also elicits neurobehavioral alterations in F1 offspring. Bioaccumulation of BDE-209 and biotransformation to low concentrations of the congener's nona- to hexaBDE was observed and the risk of mixture toxicity was raised by the authors. Hence, uptake and bioaccumulation of BDE-209 together with its biotransformation to more bioaccumulative and toxic metabolites in organisms has the potential for significant adverse effects as a result of the combined exposures (see Section 2.4.6). Similar results have been reported by Noyes (2011) and Chen (2012a).

2.2.3 Bioavailability and tissue distribution

55. The bioavailability of BDE-209 is low due to its high molecular weight that affects its passive diffusion through biological membranes (Frouin 2013, Mizukawa 2009) and its strong affinity for particles i.e. sediment and soil (Tian and Zhu 2011, see also Section 2.2.1). In spite of that and as confirmed by monitoring data from across the world (see Section 2.3 and Tables 5.1 and 5.2 UNEP/POPS/POPRC.10/INF5) and available laboratory studies, detectable and sometimes high BDE-209 levels have been measured in a wide variety of tissues, species, food webs and top predators.

56. As discussed in Section 1.1 the water solubility of BDE-209 is low and bioavailability via direct exposure through aquatic media is reported to be very limited (Ciparis and Hale 2005, Klosterhaus and Baker 2010). However, some evidence for the bioavailability of BDE-209 in medaka following direct water exposure is shown (Luo 2013). Due to its propensity for particle binding BDE-209 is considered to be bioavailable via food and through ingestion of particles, such as dust, sediment, soil or sand (ECHA 2012c). Uptake of BDE-209 through diet was evaluated in fish (Kierkegaard 1999, Stapleton 2004, 2006) and shown to range between 0.02% and 3.2% dependent on the species and whether debromination products of BDE-209 were taken into account when estimating the total uptake. Evidence for bioavailability of BDE-209 in terrestrial species is shown by the many studies showing uptake of BDE-209 into birds (Letcher 2014, Sagerup 2009, reviewed by Chen and Hale 2010), and is underpinned by biomonitoring data evidencing uptake also in other wildlife species, as well as in humans (Section 2.3). In rats, oral absorption is reported to range from 1-26%, inhalation absorption is estimated to be negligible (El Dareer 1987, Mörk 2003, Sandholm 2003, Riu 2008), and in an in vitro experiment dermal absorption was less than 20% (Hughes 2001). Furthermore an in vitro assessment using a human gastrointestinal tract model showed that BDE-209 was bioaccessible (14%) after exposure to indoor dust samples (Abdallah 2012). In the rat and cow the majority of BDE-209 administered is recovered in feces as the original compound (Kierkegaard 2007, Huwe 2008, Riu 2008, Biesemeier 2010).

57. Studies have shown that BDE-209 preferentially sequesters to blood-rich tissues such as muscle, liver, intestine, gills (fish), and to lesser extent to adipose tissue (e.g. Shaw 2012, Wan 2013, EFSA 2011, ECB 2002, 2004). The sequestration to blood rich tissues may possibly be explained by BDE-209 binding to proteins (Hakk 2002, Mörck 2003). In Chinese sturgeons, lipids did not play an important role in the distribution of BDE-209 (Wan 2013). BDE-209 was detected at relatively high concentrations in organs participating in absorption, uptake and metabolism such as the liver, gills, and intestines with liver having the highest concentration of BDE-209 followed by gills. Furthermore, the estimated partition coefficients between tissues and blood were higher than those of less brominated BDEs suggesting that the low partition ratios from blood to tissues would lead to high bioaccumulation of BDE-209, especially in absorbing organs (Wan 2013). Since this organism almost stops feeding during the migration period in the Yangtze River it can be assumed that the sturgeon was in a depuration period. A similar pattern was observed in a bioaccumulation study on harbor seals (Shaw 2012). Average hepatic ΣPBDE (tri- to octa-BDE) concentrations were similar to those of the average seal blubber ΣPBDE (mono- to hexa-BDE) as reported in Shaw (2008). In contrast, BDE-209 concentrations in liver were up to five times higher than those in blubber, which is consistent with observations that BDE-209 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 (EFSA 2011, Seyer 2010, Riu 2008). In lactating cows fed naturally contaminated silage BDE-209 was the dominating congener in feed, organs, adipose tissue and feces, but not milk (Kierkegaard 2007). 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 (Letcher 2014).

58. Human data demonstrate that BDE-209 is absorbed and distributed to fat, blood, cord blood, placenta, fetuses and breast milk (Frederiksen 2009a, Zhao 2013; UNEP/POPS/POPRC.10/INF5, Table 4.1). Maternal transfer of BDE-209 to eggs and offspring has also been reported in fish, frogs, birds, rats and reindeer (Vorkamp 2005, Lindberg 2004, Johansson 2009, Garcia-Reyero 2014, Nyholm 2008, Rui 2008, Biesemeier 2010, Cai 2011, Holma-Suutari 2014, Liu 2011c).

2.2.4 Bioaccumulation

59. In the past, bioaccumulation of BDE-209 in biota was assumed to be low, mostly attributed to the large molecular size and extreme hydrophobicity and low bioavailability of BDE-209 (Hale 2003). However, low bioaccumulation can also be explained by factors such as low uptake and/or high ability to eliminate or metabolize BDE-209 through excretion and debromination of BDE-209 (Hale 2003, Arnot 2010). Furthermore, variable results for bioaccumulation can also be explained by analytical challenges to measure BDE-209 (Ross 2009, de Boer and Wells 2006, Covaci 2003, Kortenkamp 2014) and/or debromination to lower PBDEs. As noted by ECA (2010) a complete evaluation of a substance's bioaccumulation potential should consider the bioaccumulation potential of both the parent substance

and its metabolic product(s). Environmental monitoring studies show that BDE-209 is found to be present in a variety of species as well as humans all over the world, and provide supporting evidence for bioaccumulation (see section 2.3.1 and UNEP/POPS/POPRC.10/INF, Table 5.2). The octanol-water partition coefficient (log Kow) values for BDE-209 reported in the literature are highly variable ranging from 6.27 to 12.11 depending on the measurement or estimation method used (CMABFRIP 1997, Dinn 2012, ECA 2010, Kelly 2007, Tian 2012, US EPA 2010, Watanabe and Tatsukawa 1990). While compounds with a log Kow >5 are considered bioaccumulative, chemicals (like BDE-209) with a log Kow > 7.5 are thought to be less bioaccumulative because of predicted declines in dietary absorption potential (Arnot and Gobas 2003). However, results from food web studies show that BDE-209 bioaccumulates (BMF>1) in both aquatic and terrestrial species (Yu 2011, 2013, Wu 2009a, EC 2010 and references therein).

60. The BCF for BDE-209 in fish has been estimated to be <5000 with non-appreciable aqueous uptake predicted due to its large size and low water solubility (ECHA 2012 a, ECA 2010). However, BCF is not considered to be a good descriptor of the bioaccumulation capacity of strongly hydrophobic substances, such as BDE-209. BCFs represent processes of chemical absorption by an aquatic organism from the ambient aqueous environment through its respiratory and dermal surfaces with no dietary considerations (Arnot and Gobas 2006). According to the revised OECD guidelines for bioaccumulation studies, testing via aqueous exposure may become increasingly difficult with increasing hydrophobicity. Hence, for strongly hydrophobic substances (log KOW >5 and water solubility below ~ 0.01-0.1 mg/L) a dietary test is recommended (OECD 305, 2012).

61. For terrestrial organisms log KOW and BCF are not good predictors of biomagnification for chemicals with log KOA ≥6 and log KOW>2 (Kelly 2007, 2009) and in terrestrial food chains chemicals with log KOW<5 and BCFs<5000 have been shown to biomagnify. As previously mentioned, the most important exposure route for BDE-209 in aquatic and terrestrial food webs is through the diet (Shaw 2009, Kelly 2007). The accumulated levels of BDE-209 in sediment-associated organisms and filter feeders (mussels, zoo plankton, crustacean, flat fishes, benthic invertebrates and aquatic worms) have been interpreted to be the result of uptake of particles containing adsorbed BDE-209 and not as evidence of bioaccumulation, however, uptake of particles is considered to be an exposure route for higher trophic levels in aquatic food webs (Shaw 2009 and references therein). In terrestrial ecosystems, BDE-209 adsorbs strongly to atmospheric particulates (i.e. aerosols) due to its high octanol-air partition coefficient (KOA), and will be deposited to terrestrial vegetation and soil through wet and dry deposition (Christensen 2005, ECA 2010, Mizukawa 2013, Yu 2011). This provides an exposure route for terrestrial organisms that ingest soil or plants as a food source. Therefore, when considering the bioaccumulative behaviour of BDE-209, calculated or measured BAFs, biomagnification factors (BMFs) and trophic magnification factors (TMFs) are believed to give more relevant information than calculated or measured BCFs (Shaw 2009, Kelly 2007, Powell 2013).

62. The BAF represents the bioaccumulation of chemicals in an organism by all routes of exposure, including by dietary and ambient sources. Historically, limited data have been available to allow for estimations of BAFs for BDE-209 as numerous studies measured BDE-209 concentration but did not compare these values with ambient levels. As a result, previous assessments found equivocal evidence concerning whether BDE-209 is bioaccumulative (ECHA 2012a,c, ECA 2010, US EPA 2010). Since this time, additional studies have measured BDE-209 BAFs in biota from regions captured by these assessments as well as from other areas. In particular logBAF values for aquatic fish and invertebrates range between 3.3 and 7.2 based on data published recently having logBAFs>3.7 (Frouin 2013, Mizukawa 2009) corresponding to a BAF>5000 (He 2012, ECA 2010), and fulfilling the criteria on bioaccumulation in Annex D (BAF>5000). In the above mentioned studies the BDE-209 uptake by aquatic organisms were measured and compared to water concentrations and logBAFs were estimated.

63. BMFs and TMFs from field data show that BDE-209 can biomagnify in several aquatic and terrestrial organisms and food webs (BMFs >1 and TMF>1; see UNEP/POPS/POPRC.10/INF5, Table 3.5 for details). BMFs range between 1-7 in terrestrial organisms and food webs as reported in the scientific literature (Yu 2011, Wu 2009a) and estimated from modeling (Kelly 2007). In a terrestrial food web study spanning several trophic levels BMFs ranging between 1.4 and 4.7 were reported (Yu 2011, 2013). In another field study on frog (influenced both by aquatic and terrestrial environments) focusing on biomagnification from insect to frog, BMFs ranged from 0.8-13.0 depending on gender (Wu 2009a). In aquatic organisms, BMFs ranged between 0.02 and 34. BMFs in seal blubber- and blood have been found to be in the range 0.03-0.06 and 8.3-20.8, respectively (Thomas 2005 as reported in ECA 2010). Other studies report BMFs between 0.2 and 2.2 for harbor seals (Jenssen 2007), 1.5 for marine biota (Baron 2013), and 1.28 for rainbow trout (Stapleton 2006 as reported in ECA, 2010). Furthermore BMFs between 0.1 and 34 were reported in an aquatic food web study by Law (2006). In a number of other aquatic studies BMFs from 1.2 to 5.1 (Mo 2012), 0.67 to 1.3 (Shaw 2009), 4.8 to 12.7, although there were some uncertainties regarding the food web (Tomy 2009), and between 0.02 and 5 (Burreau 2004, 2006, reviewed in ECA 2010) have been reported. In aquatic

food webs TMF values have been reported as 3.6 (Law 2006), 0.26 (Wu 2009b), 0.78 (Yu 2012, as reviewed in ECA 2010) and 0.3 (Tomy 2008). Most of the reported BMFs and TMFs for BDE-209 have all been calculated using muscle (fish, mammals, and birds), whole body (bivalves, zooplankton, and fish) or adipose tissues (fish and mammals). The differences between BMFs and TMFs reported may be species dependent and influenced by overall condition of the organism, diet, exposures and tissue analysed, metabolism, sex, and food web structure. Furthermore, trophic dilution (TMF<1) is observed in several of the above studies and this might be due to biotransformation of BDE-209 through the food webs. Trophic magnification (TMF>1) is observed for known BDE-209 biotransfomation products (Wu 2009b) and the BMF for BDE-202 ranged from 4.7 to 20 in a common kestrel/rat/sparrow food web (Yu 2011). Hence, bioaccumulation of degradation products as a result of BDE-209 exposure might be observed rather than bioaccumulation of BDE-209 itself.

64. The biota-sediment accumulation factor (BSAF) represents the steady state concentration ratio of a contaminant between an organism and sediment, and can provide further insights into bioaccumulation and biomagnification potential. Calculated sediment BSAF values for BDE-209 suggest low biomagnification potential (BSAF<1) in a number of studies (Klosterhaus and Baker 2010, He 2012, La Guardia 2012, Sellstrøm 2005,Tian and Zhu 2011, Xiang 2007, reviewed in EC 2010). But some studies show higher sediment BSAFs > 3 suggestive of bioaccumulation potential in some shellfish species (deBruyn 2009, Wang 2009). In the study by deBruyn (2009), BDE-209 concentrations were either low (BSAF≤ 1.48) or below the limits of quantification for most samples except at one reference site where the sediment BSAFs was calculated as 3.53 (deBruyn 2009). However, in a recent study in a soil invertebrate food web BSAFs for BDE-209 ranged from 0.07 to 10.5 following land-application of biosolids. In the same study, BMFs ranged from 0.07 to 4.0, however, there was some uncertainties regarding the ΔδN isotope analysis and the authors further concluded that soil contact is likely more important that trophic status in determination PBDE accumulation in soil invertebrates inhabiting sludge-applied sites (Gaylor 2014) (see UNEP/POPS/POPRC.10/INF5, Table 3.5 for details). The interpretation of BSAF values for BDE-209 is complicated by the fact that BDE-209 metabolism varies widely across species, extremely high sediment levels in some of the field studies and problems of getting clean and sediment-free samples for sediment living organisms (ECHA 2012c, La Guardia 2012).

65. Some studies have observed higher biomagnification or increased accumulation potential of BDE-209 in terrestrial organisms compared to aquatic organisms (Christensen 2005, Chen and Hale 2010b, Jaspers 2006, Kelly 2007, Voorspoels 2006a). This is expected given the physico-chemical properties of BDE-209 and the differences in toxicokinetics between terrestrial and aquatic organisms as defined by Kelly (2007). Kelly (2007) calculated higher BMFs for BDE-209 among terrestrial carnivores and humans (BMF=8) than for marine mammals (BMF=3) with the lowest BDE-209 BMF values in terrestrial herbivores and aquatic organisms (BMFs=1). This study concluded that BMFs for very hydrophobic chemicals like BDE-209 were higher in air-breathing animals than water-breathing animals due to slower respiratory elimination and slow elimination via urine that was reflective of the compound's high Koa and Kow values, respectively. Conversely, other studies show that BDE-209 absorption in some teleost fish occurs at a slow rate, which may allow for lower bioaccumulation potential and greater metabolism (or debromination) and elimination of BDE-209 than seen in terrestrial species (Mörck 2003, Stapleton 2004, Kierkegaard 1999). Available data also suggests that there are additional variables influencing the bioaccumulation of BDE-209. For instance, BDE-209 partitions strongly to sediments and soils, is highly persistent, and is the dominant or one of the most predominant PBDEs detected in abiotic compartments of the global environment (see section 2.3.1). This ubiquitous abiotic contamination sometimes at high levels may allow BDE-209 to enter food webs and reach steady state levels in biota despite the large molecular size (MW=959) and high log Kow (Stapleton 2004).

66. Little is known about the bioaccumulation of BDE-209 (or other PBDEs) in plants and in herbivores. BDE-209 was examined in a small herbivorous food chain (paddy soils, rice plants, and apple snails) from an electronic waste recycling site in South China. The BMFs for BDE-209 from the rice plant to snails ranged between 1.2 and 6.3 and clearly demonstrated that BDE-209 can bioaccumulate in the plant/herbivore food web (She 2013). On the other hand, bioaccumulation was not observed in a recent laboratory experiment where apple snails were dietary exposed to BDE-209 (reported BAF was <1) (Koch 2014). Hence from these two studies a clear conclusion of bioaccumulation in snails cannot be made. Many of the reported concentrations of BDE-209 in biota are given on a lipid-normalised basis. Although this is common practice when reporting the levels of bioaccumulative substances, in retrospect this might not be the best analytical approach for those substances that do not partition significantly to lipid (OECD 2012), as is possibly the case for BDE-209 (see section 2.2.3). Studies showing a lack of BDE-209 biomagnification in fish and mammals have been based on levels detected in muscles or adipose tissue and/or have been lipid normalised. As discussed in section 2.2.3 evidence shows that BDE-209 preferentially sequesters to blood-rich tissues,

such as the liver, intestine, muscle and gills, hence some previous studies may have targeted the wrong tissue and underestimated the bioaccumulation and biomagnification potential of BDE-209 (Stapleton 2004, Voorspoels 2006a, Wan 2013).

67. Debromination of BDE-209 to lower brominated and more bioaccumulative PBDEs after uptake of BDE-209 in organisms (see section 2.2.2) adds to the concern about the use and releases of BDE-209, since some PBDEs are already listed in Annex A of the Stockholm Convention for global elimination, and/or are vPvB and PBT substances in the EU (POPRC 2006, 2007, ECHA 2012a, ECA 2010). Studies of toxic effect and debromination to lower PBDEs have been paralleled by observations of bioconcentration and bioaccumulation of BDE-209 (Garcia-Reyero 2014, Noyes 2011, 2013, Kuo 2010). Furthermore the the presence of lower brominated PBDEs in field studies can be due to both debromination of BDE-209 and as a result of direct exposure from c-pentaBDE or c-octaBDE.

2.2.5 Potential for long-range environmental transport

68. Along with other less brominated PBDEs, BDE-209 is found in various environmental compartments in the Arctic and Antarctic including air, sediment, snow, ice, soil, sediment and biota (UNEP/POPS/POPRC.10/INF, Tables 5.1 and 5.2).

69. Several studies have reported that BDE-209 is the predominant or one of the dominating PBDEs in Arctic air (Wang 2005, Su 2007, Hermanson 2010, Hung 2010, Möller 2011b, Meyer 2012, NEA 2014, Salamova 2014). The levels of BDE-209 in the Arctic atmosphere together with studies showing a significant deposition on Arctic ice (Hermanson 2010) and snow (Meyer 2012) 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 HBCD, and substantially higher than for other PBDEs (Hermanson 2010). 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 (Dickhut 2012).

70. BDE-209 deposited to the Arctic environment is bioavailable to the organisms living there and is widespread in Arctic food webs (de Wit 2006 and 2010, ECA 2010, NCP 2013). Arctic biota samples contaminated with BDE-209 include e.g. vegetation, birds of prey, seabirds and seabird eggs, marine and freshwater fish, different amphipods, zooplankton, shrimps and clams, terrestrial and marine mammals (de Wit 2006 and 2010, Letcher 2010, Tomy 2008). Typically Arctic biota is co-exposed to BDE-209 and a multitude of other PBDEs and POPs (de Wit 2006, 2010, Letcher 2010). Biomonitoring data have shown that BDE-209 contributes significantly to the total body burden of PBDEs in some Arctic species, accounting for >50% of total PBDE burden in detritus feeding ice-amphipods (Sørmo 2006), 60% in redfish and 75% in Arctic cod (Tomy 2008). BDE-209 is also the predominant congener in moss samples from remote sites in Norway (Mariussen 2008).

71. BDE-209 is also found in air in remote areas of Asia on the Tibetan Plateau (Xiao 2012, Xu 2011). Snow pack samples in the Tartra Mountains in Slovakia showed remarkably high levels of BDE-209 (Arellano 2011). 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 (Möller 2012, Möller 2011a,b, Lohmann 2013). Both oceanic and atmospheric processes contribute to the environmental transport of BDE-209 (Su 2007, Möller 2011a,b, Breivik 2006). 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 (Breivik 2006, Wania and Dugani 2003). 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 (Wilford 2008, Meyer 2012). Furthermore particles can protect the BDE-209 molecule from photolysis and lengthen its life-time in the air to >200 days (Breivik 2006, Raff and Hites 2007 as cited in de Wit 2010). In the Arctic, the deposition of airborne particles is found to be higher during the Arctic haze season (Su 2007, AMAP 2009). In tropical Asia, long-range environmental transport of PBDEs including BDE-209 associated with gas and/or particles is assisted by the monsoon (Xu 2011).

72. 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 5x105 molecule cm-3 and a reaction rate of 1.7x10-13 cm3 molecule-1 s-1 (ECB 2002). Other applications such as EPISuite 4.1 (AOPwin module) and PBTProfiler estimate a different reaction rate (3.37x10-14 cm3 molecule-1 s-1) and therefore predict even longer half-lives of 317 days (12 h day, 1.5 x 106 OH radicals cm-3) and 470 days (24 h day, 5x105 molecules cm-3) respectively.

73. Although local sources of releases may be present (Hale 2008, Danon-Schaeffer 2007, Li 2012c), the available data from remote regions overall shows that BDE-209 is detected in these areas as a result of long-range environmental transport.

2.3 Exposure

2.3.1 Environmental levels and trends

74. BDE-209 is widely dispersed in the global environment and is found in biotic and abiotic matrices worldwide. An overview of environmental levels is reported in several reviews (de Wit 2006, 2010, ECA 2010, Letcher 2010, Law 2014) and in UNEP/POPS/POPRC.10/INF5 Table 5.1 and 5.2. In most environmental matrices BDE-209 co-exists with other PBDEs and is the main or one of the dominating PBDEs detected.

75. BDE-209 is detected in air in urban, rural and remote regions (UNEP/POPS/POPRC.10/INF5 Table 5.1), as well as in precipitation (Ma 2013, Robson 2013, Arinaitwe 2014 and references therein). In urban and rural environments detected levels range between 4.1 and 60 pg m-3 (as reviewed by Syed 2013) while concentrations in Arctic air ranges from non-detectable to 41 pg m-3 (reviewed by de Wit 2010). 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 (Xiao 2012, Möller 2011a,b, 2012). Recently, however, Lohman (2013) reported particle-bound and gas-phase BDE-209 concentrations in tropical Atlantic Ocean air as high as 43.89 and 260 pg m−3, respectively. Based on these measurements Lohman (2013) calculated the total deposition of BDE-209 to the Atlantic Ocean from air 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. As described above, both in the Arctic and the tropics, air-transport of BDE-209 is influenced by seasonal weather phenomena (Xu 2011, Su 2007, AMAP 2009). Air mass back trajectory analysis indicates that major potential source regions of BDE-209 are widely distributed in industrialized and urbanized areas in tropical Asia (Xu 2011).

76. 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 (reviewed by Wang 2010b). BDE-209 was detected in soil at landfill sites in Arctic Canada (Danon-Schaeffer 2007, Li 2012c), but PBDE soil levels outside the landfills were similar to levels measured in soil at background locations elsewhere in the Arctic (de Wit 2006, 2010), 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 (Wang 2011b, reviewed by Wang 2010b, Gao 2011, Li 2012a). 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 (de Wit 2005, NEA 2012, NERI 2003, Annex E Denmark, Ricklund 2008a,b, Earnshaw 2013). As shown by Sellstrøm (2005) and de Wit (2005) 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 (Sellstrøm 2005).

77. Reported BDE-209 levels in sediments worldwide range from non-detectable to 16,000 ng/g dw i.e. slightly higher than in soil (see Wang 2010b, Eljarrat 2007, Sellstrøm 1998b; POPRC/ INF Table 5.1). High concentrations in sediment are typically found in the vicinity of industrial sites (Wang 2010b, Eljarrat 2004, 2005, 2007, Sellstrøm 1998b). 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 (Wang 2010b, Eljarrat 2005, Marvin 2013). Levels of BDE-209 in soil and sediment in remote regions are low (de Wit 2006, 2010, CPAN 2010, 2012b, Boitsov and Klungsøyr 2013, SFT 2008a,b), 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 (Hale 2008, Danon-Schaeffer 2007, Li 2012c). The doubling time for BDE-209 in these sediments range between 5.3 and 8.4 years (Chen 2007b, Kwan 2014, Zhu and Hites, 2005, Zegers 2003; UNEP/POPS/POPRC.10/INF5 Table 5.1).

78. BDE-209 is also found in a variety of terrestrial and aquatic species globally (de Wit 2006, 2010, Letcher 2010, ECA 2010, Chen and Hale 2010, NCP 2013; see UNEP/POPS/POPRC.10/INF5 Table 5.2). Measurements include plants, seabirds such as eider, guillemot, glacous- and herring gulls, different birds of prey, fish and marine invertebrates, marine crustaceans, insects and frogs, as well as marine and terrestrial mammals. BDE-209 is found in a variety of tissues in adults, as well as in eggs of oviparous organisms.

79. In the Arctic, reported biota levels range from non-detectable to 250 ng/g lw (UNEP/POPS/POPRC.10/INF5 Table 5.2). In areas more affected by antrophogenic activities levels up to ≥12 000 ng/g lw have been reported. High BDE-209 levels have often been shown in terrestrial environments, and likely reflect the low volatility of BDE-209 and its high affinity to organic matter in dust and soils (Chen and Hale 2010, Chen 2012d). In particular, BDE-209 levels in birds and bird eggs are extensively studied, and have in some instances reported to be very high. Common kestrels from China contain very high BDE-209 levels with concentrations of 2150 and 2870 ng/g lw, in muscle and liver, respectively (Chen 2007a). One specimen contained as much as 6220 ng/g lw in the muscle and 12 200 ng/g lw in the liver. These levels are among the highest BDE-209 levels reported in wildlife (Chen 2007a), and much higher than reported elsewhere (Bustnes 2007, Fliedner 2012, Johansson 2011, Sørmo 2011, Vorspooels 2006b, Gentes 2012, Chabot-Giguère 2013, Mo 2013, Chen 2010a, Chen and Hale 2010). High levels were also reported in kingfishers, Eurasian tree sparrows and common kestrels in China (Chen 2007a, Yu 2011, Mo 2012) and peregrine falcons in Sweden (Johansson 2011). BDE-209 is also detected in several species of Arctic birds (de Wit 2006, 2010). Red foxes in urban, rural areas in Belgium had liver levels opp to 760 ng/g lw, where BDE-209 typically contributed to ~70 % of the total PBDE load (Voorpoels 2006a). Another species found to contain high BDE-209 levels is earthworm with reported maximum concentrations of 5200 ng/g lw (Sellstrøm 2005).

80. Limited data on temporal changes in environmental BDE-209 levels is available. In the few studies reporting temporal trend data from abiota in the Arctic, BDE-209 levels in Canadian Arctic air were found to increase from 2002-2005 (Su 2007, Hung 2010), while the same was not observed from 2007-2009 (NCP, 2013). For the period 2002-2005 doubling times in the range of 3.5-6.2 years were reported (Su 2007, Hung 2010). No temporal trends of BDE-209 in air for 2007-2013 can be seen at the Norwegian Arctic sites (i.e. Zeppelin and Andøya observatory) (NEA 2014). Instead the concentrations fluctuate from year to year. In contrast, levels in Antarctic ice are reported as stable and unchanged in the period 2001-2007 (Dickhut 2012). In urban and rural air and precipitation where levels are influenced by both diffuse and point sources, the pattern is more complex and suggests either no significant change (Ma 2013), an increase (Arinaitwe 2014) or a decline (Robson 2013) in BDE-209 levels over time. It should be noted that in most studies on air or precipitation no clear temporal or spatial trends in is reported. Although BDE-209 is stabilized by binding to air particles (de Wit 2010), the lack of any observable time-trends may in some cases possibly result from photolysis/debromination of BDE-209 to lower brominated PBDEs (Wang 2005, Xiao 2012, see also Meyer 2012, Robson 2013, Arinaitwe 2014). With regards to geographical trends, geographical comparisons of air monitoring data are generally hampered by the fact that most studies report episodic monitoring data.

81. Concentration- and time trends of BDE-209 in sediment have also been reported. In sediment cores from a remote lake in Switzerland BDE-209 levels increased steadily in the period from 1990s to 2001 with a doubling time of about 9 years (Kohler 2008). Sediment cores from urban/polluted areas show doubling times for BDE-209 between 5.3 and 8.4 years (Chen 2007b, Kwan 2014, Zhu and Hites, 2005, Zegers 2003; UNEP/POPS/POPRC.10/INF5 Table 5.1). Based on a study in the south of China, the time at which BDE-209 started to increase in sediments in China seems to be 10-20 years later than those in North America and Europe, which might reflect differences in historical patterns of production and use of BDE-209 in these continents (Chen 2007b). In dated sediment cores taken from lakes in Ontario, Quebec and northern New York State along a latitudinal transect in North America, BDE-209 was generally detected only in recent sediment horizons, and sedimentation fluxes were found to decline exponentially with latitude (Breivik 2006).

82. Paralleling the observation of increasing levels of BDE-209 in Arctic air, Vorkamp (2005) observed a significantly increasing temporal trend in BDE-209 concentration in eggs of peregrine falcon from southwestern Greenland collected from 1986 to 2003. The measured concentrations in this study ranged from 3.8 to 250 ng/g lw, with a median of 11 ng/ g lw. An increace in BDE-209 was also observed in peregrine falcon eggs from Sweden collected from 1974 to 2007 from <4 to 190 ng/g lw (Johansson 2011). BDE-209 levels in UK peregrine falcon eggs were found to increase from 1975 to 1995, and thereafter to decrease from 1995 to 2001 (Leslie 2011). Increasing temporal trends in BDE-209 concentrations were also reported in herring gull eggs from the Laurentian Great Lakes in North-America (Gauthier 2008). From 1982 to 2006 BDE-209 doubling times ranged from 2.1 to 3 years. In contrast, steady state levels were reported in eggs of coastal herring gulls in Germany (Fliedner 2012). In the same study, no significant temporal trend was observed between 1973 and 2001 for BDE-209 concentrations in sparrowhawk muscle tissue, although some samples with higher concentrations were seen in later years. In Norway, geographical trends in BDE-209 levels are observed in moss (Mariussen 2008). Following a transect from south to north BDE-209 levels in moss is observed to decline, indicating that BDE-209 originating in source regions south of Norway are transported towards the Arctic via atmospheric processes and deposited along the way, resulting in the observed decreasing geographical gradient. BDE-209 levels in moss in Norway also appear to have

increased over time likely reflecting similar changes in air levels (SFT 2002, Mariussen 2008, CPAN 2012b).

2.3.2 Human exposure

83. 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 (HCA 2012). Detected BDE-209 concentrations in the indoor air range from <LOQ to 651 pg/m3 (Harrad 2010) and from 63 to 10,000 ng/g in dust from Germany, Sweden and the UK (Fromme 2009). The BDE-209 concentrations in dust exceeded by far the sum of the lower brominated PBDEs that have been detected (Frederiksen 2009a, EFSA 2011, Besis and Samara 2012). The concentrations of BDE-209 in North American house dust were comparable to those in Europe (<500-2000 ng/g) (Fromme 2009). Further, the occupancy in cars and airplanes may be a significant source to PBDE exposure as reviewed by Besis and Samara (2012). 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 (Brommer 2012). 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 (Coakley 2013).

84. BDE-209 is widely present in food and is reported in concentrations ranging from ~2 to >50,000 pg/g ww as reviewed by Frederiksen (2009a). 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 (Schecter 2011, EFSA 2011 and Riviere 2014). Contributions from drinking water and outdoor air to indirect BDE-209 exposure are low compared to intakes from food and often considered negligible.

85. The internal dose, e.g. assessed using human biomonitoring, reflects an integrated exposure over time comprising various sources and pathways. 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 (Frederiksen 2009b, Gomara 2007). 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) (Zhao 2013). Biomonitoring studies on cord blood showed BDE-209 median concentrations to be in the range <1.2 to 27.1 ng/g lw (UNEP/POPS/POPRC.10/INF5, Table 4.1). 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. The extensive review by Frederiksen (2009a) 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 (UNEP/POPS/POPRC.10/INF5, Table 4.1), 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 (Frederiksen 2009a). More recent studies show similar levels (UNEP/POPS/POPRC.10/INF5, 4.1), except from the strikingly high levels (mean 220 ng/g lw) reported from Laizhou in China, a previous production area of halogenated flame retardants (He 2013). 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 (Lignell 2011). 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 (Fängström 2005a). In summary, the biomonitoring data show widespread and ongoing exposures to BDE-209 throughout the world, and confirm fetal exposure and absorption in adults.

86. 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 (Sjödin 1999, Jakobsson 2002), while a median of 35 ng/g lw were reported among rubber workers (Thuresson 2005). The widespread recycling and dismantling of e-waste under primitive conditions in China has received increasing attention. Median BDE-209 concentrations in Guiyu (Bi 2007) 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 has been observed in a study by Qu (2007), i.e. 3,436 ng/g lw which is about 3,000 times higher than usually observed in general populations. In contrast, a recent study (Yang 2013) did not find significant difference between the residents in an e-waste recycling area and the reference group.

87. 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 (EFSA 2011). 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. Lorber (2008) reviewed exposure to PBDEs in the US and 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) (HCA 2012). Food and indoor dust were the dominant sources of exposure, contributing 51 and 45% to the total intake, respectively.

88. 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 (Kortenkamp 2013). Health Canada (HCA 2012) 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. 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 (Coakley, 2013). 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 decaBDE and other PBDEs from dust and breast milk measured in this study was below US EPA Reference Dose values (7 µg/kg bw d, US EPA 2008).

89. Several studies show that toddlers and young children have higher levels of PBDEs than adults (Frederiksen 2009a), which was also seen for BDE-209 (Fischer 2006, Lunder 2010, Sahlström 2014). 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 (EFSA 2011). 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 (Chen 2009). 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 (HCA 2012). 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 (Sahlström 2014). 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.

2.4 Hazard assessment for endpoints of concern

90. National and regional assessments conducted by the EU, the United Kingdom, Canada and US have evaluated the potential for c-decaBDE/BDE-209 to induce adverse effects in wildlife and humans (e.g. ECB 2002, 2004, 2007, UK EA 2009, ECHA 2012a, HCA 2006, 2012, US EPA 2008, EFSA 2011). In addition, the toxicity of BDE-209 and other PBDEs has been the topic of several scientific papers and reviews (see e.g. Dingemans 2011, Chen and Hale 2010, Costa and Giordano 2011, Kortenkamp 2014). In the literature toxic effects are reported for soil organisms, plants, birds, fish, frog, rat, mice and humans. Reported effects of BDE-209 range from changes at biochemical- and cellular level to effects which may have more direct implications at higher-levels of biological organization including survival, growth, behavior, immune function, reproduction, development, nervous system and endocrine modulating effects. In vertebrates, the liver, the thyroid hormone (TH) axis and the nervous system appear to be the main targets for BDE-209 toxicity (for review see Costa and Giordano 2011). In both wildlife and humans, early developmental stages appear more vulnerable to BDE-209 exposure than adults. In addition, debromination of PBDEs to more toxic PBDEs is a reason for concern in several assessments (UK EA 2009, ECHA 2012a,c, ECA 2010, Kortenkamp 2014). While some studies either report no effects or effects only at high doses, other studies suggest that BDE-209 may induce adverse effects at low and/or environmentally relevant concentrations.

[1] Lower bound: values < LOD/Q has been assigned 0 concentration. Upper bound: values < LOD/Q has been assigned the LOQ/Q-concentration.

2.4.1 Toxicity to aquatic organisms

91. C-decaBDE and its main constituent BDE-209 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 (e.g. ECB 2002, 2004, 2007, UK EA 2009). 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 (ECHA 2012 a).

92. 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 nona- and BDE-209, was shown to have the potential to affect development and metamorphosis in amphibians (Schriks 2006, 2007, Balch 2006, Qin 2010). 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. In a study by Shricks (2006) 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 (Qin 2010). 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 (ECHA 2012a). 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 (Ganser 2009). In this study 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 (Liu 2011c, Wu 2009a).

93. 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 (Noyes 2011, 2013). 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 (Noyes 2013). 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. Similarly, the study by Chen (2012a) indicated that BDE-209 has the potential to cause adverse effects in zebrafish at early life stages with impacts on T3 and T4 concentrations. Li (2011) observed changes in expression of TH-associated genes 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, Thienpont (2011) and Garcia-Reyero (2014) report no visible effect on thyroid function in exposed fish embryos. However, it should be noted that Thienpont (2011), who exposed 48 hours post fertilization embryos to 960 µg/L BDE-209 for three days conclude 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. Garcia-Reyero (2014) speculated that the absence of effects on the TH-system in their study may be explained by shorter exposure and/or lower doses than those used by Noyes (2011) and Chen (2012a). Potential TH disruption in fish by several PBDEs was also investigated in vitro by Morgado (2007) 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.

94. Other effects, both chronic and acute, have also been observed in fish following exposure to BDE-209. In the above dietary study from 2013, Noyes observed a significant increase in percent cumulative mortality as well as a decline in gonadal-somatic index. Chen (2012a) observed significant decreases in body weight and survival rate of zebrafish larvae exposed to 1.92 mg/L BDE-209 via water for 14-days. Significant changes were not observed at any of the lower exposure doses tested (0, 0.08, 0.38 mg/L).

95. 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 (Kuo 2010, de Wit 2002).

96. He (2011) documented 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. Several of the effects reported by He (2011) 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).

97. Potential reproductive toxicity of BDE-209 was also demonstrated in rare minnow (Li 2011). In this study, 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 larve 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.

98. In the above mentioned study (Garcia-Reyero 2014) 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.

99. 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 (Feng 2013a, b).

100. In several of the above fish studies BDE-209 was reported to debrominate to lower brominated PBDEs (Noyes 2011, 2013 Chen 2012c, Kuo 2010, He. 2011), 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.

101. 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. Based on Noyes (2013) 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 (Chen 2012a, He 2011, Noyes 2011). The levels used in some of the experiments were comparable to levels in more polluted areas (Zhang 2010a, Wang 2011b).

2.4.2 Toxicity in soil organisms and plants

102. Toxicity data are available for soil microorganisms, plants and earthworms. Most of the published data is new and was not reviewed in any of the previous risk assessments and evaluations (e.g. ECHA 2012a, UK EPA 2009, ECA 2006). Based on a plant toxicity study by (Porch and Krueger, 2001) and two 28- and 56-day toxicity studies with earthworms, ECB (2002) reported that no effects were seen on plants at concentrations up to 5,349 mg/kg dry weight. and that a NOEC $\geq$ 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.

103. More recent studies have shown that environmentally relevant levels of BDE-209, alone and in combination with copper, another typical contaminant of soil, can affect protein levels in earthworms as well as soil enzymatic activity and alter the bacterial community structure by reducing species richness (Zhu 2010, Liu 2011a, Zhang 2012, 2013c). In these studies, protein levels, soil enzymatic activity and soil community structure/ species richness were significantly affected at concentrations as low as 1 mg BDE-209/kg soil (Zhu 2010, Liu 2011a, Zhang 2012, 2013c), suggesting that BDE-209 may induce potentally adverse effects soil organisms at lower concentrations than previously thought and close to

environmental levels found in more polluted areas (e.g. Yu 2011, Li 2013, see UNEP/POPS/POPRC.10/INF5, Table 5.1).

104. Xie (2011) observed a significant increase in hydroxyl radical levels in earthworms at 0.01-10 mg/kg of BDE-209, which is within the range of environmental levels reported in soil (Syed 2013). The effect was paralleled by oxidative damage to protein and lipids and a reduction in antioxidant capacity. In this study oxidative stress and oxidative lipid damages were observed at concentrations as low as 0.01 mg/kg (Xie 2011). In a more recent acute earthworm study by the same authors, 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-signifcant changes in avoidance response at 1000 mg/kg BDE-209, no other effects were reported suggesting that adult earthwoms have a strong tolerance for BDE-209 in soils, but that a potential toxicity exist for earthworm embryos or juveniles (Xie 2013b).

105. In ryegrass seedlings exposed to 100 mg/kg BDE-209 Xie (2013a) observed a 35% inhibition of root growth and 30% decrease of the chlorophyll b and carotenoid contents of leaves. 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. Sverdrup (2006) did not observe effects 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 speculated that the absence of toxicity could be due to the low water solubility of BDE-209.

106. In summary, BDE-209 appears not to be acutely toxic to plants and soil organisms and adverse effects are generally observed at high doses (ECB 2002, Sverdrup 2006, Xie 2013 a,b). 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 (Zhu 2010, Liu 2011a, Zhang 2012, 2013c, Xie 2011, 2013a).

2.4.3 Toxicity in birds

107. As highlighted by Chen and Hale (2010), birds exhibit some of the highest concentrations of BDE-209 reported in wildlife and may be at risk for experiencing adverse effects (ECHA 2012a, see also UNEP/POPS/POPRC.10/INF5, Table 5.2). However, a limited number of studies examining adverse effects of BDE-209 exposure to birds are available.

108. 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 were found for reproductive parameters (Gilchrist 2014). BDE-209 concentrations were not reported.

109. 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 (ECHA 2012 a). Reported concentrations in bird eggs typically range between 1-100 µg/kg ww, but up to 420 µg/kg ww have been reported (ECHA 2012a). 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.

110. A reduction in body mass was observed in European starlings exposed to BDE-209 by silica implants (van den Steen 2007).

111. Birds are reported to metabolize BDE-209 to lower brominated PBDEs, including some POP-BDEs (BDE-183) (Letcher 2014) and exposure to lower brominated PBDEs have been associated with immunomodulatory changes, developmental toxicity, altered reproductive behavior, reduced fertility and reproductive success (for overview see Chen and Hale 2010, Glichrist 2014, POPRC 2007). 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 (Marteinsson 2010). 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 behavior (Viberg 2003, 2007) and hyperactivity (Johansson 2008, Rice 2007). For a comprehensive discussion on behavioral effects in rodents see chapter 2.4.4 below.

2.4.4 Toxicity in terrestrial mammals

112. 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 (Dingemans 2011, Messer 2010, Kicinski 2012, Costa and Giordano 2011, HCA 2006, 2012, Gascon 2012, Chao 2011, Kortenkamp 2014).

113. Developmental neurotoxicity is the reported critical endpoint of several PBDEs (Blanco 2013, Branchi 2002, Eriksson 2001, Kuriyama 2005, Rice 2007, 2009, Suvorov 2009, Viberg 2003, 2004, 2007, Xing 2009, Zhang 2013a, UNEP/POPS/POPRC.10/INF5, Table 6.1) and is thought to arise through disturbance of TH system and direct toxicity to neuronal- and stem cells. Developmental neurotoxicity has also been reported for BDE-209 in some studies (Johansson 2008, Viberg 2003, 2007, Rice 2007, 2009, Fujimoto 2011, Heredia 2012, Chen 2014, Reverte 2013, 2014), but not others (Biesemeier 2011). In support of the toxicological findings in previous studies (Johansson 2008, Viberg 2003, 2007, Rice 2007, 2009), neurobehavioral effects of BDE-209 in rodents during juvenile development or adulthood have also been reported more recently (Fujimoto 2011, Heredia 2012 Chen 2014, Reverte 2013, 2014). 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 (Reverte 2013, Heredia 2012). In a more recent 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 (Reverte 2014). Moreover Chen (2014) reported that 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.

114. 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" (OECD 2007). 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 by Viberg (2003, 2007) and Johansson (2008). Other researchers (e.g. Hardy 2008, 2009, Goodman 2009, William and DeSesso 2010) have noted limitations with the former studies. Despite this, the US EPA used the studies from Eriksson and Viberg in their derivation of oral reference doses for BDE-209 (as reviewed in US EPA 2008). A study conducted by Rice (2007) did not show a consistent depression in motor activity over time in mice, however their follow-up study showed neurobehavioral long-lasting deficits when tested at 16 months (Rice 2009). Similar to the findings of Viberg (2003, 2007) and Johansson (2008), 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) (Fischer 2008a, Viberg 2003, 2007, Liang 2010). In further support of findings indicating that BDE-209 can act as a neurotoxicant in mammals, Fujimoto (2011) showed 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 (2011), 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 (2011) 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 (HCA 2012) report, where lower LOAEL and NOAEL values have been suggested instead of the value reported in the original study.

115. In line with the findings of Chen (2014), other studies show that BDE-209 can exert direct toxic effects on neuronal cells (reviewed by Dingemans 2011, UNEP/POPS/POPRC.10/INF5 Table 6.2) and interfere with neuronal signaling, and induce oxidative stress and apoptosis (Chen 2010b, Huang 2010b, Liang 2010, Al-Mousa and Michelangeli 2012, Hendriks et al. 2012, Liang 2010, Xing 2010), effects that may lead to neurotoxicity and interfere with learning and memory by affecting long-term potentiation as shown by Viberg (2008) and Xing (2009). BDE-209 is further shown to cause changes in gene expression, intracellular protein levels, and disturbance of synaptogenesis and cell differentiation (Pacyniak 2007, Viberg 2008, 2009, Zhang 2010b, Song 2013).

116. 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 (Ahmed 2008, Gilbert 2012, Dingemans 2011). In vitro (Hamers 2006, Ibhazehiebo 2011, Ren 2013b) and in vivo studies assessing TH/TSH effects due to BDE-209 administration (UNEP/POPS/POPRC.10/INF5 Table 6.3) 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 (Lee 2010b, Chi 2011, Fujimoto 2011), also no change (Wang 2010a, Zhou 2001), and increase in T3 levels has been reported (Van der Ven 2008, Wang 2011c). For T4, animal studies report both decreases in T4 levels at high dose (Rice 2007, Kim 2009, Chi 2011, Fujimoto 2011) as well as no change in T4 level (Tseng 2008, Van der Ven 2008, Wang 2010a,2011c, Lee 2010b, Zhou 2001). For TSH, two animal studies performed with BDE-209 (Kim 2009, Lee 2010b) 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 (NTP 1986). 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 offsprings have indicated that low doses of BDE-209 may adversely affect the developing thyroid organ (Kim 2009, Lee 2010b, Fujimoto 2011). 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 ciritical 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.

117. 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 (Tseng 2006, 2013, van der Ven 2008). Effects on testicular development has also been reported following exposure at post natal days 1-5 at low doses (Miyaso 2012). 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 (van der Ven 2008) and female (Hamers 2006, Gregoraszczuk 2008) 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 (Hardy 2002). Similarly, Ernest (2012) reported that a mixture composed of three commercial BDEs (52.1% DE-71, 0.4% DE-79, and 44.2% decaBDE-209) 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 (van der Ven 2008). Further, BDE-209 can inhibit estradiol-sulfotransferase in vitro (Hamers 2006), which could implicate a (local) increase of endogenous estradiol in vivo. In another in vitro study Gregoraszczuk (2008) 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.

118. Oxidative stress and impaired glucose homeostasis has 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 (Zhang 2013e). Reduced insulin levels and increased levels of tumor necrosis factor- (TNF-□) 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 (Zhang 2013e). Van der Ven (2008) also observed insulitis 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.

119. Immunotoxic effects of BDE-209 have been reported in some studies (Teshima 2008, Watanabe 2008, 2010, Zeng 2014), 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 (Zeng 2014). In contrast to these studies, van der Ven (2008) reported no immunotoxic effects on the T cells in rats.

120. Gene mutations are suggested not to occur after exposure to BDE-209 or other PBDEs (Anderson 1990, EFSA 2011, HCA 2012, JETOC 2000, Kirkland 2005, NTP 1986), although recent studies have indicated that BDE-209 may cause DNA damage through the induction of oxidative stress in vitro (Ji 2011, Tseng 2011). There is limited evidence for carcinogenicity of BDE-209 in experimental animals (EFSA 2011, HCA 2012). 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 (EFSA 2011).

2.4.5 Human toxicity

121. A number of studies have assessed the risk of BDE-209 and other PBDEs to humans. The primary focus has been on assessing the risk for developmental neurotoxicity, which is generally considered as the most critical effect in mammals.

122. The observation, as outlined in section 2.3.4, that exposure takes place already during the early phases of human development i.e. in utero via placental transfer and postnatal via mothers milk (e.g. Gómara 2007, Kawashiro 2008, Wu 2010, Miller 2012, Mannetje 2013, Coakley 2013), 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 (Gascon 2012), and that human prenatal or postnatal exposure to BDE-209 delays cognitions and potentially affects neurological development (Chao 2011). Furthermore, several epidemiological studies support that exposure to PBDEs may result in human neurodevelopmental toxicity (Harley 2011, Hoffman 2012, Herbstman 2008, Chevrier 2010, 2011, Gascon 2011, Roze 2009, Eskenazi 2013, Schreiber 2010). Some human studies also observed associations between TH/TSH levels and exposure to BDE-209 or other high congeners such as BDE≥183 (Huang 2014, Zota 2011, Wang 2010c, see UNEP/POPS/POPRC.10/INF5 Table 6.4).

123. A risk characterization and a hazard and dose-response assesment 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 (Health Canada, 2012, US EPA 2008, 2010). 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 (EFSA 2011). 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 (EFSA 2011). 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 (Fowles and Margott 2013). 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 (e.g. Pellacani 2012, Tagliaferri 2010, Llabjani 2010, Karpeta and Gregoraszczuk 2010, Hallgren and Darnerud 2002, He 2009).

2.4.6 Mixture toxicity and combined effects of multiple stressors

124. In the environment, the exposure and response to toxic compounds as well as the likelihood for adverse effects is influenced by a number of factors besides the inherent properties of the compound. Such effects include environmental temperature, salinity and pH, the physiological status of the organisms, toxicokinetic processes, food web or trophic structure, environmental transport, partitioning, transfer mechanisms and deposition (for overview see Letcher 2010, Schiedek 2007, AMAP 2011, POPRC 2013b). Climate change impact on ecosystems may also have an effect on several of these factors and hazardous chemicals can affect the ability of organisms to adapt to climate changes and endure their physical environments (AMAP 2003, POPRC 2013b, UNEP/AMAP 2011, NCP 2013). In addition, wildlife and humans are typically not only exposed to BDE-209 alone but rather to a complex mixture of multiple PBDEs as well as other POPs (de Wit 2006, 2010, Kortenkamp 2014, EFSA 2011, NCP 2013). Thus when considering the likelihood for adverse effects to humans and wildlife all these factors need to be considered and may provide additional reasons for concern.

125. 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 (Tagliaferri 2010). 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 (Karpeta 2010). 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 toxicy of PBDEs. In an in vitro study with binary mixtures of PCBs and PBDEs (BDE-47, -153, -183, or 209), Ljabljani (2010) 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/WHO (2013).

126. Further indications for possible mixture toxicity between PBDEs is provided by Kortenkamp (2014), who evaluated the likelihood and type of combined effects between BDE-209 and other PBDEs to humans and wildlife based on concentration addition using the hazard index approach. On the basis of common modes of action and common adverse outcomes, the study finds that it can be expected that BDE-209 and other PBDEs may produce combined developmental neurotoxicity both in humans and wildlife (Kortenkamp 2014). For humans, the study shows that by taking into account the combined exposure to PBDEs the tolerable combined exposures are exceeded for all age groups, particularly for small children. Though the authors indicate that further research is necessary, the mixture risk assessment nonetheless indicates that combined exposures to BDE-209 and other PBDE likely pose significant health concerns, especially for young children of age 6 months to 3 years which bear the highest PBDE exposures of all age groups. The study also indicates a risk from combined PBDE exposure to wildlife, including Arctic top predators such as the polar bear. Relative to the other PBDEs BDE-209 was found not to make a significant contribution to the overall risk to wildlife. Overall, the study shows that a consideration of BDE-209 in isolation, without taking into account co-exposure to other BDEs, would underestimate the risk. A similar concern was also indicated by Villanger (2011a,b, 2013) who demonstrated that organohalogen contaminant mixtures including several PBDEs (BDE-28, -47,-99,-100 and -153) may influence the thyroid homeostasis in Arctic marine mammals. Though the impact of BDE-209 was not assessed in these studies and only correlations were reported, the study, similar to the findings of Kortenkamp (2014), raises concerns that PBDEs due to a similar mode of action may act in concert and induce adverse toxic effects and thereby pose a threat to Arctic marine top predators and other wildlife.

127. 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 Plourde (2013) observed that 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 ∑PBDE 26.2-680 ng/g ww, plasma BDE-209 0.70-19.1 ng/g ww and ∑PBDE 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 (Sagerup 2009). 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 by Plourde (2013) which 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 (Chen, 2007a) 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 (Ciesielski 2008). However, as these studies report BDE-209 concentration on a ng/g lw basis they are not directly comparable to the findings of Plourde (2013) who reported BDE-209 concentrations in ng/g ww .

128. Additional concerns relate to multiple stressor effects i.e. possible combined effects between toxic chemicals and other factors. Iodine deficiency, a common condition worldwide (reviewed by Walker 2007), can increase the sensitivity to adverse effects from thyroid-disrupting chemicals such as BDE-209 (see Dingemans 2011). Second, exposures to thyroid-disrupting chemicals, including BDE-209 and other PBDEs, may also impair the ability of vertebrates to adequately respond to the climate change impact on their environment (Hooper 2013, POPRC 2013b). Third, climate change and elevated temperatures may increase degradation, and long-range environmental transport of BDE-209 (POPRC 2013b, IPCC 2007, NCP 2013, Xu 2011, Christensen 2014).

3. Synthesis of information

129. C-decaBDE is still produced and used as a flame retardant in many countries. BDE-209, the main component of c-decaBDE, is ubiquitous in the global environment, including biota. Monitoring data provide evidence for environmental emission from a wide range of sources, as well as long-range environmental transport over great distances.

130. High persistency of BDE-209 in soil and sediment is demonstrated in several studies. Reported half-lives in sediment and soil are high and range from >180 days to 50 years, depending on the environmental conditions.

131. Although BDE-209 is persistent in soil, sediment and air, it is known to debrominate to lower brominated PBDEs in the environment and biota. Debromination is indicated as a major concern in a number of assessments as some of the lower brominated PBDEs that are formed are known to be more bioaccumulative, toxic and persistent and to have a greater tendency to undergo long-range environmental transport than the fully brominated BDE-209. Some of the PBDEs formed are also listed POPs. Studies have shown debromination in biota and in environmental matrices. Several PBDE congeners that are not part of c-decaBDE have been identified and are considered to provide evidence for debromination. C-decaBDE and BDE-209 are also a source to highly toxic PBDD/PBDF and hexabromobenzene. PBDD/PBDF are found as impurities in c-decaBDE and may be formed unintentionally during e.g. UV-exposure, industrial-, waste- and recycling operations.

132. Both oceanic and atmospheric processes contribute to the long-range environmental transport of BDE-209, but atmospheric particle transport is believed to be the main mechanism. The estimated atmospheric half-life in air is 94 days, but the life-time can exceed 200 days. In the Arctic and other remote regions BDE-209 is found in various environmental compartments including air, sediment, snow, ice and biota.

133. BDE-209 released to the environment is bioavailable and taken up by organisms, including humans. BDE-209 is widely distributed in the global environment and high body burdens have been demonstrated in some species. Common kestrels in China contained some of the highest BDE-209 levels ever reported in wildlife, while increasing levels have been observed in peregrine falcons from Greenland, North-America and Sweden. Temporal trends indicate that the levels of BDE-209 were increasing in the Arctic atmosphere from 2002-2005 with a doubling time in the range of 3.5-6.2 years. In the period 2009-2013 no temporal trends of BDE-209 in air have been observed.

134. BDE-209 can transfer from mother to off-spring and exposure takes place during early development. Maternal transfer to eggs and offspring has been reported in fish, amphibians, birds and reindeer. In humans, exposure to BDE-209 takes place in the early phases of development in utero via placental transfer and postnatally via mother's milk. In addition infants and toddlers are reported to have higher body burdens of BDE-209 and other PBDEs than adults due to a higher exposure to dust. In humans elevated levels have been reported in electronic dismantler workers, computer technicians, injection workers at electrical application factories and people residing near production and recycling facilities.

135. The equivocacy in the available bioaccumulation data largely reflects species differences in uptake, metabolism and elimination, differences in exposure regimes and analytical challenges in measuring BDE-209. The BCF for BDE-209 in fish has been estimated to be <5000 with non-appreciable aqueous uptake predicted due to its large molecular size and low water solubility (<0.1 μg/L at 24 °C). However, the most important exposure route for BDE-209 in aquatic and terrestrial food webs is through the diet, and when considering the bioaccumulative behaviour of BDE-209, calculated or measured BAFs, BMFs and TMFs are believed to give more relevant information than calculated or measured BCFs. Although some studies do not demonstrate BDE-209 bioaccumulation and trophic dilution has been observed (TMF<1), bioaccumulation has been reported for some aquatic species (BAFs>5000) and a number of aquatic and terrestrial organisms (BMFs>1 and TMF>1).

136. There is evidence that BDE-209 can result in adverse effects to reproductive health and output in fish, earthworm, mouse and rats as well as developmental- and neurotoxic effects in amphibians, rodents and humans. For some organisms such as frogs and birds adverse effects levels reported for BDE-209 are close to or within the range of reported environmental levels. Due to debromination, organisms are moreover co-exposed to a complex mixture of PBDEs, including the already listed POPs BDEs. On the basis of common modes of action and common adverse outcomes, there is a concern that BDE-209 and other PBDEs combined may cause developmental neurotoxicity in both humans and wildlife at environmentally relevant concentrations.

137. There is an increasing concern about endocrine disruptive chemicals since they can cause adverse effects at low environmental levels, display non-monotonic dose-response curves, and the timing of exposure can be more critical than the level of exposure (UNEP/WHO 2013). Available toxicity data shows that BDE-209, similar to other PBDEs, may act as an endocrine disruptor, and interfere with TH homeostasis in fish, amphibians, rat, mice and humans, and possibly steroid hormone homeostasis. This fact, combined with debromination and co-exposure to BDE-209 and other similarly acting PBDEs, some of which are listed POPs as well as the high persistency of BDE-209 in sediments and soils increases the likelihood for chronic long-term adverse effects.

Table 3. POP characteristics of BDE-209, the main component of c-decaBDE

Criterion	Meets the criterion (Yes/No)	Remark
Persistence	Yes	Dated sediment cores indicate no degradation over a period of > 30 years (Kohler 2008). Reported half-life in sediment range between 6 and 50 years, with an average of around 14 years at 22°C and under dark conditions (Tokarz 2008). Degradation half-life in sludge-amended soil under aerobic and anaerobic conditions >360 days (Nyholm 2010, 2011, as cited in ECHA 2012 a). No degradation of decaBDE after 180 days in soil samples spiked with BDE-209 (Liu 2011a) Temporally increasing concentrations is observed in some organisms and support the picture of BDE-209 as a persistent substance (e.g. Vorkamp 2005). Debrominates to lower brominated PBDEs with PBT/vPvB and POP properties that are known to be persistent (ECHA 2012a, POPRC 2013a)
Bio-accumulation	Yes	Found in elevated concentrations in top predators (Shaw 2008, 2009, 2012, Voorspoles 2006a, Jenssen 2007, Sørmo 2006, Verreault 2005). Log K_{ow} ranging between 6.27 to 12.11 (CMABFRIP 1997, Dinn 2012, ECA 2010, Kelly 2007, Tian 2012, US EPA 2010, Watanabe and Tatsukawa 1990). BAF>5000 in aquatic organisms (Mansouri 2012, He 2012). BMFs>1 in aquatic organisms (Baron 2013, Law 2006, Jenssen 2007, Mo 2012, Shaw 2009, Tomy 2009) BMFs >1 in terrestrial organisms (Yu 2011, She 2013, Wu 2009a). TMFs> 1 in aquatic organisms (Law 2006), and in some Arctic food webs (Tomy 2009). Debrominates to lower brominated PBDEs with PBT/vPvB and POP properties that are known to bioaccumulate (ECHA 2012a, POPRC 2013A) Toxic effects are observed at low and/ or environmentally relevant concentrations in birds, fish and frog (ECHA 2012a, Plourde 2013, Kuo 2010, Qin 2010, Wu 2009a, Liu 2011c)
Potential for Long-Range Environmental Transport	Yes	Widespread in the Arctic environment and biota (de Wit 2006, 2010, ECA 2010, NCP 2013). Monitoring data on BDE-209 levels in moss from Norway show that levels decrease from southern to northern Norway (Mariussen 2008) When bound to fine air particles BDE-209 can remain airborne for hours or days, provided it is not removed by wet deposition (Wilford 2008, Meyer 2012). The estimated atmospheric half-life in air is 94 days in air, but the life-time can be > 200 days (Breivik 2006, Raff and Hites 2007). DecaBDE debrominates to lower brominated PBDEs with PBT/vPvB and POP properties that are known to undergo long-range environmental transport (ECHA 2012a, POPRC 2013a)
Adverse effects	Yes	BDE-209 exerts reproductive, developmental, endocrine and neurotoxic effects in aquatic organisms, mammals and birds. Effects on growth, survival and mortality are also reported. Key data include: ▪ Delayed metamorphosis in tadpoles with a tentative NOEC below 1 µg/L (Qin 2010 in ECHA 2012a). ▪ TH disruptive effects and mortality in fathead minnow chronic 28 d-LOEL of ~3 ng/g BDE-209 bw/day or 0.41 ng/g ww food (Noyes 2013). ▪ Mortality of up to 98% in chicken embryos after 20 days following a single egg injection with BDE-209 (LD_{50} of 44 µg/ egg or 740 µg/ kg ww, Sifleet 2009 in ECHA 2012a). ▪ Developmental neurotoxicity in rodents (e.g. Johansson 2008, Viberg 2003, 2007, Rice 2009, Fujimoto 2011, Heredia 2012, Reverte 2013, 2014). Epidemiological evidence for cognitive developmental effects in humans (Gascon 2012, Chao 2011). ▪ Several studies suggest that adverse effects may occur at BDE-209 concentations at or close to environmentally relevant levels in fish, frogs, birds and soil organisms (Kuo 2010, Wu 2009a, Liu 2011c, Qin 2010, Noyes 2013, Zhu 2010, Liu 2011a, Zhang 2012, 2013, Sifleet 2009, ECHA 2012a). ▪ Debromination to lower brominated PBDEs with PBT/vPvB and POP properties that are known to have significant adverse effects (ECHA 2012a, POPRC 2013a, POPRC6 and POPRC7). ▪ Risk to humans and wildlife resulting from combined effects between BDE-209 and other PBDEs at environmentally relevant concentrations (e.g. Kortenkamp 2014, Plourde 2013). ▪ Potential risk for low-dose effects during sensitive developmental periods as a result of endocrine disruption (UNEP/WHO 2012).

4. Concluding statement

138. C-decaBDE is a synthetic substance with no known natural occurrence that is used as a flame retardant in many applications worldwide. Releases of c-decaBDE to the environment are continuing in all regions investigated. BDE-209 (or decaBDE), the main constituent of c-decaBDE is persistent in the environment and bioaccumulates and biomagnifies in fish, birds and mammals. There is evidence for adverse effects to critical endpoints including reproduction, survival, nerve- and endocrine systems. C decaBDE is also degradaded 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. C-decaBDE with its main constituent BDE-209 is likely, therefore, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted.

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