Stockholm Convention on Persistent Organic Pollutants

Persistent Organic Pollutants Review Committee
Ninth meeting
Rome, 14–18 October 2013
Item 7 (a) of the provisional agenda[*]
Consideration of chemicals newly proposed for inclusion in Annexes A, B and/or C to the Convention: decabromodiphenyl ether (commercial mixture, c-decaBDE)

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

Note by the Secretariat

1.   The annex to the present note contains a proposal submitted by Norway to list decabromodiphenyl ether (commercial mixture, c-decaBDE) in Annexes A, B and/or C to the Convention pursuant to paragraph 1 of Article 8 of the Convention. The proposal is being circulated as submitted and has not been formally edited. The Secretariat's verification of whether the proposal contains the information specified in Annex D is discussed in document UNEP/POPS/POPRC.9/INF/8.

Possible action by the Committee

2.   The Committee may wish:

(a)   To consider the information provided in the present note;

(b)   To decide whether it is satisfied that the proposal fulfils the requirements of Article 8 of and Annex D to the Convention;

(c)   To develop and agree on, if it decides that the proposal fulfils the requirements referred to in paragraph 2 (b) above, a workplan for preparing a draft risk profile pursuant to paragraph 6 of Article 8.

---

[*] UNEP/POPS/POPRC.9/1.

Annex

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

1. Introduction

1.   Commercial decabromodiphenyl ether (c-decaBDE) is widely used as an additive flame retardant in textiles and plastics. It is a synthetic mixture of polybrominated diphenyl ethers, with the main component being the fully brominated congener decaBDE.
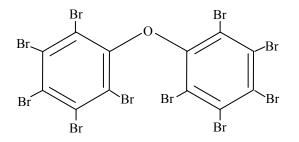
2.   The nomination report specifically addresses the information requirements and screening criteria of paragraph 1 and 2 in Annex D in the Stockholm Convention on Persistent Organic Pollutants and summarizes relevant evidence relating to the screening criteria for persistence, bioaccumulation, adverse effects and long-range transport. Some additional information relating to paragraph 3 of Annex D is also provided. The report is based on existing risk assessments in the EU and North America. In addition more recent literature from peer-reviewed scientific journals is included.

2. Chemical identity

3.   The nomination concerns commercial decabromdiphenyl ether; c-decaBDE. The commercial mixture consists primarily of the fully brominated decaBDE congener in a concentration range of 77.4-98 %, and smaller amounts of the congeners of nonaBDE (0.3-21.8 %) and octaBDE (0-0.04 %) (ECHA 2012 a, US EPA 2008). The range reflects differences in the composition of c-decaBDE mixtures available (ECHA 2012 a, UK Environment Agency 2009, US EPA 2008). Trace amounts of other compounds, thought to be hydroxybrominated diphenyl compounds can also be present as impurities, whereas total tri-, tetra-, penta-, hexa- and heptaBDEs are typically present at concentrations below 0.0039 % w/w (EC 2002, ECHA 2012 a). In this document the term decaBDE alone refers to the single fully brominated PBDE, also denoted as BDE-209.

4.   The chemical data on decaBDE are presented in Figure 1 and in Tables 1 and 2 below (ECHA 2012 a).

Figure 1. Structural formula

Table 1. Chemical identity

CAS number:	1163-19-5
CAS name:	Benzene, 1,1'-oxybis[2,3,4,5,6-pentabromo-]
IUPAC name:	1,1'-Oxybis(pentabromobenzene)
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[1]; DBBE; DBBO; DBDPO
Trade names	commercial decaBDE mixture, technical decaBDE, technical DeBDE, BDE-209, DE-83R, Bromkal 82-ODE, Bromkal 70-5, Saytex 102 E

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

Table 2. Overview of relevant physicochemical properties

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

3. Global consumption and use

5. DecaBDE is a general purpose additive flame retardant which is compatible with a wide variety of plastics/polymers and textiles. Flame retardants inhibit the ignition of materials and slow the rate at which flames spread. Additive flame retardants are physically combined with the material whereas reactive flame retardants are chemically combined. The versatility of decaBDE has resulted in a range of end uses, leading to a complex life cycle. In plastics, c-decaBDE is used for electrical and electronic equipment in housings of computers and TV sets, in the transportation and aeronautic sectors and in construction and building, such as wires and cables, pipes and carpets (BSEF 2013). In textile applications, decaBDE is used in contract textiles, mainly for public buildings and transport and in domestic furniture textiles in countries with stringent fire safety regulations (BSEF 2013).

6. According to the global demand reported by the industry c-decaBDE was the second largest brominated flame retardant and the major PBDE mixture on the market in 2001 (BSEF 2006 as cited in AMAP 2009). The most recent data from member companies of the European Flame Retardants Association (EFRA) indicates that in 2010, 7,500 – 10,000 tons of commercial decaBDE were sold in the EU (VECAP 2011). The figures do not include decaBDE imported in preparations or articles. The production/import volume in US of decaBDE is reported to be in the range of 50-100 million lbs (25,000 – 50,000 tons) in 2002 and in 2006 (US EPA Inventory Update Reporting as cited in Klosterhaus et al. 2012). Among the Asian countries, c-decaBDE is produced mainly in China, where its production was up to 13,500 tons per annum in 2001 and up to 30,000 tons in 2005 (Xia et al. 2005, Zou et al. 2007). According to a market research report by Freedonia in 2010 on the world flame retardants industry the world demand for flame retardant additives is forecast to rise 6.1 percent per

year to 2.2. million metric tons in 2014. This is partly due to more stringent safety requirements and more flammable materials. The Asia/Pasific region will continue to be the largest and fastest-growing market for flame retardants through 2014, accounting for nearly half of the world demand (Freedonia 2010).

4. National and international administrative actions on decaBDE

7. DecaBDE has been under scrutiny for its potential health and environmental impacts for a longer time, both within the scientific community and among regulators. At present several environmental and health risk/hazard assessments of decaBDE have been independently conducted on the international, regional and national levels. This has resulted in risk reduction measures in several countries.

8. Already in 1986 the German Chemicals Industry Association (VCI) voluntarily agreed to discontinue the use of decaBDE because of concerns about the potential for brominated dioxins/furans emissions from incomplete combustion (ECHA 2012 c). Later in 2008 the EU launched the ban on the use of decaBDE in electrical and electronic equipment through the Restriction of Hazardous Substances in Electrical and Electronic Equipment Directive (RoHS). Recently (December 2012) decaBDE was identified as a PBT substance (persistent, bioaccumulative and toxic) and a vPvB substance (very persistent, very bioaccumulative) and was adopted to the Candidate List of substances of very high concern (SVHCs) under the Registration, Evaluation, Authorisation and Restriction of Chemicals Regulation (REACH) in EU (ECHA 2012 b). According to OECDs compilation of risk and hazard information for PBDEs, Switzerland has harmonized their regulations on the use of decaBDE in electrical and electronic equipment with the EU RoHS directive (OECD 2008). In 2008 a national ban came into force in Norway on production, import, export, use and the placing on the market of decaBDE. The ban includes all uses except in transportation.

9. In North America the first restriction was adopted in Canada in 2008 with a ban on manufacture of PBDEs, including decaBDE, under the The Polybrominated Diphenyl Ethers Regulations (Environment Canada 2008). In August 2010, Environment Canada and Health Canada published a Final Revised Risk Management Strategy for PBDEs which reiterated the objective of reducing the concentration of PBDEs in the Canadian environment to the lowest level possible. This resulted in agreement with three large worldwide producers of decaBDE to voluntarily phase-out the export of decaBDE to Canada. The voluntary agreement included a phase-out of DecaBDE exports and sales for electrical and electronic equipment by the end of 2010, for transportation and military uses by the end of 2013 and for all other uses by the end of 2012 (Environment Canada 2010 b). Canadian authorities recently announced that they are consulting on plans to extend the current manufacturing restriction on polybrominated diphenyl ethers (PBDEs) to ban the use, sale and import of all PBDEs in the country. According to the announcement, Environment Canada is also considering a ban on PDBE use in products (Environment Canada 2010 b). Final regulatory controls are targeted for adoption in 2013 (BSEF 2013). From 2009, as the result of negotiations with US Environmental Protection Agency (EPA), three large US producers of decabromodiphenyl ether (decaBDE) announced commitments to phase out decaBDE. The companies have committed to end production, importation, and sales of decaBDE for most uses in the United States by December 31, 2012, and to end all uses by the end of 2013 (US EPA 2013). In addition limited bans have been adopted by four states in US, Washington, Oregon, Vermont and Maine (BSEF 2013).

10. In Asia limited restrictions have been adopted in China, India and Korea. In the revision of the Chinese RoHS legislation (Administrative Measure on the Control of Pollution Caused by Electronic Information Products) a restriction on the use of DecaBDE for printers, mobile and fixed phones was adopted (BSEF 2013). Korea implemented a law in 2008 which covers end-of-life and restrictions on electronic products and vehicles. Exemptions, limit values and restricted substances are the same as the EU RoHS Directive. But DecaBDE is exempted from the list of hazardous substances in polymeric applications under the Recycling of Resource in Electronic Equipment and Automobiles' Regulation (BSEF 2013).

11. In India, the e-waste (management and handling) Rules came into effect in May 2012. The chapter on the Restriction of hazardous substances under the e-waste rules restricts the use of PBDEs in electric equipment (BSEF 2013).

5. Information on decaBDE in relation to the POP screening criteria

5.1 Persistence

12. The high persistency of decaBDE in soil and sediment is demonstrated in several studies and appears to be dependent on slow biodegradation processes and reliance on photodegradation in those matrixes (ECHA 2012 a; and references therein, Environment Canada 2010 a; and references therein). Furthermore, hydrolysis is unlikely to be a relevant degradation process in the environment, explained by the very low water solubility of decaBDE, < 0.1 ug/l at 25 °C, and that the molecule does not contain any functional groups that are readily susceptible to hydrolysis (ECHA 2012 a).

13. The extrapolated primary degradation half-life in sludge-amended soil under both aerobic and anaerobic conditions was found to be >360 days assuming exponential decay (Nyholm et al. 2010 and Nyholm et al. 2011, as cited in ECHA 2012 a). Liu et al. (2011 a) concluded no degradation of decaBDE after 180 days in soil samples spiked with decaBDE. Using bacterial cultures for testing, He et al. (2006) found no decaBDE degradation in soil for up to one year with three of the four bacterial strains tested. However, after incubation with the fourth type of bacteria, decaBDE was no longer detectable in the culture after 2 months incubation. The study shows that decaBDE biodegradation in soil and other environmental compartments can be influenced by the types of bacteria present.

14. Depending on the experimental conditions used for testing, environmental half-lives of decaBDE in sediment are reported to range from hours to months and even years in the literature (ECHA 2012 a; and references therein). Two studies on freshwater sediments show that decaBDE has the potential to photodegrade relatively quickly in the aquatic environment (Tysklind et al. 2001, Søderstrøm 2003). Reported degradation half-lives in these studies were 100 hours and 53 hours, respectively (Tysklind et al. 2001, Søderstrøm 2003). But under other conditions, in deep sea sediments, where light attenuation and matrix shielding would affect overall exposure to sunlight and potential for photodegradation, the persistency of decaBDE appears to be high. The longest half-life in sediment is reported by Tokarz et al. (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 decaBDE to range between 6 and 50 years, with an average of around 14 years.

15. Noteably, according to the recent Canadian and European assessments of decaBDE it can be concluded that there is a high probability that decaBDE is debrominated in the environment to substances which themselves have persistent organic pollutants (POP) properties, or act as precursors to such substances (Environment Canada 2010 a, ECHA 2012 a and b). Degradation under environmentally realistic conditions in sediments and in aerobic soil in the presence of plants have been shown to lead to the formation of octaBDEs, nonaBDEs and listed POPs, such as tetra- to heptaBDE congeners (ECHA 2012 a; and references therein). In the EU assessment it was concluded that there is a high probability that decaBDE is debrominated in soil and sediments to such substance, in individual amounts greater than 0.1% over timescales of a year (ECHA 2012 a and b). The high persistence combined with wide distribution in the environment creates a high potential for lifetime exposure and uptake in organisms, and a pool of the substance in many localities that will act as a long-term source of degradation products through both abiotic and biotic transformation (ECHA 2012 a).

16. Debromination within biota is concluded to be an important additional pathway for the formation of lower brominated congeners in the EU and Canadian assessments (ECHA 2012 a and b, Environment Canada 2010 a). Existing evidence from field and laboratory studies show that debromination of decaBDE occurs in fish, birds and mammals (Environment Canada 2010 a; and references therein, ECHA 2012 a; and references therein). Fish can take up decaBDE in their diet, and transform it into at least hexa- and heptaBDEs. The yield of the hepta- and hexaBDE metabolites is generally low (typically below 5% of the absorbed decaBDE dose in the various studies) and the actual amounts are small, but the formation of precursors (octa- and nona-BDEs) is more extensive and these could provide an ongoing source over longer time periods to lower brominated PBDEs (ECHA 2012 a and b). *In vitro* studies suggest a similar metabolite profile in bears, whales and seals (ECHA 2012 a). Birds might be able to debrominate decaBDE to at least octaBDEs (ECHA 2012 a).

Conclusion on persistence according to the Criteria of Annex D

17. DecaBDE fulfills the criteria for persistence with half-lives in soil and sediment greater than six months (180 days).

5.2 Bioaccumulation

18.   DecaBDE has been detected in the tissues of a large number of species – including zooplankton, fish, aquatic and terrestrial invertebrates, birds and mammals worldwide (ECHA 2012 a, Environment Canada 2010 a, Daso et al. 2013, Koenig et al. 2013, Chen and Hale 2010, de Wit et al. 2010, Law et al. 2008). But it has been difficult to draw firm conclusions from the scientific information on bioaccumulation potential of decaBDE that has been considered in previous assessments by ECHA and Environment Canada (ECHA 2012 a, Environment Canada 2010 a). Nevertheless, more recent data and data not considered in the previous assessments shows a high potential for decaBDE to bioaccumulate (Wu et al. 2008, He et al. 2012, La Guardia et al. 2012, Jenssen et al. 2007, Mo et al. 2012, Yu et al. 2011, Wu et al. 2009 a, Yu et al. 2012, Kelly et al. 2007, Yi et al. 2013).

19.   The log octanol-water partition coefficient (log Kow) for decaBDE reported in the literature are high and ranges from 6.27 to 12.11 (Dinn et al. 2012, Tian et al. 2012, US EPA 2011, Kelly et al. 2007, Environment Canada 2006). The bioconcentration factor (BCF) has been calculated to be <3000 (CITI 1992). But due to the low water solubility (<0.1 µg/l at 25°C) this value was considered to be uncertain in the assessments undertaken by ECHA and Canada (EC 2002, EC 2007, ECHA 2012 a, Environment Canada 2010a). Even so BCF in fish for decaBDE was considered to be less than 5000 (ECHA 2012 a, Environment Canada 2010 a). This was considered to be underpinned by the fact that the maximum BCF value has been estimated to be equal to 2000 L/kg for substances with a log Kow higher than 9.3.

20.   The BCF value represents uptake of chemical molecules from water to organisms by passive diffusion through cell membranes. This exposure route, is believed to be less important for decaBDE (Booij et al. 2002, Shaw et al. 2009, Kelly et al. 2007), due to its large molecular size (959.2 g/mole). The most important exposure route for decaBDE in aquatic food webs is through diet (Booij et al. 2002, Shaw et al. 2009, Kelly et al. 2007). The accumulated levels of decaBDE 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 digestion of particles. Passive diffusion was considered to contribute to only a smaller fraction of the detected levels (La Guardia et al. 2012, Wang et al. 2009, Shaw et al. 2009). Only a small fraction of the molecules of DecaBDE dissolve in water and the larger fraction of the molecules are strongly associated with particles, (Booij et al. 2002; Shaw et al. 2009; Dinn et al. 2012). Therefore, when considering the bioaccumulative behaviour of decaBDE calculated or measured bioaccumulation factors (BAFs), biomagnification factors (BMFs) and trophic magnification factors (TMFs) are believed to give more relevant information than calculated or measured BCFs (Booij et al. 2002, Shaw et al. 2009, Kelly et al. 2007, Powell et al. 2013).

21.   The lipid normalised values for logBAF for fish reported in the literature range between 4.06-6.7 (Wu et al. 2008 as cited in Mansouri et al. 2012, He et al. 2012, La Guardia et al. 2012). A logBAF>3.7 corresponds to BAF>5000 (He et al. 2012, Environment Canada 2010 a). In the evaluation done by Canada the relationship between logBAF and BAF is explained more thoroughly (Environment Canada 2010 a). These data have not been considered for decaBDE in the previous assessments (Environment Canada 2010 a; ECHA 2012 a). The previous assessments underlined that the levels of decaBDE usually found in the biota is one magnitude lower than for other polybrominated diphenyl ethers, suggesting a low bioaccumulation potential for decaBDE. Important to note is however that the logBAFs reported for decaBDE in the literature fulfil the criteria on bioaccumulation in fish in Annex D of the Convention (BAF>5000), even though some of them were lower than for other brominated diphenyl ethers measured in the same species (Wu et al. 2008 as cited in Mansouri et al. 2012, He et al. 2012, La Guardia et al. 2012).

22.   The measured BMFs and TMFs from field data shows that decaBDE can biomagnify in several aquatic and terrestrial organisms and food webs (BMFs >1and TMF>1). The BMFs reported in the scientific literature range between 1.4-7 in terrestrial organisms and food webs (Yu et al. 2011, Wu et al. 2009 a) and between 0.02-34 in aquatic organisms and food webs (Jenssen et al. 2007, Mo et al. 2012, Environment Canada 2010 a; and references therein). In aquatic food webs TMF values range between 0.2 – 10.4 (Wu et al. 2009b, Yu et al. 2012, Wu et al. 2008a, Jenssen et al. 2007, Environment Canada 2010 a; and references therein). Most of the above reported BMFs and TMFs for decaBDE have all been calculated using muscle (fish, mammals, birds), whole body (bivalves, zooplankton, fish) or adipose samples (fish and mammals). The differences between the BMFs and TMFs reported may depend on interspecies differences, diet, exposure and gender, length and type of food chain etc. They will also differ depending on if they are measured for whole body or in different organs and if they are lipid-normalised or not.

23.   Furthermore, some data available suggests a higher potential for biomagnification of decaBDE in terrestrial than in aquatic organisms (Christensen et al. 2005, Chen and Hale 2010, Jaspers et al. 2006, Voorspoels et al. 2006 a). But Kelly et al. (2007) have studied the accumulation behaviour of different chemicals in a piscivourous, terrestrial and marine mammal food web in more detail by using modelling and field observations. They found that the biomagnification of decaBDE occurred in all three food webs investigated. The results showed a higher BMF for terrestrial carnivores and humans (BMF=8) than for marine mammals (BMF=3). The lowest BMF was found in terrestrial herbivores and water-respiring organisms (BMF=1). More specifically the findings by Kelly et al. 2007 indicated that substances like decaBDE, with a high log Kow of 9.9 (>5), are absorbed at very slow rates by aquatic organisms (Mörck et al. 2003, Stapleton et al. 2004, Kierkegaard et al. 1999). While they in air-breathing animals may be accumulated to a larger degree due to slow respiratory elimination and slow elimination in urine and nitrogenous wastes explained by a high log octanol-air partition coefficient (log Koa) of 13.1 (>6).

24.   In addition almost all of the studies with no findings of biomagnification potential of decaBDE in fish and mammals have been based on detected levels of decaBDE in muscles or adipose tissue and/or have been lipid normalised. Those studies may have underestimated the biomagnification potential of decaBDE, since decaBDE appears to associate more with blood-rich tissues, and is predominately found in liver, to some extent in blood and to a lesser extent in muscles and adipose tissue (Wu et al. 2009 a, Voorspoels et al. 2006 b, Wang et al. 2011 b, Yi et al. 2013).

25.   The potential of biotransformation of decaBDE to more toxic and bioaccumulative metabolites was considered to be of high concern in the assessments of decaBDE in Canada and EU and a valid reason for restrictions of the use and production of c-decaBDE. The debromination of decaBDE to already listed POPs, hexa- and heptaBDE, in the biota were considered to be confirmed by the large range of field studies and laboratory studies investigating biotransformation in the wild and in controlled laboratory settings (Environment Canada 2010 a, ECHA 2012 a and b). Recent scientific findings add to the concern regarding the bioaccumulation potential of decaBDE and debromination in organisms in the environment, since they show that the accumulation of decaBDE can lead to adverse effects in vulnerable life stages of mammals, fish and amphibians (Chen et al. 2012, He et al. 2011, Noyes et al. 2011). The levels used in the experiments were comparable to levels in more polluted areas (Zhang et al 2011 a; Wang et al. 2011). The reported debromination products were lower brominated diphenyl ethers, such as nona- and octaBDE (Chen et al. 2012, He et al. 2011) and hepta- and hexaBDE (Noyes et al. 2011, He et al. 2011). The study by He et al. (2011) showed that long-term chronic exposure to low doses of decaBDE can have adverse effects on reproductive performance in fish and the behaviour in their offspring. Moreover, accumulation of decaBDE and transfer to offspring has been demonstrated in studies of the accumulation of decaBDE in amphibians (Wu et al. 2009 a) and mammals (Zhang et al. 2011 c). The findings by Wu et al. (2009 a) showed a relatively high biomagnification (BMF=7) of decaBDE from insects to female frogs followed by a mother-to-egg transfer.

Conclusion on bioaccumulation according to the criteria in Annex D

26.   Based on the weight of evidence decaBDE fulfils the criteria on bioaccumulation under Annex D of the Convention.

5.3   Potential for long-range environmental transport

27.   As a testament of its potential for long-range environmental transport, decaBDE is found in various environmental compartments in the Arctic including air, sediment, snow, ice and biota (Meyer et al. 2012, Hermanson et al. 2010, Su et al. 2007, Knudsen et al. 2006, Bakke et al. 2008, Tomy et al. 2008, Tomy et al. 2009, Breivik et al. 2006, de Wit et al. 2006 and 2010, Hung et al. 2010, Letcher et al. 2010, AMAP 2009, Environment Canada 2010 a). Several studies have reported that decaBDE is the predominant or one of the dominating PBDEs in Arctic air (Wang et al. 2005, Möller et al. 2011, Meyer et al. 2012, Hermanson et al. 2010, Hung et al. 2010, Su et al. 2007). DecaBDE deposited to the Arctic environment is bioavailable to the organisms living there and fount to be widespread in the Arctic food webs (de Wit et al. 2006 and 2010, Environment Canada 2010 a). Biological samples from the Arctic contaminated by decaBDE include a variety of species spanning different trophic levels of terrestrial and aquatic food chains (de Wit et al. 2010). Arctic samples contaminated with decaBDE include vegetation, birds of prey, seabirds and seabird eggs, marine and freshwater fish, different amphipods, zooplankton, shrimps and clams, terrestrial and marine mammals (de Wit et al. 2006 and 2010, Tomy et al. 2008). Biomonitoring of Arctic biota have demonstrated increasing decaBDE levels in some Arctic species such as the peregrine falcon. The increasing levels were monitored in eggs from southwestern Greenland collected from 1986 to 2003 (Vorkamp et al. 2005). Biomonitoring data have also shown that decaBDE can contribute significantly to the total

body burden of PBDEs in Arctic species. DecaBDE has for example been reported to account for > 50% of total BDE burden in the detritus feeding ice-amphipod *Gamarus wilkitzkii* (Sørmo et al. 2006) and 60% in redfish and 75% in arctic cod (Tomy et al. 2008). DecaBDE is also the predominant congener in moss samples, and when present also in moose samples (Mariussen et al. 2008).

28. DecaBDE 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 5.105 molecule/cm^3 decaBDE (EC 2002). The monitored levels of decaBDE in the Arctic atmosphere together with studies showing a significant deposition on Arctic ice (Hermanson et al. 2010) and snow (Meyer et al. 2012) further underlines the potential of decaBDE to undergo long-range transport to remote regions. For example, in a study assessing a total of 19 different brominated flame retardants in ice cores samples from the Norwegian Arctic, decaBDE was found to provide the second greatest share of the deposition of brominated flame retardants from air to the Arctic ice (Hermanson et al. 2010). The deposition rate for decaBDE was found to be 320 pg/ cm/ year in the period 1995-2005 (Hermanson et al. 2010). The deposition of decaBDE was surpassed only by HBCD, and was substantially higher than for other PBDEs (Hermanson et al. 2010). Adding to this, temporal trend data show that the levels of decaBDE in the Arctic atmosphere are increasing with a doubling time in the range of 3.5-6.2 years (Su et al. 2007, Hung et al. 2010).

29. Both oceanic and atmospheric processes contribute to the environmental transport of decaBDE (Su et al. 2007, Möller et al. 2011, Breivik et al. 2006), but atmospheric particle transport is believed to be the main mechanism behind the long-range environmental transport of decaBDE. In the atmosphere, decaBDE is predicted to sorb almost entirely to atmospheric particles. Its atmospheric long-range transport therefore appears to be controlled by the atmospheric mobility of the particles to which it is attached (Breivik et al. 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 et al. 2008, Meyer et al. 2012). The deposition of air borne particles is found to be higher during the Arctic haze season (Su et al. 2007, AMAP 2009). Furthermore it is found that the particles can shield the decaBDE molecule from photolysis and lengthens its life-time in the air to > 200 days (Breivik et al. 2006, Raff and Hites 2007 as cited in de Wit et al. 2010).

Conclusion on long-range transport according to the criteria in Annex D

30. The estimated half-life in air for decaBDE and its wide occurrence in the Arctic fulfill the criteria for long-range transport in Annex D to the Stockholm Convention.

5.4   Adverse effects

31. National- and regional assessments conducted independently by the EU, the United Kingdom, Canada and US have evaluated the potential for decaBDE to induce adverse effects in wild organisms and humans (e.g. EC 2002, EC 2004, EC 2008, UK Environment Agency 2009, ECHA 2012 a, Health Canada 2006, Health Canada 2012, US EPA 2008, EFSA 2011). The toxicity of decaBDE has also been the topic of several scientific papers and reviews (see e.g. Dingemans et al. 2011, Chen and Hale 2010, Costa and Giordano 2011). In the available literature adverse effects are reported for soil organisms, birds, fish, frog, rat, mice and humans. The effects range from changes at biochemical- and cellular level to effects which may have implications at higher-levels of biological organization including survival, growth and reproduction. In both wild organisms and humans early developmental stages appears more vulnerable to decaBDE exposure than adults. In vertebrates, the liver, the thyroid hormone axis and the nerve system appears to be the main targets for decaBDE toxicity.

32. Although adverse effects have been reported, shortage of data makes it difficult to draw firm conclusions regarding the toxicity of decaBDE to soil organisms and plants (Xie et al. 2013, Xie et al. 2011, Zhu et al. 2010, Liu et al. 2011 b, Zhang et al. 2012, for overview see ECHA 2012 a). The available scientific literature provides no evidence of adverse effects to plants, but demonstrates that debromination products are detected in plants and soil following decaBDE exposure (ECHA 2012 a). A recently published study suggest that earthworm embryos or juveniles may be susceptible to adverse effects and that exposure during early developmental phases can lower the number of juveniles hatched per cocoon (Xie et al. 2013). Other scientific studies have shown that decaBDE both alone and in combination with copper, another typical contaminant of soil linked to releases from recycling plants processing e-waste, can affect enzymatic activity in soil and alter the bacterial community structure by reducing species richness (Zhu et al. 2010, Liu et al. 2011 b, Zhang et al. 2012). The study was conducted with decaBDE levels that are within the range of reported levels in contaminated soil.

33. Data on avian toxicity are scarce, but raises concerns for possible adverse effects (see reviews by Chen and Hale 2010 and ECHA 2012 a). More specifically, only one study to date has assessed the

toxicity of decaBDE in birds (Sifleet 2009). This study found a mortality of up to 98 % in chicken eggs injected with decaBDE. Birds are however also reported to metabolize decaBDE to lower brominated PBDEs, including the POP-BDEs, hexa-, hepta-, tetra- and pentaBDEs (Van den Steen al. 2007, ECHA 2012 a; and references therein). Exposure to lower brominated PBDEs in birds have been associated with immunomodulatory changes, developmental toxicity, altered reproductive behavior, reduced fertility and reproductive success (Chen and Hale 2010, POPRC 2007). According to the assessment undertaken by the EU (ECHA 2012 a), the decaBDE concentrations typically found in bird eggs in the wild are only around 2-10 times lower than the concentrations that according to Sifleet (2009) was observed to induce significant mortality (ECHA 2012 a). The reported concentrations in bird eggs are typically in the range of 1-100 μg/kg ww, but concentrations up to about 420 μg/kg ww have been reported. Hence the margin between exposure levels in wild birds and observed effect levels is not high, especially considering that the study by Sifleet (2009) does not take account of potential sub-lethal effects, and that the author noted that additional decaBDE would likely have been assimilated following hatching and resorption of the remaining yolk. With the exception of the study by Sifleet et al. (2009) and one controlled debromination study by Van den Steen et al. (2007) most other available data are from wild birds who, according to Chen and Hale (2010) exhibit some of the highest concentrations of decaBDE ever reported in wildlife. Terrestrial feeding birds appear to exhibit elevated decaBDE levels in comparison to birds preying on fish, and may hence be at greater risk of experiencing adverse effects (Chen and Hale 2010).

34.   DecaBDE has limited solubility in water and early hazard assessments of decaBDE suggested that it was not likely that significant acute or chronic toxic effects would occur in aquatic organisms at concentrations up to the limit of water solubility (e.g. EC 2002, EC 2004, EC 2007, UK Environment Agency 2009). However, as concluded in the most recent EU assessment new studies documenting adverse effects on important biological endpoints including reproduction, development, nervous system, endocrine system, growth and fitness, raise concerns for adverse effects also to aquatic organisms (ECHA 2012 a). More specifically, controlled feeding studies with fathead minnows conducted at environmentally relevant concentrations have shown that decaBDE may interfere with thyroid hormone system in juvenile fish (Noyes and Stapleton 2010, Noyes et al. 2011). This is supported by findings by Chen et al. (2012). Chen et al. (2012) moreover observed significant decreases in body weight and survival rate of zebrafish larvae exposed to decaBDE. Based on measurements of otolith increment widths there are also indications that decaBDE may affect growth rates in fish at environmentally relevant range of decaBDE exposure levels found in sediment (Kuo et al. 2010b). He et al. (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 reported effects were trans-generational i.e. they were observed in offspring of exposed parents and are according to the authors likely explained by maternal transfer to off-spring. In males, indicators sperm quality was significantly affected even at the lowest exposure dose (0.001 μM or 0.96 μg/ l). In all the above studies decaBDE was reported to debrominate to lower brominated PBDEs, thus it is also possible that other PBDE congeners besides decaBDE contributed to the effects reported in these studies.

35.   Via their influence on thyroid hormone system, PBDEs including nona- and decaBDE, have been shown to have the potential to affect development and metamorphosis in amphibians (Schriks et al. 2006 and 2007, Balch et al. 2006, Qin et al. 2010). According to the available studies nonaBDE, which is one of the main congeners present in c-decaBDE besides decaBDE can delay metamorphosis in *Xenopus laevis* tadpoles as evidenced by a significantly reduced tail tip regression following BDE-206 exposure *in vitro* (Schriks et al. 2006). In a more recent *in vivo* study a commercial decaBDE mixture consisting of 98.5% w/w decaBDE was reported to affect metamorphosis in *Xenopus laevis* tadpoles by delaying the time to forelimb emergency (Qin et al. 2010, as cited in ECHA 2012 a). The delay forelimb emergency was accompanied by histological changes in the thyroid gland and reduced expression of the thyroid receptor in tail tissue.

36.   Adverse effects of decaBDE to mammals have largely been investigated in controlled laboratory studies with captive organisms, mainly rodents, and have been the topic of several scientific reviews and government assessments (e.g. EC 2002, EC 2008, US EPA 2008, Costa and Giordano 2011, Dingemans et al. 2011, Health Canada 2006, Health Canada 2012, EFSA 2011). Data on adverse effects resulting from decaBDE exposure in mammals is also underpinned by *in vitro* studies, which provides evidence that decaBDE induces similar cellular effects as other PBDEs (Health Canada 2012). Amongst others a potential to elicit neurotoxic effects, act as an endocrine disruptor of steroid and thyroid hormone regulated processes, promote cancer, induce DNA damage, and affect metabolism has been identified *in vitro* (Ibhazehiebo et al. 2011, Li et al. 2012, Pellacani et al. 2012, Dingemans et al. 2011, Pacyniak et al. 2007, Karpeta and Gregoraszczuk 2010).

37.   Apparently, the fetal/ neonatal nervous system, the liver and the thyroid hormone axis are the primary targets for decaBDE toxicity in rodents (Costa and Giordano 2011, Dingemans et al. 2011, Health Canada 2012). Although decaBDE appears to have low acute toxicity when given by the oral, inhalation and dermal route, available mammalian toxicology data indicates that long-term exposure could result in adverse effects similar to those observed for other PBDE congeners (see e.g. Costa and Giordano 2011, Dingemans et al. 2011, Health Canada 2010). Rodent studies have for example demonstrated that decaBDE may act as an endocrine disruptor of the thyroid hormone system (see e.g. Dingemans et al.2011, Costa and Giordano 2011 for review). In rodents, decaBDE exposure can also result in decreased immune function during pregnancy and lactation (Zhou et al. 2006, Liu et al. 2012), can compromise the organisms ability to cope with infections (Watanabe et al. 2008, Watanabe et al. 2010) and negatively affect reproductive parameters such as the number of follicle- and sperm cells (Liu et al. 2012, Miyaso et al. 2012, Tseng et al. 2006).

38.   The thyroid disrupting potential of decaBDE in mammals have been the subject of several reviews/ assessments (e.g. EFSA 2011, Dingemans et al. 2011, Costa and Giordano 2011), and is considered to be of concern given that decaBDE through its interaction with the thyroid hormone system can act as neurotoxicant of the developing brain (see reviews by Dingemans et al. 2011, Costa and Giordano 2011). DecaBDE may also exert a number of direct effects on brain cells that can compromise brain function and integrity (Costa and Giordano 2011, Dingemans et al. 2011). Animal studies investigating the developmental neurotoxicity of decaBDE have reported a broad spectrum of effects ranging from no observed effects to alterations in e.g. spontaneous and cognitive behaviors, learning, memory, locomotor activity, rearing activity, reflexes and habituation following decaBDE exposure (see US EPA 2008, Dingemans et al. 2011, Costa and Giordano 2011, Health Canada 2012; and references therein). In rodents neurobehavioral effects during juvenile development or adulthood have been observed after a brief postnatal exposure to decaBDE (Johansson et al. 2008, Viberg et al. 2007, Viberg et al. 2003, Rice et al. 2007). In mice, aging appears to unmask behavioural effects not evident at a younger age (see e.g. Rice et al. 2009 and reviews by Health Canada 2012 and Costa and Giordano 2011 and references therein). Although the notion that decaBDE may be a developmental neurotoxicant has been contested (e.g. Hardy et al. 2009, Goodman 2009, Williams and de Sesso 2010), the weight of evidence of available *in vitro* and *in vivo* data altogether indicates that decaBDE has the potential to induce neurotoxic effects in mammals exposed to decaBDE during early stages of development (see reviews by Dingemans et al. 2011, Costa and Giordano 2011, Health Canada 2012).

39.   Worldwide, humans of all ages are daily exposed to decaBDE via environmental media and a range of food stuffs, including mother's milk (see Costa and Giordano 2011, Health Canada 2012, EFSA 2011 and references therein). In Europe decaBDE along with BDE-47 is the predominant PBDE congener in food (EFSA 2011). In adults, household dust and occupational exposure is thought to be the main sources of decaBDE exposure (Costa and Giordano 2011). Dust will also be a major source of decaBDE for toddlers which have a higher tendency to transfer house dust particles from their hand to their mouth. Further, for infants mouthing of hard plastic toys can be an additional exposure route (Health Canada 2012).

40.   The observation that exposure takes place already during the early phases of human development i.e. *in utero* via placental transfer or postnatally via mothers milk (Gomara et al. 2007, Kawashiro et al. 2008, Wu et al. 2010, Miller et al. 2012), proposes that the developmental neurotoxicity observed in mammalian models could have implications also for humans (Health Canada 2012, US EPA 2008, EFSA 2011, Costa and Giordano 2011). However, although PBDEs share structural similarities with other environmental pollutants such as polychlorinated biphenyls (PCBs) and organochlorines, investigation into the toxicity and carcinogenicity of decaBDE in humans remains surprisingly limited (for overview see e.g. US EPA 2008, Health Canada 2012, Health Canada 2006). Yet, evidence is slowly emerging that BDE-209 either alone or in concert with other PBDEs could act as a developmental neurotoxicant and possibly play a role as a risk factor in human disease (e.g. Dingemans et et al. 2011, Messer et al. 2010, Kicinski et al. 2012, Costa and Giordano 2011, Health Canada 2012, Health Canada 2006, Gascon et al. 2012, Chao et al. 2011). Effects of decaBDE on thyroid hormone signaling, a major timing factor for the precise regulation of brain cell growth and brain connectivity, has for example been proposed as a factor that could contribute to human neurological disorders such as autism (Messer 2010). In a recent epidemiological study assessing the linkage between PBDE levels in breast milk and neurophysiological development in infants decaBDE was significantly correlated with lower mental development scores in children 12-18 months of age (Gascon et al. 2012). The observation of a correlation between decaBDE exposure levels and lower mental development scores found in this study are consistent with results previously reported by Chao et al. (2011). Gascon et al. (2012), but not Chao et al. (2011), also reported a negative, but non-significant correlation between the total sum of PBDEs in breast milk and mental test scores (Chao et l. 2011, Gascon et al. 2012). In this context is also worth noting that epidemiological data

indicating an association between PBDE exposure at early age and neurodevelopmental toxicity have also been reported for lower brominated PBDEs (Roze et al. 2009, Herbstman et al. 2010, Gascon et al. 2011, Kicinski et al. 2012).

41.   Risk characterizations of decaBDE conducted by Health Canada (2012) and US EPA (2008), have suggested that the daily intake of decaBDE in the United States and Canada at present is not likely to result in neurodevelopmental toxicity even for the potentially most highly exposed and sensitive age group, infants. A similar conclusion was reached by European Food Safety Authority Panel on Contaminants (EFSA 2011). However, neither risk assessment take into account the potential risk that PBDEs could act in concert to induce additive or synergistic effects as suggested by the available *in vitro* data (e.g. Pellacani et al. 2010, Tagliaferri et al. 2010, Llabjani et al. 2010, Karpeta and Gregoraszczuk 2010, Hallgren and Darnerud 2002, He et al. 2009). In this context it is worth noting that the recently published WHO/ UNEP report on endocrine disruptors concludes that endocrine disruptors can work together to produce additive effects, even when combined at low doses that individually do not produce observable effects (WHO/UNEP 2012). Moreover, as pointed out by Health Canada (2012), the assessment of human health risks is limited by a scarcity of inhalation and/or dermal exposure data as well as by insufficient data on toxicokinetics of decaBDE in humans. Lastly additional risk factors were also not considered. Iodine deficiency, a common condition worldwide (reviewed by Walker et al. 2007), is said to increase the sensitivity to adverse effects from thyroid-disrupting chemicals such as decaBDE (Dingemans et al. 2011).

Conclusion on adverse effects according to the criteria in Annex D

42.   The weight of evidence of available toxicity data shows that decaBDE alone and/ or in concert with its debromination products have the potential to damage human health and/or the environment.

6.   Statement of the reasons for concern and need for global action

6.1   Reasons for global concern

43.   Based on the existing data decaBDE can be considered to meet the screening criteria in Annex D for persistence, bioaccumulation, long-range transport and adverse effects under the Stockholm Convention. Adding to this concern is the potential debromination to other POPs and the possibility of combined effects. Several assessments have concluded that there is a high probability that decaBDE is transformed in the environment and in biota to form substances or act as precursors to lower brominated PBDEs, which themselves are POPs. In addition reported *in vitro* data moreover suggests the possibility that the different PBDEs could act in concert to induce additive or synergistic effects.

44.   Adding to the above concern is that decaBDE is widespread in the global environment and in biota and the potential of biomagnification of decaBDE has been shown to be important in several food webs. In addition the temporal trends indicate that the levels of decaBDE are increasing in the Arctic atmosphere. Although the tissue levels might be relatively low in some species a study on fish suggests that chronic exposure of decaBDE at low doses can lead to adverse effects, especially in juvenile life stages. Of particular concern is that decaBDE is a potential endocrine disruptor. Endocrine disruptive chemicals produce a non-linear dose–response and can cause adverse effects even when environmental levels are low (UNEP/WHO 2012). Sensitivity to endocrine disruption is highest during tissue development and organisms in early developmental stages are more vulnerable than adults (UNEP/WHO 2012). Furthermore the high persistence of decaBDE in sediments and soils means that organisms may be exposed continuously, thus increasing the likelihood for adverse effects and the exposure of organisms will continue long after the release into the environment have stopped.

45.   The use of decaBDE is currently widespread across the globe. Although use and placing on the market of decaBDE has been restricted recently in some regions it is still produced and used as a flame retardant in many countries. The use of flame retardants is foreseen to increase globally partly due to more stringent fire safety regulations. This will especially affect the temporal trends of decaBDE in regions with no restrictions on further marketing or use of the substance and contribute to the environmental levels in regions far from sources. Furthermore, the long-range transport of decaBDE denotes that single countries or groups of countries cannot abate the pollution caused by it alone. Due to the harmful POP properties and risks related to its possible continuing production and use, international action is warranted.

7.   References

Arctic Monitoring and Assessment Programme, AMAP 2009. Arctic Pollution 2009, Oslo. 83pp.

Bakke T, Boitsov S, Brevik EM, Gabrielsen GW, Green N, Helgason LB, Klungsøyr J, Leknes H, Miljeteig C, Måge A, Rolfsnes BE, Savinova T, Schlabach M, Skaare BB, Valdersnes S. (2008). Mapping selected organic contaminants in the Barents Sea 2007 (TA-2400/2008). Norwegian Pollution Control Agency, 2008, pp.132

Balch, G. C., L. A. Vélez-Espino, et al. (2006). Inhibition of metamorphosis in tadpo of Xenopus laevis exposed to polybrominated diphenyl ethers (PBDEs). Chemosphere 64(2). 328-338.

Booij, K., B. N. Zegers, et al. (2002). Levels of some polybrominated diphenyl ether (PBDE) flame retardants along the Dutch coast as derived from their accumulation in SPMDs and blue mussels (Mytilus edulis). Chemosphere 46 (5). 683-688.

Breivik, K., F. Wania, et al. (2006). Empirical and modeling evidence of the long-range atmospheric transport of decabromodiphenyl ether. Environ Sci Technol 40(15). 4612-4618.

Bromine Science and Environmental Forum, BSEF (2013). About decabromo diphenyl ether (decaBDE), http.//www.bsef.com/our-substances/deca-bde/about-deca-bde (accessed in April 2013)

Chao HR, Tsou TC, et al. (2011). Levels of breast milk PBDEs from southern Taiwan and their potential impact on neurodevelopment. Pediatr Res 70(6). 596-600.

Chen D, Hale RC (2010). A global review of polybrominated diphenyl ether flame retardant contamination in birds. Environment International, 36, 800–811.

Chen Q, Yu L, et al. (2012). Bioconcentration and metabolism of decabromodiphenyl ether (BDE-209) result in thyroid endocrine disruption in zebrafish larvae. Aquat Toxicol 111. 141-148.

Christensen J, Mac Duffee M, MacDonald RW, Whiticar M, Ross P (2005). Persistent organic pollutants in British Columbia grizzly bears. Consequence of divergent diets. Environ. Sci. Technol. 2005, 39, 6952-6960.

CITI (1992). Biodegradation and bioaccumulation data of existing chemicals based on the CSCL Japan. Chemicals Inspection and Testing Institute Japan. Japan Chemical Ecology-Toxicology and Information Centre, October 1992.

Costa LG, Giordano G (2011). Is decabromodiphenyl ether (BDE-209) a developmental neurotoxicant? Neurotoxicology 32(1). 9-24.

Danon-Schaffer MN, Gorgy T, Li L, Grace JR. (2007). Determination of PBDEs in Canadian North landfill leachate and soils. Final Report - September 2007. Vancouver, BC, Canada.Environmental Damages Fund, Environment Canada, University of British Columbia;2007. 139 pp.

Daso AP, Fatoki OS, Odendaal JP. Occurrence of polybrominated diphenyl ethers (PBDEs) and 2,2',4,4',5,5'-hexabromobiphenyl (BB-153) in water samples from the Diep River, Cape Town, South Africa. Environ Sci Pollut Res Int. 2013 Jan 30.[Epub ahead of print] PubMed PMID: 23361180.

de Wit CA (2002). An overview of brominated flame retardants in the environment. Chemosphere 2002, 46, 583-624.

de Wit CA, Alaee M, et al. (2006). Levels and trends of brominated flame retardants in the Arctic. Chemosphere 64(2). 209-233.

de Wit CA, Herzke D, et al. (2010). Brominated flame retardants in the Arctic environment--trends and new candidates. Sci Total Environ 408(15). 2885-2918.

Dinn PM, Johannessen SC, et al. (2012). PBDE and PCB accumulation in benthos near marine wastewater outfalls. the role of sediment organic carbon. Environ Pollut 171. 241-248.

Dingemans MM, van den Berg M, et al. (2011). Neurotoxicity of brominated flame retardants. (In)direct effects of parent and hydroxylated polybrominated diphenyl ethers on the (developing) nervous system. Environ Health Perspect 119(7). 900-907.

Environment Canada (2006). Canadian Environmental Protection Act, 1999. Ecological Screening Assessment Report on polybrominated diphenyl ethers (PBDEs).

Environment Canada (2008). The Polybrominated Diphenyl Ethers Regulations (SOR/2008-218) under the Canadien Environmental Protection Act, 1999.

Environment Canada (2010a). Canadian Environmental Protection Act, 1999. Ecological State of the Science Report on Decabromodiphenyl Ether (decaBDE). Bioaccumulation and Transformation.

Environment Canada (2010b). Risk Management Strategy for polybrominated diphenyl ethers (PBDEs) European Commission, EC (2002). European Union Risk Assessment Report. Bis(pentabromophenyl ether). 1st Priority List, Volume 17. EUR 20402 EN. European Chemicals Bureau, Institute of Health and Consumer Protection, European Commission.

European Commission, EC (2004). Update of the Risk assessment of bis(pentabromephenyl ether (decabromodiphenyl ether) CAS Number. 1163-19-5, EINECS Number. 214-604-9, Final Environmental Draft of May 2004, 294 pp.

European Commission (2007). Update of the risk assessment of bis(pentabromophenyl) ether. Final Draft of October 2007. European Chemicals Bureau.

European Commission (2008). Communication from the Commission on the results of the risk evaluation of chlorodifluoromethane, bis(pentabromophenyl)ether and methenamine and on the risk reduction strategy for the substance methenamine, 2008/C 131/04, Official Journal of the European Union, 29.5.2008. Available online: http://echa.europa.eu/web/guest/information-on-chemicals/information-from-existing-substances-regulation?search_criteria=1163-19-5

European Chemicals Agency, ECHA (2012a). Support Document Bis(pentabromophenyl) ether [decabromodiphenyl ether] (Member State Committee, 29 November 2012).

European Chemicals Agency, ECHA (2012b). Agreement of the member state committee On the identification of bis(pentabromophenyl) ether [decabromodiphenyl ether], (Member State Committee, 29 November 2012).

European Chemicals Agency, ECHA (2012c). Annex XV dossier. Proposal for Identification of a PBT/vPvB Substance. Bis(pentabromophenyl)ether (decabromodiphenyl ether; decaBDE). July 2012-final.

European Food Safety Authority Panel, EFSA (2011). European Food Safety Authority Panel on Contaminants in the Food Chain (CONTAM). Scientific Opinion on Polybrominated Diphenyl Ethers (PBDEs) in Food. EFSA Journal, 9 (5), 2156. doi.10.2903/j.efsa.2011.2156. Available online: http://www.efsa.europa.eu/en/efsajournal/doc/2156.pdf

Freedonia (2010). A study report on the market research of The World Flame Retardants Industry.

Gascon M, Vrijheid M et al. (2011). "Effects of pre and postnatal exposure to low levels of polybromodiphenyl ethers on neurodevelopment and thyroid hormone levels at 4 years of age." Environ Int 37(3). 605-611.

Gascon M, Fort M, et al. (2012). "Polybrominated diphenyl ethers (PBDEs) in breast milk and neuropsychological development in infants." Environ Health Perspect 120(12). 1760-1765.

Gerecke AC, Hartmann PC, Heeb NV, Kohler H-P E , Giger W, Schmid P, Zennegg M, Kohler M (2005). Anaerobic degradation of decabromodiphenyl ether. Environmental Science and Technology, 39, 1078-1083.

Gerecke AC, Giger W, Hartmann PC, Heeb NV, Kohler H-P E, Schmid P, Zennegg M, Kohler M (2006). Anaerobic degradation of brominated flame retardants in sewage sludge. Chemosphere, 64, 311-317.

Gomara B, Herrero L, Ramos JJ, Mateo JR, Fernandez MA, Garcia JF, Gonza MJ (2007). Distribution of polybrominated diphenyl ethers in human umbilical cord serum, paternal serum, maternal serum, placentas, and breast milk from Madrid population, Spain. Environ. Sci. Technol 2007;41.6961–6968.

Goodman JE (2009). Neurodevelopmental effects of decabromodiphenyl ether (BDE-209) and implications for the reference dose." Regul Toxicol Pharmacol 54(1). 91-104.

Hale RC, Alace M, Manchester-Neesvig JB, Stapleton HM, Ikonomou MG (2003). Polybrominated diphenyl ether flame retardants in the North American environment. Environ. Int. 2003, 29, 771–779.

Hallgren S, Darnerud PO (2002). Polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs) and chlorinated paraffins (CPs) in rats-testing interactions and mechanisms for thyroid hormone effects. Toxicology 177(2-3). 227-243.

Hardy ML, Banasik M, Stedeford T. Toxicology and human health assessment of decabromodiphenyl ether. Crit. Rev. Toxicol 2009;39 S3:1–44.

He J, Robrock KR, Alvarez-Cohen L (2006). Microbial reductive debromination of polybrominated diphenyl ethers (PBDEs). Environmental Science and Technology, 40, 4429-4434.

He P, Wang AG. et al. (2009). Mechanism of the neurotoxic effect of PBDE-47 and interaction of PBDE-47 and PCB153 in enhancing toxicity in SH-SY5Y cells. Neurotoxicology 30(1). 10-15.

He J, Yang D, et al. (2011). Chronic zebrafish low dose decabrominated diphenyl ether (BDE-209) exposure affected parental gonad development and locomotion in F1 offspring. Ecotoxicology 20(8). 1813-1822.

He MJ, Luo XJ, et al. (2012). Bioaccumulation of polybrominated diphenyl ethers and decabromodiphenyl ethane in fish from a river system in a highly industrialized area, South China. Science of the Total Environment 419. 109-115.

Health Canada (2006). State of the Science Report for a Screening Health Assessment Polybrominated Diphenyl Ethers (PBDEs)[Tetra-, Penta-, Hexa-, Hepta-, Octa-,Nona- and Deca- Congeners][CAS Nos. 40088-47-9, 32534-81-9, 36483-60-0,68928-80-3, 32536-52-0, 63936-56-1, 1163-19-5]. 39.

Health Canada (2012). Human Health State of the Science Report on Decabromodiphenyl Ether (decaBDE). 32.

Herbstman JB, Sjodin A, Kurzon M, Lederman SA, Jones RS, Rauh V, Needham LL, Tang D, Niedzwiecki M, Wang RY, Perera F. Prenatal exposure to PBDEs and neurodevelopment. Environ. Health Perspect 2010;118:712–719.

Hermanson MH, Isaksson E, Forsström S, Teixeira C, Muir DC, Pohjola VA, van de Wal RS. Deposition history of brominated flame retardant compounds in an ice core from Holtedahlfonna, Svalbard, Norway. Environ Sci Technol. 2010 Oct 1;44(19).7405-10.

Hu GC, Dai JY, et al. (2010). Bioaccumulation behavior of polybrominated diphenyl ethers (PBDEs) in the freshwater food chain of Baiyangdian lake, north China. Environ Int 36(4). 309-315.

Hung H, Kallenborn R, et al. (2010). Atmospheric monitoring of organic pollutants in the Arctic under the Arctic Monitoring and Assessment Programme (AMAP). 1993-2006. Sci Total Environ 408(15). 2854-2873.

Ibhazehiebo K, Iwasaki T, et al. (2011). Disruption of thyroid hormone receptor-mediated transcription and thyroid hormone-induced Purkinje cell dendrite arborization by polybrominated diphenyl ethers. Environ Health Perspect 119(2). 168-175.

Jaspers VLB, Covaci A, Voorspoels S, Dauwe T, Eens M, Schepens P. Brominated flame retardants and organochlorine pollutants in aquatic and terrestrial predatory birds of Belgium. levels, patterns, tissue distribution and condition factors. Environ Pollut 2006;139.340–52.

Jenssen BM, Sørmo E, Bæk K, Bytingsvik J, Gaustad H, Ruus A, Skaare JU. Brominated flame retardants in north-east Atlantic marine ecosystems. Environmental Health Perspectives vol 115, Sup 1, December 2007.

Johansson N, Viberg H, Fredriksson A, Eriksson P (2008). Neonatal exposure to deca-brominated diphenyl ether (PBDE 209) causes dose response changes in spontaneous behaviour and cholinergic susceptibility in adult mice. Neurotoxicology 2008, 29.911–919.

Karpeta A, Gregoraszczuk E (2010). Mixture of dominant PBDE congeners (BDE-47, -99, -100 and -209) at levels noted in human blood dramatically enhances progesterone secretion by ovarian follic. Endocr Regul 44(2). 49-55.

Kawashiro Y, Fukata H, Omori-Inoue M, Kubonoya K, Jotaki T, Takigami H, Sakai S, Mori C. (2008). Perinatal exposure to brominated flame retardants and polychlorinated biphenyls in Japan. Endocr. J. 2008;55.1071–1084.

Kelly BC, Ikonomou MG, Blair JD, Morin AE, Gobas FAPC. (2007). Food web-specific biomagnification of persistent organic pollutants. Science 2007;317.236–9.

Kicinski M, Viaene MK, et al. (2012). Neurobehavioral function and low-level exposure to brominated flame retardants in adocents. a cross-sectional study. Environ Health 11(86). 11-86.

Kierkegaard A, Balk L, Tjarnlund U, De Wit CA, Jansson, B. (1999). Dietary uptake and biological effects of decabromodiphenyl ether in rainbow trout (Oncorhynchus mykiss).Environ. Sci. Technol. 1999, 33 (10), 1612−1617.

Klosterhaus SL, Stapleton HM, La Guardia MJ, Greig DJ (2012). Brominated and chlorinated flame retardants in San Francisco Bay sediments and wildlife. Environment International 47 (2012) 56–65.

Knudsen LB, Polder A, Føreid S, Lie E, Gabrielsen GW, Barret R, Skåre JU. (2006). Nona- and deca-brominated diphenylethers in seabird eggs from Northern Norway and Svalbard. Report TA-2174/2006, Norwegian Pollution Control Agency, 2006, pp.30

Koenig S, Huertas D, Fernández, P. (2013). Legacy and emergent persistent organic pollutants (POPs) in NW Mediterranean deep-sea organisms. Science of the Total Environment 443 (2013) 358–366.

Kuo YM, Sepulveda MS, et al. (2010). Bioaccumulation and biotransformation of decabromodiphenyl ether and effects on daily growth in juvenile lake whitefish (Coregonus clupeaformis). Ecotoxicology 19(4). 751-760.

La Guardia MJ, Hale RC, et al. (2012). In Situ Accumulation of HBCD, PBDEs, and Several Alternative Flame-Retardants in the Bivalve (Corbicula fluminea) and Gastropod (Elimia proxima). Environmental Science & Technology 46(11). 5798-5805.

Law K, Halldorson T, et al. (2006). Bioaccumulation and trophic transfer of some brominated flame retardants in a Lake Winnipeg (Canada) food web. Environ Toxicol Chem 25(8). 2177-2186.

Law RJ, Herzke D, Harrad S, Morris S, Bersuder P, Allchin CR.(2008). Levels and trends of HBCD and BDEs in the European and Asian environments, with some information for other BFRs. Chemosphere 73 (2008) 223–241.

Lee LK, He J (2010). Reductive debromination of polybrominated diphenyl ethers by anaerobic bacteria from soils and sediments. Applied and Environmental Microbiology, 76(3), 794-802.

Letcher RJ, Bustnes JO, Dietz R, Jenssen BM, Jørgensen EH, Sonne C, Verreault J, Vijayan MM, Gabrielsen GW. Exposure and effects assessment of persistent organohalogen contaminants in arctic wildlife and fish. Sci Total Environ. 2010 Jul 1;408(15).2995-3043.

Li ZH, Liu XY, et al. (2012). Effects of decabrominated diphenyl ether (PBDE-209) in regulation of growth and apoptosis of breast, ovarian, and cervical cancer cells. Environ Health Perspect 120(4). 541-546.

Liu L, Zhu W, Xiao L, Yang L, 2011a. Effect of decabromodiphenyl ether (BDE 209) and dibromodiphenyl ether (BDE 15) on soil microbial activity and bacterial community composition. Journal of Hazardous Materials, 186, 883-890.

Liu L, Zhu W, et al. (2011) b. Effect of decabromodiphenyl ether (BDE 209) and dibromodiphenyl ether (BDE 15) on soil microbial activity and bacterial community composition. J Hazard Mater 186(1). 883-890.

Liu X, Zhan H, et al. (2012). The PBDE-209 exposure during pregnancy and lactation impairs immune function in rats. Mediators Inflamm 692467(10). 15.

Llabjani V, Trevisan J, et al. (2010). Binary Mixture Effects by PBDE Congeners (47, 153, 183, or 209) and PCB Congeners (126 or 153) in MCF-7 Cells. Biochemical Alterations Assessed by IR Spectroscopy and Multivariate Analysis. Environmental Science & Technology 44(10). 3992-3998.

Mansouri, K., V. Consonni, et al. (2012). Assessing bioaccumulation of polybrominated diphenyl ethers for aquatic species by QSAR modeling. Chemosphere 89(4). 433-444.

Mariussen E, Steinnes E, et al. (2008). Spatial patterns of polybrominated diphenyl ethers (PBDEs) in mosses, herbivores and a carnivore from the Norwegian terrestrial biota. Sci Total Environ 404(1). 162-170.

Messer A. (2010). Mini-review. polybrominated diphenyl ether (PBDE) flame retardants as potential autism risk factors. Physiol Behav 100(3). 245-249.

Meyer T, Muir DC, et al. (2012). "Deposition of brominated flame retardants to the Devon Ice Cap, Nunavut, Canada." Environ Sci Technol 46(2). 826-833.

Miller MF, Chernyak SM, et al. (2012). Concentrations and speciation of polybrominated diphenyl ethers in human amniotic fluid. Science of The Total Environment 417–418(0). 294-298.

Miyaso H, Nakamura N, et al. (2012). Postnatal exposure to low-dose decabromodiphenyl ether adversely affects mouse testes by increasing thyrosine phosphorylation level of cortactin. J Toxicol Sci 37(5). 987-999.

Mo L, Wu JP, Luo XJ, Zou FS, Mai BX. (2013). Bioaccumulation of polybrominated diphenyl ethers, decabromodiphenyl ethane, and 1,2-bis(2,4,6-tribromophenoxy) ethane flame retardants in kingfishers (Alcedo atthis) from an electronic waste-recycling site in South China. Environ Toxicol Chem. 2012 Sep;31(9).2153-8.

Mörck A, Hakk H, Orn U, Wehler EK. (2003). Decabromdiphenyl ether in the rat. Absorption, distribution, metabolism, and excretion. Drug metabolism and disposition,Vol. 31, No. 7 Copyright © 2003 by The American Society for Pharmacology and Experimental Therapeutics 983/1072490 DMD 31.900–907, 2003.

Muir, D. (2011). Fate, bioavailability, and effects of decabromodiphenyl ether and non-BDE BFRs. Final Project Report, Chemicals Management Plan Research Fund. 13 June 2011. Environment Canada.

Muir D, Teixeira C, Chigak M, Yang F, D'Sa I, Cannon C, Pacepavicius G, Alaee M (2003). Current deposition and historical profi of decabromodiphenyl ether in sediment cores. Organohalogen Compd. 2003, 61, 77–80.

Möller A, Xie Z, et al. (2011). Polybrominated diphenyl ethers (PBDEs) and alternative brominated flame retardants in air and seawater of the European Arctic. Environ Pollut 159(6). 1577-1583.

Noyes PD, Stapleton HM(2010). Impacts of polybrominated diphenylethers (PBDEs) on the matabolism of thyroid hormones in early life stages of fathead minnows (Pimepha promelas). Organohalogen Compounds 72. 1334-1337.

Noyes PD, Hinton DE, et al. (2011). Accumulation and debromination of decabromodiphenyl ether (BDE-209) in juvenile fathead minnows (Pimepha promelas) induces thyroid disruption and liver alterations. Toxicol Sci 122(2). 265-274.

Nyholm JR, Lundberg C and Andersson PL (2010). Biodegradation kinetics of selected brominated flame retardants in aerobic and anaerobic soil. Environmental Pollution, 158 (6), 2235-2240.

Nyholm JR (2011). Email from Jenny Nyholm to Stephen Dungey, Environment Agency, 21 August 2011.

Organisation for Economic Co-operation and Development, OECD (2008). Brominated Flame Retardants (BFRs). Hazard/Risk Information Sheets, October 2008. http.//www.oecd.org/dataoecd/3/6/42073463.pdf

Orihel et al. (2009). Summary of major research projects at the experimental lakes area during 2008. Available online: http.//www.ontarioaquaculture.com/files/ELARES2008.pdf

Pacyniak EK, Cheng X, et al. (2007). The flame retardants, polybrominated diphenyl ethers, are pregnane X receptor activators. Toxicol Sci 97(1). 94-102.

Pellacani C, Buschini A, Galati S, Mussi F, Franzoni S, Costa LG. Evaluation of DNA damage induced by 2 polybrominated diphenyl ether flame retardants (BDE-47 and BDE-209) in SK-N-MC cells. Int J Toxicol. 2012 Jul-Aug; 31(4):372-9.

Persistent Organic Pollutants Review Committee (POPRC) (2007). Risk profile on commercial octabromodiphenyl ether. Document UNEP/POPS/POPRC.3/20/Add.6 pp. 23. Available online: http.//chm.pops.int/Convention/POPsReviewCommittee/Reviewedchemicals/tabid/781/Default.aspx

Persistent Organic Pollutants Review Committee (POPRC) (2006). Risk profile on commercial pentabromodiphenyl ether. Document UNEP/POPS/POPRC.2/17/Add.1. Available online: http.//chm.pops.int/Convention/POPsReviewCommittee/Reviewedchemicals/tabid/781/Default.aspx

Powell DE, Sesten RM, Woodburn KE, Gerhards R. (2013). Trophic magnification factors (TMF). Interrelationship with other measures used to interpret bioaccumulation in aquatic environments. Scientific advancements in bioaccumulation assessment. Helsinki Workshop, 11 mars 2013.

Qin X, Xia X, et al. (2010). Thyroid disruption by technical decabromodiphenyl ether (DE-83R) at low concentrations in Xenopus laevis. J Environ Sci 22(5). 744-751.

Raff JD, Hites RA (2007). Deposition versus Photochemical Removal of PBDEs from Lake Superior Air. Environ.Sci. Technol. 2007, 41, 6775-6731.

Rice DC, Reeve EA, Herlihy A, Thomas Zoeller R, Douglas Thompson W, Markowski VP (2007). Developmental delays and locomotor activity in the C57BL6/J mouse following neonatal exposure to the fully-brominated PBDE, decabromodiphenyl ether. Neurotoxicol Teratol 2007, 29.511–520.

Rice DC, Thompson WD, Reeve EA, Onos KD, Assadollahzahed M, Markowski VP (2009). Behavioral changes in aging but not young mice after neonatal exposure to the polybrominated flame retardant decaBDE. Environ Health Perspect 117.1903–1911. Toxicology and Pharmacology 23(3). 302-307.

Roze E, Meijer L, et al. (2009). Prenatal exposure to organohalogens, including brominated flame retardants, influences motor, cognitive, and behavioral performance at school age. Environ Health Perspect 117(12). 1953-1958.

Schenker U, Soltermann F, Scheringer M, Hungerbühler K. (2010). Modeling the environmental fate of polybrominated diphenyl ethers (PBDEs). the importance of photolysis for the formation of lighter PBDEs. Environ. Sci. Technol. 2010, 42(24), 9244-9249.

Schriks M, Zvinavashe E, Furlow DJ, Murk AJ (2006). Distruption of thyroid hormone-mediated Xenopus laevis tadpole tail tip regression by hexabromocyclododecane (HBCD) and 2,2',3,3',4,4',5,5',6-nonabrominated diphenyl ether (BDE-206). Chemosphere, 65(10), 1904–1908.

Schriks M, Roessig JM, et al. (2007).Thyroid hormone receptor isoform selectivity of thyroid hormone disrupting compounds quantified with an in vitro reporter gene assay.Environ Toxicol Pharmacol 23(3). 302-307.

Shaw SD, Berger ML, et al. (2009). Bioaccumulation of polybrominated diphenyl ethers and hexabromocyclododecane in the northwest Atlantic marine food web. Sci Total Environ 407(10). 3323-3329.

Sifleet SD, 2009. Toxicology of Decabromodiphenyl Ether in Avian Embryos. Disposition of the Flame Retardant BDE-209 in Yolk-injected Chicken Embryos (Gallus gallus). Thesis presented to the Faculty of the School of Marine Science, The College of William and Mary in Virginia, USA.

Stapleton HM, Letcher RJ, Baker JE (2004). Debromination of polybrominated diphenyl ether congeners BDE 99 and BDE 183 in the intestinal tract of the common carp (Cyprinus carpio). Environmental Science and Technology, 38, 1054-1061.

SuY, Hung H, Sverko E, Fellin P, Li H (2007). Multi-year measurements of polybrominated diphenyl ethers (PBDEs) in the Arctic atmosphere. Atmos. Environ. 2007, 41, 8725–8735.

Søderstrøm G (2003). On the combustion and photolytic degradation products of some brominated flame retardants. Department of Chemistry, Environmental Chemistry, University of Umea, Sweden.

Sørmo EG, Salmer MP, Jenssen BM, Hop H, Baek K, Kovacs KM, et al. (2006). Biomagnification of polybrominated diphenyl ether and hexabromocyclododecane flame retardants in the polar bear food chain in Svalbard, Norway. Environ Toxicol Chem 2006;25. 2502–11.

Tagliaferri S, Caglieri A, et al. (2010). Low concentrations of the brominated flame retardants BDE-47 and BDE-99 induce synergistic oxidative stress-mediated neurotoxicity in human neuroblastoma cells. Toxicol In Vitro 24(1). 116-122.

Tian SY, Zhu LY, et al. (2012). Bioaccumulation and Metabolism of Polybrominated Diphenyl Ethers in Carp (Cyprinus carpio) in a Water/Sediment Microcosm. Important Role of Particulate Matter Exposure. Environmental Science & Technology 46(5). 2951-2958.

Tokarz III JA, Ahn M-Y, Leng J, Filley T.R., Nies L, 2008. Reductive debromination of polybrominated diphenyl ethers in anaerobic sediment and a biomimetic system. Environmental Science and Technology, 42 (4), 1157-1164.

Tomy GT, Pleskach K, et al. (2008). Enantioselective bioaccumulation of hexabromocyclododecane and congener-specific accumulation of brominated diphenyl ethers in an eastern Canadian Arctic marine food web. Environ Sci Technol 42(10). 3634-3639.

Tomy GT, Pkach K, Ferguson SH, Hare J, Stern G, MacInnis G, Marvin CH, Loseto L. (2009). Trophodynamics of some PFCs and BFRs in a western Canadian Arctic marine food web. Environ Sci Technol 43.4076-4081.

Tseng, L. H., C. W. Lee, et al. (2006). Postnatal exposure of the male mouse to 2,2',3,3',4,4',5,5',6,6'-decabrominated diphenyl ether. decreased epididymal sperm functions without alterations in DNA content and histology in testis. Toxicology 224(1-2). 33-43.

Tysklind, M, Sellstrom U, Soderstrom G, de Wit C (2001). Abiotic transformation of polybrominated diphenyl ethers (PBDEs). Photolytic debromination of decabromo diphenyl ether. The Second International Workshop on Brominated Flame Retardants, BFR 2001, 14-16 May 2001, Stockholm.

UK Environment Agency (2009). Environmental risk evaluation report. Decabromodiphenyl ether (CAS no. 1163-19-5). Authors Brooke, D.N., Burns, J., Crookes, M.J. and Dungey, S.M Report to the. 290 pp.

UNEP (2007). Report of the Persistent Organic Pollutants Review Committee on the work of its third meeting. Addendum. Risk profile on commercial octabromodiphenyl ether. Third meeting of Persistent Organic Pollutants Review Committee, Geneva, 19–23 November 2007. Stockholm Convention on Persistent Organic Pollutant . UNEP/POPS/POPRC.3/20/Add.6.

UNEP (2011). Other technical work. debromination of brominated flame retardants. Update on reductive debromination of polybrominated diphenyl ethers. Persistent Organic Pollutants Review Committee. Seventh meeting, Geneva, 10–14 October 2011. UNEP/POPS/POPRC.7/INF/18

US EPA (2008). Toxicological Review of Decabromodiphenyl ether (BDE-209) (CAS No. 1163-19-5). 599. Available online: www.epa.gov/iris

US EPA 2013 (accessed in April 2013). DecaBDE Phase-out Initiative http.//www.epa.gov/oppt/existingchemicals/pubs/actionplans/deccadbe.html (EF)

US EPA (2011). U.S. EPA Office of Pollution Toxics and Syracuse Research Corporation (SRC), EPISuite 4.1, 2011. http.//www.epa.gov/opptintr/exposure/pubs/episuitedl.htm

Van den Steen E, Covaci A, Jaspers VLB, Dauwe T, Voorspoels S, EensM, et al. (2007). Accumulation, tissue-specific distribution and debromination of decabromodiphenyl ether (BDE 209) in European starlings (Sturnus vulgaris). Environ Pollut 2007;148.648–53.

VECAP, 2011. Sustainable improvement. VECAP Annual Progress Report 2011. Available online: http.//www.vecap.info/

Viberg H, Fredriksson A, Jakobsson E, Orn U, Eriksson P (2003). Neurobehavioral derangements in adult mice receiving decabrominated diphenyl ether (PBDE 209) during a defined period of neonatal brain development. Toxicol Sci 2003, 76.112–120.

Viberg H, Johansson N, Fredriksson A, Eriksson J, Marsh G, Eriksson P. (2006). Neonatal exposure to higher brominated diphenyl ethers, hepta-, octa-, or nonabromodiphenyl ether, impairs spontaneous behavior and learning and memory functions of adult mice. Toxicol Sci 92.211–218.

Viberg H, Fredriksson A, Eriksson P (2007). Changes in spontaneous behavior and altered response to nicotine in the adult rat, after neonatal exposure to the brominated flame retardant, decabrominated diphenyl ether (PBDE 209). Neurotoxicology 2007, 28.136–142.

Voorspoels S, Covaci A, Lepom P, Escutenaire S, Schepens P. (2006 a). Remarkable findings concerning PBDEs in the terrestrial top-predator red fox. Environ. Sci. Technol. 40, 2937-2943.

Voorspoels S, Covaci A, Lepom P, Jaspers VL, Schepens P. Levels and distribution of polybrominated diphenyl ethers in various tissues of birds of prey. Environ Pollut. 2006 Nov;144(1).218-27.

Vorkamp K, Thomsen M, Falk K, lie H, Møller S, Sørensen PB. (2005). Temporal development of brominated flame retardants in peregrine falcon (Falco peregrinus) eggs from South Greenland (1986-2003). Environ Sci Technol 39.8199-8206.

Walker, S. P., T. D. Wachs, et al. (2007). Child development. risk factors for adverse outcomes in developing countries. Lancet 369(9556). 145-157.

Wang Z, Ma X, et al. (2009). Congener specific distributions of polybrominated diphenyl ethers (PBDEs) in sediment and mussel (Mytilus edulis) of the Bo Sea, China. Chemosphere 74(7). 896-901.

Wang S, Zhang S, Huang H, Christie P (2011a). Behaviour of decabromodiphenyl ether (BDE-209) in soil. Effects of rhizosphere and mycorrhizal colonization of ryegrass roots. Environmental Pollution, 159, 749-753.

Wang F, Wang J, Hu G, Luo X, Mai B, Dail J. (2011 b). Tissue distribution and associated toxicological effects of decabrominated diphenyl ether in subchronically exposed male rats. International Scholarly Research Network ISRN Toxicology, Volume 2011, Article ID 989251, 8 pages.

Wang XM, Ding X, Mai BX, Xie ZQ, Xiang CH, Sun LG, et al. (2005). Polybrominated diphenyl ethers in airborne particulates collected during a research expedition from the Bohai Sea to the Arctic. Environ Sci Technol 2005; 39. 7803–9.

Wania F, Dugani CB (2003). Assessing the long-range transport potential of plybrominated diphenyl ethers. a comparison of four multimedia models. Environ. Toxicol. Chem.2003, 22, 1252–1261.

Watanabe W, Shimizu T, et al. (2008). Effects of decabrominated diphenyl ether (DBDE) on developmental immunotoxicity in offspring mice. Environ Toxicol Pharmacol 26(3). 315-319.

Watanabe W, Shimizu T, et al. (2010). Functional disorder of primary immunity responding to respiratory syncytial virus infection in offspring mice exposed to a flame retardant, decabrominated diphenyl ether, perinatally. J Med Virol 82(6). 1075-1082.

Wilford BH. et al. (2008). Decabromodiphenyl ether (deca-BDE) commercial mixture components, and other PBDEs, in airborne particulates at a UK site. Environment International, 34 (3), 412-419.

Williams AL, DeSesso JM. (2010). The potential of selected brominated flame retardants to affect neurological development. J Toxicol Environ Health B 13.411–448.

WHO/UNEP (2012). State of the science of endocrine disrupting chemicals. An assessment of the state of the science of endocrine disruptors prepared by a group of experts for the United Nations Environment Programme and World Health Organization. Edited by Åke Bergman, Jerrold J. Heindel, Susan Jobling, Karen A. Kidd and R. Thomas Zoeller. 289 pp.

Wu JP, Luo XJ, Zhang Y, Luo Y, Chen SJ, Mai BX, Yang ZY. Bioaccumulation of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) in wild aquatic species from an electronic waste (e-waste) recycling site in South China. Environ Int. 2008 Nov;34(8).1109-13.

Wu J, Luo X, Zhang Y, Chen S, Mai B. Guan Y, Yang, Z (2009 a). Residues of polybrominated diphenyl ethers in Frogs (Rana limnocharis) from a contaminated site, South China. Tissue distribution, biomagnification, and maternal transfer. Environ. Sci. Technol. 2009, 43, 5212–5217.

Wu JP, Luo XJ, et al. (2009) b. "Biomagnification of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls in a highly contaminated freshwater food web from South China." Environmental Pollution 157(3). 904-909.

Wu K, Xu X, Liu J, Guo Y, Li Y, Huo X. (2010). Polybrominated diphenyl ethers in umbilical cord blood and relevant factors in neonates from Guiyu, China. Environ. Sci. Technol 2010;44.813–819.

Xia J, Wang L, Luo H 2005. Present status and developing tendency of flame retardant. Appl. Chem. Ind. 34, 1–4.

Xiang CH, Luo XJ, et al. (2007). Polybrominated diphenyl ethers in biota and sediments of the Pearl River Estuary, South China. Environ Toxicol Chem 26(4). 616-623.

Xie X, Wu Y, et al. (2011). Hydroxyl radical generation and oxidative stress in earthworms (Eisenia fetida) exposed to decabromodiphenyl ether (BDE-209). Ecotoxicology 20(5). 993-999.

Xie X, Qian Y, et al. (2013). Effects of decabromodiphenyl ether (BDE-209) on the avoidance response, survival, growth and reproduction of earthworms (Eisenia fetida). Ecotoxicol Environ Saf 8(12). 00462-00469.

Yu LH, Luo XJ, Wu JP, Liu LY, Song J, Sun QH, Zhang XL, Chen D, Mai BX.(2011 a). Biomagnification of higher brominated PBDE congeners in an urban terrestrial food web in north China based on field observation of prey deliveries. Environ Sci Technol. 2011 Jun 15;45(12).5125-31.

Yu Y, Zhang S, Huang N, Li J, Pang Y, Zhang X, Yu Z, Xu Z. (2012). Polybrominated diphenyl ethers and polychlorinated biphenyls in freshwater fish from Taihu Lake, China. Their levels and the factors that influence biomahnification. Environmental Toxicology and Chemistry, Vol. 31, No. 3, pp. 542e, China.

Yi W, Kun Z, et al. (2013). "Distribution is a Major Factor Affecting Bioaccumulation of Decabrominated Diphenyl Ether. Chinese Sturgeon (Acipenser sinensis) as an Example." Environ Sci Technol.

Zhang Y, Wu JP, et al. (2011) a "Biota-sediment accumulation factors for Dechlorane Plus in bottom fish from an electronic waste recycling site, South China." Environment International 37(8). 1357-1361.

Zhang K, Wan Y, et al. (2010). "Tissue concentrations of polybrominated compounds in Chinese sturgeon (Acipenser sinensis). origin, hepatic sequestration, and maternal transfer." Environ Sci Technol 44(15). 5781-5786.

Zhang W, Cai Y, Sheng G, Chen D, Fu J. (2011 c). Tissue distribution of decabrominated diphenyl ether (BDE-209) and its metabolites in sucking rat pups after prenatal and/or postnatal exposure. Toxicology. 2011 Apr 28;283(1).49-54.

Zhang W, Zhang M, et al. (2012). The combined effect of decabromodiphenyl ether (BDE-209) and copper (Cu) on soil enzyme activities and microbial community structure. Environmental Toxicology and Pharmacology 34(2). 358-369.

Zhou J, Chen DJ, et al. (2006). Impact of PBDE-209 exposure during pregnancy and lactation on immune function of offspring rats. Nan Fang Yi Ke Da Xue Xue Bao 26(6). 738-741.

Zhu W, Liu L, et al. (2010). Effect of decabromodiphenyl ether (BDE 209) on soil microbial activity and bacterial community composition. World Journal of Microbiology and Biotechnology 26(10). 1891-1899.

Zou M, Ran Y, Gong J 2007. Polybrominated diphenyl ethers in watershed soils of the Pearl river delta, China. occurrence, inventory, and fate. Environ. Sci. Technol. 41, 8262–8267.