SUMMARY OF COMMENTS RECEIVED ON DRAFT ASSESSMENT PUBLISHED MAY 8, 2004

POLYBROMINATED DIPHENYL ETHERS (PBDEs)

Comments on the CEPA DSL Draft Screening Assessment Report on PBDEs were provided by:

1. Northwest Environment Watch (NEW).
2. Dept. of Geography, University of Toronto.
3. Learning Disabilities Association of Canada.
4. Sierra Legal Defence Fund (SLDF).
5. Bromine Science and Environmental Forum (BSEF). Summary: Industry comments pertaining to Decabromodiphenyl ether (CAS# 1163-19-5) on the 2004 PBDE Screening Assessment Reports by Environment and Heath Canada.
6. BSEF. Industry Comments Pertaining to Decabromodiphenyl ether (CAS# 1163-19-5), January 2004 PBDEs Screening Assessment Report Prepared by Environment Canada.
7. BSEF. Industry Comments Pertaining to Decabromodiphenyl ether (CAS# 1163-19-5), January 2004 PBDEs Screening Assessment Report Prepared by Health Canada.
8. BSEF.
9. Canadian Plastics Industry Association.

Comment[(source)]	**Response**
Support the recommendation proposed in Environment Canada's draft scientific assessment to consider some forms of PBDEs "toxic" as defined by CEPA[1,3,4], including the recommendation that consideration be given to adding tetra-hexaBDEs to the Virtual Elimination List under CEPA[1,4]. Support the recommendation that PBDEs (tetra – decaBDEs) be considered as Track 1 substances under the Toxic Substances Management Policy.[1]	Recommendation will be based on final conclusion of the Screening Assessment. All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.
Health Canada and Environment Canada should consider adding decaBDE to the Virtual Elimination List under CEPA 1999. NEW notes that this is supported by regular decaBDE detection in human breast milk from North American mothers, and evidence of transformation into the lower-brominated and potentially more toxic forms of PBDEs. [1]	Recommendation will be based on final conclusion of the Screening Assessment Reports. All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.
Health Canada and Environment Canada should consider a systematic and broad monitoring program which could help determine whether human levels decrease upon the withdrawal of penta- and octaBDE. It could help identify how PBDEs make their way into human bodies. Recent studies have revealed high levels of PBDEs – particularly decaBDE – in household dust, suggesting that this pathway may be important to monitor. [1]	All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.

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Health Canada and Environment Canada should consider a strategy for removing "PBDE-laden" products already in use, including a strategy for safely disposing of such products at the end of their lifecycles, and if deemed prudent, for speeding the removal such products from homes and offices. [1]	All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.
"…an aggressive interim plan is necessary to halt the ongoing contamination of our environment with PBDEs. We therefore ask EC to use its powers under s.94 of CEPA to order an immediate, albeit interim, phase out and ban for the use of PBDEs in all consumer products pending final regulation…We anticipate that such a Ministerial order would include a ban [on] the import into Canada of products containing PBDEs."[4]	All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.
"we…recommend that…the government of Canada assist in the effort to find safe alternatives to PBDEs." [4]	All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.
"…[i]t is implied that any [risk management] actions [focused on PBDEs] would be based on the[ir] environmental effects of PBDEs and not the human health effects, which in actual fact have more scientific, political, and regulatory urgency for Canadians." [3]	Risk Management Response: All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.
"DBDPO does not qualify under CEPA 1999 as a substance for categorization and thus should not have been subjected to a screening assessment. DBDPO does not meet the criteria for categorization. DBDPO does not qualify as GPE and it does not meet the definition of inherently toxic to humans or non-human organisms."[5,6,7]	Section 68 of CEPA 1999 provides authority for the Ministers to undertake an assessment of any substance to determine, among other issues, whether it may be toxic.
*"Substances that are Persistent or Bioaccumulative **and** Inherently Toxic to Non-Human Organisms.* DBDPO is persistent; however, it is not bioaccumulative and is not inherently toxic to non-human organisms. DBDPO is a large, poorly absorbed, rapidly eliminated molecule. Its fish bioconcentration factor is <50 (CITI 1992). Oral feeding studies in fish show 0.005% uptake of a 7.5 or 10 mg/kg/d dose (Kierkegaard et al. 1999) and 0.4% absorption of a dose of 940 ng/fish/d (Stapleton et al. 2003). No toxicity has been demonstrated in acute studies in fish, algae and daphnia (Hardy 2002; VCCEP 2002), and chronic toxicity is not anticipated due to its low water solubility (EPA PBT Profiler) and lack of toxicity in a chronic daphnia study with the OctaBDE product (EU 2004). Studies in sediment organisms, terrestrial plants, and soil organisms show no evidence of toxicity."[5,6,7] "See table 2 for summary of DBDPO's environmental toxicology." [6,7]	The PBDE Environmental Screening Assessment has reviewed and acknowledged these data provided by the reviewer.
"DBDPO's properties are clearly different from the penta- and octabromodiphenyl ether products and thus should not have been included in an assessment on those two products." [5,7]	In this screening assessment, PBDEs were assessed as a group, but consideration was also given to the commercial products and the individual congeners. A class assessment for PBDEs was conducted for the following reasons: - their constituents have an identical base structure.

Comment[(source)]	Response
	- as commercial mixtures, there is overlap with respect to their constituents. Commercial pentabromodiphenyl ether is predominantly a mixture of penta-, tetra-, and hexaBDEs. Commercial octabromodiphenyl ether contains mainly hepta-, octa-, and hexaBDE, but may also contain small amounts of nona- and decaBDE. Commercial decabromodiphenyl ether is composed mainly of decaBDE but also has a small amount of nonaBDE. - there is evidence suggesting that under some circumstances higher brominated DEs may debrominate to form lower brominated DEs in the environment and in biota. - their physical and chemical properties are not all together dissimilar, in fact, consideration of these compounds as a group is supported by trends in physical/chemical properties associated with the degree of bromination. - historically, these products have been used concurrently, are co-occurring in the environment and any environmental risk would result from their combined presence in the environment. - they are all used as additive flame retardants.
"…there is no "weight of evidence" which indicates DBDPO breaks down in the environment to lower brominated diphenyl ethers of concern…"[5]	Laboratory studies are reviewed in the Supporting Working Document and summarized in the Environmental Screening Assessment report which show that DBDE may transform to lower brominated DEs (e.g., penta- and hexaBDEs) which may be bioaccumulative and persistent by abiotic and biotic transformation. It is reasonable to conclude from these studies that some level of decaBDE debromination may be occurring in the environment.
"Environment Canada's focus in the screening assessment report on DBDPO's potential for environmental degradation is not supported by the research data or by DBDPO's predicted environmental partitioning. Environment Canada's claim of anaerobic degradation appears to be unsubstantiated – to our knowledge no anaerobic degradation of DBDPO in sediment has been observed." [5]	The screening assessment examines the physical and chemical properties of decaBDE and notes in the Supporting Working Document that the majority of DBDE released into the environment is predicted to partition to soil and sediment, with very little (approximately 1%) predicted to partition to air and water. Section 2.3.5.2.1 reviews research which shows that DBDE adsorbed to sand, soil or sediment particulates, or precipitated onto a hydrated quartz surface, can undergo a some degree of transformation to lower brominated DEs and to brominated dibenzofurans when irradiated with UV light or sunlight. Since the comments were composed, a study by Gerecke et al. (2005) has found that decaBDE is susceptible to some slow biodegradation under specific anaerobic conditions using sludge inoculum.
"Environment Canada focused heavily on laboratory studies regarding the photolysis of DBPDO in organic solvents. While this may show that photolysis can theoretically occur, this scenario is not applicable to the environment, particularly since photolysis routes are expected to be different in organic or aqueous matrixes. DBDPO in the environment will partition primarily to sediment and secondarily soil, and will bind to particulate matter. DBDPO will not be dissolved or suspended in organic solvents in these matrixes. Photolysis will not occur at substantial rates in sediments or soils.	Photolytic transformation of decaBDE may occur in both organic and aqueous media. Based on the available studies, it is reasonable to expect that decaBDE will undergo some level of photodegradation in the environment. The available literature indicates that lesser brominated DEs, PBDFs and other unidentified substances could be formed. Studies cited in the Supporting Working Document and summarized in the Environmental Screening Assessment Report describe photolytic debromination of DBDE adsorbed to sand, sediment and soil particulates. Reductive debromination is reported, with formation of nona- to hexaBDEs, as well as tetra- and pentaBDFs.

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Binding to particulates will slow photolysis. Photolysis is most likely to occur in air or water; however, DBDPO concentrations will be extremely limited in these media and binding to particulates will slow any reactions." [5] "The only evidence for degradation comes from laboratory photolysis studies that show a small fraction may undergo photolysis. This, coupled with the fact that Decabromodiphenyl ether/oxide partitions only minimally to air, indicates photolysis is not a significant degradation pathway for the substance." [5]	In one study, DBDE is precipitated onto a quartz surface, hydrated, and irradiated with UV light or sunlight. Small amounts of nona- and octaBDE are reported as degradation by-products. The screening assessment agrees that decaBDE in the environment will partition primarily to sediment and soil. Thus, it is likely that only a comparatively small proportion of decaBDE entering the environment would be susceptible to photodegradation. In the atmosphere, decaBDE would be mainly found sorbed to particulate matter. The rate of decaBDE degradation, primarily due to reaction with atmospheric hydroxyl radicals, is predicted to be slow. However, the available literature clearly shows that some level of decaBDE phototransformation is likely to occur in the environment and that lower brominated DEs and dibenzofurans, as well as unidentified products can be formed. These products maybe more bioaccumulative and toxic than their parent compound, and thus could present a risk to organisms. Therefore, while it is difficult to evaluate the significance of photodegradative processes in the overall fate of decaBDE in the environment, their potential impact on organisms is an environmental concern.
"Laboratory studies show Decabromodiphenyl ether/oxide is not degraded in sediments (Schaeffer and Flaggs 2001; de Wit 2000), which is where it primarily resides the environment (VCCEP 2002)." [5]	The screening assessment does not dispute these findings. Since the comments were composed, a study by Gerecke et al. (2005) has found that, decaBDE is susceptible to some slow biodegradation under specific anaerobic conditions using sludge inoculum.
"Sediment monitoring indicates that the lower brominated diphenyl ethers detected were derived from releases of the commercial pentaBDE or OctaBDE products, and not from Decabromodiphenyl ether/oxide (Rayne and Ikonmou 200[2], Song et al.2004)." [5]	While studies such as those of Rayne and Ikonomou (2002) and Song et al. (2004) provide evidence that the commercial PeBDE and OBDE formulations form an important, if not dominant, source of lower BDEs to the environment, their results do not preclude potentially significant contributions from DBDE. Rayne and Ikonomou (2002) placed semipermeable membrane devices in the Fraser River, BC and analyzed the resultant SPMD samples for 36 PBDEs (mono- to hexa-congeners). Their study did not involve the sampling of sediments. They found that the congener patterns observed in the SPMD samples differed significantly from those of the commercial PeBDE and OBDE mixtures. They then applied modeling and calculation procedures and found that the reconstructed congener patterns more closely approximated those of the technical mixtures. Much of the data on PBDEs in sediments focuses on the presence of the lower congeners, and in many studies, analyses were not conducted for the higher brominated, octa- to deca- BDEs. There is evidence indicating that these higher brominated PBDE congeners may be present in sediments

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	at high concentrations. In a sediment monitoring survey conducted in the UK, the highest survey levels of octaBDE were measured in sediments collected downstream from a warehouse where DBDE was stored (Allchin et al. 1999, Law et al. 1996, Environment Agency 1997 all cited in European Communities 2002). La Guardia (2001) detected high levels of nona- and decaBDE in samples of sewage sludge collected from various locations in Canada and the United States. The occurrence of high levels of nonaBDE in the samples (up to 7190µg/kg dw) is significant as the proportion present in the samples far exceeds that reported for commercial formulations. Open literature sources note that PBDE congener patterns found in the environment closely resemble those of the PeBDE and OBDE commercial products, and thus, some researchers (e.g., Rayne and Ikonomou (2002) and Song et al. (2004)) have concluded that the main sources of PBDEs in the environment are the commercial products, but they also qualify their findings to indicate that they are suggestive rather than completely conclusive. Söderström et al. (2004) also concludes that the main source of the lower brominated DEs (i.e., BDE 47, 154 and 183) found in the environment is likely predominated by emissions from the commercial PeBDE and OBDE mixtures. In their studies they note that the most commonly found PBDEs in environmental samples (BDE 47, 99 and 100) were only formed to a minor degree during their photolysis studies. However, it should be noted that most monitoring studies to date have investigated the occurrence of the main components of the commercial products and these are found to dominate the PBDEs reported in the environmental samples. Analytical standards are not available for all congeners, and thus, studies have so far not necessarily investigated the presence of all possible transformation products of decaBDE in the environment. The overall contributions of DBDE to the overall risk presented by the lower congeners is presently not known.
"DBDPO is used solely as a fire retardant to prevent ignition of flammable products or, in the event ignition occurs, to slow down the fire's growth. In any assessment of DBDPO, the reduction in fire risk to life, limb and property should be given appropriate weight." [5] "DBDPO is necessary to save human lives...In the applications in which DBDPO is used, an estimated 280 lives are saved each year in the U.S. through the use of a brominated flame retardant….children are especially vulnerable to fire deaths and injuries." [5] "The protection provided by DBDPO in terms of enhanced fire safety reduces the very real risk of death or injury that consumers face in the home from fires." [7]	All stakeholders and experts will be consulted during the risk management phase to determine the appropriate control or preventive action under CEPA 1999.

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"The benefits derived from the use of DBDPO in consumer products, particularly for children, far outweigh the insignificant potential for harm." [5, 7]	
"DBDPO…does not meet the screening assessment report's definition of 'toxic' or 'capable of becoming toxic'…Studies have shown that DBDPO is not acutely or chronically toxic to fish, algae, daphnia, sediment organisms, soil organisms or terrestrial plants. Thus, it does not have immediate or long-term harmful effects. It does not affect the ozone layer or other processes on which life depends and therefore does not meet the requirement under b). DBDPO is a large poorly absorbed molecule whose NOAEL in repeated dose studies is at least 1000 mg/kg/d (Hardy 2002), and which in Canada is primarily imported in finished articles. Thus, it does not constitute a danger in Canada to human life or health. The weight of evidence approach for screening evaluations specified in CEPA 1999 supports these conclusions." [6]	PBDEs, including decaBDE, have been assessed as a class (see above). There is evidence to suggest that decaBDE can degrade in the environment to form less brominated forms, such as penta- and hexaBDEs congeners and PBDFs, which may be more toxic and bioaccumulative than decaBDE. Although the database describing decaBDE degradation rates and products is still limited, sufficient information exists to warrant concern. DBDE has become the prevalent commercial PBDE product used in North America and the world. In North America and Europe, it is often found in concentrations which exceed those of other PBDEs in sewage sludge and sediments. Concentrations of DBDE are now exceeding mg/kg dw levels in North American sewage sludge. Increasing accumulations of DBDE in the environment have uncertain environmental implications. Evidence of debromination has led researchers to note that even slight and very long term transformation to lower brominated diphenyl ethers could have serious ecological consequences over periods spanning several decades. Some literature also indicates that DBDE will transform by aerobic biodegradation, metabolically induced transformation and reactions with reducing agents. While it has been shown that lower BDEs can be formed by these processes, other products may also be formed. It is not known whether debromination or other unknown pathway(s) of transformation will result in environmental impacts. Thus, while current concentrations measured in the environment for homologues found in commercial DBDE do not appear to exceed the known effects thresholds, the persistence of these homologues and their potential to transform to possibly more toxic and bioaccumulative forms in the environment warrants concern. The evidence provided in the Supporting Working Document and Environmental Screening Assessment is consistent with Subsection 64(a) of CEPA 1999, "… it is entering or may enter the environment in a quantity or concentration or under conditions that…have or may have an immediate or long-term harmful effect on the environment or its biological diversity…".
"Page 52. Many of the trade names listed are incorrect…Those no longer produced, include but are not limited to Berkflam, Bromkal and Adine. PBDE 209 is not a recognized trade name. Saytex 120E is not a trade name for DBDPO. Saytex 120 is an entirely different product, and no 'Saytex 120E' product exists." [6]	The identified erroneous trade names have been removed from the Supporting Working Document. Although the manufacture of some products may have been discontinued, it is relevant to list their names in addition to those currently manufactured since the open literature may make reference to these products.

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"Page 53; Table 2.6. We believe DBDPO's physical/chemical properties and environmental fate parameters are best represented in Table 3." [6]	The values described in Table 2.6 of the Supporting Working Document are consistent with those of the referenced Table 3. Table 2.6 provides measured physical and chemical parameters for DBDE; model-derived estimates (such as those obtained using EPIWIN) are summarized in Appendix B of the Supporting Working Document. Table 3 reports a log Kow of 5.625 derived by MacGregor and Nixon (1997). Our copy of the same study indicates that the measured log Kow is 6.265 (this value is reported in the environmental screening assessment) Partitioning to water, air, soil and sediment was estimated in the PBDE screening assessment using Level III Fugacity Modeling. The findings are consistent with those identified in Table 3. Table 3 indicates that DBDE is "[n]ot expected to undergo long range transport (Wania and Dugani 2002). There is no reference for this citation in the Reference List appended to the received comments. The environmental screening assessment notes that Dugani and Wania (2002) found that PBDE congeners with four to six bromine atoms have a higher long-range transport potential than the lower or higher brominated congeners. Based on this, the potential for long- range atmospheric transport of decaBDE is likely to be much lower than that of the tetra- to hexaBDEs. However, low concentrations of decaBDE have been measured in sediments collected from a remote Arctic lake (Muir et al. 2003) and this suggests that the substance is capable of being transported long distances to remote locations. Thus, evidence supports the potential long-range transport of decaBDE (see Section 2.3.6.3 of the Supporting Working Document). The degradation studies reported for ready biodegradation, anaerobic sediment degradation, and photodegradation (aqueous, solvent and solid surface) in Table 3 are currently reviewed in the Supporting Working Document. Hydrolysis is not a significant route of degradation due to the low water solubility of decaBDE, and thus, hydrolysis is not discussed in the Supporting Working Document or in the environmental screening assessment. Table 3 reports a predicted atmospheric oxidation half-life for DBDE of 169 days using EPIWIN V3.04. This is consistent with predictions by AOPWIN of 161 and 318 d for nonaBDE and decaBDE, respectively, reported in Section 2.3.5.2.1 of the Supporting Working Document. Biological endpoints relevant to wastewater treatment were not subject to the screening assessment of PBDEs since the focus of the assessment is ecological endpoint rather than those

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	relevant to sludge respiration. A brief discussion of fate in a sewage treatment plant was added to the Supporting Working Document.
"Page 56. 2.3.5.1. Environmental Partitioning. DBDPO's log Kow has been measured (Log Kow = 5.67) (MacGregor and Nixon 1997). It's estimated Log Kow, using only its chemical structure to remove variation due to analytical methodology, is 12.6 (EPIwin)." [6]	The study by MacGregor and Nixon (1997) reported a log Kow of 6.265. This value is reported in the Supporting Working Document and in the environmental screening assessment. The estimated log Kow of 12.6 obtained using EPIWIN is within the range of predicted values summarized in Appendix B of the Supporting Working Document.
"Pg 56. 2.3.5.1 Abiotic Degradation. The final paragraph implies all lower brominated diphenyl ethers are known to bioaccumulate. This is incorrect…Also, this section presumes that if DBDPO degrades abiotically, it will do so only to lower brominated diphenyl ethers. This is an erroneous assumption as laboratory studies have demonstrated that any breakdown products are typically NOT lower brominated diphenyl ethers (Zetsch 2003; Jafvert and Hua 2001)." [6]	Further discussion of the uncertainties surrounding DBDE transformation has been added to Section 2.3.5.1 of the Supporting Working Document. The available literature provides strong evidence for the transformation of decaBDE in the environment, with possible formation of lower brominated DEs and dibenzofurans. BDE 153 has been positively identified as a transformation product of decaBDE, and other unidentified PBDE congeners with 5 and 6 bromine atoms per molecule have also been identified. Based on the observed trends in physical and chemical properties of PBDEs, it is reasonable to expect that these unidentified penta- and hexaBDEs will have higher bioaccumulation potential than decaBDE. The photodegradation study of Jafvert and Hua (2001) has been reported in the Supporting Working Document and in the Environmental Screeening Assessment Report. The assessment has been modified to include greater detail on this study, particularly with regard to the formation of breakdown products.
"Page 57. The third paragraph discusses Soderstrom et al. (2003). It should be noted that the earlier version of this paper (Sellstrom et al. 1998) initiated discussion regarding the potential for DBDPO to degrade to lower brominated diphenyl ethers. Upon completion of the work and full publication, these same authors conclude that DBDPO is not the source of tetra and pentaBDE congeners in the environment."[6]	The Supporting Working Document has been edited to note that the researchers conclude that the origin of BDE 47, 99, 100 and 183 in the environment is "probably primarily from emissions of technical PentaBDE products and possibly from other degradation pathways of decaBDE. To further investigate the degradation pathways of decaBDE, combined photolytic/bacterial degradation pathways should be examined."
"Page 59 & 60. 2.3.5.3 Bioaccumulation. Examination of the CITI 1992 reference indicates that the DBDPO fish bioconcentration study was conducted for such time so as to reach equilibrium. The final sentence in the first paragraph should be deleted."[6]	Statement has been deleted.
"The paragraphs reporting Stapleton et al (2003) should emphasize that DBDPO's bioavailability was estimated to be only 0.4% of the dose. Carp had no detectable DBDPO after consuming treated food for 90 days at a dose of 940 ng/fish/d. The estimated bioavailability (0.4% of the dose) was based on detection of presumed metabolites. The presumed metabolites were not the lower brominated diphenyl ethers typically detected in wild-caught fish (e.g. BDE 47, 99, 100). What hazard/risk is Environment Canada predicting based on 0.4% absorption? Also note that Stapleton did	The Supporting Working Document states that decaBDE was not detected in the whole fish tissues during the exposure and depuration periods of the study. The researchers also did not detect BDE 47 or BDE 99 in the fish tissues, but did measure the accumulation of penta- and hexaBDEs. Notably, BDE 154 and BDE 155 were positively identified. BDE 154 is often detected in wild fish and other biota at elevated concentrations and is sometimes the third most dominant PBDE congener in body burden (Stapleton et al. 2004, Manchester-Neesvig et al. 2001 and Lepom 2002). BDE 155 is also often found in the tissues of wildlife. See Appendix C, Table C.6 in the

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not provide the purity of the test article utilized in her experiment nor could she determine whether any "transformation" of the test article occurred on the food, in the water or in the fish's digestive tract. Based on NTP (1986) and El Dareer (1987), it appears that metabolism after ingestion would most likely occur in the gut as a result of intestinal bacterial action. Any such metabolism, however, is only a small fraction of the total dose."[6]	Supporting Working Document for further information on PBDE accumulations in biota. The risk/hazard presented by the 0.44% BDE 209 adsorption, determined based on the presence of possible metabolites, was not a subject of this assessment. However, concentrations of BDE 209 in sediments are reaching very high levels and therefore even comparatively small accumulation rates could potentially result in significant uptake of the substance, particularly by bottom-feeding fish such as carp. Stapleton et al. (2004) describe the purity of the decaBDE used in the experiment as >98%. No information is provided on the identities of any impurities present. The material used was a chemical standard and not the commercial product, which is known to contain lesser brominated diphenyl ethers as impurities. The authors conducted a simple mass-balance calculation to show that the peaks observed in the chromatograms did not result from selective accumulation of minor impurities in the exposure food. The researchers illustrated this point using the example of BDE 154. They calculated the maximum amount of BDE 154 that would be accumulated in the fish assuming that it was present in the food at the analytical detection limit concentration (0.03 ng/g ww). If BDE 154 was present in the spiked food fed to carp, the maximum mass of this substance would be approximately 2 ng. Since they found that BDE 154 concentrations were as high as 35 ng after 60 d of exposure, they concluded that the peak could not have resulted from the selective accumulation of impurities in the spiked food pellets. Stapleton and Baker (2003) have also suggested that debromination may have occurred as a result of intestinal bacteria (see Section 2.1.5.3 and 2.3.5.3 in the Supporting Working Document). While some transformation may take place via this route, the researchers considered the evidence from their studies sufficient to conclude that metabolic debromination of BDE 209 by the carp was also occurring and that this process led to the accumulation of lower brominated BDE metabolites in the tissues of the organism.
"Page 61 presents data by Morck et al. (2003), but neglects to mention the impact of the specialized formulation on absorption. Please see the attached: "Relevance: DBDPO Absorption And Metabolism Reported By Morck Et Al. 2003 And Sandholm Et Al. 2003". NTP (1986) and El Dareer et al (1987) demonstrated decades ago that DBDPO could be metabolized if it was injected directly into the blood stream."[6]	The wording in the Supporting Working Document has been adjusted to emphasize that the aim of the study was to examine metabolic pathways and products rather than to measure accumulation.
"The final paragraph on page 61 under Bioaccumulation states that detection of DBDPO in falcons contradicts the belief that the DBDPO molecule is too large to be bioavailable,	The following edited text has been added to the Supporting Working Document: "DBDE was recently detected in 18 of 21 peregrine falcon, *Falco peregrinus*, eggs at concentrations ranging

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'and therefore cannot accumulate in living organisms'… However, research dating to the mid-1970s has shown that DBDPO is poorly bioavailable, but that its bioavailability is not zero (Norris et al.1973, 1974, 1975; NTP 1986, El Dareer et al 1987). Its low bioavailability is very likely related to its large molecular size (10 bulky bromine atoms) and high molecular weight (959.7)… Studies performed in fish also indicate poor bioavailability. Uptake in trout after consuming treated food for 120 days was estimated to be 0.005% of the 7.5 or 10 mg/kg/d dose (Kierkegaard et al. 1999). Carp had no detectable DBDPO after consuming treated food for 90 days at a dose of 940 ng/fish/d, with an estimated bioavailability of 0.4% of the dose based on detection of presumed metabolites (Stapleton et al. 2003). The presumed metabolites were not the lower brominated diphenyl ethers typically detected in wild-caught fish (BDE 47, 99, 100). It is difficult to conceive that a substance with an absorption of 0.005-0.4% presents a hazard, much less risk, either from some inherent property or from formation of metabolites." [6] "…Mere detection of a substance does not indicate bioaccumulation. This is particularly true with DBDPO, which is very difficult to analyze, let alone quantitate. The DBDPO concentrations infrequently detected in wild-caught specimen are at typically so low as to be at the limit of quantitation. Is Environment Canada convinced that the analytical methods used were actually specific for DBDPO? See the first paragraph on page 62 where the difficulties encountered in analyzing DBDPO are related." [6]	from 28 to 430 µg/kg lipid weight (see Section 2.3.6.6). The findings represent the first reporting of DBDE in the eggs of a top predator avian species, and provide further evidence that the substance has some bioavailability despite its large molecular size and weight. Detection of DBDE in wildlife suggests that the substance is present in the environment at concentrations or exposure frequencies which exceed the capacity of organisms to metabolize and/or eliminate it."
"Page 61. 2.3.5.4. Formation of Brominated Dibenzo-p-dioxins and Dibenzofurans. Formation of PBDFs (only) on photolysis has only been observed when DBDPO is suspended in organic solvents. This is not relevant to the environment." [6]	Studies (e.g., Söderström et al. 2004, Palm et al. 2004) have determined that PBDFs are formed as transformation products of decaBDE in aqueous solution.
"Page 62. 2.3.6.1 Atmosphere. This section cites Leisewitz et al. (2001) as reporting DBDPO air concentrations outdoors "in the vicinity of a production site". The reference is from a German governmental agency; however, DBDPO is not manufactured in Germany."[6]	Leisewitz et al. (2001) does not cite the source for these data and provides no details on the nature of the production facility. Wording in the Supporting Working Document has been adjusted to indicate that the location of the site, type of production facility and sampling year were not provided.
"Page 64. 2.3.6.5 Waste Effluent and Biosolids. This section reports detection of DBDPO in sewage sludge, but fails to note that DBDPO's settling and binding to sludge is predicted in environmental fate models. EPIwin predicts that ~94% of DBDPO in a wastewater treatment plant influent will be removed via settling to sludge. Detection of DBDPO in sludge of treatment plants with influents containing DBDPO should not be unexpected. Indeed, this indicates the treatment plant is performing the job it was intended to do – remove substances from the waste stream."[6]	The section has been edited to consider DBDE's fate (94% adsorption to and removal by sludge) as predicted by EPIWIN Ver. 3.10.

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"Page 66. 2.3.6.6 Biota. We recommend deleting the reference to Rice et al. (2002). The explanation given in the screening assessment does not adequately relate Rice's conclusions regarding the origin of the BDE 181, 183 and 190 congeners. Rice et al. states that the 'likeliest explanation' *would be discharges from manufacturing facilities that use products containing these congeners or discharges from a waste facility that may be leaking these congeners (emphasis added).* Rice et al. then goes on to discuss how these congeners are found in the OctaBDE product. Speculation re degradation of DBDPO as a source is only mentioned in passing and then in the context that others have speculated to that effect. "[6]	The reference to Rice et al. (2002) has been deleted in Section 2.3.6.6 since the subject study could not confirm that BDE 209 was ingested by the organisms. However, reference to this study is retained in Section 2.2.6.6 where text has also been edited to indicate that the possible sources for the other heptaBDEs, BDE 181 and BDE 190, found in the carp in the Des Plaines River are not obvious, especially since no commercial products have been documented as containing major quantities of these congeners. The researchers speculate that the significant presence of heptaBDE in the Des Plaines River fish may have resulted from active metabolism of BDE209 present in the river sediment (although this was not confirmed). They also suggest that differing contributions to the two river systems from municipal wastewater treatment facilities could have played a role (effluent sources were not sampled to confirm this hypothesis).
"The last paragraph under the Biota section mentions detection of DBDPO in peregrine falcon eggs as reported by Sellstrom. Sellstrom's finding is inconsistent with a larger monitoring study reported by de Boer et al. (2003). In that work, approximately 67% of the bird tissues analyzed contained no detectable DBDPO. Bird tissues were collected throughout the UK, with additional samples collected in the Netherlands. Both the UK and the Netherlands currently utilize DBDPO in various industrial processes." [6]	Findings first described in Sellström et al. (2001) and Lindberg et al. (2003) have now been published as Lindberg et al. (2004). The references in the Supporting Working Document have been updated to reflect this. A description of results from the de Boer et al. (2003) study have also been added to the text and to Appendix C of the Supporting Working Document.
"Page 67. 2.3.7.1 Pelagic Organisms. Under the discussion of Kierkegaard et al. (1999), the draft screening assessment states that the liver-to-body index and plasma lactate concentrations were higher in fish fed DBDPO-treated food, and that this suggests "delayed chronic effects". Please note that studies in other species (rat) have shown an increase in liver weight after prolonged dosing with DBDPO (e.g. NTP 90 day study). However, no adverse effects were noted on histopathology or with respect to hepatic enzymes. An increase in liver weight that is not accompanied by pathology or functional aberration is not indicative of an adverse effect. Further, NTP's lifetime studies in rats and mice at extremely high doses (up to 5% of the diet) do not suggest the existence of "delayed chronic effects". Likewise, an increase in plasma lactate is not indicative of an adverse effect. Environment Canada should also consider that only 0.005% of the DBDPO was absorbed – thus it is highly unlikely that this small amount could result in any adverse effect ether from the parent compound or any metabolites. We believe Environment Canada's conclusion of a suggestion of 'delayed chronic effects' is faulty and was reached because only a screening assessment of DBDPO's toxicology database was performed. We believe it should be recognized that the so-called 'metabolites' of DBPDO reported in Kierkegaard et al. could very likely have been present in the test article at the study's initiation, and thus are not 'metabolites' at all." [6]	Kierkegaard et al. speculated that effects observed in treated fish following a 71-d depuration period might indicate delayed chronic effects. There is no evidence that further research aimed at clarifying this issue was conducted; however the study is of acceptable quality and the results are considered valid. Text in the Supporting Working Document describing the Kierkegaard study has been adjusted to emphasize that the supposition of delayed chronic effects originates with the researchers. As well, a comment made by European Communities (2002) that the observed liver effects may have been related to a build up of the more toxic lower brominated congeners present in the commercial formulation used has been added to Supporting Working Document.

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"Page 68. 2.3.7.4 Wildlife. Hardy 2002 and NTP 1986 should be cited as references for DBDPO studies performed in rodents. The citations used by the screening assessment report, Darnerud et al. and de Wit, merely review studies previously reported. Some of the information in those reviews is inaccurately reported."[6]	Reference to Darnerud et al. (2001) and de Wit (2002) removed from the first sentence of the section. The Supporting Working Document has been updated with references to the European Communities (2002, 2004) and Health Canada (2004). Hardy (2002) has also been added as a potential reference for follow-up by readers.
"Page 69. The discussion re the NTP two-year bioassay should relate that neither the NTP (2000), OSHA (1990) nor IARC (1990) consider DBDPO to be a human carcinogen."[6]	Carcinogenesis is not an effect which is reviewed in the environmental component of the screening assessment since it is deemed more relevant to human health.
"Page 70. Hardy et al. (2002) should be added to the discussion of the BFRIP study on prenatal developmental toxicity. The study results are not accurately reported in the draft screening assessment report. In that study, the NOEL for maternal and fetal effects was 1000 mg DBDPO/kg/d administered on gestation days 0-19."[6]	A reference to Hardy et al. (2002) has been added to the Supporting Working Document and the description of study results and endpoints has been clarified. Text in Supporting Working Document now reads: "BFRIP commissioned a study in which pregnant female rats received doses of 100, 300 or 1000 mg DBDE/kg bw by gavage on days 0 to 19 of gestation (Hardy et al. 2002). The test substance used had a purity of 97.34%. No clinical signs of toxicity were observed during the test, and no treatment-related effects were seen in maternal gestational parameters (body weight, body weight gain and food consumption), liver weights or necropsy findings. As well, no effect of treatment was seen in fetal body weights, fetal sex distribution, or from the fetal external, visceral or skeletal examinations. A NOEL for maternal and developmental toxicity of 1000 mg/kg/d was reported for the study by Hardy et al. (2002). However, the European Communities (2002) noted an apparent dose-dependent decrease in the percentage of viable fetuses per implant and an increase in post-implantation loss at the two highest doses. While the European Communities (2002) considered that the observed effects were not statistically or biologically significant, they were not taken into consideration for the development of a NOAEL."
"Page 71. We fail to see the relevance of the Olsman et al. (2002) study. This study photolyzed DBPDO in an organic solvent that is a recognized method to produce a small quantity of PBDFs. This is not a pathway relevant to the environment because DBDPO will not be present in an organic solvent in that matrix. Further, PBDFs are known to be subject to photolysis, and if formed, will rapidly degrade." [6]	Reference to Olsman et al. (2002) has been removed from the report. This is fundamentally a photodegradation study whose findings have been demonstrated by other researchers already in the Supporting Working Document. Section 2.3.5.2.1 demonstrates that photodegradation of decaBDE in organic solvents and on solid matrices such as sand, soil and sediment can result in the production of small amounts of polybrominated dibenzofurans, which are inherently dioxin-like in nature.
"In the last paragraph, we recommend replacing the Darnerud and de Wit references with Hardy et al. (2002), NAS (2000), and U.S. VCCEP (2002). The recommended references are more complete and thorough."[6]	The Supporting Working Document has been updated with references for European Communities (2004) and Health Canada (2004). These replace the older references identified in the draft document (i.e., WHO (1994), Darnerud et al. (2001) and de Wit (2002)). Reference has also been made to Hardy et al. (2002).
"Page 82. 3.3.1 Pelagic Organisms. This section concludes that based on Kiekegaard et al. (1999) and laboratory studies of 'photolysis and anaerobic degradation' that it cannot	The Supporting Working Document has been edited to recognize the uncertainties in relation to the Kierkegaard et al. (1999) study. Section 3.3.1 has been edited by adding reference to the carp

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be ruled out that DBDPO may be transformed to PBDE congeners which are bioaccumulative and toxic. In reaching this conclusion, Environment Canada makes several errors. Less than 0.005% of the DBDPO administered by Kierkegaard appeared to be absorbed over the course of the 120-day study. How does Environment Canada envision that such a slight absorption will result in conversion to enough 'metabolites' to induce toxicity? This conclusion also does not consider that the weight of the evidence from multiple repeated dose studies indicates DBDPO exerts essentially no effects at doses at least as high as 1000 mg/kg/d (Hardy 2002). Finally, the only known composition of the test article used by Kierkegaard et al., FR BA300, was composed of only 77% DBDPO. The remainder of FR BA300 consisted of lower brominated diphenyl ethers. Any effects seen or 'metabolites' detected could reasonably be expected to have originated in the test article. (NOTE: FR BA300 has not been commercially produced since at least the mid-1980s.)" [6] "Further, no evidence for anaerobic degradation has been reported in the draft screening assessment report; in fact, earlier sections of the report state that 32 week and 2 year anaerobic sediment studies failed to detect evidence of degradation. Finally, the laboratory photolysis studies cited in the draft assessment typically involved organic solvents that induce photolysis via a different pathway than that of the aqueous matrix found in the environment (Norris et al. 1973, 1974, 1975). Also overlooked is the fact that the screening assessment recognizes that DBDPO partitions primarily to sediment and soil, with negligible partitioning to air and water. Air and water are the environmental matrixes in which photolysis may occur. Thus, the weight of the evidence indicates that DBDPO does not transform into congeners that are bioaccumulative and toxic." [6]	study by Stapleton et al. (2004). The uncertainties relevant to the Kierkegaard et al. (1999) and Stapleton et al. (2004) studies have been further clarified in Section 2.3.5.3. The screening assessment concurs that anaerobic biodegradation of DBDE in sediments has not been observed. However, it notes that the European Communities (2002) reviewed various data for halogenated aromatic compounds (i.e., polybrominated biphenyls (PBBs) and polychlorinated biphenyls (PCBs)) subjected to anaerobic biodegradation and concluded that this process could result in reductive dehalogenation of DBDE. Studies cited in the Supporting Working Document indicate that photolytic debromination of DBDE can occur in an aqueous medium as well as in solvents and lower brominated DEs will be formed. It is reasonable to expect that these substances will be more bioaccumulative, and potentially more toxic than their parent compound. The Supporting Working Document also notes that there are other products formed when decaBDE is hydrated or in an aqueous solution (e.g., PBDFs and unidentified substances) (see Section 2.3.5.2.1 of Supporting Working Document). The screening assessment agrees that decaBDE in the environment will partition primarily to sediment and soil. Thus, it is likely that only a comparatively small proportion of decaBDE entering the environment would be susceptible to photodegradation.
"Page 82. 3.3.2 Benthic Organisms. The second paragraph of this section again refers to possible bioaccumulation of DBDPO metabolites and cites Kierkegaard et al. 1999, Watanabe and Tatsukawa 1987, and Norris et al. 1973 and 1974. As stated previously, Kierkegaard's results do not indicate a rational concern re metabolites. Watanabe and Tatsukawa reported on photolysis of DBDPO in organic solvents, this is not applicable to benthic organisms. The Norris et al. studies did not indicate formation of hexaBDE and pentaBDE congeners as a result of photolysis. This section requires revision. In reaching its conclusions, this section ignores the fact that DBDPO has been tested in benthic organisms and shows no effects at the unrealistically high concentration of 5000 mg/kg	See responses to previous comments. The second paragraph is edited to note that "… , it is reasonable to expect that a small amount of decaBDE may transform in the environment by various means to form potentially bioaccumulative forms of PBDEs such as hexaBDEs." As noted above, various studies show that a small amount of decaBDE could be reasonably expected to transform to lower brominated PBDEs like hexaBDE which may be bioaccumulative; however, there is insufficient data to quantify the environmental risk resulting from this process. Section 3.3.2 has considered the available studies showing that no effects have been documented

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dry wt (Kruger et al. 2001a,b). In the final paragraph of this section, Environment Canada again states that DBDPO might transform due to anaerobic degradation. As previously pointed out, earlier sections of the screening assessment correctly reported that no evidence of anaerobic degradation of DBDPO was observed in 32 week (Schaeffer and Flaggs 2001) and 2 year (de Wit 2000) studies. In addition, European sediments have been monitored over a 20-year period (de Boer 2001), and do not support degradation of DBDPO. Environment Canada also concludes that DBPDO might be metabolized by environmental organisms to toxic substances. In reaching this conclusion, Environment Canada fails to consider the weight of the evidence. DBDPO is a poorly absorbed molecule. Multiple repeated dose studies show DBDPO has a NOAEL of at least 1000 mg/kg/d. The doses administered in the studies were so high that ample opportunity was allowed for the production of toxic metabolites and their potential adverse effects. The fact that this did not occur argues that DBDPO is not transformed to metabolites of concern." [6]	at nominal concentrations of 5000 mg/kg dw since it uses a corresponding measured concentration (4536 mg/kg dw; ACCBFRIP 2001a, b) to derive the ENEV used in the quotient analysis. This section concludes that DBDE concentrations will not in themselves cause adverse effects on populations of benthic organisms.
"Page 83. 3.3.3 Soil Organisms. A statement regarding potential transformation of DBDPO to lower brominated congeners is repeated here. There is no evidence to support this – only speculation that it 'might' occur. It is equally as likely that DBPDO is stable (e.g. does not degrade) or that if it degrades, it degrades to some molecule that is not a lower brominated diphenyl ether of concern." [6]	As noted above, various studies show that a small amount of decaBDE is likely to transform to form products such as lower brominated PBDEs like hexaBDE which may be bioaccumulative; however, there is insufficient data to quantify the environmental risk resulting from this process. There is uncertainty regarding all pathways of DBDE transformation in the environment. There is a lack of knowledge over all formed chemical products and potential impacts.
"Page 83. 3.3.4 Wildlife. This section does not take into consideration that 67% of birds monitored in the UK, Netherlands and Sweden contained no detectable DBDPO (de Boer et al. 2003). The reference to the Norris et al. studies on page 84 is not applicable to today's DBDPO product. Those studies were preformed using a product with only 77% DBDPO, and their results cannot be applied to today's commercial DBDPO product (>= 97% DBDPO) or the DBPDO molecule itself. Instead, we recommend Environment Canada cite the 13-week studies performed by NTP in rats and mice (NTP 1986). These studies demonstrated that doses as high as 10%of the diet were without adverse effects." [6]	A description of the results obtained by de Boer et al. are provided in S.2.3.6.6 of the Supporting Working Document. However, an EEV of 430 µg/kg lw for peregrine falcon eggs (Lindberg et al. 2004) is deemed appropriate and no changes have been made. Eggs are a good medium to use for estimating an EEV as they represent potential food exposure to wildlife, and they also represent potential exposure to a very sensitive life stage, the developing embryo. The value selected is conservative while not being the highest concentration measured in biota. As well, decaBDE was present in 18 out of 21 samples analyzed, indicating a high incidence of contamination. Increased liver and thyroid weights were reported for Sprague Dawley rats administered 80 mg DBDE/kg bw/d in food over 30 days (Norris et al. 1974, 1975). It is noted that Norris et al. (1974, 1975) used Dow FR-300-BA in their studies, which is an older commercial DBDE formulation composed of 77.4% decaBDE, 21.8% nonaBDE and 0.8% octaBDE. This product is no longer produced and current formulations of DBDE are composed of a much higher proportion of decaBDE (e.g., usually > 97%). The value, 80 mg DBDE/kg bw/d in food is nevertheless

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	considered appropriate for use as a CTV since the subject study is of acceptable quality and represents a conservative measured endpoint. Although the study used an older DBDE formulation, its constituents represent homologue groups subject to this assessment. This assessment is not limited to analyses of the commercial products, but rather PBDEs in the homologue groups with four to 10 bromine atoms/molecule. Thus, this study is deemed appropriate for use in the quotient analysis, as long as it is understood that it reflects a mixture with a greater proportion of nonaBDE (and a small fraction of octaBDE) than current DBDE formulations.
Letters summarizing comments presented in references 5-7. The senders of the letters indicate their interest in meeting with appropriate members of staff to review the draft PBDE Screening Assessment and the position on decabromodiphenyl oxide/ether.[8,9]	All stakeholders will be consulted during the risk management phase regarding the proposed control or preventive action under CEPA 1999

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