

SUPPLEMENT TO NOTICE OF OBJECTION

Ecojustice is filing this supplement to the Notice of Objection with respect to Environment Canada's proposed *Polybrominated Diphenyl Ethers Regulations* on behalf of the David Suzuki Foundation, Environmental Defence, and the Canadian Environmental Law Association. This supplement highlights the results of recent scientific studies in support of arguments presented in section III of the Notice of Objection.

I. Purpose of Supplement

On February 14, 2007, the Sierra Legal Defence Fund (now Ecojustice Canada) filed a Notice of Objection ("NOO") with respect to the proposed *Polybrominated Diphenyl Ethers Regulations* published in the *Canada Gazette Part I* on December 16, 2006 (vol. 140, no. 50). The NOO was filed pursuant to sections 332(2) and 333 of the *Canadian Environmental Protection Act* ("CEPA"), on behalf of the David Suzuki Foundation, Environmental Defence, and the Canadian Environmental Law Association. It petitions Environment Minister Hon. John Baird to establish a Board of Review to examine the basis for Environment Canada's decision to exclude hepta- through decabrominated diphenyl ethers from the proposed ban of other chemicals in this class. The NOO also urges the Minister to revise the proposed regulations to prohibit the use, sale, offer for sale, and import of *all* polybrominated diphenyl ethers ("PBDEs").

As stated in the NOO, a key reason, *inter alia*, for the Objection was:

The evidence presented in Environment Canada's screening assessments for the proposed regulation is now outdated: it included information up until October 2004 only. New evidence shows that decaBDE bioaccumulates in aquatic and terrestrial organisms including human beings. Recent studies also point to additional evidence of the debromination of decaBDE into lower-brominated congeners. The new evidence points to threats of serious damage from PBDEs, and application of the precautionary principle requires effective Government action. (p. 1)

Subsequently, significant new evidence has continued to be published in the scientific literature that further substantiates the above objection. The purpose of this Supplement is to review scientific findings published since February 14, 2007 that is relevant to Section III of the NOO on "New Evidence on Bioaccumulation and Debromination". We also include new evidence related to the health effects of decaBDE.¹

¹ As in the NOO, the focus in this Supplement is on decaBDE because it is the most highly brominated and prevalent PBDE in the environment. It is also the main component of the most widely-used PBDE commercial mixture, "DecaBDE" or "BDE 209". Conclusions regarding decaBDE also apply to hepta-, octa-, and nonaBDE.

This new evidence is found in the general literature on PBDES and DecaBDE, including the following 15 scientific papers, each of which have been published in the past number of months:

1. Allen, J.G., *et al.*, Personal Exposure to Polybrominated Diphenyl Ethers (PBDEs) in Residential Indoor Air. *Environmental Science & Technology* (2007), 41, 4574-4579.
2. Betts, K., Record-High PBDEs in Chinese Birds of Prey. *Environmental Science & Technology* (2007 March), 41, 1804-5.
3. Bi, X., *et al.*, Exposure of Electronics Dismantling Workers to Polybrominated Diphenyl Ethers, Polychlorinated Biphenyls, and Organochlorine Pesticides in South China. *Environmental Science & Technology* (2007), 41, 5647 -5653.
4. Cahill, T.M., *et al.*, Atmospheric Concentrations of Polybrominated Diphenyl Ethers at Near-Source Sites. *Environmental Science & Technology* (2007) 41, 6370-6377.
5. Chao, H-R., *et al.*, Levels of polybrominated diphenyl ethers (PBDEs) in breast milk from central Taiwan and their relation to infant birth outcome and menstrual cycle. *Environment International* (2007) 33, 239–245.
6. Gobas, F., *et al.*, Quantitative Structure Activity Relationships for Predicting the Bioaccumulation of POPs in Terrestrial Food Webs., *QSAR Comb. Science* (2003), 22, 329 at 335.
7. Harrad, S., *et al.*, Polybrominated diphenyl ethers in domestic indoor dust from Canada, New Zealand, United Kingdom and United States. *Environment International* (forthcoming).
8. Karlsson M., *et al.*, Levels of Brominated Flame Retardants in Blood in Relation to Levels in Household Air and Dust. *Environment International* (2007), 33, 62-69.
9. Kelly, B.C., *et al.*, Food Web-Specific Biomagnification of Persistent Organic Pollutants, *Science* (2007), 317, 236.
10. Lam, J.C.W., *et al.*, Assessment of Polybrominated Diphenyl Ethers in Eggs of Waterbirds from South China. *Environmental Pollution* (2007), 147, 258-267 and 265-266.
11. Nerissa, W., *et al.*, Human Exposure to PBDEs: Associations of PBDE Body Burdens with Food Consumption and House Dust Concentrations. *Environmental Science & Technology* (2007), 41, 1584 -1589.

12. Rice, D.C., *et al.*, Developmental delays and locomotive activity in the C57BL/6/J mouse following neonatal; exposure to fully brominated PBDE, decabrominated diphenyl ether. *Neurotoxicology and Teratology* (2007), 29, 511–520.
13. She, J., *et al.*, Polybrominated Diphenyl Ethers (PBDEs) and Polychlorinated Biphenyls (PCBs) in Breast Milk from the Pacific Northwest. *Chemosphere* (2007) 67, S307-S317.
14. Van den Steen, E., *et al.*, Accumulation, Tissue-Specific Distribution and Debromination of decabromodiphenyl ether (BDE 209) in European Starlings. *Environmental Pollution* (2007), 148, 648-653.
15. Wang, Y., *et al.*, Polybrominated diphenyl ether in the East Asian environment: A Critical Review. *Environment International* (2007), 33, 963-973.

We submit that this new information confirms the need for a Board of Review to examine the growing body of scientific literature on decaBDE, including recent studies not captured in the screening assessment, and more generally timing and review issues for making determinations under CEPA.

II. Additional Evidence of DecaBDE Bioaccumulation

As noted in the NOO, and subsequently published in *Environmental Science and Technology*, Kellyn Betts underlines that decaBDE can bioaccumulate to a much greater extent than believed possible -- and that the high levels of PBDEs indicate that animals eaten by the birds of prey, including small mammals, small birds, earthworms and insects, are much more contaminated than previously believed.²

There is also new evidence to show that uptake of PBDEs in human beings occurs through respiration. Bioaccumulation is an examination of the accumulation of a substance in an organism by *all* means of uptake, including uptake through respiration in air-breathing organisms. Previous examinations of uptake and bioaccumulation of PBDEs focused on uptake in relation to water, and thus had not generally considered uptake through respiration of air. The uptake of chemical substances through air respiration can be measured by the octanol-air coefficient (K_{OA}). The K_{OA} is a "crucial physical-chemical property controlling the potential of organic chemicals to biomagnify in terrestrial mammalian food-chains".³

When consideration is given to accumulation of chemicals through air respiration, it is evident that bioaccumulation of decaBDE occurs in human beings. In their recent article in *Science*, the Barry Kelly, *et al.*, analyze the results of bioaccumulation modeling on three

² Betts, K., Record-High PBDEs in Chinese Birds of Prey. *Environmental Science & Technology* (2007 March), 4196, 1804-5.

³ Gobas, F., *et al.*, Quantitative Structure Activity Relationships for Predicting the Bioaccumulation of POPs in Terrestrial Food Webs., *QSAR Comb. Science* (2003), 22, 329, at 335.

food webs.⁴ Modeling, particularly QSAR analysis, is a method of estimating bioaccumulation generally recognized in the scientific community. The three food webs analyzed were the piscivorous (water-respiring organisms only), the terrestrial (air-breathing organisms only) and the marine-mammalian food web (including water respiring and air-breathing organism). The authors found (at 238) that the "application of the bioaccumulation model to identify potentially bioaccumulative substances among commercial chemicals reveals distinct differences in the biomagnification behavior of chemicals in different food webs".⁵ They concluded that the reason for this was that organisms in different food webs have different rates of uptake and elimination *vis-à-vis* specific chemicals. The modeling found (at 237) that chemicals with high octanol-water and octanol-air coefficients, including PBDE-209 (the commercial formulation that includes mostly deca BDE), biomagnify in water-respiring and air-breathing organisms. The calculated biomagnification factor of PBDE-209 in human beings was 8. Thus modeling has shown that a factor of greater than 5 for human beings is found for BDE-209 when consideration is given to uptake through air.

As found by Karlsson, *et al.*, this is particularly relevant given the prevalence of decaBDE in indoor air and dust. They found that deca-BDE is a dominant PBDE in dust.⁶ Correlations between PBDEs in dust and breastmilk have been shown.⁷ Recently a study headed by Joseph G. Allen and published in *Environmental Science and Technology* compared PBDEs in room air to "personal air" (the "personal cloud effect"⁸) and found that the quantity of BDE 209 in personal air is on average four times higher than that in area air.⁹ It further estimated that the contribution of inhalation exposure to BDE 209 in adults accounts for 22% of BDE 209 exposure in adults. The conclusion was:

To our knowledge, our results are the first measurements of personal air concentrations of PBDEs in a non-occupational setting and the first indoor air measure of PBDEs in the United States; they include BDE 209, a congener that has not been widely reported. We found that personal air concentrations of less volatile PBDEs exceed area measurements, consistent with a personal cloud effect, and that inhalation of BDE 209 may account for a significant percent of overall PBDE exposure in U.S. adults.¹⁰

⁴ Kelly, B.C., *et al.*, Food Web-Specific Biomagnification of Persistent Organic Pollutants, *Science* (2007), 317, 236.

⁵ Biomagnification represents increasing levels of bioaccumulation in an organism because of dietary accumulation.

⁶ Karlsson, M., *et al.*, Levels of Brominated Flame Retardants in Blood in Relation to Levels in Household Air and Dust. *Environment International* (2007), 33, 62-69.

⁷ Nerissa, W., *et al.*, Human Exposure to PBDEs: Associations of PBDE Body Burdens with Food Consumption and House Dust Concentrations. *Environmental Science & Technology* (2007), 41, 1584 -1589.

⁸ The 'personal cloud effect' is the air around a human being in his or her personal space that is measured throughout the day.

⁹ Allen, J.G., *et al.*, Personal Exposure to Polybrominated Diphenyl Ethers (PBDEs) in Residential Indoor Air. *Environmental Science & Technology* (2007), 41, 4574-4579.

¹⁰ *Ibid.*

Another study performed international comparison of the levels of PBDEs in dust.¹¹ It found that "dust ingestion appears to constitute a substantial and in some instances the overwhelmingly most important exposure pathway to BDE 209" (at 6) and (at 7) that "[t]he evidence presented here adds to the weight of evidence that ingestion of indoor dust constitutes currently an appreciable source of human exposure to PBDEs" .

As a footnote, it is possible that human proximity to computers is contributing to ingestion of BDE 209. PBDE concentrations in human beings have been found to be higher when computers are turned on as compared with a time when computers are turned off.¹²

III. Additional Evidence of DecaBDE Debromination

Additional evidence of the debromination of decaBDE into substances that have been banned has also recently been published. The study "Accumulation, Tissue-Specific Distribution and Debromination of decabromodiphenyl ether (BDE 209) in European Starlings"¹³ headed by Evi Van den Steen concludes:

Our results suggest that BDE 209 is degraded to lower brominated congeners in tissues of European starlings. Although it can not be excluded that a small fraction of the octa- and nona-BDEs found in the tissues stems from the bioaccumulation of eventual trace octa- and nona-BDEs in the oil, it is mostly plausible to assume that the presence of these congeners is the result of the debromination of BDE 209. The formation of lower bromination congeners from decaBDE has previously been reported in rats (Mörk et al, 2003) and fish (Kierkegaard et al. 1999; Stapleton et al., 2004). Similar to those in fish (Kierkegaard et al. 1999; Stapleton et al., 2004), our results suggested that BDE 209 can be debrominated down to hexaBDEs.

The present study, in accordance with the previous studies (Kierkegaard et al. 1999; Mörk et al, 2003; Stapleton et al., 2004), suggested that BDE 209 can be debrominated to congeners that are also present in the penta and octa-BDE commercial mixtures, which are no longer allowed for use due to their potential toxicity (Darnerud, 2003). Thus, phasing out the penta- and octaBDE commercial mixtures could be insufficient to restrict the potential risk of lower brominated congeners. These results are a great cause of concern considering the large amounts of deca-BDE that are worldwide used (BSEF, 2003).

Given the weight of evidence on the issue, it appears established that decaBDE debrominates into lower congener, persistent, bioaccumulative and toxic PBDEs.

¹¹ Harrad, S., *et al.*, Polybrominated diphenyl ethers in domestic indoor dust from Canada, New Zealand, United Kingdom and United States. *Environment International* (forthcoming).

¹² Cahill, T.M., *et al.*, Atmospheric Concentrations of Polybrominated Diphenyl Ethers at Near-Source Sites. *Environmental Science & Technology* (2007) 41, 6370-6377.

¹³ Van den Steen, E., *et al.*, Accumulation, Tissue-Specific Distribution and Debromination of decabromodiphenyl ether (BDE 209) in European Starlings. *Environmental Pollution* (2007), 148, 648-653.

IV. Human Exposure and Health Outcomes

Several other new studies provide further evidence of decaBDE's prevalence in the environment, human exposure, and negative health outcomes. A recent report by Bixian Mai, *et al.*, on PBDE migration from landfills indicates that BDE-209 is the major overall contribution to the overall soil PBDE concentrations. Further, waste discharges from some cities have showed that 72.6 to 99.7% of the total concentrations of PBDEs were comprised of BDE-209.¹⁴

A recent critical review of studies on PBDEs headed by Yawei Wang indicates that the concentration of deca BDE in human blood has been increasing rapidly in recent years across the planet:

The time trends of PBDE levels in human blood in Japan and Norway is shown in Fig.3, from which it can be seen that the concentrations of PBDEs in human blood has increased rapidly in recent years. High levels of deca-BDE (up to 13 ng g⁻¹, lipid weight) were determined in Japanese human blood collected in 2004 (Takasuga *et al.*, 2001), which contrasts with the previous reports that found BDEs-47, 99 and 152 to be prevalent. Possible sources for the more recent prevalence of deca-BDE are food and computer rich environments, such as offices and game rooms (de Boer *et al.*, 1999).¹⁵

PBDEs in breastmilk in the Pacific Northwest of the United States and Canada are comparable to levels found elsewhere in North America, but are 20-40 times higher than the levels found in Sweden and Japan.¹⁶

The highest BDE-209 concentration reported in human beings was recently reported with a single person who showed a concentration of 3100ng/g lipid, which is 50- to 200-fold higher than in previously known occupationally exposed populations.¹⁷

Other studies have shown that PBDE levels in breast milk are correlated with adverse birth outcome such as birth weight, birth length and chest circumference, particularly for the congeners of BDE-47, BDE-99, BDE-100, and BDE-209.¹⁸

¹⁴ Mai, B.X., *et al.*, Distribution of polybrominated diphenyl ethers in sediments of the Pearl River Delta and adjacent South China Sea. *Environmental Science & Technology* (2005), 39, 3521-7, cited in Wang, Y., *et al.*, Polybrominated diphenyl ether in the East Asian environment: A Critical Review. *Environment International* (2007), 33, 963-973.

¹⁵ Wang, Y., *et al.*, *ibid.*, at 969.

¹⁶ She, J., *et al.*, Polybrominated Diphenyl Ethers (PBDEs) and Polychlorinate Biphenyls (PCBs) in Breast Milk from the Pacific Northwest. *Chemosphere* (2007) 67, S307-S317.

¹⁷ Bi, X., *et al.*, Exposure of Electronics Dismantling Workers to Polybrominated Diphenyl Ethers, Polychlorinate Biphenyls, and Organochlorine Pesticides in South China. *Environmental Science & Technology* (2007), 41, 5647 -5653.

¹⁸ Chao, H-R., *et al.*, Levels of polybrominated diphenyl ethers (PBDEs) in breast milk from central Taiwan and their relation to infant birth outcome and menstrual cycle. *Environment International* (2007) 33, 239-245.

Similarly, a study by Deborah C. Rice, *et al.* and published in *Neurotoxicology and Teratology* that examined the development of mice after neonatal exposure to decaBDE observed behavioural changes such as increased activity levels in males. The observed “effects suggest that decaBDE is a developmental neurotoxicant that can produce long-term behavioural changes following a discrete period of neonatal exposure” a finding supported by other studies.¹⁹

Another recent study, headed by James C.W. Lam, evaluated the neurotoxic potential of PBDEs in birds from the neurotoxic potential on rodents. The study derived the tentative critical concentrations (TCCs) for substances, and found that "with regard to BDE 209, log₁₀ 90th percentile exposure level (2.53), based on cattle egret and Chinese pond heron, was greater than both TCCs with and without the application of the uncertainty factor. This suggests a potential risk by BDE 209 to the cattle egret and Chinese pond heron populations"²⁰

V. Conclusion

Environment Canada’s PBDE screening assessment considered information published prior to October 2004 (information published between October 2004 and October 2005 was reviewed but generally not included in the assessment). A key reason for the Notice of Objection was that the screening assessment was out of date with respect to its conclusions concerning the bioaccumulation and debromination of hepta- through decaBDE. New information published in the scientific literature since the NOO was filed in February, 2007, reinforces this claim. Recent research findings on the prevalence of decaBDE in the environment, human exposure to this chemical, and the negative health outcomes add urgency to the Objection.

Having received no response to the NOO to date, we reiterate our request that the Minister of the Environment revise the proposed *Polybrominated Diphenyl Ether Regulations* to prohibit the use, sale, offer for sale, and import of all PBDEs; and further that a Board of Review be established to examine, *inter alia*, new evidence concerning decaBDE bioaccumulation and debromination, and whether measures should be recommended to ensure the currency of screening assessments and related determinations under CEPA.

¹⁹ Rice, D.C., *et al.*, Developmental delays and locomotive activity in the C57BL/6/J mouse following neonatal; exposure to fully brominated PBDE, decabrominated diphenyl ether. *Neurotoxicology and Teratology* (2007), 29, 511–520.

²⁰ Lam, J.C.W., *et al.*, Assessment of Polybrominated Diphenyl Ethers in Eggs of Waterbirds from South China. *Environmental Pollution* (2007), 147, 258-267 and 265-266.