



LEARNING DISABILITIES ASSOCIATION OF CANADA
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October 30/06

Director
Chemical Sector Division
Environment Canada
351 St. Joseph Blvd. 12th Floor
Gatineau, QC K1A 6H3

Re: Consultation on PBDE Risk Management Strategy

We support the conclusion by Environment Canada that the seven PBDEs be added to Schedule 1 of CEPA 99, and that they meet the criteria set out in paragraph 64 (a) of the Act. We also support that conclusion that tetra-, penta- and hexaBDE meet the criteria for persistence and bioaccumulation, as defined by the Persistence and Bioaccumulation Regulations of CEPA 1999 that their presence results from human activity and therefore they meet the conditions set out in CEPA 99 for mandatory addition to the Virtual Elimination List.

We have some concerns that this risk management plan is not going to be adequately protective of human health and development for the following reasons:

- The lengthy timeframe set out to begin mitigating actions on PBDEs
- The risk management plan does not describe in any detail possible regulatory actions on “near-field” exposures into air and dust from PBDEs contained in consumer products in the home, that are of most importance to human health risk¹. As noted in the strategy, these include common consumer products e.g. computers and soft home furnishings containing large amounts of PBDEs. These

¹ Harrad.S, Wijekesera R, Halliwell C, Baker R (2004) Preliminary assessment of U.K. human dietary and inhalation exposure to polybrominated deiphenyl ethers. Environ Sci. Technol. 38 (8) pp 2345-50.

exposures are relatively direct to humans via inhalation from indoor air and ingestion from dust particles – especially relevant for children’s exposures.

- There is a recognition that disposal is an issue but no detail or outline of a plan for action on disposal of products containing PBDEs and monitoring of leachate from disposal

Because the 2004 risk assessment for PBDEs proposed that these substances be added to Schedule 1 of CEPA and considered “toxic” based on environmental considerations, the reasons for this action plan are based principally on the effects of PBDEs on the environment and the secondary poisoning of wildlife from releases into the environment.

We cannot agree with the conclusions of the 2004 screening assessment report (health) that actions taken to protect ecosystems and wildlife will be adequate to protect human health and development. The report also noted that more scientific information was needed regarding the risk to human health, but that this research was not a priority *“unless information becomes available to indicate that measures recommended to control exposure of environmental organisms to PBDEs will not be protective of human health”*. This seems to be an astonishing Catch-22 statement that seems to say “when we know that action is needed to reduce risk to human health, we’ll begin to do more research”.

For a number of reasons, including those provided to the Ministers in our 2004 comments on the human health risk assessment for the PBDEs, we disagree with the statement in the proposed risk management strategy that worst-case estimates of the exposure of Canadians to PBDEs were much less than those which caused health effects in animals. Of major concern to our Association are the increasing number of experimental studies of the neurodevelopmental effects of PBDEs from prenatal or early life exposures^{2 3 4 5 6}, and on

² D. F. Staskal, H. Hakk, D. Bauer, J. J. Diliberto, and L. S. Birnbaum. Toxicokinetics of Polybrominated Diphenyl Ether Congeners 47, 99, 100, and 153 in Mice Toxicol. Sci., November 1, 2006; 94(1): 28 - 37.

³ H. Viberg, N. Johansson, A. Fredriksson, J. Eriksson, G. Marsh, and P. Eriksson Neonatal Exposure to Higher Brominated Diphenyl Ethers, Hepta-, Octa-, or Nonabromodiphenyl Ether, Impairs Spontaneous Behavior and Learning and Memory Functions of Adult Mice

⁴ P. R. S. Kodavanti, T. R. Ward, G. Ludewig, L. W. Robertson, and L. S. Birnbaum Polybrominated Diphenyl Ether (PBDE) Effects in Rat Neuronal Cultures: 14C-PBDE Accumulation, Biological Effects, and Structure-Activity Relationships Toxicol. Sci., November 1, 2005; 88(1): 181 - 192.

thyroid disruption during development⁷. In the total absence of data on clinical effects in children, it would be prudent to suggest that there may be effects at these levels in the most highly exposed populations. In any case exposures to animals do not depict or predict the same toxicological effects of exposures in humans. Uncertainty factors must be applied to extrapolate from animal to human and for intrahuman differences (infant to adult). There is evidence that levels of PBDEs in breast milk consumed by infants are doubling every two to five years, and are approaching the level of PCBs that have been found to have permanent adverse effects on neurodevelopment⁸. Combinations of these chemicals can have additive or even synergistic effects on thyroid economy and/or neurodevelopment. The decision to allow such a lengthy period -1 year and eight months - to design a risk management plan, and another eighteen months to implement it, in total *three years and two months before any action is taken* – is much less than protective. In 2002 the EU Commission’s conclusions on risks to humans brought about timely and precautionary action on PBDEs.

It also addressed secondary poisonings via degradation products to more toxic and bioaccumulative compounds. Regulations regarding disposal are important we agree, but details on the strategy are absent.. It is unclear whether products currently in commerce will continue to be produced with PBDEs under this plan . Regulations regarding PBDEs in consumer products should require that PBDEs not be present in all products imported

⁵ A. K. Peters, K. van Londen, A. Bergman, J. Bohonowych, M. S. Denison, M. van den Berg, and J. T. Sanderson. Effects of Polybrominated Diphenyl Ethers on Basal and TCDD-Induced Ethoxyresorufin Activity and Cytochrome P450-1A1 Expression in MCF-7, HepG2, and H4IIE Cells. *Toxicol. Sci.*, December 1, 2004; 82(2): 488 - 496.

⁶ Henrik Viberg^{*,1}, Anders Fredriksson^{*}, Eva Jakobsson[†], Ulrika Örn[†] and Per Eriksson^{*} Neurobehavioral Derangements in Adult Mice Receiving Decabrominated Diphenyl Ether (PBDE 209) during a Defined Period of Neonatal Brain Development. *Toxicol. Sci.* 76 (1) 112-120.

⁷ Tong Zhou^{*}, Michele M. Taylor[†], Michael J. DeVito[‡] and Kevin M. Crofton[‡] Developmental Exposure to Brominated Diphenyl Ethers Results in Thyroid Hormone Disruption. *Toxicological Sciences* **66**, 105-116 (2002)

⁸ Tom Muir. Are thyroid and neurodevelopmental effects related to rising PBDE levels? *Organohalogen Compounds CD proceedings* 2005.

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or manufactured in Canada. Presumably this could be accomplished via a Ministerial decision under CEPA 99.

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