

Comment on "Global Assessment of Polybrominated Diphenyl Ethers in Farmed and Wild Salmon"

The Hites et al. study on polybrominated diphenyl ethers (PBDEs) in farmed and wild caught salmon (1) reports detection of the pattern of lower brominated PBDEs typically found in fish. The primary isomers detected were tetra-, penta-, and hexabromodiphenyl ethers (e.g., BDE-47, -99, -100, -153, and -154). These isomers are associated with the PentaBDE product that is being phased out voluntarily by its sole manufacturer.

We are concerned at confusion that was created in part due to discrepancies between the original August 10, 2004, on-line version of this paper and a subsequent revision, which appeared August 27. In the first version, the authors did not report information specific to detection of decabromodiphenyl ether/oxide (DecaBDE), which represents more than 80% of the PBDEs manufactured worldwide. Subsequently, the authors were quoted as saying they had analyzed for DecaBDE, but "We didn't report it because it was found at such a low level" (2). This is consistent with DecaBDE's known properties—DecaBDE does not have the same potential as the tetra penta, and hexa compounds for uptake, and its detection in fish is uncommon. In the revised August 27, 2004, on-line version, the authors did mention DecaBDE's nondetection in salmon near the end of the Results and Discussion section, although they did not include DecaBDE in the list of congeners analyzed under Materials and Methods nor make it clear elsewhere in the paper that DecaBDE did not contribute to the sum of the PBDEs. Only the most careful of readers would have realized from the study, *Environ. Sci. Technol.*'s accompanying editorial, and the resulting press attention that detection of the major PBDE in use today (i.e., DecaBDE)—and the only PBDE that will be available as of 2005—was negligible to nonexistent.

Unfortunately, the current trend of reporting data generically as PBDEs—rather than referring to the specific isomers (e.g., BDE-47, -99, etc.) or commercial product (e.g., Penta-, Octa-, or DecaBDE) studied has led to growing and unnecessary confusion among public, professional, and media audiences. Using the generic term PBDEs suggests that all members of that class share similar characteristics, even though the scientific literature clearly demonstrates that this assumption is false. We urge authors and scientific publications to exercise more precision as they report research findings. Such rigor will greatly enhance the ability of interested parties to put research into a perspective that allows for accurate and meaningful deliberation.

DecaBDE is used to flame retard certain electronic and electrical equipment and upholstery textiles and is estimated to save 200–300 U.S. lives each year through fire prevention (3, 4). DecaBDE's toxicology has been studied extensively over the past 20 years and is very different from the other two commercial PBDE products. DecaBDE has a no-adverse-effect level of at least 1000 mg kg⁻¹ d⁻¹ in repeated dose studies (5), and the U.S. National Academy of Sciences defined an oral reference dose (RfD) of 4 mg kg⁻¹ d⁻¹ (6). Risks to human health or the environment from DecaBDE have not been identified in exhaustive assessments performed by various authorities around the world (3, 6–8). For example, the European Union recently concluded a 10-year risk

assessment of DecaBDE (6). Risks to human health and the environment were not identified. Likewise, the U.S. National Academy of Sciences concluded that DecaBDE did not present a health risk to consumers, including children, when used in upholstery textiles (6).

Fires, on the other hand, represent a serious risk in the United States. Nationwide, there is a fire-related death every 2.5 h. Someone is injured in a fire every 28 min (9). The majority of fire deaths (85%) occur in home fires. Those most vulnerable to death and injury are children, senior citizens, and those with low incomes (10). The latest estimate is that fires cost the United States \$186–305 billion, depending on whether the events of September 11 are included (11). Continued efforts to reduce the number and severity of fires are critical.

The Hites et al. paper (1) left the reader with several misconceptions that we would like to correct. DecaBDE has not been banned in the European Union. The toxicology database on DecaBDE is extensive; in 2000, the U.S. National Academy of Sciences concluded that additional studies were not needed in order ensure consumer protection. Although DecaBDE is the most "highly brominated" PBDE (to use the term in the Hites et al. paper; 1), it is not "metabolically active" and does not induce hepatic enzymes (12, 13) or bind to the Ah receptor. One study, with serious design flaws such that high false positive rates are induced, reported developmental neurotoxicity when a single dose was administered to neonatal mice (14). However, other studies found no evidence of neurotoxicity after repeated administration of much higher doses to rats during gestation (15) or to rats and mice over the course of a lifetime (16). DecaBDE is not an endocrine disruptor, does not affect thyroid hormone levels, and is not an estrogen agonist (13, 16). DecaBDE does not induce bacterial mutations, chromosome aberrations, sister chromatid exchanges, cytogenic aberrations in rat bone marrow cells, or mutations in the mouse lymphoma assay (16). DecaBDE is not listed as a carcinogen by NTP, IARC, or OSHA (17–19).

We urge greater precision in reporting results from studies on PBDE isomers. This would allow the reader to discriminate between DecaBDE and those isomers associated with the Penta- and OctaBDE products. A quality journal like *Environ. Sci. Technol.* should insist on it.

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