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EXPOSURE OF INFANTS AND CHILDREN IN THE U.S. TO THE FLAME RETARDANT DECABROMODIPHENYL OXIDE (DBDPO)

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Introduction

Decabromodiphenyl oxide (DBDPO), the most highly brominated of the polybrominated diphenyl oxides (PBDPOs), is the most widely used brominated flame retardant in the United States. It is used predominantly in hard plastic electronic consumer products and in flame-retarded backing on textiles for furniture. DBDPO was included in the U.S. Environmental Protection Agency's (U.S. EPA's) Voluntary Children's Chemical Evaluation Program (VCCEP), because PBDPOs, as a class of compounds, have been reported in human breast milk samples, although DBDPO has never been detected.

Two recent studies evaluated general population exposures to flame-retardant chemicals on textiles and the associated risks. The National Academy of Sciences (NAS) found that risks to adults or children from DBDPO were "negligible,"[1] and the Consumer Product Safety Commission stated, "It does not appear that DBDPO would present a hazard to consumers."[2] Studies conducted by the World Health Organization[3] and European Union[4] also concluded that exposures to DBDPO did not pose a health risk to the general population. Despite the conclusions of these well-respected organizations, certain governmental agencies are still concerned about DBDPO, because it is a persistent organic chemical. Furthermore, the PBDPOs are often being treated as a single chemical, rather than considering the unique physical properties and toxicity of each individual chemical within this relatively broad class.

In this assessment, children's potential exposures to DBDPO from all sources (including electronics, upholstery, breast milk, and the general environment) were characterized using data from published literature, agency reports, and information from manufacturers. An extensive literature search indicated that there are very few data on the concentrations of DBDPO in environmental media and food in the U.S., and when data were found, the concentrations are typically very low or below the detection limit. However, biomonitoring data (e.g., serum levels) for DBDPO in humans are available, and provide an alternative way to calculate intakes, which often may have lower levels of uncertainty than calculations using limited measured data. As a result, this analysis largely relies on biomonitoring data to assess exposures, and thus risks, for children exposed to DBDPO in the U.S.

Materials and Methods

This child-specific risk assessment followed the VCCEP guidance for a Tier I assessment, and all applicable USEPA guidance. Conservative assumptions were made for all input parameters, and both a reasonable estimate (RE) and an upper estimate (UE) were calculated for each pathway.

Based on the manufacture and uses of consumer products containing DBDPO, intakes from six exposure pathways were quantified:

Child (0–2 years) ingesting breast milk from a mother who is occupationally exposed to DBDPO in two different job categories:

1. A mother who manufactures DBDPO (bagging operation)
2. A mother who disassembles electronics

Additional pathways for children's exposure:

3. Child (0–2 years) mouthing DBDPO-containing plastic electronic products
4. Child (0–2 years) inhaling DBDPO particulates released from plastic electronic products
5. Child (0–2 years) mouthing DBDPO-containing fabric
6. Child (all ages) exposed to DBDPO via the general environment (e.g., soil and dust, diet, ambient air, and water).

The first two pathways, which involve intake via breast milk, are of particular interest to those who are concerned with persistent chemicals and exposures to infants. However, DBDPO, because of its large molecular size, low oral bioavailability, and rapid elimination, is expected to partition only minimally into breast milk, and has been shown to not bioaccumulate. At present, there are no published values for DBDPO in breast milk; therefore, exposures via this pathway were estimated indirectly. For the first exposure pathway, a workplace air concentration was estimated (1 to 5 mg/m³), an air-to-serum ratio was calculated, and then a serum-to-breast milk partitioning factor was selected (0.1 to 0.5, based on data from lower brominated diphenyl ethers). For the second exposure pathway, serum levels of DBDPO in Swedish disassembly workers were selected from published studies (4.8 to 9.9 ng/g lipid)[5] and combined with the aforementioned serum-to-breast milk partitioning factor to estimate breast milk concentrations.

The intake calculations for the third pathway are based on the speculative assumption that DBDPO may leach from plastic and be available for an infant to ingest through mouthing, although leaching experiments found undetectable levels of DBDPO when an acrylonitrile butadiene-styrene (ABS) pellet with DBDPO was placed in water or acetic acid.[6] Intakes were derived using the reported detection limit in water (0.075 mg/L) and the amount leached in cottonseed oil at 135°F for 7 days (1 mg/L)[6]. For the fourth pathway, intakes were based on air concentrations of DBDPO (0.052 to 0.087 ng/m³) measured in an office with computers in Sweden.[7] Intakes for the fifth pathway were drawn from the NAS study, which assumed that a child (0–2 years) mouthed fabric backcoated with DBDPO for 1 hour each day[1]. For the sixth pathway, serum levels of DBDPO in U.S. blood donors (<0.96 to 33.6 ng/g lipid)[8] were used to back-calculate exposures, assuming a one-compartment model, a half-life of 3 to 6.8 days, and an oral absorption of 1% to 2%.

Total daily intakes were calculated for three receptor populations by aggregating the following pathway-specific intakes:

1. Total aggregate intake for a nursing infant (age 0–2 years) whose mother is occupationally exposed through the manufacture of DBDPO includes intakes from pathways 1, 3, 4, 5, and 6.
2. Total aggregate intake for a nursing infant (age 0–2 years) whose mother is occupationally exposed through the disassembly of electronics includes intakes from pathways 2, 3, 4, 5, and 6.
3. Total aggregate intake for a child (age >2–18 years) includes intakes from pathway 6.

To estimate noncancer risks associated with an estimated exposure, a hazard quotient (HQ) is calculated by dividing the estimated intake by a reference dose (RfD). The RfD is an estimate of daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of adverse effects during a lifetime. The RfD for DBDPO used in this assessment, 4 mg/kg-day[1], was derived by the NAS using the National Toxicology Program's (NTP's) 2-year rat bioassay results.[9] The RfD was based on the chronic no-observed-adverse-effect level (NOAEL) of 1,120 mg/kg-day, and a composite uncertainty factor of 300. The RfD derived by the NAS study was used, rather than the U.S. EPA Integrated Risk Information System (IRIS) value, because the NTP study was more recent, used more than one species and a larger number of animals, and because the NTP study used a DBDPO product of higher purity, which is more representative of the commercial formulation currently being used.

Results

As presented in Table 1, there is a difference of up to an order of magnitude between the RE and UE exposures for the two infant scenarios, and a difference of two orders of magnitude between the RE and UE exposures for the general environment scenario. The highest estimated exposure (UE for the infant, manufacturer scenario) is 0.76 mg/kg-day, and the lowest estimated exposure (RE for the older child's general exposures) is 0.0012 mg/kg-day.

Table 1. DBDPO exposure estimates and hazard quotients

	Exposure (mg/kg-day)		Hazard Quotient[a]	
Pathway/Scenario	RE	UE	RE	UE
Pathway-specific				
Ingestion, breast milk, manufacturer	1.9×10^{-2}	3.4×10^{-1}	0.005	0.09
Ingestion, breast milk, disassembler	3.3×10^{-6}	2.5×10^{-5}	8×10^{-7}	6×10^{-6}
Ingestion, consumer electronics	4.3×10^{-6}	2.5×10^{-4}	1×10^{-6}	6×10^{-5}
Inhalation, particulates	3.1×10^{-8}	6.3×10^{-8}	8×10^{-9}	2×10^{-8}
Ingestion, mouthing fabric (NAS)	2.6×10^{-2}	2.6×10^{-2}	0.007	0.007
General exposures (all ages)	1.2×10^{-3}	3.9×10^{-1}	0.0003	0.1
Aggregate				
Infant, manufacturer	0.046	0.76	0.01	0.2
Infant, disassembler	0.027	0.41	0.007	0.1
Child, general (>2–18)	0.0012	0.39	0.0003	0.1

[a] Hazard quotient calculated using an RfD of 4 mg/kg-day, derived by the NAS[1].

The HQs, shown in Table 1 and Figure 1, for the RE scenarios range from 0.0003 to 0.01, and from 0.1 to 0.2 for the UE scenarios, with the highest HQ associated with the UE for the infant whose mother manufactures DBDPO and is employed in the bagging operation. All calculated HQs are significantly less than one, with the highest aggregate HQ being 0.2.

Discussion

The calculations presented here indicate that the potential exposures for each scenario evaluated are quite small. It must be stressed that the RE, as well as the UE, represents exposures that are greater than that actually experienced by the majority, if not all, of the U.S. population. Additional data would lower the uncertainties and overestimates in the calculations of intake. Moreover, even when using these highly conservative values, the risk calculations show that all HQs are well below 1, indicating that there is little concern for potential health risk among children associated with DBDPO in the environment, in consumer product applications, or even

from secondary occupational exposures, and suggests that more refined evaluations under the VCCEP are not likely to be needed. No published or government agency evaluations have shown a human health risk associated with DBDPO. Because multiple national and international studies have concluded that there are no health risks associated with the use of DBDPO, and because the results of this study show no apparent risks to infants and children, the totality of available evidence indicates that DBDPO is a safe product, and its use provides a clear benefit to the consumer.

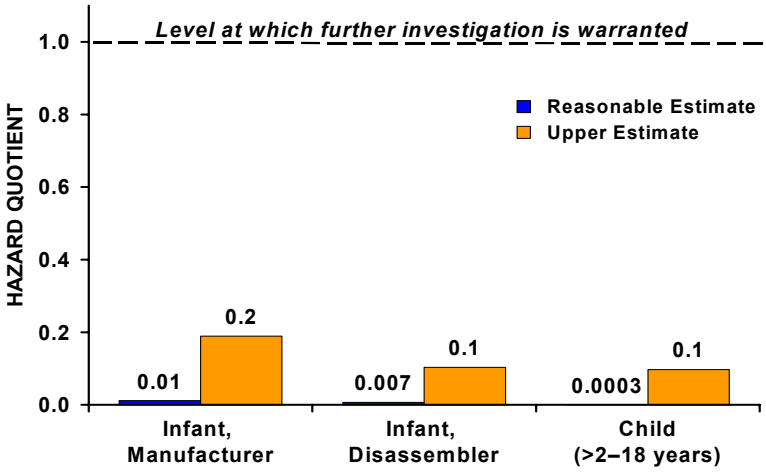

Figure 1. Hazard quotients for children's exposure to DBDPO

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