New Information on Decabromodiphenyl Ether and How it Changes Our Interpretations of Risk

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Introduction

Decabromodiphenyl ether (DBDPE), the most highly brominated of the polybrominated diphenyl ethers (PBDPEs), 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. Several U.S.[1,2] and international organizations[3,4] have formally evaluated the human health and environmental risks associated with the use of the major BFRs for consumer applications and for DBDPE specifically. These risk assessments have found DBDPE to be safe in its' current use. Most recently, DBDPE underwent an evaluation under the Voluntary Children's Chemical Evaluation Program (VCCEP) and was found to pose negligible health risks for children[5] and is currently undergoing a formal risk assessment within the European Union.

Most of these risk assessments have had to rely on extrapolations to estimate exposures among the general population[5]. Most uncertain has been the indirect methods that have had to be used to estimate infants' exposures via potential presence in breast milk. However, newly published data on levels of DBDPE in human milk volunteers in the U.S. provide a better means for calculating infants' exposures. This new data is used to put the previously conducted DBDPE VCCEP risk assessment in perspective and to help assess whether a different conclusion about risks is warranted, what new data gaps and data needs warrant further attention, and some conclusions about risks and benefits of DBDPE and how these new findings change the balance between risks and benefits of the use of DBDPE to protect consumer products.

In the VCCEP risk assessment, children's potential exposures to DBDPE 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 had indicated that few data existed on the concentrations of DBDPE in environmental media and food in the U.S., and because the concentrations are typically very low or below the detection limit. However, biomonitoring data (e.g., serum levels) for DBDPE 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 DBDPE in the U.S.

Materials and Methods

The 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 DBDPE, intakes from six exposure pathways were quantified:

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

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

Additional pathways for children's exposure:

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

The first two pathways involve intake via breast milk. At the time the VCCEP risk assessment was conducted, there were no published values for DBDPE 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/m^3), 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 DBDPE in Swedish disassembly workers were selected from published studies (4.8 to 9.9 ng/g lipid)[6] and combined with the aforementioned serum-to-breast milk partitioning factor to estimate breast milk concentrations.

The intake calculations for the third pathway were based on the assumption that DBDPE may leach from plastic and be available for an infant to ingest through mouthing, although leaching experiments found undetectable levels of DBDPE when an acrylonitrile butadiene-styrene (ABS) pellet with DBDPE was placed in water or acetic acid[7]. 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)[7]. For the fourth pathway, intakes were based on air concentrations of DBDPE (0.052 to 0.087 ng/m³) measured in an office with computers in Sweden.[8] Intakes for the fifth pathway were drawn from the NAS study, which assumed that a child (0–2 years) mouthed fabric backcoated with DBDPE for 1 hour each day[1]. For the sixth pathway, serum levels of DBDPE in U.S. blood donors (<0.96 to 33.6 ng/g lipid)[9] 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 DBDPE 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) was calculated by dividing the estimated intake by a reference dose (RfD). The RfD for DBDPE 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[10]. 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.

Results and Discussion

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. DBDPE 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, 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 DBDPE and is employed in the bagging operation. All calculated HQs are significantly less than one, with the highest aggregate HQ being 0.2.

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 DBDPE 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.

Recently reported data on DBDPE in breast milk provides a means of checking the conservatism used in estimating exposures in this assessment. Schecter et al.[11] reported a mean of 0.92 ng/g lipid and a maximum of 8.24 ng/g lipid of DBDPE in breast milk from volunteers in the U.S. In contrast, the maximum estimated breast milk concentration calculated in this VCCEP exposure assessment was 70,000 ng/g lipid[5]. Thus, it is almost certain that the estimated levels of exposure in this

VCCEP assessment are vast over-predictions of actual exposures experienced by infants in the U.S. Since the exposures estimated in this VCCEP assessment are below the RfD and are almost certainly over-estimates of exposures, a large margin of safety is indicated for this compound.

No published or government agency evaluations have shown a human health risk associated with DBDPE. Because multiple national and international studies have concluded that there are no health risks associated with the use of DBDPE, and because the results of this study show no apparent risks to infants and children, DBDPE should be considered a safe product, and its use provides a clear benefit to the consumer. The life saving benefits provided by DBDPE is well recognized. Estimates suggest that the BFRs are responsible for avoiding 280 deaths in the U.S. annually, at a minimum, and numerous more injuries[12]. Since the BFRs, and DBDPE specifically, provide a real and valuable benefit to society, a careful and serious comparison of the risks and benefits of these compounds should always be evaluated when making risk management decisions.

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