

Exposure of Infants and Children in the U.S. to the Flame Retardant Decabromodiphenyl Oxide (DBDPO)

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ABSTRACT

Decabromodiphenyl oxide (DBDPO) is a widely used brominated flame retardant in the United States. It is used predominantly in hard plastic electronic consumer products and in flame-retardant backing on textiles used in furniture. Decabromodiphenyl oxide was included in the U.S. Environmental Protection Agency's (EPA's) Voluntary Children's Chemical Evaluation Program (VCCEP). A child-specific exposure assessment of DBDPO was performed following the VCCEP guidance for a Tier 1 exposure assessment (e.g., screening-level assessment using currently available data and conservative assumptions). Exposure pathways that were considered included general environmental exposures, breast milk exposures, inhalation of DBDPO particulates in air, and mouthing DBDPO-containing consumer products. For each exposure scenario, a mid-range estimate and an upper estimate of intake were calculated. Despite the uncertainties, results indicate that the aggregate exposures for children to DBDPO for each scenario evaluated were at least one order of magnitude (most being several orders of magnitude) below the National Academy of Sciences (NAS) reference dose (RfD) for DBDPO. This analysis indicates that, using the available data, current levels of DBDPO in the U.S. are not likely to represent an adverse health risk for children.

Key Words: brominated flame retardants, decabromodiphenyl oxide, exposure assessment, children's health, risk assessment.

INTRODUCTION

Decabromodiphenyl oxide (DBDPO) is a widely used brominated flame retardant in the U.S. Global market demand for DBDPO in 1999 has been estimated at

54,800 tons, with 24,300 tons being used in the Americas (Hardy 2002). There are two manufacturing facilities for DBDPO in the U.S.; both are located in Arkansas. About 80% of DBDPO is used in the production of high-impact polystyrene electrical or electronic products, such as television cabinets (Hardy 2002). A typical DBDPO load level in high-impact polystyrene, flame retarded to meet the Underwriter Laboratories V-O standard, is about 15%. Decabromodiphenyl oxide is also added to other polymers that are used to manufacture a variety of electrical and electronic equipment, including polypropylene, acetate copolymers, and other polyester resins. These applications are important in improving the safety of consumer products since potentially flammable polymers are in close contact with possible ignition sources (electricity).

The other important application for DBDPO is in textiles and fabrics used in residential and commercial furniture and in automotive fabrics, where the DBDPO is applied in a latex back-coat to the textile. Decabromodiphenyl oxide is not used in children's clothing or sleepwear. Decabromodiphenyl oxide is typically applied to furniture fabrics at an average concentration of about 50 g/m² (European Chemicals Bureau 2002), although this can vary considerably with the type of fabric being treated and the end use. In both plastic and textile applications, DBDPO is used in combination with antimony trioxide, which increases the flame-retardant properties of DBDPO.

In the U.S., commercial furniture is required to meet fire safety standards, but only in California are fire safety standards also applied to residential furniture by law. For textile applications, DBDPO is encapsulated in a polymer and applied to the backing of the fabric, which significantly limits potential releases and exposures via direct contact with the flame-retarded surface. However, DBDPO is an additive flame retardant, which means it is physically combined with the material instead of being chemically combined, as is the case with reactive flame retardants. This creates the possibility that DBDPO may diffuse out of the treated material and increases the importance of assessing potential childhood exposures to this chemical (ECB 2002).

Four scenario-specific health assessments for DBDPO have been conducted previously. The World Health Organization (WHO) evaluated the manufacture and formulation of DBDPO into polymers and concluded that exposure of the general population to DBDPO is insignificant (WHO 1994). The National Academy of Sciences (NAS) and the Consumer Product Safety Commission (CPSC) assessed the use of DBDPO in textiles (National Academy of Sciences 2000; Babich and Thomas 2001), and the European Chemicals Bureau examined exposures to DBDPO via the general environment (ECB 2002). These four evaluations concluded that exposures via the respective pathways did not pose any adverse health risks to the general population. While all four evaluations concluded that exposures to DBDPO were minimal and not likely to pose an adverse health risk, they did not specifically estimate exposures from all possible routes of exposure to DBDPO for children.

DBDPO Exposure of Children

In recent years, regulatory agencies have raised concerns about detection of certain polybrominated diphenyl oxide/ether (PBDPO, PBDE) congeners in the environment and in human adipose tissue, blood serum, and/or breast milk. Of particular concern is the exposure of infants and young children to these chemicals and the associated risk of adverse effects during early stages of development. As a result, the USEPA included DBDPO in the Voluntary Children's Chemical Evaluation Program (VCCEP). The VCCEP was "designed to provide data to enable the public to better understand the potential health risks to children associated with certain chemical exposures" (Federal Register, December 26, 2000, 65 FR 81700), and requires sponsors to provide information on hazards and exposures, and to conduct a risk assessment and data needs assessment. Decabromodiphenyl oxide was one of the 21 compounds selected for consideration in the VCCEP.

The objective of this study was to quantify the potential levels of exposure to DBDPO experienced by children, in a manner consistent with the VCCEP Tier 1 guidance. A Tier 1 exposure assessment is "screening" in nature, relying on "readily available data" and what are generally considered conservative assumptions. If no excess risks are predicted in the Tier 1 assessment, then it may be inferred that further research is not required to assess the potential risks for this compound, and thus, a Tier II assessment would not be required. The focus of this article is the exposure assessment, and the inputs used to calculate intake, which is then combined with the recommended reference dose (RfD) to calculate risks. The toxicology data presented under the VCCEP program are described only briefly here (see <http://www.tera.org> for the VCCEP document on DBDPO), and data needs (which are an important component of the VCCEP program) are not addressed in this paper.

There are few data available on DBDPO in environmental media and food, which may be due to a variety of factors. When DBDPO is included in the analyte list, DBDPO concentrations are often below detection limits, or are detected at very low concentrations. Additionally, the analytical methods to quantify DBDPO at very low concentrations are complex. There are some biomonitoring data on DBDPO in humans. These data (blood) can reduce uncertainties associated with exposure calculations, and by definition, they account for intake via all possible avenues of exposure, whether or not the avenues are identified or clearly understood. This approach is valuable, particularly in a screening-level exposure assessment where limited data exist on the compound in the environment. 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.

METHODS

This child-specific exposure assessment for DBDPO followed the Tier I guidelines described in the VCCEP. Exposures affecting children are defined by the VCCEP as those occurring prior to conception (via either parent), during prenatal development, and postnatally to the age of sexual maturation (defined as 18–21 years of age) (USEPA 2002). The VCCEP stipulates that assessments should identify and account

for the sources and pathways of exposure, the chemical concentrations in the various media, the frequency and duration of exposure, and the number of children potentially exposed.

The VCCEP guidance suggests that a Tier 1 exposure assessment should contain screening-level information on exposure from manufacturing, supplemented with relevant screening-level data on downstream processing and use activities and specific information on children's exposures. The screening approach generally relies on measured data, existing release and exposure estimates, and other exposure-related information. The assessment is expected to generate conservative, quantitative estimates of exposure.

Although substantial data are available regarding the environmental concentrations of the lower brominated diphenyl oxides (BDPOs, PBDEs), there are relatively few data for concentrations of DBDPO (Table 1). This is because DBDPO is far less environmentally prevalent (WHO 1994), and in the few studies in which DBDPO was included as an analyte, it was usually not detected (Hardy 2000). The low prevalence cannot be ascribed to DBDPO being degraded into lower BDPOs, because there is no evidence that it is biotransformed in the environment (WHO 1994), or that it photodegrades in water (Norris *et al.* 1974, 1975). Similarly, DBDPO is not metabolized to lower BDPOs in the mammalian system (Mörck and Klasson-Wehler 2001). Therefore, breakdown products of DBDPO were not considered in this assessment. To surmount the potential uncertainties and limitations associated with few environmental data, this assessment relied primarily on biomonitoring data (i.e., serum concentrations of DBDPO).

EXPOSURE PATHWAYS AND SCENARIOS

Based on the use patterns of DBDPO, the following are six plausible pathways through which children may be exposed:

Child (0–2 years) ingesting breast milk from a mother who is occupationally exposed to DBDPO. Two possible scenarios (for which some data exist) are:

1. A mother who manufactures DBDPO, and
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,

Table 1. Measured concentrations of DBDPO in the environment, biota, and humans in the U.S.

Medium	Sample size	Concentration	Unit	Location	Reference
Sediment	—	<100–14000	$\mu\text{g}/\text{kg}$	U.S. ^a	Zweidinger <i>et al.</i> 1979
Sediment	1	13	$\mu\text{g}/\text{kg}$ dry wt	U.S., Lake Ontario	Alaee 2001
Sludge	11	<75–9160	$\mu\text{g}/\text{kg}$ dry wt	U.S.	Hale <i>et al.</i> 2001
Sludge	1	1470	$\mu\text{g}/\text{kg}$	U.S.	Hale <i>et al.</i> 2002
Air	10	0.016–26	$\mu\text{g}/\text{m}^3$	U.S. ^a	Zweidinger <i>et al.</i> 1977
Air	24	<0.0000010	$\mu\text{g}/\text{m}^3$	Rural U.S. Great Lakes	Strandberg <i>et al.</i> 2001
Air	12	0.00000020–0.00000035 ^b	$\mu\text{g}/\text{m}^3$	Urban Chicago	Strandberg <i>et al.</i> 2001
Fish (salmon)	2	<0.00065	ng/g wet wt	Alaska	Easton <i>et al.</i> 2002
Fish	20	<1.5	ng/g wet wt	3 U.S. lakes	Dodder <i>et al.</i> 2002
Fish (carp)	48	<0.00065	ng/g wet wt	U.S. rivers	Loganathan <i>et al.</i> 1995
Chicken fat	13	0.44–3.35 ^c	ng/g whole wt	Southern U.S.	Huwe <i>et al.</i> 2002
Human adipose	5	ND–0.7	ng/g lipid	U.S.	Cramer <i>et al.</i> 1990
Human serum	12	<0.96–33.6	ng/g lipid	U.S.	Stanley <i>et al.</i> 1991
Human hair	3	5 ^d	ng/g	Arkansas	Sjödín <i>et al.</i> 2001b DeCarlo 1979

ND = not detectable.

^aNear manufacturing site.

^bRange of annual average concentrations.

^cResults are uncertain because DBDPO was detected at high levels in blanks, and because reported units for blank results (ng) are inconsistent with concentration units for sample results (ng/g), making interpretation difficult.

^dMaximum of three composite samples.

6. Child (0–18 years) being exposed to DBDPO via the general environment (e.g., eating food, incidentally ingesting soil and dust, breathing ambient air, and drinking water).

These six exposure pathways were combined into three exposure scenarios, which aggregated all exposures for a given population:

1. Child (0–2 years) ingesting breast milk from a mother who manufactures DBDPO, mouthing DBDPO-containing plastic electronic products, mouthing DBDPO-containing fabric, and being exposed via the general environment,
2. Child (0–2 years) ingesting breast milk from a mother who disassembles electronics, mouthing DBDPO-containing plastic electronic products, mouthing DBDPO-containing fabric, and being exposed via the general environment,
3. Child (>2–18 years) being exposed via the general environment.

The VCCEP guidance suggests that hazard information should guide the selection of the exposure scenarios to be evaluated. Because DBDPO has not been shown to produce reproductive or developmental effects (Hardy *et al.* 2002; Norris *et al.* 1975; BFRIP 2002), exposures to prospective parents and *in utero* exposures were not included in this evaluation. A more detailed discussion of these tests is provided in the assessment submitted to VCCEP (BFRIP 2002).

EXPOSURE FACTORS, ASSUMPTIONS, AND PATHWAY-SPECIFIC INTAKES

A substantial number of parameters need to be considered in any exposure assessment. The sources of information for conducting these analyses can be drawn from numerous disciplines, including pediatrics, toxicology, physiology, health physics, industrial hygiene, and various social sciences, as well as the recent specialty areas of exposure assessment and risk assessment (Paustenbach 2002). The source of each exposure factor used in these calculations is cited in the following sections and in Tables 2–7. The symbols for the exposure factors and the formulas for calculating the daily intakes for each exposure pathway are also shown in Tables 2–7.

Exposure Pathway 1 (Via Breast Milk From Mother Who Manufactures DBDPO)

One of the most important exposure pathways for children exposed to persistent chemicals is via breast milk. Decabromodiphenyl oxide is expected to transfer only minimally into breast milk due to its low bioavailability and other properties. For example, of the amount that a mother might ingest, less than 2% is expected to be absorbed from the gastrointestinal tract (El Dareer *et al.* 1987). Further, DBDPO's large molecular size and other physio-chemical properties most likely limit the amount that can transfer from serum into breast milk (BFRIP 2002).

Table 2. Estimated DBDPO intake by an infant ingesting breast milk from a mother who manufactures DBDPO

Exposure parameters	Mid-range estimate (birth to 3 months)		Upper estimate (birth to 2 years)	
	Value	Source/comment	Value	Source/comment
C_a : DBDPO concentration in workplace (mg/m^3)	1	ECB 2002	5	WEEL (AIHA 1996)
CF_{a-s} : Air/serum transfer factor ($\mu\text{g}/\text{g}$ lipid serum) per (mg/m^3 air)	27.4	Sjödín <i>et al.</i> 1999; Sjödín <i>et al.</i> 2001a	27.4	Sjödín <i>et al.</i> 1999; Sjödín <i>et al.</i> 2001a
R_{b-m} : Breast milk to serum ratio (unitless)	0.1	Based on the fact that higher brominated DPO's do not partition into milk as effectively as lower brominated DPO's	0.5	Conservative assumption that DBDPO partitions from serum into breast milk on a lipid weight basis at the ratio that hepta-DPO does
F_{lipid} : Fraction of breast milk that is lipid (g/mL)	0.04	4% expressed as g/mL (Table 2-12, USEPA 2002)	0.04	4% expressed as g/mL (Table 2-12, USEPA 2002)
CF: Conversion factor ($\text{mg}/\mu\text{g}$)	0.001		0.001	
IR: Ingestion rate, breast milk (mL/day)	742	Mean for ages 1-6 months (Table 2-12, USEPA 2002)	980	12-month average, upper percentile (Table 2-12, USEPA 2002)
ABS: Absorption (percent)	100% ^a	Necessary to use with toxicity value	100% ^a	Necessary to use with toxicity value
BW: Body weight (kg) and 0-3 months (ME) and 0-2 years (UE)	4.36	Average of 50th percentile weights, birth through 3 months (Table 11-1, USEPA 2002)	7.84	Average of 50th percentile weights, birth through 24 months (Table 11-1, USEPA 2002)
Daily Intake ($\text{mg}/\text{kg}\cdot\text{day}$)	0.019	Calculated	0.34	Calculated

$$\text{Intake} = \frac{C_a \times CF_{a-s} \times R_{b-m} \times F_{\text{lipid}} \times CF \times IR \times ABS}{\text{BW}}$$

^aAlthough the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations, because the toxicity value is based on an ingested dose rather than an absorbed dose. See text for details.

Table 3. Estimated DBDPO intake by an infant ingesting breast milk from a mother who disassembles electronics

Exposure parameters	Mid-range estimate (birth to 3 months)		Upper estimate (birth to 2 years)	
	Value	Source/comment	Value	Source/comment
C _b : DBDPO concentration in mother's blood (ng/g lipid)	4.8	Median for computer disassembly workers in Sweden (Sjödin <i>et al.</i> 1999)	9.9	Highest level reported for computer disassembly workers in Sweden (Sjödin <i>et al.</i> 1999)
R _{b-m} : Breast milk to serum ratio (unitless)	0.1	Based on the fact that higher brominated DPO's do not partition into breast milk as effectively as lower brominated DPO's (see text)	0.5	Conservative assumption that DBDPO partitions into breast milk and serum on a lipid weight basis at the ratio that hepta-DPO does (BDE-183) (see text)
F _{bbm} : Fraction of breast milk that is lipid (g/mL)	0.04	4% expressed as g/mL (Table 2-12; USEPA 2002)	0.04	4% expressed as g/mL (Table 2-12, USEPA 2002)
CF: Conversion factor (mg/ng)	1E-06		1E-06	
IR: Ingestion rate, breast milk (mL/day)	742	Mean for ages 1-6 months (Table 2-12, USEPA 2002)	980	12-month average, upper percentile (Table 2-12, USEPA 2002)
ABS: Absorption (percent)	100% ^a	Necessary to use with toxicity value	100% ^a	Necessary to use with toxicity value
BW: Body weight, (kg)	4.36	Average of 50th percentile weights, birth through 3 months	7.84	Average of 50th percentile weights, birth through 24 months (Table 11-1, USEPA 2002)
Daily Intake (mg/kg-day)	3.3E-06	Calculated	2.5E-05	Calculated

$$\text{Intake} = \frac{C_b \times R_{b-m} \times F_{bbm} \times CF \times IR \times ABS}{BW}$$

^aAlthough the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations, because the toxicity value is based on an ingested dose rather than an absorbed dose. See text for details.

Table 4. Estimated DBDPO intake by an infant mouthing DBDPO-containing electronics

Exposure parameters	Mid-range estimate		Upper estimate	
	Value	Source/comment	Value	Source/comment
C _L : Mass of DBDPO leached from surface into liquid per day (mg/day)	0.15	Norris <i>et al.</i> 1974. No DBDPO was extracted from ABS terpolymer in water for 1 day at 120°F. This value is the limit of detection (0.075 mg/L) multiplied by the total volume (2 L) divided by the total number of days (1 day).	0.29	Norris <i>et al.</i> 1974. Extraction of DBDPO from ABS terpolymer in cottonseed oil at 135°F for 7 days. This value is the concentration of DBDPO leached (1 mg/L) multiplied by the total volume (2 L) divided by the total number of days (7 days).
CF: Conversion factor (day/min)	6.9E-04	1 day has 1440 minutes	6.9E-04	1 day has 1440 minutes
MT: Mouthing time (min/day)	32.4	Total mouthing time, average of means for ages 3–18 months (Table 6–1, p. 6–12, USEPA 2002)	97.2	Total mouthing time, average of maximums for ages 3–18 months (Table 6–1, p. 6–12, USEPA 2002)
FS: Fraction of objects with DBDPO (percent)	1%	Professional judgment (see text)	10%	Professional judgment (see text)
ABS: Absorption (percent)	100% ^a	Necessary to use with toxicity value	100% ^a	Necessary to use with toxicity value
BW: Body weight, 0–2 years (kg)	7.84	Average of 50th percentile weights, birth through 24 months (Table 11–1, USEPA 2002)	7.84	Average of 50th percentile weights, birth through 24 months (Table 11–1, USEPA 2002)
Daily Intake (mg/kg-day)	4.3E-06	Calculated	2.5E-04	Calculated

$$\text{Intake} = \frac{C_L \times CF \times MT \times FS \times ABS}{BW}$$

^aAlthough the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations, because the toxicity value is based on an ingested dose rather than an absorbed dose. See text for details.

Table 5. Estimated DBDPO intake of young children inhaling particulates released from electronics

Exposure parameters	Mid-range estimate		Upper estimate	
	Value	Source/comment	Value	Source/comment
C_a : DBDPO concentration in air (respirable); vapor attaches to dust particulates in air (ng/m^3)	0.052	Office with computers (mean of all samples, using one-half the detection limit for nondetects) Sjödin <i>et al.</i> 2001a	0.087	Office with computers (highest value reported) Sjödin <i>et al.</i> 2001a
CF: Conversion factor (mg/ng)	1.0E-06		1.0E-06	
FI: Fraction of time spent in room with TV or computer (unitless)	0.83	20 hrs in 24-hr period (Hubal <i>et al.</i> 2000)	1	24 hrs in 24-hr period; professional judgment
IhR: Inhalation rate (m^3/day)	5.65	Average of <1 yr (4.5) & 1-2 yrs (6.8), means (Table 7-13, USEPA 2002)	5.65	Average of < 1 yr (4.5) & 1-2 yrs (6.8), means (Table 7-13, USEPA 2002) ^a
ABS: Absorption (percent)	100% ^b	Necessary to use with toxicity value	100% ^b	Necessary to use with toxicity value
BW: Body weight, 0-2 years (kg)	7.84	Average of 50th percentile weights, birth through 24 months (Table 11-1, USEPA 2002)	7.84	Average of 50th percentile weights, birth through 24 months (Table 11-1, USEPA 2002)
Daily Intake ($\text{mg}/\text{kg}\text{-day}$)	3.1E-08	Calculated	6.3E-08	Calculated

$$\text{Intake} = \frac{C_a \times \text{CF} \times \text{FI} \times \text{IhR} \times \text{ABS}}{\text{BW}}$$

^aOnly means are reported.

^bAlthough the absorption of DBDPO is estimated to be less than 2%, an absorption of 100% is necessary in the intake calculations, because the toxicity value is based on an ingested dose rather than an absorbed dose. See text for details.

Table 6. Summary of NAS (2000) results for DBDPO exposures from upholstery textiles

Exposure pathway	Noncancer intake	Noncancer hazard quotient	Comment/assumptions
Dermal—Adult (mg/kg-day)	9.8E-01	0.25	<ul style="list-style-type: none"> • Adult spends 1/4 of every day sitting on furniture that has upholstery backcoated with DBDPO • 1/4 of the adult's upper torso is in contact with upholstery • Adult's skin and clothing and upholstery fabric present no barrier to DBDPO movement • Sufficient sweat is present to allow dissolution of DBDPO and transfer to the skin and into the body • All DBDPO that dissolves is absorbed immediately • Same as dermal scenario above except assumes that DBDPO only dissolves up to its solubility limit in water
Dermal—Adult (mg/kg-day)	1.33E-09	3.34E-10	
Inhalation of Particulates—Adult (mg/m ³)	4.8E-04	0.000034	<ul style="list-style-type: none"> • Adult spends 1/4 of life in a room with low air-exchange rates • Room contains relatively large amount of fabric upholstery treated with DBDPO • DBDPO treatment is gradually wearing away over 25% of its surface to 50% of its initial quantity over the 15-year lifetime of the fabric • 1% of the worn-off DBDPO is released into indoor air as small particles that may be inhaled
Inhalation of Vapors—Adult (mg/m ³)	3.8E-04	0.0000271	<ul style="list-style-type: none"> • Same as particulate scenario above except assumes that DBDPO is released by evaporation
Oral-Child, 0-2 yrs (mg/kg-day)	2.6E-02	0.0065	<ul style="list-style-type: none"> • Child mouths fabric backcoated with DBDPO for 1 h per day, daily, for 2 years

Table 7. Estimated DBDPO intake of children via the general environment

Input parameters	Mid-range estimate		Upper estimate	
	Value	Source/comment	Value	Source/comment
C _{ss} : Concentration in body, steady state (ng/g lipid)	0.96 ^a	Median (Sjödín <i>et al.</i> 2001b)	33.6 ^a	Maximum (Sjödín <i>et al.</i> 2001b)
FL: Fraction of body weight into which DBDPO partitions (kg lipid/kg BW)	0.25	Used by USEPA Dioxin Reassessment (USEPA 2000)	0.5	Upper-end estimate
CF ₁ : Conversion factor 1 (g lipid/kg lipid)	1E+03		1E+03	
CF ₂ : Conversion factor 2 (mg/ng)	1E-06		1E-06	
Ln(2): Natural log of 2 (unitless)	0.693		0.693	
t _{1/2} : Half life of chemical (days)	6.8	Mean (Sjödín 2000)	3	Lower bound on calculated confidence interval (Sjödín 2000)
ADD: Average daily dose (absorbed) (mg/kg-day)	0.00046	Calculated	0.073	Calculated
ABS: Absorption (percent)	2%	El Dareer <i>et al.</i> 1987	1%	In this equation, use of a lower ABS will result in a higher intake estimate.
Daily Intake (mg/kg-day)	1.2E-03	Calculated	3.9E-01	Calculated

$$\text{ADD}_{\text{absorbed}} = C_{\text{ss}} \times \text{FL} \times \text{CF}_1 \times \text{CF}_2 \times \frac{\text{Ln}(2)}{t_{1/2}}$$

$$\text{Intake} = \frac{\text{ADD}_{\text{absorbed}}}{\text{ABS}}$$

^aValues converted from pmol/g lipid to ng/g lipid using the formula: (pmol/g lipid) × (959 g/mol) × (1 mol/10¹² pmol) × (10⁹ ng/1 g) = ng/g lipid.

DBDPO Exposure of Children

Although some other flame retardants have been detected in breast milk, because there have been no published concentrations for DBDPO, the exposures via this pathway were estimated indirectly. To estimate the milk concentration, a workplace air concentration was identified, an air-to-serum ratio was calculated, and then a partition coefficient value was selected to represent the amount of DBDPO that might transfer from the serum into the breast milk.

Workplace exposure to DBDPO may occur during manufacturing or formulation into the resin or liquid polymer dispersion. Decabromodiphenyl oxide is manufactured in a closed system by the reaction of bromine with diphenyl oxide. The highest exposure potential is likely associated with the activities of packaging DBDPO for shipping, or of emptying bags into a hopper for product formulation. Once formulated, DBDPO is encased in a polymer matrix, significantly reducing the potential for worker exposure. The American Industrial Hygiene Association (AIHA) has established a Workplace Environmental Exposure Level (WEEL) of 5 mg/m³ for DBDPO (AIHA 1996). The U.S. Occupational Safety and Health Administration (OSHA) has not set a Permissible Exposure Level (PEL) for DBDPO. However, because it is considered a "nuisance dust," it is subject to a 5 mg/m³ limit. Early industrial hygiene surveys identified employee 8-hour, time-weighted average (TWA) exposures to DBDPO to be in the 1- to 4-mg/m³ range, with possible excursions as high as 42 mg/m³ during certain tasks (AIHA 1996). During activities that generate dust concentrations greater than 5 mg/m³, respirators are expected to be worn in the work environment (AIHA 1996).

The potential intake of DBDPO that might be incurred by a nursing infant due to the exposure of a working mother was considered the most extreme scenario for uptake. A woman engaged in bagging DBDPO during manufacture, or in emptying bags of DBDPO into hoppers for formulators and compounders, was assessed, because this task is believed to produce the highest possible exposure. Because there is a lack of workplace air data in the U.S., an upper limit of exposure (UE) of 5 mg/m³ was selected based on the WEEL. A mid-range estimate (ME) of 1 mg/m³ was based on a European Union study that concluded that the majority of workplace air levels were below 1 mg/m³ (ECB 2002). Workplace air concentrations and associated blood levels for Swedish workers in an electronics recycling plant were used to estimate an air:serum ratio, which was then combined with estimates of air concentrations for a U.S. worker to derive a hypothetical serum level. The mean air level (n = 2) was 175 ng/m³ (Sjödin *et al.* 2001a) and the median serum concentration of DBDPO (n = 19) was 4.8 ng/g lipid (Sjödin *et al.* 1999), yielding a ratio of 27.4 ($\mu\text{g DBDPO/g lipid}$) per (mg DBDPO/m^3). It is acknowledged that this is a very limited data set for calculating this ratio, but it appears reasonable based on the properties of DBDPO.

To estimate the transfer of DBDPO from serum to breast milk, data regarding the partitioning of other PBDPO congeners from serum to breast milk were used. Available data on the levels of other PBDPOs in breast milk (Ryan and Patry 2001) and serum (Sjödin *et al.* 2001b) were used to calculate a ratio of breast milk to serum (i.e., for each congener, the breast milk concentration was divided by the serum

concentration). These ratios were <1.5 for the various PBDPOs, and decreased with increasing molecular weight/bromination, with a value of 0.54 determined for hepta-BDPO (the highest brominated PBDPO for which breast milk concentrations have been published). Thus, a UE for breast milk:serum ratio of 0.5 was selected. Because the higher PBDPOs have a lower transfer rate to breast milk, using the ratio derived for hepta-BDPO may be a high-end estimate. A ratio of 0.1 was selected as an ME, because it is likely that DBDPO will partition into breast milk with even greater difficulty due to its greater degree of bromination and higher molecular weight and size. The fraction of breast milk that is lipid was assumed to be 4% (USEPA 2002). A UE breast milk ingestion rate of 980 mL/day was selected based on the upper-percentile value for a child <1 year old (USEPA 2002). No ingestion rates for 1- to 2-year-old children were presented, but they are known to decrease after the age of 9 months (USEPA 2002). Therefore, using the 12-month average value to represent the entire 2-year period would likely overestimate actual exposures. An ME ingestion rate of 742 mL/day was used, based on the mean value for children 1–6 months of age (USEPA 2002). For the ME, an infant was assumed to breastfeed from the exposed mother daily from birth to 3 months of age (Collaborative Group on Hormonal Factors in Breast Cancer 2002), and for the UE, daily from birth to 2 years. An ME of 4.36 kg and a UE of 7.84 kg for body weight were derived from the 50th-percentile weights for children from birth through 3 months and 2 years, respectively (USEPA 2002). Although the gastrointestinal absorption of DBDPO was estimated to be <2% (Hardy 2002), it was necessary to use 100% in these intake calculations, because the reference dose used in risk calculations is based on an ingested dose rather than an absorbed dose.

Using the information, assumptions, and formula presented in Table 2, the estimated daily intake for an infant exposed via ingesting breast milk from a mother who manufactures DBDPO ranged from 0.019–0.34 mg/kg-day for the ME and UE, respectively (Table 2). It is acknowledged that there is substantial uncertainty surrounding the methods used to calculate the serum levels, as well as the calculation of the percentage of DBDPO in the serum that would partition into the breast milk. However, there are enough published data on this class of chemicals and other persistent compounds to provide some level of confidence on the level of conservatism built into these calculations. These are addressed in the discussion.

Exposure Pathway 2 (Via Breast Milk From Mother Who Disassembles Electronics)

For this pathway, the mother of a breastfeeding infant is assumed to be an electronics disassembly worker. There are no U.S. data for either workplace air concentrations or serum levels of DBDPO for a disassembly worker. However, DBDPO was detected in the serum of Swedish workers engaged in dismantling electronic equipment (Sjödín *et al.* 1999; Sjödín 2000) and in Swedish computer technicians (Hagmar *et al.* 2000). Therefore, serum concentrations of DBDPO for a U.S. worker were assumed to be the same as those measured in the Swedish workers. The maximum level (9.9 ng/g serum lipid) reported by Sjödín *et al.* (1999) was used for the

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UE, and the median level (4.8 ng/g serum lipid) was used for the ME. Additionally, the DBDPO in the serum is assumed to partition into breast milk, as discussed above, which is fed to an infant daily from birth through 3 months (ME) and from birth through 2 years (UE).

Using the information, assumptions, and formula presented in Table 3, the estimated daily intake for a child exposed via ingesting breast milk from a mother who disassembles electronics ranged from 3.3×10^{-6} to 2.5×10^{-5} mg/kg-day for the ME and UE, respectively. There was less uncertainty regarding serum concentration than in the previous pathway because the values were obtained from published literature rather than calculated.

Exposure Pathway 3 (Child Mouthing Consumer Electronics Products)

Decabromodiphenyl oxide is used to flame-retard synthetic polymers in electrical and electronic equipment. A typical example of its use in the U.S. is in the cabinet backs of television sets, where DBDPO is used at a level of about 12% (WHO 1994). For this exposure pathway, DBDPO is assumed to leach from the surface of the electronic product into the saliva of the infant, and the infant is exposed by reingesting the DBDPO-containing saliva. Although it seems unlikely that children would mouth the types of electronic products that contain DBDPO, it was conservatively assumed that the possibility exists.

The available data indicate that DBDPO does not appear to leach very readily or to a significant degree from consumer electronic products. Norris *et al.* (1974) found no evidence of leaching from a pellet of acrylonitrile butadiene-styrene (ABS) terpolymer containing 4.25% DBDPO placed in 2 L of water for a full day at 120°F (detection limit = 0.075 mg/L). Similarly, no leaching was observed from the ABS pellets immersed in a 3% acetic acid solution at 120°F for 1 or 7 days. The only conditions under which any leaching was detected by these researchers were when ABS pellets containing DBDPO were suspended in cottonseed oil for 7 days at 135°F, with a resulting DBDPO concentration of 1 mg/L (Norris *et al.* 1974).

Hypothetically, the quantity of DBDPO that is leached from the plastic per day and is available to the child for uptake can be calculated by multiplying the concentration (mg/L) in the leachate by the volume (L) of leachate and dividing by the total number of days that each experiment was conducted. For the UE, using a concentration of 1 mg/L in cottonseed oil, the volume of 2 L, over a period of 7 days, this calculation yields a value of 0.29 mg/day. For the ME, using the detection limit of 0.075 mg/L, the volume of 2 L, over a period of 1 day, this calculation yields a value of 0.15 mg/day. This calculation assumes that the rate of leaching is constant over the entire period, and that a smaller volume of liquid will leach a lesser amount of DBDPO. Because DBDPO is not extracted by either water or acetic acid, and is unlikely to leach from the plastic at all when mouthed by an infant, both the UE and the ME are likely to overestimate actual exposures.

Two other input parameters required for this pathway include estimates of the amount of time an infant might mouth objects, and the fraction of those objects

mouthed that would contain DBDPO. A UE value of 97.2 min/day of total mouthing time was selected based on an average of maximum values for 3- to 18-month-old children (USEPA 2002). This mouthing time encompasses all objects mouthed, including fingers and toys. An ME value of 32.4 min/day was selected based on an average of mean mouthing times (USEPA 2002). It is not certain what percentage of items in a typical household contain DBDPO flame-retardant polymer casings. However, it is reasonable to assume that the majority of items that children mouth are their fingers, toys, and small nonelectrical products. Therefore, 10% was assumed as the UE, and 1% as the ME, based on professional judgment.

Using the information, assumptions, and formula presented in Table 4, the estimated daily intake for a child exposed via mouthing DBDPO-containing electronic products ranged from 4.3×10^{-6} to 2.5×10^{-4} mg/kg-day for the ME and UE, respectively. There was a significant amount of uncertainty surrounding the amount of DBDPO that could actually leach out of treated plastics (if at all). Some recent findings from an unpublished study of levels of DBDPO in household dust provide another means of calculating exposures via the likely route by which DBDPO might be liberated from consumer products, and these are addressed in the discussion section.

Exposure Pathway 4 (Children Inhaling DBDPO Particulates in Air)

For this exposure pathway, the intake of DBDPO by children who inhaled particulates released from plastic electronic products was evaluated. Because of its low vapor pressure and the partial encapsulation, DBDPO encased in hard plastic has a very low tendency to volatilize from this matrix into indoor air. Despite this, it has been reported that DBDPO was measured in the air (as a particle phase and not as a semi-volatile) in an office in which computers were used (Sjödin *et al.* 2001a). A respirable air concentration of 0.087 ng/m³ was selected as a UE based on the highest value reported, and 0.052 ng/m³ was selected as an ME based on the mean of all samples reported, using one-half the detection limit for nondetected concentrations from this study (Sjödin *et al.* 2001a). It is acknowledged that, in fact, the results from this study may have been an artifact, but are used here since this is a screening assessment.

An inhalation rate of 5.65 m³/day was adopted in our calculations based on the average of the mean value presented for children <1 year old (4.5 m³/day) and the mean for children aged 1–2 years (6.8 m³/day). No medians or high-end values were presented for this parameter, so the same value was used as both the UE and the ME (USEPA 2002). As a UE, it was assumed that an infant spends all 24 hours per day in a room with a TV or computer, yielding a fraction of 1. A fraction of 0.833 was selected as the ME, assuming the child spends 20 hours of a 24-hour period in a room with computers or TVs (Hubal *et al.* 2000).

Using the information, assumptions, and formula presented in Table 5, the estimated daily intake of DBDPO by a child exposed via the inhalation of particulates from plastic electronic products ranged from 3.1×10^{-8} mg/kg-day to

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6.3×10^{-8} mg/kg-day for the ME and UE, respectively (Table 5). Even adopting what we considered to be the rather conservative assumptions discussed above, the calculated intakes via this pathway were orders of magnitude less than the intakes estimated via other pathways. Therefore, inhalation of DBDPO in the household was deemed to contribute only minimally, if at all, to the total plausible daily intake. Because the intake from this pathway was so small, and these exposures would be included in the values calculated in the general exposures pathway analysis, this value was not included in the aggregate intake calculations.

Exposure Pathway 5 (Child Mouthing Fabric)

The NAS recently conducted an exposure and risk assessment for fire retardants used in upholstery textiles (NAS 2000). The results from the NAS are summarized in Table 6.

The analyses by the NAS suggest that exposure to DBDPO via contact with fabrics was minimal. They also indicated that their estimates of exposure were extremely conservative, and it was their opinion that the intake of DBDPO by children did not warrant concern from a human health risk perspective. The NAS (2000) concluded that no further evaluations were justified for DBDPO in flame-retardant upholstery textiles. For this assessment, the intake of 0.026 mg/kg-day, calculated for children's exposure to DBDPO via mouthing of upholstery, was selected and included in the aggregate intake.

Exposure Pathway 6 (Intake By Children From the General Environment)

There are only limited data on DBDPO in the environment and food items in the U.S. An exhaustive literature review revealed only a few studies reporting analyses of environmental media or food items for DBDPO content, and in most instances, DBDPO was not detectable (Table 1). Because there are such limited environmental and dietary data, the use of a standard intake calculation could significantly under- or overestimate potential exposures. For example, there are no data available for DBDPO in beef or dairy products, and the available published data for chicken fat (Huwe *et al.* 2000) are highly uncertain (because of the high detection limits and incorrect units reported in the study for the blanks). However, DBDPO has been detected in the body tissues of U.S. residents (Cramer *et al.* 1990; Stanley *et al.* 1991; Sjödin *et al.* 2001b), indicating that at least some individuals have been exposed to and have absorbed DBDPO.

To overcome some of the uncertainties and limitations that could be associated with calculating intake from a small data set on DBDPO in environmental media, intake of DBDPO via the general environment (*i.e.*, for intakes not associated with infant-specific behaviors such as breast milk ingestion or mouthing) was quantified using measured serum levels. In this approach, the serum concentrations of DBDPO reported in the literature were assumed to represent the steady-state blood level resulting from the amount of DBDPO that a child might absorb via all

possible general (*i.e.*, noninfant-specific) exposure pathways, including inhalation of indoor and outdoor air; ingestion from food or drinking water and incidental ingestion and dermal contact with soils, sediments, or dust. Using this approach, one accounts for all routes of exposure from all media, whether or not the pathway has been identified, is clearly understood, or has been adequately measured. For this reason, especially in occupational health, there has been significant emphasis placed on the importance of biological monitoring whenever it can be meaningfully applied.

The concentrations used to represent the steady-state body concentration for this calculation were taken from the one study that reported serum levels of DBDPO for individuals in the U.S. (Sjödín *et al.* 2001b). This study provided summary results for 12 samples taken from U.S. blood donors in 1988. Concentrations ranged from <1 to 35 pmol/g lipid, with a median concentration of <1 pmol/g lipid. Seven of the 12 samples were nondetects. After converting to the appropriate units, the median (0.96 ng/g lipid) and maximum (33.6 ng/g lipid) serum concentrations were used for the ME and UE calculations, respectively.

Calculating the absorbed dose of DBDPO that would produce the above-mentioned measured serum DBDPO in humans requires information on the half-life, fraction of body weight in which DBDPO partitions, and bioavailability of DBDPO in humans. The average half-life of DBDPO was reported to be 6.8 days (confidence interval of 3–12 days) in a study of workers with workplace exposure to DBDPO (Sjödín 2000). An average biologic half-life in humans of 6.8 days was used as the ME, and the lower-bound value of 3 days was used as the UE. Estimates for the fraction of body weight in which DBDPO partitions were based on the partitioning behavior of DBDPO in the various body tissues. In rats dosed orally with DBDPO, concentrations of DBDPO were higher in the liver than in adipose tissue, and concentrations in other tissues and muscle were much lower than in adipose tissue and liver (El Dareer *et al.* 1987). A value of 25% was used as the ME, representing the average body fat content for the general population. A value of 50% was used as a UE, because DBDPO does not partition in the lipid fraction exclusively. The oral bioavailability of DBDPO in rats was <2% (El Dareer *et al.* 1987). There are no data on inhalation bioavailability, and only *in vitro* data on dermal bioavailability (Hughes *et al.* 2001). Values of 2% and 1% were used as the ME and UE, respectively, for percentage absorbed (by all routes). In this instance, intake is back-calculated from an absorbed dose, so a lower absorption factor provides a higher intake estimate (*i.e.*, more conservative assumption), because the absorption factor is in the denominator of the equation.

Using the information, assumptions, and formula presented in Table 7, the estimated daily intakes for persons in the community due to exposures via all media were 1.2×10^{-3} and 3.9×10^{-1} mg/kg-day for the ME and UE, respectively. Because the majority of the serum samples tested had nondetectable levels of DBDPO, it is likely that calculated intakes may overestimate actual exposures.

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Aggregate Exposures

Aggregate exposures potentially experienced by the three populations evaluated in this exposure assessment—the infant of a mother who manufactures DBDPO (infant, manufacturer), the infant of a mother who disassembles consumer electronics (infant, disassembler), and an older child’s exposures associated with DBDPO in the environment—were determined by summing intakes from all applicable exposure pathways for each receptor (Table 8). The infant of a mother who manufactures DBDPO could be exposed via ingesting breast milk, ingesting DBDPO while mouthing electronic consumer products, ingesting DBDPO while mouthing furniture fabric, and from general environmental exposures. The infant of a mother who

Table 8. DBDPO exposure estimates and hazard quotients

Daily intakes	Exposure duration (yrs)	Exposure estimate (mg/kg-day)		Hazard quotient (RfD = 4 mg/kg-day ^e)	
		Mid-range	Upper	Mid-range estimate	Upper estimate
Pathway-specific					
Ingestion, breast milk, manufacturer	0–2	1.9E–02 ^a	3.4E–01	0.005	0.09
Ingestion, breast milk, disassembler	0–2	3.3E–06 ^a	2.5E–05	8E–07	6E–06
Ingestion, consumer electronics	0–2	4.3E–06	2.5E–04	1E–06	6E–05
Ingestion, mouthing fabric (NAS)	0–2	2.6E–02	2.6E–02	0.007	0.007
General exposures	0–18	1.2E–03	3.9E–01	0.0003	0.1
Aggregate					
Infant, manufacturer ^b	0–2	0.046 ^b	0.76 ^b	0.01	0.2
Infant, disassembler ^c	0–2	0.027 ^c	0.41 ^c	0.007	0.1
Child ^d	(>2–18)	0.0012 ^d	0.39 ^d	0.0003	0.1

^aAssumes a shorter duration for nursing (0–3 months), based on Collaborative Group on Hormonal Factors in Breast Cancer 2002.

^bThis value incorporates the intakes for ingestion of breast milk from a mother who is a manufacturer, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.

^cThis value incorporates the intakes for ingestion of breast milk from a mother who is a disassembler, plus ingestion from consumer electronic products, ingestion from mouthing fabric, and general exposures.

^dThis value incorporates the intake from general exposures. See text for details.

^eThe RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data. The RfD for DBDPO, 4 mg/kg-day, was calculated by the NAS instead of using the current 1999 IRIS RfD (0.01 mg/kg-day). NAS calculated a revised RfD for DBDPO using the NTP 2-year bioassay results (NTP 1986), which were not available at the time of the IRIS derivation (1984–1985).

disassembles consumer electronics would have the same exposures, except that the mother's breast milk would contain a different amount of DBDPO. Children who were no longer breastfeeding or mouthing consumer products (>2–18 yrs) would be exposed only via the general environment.

As presented in Table 8, there is a difference of up to an order of magnitude between the ME and UE for the two infant scenarios, and a difference of two orders of magnitude between the ME and UE for the general environment scenario. This magnitude of difference between the ME and UE is not uncommon in screening level assessments. The highest estimated exposure (UE for the infant, manufacturer scenario) is 0.76 mg/kg-day. The lowest estimated exposure (ME for the child's exposure) is 0.0012 mg/kg-day. The large differences between the ME and UE estimates are indicative of the degree of uncertainty in these calculations. Despite the uncertainties inherent in these calculations, the UE estimates are likely to be significantly higher than the true exposures experienced by children in the U.S., because high-end or maximum values were used for many, if not nearly all, of the input parameters.

TOXICITY VALUE

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) which is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a no-observed-adverse-effect level (NOAEL), lowest-observed-adverse-effect level (LOAEL), or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data. The RfD for DBDPO used in this assessment, 4 mg/kg-day, was calculated by the NAS instead of using the RfD (0.01 mg/kg-day) from the USEPA's Integrated Risk Information System (IRIS). The NAS derived this revised RfD for DBDPO using the National Toxicology Program's (NTP's) 2-year bioassay results, which were not available at the time of the IRIS derivation (1984–1985). The more recent NTP study (NTP 1986) used by the NAS has several advantages over the study on which the IRIS RfD was based. The NTP study used more than one species (both rats and mice), a larger number of animals (50 versus 25 rats/sex/dose), a higher dose range, and they also used a product of higher purity, which is more representative of the commercial formulation currently being used. The purity of the product used for toxicity testing is a particularly compelling reason to rely on the NTP study over the study used by IRIS. The primary study relied upon by IRIS (Norris *et al.* 1975) used a mixture containing 77% DBDPO, 21.8% nonabromodiphenyl oxide, and 0.8% octabromodiphenyl oxide, whereas the NTP study used material containing 94–97% DBDPO. The Agency for Toxic Substances and Disease Registry (ATSDR) has derived a Minimal Risk Level (MRL) for PBDEs as a class of compounds, not specific to DBDPO. The RfD derived by the NAS is specific to DBDPO and is thus more appropriate for this assessment.

The RfD derived by the NAS is also comparable to the acceptable daily intake of 3.2 mg/kg-day calculated by the CPSC (Babich and Thomas 2001). All available

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information, including more recent published and unpublished studies, was reviewed, but it was determined that there was no information available to suggest that the RfD should be lower than the value of 4 mg/kg-day derived by the NAS based on the NTP studies. A more detailed discussion of the animal toxicity data can be found in both the NAS assessment (2000) and the assessment presented to the VCCEP review panel (BFRIP 2002).

RISK CALCULATION

For noncancer health effects, quantitative risk estimates are typically provided in the form of hazard quotients (HQs). The HQ represents the estimated exposure for a specific chemical divided by the RfD, expressed in mg/kg-day.

$$\text{Hazard Quotient} = \frac{\text{Intake}}{\text{RfD}}$$

If an HQ value is less than one, then it can reasonably be assumed that the chemical exposure does not pose a significant risk. As HQ values increase above one, the potential for an adverse effect increases. As shown in Table 8 and Figure 1, the HQs for ME scenarios range from 0.0003 to 0.01, and the HQs for the UE scenarios range from 0.1 to 0.2, 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 for DBDPO are significantly less than one, with the highest aggregate HQ being five times lower than one (0.2). As such, HQs indicate that the estimates of exposure shown here are unlikely to present an adverse health effect.

DISCUSSION

This screening-level exposure and risk assessment for DBDPO provides a framework that can be used to assess potential health risks for compounds for which limited environmental data are available, but some biomonitoring data exist for

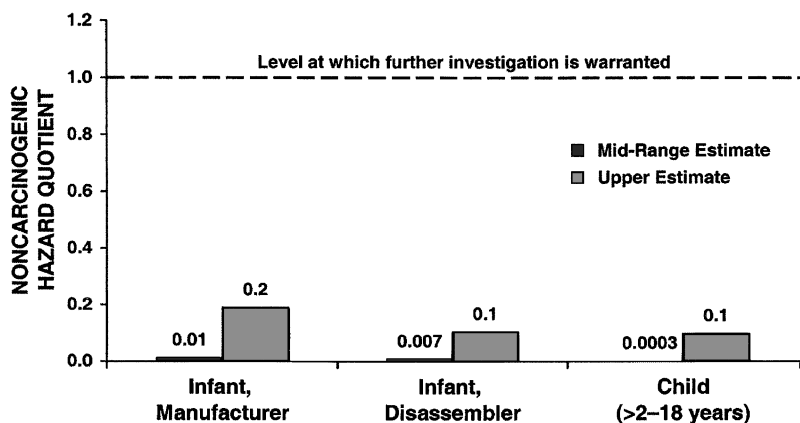


Figure 1. Hazard quotients for children's exposure to DBDPO.

humans. Relying on biomonitoring data to calculate exposures can reduce uncertainties associated with classic exposure calculations, because it bypasses the need for representative concentrations of the compound in environmental media and food, as well as the need to estimate the rates (how often and for how long) at which children, or adults, come in contact with these media. There is probably no better example of the usefulness of biological monitoring to characterize exposure than the experience with the dioxins and furans (a class of long-lived organic chemicals) (Aylward *et al.* 1996). The calculation using biomonitoring data accounts for intake via all exposure pathways, regardless of whether or not they are identified or clearly understood.

Like other screening-level risk assessments, this one was conducted in a manner that was intended to ensure that the exposures would almost certainly be overestimated, rather than underestimated. The MEs were intended to approximate, to the extent possible, the mean or median exposures for children in the U.S. Some assumptions made for the ME calculations are likely to result in estimated exposures that are greater than the actual mean or median; however, the limited availability of data on DBDPO precludes an assessment of how accurately the value represents actual exposures of children in the U.S. The UE can be characterized as being “conservative” in nature, because for nearly every input parameter, it is our view that rather extreme values were chosen, resulting in what we believe are significant overestimates of the actual exposure of the population. The limited database on DBDPO, however, prevents us from concluding with any quantitative certainty that the UE is a true “bound” on exposures. Although a quantitative uncertainty assessment could have been conducted, it was beyond the scope of this work and we expect that enough additional data will be collected within 2–3 years to make such an assessment unnecessary.

A comparison of our calculated concentrations with published data for breast milk concentrations from U.S. donors and the serum level data of DBDPO in workers in other countries suggests to us that the exposure estimates presented here are likely to significantly overestimate the true uptake by children in the U.S. For example, the concentrations of DBDPO in breast milk calculated in this exposure assessment, due to the numerous conservative assumptions that were selected, are as high as 70,000 ng DBDPO/g breast milk lipid. In contrast, the highest level of DBDPO ever measured in breast milk has been 8.24 ng/g lipid, with a mean of 0.92 ng/g lipid (Schechter *et al.* 2003). This value was obtained from a study that was published after the analysis presented here was submitted for publication. The findings from Schechter *et al.* (2003), however, were addressed by the peer-consultation panel that assessed DBDPO in early 2003. Therefore, the assumptions built into the calculations in our assessment appear to have generated estimates of intake perhaps as much as 1,000 fold greater than what is known today and may be considerably higher than what will be measured in the coming years.

Recently, some new information on DBDPO in indoor dust has been released (Rudel 2003, personal communication) that may give additional confidence that our assessment does not underestimate actual exposures. A total of five dust samples

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taken in Cape Cod, Massachusetts, were analyzed for DBDPO; four samples were from homes, and the fifth was from an office. Results ranged from 917 to 1,472 ng/g. Using the maximum dust concentration of 1,472 ng/g, and dust ingestion rates of 100 to 400 mg/day (USEPA 2002), one can estimate intakes of 1.9×10^{-5} and 7.5×10^{-5} mg/kg-day. The associated HQs are 4.7×10^{-6} and 1.9×10^{-5} . These values are several orders of magnitude lower than intakes estimated in our analysis for general environmental exposures. Dust concentrations in other areas such as California, which requires residential upholstery to meet fire safety standards, may be higher than those measured in Cape Cod. However, dust concentrations would have to be greater than 7,000,000 ng/g to equal the highest intake estimated in our so-called "general exposure pathway" (3.9×10^{-1} mg/kg-day). This large difference may indicate that dust is only a minor contributor to total environmental exposures, or it may reinforce the conclusion that the calculated intakes presented herein are overestimates of actual exposures.

There is other evidence that our approach is almost certainly yielding estimates that are greater than what will be observed in future studies. Specifically, in our assessment of breast milk ingestion from a mother who manufactures DBDPO, our highest estimated blood concentration for the mother was 137,000 ng DBDPO/g serum lipid (assuming a mother is exposed to 5 mg/m^3 DBDPO in the workplace air). This can be compared with 34.5 ng/g serum lipid, the highest reported level to date (in a worker assembling plastic-coated wires). Thus, we believe this is yet additional evidence that the compounding of conservative assumptions that is inherent in many screening-level assessments has likely occurred here as well (Nichols and Zeckhauser 1988; Paustenbach 1989; Burmaster and Harris 1993).

The VCCEP guidance suggests that the number of people potentially exposed to the compound of interest be discussed. This suggestion is consistent with generalized risk assessment guidance that the extent of the exposed population should be weighed when making decisions about the acceptability of individual levels of risk. For the two exposure pathways associated with occupational exposure, the number of people exposed is uncertain, but is expected to be relatively small. For example, there are only two DBDPO manufacturing facilities in the U.S and the available information indicates that no more than 50 employees work in the bagging operation and that none of them are women (BFRIP 2002). It is less certain how many employees (and more importantly, nursing mothers) might be involved in compounding DBDPO (incorporating DBDPO powder into a polymer matrix), disassembling consumer electronics, molding plastic casings, or handling flame-retardant textiles. Since DBDPO is used in such a wide variety of consumer products, it is likely that a majority of the U.S. population is exposed to DBDPO to some extent. Thus, the predicted exposures for the general environmental scenario could theoretically reflect exposures experienced by any portion of the U.S. population. However, more than half of the U.S. blood donors in the Sjödin *et al.* study (2001b) had nondetectable levels of DBDPO in their blood. Studies that are expected to be conducted in the near future will provide more insight on this issue.

The PBDEs have been heavily scrutinized of late because of greater frequency of detections in various environmental media. Due to public concern about the persistence of these chemicals, the European Union (EU) and California have promulgated rules that will phase out the production and use of pentabrominated diphenyl ether and octabrominated diphenyl ether products over the next 10 years. However, thus far, DBDPO has not been banned by any country, state, or government agency because there is a belief that DBDPO is different from the other PBDEs and that when properly used they yield significant fire-related benefits (e.g., preventing injuries and death) for society. Organizations that have recently evaluated the potential health risks associated with the specific use of DBDPO in consumer products have included the WHO, CPSC, the EU, and the NAS. Each of these organizations has concluded that DBDPO does not pose a health risk to humans. The assessment presented here, based on the available data, provides information that is consistent with their conclusions.

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