Exposure of Americans to Polybrominated Diphenyl Ethers. Part 1. Adult Exposures

Matthew Lorber, Office of Research and Development, United States Environmental Protection Agency, 1200 Pennsylvania Ave, NW,Washington, DC 20460

INTRODUCTION

The exposure of adult Americans to polybrominated diphenyl ethers, PBDEs, was systematically evaluated in Part 1 of this study. First, exposure media data on PBDE congeners were compiled. Then, an adult intake dose was derived using exposure factors in combination with these concentrations. The exposure pathways evaluated included food and water consumption, inhalation, and ingestion and dermal contact to house dust. These intakes were converted to a body burden using a simple pharmacokinetic (PK) model. The predicted body burdens were compared with representative profiles of PBDEs in blood and milk from the literature. Part 2 conducts similar exercises for infants and young children.

METHODS

The literature was surveyed for measurements of PBDE congeners in environmental/exposure media, in blood, and in breast milk. The congeners assessed include BDEs 28, 47, 99, 100, 138, 153, 154, 183, and 209. The final congener profiles were characterized as being representative of the US general population, where "general population" is defined as the population exposed primarily to background levels that occur in the home and normal work environments; general population exposures do not include occupational exposures. Key considerations for the final assignment of concentrations include: 1) the studies represent US conditions; 2) studies with a full suite of congeners including BDEs 183 and 209 are chosen over studies without these higher brominated congeners; 3) occupational data, while of interest, does not represent general population exposure and are not considered in this exercise; and 4) studies with a complete and appropriately background data set are chosen, even if they represent only one geographic region of the US. In the interest of expediency, the final selections of exposure media and body burden concentrations are provided in Table 1. A manuscript is being prepared which provides details and references on this and other data.

The final selected profile of exposure media congener-specific concentrations are combined with exposure factors to estimate congener-specific intake quantities in units of ng/day. The exposure factors used here were used by EPA to estimate background exposures to dioxin-like compounds in EPA's *Dioxin Reassessment* (EPA, 2003), with one important addition, as will be described shortly. In that assessment, as here, the intent was to characterize general population exposures to background concentrations of dioxin-like compounds. All of the exposure parameters and a brief description of the pathways are provided in Table 2. Further information on these assignments can be found in EPA (2003).

The important addition to the procedures originally laid out for dioxin-like compounds, applied here to PBDEs, is that inhalation and "soil-related" exposures of ingestion and dermal are assumed to occur primarily indoors rather than outdoors. This is because of the evidence that indoor air and house dust concentrations of PBDEs are significantly higher than outdoors, and indoor exposures appear critical for PBDEs. The

same is not true for dioxin-like compounds, where indoor and outdoor air and dust/soil are similar. This is because the primary sources of dioxin release into the environment are combustion-related and into the outdoor environment, whereas the primary sources of PBDE release are from their use in indoor products, and their subsequent release into the indoor environment. A weighted average of outdoor and indoor air, and outdoor soil and indoor house dust was determined by using an "indoor fraction" determined from time spent indoors, which was 0.90 for adults.

Intake doses were converted to body burdens using a simple, one-compartment pharmacokinetic model. The single compartment was body lipids, and the dissipation from that compartment was modeled using a first-order elimination rate. The steady state body lipid concentration modeled in this way is given as:

$$C_{BDE} = (D_{BDE} * ABS_{BDE})/(k_{BDE} * BL) \quad (1)$$

where C_{BDE} is the congener-specific/route-specific (CS/RS) lipid-based concentration (ng/g lwt), D_{BDE} is the CS/RS daily dose of BDE (ng/day), ABS_{BDE} is the CS/RS absorption fraction, BL is the body lipid mass (17.5 kg, or 25% of a 70-kg adult), and k_{BDE} is the congener-specific first-order dissipation rate in the body (day^{-1}). Elimination half-lives for lower brominated congeners were provided in Geyer et al (2005), while half-lives for BDEs 183 and 209 were provided in Thuresson et al (2006). These half-lives, shown in Table 2, were converted to elimination rates for use in Equation (1).

RESULTS

In addition to exposure media and body burden concentrations, Table 1 provides overall dose estimates for each congener, and predictions of body burden concentrations. Key observations and findings from this exercise include:

1) Although not described here, the literature search on exposure media concentrations and body burdens revealed that air (indoor/outdoor), soil, dust, and body burdens were significantly higher in the US as compared to Europe and elsewhere abroad, although food concentrations around the world appeared more comparable. However, there was an important finding which bears further examination, and that is, in a recent UK food survey published in 2006 (FSA, 2006), BDE 209 dominated all composite food types sampled getting as high as 3.6 ng/g wwt in meat products, and being at 0.1 to 0.3 ng/g wwt in other animal food products. In contrast, BDE 209 averaged near or under 0.1 ng/g wwt in all US animal food products.

2) The average adult intake was 518 ng/day, or 7.3 ng/kg-day, driven by indoor house dust exposures via ingestion and dermal contact; those two pathways accounted for about 86% of total intakes, with inhalation and food/water ingestion explaining the remaining 14%. Intakes for BDEs 47, 99, and 209 were equal at about 27% of total each.

3) The prediction of total body burden was low at 33.8 ng/g lwt, while it was observed at 64.0 ng/g lwt in blood and 93.7 ng/g lwt in milk in studies selected as representative of the general population. However, predictions were reasonably close to measurements for 8 of 9 congeners. The prediction of BDE 47 at 8.6 ng/g lwt did not match the observed measurements of 33.9 ng/g lwt in blood and 50.0 ng/g lwt in milk. The cause for the underprediction of BDE 47 is not known, but it could very easily be the assumed half-life in humans. At 3.0 years, it dissipated nearly twice as fast as BDE 99, which was assigned a half-life of 5.4 years.

4) The reasonable match of 8 of 9 congeners in this simple pharmacokinetic exercise lends credibility to the overall approach of estimating intakes from exposure media concentrations and contact rates, followed by use of the simple PK model to predict body lipid concentrations. Nonetheless, uncertainties exist, the most critical being limited environmental and body burden measurements more representative of regional rather than national trends, indoor contact rates with house dust, and the PK parameters of absorption of BDEs into the gut from house dust ingestion and dissipation half-life of BDEs in the body.

Disclaimer

The views expressed in this article are those of the authors and do not necessarily reflect the views or policies of the U.S. Environmental Protection Agency. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Bibliography

1. Allen et al. 2006. Personal and Indoor Air Exposure to PBDEs in US Urban Residences. Presented at, Dioxin 2006, held in Oslo, Norway, Aug 21-25, 2006..
2. CADAMP. California Ambient Dioxin Air Monitoring Network web page for polybrominated diphenyl ethers. 2006. http://www.arb.ca.gov/toxics/pbde.htm.
3. EPA. 2003. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. United States Environmental Protection Agency. NAS Review Draft. December, 2003. EPA/600/P-00/001C(a-f). http://www.epa.gov/ncea/dioxin.htm.
4. FSA, 2006. Brominated Chemicals: UK Dietary Estimates. Published by Food Standards Agency, UK, Information Sheet 10/06. http://www.food.gov.uk/science/surveillance/fsisbranch2006/fsis1006.
5. Geyer et al. 2004 *Organohalogen Compounds* 66: 3820-3825.
6. Lunder and Sharp. 2004. Mothers' Milk Record Levels of Toxic Fire Retardants Found in American mothers' breast milk. Environmental Working Group. http://www.ewg.org
7. Offenberg et al. 2006. Polybrominated diphenyl ethers in US soils. 2006. Presented at Dioxin 2006, held Aug 21-25 in Oslo, Norway.
8. Oros et al. 2005. *Env Sci and Tech* 2005: 39: 33-41.
9. Schecter et al. 2005. *J. Occup Env Med* 47: 199-211.
10. Schecter et al. 2006. *Env Health Persp* 114: 1515-1520.
11. Thuresson et al 2006 *Env Health Persp* 2006: 114: 176-181.

Table 1. Exposure pathways and factors for the PBDE intake dose estimate, and elimination rate constants and half-lives for pharmacokinetic modeling.

Exposure Factors; Units	Value	Exposure Factors; Units	Value	Congener	Elimination half-life, yr
body weight, kg	70	beef ingestion, g/d	49.7	28	6.0
soil ingestion, mg/d	50	poultry ingestion, g/d	35	47	3.0
soil dermal contact, mg/d	12	other meat, g/d	24.5	99	5.4
inhalation, m^3/d	13.3	fresh/marine fin fish, g/d	11.6	100	2.9
fraction indoor	0.875	fresh/marine shellfish, g/d	3.8	138	6.0
water ingestion, l/d	1.4	**Pharmacokinetic Parameters**		153	11.7
milk ingestion, g/d	175	ABS, absorption, soil ingestion, all congeners	0.50	154	5.8
dairy ingestion, g/d	55	ABS, absorption, dermal contact, all congeners	0.03	183	0.26
egg ingestion, g/d	16.8	ABS, absorption, food ingestion, inhalation, all cong	0.80	209	0.04

Table 2. Exposure media concentrations, body burdens, intake doses, and predicted body burdens of PBDEs.

BDE Congener	**28**	**47**	**99**	**100**	**138**	**153**	**154**	**183**	**209**	**Total**	**Reference**
I. EXPOSURE MEDIA CONCENTRATION											
Water, pg/l	3.3	42.7	27.6	7.2	0.3	3.9	2.9	4.4	42.3	134.6	Oros et al (2005)
Soil, ng/g dwt	---	1.9	3.6	0.4	---	5.7	4.8	37.4	15.3	69.1	Offerman et al (2006)
Indoor dust, ng/g	ND	1857	2352	911	181	243	156	60	2394	8154	Lunder & Sharp (2004)
Outdoor air, pg/m^3	3.0	53.0	51.0	13.0	---	3.9	4.0	1.4	25.0	154.3	CADAMP (2005)
Indoor air, pg/m^3	27	177	79	16	---	5	7	---	121	432	Allen et al (2006)
Shellfish, ng/g wwt	ND	3.6	1.2	0.9	ND	ND	ND	ND	ND	5.7	Oros et al (2005)
Finfish, ng/g wwt	0.03	0.60	0.17	0.13	0.001	0.02	0.05	0.002	0.09	1.09	Schecter et al (2006)
Beef, ng/g wwt	0.02	0.05	0.04	0.006	0.0001	0.006	0.004	0.001	0.003	0.13	Schecter et al (2006)
Pork, ng/g wwt	ND	0.08	0.12	0.015	0.001	0.02	0.01	0.009	0.02	0.28	Schecter et al (2006)
Poultry, ng/g wwt	0.0002	0.06	0.12	0.03	0.002	0.02	001	002	0.12	0.36	Schecter et al (2006)
Dairy, ng/g wwt	0.0002	0.03	0.03	0.005	<0.0001	0.004	0.002	0.002	0.04	0.11	Schecter et al (2006)
Eggs, ng/g wwt	0.0002	0.02	0.04	0.006	0.0001	0.004	0.003	0.0001	0.01	0.08	Schecter et al (2006)
II. INTAKE DOSE CALCULATIONS											
TOTAL, ng/day	1.9	133.2	148.0	57.0	10.0	15.6	10.1	4.1	137.9	517.9	
FRACTION	<0.01	0.26	0.29	0.11	0.02	0.03	0.02	0.01	0.27		
III. BODY BURDENS											
Predicted, ng/g lwt	0.3	8.6	15.6	3.3	1.1	3.7	1.1	0.02	0.1	33.8	
Blood, obs, ng/g lwt	1.4	33.9	10.8	5.2	0.2	9.9	0.8	0.4	1.4	64.0	Schecter et al (2005).
Milk, obs, ng/g lwt	3.8	50.0	10.0	12..0	---	16.0	0.8	0.3	0.8	93.7	Lunder and Sharp (2004).