Organohalogen Compounds 67: 505-508 (2005)

Estimating Human Exposure to PBDE-47 Via Air, Food and Dust Using Monte Carlo Methods

Thomas Webster[1], Veronica Vieira[1], Arnold Schecter[2]

[1]Boston University School Of Public Health
[2]University of Texas School of Public Health

Introduction

Polybrominated diphenyl ethers (PBDEs) are commonly used as fire retardants in consumer products. BDE 47 (2,2',4,4'-tetrabromodiphenylether), a semi-volatile, persistent and lipophilic compound, is typically the most prominent congener found in humans despite its minor contribution to commercial production and usage. Currently, the primary routes of exposure are unknown but are hypothesized to be dietary intake and exposure in the indoor environment, e.g., inhalation of indoor air. This study was designed to evaluate the roles of various potential routes of exposure to BDE 47. We compare estimated doses of BDE 47 via inhalation, animal-based foods and breast feeding, ingestion of dust, and dermal exposure to dust. These estimates are focused on US residents of three different ages: infants, young children and adults. In this initial evaluation, we use Monte Carlo methods based on measured levels in relevant media to provide central estimates as well as distributions.

Materials and Methods

We estimated distributions for BDE 47 concentrations in different media, relying as much as possible on US reports with individual data. We used data for indoor air measurements from Ottawa, Canada, as data from the USA were not available. These samples were collected using passive air filters which collect vapors and respirable-size particles.[1] We utilized house dust concentrations from four studies.[2-5] For breast milk concentrations, we combined data from four recent studies.[5-8] Concentrations in beef, pork and chicken were estimated from a recent US Department of Agriculture survey.[9] Concentrations in fish and dairy products were based on a study in Texas.[10] Air, dust and breast milk concentrations were approximately log-normally distributed. We assumed log-normal distributions for BDE 47 concentrations in meats and uniform distributions for the less well characterized dairy products and fish.

We used the USEPA's *Exposure Factors Handbook*[11] and related documents[12] for age-specific body weights and exposure factors: inhalation, diet (beef, pork, poultry, dairy products, fish, breast feeding), dust ingestion, and dermal exposure to house dust. We used exposure factors for three different ages: infants, young children, and adults.

Using BDE 47 dissolved in a vehicle, absorption rates in animal experiments were 86% (oral), 95% (intra-trachael), 77% (dermal).[13] No data are available for oral or dermal absorption of BDE 47 bound to dust (which presumably reduces bioavailability). We assumed the following point estimates: diet (90%), inhalation (90%), dust ingestion (50%), dermal absorption from dust (10%).

In these analyses, exposure means contact with body surfaces while dose includes route-specific absorption factors. We estimated age-specific dose for the four pathways using standard equations.[11] Dose distributions were constructed using single-stage Monte Carlo methods.[14] We ran ten thousand simulations for each age group using the Crystal Ball software package.[15] Means and various percentiles (10%, 50%, 90%) were computed for each age and pathway as well as totals.

We estimated steady state concentrations in lipid for adults assuming first order pharmacokinetics, a halflife of 4 years (extrapolated from animal data) and 30% of body weight as lipid.[16,17] Because of the long assumed half life for BDE 47, infants and young children probably do not reach a steady state body burden.

Results

Table 1 shows the estimated doses for the four pathways and three age groups. Infants receive the largest dose

followed by young children and then adults. We evaluated average contributions by the various routes at the mean dose (relative contributions may differ for individuals). Dose to infants is overwhelming due to diet. Diet dominates for adults, but dust exposure is also important. Ingestion of dust, dermal exposure to dust and diet play approximately equal roles in young children. Inhalation is a minor pathway for all groups. Total dose distributions were skewed to the right. As shown in Figure 1, the estimated total dose for adults is approximately log-normal.

Based on the median absorbed dose in Table 1, the estimated steady state concentration in adults was 5 ng/g lipid (11 ng/g for the 90th percentile dose). Assuming that lipid concentrations are equivalent in breast milk and adipose tissue, the median measured concentration of BDE 47 in adult women of child-bearing age in the four reviewed studies was 20 ng/g lipid.[5-8] The geometric standard deviation of the estimated body burdens was less than the observed value; inter-individual differences in pharmacokinetics may contribute to this additional variation .

Discussion

Our results suggest that both diet and indoor dust (ingestion and dermal) may be important sources of exposure to BDE 47. In comparison, inhalation appears relatively minor on average, although it may play a more important role in extreme cases. The balance between doses via diet and dust depends on age: diet appears most important for infants and adults; dust is relatively more important for young children. Conclusions regarding our dose estimates are limited by poor knowledge regarding exposure to indoor dust (particularly for adults), the small amount of data on PBDE concentrations in many media (particularly food and air), and the lack of data on absorption of BDE 47 bound to dust. Future research on these factors is needed. Our dose estimates could be improved by taking into account uncertainty in route-specific absorption; two-stage simulations provide a reasonable approach.[14] Nevertheless, recent epidemiological results suggests that both dust and diet may contribute to the body burdens of adults.[5]

The steady-state body burden estimated from the median dose for adults was about one fourth of the median body burden found in US women of reproductive age. This steady state estimate does not take into account uncertainty in pharmacokinetic parameters. The difference, if real, may be due to several factors including underestimation of the halflife of BDE 47 (extrapolated from animal data) and underestimation of dose. For example, we ignored exposure from non-animal based foods (for which virtually no data are available for the USA); the BDE 47 content of vegan diets was important in a British study.[18] Our results suggest that the skewed distribution of BDE 47 body burdens may be due in part to variation in exposure.

Acknowledgments

Dr. Vieira's participation in this project was supported in part by a grant from the New York Community Trust. Daniele Staskal and Linda Birnbaum provided helpful suggestions.

References

1. Wilford BH, Harner T, Zhu J, Shoeib M, Jones KC (2004). *Environ Sci Technol.* 38(20): 5312-8.

2. Stapleton HM, Dodder NG, Offenberg JH, Schantz MM, Wise SA (2005). *Environ Sci Technol.* 39(4):925-31.

3. Schecter A, Paepke O, Joseph JE, Tung KC (2005). *J Toxicol Environ Health A*. 68(7):501-13.

4. Sharp R and Lunder S (2004). *In the Dust: Toxic Fire Retardants in American Homes.* Environmental Working Group. www/ewg.org.

5. Wu N, Webster T, Herrmann T, Paepke O, Tickner J, Hale R, Harvey E, La Guardia M, Jacobs E (2005). Associations of PBDE levels in breast milk with diet and indoor dust concentrations. Submitted to Dioxin 2005.

6. Schecter A, Pavuk M, Paepke O, Ryan JJ, Birnbaum L, Rosen R (2003). *Environ Health Perspect*. 111(14):1723-9.

7. Lunder S and Sharp R (2003). *Mothers' Milk: Record levels of toxic fire retardants found in American mother's breast milk.* Environmental Working Group. www/ewg.org.

8. Williams-Derry C and Abbotts J (2004). *Flame Retardants in the Bodies of Pacific Northwest Residents.* Northwest Environment Watch. www.northwestwatch.org

9. Huwe, J (2004). Polybrominated diphenyl ethers in meat samples collected from supermarkets across the US. Presented at BFR 2004, Toronto, Ontario, Canada.

10. Schecter A, Paepke O, Tung KC, Staskal D, Birnbaum L (2004). *Environ Sci Technol*. 38(20):5306-11.

11. USEPA (1997). *Exposure Factors Handbook*. EPA/600/P-95/002Fa.

12. USEPA (2003). *CSFII Analysis of Food Intake Distributions*. EPA/600/R-03/029.

13. Staskal DF, Diliberto JJ, DeVito MJ, Birnbaum LS (2005). *Toxicol Sci*. 83(2):215-23.

14. Bogen KT, Spear RC (1987). *Risk Anal.* 7(4):427-36.

15. Decisioneering, Inc. www.decisioneering.com

16. Geyer H, Schramm K-W, Darnerud P, Aune M, Feicht E, Fried K, Henkelmann B, Lenoir D, Schmid P, McDonald T (2004). *Organohalogen Compounds* 66:3867-72.

17. McDonald TA (2004). Distribution of PBDE Levels Among U.S. Women: Estimates of Daily Intake and Risk of Developmental Effects. Presented at BFR 2004, Toronto, Ontario, Canada.

18. Harrad S, Wijesekera R, Hunter S, Halliwell C, Baker R (2004). *Environ Sci Technol.* 38(8):2345-50.

Table 1. Estimated absorbed dose (ng/kg/day) for infants, young children and adults via

four routes of exposure.

infants	10%	50%	90%	mean	mean %
dermal	0.1	0.6	3.3	1.5	1.2%
diet	20.9	66.1	259.9	117.9	95.1%
inhalation	0.0	0.0	0.2	0.1	0.1%
dust ingestion	0.2	1.3	9.7	4.5	3.6%
total*	25.1	72.3	267.0	123.9	100.0%
child	10%	50%	90%	mean	mean %
dermal	0.2	1.0	5.8	2.7	34.8%
diet	0.6	1.7	4.3	2.1	27.9%
inhalation	0.0	0.0	0.2	0.1	1.0%
dust ingestion	0.1	0.8	6.1	2.8	36.3%
total*	2.0	5.0	14.3	7.7	100.0%
adult	10%	50%	90%	mean	mean %
dermal	0.0	0.0	0.2	0.1	10.5%
diet	0.2	0.5	1.1	0.6	63.1%
inhalation	0.0	0.0	0.1	0.0	2.7%
dust ingestion	0.0	0.1	0.5	0.2	23.7%
total*	0.4	0.8	1.6	0.9	100.0%

* Means can be summed across routes; percentile data cannot.

Figure 1. Distribution of estimated total dose for adults (10,000 simulations)