Polybrominated Diphenyl Ether Concentrations in People

Ronald A. Hites

School of Public and Environmental Affairs, Indiana University, Bloomington, IN 47405, HitesR@Indiana.edu

Polybrominated diphenyl ethers (PBDEs) save lives by serving as flame retardants in a wide variety of commercial and household products. For example, polyurethane foam, which is widely used in upholstered furniture, is flammable unless it is treated with suitable flame retardants such as PBDEs. In fact, some polyurethane foam is treated with 10-30% by weight of PBDEs to make this material safe for home use.[1] Because many states and the federal government now have regulations requiring most household products, such as mattresses and electronics, to be flame resistant,[2] PBDEs have become an important commercial substance. Not surprisingly, the use of PBDEs has increased over the years, and annual sales are now exceed 70,000 metric tons.[3]

PBDEs are commercially available as three products, two of which are mixtures of several congeners.[4] The so-called penta-product contains 2,2',4,4'-tetrabromodiphenyl ether (BDE-47), 2,2',4,4',5-pentabromodiphenyl ether (BDE-99), 2,2',4,4',6-pentabromodiphenyl ether (BDE-100), 2,2',4,4',5,5'-hexabromodiphenyl ether (BDE-153), 2,2',4,4',5,6'-tetrabromodiphenyl ether (BDE-154), in a ratio of about 9:12:2:1:1.[5, 6] The octa-product contains several hexa- to nona-brominated congeners, and the deca-product is almost entirely composed of decabromodiphenyl ether (BDE-209).[4] Like most commercial chemical mixtures, the compositions of these products vary with manufacturer and with the year in which they were produced.

Despite their societal benefits, PBDEs seem to be migrating from the products in which they are used and entering the environment and people. PBDEs are now ubiquitous; they can be found in air, water, fish, birds, marine mammals, and people, and in many cases, the concentrations of these compounds are increasing over time.

A large number of samples from people have been analyzed for PBDEs. A recent review[7] gives these concentrations in ambient human tissue, blood (usually serum), and milk; these data were sorted by the year in which the sample was taken. In this case, "ambient" means samples from people who were not known to have been occupationally-exposed. ΣPBDE concentrations range from <0.03 ng/g lipid for adipose tissue from Japan in 1970 to >190 ng/g lipid for milk from Austin and Denver in the United States in 2000. A plot of all these concentrations vs. sampling year (see Figure 1) shows an exponential increase with a doubling time of ~5 years. In general, the PBDE concentrations in people have increased by a factor of ~100 during the last 30 years. The regression of

these data as a function of year is good despite the disparate sample types (blood, milk, and tissue), the different continents of origin, and the various congeners measured. This analysis shows that the North American samples are always above the regression line (in recent years by a factor of >10), and the Japanese samples are usually below the regression line (by a factor of ~5). This suggests that people in the U.S. are exposed to higher levels of these PBDE congeners than are Europeans and that the Japanese are exposed to less than the Europeans.

The variations in PBDE concentrations among populations can be examined in more detail using those published data that were given in sufficient detail to determine the statistical distribution of the concentrations. Figure 2 shows data from seven such data sets; this figure shows the 10th and 90th percentiles (the "whiskers") and the 25th and 75th percentiles (the boxes); the median and outliers are also shown. The median concentrations in the four United States samples (neonatal and maternal blood, tissue, and milk) are all about the same at ~35 ng/g lipid; the median concentration in the Vancouver (Canada) milk samples is somewhat lower than the United States value; and the Swedish neonatal and maternal blood concentrations are much lower at ~2 ng/g lipid. In other words, the concentrations of PBDEs are ~20 times higher in people from the U.S. as compared to people from Europe.

It is also interesting to note that all samples show a few high outliers. In North America, these outlier concentrations usually exceed 300 ng/g lipid, which is ~10 times the average level. The reason for these outliers is not yet clear.[8] In general, it is likely that the reason that the North American samples show such relatively high concentrations is the focused use of the penta-BDE product in North America. The U.S. and Canada used 7100 metric tons per year of the penta-BDE product, which was 95% of the world's production.[3]

Given that some PBDEs are used in plastics that end up in consumer electronics, one would expect that workers involved in assembling or disassembling these products would have an excess load of some PBDEs in their blood. In this case, the data are much less complete than for the ambient samples; thus, using these data to compare PBDE levels between exposed people and controls must be done on a congener-specific basis. For occupational samples, only BDE-47 and 153 have sufficient data to make this comparison. In these cases, the PBDE concentrations in the blood of the exposed workers were about twice that in the blood of the controls, and this difference is statistically significant.[7] These data certainly suggest that some people can accumulate higher than ambient levels of PBDEs through their work.

Acknowledgements. This work was supported by a grant from the STAR Program of the U.S. Environemtnal Protection Agency (grant R-83039701).

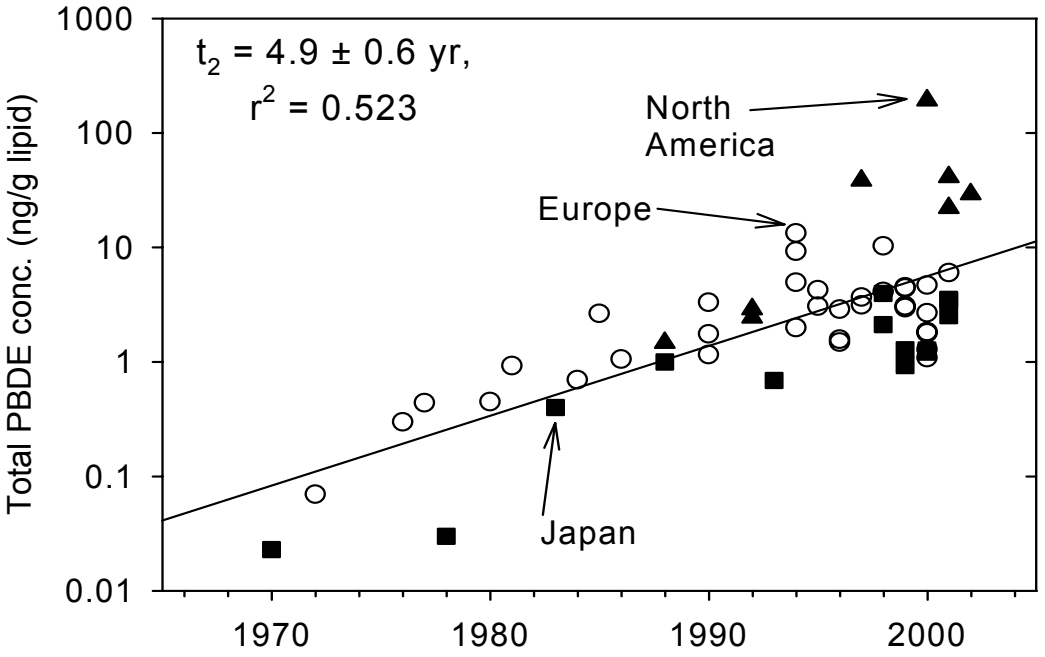

Figure 1. Total PBDE concentrations (ΣPBDE) in human blood, milk, and tissue (in ng/g lipid) shown as a function of the year in which the samples were taken. The three symbol types indicate the location from which the samples were collected. The overall regression is shown.

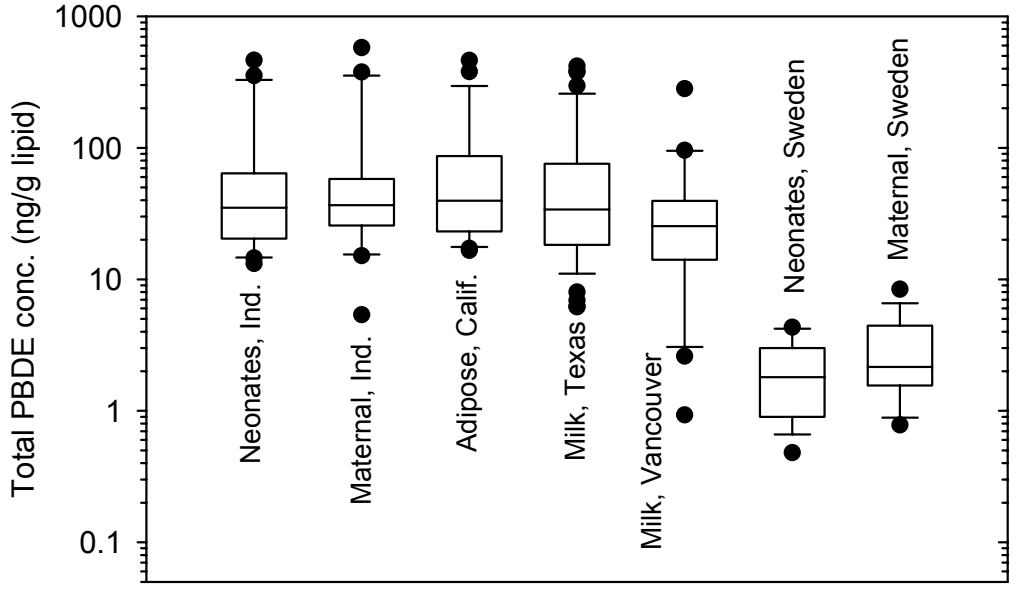

Figure 2. Distributions of the ΣPBDE concentrations in human blood, milk, and tissue from different locations. The horizontal lines indicate the 10th, 25th, 50th, 75th, and 90th percentiles of the distributions. Outliers are shown as dots. The data are taken from the

following sources: neonatal and maternal blood from Indiana,[9] adipose tissue from California,[10] milk from Texas,[11] milk from Vancouver, Canada,[12] and neonatal and maternal blood from Sweden.[13]

Reference

1 Hale, R. C.; La Guardia, M. J.; Harvey, E.; Mainor, M. *Chemosphere* **2002**, *46*, 729-735.

2 Kidder, R. C. *Flame Retard. '94* **1994**, *6*, 21-31.

3 Bromine Science and Environmental Forum, *Total Market Demand*, **2003**, available at: www.bsef.com .

4 Alaee, M.; Arias, P.; Sjödin, A.; Bergman, Å. *Environ. International* **2003**, *29*, 683-689.

5 Hale, R. C.; LaGuardia, M. J.; Harvey, E. P.; Gaylor, M. O.; Mainor, T. M.; Duff, W. H. *Nature* **2001**, *412*, 140-141.

6 Sjödin, A.; Jakobsson, E.; Kierkegaard, A.; Marsh, G.; Sellström, U. *J. Chromatog.* **1998**, *A822*, 83-89.

7 Hites, R. A. *Environ. Sci. Technol.* **2004**, *38*, 945-956.

8 van Bavel, B.; Hardell, L.; Kitti, A.; Liljedahl, M.; Karlsson, M.; Pettersson, A.; Tysklind, M.; Lindström, G. *Organohalogen Compd.* **2002**, *58*, 161-164.

9 Mazdai, A.; Dodder, N. G. Abernathy, M. P.; Hites, R. A.; Bigsby, R. M. *Environ. Health Perspect.* **2003**, *111*, 1249-1252.

10 She, J.; Petreas, M.; Winkler, J.; Visita, P.; McKinney, M.; Kopec, D. *Chemosphere* **2002**, *46*, 697-707.

11 Schecter, A.; Pavuk, M.; Papke, O.; Ryan, J. J.; Birnbaum, L.; Rosen, R. *Environ. Health Perspect.* **2003**, *111*, 1723-1729.

12 Ryan, J. J.; Patry, B.; Mills, P.; Beaudoin, N. G. *Organohalogen Compd.* **2002**, *58*, 173-176.

13 Meironyte Guvenius, D.; Aronsson, A.; Ekman-Ordeberg, G.; Bergman, A.; Noren, K. *Environ. Health Perspect.* **2003**, *111*, 1235-1241.