PERSONAL AND INDOOR AIR EXPOSURE TO PBDES IN US URBAN RESIDENCES

Allen JG[1], McClean MD[1], Stapleton HM[2], Nelson JW[1], Sanchez G[1], Fraser AJ[1], Webster TF[1]

[1]Dept. Environmental Health, Boston University School of Public Health, 715 Albany St., Boston, MA 02118 USA; [2]Nicholas School of the Environment and Earth Sciences, Duke University, Durham, NC 27708 USA

Introduction

Polybrominated diphenyl ethers (PBDEs) are a class of compounds commonly used as fire retardants in furniture containing polyurethane foam (PUF) and consumer products such as televisions. Human body burdens and environmental concentrations have increased for several decades and vary geographically, with the highest values reported in the USA.[1,2] We previously showed a strong association between PBDE concentrations in breast milk and house dust sampled from participant's homes.[3] While this implies that the indoor environment is a major source, it cannot distinguish between direct exposure to dust via ingestion and dust acting as a surrogate for PBDEs in indoor air. Current exposure estimates for North America suggest that inhalation is minor compared with diet and/or dust ingestion.[4-7] However, these comparisons are based on very uncertain estimates of ingestion of house dust.[5] Three estimates of inhalation exposure in North America[5-7] rely on Canadian indoor area air measurements sampled using passive air monitors[8], a method that may undersample respirable particles. No US indoor air measurements of PBDEs have been reported. Area air samples generally underestimate personal air exposure of both volatile compounds and particulates.[9] It is therefore possible that the inhalation route for PBDEs has been underestimated in residential settings.

Our current study has several objectives: 1) As it is difficult to directly measure PBDE exposure to dust via ingestion (or possibly dermal absorption), we collected indoor air using both personal and area air monitors. 2) In our previous study of breast milk and house dust, we analyzed researcher collected dust obtained by vacuuming measured areas of the most commonly used living areas of participant's homes.[3] Although better controlled than using dust from participant's vacuum cleaner bags, our procedure is far more laborious. The two methods can be highly correlated for some compounds[10], but it is not known if this is true for PBDEs; we therefore sampled dust from homes using both methods. 3) The relationship between PBDE concentrations in air or dust and characteristics of buildings and their contents remains an important research question.[3,4,6,8,11] Room characteristic information was collected for each room sampled.

Materials and Methods

Indoor air and dust samples were collected from 20 urban residences in the Boston, Massachusetts area. We sampled both single-family homes and apartments. At each home, one personal air sample and two area samples (bedroom, living room) were collected simultaneously during a one-week period. Area samplers were mounted on tripods. Participants were asked to turn on all pumps in the evening when they came home from work and turn them off in the morning. The personal pumps were placed near the bed at night with the sampler near the breathing zone. Air samples were collected at 2 liters/minute, calibrated at the beginning and end of sampling; pumps recorded the total time sampled, and volumes were calculated from the calibrated flows. Air was drawn through a glass fiber filter (GFF) followed by a PUF plug. Prior to use, the GFFs were baked in an oven and the PUFs were Sohxlet extracted. We included 13% field blanks and 12% replicates (attached to stationary pumps in living rooms). We collected three household dust samples from each residence: the resident's vacuum cleaner bag and two researcher-collected samples obtained by vacuuming the bedroom and living room. We employed Eureka Mighty Mite vacuum cleaners, collecting the dust in a cellulose extraction thimble (Whatman) inserted just behind the crevice tool. Using a questionnaire, we collected information on the characteristics of the residence and the contents of the bedroom and living room, including electrical appliances, carpet and foam-containing furniture.

PUFs and filters were extracted using an automatic pressurized fluid extractor (PFE). Air sampling units were disassembled and individual pufs and filters were placed into pre-cleaned 34 ml stainless steel extraction vessels. Each PUF and filter were spiked with two quantification standards, 4'-fluoro-2,3,3',4,5,6-hexabromodiphenyl ether (FBDE 160) and ^{13}C labeled BDE 209. The void volume of all cells was filled with hydromatrix (Dionex,

Sunnyvale, CA) and cells containing pufs were extracted with HPLC grade petroleum ether, whereas the cells containing glass fiber filters were extracted using HPLC grade dichloromethane. Laboratory blanks, consisting of hydromatrix filled cells, were extracted alongside the air samples. Samples were extracted by heating and pressurizing the cells to 100 °C and 1500 psi for 5 minutes with the appropriate solvent. Each sample was extracted over three cycles and collected into amber collection vials. Each extract was reduced in volume to 200 µl using a gentle stream of purified nitrogen gas, and then filtered through glass wool plugged disposable pipettes. Extracts were analyzed for PBDEs using a gas chromatograph (GC) coupled to a mass spectrometer operated in electron capture negative ionization (GC/ECNI-MS) mode. A 0.25 mm x 15 m fused silica capillary column coated with 5% phenyl methylpolysiloxane was used for the separation of PBDE congeners. On column injection was employed in the GC, and the injection port was set to track the oven temperature. The oven temperature program was held at 80 °C for 2 min followed by a temperature ramp of 12 °C/min to 140 °C, followed by a temperature ramp of 5°C/min to a final temperature of 280 °C, which was held for an additional 20 min. The transfer line temperature was maintained at 280 °C and the ion source was held at 200˚C. A suite of 27 individual BDE congeners were measured in all samples. Tri- through octaBDE congeners, and FBDE 160, were quantified by monitoring bromide ions (m/z 79 and 81). All three nonaBDE congeners and BDE 209 were quantified by monitoring molecular ion fragments (m/z 487 and 409), and BDE 209L was monitored through 495 and 415.

Masses of individual congeners on the GFFs and PUFs were separately blank corrected and converted into concentrations. Concentrations below the limit of detection (LOD) were given a value of 1/2 LOD. Data were log-transformed prior to statistical analysis. Dust analysis is still in progress.

Results

We report here all indoor air results, excluding PBDE 209 which is under analysis. Data for all congeners were log-normally distributed. Summary statistics are presented as geometric means (GM) and geometric standard deviations (GSD) by congener and sample location (Table 1). The GM concentration of PBDEs in personal air was 469.1 pg/m^3 (∑BDE equals the sum of congeners 17, 28/33, 47, 49, 66, 85/155, 99, 100, 153, 154). The dominant congeners found in each household were PBDEs 47 and 99. Log concentrations of ∑BDEs were significantly

Table 1 - Summary Statistics for PBDEs in Air*

	Personal Air (n = 20)		Bedroom (n = 20)			Main living area (n = 20)		
Congener	**GM (GSD)** (pg/m^3)	**Range** (pg/m^3)	**GM (GSD)** (pg/m^3)	**Range** (pg/m^3)	p-value†	**GM (GSD)** (pg/m^3)	**Range** (pg/m^3)	p-value‡
BDE 17	7.6 (2.3)	2 - 58.1	8.1 (2.9)	1.7 - 46.7	0.83	7 (3)	2 - 81.5	0.79
BDE 28/33	29.6 (1.7)	11.8 - 98.4	27.3 (2.3)	5.7 - 102.3	0.52	25.4 (2.3)	5.6 - 166.2	0.28
BDE 47	226.8 (2.3)	73.8 - 1393	157.9 (2.7)	31.2 - 784.3	0.03	145.1 (2.6)	30.9 - 2371.4	0.02
BDE 49	9.1 (3.2)	1.7 - 59.5	6 (2.9)	1.7 - 35.7	0.05	7.2 (3.1)	1.7 - 88.3	0.40
BDE 66	3.7 (2.1)	1.7 - 18.9	3.5 (2.1)	1.4 - 15.9	**	3.5 (2.3)	1.7 - 59.6	**
BDE 85/155	3.8 (2.6)	1.7 - 39.5	2.7 (1.7)	1.7 - 16.7	**	2.5 (1.5)	1.7 - 9.4	**
BDE 99	110.8 (2.8)	20.8 - 879.4	66.9 (2.2)	28.4 - 385.7	<0.01	60.3 (2.1)	24.6 - 552.6	<0.01
BDE 100	22.2 (2.8)	4.4 - 177.2	14.4 (2.4)	4.4 - 101.2	0.01	12 (2.3)	5.2 - 156.3	<0.01
BDE 153	8.6 (3.2)	1.6 - 73.7	4 (2.3)	1.6 - 26.7	<0.01	3.5 (1.8)	1.6 - 10.9	<0.01
BDE 154	9.1 (3.1)	2.1 - 78.9	6.1 (3.1)	2.1 - 138.7	0.03	5.2 (2.5)	2.1 - 60.5	<0.01
∑BDE	469.1 (2.2)	151.2 - 2479.6	324.7 (2.3)	92.9 - 1342.6	<0.01	288.6 (2.3)	82.1 - 3512.2	<0.01

* Sum of puf and filter

** Percent detect <60%

† Difference between personal and bedroom air using paired t-test

‡ Difference between personal and main living area air using paired t-test

higher in personal air compared to bedroom air (44% higher) and main living area air (63% higher) [see Table 1]. The ratio of concentrations in personal air and room air appears to increase with degree of bromination.

Figure 1 presents correlations of ∑BDE between sample locations. Personal air is strongly correlated with bedroom air (r = 0.785, p=0.0001) and main living area air (r = 0.592, p=0.0059) [not shown]. Bedroom air and main living area air were less highly correlated (r = 0.452, p=0.045), but there was one highly influential point that decreased the correlation.

We examined potential associations between log ∑BDE and housing characteristics using linear regression models. In our preliminary analyses, single-family homes had significantly higher levels of ∑BDE concentrations as compared to apartments/condominiums for personal air (ratio=2.6, p=0.016) and main living areas (ratio=2.6, p=0.17) but not bedrooms (ratio=1.5, p=0.33).

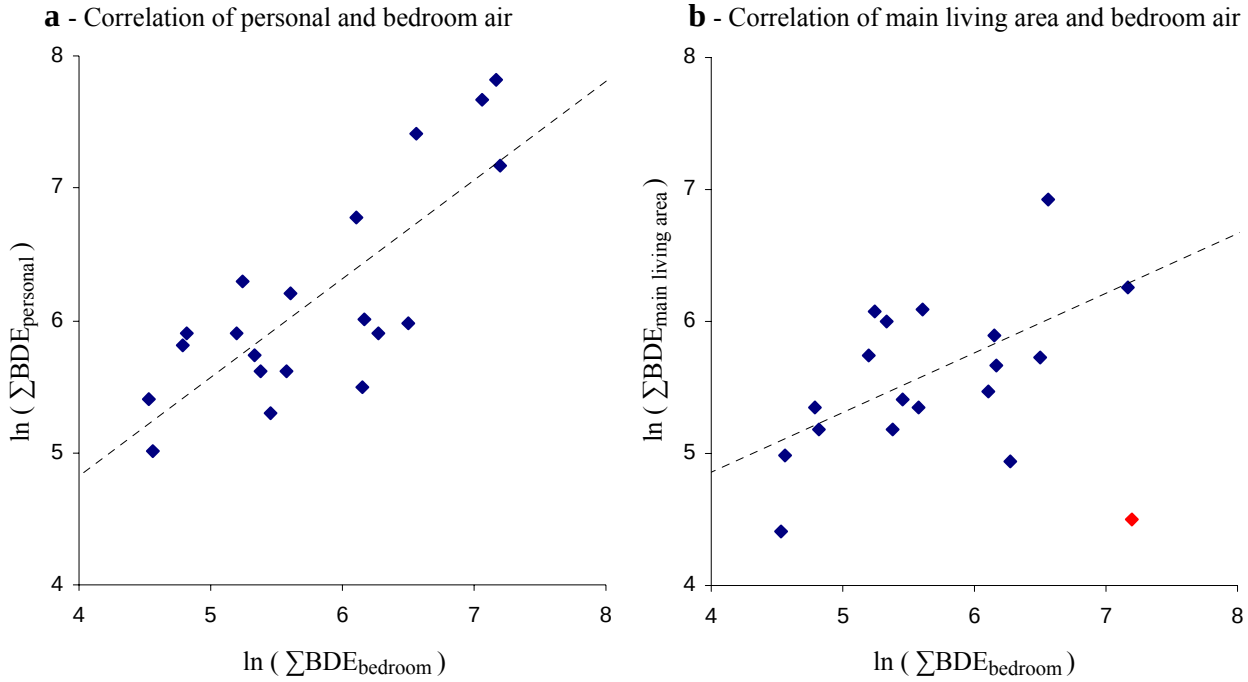
Discussion

These are the first indoor air measurements of PBDEs in the US and the first personal air measurements in a non-occupational setting. We demonstrated the feasibility of measuring PBDEs in air volumes (mean = 9.1 m^3) that are substantially lower than typically reported for non-occupational studies.

The geometric mean concentrations in personal air, bedroom air, and main living area air were 469.1, 324.7, and 288.6, respectively. These are similar to residential indoor air concentrations found in Toronto (median=436 pg/m^3, n=4; BDE 17, 28/33, 47, 85, 99, 100, 153, 154, 183) using high-volume sampling[12], and higher than that found in Ottawa (median=100 pg/m^3, n=74; BDE 17, 28, 47, 66, 71, 85, 99, 100, 153, 154) using passive air sampling which under samples particulate[8], or in Birmingham (UK) using high volume samplers (median=129, n=7; BDE 47, 99, 100, 153, 154)[11] [See figure 2]. Not surprisingly, the concentrations we measured in personal air in homes were much less than found in an occupational study of electronics recyclers.[13]

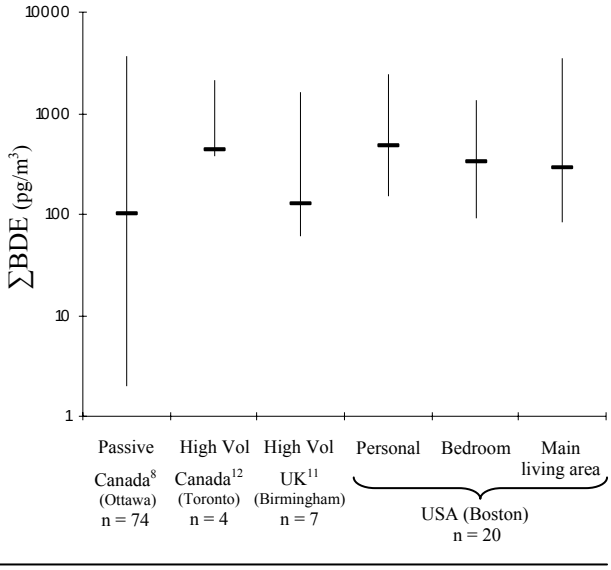
Our results indicate that PBDE concentrations in personal air from homes exceed those in bedrooms and living rooms, but by less than a factor of two. This result indicates increased concentrations of PBDEs in microenviron-

Figure 1 - Correlation of air samples by location (Sum of 10 congeners)



ments, e.g., those produced by volatilization from sources (such as foam mattresses) or resuspension of dust. By congener, the ratio of personal to room air appears to increase with degree of bromination, consistent with the understanding that personal exposures are affected by personal dust clouds.[9] Elemental analysis of air sampling filters in an earlier study of indoor air estimated that bromine concentrations in personal air samples were 90% higher than area air samples.[14] Previous measures of PBDEs in indoor air may under-represent personal exposure to PBDEs, especially the particulate phase. However, our air samplers did not exclude particles too large to inhale; depending on the size fractionation of PBDEs, our results may somewhat overestimate inhalation exposure. Our results do not report BDE 209 (still being analyzed), a congener that may be present in large amounts in suspended particulates.

Figure 2 - Personal and indoor air PBDE concentrations compared to previous indoor air studies



The geometric mean concentration of PBDEs in personal air in our study is about four times larger than the median value measured by Wilford et al using passive area air monitors[8], data used in several previous assessments of inhalation exposure in homes.[5-7] Use of our personal air GM concentration significantly increases exposure estimates for inhalation, but does not alter the conclusion that inhalation is a minor route of exposure compared with diet and/or dust, at least for the average person. Indoor air in offices may exceed that in homes[11]; future work might measure personal air in offices or other microenvironments (e.g., automobiles) with increased attention to respirable particles.

Acknowledgements
We wish to thank the study participants for their involvement and the Boston University Center for Interdisciplinary Research in Environmental Exposures and Health (CIREEH) for funding.

References

1. Norén K and Meironyté D. *Organohalogen Compounds* 1998; 38:1-4.
2. Hites RA. *Environ Sci Technol.* 2004; 38:945-56.
3. Wu N, Webster T, Herrmann T, Paepke O, Hale R, Harvey E, La Guardia M, Jacobs E. *Organohalogen Compounds* 2005; 67: 654-7.
4. Stapleton HM, Dodder NG, Offenberg JH, Schantz MM, Wise SA. *Environ Sci Technol.* 2005; 39:925-31.
5. Webster T, Vieira V, Schecter A. *Organohalogen Compounds* 2005; 67:505-8.
6. Wilford BH, Shoeib M, Harner T, Zhu J, Jones KC. *Environ Sci Technol.* 2005; 39:7027-35.
7. Jones-Otazo HA, Clarke JP, Diamond ML Archbold JA, Ferguson G, Harner T, Richardson GM, Ryan JJ, Wilford B. *Environ. Sci. Technol.* 2005; 39:5121-5130.
8. Wilford BH, Harner T, Zhu J, Shoeib M, Jones KC. *Environ Sci Technol.* 2004; 38:5312-8.
9. Wallace L. *J. Air & Waste Manage Assoc* 1996; 46:98-126.
10. Colt JS, Zahm SH, Camann DE, Hartge P. *Environ Health Perspect.* 1998; 106:721-4.
11. Harrad S, Wijesekera R, Hunter S, Halliwell C, Baker R. *Environ Sci Technol.* 2004; 38:2345-50.
12. Shoeib M, Harner T, Ikonomou M, Kannan K. *Environ. Sci. Technol.* 2004; 38:1313-1320
13. Pettersson-Julander A, van Bavel B, Engwall M, Westberg H. *J Environ Monit* 2004; 6:874-880.
14. Özkaynak H, Xue J, Spengler J, Wallace L, Pellizzari E, Jenkins P. *J Expos Anal Env Epidem.* 1996; 6:57-77.