A PRELIMINARY ASSESSMENT OF UK HUMAN EXPOSURE TO POLYBROMINATED DIPHENYL ETHERS (PBDEs)

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Introduction

Polybrominated diphenyl ethers (PBDEs) are a group of brominated compounds used as flame retardants. Despite the human health concerns surrounding these contaminants, comparatively few data are available relating to both: (a) the magnitude of human exposure; and (b) the relative significance of inhalation and diet as human exposure pathways. To our knowledge, there are currently no human exposure data available for the UK, and although daily dietary exposure to Canadian adults was estimated to be 44 ng SPBDE/person[1] there has been no systematic evaluation of the relative significance of exposure *via* inhalation of outdoor and indoor air. This latter environment is likely to be of particular importance given the number of PBDE sources indoors, and the low proportion of time spent outdoors by the population of the UK and other temperate industrialised regions. The objectives of this study were to determine concentrations of PBDE congeners 47, 99, 100, 153, and 154 in indoor air and duplicate diet samples and thereby make a preliminary evaluation of both the overall magnitude of human exposure to PBDEs and the relative significance of exposure *via* the inhalation and dietary ingestion routes.

Methods and Materials

Indoor Air

Nine sampling sites located in and around the University of Birmingham were investigated between 12th and 28th June 2001. Air samples were collected using a Graseby-Anderson Hi-vol sampler modified to hold a Teflon-coated glass fibre filter (GFF, 0.6 mm pore size) and a pre-cleaned polyurethane foam (PUF) plug. Sample volumes were typically 300 m^3, collected with the windows closed, over a period of 24 hours.

Duplicate diet samples

Accurately weighed *ca* 50 g samples of freeze-dried omnivorous duplicate diet samples collected in 1999 and 2000 by the Dept. of Nutrition and Dietetics, King's College, London were investigated. Further details of the collection, preparation and storage of these samples are available[2].

Analysis

Samples were treated with known quantities of PCB # 173 as an internal standard, prior to soxhlet extraction for *ca.* 16-24 hrs with dichloromethane:hexane (50:50 v/v). Concentrated crude extracts were washed with conc. H_2SO_4, prior to sequential further purification on: a H_2SO_4-impregnated silica multicolumn, lipid removal *via* solvent exchange between dimethyl sulfoxide and hexane (diet samples only) and florisil chromatography. After concentration and exchange of solvent to nonane, GC/MS analysis was carried out on a Fison MD-800 instrument fitted with a HP-5 trace analysis column (50 m

x 0.25 mm i.d., 0.25 mm film thickness). One ml of sample extract was injected in the splitless mode at an injector temperature of 285 °C. The oven temperature programme was: 140 °C for 2 min., 5 °C/min to 200 °C; 2 °C/min to 280 °C, 5 °C/min to 295 °C; 295 °C for 18 min. Six ions (for PBDE # 47: 485.8, 487.8; PBDEs # 99 and 100: 403.8, 405.8; and PBDEs # 153 and 154: 481.7, 483.7) were monitored in EI selected ion monitoring mode (ionisation voltage, 70 eV).

To ensure accurate and precise measurement peaks were only accepted if the following criteria were met:

1. Signal to noise ratios for the least abundant ion exceeded 3:1
2. Peaks eluted within 5 s of standards run in the same batch as the samples
3. Isotope ratios for peaks were within 15% of those obtained for standards run in the same batch as the samples

Blanks consisting of a GFF and a PUF plug for air samples (n=3), and a pre-extracted soxhlet thimble for diet samples (n=3) were analysed and found to contain concentrations of target PBDEs that were no greater than 5 % of the concentrations found in the corresponding samples. Our data are thus not corrected for blank concentrations. Recoveries of the internal standard typically exceeded 70 % and were never lower than 50 %. Analytical precision was assessed by replicate (n=3) analysis of an homogenised sediment sample, and found to be better than 22 % for all congeners, and substantially better for congeners 47 and 99.

Results and Discussion

PBDE Concentrations in Indoor Air

Table 1 shows the PBDE concentrations (PBDEs 47, 99, 100, 153 and 154) in the two categories (*i.e.* workplace and domestic) of microenvironments studied. Concentrations were generally at least 10 times lower than those reported for outdoor air sampled in Chicago in the late 1990s[3], implying that the source of PBDEs in the indoor environments studied is not infiltration of outdoor air. Furthermore, our data suggest that in Birmingham at least, ventilation of indoor air is likely to constitute a significant source of PBDEs in outdoor air. The highest concentrations were detected in a workplace environment (sample # 4) and concentrations were generally higher in workplace than domestic microenvironments, though this difference was not statistically significant. Interestingly, there was a wide variation in concentrations detected in samples 1, 2, 4, and 6, which were all obtained from different microenvironments located within the same University building (constructed in 1998). Such large intra-building concentration variations indicate that the origin of PBDEs in these microenvironments is not related to the building overall, but related to characteristics of individual microenvironments. Specifically, the samples with the highest PBDE levels (*i.e.* 4 and 6), were collected from rooms equipped with numerous computers (16 and 12 per room respectively). In addition, both rooms were fully carpeted and contained a number of polyurethane foam-padded chairs. By comparison, the least contaminated workplace sample (# 1) was collected from an uncarpeted, unfurnished room devoid of electrical appliances. The remaining workplace samples were obtained from offices containing 1-2 computers. On this limited evidence, there appears to be a correlation between computer usage and elevated atmospheric PBDE levels, although considerably more data is needed to verify this tentative hypothesis.

Inhalation of Air as a Source of Human Exposure to PBDEs

We estimated the median and range of daily human intake of PBDEs via inhalation (assuming 100% absorption of intake). The values were calculated using the following algorithm:

$$\Sigma \text{Exposure} = [(C_w F_w) + (C_h F_h) + (C_o F_o)]R_f$$

Where SExposure is the daily adult human exposure *via* inhalation (ng SPBDE person^{-1} day^{-1}), $C_{w/h/o}$ is the SPBDE concentration in workplace/home/outdoor air respectively (ng/m^3), R_f is the adult respiration rate (20 m^3/d) and $F_{w/h/o}$ is the respective fraction of day spent at workplace/home/outdoors. To estimate exposure *via* inhalation of indoor air, we used a value of 23.8 % (40 h) for time spent at the workplace, and a value of 67.9 % for time spent in the home[4]. In the current absence of data on concentrations of PBDEs in Birmingham outdoor air, we have not attempted to estimate exposure *via* inhalation of outdoor air. However, given that concentrations in outdoor air in both the UK[5] and North America[3] are considerably below those recorded in indoor air in this study, we do not believe inhalation of outdoor air to make an appreciable contribution to overall inhalation exposure. Using the above algorithm, median concentrations in workplace and domestic environments, the median lower bound (*i.e.* where a congener is below detection limit, that concentration is assumed to be zero) daily human exposure to ΣPBDEs *via* inhalation is 32.9 ng/person. Note that ΣPBDE is the sum of congeners 47, 99, 100, 153, and 154.

Dietary Ingestion as a Source of Human Exposure to PBDEs

The daily dietary intake of ΣPBDEs was calculated using data on the ΣPBDE content of the diet samples (Table 2) and food mass ingestion data for the individuals consuming each diet sample. The estimated median lower bound value (*i.e.* where a congener is below detection limit, that concentration is assumed to be zero) of 90.5 ng/person is higher than that (44 ng/person) reported previously for the Canadian population *via* a "food basket" study[1]. Our data also indicate a wide range of concentrations in the magnitude of individual exposures. While the magnitude of overall exposure and the relative significance of inhalation and dietary exposures will depend greatly on individual lifestyles, our estimates of median exposures indicate diet and inhalation to contribute 73 and 27 % respectively to an overall median daily exposure of 123 ng ΣPBDE/person. On this basis, future assessments of human exposure to PBDEs should focus on both diet and inhalation pathways.

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Table 1. PBDE Concentrations (pg/m^3) in Indoor Air Samples

Sample #/ Location type[a]	BDE 47	BDE 99	BDE 100	BDE 153	BDE 154	ΣPBDE
1/W	610	110	47	2.3	2.1	769
2/W	1360	157	73	<1.0	<1.0	1590
3/D	645	80	37	<1.0	<1.0	2350
4/W	7140	6510	1450	181	228	17900
5/W	1100	229	94	2.8	2.7	1430
6/W	4640	789	275	7	15	5730
7/D	1330	209	82	<1.0	1.4	1620
8/W	770	465	145	3.9	8.7	3010
9/D	721	133	52	<1.0	<1.0	906
Median W	1230	347	239	3.4	5.7	2300
Median D	721	133	52	<1.0	<1.0	1620

[a] W = workplace; D = domestic

Table 2. PBDE Concentrations (pg/g dry weight) in Duplicate Diet Samples and Daily Exposure Estimates

Sample #	BDE 47	BDE 99	BDE 100	BDE 153	BDE 154	Daily ΣPBDE dietary intake[a] (ng/person)
1	109	61.2	28.9	11.6	11.7	113
2	59.5	52.8	11.6	<10	<10	54.9
3	79.6	73.7	17.4	<10	<10	137
4	74.0	93.0	35.3	37.1	27.1	189
5	47.4	<10	<10	<20	<20	40.5
6	117	83.5	<10	<20	<20	99.3
7	48.0	66.4	<10	<20	<20	77.5
8	48.7	56.5	<10	<20	<20	81.6
9	31.5	53.7	<10	<20	<20	37.2
10	94.2	174	<10	<20	<20	235
Median	76.8	63.8	<10	<20	<20	90.5

[a] assuming that where a congener is below detection limit, that concentration is zero (*i.e.* lower bound estimate).