HEXABROMOCYCLODODECANES AND TETRABROMOBISPHENOL-A IN INDOOR AIR AND DUST IN BIRMINGHAM, UK: IMPLICATIONS FOR HUMAN EXPOSURE

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Introduction

Brominated flame retardants (BFRs) constitute a diverse group of compounds used to prevent or minimize fire hazards. The most widely used BFRs are tetrabromobisphenol A (TBBP-A) with a global demand of 170,000 t in 2004, alongside Decabromodiphenyl ether and hexabromocyclododecane (HBCD), for which the respective worldwide market demands in 2001 were respectively 56,100 t and 16,700 t[1].

TBBP-A is used mainly as a reactive flame retardant covalently bonded to the polymer matrix in epoxy and polycarbonate resins used in printed circuit boards and electronic equipment. It can also be used as an additive, for instance in high impact polystyrene (HIPs). TBBP-A has been identified as an endocrine disrupter due to its structural similarity to 17-β-estradiol and thyroxine (T4). It also displays a high potency to bind to human transthyretin and immunotoxicity. However, the potential toxicity of TBBP-A exposure is mitigated to some extent by its estimated human half-life of 2.2 days[2].

HBCD is used as an additive to expanded and extruded polystyrene foams for thermal insulation of buildings, back-coating of fabrics for furniture and to a lesser extent in HIPS[1]. The commercial formulations consist mainly of α-, β-, and γ-HBCD diastereomers with γ-HBCD being predominant. HBCD induces hepatic cytochrome P450 enzymes in rats and alters the normal uptake of neurotransmitters in rat brain. It can disrupt the thyroid hormone system and induce cancer through a non-mutagenic mechanism in humans[3].

To the authors' knowledge, there have been no reports of diastereomer-specific HBCD concentrations in indoor air, save for those reported recently by our group in three offices[4]. In addition, very little is known about concentrations of HBCDs in outdoor air[5]. Information on the presence of TBBP-A in indoor air and dust in the UK is scarce[6] and there is - to date - no report on human exposure to TBBP-A or HBCDs via air inhalation and dust ingestion that considers exposure in both domestic and non-domestic microenvironments, such as cars, offices, and public microenvironments (PMEs).

In light of the above, the aims of this study are: (1) to report concentrations of α-, β- and γ-HBCDs and TBBP-A in air and dust from cars, homes, offices, and PMEs; (2) to evaluate whether the previously observed shift in HBCD diastereomer pattern from predominantly γ-HBCD in the commercial formulation towards α-HBCD observed in dust samples[7] is reflected in air samples and (3) to estimate exposure of UK adults and toddlers to HBCDs and TBBP-A via both inhalation and dust ingestion.

Materials and Methods

Dust samples were collected between September 2006 and June 2007 through a standardized procedure using a vacuum cleaner; the samples represented the 25-500 μm size fraction. Further details are provided elsewhere[7]. The following microenvironment categories were selected for study: homes (n=45), offices (n=28), PMEs (3 pubs and 1 restaurant) and cars (n=20). Air samples were collected between February and December 2007 in a total of 62 microenvironments comprising 33 homes, 25 offices and 4 PMEs (3 Pubs and 1 restaurant) within the West Midlands conurbation. Outdoor air sampling (n=5) was performed in December 2007 at the Elms Road Observatory Site (EROS) in Birmingham, UK. Low volume active air samplers were used for monitoring concentrations of HBCDs in outdoor air and TBBP-A in both indoor and outdoor air while PUF disk passive samplers were employed to sample HBCDs in indoor air. Details of the active and passive air sampling devices can be found elsewhere[4].

Air samples were Soxhlet extracted while dust samples were extracted using pressurized liquid extraction (Dionex, ASE 300). The extracts were purified by passing through a SPE cartridge filled with 8 g of pre-cleaned acidified silica (44% concentrated sulfuric acid, *w/w*), prior to analysis using LC/ESI/MS/MS[7].

Descriptive statistical analysis of the data was conducted using Excel (Microsoft Office 2003) with other statistical procedures conducted using SPSS version 13.

Results and Discussion

HBCDs and TBBP-A concentrations in air from different categories of indoor microenvironments

Indoor air concentrations of HBCDs are an order of magnitude higher than those detected in outdoor air in this study (Table 1) and US outdoor air[5]. While no statistically significant difference ($p<0.05$) was observed between concentrations of ΣHBCDs in air from homes and offices, concentrations in the few PMEs sampled are substantially above those detected in homes and offices. It is interesting that concentrations of ΣHBCDs in air reported here are typically several times greater than the concentrations of Σtri-hexa-BDEs (i.e. those congeners predominant in the Penta-BDE formulation) reported in an earlier study of the same microenvironment categories in Birmingham, UK[8,9].

Table 1: Summary of HBCDs and TBBP-A concentrations (pg m^{-3}) in air from the studied microenvironments.

Location (reference)	Diastereomer/ Statistical parameter	α-HBCD	β-HBCD	γ-HBCD	Σ HBCDs	TBBP-A	Σtri-hexa-BDEs[a]
Homes, n=33, n=5 for TBBP-A	Average	59	22	170	250	16	52
	Standard deviation	77	8.7	140	240	5.2	61
	Median	37	22	120	180	15	24
	Minimum	14	5.0	39	67	8.9	4
	Maximum	430	54	710	1300	22	250
Offices, n=25, n=5 for TBBP-A	Average	43	24	120	180	16	170
	Standard deviation	20	5.9	68	90	12	280
	Median	36	23	110	170	11	71
	Minimum	18	14	43	70	4.1	10
	Maximum	87	34	370	460	33	1400
Public micro-environments, n=4	Average	250	28	550	900	26	110
	Standard deviation	110	12	140	60	6.8	72
	Median	210	24	570	900	27	140
	Minimum	180	19	360	820	17	29
	Maximum	400	46	690	960	32	160
Outdoor air, n=5	Average	3.0	1.1	33	37	0.76	21[b]
	Standard deviation	0.51	0.09	1.9	2.4	0.06	
	Median	2.9	1.0	33	37	0.74	
	Minimum	2.3	0.94	31	34	0.69	
	Maximum	3.7	1.2	35	40	0.85	

a – from reference 8; b – from reference 9.

TBBP-A concentrations in indoor air (Table 1) exceed by an order of magnitude those present in outdoor air – consistent with the presence of indoor emission sources. Unlike HBCDs, there are no substantial differences apparent between concentrations in PMEs and those in homes and offices. Concentrations of TBBP-A in both indoor and outdoor air are much lower than those of HBCDs and are similar to those of Σtri-hexa-BDEs[8].

HBCDs and TBBP-A concentrations in dust from different categories of indoor microenvironments

Concentrations of ΣHBCDs in car dust exceeded significantly ($p<0.05$) those in both homes and offices (Table 2). No significant differences were detected between ΣHBCDs concentrations in dust from homes and offices. Such differences in contamination between microenvironment categories coincide with those observed by our group for Σtri-hexa-BDEs, but not BDE-209[10].

The levels of TBBP-A found in indoor dust samples (Table 2) are slightly lower than those of Σtri-hexa-BDEs, and substantially lower than those of both HBCDs and BDE-209[10]. As noted earlier for air, this relative order of contamination is not a simple reflection of the respective production volumes of these BFRs. We believe this is consistent with the fact that TBBP-A is used predominantly as a reactive flame retardant and hence its release from treated goods is likely to be less facile than for additive FRs, such as HBCDs and PBDEs. In contrast to HBCDs (this study) and PBDEs[10], TBBP-A concentrations in car dust are significantly lower ($p<0.05$) than in

homes and offices – no significant difference was observed between concentrations in homes and offices. While there are too few samples from PMEs to draw firm conclusions, the concentrations of TBBP-A in dust from the studied PMEs are markedly higher than in cars, homes, or offices. We believe this may be attributable to the comparatively large number of electronic items (e.g. TVs and video game machines) in these PMEs (pubs and restaurants).

Table 2: Summary of HBCDs and TBBP-A concentrations (ng g^{-1}) in dust from the studied microenvironments.

Location (reference)	Diastereomer/ Statistical parameter	α–HBCD	β–HBCD	γ–HBCD	ΣHBCDs	TBBP-A	Σtri-hexa-BDEs[a]	BDE-209[a]
Homes, n=45, for TBBP-A n=35, non-detects = 1	Average	3200	1000	4200	8300	87	77	260000
	Standard deviation	11000	3900	13000	26000	71	68	580000
	Median	380	93	670	1300	62	46	8100
	Minimum	22	9.0	70	140	< DL[b]	7	<DL
	Maximum	66000	26000	75000	140000	382	250	2200000
Offices, n=28, for TBBP-A non-detects = 4	Average	610	210	760	1600	49	250	30000
	Standard deviation	780	300	910	1700	46	310	67000
	Median	220	84	470	760	36	100	6200
	Minimum	15	11	36	90	< DL	16	620
	Maximum	2900	1300	3700	6600	140	1100	280000
Cars, n=20, for TBBP-A non-detects = 10	Average	3200	1400	14000	19000	6	2300	410000
	Standard deviation	2900	1600	16000	19000	8	5700	770000
	Median	2000	740	9600	13000	2	190	100000
	Minimum	54	16	27	190	< DL	54	12000
	Maximum	8800	5200	56000	69000	25	22000	2600000
Public micro-environments, n=4	Average	1000	330	1400	2700	220	n.m.[c]	n.m
	Standard deviation	190	67	270	390	140	n.m.	n.m.
	Median	1000	310	1300	2700	230	n.m.	n.m.
	Minimum	810	270	1100	2300	52	n.m.	n.m.
	Maximum	1200	420	1700	3200	350	n.m.	n.m.

a – from reference 9; b - Method detection limit; c- not measured.

Differences in HBCD diastereomer pattern between matched indoor air and dust samples

α-HBCD made a significantly greater contribution to ΣHBCD in dust than in air, with the opposite trend observed for γ-HBCD ($p<0.05$). On average, dust composition is 33% α-HBCD, 56% γ-HBCD, while for air; this is 22% α-HBCD, and 65% γ-HBCD. The pattern in air reflects what is likely to be present in treated goods and thus is likely a consequence of volatilization from treated goods. In contrast, the greater relative abundance of α-HBCD in dust from the same microenvironments is difficult to explain, but is consistent with the existence of post-depositional processes that induces a shift from γ-HBCD to□α-HBCD in this matrix. Elucidating the cause of this difference is therefore a new research priority.

Human exposure to HBCDs and TBBP-A via air, dust and diet

The concentrations of HBCDs and TBBP-A reported in this study were used to estimate exposure of UK adults and toddlers to these BFRs via air inhalation and dust ingestion. The obtained estimates are compared to the upper bound dietary intakes of HBCDs from the whole diet by the UK population in 2004[11] (Table 3). However, the reported upper bound dietary intakes of TBBP-A by the UK population (average adult exposure = 1.6 ng/kg body weight/day)[11] appear to be largely overestimated compared to the average dietary intake of TBBP-A by the Dutch population (0.04 ng/kg body weight/day)[12]. While TBBP-A was not found above the detection limit in any of the analyzed UK food samples and the reported values are only upper bound intakes[11], the dietary exposure data from the Dutch survey appear to be more reliable since the flame retardant was detected and quantified in some of the analyzed food samples. Therefore, we have chosen to compare our estimates for human exposure to TBBP-A via air inhalation and dust ingestion to its dietary intake by the Dutch population[12] (table 3). Dust ingestion is the major pathway of exposure to HBCDs and TBBP-A for UK toddlers even at a mean dust intake scenario. Specifically, the exposure of a toddler weighing 10 kg and ingesting 200 mg dust day^{-1} contaminated at

the 95th percentile level reported in this study will exceed ten-fold the exposure received via the diet. In contrast, inhalation is indicated as only a minor pathway of exposure to HBCDs contributing <1% of total daily exposure to both adults and toddlers while for TBBP-A, inhalation exposure accounts for an average of 6% and 2% of the total daily exposure of UK adults and toddlers respectively.

Table 3: Summary of estimates of exposure (ng day^{-1}) of UK adults and toddlers to HBCDs and TBBP-A via air, dust and diet.

Air	Intake (ng day^{-1})	**Adult**					**Toddler (6-24 months)**				
		α-HBCD	β-HBCD	γ-HBCD	Σ HBCDs	TBBP-A	α-HBCD	β-HBCD	γ-HBCD	Σ HBCDs	TBBP-A
	5th %ile	0.5	0.2	1.3	2.3	0.2	0.1	0.0	0.3	0.5	0.0
	Median	0.8	0.4	2.6	3.9	0.3	0.2	0.1	0.5	0.8	0.1
	Average	1.2	0.6	3.2	5.0	0.3	0.2	0.1	0.6	1.0	0.1
	95th %ile	2.7	1.1	7.1	10.4	0.4	0.5	0.2	1.4	2.1	0.1
Dust		**Mean dust intake**									
		α-HBCD	β-HBCD	γ-HBCD	Σ HBCDs	TBBP-A	α-HBCD	β-HBCD	γ-HBCD	Σ HBCDs	TBBP-A
	5th %ile	1.8	0.6	2.6	5.8	0.4	4.2	1.3	6.6	13.9	1.3
	Median	46.6	15.3	69.6	132	1.6	145	47.2	212	404	4.4
	Average	8.4	2.5	19.9	32.5	1.3	22.8	6.3	51.8	86.9	3.3
	95th %ile	109	48.5	331	469	3.2	324	147	1043	1463	8.5
		High dust intake									
	5th %ile	4.5	1.4	6.5	14.4	1.1	16.2	5.0	26.0	55.9	5.2
	Median	116	38.3	174	329	4.0	558	179	737	1473	18.0
	Average	21.1	6.2	49.7	81.3	3.1	76.5	19.9	133	251	13.6
	95th %ile	273	121	828	1172	8.1	1251	559	3797	5429	34.9
Diet[a]	Average	203	105	112	413	2.8[b]	120	57.0	67.0	240	0.4[b]
	High level	385	231	217	840	2.8[b]	240	110	140	500	0.4[b]

a- from reference 11; b- from reference 12

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