DUST FROM UK PRIMARY SCHOOL CLASSROOMS AND DAYCARE CENTRES: ITS SIGNIFICANCE AS A PATHWAY OF EXPOSURE OF YOUNG CHILDREN TO PERFLUOROALKYL COMPOUNDS (PFCs) AND BROMINATED FLAME RETARDANTS (BFRs)

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Abstract
Recent research has highlighted the potential significance of indoor dust ingestion as a pathway of exposure to both PFCs and BFRs. In particular, it has been identified as a pathway of concern for young children. However, relatively little is known about the presence of such chemicals in dust from classrooms in child daycare centres and primary schools. This paper reports concentrations of selected PFCs and BFRs in samples of dust (n=43) from such microenvironments in the UK West Midlands conurbation. Concentrations in classrooms are generally in line with those in other UK microenvironments; although concentrations of PFOS and PFOA in UK classrooms exceed those reported in Swedish child daycare centres. Reassuringly, exposure of young children via dust ingestion to PFOS and PFOA falls comfortably below even the most stringent exposure guideline value for these compounds. However, when the data from this study are combined with data on concentrations in dust from UK homes and cars under a high-end exposure scenario, young children are exposed to BDE-99 and BDE-209 at levels that exceed a recent health-based limit value for BDE-99 derived by Netherlands researchers and the USEPA's reference dose (RfD) for BDE-209.

Introduction
Perfluoroalkyl compounds (PFCs) have found widespread industrial and consumer applications owing to their unique properties[1]. Despite recent restrictions on their production, there are increasing reports of their presence in both indoor and outdoor environments[2-5]. This presence has generated concerns about the human toxicity of some PFCs, and the UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) has recommended provisional tolerable daily intakes (TDIs) of 0.3 and 3 μg/kg bw/day for PFOS (perfluorooctane sulfonate) and PFOA (perfluoro octanoic acid) respectively[6,7]. The German Drinking Water Commission (GWDC) has also recommended guideline intake values of 0.1 μg/kg body weight/day for both PFOS and PFOA[8].
Although their applications differ, similar considerations of environmental contamination leading to both indoor and outdoor exposures and concerns about the human health impacts of such exposures exist for both recent- and current-use brominated flame retardants (BFRs). Based on the most recent global production figures available; the most widely-used BFRs are TBBP-A, Decabromodiphenyl ether, Octabromodiphenyl ether, Pentabromodiphenyl ether, and Hexabromocyclododecane[9]. Furthermore, recent restrictions on the manufacture and use of PBDEs have shifted attention to alternative BFRs. Of these, decabromodiphenylethane (DBDPE) and 1,2-bis(2,4,6-tribromophenoxy)ethane (BTBPE) have been reported previously as present in UK indoor dust[10]. While there is growing understanding of the potential human health effects of some BFRs, very few health based exposure standards exist; a situation exemplified by the current position of the UK COT that there is not yet sufficient evidence to permit setting a TDI for PBDEs[11]. Recently however, a preliminary Health Based Limit Value (HBLV) for BDE 99 was derived by Netherlands researchers[12]. This HBLV (0.23–0.30 ng/kg bw/day) is driven by impaired spermatogenesis which appears the most sensitive end-point[12]. Also pertinent given its predominance in UK indoor dust[10], is the reference dose (RfD) of daily oral exposure to BDE-209 of 7 μg/kg bw/day[13]. This RfD is that considered by the United States Environmental Protection Agency's Integrated Risk Information System (IRIS) Toxicological Evaluations to be without appreciable risk of deleterious effects.

While dietary exposure is an important pathway of human intake to BFRs and PFCs[14-16], there is an increasing weight of evidence that the ingestion of indoor dust may also play an important role[2-4,10]. Of particular concern is that normalised to body weight, young children are considered to ingest substantially more dust than adults[17]. To date however, while the exposure to PFCs and BFRs of young children arising from the ingestion of house and car dust has been evaluated, exposure received in the classroom has received comparatively little attention[18]. This paper therefore reports the concentrations of a range of PFCs and BFRs in indoor dust sampled from 43 classrooms frequented by young children (age range ~2-6 years), and estimates the potential exposure of British children to such contaminants.

Materials and Methods

Sampling

Dust samples (n=43) were collected from classrooms in daycare centres and primary schools in the West Midlands of the UK, during winter 2007 /spring 2008. Samples were collected using a portable vacuum cleaner, to which a sock with a 25 μm mesh size (Allied Filter Fabrics Ltd, Australia) was inserted into the nozzle of the device to retain the dust. The socks containing the samples were placed in resealable polyethylene bags for transportation. The samples were sieved through a 500 μm mesh and stored in the dark at 4ºC until extraction.

Analysis

Concentrations of PFCs (i.e. PFOS, PFOA, perfluoro hexanesulfonate (PFHxS), perfluorooctane sulfonamide (FOSA), N-methyl-perfluoro octanesulfonamide (MeFOSA), N-ethyl-perfluoro octanesulfonamide (EtFOSA), 2-(N-methylperfluorooctane sulfonamido)-ethanol (MeFOSE) and 2-(N-ethylperfluorooctane sulfonamido)-ethanol (EtFOSE)), α-, β-, γ-HBCD, and TBBP-A were all determined at the University of Birmingham, while those of all other BFRs (see Table 1) were measured at the University of Antwerp.

PFCs Internal standards (^{13}C-labelled) PFOS and PFOA, ^{18}O-PFHxS, ^{2}H-MeFOSA and MeFOSE) were added to aliquots of each sample (0.1 g) in a 15 mL polypropylene centrifuge tube and acetone (5 mL, HPLC grade) added for the first solvent extraction. The samples were sonicated for 15 minutes and shaken at 5 minute intervals. Samples were centrifuged to aid settling and separation of the dust, and the supernatant liquid removed. The procedure was repeated and the supernatant added to the first extract. The combined extracts were filtered through a grade 1 Whatman filter paper, prior to the addition of Celite (0.5 g). Solid phase extraction was then conducted using an Oasis WAX column, preconditioned with methanol and 0.1% formic acid (aq.). After loading, the sample column was washed with 0.1% formic acid and methanol, and dried under vacuum. The sample was eluted with 4% NH_4OH in methanol, dried under nitrogen and eluted in 100 μL methanol and 75 μL ammonium acetate (20 mM aq. in MeOH, 1/3, v/v), ready for LC-MS/MS analysis.

For QC purposes, one field blank (consisting of 0.1 g sodium sulfate "sampled" using the standard procedure) was conducted for every five samples. Concentrations of PFOS and PFOA in these blanks did not exceed 5% of the level detected in samples and results were not corrected for blank levels. To the authors' knowledge there is not currently available a standard reference material that has certified or indicative concentrations of PFOS or PFOA in indoor dust. However, average$\pm\sigma_{n-1}$ concentrations of PFOS and PFOA of 1990±78 and 673±26 ng g^{-1} respectively have been reported recently for SRM2585 (indoor dust)[18]. Our replicate (n=5) analyses of SRM2585 yielded average$\pm\sigma_{n-1}$ concentrations of 1700±210 and 730±53 ng g^{-1} for PFOS and PFOA respectively.

Samples were analysed on an API 2000 LC-MS/MS (Applied Biosystems) fitted with an electrospray ionisation source (ESI) operated in negative ion mode. Chromatographic separation of target PFCs was achieved on a Shimadzu LC fitted with a Varian Metasil Basic column (3 μm particle size, 150 x 3.2 mm). Target analytes were eluted using water/methanol with 2 mM ammonium acetate solution (9:1, v/v) (A), and methanol (B). The elution programme was 20%B for 5 minutes, then 100% B for 3 minutes, and finally 20%B for 10 minutes.

Separate aliquots of the same classroom dust samples were analysed for concentrations of BFRs. Details of the methods used for extraction, clean up and analysis of these compounds and their accuracy and reproducibility are described elsewhere[10,19].

Results and Discussion

Concentrations

Table 1 summarises the concentrations of target compounds in the analysed samples. Those of the most widely used BFRs (HBCDs, TBBP-A, BTBPE, and PBDEs) are consistent broadly with the levels detected previously in other UK indoor microenvironments[10,19]. BDE-209 and HBCDs are the principal BFRs detected. Interestingly, while concentrations of BTBPE were generally low and in line with those of the lower brominated BDEs; one sample (from a primary school classroom) contained >45 μg BTBPE kg^{-1}. To our knowledge this is comfortably the highest concentration of BTBPE reported in indoor dust. At the current time, we have no explanation for the high concentration of BTBPE in this sample; however, we note that BTBPE has been marketed recently as a replacement for the Octa-BDE formulation[20], and it is possible that concentrations of BTBPE in indoor dust may be rising in response to this action.

Concentrations of target PFCs in dust samples in this study are consistent broadly with those reported previously in indoor dust samples from other locations[2-4,18]. While PFCs were determined in dust from 10 child daycare centres in the United States[4], the authors did not report the concentrations found in a format that distinguished those in the classroom dust from the far larger number of house dust samples. However, concentrations of both PFOS and PFOA in this study exceed those reported for 10 Swedish daycare centers where the median concentrations were 31 and 41 ng g^{-1} for PFOS and PFOA, respectively[18].

Table 1: Summary of Concentrations (ng g^{-1}) of BFRs and PFCs in Classroom Dust Samples

Compound	Minimum	5th percentile	Median	Mean	95th percentile	Maximum	Median (Homes)[c]	Median (Cars)[c]
BDE 28	1.5	1.5	1.7	5.7	19	25	0.50	0.50
BDE 47	1.6	4.0	26	32	85	120	10	54
BDE 66	0.8	1.4	3.1	4.0	9.7	11	0.50	0.92
BDE 100	1.0	1.2	6.7	10	26	50	3.4	17
BDE 99	1.1	5.3	36	54	140	270	20	100
BDE 85	1.0	1.2	3.3	4.9	14	20	-	-
BDE 154	2.1	2.6	5.3	7.7	22	26	2.8	11
BDE 153	2.0	3.3	10	28	76	310	5.0	11
Σtri-hexa-BDEs	2.7	10	86	130	450	510	46	190
BDE 183	1.4	2.1	4.6	9.2	23	48	4.2	7.8
BDE 197	2.0	2.4	4.9	8.6	31	35	5.2	24
BDE 203	1.3	1.8	4.5	9.0	34	50	6.4	50
BDE 196	3.7	3.7	9.1	14	36	42	12	75
BDE 209	49	140	5000	8500	24000	88000	8200	104000
DBDPE	27	49	200	380	1200	1600	24	100
TBBPA-bdpe	22	35	180	210	420	750	-	-
BTBPE	4.6	6.5	21	1600	200	>45000[b]	5.3	5.0
ΣHBCDs[a]	72	370	4100	8900	37000	89000	1300	13000
TBBP-A	17	20	110	200	460	1400	63	11
PFOS	22	130	840	990	2400	3700	150	360
PFOA	18	23	240	300	630	1700	340	170
PFHxS	16	28	700	2300	7600	34000	230	240
MeFOSA	<BDL	<BDL	<BDL	<BDL	<DL	<DL	<DL	<DL
EtFOSA	<BDL	7	31	71	270	640	49	28
FOSA	<BDL	6	34	170	610	750	19	14
EtFOSE	<BDL	32	450	1500	4400	13000	130	130
MeFOSE	<BDL	110	880	1400	5500	8400	140	260

[a]Sum of α-, β-, and γ-HBCDs

[b]value cited as "greater than" as the GC peak was overloaded
[c]Data for HBCDs and TBBP-A taken from[19]; that for other BFRs from[10]; and that for PFCs from[21].

Sources of contamination
In contrast with previous studies of PFCs in dust from other indoor microenvironments, but in line with the only other available data on PFCs in dust from classrooms[18]; no correlation was detected between concentrations of PFOS and PFOA. Interestingly, the concentrations of PFOS and EtFOSE display a significant linear correlation ($R=0.52$; $p<0.001$), implying that these two PFCs have a common source or sources in the classrooms studied. In particular, it may be that EtFOSE is undergoing degradation to PFOS either before or after release from treated products. However, the correlation is driven predominantly by the presence of one sample that is highly contaminated with both PFOS and EtFOSE, and removal of this sample removes the correlation. In contrast, while there is no correlation between concentrations of EtFOSE and MeFOSE when all samples are considered; removal of the high PFOS and EtFOSE sample, reveals a significant correlation between EtFOSE and MeFOSE ($R=0.43$; $p<0.01$). In all sampled classrooms, we collected information about likely sources of our target compounds (e.g. number and type of electronic items, type of floor covering etc.). Analysis of these data yielded no clear insights into the origins of dust contamination with any of the target PFCs or BFRs in our samples.

We have derived estimates of the exposure of young children (age range 2-6 years) via dust ingestion under 3 exposure scenarios: (1) a low-end scenario where the child ingests 50 mg dust day^{-1} contaminated at the 5th percentile concentration, (2) a "typical" scenario where 50 mg dust day^{-1} contaminated at the median concentration is ingested, and (3) a high-end scenario where the child ingests 200 mg dust day^{-1} contaminated at the 95th percentile concentration. We have utilised the concentration data for classrooms from this study, alongside those from UK homes and cars[10,19,21] to calculate exposure under each scenario. To do so, we have assumed that dust ingestion is pro-rata to our estimates of the proportion of time spent in classrooms, homes, and cars over a year (20.1, 75.7, and 4.2% respectively). We have then divided the exposure estimates by an assumed typical child weight of 20 kg to normalise to body weight. The results for BDE-99, BDE-209, ΣHBCDs, PFOS, and PFOA are shown in Table 2, alongside the relevant HLBV, TDI, RfD, or dietary exposure estimate for comparison.

Table 2: Comparison of Exposure via Dust Ingestion with Exposure Guidelines or Dietary Exposure (all values expressed as ng/kg bw/day)

Compound	Low-end exposure (% arising in classroom)	"Typical" exposure (% arising in classroom)	High-end exposure (% arising in classroom)	Exposure guideline/dietary exposure
BDE-99	0.01 (18)	0.07 (27)	4.3 (6.5)	0.23-0.30[a]
BDE-209	2.2 (3.2)	28 (8.9)	13000 (0.4)	7000[b]
ΣHBCDs	0.55 (34)	5.9 (35)	330 (22)	24[c]
PFOS	0.10 (67)	0.75 (56)	12 (39)	300[d] 100[e]
PFOA	0.06 (19)	0.79 (15)	6.8 (19)	3000[f] 100[e]

[a]HBLV[12]
[b]RfD[13]
[c]UK average dietary exposure for toddlers aged 1.5-2.5 years[22]
[d]UK provisional TDI[6]
[e]GWDC reference value[8]
[f]UK provisional TDI[7]

Table 2 suggests that there is cause for concern regarding the exposures via dust ingestion of some young children in the UK for BDE-99 and BDE-209. While the numbers of children exposed at such levels will likely be low, our high-end exposure scenario estimates for both these contaminants exceed the relevant exposure guidelines. A far more reassuring picture emerges for PFOS and PFOA for which even our high end estimates of exposure via dust ingestion fall considerably below the provisional TDI levels set by the UK government and the German guideline exposure limits. While drawing firm conclusions on the basis of this limited dataset might

be premature, it would appear that while dust ingestion may make a substantial contribution to the exposure of young British children to PFOS and PFOA; on the evidence presented here, it does not appear to present a risk to health. Currently, there are no health-based guidelines against which exposure to HBCDs may be evaluated. However, Table 2 illustrates that dust ingestion is an important pathway of exposure to HBCDs for young British children, and emphasises the need for full evaluation of the health implications of such exposure. Table 2 also indicates the proportion of overall dust exposure arising from ingestion in the classroom. Overall, our data suggest that exposure while in the classroom is appreciable, but that it is generally less significant than that received while at home (which is usually the major contributor to overall dust exposure) or in a car.

It is also important to note that concentrations of PFHxS, MeFOSE and EtFOSE in classroom dust exceed those of both PFOS and PFOA. Both MeFOSE and EtFOSE may undergo *in vivo* metabolism to yield PFOS, thus contributing to human body burdens. Furthermore, while the toxicology of PFHxS is as yet uncharacterised, it's human half-life of 8.5 years exceeds that of PFOS and PFOA[23].

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