DUST FROM PRIMARY SCHOOL AND NURSERY CLASSROOMS IN THE UK: ITS SIGNIFICANCE AS A PATHWAY OF EXPOSURE OF YOUNG CHILDREN TO PFOS, PFOA, HBCDS, AND TBBP-A

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Introduction

Perfluorinated compounds (PFCs) have found widespread applications owing to their unique properties that have facilitated uses in the semiconductor industry, chrome plating, photography, and stain-proofing for a wide range of consumer goods[1]. Furthermore, their resistance to thermal, biological and chemical degradation (owing to the strength of the fluorine-carbon bonds) also confers environmental persistence, and a propensity for bioaccumulation. Hence, following release to the environment they are present in the human diet[2 3]. There are also concerns about the human toxicity of some PFCs, and the UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment has recommended provisional tolerable daily intakes (TDIs) of 0.3 and 3 μg/kg bw/d for PFOS (perfluorooctane sulfonate) and PFOA (perfluorooctanoic acid respectively[4 5]. The setting of such TDIs has generated a need to quantify the magnitude of exposure and the relative significance of different pathways. The UK Food Standards Agency has assessed dietary exposure of the UK population in 2004, but to date – despite reports from other countries on the concentrations of PFOS and PFOA in indoor dust [6 7 8] - no evaluation has been made of the significance of indoor dust ingestion as a pathway of exposure of the UK population to these chemicals. Studies of exposure via dust ingestion to other chemicals with significant indoor use patterns like brominated flame retardants has shown young children to be particularly exposed via this pathway[9 10], owing to the extra time that they spend in close proximity to the floor, coupled with their lower body weight. This paper therefore presents a preliminary evaluation of the contamination of indoor dust sampled from 20 classrooms frequented by young children (age range ~2-6 years), and estimates the potential exposure to PFOS and PFOA of British children. Quantification of related precursor compounds will be completed later in the project.

Classrooms for young children often contain large quantities of materials such as foam cushions, rugs and carpets, many of which have stain proofing chemicals applied during manufacture. Smart boards and computers are also a major component of classrooms and are used to aid the development of computer skills at an early age. In short, classrooms have many potential sources of PFOS, PFOA, and brominated flame retardants. PFOS and PFOA have already been detected in dust from 10 day care centres in Ohio and North Carolina, USA[8].

The ingestion of indoor dust has been highlighted previously as a potentially important exposure pathway for brominated flame retardants (BFRs)[9 10]. This study is focussed on Hexabromocyclododecane (HBCD) and Tetrabromobisphenol-A (TBBP-A). 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 high impact polystyrene (HIPS)[11]. The commercial formulations consist mainly of the α-, β-, and γ-diastereomers with γ-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[11]. 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 HIPS. It has been identified as an endocrine disrupter due to its structural similarity to 17-β-estradiol and thyroxine (T4), and 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[12]. We have shown previously that HBCDs are present at substantial concentrations in household dust from both the UK and North America and that ingestion of such dust contributes substantially to human exposure, with young children likely to be the most exposed sector of the population[9]. Elsewhere in this symposium we also address contamination of dust from a variety of microenvironments with both HBCDs and TBBP-A and estimate the resultant human exposure[13]. However,

these conclusions were based on analysis of domestic, office, and car dust only, and there are no data to our knowledge on the concentrations of HBCDs or TBBP-A in classrooms occupied by young children.

Material and Methods

Dust samples were collected from primary school and nursery classrooms 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 filtered through a 500 μm mesh and stored in the dark at 4ºC until extraction. Internal standards (^{13}C-labelled) PFOS and PFOA) 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 sulphate "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 even indicative concentrations of PFOS or PFOA in indoor dust. Hence, method accuracy and precision were evaluated by standard addition. Specifically, 0.1 g of a thoroughly homogenised indoor dust sample was "spiked" with the equivalent of 200 ng g^{-1} each of native PFOS and PFOA. This "spiked" dust aliquot was then analysed as normal, with the process repeated five times. The incurred concentration of each target compound in the dust sample was also determined, by analysis of an unfortified aliquot. After subtraction of this concentration from those determined in each of the "spiked" samples, the average recoveries of PFOS and PFOA were 102 and 96% (range 91-110% and 90-105%) respectively. The relative standard deviations of these recoveries were 6.8% (PFOS) and 6.3% (PFOA).

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 PFOS and PFOA 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 (1:1, v/v) (A), and methanol (B). The elution programme was 50%B for 5 minutes, then 100% B for 3 minutes, and finally 50%B for 10 minutes.

Separate aliquots of the same classroom dust samples were analysed for concentrations of α-, β-, γ-HBCD, and TBBP-A. Details of the methods used for extraction, clean up and analysis of these compounds are described elsewhere[9 13].

Results and Discussion

Table 1 summarises the concentrations of target compounds in the analysed samples. While concentrations of all target compounds vary within this dataset by at least an order of magnitude; those of HBCDs are the most skewed. In most samples HBCDs predominate, with the exception of a few where the levels of PFOS match or exceed them. Interestingly, the concentrations of PFOS and PFOA display a significant linear correlation, implying similar sources of these compounds in the classrooms studied.

Table 1: Summary of Concentrations (ng g^{-1}) of PFOS, PFOA, HBCDs and TBBP-A in Classroom Dust Samples

Parameter	PFOS	PFOA	ΣHBCD[a]	TBBP-A
Method Quantitation Limit	2.2	2.1	0.8	0.3
Range	85-3700	42-640	72-89000	19-440
Median	1200	220	5200	110
Mean	1300	240	13000	150

[a]Sum of α-, β-, and γ-HBCDs

Concentrations of PFOS and PFOA in dust samples in this study are consistent broadly with those reported previously in indoor dust samples from other locations[6 7 8]. For HBCDs and TBBP-A, ANOVA analysis of log-transformed concentrations from this study with those reported elsewhere in this symposium for dust samples in UK cars, offices, and homes[13], reveals the following:

1. Concentrations of HBCDs in classroom dust are significantly higher ($p<0.05$) than those in offices, but there is no significant difference in contamination between dust samples from classrooms, homes, and cars. However, after excluding the results in dust collected from classrooms with laminate floors (3 samples), concentrations in classroom dust significantly exceed those in homes.
2. Concentrations of TBBP-A in classroom dust are significantly higher ($p<0.05$) than in dust from cars and offices. There was no significant difference between concentrations in classrooms and homes even after removal of samples from classrooms with laminate floors.

While it would be unwise to draw firm conclusions on the basis of this comparatively small dataset, it appears that UK classrooms are contaminated with HBCDs and TBBP-A at concentrations significantly greater than those found in some other commonly frequented microenvironment categories. We report elsewhere that dust ingestion is an important pathway of exposure of the UK population to these BFRs, and the data presented here are consistent with that finding. Further detailed evaluation of the toxicological implications of such exposure is therefore justified.

There are currently no data on the concentrations of PFOS and PFOA in indoor dust from other UK microenvironments against which the classroom data reported here can be evaluated. Therefore we have derived estimates of the exposure of young children (age range 2-6 years) using average and high dust ingestion figures of 50 and 200 mg day^{-1} [14], and assuming 5th percentile, median, average, and 95th percentile concentrations in the classroom dust samples reported here. In the absence of concentrations in dust from other microenvironments, we have calculated the ingestion of PFOS and PFOA associated with dust from classrooms alone, assuming dust ingestion is pro-rata to the proportion of waking hours spent in the classroom per week (8 hours out of 12, five days a week). We have then divided the exposure estimates by an assumed typical child weight of 20 kg to normalise to body weight. On this basis, assuming high dust ingestion rates of dust contaminated at the 95th percentile concentration, exposure via dust ingestion is estimated to be 0.015 μg/kg bw/d for PFOS and 0.0024 μg/kg bw/d for PFOA. These estimates are substantially lower than estimates of high level *upper bound* (*i.e.* where PFOS and PFOA concentrations were below detection limits, they were assumed to be present at the detection limit) dietary exposure to the same sector of the UK population (0.4 and 0.2 μg/kg bw/d for PFOS and PFOA respectively[3]). However, they are more in line with the corresponding high level *lower bound* (*i.e.* where PFOS and PFOA concentrations were below detection limits, their concentrations were assumed to be zero) estimates of dietary exposure (0.09 and 0.009 μg/kg bw/d for PFOS and PFOA respectively[3]). Importantly, even our high end estimates of exposure via dust ingestion fall considerably below the provisional TDI levels set by the UK government. While drawing firm conclusions on the basis of this limited dataset is inadvisable, 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. However, more detailed characterisation of PFOS and PFOA concentrations in indoor dust from other microenvironments is required. Measurement of precursor compounds is also necessary, since these may be present at higher

concentrations than PFOS and PFOA in dust. Such compounds may undergo in vivo metabolism to yield PFOS and PFOA, thus contributing to human body burdens.

Acknowledgements
The authors acknowledge gratefully the NERC and Unilever for financial support of EG via studentship ref. NER/S/U/2006/14255. The support of the Egyptian government and Egyptian ministry of higher education for funding the studentship of MAA is also acknowledged.

References

1. OECD, 2006. *Organisation for Economic Co-operation and Development, series on risk management* No.22, ENV/JM/MONO(2006)36.
2. Tittlemier, S. A, Pepper, K., Seymour, C., Moisey, J., Bronson, R., Cao, X. L. and Dabeka, R.W. *J. Agric. Food Chem*, 2007; 55: 3203.
3. United Kingdom Food Standards Agency. *Fluorinated Chemicals: UK Dietary Intakes*; Food Survey Information Sheet 11/06; United Kingdom Food Standards Agency: 2006.
4. UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment, COT Statement on the Tolerable Daily Intake for Perfluorooctane Sulfonate, COT Statement 2006/09, October 2006.
5. UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment, COT Statement on the Tolerable Daily Intake for Perfluorooctanoic Acid, COT Statement 2006/10, October 2006.
6. Moriwaki, H., Takata, Y. and Arakawa, R. *J. Environ. Monit*., 2003; 5: 753.
7. Kubwabo, C., Stewart, B., Zhu, J. and Maro, L. *J. Environ. Monit.,* 2003; 7: 1074.
8. Strynar, M. J. and Lindstrom, A. B. *Environ. Sci. Technol*., doi:10.1021/es7032058.
9. Abdallah, M. A., Harrad, S., Ibarra, C., Diamond, M. L., Melymuk, L., Robson, M. and Covaci, A. *Environ. Sci. Technol*, 2008; 42: 459.
10. Harrad, S., Ibarra, C., Diamond, M. L., Melymuk, L., Robson, M., Douwes, J., Roosens, L., Dirtu, A. C. and Covaci, A. *Environ. Int.*, 2008: 34: 232.
11. Covaci A., Gerecke A.C., Law R.J., Voorspoels S., Kohler M., Heeb N.V., Leslie H., Allchin C. and De Boer J. *Environ. Sci. Technol*. 2006; 40: 3679.
12. Covaci A., Voorspoels S., Abdallah M.A., Geens T., Harrad S. and Law R. *J. Chromatogr. A* (*Submitted).*
13. Abdallah, M. A., Harrad, S. and Covaci, A. Hexabromocyclododecanes and Tetrabromobisphenol-A in Indoor Air and Dust in Birmingham, UK: Implications for Human Exposure, *ibid.*
14. Jones-Otazo, H., Clarke, J. P., Diamond, M. L., Archbold, J. A., Ferguson, G., Harner, T., Richardson, G. M., Ryan, J. J. and Wilford, B. *Environ. Sci. Technol.,* 2005; 39: 5121.