UNIVERSITY OF BIRMINGHAM

TOWARDS UNDERSTANDING THE FATE OF PERFLUOROALKYL COMPOUNDS (PFCs) WITHIN URBAN ENVIRONMENTS: IMPLICATIONS FOR HUMAN EXPOSURE.

by

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A thesis submitted to the University of Birmingham for the degree of:

DOCTOR OF PHILOSOPHY (Ph.D)

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November 2010

ABSTRACT

Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) have been detected across the globe in a variety of media. The toxicity of these compounds and other precursors has led to concern about human exposure. The purpose of this thesis is to investigate the presence of perfluoroalkyl compounds (PFCs) in UK indoor and outdoor microenvironments and the impact this may have on human exposure. Both PFOS and PFOA were chosen for analysis (via LC-ESI-MS/MS) because of their highly persistent behaviour. Additionally, perfluorohexane sulfonate (PFHxS), was chosen for its long half life in humans, along with precursors to PFOS and PFOA; perfluorooctane sulfonamides (PFOSAs) and perfluorooctane sulfonamidoethanols (PFOSEs).

Analytical methods were developed for the detection of these 8 perfluorinated compounds via LC-ESI-MS/MS for the separation and determination of levels in various samples including air, dust and soil. PFCs were detected at a high frequency in samples of air and dust from indoor environments. Significant differences were noted between the levels of contamination found in classrooms and other microenvironments (homes, offices and cars). PFOSEs (MeFOSE and EtFOSE) were the predominant PFCs detected in outdoor air samples with a median PFOSE concentration of 74 pg m^{-3}. By comparison, PFOS was the predominant PFC in soils from across the UK, at a median concentration of 560 pg g^{-1}.

The exposure of UK adults and children to these compounds via non-dietary sources was analysed revealing that the majority of such exposure for adults occurs in the home for all target PFCs. The situation is slightly different for children, who receive substantial exposure from both homes and classrooms. Under an assumed "typical" exposure scenario, adults are estimated to be exposed to 70 pg $(kg\ bw)^{-1}\ d^{-1}$ and 60 pg $(kg\ bw)^{-1}\ d^{-1}$ for PFOS and PFOA (respectively), whilst exposure for children under a

"typical" scenario is much higher at 640 pg (kg bw)$^{-1}$ d^{-1} and 470 pg (kg bw)$^{-1}$ d^{-1}. The elevated exposure of children from non-dietary sources is a consequence of their lower body weights, and greater dust ingestion rates. A simple single compartment, steady state pharmacokinetic model was also conducted to determine the likely contribution of PFOS and PFOA from non-dietary sources to concentrations in human blood serum. The results revealed non-dietary exposure can provide up to 5 % and 44 % of the mean serum concentrations in adults and children respectively. Compared to the European Food Safety Authority (EFSA) recommended tolerable daily intake (TDI) the exposure from non-dietary, plus dietary intakes for both adults and children remains well below the TDI values of 150ng (kg bw)$^{-1}$ d^{-1} and 1500ng (kg bw)$^{-1}$ d^{-1} , for PFOS and PFOA.

An international comparison of house dust was conducted and concentrations of PFCs in the UK (except for ethyl perfluorooctane sulfonamide (EtFOSA)) were found to be similar to those from France, Germany, USA, Canada and Australia and much greater than from homes in Kazakhstan and Thailand. Concentrations of EtFOSA were significantly higher in house dust samples from Australia compared to the UK, and were raised in all other countries. The reason for this is uncertain, but is speculated to derive from the use of EtFOSA as an active ingredient in Sulfluramid, which is an insecticide used for control of ants, cockroaches and termites.

ACKNOWLEDGEMENTS

Numerous thanks are owed to those who have supported, guided and inspired me throughout my studies, here at the University of Birmingham, 2006 – 2010.

I would like to return thanks and appreciation to my supervisor, Dr. Stuart Harrad, whose, advice, knowledge and encouragement has helped me in completing this project particularly throughout the highs and lows of this project.

I would also like to thank Richard Cubberley (Unilever) for his kind support and invaluable advice throughout my studies. Gratitude is also expressed to all those who aided in the wonderful task of dust sample collecting, a lot of which would have been unattainable without their help: Bibiana Baez-Brown, Matt Robson, Tim Evans, Dudsadee Muenhor and Mohamed Abdallah.

For all those who passed through the doors of room 216 throughout the 4 years for idle chat, intellectual conversations and impassioned discussions, a big thanks is expressed for all the good memories, fun and friendships made. And specifically to Dr. Dr. Dudsadee Muenhor, for her abiding friendship well into the future.

Utmost, for my family I wish to express my love and thanks for their indomitable encouragement and support. My parents have been an ancillary in the achievement of this PhD, with their immutable love and belief in me, enabling me to achieve my aspirations in life.

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ABBREVIATIONS

%RSD	Relative Standard Deviation (%)
<DL	Below Detection Limit
AFFF	Aqueous Film Forming Foam
ANOVA	Analysis of Variance
ATOFMS	Atmospheric Time of Flight Mass Spectrometer
BAF	Bioaccumulation Factor
BfR	Brominated Flame Retardant
Bw	Body Weight
C6	6 Carbon Atom Chain
C8	8 Carbon Atom Chain
COT	Committee On Toxicity
DCM	Dichloromethane
EFSA	European Food Safety Authority
EPA	Environment Protection Agency
EtFOSA	Ethyl-Perfluorooctane Sulfonamide
EtFOSE	Ethyl-Perfluorooctane Sulfonamido Ethanol
FOSA	Perfluorooctane Sulfonamide
FTCAs	Fluorotelomer Carboxylic Acids
FTOH	Fluorotelomer Alcohol
FTS	Fluorotelomer Sulfonate
FTUCA	Fluorotelomer Unsaturated Carboxylic Acid
GC-MS	Gas Chromatography - Mass Spectrometry
GFF	Glass Fibre Filter
HBCD	Hexabromocyclododecane
HDPE	High Density Polyethylene
HiVol	High Volume Air Sampler
HPLC	High Pressure Liquid Chromatography
IPE	Ion Pair Extraction
IS	Internal Standard
K_{OA}	*n*-Octanol/Air Partition Coefficient
K_{ow}	*n*-Octanol/Water Partition Coefficient
LC	Liquid Chromatography
LC-ESI-MS/MS	Liquid Chromatography Electrospray Tandem Mass Spectrometer
LLE	Liquid-Liquid Extraction
LOD	Limit of Detection
LOQ	Limit of Quantification
LoVol	Low Volume Air Sampler
LRM	Laboratory Reference Material
LSE	Liquid Solid Extraction
m/z	Mass To Charge Ratio
MeFOSA	Methyl-Perfluorooctane Sulfonamide
MeFOSAA	Methyl Perfluorooctane Sulfonamido Acetate
MeFOSE	Methyl-Perfluorooctane Sulfonamido Ethanol
MRM	Multiple Reaction Monitoring
MS	Mass Spectrometry
MS/MS	Tandem Mass Spectrometry

MTBE	Methyl-Tert Butylether
NCI	Negative Chemical Ionisation
N.D.	Non-Detect
NOEC	No Observed Effect Concentration
PAH	Poly-Aromatic Hydrocarbons
PBDE	Polybrominated Diphenyl Ether
PCB	Polychlorinated Biphenyls
PCDD/F	Polychlorinated Dibenzo-p-Dioxin/Furan
PCI	Positive Chemical Ionisation
PFAS	Perfluoroalkyl Sulfonamides
PFBS	Perfluorobutane sulfonate
PFCA	Perfluorocarboxylic acids
PFCs	Perfluorinated Compounds
PFDA	Perfluorodecanoic acid
PFDS	Perfluorodecane sulfonate
PFHxS	Perfluorohexane Sulfonamide
PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctane Sulfonate
PFOSA	Perfluorooctane Sulfonamides
PFOSE	Perfluoroctane Sulfonamidoethanols
PK	Pharmacokinetic
PLE	Pressurised Liquid Extraction
POP	Persistent Organic Pollutant
POPRC	POP Review Committee
PP	Polypropylene
PPG	Polypropylene Glycol
PTFE	Polytetrafluoroethylene
PUF	Polyurethane Foam
QA/QC	Quality Assurance / Quality Control
RRF	Relative ResponseFactor
SD	Standard Deviation
SIP	Sorbent Impregnated PUF
SPE	Solid Phase Extraction
SR	Sampling Rate
SRM	Certified Reference Material
TDI	Tolerable Daily Intake
VOC	Volatile Organic Compound
WAX	Weak-Anion Exchange

1. INTRODUCTION AND LITERATURE REVIEW

1.1. SYNOPSIS

Teflon was introduced to the commercial market in the 1950s, after being accidently discovered in 1938 by a scientist (Dr Roy J. Plunkett) whilst working on refrigerants for DuPont (Chemical Heritage Foundation, 2005). Teflon is a commercial name for polytetrafluoroethylene (PTFE), which is one of the slipperiest materials ever made, with a very low coefficient of friction in the range of 0.04 - 0.10 (Flom & Porile, 1955). This property of PTFE is what makes it so useful in numerous applications, and is created by the close affiliation and strong bonding of the fluorine to the carbon backbone. These polarised covalent bonds are some of the strongest found in organic chemistry and they also one of the shortest, resulting in the molecules being tightly packed to one another, with these properties increasing as the carbon chain length increases (Tatlow, 1984). Formulation of PTFE was varied, with changes occurring in the carbon length and addition of different functional groups, allowing the development of pefluorooctansulfonyl fluoride (POSF), which is an intermediate in the production of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). Thus perfluoroalkyl sulfonates and perfluorocarboxylates were produced at varying chain lengths for different applications, and further application of other functional groups evolved.

The application of perfluorinated compounds (PFCs) has benefited both the commercial market and industry, a list of past and present uses are noted in Table 1 and this chapter indicates the uses and functions of these chemicals, the extent to which they have been manufactured, and the physicochemical properties that allow PFCs to be utilised in a myriad of applications.

Table 1Applications of PFCs (Plastomer Technologies, 2009)

o Coatings for non-stick frying pans, and cooking pots	o Laboratory applications, tubing, piping and containers
o Waterproof and stain proof coatings for clothing	o Improves properties of paints, inks and lubricants
o Stain repellent finishes for carpets and car upholstery	o Heat resistant data cable insulation (used in jet engines)
o Improves thermoplastic wear-resistance	o UV and weatherproof coatings for graphics
o Used in aqueous film forming foam (AFFF) in fire extinguishers	o O-rings for machinery in contact with chemicals and oils.
o Semiconductor manufacturing	o Fabric protection
o Wipe-able wall paints	o Medical applications
o Automotive components used in areas in contact with gasoline and high temperatures	o Construction industry applications for sliding materials into place, on large builds.

PFCs are entirely derived from artificial synthesis and are unable to be produced in nature, however some may be biodegraded via natural mechanisms e.g. to perfluorooctane sulfonate and perfluorooctanoic acid (respectively, PFOS and PFOA). The widespread presence of PFCs is partially accounted for by their utilisation over many decades within a vast array of products. The chapter also outlines the widespread and diverse contamination of the environment, as a result of the global use of these compounds, and the media in which the chemicals have been discovered. The chapter will also examine the movement, spread and degradation of this family of compounds.

The impacts of PFCs on the human population have been studied, with many potential sources found in people's homes, work places and indoor environments (Kubwabo *et al.*, 2005, Björklund *et al.*, 2009). The presence in various environmental media implies there is concern of significant contamination potential to the food web, which has led to PFOS and PFOA being identifiable in top predators, such as polar bears, in remote arctic regions (Bossi *et al.*, 2005). As a result of contamination of water via industrial and wastewater effluent (Lin *et al.*, 2009), PFCs are able to pass through the cleaning filters in drinking water facilities, producing measurable concentrations of PFCs in regional drinking waters (Hölzer *et al.*, 2008, Skutlarek *et al.*, 2006). The presence in water also facilitates the

transportation of PFCs to oceans, and via oceanic movement towards polar latitudes (Yamashita *et al.*, 2005).

Both acute and chronic dose exposures in nature can produce toxic effects in biota (Liu *et al.,* 2009). The toxicity of compounds has been tested on various animals as well as gene and cellular bioassays. The outcome of these studies are subject to a high degree of variability, dependent on the compound half-life, organism and dose concentration, but results indicate various affects to the internal organ system, endocrine disruption and affects on foetal and embryonic development (Cui *et al.*, 2009, Kennedy *et al.*, 2004, Potera, 2009, Abbott *et al.*, 2009, Fei *et al.,* 2007). The risk associated with such sources of exposure, and contamination pathways are often described by simplistic models (Fei *et al.*, 2008, Pistocchi *et al.,* 2009, Egeghy & Lorber *et al.*, 2010), due to the complexity of the issue, and therefore the determination of risk is associated with a high degree of uncertainty.

Exposure from indoor environments and non-dietary sources can have a great effect on the contribution of chemicals to exposure, (e.g. lead and arsenic contamination), where dust intake can become the primary route of contamination for humans (Gaitens *et al.,* 2009). The exposure from non-dietary sources is investigated in this thesis, to identify the significance of indoor sources and pathways. Neglecting to take into consideration the contribution provided by the non-dietary pathways could produce a significant under-estimate of the overall daily exposure doses to PFCs.

Indoor environments receive PFCs from a plethora of sources including wall paints, floor waxes, stain-proofed carpeting, stain-proofed furnishings and textiles, waterproofed clothing and shoes (Washburn *et al*., 2005, Yamada *et al.* 2005), as well as kitchen and cooking sources, such as non-stick utensils and pots and pans and food container boxes used for heating and storing food (Begley *et al.*, 2005, Powley *et al.*, 2005, Sinclair *et al.* 2007), all of which allow PFCs to diffuse out during their lifetime. As well as these

applications, PFCs are also used as surfactants in many cleaning liquids, shampoos and other products that are used for the removal of grease, fats, dirt and oils.

Indoor dust potentially includes these chemicals because of migration, and wear and tear from the original products over time. The largest percentage of dust intake (generally) is received as an oral dose and ingested, as a result of hand-to-mouth behaviour (Stapleton *et al.*, 2008) and is hypothesised to be ingested via the general settlement of dust onto hands and settling on food. A smaller percentage is inhaled due to turbulence and re-suspension of dust, particularly indoors where each individual can create a personal dust cloud with movement and activity (Rhodes *et al.*, 1991, Conner & Williams, 2004, Allen *et al.*, 2007). There is also the possibility that PFCs may be absorbed via dermal exposure after contact with dust, or with products and surfactants containing PFCs (OECD, 2002).

The vapour:particulate phase partitioning of PFCs plays an important role in their atmospheric fate and behaviour. Potential precursor compounds to PFOA and PFOS are fluorotelomer compounds (8:2 fluorotelomer alcohol (FTOH), 6:2 fluorotelomer carboxylic acids (FTCA), etc) (Fasano *et al.*, 2006) and perfluorooctane sulfonamidoethanols such as (methyl- and ethyl-perfluorooctane sulfonamidoethanol (MeFOSE and EtFOSE, respectively)), which are more volatile and tend to have relatively shorter half-lives in the atmosphere (10 - 50 days, Martin *et al.*, 2006, Hurley *et al.*, 2004).

Indoor environments are comparatively closed systems, with higher temperatures and low ventilation rates that facilitate the build-up of chemical contaminants, resulting in the likelihood of indoor concentrations of PFCs that exceeding those of PFOS, PFOA and other semi-volatile compounds in the outdoor environment. Therefore, the potential exposure and daily doses are relatively high for humans who spend the majority of their day indoors.

1.2.PRODUCTION

PFOS was discovered in the 1930s with production beginning in the late 1940s via the Simons-electrochemical fluorination (ECF) process. Large scale production began with PFCs being utilised for their stain resistant properties, but were quickly employed for their other properties including lowering surface tension, and chemical resistance. Over the last decade however, environmental concern regarding the environmental persistence and harmful effects of PFCs has encouraged manufacturers in Europe and USA to reduce or cease production of chemicals that use or can degrade to PFOS and PFOA. Such concerns have culminated in the listing in 2009 of PFOS under the UNEP Stockholm Convention on POPs (The POPS, 2010). The EU has now regulated the importation of PFOS to uses which have no viable alternative chemicals, such as etching surfactants in semiconductor production and photolithography (Tang *et al.,* 2006, Hori *et al.,* 2004). Other milestones related to the production and use of PFCs can be found in Table 2.

Since manufacture of PFCs began, it is estimated that 96 000 t of the feedstock perfluorooctanesulfonyl fluoride (POSF) was produced between 1970 and 2002 (Paul *et al.,* 2009). This was manufactured primarily by the two major producers of PFCs, 3M and Dupont and, in 2000, 3M produced 78 % of the annual total volume (Paul *et al*., 2009). During the last 50 years, 3M has produced approximately 72 fluorinated compounds in wide-scale production. Of these, 21 comprised < 99 % of the total market demand of PFCs and were used for 6 main purposes: paper and packaging (including food), performance chemicals, aftermarket/consumer products, apparel, carpet, and home textiles (3M Speciality Materials, 2000).

Table 2 Milestones in the production and use of PFCs

Date	Chemical	Milestone	References
1947	PFOS & PFOA	Production began	3M Technical Document, 1995
1956	PFOS & POSF	Mass production began	Hekster *et al.,* 2003
1968	"Organic fluorine"	Detection of organic fluorine in human blood serum.	Taves, 1968
1985	PFDA	Identification of toxic effects on rats	Langley & Pilcher, 1985
1999	PFOS	3M identify PFOS in serum samples from their employs	Olsen *et al.,* 1999
2000	PFOS	3M announces phase-out after detection of PFOS in a wide variety of environmental media	3M Sustainability
2001	PFOS	Detection of PFOS in the Arctic	Giesy & Kannan, 2001
2002	PFOS	Ceased production by 3M	3M Sustainability
2002	PFAS	Control of production and importation of 75 PFCs began to be monitored by the US EPA, to reduce use and permit only essential uses.	US EPA, Significant New Use Rule.
2006	PFOS	Directive 2006/122/EC – restriction on PFOS in Europe.	European Commission
2009	PFOS & PFOA	Stockholm Convention – added as a POP and restricted use within Europe under section B of the convention	The POPs
2010	PFOA	U.S.A. - planned 95% reduction in facility emissions	US EPA, 2009
2015	PFOA	U.S.A. - Eliminate facility emissions	US EPA, 2009
2016	EtFOSA	U.S.A. - Phase out deadline for Sulfluramid	US EPA, Significant New Use Rule.

Production in the last 10 years has moved from North America and Europe primarily to China, where it has been increasing since 2003, and Brazil, where PFCs are incorporated into insecticide baits (Sulfluramid – containing ethyl perfluorooctane sulfonamide (EtFOSA)) (Machado-Neto *et al.*, 1999) used to manage and restore areas of the rainforest (Campoe *et al.*, 2010). Chinese production of perfluorooctane sulfonyl fluoride (POSF) increased fourfold from 2004 to 2006, by which time production exceeded 200 tons a year (Bao *et al.*, 2009). However, these raw chemicals do not all remain in China, approximately 100 tons was transported to South America and a small amount to Europe (Bao *et al.*, 2009), for use in industrial manufacturing e.g. processing of semiconductors,

metal plating, photolithography and certain aviation fuels. All of these continued uses are permitted under the new Stockholm Directive, despite the restriction of PFCs from more commercial uses in the EU.

The USA estimates that, on an annual basis, 5000 tonnesof FTOHs are currently produced worldwide (Telomer Research Council, 2002); a figure of relevance owing to their potential to undergo environmental degradation to PFASs and PFCAs.

PFCs are manufactured via two main techniques, electrochemical fluorination (ECF) and telomerisation. ECF is based upon the electrolysis of a hydrocarbon analogue of the target PFC in liquid hydrogen fluoride. A feedstock of perfluorooctanesulfonyl fluoride (POSF) is produced from ECF and undergoes reaction with other chemicals to form various PFCs. Often POSF is used as the hydrocarbon analogue in aqueous hydrogen fluoride.

Telomerisation is based upon the polymerisation of an unsaturated perfluoroalkene in the presence of perfluoroalkyl iodide and, unlike the ECF process, yields only linear PFCs (Jahnke *et al.,* 2007b). Around 34 – 40 % of the POSF feedstock goes to make linear PFCs via the ECF process, while the rest results in isomers and homologous by-products and unsaturated fluorocarbons (3M, 1999). These by-products are highly variable from site–to–site and chemical batch. They are often released from the manufacturing facilities (specifically the volatile compounds, vented through stacks), or are present in the final product (less volatile compounds) with subsequent release over time, thus resulting in a variety of PFCs reaching the environment. The production of some PFCs requires multiple ECF steps due to the hydrocarbon analogues having poor reaction yields, which increases the quantities of potential waste products produced during the manufacturing.

Production began in North America and companies expanded to northern Europe, where the uses of the chemicals were widespread (Paul *et al.* 2009). A large amount of the waste and contamination from these products has remained within the northern hemisphere, where via the movement of oceanic circulations and air masses they have been cycling northwards, towards the polar regions (Young *et al.* 2007, Wania *et al.* 2007). However,

despite this dominant use in the northern hemisphere, over the years production and use have spread and contamination is identifiable in the southern hemisphere environment and, more specifically, Antarctica, where concentrations have been identified in penguins (Tao *et al.*, 2006). Present production is primarily in China and Brazil since the increased regulations and restrictions in North America and the European Union, and therefore products are now undergoing enhanced transport to southern Asia, Australasia and South America (Paul *et al*. 2007, Armitage *et al.*, 2007).

Current uses of the primary compounds include incorporation into consumer goods and solutions applied to these goods and incorporation within industrial fluids and use as an insecticide (Sulfluramid). In cases where the application of the PFCs is for consumer use, it has been possible to encourage manufacturers to discontinue use of PFOS and PFOA for alternatives (often fluorotelomer products or shorter chained PFCs, < C8). However, other applications for PFOS continue to be used, particularly in industry, where there are currently no other alternatives for the compound. In the past decade the use of PFCs, particularly the more bioaccumulative and persistent compounds, have been scrutinised leading to the cessation or reduction of their use. This is particularly prevalent for PFOS and PFOA, but also includes compounds such as ethyl-perfluorooctane sulfonamide (EtFOSA), which is often incorporated into industrial insecticides.

1.3.PHYSICOCHEMICAL PROPERTIES

PFCs have a variety of physical traits, which render them highly resistant to biological, physical and chemical degradation, facilitating their many uses. PFCs consist of a fully fluorinated hydrocarbon backbone chain and attached to this are various functional groups (R). The perfluoroalkyl moiety $F(CF_2)_xR$ is depicted in Figure 1 and represents the basic structure of the PFCs.

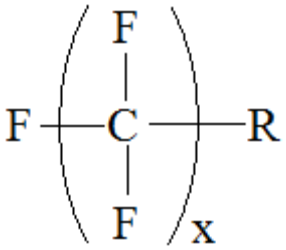


Figure 1 Perfluoroalkyl moiety

The PFC moiety displays inertness thermally, chemically and biochemically due to the strong covalent bonds present between the fluorine and carbon atoms. This F-C bond is the strongest of the organic chemistry bonds as a result of the electronic structure of both atoms, and this produces a high electronegativity and polarity.

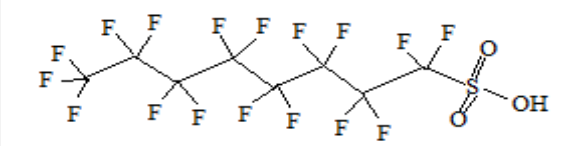
PFOS presents amphiphatic characteristics as a result of its polarity and hydrophobicity produced by the (perfluoro) carbon backbone and hydrophilicity via the perfluorinated tail. Combined, these properties make PFOS a highly versatile and useful compound (De Voogt & Saez, 2006). However, they also cause it to partition into proteins in biota (Weiss *et al.,* 2009, Jones *et al.,* 2003) rather than adipose tissue, as is the case with other halogenated persistent organic pollutants (POPs). PFCs also have the ability to lower surface tensions of solutions, but this behaviour is highly dependent upon the number of

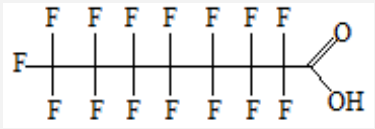
fluorine atoms present in the PFC and the equivalent free surface energy (Sakka & Ogata, 2005).

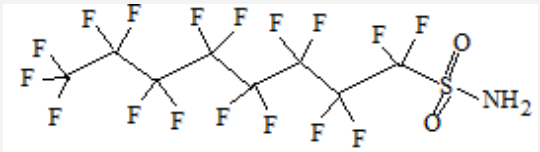
Differences between the behaviour and properties of various PFCs are induced by whether the compounds are branched or linear, the number of carbon atoms in their backbones, and also primarily whether the carbon backbones are fully or partially fluorinated (Upham *et al.*, 2009, Rayne *et al.*, 2009, Ochoa-Herrera *et al.*, 2008). The distinctive behavioural characteristics of PFCs are produced by the strong C-F bonds, which afford substantial resistance to environmental degradation (Olsen *et al.*, 2007, Latala *et al.*, 2009.).

PFOS and PFOA are more persistent than other perfluorinated compounds (including others measured in this study) because they are both formed of a fully fluorinated backbone, with a small functional group attached (Shoeib *et al.*, 2006, Prevedouros *et al.*, 2006). The fully fluorinated backbone of the compounds confers resistance to many degradation pathways because of the strong C-F bonds and the fact that the energy required to break this is not generally encountered in nature (Butt *et al.*, 2009). Some illustrative physicochemical and other relevant properties of four common PFCs found in the environment are displayed in Table 3.

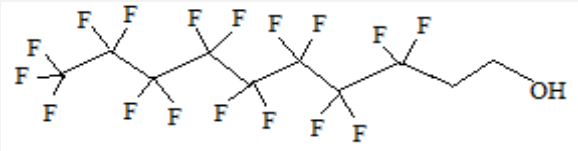
Table 3 Illustrative physicochemical and other relevant properties of PFOS, PFOA a sulfonamide and a FTOH

PFOS	**sodium perfluoro-1-octanesulfonate**
Boiling Point	Not calculable
Melting Point	> 400˚C (EFSA Journal, 2008)
Molecular Weight	500.13
Structure	
Vapour Pressure	3.31 x 10^{-4} Pa @ 20˚C (EFSA Journal, 2008)
Water Solubility	519 mg L^{-1} @ 20˚C in pure water (EFSA Journal, 2008) 12.4 mg L^{-1} in salt water (Giesy *et al*., 2010)
Log Koc	2.57 (Higgins and Luthy, 2006)
Air-water Partition Coefficient	<2 x 10^{-6} (3M Company, 2003)
Henry's Law Constant	3.05 x 10^{-9} atm. m^3 mol^{-1} pure water (EFSA Journal, 2008)
Behaviour	Hydrophilic head, hydrophobic and oleophobic tail. Persistent and bioaccumulative, resists environmental, chemical and biodegradation.
Uses	Surface-active agents in aqueous media, chemical intermediate; acid catalyst for photoresists, surfactant in aqueous fire fighting foam, surfactant for alkaline cleaners; emulsifier in floor polish; mist suppressant for metal plating baths; surfactant for etching acids for circuit boards; pesticide active ingredient for anti bait traps.
Manufacturing	Hydrolysis of perfluorooctylsulfonyl fluoride (POSF)
Production	Began in 1949 by 3M and continued until ca. 2002
Regulatory highlights	Addition of PFOS to Stockholm convention annex B in August 2009, restricting use to industrial use, where no alternatives were available. Cessation of use in chromium plating industry.
Toxicity/metabolism	Metabolism results in $C_8F_{17}SO_3^-$ (3M & EPA, 2003) Absorbed orally and distributed to plasma/the liver (Cui *et al*., 2009) Elimination from urine, faeces, childbirth and lactation (Harada *et al.,* 2005, Apelberg *et al.,* 2007) Toxicity noted in lab. animals (Sprague-Dawley rats and monkeys (Lefebvre *et al.,* 2008, Fuentes *et al.,* 2006)
TDI	COT – 300 ng (kg bw) $^{-1}$ d^{-1} (COT, 2006a) EFSA – 100 ng (kg bw) $^{-1}$ d^{-1} (EFSA Journal, 2008)
Human Half-life	5.4 years (Olsen *et al*., 2007)

PFOA	**perfluoro-1-octanoic acid**
Boiling Point	189˚C (National Toxicology Program)
Melting Point	52-54˚C (National Toxicology Program)
Molecular Weight	414.09
Structure	
Behaviour	Persistent and bioaccumulative, resists environmental, chemical and biodegradation.
Vapour Pressure	4.2 Pa @ 25˚C (EFSA Journal, 2008)
Log K_{oc}	2.06 (Higgins & Luthy, 2006)
Uses	Dielectric liquid material proposed for use to replace PCBs in transformers. Used to produce PFOA salts – processing aids in production of fluoropolymers and fluoroelastomers. Additive in aqueous fire fighting foam, cosmetics, greases and lubricants, paints, polishes and adhesives, fluorinated surfactants.
Production	ECF – used as a processing aid in telomerization for fluorotelomer production but is not incorporated into the final technical mixture (DuPont, 2008).
Regulatory highlights	Voluntary reduction of PFOA by 95 % in 2010 and complete phase out of stack emissions by the USA's top 8 producers by 2015 (US EPA Stewardship Programme, 2010).
Toxicity	Metabolic effects in newborn mice (Rosen *et al.*, 2007) Developmental toxicity in mice (Hines *et al.*, 2009)
TDI	COT – 3000 ng (kg bw) $^{-1}$ d^{-1} (COT, 2006b) EFSA – 100 ng (kg bw) $^{-1}$ d^{-1} (EFSA Journal, 2008)
Human Half life	3.8 y in serum (Olsen *et al.*, 2007)

EtFOSA	**N-ethyl perfluoro-1-octanesulfonamide**
Melting Point	88 - 90˚C
Molecular Weight	527
Structure	
Behaviour	Semi-volatile compound, with short half-life, can be degraded in the environment and via metabolisation in both humans and animals.
Vapour Pressure	7 Pa @ 25°C (Lei *et al.*, 2004)
Uses	Surfactants for protection of fabrics and papers, food packaging coatings (Begley *et al.*, 2005) and used industrially for anticorrosion and antistatic agents (O'Brien *et al.*, 2006) and insecticide – sulfluramid (Campoe *et al.*, 2010).
Manufacturing	Electrochemical fluorination

Production	>17 tonnes were manufactured in 2005 for pesticide use (OECD, 2006), though the real figure is likely to be raised with one countries reported use for 2005 being in the range of 10 - 30 tonnes and additional applications, such as an additive for toner or printer ink (OECD, 2006).
Regulations	Controls in place for use as an insecticide vary between jurisdictions. Countries restricting use include UK, N. America, Canada, Australia.
Toxicity	Immunotoxicity in rabbits (O'Brien *et al.*, 2006), suppression of immunoglobulin (l g) M production (Peden-Adams *et al.*, 2007)
Atmospheric lifetime	20 – 50 days (Martin *et al.*, 2006)

8:2 FTOH	**Perfluorooctyl ethanol**
Boiling Point	193˚C (Stock *et al.*, 2004)
Melting Point	50˚C (Stock *et al.*, 2004)
Molecular Weight	464
Structure	
Behaviour	Volatile compounds. Degrades via photolyic and bio- degradation and undergoes metabolisation in humans and animals (Fasano *et al.*, 2006).
Vapour Pressure	31 Pa @ 25˚C (Cobranchi *et al.*, 2006)
Uses	An intermediate for the manufacture of finishing products; paints, waxes, coatings, polishes and adhesives for commercial use, and uses in the semiconductor industry.
Production	Telomerization, 2000 – 2002 global production of all FTOH estimated at 5 - 6.5 x 10^6 kg y^{-1} (Telomer Research Program, 2002)
Regulatory highlights	Not currently regulated
Toxicity	Cytotoxicity in rats after bioactivation (Martin *et al.*, 2009) Developmental toxicity to neonatal mice (Liu *et al.*, 2009) Endocrine disruptor to MCF-7 breast cancer cells (Maras *et al.*, 2006)
Atmospheric Lifetime	c.a. 20 days (Ellis *et al.*, 2003)

1.4.USES

The properties mentioned in the previous section permit multiple uses of PFCs. A small array of uses is presented in Table 4, for commercial and industrial products. The hydrophobic, lipophobic and oleophobic properties of PFCs are often utilised in commercial food packaging, to provide coatings to paper and card packaging with greater resistance to oils and fats from foodstuffs. The surfactant properties of PFCs allows for numerous uses in both industrial and commercial applications. Commercial uses of PFCs include use as surfactants in washing detergents, shampoos, and cleaning agents.

Table 4 PFC containing products

Product Name	Use	Perfluorinated Ingredient	Manufacturer
Scotchgard, 3M	Stain-proof coatings	PFOS, PFOA	Ceased use of PFCs in Scotchgard in 2002
'Scotch' products, 3M		PFBS	Produced from 2001
Teflon, Dupont	Non-stick coating	PTFE	By 2010 will achieve 95 % reduction in PFOA emissions
Gore-Tex	Water and stain resistant coating for textiles	PTFE	Produced since 1970s
PTFE	Non-stick coatings, for carpets, clothing, highly resistant seals, tubing, insulating wires	PTFE	Produced since 1950s
Capstone and Zonyl fluorosurfactants (DuPont)	Additive in paints, polishes, coatings, waxes, and cleaning surfactants. Also used as a foaming agent for reduction in surface tension whilst drilling in the oil field.	FTOHs	Currently in production
Sulfluramid (CAS NO.: 4151-50-2) Trade name: FT-9	Insecticide, acaricides, pesticide ingredient and used as an intermediate for perfluoro surfactant production.	EtFOSA	17 tonnes produced in 2005 from 17 countries (OECD, 2006) Main production in China

In industry, the surface tension properties are utilised in aqueous fire fighting foam (AFFF), mist suppressants for acid baths, and surfactants in photolithography and semiconductor manufacturing.

Since the addition of PFOS to the Stockholm Convention on persistent organic pollutants (POPs), use and production has become restricted and limited (under annex B) to semiconductor industry production of photo-resists, etching components and anti-reflective coatings, use in aviation oil hydraulics, metal plating, medical devices, reservoirs of aqueous film forming foams (AFFF) containing PFOS and insect baits for ant control. Exemptions are also in place for uses in oil drilling, liquid crystal display (LCD) production, treatments for carpets, leather and upholstery, papers and packaging, rubber and plastics (The POPs, 2010).

With PFOS coming under greater restriction, utilisation of less persistent perfluorinated compounds is occurring in many products, such as paints (e.g. Dupont Capstone® paints), waxes and surface coating products. The use of FTOHs is preferable because they are less persistent, and have extremely low half-lives in biota (Dinglasan *et al.,* 2004).

PFBS is being considered as a viable substitute for PFOS, as it has been shown to contain only linear isomers in industrial production. PFOS manufacturing produces linear and branched PFOS isomers, along with a number of other waste products (Vyas *et al.,* 2003), which can all enter the environment and all behave differently.

1.5.PFCS IN THE ENVIRONMENT

Environmental impacts of PFCs can be monitored via their widespread presence in various types of biota and environmental media from around the world (Figure 2). Concern regarding the wide distribution of PFCs gained much attention when they were detected in top predators from polar regions (Begley *et al.* 2005), which is an indication of the widespread use and distribution of PFCs. The lack of use in polar regions, suggests that the pollution in polar

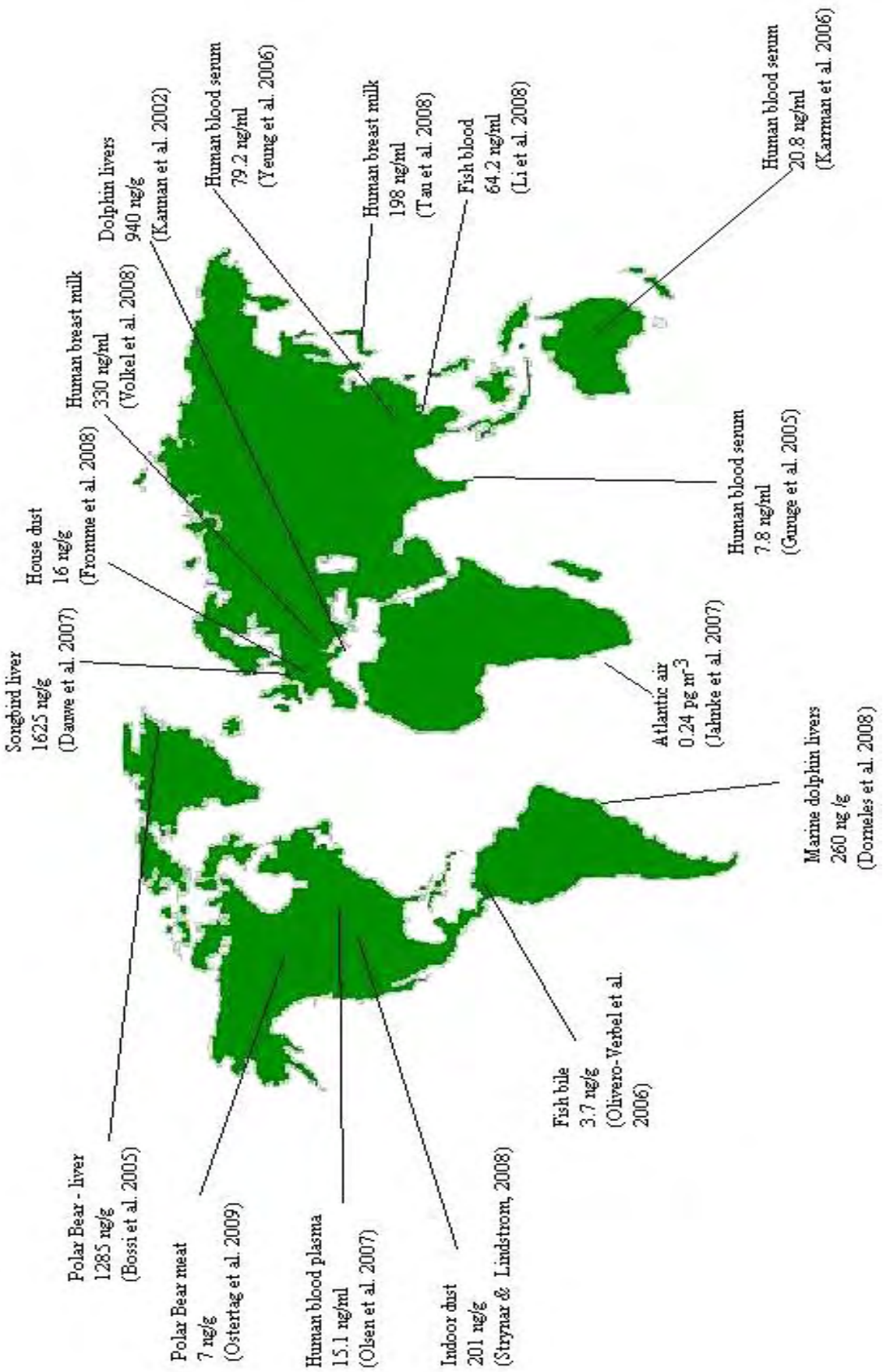


Figure 2 Illustrative graphical summary of PFC measurements from across the globe

bears (Begley *et al.* 2005) is the result of long-range atmospheric and pelagic transportation from industrialised regions, followed by contamination of polar environments.

Figure 2 provides a brief example of the detection of PFOS in a variety of media from around the globe. PFCs have been detected in the environment since the 1980s, but the extent of the global contamination became more evident from the turn of the millennia when detection techniques began to improve. As a result PFCs were identifiable in a variety of samples distributed around the world, both near and far from industrial production and populated regions. The detection of PFCs in remote or polar regions indicates that the chemicals are being transported and distributed globally.

1.6.SOURCES

Sources of PFCs include direct and indirect releases, point and diffuse releases, and have many different contributing factors. Though PFCs are an anthropogenic family of chemicals, there are some indirect sources of the more resilient, less volatile compounds as a result of environmental degradation. Sources have been identified to be greatest in urban regions, with a direct correlation to population density (Murakami *et al.,* 2008). Outdoor air receives inputs from indoor environments and industrial releases through stack emissions and migration from work facilities. Buildings can act as individual "hotspots" of contamination to the external environment with concentrations typically reaching a magnitude of 10 times higher than outdoors (Shoeib *et al.,* 2005). Sources in indoor environments are numerous, and include wear and tear from products coated with PFCs such as carpets, sofas and textiles (3M, 1999). Diffusion of the chemical compounds also occurs over time from sources such as paints and surface coatings but can also be directly dispersed into the atmospheric environment from release of PFC products used for coating consumer items (for stain resistance or waterproofing). For example, it was

calculated that approximately 44 % of emitted PFCs from such products (coated clothing and textiles) are disseminated directly into the surrounding environment (EPA Release Report, 2000).

Along with homes acting as sources to the outdoor environment, direct releases to the environment often occur from manufacturing because of the use of PFCs as intermediates within manufacturing processes, and are often released through stacks and wash effluents (Armitage *et al.*, 2009). This is particularly relevant for PFOA, where atmospheric concentrations are primarily a result of use in the fluorotelomer production (via telomerisation) process, where the compound is not incorporated into the final product and released as waste. Emissions of PFOA from US manufacture of fluorotelomers are now being controlled and are due for phase out in 2016 (US EPA, 2009). Direct releases to the environment have also occurred from uses of aqueous film forming foam (AFFFs) and will continue to be a possible source because of the present stock reservoirs in Europe. This has become particularly important for the UK, where local contamination became apparent after AFFFs were used on an oil depot fire at Buncefield in 2005 (Powell, 2006). Resultant impacts from the mass use of AFFFs included reports of physical deformation of calves and low survival rates (Anslow, 2007), as well as detection in aquatic sites further down the catchment (HPA, 2007). Rural regions receive the majority of their atmospheric sources from urban regions, as detected by back trajectories (Martin *et al.*, 2002), and the spreading of waste sludge to fertilise agricultural land (Swedish Chemical Inspectorate, 2004). Emissions to the environment also arise from the use of Sulfluramid, an insecticide widely used to prevent the spread and damage of leaf-cutter ants, and cockroaches (Campoe *et al.*, 2010, Schal *et al.*, 1992). However, this is not a relevant pathway for the UK, as Sulfluramid is not licensed for use in the country.

1.7. AIR

The presence of PFCs in air samples from around the world supports the idea of global movement of these compounds. Concentrations of PFCs in the gaseous state can even be located via oceanic atmospheric concentrations in polar regions (Dreyer *et al.,* 2009). Differences in the two hemispheres result from the main manufacturing base residing within the northern hemisphere. A concentration gradient exists between the Northern and Southern hemispheres, with oceanic atmospheric concentrations being significantly different (Dreyer *et al.,* 2009). Volatile PFCs along with PFCAs and PFASs were detected in the Canadian Arctic, with the precursors being measured in the vapour phase and the PFCAs and PFASs present in the particulate phase. However, it is possible that the PFCAs and PFASs present in Arctic waters contribute to the atmospheric concentrations via marine aerosols (Stock *et al.,* 2007).

Atmospheric concentrations are highly variable for PFCs because of differences in compound volatility and atmospheric half-life. The compounds considered to be semi-volatile include perfluorooctane sulfonamides and sulfonamido ethanols (PFOSAs and PFOSEs), and FTOH, which are common in air masses around urban centers, and industrial areas (Kim & Kannan, 2007). The concentrations of these compounds attenuate with increasing distance from source regions (Dreyer *et al.* 2010, Gewurtz *et al.,* 2009).

These chemicals tend to be distributed via atmospheric transport to colder climates, which is possible due to the relatively long atmospheric half-lives of the volatile PFCs (20 – 50 days for FTOH, PFOSEs and PFOSAs). However the chemicals can be removed from the atmosphere by wet or dry deposition which, although negligible for FTOHs, is thought to be a major pathway for the removal (in ca 10 days (Hurley *et al.,* 2004)) of particulate-bound PFC (Dreyer *et al.,* 2010). At this point the chemicals are removed from the air, the more volatile can re-volatilise, and particulates can be re-suspended, allowing further movement and transportation. Movement back into the atmosphere for PFASs and PFCAs is inhibited by high solubility in water and uptake by biota (Arp & Goss, 2009). FTOHs

have been shown to reach the Arctic (Shoeib *et al.*, 2006), prior to degradation, and are therefore thought to be a significant source of PFOS and PFOA in the Arctic.

Increases in atmospheric concentrations were noted in summer in Northern Europe, with diffuse sources emitting to the gas phase. Lohmann *et al.*, (2007) made comparisons of the log K_{OA} (octanol/ air partition coefficient) and log K_{OW} (octanol/ water partition coefficient) values for a range of POPs, in the context of their capacity for global transport. The work depicts the presence of PFOSEs, FTOH, and PFOSAs in a region on the diagram associated with primarily direct atmospheric global distribution (Figure 3).

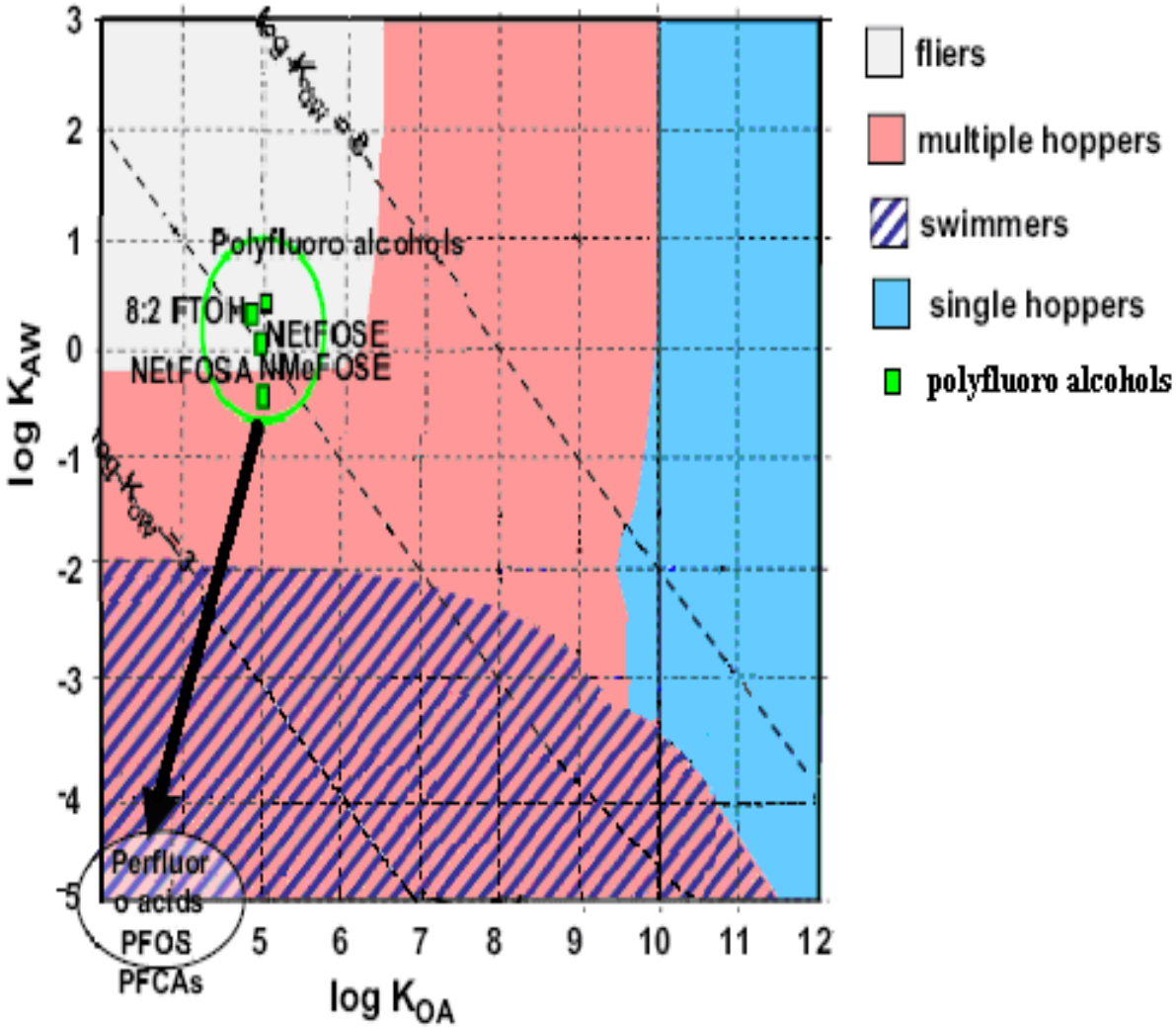


Figure 3 Major modes of POPs transportation, derived from hypothetical chemicals, with PFCs and other common present day compounds placed onto the diagram (Lohman *et al.*, 2007)

The less volatile perfluorooctane sulfonamides (PFOSAs) are located towards the region dominated by chemicals which are transported via hopping (the process of a continuous cycle of transportation and deposition through the environment). Perfluoroalkyl sulfonates (PFASs) and perfluorocarboxylic acids (PFCAs) are situated within the diagram far from the volatile PFCs, located within the region of global movement via oceanic pathways rather than hopping as a result of their low partition coefficients. This behaviour suggests PFASs and PFCAs will be easily deposited via wet and dry deposition, within an estimated time scale of around 10 days (Hurley *et al.,* 2004) and continue to be transported via aquatic pathways.

The degradation of FTOH as presented by Ellis *et al.,* (2003) suggests that degradation can occur via oxidation of the 4:2, 6:2, and 8:2 FTOH species (with the presence of chlorine atoms initiating the process). This degradation is initiated via OH radicals and the addition of O_2 to produce fluorotelomer aldehydes; continuous reactions with peroxy radicals and O_2 produce perfluorinated aldehydes or perfluorinated alkoxy radicals which can subsequently degrade to PFCAs. However, this pathway is unlikely to be relevant in polluted urban atmospheres, because of the impact of NO_X and the unzipping reaction of the FTOH, producing COF_2 molecules (Ellis *et al.,* 2004). The degradation of FTOH to PFCAs is affected by seasonal and altitudinal differences, producing a time range of FTOH (gaseous) to degrade over a matter of weeks to months (Yarwood *et al.,* 2007). The summer months see an acceleration in the rate of FTOH degradation within the northern hemisphere due to increased photolysis and presence in atmospheric free radical production, thus impacting the production of PFCA, where degradation of FTOHs provides 8% of PFCAs in summer compared to 1% in winter (Yarwood *et al.,* 2007).

Perfluoroalkyl sulfonamides and amido alcohols, and fluorotelomer olefins, which are semi-volatile, can also undergo degradation to PFCAs (Martin *et al.,* 2007, Nakayama *et al.,* 2007). These precursors have sufficiently long half-lives to undergo transport from urban and industrial regions to rural and remote locations (Nakayama *et al.,* 2007). Semi-

volatile PFCs such as FTOHs, PFOSAs and PFOSEs also have half-lives consistent with an ability to undergo widespread spatial distribution. These chemicals degrade via oxidation and peroxy radical reactions, thus FTOHs, PFOSAs and PFOSEs are not scavenged as quickly in rural regions because of lower NO_x concentrations.

Perfluorinated acids have been demonstrated to have an atmospheric half-life of approximately 130 days with respect to OH radical reaction (Hurley *et al.*, 2004). However, the atmospheric presence of these compounds is a lot shorter than this (around 10 days, (Hurley *et al*., 2004)) as a result of the wet and dry deposition which occurs because of the extremely low partition coefficients, and the persistent nature of the compounds.

An urban – rural gradient exists because of the high number of sources within urban regions and high scavenging rates by free radicals. Thus as the perfluorinated compounds are transported away from urban areas they are deposited and scaveneged from the atmospheric environment.

Concentrations in outdoor air from Europe and North America are summarised in Table 5. Rural measurements indicate generally higher concentrations of FTOHs compared to PFOSAs and PFOSEs, which is as a result of greater deposition via aerosol scavenging and wet deposition of the latter. Compared to the urban locations, rural regions generally have slightly lower values for FTOHs, PFOSAs and PFOSEs, but the urban:rural gradient is not as substantial as might be expected if concentrations were linked solely with population density (Pistocchi & Loos, 2009). Comparisons of data produced by different studies are difficult because of the differences in sampling methods, seasonality, and atmospheric conditions between studies. Rural regions within North America and Europe are likely to receive atmospheric concentrations from a large variety of back trajectories (urban centres, industry, airports, etc).. Marine atmospheric concentrations are lower than urban and rural values, apart from concentrations measured by Jahnke *et al*., 2007a in a German marine region.

Table 5 Median outdoor air concentrations (pg m^{-3})

Reference	Location	*n*, sample	6:2 FTOH	8:2 FTOH	10:2 FTOH	FOSA	Me-FOSA	Et-FOSA	Me-FOSE	Et-FOSE
Barber *et al.*, 2007	UK, rural	*n* = 2	81	100	75	<2.1[a]	5.5	7.9	24	9.2
	Ireland, rural	n = *4*	5	11	7.8			<1.6	<80	<53
Jahnke et al., 2007b	Germany rural	*n* = 6	29	85	28	n.d.	7.4	3.2	6	8.4
Martin *et al.*, 2002	Canada, rural	*n* = 2	29	32	17				35	76
Piekarz *et al.*, 2007	USA, remote	*n* = 34	4.6	24	15			<3.2	<11	<3.7
Barber *et al.*, 2007	Norway, suburban	*n* = 2	12	34	17	0.78[a]	5.5	7.9	24	9.2
	UK, urban	*n* = 2	190	240	65	<1.6[a]	6.1	9.6	24	6.4
Martin *et al.*, 2002	Canada, urban	*n* = 4	87	55	29			14	100	210
Dreyer & Ebinghaus, 2009	Germany urban	*n* = 8	26	65	20	0.4	2.6	2.8	2.6	1.2
Shoeib *et al.*, 2006	Canada, urban	*n* = 3	18	41	22				12	3.3
Shoeib *et al.*, 2004	Canada, urban	*n* = 1							32	9.8
Jahnke et al., 2007b	Germany urban	*n* = 6	56	110	29	n.d.	7	2.6	18	3.5
Shoeib et al., 2005	Canada, urban	*n* = 7						n.d.	82	88
Dreyer & Ebinghaus 2009	Germany Marine	*n* = 7	8	38	9.6	2.6	2.9	1.1	1.6	0.7
Jahnke *et al.*, 2007a	Germany Marine	*n* = 3	160	180	46		3.8	2	19	8.9
	Spain, Marine	*n* = 3	11	29	9.2		1.9	0.8	3.9	n.d.
	South Africa, Marine	*n* = 3	n.d.	2.4	0.9		0.5	n.d.	n.d.	n.d.
Shoeib *et al.*, 2006	Remote, ocean	*n* = 20	2.7	15	7.1				12	2.9
Stock *et al.*, 2007	Canadian Arctic	*n* = 10	<14	9	<1.5	20		11	31	<27

[a] Particulate measurement only

Measurements collected in a marine environment in South Africa (Jahnke *et al.*, 2007a) indicate the concentrations are extremely low, with detection of only 8:2 and 10:2 FTOH and MeFOSA, which is expected for Southern Hemisphere regions because of the low production and use rates.

The majority of compounds detected in atmospheric samples from the Southern Hemisphere will have been transported there and consist of the relatively volatile PFCs (Dreyer *et al.*, 2009, Ahrens *et al*., 2009) as there is no known production in this continent and little known importation (OECD 2002), the more volatile compounds will have been transported further distances. In comparison to this the concentrations from remote Arctic regions have measurably higher concentrations than the South African marine environment because of the lack of production and use in the Southern Hemisphere.

Concentrations in the southern hemisphere do display similar behavioural traits (cycling to the polar regions, more volatile compounds in the ocean atmospheric samples), with concentrations increasing in areas with high population density, and primarily FTOH concentrations in the Southern Ocean (Dreyer *et al.*, 2009).

Indoor atmospheric concentrations have been measured at concentrations 10 - 100 times that of outdoor concentrations (Shoeib *et al.,* 2004, Shoeib *et al.,* 2005). Elevated concentrations in indoor environments have been detected in a number of studies (see Table 6) from Canada and Norway, and substantially exceed concentrations detected outdoors (Table 5). The indoor environments tend to have higher concentrations than outdoors due to them generally being a closed system, with little ventilation in and out of the environment, and a high number of sources arising from commercial goods (Yamada *et al.* 2005). Although there are only limited results, attempts have been made to attribute differences in concentrations in indoor environments to the presence of different indoor sources. It appears that elevated indoor concentrations result from the release of PFCs from indoor products, carpeting and furnishings (Vestergren *et al.*, 2008, Trudel *et al*., 2008).

Table 6 Median indoor air concentrations (pg m^{-3})

Reference	Location	*n*, sample	6:2 FTOH	8:2 FTOH	10:2 FTOH	Me-FOSA	Et-FOSA	Me-FOSE	Et-FOSE
Shoeib et al., 2005	Canada, houses	*n* = 59					40	1800	740
Shoeib et al., 2004	Canada, houses	*n* = 4						4600	1000
Shoeib et al., 2007	Canada, houses	*n* = 52		2100	890				
Barber *et al.*, 2007	Norway, houses	*n* = 4	3000 (<40)[a]	3400 (<10)[a]	3600 (13)[a]	6600 (6)[a]	6600 (7)[a]	6000 (360)[a]	5800 (76)[a]
Jahnke *et al.*, 2007b	Norway, offices	*n* = 2	180, 250	850, 420	900, 1.6		190, 160	730, 800	300, 820

[a] vapour (particulate) concentrations

1.8. WATER

Environmental concentrations of PFCs are highly variable due to various parameters (Figure 2) including locality, environmental medium, sources, sinks, and volatility of compounds (Lohmann *et al.,* 2007, Giesy & Kannan, 2001). It is thought that the bulk of transportation of PFCs to the polar regions is oceanic (Yamashita *et al.,* 2005). Atmospheric PFCs can undergo wet and dry deposition to the oceans, as well as hopping along a transect to the poles (Stock *et al.*, 2007), but the majority of contamination originates from waste water and discharges to rivers (Simcik & Dorweiler, 2005). Along with this global transportation, the presence of PFCs in water provides the chemicals a pathway into drinking water and the food web (Schuetze *et al.*, 2010).

Concentrations displayed in Table 7 indicate a range of aqueous environments with measurable concentrations of PFCs from various regions around the world. This suggests that contamination of waterways and oceans occurs even in regions where manufacturing (the largest source to rivers) is not present.

Table 7 Concentrations of PFCs in water samples (ng L^{-1})

Reference	Location, *n* samples	Type	PFOS	PFOA	PFHxS	FOSA
RIVERS						
Senthilkumar *et al.*, 2007	Japan, *n* = 5	River water	6.5	59	<6.6	<3.7
Skutlarek *et al.*, 2006	Germany, *n* = 50	River water	220	1600		
Skutlarek *et al.*, 2006	Germany, *n* = 29	Surface water	11	240		
Jin *et al.*, 2009	China, *n* = 34	Yangtze river	4.7	5.4		
So *et al.*, 2007	China, *n* = 2	River	23	4.3	<0.67	0.15
SEAWATER						
Taniyasu *et al.*, 2005	Japan, *n* = 2	Tokyo Bay	14	35	2	0.31
Taniyasu *et al.*, 2005	Japan, *n* = 2	Tomakomai Bay	2600	64	92	350
Ahrens *et al.*, 2009	N. Atlantic, *n* = 40,	Surface open-ocean water	<0.01- – 0.3	<0.004 – 0.23	n.d.	<0.017-0.31
Ahrens *et al.*, 2009	Mid-Atlantic, *n* = 10	Surface open-ocean water	<0.01 – 0.06	<0.004 – 0.087	n.d.	<0.017 – 0.06
Ahrens *et al.*, 2009	S. Atlantic, *n* = 10	Surface open-ocean water	<0.01	<0.004	n.d.	<0.017 – 0.053
LAKES						
Boulanger *et al.*, 2004	USA, *n* = 8	Lake Erie	31	36		0.9
Boulanger *et al.*, 2004	USA, *n* = 8	Lake Ontario	55	45		1
Jin *et al.*, 2009	China, *n* = 7	Lake & Pond	4	3.9		
Loos *et al.*, 2007	Italy, *n* = 8	Lake	7.8	2.4		
OTHER						
Scott *et al.*, 2006	Canada, *n* = 119	Precipitation, rural		0.1 - 10		
Scott *et al.*, 2006	Canada, *n* = 75	Precipitation, urban & suburban		0.1 – 89		
Liu *et al.*, 2009	China, *n* = 21	Precipitation, urban	145	25	0.36 – 2.1	
Jin *et al.*, 2009	China, *n* = 13	Ground & river, rural	0.4	0.1		
Jin *et al.*, 2009	China, *n* = 15	Ground & river, urban	5.7	4.1		
Schultz *et al.*, 2006	*n* = 10	Cleaned waste water effluent	24	11	1.2	4.6
CONTAMINATED LOCATIONS						
Moody *et al.*, 2002	Canada, *n* = 6	153 days after release of 22000 L AFFF	320	20		
Hansen *et al.*, 2002	USA, *n* = 20	Upstream of fluorochemical manufacturing discharge	32	<LOQ		

Reference	Location, *n* samples	Type	PFOS	PFOA	PFHxS	FOSA
Hansen *et al*., 2002	USA, *n* = 20	Downstream of fluorochemical manufacturing discharge	110	350		
Moody *et al.*, 2003	USA, *n* = 10	Ground water at fire-training base	32000	33000	50000	
POTABLE						
Skutlarek *et al.*, 2006	Germany, *n* = 44	Drinking water	3	39		
Quinones & Snyder, 2009	USA, *n* = 3	Drinking water, utility 1	<1	<1	<1	
Quinones & Snyder, 2009	USA, *n* = 6	Drinking water, utility 2	9.4	11	2.2	
Quinones & Snyder, 2009	USA, *n* = 33	Drinking water, utility 3	1.2	<5	<1	
Quinones & Snyder, 2009	USA, *n* = 7	Drinking water, utility 4	<1	<5	<1	
Quinones & Snyder, 2009	USA, *n* = 7	Drinking water, utility 5	22	30	12	
Quinones & Snyder, 2009	USA, *n* = 5	Drinking water, utility 6	<1	n.d.	<1	
Quinones & Snyder, 2009	USA, *n* = 5	Drinking water, utility 7	57	18	6.1	
Jin *et al.*, 2009	China, *n* = 34	Tap water	1.8	3.4		
Loos *et al*., 2007	Italy, *n* = 6	Tap water	8.1	2.4		

PFCs are largely deposited out of the atmosphere within close proximity to conurbations (Mahmoud *et al.*, 2009) and urban regions constitute a large input of PFCs to water systems (Loewen *et al.*, 2005, Murakami *et al*., 2008). This input includes direct deposition and releases to rivers running past highly populated areas as well as from wastewaters, sewers and wash-off from impervious materials (buildings, streets etc) and surface runoff. Along with this deposition, urban regions also tend to pollute with direct releases from manufacturing. Rainfall in urban regions can constitute a pathway of PFCAs to water sources (Scott *et al.*, 2006, Dreyer *et al.*, 2010).

This has led to samples taken downstream of large conurbations and at the mouths of rivers having greater concentrations than rural and groundwater measurements (see). Inland river concentrations measured in Germany (Skutlarek *et al.*, 2006) also appear to be high in respect to concentrations from coastal rivers in China (So *et al.*, 2007, Jin *et al.*, 2006) and water measured downstream of fluorochemical industry waste discharges. The

relatively high concentration in the German rivers is likely to have been contributed to by the industry along the banks of the river and the high population on the river banks. Table 7 shows urban regions contain significantly higher concentrations in comparison to rural and remote locations (defined as at least 100 km from large conurbations). Urban regions showed a 2 - 10 fold increased concentration flux compared to rural regions, which is expected to derive from the degradation of fluorotelomer unsaturated carboxylic acids (FTUCAs), which were positively correlated with PFOA concentrations (Scott *et al.*, 2006). Rural regional waters become contaminated as a result of atmospheric deposition of PFCAs (Scott *et al*., 2006) following transportation and atmospheric degradation of FTOHs. Measurements of wet-precipitated PFCAs, fluorotelomer carboxylic acids (FTCAs) and FTUCAs were measured in North America by Scott *et al.,* (2006) and shown to aid movement from urban atmospheric concentrations into urban waterways.

Sources to water include a range of direct releases from industrial uses and spills (Lin *et al.*, 2009, Moody *et al.* 2002, Hansen *et al.*, 2002, see). Concentrations in rivers situated downstream of industrial uses generally remain as the regions with the highest concentrations (Pan & You, 2010). Large amounts of PFOS and PFOA were also delivered to rivers from manufacturing facilities as a waste product from the telomerisation process for fluorotelomer production (Davis *et al.*, 2007) along with other manufacturing discharges including chrome plating and semiconductor facilities (Lin *et al.*, 2009, Kelly & Solem, 2009).

Wastewaters not only contain contamination from urban wash-off, and industrial releases but from wastewater from washing of clothes, washing of indoor environments and washing of cooking utensils (Washburn *et al.* 2005, 3M Specialtiy Materials). The washing of clothes coated in PFCs aids in the transfer of PFCs into the waterways with 73 % of PFC coatings expected to wash off over a 2 year life span (3M Speciality Materials, 2000).

As sewage is treated (at waste water treatment plants), some semi-volatile compounds volatilise into the atmosphere, whilst the more persistent compounds (mainly PFAS and PFCAs) remain within the effluent (rather than partitioning to sludge) and are discharged into rivers (Becker *et al.*, 2008, Sinclair & Kannan, 2006). Discharge from wastewater treatment plants (Murakami *et al.*, 2009, Bossi *et al.*, 2008, Lee *et al.*, 2010, Boulanger *et al.*, 2005), run off from biosolid use and leaching from landfill (Cheng *et al.*, 2008, Bossi *et al.*, 2008, 3M, 2001) also occurs.

River water concentrations can be highly variable depending on their location, size and industrial inputs (Hansen *et al.*, 2002), but both urban and rural rivers can contain measurable concentrations of PFOS and PFOA. This has resulted in marine bays, situated at the mouths of rivers containing higher concentrations than other water environments (Taniyasu *et al.*, 2005). Marine bays are also likely to be influenced by the large amount of traffic and use of the bay and the population of boats and people within the area (Taniyasu *et al.*, 2005). Although Tokyo bay remains relatively low (Table 7) the trend is identified from a number of other bays in Japan (Taniyasu *et al.*, 2005), and other sites (Dorneles *et al*, 2008, Sakurai *et al.*, 2010, Li *et al*., 2008)

Thus, the PFCs head towards the oceans and oceanic transportation moves them towards the polar regions (Butt *et al.*, 2010, Yamashita *et al.*, 2005). Despite concentrations decreasing with distance from conurbations and the continents, concentrations of PFCs are still measurable in the Atlantic Ocean towards the Arctic regions (Dreyer *et al.*, 2009). The presence of perfluorinated acids has been detected in the circulatory deep oceanic waters, suggesting a theory of potential mixing and transportation to the poles (Yamashita *et al.*, 2008). This also suggests that the southern pole will be affected by PFCs, despite a reduced volume of PFCs being used in the southern hemisphere, interhemispheric transport from the north is possible (Tao *et al.*, 2006, Jahnke *et al.*, 2007a) and the increase of PFC use in the southern hemisphere is being observed, thus it is expected that concentrations in the Southern Oceans will begin to increase from present values.

All these direct source discharges end up contributing to the oceanic burden. The lowest concentrations are seen in oceans because of the amount of dilution occurring and the limited number of sources. Movement of FTOHs occurs via atmospheric trans-Pacific transport from Asia towards the Americas, a small proportion of which is a source to the American west coast (Primbs *et al.,* 2008). However, a larger proportion of the trans-Pacific plume is deposited onto the ocean surface, either as a FTOHs scavenged by aerosols or as a degradation product allowing for atmospheric concentrations to add to the burden of the oceans.

Tap water is also affected by this presence of PFCs in water, as current clean-up processes are unable to remove the more persistent compounds, particularly PFOS and PFOA. Table 7 suggests concentrations in tap water can be comparable to those in river water (Jin *et al.*, 2009, Loos *et al.*, 2007) and can lead to considerable human exposure (Hölzer *et al.*, 2008).

1.9. SEDIMENTS

Sources to sediment occur through deposition of compounds in the atmosphere, wash off from non-permeable surfaces, use of pesticides and insecticides containing PFCs, and sludge waste spreading for fertilisation of agricultural land. PFOS and PFOA behave in a hydrophobic fashion and bind with sediment, rather than remaining in the aqueous phase (Martin *et al.*, 2004, Ahrens *et al.*, 2010). Concentrations of PFCs identified in sediment from various locations around the globe are included in Table 8, and represent the presence of contamination in various soil and sediment types (Beach *et al.* 2006).

Sources in rural regions are primarily a result of atmospheric deposition, the use of waste sludge as a fertiliser and the use of PFC-containing pesticides (Scott *et al.*, 2006, Diaz-Cruz *et al.*, 2009). Concentrations in urban soil tend to be higher, because of higher population densities and associated concentration of PFC-containing consumer goods

producing higher rates of deposition to soils. Contamination in urban soils is likely to be exacerbated by the comparatively smaller proportion of soil available in urban areas (due to the number of impermeable surfaces and building denisity) to which PFCs may bind. Moreover, PFCs are susceptible to urban run-off into waterways and associated sediments. PFCs are retained strongly by sediments, because of their hydrophobic properties (and weak hydrophilic property induced from the tail of the compound), but the type of sediment (presence of cationic and anionic surfactants) as well as the pH, organic content and moisture exert a strong influence on the extent to which PFCs can undergo absorption to and desorption from sediment (Pan *et al.,* 2009, Becker *et al.,* 2008, Higgins *et al*., 2006). This can also influence the movement and transportation of PFCs globally (Pan *et al.,* 2009). It has also been shown that PFOS can undergo aquatic transport over long distances before being scavenged by sediment in estuaries because of the change in salinity (Pan & You, 2010). PFCs appear to reside mainly in upper sediment layers and in top soil, primarily as a result of such layers having high organic carbon and protein content. Biodegradation occurs in sediment for the more volatile compounds, but PFOS and PFOA are remarkably persistent in this environment (The POPs, 2010, Fromme *et al.*, 2009).

8:2 FTOH and other precursor compounds have been observed to break down in soils and sludges via microbial degradation (Parsons *et al.,* 2008). Reductive defluorination is relatively limited because of the strong C-F bonds, but the degradation of the moieties attached to the main fluorinated hydrocarbon allows PFCs to be transformed to PFCAs and PFASs. PFCs have been detected in groundwater samples (particularly around landfills) (Cheng *et al.,* 2010, Jin *et al*., 2009) primarily the perfluorooctane sulfonamides and sulfonamidoethanols and shorter chained <C6 compounds (Ahrens *et al.,* 2010), indicating the potential for movement through soil and desorption from surface sediments.

Table 8 Mean Concentrations of PFCs in sediment (ng kg^{-1})

Reference	Sediment type and location	PFOS	PFOA	PFHxS
Harino *et al.*, 2009	Marine core sediment, Japan (0-1 cm), *n* = 1	900 600 2100	<100 500 <100	
Becker *et al.*, 2008	Germany (0-15 cm), *n* = 11	110	27	
Senthilkumar *et al.*, 2007	Japan, Tenjin river, *n* = 1	11	2.1	<1.4
Clara *et al.*, 2009	Austria, Lake Constance, *n* = 5	<940	380	
	Austria, Alpine Lakes, *n* = 6	n.d.	230	
	Austria, Danube Riverbank, *n* = 8	280	1400	
Bao *et al.*, 2009	China, Hun River site 1 (0-10cm), *n* = 6	170	130	<130
	China, Taizi River, site 1, *n* = 6	290	120	n.d.
	China, Daliao River, site 1, *n* = 6	140	110	n.d.
Kumar *et al.*, 2009	USA, Savannah River, *n* = 5	300 – 800	n.d. – 200	n.d. – 300
Higgins *et al.*, 2005	USA, Bolinas Lagoon, *n* = 3	380	290	n.d.
	USA, Hayward Marsh, *n* = 3	1700	630	<200
	USA, Kirby Park, *n* = 3	201	<100	<200
	USA, Palo Alto Mudflats, *n* = 3	1500	140	n.d.
	USA, Petaluma River, *n* = 3	1200	230	n.d.
	USA, Salinas River, *n* = 3	1300	170	n.d.
	USA, San Francisquito Creek, *n* = 3	3100	250	n.d.
	USA, San Pedro Creek, *n* = 3	250	270	n.d.
	USA, Yosemite Slough, *n* = 3	290	270	<200
	USA, Baltimore Inner Harbour, *n* = 3	850	390	72
	USA, Gwynn's Run, *n* = 3	n.d.	190	n.d.
Stock *et al.*, 2007	Canada, Resolute Lake, (0-1 cm) (associated with a spillage of AFFF from the airport)	85 000	7500	3500
	Canada, Char Lake (0-1 cm)	1100	1700	n.d.
	Canada, Amituk Lake (0-1.5 cm)	62	960	1000

Table 8 indicates that the majority of studies have concentrated on aquatic sediments, with PFOS and PFOA preferring to partition out of water (Ahrens *et al.*, 2010). This supports the work by Martin *et al.*, (2004) which indicates that, in the aquatic system, the majority of uptake was via the sediment uptake by benthic organisms and entering the food web in Lake Ontario and not directly from the water. The hydrophobicity of the perfluorinated compounds, partitions PFOS and PFOA into sediment rather than water (Pan & You, 2010, Higgins *et al.* 2006) in aquatic systems. Few studies have concentrated on soil samples,

and though there is an understanding of the presence and potential routes into soils, global concentrations are not well documented.

1.10. BIOTA

With PFCs reaching water bodies, air, soils and sediments, there are an abundant number of pathways and uptake routes for their entry into the food web. With the compounds being present in the environment for over 50 years, the concentrations in biota represent the widespread distrbuition of these compounds along with the high volume of use of the chemicals.

PFCs have been detected in a wide variety of biota from around the world, ranging from those at the bottom of the food web (Martin *et al.* 2004), to top predators, and examples of some of the biota affected are detailed in Table 9. Aquatic organisms from the tidal flats of the Ariake Sea, Japan, were identified to accumulate concentrations of PFCs (particularly PFOS) from the concentrations in water (Nakato *et al.*, 2006), concentrations in fish have been identified from around the world (Table 10), as well as in dolphins and whales and other aquatic mammals. Research has shown that PFOS and other PFCs can be acutely toxic to freshwater organisms (Boudreau *et al.*, 2003, Latala *et al.*, 2009), however, this does not appear at the ambient concentrations seen in most rivers and oceans (see Table 7), with lehal concentrations (LC50) ranging between 31 – 169 mg L^{-1} for Daphnia Magna, Selenastrum Capricornutum, Calluna Vulgaris, Lemnaceae Gibba, and Daphnia Pulicaria. (Bourdeau *et al.*, 2003). Moreover, at lower concentrations and over more chronic exposure durations, concentrations are likely to remain within the organisms, and will continue up the food chain.

Their detection in various environmental media from across the world (as can be seen in Table 7 and Table 8) indicates the presence and input of PFCs in media and their

availability to be taken up by biota, including biota in remote locations. Table 9 lists concentrations in a variety of biota from various countries, including birds, mammals, and aquatic animals. PFCs have been quantified primarily in the livers of animals, because of their propensity for binding with proteins.

Table 9 Concentrations in biota (ng g^{-1})

Reference	Animal	PFOS	PFOA	FOSA
	Liver ng/g			
Guruge *et al.*, 2008	Cattle, *n* = 14	33	<LOD	<LOD
(Japan)	Chicken, *n* = 14	67	<LOD	<LOD
	Pig, *n* = 6	54	0.04	<LOD
Hart *et al.*, 2009	Sea Otter (2007), *n* = 2	2.8		<LOD
(Alaska)				
Senthilkumar *et al.*, 2007	Cormorants, *n* = 5	130	2.7	234
(Japan)	Racoon Dog, *n* = 2	26	4.8	8.9
	Eagle, *n* = 2	43	2.1	45
	Crow, *n* = 2	6.6	0.3	1
Van De Vijver *et al*., 2007	Porpoise, *n* = 31	210		
(Black Sea)				
Tanabe *et al*., 1997	Porpoise, *n* = 41	180		
(Black Sea)				
Kannan *et al*., 2006	Sea Otter, *n*= 4	55	60	<1
(Souhtern Sea)				
Taniyasu *et al.*, 2005	Beaver, *n* = 2	130	0.29	0.82
(Japan)				
Tomy *et al.*, 2004	Walrus, *n* = 5	2.4	0.34	
(Eastern Arctic)	Beluga, *n* = 5	13	1.6	21
	Glaucous gull, *n* = 5	20	0.14	
	Narwhal, *n* = 5	11	0.9	6.2
Tao *et al*., 2006	Albatrosses, *n* = 102	2.2		
(South Pole)				
Kannan *et al.*, 2002	Cormorant, *n* = 12	62	95	<38
(Baltic)				
	Blood / Serum ng/mL			
Kannan *et al.*, 2001	Polar bear	68		
(Arctic)				
Tao *et al*., 2006	Elephant seal, *n* = 59	0.53		
(South Pole)	(blood)			
Giesy *et al.*, 2001	Snapping Turtle	72		
(USA)	Bald Eagle	360		
	Albatross	18		
Houde *et al*., 2005	Bottle Nose Dolphins			
(USA)	Sarasota Bay, *n* = 13	780	6.3	4.5
	, Bermuda, *n* = 2	49	0.8	4.8
	IRL, *n* = 42	640	12	1.5
	Charleston, *n* = 47	1300	44	29
	Delaware Bay, *n* = 5	750	72	20

PFCs are also present in the blood serum (bound to proteins) in animals including fish (Table 10). Table 9 also indicates that the presence of PFCs extends throughout the food web at different trophic levels.Concentrations of PFOS detected in top predators from remote environments were one of the key pieces of evidence presented to the POPs review committee (POPRC) considering the listing of PFOS under the Stockholm Convention. The presence of PFOS in top predators indicates the potential for food chain biomagnification. It should be noted that certain PFOS isomers display greater bioaccumulation factors (BAFs) than others, (Houde *et al.,* 2008). Note also that PFOA is also present in biota but to a lesser extent than PFOS (Tomy *et al.,* 2004).

Fish can act as important indicators of the presence and availability of a pollutant in the aquatic environment, as they feed upon algae, microorganisms and small bottom feeders, all of which can contribute to the concentration within the fish (Kannan *et al.* 2007, Butt *et al.,* 2010).

The detection of PFCs in freshwater fish can indicate the presence of local sources to the waterways as well as distribution from upstream catchments and air masses by comparison, the presence of PFCs in oceanic biota can indicate that the compounds are capable of long-range environmental transport. This is indicated by the concentrations measured in various fish from the eastern Arctic Ocean by Tomy *et al.,* (2004). The presence within fish also highlights a potential human exposure pathway via fish consumption, which is of particular relevance to coastal populations and cultures with a higher than average fish component to their diet (Berger *et al.,* 2009, Nania *et al.,* 2009). Table 10 indicates the presence and bioaccumulation of PFCs in various fish species from around the world. The partitioning behaviour of PFCs with proteins is displayed by the presence of PFCs at measurable concentrations in fish muscle and liver samples.

Table 10 Concentrations of PFCs in fish (ng g^{-1})

Reference	Sample Type	Location and Samples	PFOS	PFOA	FOSA	PFHxS
Shi *et al.*, 2010	Muscle	Tibet, $n = 59$ (various types)	2.1	<DL		N.D.
Delinsky *et al.*, 2009	Muscle	Bluegill Sunfish, USA, $n = 91$	88			
Hart *et al.*, 2008	Liver	Skipjack Tuna, Eastern Asia, $n = 54$	13		1.30	
Ye *et al.*, 2008	Muscle	Carp, Mississippi River, USA, $n = 30$	28	<DL		<DL
Senthilkumar *et al.*, 2007	Liver	Mackerel, Japan, $n = 3$	1.6	10	<4.3	<8.5
Nania *et al.*, 2009		Variety of edible fish,				
	Muscle	Mediterranean Sea, $n = 26$	9	9		
	Liver	Mediterranean Sea, $n = 17$	33	8		
	Squid	Mediterranean Sea, $n = 30$	3	2.5		
Tomy *et al.*, 2004	Whole fish	Arctic Cod, $n = 6$	1.3	0.16	n.d.	
	Liver	Arctic, Redfish, $n = 7$	1.4	1.2	n.d.	
	Whole fish	Arctic, Shrimp, $n = 7$	0.35	0.17	n.d.	
Kannan *et al.*, 2002	Liver	Tuna, Baltic Sea, $n = 8$	47	<72	<38	<7

1.11. HUMAN EXPOSURE

PFCs have been detected in human blood (see Table 11), urine and milk (see Table 12) samples from various populations across the globe. Most striking is the presence of PFCs in umbilical cord blood (Inoue *et al.,* 2004) and in umbilical cord blood spots from New York state (Spliethoff *et al.,* 2008). The presence in urine, also suggests that either less than 100 % of PFCs are absorbed across the gastrointestinal tract, or that they are excreted endogenously.

Unlike other organohalogens like PCBs and brominated flame retardants (BfRs) that partition into lipids within the human body, PFCs partition preferentially into proteins due to their lipophobic tail. Therefore PFCs are easily cycled around the body in the blood, and tend to reside in protein-rich organs, such as the liver (Olsen *et al*., 2003), pancreas, lungs, and kidneys (Maestri *et al.*, 2006).

Various studies have identified relationships between body burdens and gender, age, and ethnicity, as well as spatial and temporal influences and occupational exposure. Since the elimination of PFOS in 3M products and the subsequent reduction of PFC use across North America and Europe, the concentration of PFCs in blood have decreased (Spliethoff *et al.,* 2008, Olsen *et al.,* 2007, Calafat *et al*., 2007). Differences between genders have been found to be slight (Butenhoff *et al.,* 2006) with those that do exist, attributable to female loss via child birth and lactation (Vestergren *et al.*, 2008, Calafat *et al.,* 2006, Toms *et al.,* 2009). Children tend to contain higher concentrations than teenagers or adults (Trudel *et al*., 2008, Toms *et al*., 2009). Human exposure is variable and differences have been attributed to gender, race, age and occupation (Ericson *et al.*, 2007, Apelberg *et al.*, 2007, Kato *et al.,* 2009, Vestergren *et al.,* 2008, Toms *et al.*, 2009, Wilhelm *et al.*, 2009). The presence of PFOS in the environment has led to contamination of populations, who do not live

western lifestyles, unlike with some BFRs where lifestyle is a more crucial pathway of exposure (Dallaire *et al.,* 2009). Concentrations measured in human blood have been shown to vary with location as a result of variation of use patterns in different countries (Tao *et al.,* 2008a). Regions with high usage of textiles, carpeting and surface finishing products (such as painted walls and waxed floors) in homes and other indoor microenvironments, appear to have higher concentrations in people compared to those in less developed countries where materials tend to be locally made and contain no PFC coatings. Regional differences have also been attributed to climatic factors (Dreyer *et al.,* 2010); e.g. warmer climates tend to have greater building ventilation rates allowing greater escape of PFCs to the outdoors. Also, fewer carpets are present thus excluding a potential source from the building, and non-carpeted rooms tend to retain fewer dust particles, because of different cleaning techniques and carpets retaining dust better. Differences in body burdens on ethnic grounds were reported by Calafat *et al.,* 2006, but the differences were minimal with a significant difference ($p < 0.01$) only found between non-Hispanic white Americans and Mexican-Americans, which suggests that external parameters (such as life style) are likely to be responsible rather than genetic factors.

Concentrations in blood samples have been reported as declining since 2002 in the USA (Olsen *et al.*, 2007). This is consistent with the decrease in PFOS manufacture in the USA since 2001, the date when the main producer of PFOS (3M) ceased production and USA industries signed up to a voluntary decrease in use of the chemicals. However, chemical formulations of PFCs have simply changed to more volatile or shorter chained compounds. Therefore, exposure is occurring from the more volatile compounds as well as the degradation products because of the unzipping volatile compounds (Hurley *et al.*, 2004) undergo prior to forming less

volatile compounds, particularly PFOS and PFOA. More long term assessments need to be conducted in order to develop a better understanding of the presence of PFOS and PFCs and whether the restrictions imposed on PFC use are having a positive effect on human exposure.

Humans are at the top of the food chain and have been shown to be exposed from dietary intakes, including meats and fish, as well as vegetables and other foods (HPA 2006, Tittlemier *et al.*, 2007). However, dietary intake of PFCs is also influenced by the packaging and cooking utensils used, due to leaching from the materials during cooking (Sinclair *et al.* 2007, Powley *et al.*, 2005). These can often contain PFCs due to their ability to resist fats and oils, allowing foods to be packaged in paper and cardboard without fats and oils penetrating through the packaging. Humans are also exposed via contact with air, dust and soil. The concentrations of PFCs are higher in air and dust from indoor environments compared to outdoor environments(Shoeib *et al.*, 2005), and sediment and air concentrations in urban regions are higher than concentrations from rural and remote regions (Simcik & Dorweiler, 2005). Therefore, depending upon the amount of time spent in certain microenvironments, the exposure to PFCs can vary greatly.

Concentrations detected in domestic indoor dust including PFOS and PFOA span 3 orders of magnitude (Kubwabo *et al.*, 2005, Bjorklund *et al.*, 2009). Other perfluorinated compounds have also been detected in some home microenvironments, including PFHxS, EtFOSA, and PFOSEs, with a large range in concentrations, reaching up to 4 orders of magnitude differences between individual environments (Shoeib *et al.*, 2005). Dust concentrations within homes appear to be highly variable within individual studies (Shoeib *et al.*, 2005) but also differences are noted between countries, which are likely to be the impact of different manufacturing mixtures and

use patterns, variation in room contents, and differences resulting from cultural and climatic differences.

Indoor environments contribute to a greater exposure for people because of the greater number of products (potentially) containing PFCs, the reduced dispersion caused by low ventilation rates and the greater percentage of time spent indoors. However, people living close to production facilities tend to have elevated concentrations in their blood, often as a result of increased air concentrations and, in some cases, contamination of the local drinking water (Holzer *et al.,* 2008, Wilhelm *et al.,* 2008). Urban regions tend to have higher concentrations than rural and suburban regions because of factors such as greater industrial use of PFCs and greater population density. Highest concentrations are seen in urban regions and are correlated positively with the population (Pistocchi & Loos, 2009). This is seen as a result of an increased volume of products containing PFCs in a concentrated area compared to rural regions. Also, with increased volume of products in the area, water concentrations are impacted as a result of PFCs being washed out of garments and products (73 % washed off of garments within 2 years, 3M Speciality Materials, 2000).

Newer buildings are more likely to contain higher concentrations of PFCs because of the incorporation of many new products which (potentially) contain PFCs, along with touch-resistant paints, waxes and varnishes. For example, it has been shown that 50 % of sulfonyl-based PFCs are lost from carpets over a 9-year period (3M Speciality Materials, 2000) due to wear via walking and vacuuming. Therefore, older buildings (and those less recently refurbished or redecorated) are likely to contain lower concentrations due to such loss processes over time. Also, the incidence of PFC incorporation into products has increased over the last two decades, with the

introduction of novel applications of PFCs such as incorporation into touch resistant paints, non-stick kitchen utensils and clothing.

Children and the occupationally exposed are likely to be the most exposed groups in the population, due to a variety of factors. Children tend to receive greater doses of dust on a daily basis, as a result of behavioural traits such as hand-to-mouth behaviour and also the increased proportion of their time spent in close proximity to the floor (Shalat *et al.* 2007). Children's body burdens are also influenced by their lower body weights, with neonates also potentially exposed via breast milk.

Table 11 Concentrations in human blood (ng mL^{-1})

Reference	Blood Type, samples	PFOS	PFOA	PFHxS	FOSA
Kärrman *et al.*, 2006	Serum, *n*= 3802	21	7.6	6.7	
Kubwabo *et al.*, 2004	Serum, *n= 56*	29	3.4		
Olsen *et al.*, 1999	Serum, *n*= 178	2.2			
Yeung *et al*., 2006	Serum, *n*= 85	53	1.6	1.9	1.8
Calafat *et al.*, 2006	Serum, *n= 54*	21	3.7		
Olsen *et al.*, 2006	Serum, *n= 645*	35	4.6	1.9	
Guruge *et al.*, 2005	Serum, *n*= 10 (urban)	7.8	9.54	0.78	
	Serum, *n*= 10 (rural)	0.96	0.53	0.082	
	Serum, *n*= 10 (rural)	6.3	9.1	0.83	
Olsen *et al.*, 2007	Serum, *n* = 100 (collected in 2000)	33	4.5		
Olsen *et al.*, 2007	Plasma, *n* = 40 (collected in 2005)	15	2.2		
Yeung *et al.*, 2006	Serum	43	19	4	4.1
	Serum	8.3	6.2	0.2	1.6
	Serum	12	<20	3.8	1.0
	Serum	4.3	<3	1.6	1.8
	Serum	42	21	1.3	2.1
	Serum	16	4.8	1.0	<3
	Serum	1.9	2.6	1.6	<3
	Serum	5.0	6.4	0.57	
	Serum	13	<10	2.0	4.6
	Serum	21	62	4.0	1.3
	Serum	25	6.4	5.9	7.9
	Serum	53	1.9	1.6	1.8
Emmett *et al*., 2006	Serum, *n* = 312		420		
	Serum, *n* = 48 (potential occupational exposure)		410		

Reference	Blood Type, samples	PFOS	PFOA	PFHxS	FOSA
	Serum, *n* = 18 (substantial occupational exposure)		820		
Ehresmen *et al.* 2006	Serum, *n* = 16 (occupational exposure)	130	1000	26	
Olsen *et al.*, 2004	Serum, *n* = 238	31	4.2	2.2	
Masunaga *et al.*, 2002	Serum, *n* = 26	16			
Hansen *et al.*, 2001	Serum, *n* = 65	28			
Toms *et al.*, 2009	Serum, *n* = 2420	15	6.4	3.1	<LOD
Von Ehrenstein *et al.*, 2009	Serum, *n* = 34	22	3.9	1.9	0.07
Rylander *et al.*, 2009	Plasma, *n*= 91	3.7	5.6	4	
Midasch *et al.*, 2006	Plasma, *n* = 105	22	6.8		
Children					
Spliethoff *et al.*, 2008	Serum, *n* = 10	2.3	1.4	2.5	1.1
Apelberg *et al.*, 2007	Cord Sera; *n = 299*	4.9	1.6		

Table 12 Concentrations in human milk (ng mL^{-1})

Reference	Country	PFOS	PFOA	PFHxS	FOSA
Tao *et al.*, 2008b	Japan, *n* = 24	200	67	6.5	
	Malaysia, *n* = 13	110		6.7	
	Philippines, *n* = 24	100		13	
	Indonesia, *n* = 20	67			
	Vietnam, *n* = 40	59		4.3	
	Cambodia, *n* = 24	40			
	India, *n* = 39	39			
So *et al.*, 2006	China, *n* = 19	120	110	21	
Volkel *et al.*, 2008	Germany, *n* = 57	120	77		
	Hungary, *n* = 13	370			
Tao *et al.*, 2008a	USA, *n* = 45	130	44	15	
Karrman *et al.*, 2007	Sweden, *n* = 12	0.2	<LOD	0.085	0.013

1.12. TOXICITY

Initially, the toxicity of PFOS and PFOA was expected to be minimal because of the strength of the C-F bond (Sargent and Seffl, 1970). This presence of organic fluorine was first identified in human blood serum ca 30 years after PFC manufacture began (Sargent and Seffl, 1970), but it took a further decade before the organic fluorine was identified as PFOS and PFOA. Subsequent studies indicate that both PFOS and PFOA can induce toxic effects because of the similarity in structure to various hormones, leading to the potential for endocrine disruption (Peden-Adams *et al.*, 2007, Jensen & Leffers, 2008).

PFCs have been identified to have a no observed effect concentration (NOEC) in the order of mg L^{-1} for aquatic organisms (Hanson *et al.*, 2005), which is well above any of the recorded aquatic concentrations, including those from specific contamination sites (Table 13). Despite this, PFOS has been shown to bioaccumulate in fish (Houde *et al.*, 2008) and continue to biomagnify throughout the food web (Bossi *et al.*, 2005). This suggests that top predators (e.g. polar bears and humans) in the food web are likely to incur greater toxicity and exposure doses.

Table 13 NOEC values derived by Boudreau *et al.* (2003) for aquatic species.

Species Name	Endpoint	Lethal Concentration (LC50) (mg L^{-1})	NOEC Value (mg L^{-1})
S. capricornutum	cell density	48.2	5.3
	chlorophyll	59.2	16.6
C. vulgaris	cell density	81.6	8.2
	chlorophyll	88.1	9.6
L. gibba	frond number	59.1	29.2
	wet weight	31.1	6.6
D. magna	48-h survival	130	33.1
	48-h immobility	67.2	0.8
	21-day adult survival	42.9	5.3
D. pulicaria	48-h survival	169	46.9
	48-h immobility	134	13.6

The persistent nature of PFOS and PFOA also makes them resistant to metabolism and this combined with their hydrophobicity results in their transport around the body and accumulation in tissues/organs with high concentrations of proteins. Hence, PFOS and PFOA accumulate in major body organs and produce hepatotoxicity, peroxisome proliferation, and endocrine disruption (Jensen & Leffers, 2008). PFOA has been shown to induce carcinogenic effects in breast cancer bioassays (Rosen *et al.,* 2008), while studies of occupationally exposed populations have indicated the possibility of a small increase in occurrence of intestinal, stomach, testicular and bladder cancers (Olsen *et al.,* 2003, Alexander & Olsen, 2007) as a result of chronic exposure to PFOS. An overview of the toxic effects that may be elicited by various PFCs in primarily mammalian species is given in Table 14.

Table 14 Reported toxic effects of PFCs

Animal Toxicity	Animals	Compounds	Reference
Behavioural effects	Mice	PFOS	Fuentes *et al.,* 2007, Johansson *et al.,* 2009, Fei *et al.,* 2007
Peroxisomal proliferation	Mice, mouse and human cells	PFOA, PFOS, PFOSA	Kennedy *et al.,* 2004, Takacs & Abbott, 2006, Berthiaume & Wallace, 2001, Shipley *et al.,* 2004, Seacat *et al*., 2002
Weight loss	Rats, mice	PFOS, PFOA	Cui *et al.,* 2009, Seacat *et al*., 2003, Fuentes *et al*., 2007
Embryotoxicity	Chickens, Zebrafish larvae	PFOS, FTOH	O'Brien *et al.,* 2009, Shi *et al.,* 2009, Seacat *et al.,* 2002, Liu *et al.,* 2010
Induced change in cytochrome P450s	Rats, Chickens	PFOS, PFOA	Hu *et al*., 2004, Yeung *et al.,* 2007
Immunotoxicity	Mice	PFOA	Peden-Adams *et al.,* 2008, DeWitt *et al.,* 2008
Carcingenicity	Mcf-7 breast cancer cell	PFOA	Maras *et al.,* 2006, Rosen *et al.,* 2008
Mitochondrial dysfunction	Rat, dolphin	PFDS	Starkov & Wallace, 2002, Hu *et al*., 2002, Kleszczyriski *et al.,* 2009
Neurotoxicity	Cellular and gene level, mice	PFOS, PFOA, PFOSA, PFBS	Slotkin *et al*., 2008, Johansson *et al.,* 2007

Indications are that the foetus and infants are particularly susceptible to the adverse impacts of PFC exposure (Yanai *et al.,* 2008, Fei *et al.,* 2008). Tests on rat and mouse foetuses and neonates indicate a range of effects including developmental neurotoxicity (Butenhoff *et al.,* 2009) and reduced birth weights (Washino *et al.,* 2009).

1.13. SAMPLING

1.13.1. Surveying Dust

Dust is considered to consist of solid particles approximately <500 µm diameter (Lewis *et al.,* 1999) and is produced from wear and tear of products, the degradation of materials and the deposition of airborne components, such as pollens, soil minerals and other particles, along with volatile organic compounds (VOCs), (Nilsson *et al.,* 2005). The content of dust is highly variable from place to place and dependent upon season and various environmental variables, including natural and anthropogenic surroundings (Layton & Beamer, 2009). The variation in air flow, age of building and its contents, cleaning rate, heating and humidity, and many other variables can have a large impact on the dust loading in a room and also the content of organic material (soil and dirt) in the dust. Outdoor aerosols and "tracked-in" soil can also contribute to indoor dust concentrations.

Dust can cause allergies in people, and may represent a pathway of exposure to chemicals such as heavy metals, pesticides, and various man-made chemicals, that is of particular relevance to infants (Roberts *et al.,* 2009).

The method of settled dust collection is dependent on the microenvironment the sample is collected from, and the type of dust being collected. Samplers can be fitted to people, in order to measure the volume of suspended particulates that are likely to

be inhaled, along with simple vacuuming techniques used to collect deposited dust on the floor and horizontal platforms in rooms (shelves, windowsills etc). Also, aggregated dust in window films can be used to determine concentrations of chemicals from indoor and outdoor environments (Diamond *et al.,* 2000, Gewurtz *et al.,* 2009). Some sampling techniques work on the premise of deposition rates and will collect dust in Petri-dishes over a set number of days (Hogervorst *et al.,* 2007). For settled dust, the main sampling techniques involve insertion of a collection vessel inside the vacuum cleaner (Björklund *et al.,* 2009, Kato *et al.,* 2009), or using the whole bag sample from vacuum cleaner (Moriwaki *et al.,* 2003, Kubwabo *et al.,* 2005). However, for brominated flame retardants whole vacuum cleaner bag samples have been shown to provide lower concentrations compared to researcher collected dust, as there can be a large difference between concentrations in different rooms (Allen *et al.,* 2008). Wipe sampling is also conducted for the collection of samples from windows, and also dust films on objects and shelving units (Diamond *et al.,* 2000, Thorne *et al.,* 2005, Kuusisto *et al.,* 2007).

Chemical pathways into the body can occur through multiple routes, including via contact with dust. The ingestion and inhalation of dust occurs from all environments, indoors and outdoors, due to suspension of dust and the ingestion via hand-to-mouth and object-to-mouth transfer. The major pathway for dust to enter the body is through ingestion, and the ratio of inhalation:ingestion intake is much lower for children, as they spend a greater amount of time on or at close proximity to the floor, and have behavioural traits of sucking and mouthing various objects.

House dust has been shown to consist of chemicals derived from outdoor samples, such as lead from vehicle emissions, pesticides, and particulate material (often associated with traffic and uncontrolled burning of coal and wood) (Fergusson *et al.,*

1986, Menichini *et al.* 2007). Indoor sources to dust are numerous, including those associated with building materials (PCBs and HBCDs), furnishings (PFCs) and cooking utensils and packaging (PAHs and PFCs). Indoor environments are also affected by the personal care products used by humans (e.g. deodorants, shampoos and hair products) (Potera 2009, Washburn *et al.* 2005).

Concentrations of many contaminants within indoor microenvironments tend to be higher than outdoors (Shoeib *et al.*, 2005, Shoeib *et al.*, 2004). This is driven by the low ventilation rates within indoor environments, particularly relevant to modern "energy-efficient" buildings, and also the number of potential sources that are within the indoor environment. "Hotspots" of HBCD contaminated dust have been detected within single indoor microenvironments (Harrad *et al.,* 2009), caused by migration of the chemicals of interest from particular products to the surrounding dust. These highly contaminated objects create a distinct dust profile in the room, where the dust concentrations decrease with distance from the object. This allows contributing sources to be identified and avoided during point-sampling in the room.

Currently, there is little known about the distribution of PFCs within rooms, and low mixing rates of dust in rooms may result in concentration gradients within rooms. Both intra- and inter-room variability in PFC concentrations in dust must be considered when sampling though, in an exposure assessment context, the most relevant sampling method is that which yields 'biologically-relevant' dust (Allen *et al.*, 2008, Harrad *et al.,* 2009), i.e. dust that has a high probability of being ingested or inhaled.

1.13.2. Air Monitoring

Passive and active air sampling has been used for some time to monitor POPs in the troposphere and stratosphere and indoor air. The principal idea of active samplers is to

obtain a sample via the aid of a pump to collect large volumes at any one time. These types of samplers allow volatile compounds to be collected under short sampling times (days, as opposed to weeks when passive sampling), and high volume samples (which is advantageous when concentrations are extremely low). Some types of active samplers such as an aerosol time of flight mass spectrometer (ATOFMS) can produce close to real time concentration results. This type of sampling is advantageous for measuring pollution events caused by traffic impulses, or crop spraying incidences, where the concentrations are likely to change by the hour. However, disadvantages associated with active sampling for indoor environments include the cost, noise pollution, obtrusiveness and the fact that high sampling rates can quickly exceed the volume of a room (resulting in underestimates of concentrations) (Harrad, 2009, Levy *et al.*, 2007).

The use of passive samplers as an alternative means for measurement of air concentrations is often favoured for the advantages they have over active sampling in both remote and less accessible regions. The favourable properties of passive samplers includes ease of installation, uptake rates allowing them to be left for a while prior to collection and ability to measure chronic exposure (Harrad, 2010b). These samplers are also useful within indoor environments, because they are less intrusive than active samplers (noise and comparative size). They are, however, strongly affected by air movement (especially in outdoor air), and, therefore, sampling rates are not as accurate (Harrad, 2010b).

Passive samplers work on the principle of retention of the chemical on a sorbent media such as polyurethane foam, triolein, XAD resin, or can consist of natural chemical sorbents like soil and sediment, vegetation, dust (Harrad *et al.*, 2006, Ockenden *et al.*, 1998, Choi *et al.*, 2008, Zhu & Hites, 2006, Klánová *et al.*, 2009).

The samplers are usually deployed from anywhere between a couple of weeks to several months, in order to collect measurable masses of environmental contaminants, but collected prior to reaching equilibrium, and thus they can provide a time-weighted average concentration. Passive samplers are used for collecting PFCs as well as many other organic pollutants, including brominated flame retardants (BfRs), OCPs (organochlorine pesticides), PAHs (polyaromatic hydrocarbons), and PCBs (polychlorinated biphenyls) (Abdallah & Harrad, 2010, Yusà *et al.*, 2009, Bohlin *et al.*, 2010, Hazrati & Harrad, 2007).

The majority of techniques used for the collection of PFCs include the use of XAD-4, polyurethane foam (PUF) disks and glass fibre filters (GFF), which are used in various measuring devices. The use of these sampling matrices have been noted in both passive and active sampling techniques as XAD is effective at absorbing the volatile precursor compounds, whilst the GFF can retain particulate compounds. The PUF disks are used to retain the less-volatile compounds and can be used as a support for the XAD (Shoeib *et al.*, 2008). These different sampling configurations all work under the same principle of diffuse uptake of the analytes via a porous absorbent material. The differences in the passive sampling techniques for PFCs vary in order to provide the best linear uptake to suit the sampling campaign. The underlying sampling principle is to retain vapour phase analytes on a porous media, with a linear uptake rate. This uptake rate can be a function of the transfer rate across the sampling media and air interface and is influenced by wind speed, polarity of the compounds, ambient temperature and the uniformity of the sampling medium and rate of loss (Shoeib & Harner, 2002).

The air volume (V_{AIR}) sampled is a function of the following, (Shoeib *et al.*, 2008):

Equation 1 Volume of sampled air

$$V_{AIR} = K_{SIP-A} V_{SIP} (1 - \exp\left[- \frac{A_{SIP}}{(V_{SIP})\left(\frac{k_A}{K_{SIP-A}}\right)} \right] \times t)$$

Where $K_{SIP\text{-}A}$ is the equivalent volume of air, with the same mass of analyte as 1 unit volume of the passive sampler under equilibrium conditions, the passive sampler – air partition coefficient, V_{SIP} is the volume of the passive sampler, k_A is the air-side mass transfer coefficient, A_{SIP} is the surface area of the passive sampler and t represents the extent of deployment of the sampler. Provided the sampler is deployed during the linear uptake period only, the concentration of the sampler should reflect the concentration of analytes present over the sampling period (Shoeib *et al.,* 2008).

Limitations of passive air samplers are derived from their relatively slow sampling rates, compared to active air samplers, therefore passive sampling techniques are not adequate to measure short term exposure variations, such as traffic-related diurnal variations in air quality resulting. Moreover, if deployed beyond the linear uptake period, equilbrium is reached with consequent underestimation of concentrations (Shoeib & Harner, 2002).

The vapour:particle partitioning of PFCs is a function of chain length and volatility. The presence of PFCAs and PFASs in remote regions is explained by the movement of more volatile compounds (FTOHs, PFOSAs etc) followed by scavenging by wet and dry deposition and degradation (Hurley *et al.,* 2004). Compounds which exist in both vapour and particulate phases depending on environmental conditions and temperature are the semi-volatile compounds FOSA, MeFOSA, EtFOSA, MeFOSE and EtFOSE, (Shoeib *et al.*, 2004, Shoeib *et al.* 2006) whilst FTOHs and FOSA are

not detected in the particulate phase(Dreyer & Ebinghaus, 2009, Stock *et al.*, 2007). Measured octanol – air partition coefficients (K_{OA}) by Shoeib *et al.*, (2004) ranged between 7.7 and 7.9 for three sulfonamides and were noted to be log-linearly related to absolute temperature, which suggests that, as the ambient temperature drops, PFCs tend to partition into the organic matter of particulates (Shoeib *et al.*, 2004).

1.14. EXTRACTION AND ANALYTICAL TECHNIQUES

The sample matrix can determine the type of extraction and analytical techniques employed, as there are many substances which can cause interferences during analysis. The impact of sample contamination is prevalent as there are many instrumental parts, which are made from polytetrafluoroethylene (PTFE), including tubing, seals and additional parts. PFCs were also noted to be capable of being retained on glassware (Matin *et al.*, 2004) leading to potential loses of the perfluorinated compounds during extraction and clean-up stages. Therefore, methods often include the use of polypropylene containers, and direct online extraction techniques, to reduce losses (Kuklenyik *et al.*, 2005). Polypropylene and high-density polyethylene (HDPE) have also been noted to retain PFCs from stored water samples (Taniyasu *et al.*, 2005). Optimised sample storage is also essential in order to reduce the loss of PFCs, and samples should be kept at -20°C until use to prevent degradation of the precursor compounds (van Leeuwen, 2009), with minimum head room in containers and aluminium foil lined lids to prevent degradation of FTOH to PFCAs (Szostek *et al.*, 2006).

Methods for clean-up and extraction have advanced significantly over the last decade, minimising and even eliminating matrix effects and interferences, aided by the improvement and development of high-quality mass labelled standards. Clean-up and

extraction of PFCs is dependent on the type of sample and the PFC requiring quantification. Extraction techniques include solid-phase extraction (SPE), soxhlet extraction, digestion with KOH, ion pair extraction (IPE), pressurised liquid extraction (PLE), liquid solid extraction (LSE) and liquid-liquid extraction (LLE). The extraction type can be dependent on the sample type being analysed and the need to remove contaminants such as lipids from biological samples and sulfates from soils, as these can cause matrix effects during analysis.

The two main extraction techniques that have been utilised are IPE and SPE. Biota and biological samples are often treated by IPE, which consists of ion-pairing (Orata *et al.*, 2009, Taniyasu *et al.*, 2005, Washington *et al.*, 2008) with tetra-n-butylammonium hydrogensulfate (TBA), followed by LSE with methyl-tert-butylether (MTBE), which was first devised by Hansen *et al.*, 2005. This method can be fairly laborious and therefore is less popular as it also not easily automated.

Solid-phase extraction is also widely used (Zhao *et al.*, 2007, Karrman *et al.*, 2007, Holzer *et al.*, 2008) for a number of different matrices (water, air, sediment, food), as it allows selective retention of PFCs, despite the differences which can occur between sample matrices (Kuklenyik *et al.*, 2004). Common SPE cartridges are Oasis HLB and WAX cartridges, Sep-Pak, Polaris C18 and other silica cartridges (Lindstrom *et al.*, 2009), with the exact cartridge selected dependent upon the polarity of the PFCs being eluted (van Leeuwen & de Boer, 2007). SPE retains the PFCs, while large molecules are not retained because of the relatively large particle size or are degraded with the addition of the formic acid, and constant washing of the column disposes of many interferences (Kuklenyik *et al.*, 2004). This method is particularly popular with fluid samples, as it can be easily automated, and the turnover is relatively fast.

Analytical techniques include LC-MS, GC-MS, ^{19}F NMR, LC-MS/MS, but often the chosen technique depends upon the PFCs targeted as the different polarities lend themselves to different techniques (Moody *et al.,* 2001). The detection of organic fluorine in human blood serum was noted in the 1970s, but determination of the actual contributing compounds was not possible. During the 1990s, technological improvements led to the detection and quantification of PFCs via GC-MS and HPLC-fluorescence techniques and further development has led to perfluorinated compounds being analysed by LC-ESI-MS/MS for the less volatile compounds and GC-MS for fluorotelomer alcohols. Such techniques have facilitated the determination of specific PFCs and in particular the ability to distinguish them from other organic fluorine products and the identification of isomers (Langlois & Oehme, 2005).

The improvement of analytical techniques over the last decade has allowed for the detection of many PFCs down to parts per trillion levels. This improvement in techniques has allowed for LC-MS/MS to flourish, with HPLC – MS/MS using negative ion electrospray with triple quadrupoles or ion trap to become the most widely used instruments for the detection of perfluoroalkyl compounds (Martin *et al.*, 2004). Columns used on the HPLC-MS/MS tend to be reverse phased C18 silica columns (Lindstrom *et al.,* 2009). However, the matrix suppression and enhancement artefacts associated with electrospray ionisation (ESI) are not trivial. Improved availability of mass labelled standards has aided in the elimination of these problems and has made ESI a reliable technique. Detection of more volatile PFCs like the fluorotelomers is becoming increasingly widespread as manufacturing and regulations push industry towards less persistent compounds and GC-MS techniques have been utilised for this analysis. The GC-MS techniques utilise either positive or negative chemical ionisation (PCI or NCI) (Szostek & Prickett, 2004), as they are more

suitable for such volatile compounds. However, fragmentation patterns are produced by molecular breakdown, and are most convoluted for NCI GC-MS (Ellis & Mabury, 2003), thus the samples require a suitable degree of extraction and clean-up to eradicate interferences.

1.15. CONCLUDING REMARKS

The ubiquitous presence of PFCs present in the environment, combined with their toxicity, suggests that a greater understanding is required of the potential risks they represent to human health. The complex environmental processing of PFCs is well illustrated by the fact that, in addition to direct releases of PFOS and PFOA, these two compounds can also become widespread via the transportation of more mobile precursor compounds that can undergo degradation to PFOS or PFOA as stable end-products. The detection of PFCs is possible in many environmental matrices including biota on a global scale; leaving very few regions untouched by their presence. Exposure from non-dietary sources received from various microenvironments can act as a significant pathway of exposure to PFCs (Shoeib *et al.*, 2004). Therefore, better characterisation of non-dietary exposure of adults and children will allow a greater understanding of any current risks.

1.16. AIMS

The study examines the potential implications of PFCs in urban environments as exposure to humans occurs via various media and differs with each microenvironment. It also characterises non-dietary human exposure, in particular via indoor pathways, due to the large proportion of the day spent indoors. Indoor

environments examined in this study include: homes, offices, cars and classrooms, where both air and dust have been monitored.

The overarching hypothesis of this thesis is that:

> ‘Perfluoroalkyl compounds can migrate from consumer goods and materials within which they are incorporated. This migration results in contamination of indoor air and dust, as well as the outdoor environment thereby resulting in human exposure’.

To test this overarching hypothesis, I will investigate whether perfluoroalkyl compounds are present in air and dust from a variety of indoor microenvironments, as well as outdoor air and soil. Combining these data with appropriate exposure factors, I will evaluate whether the levels at which these compounds are present are sufficient to constitute a plausible pathway of exposure to humans.

Within the framework of this overarching hypothesis, further more specific hypotheses are proposed:

1. Indoor environments are substantial reservoirs of perfluoroalkyl compounds
2. Distributions of perfluoroalkyl compounds within a room are variable.
3. There is substantial spatial and temporal variation in the distribution of PFCs in the outdoor urban environment.
4. UK soils are a substantial reservoir of PFCs

5. Classroom environments are contaminated with a signature of PFC contamination that is distinct from that observed in other indoor microenvironments.
6. The presence of PFCs in the indoor and outdoor environment results in both external and internal human exposure.
7. Due to global variations in use patterns, there is substantial international variation in the contamination of indoor dust with PFCs.

In light of the above, the aims of this project are:

I. To characterise the presence of PFCs in UK indoor air and dust samples.
II. To characterise spatial and temporal variability in concentrations of PFCs in indoor air and dust, and in soil and outdoor air. These data can provide insights into human exposure and source attribution.
III. To conduct a preliminary evaluation of the concentrations of PFCs in UK soils. Such data will provide an indication of the extent to which soil is an environmental sink for PFCs.
IV. To quantify the presence of PFCs in dust from primary school and nursery school classrooms. These data will provide an indication of the contribution of such environments to the exposure of young children to PFCs.
V. To evaluate the relative contribution of different exposure pathways to PFC concentrations in humans..
VI. To conduct a preliminary evaluation of whether concentrations of PFCs in indoor dust vary significantly between different countries.

2. METHODOLOGY

2.1. SYNOPSIS

In order to test the hypothesis and aims detailed in Chapter 1, indoor samples of air and dust were collected from the West Midlands area, along with outdoor air samples, and UK soil samples. The presence of PFCs in these matrices, constitute routes of non-dietary exposure to humans. Collection of samples was conducted according to the protocols detailed in this chapter. For both air and dust collection and extraction, the methods involved were specific for PFCs and were chosen according to ease of use and deployment, cost effectiveness and PFC sampling capability. All methodologies and analytical techniques used were developed from previous studies, and modified to best suit the analysis of dust and air for the range of PFCs in this study. The basic methodology for extraction of PFCs from dust matrices was developed via adaptation of existing methods (Taniyasu, 2005; Young & Tran, 2006). Acetone was used as the extraction solvent because PFOS was found to be more soluble in polar organic solvents (Takagi & Igarashi, 2002); while the air sampling methodology was based on the sampling techniques reported by Shoeib *et al.*, (2008) which provided effective capture of both vapour and particulate-phase semi-volatile PFCs. Sampling focused on the urban environment because of the higher concentrations of many contaminants noted in more densely populated areas (Murakami *et al.*, 2008, Herzke *et al.*, 2009), and also because urban locations provide a wide variety of easily accessible microenvironments (particularly indoor) to study.

Along with dust, air, and soil samples, paint and carpet samples were analysed, to examine their potential as sources of PFCs to indoor environments. A basic

description of the extraction techniques employed for each of these sample matrices is given in Table 15.

Table 15 Overview of sampling and extraction techniques for different sample types

Sample Type	Collection Method	Extraction Solvent and Technique
Dust	Nylon sock	Acetone Sonication
Air	Passive PUF disk, LoVol Sampling, & HiVol Sampling	Hexane:Acetone (60:40) Soxhlet
Soil	Top soil (to 5 cm depth) (Evans 2008)	Acetone Sonication
Paint	Household Brands purchased from consumer outlets	Acetone Sonication
Carpet	New and used material purchased from consumer outlets or donated by householders	Hexane:Acetone Soxhlet

The PFCs monitored are listed in Table 16 and include PFOS, PFOA, PFHxS, perfluorooctane sulfonamides (FOSA, MeFOSA and EtFOSA) and perfluorooctane sulfonamidoethanols (MeFOSE and EtFOSE). PFOS and PFOA were chosen because of their persistence within the environment, and because they are additionally the stable end-products resulting from degradation and metabolism of more volatile PFCs (FTOH, perfluorooctane sulfonamides and perfluorooctane sulfonamidoethanols, Wallington *et al.*, 2006, Plumlee *et al.*, 2009, Vestergren *et al.*, 2007, Tomy *et al.*, 2004). PFHxS was also chosen for analysis, because of its long half-life in humans (ca. 8 years (Spleithoff *et al.*, 2008)) and because it is a stable degradation end-product of more volatile PFCs, (similar to those for PFOS). It was also used as an intermediate for AFFF and post-market carpet treatments (Olsen *et al.*, 2003). The FOSAs and FOSEs were chosen for their presence in both air and dust (Shoeib *et al.*,

2004, Piekarz *et al.*, 2007), their presence as intermediate products of fluorotelomer degradation and use as manufacturing intermediates (van Zelm *et al.*, 2008, Rhoads *et al.*, 2008), and also their potential to act as precursors of PFOS and PFOA. The semi-volatile properties of PFOSAs and PFOSEs allow them to partition between the particulate and gaseous phase (Shoeib *et al.*, 2004), thus making them present in both air and dust samples at measurable concentrations.

2.2. CHEMICALS

All perfluorinated analytes used in this study were supplied by Wellington Laboratories Inc. and are detailed in Table 16. Solvents used were all of HPLC analytical grade and provided by Fisher Scientific UK Ltd, along with reagents and chemicals including, anti-bumping granules and HPLC ammonia solution. Nitrogen used for solvent reduction was oxygen free, supplied by BOC Gases and the solid phase extraction (SPE) cartridges were supplied by Waters Corp.

Table 16 Perfluorinated analytes of native standards and mass labeled internal standards

Compound	Abbreviation	Molecular Formula	Molecular Weight
Native Standards			
Sodium, perfluoro-1-octanesulfonate (L-PFOS	PFOS	$C_8F_{17}SO_3^-Na$	522.11
Perfluoro-n-octanoic acid	PFOA	$^{12}C_8HF_{15}O_2$	414.07
Sodium perfluoro-1-hexanesulfonate	PFHxS	$C_6F_{13}SO_3Na$	422.10
Perfluoro-1-octanesulfonamide	FOSA	$C_8H_2F_{17}NO_2S$	499.15
N-methylperfluoro-1-octanesulfonamide	MeFOSA	$C_9H_4F_{17}NO_2S$	513.17
N-ethyl-perfluorooctane sulfonamide	EtFOSA	$C_{10}H_6F_{17}NO_2S$	527.20
2-(N-methylperfluoro-1-octnaesulfonamido)-ethanol	MeFOSE	$C_{11}H_8F_{17}NO_3S$	557.23
2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol	EtFOSE	$C_{12}H_{10}F_{17}NO_3S$	571.25

Internal Standards			
Sodium perfluoro-1-[1,2,3,4-$^{13}C_4$]octanesulfonate	MPFOS	$^{13}C_4{}^{12}C_4F_{17}SO_3^-$ Na^+	526.08
Perfluoro-n-[1,2,3,4-$^{13}C_4$]octanoic acid	MPFOA	$^{13}C_4{}^{12}C_4HF_{15}O_2$	418.04
Sodium perfluoro-1-hexane[$^{18}O_2$]sulfonate	MPFHxS	$C_6F_{13}S^{18}O_2{}^{16}ONa$	426.10
N-methyl-d_2-perfluoro-1-octanesulfonamide	d-MeFOSA	$C_9D_3HF_{17}NO_2S$	516.19
2-(N-deuteriomethylperfluoro-1-octanesulfonamido)-1,1,2,2-tetradeuterioethanol	d_7-MeFOSE	$C_{11}D_7HF_{17}NO_3S$	564.27

2.3.SAMPLE COLLECTION

Sample collection was required to meet a number of conditions, in order to make the sampling viable. These conditions included cost, collection period and efficiency, noise regulations, collection in remote and urban areas, and minimal electricity requirement.

2.3.1. Air

Air samples were collected using passive air samplers, based on the retention of chemicals onto polyurethane foam (PUF) disks. Passive air samplers were used because of the ease of deployment, and relatively low price of the samplers. The samplers do not require a power supply, make no noise and can be left unattended for 4 - 6 weeks. This allows their deployment year-round in various environments, both indoors and outdoors, within urban or remote regions. Limitations of the sampling devices are the sampling rates of the PUF disks and the retention of volatile compounds. The length of time for deployment is a function of the atmospheric concentrations of the sampled chemicals, and the rate of uptake of the chemicals onto the disk.

Perfluorinated compounds have variable concentrations in the atmosphere, with indoor concentrations of MeFOSE and EtFOSE reaching levels an order of magnitude higher than outdoors (Shoeib *et al.*, 2004). There is also a divide between the partitioning of PFCs into the gaseous and particulate phases (Shoeib *et al.*, 2005). While semi-volatile compounds – perfluorooctane sulfonamides and sulfonamido ethanols – are present mainly in the vapour phase; others like PFOS, PFOA and PFHxS, are less volatile and present primarily in the particulate phase (Olsen *et al.*, 2005). The gas:particle partitioning behaviour of the PFCs monitored influences the choice of air sampling method used, and indeed whether air or floor dust is the most effective means of monitoring indoor contamination.

Three types of air samplers were used throughout the sampling campaign, passive foam samplers, LoVol samplers and HiVol Samplers (Table 17). All air samplers used sorbent impregnated PUF (SIP) disks (Shoeib *et al.*, 2008) for the collection and retention of PFCs. The PUF disks used initially in the passive air samplers were unable to retain the more volatile perfluorinated compounds. The addition of XAD-4 slurry (hexane + XAD-4 powder) to the PUF disks increased the retention of the sulofonamides and made it possible to retain fluorotelomer alcohols (Shoeib *et al.*, 2008).

The PUF disks (140 mm x 12 mm cylindrical disks with an average surface area of 361 cm^2 and 0.017 $g.cm^{-3}$ density, P.A.C.S UK Ltd) were washed in deionised, distilled water, dried and then precleaned for 12 hours via soxhlet extraction in dichloromethane (DCM), dried in a dessicator, and then dipped into the XAD-4 slurry according to the methodology devised by Shoeib *et al.*, 2008. The SIP disks were dried again in the dessicator, individually wrapped in solvent rinsed aluminium foil, sealed and stored in a cold room (4°C) until use. Glass filters were soxhlet extracted

in DCM for 12 hours, dried in clean aluminium foil overnight, sealed in aluminium foil and stored in the cold room prior to use. All SIP disks and glass filters were handled with talc free gloves and solvent cleaned tweezers.

Air samples were collected from indoor and outdoor microenvironments using passive samplers. The passive samplers were calibrated with the use of the active samplers set up in the same environments and run simulatenously with the passive samplers (results and individual compound sampling rates are available in Table 18).

The passive air samplers were prepared within the laboratory by washing all housing equipment with detergent and water, followed by DCM and methanol. The SIP disks were then placed into the sampler housing, 1 cm above the lip of the sampler housing, and the whole sampler was sealed within a polypropylene (PP) bag. Samples were mounted outdoors at a height of 1 m above ground level, and in homes at heights ranging between 1.0 and 1.5 m from the floor and located as centrally within the sampled microenvironment as possible.

Blank samples were produced and handled in the same way, taken to the sampling location, removed from packaging, placed in the samplers, removed and sealed in a new PP bag, and returned to the laboratory for storage and extraction.

Active air sampling allowed the collection of both the particulate phase (on glass fibre filters (GFF)) and gasous phases (on SIP disks) to be collected. GFF were precleaned via soxhlet in acetone for 8 h and dried in a dessicator. The LoVol used 37 mm Whatman filters, whilst the HiVol employed 20.3 cm x 25.4 cm Whatman filters. They were then wrapped in solvent rinsed aluminium foil, sealed and stored in the cold room until required.

The active air sampling equipment was set up on-site and the air intake placed at approximately a 1 m height, to match the passive air samplers. The active samplers

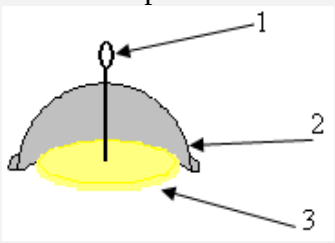
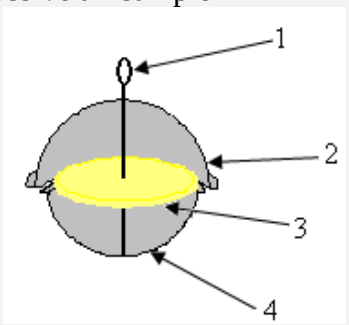
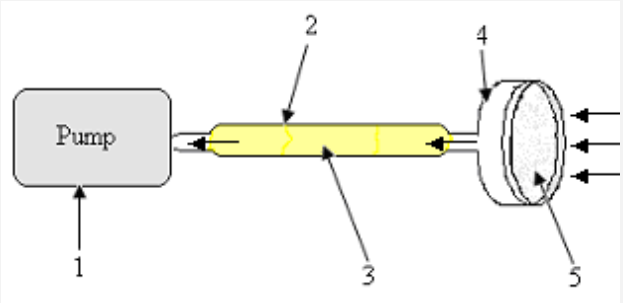
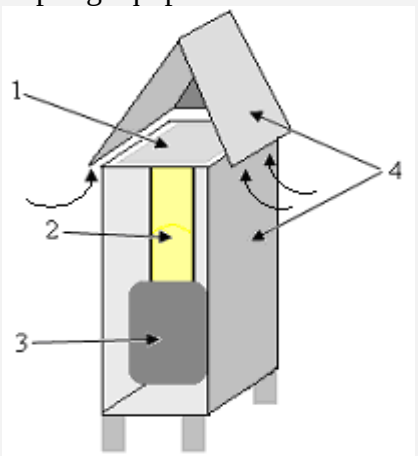
(the HiVol and LoVol) remained in the field throughout sampling campaigns and, between sampling periods (and SIP refreshment). The equipment was wiped over with DCM before each new sample was to be collected.

The LoVol samplers contained three SIP disks, which were placed within the glass housing (3 cm x 25 cm cylindrical tube) (Table 17), the front two SIP disks retaining the gaseous fraction of the PFCs and the third SIP disk, located at the back (furthest distance from air inlet) was analysed to check for any analyte breakthrough. A glass fibre filter was attached to the front of the glass tube housing, to collect the particulate fraction. This was housed in an open fronted 37 mm standard filter holder, made from polypropylene.

The HiVol sampler required a GFF to be placed at the top of the equipment for the collection of particulates, followed by two SIP plugs (10 x 8 cm cylindrical PUF disks, density ca. 0.02 g cm^{-3}) placed inside a metal tube housing, situated above the pump. The HiVol was used for the collection of 24 hour duration samples of outdoor air on a monthly basis. The HiVol sampler used was a Graseby-Andersen (maintained by Air Monitors, Gloucestershire, UK), fitted with a total suspended particulate (TSP) inlet modified to hold a standard glass fibre filter (GFF, 25 cm x 20 cm, 1 μm pore size, Whatman, UK) and two pre-cleaned and XAD coated polyurethane foam plugs (8 cm diameter x 10 cm length, 503 cm^3 volume, 0.017 g cm^{-3} density (prior to XAD)). The HiVol was run for 24 hrs at a sampling rate of 0.70 m^3 min^{-1}, yielding a total sample volume of 1008 m^3.

Field blanks were collected from the active samplers by setting up the whole equipment including new SIP disks and glass filters, followed by immediate removal of the sampling material. These samples were sealed separately, and returned to the laboratory for analysis.

Table 17 Air Sampling Equipment

Sampler Design	Equipment
Indoor passive air sampler 	1. Stainless steel bolt, with eye 2. Stainless steel housing, to prevent direct dust settlement directly onto SIP disk 3. SIP disk
Outdoor passive air sampler 	1. Stainless steel bolt, with eye 2. Stainless steel housing, for protection against the weather and direct deposition onto the SIP. 3. SIP disk 4. Stainless steel base housing, for protection from the weather and to minimise wind effects
LoVol sampling equipment 	1. Pump 2. Glass tube, covered with aluminium foil, to prevent light penetration through to SIP disks. 3. SIP disks (x3), two for collection and one breakthrough. 4. Filter holder (polypropylene) 5. Glass fibre filter.
HiVol sampling equipment 	1. Glass fibre filters. 2. SIP plugs (x2) 3. Pump 4. Shelter, to avoid rain reaching filters and pump.

Passive air samplers required longer periods of time for sampling because of the low sampling rates (SR) compared to the active air samplers. Passive samplers were left for approximately 40 days in order sample detectable quantities of PFCs without reaching equilibrium. The optimum sampling duration was derived from Shoeib *et al.*, 2008, and confirmed by the calibration study. The SR of the passive air samplers for the different PFCs monitored, ranged between 0.8 and 2.4 pg m^{-3} and 0.8 and 2 pg m^{-3} for indoor and outdoor designs. The difference in the sampling rates is because of the housing of the samplers and also the effect of the air flow rate through the sampler.

Calibration of the indoor and outdoor passive samplers was conducted alongside the use of a LoVol sampler over a period of 60 days, to assess the uptake of the SIP disks in both designs.

The LoVol sampler (depicted in Table 17) consisted of a single inlet pump (Charles-Austen, Capex L2X diaphragm pump), operated for 24 h at a flow rate of 0.004 m^3 min^{-1} to produce a single sample of c.a. 5.76 m^3 air. The particle phase was collected on a 47 mm glass filter membrane (1.0 μm pore size, Whatman, UK), which was housed in a standard open face filter holder, placed before the PUF SIPs. The PUF SIPs were used as gas phase sorbents, which consisted of three (14 cm diameter and 12 cm depth disks) inserted into a glass tube (3 cm x 25 cm cylindrical tubes). These SIPS were prepared according to the aforementioned methodology and had been treated with the XAD slurry. The system was controlled using a flow meter (Platon 50 L min^{-1}) connected to an adjustable valve. The flow meter was calibrated using a Gilibrator air flow calibrator (Gilian), directly before and after the 24 h period to ascertain that a constant flow rate was being achieved. The gaseous phase sorbents housed in the glass tube were covered in aluminium foil to prevent photodegradation. The calibration was conducted throughout September – December 2008, within a

temporarily vacant office, located at the University of Birmingham campus. The passive samplers were harvested every 10 days, over the 60 day period along with the LoVol SIP disks. However, the breakthrough SIP disk from the LoVol sampler was only collected every 20 days to assess breakthrough concentrations. Table 18 indicates the active sampler concentration (pg m^{-3}), the mass of compound collected per passive sample (pg), the breakthrough (%) and the sampling rate (SR, m^3 day^{-1}).

Table 18 Passive air sampler calibration

Compound	Active Sample (pg m^{-3})	Mass of compound collected per passive sampler (pg) 10 day	20 day	30 day	40 day	50 day	60 day	Breakthrough (pg)	S.R. (m^3 d^{-1})	R^2
Outdoor Housing										
PFOS	1.8	2	7	22	48	51	79	n.d.	0.9	0.85
PFOA	2.2	0	4	9	19	55	68	n.d.	1.05	0.71
PFHxS	1.7	6	17	20	27	56	89	n.d.	0.8	0.78
MeFOSA	9.8	0	19	130	530	870	1100	n.d.	2.0	0.76
EtFOSA	270	2900	7200	11 000	13 000	16 000	23 000	n.d.	1.3	0.96
FOSA	210	1600	3400	7400	9600	10 000	14 000	0.3	1.2	0.96
MeFOSE	410	4500	8600	15 000	17 000	28 000	34 000	0.4	1.5	0.95
EtFOSE	360	5600	14 000	20 000	27 000	30 000	36 000	0.2	1.8	0.98
Indoor Housing										
PFOS	1.6	2	5	17	32	30	62	n.d.	0.8	0.79
PFOA	2.1	0	5	16	18	28	52	n.d.	1	0.78
PFHxS	1.3	2	4	17	26	32	74	n.d.	0.9	0.71
MeFOSA	7.3	110	210	120	310	480	515	n.d.	2.4	0.81
EtFOSA	230	2600	7800	11 000	16 000	23000	25000	n.d.	1.8	0.97
FOSA	190	3500	7500	9100	12 000	15 000	20 000	n.d.	1.7	0.98
MeFOSE	400	7600	16 000	26 000	35 000	44 000	51 000	n.d.	2.1	0.99
EtFOSE	320	6500	12 000	21 000	26 000	33 000	39 000	n.d.	2.1	0.99

2.3.2. Dust

Dust samples were collected using a hand-held Black-&-Decker Dustbuster vacuum cleaner for samples in the UK and individual household vacuum cleaners for samples collected from international sampling locations. A copy of the dust collection protocol instruction sheet provided for the overseas and home owner sample collections is provided as Appendix 1.

Sampling locations for homes, offices and cars were located via aquaintances of the dust collector and often resulted in samples being collected within the same town or city. Samples collected from classrooms were located via postal contact. Around 150 letters were sent to nurseries and primary schools within the West Midlands, with a response of around 40 organisations between March 2008 and March 2009.

The house and office samples were collected from areas of 1 m^2 or 4 m^2, carpeted and uncarpeted, for 2 minutes and 4 minutes, respectively and the area sampled in cars is indicated in Figure 4. This time allowance for collecting these dust samples are derived from Heinrich *et al.* (2003) and Hyvärinen *et al.* (2003) and were defined to produce good reproducibility, whilst allowing collection of the entire dust presence. The location of the sampling area was determined by the use of the room and the area available. Samples were taken from a centrally located area in a room, providing a fair representation of the room, within an area where people are likely to frequent (rather than the corners). The volunteers for the dust collection were derived from acquaintances of the researcher in the Division of Environmental Health and Risk Management, University of Birmingham for homes, offices and cars. The number of samples collected and the type of microenvironment are detailed in Table 19.

Table 19 Microenvironments sampled for this dust study

Country	Home	Office	Car	Nursery or Primary School Classroom
UK	45	20	20	43
Australia	20		10	
Canada	19			
France	9	8		
Germany	10			
Kazakhstan	9	11		
Thailand	20			
USA	10			

Chapter 4 examines the issue of spatial variation in PFC concentrations in dust within personal rooms. Dust from school and nursery classrooms was collected from the entire room, because of different use patterns and greater occupancy rates within classrooms.

Dust samples were collected into a nylon (25 µm mesh) sock fitted within the vacuum cleaner nozzle (Figure 4). The sock was then removed from the nozzle, tied and sealed in a polypropylene bag. The samples were stored in a cold room for the maximum of a week prior to sieving. Samples were sieved through a 500 µm aluminium sieve, with hair and long fibers removed with solvent rinsed tweezers. Following sieving, samples were weighed and stored in glass jars, sealed with aluminium lined lids, and placed back into the cold room until required for extraction. Between dust collections the nozzle and head of the vacuum cleaner were wiped with an alcohol wipe, to avoid cross-contamination.

Table 20 Dust collection and surface type

Surface	Area (m^2)	Time (min)
Carpet (wall-to-wall)	1	2
Rug (larger than 1 m^2)	1	2
Bare floor	4	4

Figure 4 Dust collection area for vehicles (yellow, highlighted regions)

Figure 5 Nylon dust collection sock

2.4.SOIL SAMPLE COLLECTION

Soil samples were collected in 2005, by volunteers at each sample site across the UK (site locations mapped in Figure 30, Chapter 7). The protocol is according to Evans (2008). The samples were all collected using the same protocol, with the collection of the top 5 cm of surface soil from three randomly selected locations across the 10 by 10 metre grid sampling site (see Evans 2008 for further sampling details), followed by homogenisation and storage in an amber glass storage glass (precleaned via washing, rinsing with deionised water and DCM rinse). Care was taken to remove visible roots and debris from the sample prior to storage in the jars. The top 5 cm were collected, as the samples were archived from a previous study on polychlorinated biphenyls (PCBs) in soil and the top 5 cm is primarily where the PCBs reside (Evans, 2008). PFC concentrations in soil have been primarily detected in the top 5 cm (Naile *et al.*, 2010) or surface soil (Washington *et al.*, 2009, Li *et al.*, 2010), however independent work by Daikin America Ltd, indicated an inverse relationship between PFOS and PFOA concentrations with depth (Daikin America Ltd, 2003a and 2003b).

2.5.EXTRACTION METHODS

Extraction methods used within this study were developed individually to isolate the PFCs from the individual sampling media. The extraction methods converge to the same protocol at the later stages whilst undergoing solid phase extraction (SPE) and solvent exchange to maintain as much consistency throughout the methods as is possible. The final extraction methods are summarised in the following sections. The method development began with the use of methanol as the extraction solvent, but did not provide adequate PFC recovery from matrix spike samples. DCM was tested as an alternative, but resulted in excessive quantities of co-extracted material, which required additional purification. Finally, acetone was deemed an acceptable solvent of choice, there were no matrix effects detected and levels of co-extarctives were not excessive (a general method is described in Figure 6). Recovery of standards from matrix spiked samples were good (Table 21), and displayed acceptable reproducibilty.

Table 21 Acetone method reproducibility test using matrix spiked samples (n = 6) (ng g^{-1})

Compound	Average Spiked Concentration	Average Standard Recovery (%)	SD	RSD (%)
PFOS	190	95	4.3	2.3
PFOA	179	90	7.2	4.0
PFHxS	193	97	6.5	3.4
MeFOSA	169	85	8.3	4.9
EtFOSA	195	98	3.7	1.9
FOSA	197	99	7.4	3.7
MeFOSE	191	96	5.0	2.6
EtFOSE	194	97	4.7	2.5

2.5.1. Air

SIP disks used for passive collection of air samples were extracted via soxhlet extraction using a hexane:acetone mix (60:40 v/v) for 8 h. The majority of solvent

was removed from the SIPs and collected along with the rest of the soxhlet wash solvent. The sample was then reduced in volume using nitrogen blowdown, via a Turbovap Concentration Evaporator to approximately 5 mL. The samples were then transferred to centrifuge vials (along with the wash contents of the turbovap tubes), centrifuged to separate the XAD particles from the solvent, and the supernatant removed and placed in a 15 mL centrifuge vial. The vials were washed with acetone and then centrifuged again, with the supernatant being added to the first volume. Samples then underwent solid phase extraction (SPE) via the SPE method detailed in Figure 7.

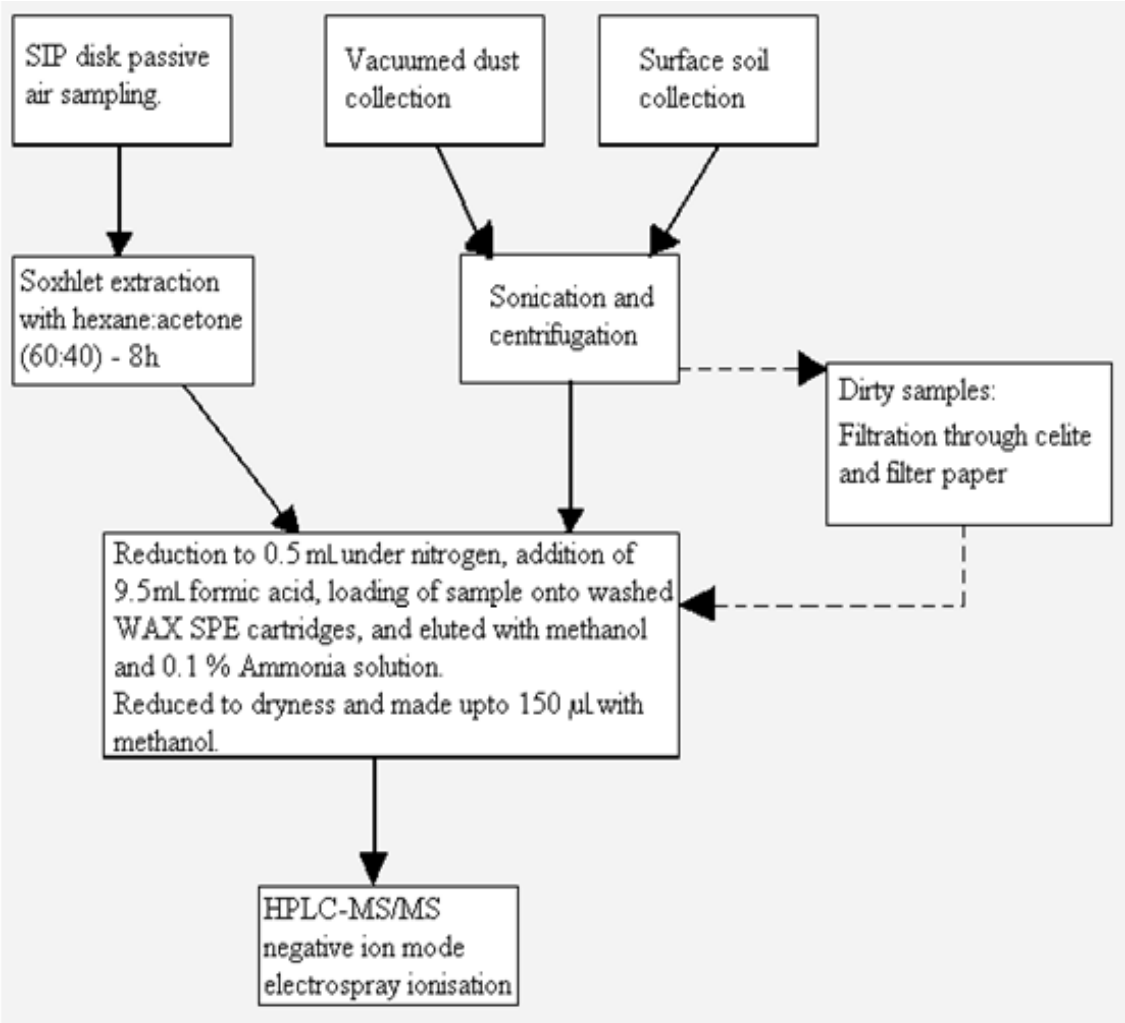


Figure 6 Extraction and purification processes for air, dust, and soil samples

2.5.2. Dust

The dust extraction method was originally produced by Taniyasu (Taniyasu, 2005) and has been adapted along with recommendations from a Waters® application notes publication (Young & Tran, 2006). The two methods were combined to produce the final dust extraction method detailed in Figure 7.

2.5.3. Soil

Soil samples were analysed via a method developed for the extraction of PFCs from sediment (Powley *et al.,* 2005). Samples (5 g, accurately weighed) were mixed with the equivalent amount of pre-extracted anhydrous sodium sulfate (Na_2SO_4) to remove any moisture. 5 ml of acetone was then added along with the internal standards and shaken for 1 h. The sample was then sonicated for 30 minutes with 20 seconds of wrist shaking every 10 minutes. The samples were then centrifuged and the supernatant removed and placed in a clean centrifuge vial. A further 5 mL of acetone was added and the shaking, sonication and centrifugation repeated.

The second aliquot was added to the first, and 3 drops of isooctane (as "keeper" solvent) was added, prior to nitrogen blowdown to 0.5 mL, at which point 9 mL of 2 % formic acid was added. The method then continued with the SPE extraction detailed in Figure 7 and section 6.

2.6. SOLID PHASE EXTRACTION

Solid phase extraction was conducted on Waters® Oasis weak anion exchange (WAX) cartridges which are able to retain strong acids, as well as perfluoroalkyl sulfonates, via the use of piperazine packing and are a mixed phase sorbent. The 1 mL

samples from the extraction procedure were made up to 10 mL with 2 % formic acid added to the SPE columns after a three-step preparation.

Extraction

- Sieve dust, to grade of 500 µm
- Take 100 mg of dust and place in 15 mL centrifuge vial
- Add 5 mL of Acetone
- Add 15 µL of internal standards (Conc. 1000 µL L^{-1})
- Shake for 10 s
- Sonicate for 5min and shake for 10 s (x3)
- Centrifuge for 5 min @ 2000 g
- Decant clear supernatant and transfer to new container
- Repeat procedure from stage 3 with leftovers
- Add two supernatants together
- Add a three drops of isooctane and reduce to approx. 0.5 mL
- Add 9 mL of 2 % formic acid
- If particulates form:
  - Add 1g of Celite.
  - Filter through a grade 1 filter and rinse with 2 mL hexane.

SPE

- Condition Oasis WAX cartridge with:
  - 4 mL 0.1 % NH_4OH in methanol
  - 4 ml methanol (100 %)
  - 4 ml 0.1 % formic acid in deionised water
- Load Sample
- Wash with 4 mL 25 mM sodium acetate buffer, pH 4
- Cover cartridge and dry under vacuum for 30 min (no water to be present, after 30 min)
- Elute with:
  - 4 mL MeOH (elutes FTOHs, PFOSA, NEtFOSA)
  - 4 mL 0.1 % NH_4OH / MeOH (elutes PFCAs, PFOS)
- Reduce under nitrogen to ~200 µl
- Transfer to vial + rinse from centrifuge tube
- Reduce to dryness
- Add 150 µl of methanol
- Store at –20°C until ready for analysis

Figure 7 Dust extraction procedure

The WAX cartridges were washed with 4 mL of 0.1 % ammonia solution, followed by pre-conditioning with 4 mL methanol and 4 mL 0.1 % formic acid, consecutively. Samples were then added to the cartridges. The cartridges were then washed with 4 mL of 25 mM acetate buffer (pH 4), and samples left to dry (to remove residual water) by leaving the cartridges pumping through a supelco vacuum manifold (using a Charles Austin, Capex L2X diaphragm pump, at a pressure of 20 kPa) for 30 minutes.

Once the SPE cartridges were dry, the samples were extracted from the solid phase, using 4 mL methanol, followed by 4 mL 0.1 % ammonium solution in methanol. The first wash (methanol) extracted the fluorotelomer alcohols and perfluorooctane sulfonamides; while the ammonia extracted the remaining compounds; perfluorocarboxylic acids and perfluorinated sulfonates.

2.7.HPLC – TANDEM MASS SPECTROMETRY

Analysis of perfluorinated compounds were conducted primarily via liquid chromatography (LC) separation and detection via mass spectrometry (MS) using a Shimadzu HPLC coupled with an API 2000 tandem mass spectrometer (MS/MS). The triple quadrupole MS/MS was operated in negative ion mode, using electrospray ionisation. The conditions of the MS/MS can be found in Table 23 and the HPLC mobile phase gradients in Table 22. The column used throughout the study was a Varian C18 Metasil Basic, 5 µm Si, 150 x 2.1 mm, 100 Å.

Table 22 LC operating condititions

Mobile Phase	A: 2 mM ammonium acetate in water/methanol 9:1 B: 100 % Methanol		
Flow Rate	0.2 mL min^{-1}		
Gradient	**Time (min)**	**A (%)**	**B (%)**
	0	90	10
	0.1	70	30
	7	25	75
	10	0	100
	12	0	100
	16	90	10
	20	90	10

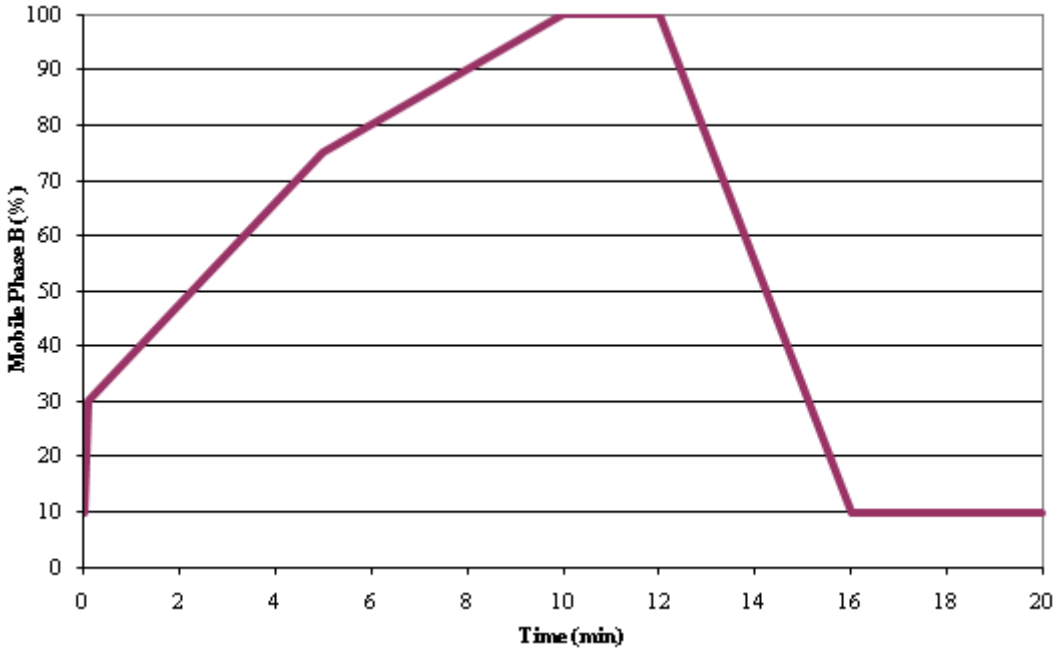


Figure 8 Mobile phase gradient profile

Details regarding the setup of the methodology for the mass spectrometer are detailed in Table 23 and Table 24. These parameters are specific for the individual mass spectrometer used and are likely to vary for other mass spectrometers. The chromatographic output from a set of 200 ng mL^{-1} standards (native and internal) is shown in Figure 9.

Table 23 MS operating conditions

Parameter	Value
Capillary Voltage	1 kV
Cone & Desolvation Gas	60 –740 L h^{-1}
Curtain Gas	50
Collision Gas	5
Temperature	250 °C
Ion Spray Voltage	-4500 V
Ion Source Gas 1, 2	45, 30
Focusing Potential	-400
Declustering Potential	-60 V
Resolution	Unit
Pause between mass ranges	5 ms
Source	Negative Electrospray Ionisation
MS/MS Operation	MRM
Run Time	20.6501 min
Total Scan Time	0.6501 s

Table 24 Monitored Ions and associated parameter values

Transitions	[M-K]$^-$ or [M-H]$^-$	Entrance Potential	Collision Energy	Collision Cell Exit Potential	RT (min)
FOSA	497.8 > 78	-10	-80	-3	13.0
MeFOSA	511.9 > 169	-10	-35	-15	13.6
EtFOSA	526 > 169	-10	-35	-18	13.7
d-MeFOSA	514.8 > 169	-10	-35	-15	13.7
MeFOSE	616 > 59.1	-12	-40	-5	13.6
EtFOSE	630 > 59.1	-12	-40	-5	14
d7-MeFOSE	623 > 59.1	-12	-40	-5	13.7
PFHxS	398.8 > 80.1	-12	-80	-12	10.2
MPFHxS	402.7 > 102.7	-12	-80	-6	10.2
PFOS	499 > 99.1	-9	-70	-8	11.3
MPFOS	502.8 > 99.1	-9	-70	-8	11.3
PFOA	412.9 > 369	-12	-11	-35	10.6
MPFOA	416.9 > 372.8	-12	-11	-35	10.6

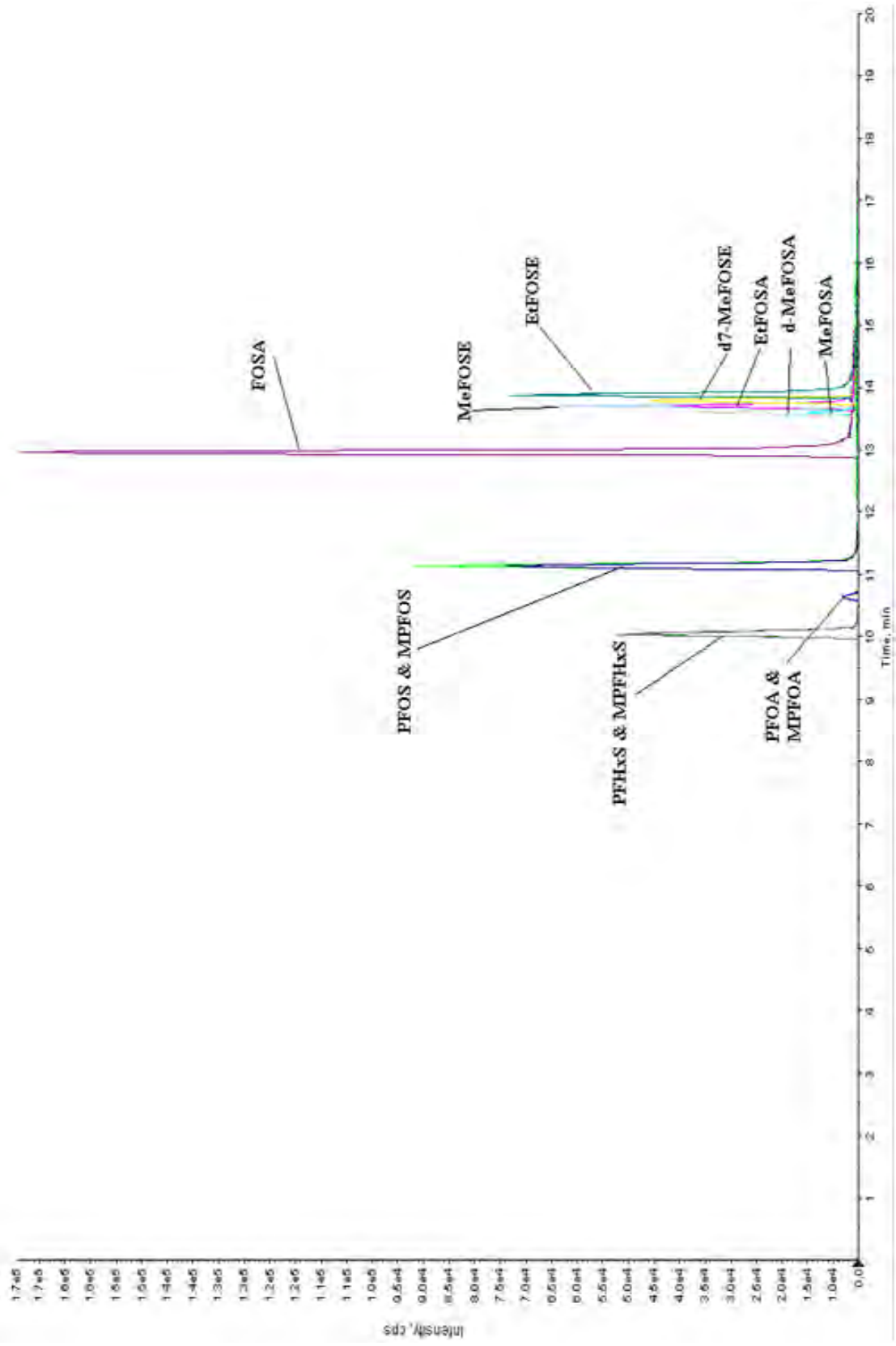


Figure 9 Chromatogram of perfluorinated compounds and retention times

2.8. QUALITY ASSURANCE / QUALITY CONTROL

Reliability of analytical data throughout the study was imperative in order to ensure that all data produced was accurate and reproducible. This section covers the QA/QC procedures that have been applied to monitor the data, from instrumentation calibration, internal standards to field and reagent blank analyses.

The LC-MS/MS was calibrated at the beginning of each sample batch analysed, after instrumentation shut-down and maintenance and after long periods of down-time. The calibration point concentrations used for all standards included 10, 20, 50, 100, 200, 1000 and 5000 ng mL^{-1}. The response of each calibration was compared to the original calibration set-up to determine the extent of any changes. This response was checked in the form of the relative response factor (RRF) for each of the native standards (NAT), which is a comparison to the response of the internal standards (IS). The response used is the area of the peak (A) with regards to the concentration (C) of the standard used, Equation 2 describes the calculation of the RRF and Table 25 details the RRFs for each native standard.

Equation 2

$$RRF = \frac{A_{NAT}}{A_{IS}} \times \frac{C_{IS}}{C_{NAT}}$$

Calculation of RRFs for each of the standards comprising the multi-point calibration, should reveal them to be essentially identical in each standard. Ideally, the relative standard deviation (*i.e.* (σ_{n-1}/average) x 100 %) of RRFs for a given target compound should not exceed 10 %.

Table 25 Calibration curve response

Calibration Standard (ng ml^{-1})	PFOS RRF	PFOA RRF	PFHxS RRF	MeFOSA RRF	EtFOSA RRF	FOSA RRF	MeFOSE RRF	EtFOSE RRF
10	1.22	0.99	1.00	0.86	0.97	1.60	0.97	1.34
20	1.22	1.10	0.91	0.89	1.20	1.54	0.99	1.28
50	1.20	1.12	0.95	0.79	1.00	1.42	1.00	1.29
100	1.23	1.10	0.94	0.82	1.10	1.48	0.95	1.27
200	1.19	1.30	0.99	0.85	1.10	1.53	0.96	1.30
1000	1.25	1.07	0.98	0.80	1.00	1.57	0.99	1.30
5000	1.26	1.05	0.94	0.80	1.10	1.58	1.00	1.29
Mean	**1.2**	**1.1**	**1.0**	**0.8**	**1.1**	**1.5**	**1.1**	**1.3**
SD	**0.0**	**0.1**	**0.0**	**0.0**	**0.1**	**0.1**	**0.0**	**0.0**
RSD (%)	**2.0**	**8.7**	**3.4**	**4.5**	**7.6**	**4.1**	**1.8**	**1.7**

However, the use of a single internal standard (d-MeFOSE) for the quantification of all perfluorooctane sulfonamides (PFOSAs) and perfluorooctane sulfonamidoethanols (PFOSEs) resulted in the RRF values for FOSA exceeding this 10 % RSD mark. The difference in the mass of FOSA compared to the IS is approximately 17 g mol^{-1} and is responsible for the difference in the analytical response. This difference can also be seen in Figure 9, where FOSA elutes approximately half a second before the other PFOSAs. Despite this variation between the native FOSA standard and the d-MeFOSE IS, the results from the calibrations were consistent during the study and remained within 25 % of the original calibration RRFs throughout.

With the calculation of the RRF taking account of analyte losses, the mass of the analyte was calculated using Equation 3, where RRF_{STD} is the relative response factor calculated from the calibration standards, M_{IS} is the mass of the internal standard added to the sample and M_S is the mass of the sample.

Equation 3 Mass of sample

$$Mass = \frac{A_{NAT}}{A_{IS}} \times \frac{1}{RRF_{STD}} \times \frac{M_{IS}}{M_S}$$

Table 26 Relative response factors for all native standards

Calibration Standard (ng ml^{-1})	PFOS RRF	PFOA RRF	PFHxS RRF	MeFOSA RRF	EtFOSA RRF	FOSA RRF	MeFOSE RRF	EtFOSE RRF
10	1.22	0.99	1.0	0.86	0.97	1.6	0.97	1.34
20	1.22	1.1	0.91	0.89	1.2	1.54	0.99	1.28
50	1.2	1.12	0.95	0.79	1.0	1.42	1.0	1.29
100	1.23	1.1	0.94	0.82	1.1	1.48	0.95	1.27
200	1.19	1.3	0.99	0.85	1.1	1.53	0.96	1.3
1000	1.25	1.07	0.98	0.8	1.0	1.57	0.99	1.3
5000	1.26	1.05	0.94	0.8	1.1	1.58	1.0	1.29
Mean	**1.2**	**1.1**	**1.0**	**0.8**	**1.1**	**1.5**	**1.1**	**1.3**
SD	**0.02**	**0.06**	**0.03**	**0.04**	**0.08**	**0.06**	**0.02**	**0.02**
%RSD	**2.0**	**8.7**	**3.4**	**4.5**	**7.6**	**4.1**	**1.8**	**1.7**

2.8.1. LC-MS/MS Tuning

The LC-MS/MS was maintained to achieve the manufacturer's standard, using the PPG standards, which are designed to tune the resolution and calibrate the mass axis. The tuning was conducted after any maintenance, and periods of shut-down. When the correct resolution is achieved for the manufacturer's standards, the response of the LC-MS/MS to the set pollutants being analysed should remain consistent. Occasionally, in order to achieve the optimum resolution of the PPG, standards parameters of the MS/MS required alteration and resulted in slight mass changes for the quantification of the native and internal standards. Therefore, calibration standards

would be re-run and RRFs checked to determine that the same resolution of target PFCs was being achieved.

Target pollutant quantification from a peak was only considered to be valid if the following criteria were met:

1. Peak signal to noise ratio exceeded 3:1
2. The RRT of the peak remained within 0.2 % of the average calibration RRT calculated from the calibration standards run for that batch of samples.

2.8.2. Internal Standards (IS)

The use of internal standards for PFCs at the time of initial study set-up was fairly limited, relative to other POPs, and therefore only five internal standards were chosen to represent the native standards. Use of the internal standards meant that no corrections were required for analyte recovery when calculating concentrations in samples. However, recoveries of internal standards were still measured for QA/QC purposes. An acceptable recovery of a given IS was considered to between 30 and 150 %. The IS was able to exceed the 100 % value for reasons such as matrix effects. As well as the recovery of the IS, the signal:noise ratio of the IS must exceed, as a minimum, 20:1, to be considered valid. The IS recoveries for all samples analysed in this study are detailed inTable 27, Table 28 and Table 29. Any samples which were identified to have an IS recovery outside the acceptable range were discarded and the sample re-extracted and analysed.

Table 27 Internal standard percentage recoveries from air samples

IS	Number	Mean (%)	Range (%)	SD	%RSD
MPFOS	211	74	34 –110	19	25
MPFOA	211	73	32 – 105	19	26
MPFHxS	211	77	31 – 103	18	23
d-MeFOSA	211	68	30 – 129	22	33
d7-MeFOSE	211	71	31 – 99	21	30

Table 28 Internal standard percentage recoveries from dust samples

IS	Number	Mean (%)	Range (%)	SD	%RSD
MPFOS	576	79	36 – 120	18	23
MPFOA	576	76	37 – 119	18	24
MPFHxS	576	76	34 – 119	19	25
d-MeFOSA	576	64	30 – 112	18	28
d7-MeFOSE	576	75	33 – 127	20	26

Table 29 Internal standard recovery percentages for soil samples

IS	Number	Mean (%)	Range (%)	SD	%RSD
MPFOS	67	78	38 – 102	14	17
MPFOA	67	83	52 – 108	13	15
MPFHxS	67	76	31 – 107	17	22
d-MeFOSA	67	74	30 – 105	18	24
d7-MeFOSE	67	77	30 – 101	15	19

2.8.3. Precision and Ongoing Monitoring of Method Performance

QA/QC for the entire method from extraction to the final analysis and result, was checked via regular analysis of an in-house laboratory reference material (LRM), obtained from a whole vacuum cleaner bag sample, sieved, homogenised and stored at –20°C. An aliquot of the LRM was analysed after every 20 samples, to provide an on-going check on method performance relative to the data obtained initially for 5 replicate analyses of the LRM. For data from a sample batch to be considered valid, the concentration of a given PFC in an ongoing LRM check analysis must fall within 30 % of the average value obtained from the initial replicate analysis.

Alongside the LRM, aliquots of SRM 2585 house dust (developed by the National Institute of Standards and Technology (NIST) and purchased from Greyhound Chromatography and Allied Chemicals) were also analysed for PFC content. From November 2009 the SRM was used additionally to the LRM, as an additional QA/QC measure. The results of the measured LRM and SRM concentrations from the entire study are detailed in Table 30.

SRM 2585 was also analysed by J. Björklund from the Department of Applied Environmental Science (ITM), Stockholm University, Sweden for both PFOS and PFOA. Average concentrations were 1990 ng g^{-1} and 673 ng g^{-1} for PFOS and PFOA (Björklund *et al.*, 2008). The results of the SRM analysis for this project indicated an agreement with Björklund, with average concentrations of 1752 ng g^{-1} and 766 ng g^{-1} for PFOS and PFOA, which are within 12 % of the results from Sweden.

Throughout the course of the project there were no available certified reference materials for PFCs, thus the reason that a LRM was used along with the SRM 2585. The reproducibility of the PFCs in both these materials provided the quality assurance for the whole analysis, and were used in accordance to the general use of a SRM.

Table 30 Concentrations (ng g^{-1}) of PFCs in both LRM and SRM in this study.

LRM, *n* = 56	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**
Range	160-240	120-190	100-160	0	120-200	100-180	410-500	240-300
Mean	197	153	128	0	169	135	446	274
SD	18	15	14	0	18	15	24	16
%RSD	9	10	11	0	11	11	5	6
SRM, *n* = 37								
Range	1700-1800	710 – 860	3200-4500	45-65	150-210	67-86	520-620	57-76
Mean	1752	766	4282	54	179	75	562	66
SD	31	38	274	5	10	5	23	4
%RSD	2	5	6	8	5	7	4	6

2.8.4. Reproducibility Tests

Reproducibility tests were conducted for each of the three methods used. These were conducted using matrix spike samples which consisted of 0.1 g sodium sulfate spiked with native standards to produce a final sample concentration of 200 ng mL^{-1}. The results are located in Table 31, displaying the percentage recoveries of the native spikes, mean recovery, standard deviation (SD) and relative standard deviation (%RSD).

Table 31 Reproducibility and accuracy test for air samples using matrix spikes at 150 pg m^{-3} (% recovery)

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
BLK	n.d	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
1	96	98	87	97	90	93	98	96
2	82	91	83	75	85	76	91	85
3	77	82	78	95	82	76	84	83
4	96	86	99	83	77	84	87	75
5	97	93	94	88	97	91	85	78
Mean	90	90	88	88	86	84	89	83
SD	9	6	8	9	8	8	6	8
%RSD	10	7	10	10	9	10	6	10

Table 32 Reproducibility and accuracy for dust samples, using matrix spikes of 150 ng g^{-1} (% recovery)

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
BLK	n.d	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
1	89	87	97	90	91	97	84	89
2	96	84	94	91	89	98	85	74
3	91	93	92	87	91	96	82	89
4	99	85	82	79	76	76	96	85
5	83	82	79	88	93	91	89	88
Mean	92	86	89	87	88	92	87	85
SD	6	4	8	5	7	9	6	6
%RSD	7	5	9	5	8	10	6	7

Table 33 Reproducibility and accuracy test for soil method with matrix spiked soil, 150 ng g^{-1} (% recovery)

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
BLK	0.1	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
1	70	75	82	80	98	81	74	100
2	85	70	97	91	75	80	86	87
3	68	70	91	90	96	95	95	92
4	75	85	80	83	83	88	77	78
5	83	88	92	73	85	80	82	76
Mean	76	78	88	83	87	85	83	87
SD	8	8	7	7	10	7	8	10
RSD (%)	10	11	8	9	11	8	10	11

Table 34 Reproducibility test for standard reference material 2585 (ng g^{-1})

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	EtFOSE	MeFOSE
BLK	1.1	0.3	1.2	n.d.	n.d.	n.d.	n.d.	n.d.
1	1800	800	3900	55	200	55	54	500
2	1700	660	4200	51	200	79	64	550
3	1400	760	4300	43	150	69	68	560
4	1700	710	4700	53	190	69	74	520
5	1900	720	4600	52	190	77	65	630
Mean	1700	730	4300	50	190	70	65	550
SD	210	53	320	4.8	21	9.7	7.3	48
RSD (%)	12		7.3	9.3	11	14	11	8.8

2.8.5. Blank Results

Field blanks were run for every 5 samples analysed, to maintain assurance that the concentrations were not introduced through the handling of the sample. The field blanks consisted of newly prepared SIPs, which were taken, placed into the sampling equipment (as with the use of a normal sampling SIP), then removed, sealed and returned to the laboratory. Blank concentrations were accepted when they remained below 5 % of the lowest concentration within the batch. If concentrations in the blank exceeded this, within a parameter of 5 – 20 % of the sample concentrations, the samples would be corrected accordingly. Any blank exceeding the 20 % threshold, required rejection of the batch. During the project no samples exceeded the 5 % parameter and therefore no transformations or rejections were conducted.

Table 35 Air sample blank concentrations (pg m^{-3}), assuming 50 m^3 of air.

Compound	Passive Air Sampler Blank Data (pg m^{-3})			SD	%RSD
	Number	Average	Range		
Indoor					
PFOS	32	0.16	<DL– 1.6	0.32	208
PFOA	32	<DL	<DL	0.27	-
PFHxS	32	<DL	<DL – 0.7	0.25	160
MeFOSA	32	<DL	<DL	0.03	-
EtFOSA	32	<DL	<DL – 0.9	0.32	250
FOSA	32	<DL	<DL – 1.1	0.4	120
MeFOSE	32	<DL	<DL – 1.2	0.33	140
EtFOSE	32	<DL	<DL – 0.9	0.42	390
Outdoor					
PFOS	15	<DL	0 – 2.2	0.63	277
PFOA	15	<DL	<DL	0.29	-
PFHxS	15	0.24	<DL – 0.8	0.35	147
MeFOSA	15	<DL	<DL	0.03	-
EtFOSA	15	<DL	<DL – 0.6	0.16	230
FOSA	15	0.1	<DL – 1.2	0.32	309
MeFOSE	15	<DL	<DL	0.06	223
EtFOSE	15	<DL	<DL – 0.7	0.18	270

Table 36 Dust sample blank concentrations (ng g^{-1}), assuming 0.5g of dust.

Compound	Dust Blank Data (ng g^{-1})			SD	%RSD
	Number	Average	Range		
PFOS	104	<DL	<DL– 7	1.01	263
PFOA	104	<DL	0 – 6	0.8	316
PFHxS	104	<DL	0 – 5	0.89	293
MeFOSA	104	<DL	0 – 1.4	0.21	583
EtFOSA	104	<DL	<DL – 9	0.99	485
FOSA	104	<DL	<DL – 2.4	0.26	645
MeFOSE	104	<DL	<DL – 12	1.3	524
EtFOSE	104	<DL	<DL	0	0

Table 37 Soil sample blank concentrations (pg g^{-1}), assuming 5g of soil.

Compound	Soil Blank Data (pg g^{-1})			SD	%RSD
Indoor	Number	Average	Range		
PFOS	10	<DL	<DL – 17	0.05	268
PFOA	10	<DL	<DL – 67	0.5	318
PFHxS	10	<DL	<DL – 30	0.36	218
MeFOSA	10	<DL	<DL	0	0
EtFOSA	10	<DL	<DL	0	0
FOSA	10	<DL	<DL	0	0
MeFOSE	10	<DL	<DL – 52	1.8	177
EtFOSE	10	<DL	<DL – 63	2.5	167

2.8.6. Limit of Detection (LOD)

The limits of detection for each individual PFC are represented in Table 38. These are defined as the quantity of the analyte which provides a signal to noise ratio of 3:1. In the majority of cases, the blank concentrations did not exceed the LOD, but in cases where the blanks contained concentrations above these levels, the blank concentration was used as the LOD. Half the LOD was used as the concentration for non-detected concentrations of PFCs in samples for the purposes of calculating descriptive statistics and, therefore, where the blank exceeded this concentration, the representation of the values below the detection limit (<DL) was half of the blank. The limit of quantification (LOQ) was determined as the lowest measurable concentration in the extracted sample, with respect to the LOD, final extract volume (FEV), volume of final extract injected (VFEI), sample size (SS) and percentage of internal standard recovery (% IS Rec), and is calculated according to the following equation (Equation 4).

Equation 4

$$LOQ = \frac{LOD \times FEV}{SS \times VFEI} \times \frac{100}{\%IS\ Rec}$$

Table 38 Limit of detection and limits of quantification for PFCs in air, dust and soil.

Compound	LOD	Sample Limit of Quantification		
		Air (pg m^{-3})	Dust (ng g^{-1})	Soil (pg g^{-1})
PFOS	0.03	1.0	1.1	16
PFOA	0.98	1.9	1.3	44
PFHxS	0.09	1.1	1.0	28
MeFOSA	0.1	2.5	1.7	11
EtFOSA	0.07	5	0.9	64
FOSA	0.02	1.8	1.2	19
MeFOSE	0.22	3.9	0.9	30
EtFOSE	0.12	2.4	0.9	18

2.8.7. Sample Storage

The precleaned sampling material was stored at 4°C in a cold room, wrapped in aluminium foil and put into an air tight container, in the dark prior to use. Samples were collected, wrapped in aluminium foil and sealed in air tight bags. The samples were stored at –20°C before extraction for both dust and air samples. Before analysis, extracted samples were stored in polypropylene vials at –20°C in the dark.

Standards were transferred from the manufacturer's snap vials to Certan vials (from LGC Promochem), which have a very low solvent evaporation rate that maintains standard integrity, (aided by storing the standards at –20°C). The compounds were removed from the freezer and allowed to reach room temperature prior to use, and then replaced in the freezer directly after use. During each use, the vials were measured after reaching ambient temperature, prior to use, to determine whether any loss occurred during the storage. At no time should the difference in the vial's weight deviate by more than 5% from that of the last measurement. At no point did the weight of the vial deviate by more than 3 % of the last recorded figure.

2.8.8. Software

Software used for the analysis of data in this study include:

- Statistics - SPSS v17.0 for Windows and Microsoft Excel, XP (T-test, ANOVA, Shaprio-Wilk test, Principle component analysis, Varimax roatation, Keyser-Meyer Olkin and Bartlett's test of Sphericity.
- Graphical Representation - Sigma plot v11.0 and Microsoft Excel, XP.
- Data Acquisition – Analyst 1.4.2 and Mass Spectrometry toolkit v3.3,
- Applied biosystems peak analysis tool. Peaks were automatically integrated, followed by a manual visual check of the peak selection.
- Microsoft Excel, 2000 and XP.

3. CONCENTRATIONS OF PFCS IN INDOOR MICROENVIRONMENTS

The hypothesis addressed in this chapter is that indoor environments are reservoirs of perfluoroalkyl compounds. Hence, this chapter's aim is to determine concentrations of PFCs in indoor air and dust from a number of commonly-frequented microenvironment categories. Three indoor microenvironment categories are studied, via homes offices and cars, because in the UK the general population spend a large percentage of their time daily within these environments (UK National Office of Statistics, 2000 & 2005). The data presented in this chapter reports for the first time concentrations of PFCs in UK indoor air and dust.

3.1. INTRODUCTION TO INDOOR PATHWAYS OF EXPOSURE

Dust and air within indoor microenvironments constitute potential pathways of human exposure. Adults and children ingest on average 20 mg and 50 mg of dust each day respectively (Jones-Otazo *et al.*, 2005) via hand to mouth behaviour, ingestion of dust with food and drink, and inhalation of suspended particulates (Sasaki *et al.*, 2003). Dust ingestion has been identified as a significant pathway of exposure to other halogenated organic chemicals such as hexabromocyclododecanes (HBCDs), influencing the daily exposure more than diet and inhalation under some exposure scenarios (Abdallah *et al.*, 2008). Within the lifetime of a product, it is common for chemicals to leach out and migrate into the surrounding environment from general use and disintegration of the product over time. The release of a chemical from the original product depends upon the way in which the particular chemical is incorporated within the product. Residual chemicals from manufacturing processes

are unlikely to be strongly bonded to the polymer, and can often be relatively free to migrate. However, chemicals that are actually bonded to a polymer will escape at a slower rate as the polymer itself abrades over time. Manufacturing of perfluorinated compounds can produce residual compounds such as MeFOSE and FTOHs (Dinglasan – Panlilio & Mabury, 2006) during the production of polymeric and surfactant solutions used for carpet cleaning, windscreen washer solutions, and surfactants for paints and lacquers. The presence of residuals can result in indoor concentrations of PFCs exceeding those found outdoors (Shoeib *et al.*, 2004). These comparatively high indoor concentrations of chemicals like PFCs, PCBs and brominated flame retardants can occur due to low ventilation rates and the presence of numerous indoor sources (Walters & Santillo, 2006, Liu *et al.*, 2009, Abdallah *et al.*, 2008).

PFCs in this study partition into both gaseous or particulate phases with PFOS, PFOA and PFHxS preferring the particulate phase (because of their low vapour pressures) (Giesy & Kannan, 2002) and the precursor compounds favouring the gaseous phase (because of their higher vapour pressures). However, all compounds have been identified in both media (air and dust) from indoor environments (Shoeib *et al.* 2005, Bjorklund *et al.*, 2009, Moriwaki *et al.* 2005, Kato *et al.*, 2009, Strynar & Lindstrom 2008).

PFCs in dust are likely to originate from the presence of micro-fibres abraded from textile and fabric furnishings, from abrasion of coatings from flooring and packaging products, and volatilisation from such goods followed by deposition to dust. The presence of threads and fibres in dust contributes to a large percentage of the total dust volume, along with pollens and other debris (Webster *et al.*, 2009).

On a daily basis, it has been estimated that > 90 % of time is spent indoors, usually within the home, work place and in vehicles travelling to and from work (Adgate *et al.,* 2004). In this study, PFC concentrations have been measured in dust from homes (n = 45); cars (n = 20) and offices (n = 20) because of the amount of time the general population spend in each microenvironment every day. Values derived from the UK government statistics reveal that adults spend 68.5 %, 16.7 %, 6.4 %, 4.4 % and 4 % at home, at work, in their car, outside, and in other microenvironments respectively (UK National Office of Statistics, 2000 & 2005). This significant amount of the day spent indoors by the general population can have a large impact upon the levels of organohalogens to which they are exposed (Wilford *et al.*, 2005, Harrad & Diamond, 2006, Abdallah *et al.*, 2008).

Levels of perfluorinated compounds in homes are likely to be influenced significantly by the specific types and presence of furnishings, fabrics, flooring materials and treatments present. By comparison, offices typically contain fewer fabrics but greater quantities of PFC-treated paper, printer inks and toners, as well as industrial cleaning products. Moreover, the greater "wear and tear" of office furniture means that the associated fabrics may be more widely and extensively stain proofed than in homes. Likewise, fabrics within modern car interiors are often stain-proofed. The home, car and office microenvironments were selected randomly from the Birmingham (UK) region, and consisted of a variety of building/vehicle ages, designs and locations.

This chapter addresses the quantification of 8 PFCs in air and dust samples from homes, cars and offices, and examines the differences found in the concentrations from various microenvironments. The presence of concentration differences between microenvironments can impact upon exposure assessments.

3.2. AIR CONCENTRATIONS

Passive air samplers were deployed in homes and offices within the Birmingham region, for a period of 30 (±2) days. The samplers were assembled as described in chapter 2 and fitted with a single SIP disk. Field blank sample results were all non-detects for all target PFCs.

Atmospheric concentrations of PFCs in homes and offices are displayed in Table 39 and Table 40 along with descriptive statistics. Results indicate the majority of PFCs are present in indoor air, but with the exception of MeFOSA, which had limited detection. The more volatile compounds: EtFOSA, FOSA, MeFOSE and EtFOSE were the most abundant in air (12 – 3100 pg m^{-3}). Despite the lower volatility, PFOS, PFOA and PFHxS were detected in > 90 % of the air samples. This may be partly attributable to the passive samples retaining deposited particulates as well as vapour phase PFCs (Hazrati & Harrad, 2007). Similar studies of UK outdoor air (using active HiVol samplers) have indicated the presence of PFOS and PFOA on filters, but none in the gaseous phase. Comparison of the median and mean from both home (1800 pg m^{-3}, 2000 pg m^{-3}, respectively) and office (1300 pg m^{-3}, 1300 pg m^{-3}, respectively) air samples indicate similar values for ΣPFC concentrations(Table 2). The individual compound descriptive statistics show that office samples have higher mean and median concentrations for the less volatile compounds, PFOS (53 pg m^{-3}, 56 pg m^{-3}), PFHxS (93 pg m^{-3}, 95 pg m^{-3}) and EtFOSA (45 pg m^{-3}, 58 pg m^{-3}), similar arithmetic means and medians for PFOA (17 pg m^{-3}, 58 pg m^{-3}), MeFOSA (<DL, 6 pg m^{-3}) and FOSA (49 pg m^{-3}, 74 pg m^{-3}), but lower office values for MeFOSE (380 pg m^{-3}, 480 pg m^{-3}) and EtFOSE (380 pg m^{-3}, 490 pg m^{-3}). This relationship could be due to the room usage in offices, with most offices sampled being well ventilated and more sparsely furnished than homes. Better ventilation in offices, facilitates more efficient

removal of more volatile compounds, while the relative lack of furnishing results in reduced PFC emissions.

Table 39 UK home air concentrations (pg m-3)

Home	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFCs
1	15	18	26	<DL	66	34	1800	1080	3000
2	16	<DL	5	<DL	52	55	2600	1900	4600
3	31	<DL	30	<DL	55	44	1500	1400	3100
4	14	15	18	<DL	280	33	3100	740	4200
5	12	<DL	0	<DL	42	24	1100	620	1800
6	9	27	22	<DL	25	1300	1200	92	2700
7	70	77	<DL	19	83	680	310	620	1900
8	60	37	9	<DL	43	61	77	1100	1300
9	<DL	30	12	<DL	67	41	860	180	1100
10	10	40	0	<DL	85	83	240	220	680
11	8	35	39	<DL	61	33	340	270	790
12	<DL	40	29	<DL	250	16	700	190	1200
13	61	67	20	6	160	23	810	470	1600
14	7	14	30	<DL	120	96	190	460	910
15	400	20	220	3	350	23	710	750	2500
16	22	14	16	7	59	160	1100	100	2400
17	1	17	24	<DL	61	100	890	710	1800
18	8	14	110	<DL	210	140	200	560	1300
19	5	440	52	<DL	300	43	710	510	2000
20	5	140	49	<DL	14	46	580	80	910
Minimum	<DL	<DL	<DL	<DL	14	16	77	80	680
5th Percentile	<DL	<DL	<DL	<DL	25	23	180	91	780
Median	11	24	23	<DL	67	45	760	590	1800
Geometric Mean	11	18	10	<DL	85	64	670	480	1700
Arithmetic Mean	38	52	36	2	120	150	950	650	2000
95th Percentile	87	150	120	7	300	710	2600	1500	4200
Maximum	400	440	220	19	350	1300	3100	1900	4600
SD	88	96	50	5	100	310	800	470	1100
%RSD	230	190	140	260	85	203	84	72	55

Table 40 UK office air concentrations (pg m^{-3})

Office	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFCs
1	78	70	330	<DL	12	100	470	720	1800
2	34	49	<DL	<DL	35	170	150	240	680
3	12	13	170	50	59	14	180	340	840
4	51	200	5	<DL	72	4	38	180	550
5	62	190	120	<DL	29	38	1200	680	2300
6	52	<DL	<DL	<DL	61	15	450	740	1300
7	44	14	12	<DL	21	23	1200	950	2300
8	89	18	84	<DL	30	65	230	210	730
9	55	15	110	<DL	150	63	620	420	1400
10	88	17	150	<DL	56	59	310	830	1500
11	79	98	49	23	30	310	190	260	1000
12	27	14	100	0	150	24	710	320	1300
Minimum	12	<DL	<DL	<DL	12	4	38	180	550
5th Percentile	20	7	<DL	<DL	17	10	99	200	620
Median	53	17	93	<DL	45	49	380	380	1300
Geometric Mean	49	25	21	<DL	45	41	330	420	1200
Arithmetic Mean	56	58	95	6	58	74	480	490	1300
95th Percentile	89	200	240	35	150	230	1200	890	2300
Maximum	89	200	330	50	150	310	1200	950	2300
SD	25	70	96	15	45	89	390	270	590
%RSD	44	120	100	250	77	120	82	56	45

The air data was tested for normality of distribution, and differences in the presence of individual PFCs was observed for homes and offices. The Shapiro-Wilk test was used, as opposed to the Kolmogorov - Smirnov test, because each microenvironment category contained less than 50 samples, and the Shapiro-Wilk has proven superior when handling small data sets (Livingston, 2004). While concentrations of most PFCs in both home and office microenvironments were revealed by the Shapiro-Wilk test to be positively skewed (apart from EtFOSE, $p < 0.023$), there were some exceptions (EtFOSE, $W = 0.914$, $p = 0.076$). Analysis of room contents and concentrations was conducted for the air concentrations and no significant relationships ($p > 0.05$), were identified from the data given on the questionnaire (this included flooring type, textiles, ventilation, electronics and time spent in room).

A t-test was conducted for home and office air samples, to compare the means for statistical differences. All data were either normally distributed or were normalized using a log transformation Significantly different air concentrations were identified ($p < 0 .05$) for PFOS, EtFOSA and MeFOSE. Offices may contain fewer volatile compounds (means of PFHxS and PFOA are also raised in office environments) due to differences in room use, and different sources.

PFOS in air concentrations were significantly higher in offices than homes ($p = 0.001$), where the opposite was true for EtFOSA ($p = 0.039$) and MeFOSE ($p = 0.033$). Although not significantly different, concentrations of volatile compounds were higher in homes than offices and the converse is seen in offices for the less volatile compounds.

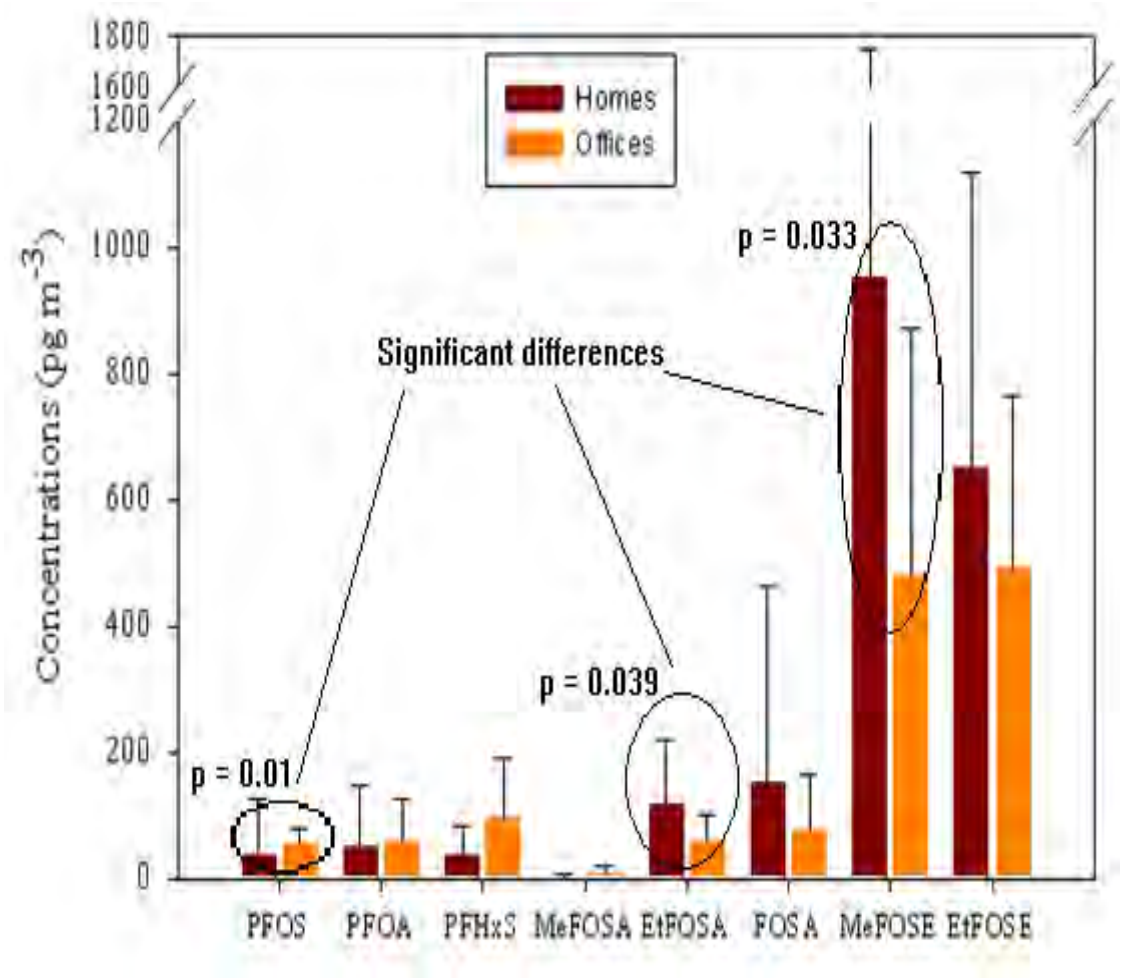


Figure 10 Comparison of arithmetic mean air concentrations in homes and offices (pg m^{-3})

PCA analysis was used to examine patterns among PFC profiles in homes and offices. Three factors (Table 41) accounted for 61 %) of the variance ((Figure 11). The PCA divides the data into two main groups. The two groups are dependent upon factor 1 (Figure 11). Cluster 1 samples have positive scores on factor 1 due to higher contents of the following compounds in the samples: MeFOSA, PFHxS, PFOS, EtFOSE and FOSA and samples in the cluster were mainly offices. Cluster 2, which contained mostly homes, had negative factor 1 scores with greater contributions of MeFOSE in the samples.

Homes in cluster 2 tended to have carpeting, electronics and general furnishings, with higher concentrations related to newer products within the rooms (as indicated by the questionnaire).Offices with larger contributions of MeFOSE fall within this category could be attributed to newer furnishings within the individual offices, but homes remain greater than offices.

MeFOSE is also present in carpet stain repellent formulae, and may be linked to homes with treated carpets (Beesoon *et al.* 2010). However, when MeFOSE concentrations in carpeted homes and offices are compared with those in non-carpeted microenvironments, there are no significant differences, probably attributable to many indoor environments containing carpets of different ages and production, and thus differences for microenvironments with and without carpets were indistinguishable.

Comparison with other studies of PFCs in indoor air (Table 42) supports this study's finding that concentrations in homes exceed those in offices. De Voogt *et al*., (2008) collected indoor air samples via HiVol samplers using glass fibre filters (GFF) to collect the particulate phase and a combination of PUF and XAD in columns for the gaseous phase.

Table 41 PCA factor loadings for air samples.

Compound	Component		
	1	2	3
PFOS	0.507	0.488	-0.073
PFOA	-0.028	0.782	0.214
PFHxS	0.695	-0.071	-0.086
MeFOSA	0.678	-0.246	0.217
EtFOSA	-0.055	0.667	-0.115
FOSA	0.242	-0.061	0.794
MeFOSE	-0.777	-0.442	0.077
EtFOSE	0.400	-0.114	-0.751

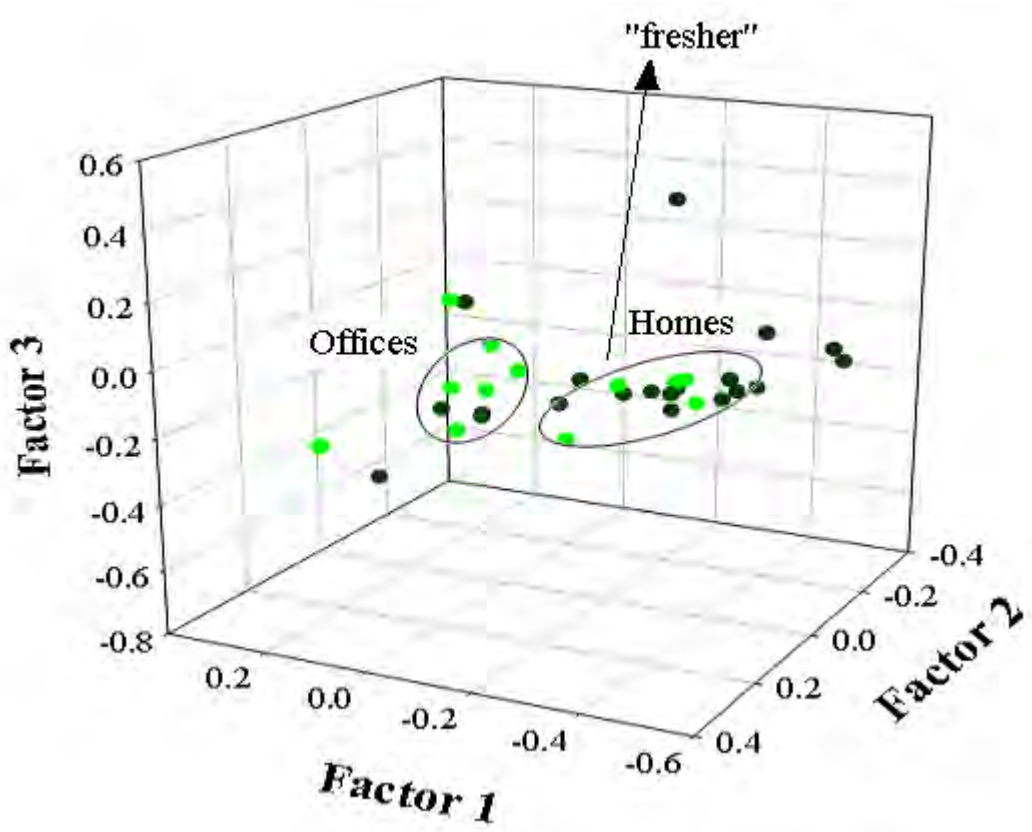


Figure 11 PCA of home (dark green) and office (light green) air samples, with variance represented by factor 1 (27%), factor 2 (18%) and factor 3 (16%).

The results from this study remain within the lower range of concentrations measured in home samples from Canada (290 – 4000 pg m^{-3}) (Shoeib *et al.*, 2004, 2005) and Europe (100 – 83 000 pg m^{-3}) (de Voogt *et al.*, 2008). The upper ranges measured by the first Canadian study are within the ranges of values measured from this study for MeFOSE and EtFOSE (77 – 3100 pg m^{-3} and 80 – 1900 pg m^{-3}, respectively) for both homes and offices. Comparisons of this study to a European study (de Voogt *et al.*, 2008) reveals that the concentrations of MeFOSE (max = 83 000 pg m^{-3}) and EtFOSE (max = 29 000 pg m^{-3}) are much higher (25 times and 15 times, respectively)than from the UK (MeFOSE = 3100 pg m^{-3} and EtFOSE 1900 pg m^{-3})compared to Tromso values.

Table 42 Comparison of indoor air samples from other countries (pg m^{-3}), ([a] range of sample concentrations)

Reference	Microenvironment	MeFOSE (pg m^{-3})	EtFOSE (pg m^{-3})	MeFOSA (pg m^{-3})	EtFOSA (pg m^{-3})
This study	Homes, n = 45	950 (77 - 3100)[a]	650 (80 – 1900)[a]	2 (0 -19) [a]	120 (14 - 350)[a]
	Offices, n = 20	480 (38 – 1200)[a]	490 (180 - 950)[a]	6 (0 - 50) [a]	58 (12 - 150)[a]
	Old Lab, n = 1	1698	1917		
Shoeib *et al.*, 2004	New Lab, n = 1	11	4.8		
	House 1, n = 1	670	360		
	House 2, n = 1	1800	990		
	House 3, n = 1	4000	930		
	House 4, n = 1	1500	290		
Shoeib *et al.*, 2005	Home, n = 59	370 – 8200[a]	230 – 7700[a]		
Jahnke *et al.*, 2007a	Office (50 m^3), n = 1	730	310		190
	Office (20 m^3), n = 1	800	820		160
de Voogt *et al.*, 2006	Indoor (gaseous), n = 4	232 – 83 000[a]	2200 – 29 000[a]	<120 - 6600[a]	<100 - 6600[a]
	Indoor (particulate), n = 4	35 – 1300[a]	< 30 –120[a]	< 5 – 6[a]	< 5 – 8[a]

		PFOS (pg m^{-3})	PFOA (pg m^{-3})	PFHxS (pg m^{-3})	FOSA (pg m^{-3})
This study	Homes, n = 20	38 (0 – 400)[a]	52 (0 – 440)[a]	36 (0 – 220)[a]	150 (16 – 1300)[a]
	Offices, n = 12	56 (12 – 89)[a]	58 (0 – 200)[a]	95 (0 – 330)[a]	74 (4 - 310)[a]
de Voogt *et al.*, 2006	Indoor (particulate), n = 4	>LOQ - 47[a]	3.4 – 6.9[a]	< 4.1[a]	< 1 – 3.5[a]

3.3. INDOOR DUST CONCENTRATIONS

Indoor dust samples were collected from homes (*n* = 45), offices (*n* = 20) and cars (*n* = 20) within the West Midlands, UK. Dust was collected as described in chapter 2. Generally domestic dust samples were collected from living rooms. However, in some cases, samples were collected from bedsits. The offices sampled ranged from closed single occupant office spaces, to open plan offices for up to 10 people. As described above for air samples, offices generally contained more electrical goods and foam chairs than homes, but fewer textiles and other consumer goods. The car samples varied greatly depending on the age and manufacturer of the car. There was a range across the samples from a 15 year old, 4 seater car, without carpet and a single analogue radio to a large people carrier, 2 years old, carpeted and upholstered throughout, and fitted with a DVD player, satellite navigation device, and digital stereo system. The differences are expected to provide large variance in compound detection and quantification.

Individual sample results and descriptive statistics for the three types of microenvironments are displayed in Table 43, Table 44, and Table 45. Results indicate the presence of PFCs in all samples, with PFOS (7400 ng g^{-1}), PFOA (6000 ng g^{-1}) and PFHxS (6100 ng g^{-1}) present at some of the highest concentrations reported in this study as a whole. The persistent PFCs, which are also degradation products of the more volatile PFCs, are the predominant perfluorinated contaminants measured in dust, owing at least in part to their low vapour pressures and consequent preferential partitioning to dust. The more volatile compounds, EtFOSA, FOSA, MeFOSE and EtFOSE also had a high detection rate in the samples (> 80 %), whilst MeFOSA quantification was similar to the air samples, with sporadic detection in samples, leading to an overall < 40 % detection rate for the compound.

Table 43 UK home dust concentrations (ng g^{-1})

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFCs
1	160	240	610	<DL	26	300	350	19	1700
2	1400	370	370	58	400	210	39	33	2900
3	95	500	220	11	23	6	98	440	1400
4	84	300	320	110	170	110	140	550	1800
5	92	470	290	<DL	9	5	470	47	1400
6	60	450	610	<DL	110	21	180	110	1500
7	630	320	150	92	50	32	100	21	1400
8	850	470	210	<DL	<DL	16	42	34	1600
9	490	520	330	<DL	<DL	23	<DL	25	1400
10	300	230	230	<DL	40	<DL	22	200	1000
11	73	400	21	<DL	840	15	240	<DL	1600
12	120	46	1500	<DL	<DL	<DL	93	1	1800
13	3	<DL	56	<DL	<DL	23	250	370	700
14	580	99	380	<DL	160	<DL	150	1020	2400
15	22	23	32	<DL	29	14	3	5	130
16	51	24	69	<DL	49	24	<DL	<DL	220
17	25	28	41	<DL	41	7	5	<DL	150
18	25	15	61	<DL	54	6	<DL	13	170
19	110	63	600	<DL	43	160	30	5	1000
20	28	43	650	<DL	38	160	31	25	970
21	33	35	67	<DL	100	95	27	32	390
22	14	74	240	64	25	20	4	41	480
23	510	250	170	<DL	11	22	17	380	1400
24	47	13	95	<DL	9	9	4	29	210
25	72	49	34	13	39	<DL	9	8	220
26	600	360	8	<DL	570	15	610	330	2500
27	180	80	140	<DL	10	80	10	17	520

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFCs
28	1200	430	210	<DL	310	240	520	900	3800
29	260	380	170	58	45	150	240	1100	2400
30	120	420	210	94	270	290	440	190	2030
31	78	22	250	22	100	42	11	9	540
32	39	17	49	32	63	<DL	360	8	570
33	82	4	130	<DL	<DL	12	<DL	<DL	230
34	380	85	430	<DL	29	43	400	270	1600
35	170	160	84	<DL	54	<DL	140	15	620
36	200	190	92	<DL	37	<DL	120	16	650
37	160	220	190	<DL	100	10	79	36	800
38	580	240	340	<DL	46	<DL	<DL	180	1500
39	210	250	350	5	69	<DL	54	30	970
40	7400	1500	6100	20	250	<DL	2500	2000	20 000
41	130	4100	770	<DL	130	37	1800	400	7300
42	140	270	2600	<DL	12	24	20	3900	7000
43	230	120	290	11	6	<DL	490	890	2000
44	1300	190	180	<DL	4	88	170	320	2300
45	960	130	230	<DL	34	150	270	360	2100
Minimum	3	<DL	8	<DL	<DL	<DL	<DL	<DL	130
5th Percentile	23	13	32	<DL	<DL	<DL	<DL	<DL	180
Median	140	190	210	<DL	41	20	93	34	1400
Arithmetic Mean	450	310	450	13	98	54	230	320	1900
95th Percentile	1300	520	1400	86	380	230	590	110	6400
Maximum	7400	4100	6100	110	840	300	2500	3900	20 000
SD	1100	620	970	28	160	80	460	680	3100
%RSD	250	200	220	210	170	150	200	210	160

Table 44 UK office dust concentrations (ng g^{-1})

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFCs
1	110	460	110	<DL	11	<DL	250	390	1300
2	670	130	130	<DL	1	<DL	920	2600	4500
3	950	160	460	<DL	18	<DL	350	90	2000
4	860	330	390	<DL	8	42	150	640	2400
5	27	410	6	<DL	11	17	470	240	1200
6	73	460	21	<DL	840	15	290	<DL	1700
7	43	380	2800	33	680	23	350	51	4400
8	870	370	5700	34	<DL	<DL	35	<DL	7000
9	26	270	680	35	<DL	<DL	150	100	1300
10	1000	660	380	<DL	46	23	420	11	2600
11	670	6000	180	19	<DL	14	190	<DL	7100
12	61	35	66	<DL	140	3	40	87	430
13	92	110	170	<DL	5	<DL	8	12	400
14	20	99	240	50	38	1	32	3	480
15	81	<DL	170	<DL	13	<DL	3	3	270
16	420	660	78	31	140	130	470	180	2100
17	430	78	480	21	32	<DL	2	500	1500
18	500	20	38	<DL	190	130	360	540	1800
19	350	300	56	<DL	<DL	23	550	370	1600
20	76	19	160	1000	170	<DL	<DL	32	1500
Minimum	20	<DL	6	<DL	<DL	<DL	<DL	<DL	270
5th Percentile	26	18	20	<DL	<DL	<DL	2	<DL	390
Median	230	280	170	<DL	15	2	220	88	1700
Arithmetic Mean	370	550	620	61	120	21	250	290	2300
95th Percentile	960	930	2900	97	690	130	570	740	7000
Maximum	1000	6000	5700	1000	840	130	920	2600	7100
SD	390	1300	1300	220	230	38	240	590	2000
%RSD	93	240	220	360	200	180	96	200	84

Table 45 UK car dust concentrations (ng g^{-1})

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFCs
1	130	14	100	<DL	28	<DL	<DL	<DL	270
2	52	29	120	<DL	17	<DL	12	<DL	230
3	81	350	240	<DL	140	47	260	140	1300
4	360	170	180	<DL	78	<DL	<DL	21	810
5	590	320	720	<DL	15	14	490	460	2600
6	430	270	290	<DL	<DL	12	320	130	1500
7	1500	6	2400	<DL	<DL	<DL	120	120	4100
8	20	51	240	<DL	370	17	330	200	1200
9	500	370	380	<DL	180	<DL	190	370	2000
10	51	<DL	46	<DL	18	68	15	33	230
11	110	13	70	14	14	130	220	8	580
12	41	15	260	<DL	22	<DL	52	11	400
13	210	21	130	<DL	11	1900	19	16	2300
14	49	12	400	9	140	9	1	110	730
15	84	84	45	130	140	16	41	150	690
16	560	85	70	7	190	150	67	45	1200
17	24	56	150	5	250	12	200	55	750
18	78	81	160	3	190	150	33	58	750
19	82	99	180	<DL	23	120	130	36	670
20	200	74	350	<DL	52	130	96	55	960
Minimum	20	<DL	45	<DL	<DL	<DL	<DL	<DL	230
5th Percentile	24	6	46	<DL	<DL	<DL	<DL	<DL	230
Median	97	65	180	<DL	40	15	82	55	780
Arithmetic Mean	260	110	330	8	94	140	130	100	1200
95th Percentile	640	350	800	20	260	240	340	380	2700
Maximum	1500	370	2400	130	370	1900	490	460	4100
SD	340	120	520	30	100	430	140	120	960
Relative SD (%)	130	120	160	350	110	300	110	120	83

A comparison of the indoor microenvironment dust sample arithmetic mean dust concentrations are displayed in Figure 12. The ∑PFC concentrations are highest in office environments (2300 ng g^{-1}), followed by homes (1900 ng g^{-1}), yet concentrations from car environments were about half of the other two microenvironments (1200 ng g^{-1}).

All indoor dust concentrations revealed positively skewed data ($W < 0.885$, $p < 0.022$), when subjected to the Shapiro-Wilk test for normality of data distribution; therefore further statistical analysis was conducted on normalised (log-transformed) data. The arithmetic mean concentrations for each of the individual compounds and for ∑PFC in each microenvironment were compared, and significant differences were identified for FOSA in cars . The car dust samples reveal a significant difference ($p < 0.001$) when analysed (using the normalized data) via ANOVA for FOSA in car dust compared to both home and office dust. FOSA is a common intermediate degradation product of MeFOSE, EtFOSE (Plumlee *et al.*, 2009) and other more volatile PFCs such as FTOH (Ellis *et al.*, 2004).

Despite no other significant differences being identified between the microenvironments, trends are still noticeable within the data; this is particularly true of the ratios of PFOS:PFOA from the three dust microenvironments. In car and home dust, PFOS (mean, 450 ng g^{-1}, 260 ng g^{-1}, respectively) is more dominant; whereas PFOA (mean 550 ng g^{-1}) is more prevalent in office dust. The presence of PFOA at higher concentrations could be indicative of the presence of precursor compounds such as FTOHs and PFOSEs (Jahnke *et al.*, 2007b, D'Hollander *et al.*, 2010) in office equipment (Wallington *et al.*, 2006).

For all compounds with the exception of FOSA, the car samples display the lowest mean concentrations (∑PFC = 1200 ng g^{-1}). Vehicle concentrations are not

significantly different from the other microenvironments, but are generally less contaminated. However, the presence of FOSA in car samples (mean = 140 ng g^{-1}) indicates opposite behaviour, with concentrations almost 3 times greater than in homes (mean = 54 ng g^{-1}), and up to 7 times greater than offices (mean = 21 ng g^{-1}). FOSA in car samples contributes (on average) 10 % of the ∑PFC concentration, whilst in homes and offices this contribution, on average, is < 5 %. The removal of sample 13 (an outlier) from the analysis, the average contribution still remains above that of offices and homes (at 7 %). The presence of FOSA in cars is likely to originate from similar sources as other indoor environments, treated upholstery, leather treatments, electronic insulating wires etc. PFCs are also combined with many lubricants and surfactants used in the engine and mechanical parts (Drobny, 2005). The car samples containing concentrations of FOSA were compared to their questionnaire data (see appendix), and no significant relationships were identified.

In comparison to concentrations found in dust samples from other countries (Table 46) the concentrations within this study are broadly similar to those reported in other studies. Comparisons of dust concentrations across different studies is complicated by various factors including analytical and sampling techniques, measurement of particulate and / or gaseous phase and identity of PFCs monitored. These parameters include whether the entire room, or a smaller area was sampled, or entire floor, or surface top dust was collected. The dust particle size range analysed is also an important parameter, as a negative relationship was identified between the particle size and the presence of PFCs by Fromme *et al.,* (2008). Other parameters that would affect the results include the analytical methods employed. However, appropriate analytical QA/QC minimises the influence of this latter factor. Concentrations from the UK (this study) span a greater range for PFOS (3 – 7400 ng g^{-1}) and PFOA (<DL -

4100 ng g^{-1}) in homes compared to other studies (displayed in Table 46). Concentrations from the UK (this study) span a greater range for PFOS (3 – 7400 ng g^{-1}) and PFOA (<DL - 4100 ng g^{-1}) in homes compared to other studies (displayed in Table 46). Removal of the extreme value for PFOS in sample 40 (7400 ng g^{-1}) resulted in UK house dust PFOS concentrations remaining within the same range of those reported for Swedish, North American and Japanese homes (Björklund *et al.*, 2009, Kato *et al.*, 2009, Kubwabo *et al.*, 2005 and Moriwaki *et al.*, 2003). Despite the range of data being

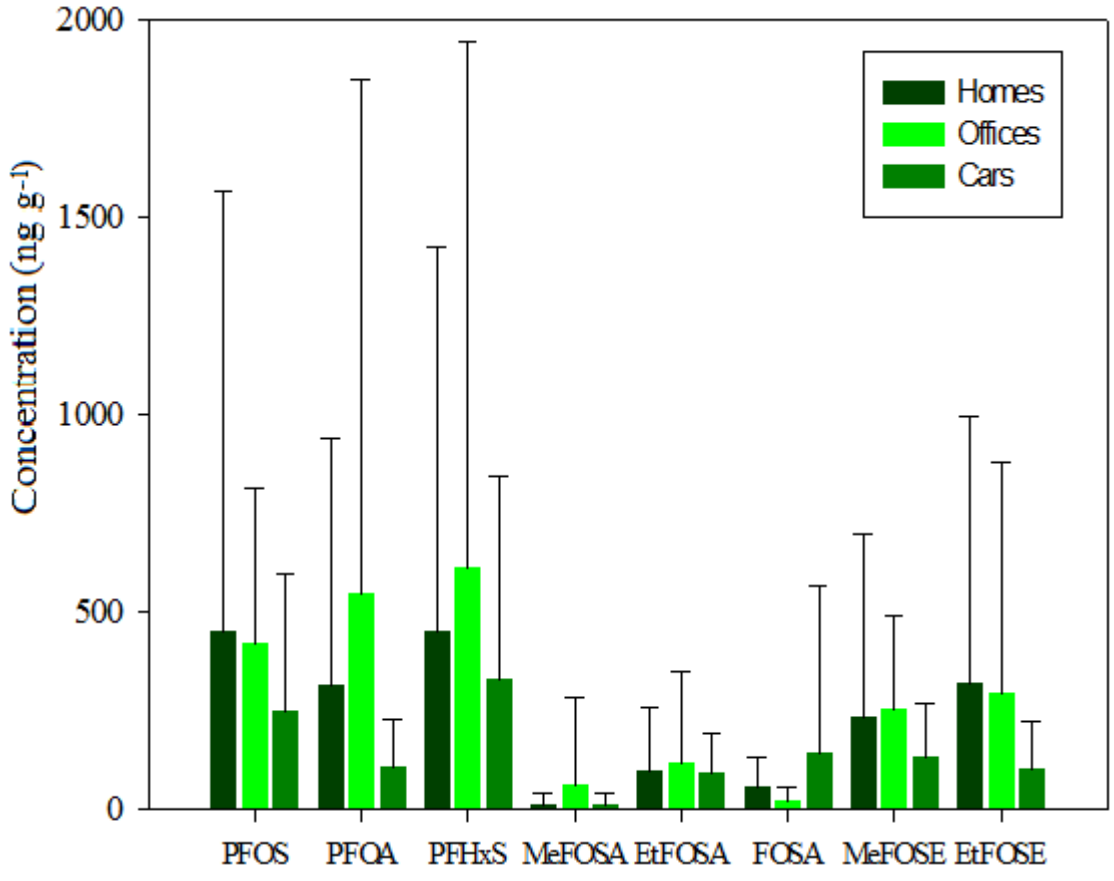


Figure 12 Comparison of arithmetic mean concentrations of PFCs detected in home, office, and car dust (ng g^{-1}, with error bars representing standard deviation)

similar to Sweden, the UK mean concentration considerably exceeds the Swedish results for PFOS and PFOA (Björklund *et al.,* 2009) suggesting UK homes are more contaminated with PFCs.

The UK mean concentrations remain between results seen from two studies in the USA (Strynar *et al.*, 2008 and Kato *et al.*, 2009). Work by Kato *et al.* (2009) indicates USA PFC concentrations remain below that of the UK, but indicate similar concentrations for MeFOSA, where the majority of samples are below the LOQ values, with 29 % and 10 % of UK and USA samples containing quantifiable concentrations.

UK EtFOSA concentrations (98 ng g^{-1}) are lower than those reported for the USA (201 ng g^{-1}), (Kato *et al.*, 2009), but are well above those reported for Canadian homes, in which EtFOSA was always not detected (Shoeib *et al*., 2005). The differences could be attributed to different use patterns of PFCs within these regions.

MeFOSE and EtFOSE concentrations measured by Shoeib *et al*., (2005) both exceed the range of concentrations identified in the UK, by 3.5 and 20 times for MeFOSE and EtFOSE respectively. This suggests that these two compounds have additional sources within Canadian homes. By comparison, UK concentrations display similar results to a USA study by Kato *et al.,* (2009) for FOSA, MeFOSE and EtFOSE, suggesting that the increased concentrations seen in Canadian homes are not indicative of the whole North American region.

The UK dust concentrations analysed via PCA, along with classroom concentrations, are discussed in chapter 7, and home dust samples will be compared to other international samples collected and analysed according to the same protocol in chapter 9.

Table 46 Comparison of mean dust concentrations (and ranges) from this study and other international dust studies (ng g^{-1}).

Reference	Type	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
This Study	Homes,	450	310	450	13	98	54	230	320
	(n = 45)	(3 – 7400)	(<DL – 4100)	(8 – 6100)	(<DL – 110)	<DL – 840)	(<DL – 300)	(<DL – 2500)	(<DL – 3900)
	Offices,	370	550	620	61	120	21	250	290
	(n = 20)	(20 – 1000)	(<DL – 6000)	(6 – 5700)	(<DL – 1000)	(<DL – 840)	(<DL – 130)	(<DL – 920)	(<DL – 2600)
	Cars,	260	110	330	8	94	140	130	1
	(n = 20)	(20 – 1500)	(<DL – 370)	(45 – 2400)	(<DL – 130)	(<DL – 370)	(<DL – 1900)	(<DL – 490)	(<DL – 460)
Kubwabo *et al.* 2005	Homes,								
	(n = 67)	2.0 - 5100	1 - 1200	2.0 - 4300					
Moriwaki *et al.*, 2003	Homes,								
	(n = 16)	11.0 - 2500	69 - 3700						
Kato *et al.*, 2009	Homes,	48	97	190	< DL	200	<DL	220	180
	(n = 39)	(32-18 000)[a]	(<DL – 9800)[a]	(48 – 44 000)[a]	(<DL - 216)[a]	(86 - 4000)[a]	(<DL - 180)[a]	(65 - 3200)[a]	(<DL – 12 000)[a]
Strynar *et al.*, 2008	Homes,								
	(n = 112)	760[a]	300[a]	870[a]					
Nakato *et al.*, 2007	Homes,								
	(n = 20)	7.0 - 41	18 - 89	n.d. – 5					
Shoeib *et al.*, 2005	Homes,								
	(n =59)					<DL		3 - 8900.	1 – 75 000
Fromme *et al.*, 2008	Homes,								
	(n = 12)	3 - 340	2 - 140						
Björklund *et al.*, 2009	Homes,								
	(n = 48)	8 - 1100	15 - 850						
	Offices,								
	(n = 10)	29 - 490	14 - 510						
	Cars (n = 5)	8.0 - 33	12.0 - 96						

[a] 25th percentile – maximum concentration

3.4. COMPARISON OF UK DUST AND AIR SAMPLES

In contrast to air samples, the dust samples display a predominance of the less volatile compounds; PFOS, PFOA and PFHxS, closely followed by contributions from more volatile compounds PFOSEs (5 – 20 %) and a minor impact from PFOSAs (< 5 %). These relationships, seen in both the home and office environments, are expected because of the basic physicochemical properties of the individual compounds.

However, the presence of the more volatile compounds within the dust samples, in some cases contributing up to 20 % of ΣPFC, is likely to derive from the presence of micro-fibres within the dust still retaining the compounds, or the compounds still remaining bound to the product as a coating. The presence of PFOS, PFOA and PFHxS is more common in the dust because of their low volatilities and persistences which allow concentrations to accumulate over time.

The contributions of individual compounds (arithmetic mean values) were expressed as percentages of ΣPFC (arithmetic mean values) to compare the distribution of PFCs within the air and dust samples for home and office microenvironments and are represented in Figure 13 and Figure 14, respectively. Both figures indicate that the air samples are dominated by the presence of MeFOSE and EtFOSE, with additional substantial contributions from PFOSAs compared to dust samples, with small contributions from the more stable and less volatile PFOS, PFOA and PFHxS. The presence of these less volatile compounds is likely to derive from the presence of suspended dust particles.

Despite their greater presence in air samples, the 3 PFOSAs contribute very little to ΣPFC in both air and dust samples (14 % and 17 %, respectively). The is likely because they are an intermediate degradation product of MeFOSE, EtFOSE and other

more volatile compounds, which readily degrade to PFCAs and PFASs. The manufacturing process is also unlikely to produce PFOSAs as residues, so this is an unlikely source to indoor environments. By comparison, PFOSEs, PFAS and PFCAs have multiple sources, as well as degradation pathways. Generally, the degradation of the more volatile compounds results in the formation of PFOSAs which are produced as intermediates (Plumlee *et al.*, 2009).

Hence, PFOS, PFOA, and PFHxS are expected to contribute more significantly to ∑PFC in the dust, as they can be emitted via direct release from the product and as a terminal product of degradation of many more volatile and more widely commercially used compounds (Vestergren *et al.*, 2008).

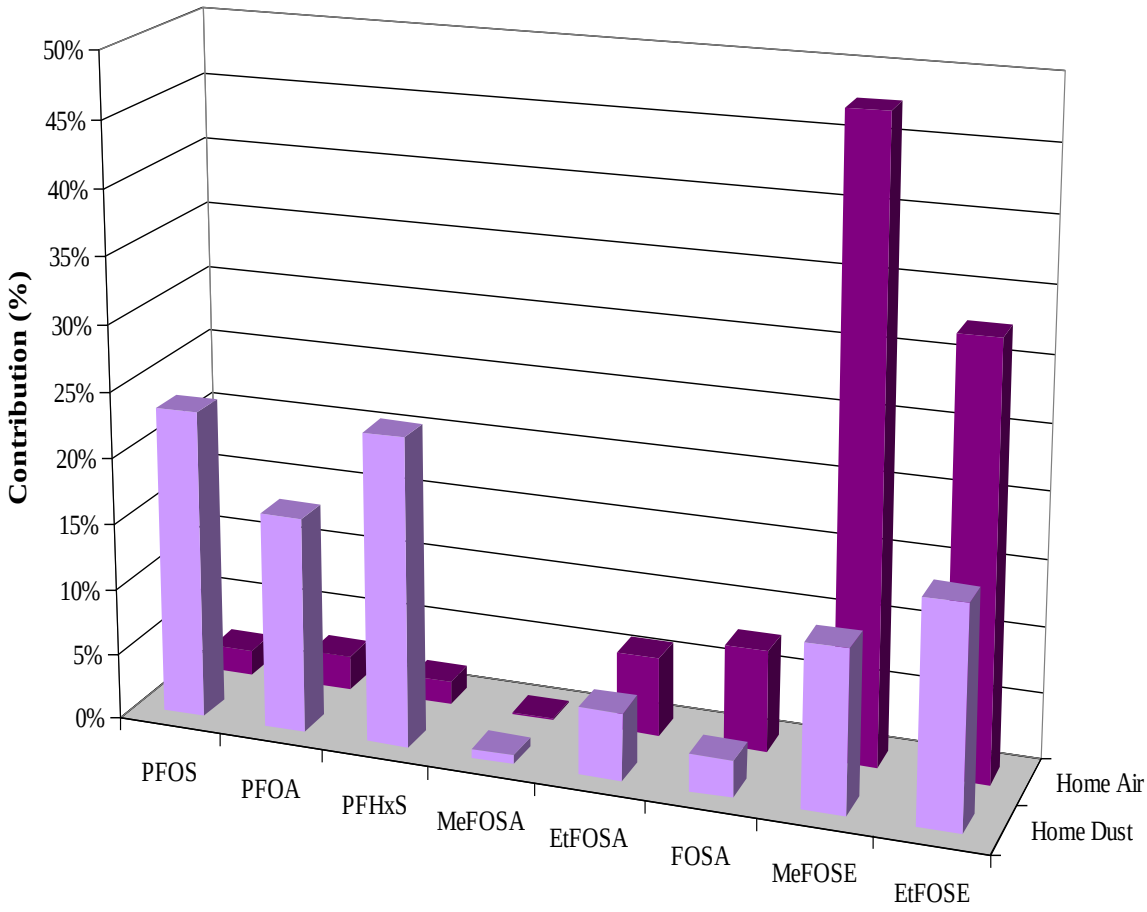


Figure 13 Comparison of contributions (%) to ∑PFC from individual compounds for both home dust and home air arithmetic mean concentrations.

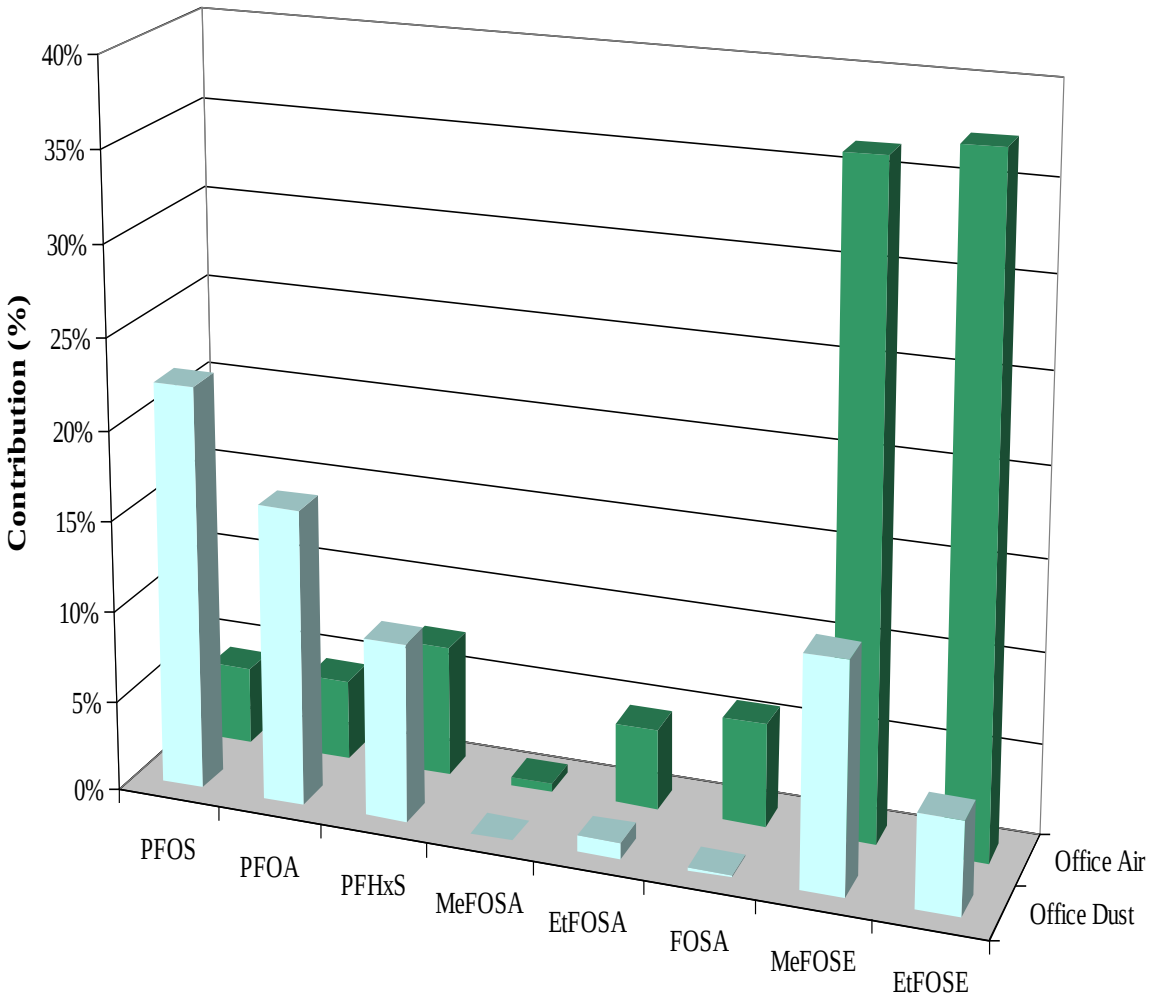


Figure 14 Comparison of contributions of individual compound arithmetic mean concentrations to ∑PFC concentrations for both office air and office dust.

The data for the 5 sample sets (home dust, office dust, car dust, home air and office air) were weighted by comparison to the ΣPFC concentration and analysed using PCA. The air samples were transformed using the vapour pressure values to better compare the results with the dust samples. The factor scores for the test are displayed in Table 47, and represent 72 % of the total variance. Results have been displayed in Figure 15, and indicate a distinct split in the data when observed for factor 2 and 4, arranging the samples into two separate groups. The groups are primarily influenced by the presence of EtFOSA, PFOS and PFOA in the samples, leaving the first group with negative factor 2 scores and the second group displaying positive factor 2 scores.

The scores are separated into the two categories because of influences from EtFOSA aerosols and dust particulate concentrations of PFOS and PFOA. Group 1 contains a large percentage of the home air, office air, and office dust and less significant presence of car and home dust, which are mainly located in group 2. The PCA depicts the presence of PFOSEs, FOSA and PFHxS to be the more prominent compounds in offices, whilst homes appear to show different signatures for the air and dust. Home environments have both scenarios present, with some homes sampled containing PFOSEs as the dominant compounds in dust, whilst others contain PFOS and PFOA as the dominant compounds. This may be related to the freshness of the signal, with the older the source the greater the amount of degradation products PFOS and PFOA, and fresher sources to home dust creating stronger contributions to the PFOSE concentrations. This pattern of behaviour is also noted with the office dust samples, where the majority of the dust is located in group 1 (fresher sources) whilst a few samples remain within group 2 (older sources). A previous studies by Fraser *et al.* (2010) indicates the presence of more volatile compounds in office air, whilst a second by D'Hollander *et al.*, (2010) detected a dominance of PFOA in the dust. Thus these two studies show similar results to these, where a large number of the office dust samples and air contain the more volatile compounds, but a few samples how the opposite behaviour with more dominant contributions from PFOS and PFOA.

The car dust fits between these two groups with some samples indicating a presence of PFOSEs, and a few with the presence of PFOS and PFOA. However, a large number of the car samples are driven to higher factor 4 values by the dominance of FOSA, thus locating the car samples outside of these two distinct groups. This relationship is more prominent in the second graph (Figure 15), which depicts factor 3 and factor 4. The majority of samples remain within a centralised region, both the

home air and dust and the office air and dust samples, but the car samples remain generally outside of this central area, and do so because of the high FOSA concentrations noted in the samples in comparison to the other environments. The car samples are not alone, and indicate that within homes and offices there is a large amount of variation between one sample and another, and the particular dominant compound within that home or office. However this second graph indicates that there are few distinct differences between the air and dust matrices found in homes and offices.

Table 47 PCA factor loadings

Compound	Component			
	1	2	3	4
PFOS	0.123	0.373	-0.361	0.011
PFOA	-0.229	0.579	0.072	-0.140
PFHxS	0.625	-0.312	-0.078	-0.154
MeFOSA	0.036	-0.196	0.546	-0.221
EtFOSA	-0.106	0.146	0.608	0.173
FOSA	-0.012	-0.049	-0.021	0.878
MeFOSE	-0.378	-0.076	0.006	-0.062
EtFOSE	-0.175	-0.244	-0.190	-0.176

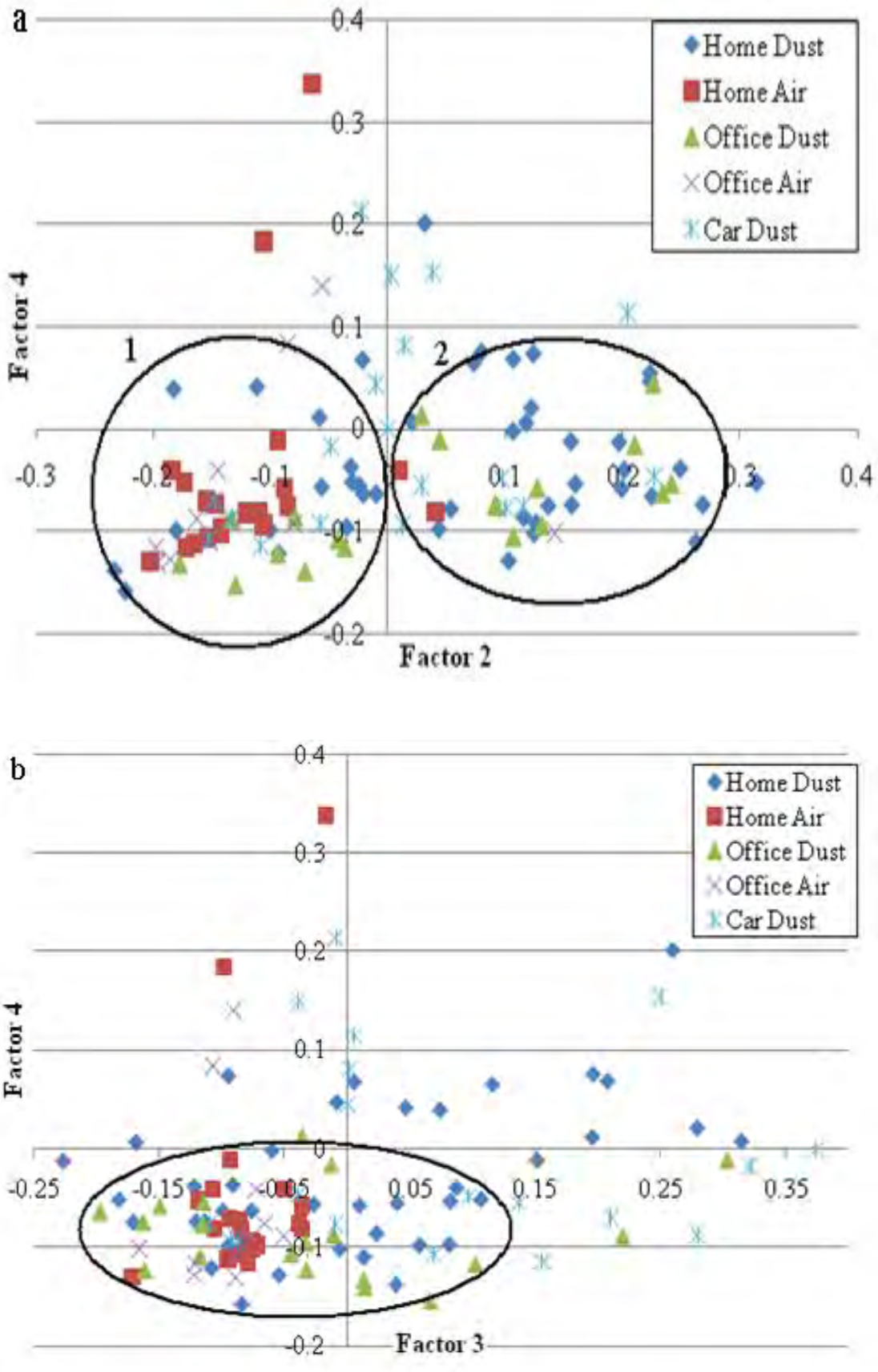


Figure 15 PCA 2D plots a, factor 2 vs. factor 4, b, factor 3 vs. factor 4.

3.5. SUMMARY AND CONCLUSIONS

This study of UK indoor microenvironments has shown that PFCs are present at measurable concentrations in both air and dust from homes, offices, and cars. This indicates that UK indoor environments can act as a reservoir to PFCs via both air and dust matrices to a quantifiable level. Presences of PFCs in these UK environments are similar in concentration to international measurements and indicate that there could be a participating contribution to human exposure (Fromme *et al.*, 2009).

Air samples are seen to contain primarily MeFOSE and EtFOSE (30 – 50 % of ∑PFC), dust samples are dominated by either PFOS and PFHxS (15 – 25 % of ∑PFC) of the PFOSEs (> 60 %), but contain a wide contribution from all 8 PFCs. The office dust matrix contained the greatest concentration of ∑PFC in dust (but not significantly), whilst home air concentrations exceeded those in offices. This may be a result of the increased ventilation and cleaning frequency (usually daily) within offices. Greater cleaning frequency can lead to less dust, and finer material in the overall total weight, which in turn can lead to a greater PFC loading within the sampled dust (Harrad *et al.*, 2009 and Kubwabo *et al.*, 2005). Office and home dust samples showed no significant differences in PFC concentrations, but, air samples indicated that EtFOSA and MeFOSE were significantly different.

Car samples contained the lowest concentrations of the 8 PFC monitored, with the lowest ∑PFC concentrations. However, while concentrations of the persistent compounds PFOS, PFOA and PFHxS are not significantly different from those in homes and offices, those of MeFOSE and EtFOSE are significantly lower in cars and FOSA is significantly higher. This distinct signature for cars has not been noted (as far as the author is aware) previously and can be considered as a signature compound for these microenvironments. This presence of FOSA may be attributable to greater

use of volatile compounds present in stain proofing chemicals applied to the upholstery (3M 2000, Danglasan-Panililio *et al.,* 2006). Alternatively, these precursor compounds could have undergone preferential photolytic degradation over time in cars due to the "greenhouse" conditions that exist in vehicle cabins.

To summarise, all the indoor microenvironments monitored within this chapter are applicable to act as reservoirs to PFCs, and continue to do so in the air and dust matrices. The data demonstrates that whilst indoor environments can act as reservoirs to these chemicals, there are distinct differences between each type of microenvironment, and thus the sources and pathways can be variable for not only each type but also individual microenvironments. The sources of PFCs to these matrices could not be determined from correlations with room contents, but are thought to be numerous, as a result of the wide use of the compounds, and for their previous unrestricted incorporation in consumer goods. The reservoir of PFCs within indoor environments also appears to be variable between the more volatile compounds and the less volatile ones, which is thought to occur from the freshness of the sources. In many of the environments containing newer products the dominant PFCs present tended to be the more volatile compounds.

The presence of PFCs in indoor air and dust suggests that human exposure is possible to occur from within indoor environments and could constitute an importantpathway for some individuals. The magnitude of such indoor exposures is examined in chapter 6. However, it is clear that each microenvironment measured is capable of delivering a different dose of PFC to each individual and thus microenvironments cannot be easily categorised and quantified as a single entity. Different contents, use, occupation and many other parameters, all affect the concentrations within rooms, and the presence and partitioning of PFCs into both air and dust.

4. INDOOR TEMPORAL AND SPATIAL VARIATIONS

The hypothesis addressed in this chapter is that there is substantial intra-room spatial and temporal variability in concentrations of PFCs in indoor dust. Stemming from this, this chapter's aim is to monitor such spatial and temporal variability in a number of indoor microenvironments. These data will be assessed both for the impact of the observed variability upon human exposure, and - in conjunction with data on room contents – for potential insights into source attribution.

This chapter studies intra-room spatial and temporal variability in concentrations of PFCs in dust within a number of indoor microenvironments. This is achieved by taking monthly samples of dust at various points within the same indoor microenvironments. Such variability has been demonstrated previously for BFRs in indoor dust and its implications for source attribution and human exposure highlighted (Harrad et al, 2008; Harrad et al, 2009). With reference to the latter, substantial within-room spatial variation can impact on human exposure assessment if the area sampled does not correspond to the most-frequented area, thereby reducing the "biological relevance" of the sample obtained (Harrad et al, 2010).

4.1. INTRODUCTION

An assessment of spatial and temporal variations (for dust and air) was conducted within selected indoor microenvironments. Spatial variations in indoor air concentrations were not monitored because of the general mixing and homogeneity of air within a room created by the ventilation, thermal mixing and turbulent mixing created by occupants in the room.

Currently, very little is understood regarding the variability of PFCs spatially and temporally. The presence of PFCs in the environment and within indoor environments (occupational and personal) has only been addressed within the last decade as a result of analytical instrumental improvements. There are still many unanswered questions relating to the behaviour and transformations of PFCs, and also their toxicity and exposure to humans. Estimates and generalisations of PFC behaviour can be assumed from the understanding of traits of other halogenated POPs, such as polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs). It is understood that PFCs share the same basic behavioural traits of other POPs, with movement in the environment towards cooler regions (Jones & de Voogt, 1999), with the pathway and movement dependent upon the volatility of the individual PFC and the ability to 'hop' through the environment (Shoeib *et al.,* 2006). However, the partitioning behaviour of PFCs varies from that of other POPs because of their differing physicochemical properties, (Arp *et al.,* 2006).

The mechanisms of movement from treated goods into the indoor environment are not directly known, but are thought to include general wear and tear of products, leaching from and degradation of the products (as seen with brominated flame retardants, ATSDR 2004). The effects of wear and tear from walking and vacuuming treated carpets can result in 50 % loss of PFCs over a 9 year period (3M, 2000). Additional sources are known to occur directly from the use of PFC containing sprays (surfactant cleaning products, paints, stain repellent carpet sprays, etc). When such products are used, only 44 % reaches the intended surface and the rest is lost to air (3M, 2000). Moreover, a 73 % loss of PFCs from treated clothing is estimated over a 2 year period, with the loss primarily via washing of the garment (3M, 2000), suggesting that the perfluorinated compounds are easily removable and not chemically bound to the

garments. Sources of PFCs have been linked (in chapter 3) to the presence and activity of occupants in the room, but sources could also be related to the clothing of the occupant. Sources of contamination to a room are numerous for PFCs and they exist on stationary objects (such as furnishings and carpet), as well as on other transported objects (such as clothing, food packaging etc). People are more likely to be affected on an individual basis within a room depending upon their activity level and behaviour. This is different for other organohalogens, where exposure can be proportional to the proximity of certain fixed objects like a TV, and which is the case for hexabromocyclododecanes (HBCDs) (Harrad *et al.,* 2009). Current research gaps noted by Harrad *et al.,* (2010b) regarding the presence of PFCs indoors include the pathways of less volatile PFCs to indoor environments, the presence of localised 'hot spots' occurring within rooms and the potential sources creating these, and emission rates from treated goods.

Due to the sampling format used for dust collection (point sampling), the impact of 'hot spots' could produce an over- or under-estimate of the concentration from the whole room. It is unlikely that the dust ingested by the occupant will be derived from the whole room content, but more accordingly from a small region from within close proximity to the occupant. Therefore, a more accurate measure of the dust ingested by an occupant may be procured via sampling the dust from a well-frequented space, to correctly estimate the 'biologically-relevant' (Allen *et al.,* 2008) dust in the room. This dust is located in the area of the room that is used regularly and would be located where people spend a high proportion of their time.

Both spatial and temporal within-room variations in PFCs concentrations could significantly impact the output of the exposure assessment (which is conducted in chapter 4). Understanding the ability of the sample to provide a precise representation

of the 'biologically relevant' dust concentration from the entire microenvironment will be determined by the significance of the variability (Harrad *et al.*, 2010b).

Point sampling is a quick and relatively non-intrusive way of collecting a sample providing a snap shot of the internal environment (Harrad *et al.*, 2010b). The sample is collected from a centrally, well frequented area of the room, where the dust is more likely to be ingested – making it 'biologically relevant'. Dust in other areas of a room may not be as likely to be ingested because of the lack of time spent in that area by occupants, hence contact with dust is reduced. Thus, inaccessible areas, such as corners, behind and under furniture were not sampled. Due to multiple uses of most rooms sampled, it is imperative to determine the relevance of this point sample for the entire room. Sampling of different areas of rooms, may also allow sources (contributing objects) to be identified.

There are many parameters within a room, which vary temporally, and are usually linked to seasonal variations including temperature, humidity, ventilation, and activity level. Human behaviour patterns also change with season; during warmer months (spring and summer) people tend to spend more time outdoors, rather than in their homes and this has been linked to more in-tread from shoes (Norra & Stuben, 2004). In addition, rooms are better ventilated during such periods. Changes to temperature, ventilation, humidity and use patterns of a room all vary seasonally, and can produce differences in source emissions, partitioning between particulate and gaseous phase and mixing within the room (Stock *et al.*, 2005).

If within-room spatial variability of dust is significant, the possibility of one person receiving greater exposure than another is possible. This is of particular interest for young children, because of the amount of time they spend on the floor, and their rate of dust ingestion. Localised exposure can be created by short-range spatial differences

in sources of perfluorinated compounds, such as placement of furnishings and textiles, along with variations across the room caused by ventilation and drafts, heating and humidity, and general wear and tear rates of flooring. Variations in localised exposure are not only dependent upon the room characteristics, but also as a result of the occupant's activity and behaviour.

Temporal variations could also influence exposure estimates. Each sample represents a single period of time, and thus variations identified in indoor air concentrations (in chapter 3) associated with variations between microenvironments and buildings could in part be a result of variations in sampling collection dates.

Processes affecting concentrations within indoor environments include the cleaning processes employed and their frequency, room use and changes in use over the year, ventilation and draughts, and changes in room contents.

With respect to cleaning processes, the frequency and the type of cleaning applied will influence the dust loading within a room and, therefore, the amount of dust, and particle size that accrues. Washing floors and carpets is the most efficient technique to remove dust from indoor environments (Schneider *et al.*, 1994, Svendsen *et al.*, 2006), as vacuuming often re-suspends and leaves smaller particulates (Hunt *et al.*, 2008), which go on to settle back down on sideboards and the floor.

Carpets are another source of variation within an indoor microenvironment, as they can act as dust reservoirs trapping dust particles within them (Roberts *et al.*, 1999), therefore newer (less worn) carpets can achieve a higher retention and re-suspension of dust particles than worn and older carpets (Svendsen *et al.*, 2006).

4.2. SAMPLING AND LOCALITY

Indoor dust samples were collected to measure within-room spatial variability of PFC concentrations. This was achieved by collecting 5 'point' samples from locations greater than 1 m apart within rooms (schematics are shown in Figure 21). All sampling was conducted according to the methodology detailed in chapter 2. Six different indoor environments were sampled in this fashion, while in a seventh room, only three "point" samples were collected (due to a small amount of floor space). In all cases, the areas sampled did not overlap and samples were collected within minutes of each other.

Temporal variability in both air and dust concentrations was monitored via monthly sampling. Seasonal variations were measured by collection of dust samples from rooms in five different buildings, over a period of one year between September 2008 and August 2009. Samples were collected according to the methodology described in chapter 2, and collected from the same area of floor each month. Passive air samplers were also located over the same time period in the five rooms monitored for seasonal variations. The passive samplers were deployed at the beginning of each month, to measure changes in air contamination throughout the period of one year.

The characteristics for each of the rooms studied in both the spatial and temporal sampling campaigns have been detailed in Table 49. During the temporal variability campaign very little changed within the rooms, with the major change being the addition of a Christmas tree during the month of December, in each of the rooms.

The variability (expressed as relative standard deviation – %RSD) in PFC concentrations associated with the preparation and analytical procedures is displayed in Table 48. The %RSD determined for the analytical procedure was determined from a reproducibility test, where 5 blank samples were spiked and the whole analytical

procedure was conducted upon these blanks. The reproducibility was assessed from the percentage recovery from the blank samples, and the %RSD for the analytical procedure is derived from these samples. Values exceeding these %RSD values in the spatial and temporal variability studies imply genuine spatial and temporal variability that is over and above that due to measurement uncertainty.

Table 48 Preparation and analytical variability (% RSD) of air and dust samples

PFC	Air sample preparation and analytical variability (%RSD)	Dust sample preparation and analytical variability (%RSD)
PFOS	10	7
PFOA	7	5
PFHxS	10	9
MeFOSA	10	5
EtFOSA	9	8
FOSA	10	10
MeFOSE	6	6
EtFOSE	10	7

Table 49 Contents of analysed rooms

Samples	Chairs	Sofas	Beds	TVs	Electronics	Textiles	Flooring	Other
Site 1s	0	2	0	1	DVD player, Digital TV box, laptop x2	Curtains, throws x2, blanket x2, pillows x4,	Carpet and rug (< 2 years)	
Site 2s	2	2	0	1	TV	Curtains, pillows x4	Carpet (c.a. 5 years)	Table and 4 chairs
Site 3s	0	5	0	0	Stereo and speakers	Curtains	Laminate flooring	social room, 2 tables
Site 4s	5	0	0	0	Stereo and speakers, gym equipment	Cushioned seating	Varnished floor boards	
Site 5s	22	0	0	1	Projector, water heater	Material blinds, piles of carpet squares	Carpet squares (2 years)	Tables and white board
Site 6s	0	2	0	0	Water cooler	Material blinds	Carpet (5 years)	Books, tables and stools
Site 7s	1	0	1	1	Stereo, laptop	Curtains, bedding materials, pillows x3, clothing	Carpet (4 years)	Student room - desk
Site 1t	1	1	0	0	Washing machine, tumble dryer, kettle, stereo	Curtains, throws x2, table cloth, pillows x3	Varnished floor boards and rugs	Boiler, table, sink, cooker
Site 2t	1	2	0	1	DVD player, surround sound	Curtains, blanket x2, pillows x10	Carpet and rugs (< 2 years)	Open fire place (wood and coal)
Site 3t	1	0	1	1	Laptop, stereo	Curtains, bedding materials, pillows x4, clothing	Carpet (4 years)	Student room – desk
Site 4t	0	2	0	1	DVD player, digital TV box, surround sound	Curtains, throw, blanket x2, pillows x 5	Carpet and rug (4 years)	Coal burning fire place
Site 5t	0	1	0	1	DVD player, surround sound, router	Curtains, blanket	Varnished floorboards and rugs	Wood burner fireplace
Site 6t	0	1	0	1	Digital TV box, DVD player	Curtains	Carpet (3 years)	
Site 7t	0	0	0	1	Stereo, weather station	Curtains, cushions x2	Carpet (5 years)	
Site 8t	1	0	1	0	Desktop computer, stereo	Curtains, bedding material, pillows x8, clothing,	Carpet and rug (8 years)	Student room - desk
Site 9t	1	1	0	1	DVD player	Curtains, pillows x3	Carpet (6 years)	
Site 10t	0	0	0	0	Piano, Stereo	Curtains, cushions x6, net curtains	Varnished floorboards and rug	

s = spatial variation t = temporal variation

4.3. TEMPORAL VARIATIONS IN PFC CONCENTRATIONS IN DUST

Temporal variability was measured via air and dust samples taken monthly from a number of different rooms over a period of 12 months, between September 2008 and August 2009. The results are detailed in Table 50.

Table 50 Temporal variations in PFC concentrations in dust (ng g^{-1} and in parentheses ng m^{-2}) and RSD

Month	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	Dust Loading
September	160 (330)	170 (330)	840 (1700)	n.d	540 (1100)	170 (330)	1500 (3000)	140 (280)	2
October	190 (340)	200 (350)	610 (1100)	n.d	370 (670)	200 (360)	1600 (2900)	120 (220)	1.8
November	140 (330)	130 (320)	690 (1600)	58 (140)	400 (950)	200 (490)	1400 (3300)	130 (320)	2.4
December	95 (100)	150 (170)	490 (540)	11 (12)	120 (130)	160 (170)	1300 (1400)	140 (160)	1.1
January	84 (100)	300 (360)	170 (200)	n.d	170 (200)	110 (130)	1400 (1600)	150 (190)	1.2
February	78 (230)	260 (750)	200 (590)	n.d	110 (310)	110 (330)	250 (720)	150 (430)	2.9
March	160 (210)	160 (210)	310 (400)	n.d	480 (630)	94 (120)	1800 (2300)	110 (140)	1.3
April	440 (700)	140 (220)	110 (180)	41 (66)	220 (360)	140 (230)	1000 (1600)	210 (340)	1.6
May	85 (120)	140 (190)	430 (610)	n.d	350 (490)	76 (110)	420 (590)	340 (480)	1.4
June	140 (370)	150 (400)	230 (610)	n.d	230 (620)	190 (510)	520 (1400)	150 (400)	2.7
August	180 (370)	210 (440)	530 (1100)	n.d	180 (390)	170 (350)	830 (1700)	180 (370)	2.1
% RSD[a]	**63**	**30**	**56**	**201**	**51**	**30**	**47**	**39**	
% RSD[b]	**69**	**59**	**76**	**235**	**66**	**61**	**59**	**51**	**33**[c]
Site 2	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**	**Dust Loading**
September	220 (260)	160 (200)	190 (230)	n.d	100 (120)	98 (120)	360 (430)	790 (950)	1.2
October	1100 (1200)	130 (140)	330 (360)	n.d	300 (330)	110 (120)	160 (180)	750 (830)	1.1
November	130 (190)	580 (860)	280 (420)	n.d	150 (230)	190 (290)	100 (160)	300 (440)	1.5
December	120 (230)	570 (1100)	270 (520)	n.d	130 (250)	110 (200)	290 (560)	1400 (2600)	1.9
January	110 (130)	920 (1100)	370 (440)	860 (1000)	100 (120)	250 (300)	600 (730)	640 (770)	1.2
February	160 (200)	440 (570)	140 (180)	n.d	100 (130)	100 (130)	290 (380)	230 (300)	1.3
March	130 (160)	500 (600)	620 (740)	n.d	170 (210)	160 (190)	810 (980)	750 (910)	1.2
April	880 (610)	120 (87)	310 (220)	n.d	180 (130)	170 (120)	1600 (1100)	400 (280)	0.7
May	550 (600)	520 (580)	160 (170)	n.d	140 (160)	420 (460)	1600 (1800)	780 (860)	1.1
June	110 (140)	490 (580)	190 (230)	130 (160)	29 (35)	220 (260)	1600 (1900)	190 (230)	1.2
July	270 (440)	410 (660)	560 (890)	n.d	11 (18)	85 (140)	380 (610)	240 (380)	1.6
August	170 (290)	240 (400)	210 (360)	n.d	840 (1400)	75 (130)	550 (930)	600 (1000)	1.7
% RSD[a]	**102**	**54**	**50**	**299**	**116**	**60**	**83**	**58**	
% RSD[b]	**85**	**58**	**57**	**299**	**144**	**52**	**70**	**80**	**24**[c]

Site 3	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	Dust Loading
September	270 (250)	410 (370)	260 (500)	n.d	110 (99)	85 (76)	880 (800)	240 (210)	0.9
October	270 (220)	290 (230)	210 (170)	n.d	840 (670)	75 (60)	930 (750)	270 (210)	0.8
November	350 (310)	400 (360)	560 (500)	n.d	550 (490)	110 (99)	870 (780)	370 (330)	0.9
December	260 (260)	400 (400)	220 (220)	n.d	240 (240)	120 (120)	900 (900)	240 (240)	1
January	250 (200)	860 (690)	640 (510)	n.d	200 (160)	130 (100)	800 (640)	100 (81)	0.8
February	170 (350)	530 (1100)	1700 (3600)	100 (220)	220 (450)	190 (400)	380 (790)	130 (280)	2.1
March	130 (75)	680 (410)	920 (550)	n.d	350 (210)	250 (150)	5600 (3400)	1500 (910)	0.6
April	180 (36)	400 (80)	410 (82)	n.d	350 (69)	93 (430)	2200 (430)	5300 (1100)	0.2
May	420 (760)	350 (630)	240 (430)	n.d	550 (980)	190 (330)	730 (1300)	300 (550)	1.8
June	120 (36)	370 (110)	320 (95)	n.d	330 (99)	130 (40)	9500 (2800)	2100 (640)	0.3
July	140 (98)	560 (390)	210 (150)	n.d	140 (95)	110 (74)	280 (1940)	630 (440)	0.7
August	230 (270)	340 (410)	330 (400)	n.d	240 (290)	79 (94)	520 (620)	840 (1000)	1.2
% RSD[a]	**40**	**35**	**82**	**346**	**62**	**41**	**126**	**147**	
% RSD[b]	**82**	**64**	**160**	**346**	**87**	**90**	**75**	**68**	**58 [c]**
Site 4	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**	**Dust Loading**
September	250 (550)	110 (240)	1200 (2600)	n.d	140 (310)	150 (320)	260 (580)	1400 (3100)	2.2
October	220 (580)	320 (820)	1800 (4700)	n.d	78 (200)	180 (480)	360 (940)	1000 (2600)	2.6
November	380 (340)	250 (230)	720 (640)	n.d	170 (150)	150 (130)	490 (440)	1400 (1200)	0.9
December	310 (1000)	320 (1000)	1300 (4200)	n.d	110 (350)	140 (450)	320 (1000)	1300 (4200)	3.2
January	290 (610)	260 (540)	1200 (2600)	n.d	140 (290)	300 (630)	120 (250)	1200 (2600)	2.1
February	580 (1200)	150 (310)	1400 (2900)	n.d	130 (260)	140 (290)	320 (672)	1000 (2100)	2.1
March	1800 (2300)	470 (610)	700 (910)	830 (1100)	170 (220)	67 (88)	520 (680)	1100 (1500)	1.3
April	450 (860)	390 (750)	1200 (2300)	n.d	180 (340)	120 (230)	350 (660)	1100 (2100)	1.9
May	480 (870)	250 (450)	1400 (2500)	n.d	170 (300)	200 (360)	250 (450)	1800 (3300)	1.8
June	210 (470)	270 (600)	1300 (2600)	27 (60)	150 (340)	190 (410)	350 (760)	1500 (3300)	2.2
July	490 (830)	230 (390)	1300 (2100)	28 (47)	140 (230)	230 (390)	350 (590)	1300 (2200)	1.7
August	420 (750)	190 (350)	1400 (2400)	31 (55)	190 (340)	140 (250)	150 (270)	1000 (1900)	1.8
% RSD[a]	**86**	**37**	**24**	**311**	**22**	**36**	**37**	**19**	
% RSD[b]	**59**	**47**	**44**	**298**	**23**	**45**	**39**	**34**	**30 [c]**

Site 5	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	Dust Loading
September	530 (480)	260 (240)	1600 (1400)	n.d	160 (140)	160 (140)	750 (680)	930 (830)	0.9
October	420 (460)	98 (110)	1500 (1700)	n.d	150 (160)	230 (260)	410 (460)	1100 (1200)	1.1
November	130 (170)	150 (190)	1000 (1300)	n.d	140 (180)	370 (470)	680 (890)	1600 (2100)	1.3
December	97 (140)	300 (420)	1200 (1700)	n.d	82 (110)	270 (380)	480 (670)	890 (1200)	1.4
January	390 (350)	240 (210)	320 (290)	n.d	220 (200)	220 (190)	900 (810)	470 (430)	0.9
February	150 (180)	76 (91)	210 (250)	n.d	72 (86)	72 (86)	1200 (1500)	540 (650)	1.2
March	420 (790)	290 (550)	420 (790)	n.d	200 (380)	69 (130)	950 (1800)	430 (810)	1.9
April	400 (320)	310 (250)	320 (250)	n.d	120 (93)	220 (180)	1100 (890)	1500 (1200)	0.8
May	69 (35)	73 (37)	1900 (930)	n.d	120 (60)	50 (25)	1300 (670)	1800 (920)	0.5
June	97 (39)	98 (39)	2700 (1100)	n.d	97 (390	80 (32)	900 (350)	430 (170)	0.4
August	120 (140)	110 (130)	230 (270)	n.d	110 (130	64 (77)	840 (1000)	880 (1100)	1.2
% RSD[a]	67	54	80		35	64	33	52	
% RSD[b]	90	86	74		74	88	60	63	38 [c]
all sites (mean)	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	Dust Loading
September	290 (410)	220 (320)	870 (1300)	n.d	210 (300)	130 (190)	750 (1100)	700 (1000)	1.4
October	440 (660)	210 (310)	900 (1300)	n.d	350 (510)	160 (240)	700 (1000)	640 (960)	1.5
November	230 (320)	300 (420)	650 (910)	12 (16)	280 (390)	200 (290)	710 (990)	760 (1100)	1.4
December	180 (310)	350 (600)	710 (1200)	2 (4)	140 (240)	160 (270)	660 (1100)	790 (1400)	1.7
January	220 (280)	510 (640)	550 (680)	170 (210)	160 (200)	200 (250)	760 (940)	420 (640)	1.2
February	230 (440)	290 (560)	730 (1400)	21 (40)	130 (240)	120 (240)	490 (950)	420 (800)	1.9
March	520 (660)	420 (530)	590 (750)	170 (210)	270 (340)	130 (160)	1900 (2400)	790 (990)	1.3
April	470 (490)	270 (280)	480 (500)	8 (9)	210 (220)	150 (160)	1300 (1300)	1700 (1800)	1
May	320 (420)	270 (350)	820 (1100)	n.d	270 (350)	190 (250)	880 (1200)	1000 (1300)	1.3
June	140 (190)	280 (370)	950 (1300)	32 (44)	170 (230)	160 (220)	2600 (3500)	880 (1200)	1.4
July	300 (380)	400 (510)	670 (860)	9 (12)	95 (120)	140 (180)	1200 (1500)	720 (920)	1.3
August	220 (350)	220 (350)	530 (850)	6 (10)	310 (500)	110 (170)	580 (920)	700 (1100)	1.6
% RSD[a]	41	30	22	177	37	20	60	40	
% RSD[b]	35	28	29	169	40	21	55	27	17 [c]

RSD[a] – %RSD of samples in ng g^{-1} RSD[b] – %RSD of samples in ng m^{-2}

[c] – relative standard deviation of dust loading (%)

RSD values are higher (19 – 150 % and 21 – 160 %, for concentrations measured in ng g^{-1} and ng m^{-2}, respectively) than what can be attributed to the variation from preparation and analytical measurements (5 – 10 %). The temporal variations are present even when the dust concentrations are compared per m^2 of the room (rather than per gram of dust), and so cannot be attributed to variations in the dust loading over time.

%RSD values are site specific, and there is little evidence of the same pattern of behaviour occurring for multiple sites. The multitude of potential sources for indoor environments and the impact of human presence in the room can mask the effect of any temporal variations created by specific parameters (temperature, humidity).

There were no specific room content variations during the sampling campaign that could be linked to variations. During the month of December, when each of the environments had the addition of a Christmas tree, the concentrations in the rooms were not distinguishable from any of the equivalent months (Figure 16).

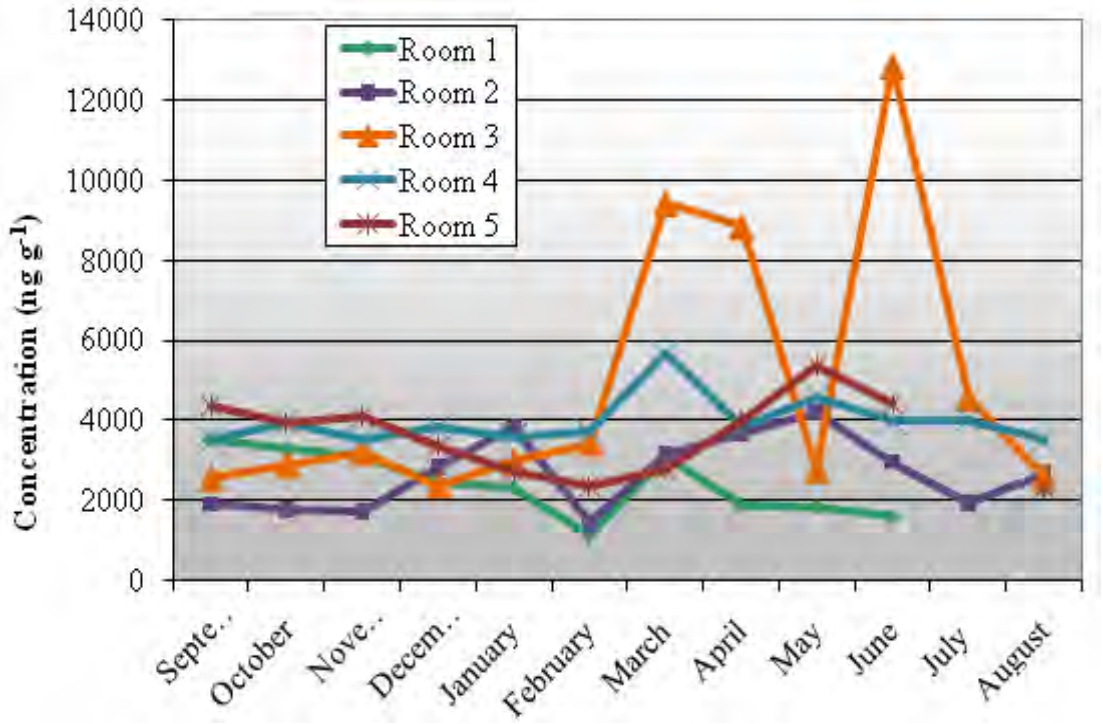


Figure 16 ΣPFC concentrations (ng g^{-1}) for each site per month

Causes of changes in MeFOSE and EtFOSE at site 3, are unknown, and maybe due to the addition of new items in the room, however no conclusive answer can be derived from the questionnaire data (PCA was not conducted due to small individual data sets). Temporal variations were assessed across rooms to determine the presence of temporal effects inducing changes simultaneously. Significant correlations were identified for site 1 PFOS concentrations and site 2 and site 3 ($p = 0.02$, $r = 0.67$, and $p = 0.04$, $r = 0.63$, respectively), however, on closer inspection, these relationships were driven by two extreme values. MeFOSE concentrations from site 5 were also correlated with site 2 ($p = 0.04$ and $r = 0.62$), whilst EtFOSE from site 1 was correlated with changes seen in sites 4 and 5 ($p = 0.04$ and $r = 0.63$, $p = 0.04$ and $r = 0.64$, respectively). These correlations indicate that there may be some common factors driving temporal variability at these sites. The relationships are not present in all five of the rooms monitored however; suggesting that different sources act upon each location.

Correlations between compounds (see

Table 51) in each location were examined and some identified, indicating the possibility that, for relationships with a positive correlation coefficient, these compounds are affected simultaneously by the same changing parameter over time.

Table 51 Correlation between individual compounds in each site

Site	Compound A	Compound B	p-value	Pearson correlation coefficient, r
1	MeFOSE	EtFOSA	<0.01	0.55
	MeFOSE	EtFOSE	<0.01	-0.7
2	PFOS	PFOA	0.04	0.6
	MeFOSE	FOSA	0.02	0.64
3	PFOS	MeFOSE	0.03	-0.62
4	PFOS	PFOA	0.05	0.58
	PFOS	PFHxS	0.05	-0.58
	MeFOSE	FOSA	0.04	-0.59
5	PFOS	EtFOSA	0.02	0.68
	MeFOSE	FOSA	0.05	-0.63

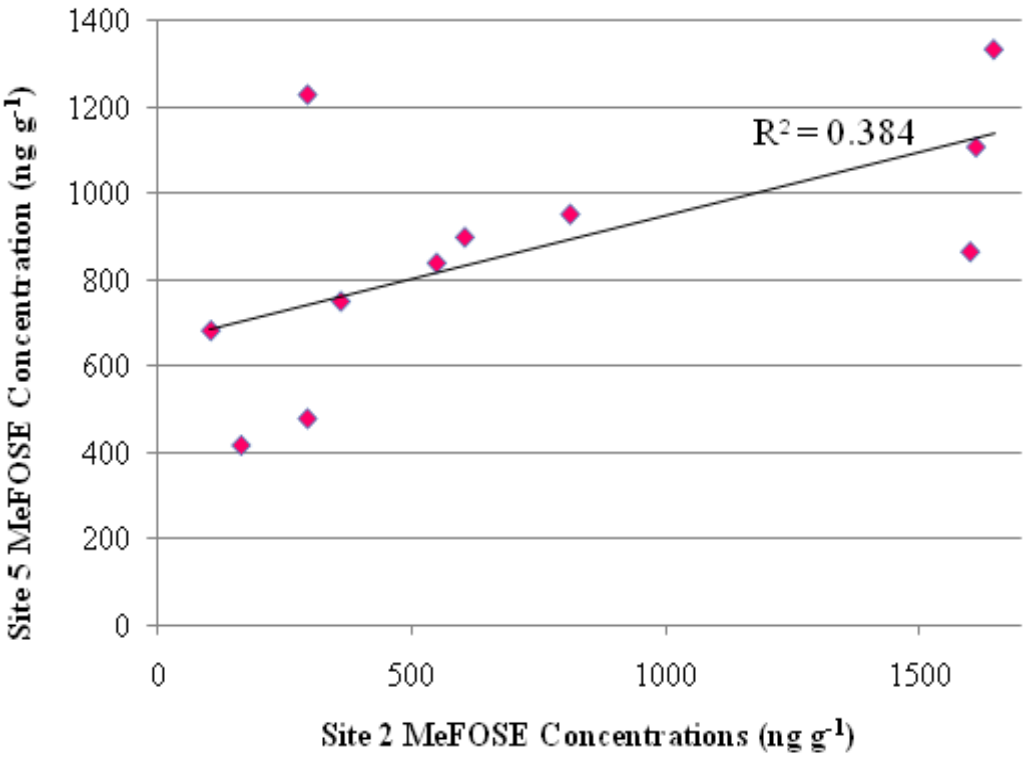


Figure 17 MeFOSE correlation between site 2 and 5 (ng g^{-1})

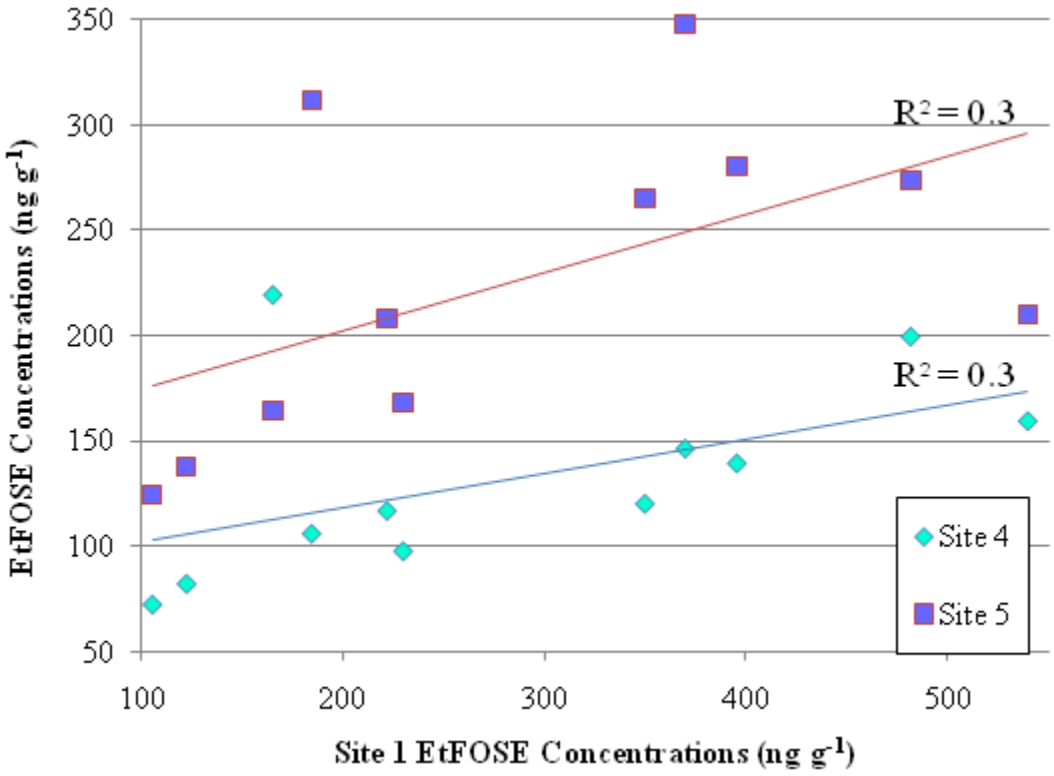


Figure 18 EtFOSE correlations between site 1 and sites 4 and 5 (ng g^{-1})

From sites 4 and 5 there is a negative relationship identified for MeFOSE and FOSA, indicating that the change in MeFOSE may induce an opposite variation in FOSA concentrations. This is in line with studies by Schenker *et al.,* (2008), who suggest the degradation of MeFOSE results in FOSA production as an intermediate degradation product. A relationship between these two compounds is not noted from any other room, apart from room 2 where they are positively correlated, suggesting that, as one compound varies, the other varies in the same direction. Despite research indicating the possibility of MeFOSE degrading to FOSA, it is also possible for more volatile compounds (Plumlee *et al.,* 2009, Martin *et al.,* 2006) to degrade via a variety of degradation steps, suggesting that the input to MeFOSE and FOSA concentrations are derived from the same source, and are not directly related by the degradation of MeFOSE to FOSA.

The general change in concentrations of individual PFCs over the sampling period did not highlight any changes induced by seasonal variations. However, concentrations of ∑PFCs, at sites 4 and 5 displayed a significant negative correlation ($p < 0.05$) with mean outdoor temperature (Met Office mean monthly weather, Figure 19 and Table 52) and site 5 is negatively correlated to average sunlight ($p < 0.01$). The correlations may not necessarily imply that concentrations are directly related to meteorological parameters, but may suggest that increases in temperature and sunlight are linked with other variables such as ventilation, time spent and type of activity within the room, which effect the overall concentrations and generation of dust and PFCs. Rainfall measurements were also significantly correlated positively with PFHxS ($p < 0.05$), though none of the other compounds, which reside primarily in the particulate form. were identified to be affected by this relationship. Again, the relationship is not likely to result from a direct partnership between rainfall and PFHxS, but is likely to occur

Table 52 Correlations of compounds with weather parameters.

Site	Parameter	Compound	p- value	r
1	Sunlight	EtFOSE	<0.05	0.58
	Temperature	EtFOSE	<0.05	-0.62
2	Temperature	PFHxS	<0.05	-0.75
	Temperature	PFOA	<0.05	-0.6
	Sunlight	MeFOSE	<0.01	0.69
	Sunlight	PFHxS	<0.05	-0.57
3	Temperature	PFHxS	<0.05	0.57
	Rainfall	PFOS	<0.05	0.58
	Rainfall	PFHxS	<0.02	0.63
4	Sunlight	PFOS	<0.05	-0.57
	Temperature	PFOS	<0.001	-0.79
	Temperature	∑PFCs	<0.05	-0.53
5	Sunlight	EtFOSE	<0.01	-0.56
	Sunlight	PFCs	<0.01	-0.79
	Sunlight	FOSA	<0.05	-0.6
	Temperature	∑PFCs	<0.05	-0.54
	Rainfall	PFHxS	<0.05	0.62

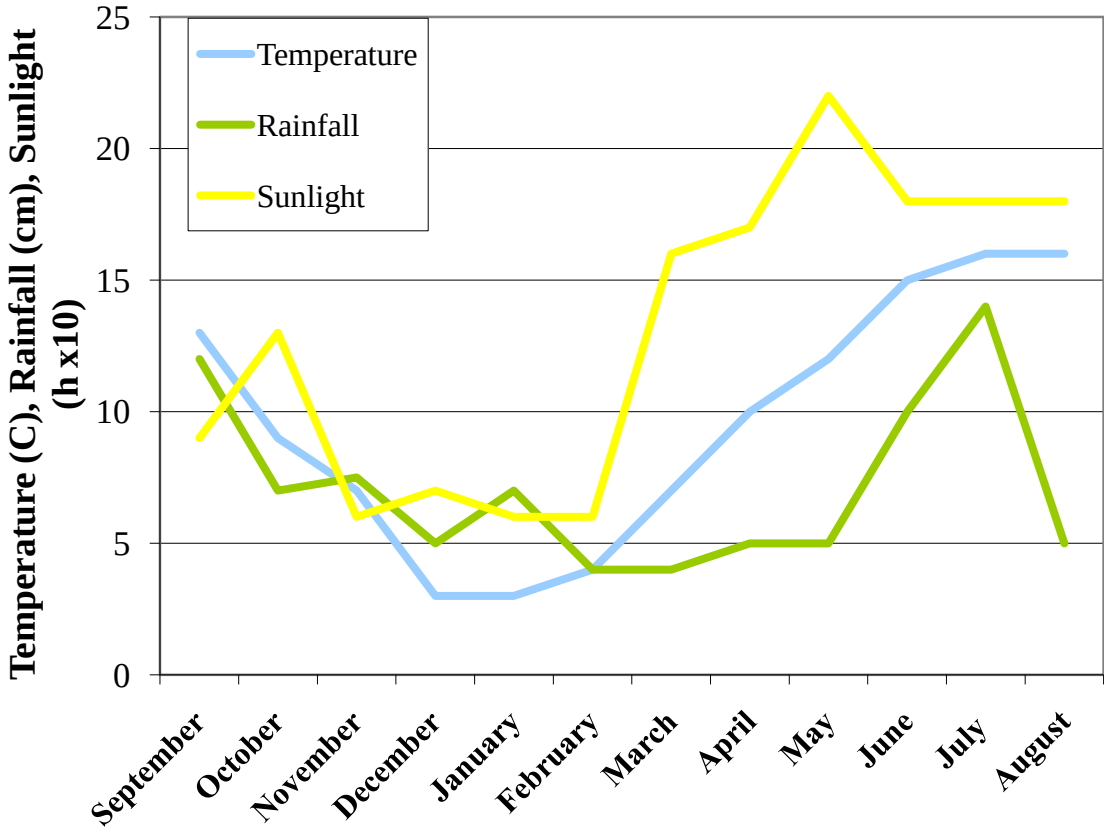


Figure 19 Weather conditions during temporal sampling campaign

from induced indoor humidity, increased occupancy and activity in the room, along with other parameters.

Dust loadings per room were compared to the mass-based PFC concentrations from individual rooms monitored over the temporal campaign. Temporal variability studies for organohalogens within rooms conducted by Harrad *et al.*, (2009), Zhang *et al.*, (2009) and Harrad *et al.*, (2008) indicate that variability can be caused by variations with time for parameters including temperature, room ventilation rate, use of room and particle concentrations, as well as the presence of individual objects and their age. These differences are created by the leaching rate from treated objects, seasonality, and associated outdoor temperature, within room ventilation, and activity levels within the room varying the suspension and dispersion of particulates. Harrad, (2010a) describes the importance of obtaining a "biologically relevant" sample, via the use of spot sample collection. In the chapter by Harrad, (2010a) it has been noted that, in certain microenvironments, spatial within-room variability can occur, surpassing the associated analytical uncertainty, and thus sampling in a well frequented area of the room is likely to provide a more 'biologically relevant' sample. Results (displayed in Figure 20) are particularly notable for sites 1, 2, & 3, for which significant ($p < 0.05$) negative correlation exists between the concentration of MeFOSE and dust loading within the room. This relationship suggests that the source(s) of MeFOSE in these rooms are independent of the sources of the dust. Hence, as dust loadings increase, the concentrations of MeFOSE in the dust are reduced or "diluted". For site 2, PFOS and site 3, EtFOSE are also both significantly negatively correlated to the room dust loading. The other compounds indicate no relationship is present with the dust loading, suggesting a constant concentration in the dust.

This suggests that there are sources of MeFOSE within the room adding to the concentrations within the room dust. This supports the idea that room dust can act as a reservoir to PFC compounds. This suggests that some sources of dust are also the sources of PFCs, which agrees with the results in chapter 6(classroom dust concentrations), where the number of occupants in the room was positively correlated to the concentration and the more wear and tear and their activity was a generator for PFCs. The relationship of PFHxS with dust loading in room 4 and room 5 indicates significant relationships with room dust loading. However, in room 4, this relationship is positive, and in room 5 negative. These different relationships are probably driven by separate sources, one which is independent of the source of dust (negative relationship) and another (positive relationship), whereby the source(s) of the dust are the same as for PFHxS (e.g. abrasion of particle/fibers from a fabric treated with PFHxS). For these compounds which appear to be coupled with dust content in the room, support the idea that concentrations of PFCS quantified in the dust are produced from the wear and tear, and leaching from sources within the room. The idea supports the hypothesis that the dust can act a reservoir to the PFCs within a room; with the compounds leaving the samples are remaining within the dust fraction.

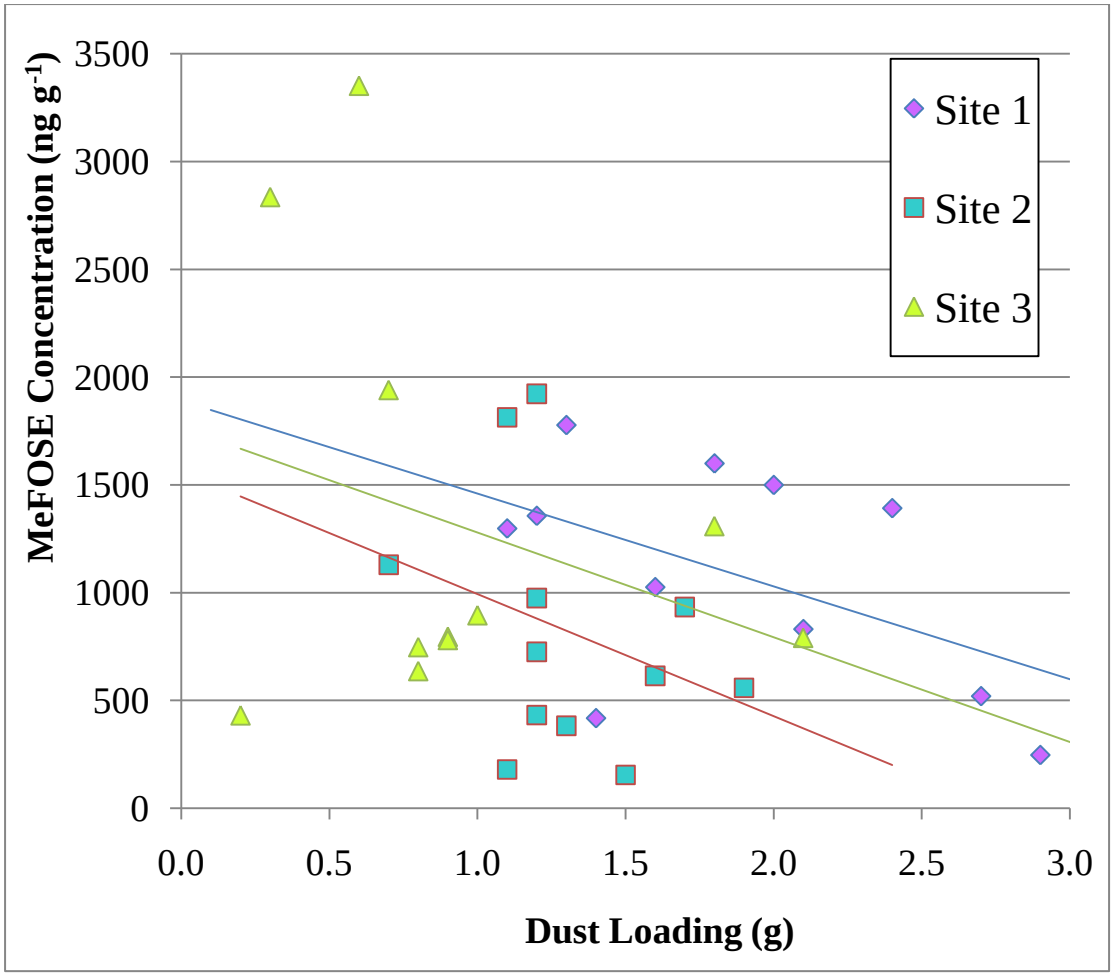


Figure 20 Site 1 dust loading correlations

4.4. TEMPORAL VARIATIONS IN CONCENTRATIONS OF PFCS IN INDOOR AIR

To the best of the author's knowledge, temporal variations in concentrations of PFCs in indoor air have not previously been reported.

Table 53 Temporal concentration variations (pg m^{-3}) in Indoor Air (with non – detects being replaced by half the LOD).

Site 1	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	∑PFC
September	<DL	<DL	12	<DL	110	230	550	230	1100
October	71	43	100	<DL	280	64	3600	3100	7300
November	44	89	24	<DL	360	120	8200	4600	13 000
January	<DL	19	21	13	120	70	1800	2900	4800
February	8	<DL	22	<DL	250	200	120	2300	2900
March	27	40	17	<DL	160	270	2600	2700	5800
April	8	33	49	<DL	220	170	1800	1500	3800
May	<DL	<DL	35	<DL	240	62	2100	1900	4300
June	5	<DL	4	<DL	83	110	580	650	1400
August	28	<DL	<DL	<DL	72	800	1800	1800	4500
% RSD	**112**	**119**	**101**	**290**	**49**	**112**	**94**	**47**	**64**
Site 2									
September	30	18	110	<DL	29	750	450	950	2300
October	25	15	49	<DL	61	110	1200	210	1700
November	62	15	30	596	130	190	420	190	1600
December	52	74	7	<DL	230	190	660	340	1500
January	53	19	30	<DL	200	200	550	540	1600
February	3	4	9	<DL	230	170	870	640	1900
April	18	10	44	<DL	1100	190	490	680	2500
May	36	<DL	50	<DL	290	100	710	580	1800
June	40	120	110	80	250	130	1200	440	2400
July	11	23	44	<DL	380	<DL	360	400	1200
August	<DL	<DL	22	<DL	170	230	190	180	790
% RSD	**69**	**135**	**77**	**291**	**105**	**93**	**52**	**51**	**30**

Site 3									
September	78	<DL	<DL	<DL	110	240	710	870	2000
October	91	<DL	8	<DL	50	770	3400	6400	11 000
November	52	16	30	<DL	130	190	420	1900	2800
December	9	34	16	<DL	110	350	590	760	1900
January	9	39	50	<DL	500	660	1800	3400	6500
February	2	8	33	<DL	150	290	3100	1300	4800
March	8	17	12	<DL	300	320	620	2000	3300
April	9	3	12	<DL	150	730	1200	740	2800
May	<DL	31	18	<DL	230	460	1300	970	3000
June	5	7	9	<DL	550	310	1100	3100	5100
August	<DL	<DL	<DL	<DL	59	200	590	750	1600
% RSD	**159**	**92**	**79**		**71**	**51**	**74**	**83**	**66**
Site 4									
September	<DL	<DL	<DL	<DL	150	290	600	890	1900
October	8	9	24	<DL	230	77	640	1500	2500
November	55	30	11	<DL	110	310	890	3100	4500
December	88	13	12	52	210	88	700	810	2000
January	69	21	49	19	890	160	6800	950	9000
February	59	28	100	<DL	230	190	430	2100	3200
March	38	34	9	5	70	480	830	800	2900
April	<DL	37	16	<DL	230	190	1700	1300	3400
May	44	14	84	<DL	120	500	1800	1900	4400
June	19	43	17	<DL	30	38	280	390	820
July	22	20	19	<DL	28	52	130	530	800
August	<DL	25	<DL	<DL	510	150	1600	830	3100
% RSD	**80**	**57**	**110**	**230**	**100**	**75**	**140**	**63**	**67**

Site 5									
September	<DL	<DL	<DL	<DL	210	140	1600	660	2600
October	11	<DL	37	<DL	370	100	1500	540	250
November	10	8	44	<DL	700	220	1900	860	3700
December	8	5	5	<DL	61	240	1200	1400	2900
January	7	9	27	<DL	150	130	1300	740	2300
February	10	36	23	<DL	150	230	930	3200	4600
March	14	4	47	<DL	270	400	490	760	2000
April	16	8	25	<DL	120	110	720	460	1500
May	4	3	3	<DL	220	200	450	390	1300
June	8	7	27	19	56	180	560	230	1100
July	8	<DL	22	<DL	250	210	410	960	1900
August	61	16	37	<DL	340	430	1200	440	2500
% RSD	**110**	**110**	**52**	**320**	**75**	**47**	**50**	**92**	**64**

The RSD values (47 – 159 %) associated with the temporal air measurements vastly exceed those reported in Table 53 for indoor dust temporal variations, and cannot be attributed to the preparation and analytical variability. The RSD values for MeFOSA are of little value, given the very low frequency with which this compound was detected.

The variation and contribution of individual compound variations in each room were reviewed for contributing parameters arising from the room contents. During the sampling campaign there were no furniture changes within any of the rooms, the major change was the presence of a Christmas tree in all homes for 2 - 3 weeks during December (and the first few days in January). Despite the addition of the trees, PFC concentrations did not vary greatly from the rest of the year. As a result, the questionnaire data associated with the sampling campaign was unable to highlight any potential sources, which could have attributed to the temporal variations.

The large RSD identified suggests that exposure assessments based on a single monthly measure of airborne contamination may not be entirely accurate. The temporal variations do not correlate between rooms, suggesting that temporal variations are chiefly respondent to individual changes in room use, room activity and other parameters. Indoor environments are affected by the weather conditions outdoors, directly and indirectly. The monthly weather conditions were analysed against the temporal variations, results indicating that ∑PFC concentrations in rooms 4 and 5 were negatively correlated (using Pearson's Correlation Coefficient) to the outdoor temperature ($p < 0.05$), with those in room 5 also significantly negatively correlated to sunlight hours ($p < 0.01$).

Temporal variations in airborne concentrations of PFCs in each room vary essentially independently for each of the rooms, indicating that the changes are likely driven by

the activity and use of the rooms. Correlations were examined between concentrations of individual PFCs in each room. For all rooms, significant correlation ($p \leq 0.05$) was identified mainly for the perfluorooctane sulfonamidoethanols (Table 54).

For all rooms the largest variations are primarily for PFOS, PFOA and PFHxS, and this is thought to derive from the low mixing of dust within the room, and the individual sources within a room.

Table 54 Correlations between Airborne Concentrations of Individual PFCs in Temporal Variability Study

Site	Compound A	Compound B	Significance, p	Pearson's Correlation Coefficient, r
Site 1	PFOA	PFOS	<0.05	0.65
		EtFOSA	<0.05	0.70
	MeFOSE	EtFOSE	<0.005	0.83
		EtFOSA	<0.05	0.69
		PFOA	<0.005	0.92
		PFOS	0.05	0.64
	EtFOSE	EtFOSA	0.02	0.73
		PFOA	<0.005	0.82
		PFOS	0.05	0.63
Site 2	EtFOSE	FOSA	<0.05	0.62
Site 3	PFOA	PFHxS	<0.05	0.66
	EtFOSE	EtFOSA	0.001	0.86
		MeFOSE	<0.05	0.62
Site 4	MeFOSE	EtFOSA	<0.01	0.73
Site 5	PFOS	FOSA	0.02	0.68
	PFOA	EtFOSE	<0.005	0.78

The RSD values tend to be driven by concentrations reported for one or two months, which show substantial variation from other months. For site 1 the concentrations in October and November for PFOS, PFOA, MeFOSE and EtFOSE strongly influence the %RSD. For site 4 the concentrations in October and January mostly drive the %RSD values, and for site 5 the FOSA, MeFOSE and EtFOSE concentrations are mainly driven by those recorded in June and July. When these months were analysed with regards to the questionnaire data, there were no discernible events like changes in room contents apparent. Potentially for site 5 the concentrations could have been

affected by the high temperatures and rainfall during these months (affecting indoor humidity, increased deposition etc.). These relationships identified at site 5, may not be evident for the other sites due to masking effects by other variables, such as ventilation, insulation, amount of sun received indoors and indoor temperatures.

4.5. SPATIAL VARIATIONS IN CONCENTRATIONS OF PFCS IN INDOOR ENVIRONMENTS

There are currently no published studies available relating to within-room spatial variations in concentrations of PFCs in settled dust. For other organohalogens, studies have indicated that specific objects within rooms can act as sources to the room, producing concentrated areas of contamination in the surrounding area around the object 'hot spots' (Harrad *et al.*, 2008, Harrad *et al.*, 2009). These spatial variations can cause a large degree of uncertainty in the exposure assessment. This is because there is concern that particular objects that act as a source could be coupled to one particular occupant of the room such as a child (e.g. a toy or a child's play mat). In this situation, exposure estimates derived from point sampling (as described in chapter 2) may not be representative.

Point sampling provides a snap shot of the concentration in the room from a specific area at a distinct point in time. The samples are collected from a well frequented area in order to obtain a representative sample of an area of floor, which has a high potential as acting as a source of dust exposure. This point sample therefore neglects dust from corners and crevices located in areas of a room, which are not well frequented and are unlikely to act as a source of dust ingestion. However, it is possible for the area selected to provide an over-estimate (if a hot spot is measured) or underestimate. Therefore, it is imperative to understand the potential for spatial deviation within room dust.

Within room spatial variation concentrations of PFCs in dust are expected to occur because of the low mixing capability of dust (compared to atmospheric mixing, Gadgil *et al.*, 2003), thus facilitating the potential for divergence in concentrations across the room.

The collection procedure for sampling within room spatial variations requires samples not to be taken from areas adjacent to one another, and a minimum of 0.5 m distance away from any other sample, so as to provide no overlap of the sampling areas and obtain representative dust samples, and dust weightings. Each of the five samples was collected on the same day within a period of 1 hour.

4.6. WITHIN-ROOM SPATIAL VARIATIONS IN CONCENTRATIONS OF PFCS IN DUST

Table 55 contains the concentrations from each of the separate areas (n = 5) monitored in the 7 rooms within which spatial variability was studied, along with the %RSD associated with each compound, and the dust loading recorded in each sample. The room-specific %RSD values are analysed against the %RSD values for dust in Table 48, which represent the associated variance created by the sample preparation (sieving and homogenisation) and the analytical procedures, thereby representing a 'benchmark' value. In the majority of cases the within room spatial variability exceeds the values in Table 48 and in such cases the observed within-room spatial variability exceeds that attributable to preparation and analytical variability combined.

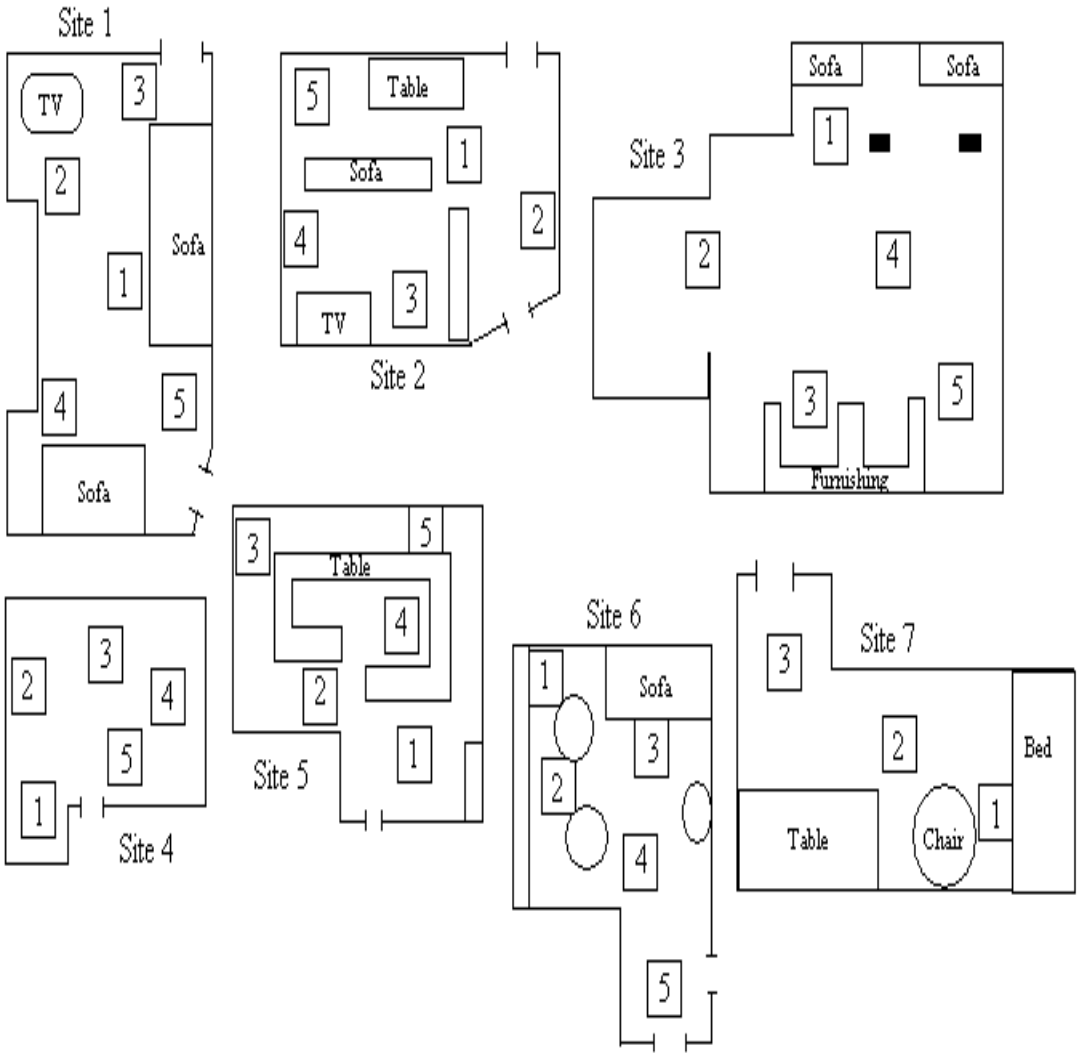


Figure 21 Diagrams (not to scale) of spatial sampling sites within rooms

Table 55 Within room spatial variability in concentrations of PFCs in dust (measured in ng g^{-1} and in parentheses ng m^{-2}) and %RSD values

Site / PFC	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	Dust Loading
Site 1s									
1	130 (410)	24 (75)	61 (200)	<DL	82 (260)	17 (55)	79 (250)	16 (52)	3.2
2	52 (110)	19 (40)	54 (110)	<DL	40 (83)	17 (35)	67 (140)	10 (21)	2.1
3	22 (20)	11 (10)	25 (22)	<DL	24 (22)	7 (7)	78 (71)	6 (6)	0.9
4	51 (61)	14 (17)	55 (66)	<DL	52 (62)	9 (11)	70 (84)	10 (12)	1.2
5	25 (50)	15 (31)	61 (120)	<DL	54 (110)	6 (13)	59 (120)	13 (26)	2
%RSD[a]	**78**	**29**	**29**	**0**	**42**	**46**	**12**	**33**	
%RSD[b]	**123**	**74**	**63**	**48**	**85**	**85**	**54**	**76**	**48**
Site 2s	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**	**Dust Loading**
1	43 (47)	18 (20)	540 (600)	<DL	6 (7)	33 (36)	130 (150)	57 (63)	1.1
2	30 (51)	73 (120)	210 (350)	<DL	10 (17)	42 (72)	110 (190)	28 (48)	1.7
3	170 (170)	39 (39)	3100 (3100)	<DL	17 (17)	24 (24)	120 (120)	71 (71)	1
4	310 (930)	79 (240)	3600 (11 000)	<DL	13 (40)	44 (130)	130 (380)	96 (290)	3
5	23 (80)	22 (76)	180 (620)	<DL	21 (74)	45 (160)	130 (450)	210 (740)	3.5
%RSD[a]	**110**	**61**	**110**	**0**	**42**	**24**	**7**	**76**	
%RSD[b]	**150**	**87**	**140**	**0**	**86**	**70**	**57**	**120**	**55**
Site 3s	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**	**Dust Loading**
1	1100 (2100)	460 (840)	110 (190)	<DL	11 (20)	60 (110)	250 (440)	390 (690)	1.8
2	710 (1000)	230 (330)	72 (100)	<DL	4 (6)	60 (84)	550 (780)	250 (340)	1.4
3	1300 (640)	160 (82)	110 (56)	<DL	10 (5)	62 (31)	1700 (850)	320 (160)	0.5
4	320 (660)	1100 (970)	24 (21)	<DL	14 (12)	63 (56)	360 (330)	52 (47)	0.9
5	1100 (2000)	100 (200)	240 (270)	<DL	18 (33)	65 (123)	740 (1400)	840 (1600)	1.9
%RSD[a]	**30**	**97**	**49**	**0**	**45**	**3**	**81**	**79**	
%RSD[b]	**59**	**82**	**79**	**0**	**78**	**47**	**55**	**110**	**46**

Site 4s	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	Dust Loading
1	1200 (830)	190 (130)	16 (11)	<DL	4 (3)	120 (86)	1100 (740)	2700 (1900)	0.7
2	930 (1300)	190 (260)	54 (76)	<DL	4 (6)	99 (140)	250 (350)	800 (1100)	1.4
3	670 (540)	130 (110)	130 (100)	<DL	1 (1)	130 (110)	920 (740	2600 (2100)	0.8
4	790 (790)	120 (120)	93 (93)	<DL	2 (2)	150 (150)	730 (730)	1800 (1800)	1
5	1100 (780)	140 (100)	140 (98)	<DL	4 (2)	100 (70)	270 (190)	1000 (700)	0.7
%RSD[a]	23	20	60	0	43	19	57	50	
%RSD[b]	33	46	50	0	63	31	48	39	32
Site 5s	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**	**Dust Loading**
1	5100 (7700)	700 (1000)	1600 (2400)	<DL	77 (120)	66 (100)	950 (1400)	12 (17)	1.5
2	1400 (3300)	310 (730)	400 (970)	<DL	13 (30)	74 (180)	620 (1500)	14 (35)	2.4
3	950 (1000)	160 (180)	460 (510)	<DL	18 (20)	73 (81)	350 (390)	90 (99)	1.1
4	1100 (2600)	300 (700)	360 (820)	<DL	11 (26)	63 (85)	1400 (3100)	280 (650)	2.3
5	2100 (1900)	230 (210)	350 (310)	<DL	20 (18)	85 (77)	780 (700)	490 (440)	0.9
%RSD[a]	82	61	85	0	100	12	47	120	
%RSD[b]	79	65	82	0	99	38	75	110	42
Site 6s	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**	**Dust Loading**
1	1900 (3100)	290 (460)	190 (300)	<DL	12 (19)	28 (45)	420 (660)	130 (200)	1.6
2	1500 (2400)	700 (1100)	60 (97)	<DL	12 (20)	29 (47)	380 (610)	120 (190)	1.6
3	1700 (790)	220 (90)	310 (120)	<DL	16 (6)	27 (11)	730 (290)	150 (60)	0.4
4	860 (1500)	330 (560)	390 (660)	<DL	8 (14)	42 (72)	150 (260)	640 (1100)	1.7
5	1100 (570)	360 (180)	42 (21)	<DL	16 (8)	32 (16)	360 (180)	68 (34)	0.5
%RSD[a]	33	49	77	0	26	19	51	110	
%RSD[b]	64	84	110	0	45	65	55	140	56
Site 7s	**PFOS**	**PFOA**	**PFHxS**	**MeFOSA**	**EtFOSA**	**FOSA**	**MeFOSE**	**EtFOSE**	**Dust Loading**
1	1500 (880)	110 (65)	480 (290)	<DL	180 (110)	55 (33)	1100 (690)	110 (65)	0.6
2	510 (200)	85 (34)	470 (190)	<DL	100 (41)	51 (20)	350 (140)	130 (50)	0.4
3	330 (300)	200 (180)	190 (170)	<DL	180 (170)	62 (56)	1600 (1400)	370 (340)	0.9
%RSD[a]	79	47	43	0	29	10	61	73	
%RSD[b]	79	83	29	0	60	49	86	110	40

The %RSD values from the individual rooms vary with each compound, and there was little evidence to suggest that the compound variations were induced by the same parameter. For sites affected by extreme values, the sample area was checked for locality. No distinct sources could be identified, although there was some indication of higher concentrations in samples taken from areas close to entrances and doorways (sites 1, 2, and 3) and from areas close to windows (sites 5 and 6). The areas around entrances and doorways are affected by the high wear and tear rates, along with potential in-tread of dust (Layton *et al.,* 2009). Likewise, areas close to windows can attract dust (Gewurtz *et al.,* 2009); moreover, heating appliances situated under windows may also attract dust. However, when the data were grouped into these categories, no significant differences were evident.

Apart from these observations, specific variations of PFC concentrations within rooms could not be attributed to any specific product. Data were examined for correlations between concentrations of individual PFCs found in samples taken from each of the five locations sampled in each room, and the relationships found are listed in Table 56. The relationships do not identify why the variations are so high, but highlight compounds that may be affected by the same factors within a given room.

Many of the correlations are identified in more than one room, suggesting that the parameters involved are not room-specific (e.g. a specific carpet). Such parameters could include proximity to doors, ventilation, heating and within room temperature gradients, or located close to where people tend to spend the majority of their time. Notably, in one or more sites, there is a correlation between the possible parent compound and a degradation compound (e.g. EtFOSE is correlated with EtFOSA, FOSA with PFOA, FOSA with PFOS etc). Thus, within a room individual relationships between compounds can exist, which do not have to be associated with

the whole room, supporting the idea of little mixing of dust within rooms and the presence of significant within-room spatial variability.

Table 56 Correlations between concentrations of individual PFCs in dust samples taken in within-room spatial variability study

Site	Compound A	Compound B	Significance, p	Pearson's Correlation Coefficient, r
1	PFOS	PFOA	<0.05	0.89
	PFOA	FOSA	<0.05	0.87
	EtFOSA	EtFOSE	<0.02	0.95
2	PFOS	PFHxS	<0.02	0.95
3	PFHxS	EtFOSE	<0.05	0.88
	EtFOSA	FOSA	<0.05	0.88
4	MeFOSE	EtFOSE	<0.001	0.99
5	PFOS	PFOA	<0.02	0.94
	PFOS	PFHxS	<0.02	0.98
	PFOS	EtFOSA	<0.005	0.98
	PFOA	PFHxS	<0.02	0.94
	PFOA	EtFOSA	<0.05	0.92
	PFHxS	EtFOSA	<0.001	0.99
6	PFOS	FOSA	<0.05	-0.89
	FOSA	EtFOSE	<0.05	0.9
7	PFHxS	EtFOSE	<0.02	-0.99

4.7. CONCLUSIONS AND FINAL REMARKS

The within-room spatial and temporal variability in concentrations of PFCs in air and dust reported here exceeds that attributable to the preparation and analytical procedures alone. Thus the presence of PFC compounds within an indoor environment are plausible to vary over both area and time. The variability caused spatially is thought to occur from a plethora of parameters including wear and tear, activity and usage, proximity to entrances, heating, and windows. The variability was not linked to any specific objects within the rooms, however due to many potential sources being present at once; distortion of product signals could be masking the

effect. Variability was noted for each of the compoundsm, and does not appear to be markedly only the degradation products that can vary but also the more volatile compounds.

The same behavior is seen within the temporal study where both air and dust matrices varied with time and was identified to occur for all of the compounds examined. The reasons for the observed within-room temporal variability are unclear, but are likely to arise from variations in room use and occupancy rather than being directly related to seasonal variations in meteorological conditions. Rather than arising from changes in room contents as suggested previously for BFRs, temporal variations in PFC concentrations appear to occur as a result of changes in room use over time, humidity, temperature and subsequent activity level in the room, ventilation and cleaning, and each indoor environment will vary independently. From the sites monitored in this study, the observed variability in PFC concentrations for each site and for each compound over time has implications for the accuracy of exposure assessments founded on single "point" measures of contamination.

With regards to the spatial variability exceeding that associated with (extraction and analytical methods), emphasises the need to sample the most biologically-relevant part of the room (the most-frequented area). This suggests also that variability in PFC concentrations between different rooms in the same building frequented by a given individual may also influence exposure assessments, and their study appears warranted.

"Dilution" of PFC concentrations in dust at high dust loadings has also been identified, specifically for the MeFOSE compound in three out of the five rooms in which temporal variability was studied. This suggests that concentrations of MeFOSE

in dust can be diluted as dust loadings increase, because the sources of MeFOSE and of the dust are independent of each other.

Use of passive samplers located at a fixed point within a room to monitor airborne PFC concentrations may underestimate exposure. The presence of a personal cloud (Rhodes *et al.*, 1991) in the case of VOCs has been shown to provide a greater exposure, than that based on the whole room air concentration (Harrison *et al.*, 2002, Allen *et al.*, 2007). Such an effect has yet to be studied for PFCs but, as with PBDEs, (Allen *et al.*, 2007), it is most likely to be pertinent for those PFCs existing primarily in the particulate phase (e.g. PFOS and PFOA), and primarily detected in dust.

5. OUTDOOR SPATIAL AND TEMPORAL VARIATIONS

The hypothesis proposed in this chapter is that there is substantial spatial and seasonal variation in the concentrations of PFCs in outdoor air. Hence, the aim of this chapter is to monitor such variability in concentrations of PFCs in ambient air.

Spatial and temporal variability in chemical contamination can impact upon human exposure, leading to some individuals being more highly exposed than others. On a microscale within an urban environment variability is hypothesised to occur because of the outgassing from inter alia homes, as well as manufacturing and industrial sources, coupled with the impact of atmospheric mixing. This chapter monitors spatial and seasonal variability in concentrations of PFCs in outdoor air from a number of urban locations within a 1.5 km area. Moreover, as addressed in chapter 3, indoor environments are a substantial reservoir of PFCs. Hence, like PCBs and BFRs it is likely that indoor PFCs undergo release to the outdoor environment (Harrad & Diamond 2006, Cahill, *et al.* 2007). This indoors to outdoor flow is variable because of seasonal changes in ventilation, along with varying transportation rates and air mass movement across cities.

5.1. INTRODUCTION

Concentrations of PFCs in outdoor air samples have been analysed for the presence of temporal and spatial variations. At present there are few publications relating to the presence of PFCs in outdoor air samples and their variability over time and space. Both natural and anthropogenic parameters may exert an influence over both spatial and temporal scales. Such factors include meteorology, population density and point

sources (Zushi & Masunaga, 2009, Lohman *et al.*, 2007, Murakami & Takada, 2008, Pistocchi & Loos, 2009).

Emissions of PFCs have been noted to occur primarily from urban sources, creating urban-rural gradients (Dreyer *et al.* 2009, Gewurtz *et al.* 2009, Jahnke *et al.* 2007). The transportation efficiency is affected by the natural and anthropogenic parameters mentioned earlier. The rate of atmospheric degradation and movement can also deviate across an urban site, depending on the proximity to sources of volatile organic compounds (VOCs), which can encourage the degradation process via the presence of free radicals OH and NOx (Yarwood *et al.*, 2007, Barber *et al.*, 2007, Ellis *et al.*, 2004, Hurley *et al.*, 2004 and Giesy *et al.*, 2005).

Recent studies by Barber *et al.*, (2007) and Jahnke *et al.*, (2007a), describe spatial variations in tropospheric concentrations across Europe and between urban and rural locations. Other spatial analyses of PFCs in the troposphere have been conducted in the context of movement around the tropics and within the Arctic circle. The latter highlights potential pathways of movement of PFCs to more remote polar regions (Stock *et al.*, 2007). On a local scale, concentration variations of PFCs have been noted across urban areas to impact upon waterways, via precipitation, creating an urban pulse of PFC concentrations (Kim & Kannan, 2007). Another examination of local scale variability was conducted in Germany (Dreyer *et al.*, 2009) at two sites 40 km apart, over a 10 month period. Substantial temporal variability was observed and indicated a significant positive correlation between temperature and atmospheric concentrations of most PFCs studied.

5.2.SAMPLING AND SITES

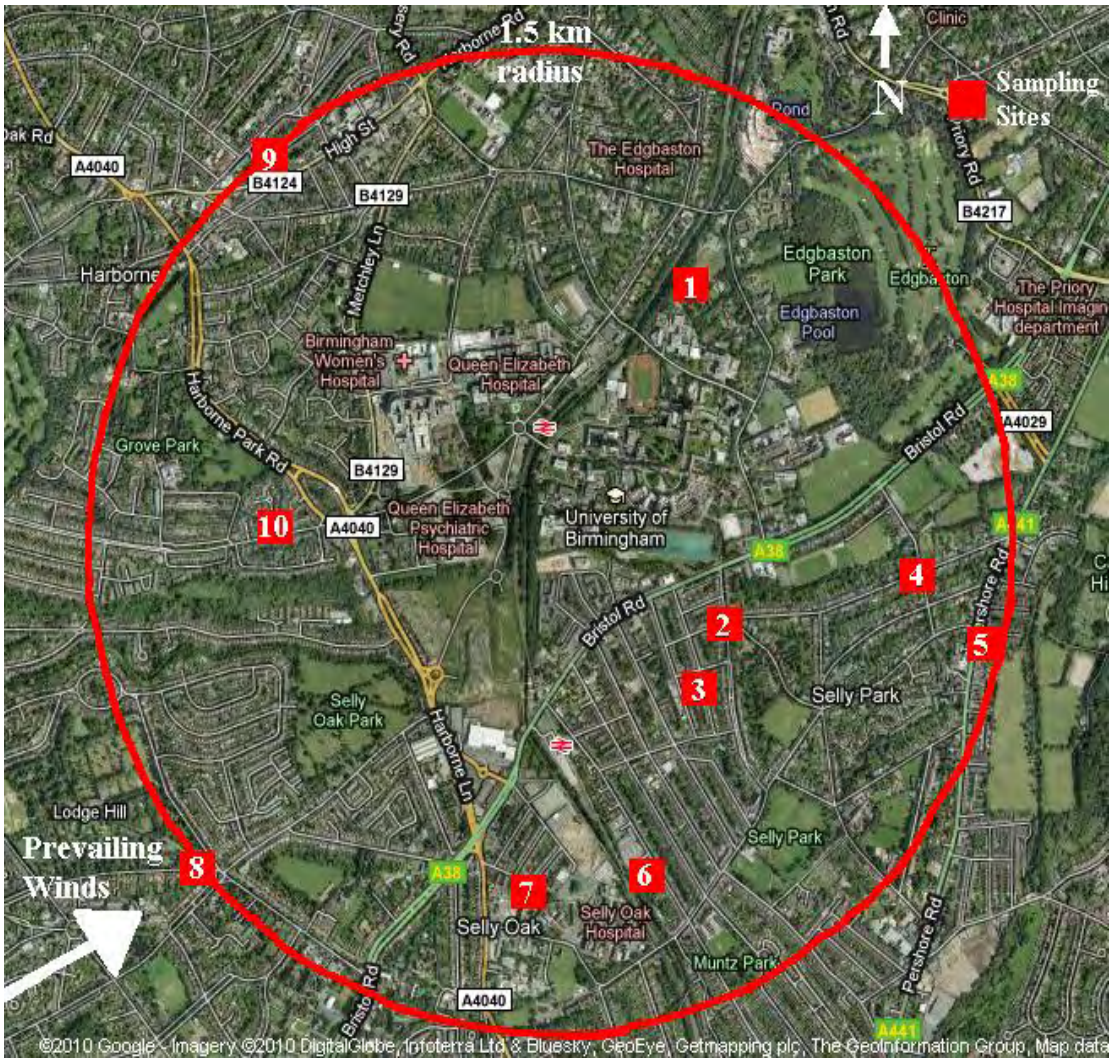


Figure 22 Location of outdoor air sampling sites for analysis of local scale spatial variability in Birmingham, UK.

Temporal variability in outdoor air was studied by deploying SIP air samplers monthly at 3 outdoor sites over the same period as the indoor temporal sampling; September 2008 – August 2009. Sites A and B were located in Birmingham, UK (numbered as site 1 and 7 in Figure 22), a large city with a population of >1 million people, whilst Site C was located in Oxfordshire (120 km SE from Birmingham, OS coordinates: 51.570868,-1.325054), at a semi-rural site. Samplers were deployed monthly and were left for a period of 30 (± 2) days.

Small scale spatial variations in outdoor air were measured in Birmingham, UK, within a 1.5 km radius, from 10 outdoor sites (Figure 22). Passive samplers were deployed in March 2009 for 30 (± 2) days at each of the ten sites. These samples were located at sites provided by acquaintances of the author, but were chosen to provide reasonable spatial coverage.

Samples for the outdoor air spatial variations were collected within (approximately) a 1.5 km radius of each sampling point, using SIPs samplers (details of SIP production and deployment are detailed in chapter 2). The samples were collected from the Selly Oak district of Birmingham city in the UK (see Figure 22). The area is situated 4.5 km southeast of the city centre, and is mainly a residential area, but includes three hospitals and the University of Birmingham main campus, and is influenced mainly by a south-westerly prevailing wind. This wind passes over small towns and rural regions before approaching the sampling area and is unlikely to be a prime source of PFCs to the urban region. Individual site concentrations are given in Table 57.

5.3.SPATIAL VARIABILITY

Spatial variations in outdoor concentrations are a result of many impacting factors, including weather and climatic conditions (Lui *et al.,* 2009 & Primbs *et al.,* 2008), and the general movement and behaviour of the contaminant (Paul *et al*., 2009). These factors can strongly influence the presence of compounds measured within a specified area. Spatial variations can occur from macro to micro scales, depending on the topography of an area and the population density (which is positively associated with an increased volume of potential PFC sources) (Kirchgeorg *et al.,* 2010, Murakami *et al.,* 2008, Pistocchi & Loos, 2009). Concentrations measured within densely built up areas, will be strongly influenced by the surrounding buildings and the protection or

vulnerability to prevailing winds and the temperature and weather conditions (microclimate) of a given location (Harrison, 2001). Thus, concentrations identified within one area, could be drastically different from that of a sample taken 200 m away, e.g. from an open park area, where atmospheric mixing is more homogenous, the area receives more sunlight and is influenced more by the prevailing winds.

These differences can occur on local scales, and therefore investigating the potential variability within a 1.5 km radius, will give a better understanding of the potential movement, and mixing of PFCs within the outdoor environment on a local scale within an urban region.

Long range movement of PFCs has been studied by Young *et al.,* (2007) Armitage *et al.,* (2009) and Wania, (2007), and has been shown to mimic that of many other atmospheric chemicals and POPs, which begin to cycle towards the Polar Regions, with the potential for LRAT.

Within urban areas, many chemicals become deposited onto the ground but, with little vegetation and soil, the chemicals are easily and quickly washed away, ending up in the local waterways (Kim & Kannan, 2007). However, those chemicals that do not get deposited (semi-volatile compounds such as the FTOHs), tend to move out towards rural regions (where concentrations are generally lower (Primbs *et al.,* 2008)), whilst interacting within the atmosphere and undergoing atmospheric degradation (Ellis *et al.,* 2004).

Table 57 Concentrations of PFCs in outdoor air, individual site results (pg m^{-3})

Location	OS Coordinates	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	52.454179, -1.929216	4.1	<DL	7.2	<DL	<DL	10	130	120
2	52.445495, -1.928616	1.3	2.7	11	<DL	59	15	13	34
3	52.443717, -1.929817	4.2	<DL	14	<DL	72	27	110	91
4	52.447117, -1.917973	1.6	<DL	30	41	170	27	72	110
5	52.445862, -1.913939	1.4	1.2	<DL	<DL	27	2.9	80	76
6	52.438014, -1.931362	<DL	<DL	<DL	<DL	54	<DL	3.4	38
7	52.437647, -1.937628	<DL	<DL	<DL	<DL	170	25	37	85
8	52.437647, -1.95857	<DL	3	<DL	<DL	150	10	64	20
9	52.459618, -1.951017	2.7	2.9	<DL	<DL	86	12	43	37
10	52.449131, -1.950374	6.1	20	4.9	12	98	4.6	27	120
%RSD		85	190	140	240	66	74	63	53

Sampling locations refer to those given in Figure 1
Samples <DL were included in the statistical assessment, and were represented by LOD/2.

Larger %RSD are produced by the less volatile compounds, PFOS, PFOA and PFHxS, and also for MeFOSA, which was detectable in 2 out of 10 samples (producing the largest RSD), all of which are likely to be present in the particulate phase (Barber *et al.,* 2007). These samples did not indicate a significant difference between any of the sites, and the apparently larger variations may be a result of the relatively low concentrations detected in the samples. As these compounds reside primarily in the particulate phase they may undergo less facile atmospheric mixing.

The semi-volatile compounds (MeFOSE, EtFOSE, EtFOSA and FOSA) exhibit slightly less spatial variation (%RSDs = 53 – 74 %). This may indicate better mixing of these primarily vapour phase compounds However, the more persistent compounds should show less spatial variability because of no atmospheric degradation pathways acting upon them, but this is not seen in the data, and may indicate the need for greater sampling numbers, to identify any true patterns of behaviour.

PFOA displays the greatest RSD (190 %) (ignoring the MeFOSA values because of low detection), with a range from <DL to 20 pg m^{-3}. Concentrations of PFOA in urban air have been associated with traffic volumes in cities by Harada *et al.,* (2005) and Kim & Kannan (2007). While PFOA has been reported previously to be the major contributor to particulate phase PFCs (Barber *et al.,* 2007 and Harada *et al.,* 2005), in this study, PFHxS was the predominant particulate phase PFC in most samples.

Elevated concentrations of less volatile PFCs have been detected in road dust from areas close to high traffic density (Yarwood *et al*., 2007). Moreover, concentrations of the more volatile compounds could also be effected by the proximity of the samplers to busy roads. Fluorotelomer compounds are degraded via reaction with NOx (Ellis *et al.,* 2004, Chiappero *et al.,* 2008, Sulbaek Andersen *et al.,* 2005), which can be abundant in locations with heavy-traffic, the degradation products consisting of the perfluorooctane sulfonamides and

sulfonamidoethanols. Locations closest to busy roads include: 4, 5 and 7. Their ΣPFC concentrations consist of the three highest values for the data set, and they are significantly different from the other sampling locations ($p = 0.02$, $F = 8.2$), when tested using an independent t-test and divided into two sampling groups: a, those situated alongside main 'A' roads, and b, those situated on routes with less traffic.

Correlations between compounds were examined. Significant correlations ($p < 0.05$) were identified for PFOS and PFOA, PFOS and EtFOSE, PFHxS and FOSA ($r > 0.64$) (see Figure 24). The origin(s) of these relationships are not fully understood, but in the case of PFOS and PFOA they may indicate common sources and/or common atmospheric transportation mechanisms. The other two correlations observed are between a primarily particulate-phase compound (PFOS and PFHxS) and a more volatile compound (EtFOSE and FOSA). In such cases, it may be that the former is a degradation product of the latter, giving rise to the correlations. Such correlations, between precursor compounds and the degradation products have been found previously by Nilson *et al.*, (2010), Fraser *et al.* (2010), and Freberg *et al.*, (2010).

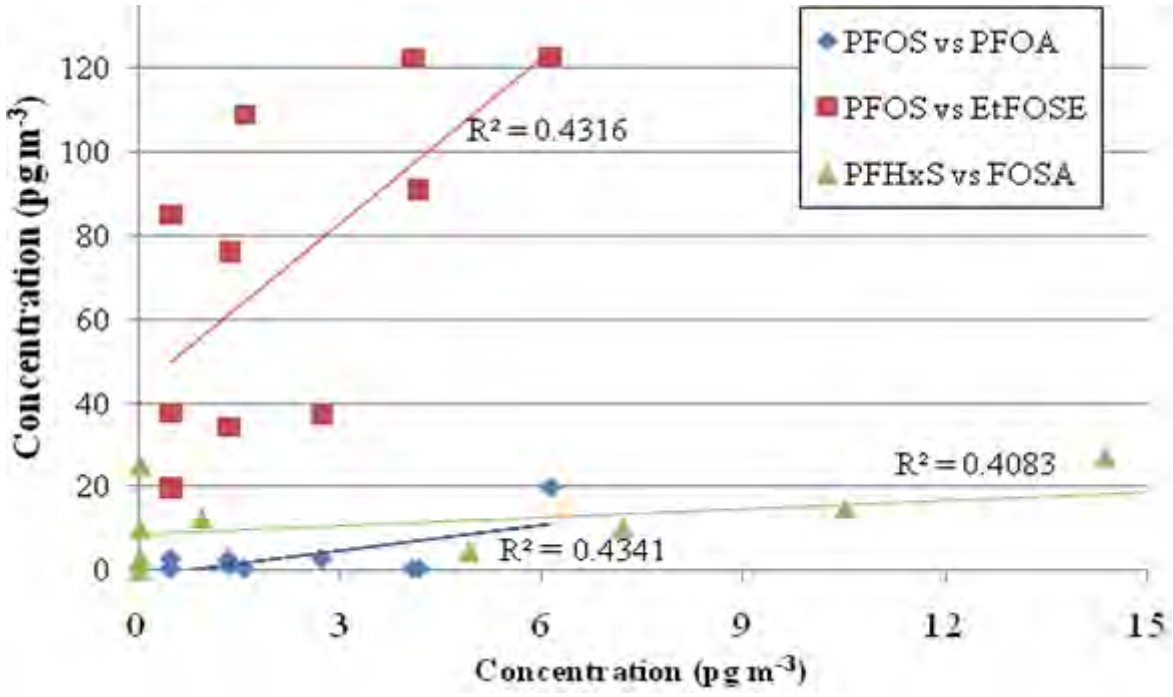


Figure 23 Correlations between different compounds (pg m^{-3})

5.4. OUTDOOR TEMPORAL VARIATIONS

Table 58 Variation in PFC concentrations in samples in temporal variability study (pg m^{-3})

Month	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
Site A				Urban background				
September	0.71	<DL	12	<DL	81	20	120	41
October	1.9	<DL	11	<DL	99	60	50	37
November	<DL	11	3.8	<DL	34	60	49	43
December	<DL	3.8	2.0	22	71	81	56	62
January	<DL	<DL	420	<DL	<DL	<DL	26	130
February	<DL	<DL	<DL	<DL	<DL	<DL	350	200
April	<DL	<DL	<DL	<DL	72	39	95	52
May	<DL	<DL	<DL	<DL	63	36	58	31
June	<DL	<DL	1.3	<DL	58	25	54	32
August	<DL	2.1	1.4	<DL	67	51	27	35
%RSD	**240**	**220**	**290**	**320**	**61**	**72**	**110**	**83**
Site B				Urban				
September	<DL	46	1.5	<DL	37	6.5	28	99
October	<DL	7.9	8.3	<DL	13	37	31	110
November	<DL	56	<DL	<DL	68	30	56	71
December	5.9	<DL	3.8	<DL	78	39	110	36
January	1.9	<DL	1.9	<DL	100	28	73	93
February	10	46	5.3	<DL	94	4.5	170	190
March	<DL	<DL	5.6	<DL	79	9.5	56	69
April	<DL	2.5	<DL	16	71	3000	68	98
May	7.1	<DL	12	<DL	40	45	39	120
June	<DL	<DL	<DL	<DL	72	45	55	85
August	16	24	20	43	140	6.5	60	100
%RSD	**140**	**140**	**120**	**250**	**48**	**300 (73)**	**60**	**39**
Site C				Semi-rural				
September	<DL	<DL	<DL	<DL	45	65	77	100
October	<DL	<DL	1.6	<DL	11	16	60	28
November	<DL	1.4	4.9	<DL	19	33	53	25
December	<DL	5.6	<DL	<DL	55	100	21	34
January	<DL	<DL	1.3	<DL	26	31	72	36
February	<DL	1.5	<DL	<DL	30	5.3	29	63
March	<DL	<DL	1.0	2.0	67	85	49	32
April	<DL	<DL	<DL	<DL	120	60	98	27
May	<DL	1.4	4.9	<DL	99	32	53	25
June	<DL	4.6	1.5	<DL	37	6.5	28	99
July	<DL	<DL	20	<DL	24	5.8	120	110
August	0.6	8.5	8.8	<DL	150	29	100	31
%RSD	**350**	**150**	**160**	**350**	**77**	**81**	**49**	**65**

On a local scale FTOH concentrations have been found to increase during periods of calm winds (Primbs *et al*., 2007) suggesting that, during months when wind speeds are lower (summer months), FTOH concentrations may increase and therefore could also cause an increase in the degradation products. A summer increase in concentrations was also noted by Dreyer *et al.,* (2009), but could not be related to local atmospheric conditions.

Temporal variations in PFC concentrations in outdoor air are displayed in Table 58. Like the results of the spatial variability study, variability (120 – 350 %), of the degradation compounds, PFOS, PFOA and PFHxS, along with MeFOSA, is greater than the precursor compounds. The relationship is presumably driven by the low concentrations of the degradation compounds and the comparatively small proportion of samples containing detectable concentrations, thus increasing the %RSD.

Table 59 Correlations between individual PFCs at a given site

Site	Compound A	Compound B	Significance, p	Pearson's correlation coefficient, r
A	EtFOSA	EtFOSE	<0.005	-0.81
	MeFOSE	EtFOSE	0.01	0.763
	EtFOSA	FOSA	<0.05	0.635
C	MeFOSE	PFHxS	0.03	0.632

Table 60 Compound correlations between sites

Compound	Site 1	Site 2	Significance, p	Pearson's correlation coefficient, r
PFHxS	B	C	0.011	0.729
MeFOSE	A	B	0.014	0.741

Table 61 Compound correlations with meteorological conditions

Compound	Site	Parameter	Significance, p	Pearson's correlation coefficient, r
PFHxS	C	Temperature	0.048	0.579
MeFOSE	A	Temperature	0.033	-0.673
	C	Rainfall	0.001	0.822
EtFOSE	B	Rainfall	0.014	-0.685
	A	Sunshine	0.038	-0.604

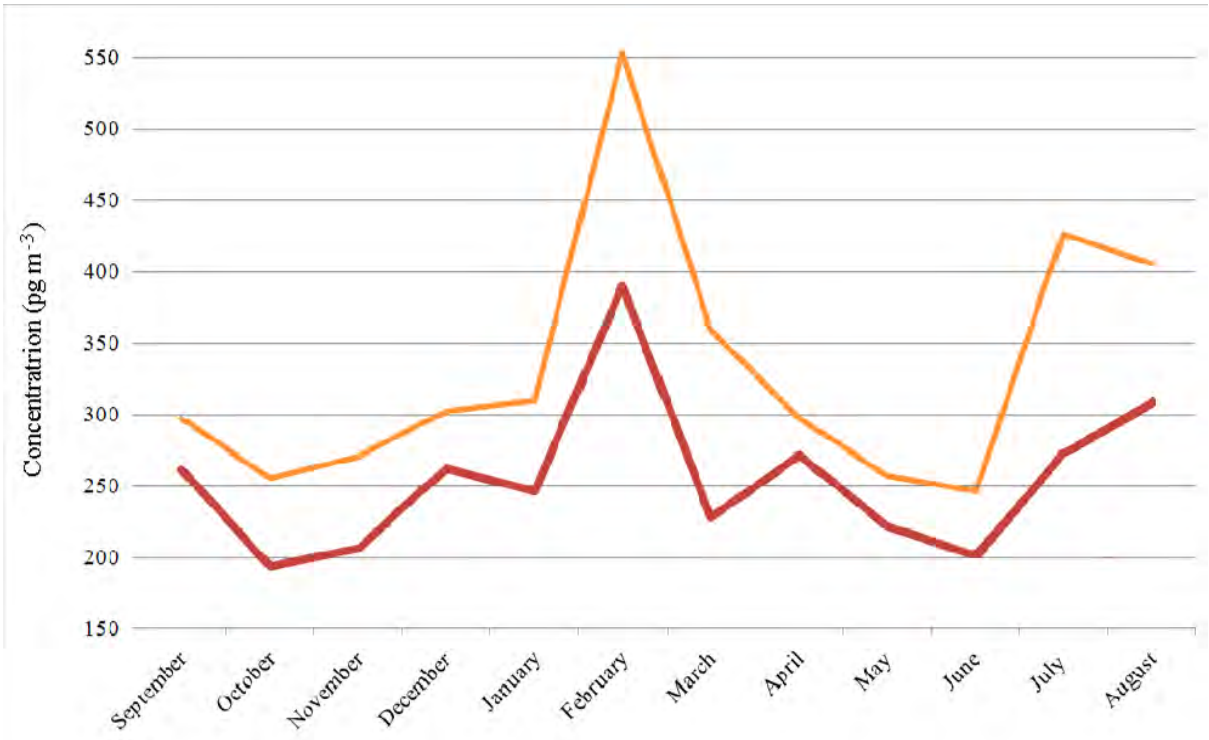


Figure 24 Mean ∑PFC monthly outdoor seasonal air concentrations (red) (pg m^{-3}) and the standard deviation (orange).

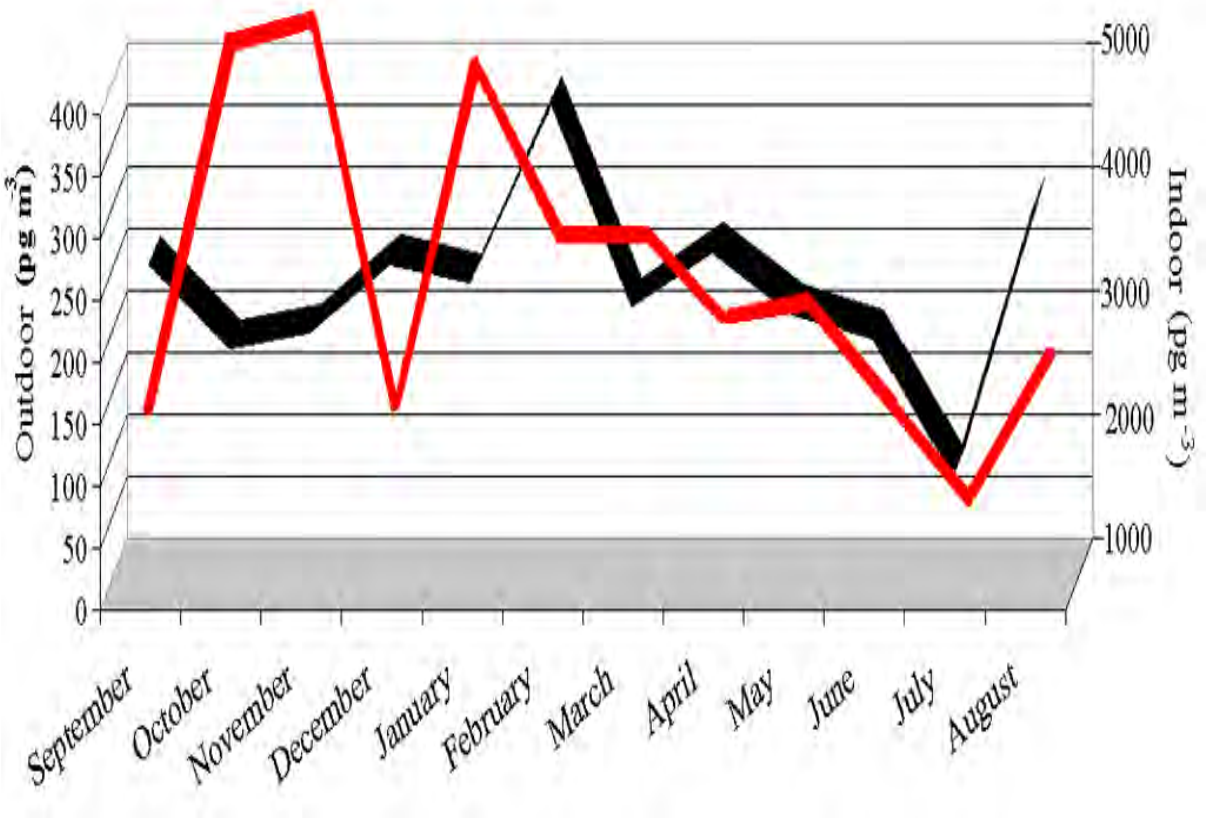


Figure 25 Indoor (red) and outdoor (black) average ∑PFC concentrations (pg m^{-3})

The less volatile and more stable compounds (PFOS, PFOA, PFHxS and MeFOSA) were either not detected (n.d.) in the samples, or were present at low concentrations. The variability for these compounds, due to this lack of detection for some samples, and the low concentrations, results in a large variability ranging between 120 – 350 %RSD for the three sites. Semi-volatile compounds were much more widely distributed in the samples, and their presence was detected in >97 % of the samples. The variability seen in the sites over the sampling campaign ranged between 39 – 110 %RSD (after the removal of site B's FOSA outlier). The outlier seen in the FOSA concentrations for April, from site B is extremely high in respect to concentrations detected in other samples from across the UK and Europe (Barber *et al.,* 2007, Dryer *et al.,* 2009, Jahnke *et al*., 2007a), and has been removed for statistical analysis to reduce the bias from this one sample. The cause of this extreme value is unknown, but could have arisen from an industrial output, particularly with the number of hospitals within close proximity to site B and the incineration of medical waste, however no cause can be concluded upon. The %RSD values for the temporal variations are similar to that measured in Northern Germany by Dreyer *et al.,* (2009), where a number of PFCs were measured over an 8 month period, and produced %RSD values > 100 % for each compound. The variability in the study by Dreyer *et al.,* (2009) was attributed to air mass origin, with peaks in concentration associated with two directions of air mass and is a variable sufficient to be the cause of the variability seen in this work.

The three sites do not appear to be influenced by the same parameters, and appear affected more by site-specific factors, except for a peak in the February ∑PFC concentration. The pattern of outdoor air concentrations resembles that of indoor air with MeFOSE and EtFOSE prominent, but with an additional predominance in the outdoor samples of EtFOSA and FOSA. Over the 12 month period, variations in outdoor air concentrations do not match those for indoor air, but show a general increase in concentrations from December to February

(Figure 24). Followed by a marked decrease in March leveling out (to approximately 200 pg m^{-3}) through June to December. The increasing concentrations starting during the three months starting from December could be a result of the increased concentrations within indoor environments, which begin from September, producing a lag effect on the outdoor concentrations. This would also explain the drop experienced in March, being an impact of the decrease in indoor air concentrations, which occur in December. The ΣPFC concentration differences between May to December are significantly different (t-test; p = 0.01) to those from January to April. From March onwards, the outdoor and indoor air concentrations indicate similar behavior, with decreasing concentrations, suggesting that they are both influenced by the same parameters, and do not indicate any lag in responses. However during the winter months ventilation from indoor environments will be minimal, and may be a cause of this lag effect.

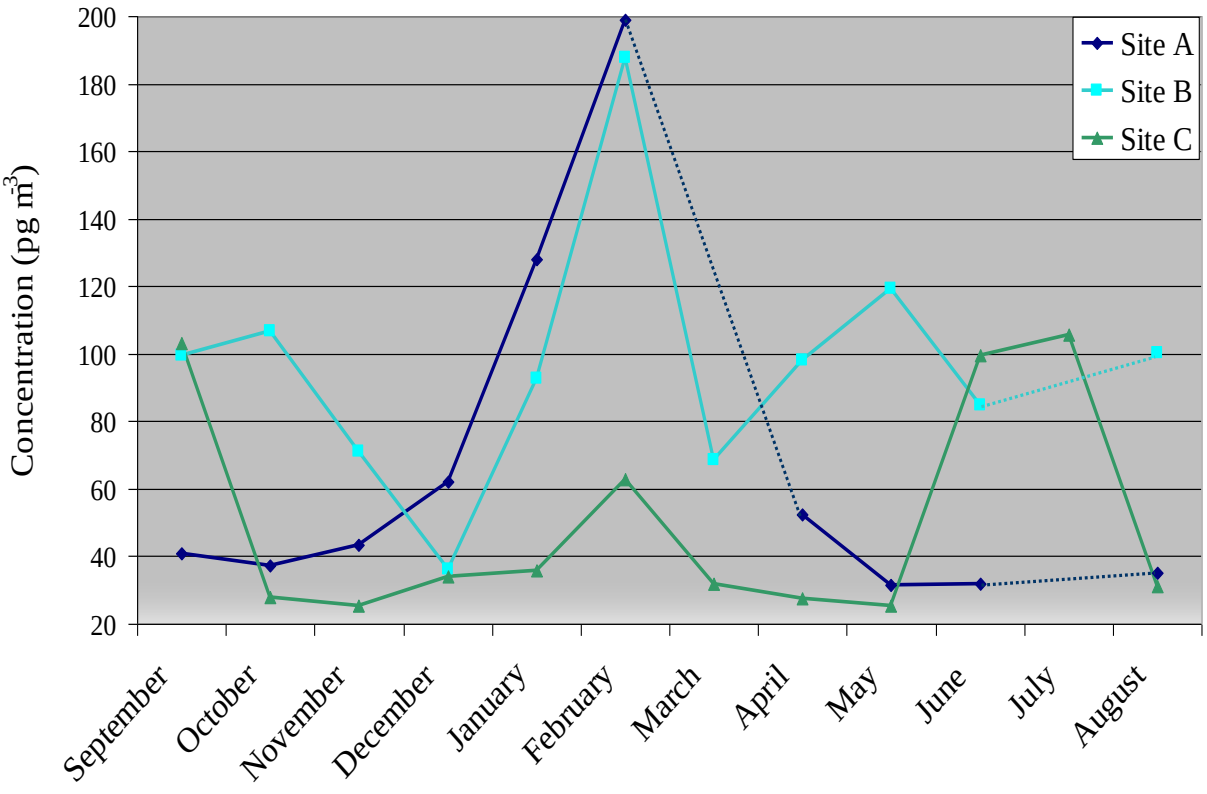


Figure 26 Temporal variations in ΣPFC concentrations in outdoor air at three sites (pg m^{-3}), missing data at sites A and B for July and at site C for March.

In Figure 26 concentrations of ΣPFCs at the three sites appear to all be affected by an event in February. This peak event was principally driven by MeFOSE and EtFOSE for site A and B, as well as by EtFOSA at site C, and to a lesser extent at site B. Indoor air was seen to rise the month before indoor microenvironments, and could be venting from indoors, impacting the outdoor air (see Figure 25).

Back trajectories were examined for the month of February and July (Figure 27) and compared to the trajectories from the other months. Air mass back trajectories were computed using the Hysplit 4.8 model, using the National Centres for Environmental

Prediction (NCEPs) Global Data Assimilation System (GDAS) data with 1º latitude/longitude resolution provided by the National Oceanic and Atmospheric Administration (NOAA)-Air Resources Laboratory (Draxler & Rolph, 2003). Hysplit was used to model 96 h back trajectories with 6 h intervals at a height of 1.5 m above ground level (this was the height of the samplers) for the individual days over the sampling period. Results for the trajectories are displayed in Figure 27, for the month of February (highest concentrations) and July (lowest concentrations). The back trajectory for February is driven across long distances (fast, movement), spending a high proportion of time over Eastern continental Europe, picking up and transporting potential source releases. The air masses reaching the UK from Eastern Europe have also been linked with the transboundary movement of air pollutants causing acidification of lakes in the UK (Krewitt *et al.*, 1998), suggesting that this is a plausible mode of transportation of PFC plumes to the UK. A number of trajectories are also based around the UK, France and the Netherlands, spending the 96 h prior to arrival over populated areas, and high traffic density air space and coastal regions. For July the air mass trajectories spend a large amount of time over the UK land and coastal regions with slow moving, low altitude air masses arising from the South West, over the mid-Atlantic. The main scenario which can be drawn from this is that, where concentrations were high in February, the air masses

traversed across continental Europe prior to reaching the site, whilst during the low concentration from July, air masses originated from short distances out in the Atlantic ocean. Other months saw air masses originating from a variety of locations with relatively long trajectories, and a greater presence of air masses from the Eastern Atlantic ocean, as well as Europe.

In comparison to the high February concentrations and the back trajectories travelling long distances (primarily across land), the July air masses tend to originate from the South West or due West of the UK in the Atlantic Ocean. The low concentrations measured, can be because the air masses traversed few contamination sources during the approach to the sampling sites and their low speeds. In comparison to the rest of the year, air masses for the two months, February and July, are distinctly different, and suggest that the anomalous PFC concentrations observed in these months is driven by the back trajectories of the air masses.

Apart from the February and July events coinciding for the three sites, inter-site correlations for different PFCs were sparse. Those that were detected (see Table 59) (compounds that were detected in < 50 % of the samples were not included in the analysis) indicated that there was some correlation between stable compounds like PFOS and PFOA, and their precursors. At site A, EtFOSA was correlated with FOSA (positively) and EtFOSE (negatively). EtFOSE can degrade via the presence of free radicals (Schenker *et al.,* 2008) to EtFOSA, which can proceed to degrade to FOSA (Armitage *et al.,* 2009). The atmospheric lifetimes of EtFOSE, EtFOSA and FOSA are of the order of 20 - 50 days (Martin *et al.,* 2006).

The degradation pathways suggest degradation of EtFOSE to ethyl perfluorooctane sulfonamide acetate that during events of high OH and other free radical concentrations there is (EtFOSAA) and EtFOSA, and also EtFOSAA degradation to EtFOSA, see Figure 28 (Plumlee *et al.,* 2009). EtFOSAA and EtFOSA may also undergo degradation to FOSA (Plumlee *et al.,* 2009).

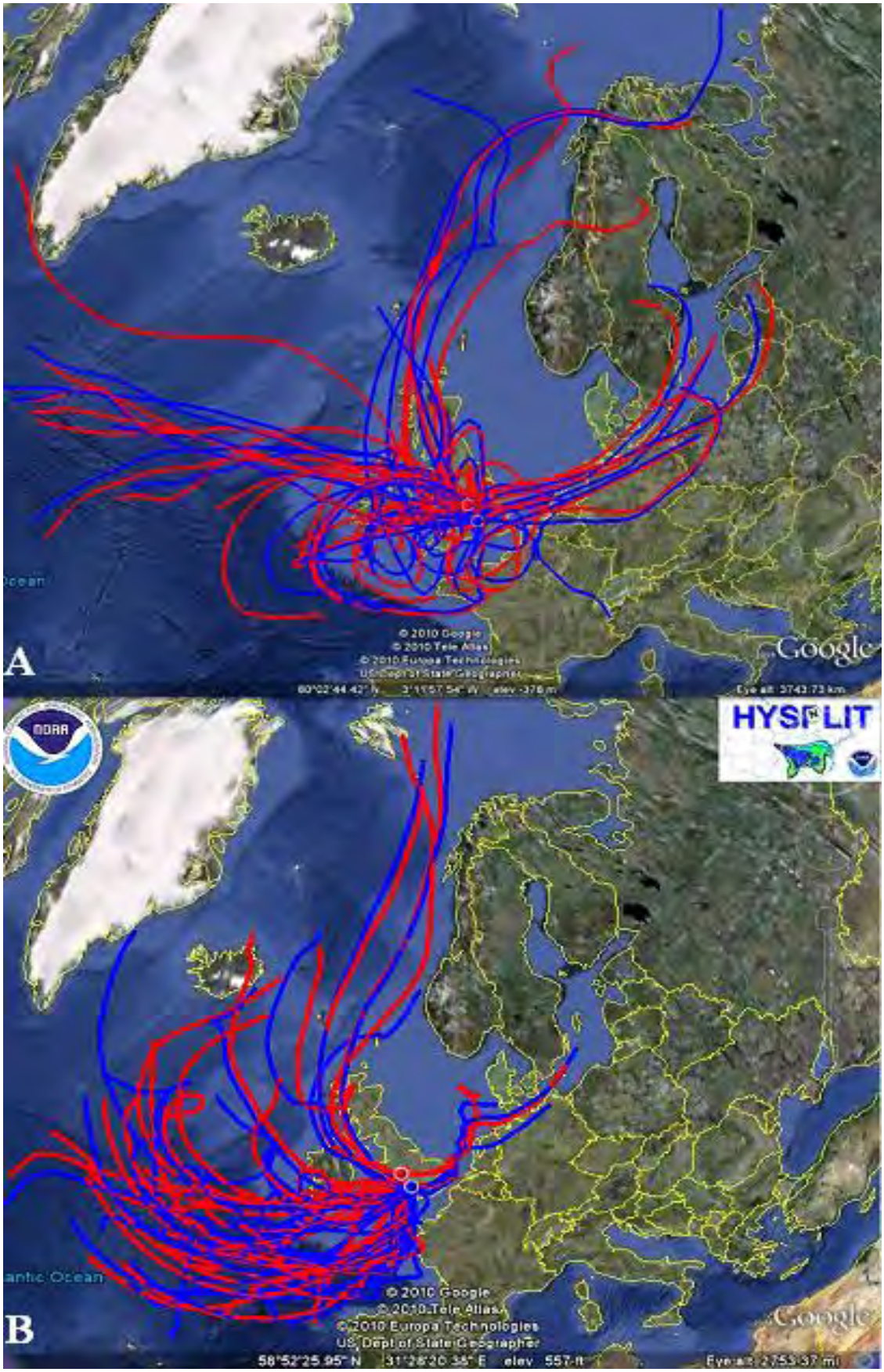


Figure 27 A, February and B, July 96 h back trajectories (red – Birmingham trajectories, blue- Harwell trajectories)

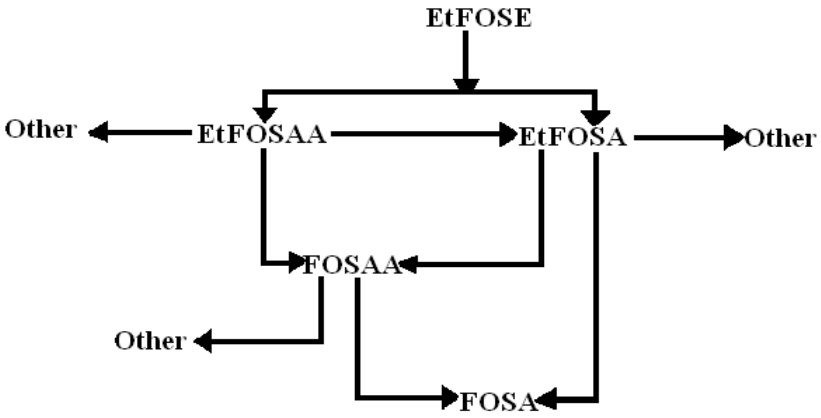


Figure 28 Degradation routes via OH radicals for selected PFCs (adapted from Plumlee *et al.* 2009).

The presence of perfluorooctane sulfonamides and sulfonamidoethanols could also be related via the emission sources as common residual compounds from fluorotelomer chemicals (Dinglasan-Panlilio & Mabury, 2006). The presence of a positive correlation between MeFOSE and EtFOSE can indicate the presence of the compounds derives from the same sources or that they are both affected by the same parameter. When these two compounds (from site A) were analysed with meteorological conditions, both displayed a negative correlation with one such parameter (see Table 61); EtFOSE a negative correlation with sunlight, and MeFOSE a negative relationship with temperature. Though these parameters are different, it does appear that perfluorooctane sulfonamides are influenced by meteorology. In addition to these relationships at site A, at site C the precursor compound MeFOSE is positively correlated with temperature and a degradation product (PFHxS) positively correlated with rainfall.

A further observation is that concentrations of PFHxS and MeFOSE at site B were correlated positively with those from site C. Despite these relationships having strong R values (0.73 and 0.74 for PFHxS and MeFOSE respectively), the fact that sites B and C are 120 km apart means these correlations are hard to explain. Seasonal variability has been noted by Stock *et al.,* (2005) in the partitioning behaviour, with MeFOSE principally remaining in the gaseous

phase in the summer, with partitioning to particles in winter months. Klánová *et al.*, (2008) also identified this behaviour of particulates, with greater collection of particulate matter during periods of colder ambient temperature. However, the observed MeFOSE concentrations in this study are not significantly different in the summer than the winter (Figure 29). This may be due to factors in addition to temperature influencing the data.

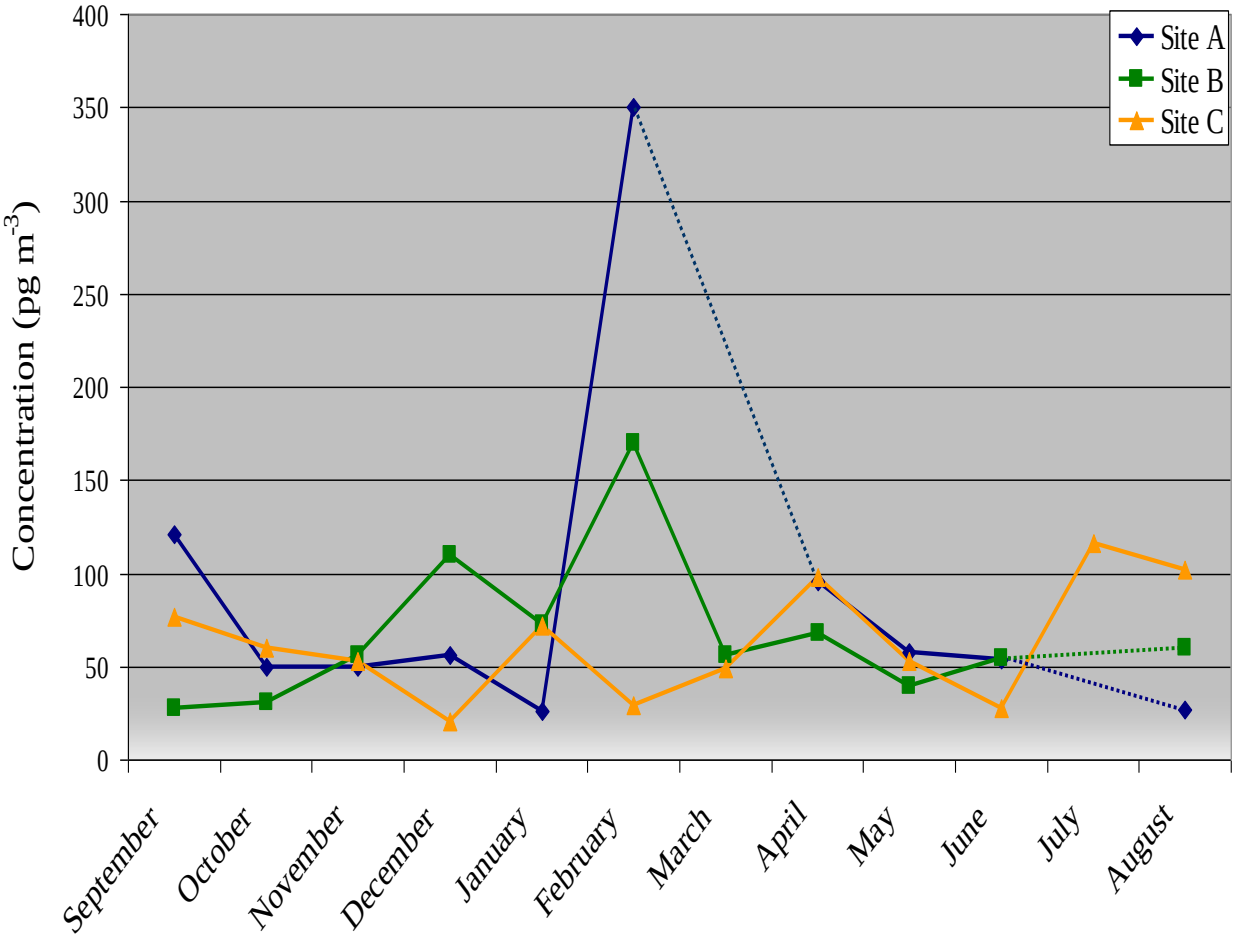


Figure 29 MeFOSE temporal concentrations (pg m^{-3})

Average climatic conditions were compared to the sites and relationships for PFHxS, MeFOSE and EtFOSE, from some of the sites, were identified (Table 59). PFHxS from site C was positively correlated with monthly average temperature (r = 0.58). However, particulate phase concentrations have been shown to be inversely related with temperature because of partitioning into the gaseous phase (Shoeib *et al.*, 2006), yet within this study both particulate

and varpour phase were measured, due to particulate deposition onto the SIP disks. Concentrations of PFHxS have only been measured in Europe in the particulate phase (Barber *et al.,* 2007, Dreyer *et al.,* 2009b) but concentrations of organohalogens such as PCBs, PCB-like dioxins and furan, and PBDEs (Su *et al.,* 2007, Ogura *et al.,* 2004, Mandalakis *et al.,* 2008) have been shown to be correlated positively with temperature as a result of an increase in diffuse sources, such as volatilisation from lakes, soil and vegetation (Klánová *et al.,* 2006).

At site A, negative correlations were identified between MeFOSE and temperature ($r = 0.6$, $p = 0.04$), and between EtFOSE and hours of sunshine ($r = 0.58$, $p = 0.04$). Though these two compounds were positively correlated with one another at site A, they appear to have different responses to the meteorological conditions. The correlations for the two compounds are related with temperatures, often improving with increasing hours of sunshine. The impact of meteorological conditions can be masked by the effects of simultaneous variations in other parameters, and, despite the presence of such relationships not being present in the statistical analysis, they may still be influencing concentrations. Further correlations were identified for MeFOSE and EtFOSE at site C and B respectively that were related to rainfall. MeFOSE at site C was correlated negatively with rainfall ($r = 0.5$, $p = 0.05$), whilst at site B EtFOSE is correlated positively with rainfall ($r = 0.59$, $p = 0.03$). The difference in these relationships with rainfall is not clear. A negative correlation is explicable in terms of enhanced scavenging and wet deposition (Dreyer *et al.,* 2010). In contrast the positive relationship between ETFOSE and rainfall is less easy to explain and indicates either that other factors associated with rainfall are influencing EtFOSE concentrations, or that this apparent correlation is an artifact.

5.5. CONCLUSIONS

The presence of both temporal and spatial variability in the data indicates a difference in atmospheric behaviour between the less volatile and stable PFCs (group 1; PFOS, PFOA, PFHxS and MeFOSA) and the more volatile precursor compounds (group 2; EtFOSA, FOSA, MeFOSE and EtFOSE). For example, group 1 (having higher concentrations of the less-volatile compounds) reside principally in the particulate phase and prefer to deposit out of the atmosphere via wet and dry deposition (Dreyer *et al.*, 2010, Prevedouros *et al*., 2006 and Kim & Kannan, 2007). Group 2, containing lower concentrations of the less-volatile compounds indicate that they are more likely to undergo mixing, which is consistent with their vapour pressures. Whilst on a microscale it is seen in the data that considerable variability is possible, however there is greater variation between the less volatile compounds at sites, than seen in the more volatile compounds. This bevahiour is also true for the variability in temporal concentrations, and was noted at each of the three sites. The reasoning behind this behavior is the volatility of the compounds, whilst those compounds that are more volatile are more efficiently mixed, the less volatile compounds are not due to being present as primarily suspended particulates.

The difference between the two groups is illustrated by the %RSD for both the spatial and temporal data sets. For group 1, the %RSD values range between 85 – 350 % for both spatial and temporal variability, whilst group 2 RSD values range between 39 -110 %. Group 2 compounds primarily remain in the gaseous phase and undergo more atmospheric mixing, transportation and dispersion (Lohman *et al.*, 2007). Their presence in the gaseous phase, led them to be a lot more variable in terms of short-range microscale spatial variability (1.5 km radius) and microscale meteorology. The compound variability was between 53 - 74 %, much less than that seen in a German study of spatial and temporal variability (Dreyer *et al.*, 2009).

The %RSD values for temporal variation were also less than in the German study (Dreyer *et al.,* 2009) with values remaining between 39 – 110 %.

The spatial variability was influenced strongly by the proximity of the sampler to a main road, with high congestion rates. The three sites located close to main roads displayed statistically higher concentrations of PFCs than in the other samples, and it is possible that increased concentrations could be driven by raised degradation rates of FTOHs (Ellis *et al.,* 2004) and suspension and turbulence of air created in the street canyons (Yarwood *et al.,* 2007). The mechanisms involved in this are increased degradation rates caused by vehicle emissions and the presence of free radicals, along with the proposed presence of PFCs in vehicle oils and other surfactants used (Drobny *et al.* 2005).

Temporal variability for group 1 compounds (PFOS, PFOA, PFHxS, MeFOSA) exceeded substantially their spatial variability. The data ranged between 120 – 350 % with minimal detection of the compounds (the group has a mean of 11.4 pg m^{-3}). The broadrange of values is not fully understood, but in some samples the presence of MeFOSA was present and these samples ranges between 2 - 43 pg m^{-3}, however were compared to the non-detected samples represented at half the limit of detection. RSD values for group 2 compounds (EtFOSA, FOSA, MeFOSE and EtFOSE) fell in the range 39 – 110 %, which also exceeds their spatial variability. The data were analysed for evidence of temporal changes affecting concentrations. The sites revealed a small amount of correlation (significant, $p < 0.05$) between compounds within sites, between sites and with meteorological conditions. The outcome of this analysis indicates that some differences exist in behaviour between sites and between different PFCs. Factors influencing PFC concentrations in a temporal fashion, were the meteorological conditions, the potential degradation pathways of precursor compounds and as seen with the spatial variability, the locality of the samplers and the surrounding environment and the air mass trajectory profiles.

No particular seasonal influences on PFC concentrations were observed. Site A showed a decrease in concentrations for the final quarter of the campaign; this was not seen in the other sites, including the other Birmingham site (B), 1.5 km from site A. Though the concentrations of PFCs in the air is highly influenced by the air mass back trajectory, and the presence of numerous sources. Within the data it is possible to identify increased concentrations associated with back tracjectories originating over European land masses, whilst lower concentrations are usually derived from air masses originating from the Atlantic (where fewer sources are present).

The temporal variability observed exceeded that attributable to the analytical methods, and thus the data in this chapter suggest that assessment of exposure via inhalation of outdoor air will be influenced by when sampling is conducted to improve the accuracy of the exposure assement and determination of whether outdoor inhalation is a significant pathway of exposure to PFCs. Exposure assessments should thus be based on a large sample set, collected from a number of locations throughout the year. This should therefore reduce the impact of peak events (seen in February).

A final observation is that higher concentrations were detected in samples collected at locations closer to busy roads. This suggests that exposure via inhalation may be greater for subjects spending more time near to highly trafficked roads, and that on the microscale there is likely to be a difference in the concentrations created by this relationship.

6. UK SOILS

The hypothesis to be addressed in this chapter is that UK soils are a substantial reservoir of PFCs. Hence, this chapter's aim is to determine concentrations of PFCs in soil samples from a number of UK locations. This chapter reports for the first time concentrations of PFCs in UK soils. These data will provide an indication of the extent to which soil represents a reservoir of PFCs. This is important as contaminants in soil are available for human exposure via a variety of pathways including groundwater, uptake via plants and animals grown/reared for food, and via direct ingestion of soil.

6.1. INTRODUCTION

This chapter details an investigation of the concentrations of PFCs in surface soil samples from across the UK. As there is currently no published data available for the UK on this topic, these data will provide a valuable benchmark for future studies. Soils have been noted as a potential sink for PFCs (Nakata *et al.*, 2006), as well as a pathway to groundwater contamination (Moody *et al.*, 2003), vegetation and the food web (Stahl *et al.*, 2009). The importance of soil concentrations when considering human exposure to PFCs is primarily the impact via the food web or movement into drinking water derived from groundwater supplies. However, a small amount of soil is also ingested from hand-to-mouth contact when outdoors, and via inhalation of suspended soil particulates (Scott & Proctor, 2008). Two additional pathways to humans include direct ingestion from soil-covered vegetation (though this is very minimal) and the second is via direct ingestion, following a behavioural disorder (Pica) – which drives people (particularly children) to eat soil and dirt (the exposure assessment will only take account of the behaviour of the general population).

There are three primary sources of PFCs to soil, the first being atmospheric deposition (Cousins *et al.,* 1999), the second is the anthropogenic application of materials containing PFCs (biosolids and insecticides (Holzer *et al*., 2008, Wilheim *et al.,* 2007)), and the third is leaching from landfill sites and contaminated water (Eggen *et al.* 2010, Moody *et al.* 2002). Little is presently known about the current concentrations in soils and the exchange between external media and soil. Present research addresses the retention capability of PFCs in soil, and the microbial degradation that can occur (Gledhill *et al.*, 2000). At present it is believed that biota within soils acts as a sink to PFOS and other PFCs, rather than them being emitted to the atmospheric environment (Sanderson *et al.* 2002).

The ability of soil to retain chemicals is dependent upon a large number of parameters, which affect air – soil partitioning, soil – water partitioning, rate of volatilisation, lifetime and microbial degradation, as well as leaching and transportation (Strynar *et al.*, 2009). These parameters include: organic matter content, moisture content, structure, porosity, acidity, texture, and relative humidity of the soil (Jia *et al.*, 2010, Liu & Lee, 2005, Higgins & Luthy, 2006, Johnson *et al.*, 2007).

For other organohalogen compounds the physico-chemical properties of the compounds have determined the exchange between various environmental compartments (Lohmann *et al.,* 2007). As discussed in chapter 5, atmospheric transportation of PFCs is influenced strongly by the volatility of the compound, and thus the gas – particulate partitioning.

6.2.PFCS IN SOILS

The environmental fate of PFCs and other organohalogens can be significantly influenced by biodegradation (Dinglasan *et al.,* 2004). Analysis of sewage sludge has also been studied as a viable removal route of PFCs from waste water, and also with respect to the impact of spreading sludge on agricultural land (Díaz-Cruz *et al*., 2009). Higgins *et al.*, (2005)

identified the primary PFC content of sewage sludge to consist of PFOS, PFDS, MeFOSAA, PFOA and PFDA, at concentrations of low ng g^{-1} to low µg g^{-1}. The predominant presence of longer-chain PFCs, including PFOS and PFOA was also noted in soil and sediment from South Korea and Japan (Naile *et al.*, 2010, Nakata *et al.*, 2006). PFOS was noted by Jia *et al.*, (2010) to be well retained by humic acids, with high sorption capacity between 5 – 35ºC, which is of particular relevance to the retention of PFOS in UK soils.

More volatile compounds are also retained by soil, and reach the soil interface via wet and dry deposition. Soils dried indoors were exposed to FTOH, producing measurable concentrations of 6:2 – 14:2 FTOH compounds (Ellington *et al.*, 2009). FTOH are also transmitted to soils via the use of contaminated biosolid sludge on agricultural land, as well as wet and dry deposition (Dreyer *et al.*, 2010)

Table 62 indicates the potential for PFOS to be retained by soil (distribution coefficient, K_d) and the higher values for the desorption kinetics (desorption coefficient, K_{des}) suggesting the limited movement of PFOS once present in the soil (Beach *et al.*, 2006).

Table 62 Beach *et al.* (2006) adsorption and desorption of PFOS to soils, sediments, and sludge[a].

Soil type	**Adsorption Kinetics K_d (L/kg)**	**Desorption Kinetics K_{des} (L/kg)**
Clay	18.3	47.1
Clay loam	9.72	15.8
Sandy loam	35.3	34.9
River sediment	7.42	10.0
Domestic sludge	<0.120	<23.7

[a]Values of K_d and K_{des} are averaged values.

6.3.BIODEGRADATION OF PFCS

The main factor affecting the bio-degradation and remediation of PFCs is their strong carbon-fluorine bonds, which limit the type of microbes that can utilise the compounds. In order for microbes to utilise the PFC, they must first be able to remove the PFC functional group (Parsons *et al.,* 2008). The limiting factor is the amount of energy required to split the carbon-fluorine bond for microbial dehalogenation to occur as seen with other organohalogen contaminants (e.g. PCBs, dioxins and furans, and PBDEs) (Dolfing, 2003, Bunge & Lechner, 2009, Robrock *et al.*, 2008).

The biodegradation of more volatile compounds, such as FTOH and polyfluoroalkyl phosphates (PAPs) can be achieved (Wang *et al.,* 2005, Lee *et al.,* 2010) in relatively short time scales (half life = 0.2 days mg^{-1} of initial mass protein, (Dinglasan *et al.,* 2004)). Degradation can result in metabolites, such as PFCAs (Liu *et al.,* 2010), and less volatile compounds, which are more persistent within the environment than the parent PAPs (Conder *et al.,* 2008, Butt *et al*., 2009).

The biodegradation of PFOS in soils has been shown to be facilitated by biomolecules (vitamin B12), which also facilitate the dechlorination of PCBs (Assaf-Anid *et al.,* 1992)) under anaerobic conditions (Ochoa-Herrera *et al.,* 2008). However, this process is favoured for branched PFOS molecules, leaving the more bioaccumulative L-PFOS structure present in the environment (Houde *et al.,* 2008). Other research has indicated that under general conditions biodegradation of PFOS cannot occur under aerobic conditions (Gledhill and Markley, 2000).

The biodegradation of the more resistant compounds, PFAS and PFCAs, is less well understood, however, it is thought to be dependent upon the length of the carbon chain.

Recent work by Liou *et al.*, (2010) indicated that under conditions which induce degradation in PCBs (methanogenesis and other inducers of co-metabolism, (Ye *et al.*, 1995)), no alteration of the PFOA structure occurred.

6.4.BIOREMEDIATION OF PFCS

Research by Pan *et al.* (2008) indicated that the presence of cationic surfactants could increase the sorption of PFOS to sediment, immobilizing the compound and retaining it within the soil matrix. The opposite behaviour was possible in the presence of anionic surfactants which promote the movement via solubility of the compound in sediments. This behaviour of PFOS with respect to surfactants is facilitated by the presence of hydrophilic and hydrophobic moieties, which are also responsible for the variety of uses of PFOS in manufacturing. The ability to retain and release PFOS in soils allows the surfactants to be applied to contaminated soils to facilitate clean-up (Pan *et al.*, 2008). Future applications via bioremediation using plants (phytoremediation) may be an applicable removal technique (Stahl *et al.*, 2009, Beach *et al.*, 2006).

6.5.MOVEMENT IN SOIL

The movement of PFCs from soils can be affected via leaching into groundwater (Moody *et al.*, 2003, Murakami *et al.*, 2009, Cheng *et al.*, 2008, Davis *et al.*, 2007), runoff into waterways (Hölzer *et al.*, 2008) and uptake by vegetation (Stahl *et al.*, 2009).

The movement of PFCs from agricultural land to local waterways, has been seen in both Germany and the USA, where contaminated human biosolids were used as fertiliser (Wilhelm *et al.*, 2009, Renner, 2009). In this way, the runoff from contaminated agricultural land enters the local surface waters (Gottschall *et al.*, 2010), and thus into the drinking water

supply, where concentrations can be raised significantly (Hölzer *et al.,* 2008, Hölzer *et al.,* 2009).

The retention of PFCs in soil is dependent upon the properties of the soil, but groundwater concentrations have been identified to contain the shorter chain molecules; greater retention occurring with chain length (Murakami *et al.,* 2009). Five years after the last known use of AFFF, concentrations of C6 and C8 compounds were found in groundwater surrounding a fire-training area in the USA, indicating the current presence of PFOS, PFOA, PFHxS and PFHxA (perfluorohexanoic acid) (Moody *et al.*, 2003). PFOS cane be retained by surface soils, preventing it from directly reaching the groundwater (Moody & Luthy, 2006), however the percultaion of other more volatile PFCs through soils whilst undergoing microbial degradation and transformation to PFOS, resulting in contamination of the groundwater.

The uptake of PFOS from soil through vegetation and megadriles is a pathway of the compound out of the soil, with plants retaining concentrations 1 – 2 fold greater than in soils (Beach *et al.*, 2006, Stahl *et al.*, 2009).

6.6.SAMPLE COLLECTION

UK archived soil samples (n = 42) were collected from surface soils across the country during the summer of 2004 (see Figure 30). Each sample was collected from a 10 by 10 m grid; a random three grid points were selected and samples from the top 5 cm were collected. The three samples from the grid were then homogenised and stored together in amber glass jars (pre-cleaned with DDW and DCM and sealed with aluminium foil lined lid) and stored in a cold room at 4°C until use. For further details regarding the extraction and analysis methods, see chapter 2. Soil sample concentrations and descriptive statistics are shown in Table 62, which indicate that the main compounds identified in the soil are PFOS, PFOA and PFHxS. The results from the UK soils indicate that PFCs are clearly entering soil, although

the exact pathways via which this occurs are not clear However, they are likely to arise from processes such as wet and dry atmospheric deposition, and spreading of biosolids (on agricultural land).

Figure 30 Soil sample locations

6.7.RESULTS AND DISCUSSION

Table 63 Soil concentrations (pg g^{-1})

Sample	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	∑PFC
1	260	22	1000	<DL	<DL	<DL	<DL	<DL	1500
2	2400	1700	<DL	<DL	<DL	<DL	<DL	<DL	4200
3	1500	1700	780	<DL	<DL	<DL	<DL	<DL	4100
4	910	900	<DL	<DL	<DL	<DL	<DL	<DL	2000
5	510	990	420	<DL	<DL	<DL	<DL	<DL	2100
6	410	1900	650	<DL	<DL	<DL	<DL	<DL	3100
7	370	<DL	480	<DL	<DL	<DL	<DL	<DL	1000
8	120	<DL	<DL	<DL	<DL	<DL	<DL	<DL	300
9	170	1700	<DL	<DL	<DL	<DL	<DL	<DL	2000
10	310	2600	<DL	<DL	<DL	<DL	<DL	<DL	3000
11	580	4600	470	<DL	<DL	<DL	<DL	<DL	5800
12	280	1400	780	<DL	450	<DL	120	70	3200
13	630	8000	1000	<DL	210	<DL	93	53	2900
14	540	1100	<DL	<DL	<DL	<DL	<DL	<DL	1800
15	2400	<DL	<DL	<DL	<DL	<DL	<DL	<DL	2600
16	1300	1100	<DL	<DL	<DL	<DL	<DL	<DL	2600
17	<DL	5200	280 000	51 000	<DL	<DL	<DL	1000	340 000
18	680	<DL	1100	<DL	<DL	<DL	550	150	2600
19	840	<DL	<DL	<DL	<DL	<DL	<DL	<DL	1000
20	1600	<DL	670	<DL	<DL	<DL	270	69	2800
21	1200	1100	1000	<DL	<DL	<DL	<DL	28	3400
22	1900	1500	<DL	<DL	<DL	<DL	<DL	<DL	3500
23	130	<DL	<DL	<DL	<DL	<DL	<DL	<DL	310

24	770	2700	<DL	<DL	<DL	<DL	<DL	<DL	3800
25	<DL	<DL	370	<DL	<DL	<DL	<DL	<DL	540
26	350	<DL	<DL	<DL	<DL	<DL	<DL	<DL	520
27	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL	180
28	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL	180
29	300	6900	<DL	<DL	<DL	<DL	<DL	<DL	7300
30	1100	2500	<DL	<DL	<DL	<DL	<DL	<DL	3700
31	840	2300	<DL	<DL	<DL	<DL	<DL	<DL	3300
32	220	<DL	<DL	<DL	<DL	<DL	<DL	<DL	390
33	580	<DL	<DL	<DL	<DL	<DL	<DL	<DL	750
34	220	<DL	560	170	160	<DL	32	<DL	1200
35	2200	6700	1600	<DL	<DL	<DL	63	20	11 000
36	2200	5200	400	200	2300	<DL	<DL	51	10 000
37	510	2000	<DL	<DL	<DL	<DL	<DL	<DL	2600
38	910	53 000	<DL	<DL	<DL	<DL	<DL	<DL	54 000
39	9200	7900	940	<DL	<DL	<DL	<DL	<DL	18 000
40	<DL	<DL	620	<DL	<DL	<DL	33	18	800
41	150	<DL	13 000	<DL	<DL	<DL	<DL	<DL	13 000
42	1700	11 000	<DL	<DL	<DL	<DL	<DL	<DL	13 000
Detection rate (%)	37	26	19	3	5	1	7	9	
Minimum	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL	180
5th Percentile	8	22	14	55	32	12	24	15	300
Median	560	1100	14	55	32	12	24	15	2700
Geo-Mean	390	380	100	69	43	12	30	21	2600
Arth-Mean	960	3100	7300	1300	110	12	47	47	13 000
95th Percentile	2400	7900	1600	170	210	12	120	70	18 000
Maximum	9200	53 000	280 000	51 000	2300	20	550	1000	340 000
%RSD	150	270	590	620	330	10	190	330	410

Results indicate that there is a dearth of the more volatile compounds (MeFOSA, EtFOSA, FOSA, MeFOSE and EtFOSE) in the majority of samples, but a greater number of samples contain PFOS (88 % of samples), PFOA (59 % of samples) and PFHxS (45 % of samples). The lack of measurable concentrations of the other PFCs in this study's soil samples may be attributable to degradation of the compounds over time during storage (due to the time extent between collection and analysis – 6 years); however, all samples were stored in sealed amber glass jars at a constant 4˚C between sampling and analysis. It is also possible that biodegradation may have occurred within the soil prior to sample collection. However the low presence of these compounds is,it is more widely accepted to have been caused by the more volatile nature of some PFCs (MeFOSA, EtFOSA, FOSA, MeFOSE and EtFOSE) drives soil:air partitioning that favours their presence in air rather than soil (Rhoades *et al.*, 2008, Shoeib *et al.,* 2004).

However, in recent studies by Higgins *et al.,* (2005), Nakata *et al.,* (2006) and Naile *et al.,* (2010), only PFCA and PFAS were identified in soil. The presence of these compounds has been attributed to their persistent structure (C-F bonds) and the lack of microbial degradation pathways. In contrast, more volatile PFCs are thought to be more accessible to microbial degradation, re-volatilisation and leaching to ground and surface waters (Moody *et al.,* 2003).

In this study, the most frequently detected compound was PFOS, followed by PFOA > PFHxS > EtFOSE > MeFOSE > EtFOSA >MeFOSA > FOSA. The highest concentrations were detected for PFHxS, PFOA and PFOS, (9.2 – 280 ng g^{-1}) reaching concentrations similar to those found in household dust (from this study, see chapter 3). However, the mean and median concentrations remain relatively lower than indoor dust by 1 – 2 orders of magnitude.

Statistical analysis was restricted to the three most predominant compounds (PFOS, PFOA and PFHxS), owing to the limited detection rate for the other compounds. No significant correlations were found between these three compounds, which suggest that the sources and/or edaphic behaviour of these compounds are different.

The highest recorded concentrations for all the analytes occur in the urban soil (samples collected from towns or cities), and these soils also had detectable concentrations of the more volatile precursor compounds including MeFOSE, EtFOSE, MeFOSA and EtFOSA. The source to these sites is likely to be a combination of sources, due to the urban environment. The data was analysed for any significant differences between the urban and rural sites using an independent T-test, and was conducted upon log-normalised data in SPSS.17. Results indicated that there was no significant difference between sites from rural and urban regions for both individual compounds and the ΣPFC concentrations (see Figure 31). However, the maximum concentrations for each of the PFCs studies are all found in urban soils.

Degradation of PFOS in soils and sediments is isomer-selective, resulting in branched PFOS being more susceptible to degradation (Ochoa-Herrera *et al.*, 2010). Linear PFOS is much more stable and higher abundances of PFOS have been identified in humans and wildlife (Benskin *et al.*, 2009, Rylander *et al.*, 2009, Houde *et al.*, 2008). This is because only the branched PFOS is susceptible to degradation by vitamin B_{12} (Torres *et al.*, 2009).

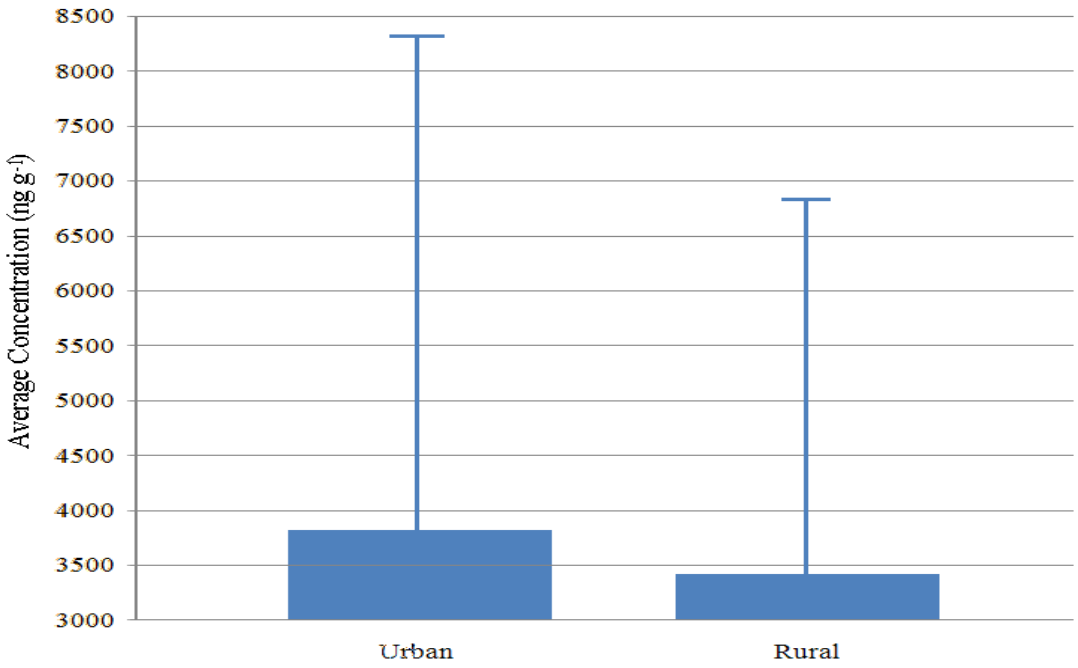


Figure 31 Urban vs rural soils (average concentrations, ng g^{-1})

6.8. COMPARISON WITH PFC CONCENTRATIONS REPORTED IN OTHER SURVEYS OF SOIL.

Table 64 Comparisons of mean soil concentrations (ng g^{-1})

Reference	Soil	Depth	PFOS	PFOA	PFHxS
This study		< 5 cm	0.96	3.1	7.3
Washington *et al.*, 2007	Wooded picnic site soil			66	
	Agricultural soil			260	
	Commercial top soil			330	
Washington *et al.*, 2009	Alabama background soil			0.064 & 0.23	
Naile *et al.*, 2010	S. Korea, coastal region,	< 5 cm	<2	<DL	<DL
Li *et al.*, 2010	Shanghai, China (range of values)		8.6 – 10	3.3 – 47	n.d. – 0.28
Contaminated Site					
Daikin, 2003a	Alabama, manufacturing site	< 5 cm	790		
Daikin, 2003b	Alabama, manufacturing site	<2 feet		530, 1300, 22.7, 48.9, 52.2	

The concentrations of PFCs in soil from this study are relatively low (by two or three orders of magnitude) compared to those reported by Washington *et al.* (2007), where samples were collected from US EPA government land in Georgia. This difference could be put down to the sorption of PFOS to soil being dependent upon the environmental conditions and the presence of naturally occurring cationic and anionic surfactants (Pan *et al.*, 2008). However, subsequent studies indicated that the concentrations measured in this study were similar to concentrations seen in the USA, Korea and China (Washington *et al.*, 2009, Naile *et al.*, 2010 and Li *et al.*, 2010). PFHxS is an exception, when compared to concentrations from China (Li *et al.*, 2010), the concentrations in this study are an order of magnitude higher. The concentration variability seen in the samples from China (Li *et al.*, 2010) indicates a very small %RSD for PFOS but greater variability for PFOA (6 % and 44 %, respectively). In comparison, the smallest variability is identified in the PFOS samples compared to the PFOA samples from this study (Results and Discussion
Table 63) and agrees with the study by Li *et al.*, (2010).

The %RSD values indicate soil concentrations are more variable than the outdoor air samples from this study, which is to be expected because of the slow mixing time in soils compared to air. This variance is not entirely the result of analytical variability (< 15 %), but will be associated with the presence of local sources, as well as edaphic factors such as: pH, mineral content, organic content, moisture, etc (Arshad & Coen, 1992, Johnson *et al.*, 2007, Higgins & Luthy, 2006). In comparison to outdoor air concentrations, the %RSD of PFOS and PFOA from the soil samples indicate much greater variability within the soils (170 % and 250 %). This variability extends throughout the other compounds for soil in comparison to outdoor air samples.

The comparison between UK soils and those studied by Washington *et al.,* (2007) and the contaminated Alabama sites, in the Daikin reports, indicates that the UK soils are not receiving any direct input of PFCs to the soils and are unlikely to have been impacted by industry. The concentrations noted in Washington *et al.* (2007) are comparable to concentrations noted at the contaminated Daikin site, and are likely to also be contaminated from local PFC manufacturing facilities. This is also indicated by the PFOA concentrations noted in Washington *et al.*, (2009), which are below the range seen in both this study and a study from Shanghai (Li *et al.*, 2010).

6.9. CONCLUSION

With 88 % of soils indicating the presence of PFOS, it can be deduced that UK soils are a substantial sink for some PFCs, in particular, for the less volatile species PFOS, PFOA and PFHxS. The lower concentrations of the more volatile compounds suggest that either soil is not a sink of these compounds and they partition quickly out of the medium (to water, biota and air), or that they are more easily biodegradable to more persistent compounds PFCAs and PFAS.

PFOS, PFOA and PFHxS have a range of concentrations in UK soils, which at the higher end rivals that of concentrations within indoor dust. However, the median values of each of these compounds remains well below UK indoor dust median concentrations and is likely to occur from dilution within the environment. The

The presence of PFOS, PFOA and PFHxS in the UK samples agrees with studies by Naile *et al.,* 2010, Nakata *et al.,* 2006, Higgins *et al.,* 2005 and Jai *et al.,* 2010, with similar concentrations (low ng g^{-1}).The UK soils do not appear to be contaminated more than other countries as seen in background sites. .It is likely, that UK soils are

receiving concentrations from diffuse pathways, as concentrations were not significantly different across the whole of the UK, in both urban and rural locations.

The low concentrations in comparison to work by Washington *et al.* (2007), indicate that none of the UK sites studied were directly contaminated from industrial use and manufacturing of the compounds. However, use of PFCs continues within the UK, including PFOS, under the exemptions of the Stockholm Convention (The POPs, 2010). Their continued use may produce concentrations higher than examined in this study, from areas close by to semiconductor and metal plating industries and also around air fields, within the UK. However, it is unlikely that UK soil concentrations would reach levels in future, seen in Alabama (Washington *et al.*, 2007), as there is no PFC production facility in the UK.

What cannot be deduced from this work and which therefore requires further research includes:

- determination of pathways of PFCs into UK soils
- sources to UK soils
- degradation occurring in the soil
- linear: branched PFOS ratios in soil and comparison to other microenvironments
- movement of PFCs out of soils
- potential human exposure from biosolid use on agricultural land

However, this study (one of very few worldwide to characterise PFC contamination of soil from non-industrially-contaminated sites) does show that soil represents an important sink for stable end-product PFCs like PFOS, PFOA, and PFHxS, but is less important for the more volatile precursor PFCs which are more likely to partition to

the atmosphere (see Chapter 3 and 5). Moreover, the fact that PFCs are present at quantifiable concentrations in soil, and at the high end are comparable to those found in indoor dust, means that there is a potential for soil to act as a human exposure pathway, both via direct ingestion and as a result of uptake into vegetation and local water sources and subsequently contaminating our food. The risk associated with direct soil ingestion is not likely to rival that of indoor dust exposure, because of the relatively short periods of time spent outdoors (by the general population), but the extent of such exposure will be examined in chapter 8.

7. PFCS IN CLASSROOMS: IMPACTS ON EXPOSURE OF CHILDREN

The hypothesis to be addressed in this chapter is that dust from UK primary school and nursery school classrooms constitutes an important pathway of exposure to PFCs for young children, and that the pattern of contamination is distinct from those observed in other UK indoor microenvironment categories. The aim of this chapter is thus to quantify the presence of PFCs in dust from a number of classrooms.

Concentrations of PFCs in dust from UK classrooms are reported for the first time in this chapter. It is hypothesised that PFC contamination in classroom dust will display a signature distinct from that seen in other UK indoor microenvironments. The rationale for this is that classrooms contain a distinct inventory of PFC-treated goods and materials. It is also hypothesised that classrooms will contribute substantially to the overall dust exposure of young children, in line with previous observations for PCBs and BFRs (Harrad *et al.* 2010c).

7.1. INTRODUCTION AND IMPORTANCE OF CLASSROOMS AS A VECTOR OF EXPOSURE TO PFCS

Classroom environments are an additional microenvironment analysed for the presence of PFCs. The time spent in classrooms by young children is comparable to that of adults in offices and results will be used in conjunction with concentrations in other relevant microenvironment categories to evaluate the exposure of children (chapter 8). Exposure of children via dust ingestion is likely to exceed that of an adult, due to the higher dust ingestion rates and lower body weight of children. Behavioural traits of young children increase their dust ingestion rates, for example, greater hand-to-mouth behaviour, and their propensity to spend time playing on the floor. The low

body weight of children (in comparison to adults) results in higher exposures when normalised to body weight, coupled with the fact that fast growth rates and development of their bodies can result in children being at higher risk of adverse effects of exposure to chemicals than teenagers and adults (USEPA, 2004). In addition to dust ingestion, other contributors to the body burdens of children include transfer from mother to baby through placental transfer (Inoue *et al.,* 2004, Apelberg *et al.,* 2007, Midasch *et al.,* 2007) and postnatal exposure via breast-feeding (Kärrman *et al.,* 2007 Völkel *et al.,* 2007, Liu, *et al.,* 2010).

Concentrations in blood samples from neonates between 1997 and 2007 from New York (Spliethoff *et al.,* 2008) identified PFOS and PFOA concentrations in ≥ 90 % of the 2640 samples analysed. PFOS is also regularly found in breast milk from mothers around the world (Calafat *et al.,* 2003, Völkel *et al.,* 2008, Fei *et al.,* 2008) producing a pathway for babies via their main diet. A recent study from Australia (Toms *et al.,* 2009) indicated that children had concentrations of PFCs in their blood serum that either matched or exceeded the concentrations in adults. These concentrations of PFCs are believed to have toxic effects on the exposed children and have been noted to cause various developmental defects, including suppression of immune responses (Yang *et al.,* 2002, Yeung *et al.,* 2007) and changes to head circumference of neonates (Apelberg *et al.,* 2007).

There have been two reported studies of dust from daycare centers in USA and Sweden. Unfortunately, the USA data does not distinguish between concentrations found in homes from those found in daycare centers and, therefore, are not specifically comparable. Conversely, the Swedish study (Björklund *et al.,* 2009), does separate this data and it will be used later on in the study as a comparison to the UK data.

The identification of chemicals in children's environments and products is of rising concern, with new regulations being introduced within the EU regarding toy safety (Directive 2009/48/EC). The regulations have now begun to prevent the use of certain chemicals that can cause endocrine disruption, have reproductive implications, and that have carcinogenic properties and toxic effects. The presence of PFCs in products that could affect children is much wider than in the basic room furnishings, food containers and cooking utensils, including childrens clothing, toys, cots and car seats.

7.2. CONCENTRATIONS OF PFCS IN CLASSROOM DUST

Dust samples were collected from 42 school and nursery classrooms from across the West Midlands (UK). The institutions were contacted via a letter, detailing the sample collection methods, use of the sample and the impact of the data gathered from the samples. The response from institutions was 32 %, and those that gave permission were contacted and appointments arranged for appropriate sampling times. Often, samples were collected, either during the school day, or shortly after the children had finished. Samples were collected according to the procedure detailed in chapter 2, with the exception that, in order to eliminate spatial variations in contamination, the entire room was vacuumed. This was considered justifiable as classrooms were utilised in all areas, therefore children would be exposed to dust in all parts of the room. Concentrations of PFCs from UK classrooms and descriptive statistics are summarised in Table 65. Results that were below the detection limit or were non-detects are represented in all statistical analysis by half the limit of detection (Maertens *et al.* 2007). MeFOSA was not detected in any samples (the MeFOSA limit of detection = 0.1 ng g^{-1}, and the limit of detection for all compounds can be found in chapter 2) with FOSA present in only 12 % of samples. Mean recoveries (± standard

deviation) for the internal standards in these dust samples were 70 % (± 20) for PFOS, 74 % (± 17) for PFOA, 70 % (± 21) for PFHxS, 59 % (± 20) for MeFOSA, EtFOSA, and FOSA, and 76 % (± 21) for MeFOSE and EtFOSE. Concentrations of PFHxS in classroom dust were highly variable (relative standard deviation of 220 %) with PFOS and PFOA showing relative standard deviations well under half of that of PFHxS (85 % and 98 % respectively). The variability for other monitored PFCs was similar to that of PFHxS. This is likely to be a result of the relative stability (shorter half-lives) of these precursor compounds and greater variation in their sources. Individual classroom contamination patterns can be observed in Figure 31 and the ratios of classrooms with and without certain sources are displayed in Figure 32.

Concentrations of PFOS and PFOA were determined in dust from Swedish daycare centres by Björklund *et al.,* (2009). Results revealed median (range) concentrations to be 31 ng g^{-1} (23 - 65 ng g^{-1}) and 41 ng g^{-1} (31 - 110 ng g^{-1}), for PFOS and PFOA respectively. Table 2 shows the equivalent values in the UK samples reported here to be 840 ng g^{-1} (22 – 3700 ng g^{-1}) and 240 ng g^{-1} (18 - 1600 ng g^{-1}). Concentrations of PFOS and PFOA in this study are significantly higher than in the Swedish study. Furthermore, in contrast to the Swedish study, the predominant compound is PFOS, a pattern seen also in samples from Canada (Kubwabo *et al.,* 2005) and the USA (Strynar & Lindstrom, 2008), but not Japan (Moriwaki *et al.,* 2003) or in the house dust samples reported in this study (chapter 3). Along with the predominance of PFOS in the samples, the other international studies cited here indicated concentrations to fall within a similar range to the UK classrooms of 10 – 4000 ng g^{-1} for both PFOS and PFOA. Differences between this study and the Swedish data could potentially arise to some degree from the sampling techniques employed to collect the dust. This study collects dust at floor level, using a standard vacuum cleaner nozzle and

collection sock which retains particles from 28 μm upwards. In contrast, the dust samples collected by Björklund *et al.,* (2009) were from around 1 m height in the rooms, from shelves and window ledges, for example. The collection technique uses styrene- acrylonitrile filters to collect the dust which was then scraped from the filter for analysis. This may result in a different particle size range being analysed, tending towards the lower end of the scale, due to smaller particles being lighter and much more likely to be carried by the air circulation within the room. However, while the impact of dust collection method on PFC concentrations is unknown, it is considered unlikely to explain the order-of-magnitude differences in concentration between the two studies.

Sample 6 contained the highest ΣPFC concentration (36 400 ng g^{-1}) from the entire thesis. This could be due to the building being in the last stages of renovation (carpet being laid in upstairs rooms). The building was over 100 years old, and hence the building itself is an unlikely source of the compounds. However, the rooms had recently been painted, floors waxed and new carpets laid on the upper floors and staircases. The main contaminant measured was PFHxS (34 000 ng g^{-1}), and this was the highest level recorded from all the microenvironments sampled. Paint and coatings applied for the protection of a surface often contain PFCs. The commercial mixtures have changed over the years, with movement away from the more persistent compounds (PFOS, PFOA, PFHxS) towards more volatile compounds (PFHxA and 6:2 fluorotelomer sulfonates, Dupont™ 2008), which are less persistent (Dupont Surface Protection Solutions, 2008). However, the high concentrations could still be derived from these surface coating sources, as reserves of paints are still in commercial circulation. It is also likely that the degradation of FTOHs and MeFOSE could result in indoor concentrations of PFOS, PFOA and PFHxS. The carpet may

also be a significant source to the room. Though the room sampled was not carpeted, the installation of the carpeting in other areas of the building may have resulted in contamination throughout the building. PFHxS was a popular PFC used in protective coatings for carpets (Olsen *et al*., 2003).

The lowest ΣPFC concentrations were detected in classroom sample 34, which only had 1/3rd of the room carpeted, and no other textiles. This room also contained no electronic products or televisions (where PFCs are incorporated into the manufacturing process and thought to leave a small amount of residuals on the circuit boards), no foam chairs (PFCs are often used on commercial seating to protect the material and extend the lifetime of the product) and the majority of the furniture was wooden. It is likely that carpet cleaning products containing PFCs are still in use and that these constitute a minor source to the room. It would be likely that other contributions would include the children's clothing that contains PFCs, as well as varnishes and coatings on the wooden furniture.

The lowest concentration of PFOS was found in sample 21, from a classroom, which like sample 6 had no carpet, but laminate floor boards. The classroom was also deficient in textiles and mainly contained wooden furniture, books, toys and paper displays. Interestingly, the low PFOS concentrations were not reflected in the pattern seen for the other PFCs – for example, concentrations of both PFHxS and MeFOSE exceed the 95th percentile. Degradation of PFHxS or PFOA precursor compounds but not of PFOS precursors may explain this pattern.

Table 65 School & nursery classroom dust concentrations (ng g^{-1})

Number	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFCs
1	610	100	400	<DL	57	<DL	170	<DL	1300
2	1700	240	190	<DL	53	<DL	2100	340	4500
3	1900	270	3200	<DL	38	<DL	<DL	51	5400
4	110	310	7700	<DL	25	<DL	<DL	27	8200
5	730	140	590	<DL	70	<DL	880	2300	4700
6	960	510	34 000	<DL	74	<DL	680	170	36 000
7	590	160	1700	<DL	10	<DL	640	1200	4300
8	1300	430	3600	<DL	29	<DL	1200	320	6900
9	650	170	260	<DL	8	<DL	490	520	2100
10	2300	640	78	<DL	130	<DL	1600	1800	7300
11	980	200	750	<DL	8	<DL	600	600	3100
12	750	290	840	<DL	250	<DL	250	58	2400
13	3700	210	47	<DL	130	<DL	960	13 000	18 000
14	1500	310	6100	<DL	20	<DL	170	10	8100
15	220	480	660	<DL	640	<DL	210	33	2200
16	1700	370	620	<DL	47	<DL	930	<DL	3600
17	800	55	3600	<DL	8	<DL	590	610	5600
18	2400	460	470	<DL	120	<DL	120	170	3800
19	200	300	3800	<DL	20	<DL	72	81	4500
20	2100	45	930	<DL	30	<DL	180	<DL	3300
21	22	330	7900	<DL	25	<DL	6900	470	16 000
22	2400	340	4600	<DL	11	<DL	1000	1300	9600
23	860	35	71	<DL	33	<DL	8400	4400	14 000
24	900	120	1700	<DL	2	<DL	1600	3700	8000

25	720	590	1400	<DL	31	<DL	1100	690	4500
26	240	110	140	<DL	30	<DL	1100	260	1900
27	2000	240	3300	<DL	23	<DL	2400	4200	12 000
28	820	360	220	<DL	23	<DL	800	4400	6600
29	870	240	1700	<DL	<DL	<DL	1900	2400	7100
30	980	460	930	<DL	1	<DL	2400	3300	8000
31	900	210	16	<DL	100	<DL	250	100	1600
32	160	99	730	<DL	27	750	<DL	<DL	1800
33	1200	460	1900	<DL	12	<DL	230	940	4700
34	370	110	27	<DL	<DL	<DL	28	93	630
35	1100	1700	1300	<DL	12	<DL	2500	420	7000
36	180	540	38	<DL	73	<DL	5200	<DL	6000
37	160	28	450	<DL	35	<DL	<DL	390	1100
38	980	74	400	<DL	400	<DL	<DL	<DL	1900
39	190	18	21	<DL	<DL	<DL	950	120	1300
40	430	1000	450	<DL	<DL	<DL	170	61	2100
41	170	18	530	<DL	60	3	1100	3500	5400
42	130	23	310	<DL	50	<DL	610	430	1500
Minimum	22	18	16	<DL	<DL	<DL	<DL	<DL	630
5th Percentile	130	23	28	<DL	7	6	110	32	1300
Median	840	240	700	<DL	31	34	880	450	4600
Geometric Mean	640	190	650	<DL	33	38	690	450	4300
Arithmetic Mean	990	300	2300	<DL	71	170	1400	1500	6100
95th Percentile	2400	630	7600	<DL	270	610	5500	4400	16 000
Maximum	3700	1600	34 000	<DL	640	750	8400	13 000	36 000
SD	8400	300	5400	<DL	120	320	1800	2500	6200
% RSD	85	98	230	<DL	170	190	130	170	100

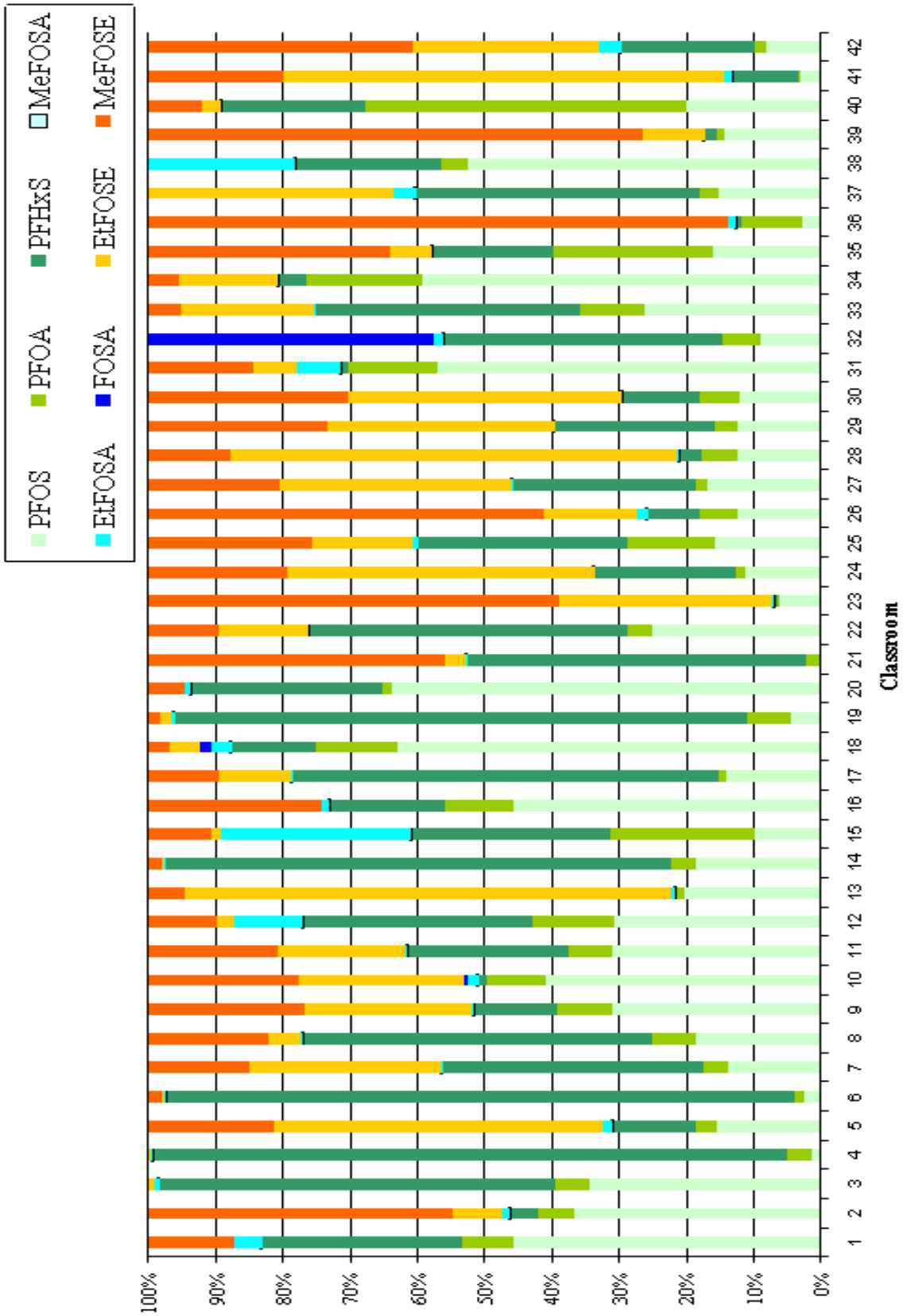


Figure 32 PFC contamination patterns (concentrations of individual PFCs expressed as a percentage of ΣPFC) in nursery and primary school dust samples.

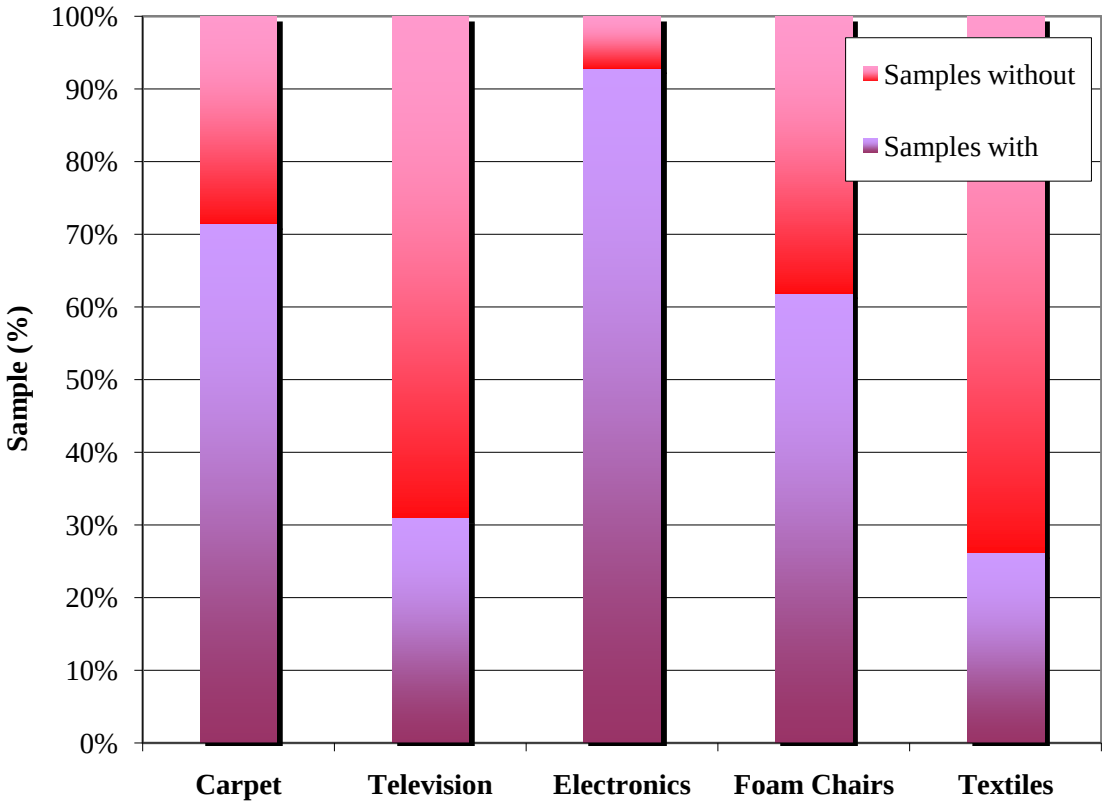


Figure 33 Presence/absence of potential s ources of PFCs in sampled classrooms

7.3.STATISTICAL RELEVANCE OF RESULTS

PFC concentrations were tested for normality of distribution, and were all revealed to display a skewed distribution. According to the Shapiro-Wilk test, all PFC categories were significantly skewed ($p < 0.001$), and therefore all statistical tests were conducted on log normalised data (base 10). Correlations between concentrations of different compounds in the same samples were examined with two significant correlations found. The first is a significant ($p = 0.01$) positive correlation between PFOS and PFOA (Figure 34), where Pearson's correlation coefficient, $R = 0.48$. This relationship was also identified by Moriwaki *et al.,* (2007) in Japanese house dust ($R = 0.6$), by Björklund *et al.,* (2009) in Swedish daycare dust ($R = 0.61$), and in German human plasma by Midasch *et al.,* (2007) ($R = 0.82$). Such relationships imply that the sources or pathways are similar for both compounds.

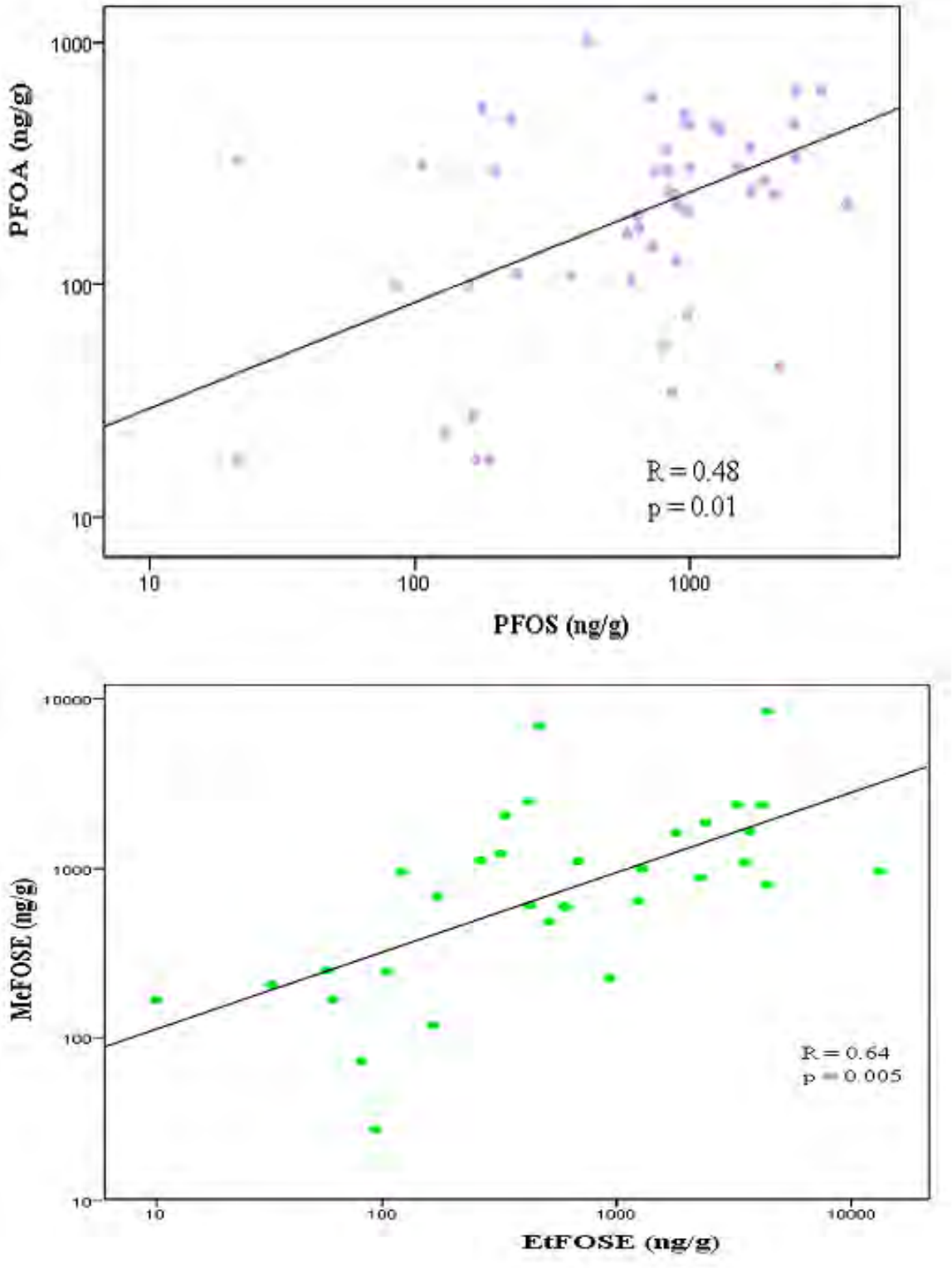


Figure 34 Correlation between concentrations in dust of (a) PFOS and PFOA and (b) MeFOSE and EtFOSE (with non-detect values omitted) on a logarithmic scale.

The correlation identified in the dust samples suggests the same behaviour applies for these samples and that they could be created by the degradation of precursor compounds (Lange, 2001).

The second significant correlation (R = 0.64) identified in these dust samples was between MeFOSE and EtFOSE (Figure 34), after removal of samples in which one or both compounds were below the detection limit. This suggests that these compounds arise from similar sources, releases and chemical behaviour within the indoor microenvironment. The potential major sources of these two compounds are likely to be protective surface coatings, often applied to paper products, carpets and furnishings, paints and waxes. However, it is important to note that inclusion of the non-detect values renders the correlation insignificant ($p = 0.298$).

7.4. INFLUENCE OF ROOM CONTENTS ON PFC CONCENTRATIONS IN DUST

An independent sample t-test was applied to compare PFC concentrations in dust from classrooms containing carpet versus those with no carpet, televisions versus no televisions, electronics versus no electronics, curtains versus no curtains, and nurseries versus primary schools. The data regarding number of samples in each category, along with mean and standard deviation for each PFC is outlined in Table 66. Analysis of the data using t-tests revealed a number of significant differences. The first is that concentrations of MeFOSE and EtFOSE are significantly higher ($p < 0.01$) in rooms containing electronic goods than those without such items. However, as there were only 3 classrooms without electronics, further monitoring is required to evaluate whether this apparent difference is genuine. All other microenvironments tested in this work all contained electronic goods, and therefore there are no other

sources of data to draw upon for a reliable examination of the impact of electronics on the concentrations of PFCs in indoor environments. However, the results do indicate that there may be residues and by products remaining on electronic circuitry from the manufacturing processes, for example, the PFOS in the photoresist formulations used in photolithography. Currently, the two main uses of PFOS in the semiconductor industry are for anti-reflective coatings and photoresists. Prior to European regulation (EC 1907/2006 annex XVII to REACH, and directive 76/769/EEC, PFOS was used more widely in industry and these products may still be in use, and constitute sources of PFCs. For example, PFOS is utilised as a surfactant in chromium plating, and although most is expected to be removed by the washing process, some may remain on the plated substrates.

There were two other significant differences detected involving FOSA: (a) when comparing classrooms with curtains and other textiles and (b) when comparing classrooms with foam chairs with p values of 0.002 and 0.015, respectively, with those rooms containing the curtains and foam chairs having higher concentrations. However, this relationship is questionable because of the low detection rate of FOSA in the samples (12 %).

A third relationship ($p = 0.01$) was identified whereby concentrations of PFOA were higher in classrooms with carpets ($n = 30$) than in those without ($n = 12$). A similar relationship has been reported by Kubwabo *et al*., (2005) and Gerwurtz *et al.,* (2009), whereby concentrations of PFOS, PFOA, PFHxS, PFOSA and perfluorobutane sulfonate (PFBS) were higher in carpeted than in non-carpeted Canadian homes. Finally, younger properties (< 20 y old) displayed higher concentrations of ∑PFCs than older houses (> 60 y old). This difference may be attributed to the greater percentage of carpeting in the houses (Strynar & Lindstrom, 2008).

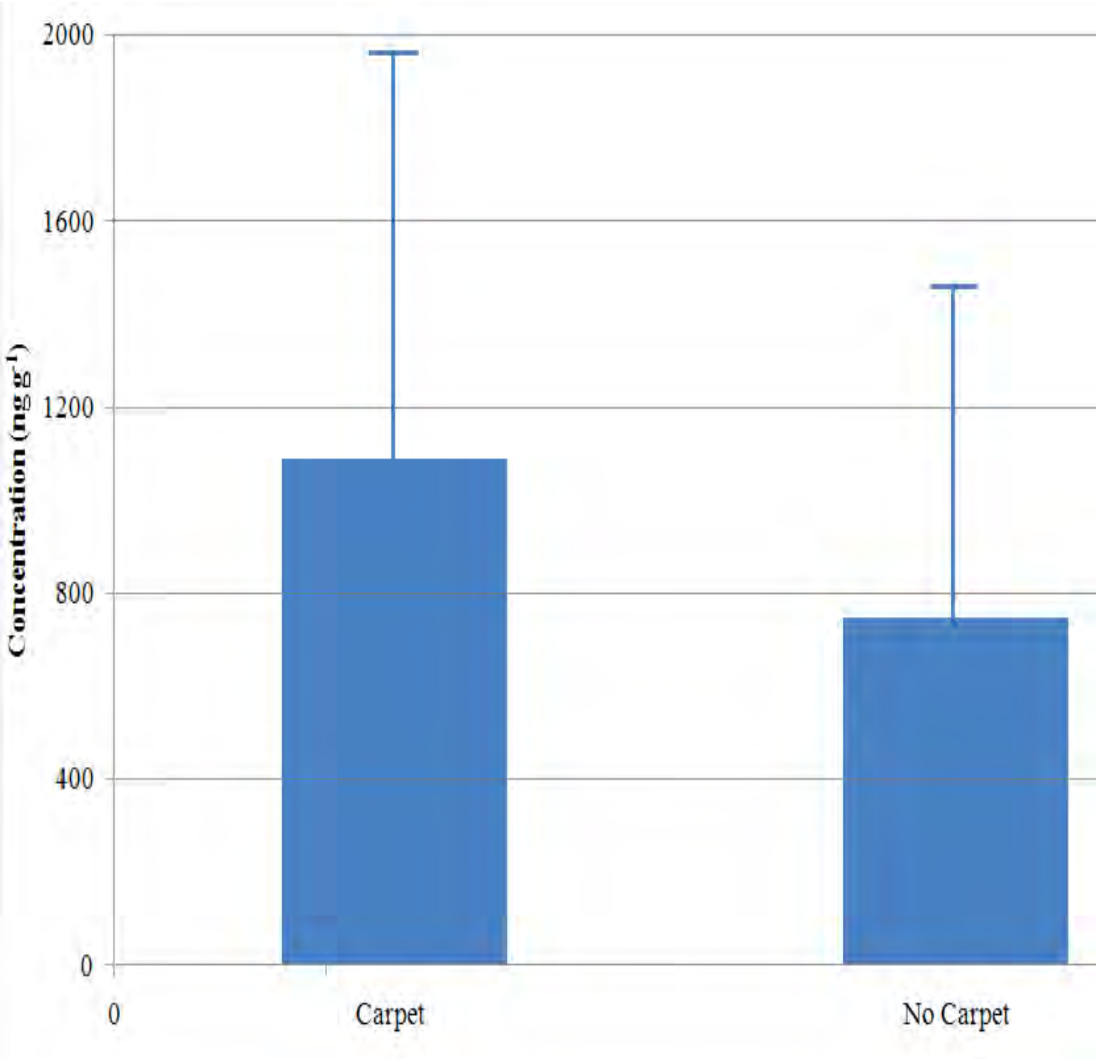


Figure 35 Average concentrations of PFCs in carpeted and non-carpeted classrooms (with Standard deviation). These categories indicated a significant difference (p = 0.01) between results.

Carpet samples have been analysed by Gewurtz *et al.*, (2009), and Mawn *et al.*, (2005) for their PFC content and the implications for exposure. Gewurtz *et al.*, (2009) detected a wide range of ∑PFCs in new carpets creating a range of concentrations from 19 – 32 500 pg cm^{-2}. The study by Mawn *et al.*, (2005) also indicated that the extraction of PFOA from carpet samples with saliva produced comparable concentrations (3 ng g^{-1}) to the extraction with solvent. This is an indication of a potential source for young children.

Comparisons of classroom ΣPFC concentrations were also applied with t-tests for identification of any significant differences in the means of classrooms when compared with the questionnaire results. The results indicated that there were very few significant relationships within the data and the room contents. There is a significant relationship ($p = 0.014$) identified in the independent t-test between classrooms with curtains and textiles (often used for displays) ($n = 11$) and those without ($n = 31$) for ΣPFC concentrations. The identification of this pattern of behaviour suggests that the presence of textiles within the room is a likely source of PFCs to the room. Materials are often coated with PFCs to provide them with greater stain and grease resistance.

There was also a weak significant difference ($p < 0.1$) between concentrations of FOSA in classrooms with foam chairs and cushioning ($n = 7$) and those without ($n = 8$). This suggests that the foam may retain the PFCs and potentially act as a secondary source (Zhang *et al.*, 2009). Alternatively, it may be that the textile covering of the foam furniture is the real source.

Table 66 Mean concentrations (and standard deviation) associated with room contents (ng g^{-1})

Content	Samples	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE	ΣPFC
Carpet	n = 30	1100 (880)	300 (170)	2600 (6200)	<DL	68 (120)	29 (140)	1100 (1700)	1400 (2600)	6600 (6700)
No carpet	n = 12	750 (710)	310 (500)	1600 (2300)	<DL	54 (110)	0.3 (0.9)	1200 (2000)	890 (1400)	4900 (4700)
Television	n = 13	900 (590)	380 (410)	2000 (2400)	<DL	41 (66)	1.2 (4.4)	1000 (910)	1080 (1500)	5400 (2300)
No television	n = 29	1000 (930)	270 (230)	2500 (6300)	<DL	75 (130)	29 (140)	1300 (2100)	1300 (2600)	6500 (7400)
Electronics	n = 39	1100 (830)	300 (300)	2300 (5500)	<DL	65 (120)	22 (120)	960 (1400)	1200 (2400)	5900 (6300)
No Electronics	n = 3	120 (86)	290 (260)	2800 (4400)	<DL	52 (25)	1 (1.7)	4400 (3000)	1300 (1900)	9000 (5700)
Foam Chairs	n = 26	1200 (920)	290 (320)	1400 (1800)	<DL	83 (140)	33 (150)	1200 (1700)	1400 (2800)	5500 (4100)
No foam chairs	n = 16	610 (450)	320 (270)	3700 (8700)	<DL	34 (31)	0.2 (0.7)	1300 (2000)	1030 (1500)	7100 (8700)
Textiles	n = 11	650 (500)	240 (200)	4400 (10 000)	<DL	85 (130)	<DL	1200 (1900)	380 (370)	6900 (10 600)
No textiles	n = 31	1100 (900)	330 (320)	1600 (2000)	<DL	57 (110)	28 (130)	1200 (1700)	1600 (2600)	5900 (3900)

*<DL, these results were replaced with the LOD/2

7.5.ANOVA

The influence of building age on PFC concentrations was tested using one-way ANOVA. Buildings were arranged into 4 classifications: 2000 – 2009 (n = 9), 1970 – 1999 (n = 19), 1930 – 1969 (n = 3), and pre-1930 (n = 11). Results indicated that there were no significant differences between concentrations in the different building age groups. What is likely to impact the concentrations is the age of renovation and remodeling of rooms, due to factors like paint, carpets and other furnishings containing PFCs. Stain resistant paints are likely to be used in classrooms; PFCs are added to paint to make them more resistant and hard wearing. Also, in regards to the flooring of the room, carpets and floor waxes contain PFCs and therefore may contribute to the concentrations in a room, especially in newer products. Kubwabo *et al.,* (2005) noted a significant positive correlation (using Spearman's Rank) between percentage of carpeted area within a building and PFOS ($p = 0.001$), PFOA ($p = 0.002$) and PFHxS ($p = 0.003$) concentrations. Interestingly, in contrast to our study, a significant positive relationship (PFOS $p = 0.005$, PFOA $p = 0.006$ and PFHxS $p = 0.09$) was found between the building age and the PFC content. However, this relationship was thought to arise from the fact that older buildings had fewer carpeted areas than newer buildings.

7.6.PRINCIPAL COMPONENT ANALYSIS (PCA)

The classroom data was expressed as a percentage of the total ΣPFC per classroom sample to produce a weighted data set (Figure 37). This data was used in SPSS (version 17.0) to conduct the PCA analysis. The data showed a statistical significance ($p < 0.001$) for the Bartlett's Test of Sphericity, indicating significance of the

correlation of relationships between the compound concentrations. The Kaiser-Meyer-Olkin measurement returned a value of < 0.6, the factor analysis was conducted despite this result, and it has been possible to represent the variables into a smaller set of component factors. The PCA results are displayed in four factor components, factor 1, 2, 3 and 4 explaining 26 %, 21 %, 13 % and 13 % (cumulatively 73 %) of the variance, and plotted against one another after a Varimax rotation was performed.

Table 67 Factor Coefficient Values Obtained for PCA Analysis

Compound	Factor Score			
	1	2	3	4
PFOS	0.81	-0.14	0.12	-0.14
PFOA	0.32	0.16	0.63	-0.06
PFHxS	-0.52	-0.77	0.28	0.06
MeFOSA	0.70	0.14	0.19	0.32
EtFOSA	0.40	-0.14	0.38	-0.15
FOSA	-0.01	-0.09	0.01	0.95
MeFOSE	-0.32	0.90	0.06	-0.07
EtFOSE	0.02	0.19	-0.89	-0.14

PC1 is dominated in a positive direction by high abundances of PFOS and MeFOSA, and in a negative direction by high concentrations of PFHxS. PC2 is dominated in a negative direction by PFHxS, and in a positive direction by MeFOSE; factor 3 scores are driven in a negative direction by high concentrations of EtFOSE, and positively by high values of PFOA. Finally, factor 4 is dominated in a positive direction by high abundances of FOSA.

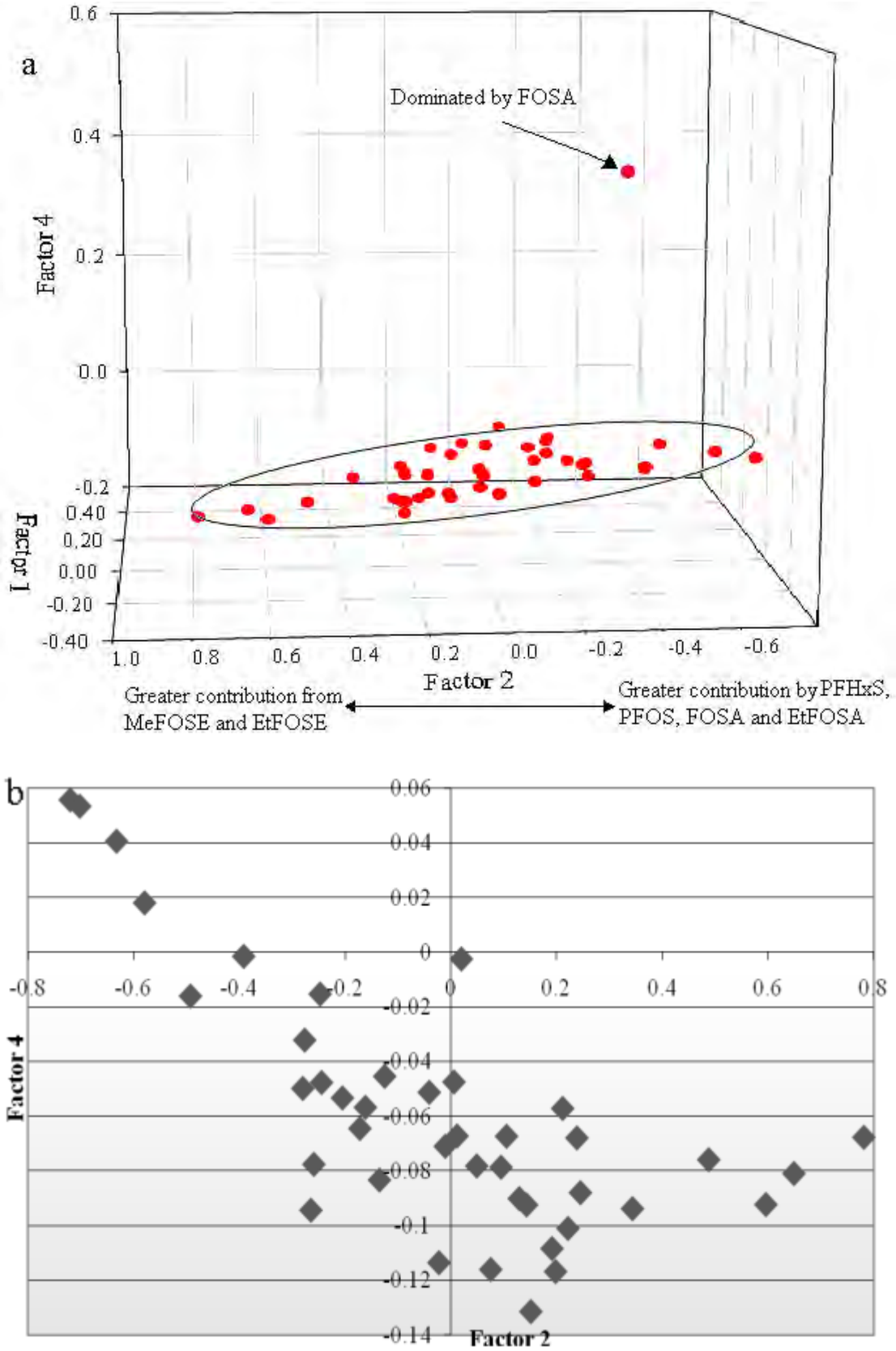


Figure 36 Factor Scores for Schools Data, graph a, 3D representation of factors 1, 2, and 4, and graph b depicting the difference in factor 2 scores created by concentrations of MeFOSE and PFHxS.

Despite differences in compound concentrations, the majority of samples remain within a small area of the factor scores. Figure 4 shows that factor 2 provides the greatest separation of the classroom dust samples. There is one obvious outlier sample in Figure 4, which displays a much higher value for factor 4 than any of the other samples. This sample has an unusually high abundance of FOSA.

While not plotted on Figure 4, factor 3 scores provided little discrimination between classroom dust samples, and simply highlighted differences between samples according to their abundance of EtFOSE.

Figure 37 displays the mean distribution of PFC compounds in the dust samples and indicates that the greatest contributions arise from PFOS and PFHxS, followed by the PFOSEs and PFOA, with small contributions from EtFOSA and FOSA.

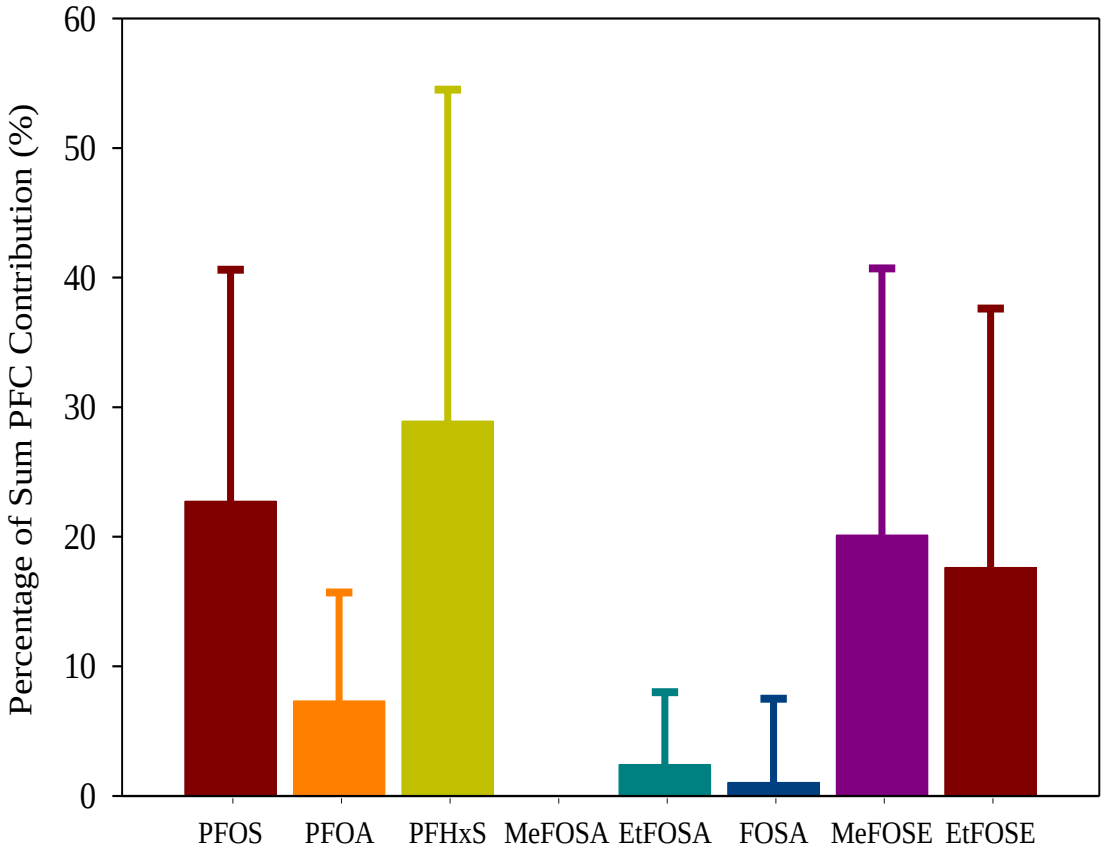


Figure 37 Mean contribution to ΣPFC from classroom samples (%), error bars representing standard deviation.

7.7. CLASSROOM COMPARISON TO MICROENVIRONMENTS

Comparing results from this study with a Swedish study (Björklund *et al.*, 2009), the average concentrations of both PFOS and PFOA are higher in UK classrooms than in Sweden. The ratio of these two compounds varies also, with PFOA being predominant in Swedish daycare centers', but PFOS predominating in the UK, similar to the UK homes. Concentrations of PFCs were identified in all samples from all microenvironments. When the classroom concentrations are compared to those from UK homes, offices and cars, large differences are revealed in the concentrations of some PFCs measured in classrooms compared to other microenvironments. It is clear in Figure 38 that concentrations of PFOS, PFHxS, MeFOSE and EtFOSE in classroom dust are higher than the measured concentrations from other UK indoor environments. The means were tested using ANOVA, with Tukey post-hoc tests, and the results revealed significant differences between the means of classrooms and some or all microenvironments. Concentrations of PFOS, PFHxS, MeFOSA and FOSA were significantly greater ($p < 0.001$) in classrooms than those in homes, offices and cars. By comparison, concentrations of PFOA in classroom dust were significantly higher than in car dust ($p = 0.006$), whilst EtFOSE in classroom dust exceeded home concentrations ($p = 0.015$). No other significant differences between PFC concentrations from the different microenvironment categories were detected. Despite there being a visibly large difference in the median and mean concentrations from classroom microenvironments, there are few significant differences between microenvironment categories in concentrations of the precursor compounds EtFOSA, FOSA, MeFOSE and EtFOSE, suggesting that the sources of these compounds are similar in each environment. The largest differences are apparent for PFOS

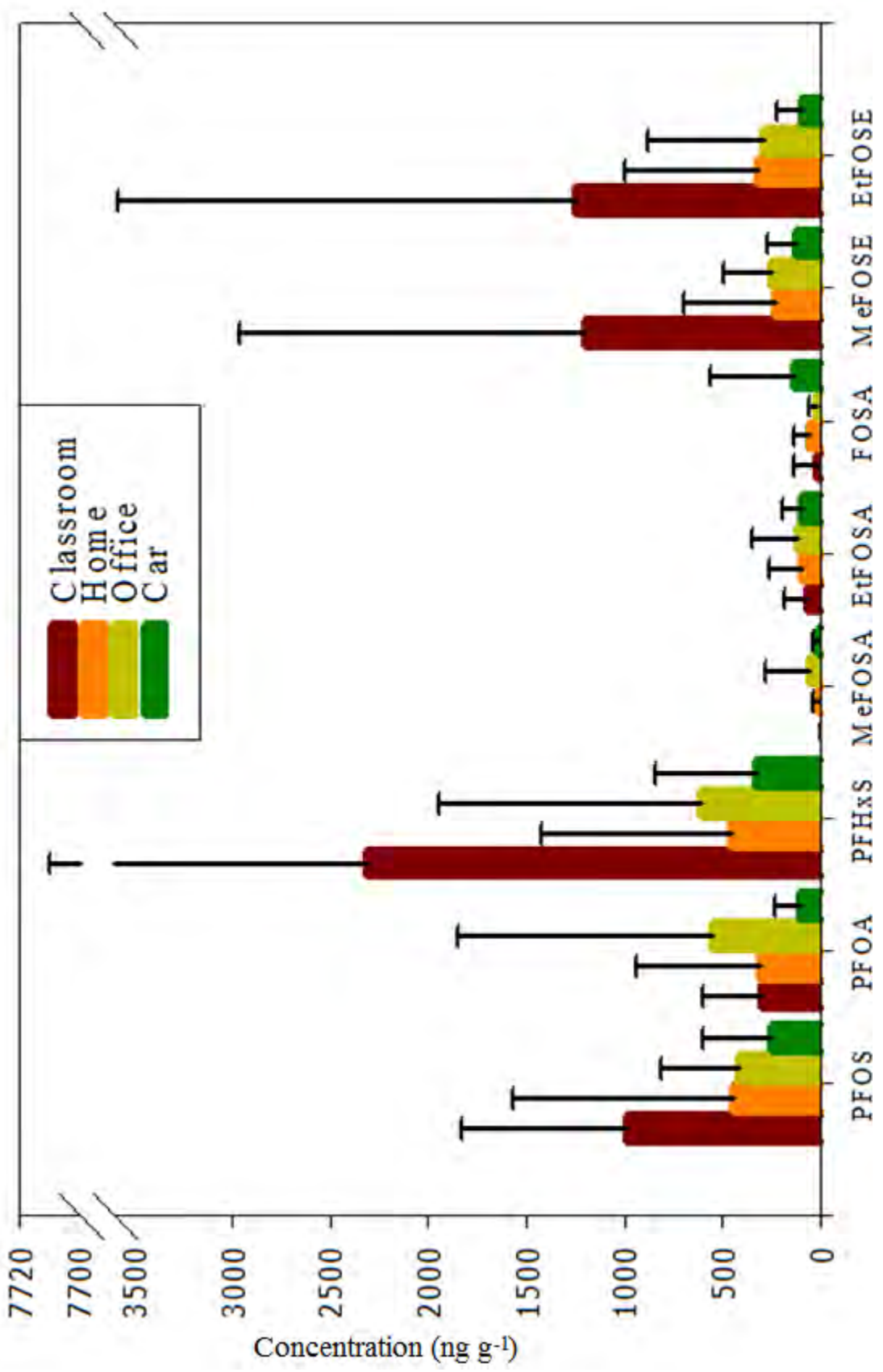


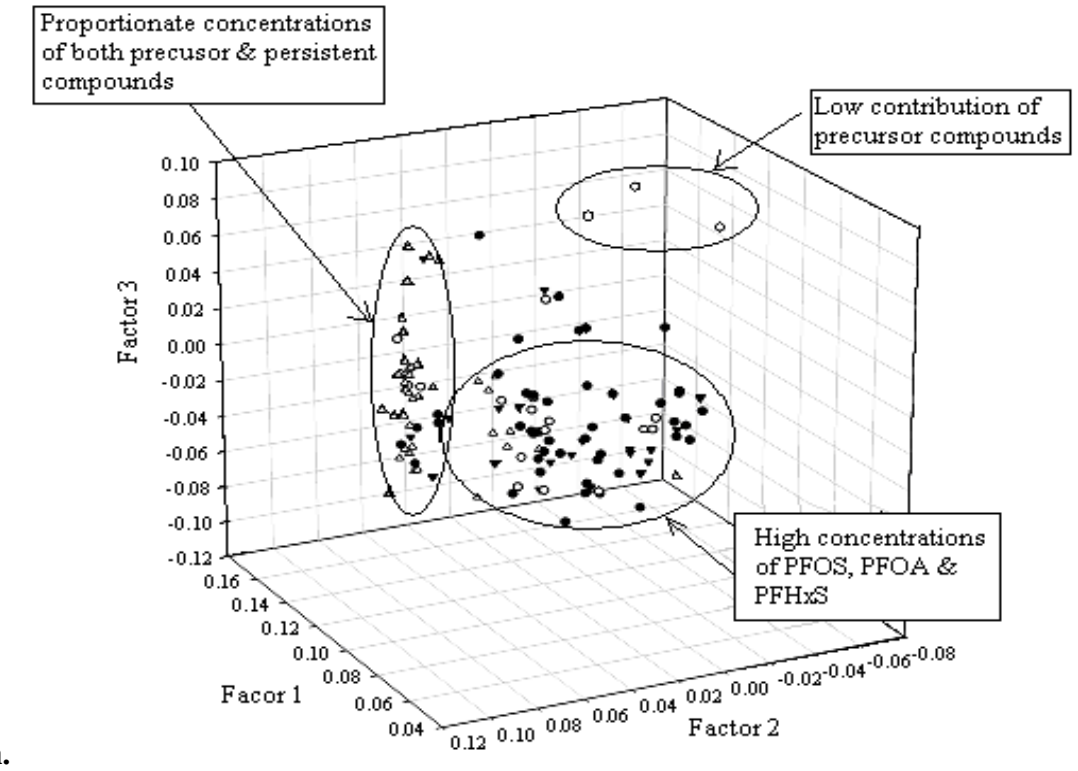
Figure 38 Comparison of average concentrations (ng g^{-1}) (± standard deviation) from 4 microenvironments

and PFHxS, which display significant differences for all the environments compared to the classroom concentrations. MeFOSE and EtFOSE are also higher in classrooms than other microenvironments, but are not significantly different. This indicates possible additional sources in classrooms that are present in the other environments, but in higher frequency of use within the classroom environments. Indications from the collated questionnaire data do not specify any exceptionally different products within the classroom environments, but did indicate a higher frequency of objects, and generally newer products. However, classrooms were the most highly occupied indoor environments studied and therefore, tended to contain more furniture (primarily tables, chairs, and chests of drawers). Classrooms also tended to contain more textile products (sheets) used for decoration and displays and dressing-up clothes, more paper use, again for displays and decoration, as well as books. The data was analysed for the correlation of the number of people in classrooms and the concentrations. However, this did not reveal any significant relationships, though it was thought that increased occupancy within the room could cause increased wear and tear, as well as additional sources from clothing (coats, shoes, bags, etc).

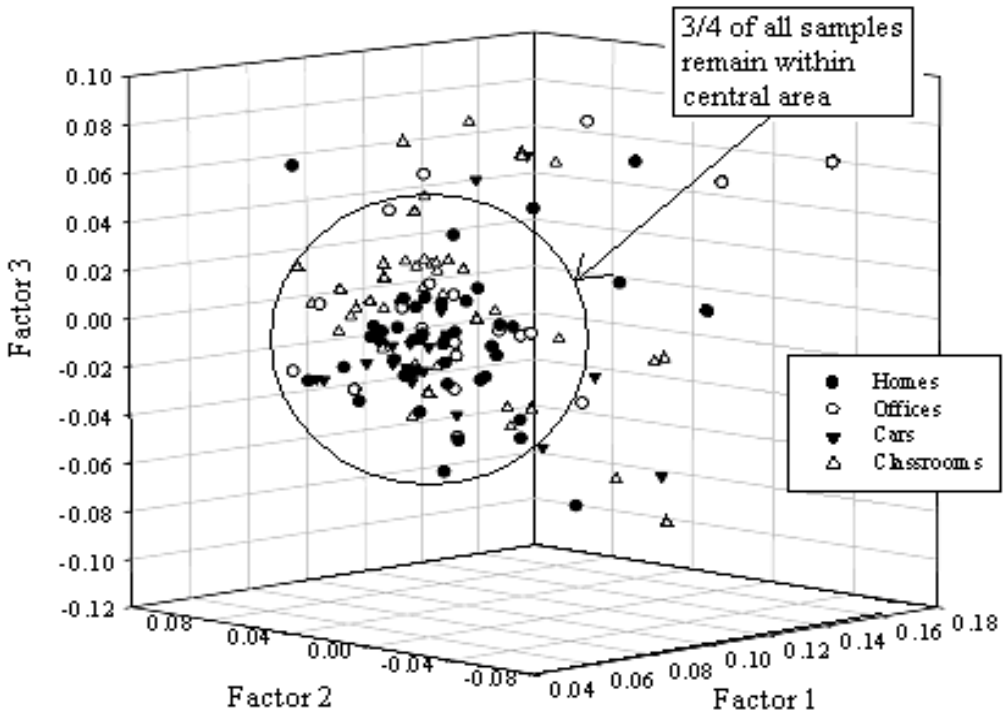
The MeFOSA results are excluded from this comparison, as there were too few detectable concentrations within the data for the results to be considered significant.

Concentrations of PFCs normalised to ΣPFC in all samples from all microenvironments studied were subjected to principal components analysis (PCA). The results of the PCA revealed 3 components, which in total represented 66 % of the total results. The results from the PCA were rotated using Varimax rotation to produce factor scores for each compound, which were applied to the individual samples analysed from each microenvironment. The individual samples were weighted (as a proportion of the ΣPFC concentration for each sample). Details of the

PCA factor scores obtained for this dataset are displayed in Table 68 and the rotated results in Figure 39 a, and b.



a.



b.

Figure 39 Classroom PCA results from two different angles, a, and, b.

Table 68 Microenvironment PCA factor scores

Parameter	Components		
	Factor 1	Factor 2	Factor 3
PFOS	0.347	0.060	-0.060
PFOA	0.320	-0.102	0.019
PFHxS	0.350	-0.141	0.026
MeFOSA	-0.025	-0.544	0.256
EtFOSA	-0.074	0.256	-0.718
FOSA	-0.234	-0.233	-0.023
MeFOSE	-0.090	0.208	0.418
EtFOSE	-0.110	0.487	0.106
% Variance Explained	30.07	18.75	17.29

The results from the PCA indicate that there is a distinct difference in the results from the classroom microenvironments. Figure a, is dominated by two groups of scores, the first located on the left, and dominated by classrooms, whilst the second (on the right) are dominated by homes and cars, whilst the distribution of offices varies between the two groups. The two groups are created primarily by differences in the presence and dominance of the prescursor compounds. The more equal the distribution of all eight compound concentrations in samples,the more positive the PCA value for both factors 1 and 2. The classrooms are distributed in the small cluster because of the high abundance of MeFOSE and EtFOSE in the samples driving the factor 2 scores in this positive direction.

The majority of homes and cars are located centrally (as indicated in Figure 39, a), with comparably lower factor 1 and factor 2 concentrations, and mainly negative factor 3 values which are predominantly derived from EtFOSA. The points are located in this cluster because of a higher contribution from the persistent compounds (PFOS, PFOA and PFHxS), and are not dominated by any one compound, but are

more evenly spread between these three PFCs. This region on Figure 39 a. also includes samples from all the microenvironments, including samples from the classrooms, suggesting the contents within the room are a better indicator of concentration and sources of PFCs than the classification of room function, e.g. different microenvironments.

There are 3 samples from the offices which display very high factor 3 scores. It appears that these samples are all influenced strongly by one extremely high compound concentration. In the case of these three samples, the high concentrations are PFOA, PFHxS and MeFOSA. These samples also all have lower than average concentrations of most precursor compounds (excluding MeFOSA), and the results displayed are primarily ruled by these individual extreme values.

Displaying the results from a different angle on the graph, Figure 39 b., indicates the similarity in results, with over 75 % of the results remaining within the cluster. The main control acting on the samples to produce this pattern is the high concentrations of the persistent compounds, PFOS, PFOA and PFHxS in each sample. These compounds are predominant in most samples, and have greater influence on the samples, though they are counter-balanced by the 5 precursor compounds. The samples are, however, those that contain MeFOSA, EtFOSA and FOSA concentrations that exceed the geometric means.

7.8. CONCLUSION

The concentrations measured in classrooms suggest that concentrations of some PFCs in dust from primary school and nursery classrooms exceed those in offices, homes and cars, with significant differences arising for PFOS, PFOA, PFHxS and MeFOSE. Comparisons of classroom dust concentrations with those from other environments

(Figure 38) suggest that there are additional sources present in classroom environments creating these significant differences. There is also a significant difference between the concentrations and detection rate of MeFOSA in classrooms. The detection rate of this compound in classrooms was much lower than in all the other microenvironments.

PCA analysis of the PFC contamination pattern, as shown Figure 39 a. indicates that the principal distinguishing feature of classroom samples is their factor 2 scores, driven by a greater presence of MeFOSE and EtFOSE. Such higher contributions from precursor compounds are indicative of fresher sources, as the precursor compounds are more volatile. This suggests that the sources of PFCs in classrooms are from frequently conducted activities such as the application of surfactant cleaning products (Walters & Santillo, 2006) or are in greater abundance, such as the emissions from stain-proofed clothing (Danish EPA Report, 2008), but are no different from the sources within other environments.

There is no great difference seen in the PCA results for classrooms and other environments, Figure 39 b, indicating that the sources and contamination of classrooms do not create a different signature of compounds compared to the other environments monitored. The higher concentrations are likely to derive from greater numbers of potential source items present within classrooms rather than different types of sources. In real terms, this may equate to greater use in classrooms of cleaning surfactants and stain resistant coatings on many surfaces (furnishings, carpet and flooring, wall paints and finishes); as well as a higher volume of paper and textiles compared to other indoor microenvironments. All these products commonly contain PFCs and increased use in classrooms would explain the higher concentrations detected.

It can therefore be concluded that the dust concentrations within UK classroom environments do not have a distinct signature of PFCs in comparison to other UK indoor environments. However, the classroom environments do contain significantly greater concentrations, and therefore should be treated as a separate indoor environment when considering child exposure to PFCs in order to conduct a more accurate exposure assessment for this population group.

8. NON-DIETARY EXPOSURE TO PFCS

The hypothesis addressed in this chapter is that the presence of PFCs in the indoor and outdoor environment constitute pathways of external and internal human exposure Leading from this, the aim of this chapter is to determine whether concentrations of PFCs in indoor air and dust, and soil and outdoor air constitute substantial pathways of external human exposure, and thus influence human body burdens.

Contaminated dust and air have been reported previously as important pathways of human exposure to lead, heavy metals and various other chemicals to human exposure (Mielke *et al.* 2010, Hogervorst *et al.*, 2007, Allen *et al.*, 2008, Abdallah & Harrad, 2009, Dirtu & Covaci, 2010). Consistent with observations of significant linear correlation between concentrations of HBCDs in indoor dust and human blood serum (Roosens et al, 2009); correlations between concentrations of PFCs in air and dust with those in human blood serum have also been identified recently (Freberg *et al.* 2010, Haug *et al.* 2010, Nilsson *et al.*, 2010, Fraser *et al.*, 2010).

Further, this chapter uses concentrations of PFOS and PFOA in relevant matrices to derive estimates of human exposure that are compared to the current EFSA tolerable daily intake values (TDIs) for these compounds. These are a threshold value, which if surpassed are expected to produce adverse health effects. Due to the uncertainty involved within the assessment parameters, three scenarios of exposure have been used to deem whether at any of these levels the body burden of people could cross the TDI threshold. In addition, exposure estimates for all target PFCs are compared to

estimates from other studiesof exposure via the diet and drinking water, to evaluate the relative significance of non-dietary exposure.

8.1.EXPOSURE PATHWAYS

This chapter details an assessment of exposure to PFCs via indoor environments. Exposure via air inhalation, dust ingestion and dermal uptake are evaluated, and compared to each other, to dietary exposure, and to relevant exposure guidelines. The assessment in this study is conducted for (a) adults and (b) young children (2 - 6 years) within the UK. The exposure scenarios generated in this chapter are compared to the recommended tolerable daily intake (TDI), above which health risks are expected to occur.

Exposure to PFCs can be categorized as dietary, drinking water and non-dietary (inhalation, ingestion of no-dietary particles and dermal uptake) exposure. At present it is understood that diet tends to be a major pathway of POPs to humans (Linares *et al.* 2010). However in cases such as with TBBPA (Abdallah *et al.* 2008) the exposure from non-dietary pathways can begin to exceed that of dietary. For cases of exposure regarding PFCs, the differentiation between dietary and non-dietary can become blurred with dietary sources adding to non-dietary sources indoors. Examples of this are from foods cooked and packaged in containers coated with PFCs (microwave popcorn bags, ready meals, etc), which allow PFCs to move from the container during the cooking process into the air and surrounding environment (Jogsten *et al.* 2009, Tittlemier *et al.* 2007). Therefore the presence of PFCs from dietary sources indicates the availability of the source to integrate into the non-dietary sources within the microenvironment.

There are three current TDI exposure guidelines within the EU for PFOS and PFOA. These are: (1) the UK Committee on Toxicology (COT) (300 ng PFOS kg^{-1} day^{-1} and 3000 ng PFOA kg^{-1} day^{-1}) (COT 2006a, COT 2006b); (2) the German Federal Institute for Risk Assessment (100 ng PFOS kg^{-1} day^{-1} and 100 ng PFOA kg^{-1} day^{-1}) (Bfr, 2006); and (3) the European Union Environment and Food Safety Authority (EFSA, 2008) (150 ng PFOS kg^{-1} day^{-1} and 1500 ng PFOA kg^{-1} day^{-1}) (EFSA, 2008). The discrepancy noted between the German PFOA TDI and the recommended EFSA value is due to the application of different safety margins applied and also the German values, using high concentrations for drinking water exposure due to contamination event (Hölzer *et al.*, 2008). Since October 2009, the UK FSA (Food Safety Authority) has adopted the EFSA TDI values, and hence the results in this study will be compared primarily to these values. TDIs are available only for PFOS and PFOA, as these are the most persistent perfluorinated compounds, which began to be manufactured in the 1970s and are now primarily a result of precursor degradation and intermediates in manufacturing processes. They are also widespread in the environment and have very long half-lives in people (OECD, 2002), leading to measurable concentrations within human serum and plasma, urine, and breast milk (Kärrman *et al.*, 2007, Harada *et al.*, 2005, Tao *et al.*, 2008). However, other PFCs are of potential concern because of their widespread use in human microenvironments. Moreover, such compounds like fluorotelomers FTs, PFOSAs, and PFOSEs have been shown to degrade within the body to perfluorinated sulfonamides (PFAS) and perfluorinated carboxylic acids (PFCAs) like PFOS and PFOA (Wang *et al.*, 2005), and may thus contribute substantially to body burdens of such stable end products.

PFC exposure via indoor pathways is, amongst other things, proportional to the amount of time spent in different microenvironment categories. Exposure calculations

take into account the proportion of each day spent in each separate microenvironment, averaged over a year. This exposure assessment includes potential exposures via dust ingestion, inhalation, and dermal uptake, as well as soil ingestion and dermal uptake. Figure 40 is a schematic diagram of the movement of PFCs from emission sources to human receptors.

Ingestion of dust and soil occurs from the transfer of particles via hand-to-mouth behaviour and the hypothesized dust deposition onto food and drink from indoor and outdoor environments. The dust may adhere to fingers and skin, and be removed once the fingers are placed into the mouth, or licked. Chemicals can accumulate in dust, due to low ventilation and removal rates, and the chemical's persistence in the environment (Lohman *et al.*, 2007), which is one of the factors linked to the exposure of indoor dust. Vacuuming of a room can cause dust to become re-suspended via the disturbance of the floor by various motions (e.g. of vacuum cleaner and people), and therefore not removed, but remaining, to resettle later on. This dust consists of various products including pollen, fungal spores, soil, fibres and threads, pet dander, trace metals, particles from indoor VOCs etc (EPA Exposure Factors Handbook, 1997). PFCs in household goods, clothing and from in-house use of surfactants are removed to the surrounding environment via general use, and wear and tear.

Ingestion rates are highly variable from one person to the next because of variable factors including age, activity, dust or soil availability (i.e. the amount present in the environment) and location. Ingestion is a pathway for dust to enter the body, and is thus a major pathway for some chemicals to enter the body (e.g. child lead exposure, HBCDs (Sayre *et al.*, 1974, Roosens *et al.*, 2009) surpassing that of dietary intake under some scenarios for some chemicals.

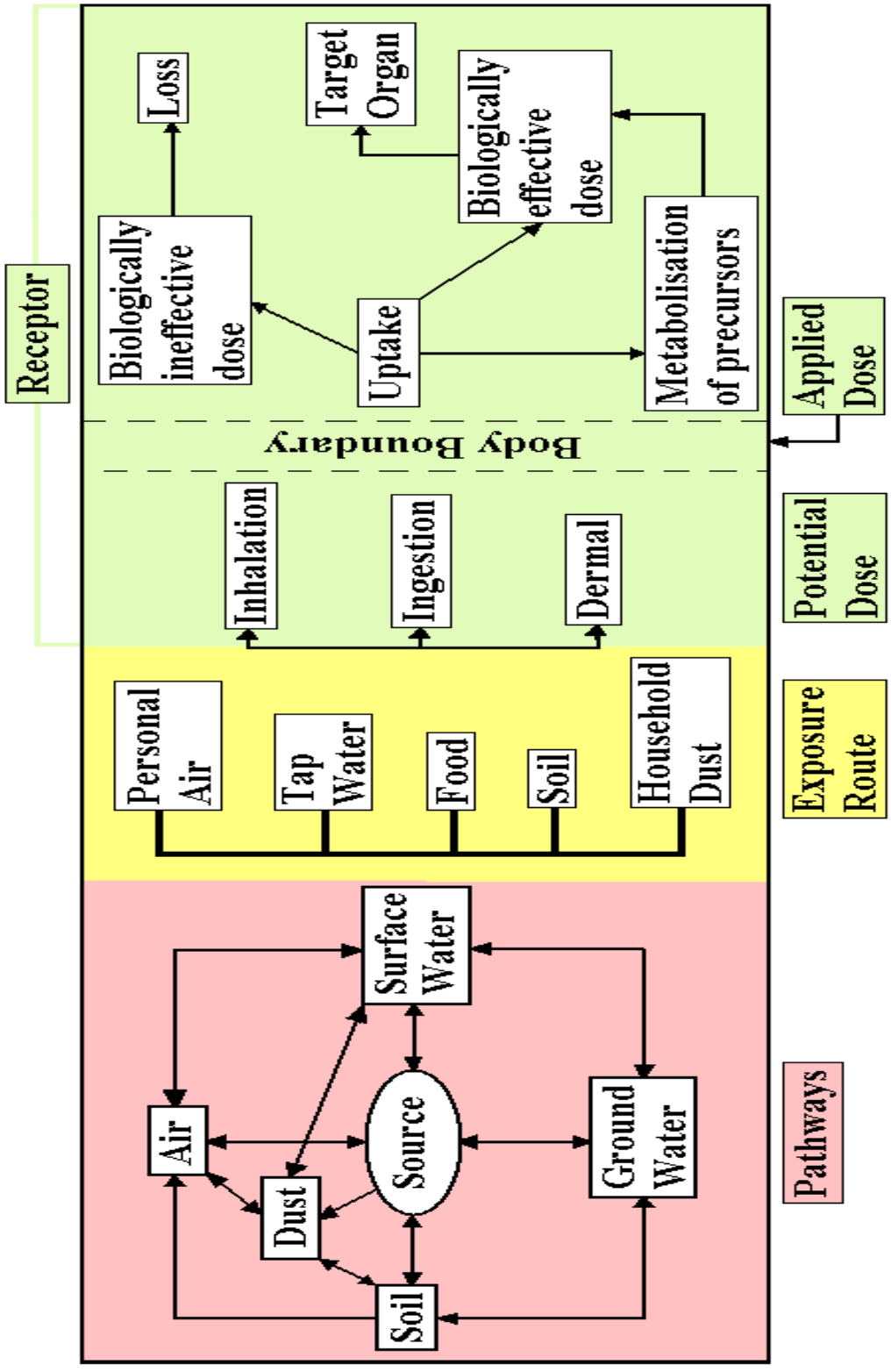


Figure 40 Exposure diagram, adapted from the World Health Organisation Environmental Health Criteria 214, figure 21.

Airborne contamination with PFCs occurs as a result of volatilisation of PFCs from treated goods, with perhaps some contribution from particulates abraded from such goods, as has been reported previously for involatile BFRs (e.g. decabromodiphenyl ether (BDE 209), Webster *et al.*, 2009). Land-based air measurements indicate higher contributions from urban regions (Stock *et al.,* 2004, PERFORCE, 2006), which suggests that the contributing sources derive from not only manufacturing, but also from sources such as the use and disposal of products containing PFCs throughout the life cycle of the chemical. Concentrations within indoor environments exceed those for outdoors for fluorotelomer alcohols and perfluorosulfonamides (Shoeib *et al.*, 2005 and Barber *et al.,* 2007) and this is also evident from the results in this study. The difference between indoor and outdoor concentrations indicates that indoor environments are the principal contributor to overall non-dietary exposure. The majority of time throughout the day is spent indoors, where exposure to higher air concentrations and particulate concentrations occurs (Shoeib *et al.*, 2005).

8.2.EXPOSURE ASSESSMENT

A deterministic approach was taken to determine the exposure received from various environments and pathways of exposure. The exposure assessment was conducted for three scenarios to create a "low", "typical" and "high" exposure value and the chosen parameters for each scenario are described in Table 69. This deterministic approach has been used previously for estimating exposure to brominated flame retardants (Abdallah & Harrad, 2009, Harrad *et al.*, 2008). However, the scenarios used in this study are simply an estimate of the likely range, and a large amount of variability is created by the uncertain nature of the dust ingestion rates for both adults and children.

Studies of dust ingestion rates are few in number, and consist of estimates derived from soil and dust ingestion scenarios (RIVM, 2008, Health Canada, 2004), with few originating from primary data collection (Stanek & Calabrese, 1995, US EPA, 2002, US EPA 1997, Roberts & Dickey, 1995, Lewis *et al.*, 2001). Uncertainties are also introduced into the exposure assessment by the assumption of average times spent in various microenvironments, and typical activity patterns of individuals (detailed in Table 70), along with the variability in dust ingestion rates. The internal uptake rate of the chemicals into the body is also an ambiguous point, where the behaviour of PFCs differs drastically from other mammals and animals (Harada *et al.* 2005), making it difficult to use in vivo derived results (Kennedy *et al.*, 2004, Vestergren *et al.*, 2008).

Table 69 Deterministic modelling scenarios.

Scenario	Sample Concentration Value	Uptake Rate
Low	5th Percentile	Mean
Typical	Median	Mean
High	95th Percentile	95th Percentile

The exposure assessment (expressed in pg (kg bw)$^{-1}$ d^{-1}) was calculated according to equation 2, which is based on the algorithm from Currado & Harrad, (1998), for the estimation of both adult and child exposure (Equation 5), where C represents the concentration in the workplace (w), home (h) and office (o) or other microenvironment (x) and *F* denotes the fraction of time spent daily in each of the environments, coupled with the respiration rate (R_r) for either adults or children.

Equation 5 Exposure algorithm (Currado & Harrad, 1998)

$$\sum exposure = ([C_w F_w] + [C_\square F_\square] + [C_o F_o] + \ldots\ldots [C_x F_X]) R_r$$

Equation 6 Exposure assessment model

$$Exposure = \frac{C \times I \times U \times EF}{\mathrm{BW}}$$

Equation 2 shows exposure is a function of the concentration (C) in the exposure medium, the intake of the exposure matrix (I) and the uptake (U) of the matrix across the internal boundary (e.g. gastrointestinal uptake) to produce an internal dose. The equation is altered depending on the fraction of time spent in the specific environment (EF), and then expressed relative to body weight (BW). This basic equation was used as the foundation of the exposure assessments conducted here and the parameters are detailed in Table 70.

All parameters used within the assessment were derived from various studies and are provided in Table 70. The exposure assessment conducted assumes that all input parameters are independent and therefore the parameters have been selected from various relevant studies.

Dermal exposure is estimated using the algorithm shown in Equation 7, and incorporates surface area (SA) along with the dermal uptake rate (Ud) across the skin. The surface area, SA, of skin which comes into contact with soil or dust has been associated with only the hands, as the hands are likely to be frequently coming into contact with dust and to an extent soil (U.S. EPA, 2008). The dermal absorbance differs between adults and children, because of the tendency of children to touch and handle more objects, and the greater amount of time children spend on the floor and spend with their hands on the floor. All parameters used are listed in Table 70.

Equation 7 Dermal exposure assessment

$$Dermal\ Exposure, Ed\ = \frac{C\ \times Ud\ \times SA\ \times EF}{BW}$$

In the absence of conclusive data, the proportion of exposure via dermal contact that occurs in the home versus other microenvironments is assumed identical to that used for estimating exposure via inhalation and dust ingestion. The effect of this assumption on the accuracy of the estimate of actual dermal contact is unknown, but it is likely that dermal contact of dust occurs throughout the day from the majority of objects that are touched. Therefore, even after washing, the skin is likely to come into contact with dust and PFC treated surfaces within a very short time scale. Dermal exposure estimates for individual microenvironments are expressed in Table 70, alongside an overall estimate of dermal exposure (E_d).

Table 70 Variable parameters assumed for the deterministic modeling, ($_A$ - adult, $_C$ - Child)

Parameter	Abbreviation	Value	Reference
Adult body weight	BW_A	70 kg	Jeffries, 2009
Child body weight	BW_C	20 kg	Freeman et al., 1995
Adult dust ingestion, mean (high)	IG_{DA}	20 (50) mg day^{-1}	Jones-Otazo *et al.*, 2005
Child dust ingestion, mean (high)	IG_{DC}	50 (200) mg day^{-1}	Jones-Otazo *et al.*, 2005
Adult Soil ingestion, mean (95^{th} percentile)	IG_{SA}	10 (331) mg day^{-1}	Stanke & Calabrese, 2000
Child Soil ingestion, mean (95^{th} percentile)	IG_{SC}	31 (106) mg day^{-1}	Stanke & Calabrese, 2000
Dust intestinal uptake	U_I	95 %	Johnson *et al.*, 1979 Seacat *et al.*, 2002
Adult time pattern	EF	Home – 68.5 % Office – 19.7 % Car – 7.4 % Outdoors – 5.4 %	UK National office of statistics, 2000 & 2005
Child time pattern	EF	Home – 75.7 % Classroom – 17.9 % Car – 4.2 % Outdoors – 2.2 %	UK National office of statistics, 2000 & 2005
Adult respiratory rates, mean (95^{th} percentile)	IR_A	13.8 (21) m^3 day^{-1}	US EPA, 2008
Child respiratory rates, mean (95^{th} percentile)	IR_C	8.7 (14.97) m^3 day^{-1}	US EPA, 2008
Adult dermal contact area (hands)	SA_A	990 cm^2	US EPA, 2008
Child dermal contact area (hands)	SA_C	360 cm^2	US EPA, 2008
Dermal uptake mean (high)	U_D	0.005 (0.05) mg cm^{-2}	EFSA Journal, 2008

8.3.RESULTS

The exposure assessment was conducted for adults (> 18 y) and children (1 - 6 y), to calculate the exposure they receive via dermal, ingestion and inhalation pathways from home environments, offices or classrooms, cars and outdoors. The results are displayed in Table 71 and Table 72, for adult and child exposures. The results indicate that under the "low" scenario for adults the exposure from all microenvironments and uptake pathways remains in the range of 1 – 30 pg (kg bw)$^{-1}$ d^{-1}, whilst child "low" exposure has a much wider range over 1 - 90 pg (kg bw)$^{-1}$ d^{-1}. This large difference between exposures is seen throughout the exposure scenarios and indicates that children can be considerably more exposed than adults, especially when normalised to their lower body weights.

Summaries of the exposure estimates for both UK adults and children in Table 71 and Table 72 individually express the associated pathways of exposure for each individual microenvironment. It is evident that the inhalation pathways are a less significant factor in exposure than dust, and this primarily arises from the concentration of PFCs measured in air remaining within the pg m^{-3} region, whilst dust samples were on an order of magnitude higher within the ng g^{-1} range.

Table 71 Adult daily exposure

Microenvironment	Pathway	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
Homes	**Ingestion - Dust**								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	0.03	0.04	0.04	<0.01	0.01	<0.01	0.02	0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.59	0.24	0.64	0.04	0.18	0.11	0.27	0.49
	Inhalation - Air								
pg (kg bw)$^{-1}$ d^{-1}	low	0.07	0.07	0.01	0.01	3.3	3	25	12
pg (kg bw)$^{-1}$ d^{-1}	typical	1.5	3.2	3.1	0.01	9	6.1	100	80
pg (kg bw)$^{-1}$ d^{-1}	high	18	32	25	1.6	64	150	560	310
	Dermal - Dust								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.02	0.02
ng (kg bw)$^{-1}$ d^{-1}	high	0.04	0.09	0.12	0.02	0.07	0.11	0.58	0.43
Offices	**Ingestion - Dust**								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	0.02	0.02	0.01	<0.01	<0.01	<0.01	0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.14	0.12	0.39	0.01	0.09	0.02	0.08	0.1
	Inhalation - air								
pg (kg bw)$^{-1}$ d^{-1}	low	0.79	0.29	<0.01	<0.01	0.66	0.38	3.9	7.6
pg (kg bw)$^{-1}$ d^{-1}	typical	2.1	0.67	3.6	<0.01	1.8	1.9	15	15
pg (kg bw)$^{-1}$ d^{-1}	High	5.4	12	15	2.1	8.9	14	73	54
	Dermal - Dust								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.15	0.13	0.41	0.01	0.1	0.02	0.08	0.1

Microenvironment	Pathway		PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE
Cars	**Ingestion - Dust**								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.03	0.02	0.04	<0.01	0.01	0.01	0.02	0.02
	Dermal - Dust								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.03	0.02	0.04	<0.01	0.01	0.01	0.02	0.02
Outdoors	**Ingestion - Soil**								
pg (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	high	0.58	1.9	0.39	0.04	0.05	<0.01	0.03	0.02
	Inhalation - Air								
pg (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	0.13	0.01	0.15	0.28
pg (kg bw)$^{-1}$ d^{-1}	typical	8.9	2.6	7.4	<0.01	0.32	0.36	4.8	9.4
pg (kg bw)$^{-1}$ d^{-1}	high	40	11	130	<0.01	4.6	10	73	92
	Dermal - Soil								
pg (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	high	0.31	1	0.21	0.02	0.03	<0.01	0.02	0.01
Daily Total	**low**	**0.01**	**<0.01**	**0.01**	**<0.01**	**<0.01**	**<0.01**	**0.03**	**0.03**
(ng (kg bw)$^{-1}$ d^{-1})	**typical**	**0.07**	**0.06**	**0.07**	**<0.01**	**0.02**	**0.01**	**0.17**	**0.14**
	high	**1**	**0.68**	**1.8**	**0.09**	**0.54**	**0.45**	**1.8**	**1.6**

Table 72 Child daily exposure

Microenvironment	Pathway	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
Homes	**Ingestion - Dust**								
ng (kg bw)$^{-1}$ d^{-1}	low	0.04	0.02	0.06	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	0.25	0.34	0.38	<0.01	0.07	0.04	0.17	0.06
ng (kg bw)$^{-1}$ d^{-1}	high	9.1	3.7	9.9	0.62	2.7	1.7	4.2	7.6
	Inhalation - Air								
pg (kg bw)$^{-1}$ d^{-1}	low	0.16	0.16	0.01	0.02	8.1	7.4	60	30
pg (kg bw)$^{-1}$ d^{-1}	typical	3.6	7.8	7.5	0.02	22	15	250	200
pg (kg bw)$^{-1}$ d^{-1}	high	49	86	66	4.2	170	400	1500	830
	Dermal - Dust								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.01	0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	0.01	<0.01	<0.01	<0.01	0.03	0.03
ng (kg bw)$^{-1}$ d^{-1}	high	0.06	0.13	0.17	0.02	0.1	0.16	0.82	0.6
Classrooms	**Ingestion - Dust**								
ng (kg bw)$^{-1}$ d^{-1}	low	0.06	0.01	0.01	<0.01	<0.01	<0.01	0.01	0.05
ng (kg bw)$^{-1}$ d^{-1}	typical	0.36	0.1	0.3	<0.01	0.01	0.01	0.19	0.38
ng (kg bw)$^{-1}$ d^{-1}	high	4.1	1.1	13	<0.01	0.46	1	7.5	9.4
	Dermal - Dust								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	0.01	<0.01	0.01	<0.01	<0.01	<0.01	0.01	0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.39	0.1	1.2	<0.01	0.04	0.1	0.71	0.89
Cars	**Ingestion - Dust**								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	0.01	0.01	0.02	<0.01	<0.01	<0.01	0.01	0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.25	0.14	0.32	0.01	0.1	0.09	0.14	0.15

Microenvironment	Pathway	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
	Dermal - Dust								
ng (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ng (kg bw)$^{-1}$ d^{-1}	high	0.02	0.01	0.03	<0.01	0.01	0.01	0.01	0.01
Outdoors	**Ingestion - Soil**								
pg (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	0<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	typical	0.02	0.04	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	high	0.27	0.88	0.18	0.02	0.02	<0.01	0.01	0.01
	Inhalation - Air								
pg (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	0.12	0.01	0.13	0.25
pg (kg bw)$^{-1}$ d^{-1}	typical	8	2.3	6.7	<0.01	0.29	0.33	4.3	8.5
pg (kg bw)$^{-1}$ d^{-1}	high	40	10	126	<0.01	4.5	10	72	91
	Dermal - Soil								
pg (kg bw)$^{-1}$ d^{-1}	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	typical	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
pg (kg bw)$^{-1}$ d^{-1}	high	0.05	0.16	0.03	<0.01	<0.01	<0.01	<0.01	<0.01
Daily Total	**low**	**0.1**	**0.04**	**0.07**	**<0.01**	**0.01**	**0.01**	**0.08**	**0.09**
(ng (kg bw)$^{-1}$ d^{-1})	**typical**	**0.64**	**0.47**	**0.73**	**<0.01**	**0.12**	**0.07**	**0.65**	**0.69**
	High	**14**	**5.2**	**25**	**0.66**	**3.6**	**3.5**	**15**	**19**

8.4.MICROENVIRONMENT EXPOSURE COMPARISON

The importance of measuring a variety of microenvironment types is illustrated by the graphs in Figure 41 and Figure 42, which show the contributions from each environment to adults and children. Both indoor and outdoor exposure has been calculated, with indoor exposure including dust ingestion and dermal uptake and indoor air inhalation. Outdoor exposure includes soil ingestion and dermal absorption and outdoor air inhalation. All exposure scenarios were calculated on a pro-rata basis, with time fractions described in Table 70. It is clear that, while some microenvironments make important contributions to exposure to some people, they do not for others. Improvements in the deterministic modelling approach could be achieved by determining more accurately the proportion of time individuals spend in different microenvironments. The importance of individual microenvironments towards exposure is also strongly influenced by the scenario of exposure used. For adults, this can be identified in the outdoor exposure scenarios, where the contribution of the outdoor environment is unimportant apart from under the “high” exposure.

Results for adults indicate that their major source of non-dietary exposure is derived from home environments, due to the amount of time people spend in them. This time fraction is also why exposure from offices is relatively high compared to that received from car and outdoor environments.

For child exposure under each of the three scenarios, exposure is dominated by the two microenvironments children spend the majority of their time in; homes and classrooms. This dominance of the two microenvironments is exemplified in the “high” exposure, where the exposure is 10 times higher than the “typical” scenario. This is especially prevalent for the classrooms, considering the shorter amount of time spent in them compared to homes. For

both car and outdoor environments, the exposure to children under the "low" and "typical" scenarios is negligible. For children (Figure 42) the exposure received from classroom environments is 43 % of the total exposure, whilst homes is 53 %. Despite only a 3rd of a Childs time being spent in the classroom, the exposure is comparable to that received from homes, where 70 % of the time is spent. Results for classroom exposure are raised, along with PCB and PBDE exposure from these classrooms (Harad *et al.* 2010), this is particularly true for PFHxS child exposure.

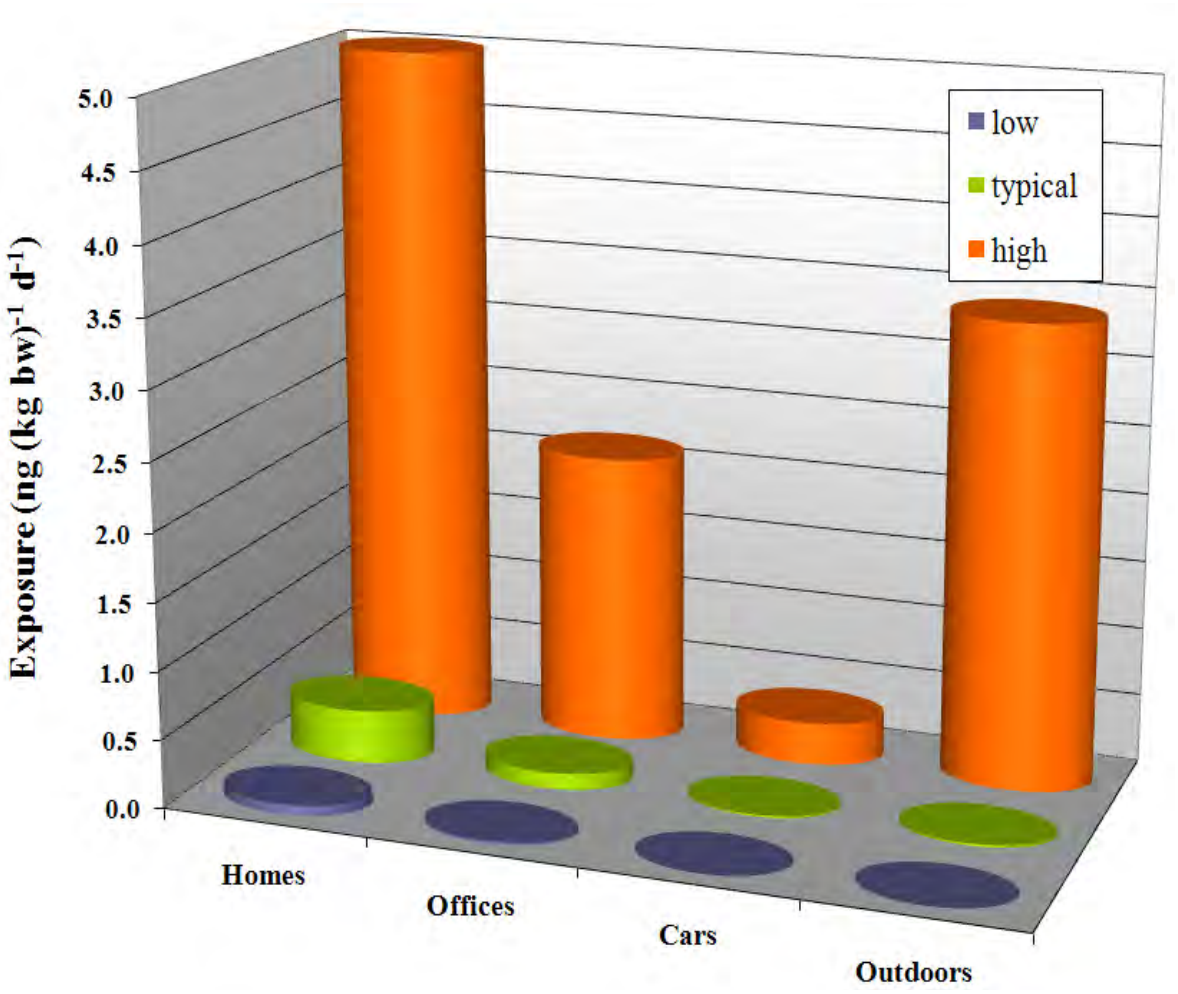


Figure 41 Adult microenvironment specific exposures (ng (kg bw)$^{-1}$ d^{-1}).

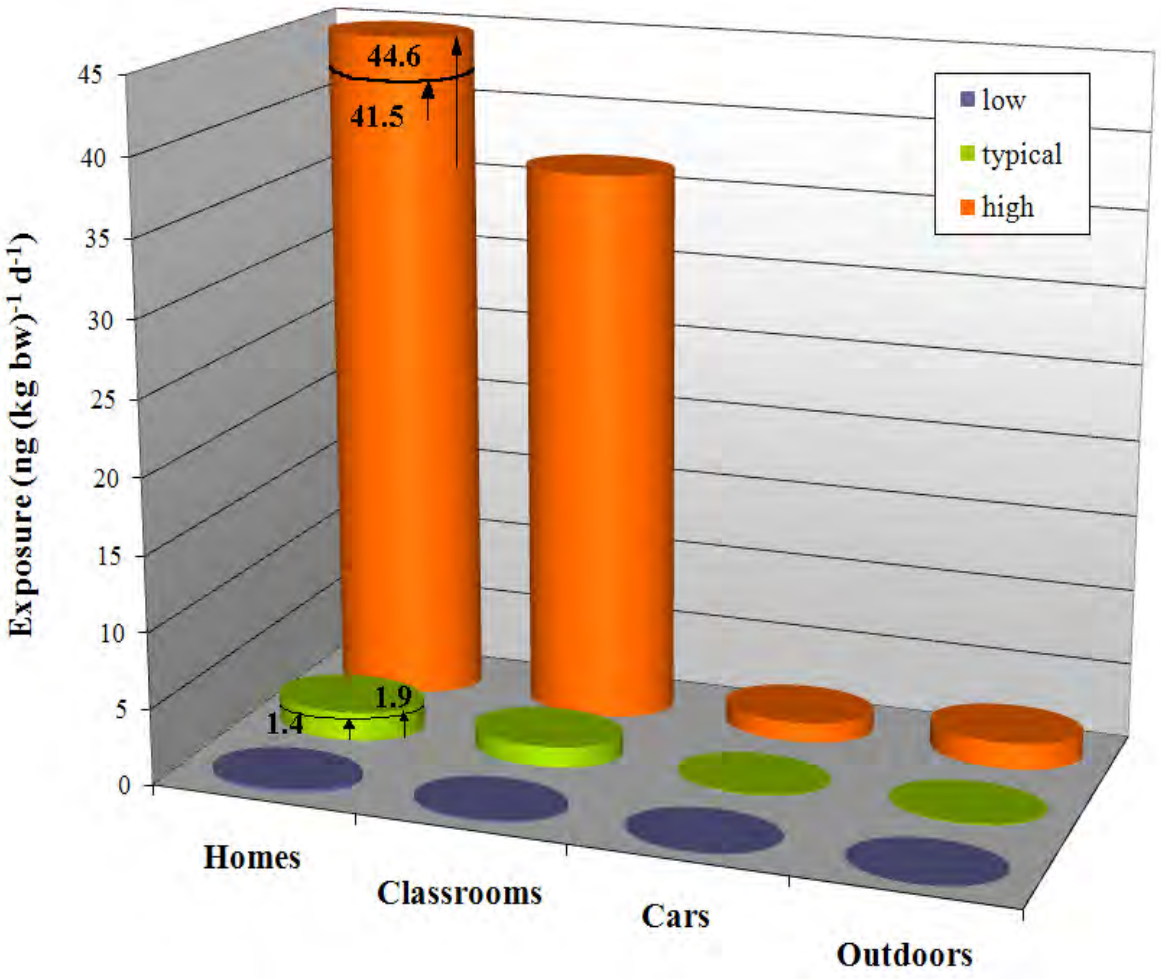


Figure 42 Child microenvironment specific exposures (ng (kg bw)$^{-1}$ d^{-1}). Home exposure is represented by dermal and ingestion uptake upto the line (dermal + ingestion) and with the additional inhalation pathway to the top of the bar (dermal + ingestion +inhalation).

8.5.EXPOSURE AND PATHWAYS

The contributing pathways of exposure for both adults and children differ considerably, with ingestion of indoor dust and outdoor soil acting as the major pathway for adults and children under the "high" exposure scenario. For adults, this ingestion pathway becomes comparable to inhalation at around 0.2 ng (kg bw)$^{-1}$ d^{-1} for the "typical" scenario. The differences noted in the pathway contributions in Figure 43 are caused by the different ingestion rates used. For instance, ingestion rates for the "high" scenario are 1.5 times higher than the "typical"

scenario, but the difference in the inhalation scenarios is only 0.5 times different. This has led, for example, to dermal exposure superseding inhalation in the "high" exposure scenario.

Due to behavioural differences and a higher intake than adults, ingestion is the dominant exposure pathway for children under all three exposure scenarios. For children, this also been seen for HBCDs and PBDEs (Fraser *et al.*, 2009, Roosens *et al.*, 2009). The child "typical" exposure is an order of magnitude higher than the adult "typical" exposure, which is a result of the slightly longer fraction of time spent at home by children, but also the higher ingestion rates, for children.

The uptake difference is also the reason for a difference in the dermal exposure estimates and the variability in contribution to overall exposure. Dermal uptake is strongly influenced by the surface area of the hand, and considering this was the only area of the body modeled for exposure, the difference between adult and child exposure is substantial.

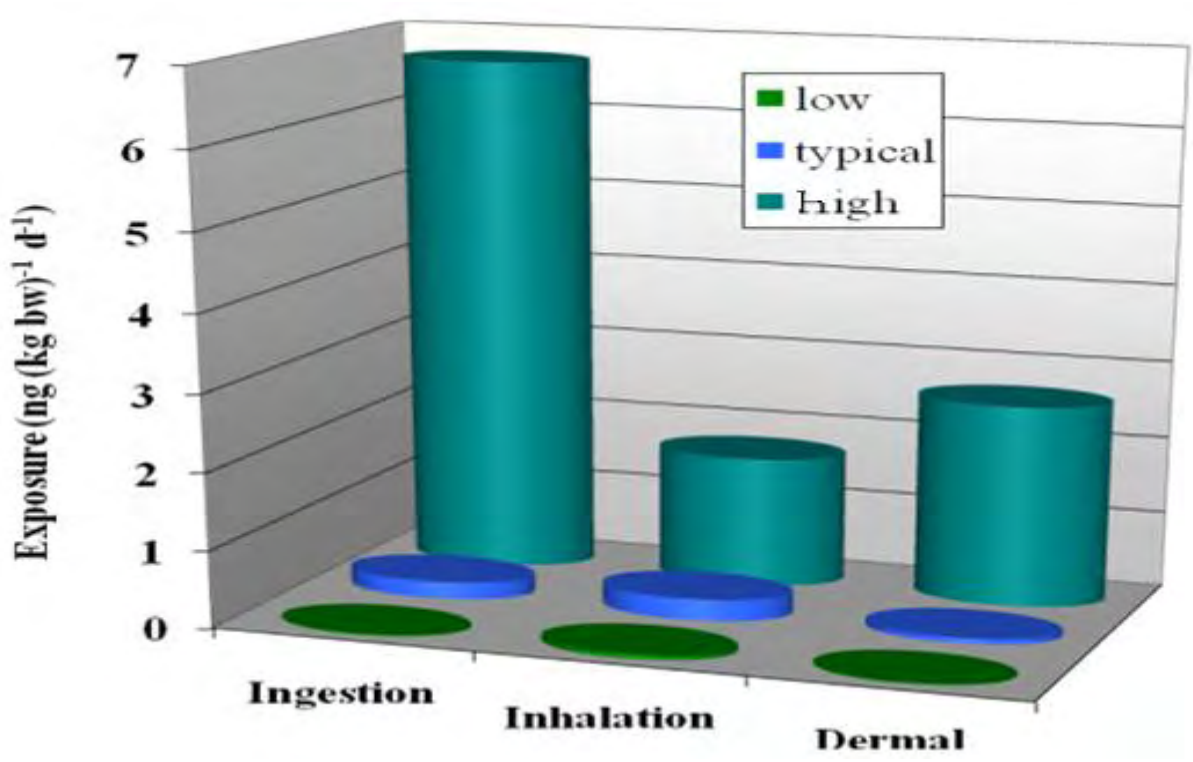


Figure 43 Adult exposure via different pathways (ng (kg bw)$^{-1}$ d^{-1}).

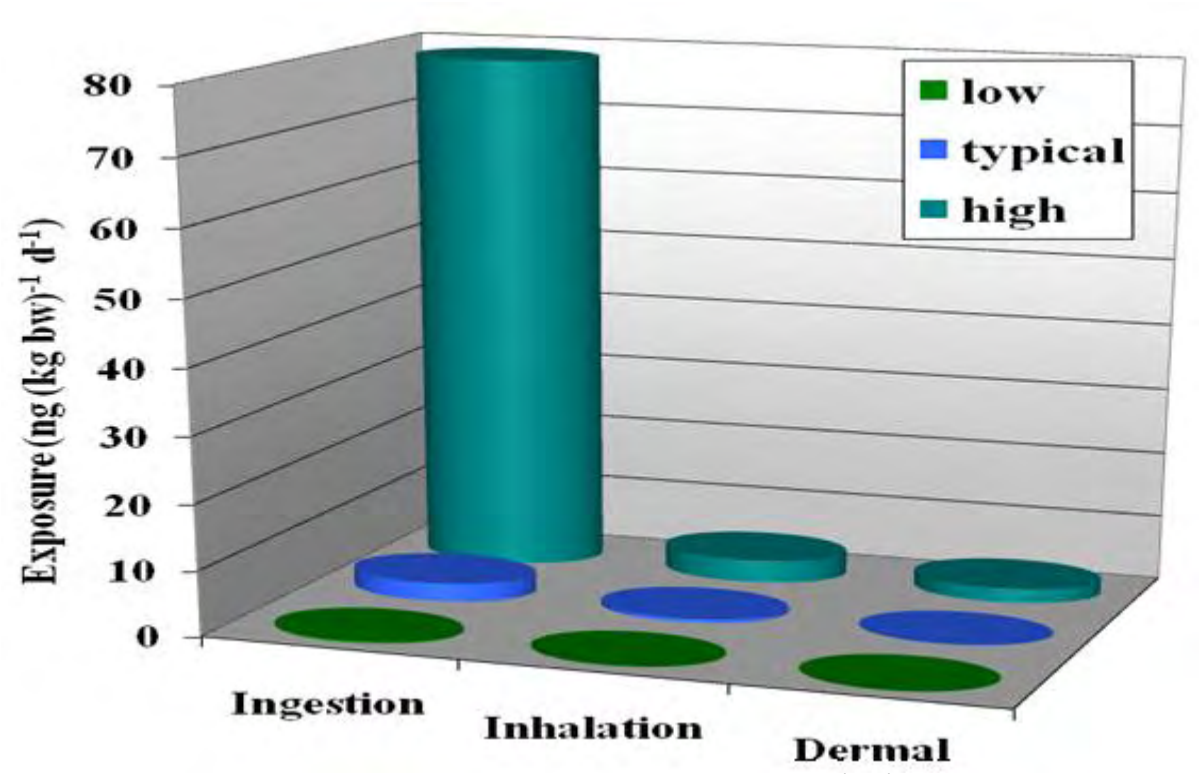


Figure 44 Child exposure via different pathways (ng (kg bw)$^{-1}$ d^{-1})

8.6.TOLERABLE DAILY INTAKES

Comparison of the above estimates of non-dietary exposure with the TDI values recommended (Table 73) indicates that adult non-dietary exposure amounts to < 0.01 % of the German, EU and UK TDI values for PFOS and PFOA. Whilst the child exposure derived from this study contributes 0.6 %, 0.4 % and 0.2 % for PFOS, and 0.5 %, < 0.01 % and < 0.01 % for PFOA in comparison to the German, EU and UK TDIs. Based on the EFSA TDI estimates, the pathways of exposure monitored in this study have minimal impact for both PFOS and PFOA for adults. German dietary intakes are estimated at a mean (and 95th percentile) of 1.5 (4.5) ng (kg bw)$^{-1}$ day^{-1} for PFOS and 2.9 (11.5) ng (kg bw)$^{-1}$ day^{-1} PFOA, for adults. In relation to exposure determined from this study, the results for children indicate that exposure from dust, air and soil could be just as significant as dietary intake and in the "high" scenario, exceed dietary exposure. The ∑PFC non-dietary "typical" (and "high")

intake for children is 3.4 (86) ng (kg bw)$^{-1}$ day^{-1} and lies within the range estimated by Fromme *et al.,* (2009) of 1.75 - 350 ng (kg bw)$^{-1}$ day^{-1}, but the PFCs included in this estimate vary from the ones used for the ∑PFC exposure in this study. For adults, the non-dietary exposure is not as significant but will contribute as much as 7 ng (kg bw)$^{-1}$ day^{-1} to their daily exposure, and body burdens.

In total the exposure to PFOS and PFOA through dietary, drinking water and non-dietary pathways combined (see Table 73 TDI, dietary, and water intake (ng (kg bw)$^{-1}$ d-1)Table 73), does not inhibit upon the current set TDI values. For both adults and children the total exposure expected to be received by the general population is not believed to cause ill-health, because it remains well below the TDI thresholds.

Due to continued research and the effects of PFCs on endocrine disruption, and peroxisome proliferation (Berthiamue & Wallace, 2002, Austin *et al.* 2003) it is possible that the TDI values will continue to fall, and exposure via the individual exposure pathways may become more important aspects in terms of health, than previously thought. This is particularly important for the pathway through non-dietary exposure, as the estimated exposure through diet is also likely to reduce over time, due to improvements in analytical techniques. The present dietary measurements include a large number of non-detected samples, which are represented at the LOD, which can introduce over estimated of the true concentrations (Trudel *et al.* 2008, Tittlemier *et al.*, 2007, FSA 2009). Therefore with the likelyness of TDIs to continue to drop and the expected decreases in estimated dietary exposures, the non-dietary pathways may have a greater impact on human health than presently expected.

In comparison to drinking water exposure (Table 73), this study indicates that non-dietary sources are a much more significant pathway. However, recent incidents associated with manufacturing facilities and waste treatment disposal have led to drinking water displaying considerably higher concentrations (Little Hocking = 3.55 ng mL^{-1} (Tillett, 2007), New Jersey

= 0.19 ng mL^{-1} (Post *et al.*, 2009), (Hölzer *et al.*, 2009)) than that seen in the general environment (1 - 100 ng L^{-1}, Quinones & Snyder, 2009, Jin *et al.*, 2009, Loos *et al.*, 2007).

The contributions of non-dietary sources, diet and drinking water to exposure in comparison to the TDI under "typical" and "high" exposure scenarios, are displayed in Figure 45 and Figure 46. They indicate that, under all scenarios for adults, the diet is the major contributor to exposure, for both PFOS and PFOA.

The situation for children is different, and whilst diet prevails as the most contributing pathway under the "typical" scenario, PFOS under the "high" scenario is primarily driven via non-dietary pathways. Under this scenario, non-dietary exposure for PFOS accounts for 75 % of the estimated total daily exposure.

Table 73 TDI, dietary, and water intake (ng (kg bw)$^{-1}$ d^{-1})

Country	Reference	Adult (ng (kg bw)$^{-1}$ d^{-1})			Child (ng (kg bw)$^{-1}$ d^{-1})		
		∑PFC	PFOS	PFOA	∑PFC	PFOS	PFOA
Tolerable Daily Intakes							
UK	COT 2006a,b		300	3000		300	3000
EU	EFSA, 2008		150	1500		150	1500
Germany	BfR, 2006		100	100		100	100
Total Non-Dietary Daily Intake		0.55	0.07	0.06	3.38	0.64	0.47
Total Daily Dietary Intake							
Germany	Fromme *et al.*, 2009		1.5 (4.5)[A]	2.9 (11.5)[A]			
Japan	Kärrman *et al.*, 2009		0.35 – 5.04	0.45 – 2			
UK	FSA, 2009		1 –10*	<0.05 – 10*	0.8 – 40*	<0.05 – 40*	<0.05 – 30*
Canada	Tittlemier *et al.*, 2006	4			1.05		
EU	EFSA, 2008		60 - 200	2 - 6			
Total Daily Tap Water Intake							
Germany	Fromme *et al.*, 2009		0.023 (0.13)[A]	0.022 (0.087)[A]			
Spain	Ericson *et al.*, 2007		0.1	0.12			

*Lower – Upper bound estimated average total dietary intakes, 2007 - 2008. [A] 95th percentile estimate

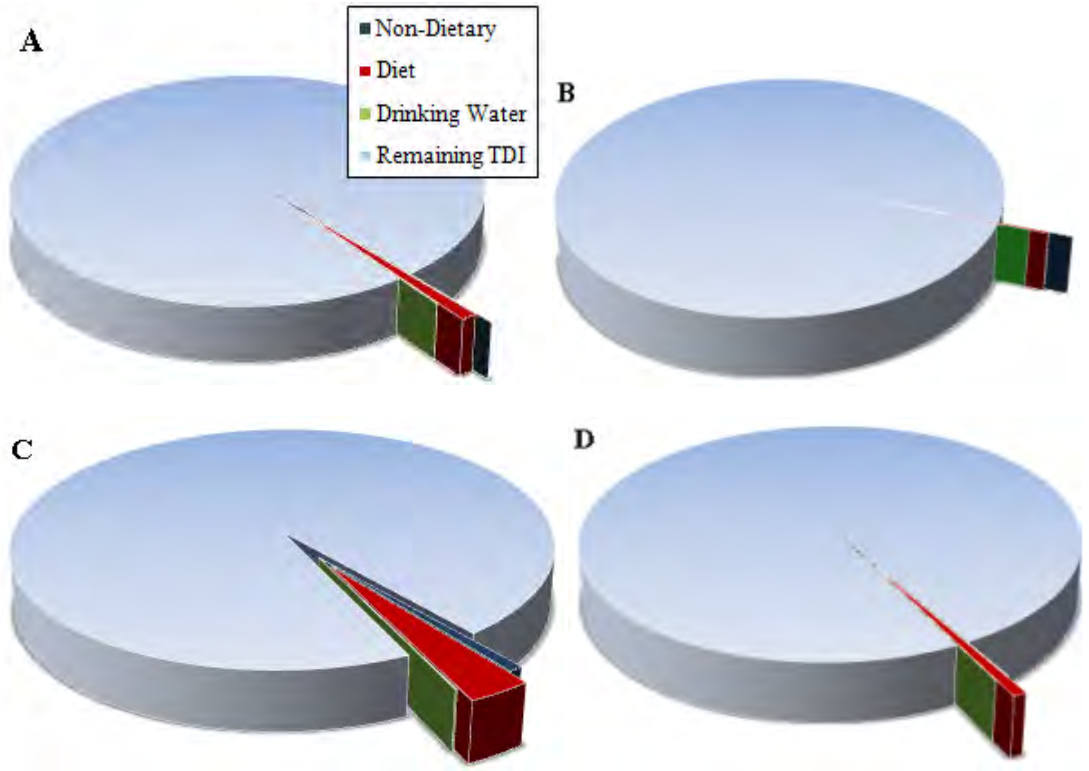


Figure 45 Adult exposure and contribution towards EFSA TDI, a, PFOS "typical" exposure, b, PFOA "typical" exposure, c, PFOS "high" exposure, d, PFOA "high" exposure.

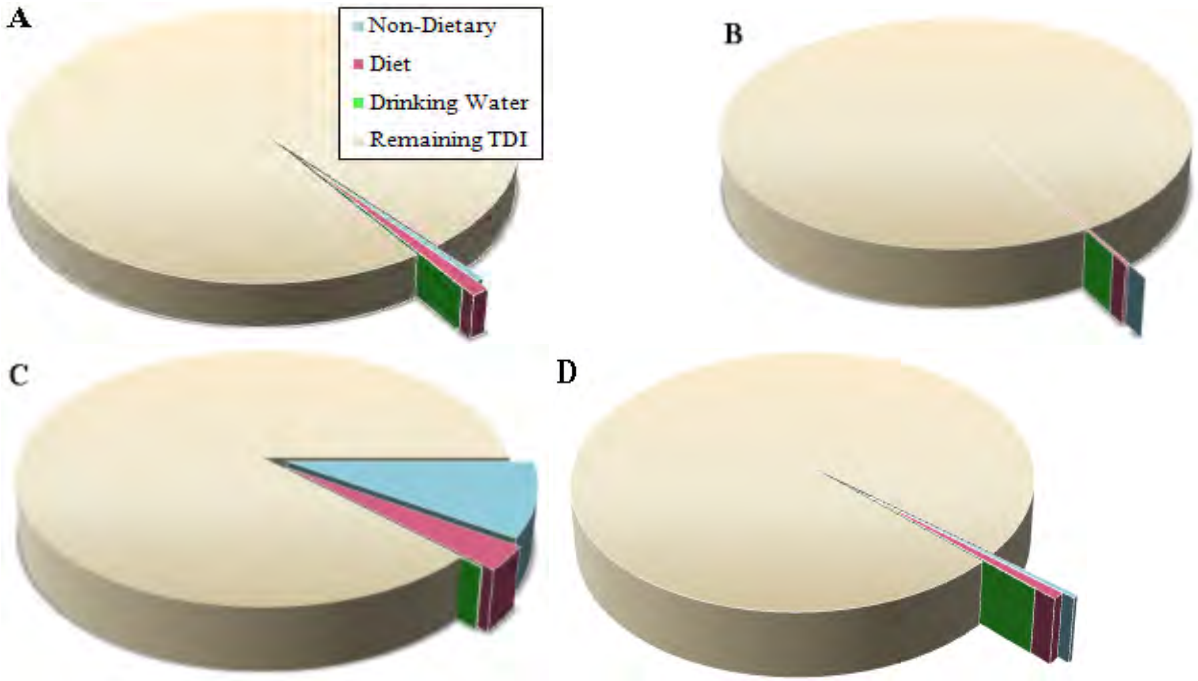


Figure 46 Child exposure and contribution towards EFSA TDI, a, PFOS "typical" exposure, b, PFOA "typical" exposure, c, PFOS "high" exposure, d, PFOA "high" exposure.

By comparison for PFOA, non-dietary exposure accounts for just 30 % of the daily estimate of overall exposure.In all cases, for both adults and children, it is clear that the least contributing pathway is from the drinking water. However, in cases of contamination (Hölzer *et al.*, 2009, Post *et al.*, 2009), the contribution can exceed the exposure from both dietary and non-dietary pathways (exposures of 5 – 270 ng (kg bw)$^{-1}$ day^{-1}).

A Risk Index (RI) was calculated from the TDI, the exposure to PFCs from non-dietary, dietary and drinking water pathways, to assess the potential risk associated with the presence of these chemicals. The RI was conducted according to a method by RIVM, 2008, and the equation is displayed in Equation 8, where the non-dietary exposure is coupled with background exposure (which is assumed to be diet and drinking water). The dietary and drinking water concentrations were derived from Fromme *et al.*, (2009), Germany (which has similar concentrations in indoor dust measured in Chapter 9), the mean concentrations were used for the prediction of the "low" and "typical" scenario and the 95th percentile was used for the "high" scenario. Results are presented in Table 74 and indicate that values for all three scenarios remain < 0.04 and < 0.12 for adults and children (respectively), which indicates a very low amount of associated risk. RI values > 1 indicate that the TDI has been surpassed and that the risk associated is highly relevant, and values > 0.8 indicate that safety procedures may have to be taken to prevent the value from rising in the future, according to an endpoint derived from the tolerable daily intake.

Equation 8 Risk index calculation (RIVM, 2008)

$$RI = \frac{non-dietary + background}{TDI}$$

Table 74 Risk index results for PFOS and PFOA and Precursor Compounds for adults and children.

RI	*Adult PFOS*	*Adult PFOA*	*Adult Precursors*	*Child PFOS*	*Child PFOA*	*Child Precursors*
Low	0.01	<0.01	0.01	0.01	<0.01	0.01
Typical	0.01	<0.01	0.01	0.01	<0.01	0.03
High	0.04	0.01	0.08	0.12	0.01	0.6

The results of the RI are low for PFOS and PFOA, even at the "high" exposure scenario for adults and children, but remain higher than RI values for pesticides found in house dust (RIVM, 2008). The values indicate that, presently, the uptake of PFOS and PFOA from all three major pathways on a daily basis is unlikely to put the general population at risk of the associated adverse effects.

The metabolisation of precursor compounds to PFOS has been identified in rats and other in-vivo experiments to result in additional concentrations of PFOS in the organism (Benskin *et al.*, 2009, Vestergren *et al.*, 2008, Tomy *et al.*, 2004), suggesting that the presence of PFOSAs and PFOSEs detected in the samples will have an additional affect upon the body burdens for PFOS (little evidence was found for the biotransformation resulting in measurable concentrations of PFOA (Tomy *et al.*, 2004)). Thus, a highly conservative estimate was also derived from the assumption that 100 % of the PFOS precursor compound dose would be metabolised directly to PFOS, and provided a RI value of 0.08 and 0.6 for adult and child (respectively) "high" scenarios. Even under these highly conservative scenarios, the contribution of precursor PFCs to exposure, and the associated risk, still remains below the level of concern (> 0.8). Though it is important to state that under a probabilistic distribution the "high" exposure for children appears to be approaching that of the level of concern (20 % below), and could surpass this value in the future if the TDI values are reduced further.

8.7.PHARMACOKINETIC MODELING

A simple steady state, first order, one compartment pharmacokinetic (PK) model has been used to predict the relative concentrations in blood which are attributed to non-dietary sources. The model is based upon a first-order PK model (Equation 9), which uses dose, elimination rate and volume of distribution to predict the concentration within the blood (Egeghy & Lorber, 2010). The steady state model is based upon the PK model in Equation 10, which depicts changes in blood concentrations over time. The ability of the model to accurately ascertain the non-dietary source contribution, for PFOS and PFOA in serum blood concentrations is improved by the long period of time people have been exposed to background concentrations (Egeghy and Lorber, 2010) and the slow elimination rates (Thompson *et al.*, 2010). At present the elimination rates, from within the body are not well represented, and indicate variability within people depending upon, age, lifestyle, sex, ethnicity, diet and occupation (Calafat *et al.* 2007). At present, it is known that PFCs partition into protein rich matrices within the body. Therefore it is expected (in respect to behavior in mammalian test subjects, (OECD 2002, Bossi *et al.*, 2005)) that the liver, and other organs will act as reservoirs to the compounds. However the presence in blood can be used to represent the presence in the body and the potential body burden it may be creating. The use of a PK model provides an insight into the potential harm and health effects that compounds from non-dietary sources may be creating from the body burdens induced via exposure. The movement of PFCs around the body is primarily driven by the partitioning of the compounds to proteins, and thus partitioning into the blood and major organs (Hundley *et al.*, 2006, Kelly *et al.*, 2009).

Parameters within the model are DP, the daily absorbed dose (ng (kg bw)$^{-1}$ day^{-1}), Vd, volume of distribution is a ratio of the amount of the compound in the body compared to the concentration in blood serum (mL kg bw^{-1}), kP, the first order-elimination rate from the body

(day^{-1}), and CP, the serum concentration (ng mL^{-1}). Values for these parameters were defined in Thompson *et al.* 2010, where they were calibrated against blood samples from Australia and USA. The Vd is the most sensitive parameter within the model (Thompson *et al.* 2010) and was previously provided in a study by Egeghy & Lorber, (2010), over a range expanding two orders of magnitude. A study by Egeghy & Lorber (2010) indicated that the true value of Vd was likely to remain within the lower range (around 200 mL kg bw^{-1}), and calibrations by Thompson *et al.* (2010) revealed the same relationship, and derived a Vd value of 230 mL kg bw^{-1} and 170 mL kg bw^{-1} for PFOS and PFOA, which have been used within this study.

Equation 9

$$DP = CP \times kP \times Vd$$

Equation 10

$$d(CP) / (dt) = DP(t) / ((Vd - kP)) \times CP(t)$$

8.8.PHARMACOKINETIC RESULTS

Results of the pharmacokinetic model are detailed in Table 75, Table 76, and Table 77. They indicate that the concentrations for adults and children vary considerably, depending upon the pathway and microenvironment involved. The impact of non-dietary sources on the blood concentrations indicate that a high proportion of the concentration could be accounted for via the sources used in this study.

The mean blood serum concentration was determined from the studies tabulated in chapter 1. These were from studies of the general population from regions within North America, Asia and Australasia over the past 10 years. The mean, median and ranges of the blood samples are detailed in Table 77, and they indicate that adult blood concentrations calculated to occur from the non-dietary pathways remain within the measured blood ranges for all three scenarios of exposure (low, typical and high). The child blood concentrations modeled also remain within the blood sample concentrations for all scenarios apart from “high” exposure of PFOS.

Table 75 Predicted contributions of non-dietary exposure to blood serum concentrations in adults (ng mL^{-1})

Microenvironment	Scenario	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
Homes	low	0.08	0.02	0.09	<0.01	0.06	0.05	0.43	0.32
	typical	0.43	0.29	0.69	<0.01	0.27	0.18	2	1.5
	high	9.4	2.7	11	0.85	4.5	5.4	20	18
Offices	low	0.04	0.01	0.02	<0.01	0.01	0.01	0.06	0.11
	typical	0.41	0.14	0.22	<0.01	0.04	0.03	0.42	0.3
	high	4.2	2	12	0.42	2.8	0.7	3.3	3.7
Cars	low	0.01	<0.01	0.02	<0.01	<0.01	<0.01	<0.01	<0.01
	typical	0.04	0.01	0.07	<0.01	0.01	0.01	0.03	0.02
	high	0.94	0.27	1.2	0.03	0.38	0.35	0.51	0.56
Outdoors	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
	typical	0.13	0.02	0.11	<0.01	<0.01	0.01	0.07	0.14
	high	0.59	0.09	1.9	<0.01	0.07	0.15	1.1	1.3
	low	**0.12**	**0.03**				**1.3**		
All Environments	**typical**	**1**	**0.47**				**7.1**		
	high	**15**	**5**				**110**		

Table 76 Predicted contributions of non-dietary exposure to blood serum concentrations in children (ng mL^{-1})

Microenvironment	Scenario	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
Homes	low	0.62	0.18	0.84	<0.01	0.13	0.12	0.97	0.63
	typical	3.7	2.6	5.8	<0.01	1.4	0.78	6.4	4.1
	high	130	29	150	9.4	43	32	95	130
Classrooms	low	0.83	0.08	0.18	<0.01	0.05	0.04	0.2	0.7
	typical	5.4	0.78	4.5	<0.01	0.2	0.22	2.9	5.7
	high	65	8.7	210	<0.01	7.4	16	120	150
Cars	low	0.04	<0.01	0.07	<0.01	<0.01	<0.01	<0.01	<0.01
	typical	0.15	0.05	0.27	<0.01	0.06	0.02	0.12	0.08
	high	4	1.1	5.1	0.13	1.6	1.5	2.2	2.4
Outdoors	low	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
	typical	0.12	0.02	0.1	<0.01	<0.01	<0.01	0.06	0.12
	high	0.58	0.08	1.8	<0.01	0.07	0.15	1.1	1.3
All Environments	**low**	**1.5**	**0.26**				**5.4**		
	typical	**9.3**	**3.4**				**42**		
	high	**200**	**39**				**1200**		

Table 77 Exposure scenario pharmacokinetic results (ng mL^{-1})

Scenario	PFOS	PFOA	PFOS (derived from PFOS & precursors)
Adult			
Low	0.12	0.03	1.3
Typical	1.0	0.47	7.1
High	15	5.0	110
Child			
Low	1.5	0.26	5.4
Typical	9.3	3.4	42
High	200	39	1200
Blood Samples*			
Mean Serum	21	8.7	
Median	19	4.7	
Range	0.96 – 53	0.53 - 62	

*values derived from following studies: Kärrman *et al.,* 2006, Kubwabo *et al.,* 2004, Olsen *et al.,* 1999, Yeung *et al.,* 2006, Calafat *et al.,* 2006, Olsen *et al.,* 2006, Guruge *et al.,* 2005, Olsen *et al.,* 2007, Olsen *et al.,* 2004, Masunaga *et al.,* 2002, Hansen *et al.,* 2001, Toms *et al.,* 2009, Von Ehrenstein *et al.,* 2009.

The PK model indicates that, under typical scenarios, the contribution of PFOS and PFOA from non-dietary sources to the mean blood concentration is around 5% for both compounds. Low end scenario concentrations for both adults and child PFOS concentrations indicate a contribution of around 5 – 10 %, while the PFOA modeled concentrations contribute around 1.5 – 4.5 %.

The mean blood sample concentrations used for comparison were also put into the PK model to determine the exposure rate required to produce such a concentration. The exposure required to induce 21 ng mL^{-1} and 8.7 ng mL^{-1} of PFOS and PFOA, is 1.45 ng $(kg\ bw)^{-1}\ d^{-1}$ and 1.18 ng $(kg\ bw)^{-1}\ d^{-1}$, respectively. This is in close agreement with the lower end modeled intake (of 1.6 ng $(kg\ bw)^{-1}\ d^{-1}$) by Egeghy and Lorber, (2010), and within the range of the estimated intake defined for PFOS by Thompson *et al.*, (2010) derived from concentrations in two American blood sample data sets, which were 1.1 – 2.1 ng $(kg\ bw)^{-1}\ d^{-1}$

and 1.6 – 3.8 ng (kg bw)$^{-1}$ d^{-1}, for PFOS and PFOA (respectively). The variability within the PK results from different studies is expected because of the simplistic nature of the model, and the large uncertainty surrounding many of the parameters involving intake and uptake of PFCs for forward and backward model calculations.

PFOS and PFOA were calculated in the PK model because of their persistence and movement into the blood (Han *et al.*, 2003), whereas precursor compounds are open to metabolisation once taken up by the body. In a recent study, Egeghy & Lorber, (2010) assumed the precursor compounds PFOSAs and PFOSEs undergo 100 % conversion to PFOS of the compounds, so that a conservative estimate of their contribution to body burdens of PFOS and PFOA may be made. The results indicate that there would be a large impact on the blood concentrations, with the majority of the blood sample concentration being derived from these non-dietary sources of precursor (to PFOS) compounds under the typical exposure scenario. This also suggests that the main presence of PFOS in blood is could be formed from the precursor compounds, and not direct intake of PFOS. Egeghy and Lorber, (2010) indicated in general there to be a more equal relationship between the concentration of PFOS derived from the main compound and the precursor input.

The PK model was applied to each type of environment and pathway of exposure for both adults and children and indicates differences in the contribution from various environments, variability between the pathway contributions and a difference between dominant contributing factors to adult and child exposure (Figure 47 - Figure 50).

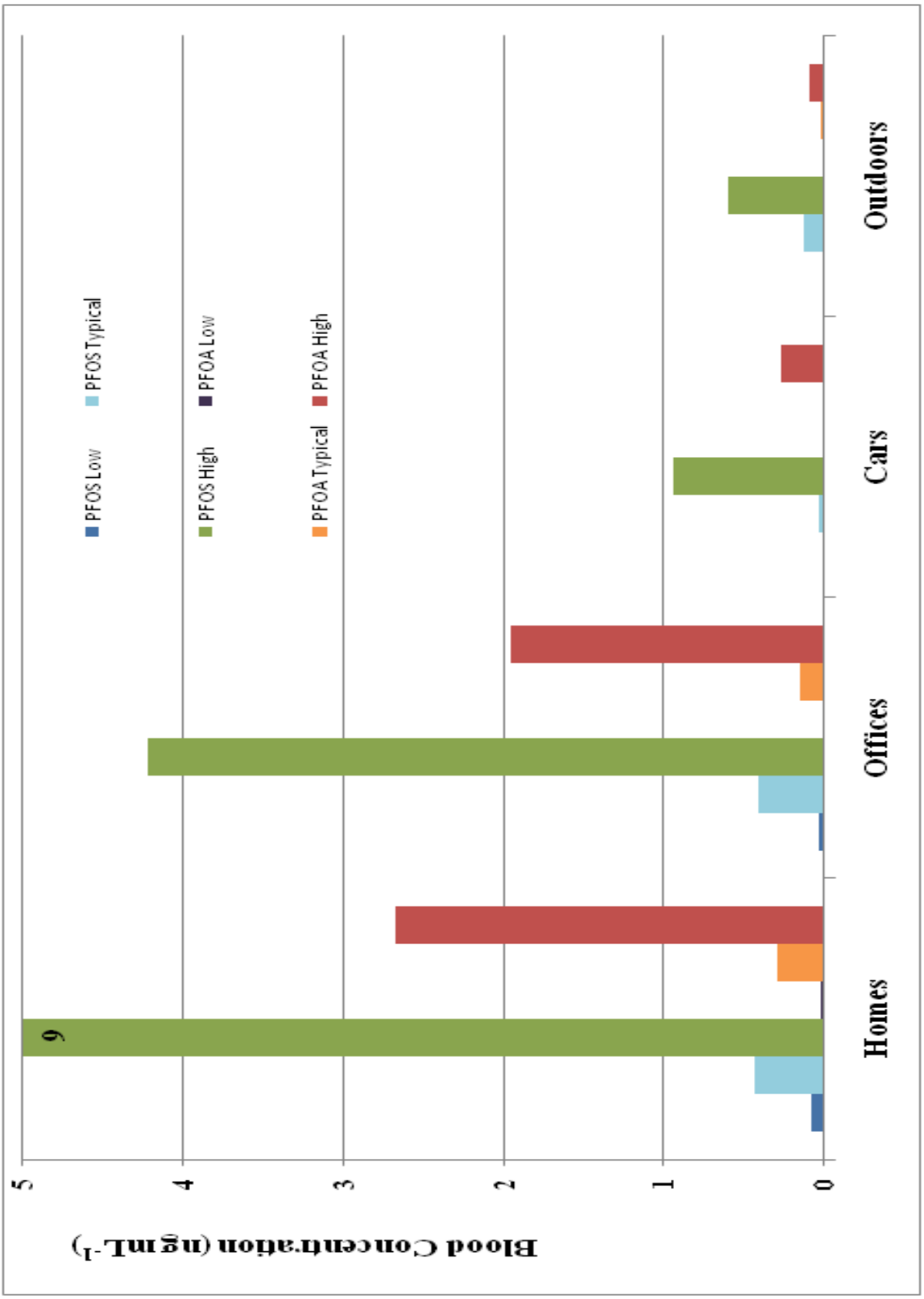


Figure 47 Adult contributions to blood concentrations (ng mL^{-1}) from independent microenvironments.

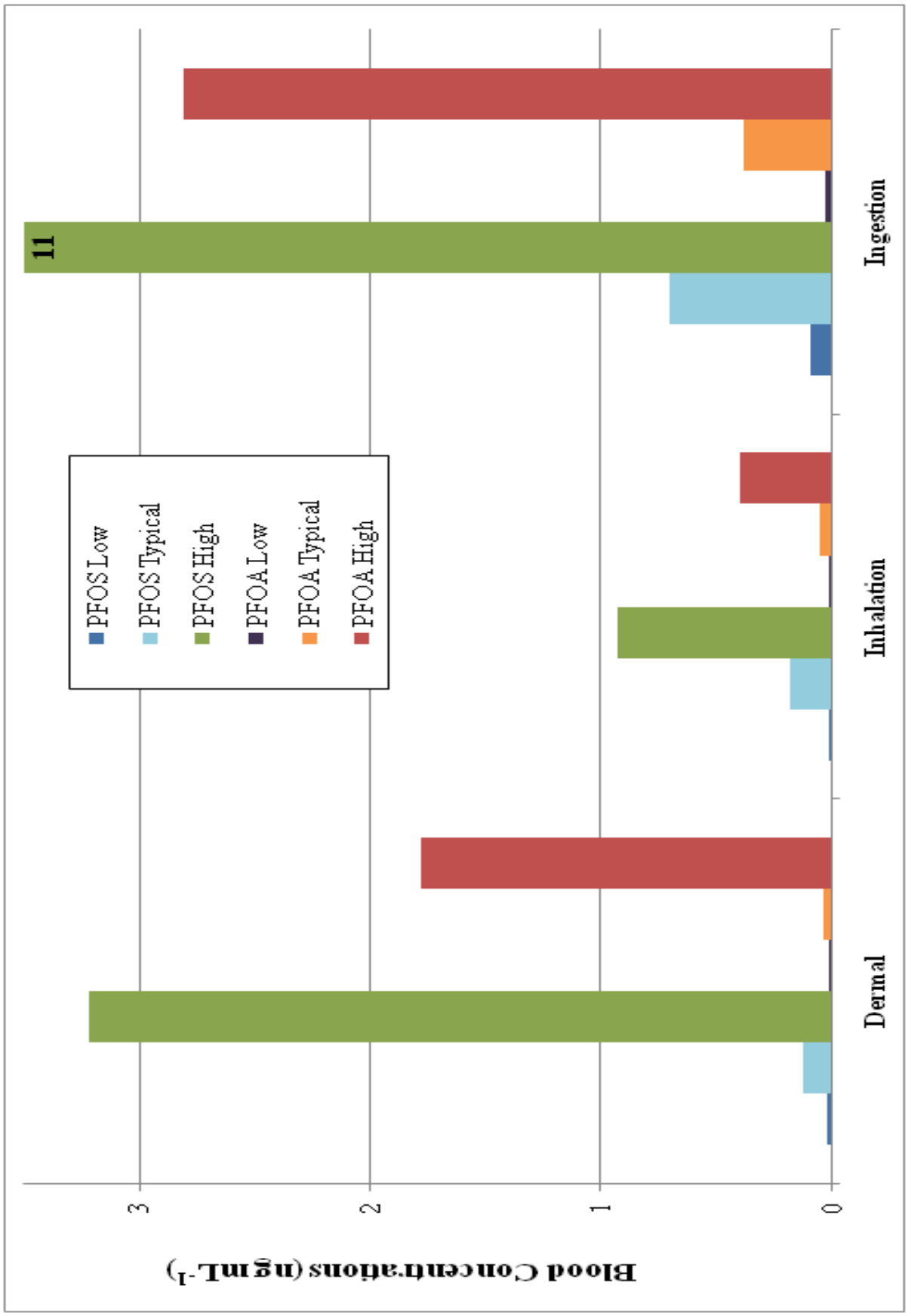


Figure 48 Adult blood concentrations from different uptake pathways (ng mL^{-1}).

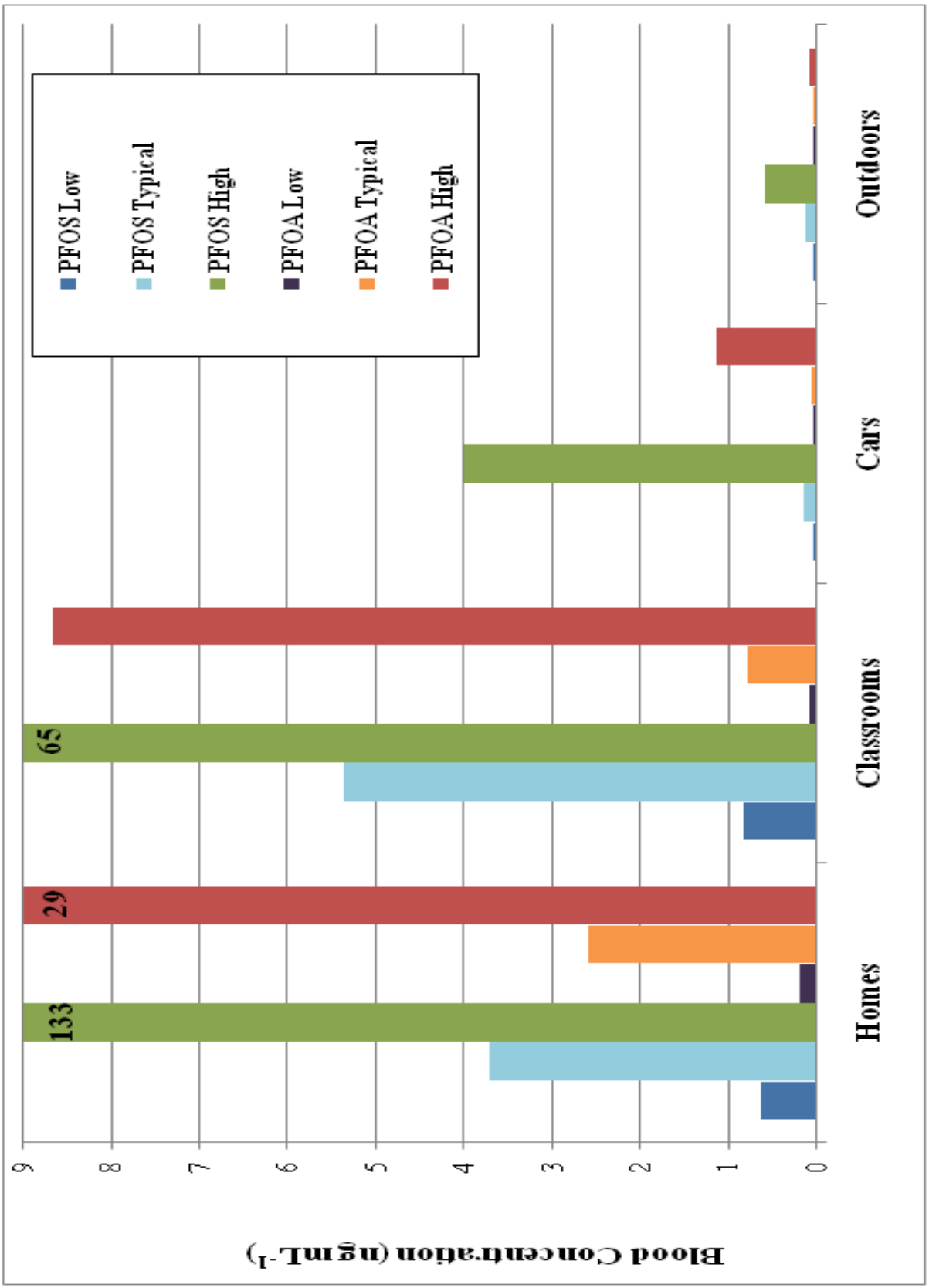


Figure 49 Child blood concentrations derived from microenvironment contributions (ng mL^{-1})

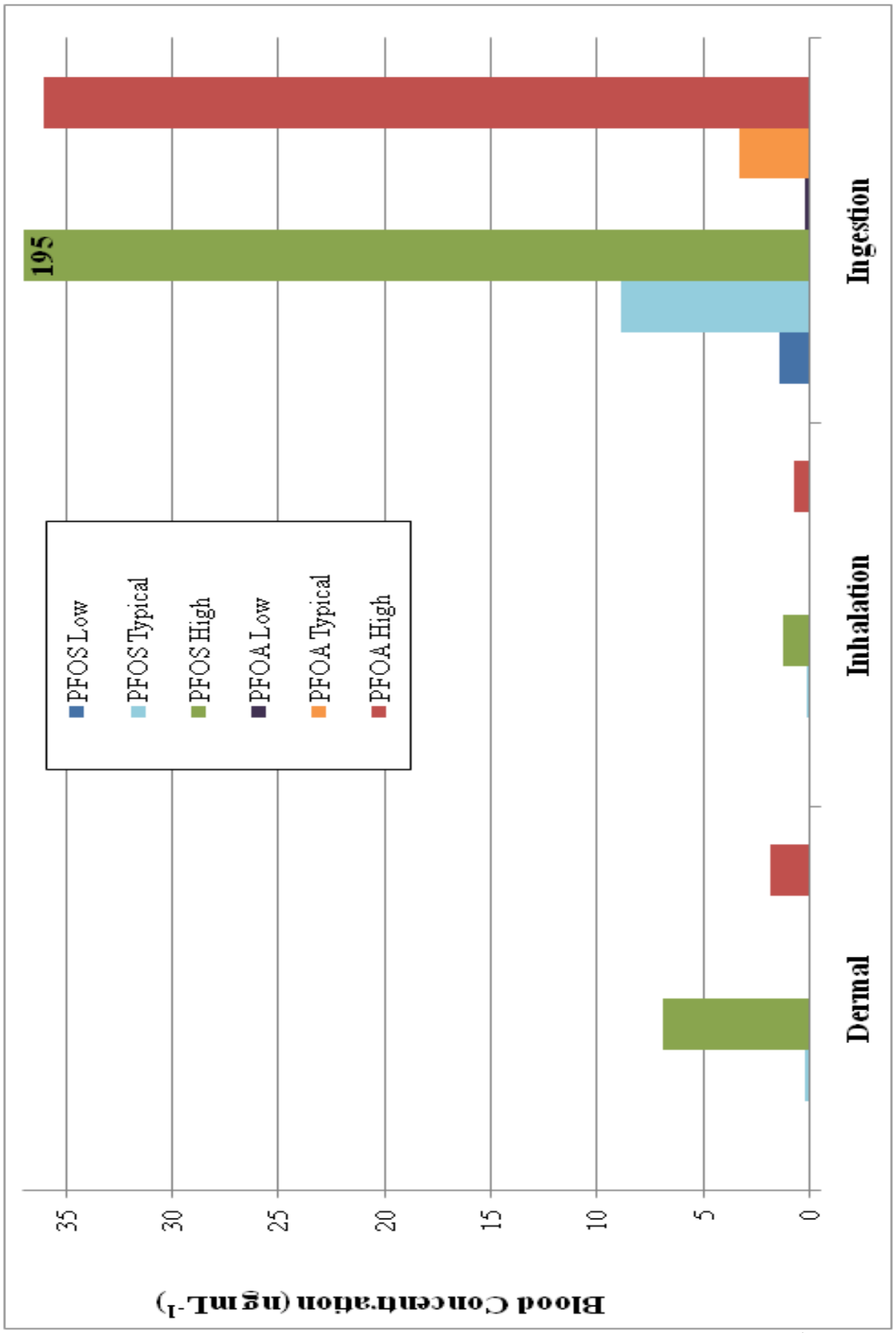


Figure 50 Child blood concentrations from different uptake pathways (ng mL^{-1}).

Figures 8 – 11 indicate that, at "high" exposure scenarios for children, the predicted serum concentrations arising from non-dietary exposure vastly exceed the measured ranges of PFOS and PFOA concentrations found in serum from around the world (Calafat *et al.,* 2007, Kärrman *et al.*, 2006). This suggests that the "high" exposure scenario may be an over representation of the feasible intake that is occurring in the environment. It is also possible that UK exposures may exceed those in the other countries for which serum concentrations are available.

8.9. COMPARISON OF EXPOSURE PATHWAYS TO SERUM CONCENTRATION

Contributions of non-dietary exposure towards human blood concentrations have been compared with the contributions from diet and drinking water. Concentrations from a German exposure study (Fromme *et al.,* 2009); have been used to assess the contribution of PFOS and PFOA from diet and drinking water to serum concentrations. These values are displayed in Table 78. The concentrations in serum amounting from adult mean diet and drinking water exposure are 21.7 ng mL^{-1} and 0.33 ng mL^{-1} for PFOS and 20.6 ng $mL^{-1}$and 0.16 ng mL^{-1} for PFOA.

Table 78 Diet and contributions to serum concentrations (ng mL^{-1})

Compound	Diet Exposure (ng $(kg\ bw)^{-1}d^{-1}$)	Drinking Water Exposure (ng $(kg\ bw)^{-1}d^{-1}$)	Serum Concentration from Diet (ng mL^{-1})	Serum Concentration from Drinking Water (ng mL^{-1})
PFOS	1.5 (4.5)	0.023 (0.13)	21.7 (65.2)	0.33 (1.9)
PFOA	2.8 (11.5)	0.022 (0.087)	20.6 (84.6)	0.16 (0.64)

The contribution of diet and water coupled with the modeled non-dietary serum concentrations results in predicted concentrations of PFOS and PFOA in adult blood serum of 23 and 21 ng mL^{-1} for mean intake and 99 and 90 ng mL^{-1} for high intake. The contributions

of dietary, drinking water and non-dietary sources to the concentrations in children's blood serum amount to 31 and 24 ng mL^{-1} for PFOS and PFOA mean intake and 280 and 120 ng mL^{-1} (respectively) for high intake. The mean predicted concentrations for adults fall within the range of those measured by Olsen *et al.*, 2004, Yeung *et al.*, 2006, Hansen *et al.*, 2001.

Figure 51 indicates the relative contributions of non-dietary, dietary and drinking water exposure to predicted serum concentration. For adults, Figure 51 A-D show that, regardless of the scenario, diet is the dominant exposure pathway. Under the "typical" exposure scenario, non-dietary sources contribute to < 5 % of predicted PFOS and PFOA serum concentrations, whilst drinking water contributes < 1.5 %. For children, the contribution of non-dietary sources to predicted serum concentrations is more significant contributing 30 % and 14 % for PFOS and PFOA respectively. This contribution rises considerably for children under the "high" exposure scenario, with non-dietary exposure contributing 75 % and 31 % of predicted serum concentrations of PFOS and PFOA. However, a high exposure diet could also contribute to the majority of serum concentrations, and in extreme cases exceed the present serum concentrations (according to data from the UK Food Safety Authority, COT 2006).

Interestingly, the predicted serum concentration of PFOS arising from the "high" exposure scenario exceeds that measured in occupationally exposed individuals (130 ng mL^{-1}). In contrast, predicted serum concentrations of PFOA (even under the "high" exposure scenario) do not approach the concentrations reported in the serum of occupationally exposed workers (range 420 – 1000 ng mL^{-1}) measured by Emmett *et al.*, (2006) and Ehresmen *et al.*, (2006). They do, however, exceed the concentration range reported for the general population (Calafat *et al.*, 2007, Kärrman *et al.*, 2006). Again, caution is advised as, currently, no data exist on concentrations of PFCs in blood serum of either occupationally or non-occupationally exposed UK adults or children.

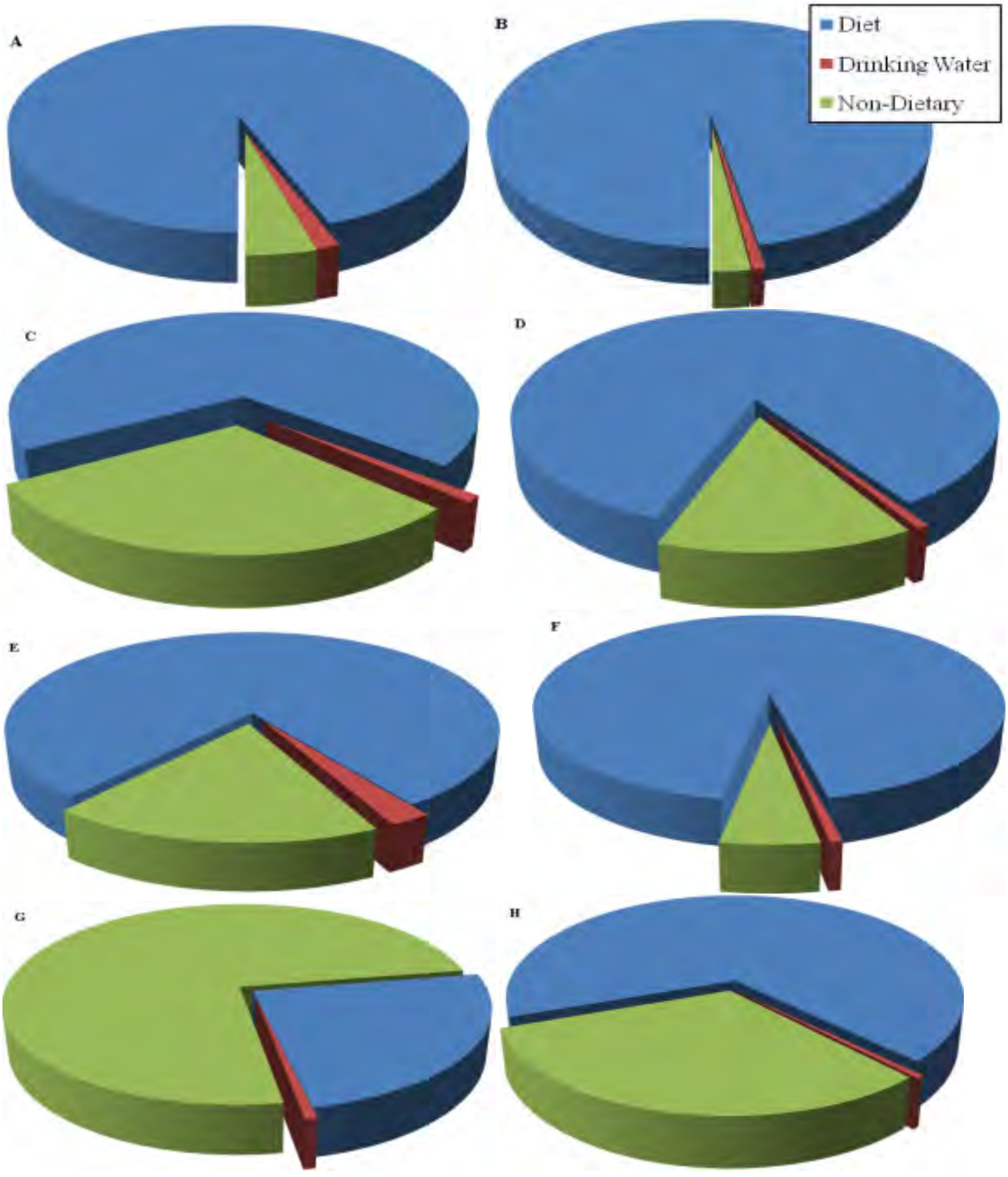


Figure 51 Contributions to predicted serum concentrations of exposure via dietary, drinking water and non-dietary sources. a, adult "mean" PFOS concentrations, b, adult "mean" PFOA concentrations, c, children "mean" PFOS concentrations, d, child "mean" PFOA concentrations, e, adult "high" PFOS concentrations, f, adult "high" PFOA concentrations, g, child "high" PFOS concentrations, h, child "high" PFOA concentrations.

8.10. CONCLUSIONS

The predicted blood serum concentrations of PFCs derived here are a simplified representation of the true values likely to be present in the UK population. Clearly, for individuals leading very different lifestyles to those assumed here, the predicted serum values reported above are likely to be inaccurate. This is particularly pertinent for individuals who may be exposed occupationally, such as metal plating factory workers, carpet and furniture salesman and firemen, etc. However, more sophisticated modeling also introduces a large amount of uncertainty because of a number of values which have not been established including variable uptake rates and excretion rates with rising concentrations. The steady state one compartmental model used here is appropriate for studying long term, low dose exposures (Egeghy & Lorber, 2010), for chemicals which have been present in the environment for a long period (chronic exposure).

A conclusion from the work reported in this chapter is that the exposure of children to PFOS and PFOA via non-dietary sources constitutes 10 – 30 % of the TDI under all the measured scenarios ("low", "typical" and "high"), and in some instances may be comparable to estimates of UK dietary exposure (Table 73). Moreover, it may exceed that in other countries (Fromme *et al.*, 2009). If the precursor compounds are included in the comparison (assuming 100 % metabolisation) the contribution to the PFOS TDI value is an additional 0.1 – 25 % and 0.1 – 3 %, for children and adults respectively, which is in agreement with a study by Vestegren *et al.,* (2008), suggesting that, for some people within the population, there will be a significant effect from the precursor compounds on the body burden of PFOS received from non-dietary sources.

Reassuringly, current data suggests that the exposure of UK children to PFOS and PFOA via both dietary and non-dietary pathways is well within the EFSA TDI values. A cautionary note, however, is that at the current time it is not known to what extent external exposure to

other PFCs ("precursor compounds") contributes via metabolism to body burdens of PFOS and PFOA and if the TDI were to drop in the future and better detection of food samples, the effect of precursor metabolisation may be causing a greater impact upon body burdens than presently recognised. This may be pertinent in view of the substantial exposure to such precursors reported here and in other studies. European studies reveal an average PFOS blood serum concentration in Germans of 11.5 ng mL^{-1} (Fromme *et al.,* 2009), which is higher than can be explained solely via non-dietary intake of PFOS. Yet applying a conservative estimate of 100 % biotransformation of these precursors to PFOS creates serum concentrations of 7 ng mL^{-1} and 42 ng mL^{-1}, for adults and children, which does not exceed maximum measured concentrations of PFOS in human blood serum (Calafat *et al.*, 2007 and Kärrman *et al.*, 2006).

The substantial contribution of non-dietary sources to PFOS is thought to derive from the degradation of precursors and, despite the restriction of production and use of PFOS within the European Union, it is thought that concentrations will remain at relatively high levels (compared to other PFCs) in dust, because of such precursor degradation. Our exposure estimates are consistent with previous findings for PFOS and PFOA that while diet is the main exposure pathway for most of the population; under high dust ingestion scenarios, the ingestion of dust can be an important exposure pathway, especially for young children (Björklund *et al.* 2009) and particularly for PFHxS.

Though at this present time, the exposure assessment indicates that non-dietary exposure modeled from the concentrations measured within this study, do not currently put children or adults at risk, according to the EFSA TDI values for PFOS and PFOA. Therefore, the presence of these compounds within homes, offices, cars, classrooms and outdoor environments are not at concentrations high enough to cause internal doses of PFOS and PFOA to exceed threshold values, (even under conservative assessments), above which toxic

effects occur. This remains true when coupled with dietary exposure. The two distinct pathways combined, do not exceed the TDI threshold, and therefore no associated risk is present for children or adults within the general UK population.

Further work that appears necessary would be to determine the relative contributions of non-dietary sources to male and females, as a number of studies have concluded that the concentrations in blood vary according to gender (Toms *et al.*, 2009). This is especially of interest for male children, due to gender-specific behavioural patterns at early infant stages (Pellegrini *et al.*, 2007).

9. COMPARISON OF DUST FROM INTERNATIONAL INDOOR MICROENVIRONMENTS

This chapter tests the hypothesis that there is substantial international variation in PFC contamination of indoor dust. Hence its aim is to evaluate concentrations of PFCs in indoor dust from homes in a number of countries.

As observed elsewhere for PBDEs and other flame retardants (Wilford *et al.* 2010; Harrad et al, 2008b), international variations in use of PFCs is likely. Such variations in use are hypothesised to be reflected in international differences in levels and patterns of PFC contamination of indoor dust, in line with those observed for concentrations in human blood serum (Wilhelm *et al.*, 2009). The chapter quantifies concentrations of PFCs in house dust from 8 different countries, and compares the levels and signatures, to determine whether significant differences exist.

9.1. SYNOPSIS

House dust samples from eight countries were taken from, specifically, the UK (n = 45), France (n = 9), Germany (n = 10), Australia (n = 20), USA (n = 10), Canada (n = 19), Kazakhstan (n = 9) and Thailand (n = 20), along with dust samples from office environments in the UK (n = 20) and France (n = 9), as well as dust from cars in the UK (n=20), Australia (n = 10) and Kazakhstan (n = 11). This chapter compares the data from different countries, and identifies differences and similarities between PFCs in Europe, N. America, Asia and Australia.

The presence of PFCs in indoor environments is becoming more apparent with multiple studies reporting concentrations within indoor environments (Shoeib *et al.*, 2005, Kato *et al.*, 2009, Kubwabo *et al.*, 2005, Moriwaki *et al.*, 2003), and confirming the inhalation and ingestion of indoor air and dust to be a potential pathway of human exposure (Trudel *et al.*, 2008).

Recent studies have quantified PFOS and PFOA concentrations in dust from the USA, Canada and Scandinavia (Kato *et al.*, 2009, Strynar & Lindstrom *et al.*, 2008, Kubwabo *et al.*, 2005, Björklund *et al.*, 2009). These studies indicate that the highest concentrations are in the USA, and differences have also been reported in concentrations of PFOS and PFOA in human milk from across Asia (Tao *et al.*, 2008). However, a systematic international comparison of indoor dust contamination has not yet been conducted for PFCs. This may be a significant gap in knowledge as such contamination is expected to vary with region according to uses and lifestyles (Trudel *et al.*, 2008). Applications have led to PFC concentrations in indoor air exceeding, by an order of magnitude, those measured in outdoor air (Shoeib *et al.*, 2005). Moreover, PFCs have been detected in a small but increasing number of surveys of indoor dust (Björklund *et al.*, 2009; Kubwabo *et al.*, 2005; Moriwaki *et al.*, 2003; Shoeib *et al.*, 2005; Strynar and Lindstrom, 2008). As a result, the indoor environment is a recognised vector of exposure to PFCs via inhalation and dust ingestion that is in addition to exposure via the diet and drinking water (Björklund *et al.*, 2009; Fromme *et al.*, 2009; Vestergren *et al.*, 2009).

PFOS and PFOA concentrations have been reported in dust from Swedish cars, child daycare centers', and offices, have been reported (Björklund *et al.*, 2009), as well as in houses and apartments. While offices and apartments had the highest median concentrations of PFOS and PFOA, major differences between microenvironment categories were not evident.

Key objectives are thus to: examine differences in PFC contamination of house dust from a number of different countries in order to facilitate preliminary assessment of international differences in PFC contamination of house dust in the context of source attribution.

9.2.INTERNATIONAL RESULTS

Dust samples were collected from the eight countries according to the sampling details given in chapter 2, and were collected via acquaintances of the research team in the Division of Environmental Health and Risk Management at Birmingham University. Descriptive statistics summarising the results for each country sampled are displayed in Table 79 for homes and Table 80 for cars and offices, and raw data can be found in Appendix B.

Table 79 Summary of concentrations of PFCs in house dust from 8 different countries (ng g^{-1}).

PFOS	UK	France	Germany	Australia	USA	Canada	Kazakhstan	Thailand
Minimum	3.5	54	47	6.5	110	42	<DL	3
5th Percentile	23	88	54	16	120	46	6.6	4
Median	140	160	170	170	310	140	28	16
Geo-Mean	150	190	190	190	320	160	25	20
Arth-Mean	450	330	310	1000	420	290	40	32
95th Percentile	1300	1100	1020	6900	890	1100	78	97
Maximum	7400	1700	1200	8100	930	1300	81	130
SD	1100	510	380	2200	310	350	28	33
%RSD	250	150	120	220	74	120	72	100
PFOA	**UK**	**France**	**Germany**	**Australia**	**USA**	**Canada**	**Kazakhstan**	**Thailand**
Mimimum	<DL	15	19	15	27	<DL	<DL	<DL
5th Percentile	13	16	30	19	33	<DL	<DL	1
Median	190	31	290	120	240	69	<DL	18
Geo-Mean	120	36	180	150	190	34	2.2	14
Arth-Mean	310	52	290	530	360	270	34	45
95th Percentile	510	150	640	2800	1200	460	170	150
Maximum	4100	220	720	2900	1800	4000	260	290
SD	620	63	240	860	510	870	87	69
%RSD	200	120	80	160	140	330	260	150
PFHxS	**UK**	**France**	**Germany**	**Australia**	**USA**	**Canada**	**Kazakhstan**	**Thailand**
Minimum	7.6	54	16	19	86	<DL	57	3
5th Percentile	32	56	28	58	89	<DL	69	3
Median	210	77	150	180	240	120	200	16
Geo-Mean	200	110	160	160	230	35	150	16
Arth-Mean	450	130	290	240	270	150	180	25
95th Percentile	1400	270	760	500	480	350	340	76
Maximum	6100	320	790	1100	560	850	410	84
SD	970	90	280	240	150	190	110	24
%RSD	220	70	97	99	55	130	60	96

MeFOSA	UK	France	Germany	Australia	USA	Canada	Kazakhstan	Thailand
Minimum	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
5th Percentile	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Median	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
Geo-Mean	<DL	<DL	<DL	1.8	<DL	<DL	0.8	<DL
Arth-Mean	13	5.4	1.7	360	15	39	67	1.6
95th Percentile	86	25	8.9	1700	80	170	250	9.2
Maximum	110	31	16	3000	130	470	250	13
SD	28	11	5.1	770	40	110	110	3.6
%RSD	210	210	310	210	270	290	160	220
EtFOSA	UK	France	Germany	Australia	USA	Canada	Kazakhstan	Thailand
Minimum	<DL	23	36	<DL	41	<DL	99	<DL
5th Percentile	<DL	39	44	<DL	43	<DL	100	<DL
Median	40	130	120	930	99	550	200	26
Geo-Mean	23	120	130	430	100	180	210	22
Arth-Mean	98	150	190	2000	140	1300	230	140
95th Percentile	380	300	510	6800	310	5000	400	710
Maximum	840	320	730	8600	380	7900	440	940
SD	160	100	200	2500	110	2000	110	250
%RSD	160	67	110	120	80	150	49	180
FOSA	UK	France	Germany	Australia	USA	Canada	Kazakhstan	Thailand
Minimum	<DL	<DL	<DL	<DL	11	<DL	<DL	<DL
5th Percentile	<DL	<DL	2.1	<DL	17	<DL	<DL	1.9
Median	20	3	47	1.2	69	7.8	<DL	8.5
Geo-Mean	5.3	1.1	20	<DL	55	1.1	<DL	6.6
Arth-Mean	54	3.4	56	25	67	190	<DL	13
95th Percentile	230	7	120	140	110	500	<DL	32
Maximum	300	7	130	140	110	2700	<DL	41
SD	80	2.6	42	48	35	610		12
%RSD	150	75	75	190	53	320		88

MeFOSE	UK	France	Germany	Australia	USA	Canada	Kazakhstan	Thailand
Minimum	<DL	<DL	2.9	<DL	58	<DL	<DL	<DL
5th Percentile	<DL	<DL	6.6	3.1	59	<DL	1.8	<DL
Median	93	13	75	38	88	46	10	1.5
Geo-Mean	40	3.6	52	33	100	20	7	1.3
Arth-Mean	240	19	130	84	120	120	11	4.8
95th Percentile	590	57	480	320	270	380	27	18
Maximum	2500	61	700	400	310	470	30	23
SD	460	22	210	110	83	150	9.7	6.6
%RSD	200	120	150	130	69	130	90	140
EtFOSE	UK	France	Germany	Australia	USA	Canada	Kazakhstan	Thailand
Minimum	<DL	<DL	12	<DL	44	<DL	13	<DL
5th Percentile	<DL	<DL	19	3.8	76	<DL	13	<DL
Median	34	13	120	20	140	52	38	1.5
Geo-Mean	41	1.9	79	19	160	11	41	3.3
Arth-Mean	320	19	100	60	210	170	61	9.7
95th Percentile	1100	53	170	160	540	710	160	32
Maximum	3900	55	180	440	700	1700	190	34
SD	680	22	5400	100	190	390	61	12
%RSD	210	120	53	160	89	230	99	120

Table 80 Summary of concentrations of PFCs in office and car dust from different countries (ng g^{-1}).

PFOS	Offices		Cars		
	UK	France	UK	Australia	Kazakhstan
Minimum	20	33	20	21	30
5th Percentile	26	45	24	30	38
Median	390	180	98	59	130
Geo-Mean	210	140	130	61	130
Arth-Mean	420	180	260	68	180
95th Percentile	1100	290	630	120	440
Maximum	1100	290	1500	130	510
SD	390	92	340	32	150
%RSD	93	53	130	48	83
PFOA	**Offices**		**Cars**		
	UK	**France**	**UK**	**Australia**	**Kazakhstan**
Minimum	<DL	9.0	<DL	<DL	17
5th Percentile	18	9.3	5.7	<DL	23
Median	280	18	65	46	81
Geo-Mean	160	23	45	17	97
Arth-Mean	550	45	110	58	180
95th Percentile	930	160	350	150	510
Maximum	6000	220	370	220	610
SD	1300	72	120	62	200
%RSD	240	160	120	110	110
PFHxS	**Offices**		**Cars**		
	UK	**France**	**UK**	**Australia**	**Kazakhstan**
Minimum	6.0	16	45	30	55
5th Percentile	20	36	46	35	75
Median	170	170	180	140	230
Geo-Mean	180	120	190	110	220
Arth-Mean	610	150	330	130	320
95th Percentile	2900	230	800	200	880
Maximum	5700	250	2400	210	1100
SD	1300	75	510	56	310
%RSD	220	51	160	43	99
MeFOSA	**Offices**		**Cars**		
	UK	**France**	**UK**	**Australia**	**Kazakhstan**
Minimum	<DL	<DL	<DL	<DL	<DL
5th Percentile	<DL	<DL	<DL	<DL	<DL
Median	<DL	<DL	<DL	<DL	23
Geo-Mean	<DL	<DL	<DL	<DL	2.9
Arth-Mean	61	52	8.6	<DL	130
95th Percentile	97	260	20	<DL	450
Maximum	1000	390	130	<DL	550
SD	220	140	30		190
%RSD	360	260	350		150

EtFOSA	Offices		Cars		
	UK	France	UK	Australia	Kazakhstan
Minimum	<DL	40	<DL	<DL	69
5th Percentile	<DL	40	<DL	34	79
Median	15	65	40	92	360
Geo-Mean	9.5	70	28	61	410
Arth-Mean	120	80	93	150	730
95th Percentile	690	150	260	410	2300
Maximum	840	180	370	570	2600
SD	230	47	100	150	840
%RSD	200	59	110	100	120
FOSA	**Offices**		**Cars**		
	UK	**France**	**UK**	**Australia**	**Kazakhstan**
Minimum	<DL	<DL	<DL	<DL	<DL
5th Percentile	<DL	1.4	<DL	<DL	<DL
Median	2.1	5.0	15	<DL	2.0
Geo-Mean	<DL	2.7	4.1	<DL	1.2
Arth-Mean	21	5.9	140	<DL	48
95th Percentile	130	12	240	<DL	180
Maximum	130	13	1900	<DL	200
SD	38	3.9	420		72
%RSD	180	66	300		150
MeFOSE	**Offices**		**Cars**		
	UK	**France**	**UK**	**Australia**	**Kazakhstan**
Minimum	<DL	<DL	<DL	2.0	<DL
5th Percentile	1.5	<DL	<DL	3.5	<DL
Median	220	40	82	6.0	14
Geo-Mean	81	13	37	7.2	5.1
Arth-Mean	250	65	130	12	49
95th Percentile	570	210	340	41	190
Maximum	920	290	490	73	290
SD	240	93	140	20	90
%RSD	96	140	110	170	180
EtFOSE	**Offices**		**Cars**		
	UK	**France**	**UK**	**Australia**	**Kazakhstan**
Minimum	<DL	<DL	<DL	<DL	1.1
5th Percentile	<DL	<DL	<DL	<DL	2.0
Median	88	<DL	55	8.0	38
Geo-Mean	31	<DL	32	1.6	28
Arth-Mean	290	15	100	8.3	110
95th Percentile	730	70	380	21	430
Maximum	2600	95	460	24	590
SD	590	33	120	8.7	180
%RSD	200	220	120	110	170

9.3. CONCENTRATION COMPARISONS

Table 79 and Table 80 report the average, median, and range of concentrations of PFCs in house, office and car dust from each country, as well as in UK office, car, and classroom dust. Table 1 indicates differences between concentrations of PFCs in house dust from different countries. ANOVA analysis of log-transformed data reveals a number of statistically significant ($p < 0.05$) differences, displayed in Table 81. The most salient features are that: (a) most PFCs are significantly lower in Kazakhstani and Thai dust than in other countries, (b) both MeFOSE and EtFOSE are significantly lower in Canadian dust than in UK and US dust, (c) in Canadian dust, PFHxS is significantly lower than in UK house dust, and (d) EtFOSA in Australian dust is significantly higher than in UK house dust.

The significantly lower concentrations of most PFCs in Kazakhstani and Thai dust suggest PFC use in these countries is lower than in Australia, Europe, and North America. More intriguing are the lower levels of some PFCs in Canadian dust compared to UK and US house dust, indicating that the FOSEs and PFHxS are not used widely in Canada. Also pertinent are the significantly higher concentrations of EtFOSA in Australian house dust compared to UK house dust. While EtFOSA was deregistered as an insecticide (as Sulfluramid for ant control) in Australia in January 2004, it was licensed for use for about 2 years before this. By comparison, EtFOSA appears never to have been registered for such use in the UK (Risk & Policy Analysts, 2004). It is therefore possible that the higher levels in Australian samples reflect past or recent use of EtFOSA as an insecticide. More puzzlingly, EtFOSA concentrations are elevated (but not significantly compared to other countries studied) in Canadian samples, as EtFOSA has never been registered for insecticide use in Canada. Moreover, the fact that EtFOSA concentrations in US samples are in line with all other countries except Australia and Canada, is at odds with the fact that, while use of Sulfluramid

is being phased out in the US, it has been licensed for such use in the recent past. Overall, the data suggests further study is warranted of the impact of Sulfluramid on EtFOSA contamination of indoor dust and other matrices relevant to human exposure. The 2006 OECD report on global PFC usage states that 17 t of EtFOSA was used as an ingredient in insecticide products at a concentration of 0.01 – 1 % (OECD, 2006). Such products were for industrial uses in highways; railroads, pipelines and high-voltage lines, as well as consumer uses like granulated bait for amateur gardening. In addition, > 1 t of the –N-[3-(trimethoxysilyl)propyl derivative of EtFOSA was reported to be used as an additive in toner or printing inks. The OECD reports provides no information on which countries have used EtFOSA or its –N-(3-trimethoxysilyl)propyl derivative.

Table 81 Significant differences between countries and contributions of PFCs

PFC	Significant Differences[a]
PFOS	UK, Australia, Canada, France, Germany & US > Kazakhstan
	UK, Australia, Canada & US > Thailand
PFOA	UK, Australia, Canada, Germany & US > Kazakhstan
	UK & Australia > Thailand
PFHxS	UK, Australia, Canada, Germany & US > Thailand
	UK > Canada
MeFOSA	No significant differences
EtFOSA	Australia > UK
FOSA	UK, Germany, Thailand & US > Kazakhstan
MeFOSE	UK & US > Canada & Thailand
EtFOSE	UK & US > Kazakhstan and Thailand
	Germany > Kazakhstan

[a]Significant at $p < 0.05$. Only significant differences shown.

Table 82 includes concentrations reported in previous surveys of indoor dust. Broadly, concentrations in this study are within the range of those reported previously (Björklund *et al.*, 2009; Kato *et al.*, 2009; Kubwabo *et al.*, 2005; Moriwaki *et al.*, 2003; Shoeib *et al.*, 2005;

Strynar and Lindstrom, 2008). Most previous studies have focused on PFOS, PFOA, and PFHxS. However, while EtFOSA was detected at a median of 550 ng g^{-1} in house dust from Toronto; it was not detected in any such samples collected in 2002-03 in Ottawa (n = 67) (Shoeib *et al*, 2005). There is no definitive explanation for this difference between the two studies, although EtFOSA was detected in over 90 % of indoor air samples in Ottawa at an average concentration of pg m^{-3} (Shoeib *et al.*, 2005). Moreover, the only other study of EtFOSA in indoor dust of presently available, reports concentrations that are consistent with this UK data (Kato *et al*., 2009). The Ottawa study (Shoeib *et al.*, 2005) appears to be the only other data available on MeFOSE and EtFOSE in indoor dust. Here, Toronto data from this study reports median concentrations about an order of magnitude below those in Ottawa. As with EtFOSA, there is no definitive explanation for these differences between the two studies, and these apparent discrepancies may simply be attributable to small sample numbers, with larger surveys revealing different patterns of contamination. A possible alternative explanation is a temporal change in PFC use patterns.

Table 82 International dust concentrations from a variety of studies

Location, microenvironment category (reference)	Statistical parameter/ PFC	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
Ottawa, Canada, homes (Shoeib *et al.*, PFOS, PFOA, and PFHxS)	Mean	444	106	392	14	<DL	-	412	2200
	Median[a]	38	20	23	7.9	<DL	-	113	138
	Minimum	2.3	1.2	2.3	0.7	<DL	-	3.3	1.4
	Maximum	5065	1234	4305	44	<DL	-	8860	75 440
Japan, homes (Moriwaki *et al.*, 2003) (n=16)	Mean	200	380	-	-	-	-	-	-
	Median	25	165	-	-	-	-	-	-
	Minimum	11	69	-	-	-	-	-	-
	Maximum	2500	3700	-	-	-	-	-	-
Stockholm, Sweden, homes (Björklund *et al.*, 2009) (n=10 houses, n=38 apartments)	Mean	49 (175)[b]	55 (142)	-	-	-	-	-	-
	Median	39 (19)	54 (93)	-	-	-	-	-	-
	Minimum	15 (8)	15 (18)	-	-	-	-	-	-
	Maximum	120	98 (850)	-	-	-	-	-	-
Ohio and North Carolina, US, homes and child daycare centers (Strynar & Lindstrom, 2008) (*n* = 10 child daycare centers, *n* = 102 homes)	Mean	761	296	874	-	-	-	-	-
	Median	201	142	46	-	-	-	-	-
	Minimum	<8.9	<10	<13	-	-	-	-	-
	Maximum	12 100	1960	35 700	-	-	-	-	-
Australia (n = 10), Germany (n = 10), UK (n = 9), Atlanta, GA, US, homes (Kato *et al.*, 2009) (n = 10)[c]	Mean	nr	nr	nr	nr	nr	nr	-	-
	Median	480	97	190	<2.6	200	<2.6	-	-
	Minimum	<2.6	<2.6	<2.6	<2.6	<2.6	<2.6	-	-
	Maximum	18 000	9800	44 000	220	4000	180	-	-

[a]geometric mean for MeFOSE, EtFOSE, FOSA, MeFOSA, and ETFOSA
[b]values in parentheses are for apartments
[c]concentrations not reported separately for individual countries
nr = not reported

9.4.POTENTIAL SOURCES OF PFCS IN DUST

Previous studies have examined their data for correlations between concentrations of individual PFCs on the premise that significant positive correlations are indicative of the existence of a common source or sources of the PFCs in question. Specifically, PFOS was found to correlate significantly with PFOA in dust from the US (Strynar & Lindstrom, 2008), from Australia, Germany, UK, and the US (Kato *et al.*, 2009), Canada (Kubwabo *et al.*, 2005), Japan (Moriwaki *et al.*, 2003), and Sweden (Björklund *et al.*, 2009). PFHxS has also been reported to be significantly correlated with PFOA (Kubwabo *et al.*, 2005, Kato *et al.*, 2009), and PFOS (Kubwabo *et al.*, 2005).

The data was examined for the presence of such correlations for house dust from Australia, Canada, Thailand, and the UK, as well as UK cars, classrooms, and offices as the sample numbers in these datasets ($n \geq 20$) were deemed sufficient for any correlations to have statistical significance. Correlation analysis was conducted on log-transformed data. Pearson correlation coefficients were obtained for each dataset. Presumably, owing to the low concentrations detected, no significant correlations were detected in Thai samples. In Australian and UK house dust, a significant correlation was detected between PFOS and PFOA. Interestingly, this correlation was not detected for UK cars, classrooms, or offices. This is consistent with a Swedish study that reported a significant correlation between PFOS and PFOA in dust from houses, apartments, and offices, but not for child daycare centers' (Björklund *et al.*, 2009). Other significant positive correlations observed for some (see Table 81), but not all, datasets are for: PFHxS with PFOS and PFOA; PFOS with MeFOSA; EtFOSA with PFOS, PFOA, and PFHxS; FOSA with PFOS and PFOA; MeFOSE with PFOA, EtFOSA, and FOSA; and EtFOSE with PFOS, PFOA, PFHxS, and MeFOSE. Where such correlations exist, this suggests the presence of common sources of the PFCs involved

(as is potentially the case for PFOS, PFOA, and PFHxS – noted is the use of PFHxS as a PFOS "substitute" (POPRC 2010)) or that one of the correlated PFCs may be a precursor compound of the other (which may be so where the FOSEs and FOSAs may degrade to PFOS (D'Eon *et al.*, 2006) or PFOA (Martin *et al.*, 2006), respectively.

Table 83 Correlations of PFCs in dust from various countries

Compound A	Compound B	Country
PFHxS	PFOS,	Kazakhstan, UK, Australia,
PFHxS	PFOA	UK, Germany, Canada,
PFHxS	EtFOSA	Kazakhstan, Canada,
PFOS	MeFOSA,	Australia, Canada
PFOS	PFOA	UK, Australia,
EtFOSA	PFOA,	UK, USA, Canada,
FOSA	PFOA	Canada, Australia,
EtFOSE	PFOA	UK, Germany,

As a complementary approach, the data was examined for differences in PFC contamination patterns via PCA. The data examined was from different UK microenvironments and for homes from different countries as two distinct datasets. No distinct differences in contamination patterns were discernible between dusts from different UK microenvironment categories. Differences were distinguishable between dusts from homes from different countries however. The first two principal components (PCs) accounted for 21 % and 17 % respectively of the total variance within the dataset. Both PCs were driven in negative directions by of the presence of EtFOSA in large concentrations for many of the samples. In contrast, highly positive values of PC1 resulted from elevated proportions of MeFOSE and EtFOSE, while PC2 was driven in a positive direction by greater abundance of PFHxS. Figure 1 plots the PC1 and PC2 scores obtained for each house dust sample. Most striking is that the majority of Australian and Canadian house dust samples occupy the bottom left quadrant of component space with strongly negative scores for both PCs. This reflects the high relative abundance of EtFOSA in these samples, and supports the hypothesis that there

is a distinct source of this PFC in most of the dust samples from these countries. By comparison, European and US samples are situated predominantly in the upper two quadrants due to their high proportions of PFHxS, MeFOSE, and EtFOSE. Samples from Kazakhstan predominantly occupy space in the top left quadrant as a consequence of relatively low abundances of FOSEs, alongside strong contributions from PFHxS and EtFOSA. The contamination pattern in Thai dusts is in some cases similar to Australian and Canadian samples, due to high relative abundances of EtFOSA, with others resembling the pattern in European dusts. Overall, this study suggests that there are significant differences in PFC use patterns across the world, even between contiguous territories like Canada and the US, and that extrapolation of exposure assessments for one country or region to others should be conducted with caution.

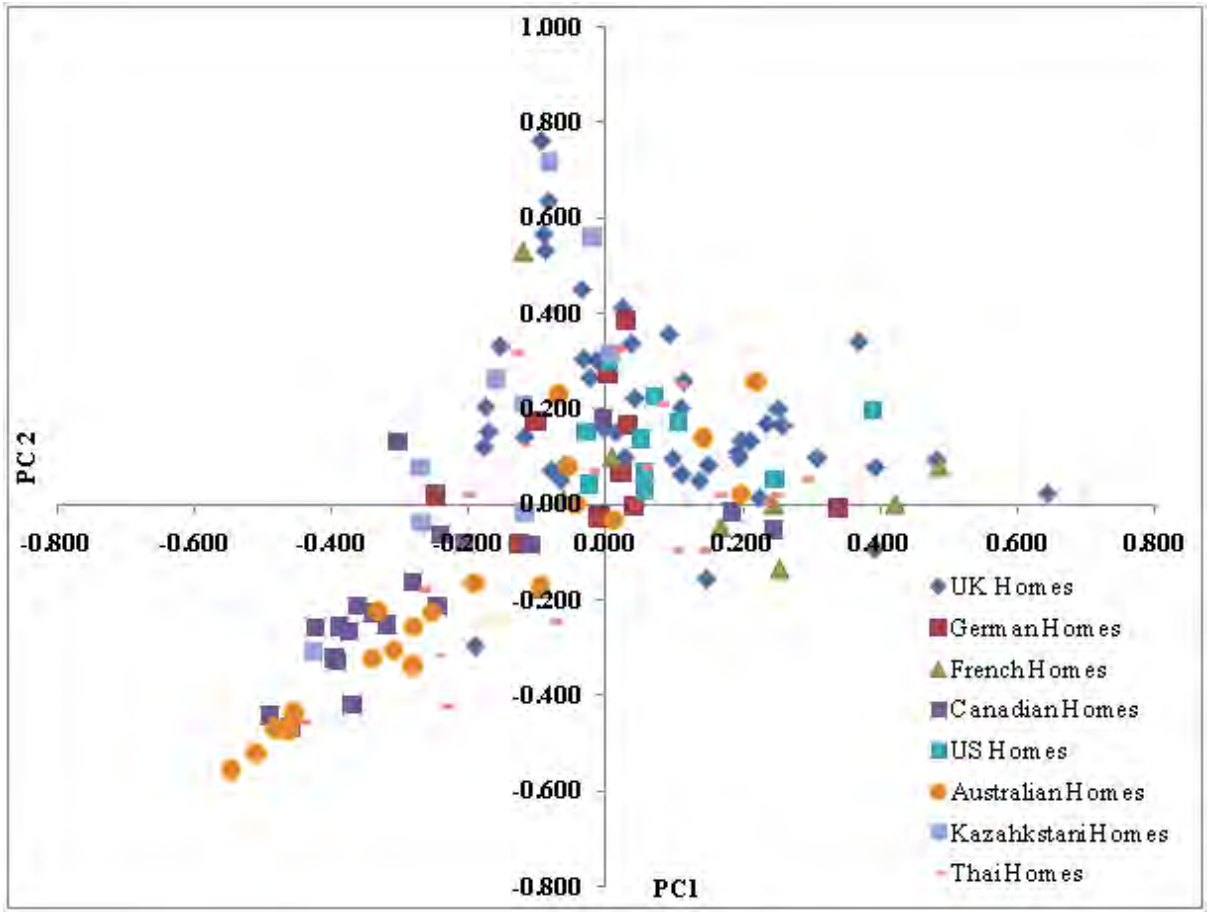


Figure 52 PCA of international homes

EtFOSA was measured in Canadian samples at a median concentration of 550 ng g^{-1} from Toronto, however it was not detected in any of the Ottawa samples (n = 67) from 2002 - 03 (Shoeib *et al.,* 2005). The explanation for the differences in the two studies cannot be provided, however, the EtFOSA was detected within indoor environments in > 90 % of the air samples (Shoeib *et al.*, 2005). At present, there is only one other study that has reported EtFOSA levels within indoor dust from America (Kato *et al.,* 2009) and it reports concentrations that are consistent with this study. The significantly high concentrations in EtFOSA from Australia are hard to define, including where the compounds are sourced from, and current thinking is leading to the presence of EtFOSA in Sulfluramid (insecticide) use within this region.

9.5. CONCLUSION

PFCs were identified in all samples from the 8 countries, which included homes, and some offices and cars. ΣPFC concentrations were highest in Australian homes (2300 ng ΣPFC g^{-1}), and cars (1300 ng ΣPFC g^{-1}). The high concentrations identified in two Australian samples (house 5 and house 8) have an individual presence in the data because of the relatively low number of analysed samples, compared to the UK. Australian samples indicated high concentrations of the three most predominant compounds, PFOS, PFOA and PFHxS with ranges of 7 – 8100 ng PFOS g^{-1}, 15 – 2900 ng PFOA g^{-1} and 19 – 1100 ng PFHxS g^{-1}. Concentrations from the UK, Germany, France, Australia, US, and Canada; remain within a similar range for all measured PFCs, whilst the detection of MeFOSA was noted in less than 25 % of all samples. The USA home samples produce the highest PFOS concentration at 330 ng PFOS g^{-1} and the German home samples provide the highest PFOA median concentration of 300 ng PFOA g^{-1}. It is evident from the original data that the presence of PFCs in

Kazakhstan and Thailand, differs greatly from the Western countries, and sample concentrations of PFOS and PFOA do not exceed 150 ng g^{-1} and 300 ng g^{-1} (respectively) for both countries.

The significantly lower concentrations of most PFCs in Kazakhstani and Thai dust suggest PFC use in these countries is lower than in Australia, Europe, and North America. More intriguing are the lower levels of some PFCs in Canadian dust compared to UK and US house dust, indicating that the FOSEs and PFHxS are not used widely in Canada. Also pertinent are the significantly higher concentrations of EtFOSA in Australian house dust compared to UK house dust. While EtFOSA was deregistered as an insecticide (as Sulfluramid for ant control) in Australia in January 2004, it was licensed for use for about 2 years before this. By comparison, EtFOSA appears never to have been registered for such use in the UK (*28*). It is therefore possible that the higher levels in Australian samples reflect past or recent use of EtFOSA as an insecticide. More puzzlingly, EtFOSA concentrations are elevated (but not significantly compared to other countries studied) in Canadian samples, as EtFOSA has never been registered for insecticide use in Canada. Moreover, the fact that EtFOSA concentrations in our US samples are in line with all other countries except Australia and Canada is at odds with the fact that while use of Sulfluramid is being phased out in the US, it has been licensed for such use in the recent past. Overall, our data suggest further study is warranted of the impact of Sulfluramid on EtFOSA contamination of indoor dust and other matrices relevant to human exposure.

10. CONCLUSIONS AND SUMMARIES

10.1. CHAPTER AND RESULTS SYNOPSIS

The work reported in this thesis has provided an insight into the presence of PFCs in (primarily UK) indoor microenvironments and the associated potential for exposure to adults and children. The majority of a person's day is spent indoors and therefore there is little opportunity to avoid such non-dietary indoor exposures.

PFCs represent a group of compounds, which consist of fluorinated hydrocarbons, often attached to functional groups. The strong carbon – fluorine bond provides the compounds with a high level of resistance to fats, oils, water, dirt and degradation (photochemical, biological and chemical). These compounds are therefore utilised in consumer products to provide stain proof and water resistant coatings for furnishings, textiles and clothing, greaseproof lining in food containers and cooking instruments, and resistant coatings in paints, varnishes and waxes, which can lead to dermal and dietary exposure. Along with these uses, the compounds are widely deployed in the manufacture of metal plated goods, semiconductors, electrical and heat resistant tubes and wire coatings.

The versatility of PFCs has led to their widespread presence in the environment, and the exposure of many species, including those at the top of the food webs. Concentrations of PFCs have been noted in a number of different media including water, air, soil, sludge, biota, blood, milk and fish, with particular concern associated with their documented presence in remote regions, including the poles, and high altitudes.

Toxic effects have been linked to the compounds, for many types of biota including, fish, birds and mammals, and they are able to enter the food web through water, diet and air uptake by plants and animals, resulting in bioaccumulation in top predators.

Within the last decade, restrictions and precautionary measures have been taken to try and limit the use of certain persistent PFCs and consequently reduce their environmental impact. These restrictions include the listing of PFOS under the Stockholm Convention (The POPs, 2010), production phase outs and the use of more volatile PFCs (e.g. FTOHs) as alternatives.

This study has addressed the presence of PFCs in UK urban indoor and outdoor microenvironments and the resultant potential for human exposure. Results have indicated that PFCs are present within indoor environments in both air and dust and this is expected to contribute to daily exposure. Measurable concentrations of PFCs are also reported in outdoor air and soil from across the UK; with both matrices constituting an additional source of non-dietary human exposure. The aims of the project have been addressed as outlined below.

10.2. HYPOTHESIS 1: INDOOR ENVIRONMENTS ARE SUBSTANTIAL RESERVOIRS OF PERFLUOROALKYL COMPOUNDS

This study of UK indoor microenvironments indicates PFCs to be present at measurable concentrations in both air and dust from homes, offices, cars and classrooms within a range of 200 – 10 000 pg $\sum$PFC m^{-3} and 100 – 40 000 ng $\sum$PFC g^{-1}, respectively. Similar results have been seen for indoor dust from various countries including Japan, USA, Canada, Germany and Australia (Moriwaki *et al.* 2003, Kato *et al.* 2008, Tittlemier *et al.* 2007, Fromme *et al.* 2008, Thompson *et al.* 2010). Like other halogenated POPs the presence of these compounds in dust and air are thought to derive from emissions from consumer goods (Washburn *et al.* 2005) and indicate that there is a potential pathway from consumer goods into dust and air, and that these indoor matrices act as a substantial reservoir of PFCs.

Detection frequencies of the 8 PFCs monitored were greater in dust samples than in indoor air. All PFCs were identified in at least one dust sample. Domestic indoor dust has also been shown to retain chemicals such as lead, heavy metals, alkylphenols, brominated flame

retardants, organotin compounds, phthalates and chlorinated paraffins (Santillo *et al.*, 2003). While not monitored in this study, it is pertinent to note that other PFCs such as FTOHs, PAPs, FTACs and others have been detected in indoor air (Lee *et al.*2010, Shoeib *et al.* 2008, Langer *et al.* 2010).

PFC concentrations in dust from primary school and nursery classrooms were found to exceed those in offices, homes and cars. This may suggest the existence of additional sources of PFCs in classroom environments. However, examination of the PFC contamination pattern does not indicate a radical difference to that observed for other indoor microenvironment categories and therefore the higher concentrations are likely due to greater numbers of potential source items within classrooms. PFC concentrations were not correlated with the number of children within the classroom.

Classroom contamination and the 'health' of the room has been considered widely in the past, with previous concerns including chalkboard dust in the air, CO_2 and microbial dust, VOCs and particulate matter (Fox *et al.* 2003, Hodgson *et al.* 2004, Fromme *et al.*2008b). Concern about exposure of children in classrooms has developed throughout the decades, resulting in guidelines and monitoring of some chemicals being routine in school environments (asbestos, atmospheric concentrations of formaldehyde and VOCs in portable classrooms, Hodgson *et al.* 2002, and PCBs, New York Department of Education 2010). The higher concentrations from classrooms noted in this thesis was also observed for PBDEs, PCBs, TBBP-A, and HBCDs for the same samples (Harrad *et al.* 2010c). Suggesting that it is a common pattern of behaviour, and concentrations could be raised by similar variables.

The substantial differences between microenvironments noted in this study indicate that a better assessment of daily human exposure can be achieved by monitoring a variety of microenvironments, rather than just homes. Also, the differences seen between homes, cars

and offices in comparison to schools suggest that children should be considered independently from adults when considering human exposure.

Office environments contained higher mean concentrations of ∑PFCs in dust (2300 ng g^{-1}), compared to homes (1900 ng g^{-1}) but the mean concentrations of ΣPFCs in domestic air (2000 ng m^{-3}) exceeded those in offices (1300 ng m^{-3}). Office air contains significantly higher concentrations of PFOS than air in homes ($p = 0.001$), but significantly lower concentrations of EtFOSA and MeFOSE than in domestic air ($p = 0.04$ and $p = 0.003$, respectively). Also of interest, office dust contains a higher proportion of PFOA than other indoor microenvironments, suggesting PFOA to be a signature of office environments, consistent with reports by D'Hollander *et al.* 2010 of significantly higher concentrations of PFOA in offices compared to homes.

Cars have previously been noted to retain chemicals used in the production and manufacturing. Studies of VOCs in car cabins showed elevated concentrations in new vehicles, slowly declining with time (Chien, 2007) along with a variety of heavy metals (Healthycar.org) and other toxic chemicals including BFRs, ozone and particulate matter (Harrad *et al.*2008, Reidiker *et al.*, 2003). In this study, car samples contained the lowest concentrations of the 8 PFCs monitored in dust, with the lowest ∑PFC dust concentrations. However, while concentrations of the persistent compounds PFOS, PFOA and PFHxS are not significantly different from those in homes and offices, those of MeFOSE, and EtFOSE are significantly lower in cars, whilst FOSA is significantly higher in cars than homes, offices and classrooms.

10.3. HYPOTHESIS II: DISTRIBUTIONS OF PERFLUOROALKYL COMPOUNDS WITHIN A ROOM ARE VARIABLE.

Spatial and temporal variability of PFCs in air and dust from indoor microenvironments are not attributable to variability associated with the preparation and analytical procedures alone. The concentration of a PFC in a sample of dust taken from a single point within a room may not be representative of the concentration in the room as a whole.

Thus, exposure analyses based upon such single "point" samples may not provide an entirely accurate measure of exposure, depending on the area of the room used by each individual occupant. This has implications for determining an individual's exposure, and the extent to which a given dust sample reflects accurately such exposure may be expressed as it's "biologically relevance" (Harrad *et al.* 2010).

In contrast to observation for brominated flame retardants (Harrad *et al.* 2009), in this study the presence of specific objects was not observed to correlate with within-room spatial variation in dust concentrations. Instead, such variability as is observed is thought likely due to spatial variations in dust loadings and "wear and tear" of PFC-containing items within the room. The lack of correlation between PFC concentrations in dust and the presence of specific potential source items may be attributable to the large scale use of PFCs in materials, textiles, upholstery, clothing and cleaning surfactants (Danish Environmental Protection Agency, 2008). It is thought that the cumulative impact from emissions from such sources may disguise the signatures of individual sources.

The spatial variability noted in each of the rooms sampled in this study for PFCs is generally greater than the variability seen for HBCDs (average RSD for homes = 14 %) in the UK by Harrad *et al.* (2009), but comparable to that observed for PBDEs (Harrad *et al.* 2008). Furthermore, a negative correlation between PFC concentration and dust loading was noted for MeFOSE and EtFOSE, at various sites. Such "dilution" of contaminant concentrations in

dust at high dust loadings has been observed previously at some locations for both PBDEs (Harrad et al, 2008), and HBCDs (Harrad et al, 2009).

Within-room temporal variability in concentrations of PFCs in dust is revealed as likely to arise from variations in room use and occupancy, rather than from seasonal variation in e.g. temperature or room ventilation rate. Temporal variations in PFC concentrations appear to occur as a result of changes in room use and room contents over time.

Use of passive samplers located at fixed points within a room to monitor airborne PFC concentrations may underestimate exposure via inhalation. The presence of a personal cloud (Rhodes *et al.,* 1991) in the case of VOCs has been shown to provide a greater exposure, than that based on the whole room air concentration (Harrison *et al.,* 2002). Such an effect has yet to be studied for PFCs but, as with PBDEs (Allen *et al.,* 2007, Hazrati & Harrad, 2006), it is most likely to be pertinent for those PFCs existing primarily in the particulate phase (e.g. PFOS and PFOA).

Temporal variability of both air and dust indicated that concentrations of PFCs can vary over time, as seen in Chapter 4. However, what is not fully understood is the cause of the observed variability, which does not correlate with exterior weather conditions (temperature, sunlight, rainfall). No indoor parameters were monitored at the time and variability within each of the rooms, were independent from one another and not linked to seasonality. The variability was not linked to any additional sources entering the room environments as seen with PBDEs (Hazrati & Harrad, 2006).

The presence of temporal variability has implications for human exposure estimates, where exposure estimates are based upon samples being taken at a single point in time.

10.4. HYPOTHESIS III: THERE IS SUBSTANTIAL SPATIAL AND TEMPORAL VARIATION IN THE DISTRIBUTION OF PFCs IN THE OUTDOOR URBAN ENVIRONMENT.

Spatial and temporal concentrations in outdoor urban air were monitored and display considerably more variability (> 15 %) than that accountable for by extraction and analytical procedures.

For outdoor air, both spatial and temporal variability is dependent upon the volatility of the compound. The less volatile compounds (PFOS, PFOA, PFHxS), as well as MeFOSA, have %RSD ranges between 85 – 350 %, while the more volatile compounds (EtFOSA, FOSA, MeFOSE and EtFOSE) have RSD values that range between 39-110 %. Whilst these ranges overlap there is a substantially greater range for the less volatile PFCs. However the temporal variability in PFC concentrations observed in this study remains below that reported in a German study (Dreyer *et al.,* 2009).

Greater spatial variability was seen for the less volatile compounds, indicating less mixing on an urban scale, and suggesting that these compounds are more strongly influenced by wind speeds, suspension and surrounding environments. The spatial variability of PFCs in outdoor air appears influenced strongly by the proximity of the sampler to busier roads with high congestion rates. The three sites located close to main roads displayed PFC concentrations that were statistically distinct from those at other sites, and support the idea of increased concentrations driven by raised degradation rates of FTOHs (Ellis *et al.,* 2004) and suspension and turbulence of air created in the street canyons (Yarwood *et al.,* 2007). Traffic-related sources of PFCs are their use in oils and hydraulic liquids; and the higher concentrations of NOx and VOCs may promote FTOH degradation. This is also supported by the correlation between the more volatile compounds and degradation products identified at these sites.

This spatial variability – which is particularly marked for less volatile PFCs - will influence human exposure for individuals frequenting highly-trafficked locations (Harada *et al.* 2005, Yarwood *et al.* 2007). This study also reveals significant correlations between airborne concentrations of PFOS and PFOA at many sites. This is consistent with there being a common source or sources of these two PFCs.

The temporal variability in PFC concentrations in outdoor air is related largely to seasonal and climatic variations (significant relationships were identified for some months – see chapter 5). However two notable events in the dataset demonstrate that during a month with high PFC concentrations, the air mass associated with the most highly contaminated samples had travelled primarily from continental Europe. The opposite effect was noted in a month with the lowest PFC concentrations where the air masses derived from the south west, mid-Atlantic region, with slow flow rates, and little time spent over land masses.

Along with air mass trajectory the concentrations of PFCs in outdoor air were significantly correlated with meteorological conditions. The meteorological conditions were specific to each location and varied the measured concentrations by individual amounts, thus the three sites monitored varied independently of the others. The influence of temperature, rainfall and sunlight have been proven to affect concentrations of other atmospheric contaminants (Klánová *et al.* 2006, Su *et al.* 2007, Dreyer *et al.* 2010) including PCBs, dioxins and furans, PBDEs and PAHs.

Correlations between atmospheric concentrations of PFCs and meteorological conditions were present primarily for the more volatile compounds, particularly MeFOSE and EtFOSE. These compounds have been demonstrated to degrade via OH radicals (Plumlee *et al.*2009), and thus are correlated to degradation products (such as PFOS), at the sites, a relationship which was also noted by Stock *et al.* (2005).

10.5. HYPOTHESIS IV: UK SOILS ARE A SUBSTANTIAL RESERVOIR OF PFCs.

Analysis of UK soil samples affirms the presence of PFCs within soil from across the UK; 88 % of UK soils contained detectable concentrations of PFOS, suggesting that UK soils represent a reservoir of PFCs, especially the less volatile compounds like PFOS, PFOA and PFHxS. The lower relative abundance of the more volatile compounds in soil suggests that either soil is not a sink for these compounds, or that they are easily biodegraded; in doing so potentially adding to the concentrations of PFCA and PFAS end-products like PFOS and PFOA.

The presence of PFOS, PFOA and PFHxS in UK soils is in accordance with concentrations noted in background studies (low ng g^{-1}), by Naile *et al.*, 2010, Nakata *et al.*, 2006, Higgins *et al.*, 2005 and Jai *et al.*, 2010, indicating that UK soils are no more contaminated than other countries. Moreover, the concentrations observed in UK soils are low in comparison to those reported by Washington *et al.*, (2007), indicating that the sites monitored in this study to be influenced strongly by industrial sources. Instead, PFCs in UK soils are expected to derive from diffuse pathways, given the relatively inter-site variability in concentrations. In particular, while EtFOSA is a known active constituent of the pesticide Sulfluramid, it has never been authorized for use in the UK.

Concentrations of PFCs in some UK soil samples approach those found in indoor dust, with concomitant potential for soil to act as an exposure pathway to humans, both directly via ingestion, and indirectly via the food web and local water sources. The latter indirect pathways are likely to present the most significant sources of human exposure.

From this study it appears that UK soils are a likely substantial reservoir of PFOS, PFOA and PFHxS, but less so for more volatile PFCs. This is thought due to the more volatile PFCs

leaching to ground water, volatilising to air, and undergoing microbial degradation in the soil matrix (Parsons *et al.* 2008, Beach *et al.*, 2006, Moody *et al.* 2003).

10.6. HYPOTHESIS V: CLASSROOM ENVIRONMENTS ARE CONTAMINATED WITH A SIGNATURE OF PFC CONTAMINATION THAT IS DISTINCT FROM THAT OBSERVED IN OTHER INDOOR MICROENVIRONMENTS.

PFC concentrations in classroom dust were examined to determine whether there were significant differences between PFC contamination of these rooms and that observed in homes, offices and cars. Results revealed that concentrations in classroom environments were generally higher than in other microenvironments (homes, offices and cars), and that concentrations in classroom dust were significantly elevated for PFOS, PFHxS, MeFOSA and FOSA ($p < 0.001$) compared to all the other microenvironments studied. Moreover, concentrations of PFOA and EtFOSE in classroom dust exceeded significantly those in cars and homes respectively. While the absolute concentrations of these PFCs in classrooms are higher, the PCA suggests that the relative contributions to ΣPFCs suggesting that there are no signature compounds present, which are indicative of classroom microenvironments.

Principal component analysis shows however, that while the absolute concentrations of these PFCs are significantly elevated in classrooms their relative contribution to ΣPFC concentrations is indistinguishable from that observed for other microenvironments. This suggests that whilst these compounds are present in other environments there are more sources present within classrooms. Similar to homes, office and cars, the classroom data indicate few relationships between room contents and PFC concentrations, suggesting that a plethora of sources are present. Classrooms containing carpets were noted to be significantly different than those without carpets, but due to a small sample group of classrooms without carpets, the relationship cannot be deemed conclusive. However, a previous study by

Gerwurtz *et al.* (2009), noted a relationship with carpeted rooms and PFCs and that concentrations are likely to arise due to the use of stain repellent surfactants used to prolong the life of the carpets. The study by Gerwurtz *et al.* (2009), supports the relationship noted between carpeted classrooms and ΣPFCs.

Another distinct relationship noted in classrooms was between the textile content of the room, and ∑PFC concentrations. This suggests that the textiles may release PFCs as a result of the stain and water proof coatings added to the textiles. This is particularly relevant for textiles used to protect furniture from paints and food.

Significant positive correlations were noted between concentrations of PFOS and PFOA and EtFOSE and MeFOSE in classroom dust. Such correlations are consistent with the existence of similar sources for the correlated compounds. The correlation between PFOS and PFOA has also been noted by Moriwaki *et al,* (2007) in homes and Björklund *et al.* (2009) in daycare centres, suggesting that this relationship is common to indoor environments and is also reflected in human serum (Midasch *et al.*2007). However, this relationship was not noted in this study for the other UK microenvironments studied.

10.7. HYPOTHESIS VI: THE PRESENCE OF PFCs IN THE INDOOR AND OUTDOOR ENVIRONMENT RESULTS IN BOTH EXTERNAL AND INTERNAL HUMAN EXPOSURE.

The exposure estimates generated in this study are derived from a simplified representation of the true exposures received by the UK population. The large range in exposure to PFCs assumed via the “low”, “typical” and “high” exposure scenarios indicates highly variable individual exposures ranging from insignificantly low levels for adults (under the low scenario) to major contributions to exposure for young children (via the high scenario).

The estimated “high-end” exposure of children via non-dietary sources constitutes 9 % and 0.4 % of the TDI respectively for PFOS and PFOA. This suggests that, even under highly conservative estimates, exposure to non-dietary PFOS and PFOA will not exceed the EFSA TDIs, and is thus unlikely to impact adversely upon health. Moreover, these TDIs are still not exceeded even when dietary exposure is included. However, as a cautionary note, one should bear in mind that the TDI may – as has been the case for dioxin-like chemicals - fall in the future, as new toxicological information emerges (Berthiaume & Wallace, 2001).

This study also shows that in comparison to exposure received via the diet, that under high-end exposure scenarios, the magnitude of non-dietary exposure (i.e. ingestion of dust and inhalation of air) can become comparable to that received via the diet for young children. This suggests that PFC contamination of indoor air and dust is sufficient to contribute significantly to human exposure.

Although not examined in this study, the concentrations of PFCs in air and dust have been correlated to those in serum; thereby indicating non-dietary exposure to be significant for some individuals (Freberg *et al.* 2010, Fraser *et al.* 2010, Haug *et al.* 2010).

The influence of external exposure to precursor compounds on internal doses of PFOS and PFOA is unknown, but assuming 100 % uptake and in vivo metabolisation of such compounds to PFOS and PFOA, the safety margin between exposure and the TDI is narrowed considerably, particularly for children. If the TDI is lowered in the future, then this safety margin will be further eroded, raising the possibility of exceedances of the TDI for some individuals.

The internal dose exposure expected to be derived from non-dietary exposure was modeled using a pharmacokinetic model. Under typical exposure scenarios, non-dietary exposure is projected to contribute approximately 5 % (for both compounds) to concentrations of PFOS

and PFOA in human blood serum for adults, and a larger proportion (≤ 44 %) to concentrations in the blood serum of children.

The above leads to the conclusion that PFCs within air, dust, and soil indoor and outdoor environments can result in internal exposure, on a scale similar to the dose received from dietary sources. Finer analysis reveals that while dust ingestion is the most significant non-dietary source for PFOS and PFOA; inhalation of air is more important for the more volatile PFCs.

10.8. HYPOTHESIS VII: DUE TO GLOBAL VARIATIONS IN USE PATTERNS, THERE IS SUBSTANTIAL INTERNATIONAL VARIATION IN THE CONTAMINATION OF INDOOR DUST WITH PFCs.

PFCs were measured in dust from Australian, Canadian, French, German, Kazakhstani, Thai, UK, and US homes. Some PFCs (PFOS, PFOA, PFHxS, FOSA, PFOSEs and ∑PFC) were significantly lower ($p < 0.05$) in Kazakhstan and Thailand than elsewhere. The differences noted for Kazakhstan and Thailand are associated with lifestyle differences, whereby the consumer goods common in other more industrialised countries were often absent from the homes sampled.

For the other countries studied, differences were noted for MeFOSE and EtFOSE, which were significantly lower in Canada than in the UK and the US. The reasons for this are unknown, as commercial use would be expected to be similar for each of the countries, based on culture and lifestyles. PFHxS was also significantly lower in Canada than the UK. Combined, this suggests that use patterns of these compounds differ between Canada and the UK. On a similar note, concentrations of EtFOSA were significantly higher in Australian than in UK domestic dust. High relative abundances of EtFOSA were also noted in Canada, Kazakhstan and Thailand. A possible explanation for the elevated presence of this compound in some homes is the use of the insecticide Sulfluramid.

The differences in concentrations of certain PFCs found in house dust samples from different countries indicates that extrapolation of human exposure assessments conducted for one country to another is not recommended. Importantly, this study provides no evidence that the current EFSA TDIs are being exceeded in any of the countries studied as a result of exposure via ingestion of house dust.

10.9. RECOMMENDATIONS FOR FURTHER WORK

The historic and continued manufacture and use of PFCs means that they will continue to maintain an environmental presence for the foreseeable future. Despite considerable research in recent years, there remain numerous research gaps relating to the environmental impacts, movement, and toxicity of PFCs. Such gaps include:

- Better understanding of the distribution of PFC profiles in the environment, their uptake via biota and environmental transport and fate.
- Better comprehension of the relative human metabolism and toxicity of straight and branched chain PFOS isomers (Houde *et al.*, 2008, Kärrman *et al.*, 2009).
- Analysis of the actual migration of PFCs from their sources and the mechanisms involved.
- Analysis of the distribution of FTOHs in UK indoor environments and their possible contribution to contamination by PFOS and PFOA.
- Research into the toxicological impact of PFCs in conjunction with multi-chemical analysis on a range of scales – e.g. from bioassays to epidemiological studies.
- The provision of a more comprehensive and accurate representation of the contribution of different microenvironments to overall human exposure, by quantification of PFC contamination in a greater variety and number of locations.

- Examination of the impacts of recent control measures with respect to PFC production and use. For example whether concentrations of PFOS and PFOA are reducing in the environment or whether the gap in the market left by restrictions on PFOS and PFOA has been filled by more volatile compounds with the ability to degrade to PFOS and PFOA.
- Investigation of the possible impact of traffic on PFC contamination in outdoor air.
- More detailed study of the presence of Sulfluramid in the environment and its role as a source of EtFOSA to the environment and of human exposure.

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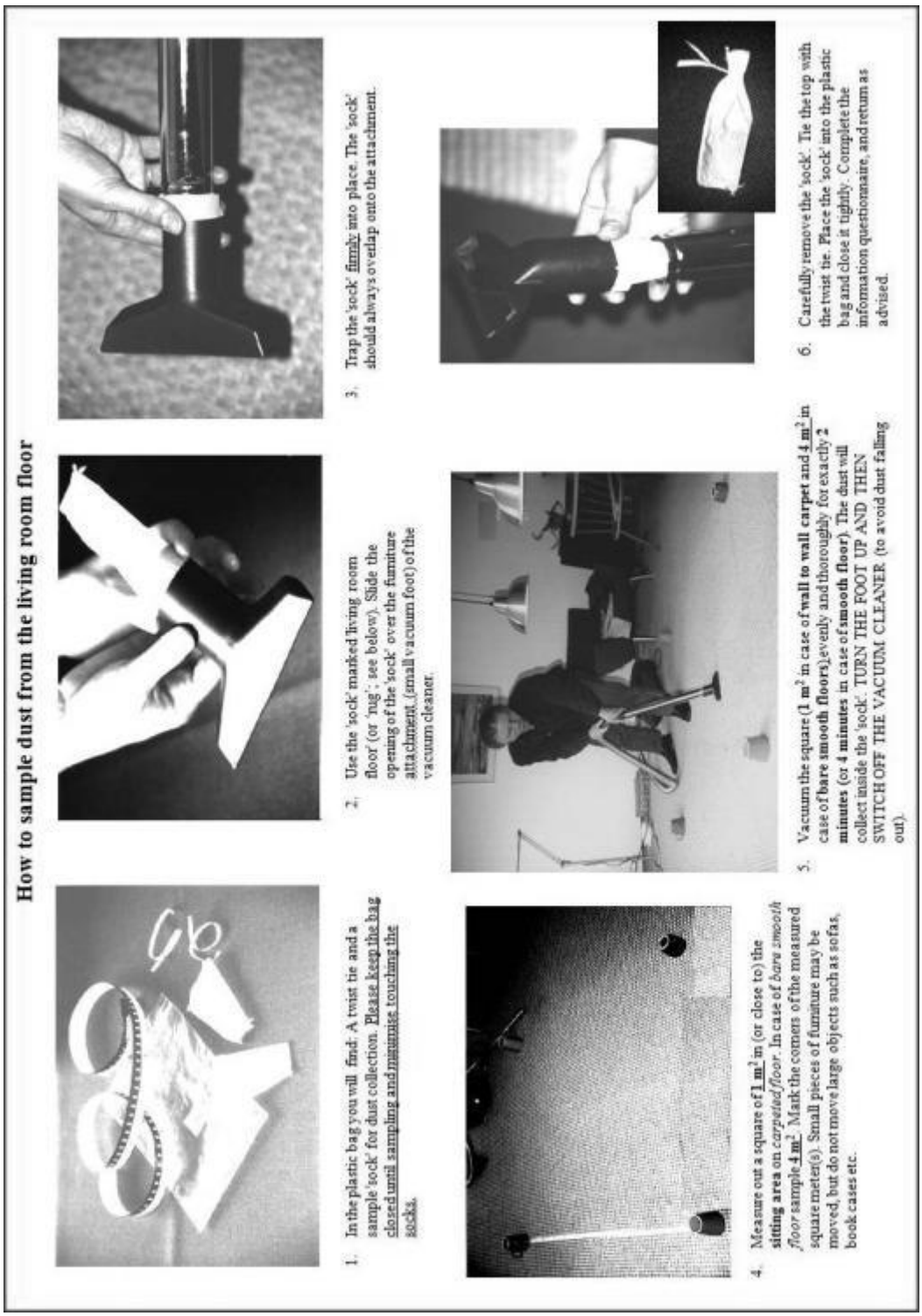
12. APPENDIX 1

A) HOUSE DUST COLLECTION PROTOCOL



B) HOUSE SAMPLING QUESTIONNAIRE

Sample ID (for lab use only):

Address from which sample taken (voluntary information, you do NOT HAVE to provide this to participate in the study, it merely facilitates future contact in case of queries – please include an email address if you have one):

Please indicate here if you would like to be informed of the results for your sample:

Construction year of the building (if known):

Date Sample taken:

Approximate time since last vacuumed:

(please leave at least 2 days between date of last vacuuming and taking sample)

ROOM SPECIFICATIONS:

Please complete the details below for the living room sampled

1. **Room ventilation**: ☐ Natural ☐ Mechanical (air-conditioned)
2. **Number of foam containing chairs (plus year of manufacture/purchase if known):**
3. **Number of foam containing sofas (plus year of manufacture/purchase if known):**
4. **Number of PCs/laptops (plus year of manufacture/purchase if known):**
5. **Number of TVs (plus year of manufacture/purchase if known):**
6. **Number of other items of electrical equipment (Printer, home cinema, video, DVD player, audiocassette, microwaves etc.) (plus year of manufacture/purchase if known):**
7. **Is the room carpeted? (If so, what is the year of manufacture/purchase if known):**
8. **Do the windows have curtains (as opposed to blinds)? (If so, what is the year of manufacture/purchase if known):**
9. **Have any of the textile surfaces in the room (e.g. furniture/curtains been "stain-proofed")? If so, please specify which items and how many?**
10. **Approximately how frequently do you vacuum this room?**
11. **Please add any further information you feel may be relevant.**

C) CAR SAMPLING QUESTIONNAIRE

Sample ID (for researcher's use only):

Date:

Car manufacturer and model:

Year of manufacture:

No. of seats:

Car ventilation: **Natural** ☐ **Air conditioned**☐

Type of seat cover: **Fabric** ☐ **Leather** ☐

Electronics inside the car (please tick box if appropriate:

Stereo ☐

Speakers ☐ **No.:**

GPRS ☐

DVD PLAYER (BUILT-IN) ☐ NO.:

Other electronic devices either built-in or used regularly (e.g. portable DVD player) (please specify):

Approximate time since vehicle last vacuumed:

Manufacturer, model number and date of manufacture (if known) of child seat(s). (If more than one, please give details of each) :

Approximate time (hours per week) spent in the car by:

(a) family adults
(b) children

13. APPENDIX 2

A) GERMAN HOUSE DUST SAMPLES

House	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	140	460	140	<DL	190	84	96	110
2	200	370	78	<DL	220	<DL	47	120
3	110	290	110	<DL	120	130	700	180
4	62	19	16	<DL	53	5	3	11
5	1200	95	42	<DL	96	44	54	120
6	47	44	160	16	120	24	11	49
7	800	730	790	<DL	93	100	120	120
8	190	550	720	<DL	36	79	98	150
9	230	300	470	<DL	730	49	11	28
10	160	98	410	<DL	250	43	210	120

B) FRENCH HOUSE AND OFFICE DUST SAMPLES

House	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	140	38	320	<DL	23	<DL	<DL	<DL
2	160	15	63	<DL	75	5	190	<DL
3	260	21	58	17	220	<DL	<DL	550
4	200	50	160	<DL	130	3	<DL	140
5	54	31	71	31	320	7	170	<DL
6	160	220	54	<DL	130	2	130	300
7	1700	31	210	<DL	280	3	68	<DL
8	140	18	77	<DL	120	7	510	240
9	160	49	170	<DL	64	4	610	500

Office	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	150	42	250	<DL	47	6	61	<DL
2	33	32	110	29	110	13	<DL	<DL
3	67	10	16	<DL	40	<DL	<DL	<DL
4	290	11	72	<DL	76	4	62	<DL
5	220	20	190	<DL	54	7	45	24
6	280	15	180	<DL	97	4	34	<DL
7	200	220	160	390	180	9	32	95
8	160	9	200	<DL	40	4	290	<DL

C) U.S.A HOUSE DUST SAMPLES

House	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	510	370	390	0	220	11	60	120
2	180	1800	160	0	380	25	110	120
3	720	300	200	19	160	45	130	170
4	380	73	350	0	44	110	71	44
5	830	40	250	130	46	73	71	120
6	110	280	93	0	63	93	310	120
7	930	120	86	0	120	99	58	170
8	240	190	560	0	230	40	100	340
9	120	27	390	0	41	65	230	700
10	170	490	220	0	79	100	74	220

D) CANADIAN HOUSE DUST SAMPLES

House	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	1300	110	34	<DL	340	27	<DL	<DL
2	110	130	40	<DL	410	30	<DL	5
3	160	86	26	<DL	1200	<DL	<DL	5
4	180	280	94	150	820	300	<DL	5
5	42	87	16	<DL	<DL	17	2	<DL
6	1100	4000	55	470	4800	240	47	66
7	46	45	240	<DL	250	<DL	<DL	<DL
8	710	<DL	200	<DL	500	<DL	14	6
9	92	18	150	<DL	1600	<DL	5	4
10	150	83	850	<DL	7900	2700	4	17
11	560	210	330	<DL	3400	380	5	18
12	64	<DL	<DL	<DL	<DL	<DL	34	<DL
13	60	<DL	<DL	<DL	230	16	15	<DL
14	380	43	210	160	1700	<DL	1	7
15	420	50	200	<DL	420	<DL	3	1
16	53	<DL	<DL	<DL	<DL	<DL	10	8
17	53	78	220	<DL	880	11	3	<DL
18	160	67	150	<DL	620	<DL	15	15
19	56	49	68	<DL	350	4	4	6
20	140	72	160	<DL	610	13	3	4

E) AUSTRALIAN HOUSE AND CAR DUST SAMPLES

House	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	27	48	73	<DL	<DL	<DL	67	19
2	17	510	88	<DL	<DL	<DL	170	5
3	7	120	19	<DL	840	<DL	3	11
4	290	44	60	<DL	300	4	130	4
5	6800	2900	440	3000	6800	47	19	6
6	34	19	230	<DL	8600	<DL	9	7
7	92	24	67	<DL	2100	4	15	5
8	8100	2780	220	<DL	930	140	11	7
9	160	550	1100	<DL	550	2	400	0
10	44	19	95	<DL	1400	<DL	16	20
11	34	190	89	<DL	970	<DL	95	100
12	190	24	230	990	480	<DL	<DL	440
13	620	54	330	<DL	1400	<DL	22	20
14	340	15	340	<DL	4100	20	52	90
15	79	32	150	74	930	<DL	33	110
16	180	360	110	120	100	<DL	320	140
17	110	120	88	210	720	18	32	33
18	1000	910	360	74	330	11	80	40
19	840	860	250	1100	5100	120	43	24
20	1500	1000	470	1600	4600	140	150	130

Car	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	360	35	99	340	1900	<DL	21	5
2	47	17	230	67	570	2	44	230
3	170	29	180	550	350	<DL	7	58
4	240	380	55	<DL	370	<DL	1	3
5	140	63	300	47	780	2	<DL	16
6	30	610	110	<DL	310	<DL	<DL	1
7	89	81	280	270	2600	200	62	75
8	110	350	230	<DL	69	150	<DL	110
9	120	130	600	<DL	260	64	59	18
10	510	81	1100	<DL	91	67	290	590

F) KAZAKHSTANI HOUSE & CAR DUST SAMPLES

House	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	59	60	161	<DL	190	<DL	2	2
2	<DL	<DL	1	<DL	<DL	<DL	<DL	<DL
3	69	<DL	120	<DL	570	<DL	6	8
4	44	<DL	30	<DL	<DL	<DL	7	<DL
5	86	81	180	<DL	89	<DL	9	<DL
6	56	73	120	<DL	70	<DL	7	15
7	<DL	<DL	3	<DL	<DL	<DL	<DL	<DL
8	130	<DL	100	<DL	180	<DL	6	<DL
9	64	220	140	<DL	250	<DL	73	24

Car	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	59	46	156	<DL	72	<DL	5	17
2	120	78	210	<DL	68	<DL	8	8
3	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
4	15	25	48	<DL	64	<DL	2	1
5	1800	710	4300	69	42	13	550	80
6	1000	600	4400	50	51	22	80	89
7	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
8	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
9	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
10	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL
11	<DL	<DL	<DL	<DL	<DL	<DL	<DL	<DL

G) THAI HOUSE DUST SAMPLES

House	PFOS	PFOA	PFHxS	MeFOSA	EtFOSA	FOSA	MeFOSE	EtFOSE
1	27	72	31	<DL	33	18	<DL	1
2	15	43	55	4	19	7	<DL	<DL
3	16	12	16	<DL	<DL	3	11	1
4	65	140	10	<DL	300	20	140	18
5	95	<DL	11	<DL	280	24	<DL	57
6	63	5	6	<DL	5	<DL	6	22
7	10	2	16	<DL	24	28	18	1
8	16	19	4	<DL	8	2	1	32
9	9	24	21	<DL	100	41	42	100
10	8	62	3	<DL	940	12	20	270
11	15	9	15	<DL	54	2	6	1
12	44	45	84	0.05	8	5	2	1
13	6	2	6	9	4	2	6	19
14	31	94	14	13	700	5	10	1
15	130	4	3	0.05	<DL	31	<DL	13
16	3	290	43	0.05	120	10	1	350
17	60	1	75	<DL	230	24	1	270
18	9	17	52	<DL	13	18	23	1
19	4	52	22	<DL	7	5	<DL	21
20	22	2	17	6	27	7	<DL	1