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ARTICLE *in* ENVIRONMENTAL SCIENCE & TECHNOLOGY · FEBRUARY 2011

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Indoor Sources of Poly- and Perfluorinated Compounds (PFCS) in Vancouver, Canada: Implications for Human Exposure

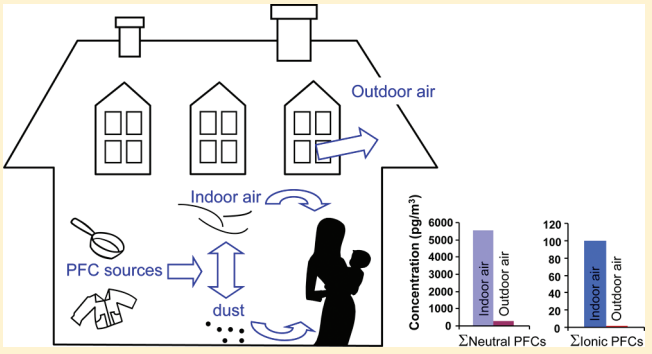
Mahiba Shoeib,*[,†] Tom Harner,[†] Glenys M. Webster,[‡] and Sum Chi Lee[†]

[†]Atmospheric Science and Technology Directorate, Environment Canada, Toronto, Ontario, Canada

[‡]School of Environmental Health, University of British Columbia, Vancouver, British Columbia, Canada

Supporting Information

ABSTRACT: Perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) are widely detected in human blood and serum and are of concern due to their potential toxicity. This study investigated the indoor sources of these compounds and their neutral precursors through a survey of 152 homes in Vancouver, Canada. Samples were collected of indoor air, outdoor air, indoor dust, and clothes dryer lint and analyzed for neutral [i.e., fluorotelomer alcohols (FTOHs), perfluorooctane sulfonamide (FOSA), and perfluorooctane sulfonamidoethanol (FOSE)] and ionic [i.e., PFOS and perfluoroalkyl carboxylates (PFCAs)] poly- and perfluorinated compounds (PFCs). Indoor air was dominated by 8:2 FTOH with a geometric mean concentration (pg/m^3) of 2900. Among the FOSAs and FOSEs, MeFOSE exhibited the highest air concentration with a geometric mean of 380 pg/m^3. PFOA was the major ionic PFC and was detected in all indoor air samples with a geometric mean of 28 pg/m^3, whereas PFOS was below the detection limit. The results for the ionic PFCs in indoor air are the first for North America. The pattern of the neutral PFCs in house dust was also dominated by 8:2 FTOH, with a geometric mean of 88 ng/g. Dusts were enriched (relative to air) with sulfonamidoethanol (FOSE) which comprised ~22% of the total neutral PFC content compared to only ~3% in air. PFOS and PFOA were the most prominent compounds detected in dust samples. Levels of neutral PFCs in clothes dryer lint were an order of magnitude lower compared to house dust. Human exposure estimates to PFCs for adults and children showed that inhalation was the main exposure route for neutral and ionic PFCs in adults. For toddlers, ingestion of PFCs via dust was more relevant and was on the order of a few mg/day. Results from this study contribute to our understanding of exposure pathways of PFCs to humans. This will facilitate investigations of related health effects and human monitoring data.



INTRODUCTION

Perfluoroalkyl sulfonates (PFSAs) and perfluoroalkyl carboxylates (PFCAs) are widely detected in wildlife and humans around the world,[1,2] and are typically dominated by the eight-carbon members perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). In addition to their ability to be transported great distances, these chemicals are recognized for their persistence, bioacummulation, and toxicity. In humans, associations between PFOS or PFOA levels and sperm quality,[3] reduced birth weight,[4] and changes in adult thyroid hormone levels[5] were reported. Because PFOS and PFOA cross the placenta[6] and are found in breast milk,[7] exposures to the developing fetus and infants are of particular concern. These properties of PFOS have led to its recent listing under the Stockholm Convention on persistent organic pollutants (POPs).[8] Consequently, it will be included as a target compound under the global monitoring plan (GMP) that includes core media of air and human tissues.[9]

There is some uncertainty regarding the fate and exposure pathways of humans to PFOS and PFOA. Exposure may occur through a number of mechanisms including (i) directly, due to the manufacture and use of PFOS and PFOA in commercial products;[10−13] (ii) abiotic breakdown (to PFOS and/or PFOA) of "precursor" compounds that are also released during PFC production and/or from commercial products (examples include fluorotelomer alcohols (FTOHs), perfluorooctane sulfonamides (FOSAs), and perfluorooctane sulfonamidoethanols (FOSEs) that degrade to PFOS and/or PFOA);[14,15] and (iii) metabolic transformation of precursors that have already been absorbed into the body.[16,17]

Human exposure pathways to PFOS, PFOA, and relevant precursors are also numerous and include exposures through diet and drinking water, air inhalation, dust ingestion, and absorption

Special Issue: Perfluoroalkyl Acid

Received: October 21, 2010
Accepted: February 1, 2011
Revised: January 19, 2011
Published: February 18, 2011

through direct skin contact with consumer products.[13,18] Although diet is thought to be a key exposure pathway to PFOS and PFOA[19] the indoor environment (inhalation of air and dust ingestion) is also believed to be important, especially since people spend more than 90% of their time indoors. Previous studies have shown that indoor air concentrations of poly- and perfluorinated compounds (PFCs) were 1 to 2 orders of magnitude higher than outdoor values.[20−22] Concentrations of PFCs in house dust may also serve as an important exposure pathway for toddlers who ingest larger quantities of dust through increased hand-to-mouth contact and related behavior.[23,24] PFC exposure route by toddlers and pregnant women (i.e., to fetuses in utero) is a concern due to possible developmental effects associated with PFCs.[25] The Vancouver-based Chemicals, Health and Pregnancy study (CHirP) aims to identify the main PFC exposure for pregnant women, including exposures via the air, dust, dryer lint (a possible surrogate for dermal exposure), drinking water, and a wide range of exposure data collected by questionnaire. A second goal is to examine the impact of maternal PFC levels on maternal thyroid hormones, which play a critical role in fetal brain development. So far, no comprehensive study investigating the exposure of a pregnant female cohort to PFCs has been reported for Canada.

This study reports on the concentrations of PFCs in indoor air, house dust, and clothes dryer lint in samples collected during 2007−2008 from up to 152 homes of pregnant women residing in Vancouver, Canada who were participants in the CHirP study. The results will be used to evaluate human exposure to these chemicals, and in future work, will be combined with questionnaire data and the results of PFC concentrations measured in serum samples collected from the study participants.

MATERIAL AND METHODS

Chemicals. The target analytes included seven neutral PFC compounds (fluorotelomer alcohols, perfluorooctane sulfonamides, perfluorooctane sulfonamidoethanols) and nine ionic PFCs (perfluorooctane sulfonate (PFOS) and C6 to C12 and C14 perfluoroalkyl carboxylates (PFCAs). Details are in Table S1 of the Supporting Information. All solvents used to process samples were HPLC grade.

Sample Collection. During 2007−2008, 152 women from the city of Vancouver, Canada who were enrolled in the CHirP study made their home available for this survey. Details on sample collection and storage are provided in the Supporting Information. Briefly, dust was collected from all 152 homes. Indoor air samples were collected from a subset of homes (n = 59). Indoor air was sampled using sorbent impregnated PUF (SIP) disk passive samplers[26] that were deployed for ∼4 weeks in participants' bedrooms. For purposes of comparison, outdoor air concentrations were also measured. Because outdoor air is more homogeneous, only six outdoor SIP passive samplers were deployed in selected participants' yards to assess average outdoor air concentrations for the metropolitan area. The outdoor samplers were deployed for ∼3 months. The sampling rate of the SIP disk passive sampler has been previously reported for PFCs.[26] To ensure the validity of the derived sampling rates, low-volume air samplers were deployed in a subset of homes (n = 5). To assess PFCs associated with clothing and the dermal absorption exposure pathway, lint samples were collected from the dryer machines of 63 participants. Detailed exposure assessment questionnaires were administered that included information on, *inter alia*, participant's diet, time activity patterns, time spent at work, home characteristics, and the use of PFC-containing consumer products (e.g., carpet care liquids, nonstick cookware, dietary type, packaged fast foods, and waterproof clothing). The role of these exposures, as well as PFC levels in air, dust, and lint on maternal body burdens will be explored in a following publication.

Sample Analysis. Details regarding the processing and analysis of air, dust, and lint samples are presented in the Supporting Information. All sample extracts were reduced to a final volume of 0.5 mL. Analysis of air, dust, and lint sample extracts for neutral PFCs (FTOHs, FOSA, and FOSEs) was by gas-chromatography−positive chemical ionization mass spectrometry (GC-PCIMS). Analysis details are given elsewhere.[27]

Quantification of the neutral PFCs was normalized against responses of their mass-labeled counterparts (added prior to extraction).

For ionic PFCs, concentrations of PFOS and PFCAs were determined by high-performance liquid chromatography (HPLC) using an Agilent LC 1100 connected with tandem mass spectrometry (MS/MS). Air extracts were analyzed on an API 3000A and dust extracts were analyzed on an API 2000 Q Trap (Applied Biosystems/MDS Sciex). Details on LC columns, mobile phases, flow rate, and injection volume are presented in the Supporting Information. Analyte responses were normalized to the response of ^{13}C-PFOA that was added to samples prior to extraction.

Blank levels for air samples were assessed using field/travel blanks (n = 12) that were collected by placing the SIP disk in the appropriate housing (indoor or outdoor) then removing it after 1 min. A field blank and a laboratory blank were processed with each set of extracts. Blank levels for dust and lint were assessed using sodium sulfate (n = 12) that was sieved, stored, and processed/analyzed as for samples.

RESULTS AND DISCUSSION

Quality Control/Quality Assurance. Results from field blanks for air samples were used to assign method detection limits (MDL), calculated as the mean of the field blank values plus 3 standard deviations. MDL values for SIP disks ranged from 0.5 to 14 pg/m^3 for neutral PFCs whereas blank levels of ionic PFCs were mostly below detection (Table S2 in the Supporting Information). Of the ionic PFCs, only PFHpA and PFOA were detected in most blanks. One anomalous field blank was elevated in PFHpA and PFOA and was removed from data treatment. If not removed, it would have resulted in MDL values of 7.6 and 9.3 pg/m^3 instead of 0.33 and 0.47 pg/m^3, respectively (Table S2).

Neutral PFCs were not detected in the sodium sulfate blanks except for 8:2 FTOH (Table S4). When compounds were not detected in blanks, $^2/_3$ of the instrumental detection limits (IDLs) were used for calculating the MDLs. Instrument detection limits (IDLs) were calculated from the lowest standard, extrapolating to the corresponding amount of analyte that would generate a signal-to-noise ratio of 3:1 (see Tables S2−S5).

All samples were normalized against recoveries of labeled compounds added prior to extraction. For the neutral PFCs, recoveries of the labeled compounds calculated against NN Me_2FOSA injected standard were variable. For instance, for air samples, recoveries ranged from (103 ± 45) % for FTOHs to (158 ± 90) % for MeFOSE. This underscores the importance of using mass-labeled compounds for quantification in order to correct for analyte loss and/or signal suppression or enhancement

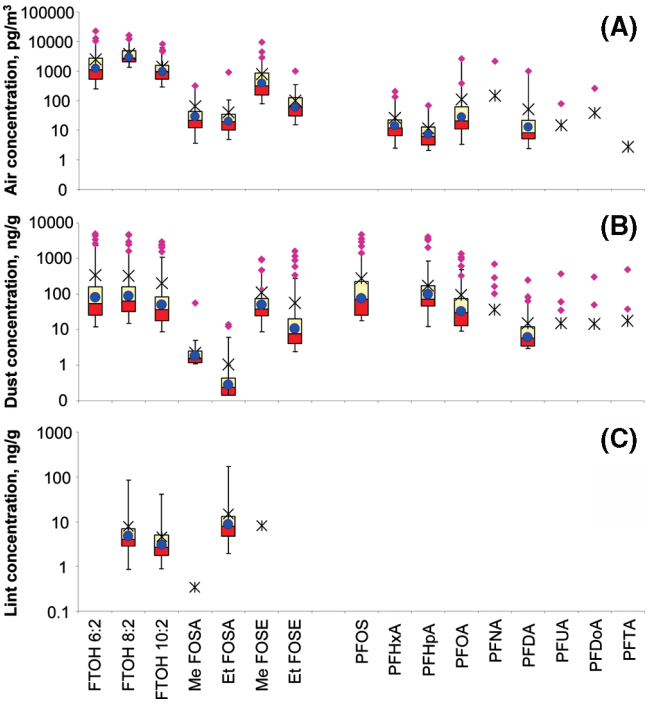


Figure 1. Box and whisker plots showing the distribution of neutral and ionic PFCs in indoor samples from Vancouver, Canada. (A) Indoor air concentrations in pg/m^3, (B) dust concentrations in ng/g, and (C) lint concentrations in ng/g. The lower and upper ends of the box are the 25th and 75th percentiles of the data. The horizontal solid line within the box is the median value and the symbols × and ● are the arithmetic and geometric mean values, respectively. The whiskers define the 5th and 95th percentile and values greater than this are shown individually. Note: for compounds that were detected in less than 10% of samples, only the mean value is presented as the other statistical metrics are subject to bias introduced by the substitution of $^2/_3$ of the MDL value.[42].

on a sample-by-sample basis. Other studies have also reported similar variability and high recoveries.[28] All neutral compounds were recovery corrected to their counterpart labeled compounds.

In the case of the ionic PFCs, individual mass-labeled standards were not available. All ionic target analytes were normalized based on the response of ^{13}C PFOA that was added to each sample prior to extraction (i.e., recovery corrected to labelled PFOA). All data reported in this study are not blank corrected.

The extraction efficiency of the duplicate sonication for dust and lint was tested by conducting a third sonication cycle. Results confirm that >95% of the total analyte was extracted in the first two cycles.

PFCs in Indoor and Outdoor Air. To derive air concentrations from the amounts of chemical accumulated in the SIP disks, it was necessary to first evaluate and confirm the SIP disk sampling rate. Calibration of the SIP samplers was accomplished by codeploying active, low-volume samplers in a subset of 5 homes for integrated 4-week samples. Results indicate a SIP disk sampling rate of ∼5 m^3/day for FTOHs and ∼4 m^3/day for the FOSAs, FOSEs, and ionic PFCs (see Figure S1). The derived sampling rates for the FTOHs are consistent with previous calibration results;[26] however, FOSA is about ∼1.5 times and FOSE is about ∼2.5 times higher in the current study compared with the previous calibration study.[26] It is notable that the SIP disks results agreed well with high-volume air samples during an international calibration exercise targeting PFCs in outdoor air.[29]

Neutral PFCs in Air. SIP disk-derived indoor air concentrations are shown in Figure 1 and Table S2 (n = 59). Indoor air samples were dominated by FTOHs that accounted for 92% of total neutral PFCs. The 8:2 FTOH and 10:2 FTOH were above the method detection limit (MDL) in all samples while 6:2 FTOH exceeded MDL in 97% of samples. Total FTOHs air concentration (pg/m^3) ranged from 890 to 47 000. The highest concentration among the FTOHs was 8:2 FTOH, with a geometric mean of 2900, followed by 6:2 FTOH at 980 and 10:2 FTOH at 950. Interestingly, 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH air concentrations that were above the 95th percentile were obtained from the same homes which may indicate a fresh and common source of FTOHs. In general, 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH were positively correlated (r^2 = 0.78, p < 0.001) suggesting that they originate from the same source.

A similar magnitude of FTOH indoor air concentrations was observed in Ottawa, Canada in 2002 during a survey of ∼52 homes.[21] In the Ottawa study, 8:2 FTOH and 10:2 FTOH also showed strong correlation suggesting a common source. The indoor air concentrations of FTOHs observed in Vancouver (this study) and Ottawa are similar to results from homes in Tromso, Norway where active samplers were employed.[22,30] For example, the geometric mean air concentration for 8:2 FTOH (pg/m^3) was 2070 in Ottawa and 3424 in Tromso. This suggests that levels of FTOHs may be broadly similar across homes in industrialized regions.

Among FOSA/FOSE detected in indoor air, MeFOSE was dominant with a geometric mean of 380 pg/m^3. The air concentration of EtFOSE was ∼6 times lower at 60 pg/m^3 followed by MeFOSA and EtFOSA at 30 and 20 pg/m^3, respectively. In contrast to the good agreement observed for the FTOHs between this study and the Ottawa survey,[21] the levels of MeFOSE and EtFOSE in Vancouver homes were 5−10 times lower compared to homes in Ottawa. This may reflect a geographic difference (western vs eastern Canada) in indoor sources. These differences may also represent a temporal decrease (2002 vs 2007−2008) in indoor air concentrations of the sulfonamidoethanol compounds in response to their ban in North America for more than a decade. MeFOSE, MeFOSA, and EtFOSA were positively correlated (r^2 = 0.74, p < 0.001) however EtFOSE was neither correlated to MeFOSE or to any of the other FOSAs (r^2 = 0.02, p > 0.4), indicating that EtFOSE is likely used in different applications. Much higher indoor air concentrations of FOSAs and FOSEs were reported in the Norwegian study conducted in April−June, 2005, with concentrations as high as 1200 pg/m^3.[30]

Results for outdoor air (n = 6) are summarized in Table S3. In general, outdoor air concentrations of the neutral PFCs were ∼17 times lower compared to indoor values, highlighting the importance of the indoor exposure pathway. Higher indoor vs outdoor air concentrations of PFCs have been observed in other studies.[21,22] The outdoor air profile was also dominated by 8:2 FTOH with geometric mean (pg/m^3) of 151. Similar to indoor air, MeFOSE was dominant among the FOSA/FOSE compounds in outdoor samples.

Ionic PFCs in Air. A subset of SIP disk indoor samples (n = 39) was analyzed for ionic PFCs, representing the first indoor air values reported for North America. PFOA was the dominant ionic PFC, detected in all indoor air samples with a geometric mean (pg/m^3) of 28 followed by PFHxA and PFHpA at 9.7 and 5.1, respectively (Figure 1, Table S2). PFOS was below the method detection limit in all indoor samples. This could be due to a low abundance in indoor air or because it is mainly associated

with air particles which are not captured efficiently by the passive sampler.[31] PFHxA and PFHpA were detected in >90% of the samples and the frequency of detection decreased with increasing chain length. The sum of ionic PFCs was about 50 times lower than the sum of neutral PFCs in indoor air, reflecting the lower volatility of the ionic compounds. In Tromso homes, the particle-phase profile of ionic PFCs was markedly different and dominated by PFHpA followed by PFOA at 17.1 and 4.4 pg/m^3, respectively.[22] Gewurtz et al. report ionic PFCs in the indoor window film in Toronto with a profile dominated by PFOA, PFDA, PFNA, and PFOS.[32] The authors conclude that concentrations and profiles can vary throughout a building depending on sources and indoor ventilation patterns. The ionic PFCs in outdoor air were below the detection limit except for PFHpA and PFOA that were detected in 4/6 samples (Table S3) with mean concentrations (pg/m^3) of 1.7 and 2.5, respectively.

PFCs in House Dust. Results from the analysis of 140 indoor dust samples collected from home vacuum cleaners are presented in Figure 1 and Table S4. Dust samples that were collected by broom (n = 12) were not sufficient for sieving and were excluded from the study. In some cases (n = 15) samples were collected from two or three different vacuum cleaners from the same house and were analyzed separately to check for variability. These results were averaged and represented as a single value for the home (Table S4). Within the same home, the variability (calculated as the % SD for replicate samples) in PFC concentrations across replicate dust samples ranged from <10% to up to 250%, and followed the order 8:2 FTOH < 10:2 FTOH < MeFOSA < MeFOSE. Colt et al. have validated the use of vacuum cleaner bags for assessing house dust concentrations of semivolatile organic chemicals.[33]

Neutral PFCs in Dust. As observed for indoor air, the pattern of the neutral PFCs in house dust was also dominated by 8:2 FTOH, with a geometric mean (ng/g dust) of 88. This distribution pattern is similar to what was observed for air indicating coupling of the FTOH burdens in indoor dust and air. However, the relative abundance of FTOHs vs sulfonamides was different in dust compared to air. The dust samples were relatively enriched with the FOSEs which made up ∼22% of the total neutral PFCs, compared to only ∼3% in air. This may reflect greater volatilization of FTOHs that have higher vapor pressures and lower K_{oa} values compared to the sulfonamidoethanols.[34,35] Among the neutral PFCs, MeFOSA and EtFOSA were found at the lowest levels in house dust, representing only ∼1% of the total neutral PFCs. Similar to air, a significant positive correlation exists for 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH dust concentrations (r^2 = 0.94, p < 0.001) suggesting a common source for these compounds. Comparable dust FTOH concentrations were reported in Ottawa, Canada,[36] and in North Carolina and Ohio homes and daycares.[23]

MeFOSE was the dominant sulfonamide compound in house dust with a geometric mean of 51 ng/g followed by EtFOSE at 8.5 ng/g, while EtFOSA and MeFOSA were about an order of magnitude lower at 0.1 and 1.8 ng/g, respectively (Table S4). Unlike the observation for air, dust concentrations of sulfonamides showed a greater deviation between mean and median values indicating greater departure from a normal distribution (Figure 1, Tables S2 and S4). A significant correlation was observed for MeFOSE and MeFOSA (r^2 = 0.61, p < 0.001) however EtFOSE was not correlated to either MeFOSE or the FOSA compounds, consistent with the air results. Also consistent with the air results, MeFOSE and EtFOSE dust concentrations from this study were about a factor of 2 lower than for the survey of Ottawa homes in 2003 with geometric means of 110 and 120 n/g, respectively.[21] Goosey et al. report FOSA and FOSE from BDL to an extremely high value of 13 000 ng/g in dust samples collected in 2007−2008 from 43 classrooms and daycare centers in the West Midlands of United Kingdom.[37]

Ionic PFCs in Dust. PFOS and PFOA were the most prominent of the ionic PFCs and were detected in all house dust samples with concentrations (ng/g) ranging from 1.5 to 4700 and 2.0 to 1400, respectively (Figure 1, Table S4). Other PFCAs were less frequently detected; PFNA was above MDL in 70% of dust samples and PFTA was above MDL in 40% of samples (Table S4). Similar to the results for indoor air, dust concentrations of ionic PFCs that were above the 95th percentile were generally obtained from the same homes. The median and maximum total PFCs (neutral and ionic) concentrations were 370 and 25 500 ng/g, indicating that typical loading of perfluorinated compounds is ∼0.4 μg and could reach ∼25 μg per 1 g of dust with 50% to 60% distributed between neutral and ionic. Statistical regressions showed that PFOA and PFOS were significantly and positively correlated (r^2 > 0.63, p < 0.001). These findings are consistent with other studies on house dust.[23,24,37]

Correlations of neutral and ionic PFCs in house dust showed variable results. For instance, 8:2 FTOH was not correlated with PFOA or PFOS (r^2 < 0.001, p > 0.2) suggesting different sources of these compounds. The lack of correlation between 8:2 FTOH and PFOA in indoor air was unexpected since PFOA occurs as a residual in fluorotelomer-based products.[11] Another possible reason for expecting a correlation between 8:2 FTOH and PFOA is that 8:2 FTOH is a precursor for the formation of PFOA. However, studies have shown that this reaction pathway is restricted in urban air (including indoor air) due to the presence of elevated NO in urban air.[38,39] MeFOSE was significantly and positively correlated with PFOA (r^2 > 0.21, p > 0.001) and PFOS (r^2 < 0.26, p > 0.001), despite considerable scatter in the data. It has been reported that the electrochemical fluorination (ECF) process used to manufacture perfluoroalkyl sulfonate-based products also produces PFOA with ∼30% branched isomers.[11,40] However, the analysis of linear and branched PFOA to elucidate the origin of PFOA (i.e., ECF vs telomerization which produces only straight chain isomers) was beyond the scope of this study and all data reported in this study represent total PFOA and PFOS (i.e., branched and linear).

PFOS and PFOA concentrations in house dust from this study are comparable to results from the Ottawa survey in 2003.[36] The dominance of PFOS and PFOA in dust samples has been reported in other studies from around the world with highest concentration observed in the UK and U.S. (Table S6) and likely attributed to higher use of these chemicals in commercial products in these countries. The year in which samples were collected could be an important factor in determining concentrations in house dust as discussed for air. For instance, regulation of PFOS and related compounds in North America may partly explain the lower air concentrations of FOSEs observed in more recent indoor studies.

To evaluate whether PFCs in air and dust have the same source, paired dust and air concentrations for the same homes were evaluated for 8:2 FTOH and MeFOSE. A significant positive correlation was obtained for MeFOSE (r^2 = 0.54, p < 0.001) indicating a close coupling between air and dust. In the case of 8:2 FTOH the correlation was significant but less strong (r^2 = 0.11, p < 0.02). This suggests that 8:2 FTOH in indoor air

may be influenced by sources other than just indoor dust (or that this FTOH may be degraded at different rates in air compared to dust).

PFCs in Clothes Dryer Lint. Lint extracts (n = 63) were analyzed for neutral PFCs; unfortunately, ionic PFCs were not analyzed in these samples due to problems during extraction. Results are presented in Figure 1 and Table S5. Duplicate extractions and analyses were performed on 6 lint samples to check the distribution of PFCs in different subsamples from the same home. Differences between duplicate samples ranged from 1% to 228% with an average of 23%. The highest variation across duplicate samples was observed for 8:2 FTOH. These differences may reflect variable PFC contents between laundry batches.

The 8:2 FTOH, 10:2 FTOH, and MeFOSE were detected in >90% of the lint samples, EtFOSE and MeFOSA were detected in ~70% of samples, and 6:2 FTOH and EtFOSA were below the method detection limit in all samples. As observed for air and dust, the highest concentration of neutral PFCs was for 8:2 FTOH, with a geometric mean of 3.5 ng/g (Table S5). Interestingly, the neutral ΣPFCs for lint was 1 order of magnitude lower than that for dust. This relatively low concentration in lint could be due to a number of factors including (i) the high temperatures used during drying that drive absorbed PFCs into the gas-phase and elimination via the dryer exhaust; (ii) generally low concentrations of PFCs in dryer lint (i.e., clothing) compared to house dust. Published reports on levels of FTOHs in clothing material has been limited so far to ski jackets.[41]

Human Exposure to PFCs. Concentrations of PFCs in air and dust samples from this study were used to estimate the exposure of adults and children to neutral and ionic PFCs. Two exposure scenarios were assumed: a mean and high scenario, representing low and high intake rates. Calculations for each scenario were performed using four concentration levels of target compounds in air and dust, representing (1) the 5th percentile (low exposed group), (2) the median (representing most of the population), (3) the 95th percentile, and (4) maximum concentrations, representing the worst case that would apply to a small portion of the population. For compounds that were detected in less than 10% of samples, only the mean value was calculated as other statistical metrics are subject to bias by the substitution of 2/3 MDL for nondetects.[42]

The dust ingestion rate (E_{Ingest}, ng/day) was calculated using

$$E_{Ingest} = C_{dust} \times Q_{dust} \times F_{uptake} \quad (1)$$

where C_{dust} is the concentration of Σneutral or Σionic PFCs in house dust (ng/g) and Q_{dust} is the dust ingested rate (g/day) of 4.16 and 100 mg/day for the mean scenario, and 55 and 200 mg/day for the high scenario for adult and toddler, respectively.[43] F_{uptake} is the uptake fraction of a compound. Based on previous studies of gastrointestinal absorption[44,45] we used the value of 0.8 for the ionic PFCs and 0.38 for neutral PFCs for the uptake fraction. It is noteworthy that earlier exposure studies based their calculations on 100% absorption.[21,23,24]

Similarly, the inhalation exposure (E_{Inhal}, ng/day) was calculated using

$$E_{Inhal} = C_{air} \times V_{air} \times F_{uptake} \quad (2)$$

where C_{air} is concentration of Σneutral or Σionic PFCs in indoor air (ng/m^3), V_{air} is the volume inhaled (m^3/day; 20 L/min for adults and 13 L/min for children),[46] and F_{uptake} is uptake fraction. As was done for ingestion and for consistency, we used

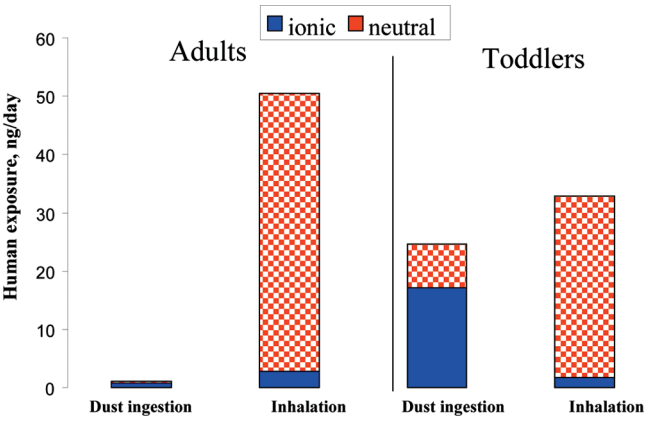


Figure 2. Exposure to ionic and neutral PFCs (ng/day) for adults and toddlers based on median air and dust concentrations (mean scenario). Tabulated results are given in Table S7.

0.8 and 0.38 as uptake fractions for ionic and neutral PFCs, respectively, for the mean scenario, and a value of 1.0 for both ionic and neutral PFCs in high the scenario. We acknowledge that there is uncertainty with the results of these exposure calculations given the lack of directly measured specific uptake fractions for the study chemicals.

Table S7 summarizes the exposure results for adults and toddlers for the different scenarios. Figure 2 shows the mean exposure scenario using the median air and dust concentrations (most of the population). For adults, inhalation is the dominant exposure pathway (ng/day) for both ionic (2.7) and neutral (48) PFCs, compared to only 0.7 and 0.3 for ingestion, respectively. When compared to the estimated Canadian adult dietary intake of 110 ng/day for PFOS and 70 ng/day for PFOA[47] the intake of total PFCs by inhalation and dust ingestion using the mean scenario from this study is substantial and represents about 28% of the dietary contribution (representing just PFOS and PFOA, as to our knowledge no other PFCs have been consistently reported in diet samples). Exposure to neutral PFCs and subsequent biotransformation to PFOS and PFOA[16,17] may help to explain human body burdens of these chemicals.

In the case of toddlers who ingest more dust due to hand-to-mouth contact, dust ingestion becomes a very important pathway. For instance, under the mean scenario, and median air and dust concentrations, intake of ionic PFCs was 17 ng/g via dust ingestion compared to ~2 ng/g for inhalation (Figure 2, Table S7). For neutral PFCs, inhalation still dominates and accounts for ~90% of total neutral PFCs. Toddlers are accumulating about the same total amount of PFCs as adults (~52 ng/day) but preferentially through different routes. Zhang et al. reported higher PFOA concentrations in blood of nonadults compared to adults, whereas PFOS showed the opposite behavior.[2] The higher PFOA in nonadults was explained by exposures through breast milk and dust ingestion in infants and toddlers.

The main implications of this work are the following: (i) Neutral and ionic PFCs are abundant in indoor air, indoor dust, and dryer lint and this presents exposure pathways for humans. (ii) For adults, intake via inhalation is important, especially for the neutral PFCs, whereas for toddlers, intake via dust ingestion becomes more relevant. and (iii) Exposure to PFOS and related chemicals through the indoor environment is important for understanding the long-term trend of these chemicals in human populations. This will be required with the recent addition of

PFOS to the global monitoring plan of the Stockholm Convention on POPs.

The results of this study provide some insight for designing future work. For instance we recognize that (i) it is important to use labeled standards for each target compound to reduce analytical uncertainty; (ii) more data are needed over time to investigate temporal trends in indoor concentrations of PFCs that may reflect changes in use patterns and effectiveness of regulation efforts; and (iii) future studies should include additional target analytes to capture changes in use/production patterns. For instance, perfluorobutanoic acid and perfluorobutane sulfonate (PFBA and PFBS) are expected to increase in indoor samples in response to preferential production and commercial use of these shorter chain PFCs.

■ ASSOCIATED CONTENT

Supporting Information. Additional discussion, a figure and data tables. This material is available free of charge via the Internet at http://pubs.acs.org.

■ AUTHOR INFORMATION

Corresponding Author
*Phone +416 739 5961; e-mail mahiba.shoeib@ec.gc.ca.

■ ACKNOWLEDGMENT

We thank all study participants and students involved in sample collection and extraction. This work was funded by Health Canada (Myriam Hill) and partial funding was also provided by Environment Canada's Chemicals Management Plan (CMP).

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NOTE ADDED AFTER ASAP PUBLICATION

There were two new references added to the reference list in the version of this paper published February 18, 2011. The correct version published May 31, 2011.