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Per- and polyfluoroalkyl substances in paired dust and carpets from childcare centers

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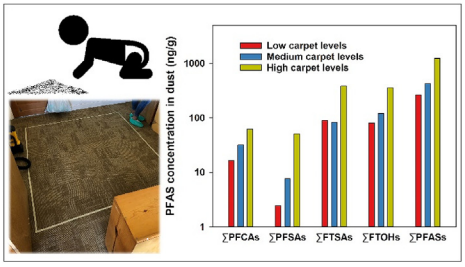
HIGHLIGHTS

- 42 per- and polyfluoroalkyl substances (PFASs) were analyzed in childcare samples.
- Two short-chain fluorotelomer-based PFASs dominated both carpets and dust.
- Strong associations were found between PFAS levels in carpet and dust pairs.
- PFASs of emerging concern significantly contributed to children exposure.

GRAPHICAL ABSTRACT



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ABSTRACT

Carpets can be a significant source of per- and polyfluoroalkyl substances (PFASs) in the indoor environment and may be an especially important source of exposure for children and toddlers. Most previous studies focused on measuring indoor dust only. In this study, we measured PFAS concentrations in paired carpet and dust samples from 18 California childcare centers in 2018 to investigate carpet as a contributor to PFASs in dust. Median total PFAS concentrations (∑PFASs) in carpets and dust were 471 ng/g and 523 ng/g, respectively. 6:2 FTOH and 6:2 FTSA were the two dominant PFASs, collectively accounting for over 50% of the ∑PFASs in both media. Other frequently detected PFASs included C_4–C_{14} perfluoroalkylcarboxylic acids, C_4–C_8 perfluoroalkylsulfonic acids, PFDS, 4:2 FTSA, 8:2 FTSA, FOSA, MeFOSE, EtFOSE, 8:2 FTOH, and 10:2 FTOH. We found strong associations between PFAS levels in carpet and dust pairs, suggesting that carpets can be a source and a sink for PFASs. The estimated total perfluoroalkyl acids (PFAA) intake via dust ingestion for children was 0.023, 0.096, and 1.9 ng/kg body weight/day in the low-, intermediate-, and high-exposure scenarios, respectively. Our data suggest that PFASs of emerging concern are playing an increasingly important role in indoor exposure to PFASs.

1. Introduction

Per- and polyfluoroalkyl substances (PFASs) are a large family of man-made chemicals consisting of aliphatic chain(s) in which all or most carbons are fluorinated, excluding the terminal functional group, like carboxylate, sulfonate, or alcohol (Buck et al., 2011). They have been used in industrial and commercial applications since the 1950s due to their useful hydrophobic and lipophobic properties, as well as their high chemical and thermal stability (Jian et al., 2017). Prior to 2003, PFAS production primarily consisted of C_8 compounds such as perfluorooctanesulfonic acid (PFOS),

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perfluorooctanoic acid (PFOA), and related chemistries. However, increasing concern about the toxicity and bioaccumulative potential of long-chain PFASs resulted in the phase-out of C_8 compounds by 3 M during 2000–2002 (USEPA, 2000) and by DuPont and seven other major manufacturers by 2015 (USEPA, 2006). PFOS and its salts and precursors were added to Annex B of the Stockholm Convention 2009 (Stockholm Convention). Additionally, a stewardship agreement was reached between the US Environmental Protection Agency (USEPA) and eight major PFAS manufacturers to reduce production of PFOA and related compounds by 95% by 2010, followed by their elimination after 2015 (USEPA, 2006). In the 2014 company progress reports, all participating companies stated that they successfully met the PFOA Stewardship Program goals (USEPA, 2014). As a result, manufacturers shifted towards short-chain (number of fluorinated carbons $\leq$ 6) and ether-based PFASs. These alternative PFASs share the extreme persistence of the long-chain compounds but are more mobile in surface water and groundwater. Knowledge of their toxicity is emerging.

Food, drinking water, indoor air and dust are the main routes of human exposure to PFAS (D'Eon and Mabury, 2011; Gebbink et al., 2015), and the relative contribution of each route depends on the exposure scenario being considered. Indoor dust, due to its abundance, accessibility, and capacity to sorb contaminants from surrounding media (e.g., flooring, consumer products, and indoor air), has been widely used as a representative medium for assessing human exposure to various contaminants indoors, including PFASs (Watkins et al., 2011; Kara′skova′ et al., 2016). Carpeted floors have been previously linked to higher PFAS contamination in indoor environments (Kubwabo et al., 2005; Gewurtz et al., 2009; Harris et al., 2017; Winkens et al., 2018). The application (during manufacturing or use) of stain- and soil-repellents containing PFASs and carpets acting as a sink for PFASs from other sources can result in the presence of PFASs in carpets (Kubwabo et al., 2005; Knobeloch et al., 2012; Karásková et al., 2016). Either way, carpets may cause direct consumer exposure to PFOA, PFOS, and other PFAS via hand-to-mouth behavior, which is particularly important for infants, toddlers, and children (Trudel et al., 2008).

Vestergren et al. reported that, although human exposure to PFOA and PFOS through household dust was modest compared to that via food intake, for other PFAS, including perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), perfluorotetra decanoic acid (PFTeDA), perfluorononanoic acid (PFNA), and perfluorotridecanoic acid (PFTrDA), dust ingestion contributed to 27–49% of the total exposure (Vestergren et al., 2012). An exposure assessment for children demonstrated that ingestion of settled dust constituted 36% of their total PFOS exposure, comparable to the dietary intake (42%) (Egeghy and Lorber, 2011). Epidemiological research identified adverse health effects on immunity, cardiometabolism, neurodevelopment, thyroid, kidney and puberty onset caused by children's exposure to PFASs (Braun, 2017; Rappazzo et al., 2017), and hence it is important to evaluate PFAS exposure for children via dust ingestion in the indoor environment. Then major source(s) of PFASs in indoor dust should be identified and children's exposure reduced.

Only a few studies measured indoor dust from children' bedrooms and schools (Strynar and Lindstrom, 2008; Bjorklund et al., 2009; Goosey and Harrad, 2011; California Environmental Protection Agency, 2012; Winkens et al., 2018; Giovanoulis et al., 2019). To our knowledge, only two of those focused on PFASs in dust from U.S. childcare centers. For example, Strynar and Lindstrom measured perfluoroalkylcarboxylic acids (PFCAs), perfluoroalkylsulfonic acids (PFSAs) and fluorotelomer alcohols (FTOHs) in dust samples from 10 Ohio and North Carolina daycares in 2000–2001, and they found that PFOA and PFOS dominated (Strynar and Lindstrom, 2008). A report by the California EPA demonstrated that PFOA, perfluorodecanoic acid, and PFOS were the most commonly detected perfluoroalkyl acids in dust samples collected from California childcare centers in 2010–2011 (California Environmental Protection Agency, 2012). These two studies targeted a limited number of individual PFASs, mostly legacy compounds (e.g., long-chain perfluoroalkyl acids) and samples were collected ~10 or 20 years ago.

Recently, as a result of manufacturers utilizing telomerization processes to synthesize PFAS products, the presence of fluorotelomer-based PFASs in the environment has increased. In addition, it became clear in the last few years that some of these compounds (e.g. FTOHs and fluorotelomer acids) may undergo biotransformation and environmental degradation to form perfluoroalkyl acids (PFAAs) (Buck et al., 2011). None of the studies on childcare centers mentioned above applied recently-established PFASs' uptake and biotransformation factors (Gebbink et al., 2015) into their estimations of PFAS intakes via dust ingestion.

Given the rapid market shifts and large number of PFASs present in the market and the limited information on the environmental fate of PFASs of emerging concern, we are revisiting the question of children's exposures to PFAS via indoor dust with a broader list of target analytes. In this study, we measured PFAS concentrations in paired carpet and dust samples collected from 18 California childcare centers in 2018. To our knowledge, this is the first time that PFAS have been analyzed in carpet and dust samples collected simultaneously. We targeted 42 individual chemicals, including PFCAs, PFSAs, and PFAA precursors. Our objectives were to measure emerging and legacy PFASs in indoor dust and carpets from childcare centers, investigate the association of PFASs in carpet with those in indoor dust, and estimate PFAA exposure risks via dust ingestion for children at ages 2–6.

2. Materials and methods

2.1. Chemicals and reagents

The detailed list of targeted PFAS analytes and the mass-labelled PFASs used as surrogate and internal standards is provided in Tables S1 and S2. The authentic standards, including 12 PFCAs, 10 PFSAs, 3 fluoroalkylsulfonamides (FASAs), 2 fluoroalkylsulfonamidoethanols (FASEs), 3 fluorotelomer carboxylic acids (FTCAs), 3 fluorotelomer sulfonic acids (FTSAs), 4 FTOHs, 3 fluorotelomer acrylates (FTAcrs), and 2 fluorotelomer methacrylates (FTMAcrs), as well as their the isotopically labelled PFASs were purchased from Wellington Laboratories (Guelph, ON, Canada), Sigma-Aldrich (St. Louis, MO), or Matrix Scientific (Columbia, SC). All individual standards were $\geq$95% purity ($\geq$98% for mass-labelled standards). Envi-Carb (graphitized non-porous carbon) and centrifugal filters (nylon membrane, 0.2 μm) were obtained from Sigma-Aldrich and VWR International (Radnor, PA), respectively. All solvents were HPLC grade or higher and they were purchased from Fisher Scientific (Hanover Park, IL).

2.2. Sample collection

A total of 29 carpet and 28 dust samples were collected from 18 California childcare centers in the summer of 2018. All instruments used during sample collection were pre-cleaned with water and isopropanol. Indoor dust was collected using a Eureka Mighty Mite (Model 3670) vacuum cleaner equipped with nylon socks (25 μm pore size, Allied Filter Fabrics, Australia) mounted on the attachment tube. An area of 2 m × 2 m (4 m^2) in the center of each classroom was vacuumed for 5 min. The dust collected was then weighed and kept in the sock, which was tied with a rubber band

and wrapped in aluminum foil. Carpet fibers were sampled along with dust collection. The top fibers (visually dust-free) of a corner of the selected carpet were snipped off using scissors. If the carpet did not have plush fibers, a corner of the carpet including its backing was cut using scissors. The carpet collected was then weighed and kept in a Ziploc bag. Both dust and carpet samples were stored at −20 °C until analysis.

2.3. Sample analysis

The sample treatment procedures used in this study followed previously established methodologies with slight modifications (Reiner et al., 2015; Karásková et al., 2016; Winkens et al., 2018). Dust (sieved through a 150 μm sieve to remove coarse materials [i.e. fibers and debris] that are less likely to be ingested by children in the same manner as dust) and carpet (fibers) samples were treated using the same protocol. A 50–100 mg sample was spiked with 20 ng each of surrogate standard (used for assessing method performance), and extracted with 3 mL of 4:1 hexane/isopropanol twice and then with 2 × 3 mL of 1:1 methanol/acetonitrile. A pre-weighed whole nylon sock was extracted for the dust field blanks using ten times the volume of solvent used for real samples, since a nylon sock was approximately 10 times heavier than the dust or carpet samples (approximate weight of socks was 850 mg). Each extraction step was performed using sonication for 30 min followed by centrifuge at 3000 rpm for 5 min. The supernatants were combined and concentrated under nitrogen till ~5 mL. The sample was cleaned-up by adding 100 mg of Envi-Carb to the extract, which was vortexed for 1 min and centrifuged for 5 min. The resulting sample was reduced to 500 μL with nitrogen blowdown, and then filtered using a centrifuge filter. The filtrate was transferred into a 1-mL polypropylene vial and spiked with 50 ng each of the internal standards used for quantitation.

FASAs and ionic PFASs, i.e., PFCAs, PFSAs, FTSAs, and FTCAs, were analyzed by ultrahigh performance liquid chromatography coupled to a triple quadrupole mass spectrometer (Agilent 1290 Infinity II UPLC − 6470 QQQ-MS), operated in the negative electrospray ionization mode (ESI^-). A gas chromatographic mass spectrometer, operated in the positive chemical ionization mode (Agilent 7890 GC–5977 B PCI-MS), was used to determine FASEs, FTOHs, FTAcrs, and FTMAcrs. Detailed compound-dependent parameters are included in Tables S1 and S2.

2.4. Quality assurance and control

Twenty dust field blanks were prepared following the same sampling protocol applied for the dust collection except that no dust was vacuumed. Also, a procedural blank and a matrix spike were processed along with every 9–12 samples. PFAS compounds detected in procedural blanks and dust field blanks were included in Table S3. Repeatability of the method was evaluated by analyzing 10% of our samples twice. The relative standard deviations of individual PFAS levels between the duplicate measurements were all <25%. The method detection limits (MDLs) for carpet were defined as the average procedural blank level + 3 × standard deviation (n = 6) or the amount of chemicals generating a signal-to-noise ratio of 5 if compound was not detected in the procedural blanks. The MDLs for dust were defined as the average dust field blank level +3 × standard deviation (n = 20) or the PFAS amount generating a signal-to-noise ratio of 5 if a PFAS was not detected in the dust field blanks (See Table S3). Given the relatively high 6:2 FTSA residue in dust field blanks, probably from the sampling devices (e.g. the vacuum cleaner and nylon socks) or plastic items used during processing, the 6:2 FTSA concentrations in these dust samples should be interpreted cautiously. The recoveries of surrogate standards (average ± standard deviation) were 87 ± 12%, 83 ± 12%, 84 ± 22%, 83 ± 5.2%, 87 ± 4.5%, 93 ± 6.1%, 159 ± 47%, 146 ± 45%, 81 ± 5.2%, 51 ± 16%, 82 ± 33%, 61 ± 15%, 81 ± 13%, and 99 ± 24% for M3PFBA, M3PFBS, MPFHxA, MPFHxS, MPFOA, MPFOS, M2-8:2 FTCA, M2-8:2 FTSA, MPFUnDA, dMeFOSA, M2PFTeDA, M4-4:2 FTOH, M2-8:2 FTOH, and dMeFOSE, respectively. Matrix spike recoveries for individual PFASs were in the range 54–169% (Table S3). To compensate for possible losses of analytes during our experimental procedures and for potential matrix effects on instrumental analyses, a surrogate standard was assigned to each PFAS based on their similarity in chemical structure and their chromatographic retention time (Table S4). These surrogate standard spike recoveries were in the range 73.4–111%. Given that all of these relative recoveries were more rational than those for the absolute recoveries (Table S3), the concentration reported in this study were surrogate adjusted.

2.5. Data analysis and exposure assessment

Statistical analyses and plotting were performed using SigmaPlot 14.0 (Systat Software Inc.), SPSS 20 (IBM Corporation), or OriginPro 2017 (OriginLab Corporation). Cells containing values below MDLs (considered not-detected) were substituted with MDL/sqrt (2) for all statistical analyses (i.e., median, Mann-Whitney test for comparison of group means, Kendall's tau correlation test, and linear regression model) and for exposure estimates. Previous studies showed that this substitution provides acceptable results both for summary statistics and group comparisons (Antweiler and Taylor, 2008; Antweiler, 2015). Only PFASs detected in ≥50% of the samples were included in exposure estimation (Gebbink et al., 2015; Winkens et al., 2018), while a detection frequency cutoff of 68% was used for the linear regression model to make sure that all the values in the medium- and high-tertiles were greater than the corresponding MDLs (Stapleton et al., 2014; Hoffman et al., 2015). Prior to data analyses, all values were logarithmically transformed to ensure normal distribution and homogenous variance across groups, which were confirmed by the Shapiro-Wilk test and the Brown-Forsythe test, respectively. The level of significance was set at $\alpha = 0.05$.

To estimate children's daily intake of PFAAs and precursors via oral dust ingestion, we used the concentrations of PFAAs (C_{PFAA}) and their precursors ($C_{precursor}$) in childcare center dust and followed the equation suggested by Gebbink et al. (2015):

$$\begin{aligned}&\textit{Total estimated daily PFAA intake via dust ingestion}\\&= \sum\left(\frac{C_{PFAA}\times q_{dust}\times f_{\mathrm{time_{in}}}}{m_{bw}}\times F_{uptake}\right)+\sum\left(\frac{C_{precrsor}\times q_{dust}\times f_{time_{in}}}{m_{bw}}\times F_{uptake}\times F_{biotransf.}\right)\\&= \sum \textit{Direct PFAA intake}+\sum \textit{Indirect PFAA intake}\end{aligned}$$

where q_{dust} is quantity of ingested dust (g/d); m_{bw} is body weight (kg); f_{time_in} is fraction of time spent indoors; F_{uptake} is gastrointestinal uptake fraction; $F_{biotransf.}$ is biotransformation factor of precursor compounds. The total PFAA intake via dust ingestion equals the sum of direct intake of PFAAs and indirect PFAA intake from biotransformation of precursors. To simulate three typical exposure scenarios, i.e., low-, intermediate-, and high-exposure, different values were applied. F_{uptake} and $F_{biotransf.}$ assigned to individual PFCAs, PFSAs, FTOHs, and FASEs were based on their

number of fluorinated carbons. Though no parameters were available for FTSAs, it has been reported that FTSAs can undergo biotransformation via unsaturated telomer carboxylic acids (FTUCAs) (Wang et al., 2011). Therefore, to assess the contributions of FTSAs to the total PFAA intake, we used the F_{uptake} and $F_{biotransf.}$ proposed for FTUCAs (Gebbink et al., 2015). Detailed information on the parameters used for individual PFASs for exposure assessment are provided in Table S5.

3. Results and discussion

3.1. PFASs in carpets and dust

Forty out of 42 targeted PFAS compounds were detected in carpet samples (See Table 1 for summary statistics and Table S6 for individual PFAS levels in each carpet sample), and 24 were observed in more than half of the samples. The total PFAS concentrations (∑PFASs, the sum of 42 target compounds) ranged between 32.2 ng/g and 8500 ng/g. PFASs may be added to carpets not only during manufacturing in the form of stain- and soil-repellents but also during use (e.g., carpet cleaning), and sometimes the latter is the more important source for PFASs in carpets. No significant differences in PFAS concentrations were detected between fibers and carpets with backing (all $p > 0.05$).

PFASs were also detected in all the dust samples, with total concentrations ranging between 46.9 ng/g and 6470 ng/g (see Tables 2 and S7). Only 10:2 FTCA, 4:2 FTOH, 8:2 FTMAcr were not detected in any of the dust samples. Several studies have measured PFASs in residential or office indoor dust, while data on PFASs in daycare dust remain scarce.

3.2. PFCAs and PFSAs

In carpet samples, the range of total PFCA concentrations (∑PFCAs, sum of 13 compounds) was 5.13–884 ng/g and the median 37.1 ng/g. The most frequently detected PFCAs were PFNA (100%), PFOA (100%), PFPeA (100%), PFTrDA (100%), PFBA (97%), PFHxA (97%), PFHpA (93%), and PFTeDA (90%). Outstanding PFBA concentrations observed in some carpet samples are noteworthy (Table S6). Some studies suggest that since short-chain PFASs are inferior to their long-chain counterparts in terms of technical performance, much larger amounts are needed to guarantee similar performances (Lindstrom et al., 2011). The total PFSA concentrations (∑PFSAs, sum of 10 compounds) were in the range 0.76–339 ng/g and the median, at 8.25 ng/g, was lower than that for ∑PFCAs. PFBS, PFHxS, PFOS, and PFDS were the more frequently detected (DF ≥ 60%) (Table 1).

The carpet PFOA concentrations reported here, with a median of 6.13 ng/g, were generally lower than those reported for carpets collected in the United States (U.S.) during 2007–2011, with a median of 15.2 ng/g, and for Canadian composite carpet (all manufactured before 2005) (USEPA, 2009; Liu et al., 2014; Kim et al., 2015). This decline is likely the result of the phase-out of PFOA by 2015 as well as differences in type of samples (USEPA, 2006, 2014). In contrast, lower levels of PFOA and PFOS were found in carpets purchased in Norway during 2012–2013 than in our study (Herzke et al., 2012; Vestergren et al., 2015), perhaps due to their lesser historical use of fluorinated stain- and soil-repellents in Europe.

In dust samples, ∑PFCAs ranged between 8.37 and 386 ng/g. C_4–C_{14} PFCAs were detected in ≥80% of the dust, while PFHxDA (C_{16}) was only quantifiable in five samples (Table 2). PFBA, PFHxA, PFOA, and PFNA were detected in virtually all of the dust samples, and they were the predominant PFCAs. ∑PFSAs, in the range of 1.29–190 ng/g, were less abundant than ∑PFCAs. The most frequently-detected PFSAs in dust were PFBS (DF: 100%), PFOS (100%), and PFDS (89%), with median concentrations of 0.53, 4.64, and 1.35 ng/g, respectively. The production of PFOS and its related compounds has been discontinued since 2000–2002 by major U.S. manufacturer, but legacy PFSAs are still continuously released from consumer products still in use, manufactured prior to the phase-

Table 1
Detection frequencies (DF, %), median and range for PFAS concentrations (ng/g) in carpet samples from childcare centers (n = 29). 10:2 FTAcr and 6:2 FTMAcr were not detected. Values in parentheses represent the number of fluorinated carbons. Non-detect values were replaced by MDL/sqrt (2) for median calculations but not for the range. Results for individual samples are presented in Table S6.

	DF	Median	Range		DF	Median	Range
PFSAs				***PFCAs***			
PFPrS (3)	31	0.05	ND[a] - 13.7	PFBA (3)	97	8.09	ND - 858
PFBS (4)	100	0.53	0.06–280	PFPeA (4)	100	4.62	0.14–115
PFPeS (5)	48	0.05	ND - 3.73	PFHxA (5)	97	10.0	ND - 91.0
PFHxS (6)	90	1.31	ND - 18.1	PFHpA (6)	93	2.22	ND - 79.9
PFHpS (7)	55	0.07	ND - 2.74	PFOA (7)	100	6.13	1.32–178
PFOS (8)	100	2.32	0.18–298	PFNA (8)	100	2.70	0.26–85.6
PFNS (9)	21	0.06	ND - 0.35	PFDA (9)	79	0.96	ND - 53.0
PFDS (10)	69	0.24	ND - 30.0	PFUnDA (10)	66	1.48	ND - 40.2
PFECHS (8)	3	0.02	ND - 0.04	PFDoDA (11)	72	0.59	ND - 22.4
Cl-PFOS (8)	14	0.04	ND - 1.10	PFTrDA (12)	100	0.57	0.26–11.0
				PFTeDA (13)	90	0.97	ND - 13.0
Neutral PFASs				PFHxDA (15)	14	1.61	ND - 3.51
FOSA (8)	59	0.04	ND - 1.63				
MeFOSA (8)	24	0.11	ND - 2.37	***Fluorotelomer acids***			
EtFOSA (8)	3	0.14	ND - 0.51	4:2 FTSA (4)	72	0.28	ND - 10.4
4:2 FTOH (4)	3	3.61	ND - 12.0	6:2 FTSA (6)	52	42.7	ND - 2800
6:2 FTOH (6)	100	69.0	7.78–2410	8:2 FTSA (8)	90	0.78	ND - 14.4
8:2 FTOH (8)	100	18.8	12.5–2140	6:2 FTCA (6)	52	1.87	ND - 81.7
10:2 FTOH (10)	66	7.29	ND - 3520	8:2 FTCA (8)	38	2.08	ND - 44.2
MeFOSE (8)	24	2.26	ND - 122	10:2 FTCA (10)	7	0.52	ND - 10.3
EtFOSE (8)	7	2.47	ND - 28.9				
6:2 FTAcr (6)	10	0.23	ND - 67.1	∑PFCAs	100	37.1	5.13–884
8:2 FTAcr (8)	10	0.28	ND - 1.27	∑PFSAs	100	8.25	0.76–339
8:2 FTMAcr (8)	17	0.24	ND - 1.11	∑PFASs	100	505	32.2–8500

[a] ND = not-detected.

Table 2
Detection frequencies (DF, %), median and range for PFAS concentrations (ng/g) in indoor dust from childcare centers (n = 28). 10:2 FTCA, 4:2 FTOH, and 8:2 FTMAcr were not detected. Non-detect values were replaced by MDL/sqrt (2) for median calculations but not for the range. Results for individual samples are presented in Table S7.

	DF	Median	Range		DF	Median	Range
PFSAs				***PFCAs***			
PFPrS (3)	11	0.05	ND[a] - 0.24	PFBA (3)	96	4.54	ND - 326
PFBS (4)	100	0.53	0.19–185	PFPeA (4)	100	1.38	0.28–5.89
PFPeS (5)	50	0.06	ND - 0.58	PFHxA (5)	100	4.57	1.10–39.3
PFHxS (6)	75	1.35	ND - 11.9	PFHpA (6)	100	2.07	0.47–21.2
PFHpS (7)	75	0.08	ND - 0.60	PFOA (7)	100	4.92	1.40–26.6
PFOS (8)	100	4.64	0.38–44.2	PFNA (8)	100	3.19	0.87–17.2
PFNS (9)	11	0.09	ND - 0.49	PFDA (9)	93	1.07	ND - 20.1
PFDS (10)	89	1.35	ND - 56.7	PFUnDA (10)	86	2.20	ND - 10.9
PFECHS (8)	11	0.02	ND - 2.53	PFDoDA (11)	93	1.30	ND - 17.1
Cl-PFOS (8)	7	0.04	ND - 0.46	PFTrDA (12)	100	1.00	0.38–5.72
				PFTeDA (13)	100	1.62	0.62–12.2
Neutral PFASs				PFHxDA (15)	18	2.02	ND - 9.66
FOSA (8)	68	0.05	ND - 0.40				
MeFOSA (8)	4	0.11	ND - 0.72				
EtFOSA (8)	4	0.14	ND - 0.34	***Fluorotelomer acids***			
6:2 FTOH (6)	100	88.2	21.6–571	4:2 FTSA (4)	57	1.12	ND - 39.6
8:2 FTOH (8)	100	32.1	13.6–297	6:2 FTSA (6)	54	203	ND - 5230
10:2 FTOH (10)	89	28.0	ND - 356	8:2 FTSA (8)	96	1.36	ND - 10.7
MeFOSE (8)	50	3.03	ND - 123	6:2 FTCA (6)	36	1.99	ND - 189
EtFOSE (8)	54	4.14	ND - 98.0	8:2 FTCA (8)	14	3.23	ND - 37.6
6:2 FTAcr (6)	11	0.23	ND - 7.34				
8:2 FTAcr (8)	11	0.28	ND - 22.8	∑PFCAs	100	35.7	8.37–386
10:2 FTAcr (10)	4	0.29	ND - 5.57	∑PFSAs	100	9.35	1.29–190
6:2 FTMAcr (6)	7	0.22	ND - 0.40	∑PFASs	100	572	46.9–6470

[a] ND = not-detected.

out, or imported from countries where they are still produced (Xie et al., 2013; Kim et al., 2015; Gewurtz et al., 2018).

Giovanoulis et al. reported PFOA, PFNA, and PFOS concentrations in the Swedish daycare dust collected in 2018, comparable to those reported here (Giovanoulis et al., 2019). However, concentrations for long-chain PFASs (fluorinated C ≥ 7) were higher in daycare dust from the U.S. (2000–2001), United Kingdom (2007–2009), and Sweden (2006–2007) (Strynar and Lindstrom, 2008; Bjorklund et al., 2009; Goosey and Harrad, 2011), probably because samples were collected 10–20 years ago. Additionally, compared to carpet dust samples from California childcare centers from 2010 to 2011 (California Environmental Protection Agency, 2012), we found higher concentrations for C_4–C_6 PFAAs, but lower or comparable concentrations for C_8–C_{10} PFAAs, indicative of a market shift from long-chain to short-chain PFASs in the last decade.

3.3. Fluorotelomer acids

All FTSAs and FTCAs were found in our carpet samples with detection frequencies greater than 50%, except for 8:2 FTCA (DF: 38%) and 10:2 FTCA (7%) (Table 1). 6:2 FTSA was detected in these samples with a median concentration of 42.7 ng/g. Herzke et al. also observed 6:2 FTSA at a concentration of 1.35 μg/m^2 in a Norwegian carpet sample (Herzke et al., 2012). This is the first report of FTCAs in carpet samples. The abundance of fluorotelomer acids observed in our carpet samples is consistent with the fact that, following the phase-out of PFOA and PFOS production by electrochemical fluorination, the majority of manufacturers have switched to telomerization processes to synthesize PFAS products (Buck et al., 2011; Washington State Departments of Ecology and Health, 2019).

All three FTSAs were detected in ≥50% of dust samples, while FTCAs detection frequencies were lower than 40% (Table 2). 6:2 FTSA was the most abundant fluorotelomer acid, followed by 8:2 FTSA and 4:2 FTSA. In contrast to our study, 6:2 FTSA was rarely detected in two recent studies of dust from children's bedrooms in Finland and daycare facilities in Sweden (Winkens et al., 2018; Giovanoulis et al., 2019), and lower 6:2 FTSA levels were reported for house dust from Canada, and some European and Asian countries (Eriksson and Kärrman, 2015). The compositional differences in samples between Europe and North America and the influence of flooring materials on indoor PFAS contamination have been well-documented by numerous studies (Jian et al., 2017). Additionally, the outstanding 6:2 FTSA concentrations in dust are not completely unexpected considering that 6:2 FTSA has been recently employed as a PFOS alternative (Buck et al., 2011; National Associaton for Surface Finishing, 2019) and that indoor dust levels for PFOS were historically high (Strynar and Lindstrom, 2008). This compound should be included in the list of target chemicals in the future.

3.4. Neutral PFASs

Neutral PFASs were generally detected in ≤40% of carpet samples, except for FOSA and three FTOHs (i.e., 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH), which had higher detection frequencies (see Table 1). FTOHs, and in particular 6:2 FTOH, were the most abundant PFAS in the carpets. 6:2 FTOH median concentration of 69.0 ng/g was higher than those of ∑PFCAs and ∑PFSAs. Previous studies confirmed the use of FTOHs in carpet protection products (Dinglasan-Panlilio and Mabury, 2006), and also observed the prevalence of FTOHs in carpets (Herzke et al., 2012; Kotthoff et al., 2015; Liu et al., 2015; Vestergren et al., 2015). Our FTOH results were comparable to those reported for several US carpets (Liu et al., 2015), but higher than the 6:2 FTOH and 8:2 FTOH levels in Norwegian carpets (Herzke et al., 2012; Vestergren et al., 2015). Herzke et al. reported an average 6:2 FTOH/8:2 FTOH ratio of 0.68 in carpets purchased in Norway during 2006–2009 (Herzke et al., 2012), which is significantly lower than ours (median ratio = 3.9). This difference suggests that current carpet stain- and soil-repellents tend to contain higher levels of shorter-chain FTOH and/or that there are regional differences in the composition of products.

The most frequently detected neutral PFASs in dust samples

were 6:2 FTOH (median: 88.2 ng/g), 8:2 FTOH (32.1 ng/g), and 10:2 FTOH (28.0 ng/g) (Table 2). As expected, the median concentrations of neutral PFASs with 8–10 fluorinated carbons, e.g. 8:2 FTOH, 10:2 FTOH, EtFOSE, and FOSA, in dust samples were generally lower than those reported for daycare dust collected before 2010, while the median dust concentration for 6:2 FTOH was higher than in previous studies (Strynar and Lindstrom, 2008; Goosey and Harrad, 2011). This trend is consistent with the shift from long-chain PFASs to short-chain alternatives. Giovanoulis et al. measured FTOHs in Swedish daycare dust in 2018 and reported medians for 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH of 4.1, 18.3, and 12.4 ng/g, respectively, which are lower than our measurements (Giovanoulis et al., 2019).

3.5. Associations between carpet and dust

The PFAS compositional patterns were generally similar between carpets and the dust, except carpets had significantly greater contributions of C_5–C_8 PFCAs than dust (all $p < 0.05$), while 6:2 FTSA, 8:2 FTSA, MeFOSE, and EtFOSE were more abundant in dust (Fig. 1 and Table S8). These differences suggest that the C_5–C_8 PFCAs may be present in carpet as a result of PFAS-containing stain- and soil-repellents, while FTSAs and the FOSEs in dust may be originating from other indoor sources (Karásková et al., 2016). Additionally, chemical-specific variations in sorption to carpet and/or dust may also play a role in these differences.

In general, close associations existed among PFASs within each sub-category in both carpets and dust (see Tables S9 and S10). For example, in carpets, all individual PFCAs were strongly correlated with each other with few exceptions (all $p < 0.05$), indicating that they may originate from similar technical mixtures of different chain lengths (Buck et al., 2011; Winkens et al., 2018). Significant associations of PFCAs with FTOHs and FTSAs were frequently observed in both media, consistent with the fact that FTOHs and FTSAs can degrade to PFCAs (Ellis et al., 2004; Wang et al., 2011, 2014; Yang et al., 2014). Moreover, MeFOSE and EtFOSE, both PFOS precursors, were strongly correlated with PFOS in dust (both $p < 0.01$).

The relationship between PFASs in paired carpet and dust samples was further explored using a generalized linear regression model (Hoffman et al., 2015). For this analysis, carpet concentrations were categorized into tertiles [i.e., low or reference (n = 9), medium (n = 10), and high (n = 9)], and tested as predictors of dust PFASs. In general, PFASs levels in carpets were closely associated with their concentrations in dust (Table 3). The concentration of PFAS in dust from childcare centers with high ∑PFASs in their carpets was on average 5.21 times (95% CI: 1.98, 13.7) greater than dust from centers with the low PFAS carpet levels. These results agree well with the highly significant correlation for ∑PFASs in carpets and dust (Kendall's tau = 0.38, $p = 0.004$), suggesting that carpets can be both a source and a sink for PFASs. An in depth analysis of the composition of dust using techniques like scanning electron microscope (SEM) would be helpful in shedding some light on this issue but they were beyond the scope of this study. The PFAS partitioning between carpets and dust potentially involves transfers in both directions, although our data can't identify which transfer direction is of higher relative importance. The partitioning processes can also change with time and environmental conditions, including human activities, making the concentrations of PFAS in carpets not at steady state. Future studies should look further to determine if the presence of PFASs in carpets is driven by their addition during manufacturing or their accumulation from various indoor sources.

Although dust and carpet pairs were not studied together before, carpeted floors were suspected to be associated with elevated PFAS contamination in indoor environment (Gewurtz et al., 2009; Beesoon et al., 2012; Fraser et al., 2012; Karásková et al., 2016). For example, PFOS, PFOA and PFHxS levels in

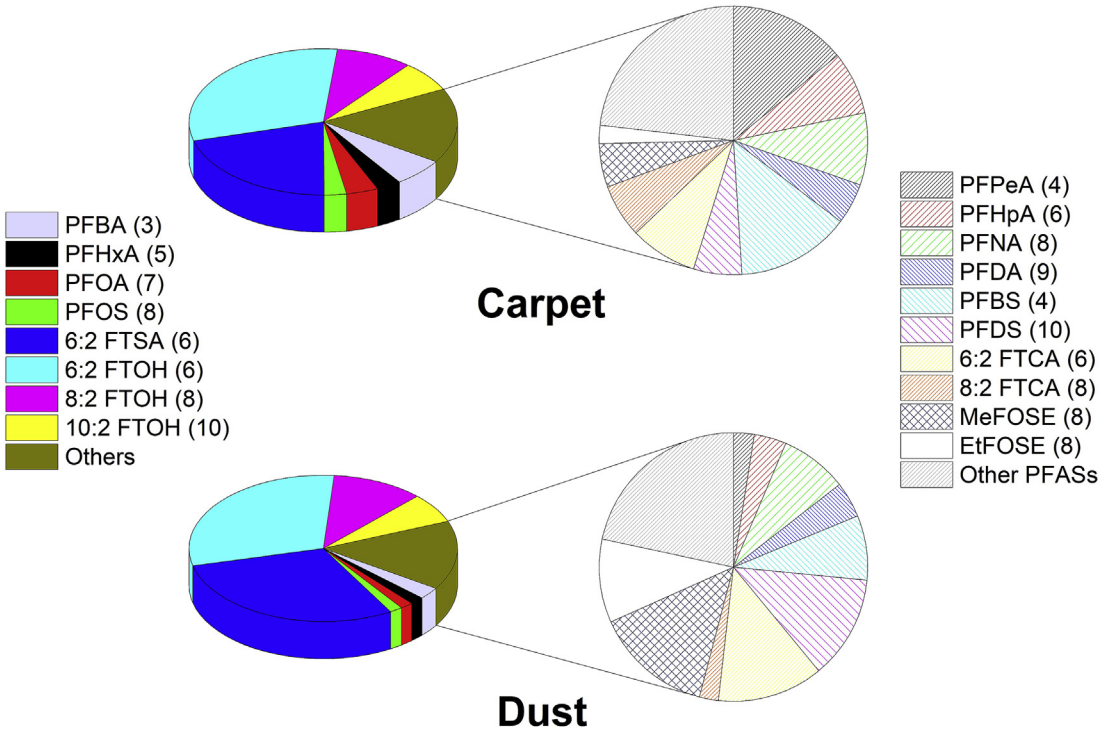

Fig. 1. Compositional profile of PFASs in carpets and dust collected from childcare centers. The data represent the mean composition of each PFAS compound. Values in parentheses represent the numbers of fluorinated carbons.

Table 3
Regression analyses for carpet PFAS levels as predictors of dust PFAS concentrations. Analyses were restricted to PFAS compounds with detection frequencies ≥68% in both media. Values in bold indicate a significant difference from the reference group. Dust levels for carpets in the lowest of the three tertiles (low carpet levels, n = 9) were treated as reference.

	Low carpet levels (n = 9)	Mid carpet levels (n = 10)		High carpet levels (n = 9)	
		Coefficient (95% CI)	*p*-Value	Coefficient (95% CI)	*p*-Value
PFBA	Reference	0.86 (0.30, 2.42)	0.765	**3.27 (1.13, 9.47)**	**0.031**
PFPeA	Reference	1.77 (0.90, 3.45)	0.092	**2.44 (1.23, 4.85)**	**0.013**
PFHxA	Reference	**2.91 (1.30, 6.51)**	**0.011**	**2.54 (1.11, 5.80)**	**0.028**
PFHpA	Reference	0.94 (0.42, 2.11)	0.877	**3.05 (1.33, 7.00)**	**0.010**
PFOA	Reference	1.42 (0.76, 2.63)	0.258	**3.88 (2.06, 7.34)**	**< 0.001**
PFNA	Reference	**2.14 (1.08, 4.25)**	**0.031**	**2.84 (1.40, 5.73)**	**0.005**
PFDA	Reference	1.01 (0.29, 3.47)	0.988	1.57 (0.44, 5.56)	0.472
PFDoDA	Reference	1.28 (0.53, 3.08)	0.572	**4.07 (1.65, 10.1)**	**0.004**
PFTrDA	Reference	0.90 (0.55, 1.47)	0.659	**2.79 (1.68, 4.64)**	**< 0.001**
PFTeDA	Reference	1.60 (0.92, 2.76)	0.092	**3.75 (2.13, 6.58)**	**< 0.001**
PFBS	Reference	1.44 (0.35, 5.86)	0.602	**5.46 (1.29, 23.1)**	**0.023**
PFHxS	Reference	0.92 (0.49, 1.74)	0.791	1.83 (0.95, 3.53)	0.068
PFOS	Reference	**2.81 (1.16, 6.81)**	**0.024**	**6.22 (2.51, 15.4)**	**< 0.001**
PFDS	Reference	2.85 (0.76, 10.8)	0.116	**11.8 (3.04, 46.2)**	**0.001**
8:2 FTSA	Reference	1.04 (0.44, 2.44)	0.925	**3.59 (1.50, 8.60)**	**0.006**
6:2 FTOH	Reference	1.56 (0.93, 2.61)	0.090	**6.33 (3.73, 10.8)**	**< 0.001**
8:2 FTOH	Reference	1.16 (0.58, 2.31)	0.658	**2.77 (1.37, 5.61)**	**0.006**
∑PFASs	Reference	1.95 (0.76, 5.01)	0.158	**5.21 (1.98, 13.7)**	**0.002**

Canadian residential dust exhibited significant correlations to the percent carpeting (all $p < 0.01$) (Kubwabo et al., 2005). Further research is needed to determine the contribution of fluorinated carpet stain- and soil-repellents to PFAS levels in carpet and dust.

3.6. PFAA exposure for children via dust ingestion

Using our dust PFAS data and the parameters described in Table S5 and in previous studies (USEPA, 2008; Gebbink et al., 2015), we estimated the daily PFAA intake via dust ingestion by children ages 2–6 who attend childcare centers. We developed estimates for both direct intake and indirect intake (via biotransformation of PFAA precursors).

The total estimated PFAA intakes via dust ingestion were 0.023, 0.096, and 1.9 ng/kg bw/day for the low-, intermediate-, and high-exposure scenarios, respectively (Table S11). PFCAs and PFSAs with significant contributions were PFBA, PFHxA, PFOA, PFOS, PFNA, and PFBS. These data show that PFASs of emerging concern, particularly short-chain PFCAs, are important contributors to children's PFAS exposure via dust ingestion (Fig. 2). Exposure to these alternative PFASs is associated with a variety of adverse toxicological outcomes including birth defect, impaired immunity, endocrine disruption, transcriptional effects, hepatic and neurodevelopmental toxicities (Gomis et al., 2018; Gao et al., 2019; Huang et al., 2019).

In addition to the direct route, PFAA precursors contribute to total PFAA intake indirectly via *in vivo* biotransformation. For example, FTOHs and fluorotelomer acids can undergo biodegradation and form PFCAs (Wang et al., 2011; Butt et al., 2014; Rand and Mabury, 2017). Fluorotelomer (meth)acrylates are subjected to *in vivo* biotransformation, generating FTCAs and FTOHs, which can be further biotransformed to PFCAs (Butt et al., 2010). Additionally, previous *in vitro* and *in vivo* studies have confirmed that the FASAs and FASEs we targeted are precursors to PFOS (Tomy et al., 2004; Xu et al., 2004; Xie et al., 2009).

The major contribution to indirect PFAA intake was from 6:2 FTSA, 6:2 FTOH, MeFOSE, and EtFOSE. MeFOSE and EtFOSE contributed 24–54% of the total estimated PFOS exposure from dust ingestion (Table S11). Winkens et al. reported that the contribution by metabolism of EtFOSE (median percentage to total PFOS intake: 56%) was greater than the direct exposure for PFOS via

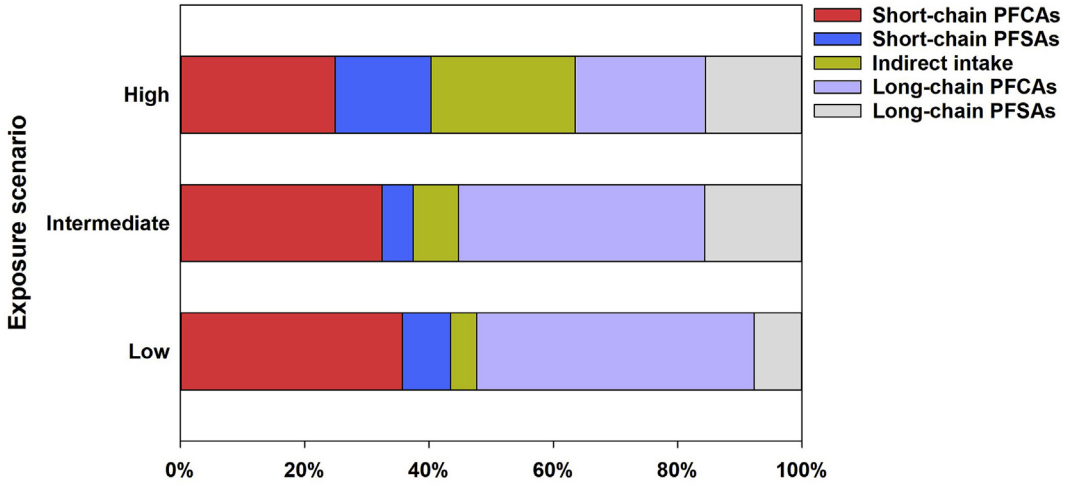

Fig. 2. Percent of total PFAA intake via dust ingestion of PFCAs, PFSAs, as well as PFAAs generated via biotransformation of precursors, by children at ages 2–6 via dust ingestion. Short- and long-chain PFAAs, respectively, have ≤6 and ≥ 7 fluorinated carbons.

dust sampled from children's bedrooms in Finland (Winkens et al., 2018). 6:2 FTOH and 6:2 FTSA can both biodegrade to PFCAs (Wang et al., 2011; Butt et al., 2014), and their aggregated contributions to total intake of C_4–C_7 PFCAs in the intermediate and high-exposure scenarios were 8.9% and 37% respectively. Though indirect routes accounted for only 4.3% and 7.3% of total PFAA intake in the low- and intermediate-exposure scenarios, respectively, their contribution reached 23% in the high-exposure scenario, mainly due to the outstanding contributions by 6:2 FTSA and FOSEs (Table S11). We suspect that these values are underestimated since other PFAA precursors not monitored in the present study, e.g. polyfluoroalkyl phosphate esters, can also contribute to the PFAA exposure (De Silva et al., 2012; Eriksson and Kärrman, 2015).

4. Implications

In response to concerns about potential health risks associated with PFAS exposure, government agencies have developed reference doses or acceptable daily intake levels for several PFASs. The US EPA uses oral non-cancer reference doses of 20 ng/kg bw/day for both PFOA and PFOS (USEPA, 2017) and has proposed a draft chronic reference dose of 10,000 ng/kg bw/day for PFBS (USEPA, 2018). The minimal risk levels reported by the Agency for Toxic Substances and Disease Registry were 3, 2, 20, and 3 ng/kg bw/day for PFOA, PFOS, PFHxS, and PFNA, respectively (Agency for Toxic Substances and Disease Registry, 2018). The tolerable daily intake of PFOA and PFOS suggested by the European Food Safety Authority were 0.8 and 1.8 ng/kg bw/day, respectively (European Food Safety Authority, 2008; Knutsen et al., 2018). Though our results were below these reference doses, dust ingestion represents only one of the numerous PFAS exposure routes. Given the ubiquitous distribution of PFASs around us, other exposure pathways, e.g. food and drinking water intake, and air inhalation, will also contribute to PFAS build-up in humans (Jian et al., 2017; Winkens et al., 2018; Hu et al., 2019). For example, a previous study using air and dust from children's bedrooms reported that the PFAA intake from air inhalation was only slightly lower than that from the dust ingestion (Winkens et al., 2018). For general adult population, the relative contributions of dust ingestion to the total PFAA intake through different exposure pathways (i.e. diet, water, air, and dust) ranged from 4.8% to 40.3% (Gebbink et al., 2015). Additionally, other PFASs that were not monitored in this study may increase the daily PFAA intake.

CRediT authorship contribution statement

Yan Wu: Conceptualization, Methodology, Formal analysis, Investigation, Writing - original draft. **Kevin Romanak:** Methodology, Formal analysis. **Tom Bruton:** Writing - review & editing, Conceptualization. **Arlene Blum:** Writing - review & editing, Conceptualization. **Marta Venier:** Supervision, Project administration, Funding acquisition, Writing - review & editing.

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Appendix A. Supplementary data

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References

Agency for Toxic Substances and Disease Registry, 2018. PFAs toxicological profile key messages. Available at: https://www.atsdr.cdc.gov/docs/PFAS_Public_KeyMessages_June20_Final-508.pdf. (Accessed 4 September 2019).
Antweiler, R.C., 2015. Evaluation of statistical treatments of left-censored environmental data using coincident uncensored data sets. II. Group comparisons. Environ. Sci. Technol. 49, 13439–13446.
Antweiler, R.C., Taylor, H.E., 2008. Evaluation of statistical treatments of left-censored environmental data using coincident uncensored data sets: I. Summary statistics. Environ. Sci. Technol. 42, 3732–3738.
Beesoon, S., Genuis, S.J., Benskin, J.P., Martin, J.W., 2012. Exceptionally high serum concentrations of perfluorohexanesulfonate in a Canadian family are linked to home carpet treatment applications. Environ. Sci. Technol. 46, 12960–12967.
Bjorklund, J.A., Thuresson, K., De Wit, C.A., 2009. Perfluoroalkyl compounds (PFCs) in indoor dust: concentrations, human exposure estimates, and sources. Environ. Sci. Technol. 43, 2276–2281.
Braun, J.M., 2017. Early-life exposure to EDCs: role in childhood obesity and neurodevelopment. Nat. Rev. Endocrinol. 13, 161–173.
Buck, R.C., Franklin, J., Berger, U., Conder, J.M., Cousins, I.T., de Voogt, P., Jensen, A.A., Kannan, K., Mabury, S.A., van Leeuwen, S.P., 2011. Perfluoroalkyl and polyfluoroalkyl substances in the environment: terminology, classification, and origins. Integrated Environ. Assess. Manag. 7, 513–541.
Butt, C.M., Muir, D.C., Mabury, S.A., 2010. Biotransformation of the 8:2 fluorotelomer acrylate in rainbow trout. 1. In vivo dietary exposure. Environ. Toxicol. Chem. 29, 2726–2735.
Butt, C.M., Muir, D.C., Mabury, S.A., 2014. Biotransformation pathways of fluorotelomer-based polyfluoroalkyl substances: a review. Environ. Toxicol. Chem. 33, 243–267.
California Environmental Protection Agency, 2012. Environmental exposures in early childhood education environments. Available at: https://ww3.arb.ca.gov/research/apr/past/08-305.pdf. (Accessed 30 July 2019).
Stockholm Convention. All POPs listed in the Stockholm convention. Available at: http://chm.pops.int/TheConvention/ThePOPs/tabid/2509/Default.aspx. (Accessed 29 July 2019).
D'Eon, J.C., Mabury, S.A., 2011. Exploring indirect sources of human exposure to perfluoroalkyl carboxylates (PFCAs): evaluating uptake, elimination, and biotransformation of polyfluoroalkyl phosphate esters (PAPs) in the rat. Environ. Health Perspect. 119, 344–350.
De Silva, A.O., Allard, C.N., Spencer, C., Webster, G.M., Shoeib, M., 2012. Phosphorus-containing fluorinated organics: polyfluoroalkyl phosphoric acid diesters (diPAPs), perfluorophosphonates (PFPAs), and perfluorophosphinates (PFPIAs) in residential indoor dust. Environ. Sci. Technol. 46, 12575–12582.
Dinglasan-Panlilio, M.J., Mabury, S.A., 2006. Significant residual fluorinated alcohols present in various fluorinated materials. Environ. Sci. Technol. 40, 1447–1453.
Egeghy, P.P., Lorber, M., 2011. An assessment of the exposure of Americans to perfluorooctane sulfonate: a comparison of estimated intake with values inferred from NHANES data. J. Expo. Sci. Environ. Epidemiol. 21, 150–168.
Ellis, D.A., Martin, J.W., De Silva, A.O., Mabury, S.A., Hurley, M.D., Sulbaek Andersen, M.P., Wallington, T.J., 2004. Degradation of fluorotelomer alcohols: a likely atmospheric source of perfluorinated carboxylic acids. Environ. Sci. Technol. 38, 3316–3321.
Eriksson, U., Kärrman, A., 2015. World-wide indoor exposure to polyfluoroalkyl phosphate esters (PAPs) and other PFASs in household dust. Environ. Sci. Technol. 49, 14503–14511.
European Food Safety Authority, 2008. Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts scientific opinion of the panel on contaminants in the food chain. EFSA Journal 6.
Fraser, A.J., Webster, T.F., Watkins, D.J., Nelson, J.W., Stapleton, H.M., Calafat, A.M., Kato, K., Shoeib, M., Vieira, V.M., McClean, M.D., 2012. Polyfluorinated compounds in serum linked to indoor air in office environments. Environ. Sci. Technol. 46, 1209–1215.
Gao, K., Zhuang, T., Liu, X., Fu, J., Zhang, J., Fu, J., Wang, L., Zhang, A., Liang, Y., Song, M., Jiang, G., 2019. Prenatal exposure to per- and polyfluoroalkyl substances (PFASs) and association between the placental transfer efficiencies and dissociation constant of serum proteins-PFAS complexes. Environ. Sci. Technol. 53, 6529–6538.
Gebbink, W.A., Berger, U., Cousins, I.T., 2015. Estimating human exposure to PFOS isomers and PFCA homologues: the relative importance of direct and indirect (precursor) exposure. Environ. Int. 74, 160–169.
Gewurtz, S.B., Bhavsar, S.P., Crozier, P.W., Diamond, M.L., Helm, P.A., Marvin, C.H., Reiner, E.J., 2009. Perfluoroalkyl contaminants in window film: indoor/outdoor, urban/rural, and winter/summer contamination and assessment of carpet as a possible source. Environ. Sci. Technol. 43, 7317–7323.
Gewurtz, S.B., Martin, P.A., Letcher, R.J., Burgess, N.M., Champoux, L., Elliott, J.E., Idrissi, A., 2018. Perfluoroalkyl acids in European starling eggs indicate landfill and urban influences in Canadian terrestrial environments. Environ. Sci. Technol. 52, 5571–5580.
Giovanoulis, G., Nguyen, M.A., Arwidsson, M., Langer, S., Vestergren, R., Lagerqvist, A., 2019. Reduction of hazardous chemicals in Swedish preschool dust through article substitution actions. Environ. Int. 130, 104921.
Gomis, M.I., Vestergren, R., Borg, D., Cousins, I.T., 2018. Comparing the toxic potency *in vivo* of long-chain perfluoroalkyl acids and fluorinated alternatives. Environ. Int. 113, 1–9.

Goosey, E., Harrad, S., 2011. Perfluoroalkyl compounds in dust from Asian, Australian, European, and North American homes and UK cars, classrooms, and offices. Environ. Int. 37, 86–92.
Harris, M.H., Rifas-Shiman, S.L., Calafat, A.M., Ye, X., Mora, A.M., Webster, T.F., Oken, E., Sagiv, S.K., 2017. Predictors of per- and polyfluoroalkyl substance (PFAS) plasma concentrations in 6-10 year old American children. Environ. Sci. Technol. 51, 5193–5204.
Herzke, D., Olsson, E., Posner, S., 2012. Perfluoroalkyl and polyfluoroalkyl substances (PFASs) in consumer products in Norway - a pilot study. Chemosphere 88, 980–987.
Hoffman, K., Garantziotis, S., Birnbaum, L.S., Stapleton, H.M., 2015. Monitoring indoor exposure to organophosphate flame retardants: hand wipes and house dust. Environ. Health Perspect. 123, 160–165.
Hu, X.C., Tokranov, A.K., Liddie, J., Zhang, X., Grandjean, P., Hart, J.E., Laden, F., Sun, Q., Yeung, L.W.Y., Sunderland, E.M., 2019. Tap water contributions to plasma concentrations of poly- and perfluoroalkyl substances (PFAS) in a nationwide prospective cohort of U.S. women. Environ. Health Perspect. 127, 67006.
Huang, R., Chen, Q., Zhang, L., Luo, K., Chen, L., Zhao, S., Feng, L., Zhang, J., 2019. Prenatal exposure to perfluoroalkyl and polyfluoroalkyl substances and the risk of hypertensive disorders of pregnancy. Environ. Health 18, 5.
Jian, J.M., Guo, Y., Zeng, L., Liang-Ying, L., Lu, X., Wang, F., Zeng, E.Y., 2017. Global distribution of perfluorochemicals (PFCs) in potential human exposure source-A review. Environ. Int. 108, 51–62.
Karásková, P., Venier, M., Melymuk, L., Bečanová, J., Vojta, Š., Prokeš, R., Diamond, M.L., Klánová, J., 2016. Perfluorinated alkyl substances (PFASs) in household dust in Central Europe and North America. Environ. Int. 94, 315–324.
Kim, M., Li, L.Y., Grace, J.R., Benskin, J.P., Ikonomou, M.G., 2015. Compositional effects on leaching of stain-guarded (perfluoroalkyl and polyfluoroalkyl substance-treated) carpet in landfill leachate. Environ. Sci. Technol. 49, 6564–6573.
Knobeloch, L., Imm, P., Anderson, H., 2012. Perfluoroalkyl chemicals in vacuum cleaner dust from 39 Wisconsin homes. Chemosphere 88, 779–783.
Knutsen, H.K., Alexander, J., Barregård, L., Bignami, M., Brüschweiler, B., Ceccatelli, S., Cottrill, B., Dinovi, M., Edler, L., Grasl-Kraupp, B., Hogstrand, C., Hoogenboom, L., Nebbia, C.S., Oswald, I.P., Petersen, A., Rose, M., Roudot, A.C., Vleminckx, C., Vollmer, G., Wallace, H., Bodin, L., Cravedi, J.P., Halldorsson, T.I., Haug, L.S., Johansson, N., van Loveren, H., Gergelova, P., Mackay, K., Levorato, S., van Manen, M., Schwerdtle, T., 2018. Risk to human health related to the presence of perfluorooctane sulfonic acid and perfluorooctanoic acid in food. EFSA Journal 16.
Kotthoff, M., Muller, J., Jurling, H., Schlummer, M., Fiedler, D., 2015. Perfluoroalkyl and polyfluoroalkyl substances in consumer products. Environ. Sci. Pollut. Res. Int. 22, 14546–14559.
Kubwabo, C., Stewart, B., Zhu, J., Marro, L., 2005. Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada. J. Environ. Monit. 7, 1074–1078.
Lindstrom, A.B., Strynar, M.J., Libelo, E.L., 2011. Polyfluorinated compounds: past, present, and future. Environ. Sci. Technol. 45, 7954–7961.
Liu, X., Guo, Z., Krebs, K.A., Pope, R.H., Roache, N.F., 2014. Concentrations and trends of perfluorinated chemicals in potential indoor sources from 2007 through 2011 in the US. Chemosphere 98, 51–57.
Liu, X., Guo, Z., Folk, E.E., Roache, N.F., 2015. Determination of fluorotelomer alcohols in selected consumer products and preliminary investigation of their fate in the indoor environment. Chemosphere 129, 81–86.
National Associaton for Surface Finishing, 2019. 6:2 Fluorotelomer sulfonate (6:2 FTS): toxicology at a glance. Available at: https://nasf.org/wp-content/uploads/2019/04/Summary-of-Toxicology-Studies-on-6-2-FTS-and-Detailed-Technical-Support-Documents.pdf. (Accessed 14 September 2019).
Rand, A.A., Mabury, S.A., 2017. Is there a human health risk associated with indirect exposure to perfluoroalkyl carboxylates (PFCAs)? Toxicology 375, 28–36.
Rappazzo, K.M., Coffman, E., Hines, E.P., 2017. Exposure to perfluorinated alkyl substances and health outcomes in children: a systematic review of the epidemiologic literature. Int. J. Environ. Res. Publ. Health 14.
Reiner, J.L., Blaine, A.C., Higgins, C.P., Huset, C., Jenkins, T.M., Kwadijk, C.J., Lange, C.C., Muir, D.C., Reagen, W.K., Rich, C., Small, J.M., Strynar, M.J., Washington, J.W., Yoo, H., Keller, J.M., 2015. Polyfluorinated substances in abiotic standard reference materials. Anal. Bioanal. Chem. 407, 2975–2983.
Stapleton, H.M., Misenheimer, J., Hoffman, K., Webster, T.F., 2014. Flame retardant associations between children's handwipes and house dust. Chemosphere 116, 54–60.
Strynar, M.J., Lindstrom, A.B., 2008. Perfluorinated compounds in house dust from Ohio and North Carolina, USA. Environ. Sci. Technol. 42, 3751–3756.
Tomy, G.T., Tittlemier, S.A., Palace, V.P., Budakowski, W.R., Braekevelt, E., Brinkworth, L., Friesen, K., 2004. Biotransformation of *N*-ethyl perfluorooctanesulfonamide by rainbow trout (*Onchorhynchus mykiss*) liver microsomes. Environ. Sci. Technol. 38, 758–762.
Trudel, D., Horowitz, L., Wormuth, M., Scheringer, M., Cousins, I.T., Hungerbuhler, K., 2008. Estimating consumer exposure to PFOS and PFOA. Risk Anal. 28, 251–269.
USEPA, 2000. EPA and 3M announce phase out of PFOS. Available at: https://archive.epa.gov/epapages/newsroom_archive/newsreleases/33aa946e6cb11f35852568e1005246b4.html. (Accessed 29 July 2019).
USEPA, 2006. 2010/15 PFOA stewardship program. Available at: https://www.epa.gov/sites/production/files/2015-10/documents/pfoaguidance.pdf. (Accessed 29 July 2019).
USEPA, 2008. Child-specific Exposure Factors Handbook. Available at: http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=484738. (Accessed 27 July 2019).
USEPA, 2009. Perflourocarboxylic acid content in 116 articles of commerce. Available at: https://cfpub.epa.gov/si/si_public_record_report.cfm?Lab=NRMRL&dirEntryId=206124. (Accessed 3 July 2019).
USEPA, 2014. EPA's Non-CBI summary tables for 2014 company progress reports. accessed March 30, 2020Available at: https://www.epa.gov/sites/production/files/2016-07/documents/2014_pfoa_stewardship_summary_table.pdf.
USEPA, 2017. Technical fact sheet – perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). Available at: https://www.epa.gov/sites/production/files/2017-12/documents/ffrrofactsheet_contaminants_pfos_pfoa_11-20-17_508_0.pdf. (Accessed 4 September 2019).
USEPA, 2018. Fact sheet: draft toxicity assessments for GenX chemicals and PFBS. Available at: https://www.epa.gov/sites/production/files/2018-11/documents/factsheet_pfbs-genx-toxicity_values_11.14.2018.pdf. (Accessed 14 September 2019).
Vestergren, R., Berger, U., Glynn, A., Cousins, I.T., 2012. Dietary exposure to perfluoroalkyl acids for the Swedish population in 1999, 2005 and 2010. Environ. Int. 49, 120–127.
Vestergren, R., Herzke, D., Wang, T., Cousins, I.T., 2015. Are imported consumer products an important diffuse source of PFASs to the Norwegian environment? Environ. Pollut. 198, 223–230.
Wang, N., Liu, J., Buck, R.C., Korzeniowski, S.H., Wolstenholme, B.W., Folsom, P.W., Sulecki, L.M., 2011. 6:2 fluorotelomer sulfonate aerobic biotransformation in activated sludge of waste water treatment plants. Chemosphere 82, 853–858.
Wang, Z., Cousins, I.T., Scheringer, M., Buck, R.C., Hungerbuhler, K., 2014. Global emission inventories for C4-C14 perfluoroalkyl carboxylic acid (PFCA) homologues from 1951 to 2030, Part I: production and emissions from quantifiable sources. Environ. Int. 70, 62–75.
Washington State Departments of Ecology and Health, 2019. Per- and polyfluorinated alkyl substances chemical action plan (PFAS CAP) – 2019 updates (accessed March 30, 2020)Available at: https://www.ezview.wa.gov/Portals/_1962/Documents/PFAS/Chemistry-2019-PFAS-CAP.pdf.
Watkins, D.J., McClean, M.D., Fraser, A.J., Weinberg, J., Stapleton, H.M., Sjodin, A., Webster, T.F., 2011. Exposure to PBDEs in the office environment: evaluating the relationships between dust, handwipes, and serum. Environ. Health Perspect. 119, 1247–1252.
Winkens, K., Giovanoulis, G., Koponen, J., Vestergren, R., Berger, U., Karvonen, A.M., Pekkanen, J., Kiviranta, H., Cousins, I.T., 2018. Perfluoroalkyl acids and their precursors in floor dust of children's bedrooms - implications for indoor exposure. Environ. Int. 119, 493–502.
Xie, W., Wu, Q., Kania-Korwel, I., Tharappel, J.C., Telu, S., Coleman, M.C., Glauert, H.P., Kannan, K., Mariappan, S.V., Spitz, D.R., Weydert, J., Lehmler, H.J., 2009. Subacute exposure to *N*-ethyl perfluorooctanesulfonamidoethanol results in the formation of perfluorooctanesulfonate and alters superoxide dismutase activity in female rats. Arch. Toxicol. 83, 909–924.
Xie, S., Wang, T., Liu, S., Jones, K.C., Sweetman, A.J., Lu, Y., 2013. Industrial source identification and emission estimation of perfluorooctane sulfonate in China. Environ. Int. 52, 1–8.
Xu, L., Krenitsky, D.M., Seacat, A.M., Butenhoff, J.L., Anders, M.W., 2004. Biotransformation of *N*-ethyl-*N*-(2-hydroxyethyl)perfluorooctanesulfonamide by rat liver microsomes, cytosol, and slices and by expressed rat and human cytochromes P450. Chem. Res. Toxicol. 17, 767–775.
Yang, X., Huang, J., Zhang, K., Yu, G., Deng, S., Wang, B., 2014. Stability of 6:2 fluorotelomer sulfonate in advanced oxidation processes: degradation kinetics and pathway. Environ. Sci. Pollut. Res. Int. 21, 4634–4642.