Environment International 119 (2018) 493–502

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Perfluoroalkyl acids and their precursors in floor dust of children's bedrooms – Implications for indoor exposure

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ARTICLE INFO

Handling Editor: Heather Stapleton

Keywords:
Per- and polyfluoroalkyl substance (PFAS)
Polyfluoroalkyl phosphoric acid ester (PAP)
Fluorotelomer alcohol (FTOH)
Child exposure
Estimated daily intake (EDI)
Standard reference material (SRM 2585)

ABSTRACT

We analysed floor dust samples from 65 children's bedrooms in Finland collected in 2014/2015 for 62 different per- and polyfluoroalkyl substances (PFASs) with a simple and highly efficient method. Validation results from the analysis of standard reference material (SRM) 2585 were in good agreement with literature data, while 24 PFASs were quantified for the first time. In the dust samples from children's bedrooms, five perfluoroalkyl carboxylic acids (PFCAs) and perfluorooctane sulfonic acid (PFOS) were detected in more than half of the samples with the highest median concentration of 5.26 ng/g for perfluorooctanoic acid (PFOA). However, the dust samples were dominated by polyfluoroalkyl phosphoric acid esters (PAPs) and fluorotelomer alcohols (FTOHs) (highest medians: 53.9 ng/g for 6:2 diPAP and 45.7 ng/g for 8:2 FTOH). Several significant and strong correlations (up to $\rho = 0.95$) were found among different PFASs in dust as well as between PFASs in dust and air samples (previously published) from the same rooms. The logarithm of dust to air concentrations (log $K_{dust/air}$) plotted against the logarithm of the octanol-air partition coefficient (log K_{oa}) resulted in a significant linear regression line with $R^2 > 0.88$. Higher dust levels of PFOS were detected in rooms with plastic flooring material in comparison to wood ($p < 0.05$). Total estimated daily intakes via dust (EDI_{dust}) and air (EDI_{air}) of perfluoroalkyl acids (PFAA), including biotransformation of precursors to PFAAs, were calculated for 10.5-year-old children. The total EDI_{dust} for PFOA and PFOS were estimated to be 0.007 ng/kg bw/day and 0.006 ng/kg bw/day, respectively, in an intermediate exposure scenario. The sum of the total EDIs for all PFAAs was slightly higher for dust than air (0.027 and 0.019 ng/kg bw/day). Precursor biotransformation was generally important for total PFOS intake, while for the PFCAs, FTOH biotransformation was estimated to be important for air, but not for dust exposure.

1. Introduction

The group of per- and polyfluoroalkyl substances (PFASs) has many sub-classes with > 3000 substances (KEMI, 2016). Perfluoroalkane sulfonic acids (PFSAs) can be produced by electrochemical fluorination (ECF) and perfluoroalkyl carboxylic acids (PFCAs) can be produced by ECF or telomerisation. ECF produces PFASs of different chain lengths as impurities, as well as a mixture of linear and branched isomers, whereas in telomerisation production, PFASs with linear perfluoroalkyl chains are produced (Paul et al., 2009; Prevedouros et al., 2006). Regulatory constraints, like restrictions of several PFCAs and PFSAs (ECHA, 2013, 2015, 2017; US EPA, 2000, 2006), as well as voluntary phase out programs have led to a production shift from long-chain to short-chain perfluorinated substances (i.e. < C_6 for PFSAs and < C_8 for PFCAs) (Buck et al., 2011). Today, the majority of the western world production of PFASs is based on telomerisation, although the 3M Company continues to use ECF to manufacture short-chain (C4) fluorochemical products in Europe and the US (Buck et al., 2011; Ritter, 2010; Wang et al., 2014). The diversity of PFASs is constantly increasing (Wang et al., 2017) and large-scale production of long-chain perfluoroalkyl acids (PFAAs) by ECF is still ongoing in China, India and Russia (Wang et al., 2014).

PFASs have many indoor use areas; they are for example widely applied for their water and oil/stain repellent characteristics in textiles

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https://doi.org/10.1016/j.envint.2018.06.009
Received 23 February 2018; Received in revised form 8 June 2018; Accepted 8 June 2018
Available online 24 July 2018

(Berger and Herzke, 2006; Herzke et al., 2012; Kotthoff et al., 2015) and in food packaging (Begley et al., 2005) or for their spreading and wetting characteristics in floor treatment products (Kotthoff et al., 2015), in paints (Herzke et al., 2012; Ritter, 2010) and cosmetics (Fujii et al., 2013) and they even occur as production residuals in cookware (Begley et al., 2005; Herzke et al., 2012). During product use, particles containing the less volatile PFASs can e.g. be abraded from aging products, while the volatile and semi-volatile PFASs in products can also outgas and partition to particles in the surrounding air, which both can be deposited to dust. Therefore, PFASs have been detected in dust and air samples of indoor environments worldwide (e.g. Haug et al., 2011; Kim et al., 2012; Shoeib et al., 2005; Tian et al., 2016; Winkens et al., 2017a).

Dust is likely to represent a more important exposure pathway for children than adults due to children's higher exposure factors related to their body weights, such as hand-to-mouth frequency, object-to-mouth frequency and dust ingestion rate, but even because of their proximity to the floor while crawling and playing on the floor and thereby stirring up dust (US EPA, 2011; Winkens et al., 2017b; Wu et al., 2018). The reported estimated daily intakes (EDIs) for PFASs via dust ingestion in the literature reflect this, as they are higher for toddlers or children than for adults (Björklund et al., 2009; Ericson Jogsten et al., 2012; Shoeib et al., 2005; Tian et al., 2016; Zhang et al., 2010).

There are many studies that have measured PFASs in sieved and settled dust of various indoor environments, including cars, offices, homes and day care centres (Björklund et al., 2009; D'Hollander et al., 2010; Eriksson and Kärrman, 2015; Goosey and Harrad, 2011; Haug et al., 2011; Karásková et al., 2016; Shoeib et al., 2011). In our study, we present a recent dataset for a large number of floor dust samples (n = 65) and for a long list of PFAS analytes (n = 62). The floor dust samples were taken during 2014/2015 in children's bedrooms, a child-specific indoor microenvironment, in which they spend the majority of their time indoors while sleeping and playing. With this individual-based data on PFASs and the recorded bodyweights of the children, we calculate the individuals' estimated daily intakes, rather than extrapolating from data for other indoor environments. We also test correlations between the different PFASs analysed in the bedroom dust, as well as between PFASs measured in dust and in air of the same rooms during the same sampling campaign (n = 55) (Winkens et al., 2017a). Furthermore, we attempt to investigate statistical relationships between PFAS levels in dust and characteristics of children's homes (e.g. floor material of the bedrooms). To assure the accuracy of our fast extraction and analytical method we analysed standard reference material (SRM) 2585 for PFASs and present data on 24 PFASs that were previously not reported in this SRM.

2. Material and methods

2.1. Solvents, standards and laboratory routines

Solvents of highest purity were used for rinsing, extraction and instrumental analysis: methanol (LiChrosolv®, Merck Millipore), ethyl acetate (SupraSolv®, Merck Millipore), acetonitrile (Chromosolv™, Honeywell). Pasteur pipettes for extract transfer, GC-vials and other glassware were baked (450 °C, 5 h) before use. Any labware contacting the samples/extracts was rinsed with the relevant solvent(s). GC-MS standards were purchased from Wellington Laboratories (Ontario, Canada), except for 6:2 fluorotelomer acrylate and fluorotelomer methacrylate (6:2 FT(M)AC) (Fluoryx, Inc., San Leandro, USA) and 7:1 FTOH (Sigma Aldrich). For more details on LC-MS/MS standards and acronyms see Table S1 in the supplements.

2.2. Dust sample collection

During 2014/2015 dust samples were collected in 65 private households in the children's bedroom from the birth cohort study, LUKAS2, with a larger number of participants (Karvonen et al., 2009), in the area of Kuopio in Eastern Finland. For 55 rooms, paired dust and air samples were available; dust samples were collected at the end of the air sampling period of 3 weeks (Winkens et al., 2017a). Housing characteristics were recorded during the sampling visits of the homes of 32 female and 30 male children, who were included in the statistical analyses (see Table S2 for housing characteristic). The participants were instructed not to vacuum clean the room at least a week before sampling. A polyester sampling sock (allied filter fabrics PTY Ltd., Australia) was imposed into the nozzle of a vacuum cleaner (Volta U4406), with which the entire floor of the child's bedroom was vacuum cleaned. Each collected sample was scraped off the sock, folded into aluminium foil and thereafter kept in a small sealable polyethylene plastic bag, which was shipped cooled and stored at −21 °C until extraction.

2.3. Dust sieving, extraction and analysis

Each dust sample was sieved through a Retsch Analysis sieve with 0.5 mm mesh size (10 cm diameter, DIN 4188) for several minutes. Hair, grass and big fibres passing through the meshes were removed with a pair of rinsed tweezers. The resulting sample was weighed into a 15 mL falcon centrifuge tube (polyethylene terephthalate, PET). The entire sieved sample was used for extraction unless it exceeded 200 mg (average sample weight for extraction 110 mg, range 11.0–206 mg). The dust was stored capped at −21 °C until extraction. In between each sample, the sieve was thoroughly rinsed under running deionised water from the tap, followed by methanol, and dried with nitrogen gas.

Two subsequent fast extractions were used for the volatile GC-fraction and non-volatile LC-fraction, respectively, which were based on a combination of two different previously published methods (Björklund et al., 2009; Bohlin-Nizzetto et al., 2015). Prior to extraction, the samples were spiked with mass labelled (ML) internal standards (50 µL of 10 pg/µL ML-PFCAs, PFSAs, PAPs and FOSAs and FOSAAs (see Table S1); 50 µL of a mixture with 375 pg/µL (ML-Et/MeFOSE) and 125 pg/µL (ML-Et/MeFOSA) as well as 90 µL of 300 pg/µL of a ML-FTOH mixture, list with acronyms see Table S1). After about 15 min, 3 mL ethyl acetate and approximately 11 mg (10% of the average dust amount) of baked (450 °C for 12 h) Supelclean™ ENVI-Carb™ SPE Bulk Packing (120–400 mesh, Supelco) were added to the dust sample and the sample was vortexed. The dust was extracted in an ultra-sonication bath (ultrasonic cleaner USC-TH, VWR) for a total of 15 min and vortexed once in between. After centrifugation (at 4000 rpm, for 5 min), the supernatant was collected in a new 15 mL Falcon tube and the extraction procedure was repeated once again except for the addition of the ENVI-Carb™. The combined extract (6 mL) was blown down under a gentle stream of nitrogen gas and low heat (40 °C) to approximately 450 µL, and then transferred quantitatively (rinsing with approx. 150 µL ethyl acetate) into the filter of a pre-weighted reaction tube belonging to the filter (VWR Centrifugal Filter, Modified Nylon 0.2 µm, 500 µL). After centrifugation, the filter was discarded and 90 µL of volumetric/recovery GC standard was added (200 pg/µL 7:1 FTOH). The total extract weight and the weight of the remaining extract was noted after transferring 200 µL into a GC vial for later analysis. The weights were recorded in order to calculate the exact recovery of the internal standards in the LC-fraction.

For the LC-fraction, the dust and black carbon pellet was extracted with 3 mL of a basic methanol solution (0.3% ammonium hydroxide, p.a. Fluka) for 15 min in the ultra-sonication bath. The supernatant was transferred into the falcon centrifuge tube (reused from the previous GC-supernatant procedure to save plastics) and blown down to 450 µL. The remaining volume of the GC-fraction in the reaction tube (see end of the section above) was blown down to dryness and directly re-suspended with the concentrated 450 µL basic methanol extract (plus 150 µL for rinsing). A volumetric/recovery standard (50 µL 10 pg/µL M8PFOA and M8PFOS) was added to the extracts. Before analysis, the reaction tube was centrifuged and only 100 µL of the entire extract were

transferred into a LC-vial with 100 µL 4 mM aqueous ammonium acetate (p.a., Merck) solution.

The GC-fractions were run on a TRACE™ GC (Thermo Scientific) coupled to an ISQ™ MS (Thermo Scientific) and the LC-fractions were run on an Ultra Performance Liquid Chromatograph (UPLC, Acquity™, Waters), coupled to a Xevo™ TQ-S tandem mass spectrometer (MS/MS, Waters Corp., Milford, MA, USA). For more details on instrumental analysis, see supplemental material. Peak integration was performed in Xcalibur (2.2 SP.1.48, Thermo Fisher Scientific Inc.) for GC-MS and MassLynx/TargetLynx (V4.1 SCN810, Waters Inc.) for LC-MS/MS.

2.4. Data evaluation and quality assurance and quality control

Due to known sample mix-up and losses of three LC-fraction extracts, only 62 samples were available for both fractions. The data were further processed in Microsoft Excel for calculation of internal standard recoveries and analyte concentrations (ng/g dust), for which the absolute amount was divided by each dust sample's weight. When mentioning a branched (br-)PFAS, the sum of the branched isomers was integrated as one signal and quantified by applying the calibration curve of the linear (l-) isomer as well as the l-isotopically labelled isomer as internal standard. When not mentioning l- or br- specifically, the linear compound is referred to. The substances 6:2 FTAC and 6:2 FTMAC had no corresponding mass-labelled standard and were therefore quantified using the internal standard with the closest retention time, ML-4:2 FTOH and ML-6:2 FTOH, respectively. For LC-MS/MS, see ML-standards in Table S1. The PFTeDA standard curve was applied for PFPeDA quantification, 6:2 diPAP for 4:2/6:2 diPAP; 8:2 diPAP for 6:2/10:2, 6:2/12:2 and 8:2/10:2 diPAP and 10:2 diPAP for 6:2/14:2 and 8:2/12:2 diPAP. The quantified amounts of compounds without corresponding internal standards, as well as the quantified sum of branched isomers, should be seen as semi-quantitative.

At least one method blank (identical treatment as samples, only dust matrix missing, total n = 7) and one standard reference material sample (SRM 2585, from the National Institute of Standard and Technology (NIST, U.S.)) were extracted with each dust sample batch. The method precision was calculated based on the replicate analysis of the SRM (n = 6, all approx. 110 mg not sieved) and expressed as relative standard deviation (RSD) in percent. Except for 6:2 FTOH, 8:2 FTOH and 10:2 FTOH in some ethyl acetate GC-MS injections (S/N ≥ 3), no other compounds were detected in solvent injections. For more information on standard curves and for method detection limits (MDLs) see supplemental information (section “Instrumental analysis, standard curves and MDLs” and Table S5). None of the reported concentrations were blank corrected.

The recoveries were calculated using the calibration curve of the mass-labelled compounds against the volumetric standards (7:1 FTOH for GC-MS; M8PFOS and M8PFOA for LC-MS/MS) for each method blank, dust and SRM sample. The recoveries of the internal standards in the GC-fractions of the dust samples ranged between 70 and 88% (ML-4:2 FTOH and ML-EtFOSE) and was nearly identical to the recovery from the SRM samples (69% ML-4:2 FTOH up to 91% ML-EtFOSE, Table S3), with a maximal absolute standard deviation of 5.7% among the dust samples (Table S3). Slightly higher recoveries were achieved for the blanks (mean 82–100%, maximal absolute SD 7.8%). LC-fraction recoveries of the PFSAs and PFCAs in dust ranged between 47% for the long-chain ML-PFUnDA and 118% for ML-PFBA and absolute standard deviations ranged up to 22% (Table S4). In blanks, especially the short-chain PFCAs had lower recoveries (e.g. 77 ± 12% ML-PFBA), whereas these were higher in SRM 2585 (162 ± 23%, Table S4). The recovery of the LC-MS/MS PFAS precursors ranged between 54 and 123% (ML-EtFOSA and ML-8:2 diPAP) for the dust samples, which compared well to the SRM recoveries. However, the PAPs had low recoveries in the blanks (Table S4). An explanation for this could be that the internal standards of the PAPs sorbed strongly to the ENVI-Carb™, as no competing sample matrix was present in the blanks.

2.5. Statistics, data presentation and calculations

Statistical analyses were conducted with the help of JMP 12.0.1 software. Non-parametric Spearman rank tests were used to examine correlations (coefficient = ρ) among the concentrations of the different compounds in dust samples, between air (previously published in Winkens et al., 2017a) and dust concentrations, as well as between dust concentrations and characteristics of the children's homes (room volume, room area, room heights, house area, distance from Kuopio, Table S2). Non-parametric Wilcoxon/Kruskal-Wallis Rank Sums tests were conducted for categorical parameters (house type, floor level, floor material, sharing/own room), as the dust concentrations were not normally distributed for all compounds and categories (Shapiro Wilk W Test, $p > 0.05$). The Wilcoxon Method for each pair was chosen as posthoc test (all tests $\alpha = 0.05$). Categories with $n \leq 3$ were excluded from the statistics (e.g. floor material: tiles and cork, each n = 1, Table S2). Data are presented with the help of RStudio (version 0.99.902, Inc.), JMP 12.0.1 and Microsoft Excel. The figures were partly modified in Inkscape 0.91.

In order to test if the distributions of PFASs between dust and air are controlled by an organic carbon-air partitioning process, we plotted for compounds with ≥50% detection frequency the logarithm of the ratio between the dust concentration and the air concentration (log $K_{dust/air}$) against the log K_{oa}. The air concentrations were previously published in Winkens et al. (2017a). The log K_{oa} values for the PFASs originate from Wang et al. (2011), are for the neutral (non-ionised) forms of the substances and are based on COSMOtherm calculations.

In order to calculate the estimated daily intake via oral dust intake (EDI_{dust}) expressed in ng/kg bw/day for each 10.5-year-old child individually, we used the dust concentration of the room (only for compounds with an overall detection frequency of ≥50%, < LOD treated as LOD/square root of 2) and bodyweight (bw) of each individual using the following equation:

$$EDI_{dust}\ [ng/kg\ bw/day] = \frac{dust\ concentration\ [ng/g] \times intake\ [g/day]}{\text{bodyweight [kg]}} \times F_{uptake}[-] \times F_{biotransf}[-] \quad (1)$$

For several factors in the equation different values were chosen to simulate different exposure scenarios, referred to as low, intermediate and high exposure scenario. The uptake factor (F_{uptake}) describing the fraction of the total PFAS amount that is taken up into the body, as well as the biotransformation factor ($F_{biotranf}$) of precursors to single PFAAs, were mostly based on modelling and rodent studies, which pose an uncertainty (see supplements). The intake of indoor dust per day originated from the exposure handbook and was 0.06 g/day in the low and intermediate exposure scenario and 0.1 g/day in the high exposure scenario (US EPA, 2008), assuming that PFAS levels in dust of other indoor environments would be similar. For more details on assumptions and different scenarios, see supplements.

For the EDI calculations via air (EDI_{air} in ng/kg bw/day), we used the same formula (Eq. (1)) and biotransformation factors as for EDI_{dust}. The dust concentration was replaced with the air concentration [ng/m^3] of each child's sleeping room (Winkens et al., 2017a) and the intake of air with the recommended long term inhalation rate for 6- to < 11-year-olds of 12.4 (mean in exposure handbook, applied for low and intermediate exposure scenario) and 18.7 m^3/day (95th percentile in exposure handbook, applied for high exposure scenario) (US EPA, 2008). The fraction of the time per day spent indoors ($F_{indoors}$) was included by multiplication into Eq. (1) (US EPA, 2008) and kept constant at 0.864 for all scenarios (see supplements and US EPA (2008)). For all scenarios and PFASs, the uptake via air was assumed to be 100% ($F_{uptake} = 1$), as assumed by others (Padilla-Sánchez et al., 2017; Shoeib et al., 2005) and initially suggested for PFOA by Kennedy et al. (2004).

3. Results and discussion

3.1. Standard reference material, SRM 2585

Within the LC-fraction, the highest mean levels exceeding 1000 ng/g were detected for monoPAP, l-PFOS and l-PFHxS (1760, 1290 and 1120 ng/g, respectively). Among the PFCAs, l-PFOA had the highest mean concentration (660 ng/g) followed by PFHpA (360 ng/g). Of the FOSAs and FOSAAs, only l-EtFOSAA had concentrations in the several hundred ng/g range (mean 444 ng/g). The relative standard deviations (RSDs) for the PFCAs and PFSAs ranged between 3.6 and 19% (highest for PFTeDA). The precision was similar for all monoPAPs and most diPAPs, with the exception of 4:2 diPAP, 8:2/12:2 diPAP and 10:2 diPAP (RSDs = 22, 20 and 37%). FOSA and FOSAA RSDs ranged from 12 to 29%, which was even exceeded by 6:2 fluorotelomer sulfonic acid (6:2 FTSA, 35%). The lower precision among the FTSAs was likely partly due to the use of a different internal standard (M8FOSA).

For the GC-fraction, the dust concentration was the highest for FTOHs and FOSEs, in the order: 8:2 FTOH > 6:2 FTOH > EtFOSE > 10:2 FTOH > MeFOSE (means between 5220 and 2790 ng/g, Table S7). EtFOSA, MeFOSA and 4:2 FTOH were in the lower ng/g dust range (5.71, 32.7 and 80.1 ng/g, respectively). The RSD between the six replicate SRM samples in the GC-fraction was ≤9.3% for the FOSA/Es and up to 21% for 4:2 FTOH (Table S7). The comparison of the RSDs suggests a higher precision for GC-MS data for FOSAs compared to LC-MS data. The concentration of EtFOSA compared well between both analytical methods (5.71 ng/g GC-MS and 7.26 ng/g LC-MS/MS). 6:2 FTAC and 6:2 FTMAC (GC-fraction), and 3:3 acid, 5:3 acid, 7:3 acid, ADONA, 9Cl-PF3ONS and 11Cl-PF3OUdS (LC-fraction) were below the MDLs in all SRM 2585 samples.

SRM 2585 is not certified for PFASs and we report concentrations for 24 PFASs that have not been reported for this SRM in literature previously (Björklund et al., 2009; Goosey and Harrad, 2011; Padilla-Sánchez and Haug, 2016; Reiner et al., 2015). Most studies appear to have outliers for some analytes when compared to the other studies (see Table 1).

Concentrations from the present study compare well to literature values, especially for the PFCAs and PFSAs. However, the concentration of 6:2 FTSA was a factor of 3–4 higher in comparison to two other studies and a factor of 2–3 lower for 8:2 diPAP (Table 1). The differences to the two other studies were biggest for the concentrations of EtFOSE. Different extraction methods and analytical techniques can only partially explain these differences. The EtFOSA results support this statement, as they compared well between the GC- and LC-MS method in the present study and with a study by Padilla-Sánchez and Haug (2016), but compared poorly to data from another study (Goosey and Harrad, 2011). The use of different internal standards among studies could be a reason for discrepancies, as they might result in varying compensations for recovery losses and matrix effects. In the present study, a matching mass-labelled standard was used for quantification of EtFOSE, but not for 6:2 FTSA, for which M8FOSA was used.

3.2. PFCAs and PFSAs in dust of children's bedrooms

Among the PFCAs, the most frequently detected were: l-PFOA > PFDA > PFDoDA > PFNA > PFHxA (in between 52 and 76% of the samples, Table S8). Among the PFSAs, only l-PFOS was detected in more than half of the samples (51%), followed by l-PFHxS with a 33% detection frequency (Table S8). The highest median concentration in dust was found for l-PFOA (5.26 ng/g), followed by PFHxA (2.33 ng/g), PFDA and PFNA (1.58 and 1.05 ng/g, Fig. 1). The l-PFOS median concentration was 0.95 ng/g. The overall maximal concentrations among the PFAAs in the dust samples were recorded for l-PFOA and PFDA with 82.7 and 67.0 ng/g, respectively (Fig. 1, Table S8).

In comparison to the SRM 2585, the floor dust samples have up to three orders of magnitude lower median concentrations for the PFSAs and up to two orders of magnitude lower concentrations for the PFCAs. This difference might reflect the phase-out and shift in production towards different compounds, which happened in between the two sampling points of the SRM (1993/1994) and the floor dust samples (2014/2015). Additionally, relatively more branched isomers were detected in the SRM sample, which occurred as impurities during the former electrochemical fluorination production. These are possible clues for temporal changes. Though, the different global origin (mostly North-USA vs. Finland) and differences of sample acquisition (SRM = vacuum cleaner bags of different homes, motels and hotels etc. in comparison to exclusively floor dust of children's sleeping room), as well as the different particle size fractions do not allow for hypothesising a purely time-causal relation of the contamination patterns.

3.3. Precursors and other PFASs in dust of children's bedrooms

Only l-EtFOSAA and 6:2 diPAP were detected in every sample (Table S8). Considering solely the 86% of the households exceeding the detection limit for br-EtFOSAA, the branched fraction accounted for 3.7 to 47% of the total EtFOSAA amount (median 15%). All diPAPs except for 4:2 and 4:2/6:2 were detected in ≥60% of the samples. 6:2 monoPAP was more frequent (87%) than the longer-chain monoPAPs. 8:2 FTSA was detected in half of the samples and 6:2 FTSA only in one, which was likely based on the outstanding high MDL for 6:2 FTS (82.9 ng/g) based on contamination and a high range between the blanks (Table S5 and S8). Among the GC analytes, the detection frequency was highest for 8:2 FTOH and 6:2 FTOH (80 and 72%), followed by 10:2 FTOH and EtFOSE (65 and 57%, respectively, Table S9). MeFOSE and 6:2 FTMAC were detected equally frequently (43%), but in different samples (Table S9). The following PFASs were not detected in any dust sample above the MDL in LC-MS/MS analysis: 4:2 monoPAP, 4:2 diPAP, br-FOSA, br-MeFOSAA, MeFBSA, br- and l-EtFOSA, 4:2 FTSA, 3:3 acid, 5:3 acid, 7:3 acid, ADONA and 11Cl-PF3OUdS; in GC-MS analysis: 6:2 FTAC, 4:2 FTOH and EtFOSA.

Among FTOHs, median concentrations of 8:2 FTOH were highest (45.7 ng/g), followed by 6:2 FTOH and 10:2 FTOH (26.6 and 15.8 ng/g, Fig. 1, Table S9). Nearly identical maximal concentrations were reached for 8:2 FTOH and 6:2 FTOH (298 and 288 ng/g), which did not originate from the same household. One of the households had close to maximum values for both FTOHs, whereas the household with maximal 8:2 FTOH concentration reached median levels for 6:2 FTOH. The diPAPs ranged between 0.88 and 53.9 ng/g for 8:2/10:2 and 6:2 diPAP (Table S8). The far higher arithmetical mean (137 ng/g) in comparison to the median for 6:2 diPAP indicates a skewed distribution with several households having several hundreds of ng/g, with a maximum of 1360 ng/g (Fig. 1, Table S8). This was the overall highest concentration in the bedroom dust for any compound. The dominance of FTOHs and PAPs shows the need of their inclusion in future dust analyses, as the focus is often on PFAAs.

3.4. Comparison of dust levels to previous studies

The concentrations and ranges of the different PFAAs are generally in the same order of magnitude as in other studies throughout Europe that were conducted close in time to our 2014/2015 sampling campaign. For a summary of recent dust studies see tables in Karásková et al. (2016). The dust concentration of PFOS is lower or similar in the present study (median 0.95 ng/g) compared to both studies conducted in 2013 in the Czech Republic (10.3 and 1.5 ng/g) (Karásková et al., 2016; Lankova et al., 2015) and 2013/2014 in Greece and Sweden (7.2 and 2.8 ng/g) (Eriksson and Kärrman, 2015). The l-PFOA concentration was higher in the current study (median 5.62 ng/g) compared to the Czech Republic (2.0 and 2.4 ng/g), but lower in comparison to Greece and Sweden (12.8 and 14.4 ng/g), which was in accordance with the trends of PFNA and PFDA. In a large indoor study conducted in Norway

Table 1
Comparison of PFAS concentrations in SRM 2585 to data from different studies; compounds highlighted in bold have a good agreement with literature values (within standard deviation (SD) range), those in italics differ from literature values (more than two times higher/lower than mean ± SD); (LC) = measured by LC-MS(/MS) in literature, whereas in the present study also GC-MS (GC) was applied.

SRM 2585 [ng/g]	Present study [mean ± SD]		Björklund et al., 2009 [mean ± SD]	Goosey and Harrad, 2011 [mean ± SD]	Reiner et al., 2015 [averages]	Padilla-Sánchez and Haug, 2016 [mean ± SD]***
PFBS	**21.3**	**± 2.87**			**18.8–130****	*5 ± 1.80*
l-PFHxS	**1120**	**± 148**				**1153 ± 104**
sum-PFHxS	**1420**	**± 191**		*4282 ± 274*	**1400***	
l-PFOS	**1290**	**± 185**				**1401 ± 84.2**
sum-PFOS	**1860**	**± 256**	**1990 ± 78**	**1752 ± 31**	**2280***	
PFDS	**403**	**± 32**			**375–902****	*188 ± 45.1*
PFBA	**229**	**± 25.2**			**249***	
PFPeA	**235**	**± 24.4**			**226***	
PFHxA	**349**	**± 33.2**			**251–474****	**324 ± 32.4**
PFHpA	**360**	**± 36**			**220–440****	240 ± 69.6
l-PFOA	**660**	**± 23.6**				**599 ± 53.9**
sum-PFOA	**747**	**± 26.6**	673 ± 26	**766 ± 38**	**430–760**	
PFNA	**90.1**	**± 12**			**101***	**85 ± 9.35**
PFDA	**66.9**	**± 10.7**			**23.9–65.7**	**54 ± 12.4**
PFUnDA	**41.5**	**± 4.21**			**26.1–55.8**	34 ± 4.42
PFDoDA	**37.9**	**± 4.15**			**34.3–40.6**	**31 ± 4.34**
PFTrDA	**29.6**	**± 4.4**			**27.9–29.3**	*4 ± 1.48*
PFTeDA	30.8	± 5.88			10.7–22.4	*3 ± 1.23*
l-FOSA	**6.77**	**± 1.42**		*75 ± 5*	**7.78–11.6**	
l-EtFOSA	7.26	± 1.02		*179 ± 10*		3 ± 1.50
l-MeFOSAA	*64.5*	*± 18*			*150 ± 12***	
l-EtFOSAA	444	± 54.6			675 ± 77**	
6:2 FTSA	*404*	*± 139*			*90 ± 9.5***	
8:2 FTSA	**212**	**± 50.6**			**169 ± 58****	
6:2 monoPAP	**208**	**± 26.1**				**245 ± 19.6**
8:2 monoPAP	190	± 27.1				132 ± 19.8
6:2 diPAP	**675**	**± 28.3**			421 ± 83**	**742 ± 89.0**
8:2 diPAP	227	± 27.3			868 ± 30**	559 ± 151
EtFOSA (GC)	**5.71**	**± 0.28**		*179 ± 10 (LC)*		**3 ± 1.50 (LC)**
MeFOSA (GC)	32.7	± 2.61				50 ± 4.0 (LC)
EtFOSE (GC)	*4200*	*± 338*		**66 ± 4 (LC)**		**55 ± 13.8 (LC)**
MeFOSE (GC)	**2790**	**± 180**		*562 ± 23 (LC)*		**2306 ± 254 (LC)**

* [Weighted average] of interlaboratory study (5 subsets, each n = 6 − 9 SRM samples, different extraction methods and partly different labs), see Table 6 in that study

** [Mean-mean] range of mean values in case 4 − 2 interlaboratory sets investigated that compound; or [mean ± standard deviation (SD)] in case only one interlaboratory subset investigated this compound, see Table 3 in that study.

*** For Padilla-Sánchez & Haug: SDs were calculated based on reported relative SDs (RSD) with the equation SD = RSD × mean/100.

6–7 years before the present study, concentrations were higher for all PFCAs and PFOS (Haug et al., 2011). EtFOSA was not detected in the present study, whereas it was detected in 6 out of 41 dust samples from bookshelves and window sills by Haug et al. (2011). As the Northern Europeans have a similar culture and thus likely a similar consumer behaviour and products in their households, we expect similar trends in PFAS contamination in Norway and Finland. Therefore, the differences between the results reported by Haug and co-workers and our study may reflect the 3M phase-out of long-chain ECF products between 2000 and 2002 and the decreased usage of PFOS and long-chain PFCAs in recent years. The concentrations in the present study are lower than those measured in North America in 2013/2014 (Eriksson and Kärrman, 2015) and 2013 (Karásková et al., 2016), which fits the significant differences found between the USA and the Czech Republic in Karásková et al. (2016). De Silva et al. (2012) analysed PAPs in 102 dust samples in Canada (sampling 2007–2008) and reported for most compounds one to two orders of magnitude higher concentrations compared to concentrations in the present study. Especially the median of 8:2/10:2 diPAP was much higher in the Canadian study compared to the present study (213 ng/g vs. 0.88 ng/g). Both sampling location and time point probably led to the discrepancies based on differences related to the production of PFASs and their presence in consumer products as well as on differences related to consumer behaviour. 8:2 FTSA was the most frequently detected FTSA in Norwegian children's bedrooms (n = 6, sampled in 2015 (Bohlin-Nizzetto et al., 2015)) and the median level (9.5 ng/g) was in the same order of magnitude as in the present study. It has to be considered though that the data for FTSA were semi-quantitative in the current study.

3.5. Correlation analysis between PFASs in dust

Concentrations in dust between the individual diPAPs were highly positively correlated ($\rho \geq 0.75$) and most of the correlations were highly significant ($p < 0.001$; Fig. S1); which was also shown by Eriksson and Kärrman (2015). A similarly strong correlation existed among the FTOHs, which was strongest between 8:2 and 10:2 FTOH ($\rho = 0.91$, $p < 0.001$). The PFCAs also correlated with each other ($\rho \geq 0.52$, $p < 0.001$), as did the linear and sum-branched isomers of EtFOSAA ($\rho = 0.87$; $p < 0.001$, Fig. S1). In summary, several substances with different chain-length, but within the same PFAS sub-class correlated with each other. This indicates that they likely originate from the same products, as they occur in mixtures of different chain lengths or in different isomer forms from ECF production (Buck et al., 2012).

The frequent presence of br-EtFOSAA as well as the highly significant and positive correlation between l-PFOS and l-PFOA ($\rho = 0.43$, $p < 0.001$) indicate that a fraction of the PFASs in dust originated from ECF production. In ECF usually up to 30% branched isomers were produced (Prevedouros et al., 2006). In the samples of the current study, the br-EtFOSAA fraction was 15%. It is unknown why the fraction of branched isomers of EtFOSAA (15%) is lower than usually found in the ECF process (30%). There was hardly any correlation among the

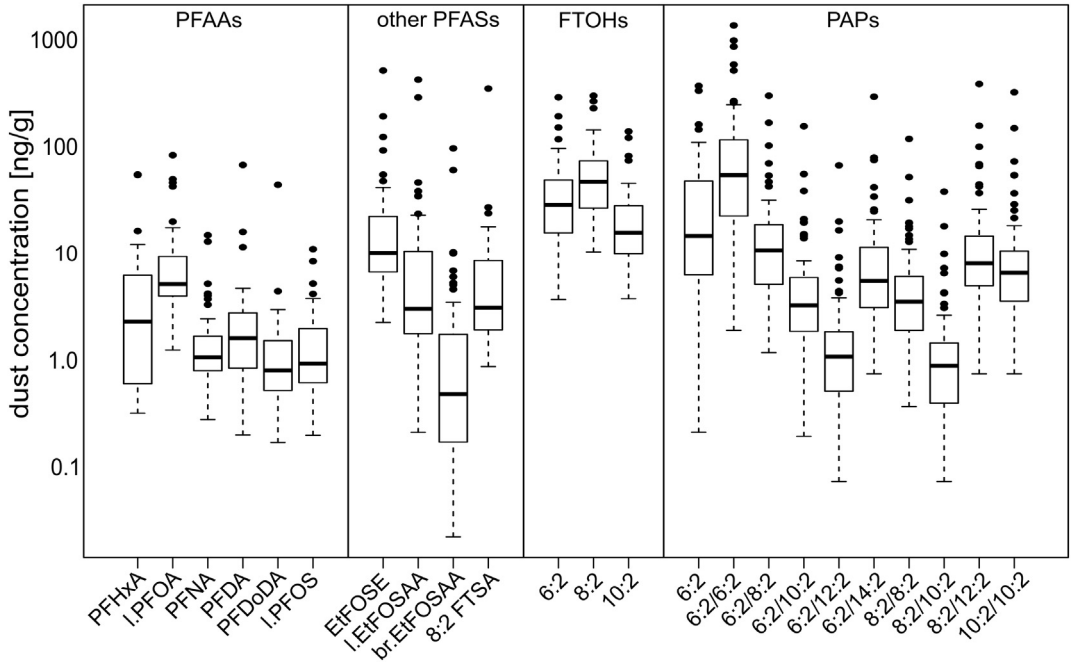

Fig. 1. PFAS concentrations (ng/g) in dust from children's bedrooms (n = 62); note the log scale; the median values are the thick horizontal lines within the boxes; each box is the inner quartile (IQ), whiskers are the quartile ± 1.5*IQ range, respectively; black circels are data points for single homes outside these ranges; substances with < 50% detection frequency were excluded; values below MDL were treated as MDL/sqrt(2).

PFCAs and the PAPs, despite PFCAs being environmental degradation products from (Lee et al., 2014) or impurities in PAP formulations (D'eon and Mabury, 2011; Fujii et al., 2013). The significant and weakly positive correlation among some PFCAs and FTOHs in dust is expected ($\rho \leq 0.51$, $p < 0.001$), due to their common origin in the telomerisation process (Wang et al., 2014). 8:2 FTSA was positively correlated with the long-chain PFCAs ($\rho = 0.42$–0.52, Fig. S1), which was previously reported for 6:2 FTSA and PFCAs with similar correlation coefficients (Eriksson and Kärrman, 2015). The assumption that could explain these correlations is that FTSAs can degrade to PFCAs (Wang et al., 2014).

3.6. Correlation among dust and indoor air of the same room

There was a significant but weak positive correlation ($\rho = 0.30$–0.41, $p < 0.05$) between 6:2 FTOH air concentrations (Winkens et al., 2017a) and the concentrations of most diPAPs in dust (Fig. S2), which suggests a common origin from similar products, as they share the same intermediates (fluorotelomer iodides) in the telomerisation process (Wang et al., 2014). The FTOHs in air correlated, also mostly significantly, with br- and l-EtFOSAA in dust and there was a strong positive correlation between 8:2 and 10:2 FTOH in dust and br- and l-PFOS in air (Fig. S2). The latter was also reported previously (Haug et al., 2011), even though these correlations do not seem to be causal relationships as the respective PFASs have distinct production processes. The correlation between several PFCAs in dust, with the exception of PFNA, with nearly all the FTOHs in air could be causal due to their common origin in the telomerisation process and the possible degradation of FTOHs to PFCAs. Indoor degradation of FTOHs is supported by the measurement of fluorotelomer unsaturated acids in a previous study (Yao et al., 2018). PFNA was the only frequently detected PFCA in dust with an uneven number of carbon atoms, which usually do not occur during telomerisation, but only via ECF and this may explain why PFNA in dust did not correlate with FTOHs in air. PFDA and PFDoDA in dust correlated nearly with all PFCAs in air (Fig. S2). It is likely that the same release mechanism from the same products occurred (Prevedouros et al., 2006) for the PFCAs. Based on their physicochemical properties, long-chain PFCAs partition rather to dust than air; therefore, PFDA and PFDoDA were likely the only significantly correlating PFCAs on the dust side. EtFOSE and l-PFOS in dust correlated solely positively ($\rho = 0.23$–0.37, $p < 0.05$) with other compounds in air originating from the ECF process (EtFOSE, MeFOSE, br- and l-PFOS, Fig. S2). Similar correlation coefficients ($\rho = 0.36$–0.42, $p < 0.05$) between PFOS in dust and FOSEs and MeFOSA in air were reported by Haug et al. (2011).

The plot of log $K_{dust/air}$ against the logarithm of the octanol-air partition coefficient (log K_{oa}) resulted in a linear regression line (pairwise comparison $r = 0.94$, $p < 0.001$), which fitted the data of the different PFASs well with a $R^2 > 0.88$ (Fig. 2). This suggests that the distribution of PFASs between indoor air and floor dust is indeed controlled by a partitioning between the gas phase PFASs in the air and the PFASs sorbed to the organic phases in the dust. A similar phenomenon has been observed for gas-particles partitioning of PFASs in outdoor air and has even been accurately modelled (Ahrens et al., 2012). Despite the plausible explanation of the partitioning by Ahrens et al. (2012), the

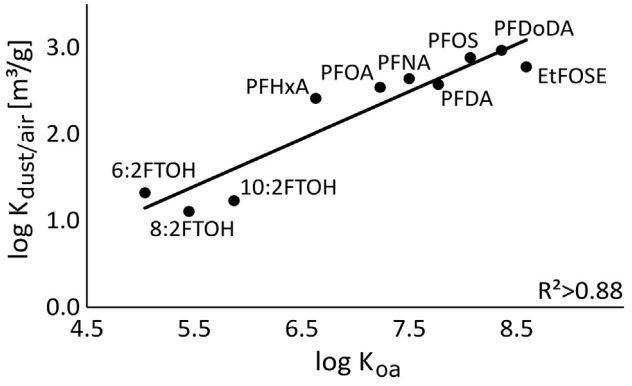

Fig. 2. log $K_{dust/air}$ [m^3/g] plotted against the log K_{oa}. The dust and air concentrations were measured in the same rooms (n = 55, median values) (Winkens et al., 2017a); log K_{oa} by Wang et al. (2011); $R^2 > 0.88$; $y = 0.547x - 1.61$.

adsorption and/or absorption behaviour of PFASs on particle surfaces is poorly understood and needs further experimental investigation. The slope of the log $K_{dust/air}$ against the log K_{oa} plot was < 1.0 (i.e. 0.55). This could indicate a lack of achievement of equilibrium of PFASs between air and dust based on a) sampling artefacts, i.e. sampling of fine particles with the passive air samplers and thus overestimating gas-phase concentrations; b) the non-equivalence of the organic matter in dust and octanol, i.e. differences in solvation properties and/or c) a short residence time of the air due to a fast air exchange rate (Chan et al., 2005; Isaacs et al., 2013).

In previous studies, good correlations were found for similar plots for several semivolatile organic compounds (SVOC, Weschler and Nazaroff, 2010) and flame retardants (Cequier et al., 2014). We plotted log $K_{dust/air}$ and log K_{oa} for flame retardants (FRs) from another study (Cequier et al., 2014). Their intercept was similar to the one for PFASs, although the slope was flatter for FRs (see Fig. S3). The FRs fitted the linear regression less well compared to the PFASs (FRs: $R^2 > 0.44$, Fig. S3). A reason for this might be that the FRs need a longer time to reach equilibrium because of their higher log K_{oa} values compared to PFASs, as reasoned by Weschler and Nazaroff (2010).

The good correlation for PFASs (Fig. 2) also showed that the extrapolation from either air to dust concentrations or vice versa might be possible based on one of the two indoor samples and the log K_{oa}, as suggested for other compounds (Cequier et al., 2014). As dust sampling, extraction and analysis is usually less invasive and time consuming than air sampling, for future indoor PFAS monitoring studies, dust analysis might be sufficient for predicting air concentrations. However, more research is needed for corroboration of the prediction certainty ($R^2 > 0.88$) from the log K_{oa} to the log $K_{dust/air}$. Studies should investigate the relationships for a larger range of PFASs and different kinds of dust samples (i.e. settled dust, vacuum cleaner bag dust, different size fractions).

3.7. Associations between dust concentrations and other housing characteristics

The dust concentration of l-PFOS in rooms with plastic (i.e. PVC/vinyl) floor material was significantly higher than in rooms with wooden floor material ($p < 0.01$ Wilcoxon each pair; Kruskal-Wallis all floor categories $p = 0.03$; Fig. S4). The mean dust concentration of l-PFOS was 2.76 ng/g in the rooms with plastic floor material in comparison to 1.00 ng/g in the rooms with wooden floor material. Different surface cleaning, coating or polishing products for different floor materials could have led to this discrepancy as PFASs are known ingredients in such products (Danish EPA, 2005; Herzke et al., 2012; Kotthoff et al., 2015; Liu et al., 2015). As PFOS was phased out around the 2000s, the age of the floor material or the floor treating products could also have had an influence.

There were some more housing parameters that were associated to the dust concentrations. However, the causality of these relations is quite questionable and should be seen with caution, but these associations are for completeness exploited in the supplements.

3.8. Estimated daily intake for 10.5-year-old children via dust and air

The total intake of a PFAA substance is referred to as the sum (ΣPFAA substance) of (1) the direct intake of the PFAA itself and (2) the PFAA amount that is generated indirectly after the intake of several precursor substances (via the same pathway) that are assumed to subsequently be biotransformed to the PFAA (see Tables S11–S14, total intake = ΣPFAA).

In the low and intermediate exposure scenarios, EDI_{dust} was the highest for the Σl-PFOA intake among single ΣPFAAs, with median values of 0.006 ng/kg bw/day and 0.007 ng/kg bw/day, respectively (Tables S11 and S12, Fig. 3). The EDI_{dust} of 8:2 FTSA in these two scenarios (0.004 and 0.005 ng/kg bw/day) was similar to the Σl-PFOS intake (0.003 and 0.006 ng/kg bw/day). For the high exposure scenario, the Σl-PFOS intake was slightly higher than the Σl-PFOA intake (median values 0.015 vs. 0.014 ng/kg bw/day, Table S13). 56% (median) of the Σl-PFOS intake in the high exposure scenario originated only from the biotransformation of EtFOSE, in the low exposure scenario l-PFOS and EtFOSE contributed equally with 40%.

In the low exposure scenario, the direct intake contributed the largest fraction of the ΣEDI_{dust} for the single PFCA homologues (median 93–100%, Table S11, Fig. 3). This proportion decreased for the high exposure scenario; in the case of PFHxA with a contribution of 69%, but the longer chain PFCAs still had high contributions of direct intakes (≥88%, lowest for PFDA, Table S13). The 6:2 diPAP biotransformation contributed with up to 12% to the largest proportion among precursors to the ΣPFHxS intake in all exposure scenarios (followed by 6:2 FTOH and 6:2 monoPAP, Tables S11–S13).

The EDIs suggest that precursor degradation did not play a major role for PFCA intake via dust. The PFOS intake via dust, however, was strongly driven by precursor biotransformation (i.e. intermediate scenario 69%, Fig. 3). Precursors were found to contribute 41–68% to ΣPFOS uptake via all investigated exposure pathways in another study (Vestergren et al., 2008).

For EDI_{air}, the ΣPFOS intake at the high exposure scenario was as high as 0.011 ng/kg bw/day (median, Table S14, Fig. 3). For the ΣPFOS intake via air, the precursors contributed with 90% to the ΣPFOS intake at the intermediate scenario; MeFOSE had the highest contribution with 71% of the ΣPFOS intake. Considering only the single substance, the intake of l-PFOA was the highest at the high exposure scenario (median value 0.007 ng/kg bw/day, Table S14). The ΣPFOA intake was the highest of all ΣPFAAs with median values of 0.007 and 0.020 ng/kg bw/day for the intermediate and high exposure scenario, respectively (Table S14). The precursor metabolism contributed with 38% to the ΣPFOA intake via air inhalation at the intermediate scenario. Generally, for the single substance, the ΣPFCA and the ΣPFOS EDIs were very similar for dust and air intake at the intermediate scenario, which was surprising as dust was previously hypothesised to have a high contribution to the EDI of infants, toddlers and children in intermediate and high exposure scenarios (Trudel et al., 2008). However, the children in our study were 10.5 years old and PFAS production changes that occurred between our study and Trudel et al. (2008) have likely affected these EDIs of both media, as well as advances in analytical methods (Vestergren et al., 2012). However, PFCAs did contribute to a higher degree to the ΣPFCA intake via dust in comparison to air. In other words, the absolute and relative contribution of FTOHs to the ΣPFCA intake was much higher in air than in dust (e.g. intermediate scenario: for air ΣPFOA = 0.007, contribution of 8:2 FTOH = 0.003 ng/kg bw/day, Table S14; for dust ΣPFOA = 0.007, contribution of 8:2 FTOH = 0.00008 ng/kg bw/day, Table S12). Therefore, the investigation of more precursor compounds in air would have been interesting and might play a role for ΣPFCA exposure. Further, the outdoor air concentration was assumed to be negligible and the indoor air concentration of the bedroom to be representative for all indoor environments, which pose an uncertainty. However, 8:2 FTOH contributed in another air study to the exactly same absolute amount to the PFOA intake as in the present study (both: 0.003 ng/kg bw/day) (Padilla-Sánchez et al., 2017). The FTOHs also contributed the same amount to intakes of PFHxA, PFNA and PFDA (0.001, 0.003 and 0.008 ng/kg bw/day) when comparing our air intakes (Table S14, see intermediate scenario, FTOHs) to their personal air intakes. The estimated daily intakes via residential measurements were 1.7–2.0 times higher than personal air intakes (Padilla-Sánchez et al., 2017). The higher FTOH concentrations and the larger body weights for the adults in their study might have counterbalanced each other and thus their adult and our child data became comparable.

When summing up the EDIs for all PFAS compounds (sum of ΣPFAAs in Fig. 3, or in Tables S12 and S14), the EDI_{dust} is slightly higher than the EDI_{air} for the 10.5-year-olds (e.g. median values at

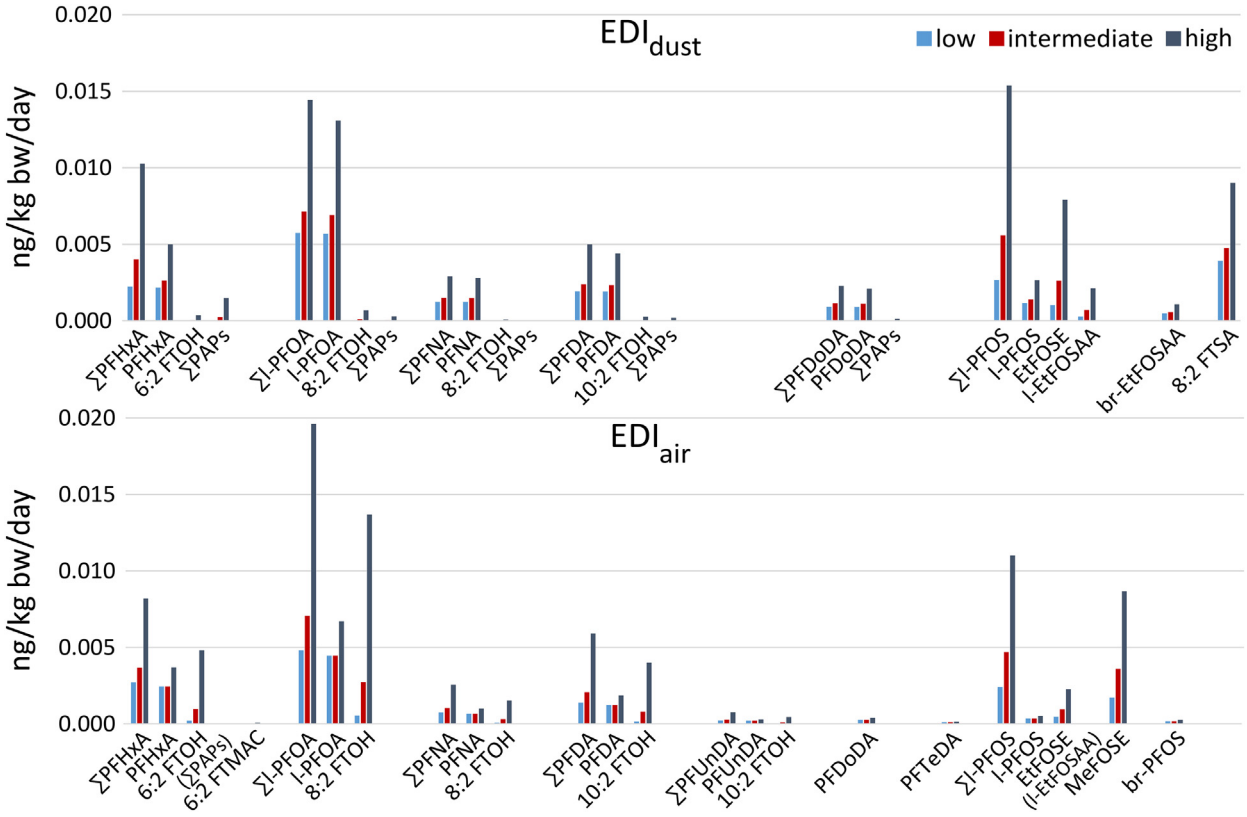

Fig. 3. Estimated daily intakes via dust (top figure) and air [ng/kg bw/day] (figure below); intakes for the total PFAA (ΣPFAA), include the contribution of different precursors (precursor bars indicate the contribution to ΣPFAA intake) at three different exposure scenarios: low, intermediate, high; median values are shown for each scenario; compound names in brackets were not measured in air (but are displayed for better comparison with dust); shown PFASs were detected in > 50% of the samples.

intermediate exposure scenario: 0.027 and 0.019 ng/kg bw/day, i.e. 40% higher and at high exposure scenario: 0.060 and 0.049 ng/kg bw/day, 23% higher). The intermediate EDI_{dust} was an order of magnitude lower compared to toddlers' intake (0.21 ng/kg bw/day) and an order of magnitude higher in comparison to adults' in the Czech Republic (0.002 ng/kg bw/day) (Karásková et al., 2016). This seems logical due to the different body weights and different assumptions made for the EDI calculation. Younger children have a higher intake of dust related to bodyweight (Winkens et al., 2017b).

In the present study, the worst case scenario (95th percentile in the high exposure scenario) for the sum of all ΣPFAAs showed a 2.0-times higher EDI_{dust} than EDI_{air} (0.304 and 0.154 ng/kg bw/day). The sum of the dust and air EDI ($EDI_{\Sigma dust+air}$) of all ΣPFAAs for the worst case was 0.458 ng/kg bw/day, which is well below the tolerable daily intakes for PFOA (1500 ng/kg bw/day) and PFOS (150 ng/kg bw/day) set by EFSA (2008). However, these values are based on adult rodents', respectively adult monkeys' toxicity studies and not on developmental stages as well as on oral uptake only. We used this simple approach, as TDIs are neither available based on inhalation nor for other PFASs. Further, we acknowledge that different entry pathways (i.e. inhalation vs. ingestion) might require different toxicity endpoints, besides the fact that the different PFASs might have different toxicological effects. The non-binding minimal risk levels (MRLs) that were recently set by the agency for toxic substances and disease registry (ATSDR, 2015) are far lower (MRL: PFOA 20 ng/kg bw/day and PFOS 30 ng/kg bw/day). Compared to the worst case sum PFAS intake via air and dust (0.458 ng/kg bw/day), the MRLs are still a factor of approx. 50 higher. However, it has to be considered that other pathways such as dermal uptake, drinking water and food ingestions were not included in this study, but would also contribute to the total EDI. Though the dermal uptake via dust was previously calculated to contribute < 1% to the total uptake of each, PFOA and PFOS, in any exposure scenario (Trudel et al., 2008; Vestergren et al., 2008). To conclude, taking the total PFAA intake for single PFAAs including their precursors' biotransformation into account, air seems to be of higher importance than previously assumed and of similar importance as dust. Considering the sum of all PFAAs, EDI_{dust} was double as high as EDI_{air}. It is necessary to remember that the EDIs are dependent on the chosen parameters in the equation and the type and number of analysed precursor compounds. More research is needed on the quantitative exposure via multiple pathways for several childhood stages in order to clearly map the importance of different exposure pathways. Additionally, uptake and biotransformation rates in children should be investigated to confirm that rates are similar to adults', which is commonly assumed for EDI calculations.

Declaration of interest

None.

Acknowledgements

We thank all participating families of the CEEP study; Sirpa Räsänen (THL) for sampling; Michael Strandell, Oskar Sandblom and Ulla Eriksson for support in instrumental troubleshooting; Raed Awad for the addition of some more LC-MS/MS compound ions; Jonathan Benskin for advice and discussions; Jonathan Martin and Anna Sobek for their valuable comments on the manuscript (all ACES); The IVL Swedish Environmental Research Institute for sharing the SRM; The Swedish Research Council FORMAS (Grant 2012-3283-23680-71) for funding.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2018.06.009.

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