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DOI:

[10.1021/acs.est.9b04604](https://doi.org/10.1021/acs.est.9b04604)

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Document Version

Peer reviewed version

Citation for published version (Harvard):

Harrad, S, Wemken, N, Drage, D, Abdallah, M & Coggins, M 2019, 'Perfluoroalkyl substances in drinking water, indoor air and dust from Ireland: implications for human exposure', *Environmental Science and Technology*, vol. 53, no. 22, pp. 13449-13457. <https://doi.org/10.1021/acs.est.9b04604>

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Perfluoroalkyl Substances in Drinking Water, Indoor Air and Dust from Ireland: Implications for Human Exposure

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12 **Abstract**

13 Perfluoroalkyl substances (PFASs) were measured in air and dust from cars, homes, offices, and
14 school classrooms in Ireland, along with drinking water from homes and offices. Perfluorooctanoic
15 acid (PFOA) dominated air and drinking water, while perfluorobutane sulfonate (PFBS)
16 dominated dust. This is the first report of PFOA, perfluorooctane sulfonate (PFOS),
17 perfluorohexane sulfonate (PFHxS), PFBS, and perfluorononanoic acid (PFNA) in air inside cars
18 and school classrooms. PFOS concentrations in classroom air exceeded significantly ($p \leq 0.05$)
19 those in homes. Atmospheric concentrations of PFOA, PFNA, and methyl perfluorooctane
20 sulfonamido ethanol (MeFOSE) ($p \leq 0.05$) were significantly higher in cars containing child car
21 seats than in cars without. PFOS, PFOA, PFBS, and PFHxS were all detected frequently in
22 drinking water but concentrations of PFASs were low, and although Σ PFASs were 64 ng/L in one
23 bottled water sample, this fell below a Swedish Action Level of 90 ng Σ PFASs/L. The Irish
24 population's exposure to PFOS and PFOA via non-dietary sources is well below estimates of
25 dietary exposure elsewhere in Europe. Moreover, even under a high-end exposure scenario, it falls
26 below the European Food Safety Authority's (EFSA) provisional tolerable weekly intakes for
27 PFOS and PFOA.

28 **Introduction**

29 Perfluoroalkyl substances (PFASs) possess beneficial industrial properties like oil and water
30 repellency, physical and chemical stability, and surface-active properties¹. Such properties mean
31 PFASs have found wide use in a variety of applications such as in carpets, clothing, paper and
32 packaging to impart dirt, grease, oil, stain, and water repellence, as well as in aqueous fire-fighting
33 foams (AFFFs) etc¹. In an environmental context however, the strong C-F bond means that PFASs
34 are resistant to thermal, chemical and biological degradation² and are capable of bioaccumulation
35 and long-range environmental transport, illustrated by their presence in the Arctic³⁻⁵. Combined
36 with concerns about toxicity⁶, perfluorooctane sulfonate (PFOS) and its salts, as well as
37 perfluorooctane sulfonyl fluoride (POSF) were in 2009 listed as persistent organic pollutants
38 (POPs) under the Stockholm Convention⁷. Moreover, the EU has listed perfluorooctanoic acid
39 (PFOA), perfluorononanoic acid (PFNA), and perfluorohexane sulfonate (PFHxS) as substances
40 of very high concern⁸ and the European Food Safety Authority (EFSA) has published challenging
41 provisional tolerable weekly intake (TWI) values for PFOS and PFOA of 13 ng/kg bw/week and
42 6 ng/kg bw/week respectively⁹. EFSA is currently evaluating the evidence for human health effects
43 arising from exposure to a range of other PFASs. Given these concerns, it is thus important to
44 measure human exposure in order to assess the risk that such exposure presents to human health.
45 Current understanding of the pathways of human exposure to PFASs is that whilst diet is the major
46 pathway for most individuals, indoor air and dust play minor but not insignificant roles¹⁰, while
47 drinking water can also be an important source of exposure to PFASs¹¹. While PFASs were not
48 detected in previous studies of Irish foodstuffs¹² and human milk samples collected in 2010 from
49 Ireland¹³, the detection limits of these surveys were quite high – i.e. 1 µg/kg fresh weight in foods,
50 and 0.5 µg/L and 1.0 µg/L for PFOS and PFOA respectively in human milk – and the quantifiable

presence of PFASs in food and human milk from other western European countries⁹ suggests strongly that the Irish population is exposed to PFASs. Consequently, the objective of this study was to measure concentrations of PFASs in indoor air and settled floor dust from Irish cars, homes, offices, and school classrooms. Concentrations of the same compounds were also measured in samples of tap water from homes and offices as well as in bottled water purchased from supermarkets. As these are the first such data for Ireland, concentrations were compared with those in previous studies in other countries to place Irish data in an international context. In addition, we estimated exposure of Irish adults and toddlers to our target PFASs via drinking water, inhalation, and dust ingestion and compared our exposure estimates with those in other countries and via dietary ingestion as well as with relevant health-based limit values (HBLVs).

Materials and Methods

Sampling strategy and sample collection

Project ethical approval was obtained from the Research Ethics Committee of the National University of Ireland, Galway (Ref 16/May/02). Prior to sample collection, participants completed a questionnaire to gather information on the year of construction of the building or car, along with other factors that could plausibly influence concentrations of PFASs in a sample. These included: type of flooring, presence or absence of child seats in cars, car manufacturer and model, and whether the room/car had been stain proofed etc. Samples of air, dust, and water were collected from three counties: Dublin, Galway, and Limerick, with sample numbers split approximately equally from each county.

Air samples were collected between August 2016 and January 2017 in cars (n=31), homes (living rooms, n=34), offices (n=34) and school classrooms (n=28) using double-bowl passive air

samplers containing an XAD-sorbent impregnated polyurethane (SIP) foam disk (further information is supplied as Supporting Information (SI)). Each sampler was deployed for ~60 days at a height of 1-2 m. Sampling rates (m^3/day) for PFASs are provided as Table SI-1¹⁴. Concentrations of an individual PFAS in air were calculated by dividing the mass of that PFAS detected in the PUF disk by the sampling rate multiplied by the number of days the sampler was deployed.

Dust was collected in the same cars (n=31), homes (n=32), offices (n=33) and classrooms (n=32) between August 2016 and January 2017. Samples were collected at the end of the air sampling period using a standard protocol¹⁵ under normal room/vehicle use conditions to reflect actual human exposure (detailed information provided as SI). Dust was sieved through a pre-cleaned 500 μm mesh sieve, homogenized thoroughly, transferred to clean glass vials and stored at -20 °C until analysis.

Tap water samples from buildings connected to a municipal water supply were collected between October 2016 and January 2017 from the same homes (n=34) and offices (n=32) from which air and dust samples were obtained. Tap water was collected in a glass bottle fitted with a polypropylene lid (Azlon Fisher Scientific). Prior to sampling, the bottles were washed with soap and warm water and rinsed sequentially with acetone, hexane and methanol. In addition, 10 samples of bottled water were purchased from shops in Galway city in late 2016. As data for these preliminary bottled water samples indicated PFASs concentrations in bottled water to exceed that of tap water; further bottled water samples (n=21) were purchased for analysis in May 2018. Additional tap water samples (n=25) were obtained from homes with private water supplies in various locations within the Republic of Ireland in May 2018.

95 **Analytical protocols**

96 Target PFASs in this study were: PFOA, PFOS, PFNA, PFHxS, PFBS, perfluorooctane
97 sulfonamide (FOSA), its methyl and ethyl derivatives (MeFOSA and EtFOSA), as well as methyl
98 and ethyl perfluorooctane sulfonamido ethanols (MeFOSE and EtFOSE).

99 *Extraction and Clean-up*

100 Drinking water samples were processed in accordance with a previously reported method¹⁶
101 (Ericson et al 2008), with dust and air samples extracted and purified in accordance with the
102 procedures reported previously by Goosey and Harrad^{14,15}. Water samples were extracted via solid
103 phase extraction (SPE) using Oasis-WAX cartridges (6 mL, 150 mg, Waters). SPE cartridges were
104 first conditioned with 6 mL methanol (0.1% NH₄OH) followed by 6 mL MilliQ water. Aliquots
105 (500 mL) of samples were spiked with 30 µL of an internal standard solution in methanol
106 containing 1 ng/µL of M8PFOS, M8PFOA, M8FOSA, MPFHxS, MPFNA, d-N-MeFOSA, d-N-
107 MeFOSE (Wellington Laboratories). Where possible, native PFASs were quantified relative to the
108 corresponding isotopically-labeled internal standard, with PFBS, EtFOSA, and EtFOSE quantified
109 relative to MPFHxS, d-N-MeFOSA, and d-N-MeFOSE respectively. Samples were thoroughly
110 mixed and then loaded onto the pre-conditioned SPE cartridges at approximately 1 drop per second.
111 Cartridges were then dried under vacuum for approximately 30 minutes before target PFAS were
112 eluted with 6 mL MeOH (0.1% NH₄OH). Samples were concentrated to approximately 0.5 mL,
113 passed through a 0.2 µm syringe filter, before further concentration to 200 µL and transfer to
114 autosampler vials ready for analysis.

115 For dust, 200 mg was weighed into a clean glass tube and spiked with 30 µL of internal standard
116 solution. 5 mL of hexane:acetone (1:1, v/v ratio) was added and samples vortexed for 2 minutes,

prior to 30 minutes ultrasonication. Tubes were centrifuged for 5 minutes at 3500 RPM and supernatant collected into a separate glass tube. This procedure was repeated twice and all three extracts combined. Crude extracts were concentrated to near-dryness and reconstituted in 1 mL hexane:acetone (1:1, v/v ratio). 50% of the extract was retained for analysis of brominated flame retardants (BFRs)¹⁷, while the rest was solvent exchanged into 0.5 mL methanol.

Air samples were extracted via pressurized liquid extraction (PLE) on an ASE 350 (Dionex). XAD-3 coated PUFs were packed into 66 mL extraction cells using clean forceps, spiked with 30 µL of internal standards and extracted with acetone at 90 °C under 1500 psi pressure, with a heating time of 5 minutes, static time of 4 minutes and a flush volume of 40% with 3 static cycles. Extracts were transferred to glass tubes, concentrated to near-dryness on a Turbovap II (Zymark). Following removal of 50% of the crude extract for analysis of BFRs¹⁷, extracts were reconstituted in 0.5 mL methanol.

Both air and dust sample extracts required further purification. Crude extracts were loaded onto an ENVI-Carb SPE cartridge (3 mL, 500 mg, Sigma Aldrich), pre-conditioned with 6 mL methanol (0.1% NH₄OH) and 3 mL methanol. Target PFASs were eluted with 3 mL of methanol (0.1% NH₄OH). Eluates were concentrated to approximately 0.5 mL, passed through a 0.2 µm syringe filter, concentrated to 100 µL and transferred to autosampler vials ready for analysis.

Chemical Analysis

PFASs were analyzed on a Sciex Exion HPLC coupled to a Sciex 5600+ triple TOF MS. Ten microliters of extract were injected onto a Raptor C18 column (1.8 µm particle size, 50 mm length, 2.1 mm internal diameter, Restek). Details of the LC conditions employed are provided as Table SI-2. The TOF-MS is equipped with a Turbo V source operated in negative mode using

electrospray ionization at a voltage of -4,500 V. The curtain gas and nebulizer gas (source gas 1) were both 25 psi, whilst the drying gas (source gas 2) was 35 psi. The CAD gas was set to medium and temperature was 450 °C. Mass spectrometric data was acquired using automatic information dependent acquisition (IDA) with two experiment types: (i) survey scan, which provided TOF-MS data; and (ii) dependent product ion scan using a collision energy of -40V and a collision a spread of 30 V. Individual PFAS was quantified in Multiquant 2.0 using MS/MS transitions and retention time for identification (Table SI-3).

Quality Assurance/Quality Control

A reagent blank (comprising either Na₂SO₄ or Milli-Q water according to sample type) was analysed with every batch of 10 samples. None of the target compounds were detected above 5% of the concentration of samples, therefore no blank correction was necessary. Limits of quantification (LOQs) were estimated based on a signal to noise ratio of 10:1 in the lowest level calibration standard.

For air and dust, every 20th sample analysed was an aliquot of SRM-2585 (NIST) – a house dust which has been previously analysed for the majority of target compounds (n = 14)¹⁸. Good agreement with previously published values was obtained (see Table SI-4). For water samples, the method was validated by spiking 500 mL MilliQ samples (n=6) with target compounds and analysing for target PFAS. All target compounds were recovered at 80-120% of their spiked concentrations with <15% relative standard deviation.

Statistical Analysis

Statistical analysis was conducted using Excel (Microsoft Office for Mac) and IBM SPSS Statistics for Mac 25.0 (Chicago, IL, U.S.A.). ANOVA analyses were followed by a Scheffe post hoc test.

For calculating descriptive statistics, where analyte peaks <LOD, concentrations of target compounds were assumed to equal $df \times LOD$, where df = the detection frequency for that compound expressed as a fraction. Statistical analysis of air and dust data was performed on log-transformed concentrations, as concentrations in these data sets were revealed to be log-normally distributed using Kolmogorov-Smirnov test and visual inspection of quantile-by-quantile graphic plots. Concentrations of PFASs in water samples displayed a normal distribution and thus were analysed without transformation. A p -value <0.05 was considered significant.

Results and discussion

Concentrations of PFASs in drinking water

Except for FOSA, EtFOSA, and EtFOSE, all target PFASs were detected in Irish drinking water (Table 1). The most frequently detected was PFOA ($df > 83\%$ in all water categories). PFNA was also detected in all three water types ($df > 19\%$). All other target PFASs were not detected in at least 1 water type. Also included in Table 1 are median concentrations of PFASs in drinking water in a range of previous studies from elsewhere in the world – with further comparative data available elsewhere³³. This comparison shows concentrations in Irish tap water are amongst the lowest reported to date for all of our target PFASs. With respect to bottled water, fewer comparative data exist, but those reveal concentrations in Ireland to be in the middle of the range reported worldwide. We compared our data on PFASs in drinking water against two stringent drinking water guidelines. The first of these was promulgated by Swedish authorities and specifies a limit of 90 ng/L for Σ PFASs (which include some of those targeted in our study)⁴². Inspection of our data reveals this limit is not exceeded for any sample in our study but was approached in one sample of bottled water where Σ PFASs = 64 ng/L. Moreover, the US EPA has specified a health-based limit value

of 70 ng/L for the sum of PFOS and PFOA concentrations^{43,44}, but in no sample in this study did PFOA and PFOA concentrations approach this limit. While this comparison with current limit values is reassuring, a recent study suggested much lower benchmark concentrations of 1 ng/L for PFOS and PFOA in drinking water based on immunotoxic effects in children⁴⁵. Consequently, while this lower benchmark concentration lacks legislative authority, it is exceeded for a small number of samples in our study and continued monitoring of PFASs in drinking water is advised.

Comparison between bottled water and tap water from municipal and private water supplies

Using ANOVA, we examined our data for significant ($p \leq 0.05$) differences in concentrations of individual target PFASs in: (a) tapwater from homes and offices connected to municipal water supplies, (b) tapwater from homes connected to private water supplies, and (c) bottled water. This analysis revealed: (i) concentrations of PFOA in tap water from private supplies exceeded significantly those in tap water from municipal supplies, (ii) those of PFOS and PFBS in bottled water exceeded those in tapwater from both municipal and private supplies, and (c) MeFOSA concentrations in tapwater from private supplies exceeded those in tapwater from municipal supplies. We are unable to explain these differences in concentrations of some PFASs between different sample types.

Concentrations of PFASs in indoor air

All target PFASs were detected in indoor air from Irish cars, classrooms, homes and offices (Table 2). The most frequently detected PFAS was PFOA (detection frequency (df) >85% in all microenvironment categories), followed by MeFOSE (df>64%), PFBS and PFOS (df>41% for

both), and PFNA (df=18% in homes but >90% in the other three microenvironment categories). FOSA, EtFOSA, and EtFOSE were infrequently detected. In terms of concentrations, PFOA again predominated (median >56 pg/m³ in all microenvironments), with only PFOS and PFBS also present at median concentrations >10 pg/m³ in any microenvironment category (13 and 21 pg/m³ in cars). Table 2 also provides data from selected other studies worldwide. To our knowledge, our data are the first for PFASs in air from car interiors. The majority of previous data exist for homes. In general, our data are at the low end of those for domestic air, with the notable exception of PFOA, for which the median concentration in Irish homes exceeds that reported in the 5 other studies reporting concentrations of PFOA in home air. While we could find only two other studies reporting concentrations of PFASs in office air; our data for Ireland are lower than these other studies, with the exception of PFNA and PFOA where Irish median concentrations are highest. Our study appears the first report of PFOA, PFOS, PFBS, PFHxS, and PFNA in school classroom air; EtFOSE and MeFOSE were measured (but both below the limit of detection) in German school classrooms²⁰, while data were reported for a good range of PFASs in Czech University classrooms²⁶. In general, our data are not markedly dissimilar to those of the Czech study for most target PFASs, but as with all other microenvironments studied here, the median concentration of PFOA is higher in Irish classrooms.

Concentrations of PFASs in indoor dust

Table 3 summarizes the concentrations of target PFASs in dust from Irish cars, classrooms, homes and offices. The most frequently detected PFAS was PFBS (df>75% in all microenvironment categories), followed by PFOA (df>66%) and PFOS (df>53%). EtFOSA and MeFOSA were detected only rarely and not at all in office dust. PFBS displayed the highest median concentrations in all microenvironment categories, followed by PFOS in home and office dust and PFOA in car

and classroom dust. Table 3 provides median concentrations from selected previous studies to provide context for our data. As with indoor air, most data exist for homes, with concentrations in Irish homes amongst the lowest worldwide. While fewer previous data exist for other microenvironment categories, concentrations in dust from Irish cars, offices, and classrooms are at the low end of the range reported elsewhere.

Factors influencing concentrations of PFASs in indoor air and dust

We inspected the questionnaire data provided by the sample donors for insights into possible influences on concentrations of PFASs in our indoor air and dust samples. To do so, we examined linear correlations between the year of building/car construction and log-transformed PFAS concentrations. In addition, we used t-tests of log-transformed data to evaluate whether PFASs concentrations in buildings/cars built before 2005 differed from those built post-2005. We also used a t-test to compare log-transformed PFAS concentrations in samples containing a putative source (e.g. carpet³¹, child seat, application of stain proofing agents etc.) with other samples where the putative source was not present. Further, we employed ANOVA with a post hoc Scheffe test to evaluate whether the county (Dublin, Galway, or Limerick) from which samples from a given microenvironment were taken, exerted a significant influence on concentrations of PFASs. Likely due to the multiple influences on PFAS concentrations, we found only one significant difference. This was for car air, with concentrations of PFOA, PFNA significantly higher ($p \leq 0.05$) in cars containing child seats ($n=12$) than those that did not ($n=17$). Moreover, when a sample from car not containing a child seat that contained an unusually elevated concentration of MeFOSE (160 pg/m^3) was excluded; concentrations of MeFOSE were significantly ($p \leq 0.05$) higher in air from cars containing child seats. Specifically, median concentrations for PFOA were 242 and 63 pg/m^3 in cars containing child seats and in those without respectively, with the corresponding data for

PFNA and MeFOSE being 5.7 and 1.5 pg/m³, and 6.9 and 1.3 pg/m³ respectively. This may indicate the use of these PFASs to stain proof the fabrics used on such child car seats. While we are unaware of direct evidence of the application of PFAS to stain proof child car seats, application of PFASs to stain proof fabrics is well documented². As a caveat, we note that we observed no significant difference in concentrations of any of our target PFASs in dust from cars regardless of the presence or absence of a child seat, although the absence of any difference for dust may be due to the lower concentrations and detection frequencies for PFOA, PFNA, and MeFOSE in dust compared to air. Alternatively, it is possible that the elevated concentrations in air from cars with child seats may arise from the use of volatile precursors of PFOA and PFNA that undergo degradation to the parent PFAS on the PUF disk during sampling.

Comparisons between indoor microenvironments

Our previous studies of UK indoor air and dust revealed differences in concentrations of PFASs between different microenvironment categories^{14,15}. Such differences are likely due to the different types and abundance of PFAS sources in these different types of microenvironment. We therefore examined our data for such differences.

Indoor air

We compared log-transformed concentrations of PFASs in Irish car, classroom, home and office air using ANOVA followed by a Scheffe post hoc test. The following significant ($p < 0.05$) differences were detected: (a) concentrations of PFHxS in office air exceed those in all other microenvironment categories, and (b) for PFOS, concentrations in classroom air exceeded that in homes. No other significant differences in concentrations of PFASs in different microenvironments were detected ($p > 0.05$).

273 *Indoor dust*

274 For indoor dust, statistical analysis revealed log-transformed concentrations of PFBS in classroom
275 dust exceeded significantly those in cars. In addition, concentrations of PFNA were significantly
276 higher in offices than in classrooms ($p \leq 0.05$). No significant differences in concentrations between
277 different microenvironments were found for other PFASs.

278

279 **Exposure to PFASs via drinking water, inhalation, and dust ingestion**

280 Exposure of the Irish population to PFASs via drinking water, inhalation and dust ingestion was
281 estimated for adults and young children based on concentrations reported here. The algorithms and
282 assumptions applied to estimate exposure via different routes and under three scenarios of low-
283 end, “typical”, and high-end exposure are provided as supporting information. These data facilitate:
284 (a) evaluation of the relative importance of different exposure pathways for different chemicals,
285 and (b) risk assessment by comparison of exposure with existing or future health-based limit values.
286 Table 4 summarises exposures for our target PFASs via all three pathways combined. It is
287 important to bear in mind that these represent central estimates and that individual exposures may
288 vary substantially around these, depending on factors such as age and behavioural traits like hand-
289 to-mouth activity. To place these exposure estimates in context, we compare them with previously
290 reported estimates of dietary exposure for other European countries combined⁹ and the UK⁴⁶, in
291 the absence of such data currently for Ireland. Table 4 also expresses the relative percentage
292 contribution of each pathway for both adults and toddlers under our typical exposure scenarios for
293 each pathway. In addition, these data are illustrated graphically for the four target PFASs for which
294 typical total exposure is highest, i.e. PFOA, PFOS, PFBS, and MeFOSA (Figure 1). It is striking
295 that for young children, drinking water is the major pathway (>70% of the three pathways

considered in this study) for 6 out of our 10 target PFASs for the three pathways monitored in this study. Likewise, drinking water is the most important exposure pathway (>65% total exposure) for adults. An important caveat to this, is that for the 2 PFASs of highest current toxicological concern – PFOS and PFOA; inhalation and dust ingestion contribute substantially to exposure. Moreover, dust ingestion is the principal contributor to non-dietary exposure of children to PFBS.

It is important to note that compared to estimates of dietary exposure for European countries, the three exposure pathways studied here constitute <10% of overall exposure under typical exposure scenarios. Moreover, even our high-end estimates of exposure via the non-dietary sources assessed here are lower than typical estimates of dietary exposure in other European countries^{9,46}. We also compared our estimates of non-dietary exposure with EFSA's provisional tolerable weekly intake values of 6 ng/kg bw/week for PFOA and 13 ng/kg bw/week for PFOS⁹. Crucially, even our high-end estimates of Irish non-dietary exposure to both PFOA and PFOS are below these provisional EFSA TWI values. In the absence currently of estimates of dietary exposure of the Irish population to PFOA and PFOS, we cannot definitively assess whether the EFSA TWI values would be exceeded for Irish adults and children when dietary intake is added to our exposure estimates. However, the data on dietary exposure in other European countries suggests that the overall exposure of the Irish population may exceed the TWI values for PFOA and PFOS for some individuals. Finally, we note that the highest non-dietary exposure estimates in our study are for PFBS, for which no health-based limit value currently exists, and thus EFSA's on-going assessment of the risks to human health from PFBS and other PFASs is welcome.

The limitations of this study are the convenience nature of the sampling. Thus, the samples analysed are not necessarily representative of Ireland. In addition, samples taken represent a snapshot of contamination at a particular point in space and time. Its strengths are that it is one of

319 the most comprehensive assessments of non-dietary human exposure to a range of PFASs
320 anywhere to date, and provides the first data worldwide on PFOS, PFHxS, PFBS, and PFNA in air
321 inside cars and school classrooms. It also highlights significantly higher concentrations in air of
322 PFOA, PFNA and MeFOSE in cars containing child seats than those without. Non-dietary
323 exposure in this study is well within current health based limit values for PFASs. However, when
324 added to dietary exposure, the contribution of such non-dietary exposure may lead to exceedances
325 of the provisional EFSA TWI values for PFOS and PFOA⁹ and possible future TWIs for other
326 PFASs such as PFBS.

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335 **Author Contributions**

336 The manuscript was written through contributions of all authors. All authors have given approval
337 to the final version of the manuscript.

338 **Acknowledgments**

339 This project (ELEVATE, reference 2016-HW-MS-8) is funded under the EPA Research
340 Programme 2014-2020. The EPA Research Programme is a Government of Ireland initiative
341 funded by the Department of Communications, Climate Action and Environment. We gratefully
342 acknowledge all the participants for their permission to collect air, dust, and water samples.

343 **Supporting Information**

344 Tables reporting air sampling rates, LC and MS conditions, as well as method accuracy data;
345 along with details of the algorithms and assumptions used to estimate human exposure are
346 provided as supporting information. This material is available free of charge via the Internet at

347 <http://pubs.acs.org>.

Figures

Figure 1. Relative contribution (expressed as %) of different target PFASs to the overall daily exposure (ng/day) of Irish toddlers and adults via drinking water, inhalation and dust ingestion under typical exposure scenarios

354

355

356 Tables

357 **Table 1: Descriptive Statistics for Concentrations (ng/L) of target PFASs in Irish drinking**
 358 **water (only those with DF>20% in at least one sample type shown)**
 359

	PFOA	PFOS	PFBS	MeFOSA	PFNA	MeFOSE
Tapwater (mains supply)						
DF (% , this study)	83	6.0	8.0	0	27	0
Minimum (this study)	0.04	<0.15	<0.2	<0.2	<0.05	<0.5
Median (this study)	0.23	<0.15	<0.2	<0.2	<0.05	<0.5
Average (this study)	0.31	<0.15	0.52	<0.2	<0.05	<0.5
Maximum (this study)	1.76	0.76	15.06	<0.2	0.42	<0.5
Turkey median^{33 a}	0.19	0.28	0.25	-	0.13	-
France median³⁴	3	5	<1	-	<1	-
USA median³⁵	4.2	1.6	1.2	-	0.74	-
Netherlands median³⁶	4.0	1.3	7.3	-	<0.5	-
Catalonia, Spain median¹⁶	0.65	0.41	<0.27	-	<0.42	-
Central Europe median³⁷	2.6	1.3	2.7	-	1.4	-
Canada median³⁸	0.31	0.64	0.16	-	0.15	-
Brazil median^{39 a}	10	5.8	1.3	-	12	-
Australia median⁴⁰	<0.5	<0.66	<0.14	-	-	-
China arithmetic mean^{41 b}	0.02-61	0.06-190	0.03-7.8	-	0.03-20	-
Tapwater (private supply)						
	PFOA	PFOS	PFBS	MeFOSA	PFNA	MeFOSE
DF (% , this study)	100	0	0	56	48	0
Minimum (this study)	0.35	<0.15	<0.2	<0.2	<0.05	<0.5
Median (this study)	0.61	<0.15	<0.2	<0.2	<0.05	<0.5
Average (this study)	0.59	<0.15	<0.2	0.30	0.08	<0.5
Maximum (this study)	1.3	<0.15	<0.2	2.7	0.49	<0.5
Bottled water						
	PFOA	PFOS	PFBS	MeFOSA	PFNA	MeFOSE
DF (% , this study)	87	29	29	19	19	42
Minimum (this study)	<0.05	<0.15	<0.2	<0.15	<0.15	<0.02
Median (this study)	0.44	<0.15	<0.2	<0.15	<0.15	0.03
Average (this study)	0.45	0.50	3.7	<0.15	<0.15	0.05
Maximum (this study)	1.3	7.1	51	0.8	0.2	0.1
Turkey median^{33 a}	0.10	<LOD	0.20	-	0.15	-
Catalonia, Spain median¹⁶	0.34	<0.24	<0.27	-	<0.42	-
Central Europe median³⁷	1.6	1.5	2.6	-	3.0	-
Canada median³⁸	<0.07	<0.03	<0.02	-	<0.03	-
Brazil arithmetic mean^{39 a}	7.6	<1.2	3.5	-	10	-

360 ^a only concentrations >limit of detection used to calculate median concentrations

361 ^b range of arithmetic mean concentrations at a number of sampling locations

362 **Table 2. Descriptive Statistics for Concentrations (pg/m³) of target PFASs in Irish indoor**
363 **air (only those with DF>20% in at least two microenvironment categories shown)**

	PFOA	FOSA	PFHxS	PFOS	PFBS	EtFOSE	PFNA	MeFOSE
Homes								
DF (% , this study)	85	41	21	41	53	24	18	71
Minimum (this study)	<0.3	<0.2	<0.4	<0.4	<0.4	<0.2	<0.3	<0.2
Median (this study)	56	<0.2	<0.4	<0.4	1.0	<0.2	1.7	3.9
Average (this study)	72	0.62	<0.4	14	22	2.2	2.1	14
Maximum (this study)	386	9.0	0.46	208	270	38	13	158
UK median¹⁴	24	45	23	11	-	540	-	760
Norway, median¹⁹	-	-	-	-	-	78	-	265
Germany, median²⁰	-	-	-	-	-	66	-	217
Canada median²¹	21	-	-	<0.02	-	56	89 (average)	320
Korea, median²²	-	-	-	-	-	59	-	89
Finland, median²³	15	-	<0.52	1.9	<1.0	17	2.4	56
Australia, median²⁴	14	-	4.3	9.7	1.3	-	3.0	-
Nepal, median²⁴	<2	-	<2	<2	<2	-	<2	-
Cars								
	PFOA	FOSA	PFHxS	PFOS	PFBS	EtFOSE	PFNA	MeFOSE
DF (% , this study)	100	29	23	94	90	26	90	74
Minimum (this study)	1.2	<0.2	<0.4	<0.4	<0.4	<0.2	<0.3	<0.2
Median (this study)	76	<0.2	<0.4	13	21	<0.2	2.1	2.9
Average (this study)	162	0.53	0.15	22	54	0.69	5.2	13
Maximum (this study)	790	7.9	0.55	152	264	6.0	24	160
Offices								
	PFOA	FOSA	PFHxS	PFOS	PFBS	EtFOSE	PFNA	MeFOSE
DF (% , this study)	91	47	44	65	41	29	91	68
Minimum (this study)	<0.3	<0.2	<0.4	<0.4	<0.4	<0.2	<0.3	<0.2
Median (this study)	96	<0.2	<0.4	8.9	0.16	<0.2	2.5	3.6
Average (this study)	153	3.6	0.40	89	37	4.9	3.7	52
Maximum (this study)	1210	58	1.4	1290	313	94	18	714
UK median¹⁵	18	59	84	55	-	420	-	310
Belgium, median²⁵	2.9	-	0.2	2.2	0.2	-	0.4	-
Classrooms								
	PFOA	FOSA	PFHxS	PFOS	PFBS	EtFOSE	PFNA	MeFOSE
DF (% , this study)	89	29	25	64	54	18	93	64
Minimum (this study)	<0.3	<0.2	<0.4	<0.4	<0.4	<0.2	<0.3	<0.2
Median (this study)	89	<0.2	<0.4	9.3	2.2	<0.2	2.5	1.9
Average (this study)	210	0.24	<0.4	188	36	1.3	3.5	12
Maximum (this study)	728	1.3	2.3	1590	202	16	15	82
Germany median²⁰	-	-	-	-	-	<LOD	-	<LOD
University classrooms Czech Republic, median²⁶	5.3	0.93	0.70	2.0	0.41	3.2	1.8	5.8

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Table 3. Descriptive Statistics for Concentrations (ng/g) of target PFASs in Irish indoor dust (only those with DF>20% in at least two microenvironment categories shown)

	PFOA	PFHxS	PFOS	PFBS	PFNA	MeFOSE
Homes						
DF (% , this study)	66	47	63	81	9.0	31
Minimum (this study)	<0.05	<0.1	<0.1	<0.25	<0.05	<0.1
Median (this study)	0.42	<0.1	0.96	10	<0.05	<0.1
Average (this study)	4.7	1.4	6.0	17	0.52	1.9
Maximum (this study)	83	9.9	140	110	14	42
UK median¹⁵	190	210	140	-	-	220
Belgium, Italy, Spain median²⁷	1.4	0.13	0.28	0.40	0.04	-
Czech Republic median²⁸	2.0	2.0	10	<LOD	<LOD	<LOD
Canada median²⁸	8.2	1.9	9.1	<LOD	4.4	<LOD
USA median²⁸	9.0	8.7	14	0.9	3.9	1.0
S. Korea median²⁹	4.5	0.0	11	0.3	1.4	2.0
Cars						
	PFOA	PFHxS	PFOS	PFBS	PFNA	MeFOSE
DF (% , this study)	84	47	69	75	41	31
Minimum (this study)	<0.05	<0.1	<0.1	<0.25	<0.05	<0.1
Median (this study)	1.8	<0.1	1.3	3.6	0.05	<0.1
Average (this study)	3.2	6.2	7.6	12	0.55	0.63
Maximum (this study)	14	49	82	170	3.1	4.2
UK median¹⁵	65	180	97	-	-	82
Sweden median³⁰	33	-	12	-	-	-
USA geometric mean³¹	11	Not quantified (NQ)	16	<LOD	15	NQ
Offices						
	PFOA	PFHxS	PFOS	PFBS	PFNA	MeFOSE
DF (% , this study)	69	44	81	88	34	31
Minimum (this study)	<0.05	<0.1	<0.1	<0.25	<0.05	<0.05
Median (this study)	0.95	<0.1	2.0	8.1	<0.05	<0.05
Average (this study)	23	2.7	91	19	8.6	27
Maximum (this study)	380	57	2700	98	120	740
UK median¹⁵	290	170	230	-	-	220
Sweden median³⁰	70	-	110	-	-	-
USA geometric mean³¹	32	NQ	15	NQ	63	NQ
Belgium median²⁵	2.9	0.2	2.2	0.2	0.4	-
Classrooms						
	PFOA	PFHxS	PFOS	PFBS	PFNA	MeFOSE
DF (% , this study)	75	38	53	97	6.0	22
Minimum (this study)	<0.05	<0.1	<0.1	<0.25	<0.05	<0.1
Median (this study)	0.46	<0.1	0.39	15	<0.05	0.02
Average (this study)	2.2	5.1	3.1	17	<0.05	0.57
Maximum (this study)	31	120	21	49	0.71	5.3
UK median¹⁵	240	700	840	-	-	660
Sweden median³²	7.7	<0.3	49	<0.5	1.1	-

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Table 4. Estimates of exposure (pg/kg body weight/day) of Irish Adults and Young Children to PFASs via non-dietary sources (i.e. air, dust, and drinking water combined), relative significance (%) of each pathway under a typical exposure scenario^a, and comparison with European dietary exposure estimates

	PFOA	FOSA	PFHxS	PFOS	PFBS	EtFOSA	MeFOSA	PFNA	MeFOSE
Adult Low non-dietary sources^b	1.4	2.9	0.39	0.57	0.77	0.01	0.81	0.39	0.17
Adult Typical non-dietary sources	30	2.9	0.57	1.6	3.8	0.01	1.1	1.6	4.1
Adult High non-dietary sources^c	132	5.5	9.9	71	282	1.2	15	18	49
Child Low non-dietary sources^b	4.7	10	1.4	1.9	5.7	0.03	2.9	1.3	0.53
Child Typical non-dietary sources	53	10	2.0	4.9	51	0.04	3.8	4.1	11
Child High non-dietary sources^c	329	19	102	227	1252	3.5	110	26	69
EFSA Provisional TWI⁹	857	-	-	1857	-	-	-	-	-
% Air Adult (Child)	62.5 (22.7)	0.8 (0.1)	5.3 (0.8)	48.7 (9.8)	12.3 (0.8)	84.6 (14.2)	0.2 (0)	33.8 (8.7)	26.3 (5.8)
% Dust Adult (Child)	0.6 (3.6)	0.1 (0.2)	2.3 (8.3)	21.1 (55.2)	70.2 (94.7)	15.4 (85.8)	0 (1.7)	0.2 (0.5)	0.2 (0.9)
% Water Adult (Child)	36.9 (73.7)	99.1 (99.6)	92.4 (90.9)	30.2 (35)	17.4 (4.5)	0 (0)	99.8 (98.3)	66 (90.8)	73.5 (93.3)
Typical^d Dietary exposure Adult Europe⁹	320	-	-	610	-	-	-	-	-
Typical^d Dietary exposure Toddlers Europe⁹	2010	-	-	750	-	-	-	-	-
Typical Dietary exposure Adult UK⁴⁶	3900	-	-	1800	-	-	-	-	-
Typical Dietary exposure toddlers (1-4.5 years) UK⁴⁶	9600	-	-	4500	-	-	-	-	-

373 ^a typical exposure scenario assumes adults and toddlers inhale air and ingest dust contaminated at the median
374 concentration and assuming an average dust ingestion rate (20 mg/day and 50 mg/day for adults and toddlers
375 respectively¹⁵)

376 ^b low exposure scenario assumes adults and toddlers inhale air and ingest dust contaminated at the 5th percentile
377 concentration and assuming an average dust ingestion rate (20 mg/day and 50 mg/day for adults and toddlers
378 respectively¹⁵)

379 ^c high exposure scenario assumes adults and toddlers inhale air and ingest dust contaminated at the 95th percentile
380 concentration and assuming a high dust ingestion rate (50 mg/day and 200 mg/day for adults and toddlers
381 respectively¹⁵)

382 ^d median lower bound estimates

383 N.B. Typical UK dietary exposure to ΣPFASs in 2012 was 60000 pg/kg body weight/day and 1400000 pg/kg body
384 weight/day for adults and toddlers (1-4.5 years) respectively⁴⁶

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532 For table of contents only (TOC art)

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