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# Concentrations of Perfluoroalkyl substances in human milk from Ireland: Implications for adult and nursing infant exposure

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# Abstract

Concentrations of 10 perfluoroalkyl substances (PFASs) were measured in 16 pools of human milk from Ireland. Only four PFASs were detected (PFOA, PFNA, PFHxS and PFOS), with concentrations dominated by PFOA which was detected in all samples at a median of 0.10 ng/mL. Concentrations and the relative abundance of PFASs in Ireland are within the range reported for other countries. Estimated exposures for nursing infants to perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) do not suggest a health concern. A one compartment pharmacokinetic model was used to predict the intakes of PFOS and PFOA required to support the observed concentrations in human milk. This suggests current adult exposure in Ireland to PFOS is below the provisional tolerable weekly intake (TWI) proposed by EFSA. In contrast, the model predicts that the maximum concentration detected in human milk in this study, implies a level of adult exposure that would exceed EFSA's provisional TWI for PFOA. As exposure of the Irish population to PFASs via drinking water, indoor air and dust is well-characterised, current understanding suggests that the major contributor to overall exposure of the Irish population is via the diet and/or less well-studied pathways like dermal uptake from PFAS-containing fabrics and cosmetics.

## Highlights

- PFOA, PFOS, PFNA, and PFHxS detected in Irish human milk
- Concentrations within the range of studies elsewhere
- Exposures of nursing infants to PFOS and PFOA not of health concern
- Modelled adult intakes of PFOA in some instances exceed provisional EFSA TWI
- Measurement of Irish exposure via the diet and dermal uptake recommended

**Keywords**

Human biomonitoring

PFASs

PK modelling

PFOS

PFOA

## Introduction

Perfluoroalkylated substances (PFAS) is a collective term for a large group of fluorinated compounds, including perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). PFOS and PFOA were widely used for stain proofing and water resistant coatings for fabrics and carpets, paper products (including food grade products), and firefighting foams (Buck et al, 2011). Although imparting beneficial longevity in the context of their commercial application, the strength of the C-F bond renders PFASs resistant to thermal, chemical and biological degradation and capable of bioaccumulation and long-range environmental transport, exemplified by their detection in the Arctic (Chaemfa et al, 2010; Sonne, 2010; Zhao et al, 2012). Coupled with toxicological concerns (Lindstrom et al, 2011), such properties have resulted in PFOS and its salts, as well as perfluorooctane sulfonyl fluoride (POSF) being listed as persistent organic pollutants (POPs) under the United Nations Environment Programme's Stockholm Convention in 2009 (Stockholm Convention, 2009). Currently, PFOA is recommended for listing under this Convention, while the $C_6$ analogue of PFOS - perfluorohexane sulfonate (PFHxS) - is under review for listing, and a potential proposal exists at the EU level to consider for listing, $C_{10}$-$C_{14}$ analogues of PFOA (including perfluorononanoic acid (PFNA) and its salts. Moreover, the European Union has identified PFOA, PFNA, and PFHxS as substances of very high concern (ECHA, 2019), while the European Food Safety Authority (EFSA) has promulgated provisional tolerable weekly intake (TWI) values for PFOS and PFOA of 13 ng/kg bw/week and 6 ng/kg bw/week respectively (EFSA, 2018). Furthermore, EFSA is currently evaluating the evidence for human health effects arising from exposure to a range of other PFASs.

Current understanding of the pathways of human exposure to PFASs is that whilst diet constitutes the principal pathway for most individuals, indoor air and dust play minor but potentially significant roles (Harrad et al, 2010), with drinking water representing a

69 potentially important additional source of exposure to PFASs (Jian et al, 2017). As part of the
70 ELEVATE project funded by the Environmental Protection Agency of Ireland, we recently
71 reported concentrations of brominated flame retardants (BFRs), PFOS, PFOA, PFHxS,
72 PFNA, and other PFASs in drinking water, and in indoor air and dust from cars, homes,
73 offices and school classrooms in the Republic of Ireland (Harrad et al, 2019b; Wemken et al,
74 2019). *Inter alia*, by multiplying our data on concentrations of PFASs by exposure factors
75 (e.g. daily air inhalation rates etc), we evaluated the relative contribution of these different
76 exposure pathways of PFASs. An alternative approach to elucidating the relative significance
77 of different exposure pathways is the application of simple pharmacokinetic (PK) models.
78 Such models have been used to predict the body burdens of PFOS and PFOA in Australians
79 based on intake data from different exposure pathways (Thompson et al, 2010). Comparison
80 of these predicted body burdens with observed body burdens for the population in question
81 highlight discrepancies between predicted and observed body burdens and facilitate
82 identifications of gaps in understanding that might account for such discrepancies. Moreover,
83 they may also be employed to derive estimates of exposure via a specific pathway about
84 which data are lacking, provided that body burdens are known, and other exposure pathways
85 are well-characterised.
86 While a previous study measured concentrations of PFOS and PFOA in human milk samples
87 collected in 2010 from Ireland (Pratt et al, 2013); the detection limits of this study were quite
88 high – i.e. 0.5 ng/mL and 1.0 ng/mL for PFOS and PFOA respectively in human milk. As a
89 consequence, neither PFOS nor PFOA were detected in any of the 11 pooled samples
90 analysed, thereby limiting the application of these data in a PK model. In the current study,
91 we therefore collected samples of human milk from 92 Irish primiparas, and pooled these to
92 provide 16 samples which were analysed for concentrations of PFASs. It is important to note
93 that the previous study of human milk in Ireland also provided data on concentrations of

94 brominated flame retardants (BFRs) in pooled samples (Pratt et al., 2013). Comparability of
95 the design of the current study with this previous study was thus necessary to facilitate
96 elucidation of temporal trends in BFR concentrations in human milk in Ireland (Wemken et
97 al, 2020). Hence, while analysis of individual human milk samples can reveal different
98 information to analysis of pooled samples, coupled with the fact that the PK model of
99 Thompson et al (2010) used estimates of PFAS body burdens derived from measurements in
100 blood serum (as the most widely used human biomarker of PFAS exposure); we adapt this
101 PK model to make use of our concentrations in human milk. Specifically, given that no
102 estimates exist of the dietary exposure of the Irish population, we apply the model here in
103 conjunction with our data on human milk and our previously-reported estimates of non-
104 dietary exposure. In this way, we predict the level of exposure required to support our
105 observed human milk concentrations and by subtracting non-dietary exposure, derive
106 estimates of the maximum level of dietary exposure. Moreover, given the elevated detection
107 limits achieved in the previous study of PFASs in Irish human milk, our study constitutes in
108 effect the first such data for Ireland, and the concentrations detected are compared with those
109 in previous studies in other countries to place Irish data in an international context. Our data
110 on PFASs in human milk are also interpreted to provide insights into the exposure of nursing
111 infants to PFASs in Ireland.
112
113 **MATERIALS AND METHODS**
114 **Human milk sample collection**
115 With slight deviations, human milk sampling and donor recruitment in this study was
116 conducted in accordance with the 4$^{th}$ WHO UNEP guidelines for developing a survey of
117 human milk for persistent organic pollutants (WHO (World Health Organisation), 2007) and
118 was consistent with procedures followed in a previous study of PFASs and BFRs in Irish

119 human milk (Pratt et al., 2013). Study protocols and design were approved by the Clinical
120 Research Ethics Committee of the Galway University Hospital (Ref: C.A. 1578) and the
121 Research Ethics Committee of the Coombe Womens and Infants University Hospital in
122 Dublin (No. 30-2016).
123 Breast milk samples were collected between 3 to 8 weeks postpartum from primiparas who
124 were in good health and exclusively feeding one infant. Participants were required to have
125 resided at their present address for a minimum of five years before sample collection. While
126 WHO Guidance stipulates that participants should be not older than 30 years; in Ireland, 65%
127 of primiparas are aged 30 – 40 years old (Central Statistics Office, 2018), and thus
128 recruitment selection criteria were amended allow recruitment of mothers up to and including
129 40 years of age. This was consistent with the previous Irish study that included mothers up to
130 and including 41 years old (Pratt et al., 2013). Eligible participants signed a consent form and
131 filled out a questionnaire to provide contextual information.
132
133 Mothers were recruited when attending breast feeding clinics at the same two Irish maternity
134 hospitals from which mothers were recruited in the study of Pratt et al (2013), namely
135 University Hospital Galway (UHG) and the Coombe Womens and Infants University
136 Hospital (Coombe), Dublin. Breast milk samples of between 30 and 60 mL were collected
137 from each participant in clean polypropylene bottles and stored at – 18 °C until analysis.
138 In total, 92 breast milk samples were collected (UHG n=59; Coombe n=33). Samples were
139 thawed at room temperature and vortexed to homogenise before pooling in equal parts by
140 volume. Contextutal data provided by the mothers in response to the study questionnaire (see
141 Supplementary Data) were used to inform the creation of sixteen sample pools depending on
142 their place of birth (Ireland, UK, EU, or non-EU), place of residence for the last five years
143 (urban or rural) with two pools created that comprised samples from mothers indicating that

144 they consumed fish at least twice a week (fish-consumer pools). Each pool contained aliquots
145 of 30 mL of milk from each individual constituent sample (15 mL for the fish-consumer
146 pools as there was less milk available from the individual donors to these pools), with the
147 number of individual samples per pool ranging between 3 and 10. Following pooling, milk
148 was freeze dried at -50 °C for 72 hours (using a Christ beta 1-8 LSC plus freeze drier) to
149 prepare for analysis.

150 **Sample preparation and analysis**

151 **Extraction & Clean-up**

152 Extraction of breast milk samples was performed based on methods previously published by
153 Kärrman et al. (2006). For consistency with our measurements of PFASs in Irish drinking
154 water, indoor air and dust (Harrad et al, 2019b); in addition to PFOS, PFOA, PFNA, and
155 PFHxS, we measured the following other PFASs: perfluorobutane sulfonate (PFBS),
156 perfluorooctane sulfonamide (FOSA), its methyl and ethyl derivatives (MeFOSA and
157 EtFOSA), as well as methyl and ethyl perfluorooctane sulfonamido ethanols (MeFOSE and
158 EtFOSE). Five mL of breast milk were added to a centrifuge tube and spiked with 20 µL of
159 an internal standard solution (containing 1 ng/µL of M8PFOS, M8PFOA, M8FOSA,
160 MPFHxS, MPFNA, d-N-MeFOSA, d-N-EtFOSA in methanol). Five mL of formic acid (50%
161 in $H_2O$) was added and the sample was vortexed for 2 minutes. The entire mixture was
162 transferred on to an Oasis WAX (6 mL/150 mg, Waters) solid phase extraction (SPE)
163 cartridge, preconditioned with 6 mL MeOH (0.1% $NH_4OH$) and 6 mL MilliQ water. After
164 allowing samples to load at 1 drop/second, cartridges were rinsed with 6 mL of 25 mM
165 sodium acetate buffer (pH 4) and 6 mL of $H_2O$, before drying under vacuum for 10 minutes.
166 Target analytes were eluted with 6 mL of MeOH (0.1% $NH_4OH$). Extracts were concentrated
167 to 1 mL and passed through a 0.2 µm syringe filter before further concentration to 100 µL in
168 methanol and transfer to autosampler vials ready for analysis.

169

170 **Instrumental Analysis**

171 PFASs were analysed on a Sciex Exion HPLC coupled to a Sciex 5600+ triple TOF MS. A
172 full description of the instrumental methodology is reported elsewhere (Harrad et al. 2019a).
173 Briefly, 10 µL of extract were injected onto a Raptor C18 column (1.8 µm particle size, 50
174 mm length, 2.1 mm internal diameter, Restek). At a flow rate of 0.4 mL/minute a mobile
175 phase gradient was ramped from 80 % Mobile Phase A (5 mM ammonium formate in water),
176 20% mobile phase B (5 mM ammonium formate in MeOH) to 95 % mobile phase B over 6
177 minutes. This was held for 0.5 minutes before equilibrating back to 20 % mobile phase B for
178 1.5 minutes. The triple TOFMS was operated in MS/MS mode equipped with a Turbo V
179 source which was operated in negative mode using electrospray ionisation at a voltage of -
180 4,500 V. The curtain gas was set at 25 psi, whilst the nebulizer gas (source gas 1) was set at
181 25 psi and the drying gas (source gas 2) at 35 psi. The CAD gas was set to medium and
182 temperature was 450 °C. The MS data was acquired using automatic information dependent
183 acquisition (IDA) with two experiment types: (i) survey scan, which provided TOF-MS data;
184 and (ii) dependent product ion scan using a collision energy of -40V and a collision a spread
185 of 30 V. Quantification of individual PFAS was performed in Multiquant 2.0 using the
186 MS/MS transitions and retention times reported in Table SD-1 for identification.

187

188 **Quality Assurance/Quality Control**

189 A reagent blank was analysed with every batch of samples. None of the target compounds
190 were detected in blank samples at concentrations above 5 % of any of the sample
191 concentrations. Therefore, results were not corrected for blank residues and method limits of
192 quantification (LOQ) were estimated based on S/N = 10:1. Average LOQs ranged from 0.01
193 to 0.1 ng/mL for PFAS (Table SD-2). In the absence of a certified reference material,

194 replicate 5 mL aliquots (n=5) of bovine milk were spiked with 5 ng of target analytes. All
195 analyses produced an average recovery of target analytes of 80-120 % with a relative
196 standard deviation of ≤15% as detailed in Table SD-3.
197
198 **Estimation of the intake of PFASs by nursing infants in Ireland**
199 To estimate the intake of PFASs by 1 month old nursing infants consuming human milk in
200 this study we used Equation 1:
201 $Di = \frac{C_{PFAS} x DV_{breast\ milk}}{BW} = ngkg^{-1}bw\ day^{-1}$ (equation 1)
202 Where $D_i$ is the estimated daily intake normalised to body weight (ng/kg bw/day); $C_{PFAS}$ is
203 the concentration of a given PFAS in human milk (ng/mL); $DV_{breast\ milk}$ is the daily volume of
204 breast milk consumed (mL/day) and BW represents the body weight (kg). For both these
205 parameters, U.S. EPA guidelines (USEPA, 2002) were used, specifically, an average intake
206 of 702 mL milk per day for a 1 month old infant weighing 4.14 kg.
207 **First order Pharmacokinetic (PK) model for PFASs**
208 A simple, one-compartment, first order pharmacokinetic (PK) model based upon that
209 reported by Thompson et al (2010) was used to investigate the relationship between predicted
210 exposure intakes via various pathways and concentrations in human breast milk. In this
211 instance, we apply the model to predict the level of exposure that would be required to
212 support the measured concentrations in human milk.
213 The model is expressed as equation 2:
214 $\frac{d(CP)}{dt} = (\frac{DI(t)}{Vd} - kP\ x\ CP(t)$ (equation 2)
215 Where CP is the concentration (ng/mL) of the target PFASs in serum; Vd is the volume of
216 distribution (mL serum/kg bw), DI is the daily absorbed intake (ng/kg bw/day) = daily intake
217 multiplied by the absorption efficiency, and kP is the first order elimination rate from the

218 body ($day^{-1}$). This equation can be rearranged, assuming steady state conditions, to yield
219 equation 3:
220 $DI = CP\ x\ kP\ x\ Vd$ (equation 3)
221 The volume of distribution is defined as the amount of a substance in the body divided by its
222 concentration in the serum or blood (Vd [mL/kg bw] = mass in body [ng/kg bw]/
223 concentration in serum or blood [ng/mL]). The values used here are those reported by
224 Thompson et al (2010), namely 230 and 170 mL/kg bw for PFOS and PFOA respectively.
225 The elimination rate constant kP = $\ln 2/t_{1/2}$, with the values used here being 0.000352 and
226 0.000826 $day^{-1}$ for PFOS (Bartell et al (2010) and PFOA (Olsen et al, 2007) respectively.
227 While an absorption efficiency of 91% was assumed for both PFOS and PFOA by Thompson
228 et al (2010); other studies (Alves et al. 2017; Li et al, 2015) have reported lower values of 11-
229 99% for PFOA - with most solid foods below 70% - and 62 ± 5.6% for PFOS in fish. On this
230 basis, we apply here an intermediate absorption efficiency value of 81%. Additionally,
231 partition coefficients between serum samples and breast milk samples were used to estimate
232 PFAS concentrations in serum equivalent to their measured concentrations in breast milk.
233 Specifically, we assumed that breast milk concentrations were 1.5% and 3.8% of those in
234 serum for PFOS (EFSA, 2018) and PFOA (Haug et al, 2011) respectively.
235

236 **Statistical analysis**

237 Statistical analysis was performed using Excel for Mac version 16.27. For the purposes of
238 statistical analysis, where the concentration of a given PFAS in a sample was <LOQ, the
239 concentration was assumed to equal the fractional detection frequency x LOQ.
240

241 **RESULTS & DISCUSSION**

242 **Concentrations and relative abundance of PFASs in human milk from Ireland**

A summary of concentrations and detection frequencies (DFs) for those target PFASs detected in at least one pooled human milk sample in this study are presented in Table 1 (the full data set is presented in Table SD-4). Concentrations of the other PFASs targeted, i.e. FOSA, EtFOSA, MeFOSA, EtFOSE, MeFOSE and PFBS were all below detection limits (< 0.05-0.1 ng/mL) in every pooled sample and are thus not discussed further. Of those PFASs that were detected, PFOA was present in all samples, followed by PFNA (69%), PFOS (62%) and PFHxS (31%). Consistent with possessing the highest detection frequency, PFOA was the PFAS present at the highest concentration in this study (0.016 – 0.344 ng/mL, median 0.10 ng/mL). Table 1 compares our data with those from selected other studies. Such comparison reveals both the relative abundance and absolute concentrations in Irish human milk to fall within the range reported previously elsewhere in the world. In terms of temporal trends, while no PFAS were detected in the previous Irish human milk survey which analysed pooled samples collected in 2011 (Pratt et al, 2013), the detection limits in this previous study exceeded even the maximum concentrations reported here and thus no meaningful temporal trend can be elucidated for Ireland. We also inspected our questionnaire data on possible factors that might influence PFAS concentrations in our samples for possible explanations for the observed variation in PFAS concentrations between different pooled samples. However, no such relationships were evident – e.g. no obvious differences were observed between those comprising donors from rural as opposed to urban locations.

**Nursing infants' intake of PFASs via breast milk**

Table 2 provides estimated intakes of our target PFASs based on a 1 month old infant weighing 4.14 kg and consuming 702 mL/day of breast milk containing PFASs at the median and 95$^{th}$ percentile concentrations reported in this study. As noted earlier, EFSA have proposed provisional tolerable weekly intake (TWI) values for PFOS and PFOA of 13 and 6

ng/kg bw/week respectively (EFSA, 2018). However, direct comparisons between our estimates of exposure of 1 month old nursing infants to PFOS and PFOA and these provisional TWI values are problematic. This is because the TWIs are derived on the basis of steady state concentrations in blood serum and for PFOA a toxicological end point of increased serum cholesterol *in adults*. For PFOS, the critical toxicological end point identified by EFSA was decreased antibody response post vaccination in children. With respect to this, EFSA pinpointed the serum concentration in 5 year old children above which the risk of this adverse effect was of concern, to be 10.5 ng/mL. Reassuringly, the human milk concentrations reported here do not indicate a health concern based on comparison with the concentrations used in modelled breast feeding scenarios carried out by EFSA. Specifically, even consumption over 6 months of the maximum concentration of PFOS in human milk in this study (0.12 ng/mL) was predicted to result in a serum concentration below 10.5 ng/mL (EFSA, 2018). Notwithstanding this reassuring assessment, further measures to reduce the exposure of the Irish population to PFASs are recommended to reduce concentrations of these contaminants in human milk.

**Modelling of daily intakes of PFOS and PFOA required to support observed human body burdens in Ireland**

Equation 3 was used to derive values of daily absorbed intake (DI) that would be required to support our observed concentrations of PFOS and PFOA in human milk. These represent the sum of exposures from all pathways. From these DI values we subtracted our recently reported daily intakes for the Irish population via inhalation of indoor air, ingestion of indoor dust, and consumption of drinking water (Harrad et al., 2019b). Table 3 shows the results of this modelling exercise and demonstrates that for PFOS, even based on the maximum concentrations in human milk in this study, the additional exposure required to support such a

293 body burden is - at 728 pg/kg bw/day - below the provisional EFSA TWI value that is
294 equivalent to 1857 pg/kg bw/day. The situation is less reassuring for PFOA. As shown in
295 Table 3, while average and median body burdens do not suggest additional exposures of
296 concern; the maximum PFOA concentration in human milk in this study, suggests additional
297 exposure of 1478 pg/kg bw/day, which is approximately twice EFSA's provisional TWI for
298 PFOA. It is important to stress at this point the uncertainties inherent in the PK model
299 employed here. Specifically, while we consider here only recent exposures via air, dust, and
300 drinking water; given the long human half-lives of PFOS and PFOA, and likely temporal
301 changes in their concentrations in the environment, the body burdens indicated by
302 concentrations in human milk will reflect a complex integral of both recent and past
303 exposures. Moreover, more research is required to enhance our knowledge of the human half-
304 lives, absorption efficiencies, and partitioning ratios between breast milk and serum for
305 PFASs. Based on current understanding of human exposure to PFOS and PFOA, the major
306 contributor to our predicted additional exposures is likely to be the diet. However, we
307 highlight that other exposure pathways such as dermal uptake of PFASs from fabrics and
308 cosmetics may also contribute considerably to human exposure. Research to characterise the
309 exposure of the Irish population to PFASs via the diet and dermal uptake is thus
310 recommended.

311

312 **Conclusions**

313 PFOA, PFOS, PFNA, and PFHxS are present in Irish human milk, indicating ubiquitous
314 exposure of the Irish population to these contaminants. This evidence of population-level
315 exposure to PFNA and PFHxS adds urgency to the EFSA's ongoing assessment of the risks
316 of exposure to PFASs additional to PFOS and PFOA. Concentrations in human milk in
317 Ireland fall within the range of those reported previously for other countries, and exposure to

PFASs of Irish nursing infants via consumption of human milk does not appear to constitute a health concern. Also reassuring, application of a simple PK model predicts that even at the maximum concentration of PFOS detected in human milk in this study, the level of exposure required to support this body burden in mothers is below EFSA's provisional TWI. In contrast, applying the same approach to PFOA, suggests that the maximum concentration of PFOA in human milk reported here, is consistent with maternal exposure above the provisional TWI for this compound. These findings suggest detailed study of dietary and dermal exposure to PFOS, PFOA and other PFASs in Ireland is required. Further research is also recommended to enhance scientific knowledge of factors such as: partitioning ratios between human milk and blood serum, as well as bioavailability and human half-lives for PFASs.

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**APPENDIX A. SUPPLEMENTARY DATA**

Supplementary data to this article can be found at…

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**Table 1: Descriptive statistics[a] for concentrations (ng/mL) of PFASs in Irish human milk from primiparas (ng/mL; n=16 pooled samples) and comparison with concentrations from other studies worldwide**

| Parameter (Country, year of sample collection, reference) | PFOA | PFHxS | PFOS | PFNA |
|---|---|---|---|---|
| Detection frequency, % (this study) | 100 | 31 | 62 | 69 |
| Arithmetic Mean (this study) | 0.13 | <0.04 | 0.038 | 0.026 |
| Median (this study) | 0.10 | <0.04 | 0.02 | 0.014 |
| Minimum (this study) | 0.016 | <0.04 | <0.02 | <0.01 |
| Maximum (this study) | 0.35 | 0.087 | 0.12 | 0.1 |
| 5$^{th}$ percentile (this study) | 0.04 | <0.04 | <0.02 | <0.01 |
| 95$^{th}$ percentile (this study) | 0.35 | 0.08 | 0.085 | 0.075 |
| Median (S. Korea, 2013; Kang et al, 2016) | 0.07 | - | 0.050 | <0.022 |
| Range of medians (from 13 countries, 1995-2011[b]; Fång et al, 2015) | - | - | 0.04-0.20 | - |
| Median (Belgium, 2009-2010; Croes et al, 2012) | 0.07 | <0.01 | 0.10 | <0.01 |
| Arithmetic mean (Sweden, 2008; Sundström et al, 2011) | 0.074 | 0.014 | 0.075 | - |
| Median (China, 2009; Liu et al, 2011) | 0.12 | - | 0.042 | 0.019 |
| Median (S. Korea, 2011; Lee et al, 2018) | 0.039 | - | 0.047 | 0.015 |
| Median (Spain, 2014; Guzman et al, 2016) | 0.049 | - | - | 0.066 |
| Arithmetic Mean (Italy, 2010; Barbarossa et al, 2013) | 0.076 | - | 0.057 | - |
| Median (Czech Republic, 2010; Lankova et al, 2013) | 0.044 | <0.006 | 0.047 | <0.006 |

[a] Values below LOQ were assumed to = LOQ*fractional detection frequency

[b] denotes range of years in which covered studies were published

**Table 2: Estimated exposure[a] (ng/kg bw/day) of a 1-month old nursing infant to PFASs in Irish human milk**

| PFAS | 95th percentile | Median |
|---|---|---|
| PFOA | 59 | 18 |
| PFHxS | 14 | 2.1 |
| PFOS | 14 | 3.5 |
| PFNA | 13 | 2.4 |

[a] Assuming a daily breast milk intake of 702 mL/day, a body weight of 4.14 kg (U.S. EPA, 2002), and consumption of breast milk contaminated at either the median or 95th percentile concentration in this study

**Table 3: Predicted daily intakes of PFOS and PFOA (pg/kg bw/day) required to support observed concentrations in Irish human milk**

| PFAS | Human milk concentration (ng/mL) | Predicted total intake[a] | Non-dietary intake[b] | Predicted additional intake[c] | EFSA "TDI"[d] |
|---|---|---|---|---|---|
| PFOS | Average | 245 | 1.6 | 244 | 1857 |
| | Median | 136 | 2.0 | 134 | 1857 |
| | Minimum | 67 | 0.6 | 66 | 1857 |
| | Maximum | 799 | 71 | 728 | 1857 |
| PFOA | Average | 591 | 30 | 561 | 857 |
| | Median | 474 | 30 | 444 | 857 |
| | Minimum | 73 | 1.4 | 72 | 857 |
| | Maximum | 1610 | 132 | 1478 | 857 |

[a]Sum of intakes from all pathways

[b]Measured data from Harrad et al (2019b) covering inhalation of indoor air and ingestion of indoor dust and drinking water

[c]Sum of intakes from all pathways minus inhalation of indoor air and ingestion of indoor dust and drinking water

[d]EFSA's tolerable weekly intake converted for the purposes of comparison only to tolerable daily intake

**Supplementary Material**

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