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Elucidating Levels and Pathways of Human Exposure in Ireland to Brominated Flame Retardants and Perfluoroalkyl Substances

Authors: Stuart Harrad, Daniel Drage, Mohamed Abdallah, Nina Wemken and Marie Coggins

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Government of Ireland

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EPA RESEARCH PROGRAMME 2014–2020

Elucidating Levels and Pathways of Human Exposure in Ireland to Brominated Flame Retardants and Perfluoroalkyl Substances

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by

University of Birmingham and National University of Ireland Galway

Authors:

Stuart Harrad, Daniel Drage, Mohamed Abdallah, Nina Wemken and Marie Coggins

ENVIRONMENTAL PROTECTION AGENCY
An Ghníomhaireacht um Chaomhnú Comhshaoil
PO Box 3000, Johnstown Castle, Co. Wexford, Ireland

Telephone: +353 53 916 0600 Fax: +353 53 916 0699
Email: info@epa.ie Website: www.epa.ie

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DISCLAIMER

This report is based on research carried out/data from 1 March 2016 to 19 September 2019. More recent data may have become available since the research was completed.

The EPA Research Programme addresses the need for research in Ireland to inform policymakers and other stakeholders on a range of questions in relation to environmental protection. These reports are intended as contributions to the necessary debate on the protection of the environment.

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Project Partners

Stuart Harrad
School of Geography, Earth and Environmental Sciences
University of Birmingham
Birmingham B15 2TT
UK
Tel.: +44 121 414 7298
Email: s.j.harrad@bham.ac.uk

Daniel Drage
School of Geography, Earth and Environmental Sciences
University of Birmingham
Birmingham B15 2TT
UK
Tel.: +44 121 414 7243
Email: d.s.drage@bham.ac.uk

Mohamed Abdallah
School of Geography, Earth and Environmental Sciences
University of Birmingham
Birmingham B15 2TT
UK
Tel.: +44 121 414 5527
Email: m.abdallah@bham.ac.uk

Nina Wemken
Centre for Climate Air Pollution Studies
School of Physics
National University of Ireland Galway
Galway
Ireland
Tel.: +353 91 495289
Email: n.wemken1@nuigalway.ie

Marie Coggins
Centre for Climate and Air Pollution Studies
School of Physics
National University of Ireland Galway
Galway
Ireland
Tel.: +353 91 495056
Email: marie.coggins@nuigalway.ie

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Executive Summary

Brominated flame retardants (BFRs) and perfluoroalkyl substances (PFASs) have found extensive use in consumer applications such as electrical and electronic goods, soft furnishings and building insulation foam to impart properties such as flame retardancy and stain resistance. Such use has led to environmental contamination and human exposure. Owing to concerns about their environmental persistence and ability to bioaccumulate and the potential adverse health effects in humans and wildlife, some BFRs and PFASs have been listed under the Stockholm Convention on Persistent Organic Pollutants (POPs), an international treaty designed to eliminate POPs from the environment. Previous studies have revealed low levels of BFRs and PFASs in Irish foodstuffs and human milk. However, no such data existed for Ireland prior to this project on the presence of both BFRs and PFASs in indoor air and dust or of PFASs in drinking water. This project therefore measured selected BFRs and PFASs in indoor air and dust from Irish homes, offices, cars and school classrooms ($n = 30$ per microenvironment category). The same contaminants were measured in 16 samples of human milk donated by Irish mothers, created from samples from 92 individuals. PFASs were also measured in samples of Irish tap water ($n = 85$) and bottled water ($n = 31$).

Comparison of concentrations of BFRs in human milk in this study with those in a previous Irish study conducted in 2011 reveal that restrictions on the manufacture and use of hexabromocyclododecane and both the penta- and octa-bromodiphenyl ether (BDE) products appear to have been successful in reducing concentrations in Irish human milk. Probably as a consequence of the more recent ban on the manufacture and use of the deca-BDE product, concentrations in human milk in this study show no significant decline compared with 2011. Moreover, while in 2011, decabromodiphenyl ethane (DBDPE) – a likely replacement for deca-BDE – was not detected in any human milk sample, DBDPE was detected in three samples in this study. This indicates increasing use of DBDPE as a "drop-in" replacement for deca-BDE, and this is supported by our findings that concentrations of DBDPE in both indoor air and dust in this study are the highest reported to date anywhere. While this is likely to be because this study is one of the few conducted since the listing of deca-BDE under the Stockholm Convention and future studies elsewhere will probably reveal similarly elevated concentrations of DBDPE, it suggests that further research into exposure to DBDPE and its health effects is a priority.

With respect to PFASs, perfluorooctanoic acid (PFOA) dominated air and drinking water, while perfluorobutane sulfonate (PFBS) dominated dust. Perfluorooctane sulfonate (PFOS) concentrations in classroom air exceeded significantly those in homes. Concentrations of PFOA, perfluorononanoic acid (PFNA) and methyl perfluorooctane sulfonamidoethanol (MeFOSE) in air were significantly higher in cars containing child car seats than in cars without them. PFOS, PFOA, PFBS and perfluorohexane sulfonate (PFHxS) were all detected frequently in drinking water but concentrations of PFASs were low, and although concentrations of ∑PFASs were 64 ng/L in one bottled water sample, this fell below a Swedish action level of 90 ng ∑PFASs/L. The Irish population's exposure to PFOS and PFOA via non-dietary sources is well below estimates of dietary exposure elsewhere in Europe. Moreover, even under a high-end exposure scenario, it falls below the European Food Safety Authority (EFSA) provisional tolerable weekly intakes (TWIs) for PFOS and PFOA. Concentrations of PFOA, PFOS, PFHxS and PFNA in Irish human milk are within the range of those reported elsewhere in the world. Other PFASs were not detected in human milk. Reassuringly, concentrations of PFOS and PFOA in Irish human milk currently do not indicate a health concern, based on breastfeeding exposure scenarios carried out by EFSA. Application of a simple pharmacokinetic model suggests that current adult exposure in Ireland to PFOS is below EFSA's provisional TWI. 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. Given that the health effects of PFASs other than PFOS and PFOA are currently under review by EFSA and that this study found that non-dietary

exposure of children to PFBS exceeds that of the other PFASs targeted in this study, it is recommended that, as well as continuing to measure PFOS and PFOA, future research should also monitor exposure to other PFASs such as PFBS, as well as PFHxS and PFNA, that were detected in human breast milk.

1 Introduction

1.1 Sources and Applications of Brominated Flame Retardants and Perfluoroalkyl Substances

Brominated flame retardants (BFRs) such as hexabromocyclododecanes (HBCDDs) and polybrominated diphenyl ethers (PBDEs) have found extensive use worldwide as flame retardants (FRs) in a wide variety of commercial, domestic and industrial applications. Applications of PBDEs include electrical and electronic equipment (e.g. televisions, personal computers, small domestic appliances) and soft furnishings (e.g. sofas, mattresses, curtains, pillows). In the former case, PBDEs were added both to the polymer casing for electronics (e.g. high-impact polystyrene or acrylonitrile butadiene styrene) and to internal circuit boards. In the latter case they were added to both the foam fillings and the fabric covers of soft furnishings such as sofas and chairs in domestic, office or vehicular environments. With respect to HBCDDs, the most important application (96% of all uses in the EU) is its widespread use as an FR in expanded and extruded polystyrene (EPS/XPS) used in building insulation foam in the construction industry. As of 2001 (the last reliable figures publicly available), Europe accounted for 2%, 16%, 14% and 57% of the annual global demand for penta-bromodiphenyl ether (BDE), octa-BDE, deca-BDE and HBCDDs, respectively (BSEF, 2003). However, evidence of their toxicity (see section 1.2), combined with their environmental persistence and bioaccumulation potential, has resulted in the listing of penta- and octa-BDE (2009), HBCDDs (2013) and deca-BDE (2017) as persistent organic pollutants (POPs) under the Stockholm Convention (UNEP, 2017), leading to restrictions on their manufacture and use. These restrictions have created a market demand for replacement FRs, such as decabromodiphenyl ethane (DBDPE), marketed as a replacement for deca-BDE (Arias, 2001).

Perfluoroalkyl substances (PFASs) are a family of synthetic chemicals characterised by a fully fluorinated hydrophobic linear carbon chain, to which are attached different hydrophilic functional groups. These chemicals have been manufactured by the 3M Company, as well as by other companies such as Dupont, and have been produced and used in commercial products and industrial processes for many years (Lindstrom *et al*., 2011). PFASs possess low molecular polarisability, short carbon to fluorine (C-F) bond length and large C-F bond binding energy. Such characteristics govern the oil and water repellency, physical and chemical stability, and surfactant properties of PFASs (Zushi *et al*., 2012). These properties mean that PFASs such as perfluorooctane sulfonate (PFOS) have found wide use in a variety of applications, with historical production peaking at the end of the 20th century in North America and Europe (Paul *et al*., 2009). The history of PFAS production is difficult to portray accurately due to the proprietary nature of this information (Lindstrom *et al*., 2011), but the 3M Company was the first main producer of perfluorooctane sulfonyl fluoride (POSF) (an intermediate product for the synthesis of PFOS), with the total cumulative production estimated to be approximately 96,000 tonnes in the peak years between 1970 and 2002 (Paul *et al*., 2009). In 2002, the 3M Company discontinued its production; however, other companies commenced manufacture at this point to meet existing market demands, with an estimated 1000 tonnes being produced annually since 2002 (Paul *et al*., 2009). The major applications of POSF derivatives have been (1) in carpets to impart stain and dirt repellence, (2) in apparel to provide water repellence, (3) in paper and packaging to afford oil and grease repellence, (4) in performance chemicals such as hydraulic fluids for aviation and (5) in aqueous fire-fighting foams (AFFFs). AFFFs are perhaps the most prominent method of widespread environmental dispersal, with use for oil drilling and military fire-fighting practice (Paul *et al*., 2009).

1.2 Health Concerns Related to BFRs and PFASs

Human exposure to some BFRs has been associated with many adverse effects such as endocrine disruption, liver microsomal enzyme induction, immunotoxicity, neurotoxicity and carcinogenicity (Darnerud, 2008; Vonderheide *et al*., 2008). Animal

studies have also shown neurodevelopmental and behavioural outcomes of exposure to PBDEs, such as hepatic abnormality, endocrine disruption and possibly cancer (Birnbaum and Staskal, 2004; Darnerud, 2008; Hakk, 2010; Wikoff and Birnbaum, 2011). In animals, HBCDDs were found to induce hepatic cytochrome P450 enzymes and alter the normal uptake of neurotransmitters, while in humans they have been reported to trigger cancer through non-mutagenic mechanisms and disruption of the thyroid hormone system (Law *et al.*, 2005; Covaci *et al.*, 2006; Darnerud, 2008). Limited toxicological data exist for DBDPE; however, it is structurally very similar to BDE-209 and may therefore display comparable adverse effects (Hardy *et al.*, 2002; Nakari *et al.*, 2009).

Concerns also exist about the potential adverse human health impacts of some PFASs (EEA, 2019; FIDRA, 2020). In 2018 the European Food Safety Authority (EFSA) highlighted particular concerns that elevated exposure to PFOS and perfluorooctanoic acid (PFOA) led to raised cholesterol levels in blood serum in adults (EFSA, 2018). Moreover, the same assessment by EFSA also concluded that elevated exposure to PFOS was linked to decreased antibody response following vaccination in children. In a new draft opinion, published in 2020 and currently undergoing public consultation, EFSA has proposed a new group tolerable daily intake (TDI) for four PFASs, based on effects on the immune system (EFSA, 2020).

As a consequence of the health concerns about the BFRs targeted in this study, a variety of jurisdictions around the world have evaluated the risk to human health, leading in some instances to health-based limit values (HBLVs). For BFRs, EFSA (2011a,b) has delivered scientific opinions that recommend benchmark doses (BMDs) for a number of PBDEs and HBCDDs and have concluded that dietary exposure in the EU to HBCDDs and BDEs-47, -153 and -209 is not of concern. However, the lower BMD for BDE-99 [12 µg/kg body weight (bw)/day] raised a potential health concern. While additional exposure to PBDEs via house dust was not explicitly considered, it was taken into account for HBCDDs, but was considered unlikely to raise a health concern. Outside the EU, the US Environmental Protection Agency (US EPA) has promulgated reference doses (RfDs) for some PBDEs. Specifically, these values are 100 ng/kg bw/day for both BDE-47 and BDE-99 (US EPA, 2008a,b), with a higher value (7000 ng/kg bw/day) for BDE-209 (US EPA, 2008c). There are no non-occupational HBLVs that relate to PBDEs, HBCDDs or PFASs in indoor air or dust of which we are aware. Likewise, the very low water solubility of PBDEs and HBCDDs is consistent with the absence of any exposure limits related to their presence in drinking water.

Based on the toxicological evidence available to date, chronic exposure guidelines are being developed for PFOS by the US EPA and other jurisdictions for water and food. A review of global guidelines and regulations can be found in Zushi *et al*. (2012), and some especially pertinent illustrative examples are discussed briefly here. The risk arising from exposure to PFOS for human adults has been evaluated as low based on the margin of exposure, derived from the ratio of the provisional TDI and the level of intake (Zushi *et al*., 2012). Moreover, drinking water guidelines exist for both PFASs. The first of these is promulgated by Swedish authorities and specifies a limit of 90 ng/L for ∑PFAS, while the second is a HBLV of 70 ng/L for the sum of PFOS and PFOA concentrations published by the US EPA (US EPA, 2016a,b). In a parallel approach to limit values for external exposure via ingestion of food and water, the Biomonitoring Commission of the German Federal Environmental Agency used the 95th percentile concentration values of two German studies (Midasch *et al*., 2006; Fromme *et al*., 2007) to establish reference values for PFOA and PFOS in the plasma of children and adults. These reference values specify a maximum permissible presence of PFOS of 10 µg/L for children, 20 µg/L for adult females and 25 µg/L for adult males (Wilhelm *et al*., 2009). These may in the future form a basis for limit values in other human matrices such as breast milk.

In mid-2018, EFSA proposed provisional tolerable weekly intakes (TWIs) for both PFOS and PFOA. At 13 ng/kg bw/week and 6 ng/kg bw/week for PFOS and PFOA, respectively (EFSA, 2018), these are substantially lower than those mentioned above. Moreover, the EU has listed PFOA, perfluorononanoic acid (PFNA) and perfluorohexane sulfonate (PFHxS) as substances of very high concern (ECHA, 2019c).

In February 2020 EFSA published a new draft opinion, in which additional PFAS were assessed, and the two TWIs set for PFOS and PFOA in 2018 were re-assessed, considering new scientific knowledge that

had become available in the meantime. A single group TWI of 8 ng/kg bw/week for PFOA, PFNA, PFHxS and PFOS, based on effects on the immune system observed in humans, is proposed (EFSA, 2020). This draft opinion was subject to public consultation (closing 20 April 2020). If adopted, it will supersede the provisional TWIs for PFOS and PFOA set in 2018.

1.3 International Action to Limit the Environmental Health Impacts of BFRs and PFASs

Over the last decade, the widespread use of PBDEs and HBCDDs has been the subject of concern, owing to their documented presence in the environment, including in human tissues, coupled with evidence of their toxicity as outlined in the previous section. At a global level, this concern is exemplified by the listing of HBCDDs and the penta-, octa- and deca-BDE commercial formulations under the United Nations Environment Programme (UNEP) Stockholm Convention on POPs. Within the EU, the manufacture and new use of penta- and octa-BDE has been banned since the mid-2000s, with that of deca-BDE restricted severely since 2008. Since the listing of HBCDDs under the Stockholm Convention in 2013, use of this BFR has also been restricted, although there was a time-limited derogation that up to the end of 2018 permitted its use within the EU in EPS and XPS for building insulation.

Likewise, the strong C-F bond means that PFASs are resistant to thermal, chemical and biological degradation (Kissa, 2001) and are capable of bioaccumulation and long-range environmental transport, exemplified by their detection in the Arctic (Chaemfa *et al*., 2010). As well as PFOS and its salts being listed as POPs under the Stockholm Convention, PFOA was recently listed under Annex A of the Convention. In addition, the European Commission recently undertook a public consultation about the possible addition of PFOA to the EU POPs Regulation (EC, 2019). Moreover, PFHxS is under review for listing, and a potential proposal exists at the EU level to consider C_{10}-C_{14} analogues of PFOA [including PFNA and its salts – which have been listed under the REACH (Registration, Evaluation, Authorisation and Restriction of Chemicals) Regulation as substances of very high concern recommended for restriction] – for listing under the Stockholm Convention (ECHA, 2019c).

1.4 Human Exposure to BFRs and PFASs

The aforementioned environmental presence of BFRs and PFASs raises concerns about potential human exposure, further exacerbated by the evidence of potential adverse human health impacts as summarised in section 1.2. Studies worldwide report the presence of PBDEs, HBCDDs and PFASs in various human tissues, including blood serum, placenta, liver and adipose tissue, and in breast milk (Tao *et al.*, 2008; Frederiksen *et al*., 2010; Abdallah and Harrad, 2011, 2014; Pratt *et al.*, 2013). These biomonitoring data provide a direct measurement of the human body burden of these contaminants resulting from various external exposure pathways (e.g. inhalation, dermal exposure, ingestion of dust and diet, and consumption of water). Current understanding is that non-occupational human exposure to PBDEs and HBCDDs occurs mainly via a combination of diet, air and indoor dust (either via ingestion or dermal contact) (Lorber, 2008; Abdallah and Harrad, 2011, 2014; Trudel *et al.*, 2011). Similar pathways are thought to drive human body burdens of PFOS and other PFASs, with additional contributors for PFASs being drinking water (Ericson *et al.*, 2008; Thompson *et al.*, 2011) and indirect exposure via metabolism of so-called “PFOS or PFOA-precursors” to yield PFOS/PFOA as the ultimate end-product (Miralles-Marco and Harrad, 2015). To date, approaches to elucidating the extent to which these various external exposure pathways contribute to human body burdens consist of two main strands. The first examines correlations between contaminant concentrations in food or indoor dust and their concentrations in human milk or serum. While significant correlations have been reported in some studies (Wu *et al*., 2007; Roosens *et al*., 2009a; Coakley *et al.*, 2013), similar correlations could not be established in other studies (Fromme *et al.*, 2009; Roosens *et al*., 2009b). The failure to establish correlations between external and internal exposure metrics is likely to be due to insufficient sample numbers, coupled with the fact that the long human residence times of POPs means that body burdens at any one time are a complex integral of exposure arising from multiple pathways and over long periods, with the result that recent external exposure via a given pathway may not necessarily correlate with current body burdens (Harrad *et al.*, 2010a). The second strand comprises the application of simple

pharmacokinetic (PK) models. Such models have been used to predict the body burdens of PBDEs in Americans and Britons, HBCDDs in Britons and PFOS in Australians using intake data from different exposure pathways (Lorber, 2008; Thompson *et al.*, 2010; Abdallah and Harrad, 2011, 2014). The predicted body burdens were then compared with the reported concentrations in human matrices for the population in question and the relationships between external and internal exposure discussed.

In summary, there are multiple exposure pathways that contribute to human body burdens of BFRs and PFASs. While the relative contributions of these different pathways vary between individuals and countries, to fully understand the origins of current body burdens of these contaminants, we must characterise exposure by a variety of pathways.

1.5 Human Exposure to BFRs and PFASs in Ireland

At the start of this project, monitoring of human exposure in Ireland to BFRs and PFASs consisted primarily of two strands. The first comprised biomonitoring data on concentrations of these and related contaminants in 11 samples of human milk from four locations (three in Dublin and one in Galway). Each sample consisted of 10 or 11 samples from individual donors (Pratt *et al.*, 2013). Comparison of concentrations of BFRs and PFASs in Irish human milk with those from the USA and selected EU Member States suggests that human body burdens in Ireland are broadly in line with those elsewhere.

The second strand comprises surveillance by the Food Safety Authority of Ireland (FSAI) of concentrations of BFRs and PFASs in the Irish diet. This consists of a number of reports (FSAI, 2005, 2010a,b), along with peer-reviewed publications (Tlustos *et al.*, 2005, 2007; Fernandes *et al.*, 2009; Pratt *et al.*, 2013). These FSAI-funded studies are complemented by the EPA-funded Irish studies of dioxin levels in the Irish environment, which monitor contaminant trends in Irish cows' milk and have been expanded to include some BFRs. These data on concentrations in human foodstuffs were used by Trudel *et al.* (2010) to derive estimates of human dietary exposure to PBDEs for the Irish population. Comparison of these estimates for Ireland with those for Belgium (Roosens *et al.*, 2009b), the UK (Harrad *et al.*, 2004; D'Silva *et al.*, 2006) and the USA (Schecter *et al.*, 2010) shows Irish dietary exposure to be within the range of that recorded in these comparator countries. Currently, no such estimates of Irish dietary exposure to HBCDDs and PFASs appear to be available. Data available to date for other countries are also limited and quite variable, but it would seem reasonable to hypothesise that such exposure would fall somewhere in the range of the data reported for these contaminants in Belgium, the UK and the USA. The fact that concentrations of PFASs reported in all Irish foodstuffs analysed to date fall below detection limits (FSAI, 2010a) suggests that dietary exposure to PFOS in Ireland would be closer to the 30–200 ng/day range reported for the UK (FSA, 2006a), rather than the higher estimate reported for Belgium (Cornelis *et al.*, 2012). However, we note the relatively high limits of quantitation (LOQs) for PFASs in the FSAI study, and further measurements in Irish foodstuffs with lower LOQ values would be needed to definitively quantify such exposure. Likewise, HBCDDs were detected in between 0% (bovine and porcine liver) and 83% (porcine fat) of Irish foodstuffs analysed (FSAI, 2010b). This, combined with the fact the detection limits for HBCDDs in the UK study cited were comparatively high (FSA, 2006b), suggests that Irish dietary exposure to HBCDDs is likely to be closer to the 1.2–20 ng/day estimate range for Belgium (Roosens *et al.*, 2009a) and the USA (Schecter *et al.*, 2010). Again, as noted for PFASs above, further analyses of Irish foods for HBCDD content with LOQ values sufficiently low to minimise "not detects" would appear desirable.

Prior to the commencement of this project there appeared to be no data for Ireland on concentrations of BFRs and PFASs in indoor air and dust, and of PFASs in drinking water. Moreover, the available data on PFASs in Irish human milk demonstrate only that concentrations of PFASs were below what the study authors acknowledged were "relatively high" detection limits of between 0.5 and 5.0 µg/mL (Pratt *et al.*, 2013).

1.6 Objectives

This project addressed the data gaps identified above by conducting the first study of concentrations of PBDEs, HBCDDs and PFASs in indoor air and dust from common microenvironments frequented by the Irish population (cars, primary school classrooms, homes and offices), along with the inaugural study of concentrations of PFASs in Irish tap water and bottled

water. Data produced by these studies have been combined with the existing database on Irish dietary exposure to facilitate an evaluation of the relative contribution of these different exposure pathways to current body burdens in the Irish population. To provide the most up-to-date information on body burdens, a study was conducted that measured concentrations of POP-BFRs and PFOS in 16 pooled samples of human milk from Dublin and Galway. By replicating as far as possible the sampling strategy of an earlier study (Pratt *et al.*, 2013) that measured BFRs in 11 pooled samples of Irish human milk, we have also examined the evidence for possible temporal reductions in human body burdens in Ireland in response to actions (both legislative and voluntary) designed to reduce human exposure over the last decade.

Against this backdrop, the overriding objectives of this project were to:

- evaluate the relative contributions of different exposure pathways (diet, drinking water, indoor air and dust) to current body burdens of BFRs and PFASs in the Irish population;
- establish current body burdens of these contaminants in the Irish population;
- compare these current body burdens with those determined previously for Ireland to assess the impact of recent restrictions on the manufacture and use of these contaminants.

2 Methods

2.1 Ethics

Ethics approval for the collection and analysis of samples of indoor air, dust and drinking water was obtained from the Research Ethics Committee of the National University of Ireland, Galway (Ref. 16/May/02). Separate ethics approval for the collection and analysis of human milk samples was provided by the Clinical Research Ethics Committee of the Galway University Hospital (Ref. C.A. 1578) and the Research Ethics Committee of the Coombe Women & Infants University Hospital in Dublin (Ref. No. 30-2016).

2.2 Sampling

Donors of indoor air, dust and drinking water samples were recruited via the project website (www.nuigalway.ie/elevate), through articles in the national press and through acquaintances of the authors.

2.2.1 Sampling of indoor air and dust

Indoor dust sampling

Project sampling protocols followed previously published methodologies (Abdallah *et al.*, 2008). Home and office participants were asked not to clean their cars for 2 weeks or their living rooms for 2 days prior to sampling. Due to school policy, classroom floors are cleaned on a daily basis, so samples were taken from classrooms at the end of the school day.

Air sampling

Air samples were collected by deploying passive air samplers for approximately 60 days in order to sample the maximum volume of air while remaining in the linear uptake phase of the polyurethane foam (PUF) disc samplers (Hazrati and Harrad, 2007). The sampling apparatus consisted of two parts: a sorbent (XAD-3) impregnated PUF disc (diameter: 140 mm; thickness: 12 mm; surface area: 360.6 cm^2; density: 0.02 g/cm^3; PACS Leicester, UK), pre-cleaned via Soxhlet extraction with dichloromethane for 8 hours (Shoeib *et al.*, 2008). The PUF was partially enclosed in two stainless-steel housings (top diameter 26 cm; bottom diameter 18 cm) and mounted according to previous studies (Abdallah and Harrad, 2010). Samplers were placed on elevated surfaces in homes, offices and schools and on the floor behind the passenger or driver's seat in cars.

Indoor air and dust samples were collected from cars ($n = 31$), homes ($n = 34$), offices ($n = 34$) and school classrooms ($n = 28$). Samples were collected between August 2016 and January 2017 in three Irish counties (Galway, Limerick and Dublin).

2.2.2 Sampling of drinking water

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 that PFAS concentrations in bottled water exceed those in tap water, additional 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 Ireland in May 2018.

2.2.3 Sampling and preparation of pooled samples of human milk

With minor deviations, human milk sampling and donor recruitment adhered to the fourth World Health Organization (WHO) UNEP guidelines for developing a survey of human milk for POPs (WHO, 2007) and also those followed in a previous study that measured BFRs in Irish human milk (Pratt *et al.*, 2013). Comparability of study design with this previous study was important to facilitate elucidation of temporal trends in BFR concentrations in human milk in Ireland. Study protocols and design were approved by the Clinical Research Ethics Committee of the Galway University Hospital (Ref. C.A. 1578) and the Research

Ethics Committee of the Coombe Women & Infants University Hospital in Dublin (Ref. No. 30-2016).

Breast milk samples were collected between 3 and 8 weeks post partum from primiparas in good health and exclusively feeding one infant. Participants had to have resided at their current address for at least 5 years prior to sample collection. Although the WHO guidance stipulates that participating mothers should be not older than 30 years, in Ireland, 65% of first-time mothers are aged 30–40 years (Central Statistics Office, 2018), and, therefore, the recruitment selection criteria were amended to include mothers up to and including 40 years of age. This was in line with the previous Irish study, which included mothers up to and including 41 years of age (Pratt *et al*., 2013).

Mothers were recruited while attending breastfeeding clinics at the same two Irish maternity hospitals from which mothers were recruited in a previous study, namely University Hospital Galway (UHG) and the Coombe Infant and Maternity Hospital (CIMH), Dublin. Breast milk samples of between 30 and 60 mL were collected from each participating mother in clean polypropylene bottles and stored at –18°C until further analysis.

A total of 92 breast milk samples were collected (UHG, $n = 59$; CIMH, $n = 33$). Samples were thawed and then pooled before analysis. Data provided by the mothers via a questionnaire were used to inform the creation of 16 sample pools depending on mothers' place of birth (Ireland, UK, EU or non-EU) and place of residence for the last 5 years (urban or rural), with two pools created that comprised samples from mothers who indicated that they consumed fish at least twice a week (fish-consumer pools). Each pool contained aliquots of 30 mL of milk from each individual constituent sample (15 mL for the fish-consumer pools as there was less milk available from the individual donors to these pools), with the number of individual samples per pool ranging between 3 and 10. Following pooling, milk was freeze dried at –50°C for 72 hours to prepare it for analysis.

2.3 Methods for Determination of Concentrations of BFRs and PFASs

Full details of the methods used are provided in the peer-reviewed publications emerging from this project (see Appendix 1). In summary, however, known masses/volumes of samples were treated with isotopically labelled internal standards before solvent extraction. Following extraction, sample extracts were purified via column chromatography before being subject to gas chromatography–mass spectrometry (applied to most BFRs) and liquid chromatography–mass spectrometry (which was applied to HBCDDs and PFASs).

BFRs targeted in this study were DBDPE, PBDE-28, -47, -99, -100, -153, -154, -183 and -209, plus α-, β- and γ-HBCDD.

Target PFASs in this study were PFOA, PFOS, PFNA, PFHxS, perfluorobutane sulfonate (PFBS), perfluorooctane sulfonamide (FOSA) and its methyl and ethyl derivatives (MeFOSA and EtFOSA), as well as methyl and ethyl perfluorooctane sulfonamidoethanols (MeFOSE and EtFOSE).

2.4 Algorithms Used to Derive Estimates of Human Exposure to BFRs and PFASs via Indoor Air, Dust and Drinking Water

We assumed a 100% absorption of intake of BFRs and PFASs. Average adult and toddler dust ingestion rates of 20 mg/day and 50 mg/day, respectively, were used (Jones-Otazo *et al.*, 2005), with high dust ingestion rates of 50 mg/day and 200 mg/day used for adults and children, respectively (Jones-Otazo *et al.*, 2005). Air inhalation rate figures for adults and toddlers were assumed to be on average 20 m^3/day and 3.8 m^3/day, respectively (Abdallah and Harrad, 2008). To calculate exposures normalised to body weight, adult and child body weights of 70 kg and 20 kg, respectively, were assumed (Abdallah and Harrad, 2008).

2.4.1 Inhalation exposure

The algorithm given in equation 2.1 (Abdallah and Harrad, 2008) was used to estimate adult inhalation exposure to all target BFRs and PFASs:

$$\Sigma_{\text{Inhalation exposure}} = [(C_H F_H) + (C_O F_O) + (C_C F_C)]\, RR \qquad (2.1)$$

where $\Sigma_{\text{Inhalation exposure}}$ is the daily adult exposure via inhalation (pg/day); C_H, C_O and C_C are the concentrations (pg/m^3) of BFRs and PFASs in homes, offices and cars, respectively; F_H, F_O and F_C are the average fraction of time spent in each microenvironment [0.72, 0.238 and 0.042 for homes,

offices and cars, respectively (Abdallah and Harrad, 2008)]; and *RR* is the daily respiration rate for adults (m^3/day).

Child inhalation exposure was calculated using equation 2.2:

$$\Sigma_{\text{Inhalation exposure}} = [(C_H F_H) + (C_S F_S) + (C_C F_C)]\, RR \qquad (2.2)$$

where $\Sigma_{\text{Inhalation exposure}}$ is the daily human exposure via inhalation (pg/day); C_H, C_S and C_C are the concentrations (pg/m^3) of BFRs and PFASs in homes, school classrooms and cars, respectively; F_H, F_S and F_C are the average fraction of time spent in each microenvironment [0.757, 0.201 and 0.042 for homes, school classrooms and cars, respectively (Abdallah and Harrad, 2008)]; and *RR* is the daily respiration rate for children (m^3/day).

2.4.2 Exposure via dust ingestion

Similar algorithms to equations 2.1 and 2.2 were used to estimate adult and child exposure to BFRs and PFASs via ingestion of indoor dust using the same fractions for time spent in different microenvironments cited above.

2.4.3 Exposure via drinking water

Exposures via drinking water (E_{DW}) pg/day were calculated for PFASs thus:

$$E_{DW} = C_{DW} \times W_{IR} \qquad (2.3)$$

where C_{DW} is the PFAS concentration in all water samples combined (pg/L) and W_{IR} is the water ingestion rate (2 L/day), although it is acknowledged that this volume may be lower for children.

2.5 Pharmacokinetic Modelling of the Relationship between External and Internal Exposure

2.5.1 Pharmacokinetic modelling for BFRs

A single-compartment, first-order PK model (Abdallah and Harrad, 2011) was used to investigate the relationship between predicted exposure intakes via various pathways and concentrations in human milk. Target BFRs were assumed to accumulate in lipids (the assumed single compartment in the model).

Hence, the change in concentration in lipid of a given BFR over time can be calculated using equation 2.4:

$$\frac{\delta C_{BFR}}{\delta t} = \frac{I_{BFR}(t) \times AF_{BFR}}{BL(t)} - K_{BFR} \times C_{BFR}(t) \qquad (2.4)$$

where C_{BFR} represents the BFR concentration normalised to milk lipid (ng/g lw); I_{BFR} stands for the daily intake of the BFR (ng/day); AF_{BFR} is the absorption fraction of the BFR; *BL* represents the body lipid mass (g) and K_{BFR} stands for the first-order dissipation rate of the BFR (per day).

Equation 2.4 can be changed thus, assuming constant K_{BFR}, to yield equation 2.5:

$$C_{FR}(t) = C_{FR}(0) \times e^{(-K_{FR} \times t)} + \left[\frac{I_{FR}(t) \times AF_{FR}}{BL(t)}\right] \times \left[\frac{1 - e^{(-K_{FR} \times t)}}{K_{FR}}\right] \qquad (2.5)$$

where $C_{FR}(0)$ represents the target FR body lipid concentration at time point 0 (initial concentration before intake). Assuming a constant intake and body lipid mass (i.e. steady state), the steady-state BFR lipid concentration can be calculated using equation 2.6:

$$C_{BFR} = \frac{I_{BFR}(t) \times AF_{BFR}}{BL(t) \times K_{BFR}} \qquad (2.6)$$

Note that the assumption of steady-state conditions is an inherent uncertainty with this model.

Dust and diet absorption fractions and human half-lives for PBDEs and HBCDDs used in equation 2.6 were taken from the literature (Geyer *et al*., 2004; Thuresson *et al*., 2006; Lorber, 2008; Abdallah and Harrad, 2011; Abdallah *et al*., 2012). In the absence of experimental data to the contrary, inhaled BFRs were assumed to be 100% bioavailable. Body lipid mass was calculated assuming that an adult weighs 70 kg, of which 25% is lipid (US EPA, 2011). K_{BFR} was determined as $0.693/t_{0.5}$, where $t_{0.5}$ is the half-life of the target BFRs in the body lipid compartment.

Daily intakes for the Irish population via inhalation and dust ingestion were taken from measurements made in this study, while estimated daily dietary intakes of PBDEs for the Irish population were obtained from the FSAI based on foodstuffs collected in 2015 (Tlustos *et al*., 2005, 2006; Garcia Lopez *et al*., 2018). As no Irish

dietary data were available for DBDPE and HBCDDs, UK dietary estimates were used (FSA, 2006a; Tao *et al.*, 2017).

2.5.2 Pharmacokinetic modelling for PFASs

For PFASs, a one-compartment, first-order PK model based on that reported by Thompson *et al.* (2010) was used to investigate the relationship between predicted exposure intakes via various pathways and concentrations in human breast milk. In this project, we applied the model to predict the level of exposure that would be required to support the measured concentrations in human milk.

The model is expressed as equation 2.7:

$$\frac{d(CP)}{dt} = \frac{DI(t)}{Vd} - kP \times CP(t) \quad (2.7)$$

where CP is the concentration (ng/mL) of the target PFASs in serum; Vd is the volume of distribution (mL serum/kg bw); DI is the daily absorbed intake (ng/kg bw/day), i.e. the daily intake multiplied by the absorption efficiency; and kP is the first-order elimination rate from the body (per day). This equation can be rearranged, assuming steady state conditions, to yield equation 2.8:

$$DI = CP \times kP \times Vd \quad (2.8)$$

The volume of distribution is defined as the amount of a substance in the body divided by its concentration in the serum or blood (Vd [mL/kg bw] = mass in body [ng/kg bw]/concentration in serum or blood [ng/mL]). The values used here are those reported by Thompson *et al*. (2010), namely 230 mL/kg bw and 170 mL/kg bw for PFOS and PFOA, respectively. The elimination rate constant $kP = \ln 2/t_{0.5}$, with the values used here being 0.000352 and 0.000826 per day for PFOS (Bartell *et al*., 2010) and PFOA (Olsen *et al*., 2007), respectively. While an absorption efficiency of 91% was assumed for both PFOS and PFOA by Thompson *et al*. (2010), other studies (Li *et al.*, 2015; Alves *et al*., 2017) have reported lower values of 11–99% for PFOA – with most solid foods below 70% – and 62% ± 5.6% for PFOS in fish. On this basis, we apply here an intermediate absorption efficiency value of 81%. In addition, partition coefficients between serum samples and breast milk samples were used to estimate PFAS concentrations in serum equivalent to their measured concentrations in breast milk. These values were 1.5% and 3.8% for PFOS (EFSA, 2018) and PFOA (Haug *et al.*, 2011a), respectively.

2.6 Estimation of Exposure of Nursing Infants to BFRs and PFASs

In order to evaluate a nursing infant's dietary intake of the target BFRs in this study we used equation 2.9:

$$D_i = \frac{C_{BFR} \times F_{lipid}}{BW} = ng / \text{kg bw} / \text{day} \quad (2.9)$$

where D_i is the estimated dietary intake normalised to infant body weight (ng/kg bw/day); C_{BFR} is the median concentration of the given BFR in human milk (ng/g lw); F_{lipid} is the daily lipid intake via breast milk (g/day); and BW represents the child's body weight. To calculate F_{lipid}, US EPA guidelines (US EPA, 2011) of an average intake of 702 mL of milk per day for a 1-month-old infant weighing 4.14 kg were used. The median lipid content analysed was 3.47 g per 100 mL of breast milk, resulting in a daily lipid intake of 24.4 g lipid per day.

As PFAS concentrations in human milk in this study are reported in units of ng/mL, to estimate the intake of PFASs by 1-month-old nursing infants consuming human milk in this study, we adapted equation 2.1 to yield equation 2.10:

$$D_i = \frac{C_{PFAS} \times DV_{\text{breast milk}}}{BW} = ng / \text{kg bw} / \text{day} \quad (2.10)$$

where D_i and BW are as defined for equation 2.9; C_{PFAS} is the concentration of a given PFAS in human milk (ng/mL); and $DV_{\text{breast milk}}$ is the daily volume of breast milk consumed (702 mL/day).

3 Results

3.1 Concentrations of BFRs in Indoor Dust

Table 3.1 summarises the concentrations of the BFRs targeted in this study that were detected in indoor dust from Irish cars, classrooms, homes and offices. All 13 PBDE congeners, DBDPE and HBCDDs were detected in all microenvironments (Table 3.1). After BDE-209 [detection frequency (DF) = 100%] and HBCDDs (99% DF), DBDPE and BDE-47 had the highest DFs (98% each). The following PBDE congeners had DFs of > 60%: PBDE-196, -197, -183 and -99. Congeners BDE-17, -28, -49, -66, -100, -154 and -153 had DFs of between 3 and 50%.

3.2 Concentrations of BFRs in Indoor Air

Table 3.2 summarises the concentrations of the BFRs targeted in this study that were detected in indoor air from Irish cars, classrooms, homes and offices. Seven of our target PBDEs, along with DBDPE and HBCDDs, were detected in all microenvironments (Table 3.2). BDE-209 had the highest DF (96%), followed by HBCDDs (81%) and DBDPE (65%). BDE-209, -99 and -47 had DFs of > 90% in all microenvironments, whereas BDE-100, -28, -183, -154 and -153 were detected in < 85% of air samples, with BDE-197, -196, -49 and -17 not detected in any sample.

3.3 Concentrations of BFRs in Human Milk

Table 3.3 summarises the concentrations of the BFRs targeted in this study that were detected in the 16 pooled human milk samples analysed. Apart from BDE-28, -99, -100 and -154 (which were below their LOQs of 0.06, 0.2, 0.2 and 0.12 ng/g lw in all pools), all target PBDE congeners and HBCDD diastereomers were detected in at least one sample pool, with BDE-47 and -153 and α-HBCDD present in all. BDE-209 was detected in 81% of samples, and, with respect to concentration, was the dominant PBDE congener, accounting on average for 65% of $\sum PBDE_{28:209}$, followed by BDE-153 (18%) and BDE-47 (15%).

3.4 Concentrations of PFASs in Indoor Dust

Table 3.4 summarises 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.

3.5 Concentrations of PFASs in Indoor Air

Table 3.5 summarises the concentrations of the PFASs targeted in this study that were detected in indoor air from Irish cars, classrooms, homes and offices. The most frequently detected PFAS was PFOA (DF of > 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/m^3 in all microenvironments), with only PFOS and PFBS also present at median concentrations of > 10 pg/m^3 in any microenvironment category (13 and 21 pg/m^3, respectively, in cars).

3.6 Concentrations of PFASs in Drinking Water

Table 3.6 summarises the concentrations of the PFASs targeted in this study that were detected in drinking water in Ireland. Except for FOSA, EtFOSA and EtFOSE, all target PFASs were detected in Irish drinking water (Table 3.6). 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 one water type.

Table 3.1. Summary of concentrations of BFRs (ng/g) in indoor dust from Irish homes, cars, offices and schools, together with median concentrations from selected other studies (median, mean)

Location	Statistical parameter	DBDPE	BDE-209	BDE-47	BDE-99	BDE-183	∑tri-octa-BDEs	α-HBCDD	β-HBCDD	γ-HBCDD	∑HBCDDs
Homes											
	n	29	29	29	29	29	29	26	26	26	26
	Median	4200	13,000	7.6	13	1	49	200	100	200	490
	Range	410–460,000	140–650,000	0.6–240	<0.2–500	<0.3–33	10–940	0.31–28,000	0.12–12,000	0.83–5600	1.3–43,000
	Mean	39,000	58,000	26	45	4.1	130	1500	680	670	2900
UK (Tao *et al.*, 2016)		<10 (<10–97)	4500 (160–370,000)	13 (0.15–1700)	12 (0.05–1700)	<1.0 (<1.0–12)		<2.6 (<2.6–400)	<2.2 (<2.2–160)	110 (16–1400)	110 (19–1500)
USA (Dodson *et al.*, 2012; Venier *et al.*, 2016)		150 (ND-3100)	2200 (75–7500)	270 (20–1300)	340 (20–2800)	11 (ND–37)		62 (17–910)	16 (7–230)	73 (13–790)	160 (39–1800)
Czechia (Kalachova *et al.*, 2012)		140 (<20–1700)	375 (41–5500)	8.9 (<0.1–11)	11.6 (<0.1–95)	3.9 (<0.8–460)		26 (<0.3–280)	7.1 (<0.3–57)	61 (<0.3–740)	93 (<0.3–950)
Sweden (Sahlström *et al.*, 2015)		150 (943–1500)	310 (140–310,000)	21 (6.5–460)	17 (<0.74–300)	–		56 (14–1400)	18 (3.4–730)	37 (2.5–4000)	110 (20–6000)
China (Wang *et al.*, 2018)		560 (220–3100)	150 (69–410)	1.3 (0.67–4.2)	4.9 (1.2–25)	0.37 (0.21–3.7)	8.0 (5.1–37)	64 (34–510)	21 (9.8–120)	64 (30–370)	160 (74–1000)
Australia (McGrath *et al.*, 2018)		1600 (ND–9000)	1100 (290–13,000)	56 (ND–2800)	74 (16–58,000)	<MQL (ND–26)	(18–11,000)				
Brazil (Cristale *et al.*, 2018)		400 (150–740)	410 (160–1200)	8.0 (4.5–1400	153 (20–290)						
Cars											
	n	28	28	28	28	28	28	29	29	29	29
	Median	7700	26,000	24	50	4.1	150	330	250	490	1300
	Range	<13–190,000	14–680,000	<0.1–130	<0.2–270	<0.3–92	0.094–690	3.4–3700	4.8–2600	2.4–17,000	2400–20,000
	Mean	23,000	82,000	31	70	9.8	200	650	410	180	2800
UK (Harrad *et al.*, 2006, 2008)		100 (<DL–2900)	100,000 (12,000–2,600,000)	54 (19–7500)	100 (23–80,00)	7.8 (<DL–67)		20,00 (54–88,00)	740 (16–5200)	9600 (27–56,000)	13,000 (190–69,000)
Germany (Brommer *et al.*, 2012)		13,00 (110–6500)	940 (220–3100)	17 (2.1–43)	32 (1.3–88)	3.7 (1.3–<0.2–17)					
Czechia (Kalachova *et al.*, 2012)		99 (<20–3600)	170 (<5–33,000)	2.2 (<0.1–280)	<0.1 (<0.1–280)	<0.8 (<0.8–15)		9 (<0.3–45)	<0.3 (<0.3–44)	25 (<0.3–240)	33 (<0.3–240)
Greece (Besis *et al.*, 2017)		856 (33–5200)	2,800 (110–38,000)	9.1 (0.63–9000)	12 (1.4–11,000)	1.4 (LOD–1200)	(23–17,000)	90 (<LOQ–1300)	16 (<LOQ–290)	46 (<LOQ–260)	155 (<LOQ–1800)
Brazil (Cristale *et al.*, 2018)		1400 (420–3800)	1600 (300–4000)	31 (4.3–190)	100 (8.5–350)						

Table 3.1. Continued

Location	Statistical parameter	DBDPE	BDE-209	BDE-47	BDE-99	BDE-183	∑tri-octa-BDEs	α-HBCDD	β-HBCDD	γ-HBCDD	∑HBCDDs
Offices											
	n	31	31	31	31	31	31	32	32	32	32
	Median	6100	3500	7.7	7	3.2	30	220	96	630	380
	Range	<13–1300,00	560–150,000	0.8–130	<0.2–160	<0.3–190	2.8–770	<0.1–4400	18–710	8.8–3300	84–5200
	Mean	12,000	4200	16	26	11	77	520	160	170	850
UK (Tao *et al.*, 2016)		<10 (<10–54)	26 (2.3–350)	6 (0.15–380)	7.9 (1.2–42)	<1.0 (<1.0–3.8)		5.4 (<2.6–31)	<2.2 (<2.2–15)	34 (3.1–320)	41 (5.5–360)
France (Abdallah *et al.*, 2016)								2700 (540–6400)	440 (140–1500)	1300 (312–6400)	4700 (1100–10,000)
China (Wang *et al.*, 2018)		1000 (580–1600)	490 (220–2900)	1.5 (0.49–17)	1.6 (0.82–32)	0.69 (0.58–5.4)	10 (6.2–64)	100 (60–160)	35 (18–54)	93 (32–190)	260 (110–390)
Australia (McGrath *et al.*, 2018)		1900 (ND–10,000)	1500 (ND–7200)	220 (40–540,0000	230 (46–1,000,000)	<MQL	(ND–920)				
Brazil (Cristale *et al.*, 2018)		2000 (840–5000)	4200 (1800–25,000)	13 (7.5–34)	30 (12–53)						
Schools											
	n	32	32	32	32	32	32	30	30	30	30
	Median	10,000	8100	5	5.1	<0.3	30	420	180	130	800
	Range	620–540,000	200–71,000	1.3–35	<0.2–240	<0.3–26	2.5–290	12–4100	71–2300	28–6700	250–10,000
	Mean	48,000	17,000	9	20	2	50	680	350	620	1700
UK (Harrad *et al.*, 2010b)		98 (<20–2500)	5000 (49–88,000)	26 (1.6–120)	36 (1.1–270)	1.2 (<2–48)	100 (23–1000)	1400 (24–100,000)	550 (14–67,000)	1700 (34–72,000)	4100 (72–89,000)
Japan (Mizouchi *et al.*, 2015)		125 (9.0–800)	1000 (200–4800)	8.9 (0.68–73)	7.9 (0.58–60)	13 (0.22–110)		340 (18–1700)	64 (2.4–340)	104 (0–500)	510 (20–2300)
Sweden (Larsson *et al.*, 2018)		34 (<2.2–420)	54 (<4.1–1200)								
Sweden (Persson and Wang, 2019)		150 (<0.58–300)	69 (<1.9–130)	30 (11–49)	44 (19–68)	5.7 (<2.2–<9.2)		310 (170–4500)	72 (52–88)	129 (98–160)	510 (380–640)
Brazil (Cristale *et al.*, 2018)		300 (210–700)	420 (94–1200)	8.2 (3.2–30)	33 (30–36)						

DL, detection limit; LOD, limit of detection; LOQ, limit of quantitation; MQL, minimum quantification level; ND, not detected.

Source: Wemken *et al.* (2019). Reproduced with permission of the American Chemical Society.

Table 3.2. Summary of concentrations of BFRs (pg/m³) in indoor air from Irish homes, cars, offices and schools and median concentrations from selected previous studies

Location	Statistical parameter	DBDPE	BDE-209	BDE-47	BDE-99	BDE-183	∑tri-octa-BDEs	α-HBCDD	β-HBCDD	γ-HBCDD	∑HBCDDs
Homes											
	n	28	28	28	28	28	28	32	32	32	32
	Median	48	410	2.1	6.1	<1.1	19	<0.3	<0.3	10	20
	Range	15–7000	<7.5–5500	<0.43–28	<0.43–330	<1.1–7.5	5.6–330	<0.3–57	<0.3–34	<0.3–3500	0.9–2500
	Mean	390	880	4.9	37	0.8	50	10	3.5	100	110
UK (Tao *et al.*, 2016)		<10 (<10–97)	170 (23–3800)	13 (0.15–1700)	12 (0.05–1700)	<1.0 (<1.0–12)		<2.6 (<2.6–400)	<2.2 (<2.2–160)	110 (16–1400)	110 (19–1500)
Sweden (Thuresson *et al.*, 2012)			310 (43–1100)								2 (<1.6–33)
Canada (Venier *et al.*, 2016)		9.2 (NA–74)	49 (ND–220)	39 (54–760)	5.3 (1.3–73)	1.3 (ND–1.6)					
Czechia (Venier *et al.*, 2016)		–	9.4 (ND–15)	1.6 (0.56–16)	0.29 (0.16–1.4)	0.12 (ND–0.23)					
USA (Venier *et al.*, 2016)		42 (ND–71)	260 (ND–5500)	52 (4.5–820)	15 (ND–1300)	2.5 (ND–5)					
Cars											
	n	29	29	29	29	29	29	32	32	32	32
	Median	160	200	1.9	2.1	<1.1	11	8.9	<0.3	18	25
	Range	<15–3200	<7.5–7100	0.79–19	<0.43–150	<1.1	6.5–200	0.3–170	0.3–62	0.3–2300	0.9–2300
	Mean	340	660	3.2	9.1	0.58	19	13	6.2	180	200
UK (Harrad *et al.*, 2006; Abdallah and Harrad, 2010)				14.8 (2.9–4700)	12 (0.0–2300)			2000 (54–8800)	740 (16–5200)	9600 (27–56,000)	13,000 (190–69,000)
Sweden (Thuresson *et al.*, 2012)			400 (160–2500)								0.0 (<1.6)
Offices											
	n	31	31	31	31	31	31	32	32	32	32
	Median	<15	240	3.4	4.2	0.55	15	<0.3	<0.3	9.6	14
	Range	<15–2800	<7.5–1600	<0.43–4800	<0.43–880	<1.1	5.7–6200	0.3–1500	0.3–710	0.3–1500	0.9–2800
	Mean	240	420	160	48	0.54	230	86	42	90	220
UK (Tao *et al.*, 2016)		<10 (<10–54)	26 (2.3–350)	6 (0.15–380)	7.9 (1.2–42)	<1.0 (<1.0–3.8)		5.4 (<2.6–31)	<2.2 (<2.2–15)	34 (3.1–320)	41 (5.5–360)
Sweden (Thuresson *et al.*, 2012)			3200 (68–5800)								0.00 (<1.6)
Schools											
	n	31	31	31	31	31	31	32	32	32	32
	Median	220	410	2.3	3.1	<1.1	12	16	<0.3	22	38
	Range	<15–3800	<7.5–21,000	1.5–29	<0.43–99	<1.1–1.4	7.0–150	0.3–210	0.3–4600	0.3–1500	0.9–6300
	Mean	460	1600	5.2	9.5	0.54	21	33	160	96	280
Sweden (Larsson *et al.*, 2018)		<7.6	<32	17	<14	<2.1		<0.57	<0.41	<1.0	<2.0
Norway (Cequier *et al.*, 2014)		8.3	<MLD	130	23	<MLD	180				
South Korea (Lim *et al.*, 2014)		–	0.21 (ND–3.6)	0.40 (ND–17)	0.28 (ND–13)	0.015 (ND–0.15)					

MLD, method limit of detection; NA, not available; ND, not detected.

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Table 3.3. Descriptive statistics for PBDE, DBDPE and HBCDD concentrations (ng/g lipid weight) in 16 pooled human milk samples from Ireland and comparison with selected other studies

Country/reference	Statistical parameter	BDE-28	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	$\sum PBDE_{28:183}$	BDE-209	$\sum PBDE_{28:209}$	DBDPE	α-HBCDD	β-HBCDD	γ-HBCDD	∑HBCDDs
Ireland (this study)	DF (%)	0	100	0	0	100	0	6	NA	81	NA	19	100	88	81	NA
	Average	<0.06	0.64	<0.2	<0.2	0.78	<0.12	<0.3	1.4	2.8	4.3	0.74	1.5	0.22	0.27	2
	Median	<0.06	0.50	<0.2	<0.2	0.71	<0.12	<0.3	1.4	1.4	2.5	<2.5	1.7	0.34	0.21	1.8
	Minimum	<0.06	0.16	<0.2	<0.2	0.44	<0.12	<0.3	0.61	<0.6	1.7	<2.5	0.66	<0.05	<0.05	0.83
	Maximum	<0.06	1.8	<0.2	<0.2	1.5	<0.12	0.4	2.5	23	24	4.6	3	0.53	1.5	3.6
Ireland (Pratt *et al.*, 2013)	Median	0.09	1.1	0.27	0.31	1.0	0.02	0.03	2.8	0.77	3.6	<2.2–<2.5	2.6	0.3	0.4	2.9
UK (Abdallah and Harrad, 2011, 2014)	Median	NA	2.8	0.71	0.45	1.10	0.30	NA	5.0	0.25	NA	<0.78	4.91	0.32	0.73	3.83
UK (Tao *et al.*, 2017)	Median	0.14	2.2	0.77	0.53	1.8	0.12	NA	5.8	<0.22	NA	<0.78	1.9	0.23	0.73	2.9
Denmark, Finland, France (Antignac *et al.*, 2016)	Median	0.19	1.99	0.76	0.45	1.20	0.07	0.07	4.90	0.34	NA	–	0.31	–	–	–
		0.33	3.12	0.54	0.42	0.77	0.05	0.03	5.19	NA	NA	–	0.31	–	–	–
		0.04	0.43	0.10	0.10	0.54	0.03	0.05	0.21	1.47	NA	–	0.56	–	–	–
Germany (Hoopmann *et al.*, 2009)	Median	NA	0.29	0.10	0.10	0.52	NA	NA	NA	NA	–	–	–	–	–	–
Canada (Zhou *et al.*, 2014)	Median	NA	NA	NA	NA	16	NA	NA	NA	NA	NA	<1.7	–	–	–	–
USA (Carignan *et al.*, 2012)	Geometric mean	–	–	–	–	–	–	–	–	–	–	–	0.71	0.08	0.20	1.02
USA (Wu *et al.*, 2007)	Median	0.9	13.9	2.4	2.4	3.0	0.2	0.1	30.2	<DL	NA	–	–	–	–	–
New Zealand (Mannetje *et al.*, 2013)	Median	0.22	2.54	0.53	0.54	0.72	0.04	0.07	NA	0.35	NA	–	–	–	–	–
China (Shi *et al.*, 2016)	Median	–	–	–	–	–	–	–	–	–	8.06	–	–	–	–	–

DF, detection frequency; DL, detection limit; NA, not available.

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Table 3.4. Descriptive statistics for concentrations (ng/g) of target PFASs in Irish indoor dust (only those with a DF of >20% in at least two microenvironment categories are 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 median (Goosey and Harrad, 2011)	190	210	140	–	–	220
Belgium, Italy, Spain median (de la Torre *et al.*, 2019)	1.4	0.13	0.28	0.40	0.04	–
Czechia median (Karásková *et al.*, 2016)	2.0	2.0	10	<LOD	<LOD	<LOD
Canada median (Karásková *et al.*, 2016)	8.2	1.9	9.1	<LOD	4.4	<LOD
USA median (Karásková *et al.*, 2016)	9.0	8.7	14	0.9	3.9	1.0
South Korea median (Tian *et al.*, 2016)	4.5	0.0	11	0.3	1.4	2.0
Cars						
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 median (Goosey and Harrad, 2011)	65	180	97	–	–	82
Sweden median (Björklund *et al.*, 2009)	33	–	12	–	–	–
USA geometric mean (Fraser *et al.*, 2013)	11	NQ	16	<LOD	15	NQ
Offices						
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 median (Goosey and Harrad, 2011)	290	170	230	–	–	220
Sweden median (Björklund *et al.*, 2009)	70	–	110	–	–	–
USA geometric mean (Fraser *et al.*, 2013)	32	NQ	15	NQ	63	NQ
Belgium median (D'Hollander *et al.*, 2010)	2.9	0.2	2.2	0.2	0.4	–
Classrooms						
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 median (Goosey and Harrad, 2011)	240	700	840	–	–	660
Sweden median (Giovanoulis *et al.*, 2019)	7.7	<0.3	49	<0.5	1.1	–

DF, detection frequency; LOD, limit of detection; NQ, not quantified.

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Table 3.5. Descriptive statistics for concentrations (pg/m^3) of target PFASs in Irish indoor air (only those with a DF of >20% in at least two microenvironment categories are 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 median (Goosey and Harrad, 2012)	24	45	23	11	–	540	–	760
Norway median (Haug *et al.*, 2011b)	–	–	–	–	–	78	–	265
Germany median (Fromme *et al.*, 2015)	–	–	–	–	–	66	–	217
Canada median (Shoeib *et al.*, 2011)	21	–	–	<0.02	-	56	89 (average)	320
South Korea median (Kim *et al.*, 2012)	–	–	–	–	–	59	–	89
Finland median (Winkens *et al.*, 2017)	15	–	<0.52	1.9	<1.0	17	2.4	56
Australia median (Eriksson *et al.*, 2015)	14	–	4.3	9.7	1.3	–	3.0	–
Nepal median (Eriksson *et al.*, 2015)	<2	–	<2	<2	<2	–	<2	–
Cars								
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								
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 median (Goosey and Harrad, 2012)	18	59	84	55	–	420	–	310
Belgium median (D'Hollander *et al.*, 2010)	2.9	–	0.2	2.2	0.2	–	0.4	–
Classrooms								
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 median (Fromme *et al.*, 2015)	–	–	–	–	–	<LOD	–	<LOD
University classrooms Czechia median (Karásková *et al.*, 2018)	5.3	0.93	0.70	2.0	0.41	3.2	1.8	5.8

DF, detection frequency; LOD, limit of detection.

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Table 3.6. Descriptive statistics for concentrations (ng/L) of target PFASs in Irish drinking water (only those with a DF of >20% in at least one sample type are shown)

	PFOA	PFOS	PFBS	MeFOSA	PFNA	MeFOSE
Tap water (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 (Endirlik *et al*., 2019)[a]	0.19	0.28	0.25	–	0.13	–
France median (Boiteux *et al*., 2012)	3	5	<1	–	<1	–
USA median (Boone *et al*., 2019)	4.2	1.6	1.2	–	0.74	–
Netherlands median (Brandsma *et al*., 2019)	4.0	1.3	7.3	–	<0.5	–
Catalonia, Spain median (Ericson *et al*., 2008)	0.65	0.41	<0.27	–	<0.42	–
Central Europe median (Gellrich *et al*., 2013)	2.6	1.3	2.7	–	1.4	–
Canada median (Kaboré *et al*., 2018)	0.31	0.64	0.16	–	0.15	–
Brazil median (Schwanz *et al*., 2016)[a]	10	5.8	1.3	–	12	–
Australia median (Thompson *et al*., 2011)	<0.5	<0.66	<0.14	–	–	–
China arithmetic mean (Zhang *et al*., 2019)[b]	0.02–61	0.06–190	0.03–7.8	–	0.03–20	–
Tap water (private supply)						
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						
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 (Endirlik *et al*., 2019)[a]	0.10	<LOD	0.20	–	0.15	–
Catalonia, Spain median (Ericson *et al*., 2008)	0.34	<0.24	<0.27	–	<0.42	–
Central Europe median (Gellrich *et al*., 2013)	1.6	1.5	2.6	–	3.0	–
Canada median (Kaboré *et al*., 2018)	<0.07	<0.03	<0.02	–	<0.03	–
Brazil arithmetic mean (Schwanz *et al*., 2016)[a]	7.6	<1.2	3.5	–	10	–

[a]Only concentrations >LOD used to calculate median concentrations.

[b]Range of arithmetic mean concentrations at a number of sampling locations.

DF, detection frequency; LOD, limit of detection.

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3.7 Concentrations of PFASs in Human Milk

A summary of concentrations and DFs for those target PFASs detected in at least one pooled human milk sample in this study is presented in Table 3.7. 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 omitted from Table 3.7. 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 DF, PFOA was the PFAS present at the highest concentration in this study (range 0.016–0.344 ng/mL; median 0.10 ng/mL).

Table 3.7. 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	0.053	0.034
Median (this study)	0.10	< 0.04	0.053	0.016
Minimum (this study)	0.016	< 0.04	< 0.02	< 0.01
Maximum (this study)	0.35	0.087	0.12	0.1
5th percentile (this study)	0.04	< 0.04	< 0.02	< 0.01
95th percentile (this study)	0.35	0.08	0.10	0.08
Median (South Korea, 2013; Kang *et al.*, 2016)	0.07	–	0.050	< 0.022
Range of medians (from 13 countries, 1995–2011; 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 (South Korea, 2011; Lee *et al.*, 2018)	0.039	–	0.047	0.015
Median (Spain, 2014; Guzmàn *et al.*, 2016)	0.049	–	–	0.066
Arithmetic mean (Italy, 2010; Barbarossa *et al.*, 2013)	0.076	–	0.057	–
Median (Czechia, 2010; Lankova *et al.*, 2013)	0.044	< 0.006	0.047	< 0.006

[a]Values below LOQ were assumed to = LOQ × fractional DF.

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4 Discussion

4.1 BFRs in Indoor Air and Dust

4.1.1 DBDPE

To the authors' knowledge, our data on concentrations of DBDPE in indoor dust are both the most recent and contain the highest concentrations reported globally. Table 3.1 reveals that the concentrations of DBDPE in this study exceed markedly those reported for similar microenvironments across Europe, China and Australia (Qi *et al.*, 2014; Tao *et al.*, 2016; McGrath *et al.*, 2018; Persson and Wang, 2019). The substantially elevated DBDPE concentrations reported here (Table 3.1) suggest that DBDPE may have been used as an FR in soft furnishings to meet Irish fire safety requirements for domestic furniture, which differ from other EU Member States (except for the UK) (Irish Statute Book, 1995; Hagen *et al.*, 2017; Fire Safety Advice Centre, 2019). DBDPE is thought to have replaced deca-BDE in plastics and textiles with a wide range of uses in the transport, building, construction and domestic sectors (ECHA, 2019b). DBDPE registrations under REACH (ECHA, 2019b) now exceed in weight BDE-209 registrations; 13 importers or manufacturers report a combined annual tonnage of between 10,000 and 100,000 tonnes of DBDPE in 2018, compared with the total range of 1000–10,000 for BDE-209 in 2014 (ECHA, 2019a). Concentrations of DBDPE in UK indoor dust increased between 2006/07 and 2015 (Harrad *et al.*, 2006; Tao *et al.*, 2016). While no comparative Irish data exist, we hypothesise that similar increasing temporal trends are occurring in Ireland and thus compared our Irish data with those in the most recent UK survey. Median DBDPE concentrations in Irish homes, cars, schools and offices exceed by 100, 77, 610 and 80 times those in UK homes (reported in 2016; Tao *et al.*, 2016), cars (sampled in 2003–2005; Harrad *et al.*, 2006), schools (sampled in 2007–2008; Harrad *et al.*, 2010b) and offices (sampled in 2016; Tao *et al.*, 2016), respectively.

Median concentrations of DBDPE reported here for Irish homes exceed those reported for Swedish homes (Thuresson *et al.*, 2012), Australian homes (also sampled in 2016; McGrath *et al.*, 2018) and homes in Beijing (Wang *et al.*, 2018). Median DBDPE concentrations reported for Irish cars also exceed those reported for cars in Greece (Besis *et al.*, 2017), while concentrations in Irish offices exceed those in Australian ones (McGrath *et al.*, 2018). Moreover, median concentrations in Irish schools exceed those recently reported for low-energy Swedish preschools (300 times higher; Larsson *et al.*, 2018) and preschools in Stockholm (100 times higher; Persson *et al.*, 2019).

4.1.2 PBDEs

In agreement with other international studies, BDE-209 was the most abundant PBDE congener detected across all microenvironments (Harrad *et al.*, 2008; Kalachova *et al.*, 2012; Venier *et al.*, 2016), contributing to > 99% of ∑PBDEs for homes, cars and schools, and 98% in offices. This is unsurprising given that BDE-209 was the most commonly used PBDE congener from 2000 until 2008 (ECHA, 2014), after which its use started to decline following restrictions, its inclusion in the Restriction of Hazardous Substances Directive [Directive (EU) 2017/2102] and its classification as a substance of very high concern under REACH (ECHA, 2019a). An estimated 10% of the total deca-BDE imported into the EU between 2000 and 2005 was imported into Ireland (ECHA, 2019a). Although no Irish statistics on the use of deca-BDE in textiles exist, UK data are thought to closely represent usage patterns in Ireland, and an estimated three-quarters of the deca-BDE used to treat UK textiles was used in domestic furniture (European Chemicals Bureau, 2003), while 95% of all upholstered materials used in the UK were treated with flame retardants to comply with the UK FFFSR [UK Furniture and Furnishings (Fire Safety) Regulation] (ECHA, 2012). When compared with UK data (Harrad *et al.*, 2010a; Tao *et al.*, 2016), median BDE-209 concentrations in Irish homes (13,000 ng/g) are higher, while concentrations in Irish offices (3500 ng/g) and schools (8100 ng/g) are consistent with UK median concentrations reported in 2015 and 2007/08, respectively (Harrad *et al.*, 2010a; Tao *et al.*, 2016). In contrast, concentrations in UK cars (Harrad

and Abdallah, 2011) between 2003 and 2005 were four times higher than those in Irish cars (median 26,000 ng/g), perhaps reflecting a downward trend in deca-BDE use in vehicle upholstery.

Comparisons with other countries reveal that BDE-209 concentrations in Irish indoor environments exceed those reported for Greece (Besis *et al.*, 2017), Germany (Brommer *et al.*, 2012) and Czechia (Kalachova *et al.*, 2012). BDE-209 concentrations in Irish homes, offices and cars exceed recent values for Australia (McGrath *et al.*, 2018), as well as those reported for homes and offices in Beijing (Wang *et al.*, 2018). BDE-209 median concentrations in Irish schools also exceed those in Brazil (median 420 ng/g) (Cristale *et al.*, 2018).

Detection frequencies across all microenvironments for BDE-47 and BDE-99 (97% and 71%) were high but these congeners were present in lower concentrations than BDE-209. BDE-47 and BDE-99 are typically associated with the penta-BDE mixture more widely used in North America (BSEF, 2003) than Europe. Concentrations of both congeners in Irish offices, homes and cars are exceeded by those in Australia, the USA and Canada (Venier *et al.*, 2016; McGrath *et al*., 2018) but are comparable to those for Czechia and the UK (Venier *et al.*, 2016; Harrad and Abdallah, 2011).

4.1.3 HBCDDs

HBCDDs were detected in all dust samples in this study, with concentrations in Irish homes (median 490 ng/g) exceeding those for the UK (110 ng/g) in 2015 (Tao *et al.*, 2016) and in other international studies (Kalachova *et al.*, 2012; Sahlström *et al.*, 2015; Dodson *et al.*, 2012). ∑HBCDD concentrations in office, school and car dust (medians 380 ng/g, 800 ng/g, 490 ng/g, respectively) are lower than those for the UK (medians 4100 ng/g, 4100 ng/g, 13,000 ng/g, respectively) (Harrad *et al.*, 2010a; Tao *et al.*, 2016) for samples collected in 2015 and 2008, which may reflect a downward trend in HBCDD use in response to recent restrictions.

∑HBCDD median concentrations in office dust are exceeded by those in France in 2016 (median 4700 ng/g) (Abdallah *et al.*, 2016). There are few published data on concentrations of HBCDDs in schools; results from this study (median 800 ng/g) are consistent with those for Japan (510 ng/g) (Mizouchi *et al.*, 2015), but the Japanese study was conducted before HBCDD's listing under the Stockholm Convention in 2013. Concentrations in Irish cars are nearly 40 times higher than those in Czechia (Kalachova *et al.*, 2012) and 8 times higher than those in Greece (Besis *et al.*, 2017).

4.2 BFRs in Indoor Air

There is a dearth of data regarding concentrations of BFRs in indoor air published over the last 5 years and so limited comparisons can be made with our data (Covaci *et al.*, 2006, 2009; Frederiksen *et al.*, 2009) – see Table 3.2.

4.2.1 DBDPE

This study reports the most recent indoor air data anywhere for DBDPE in homes, schools and offices and the first data for cars. DBDPE was detected in 65% of air samples; these concentrations are lower than those in Canada (85%) (Venier *et al.*, 2016) but higher than those in the UK in 2016 (Tao *et al.*, 2016) (DF 20%). Similar to our indoor dust data, concentrations in Irish indoor air are also mostly higher than those reported internationally. Concentrations of DBDPE in Irish homes and offices are > 10 and > 30 times higher than 2016 UK data (cars and schools were not included in the UK study) (Tao *et al.*, 2016).

Concentrations of DBDPE in Irish homes are comparable to those in US homes (median 42 pg/m^3) but exceed those reported for Canadian and Czech homes (Venier *et al.*, 2016). Concentrations in Irish offices exceed those in Spanish offices (Reche *et al.*, 2019), while those in schools exceed those reported for Swedish preschools in 2016–18 (Larsson *et al.*, 2018) and Norwegian schools sampled in 2012 (median 8.3 pg/m^3; Cequier *et al.*, 2014).

4.2.2 PBDEs

The relative abundance of individual congeners in indoor air in this project was: BDE-209 > BDE-99 > BDE-47 > BDE-183. The median concentrations of PBDEs in indoor air for all microenvironments were 7.0 pg/m^3 and 300 pg/m^3 for ∑tri-octa BDE (consisting of BDE-17, -28, -47, -49, -66, -99, -100, -153, -154 -183, -196 and -197) and BDE-209, respectively.

BDE-209 was the predominant PBDE congener in all microenvironments, representing 95%, 97%, 64% and 99% of ∑PBDE for homes, cars, offices and schools, respectively – similar to the UK (Tao *et al.*, 2016), Sweden (Thuresson *et al.*, 2012) and Germany (Fromme *et al.*, 2009). Concentrations of BDE-209 in Irish homes are consistent with those for the UK in 2015 (Tao *et al.*, 2016), but those in Irish offices exceed by a factor of 2 those recently recorded in the UK (Tao *et al.*, 2016). Concentrations of BDE-209 in Irish homes and cars are lower than those in Sweden (Thuresson *et al.*, 2012; Newton *et al.*, 2015).

It is difficult to make comparisons between the ∑tri-octa BDE concentrations reported here and elsewhere due to the different congener compositions studied (i.e. the exact PBDE congeners monitored varies between studies). Nonetheless, ∑tri-octa BDE median concentrations in Irish homes (5.6–330 pg/m^3) were lower than those reported in the UK (median 13–2600 pg/m^3) (Tao *et al.*, 2016), whereas those in Irish offices (median 15 pg/m^3; range 5.7–6200 pg/m^3) exceed those in the UK (median 20–150 pg/m^3) (Tao *et al.*, 2016). Lowest median concentrations in this study were in schools (7.0–150 pg/m^3) and were lower than those in South Korea (< DL–33,500 pg/m^3) (Lim *et al.*, 2014) and Norway (< DL–150 pg/m^3) (Cequier *et al.*, 2014).

Median concentrations of BDE-47 (2.1 pg/m^3) and BDE-99 (6.1 pg/m^3) are exceeded by those in the USA (median 52 pg/m^3 and 15 pg/m^3, respectively; Venier *et al.*, 2016). This probably reflects greater use of the penta-BDE formulation in the USA than in Europe (BSEF, 2003).

4.2.3 HBCDDs

The median ∑HBCDD concentrations in Irish homes (20 pg/m^3) were five times lower than those in the UK (110 pg/m^3) (Tao *et al.*, 2016), while median concentrations in Irish cars (25 pg/m^3) are almost 500 times lower, and in Irish offices (14 pg/m^3) were less than half those in the UK in 2008 (Abdallah *et al.*, 2008) (13,000 pg/m^3) and 2015 (Tao *et al.*, 2016) (41 pg/m^3). This may reflect a decreasing trend in HBCDD use. Nonetheless, median concentrations in all Irish microenvironments still exceed those in Sweden (< 2 pg/m^3) in 2006 (Thuresson *et al.*, 2012).

4.3 Differences between Microenvironment Categories

Several studies observed differences in concentrations of BFRs between different microenvironments (e.g. homes compared with offices) (Newton *et al.*, 2015; Tao *et al.*, 2016; McGrath *et al.*, 2018). We therefore examined our data to establish if there were any significant ($p < 0.05$) differences in BFR concentrations between the microenvironments sampled.

4.3.1 Indoor dust

There were no significant differences in DBDPE concentrations between different microenvironments. For PBDEs, concentrations of BDE-209 were significantly lower in offices (median 3500 ng/g) than in homes (median 13,000 ng/g; $p < 0.05$), cars (median 26,000 ng/g; $p < 0.05$) and schools (median 8100 ng/g; $p < 0.05$). This may suggest declining use of this BFR in offices (and to a lesser extent schools), where faster turnover of electronic and electrical goods than in homes is anticipated. Concentrations of BDE-99 in cars (median 50 ng/g) exceed significantly those in offices ($p < 0.05$) and schools ($p < 0.05$) but are statistically indistinguishable from those in homes. Moreover, BDE-183 concentrations are significantly higher in cars (median 4.1 ng/g) than in offices (median 3.2 ng/g; $p < 0.05$). Abdallah *et al*. (2008) and McGrath *et al*. (2018) made similar observations. Higher concentrations of PBDEs have been associated with interiors of vehicles, due to the increased volume of synthetic surfaces, increased volatilisation of BFRs due to high temperatures in unoccupied cars as well as to the smaller air volume and reduced ventilation within cars. No other significant differences were observed between microenvironments.

4.3.2 Indoor air

Differences in BFR concentrations in air were observed between the different microenvironments studied. DBDPE and BDE-209 concentrations in schools were significantly higher than in offices ($p < 0.05$ for both). DBDPE concentrations in schools (median 220 pg/m^3) exceeded significantly those in homes (median 48 pg/m^3; $p < 0.05$); while BDE-209 concentrations (median 410 pg/m^3) in schools exceeded significantly those in cars (median 200 pg/m^3; $p < 0.05$).

Concentrations of BDE-47 in homes (median 2.1 pg/m^3) were significantly lower than those in offices (median 3.4 pg/m^3; $p < 0.05$). BDE-99 concentrations in homes (median 6.1 pg/m^3) exceeded significantly those in schools and cars (median 3.1 pg/m^3 and 2.1 pg/m^3, respectively; $p < 0.05$).

The number of putative sources, the cleaning pattern and the location of the sampler relative to putative sources have been reported in some (but not all) studies to influence BFR concentrations (Abdallah *et al.*, 2008; Harrad *et al.*, 2009; McGrath *et al.*, 2018). The contextual information recorded for participating schools, offices, homes and cars was thus examined but provided no insights into the concentration trends observed.

4.4 Regional Differences between Microenvironments

The study included samples collected from different regions (Limerick, Galway and Dublin) in Ireland. We examined our data to establish if there were any regional differences in BFR concentrations between each of the microenvironments sampled.

4.4.1 Indoor dust

Concentrations of DBDPE in dust from Limerick schools (median 24,000 ng/g) exceeded significantly those in Galway schools (median 1500 ng/g; $p < 0.001$) but were not significantly higher than in Dublin schools (median 14,000 ng/g). Two notably high concentrations of DBDPE (medians 230,000 ng/g and 540,000 ng/g) were found in two Limerick schools; however, the numbers of items of electronic and electrical equipment such as interactive white boards, laptops, compact disc players and foam-containing furniture within all participating schools were similar. Moreover, the purchase of school furniture and electrical equipment is governed by Irish national policy and not by individual schools or regions. We are therefore unable to explain the significantly higher DBDPE concentrations in Limerick schools, which may be attributable to the small sample numbers involved (≈10 schools from each region).

4.4.2 Indoor air

Some statistically significant regional differences in car and school BDE-209 concentrations were observed. In relation to cars, 10–12 cars were sampled in each region; Dublin car concentrations (median 3.8 pg/m^3) were significantly lower than those in Galway (median 300 pg/m^3) and Limerick (median 530 pg/m^3) cars ($p < 0.001$, $p < 0.001$), which could not be explained by the contextual data. Most cars in this study were manufactured in Germany or Asia. A recent survey of BFRs in Irish waste detected high BDE-209 concentrations in end-of-life vehicles manufactured in both Germany and Asia (Drage *et al.*, 2018). The cleaning pattern of the cars did not influence the BDE-209 air concentration and neither did the presence of a child seat or air conditioning.

Some significant ($p < 0.05$) regional differences were observed in concentrations of BDE-209 in schools. Significantly higher BDE-209 concentrations were detected in Galway schools (median 930 pg/m^3) and significant lower concentrations in Dublin schools (median 150 pg/m^3). Higher concentrations (not statistically significantly) were observed in Galway schools in older school buildings (built before 1983), whereas we could not see this trend in Limerick and Dublin. Nearly all classrooms contained one or more foam-containing chairs, although the age of the chairs was difficult to establish.

4.5 Sources of BFRs in Indoor Air and Dust

Our data were analysed to explore associations between BFR concentrations and factors such as the number of electronics present in the room and type of floor surface, etc. However, similar to several other international studies, few obvious trends were found (Cunha *et al.*, 2010; Kalachova *et al.*, 2012; Abdallah *et al.*, 2016). This is most likely to be due to the convenience sampling approach used and the probably variable BFR presence in putative sources.

Concentrations of HBCDDs in office dust were positively correlated with the number of electronics present ($p < 0.01$). Higher air concentrations of ∑HBCDDs ($p < 0.05$) were found in homes (13 out of 32) with carpets. Similar observations have been made in two UK studies (Abdallah *et al.*, 2008; Tao *et al.*, 2016). As HBCDDs were not prevalent in Irish

waste electronics or carpets (Drage *et al.*, 2018), the cause of these correlations is unclear. A positive significant correlation ($p < 0.01$) was found between concentrations of BDE-209 in air and the number of electronics in schools. BDE-209 was widely used in electronic and electrical items up until 2008, and high levels of this BFR have also been detected in waste information technology and telecommunication items in Ireland (Drage *et al.*, 2018).

4.6 Temporal Trends of BFR Concentrations in Indoor Air and Dust

This is the first study of BFR concentrations in Irish indoor air and dust and, therefore, no comparisons can be made with previous Irish data. Correlations between BFR concentrations and year of building construction, car registration and the age of electronics present in the environment were examined, as were differences in concentrations from different age categories (e.g. homes built before and after a given year). Year of home construction was significantly negatively correlated with concentrations of ∑HBCDDs ($p < 0.01$) – i.e. newer homes had lower concentrations – which possibly reflects the impact of recent restrictions on the use of HBCDDs in building insulation materials.

In contrast, significantly higher concentrations of BDE-209 in air ($p < 0.05$) were found in offices containing electronics purchased after 2013 ($n = 16$; 540 pg/m^3) compared with pre 2013 ($n = 10$; average: 250 pg/m^3). Given the recent restrictions on BDE-209 use, this observation is puzzling.

Less surprisingly, homes with a greater number of electronics purchased before 2009 (pre 2009, $n = 8$; post 2009 $n = 21$) had significantly higher concentrations of ∑tri-octa BDE, suggesting a positive impact from legislative restrictions on octa- and penta-BDE.

Year of car registration and concentrations in dust of BDE-47 ($p < 0.01$), BDE-99 ($p < 0.05$) and ∑tri-octa BDE ($p < 0.05$) were negatively correlated. Moreover, concentrations in dust collected from cars ($n = 19$) registered after the listing of penta- and octa-BDE under the Stockholm Convention in 2009 were significantly lower than those in dust from cars registered pre 2009 ($n = 10$) for congeners BDE-47 ($p < 0.01$; average 42 ng/g, cf. 8.6 ng/g), BDE-99 ($p < 0.01$; 89 ng/g, cf. 31 ng/g) and ∑tri-octa BDE ($p < 0.01$; average concentration pre 2009 255 ng/g, post 2009 76 ng/g).

4.7 Exposure Assessment for BFRs in Indoor Air and Dust

BFR concentrations in indoor air and dust were used to estimate exposures of Irish adults, toddlers and school children, via inhalation of airborne BFRs and ingestion of BFRs in dust (Table 4.1) using the algorithms described in section 2.4. Two exposure scenarios were considered: a “typical” exposure scenario using median BFR concentrations and a “high-end” exposure scenario, assuming ingestion/inhalation of the 95th percentile BFR concentrations. In addition, a higher dust ingestion rate (50 mg/day for adults and 200 mg/day for children) was used for the high-end scenario calculation. High-end exposure scenario estimates for DBDPE (adult 120 ng/kg bw/day, toddler 2500 ng/kg bw/day) and BDE-209 (adult 100 ng/kg bw/day, toddler 2500 ng/kg bw/day) exceed the equivalent high-end exposures reported recently for the UK (Tao *et al.*, 2016) (adult 3.4 ng/kg bw/day, 57 ng/kg bw/day; toddler 33 ng/kg bw/day, 1900 ng/kg bw/day for DBDPE and BDE-209, respectively). By comparison, ∑HBCDD exposure estimates for adults (7.8 ng/kg bw/day) and toddlers (170 ng/kg bw/day) are below UK results (adult 22 ng/kg bw/day, toddler 750 ng/kg bw/day) (Tao *et al.*, 2016). The high-end exposure estimates (1100 ng/kg bw/day) for BDE-209 and (86 ng/kg bw/day) ∑HBCDDs calculated for school children (aged 4–6 years) are below UK values of 330 ng/kg bw/day and 1300 ng/kg bw/day, respectively (Harrad *et al.*, 2010a).

High-end estimates of exposure to BDE-209 for Irish adults, toddlers and school children (Table 3.8) are 100 ng/kg bw/day, 2500 ng/kg bw/day and 1100 ng/kg bw/day, respectively, and below the US EPA RfD value for adults of 7000 ng/kg bw/day (US EPA, 2008c). Those for BDE-47, BDE-99 and ∑HBCDDs are also below US guideline values (NRC, 2000; US EPA, 2008a,b). Our estimates of typical adult exposure via inhalation and dust ingestion exceed Irish dietary exposure estimates for BDE-209 (0.3 ng/kg bw/day) but fall below those for ∑PBDEs (2.4 ng/kg bw/day) (Trudel *et al.*, 2010).

Table 4.1. Estimates of exposure (ng/kg bw/day) of Irish adults, toddlers and school children to BFRs via indoor air, inhalation and dust ingestion under typical[a] and high-end[b] exposure scenarios[c]

		α-HBCDD	β-HBCDD	γ-HBCDD	∑HBCDDs	DBDPE	BDE-209	BDE-47	BDE-99	BDE-183	∑tri-octa-BDEs
Adult											
Air	Median	0.00035	0.00025	0.0039	0.0071	0.016	0.13	0.0010	0.0022	0.00040	0.0067
	High	0.073	0.037	0.34	0.46	1.2	1.7	0.012	0.10	0.0023	0.10
Dust	Median	0.075	0.029	0.085	0.14	1.3	3.1	0.0023	0.0036	0.00045	0.013
	High	2.4	0.83	4.1	7.4	120	99	0.13	0.14	0.017	0.35
Total	Median	0.075	0.029	0.088	0.14	1.3	3.2	0.0033	0.0058	0.00084	0.020
	High	2.5	0.87	4.4	7.8	120	100	0.14	0.25	0.020	0.45
UK (Tao *et al.*, 2016) total high		6.1	3.4	13	22	3.4	57	0.64	0.81	0.093	1.7
Toddler											
Air	Median	0.00019	0.000055	0.0037	0.0073	0.019	0.14	0.0008	0.0021	0.00020	0.0067
	High	0.020	0.0066	0.35	0.37	1.4	1.9	0.0083	0.11	0.0025	0.11
Dust	Median	0.98	0.51	1.0	2.5	21	64	0.039	0.069	0.0054	0.25
	High	49	18	98	170	2500	2500	3.2	3.3	0.31	7.6
Total	Median	0.98	0.51	1.0	2.5	21	64	0.040	0.071	0.0056	0.26
	High	49	18	99	170	2500	2500	3.2	3.4	0.31	7.7
UK (Tao *et al.*, 2016) total high		200	120	430	750	33	1900	15	24	2.3	100
School child											
Air	Median	0.0008	0.00003	0.0028	0.0052	0.019	0.090	0.00048	0.0012	0.00012	0.0039
	High	0.017	0.074	0.17	0.25	0.62	1.2	0.0042	0.044	0.0010	0.045
Dust	Median	0.61	0.30	0.47	1.42	14	29	0.018	0.030	0.0022	0.12
	High	25	11	48	86	1400	1100	1.3	1.4	0.14	3.6
Total	Median	0.61	0.30	0.47	1.4	14	30	0.019	0.031	0.0023	0.12
	High	25	11	48	86	1400	1100	1.3	1.4	0.14	3.7
UK (Harrad *et al.*, 2010b) dust high					330		13,000		4.3		
RfDs (US EPA, 2008a,b,c; NRC, 2000)					**200,000**		**7000**	**2000**	**100**	**3000**	

[a]Typical exposure scenario suggests adult and toddler exposure to air inhalation and dust ingestion at the median concentration at the average ingestion rates (air: 20 m^3/day for adults and 3.8 m^3/day for toddlers and school children; dust: 20 mg/day for adults and 50 mg/day for toddlers and school children.

[b]High-end exposure scenario suggests adult and toddler exposure to air and dust ingestion at the 95th percentile concentration using high ingestion rates (adult: 50 mg/day; toddlers and school children: 200 mg/day).

[c]All values expressed as ng/kg bw/day, assuming a body weight of 70 kg for adults, 10 kg for toddlers and 20 kg for school children.

Source: Wemken *et al.* (2019). Reproduced with permission of the American Chemical Society.

4.8 PFASs in Indoor Air, Dust and Drinking Water

4.8.1 Indoor dust

Table 3.4 provides median concentrations from selected previous studies to provide context for our data relating to concentrations in Irish indoor dust. As with indoor air, most data exist for homes, with concentrations in Irish homes among 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.

4.8.2 Indoor air

Table 3.5 provides data on concentrations of PFASs in indoor air from selected other studies worldwide. To our knowledge, the data reported in this project for Ireland are the first for PFASs in air from car interiors anywhere in the world. 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 five 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 show lower concentrations than in these other studies, with the exception of PFNA and PFOA, where Irish median concentrations are highest. Our study appears to be the first report of PFOA, PFOS, PFBS, PFHxS and PFNA in school classroom air; EtFOSE and MeFOSE were measured (but were both below the limit of detection) in German school classrooms (Fromme *et al.*, 2015), while data were reported for a good range of PFASs in Czech university classrooms (Karásková *et al.*, 2018). 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.

4.8.3 Drinking water

As well as the concentrations of PFASs detected in this study, Table 3.6 includes for comparison median concentrations of PFASs in drinking water in a range of previous studies from elsewhere in the world – with further comparative data available elsewhere (Endirlik *et al.*, 2019). This comparison shows concentrations in Irish tap water to be among 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 with two stringent drinking water guidelines. The first of these was promulgated by Swedish authorities and specifies a limit of 90 ng/L for ∑PFASs (which include some of those targeted in our study) (Livsmedelsverket, 2016). Most recently, a proposal to revise the Drinking Water Directive was agreed (Council of the European Union, 2020). This specifies a limit in drinking water of 100 ng/L based on the sum of a range of PFASs that include some but not all of those targeted in our study. Inspection of our data reveals that neither of these limits is exceeded for any sample in our study, but it was approached in one sample of bottled water where ∑PFASs = 64 ng/L. Moreover, the US EPA has specified an HBLV of 70 ng/L for the sum of PFOS and PFOA concentrations (US EPA, 2016a,b), 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 children (Grandjean and Budtz-Jorgensen, 2013). 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.

4.9 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 correlations between the year of building/car construction and PFAS concentrations. In addition, we evaluated whether PFASs concentrations in buildings/cars built before 2005 differed from those built post 2005. We also compared PFAS concentrations in samples

containing a putative source [e.g. carpet (Fraser *et al.*, 2013), child seat, application of stainproofing agents] with other samples where the putative source was not present. Finally, we evaluated whether the county (Dublin, Galway or Limerick) from which samples from a given microenvironment were taken exerted a significant influence on concentrations of PFASs. Probably due to the multiple influences on PFAS concentrations, we found only one significant difference. This was for car air, with concentrations of PFOA and PFNA significantly higher ($p \leq 0.05$) in cars containing child seats ($n = 12$) than in those that did not ($n = 17$). Moreover, when a sample from a car not containing a child seat that contained an unusually elevated concentration of MeFOSE (160 ng/g) 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 ng/g in cars containing child seats and 63 ng/g in those without them, with the corresponding data for PFNA and MeFOSE being 5.7 and 1.5 ng/g, and 6.9 and 1.3 ng/g, respectively. This may indicate the use of these PFASs to stainproof the fabrics used on such child car seats. While we are unaware of direct evidence of the application of PFASs to stainproof child car seats, application of PFASs to stainproof fabrics is well documented (Kissa, 2001). 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 DFs for PFOA, PFNA and MeFOSE in dust compared with air.

4.10 Comparisons of Concentrations of PFASs in Indoor Air and Dust between Different Indoor Microenvironments

Our previous studies of UK indoor air and dust revealed differences in concentrations of PFASs between different microenvironment categories (Goosey and Harrad, 2011, 2012). Such differences are likely to be due to the different types and abundance of PFAS sources in these different types of microenvironment. We therefore examined our data for such differences.

4.10.1 Indoor air

The following significant ($p < 0.05$) differences were detected: (1) concentrations of PFHxS in office air exceeded those in all other microenvironment categories; and (2) for PFOS, concentrations in classroom air exceeded those in homes. No other significant differences in concentrations of PFASs in different microenvironments were detected ($p > 0.05$).

4.10.2 Indoor dust

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

4.11 Comparison between Concentrations of PFASs in Bottled Water and Tap Water from Municipal and Private Water Supplies

We examined our data for significant ($p \leq 0.05$) differences in concentrations of individual target PFASs in (1) tap water from homes and offices connected to municipal water supplies, (2) tap water from homes connected to private water supplies and (3) bottled water. This analysis revealed that (1) concentrations of PFOA in tap water from private supplies exceeded significantly those in tap water from municipal supplies, (2) concentrations of PFOS and PFBS in bottled water exceeded those in tap water from both municipal and private supplies, and (3) MeFOSA concentrations in tap water from private supplies exceeded those in tap water from municipal supplies. We are unable to explain these differences in concentrations of some PFASs between different sample types.

4.12 Exposure to PFASs via Drinking Water, Inhalation and Dust Ingestion

Exposure of the Irish population to PFASs via drinking water, inhalation and dust ingestion was estimated for adults and young children based on concentrations

reported here. The algorithms and assumptions applied to estimate exposure via different routes and under three scenarios of "low-end", "typical" and "high-end" exposure are described in section 2.4. These data facilitate (1) evaluation of the relative importance of different exposure pathways for different chemicals; and (2) risk assessment by comparison of exposure with existing or future HBLVs. Table 4.2 summarises exposures for our target PFASs via all three pathways combined. To place these exposure estimates in context, we compare them with previously reported estimates of dietary exposure for other European countries combined (EFSA, 2018) and the UK (D. Mortimer, Food Standards Agency, July 2019, personal communication), in the absence of such data currently for Ireland. Table 4.2 also expresses the

Table 4.2. Estimates of exposure (pg/kg bw/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 TWI (EFSA, 2018)	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 Europe (EFSA, 2018)	320	–	–	610	–	–	–	–	–
Typicald dietary exposure toddlers Europe (EFSA, 2018)	2010	–	–	750	–	–	–	–	–
Typical dietary exposure adult UK (D. Mortimer, 2019, personal communication)	3900	–	–	1800	–	–	–	–	–
Typical dietary exposure toddlers (1–4.5 years) UK (D. Mortimer, 2019, personal communication)	9600	–	–	4500	–	–	–	–	–

NB Typical UK dietary exposure to ∑PFASs in 2012 was 60,000 pg/kg bw/day and 140,0000 pg/kg bw/day for adults and toddlers (1–4.5 years), respectively.

[a]Typical exposure scenario assumes adults and toddlers inhale air and ingest dust contaminated at the median concentration and assuming an average dust ingestion rate (20 mg/day and 50 mg/day for adults and toddlers, respectively).

[b]Low exposure scenario assumes adults and toddlers inhale air and ingest dust contaminated at the 5th percentile concentration and assuming an average dust ingestion rate (20 mg/day and 50 mg/day for adults and toddlers, respectively).

[c]High exposure scenario assumes adults and toddlers inhale air and ingest dust contaminated at the median concentration and assuming a high dust ingestion rate (50 mg/day and 200 mg/day for adults and toddlers, respectively).

[d]Median lower bound estimates.

Source: Harrad *et al.* (2019). Reproduced with permission of the American Chemical Society.

relative percentage contribution of each pathway for both adults and toddlers under our typical exposure scenarios for each pathway. In addition, these data are illustrated graphically for the four target PFASs for which typical total exposure is highest, i.e. PFOA, PFOS, PFBS, and MeFOSA (Figure 4.1). It is striking 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. A slight caveat to this is that our assumption that children drink 2 L of water daily may be an overestimate. Likewise, drinking water is the most important exposure pathway (>65% total exposure) for adults. An important caveat to this is that for the two 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 with 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 (EFSA, 2018; D. Mortimer, 2019, personal communication). We also compared our estimates of non-dietary exposure with EFSA's provisional TWI values of 6 ng/kg bw/week for PFOA and 13 ng/kg bw/week for PFOS (EFSA, 2018). 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

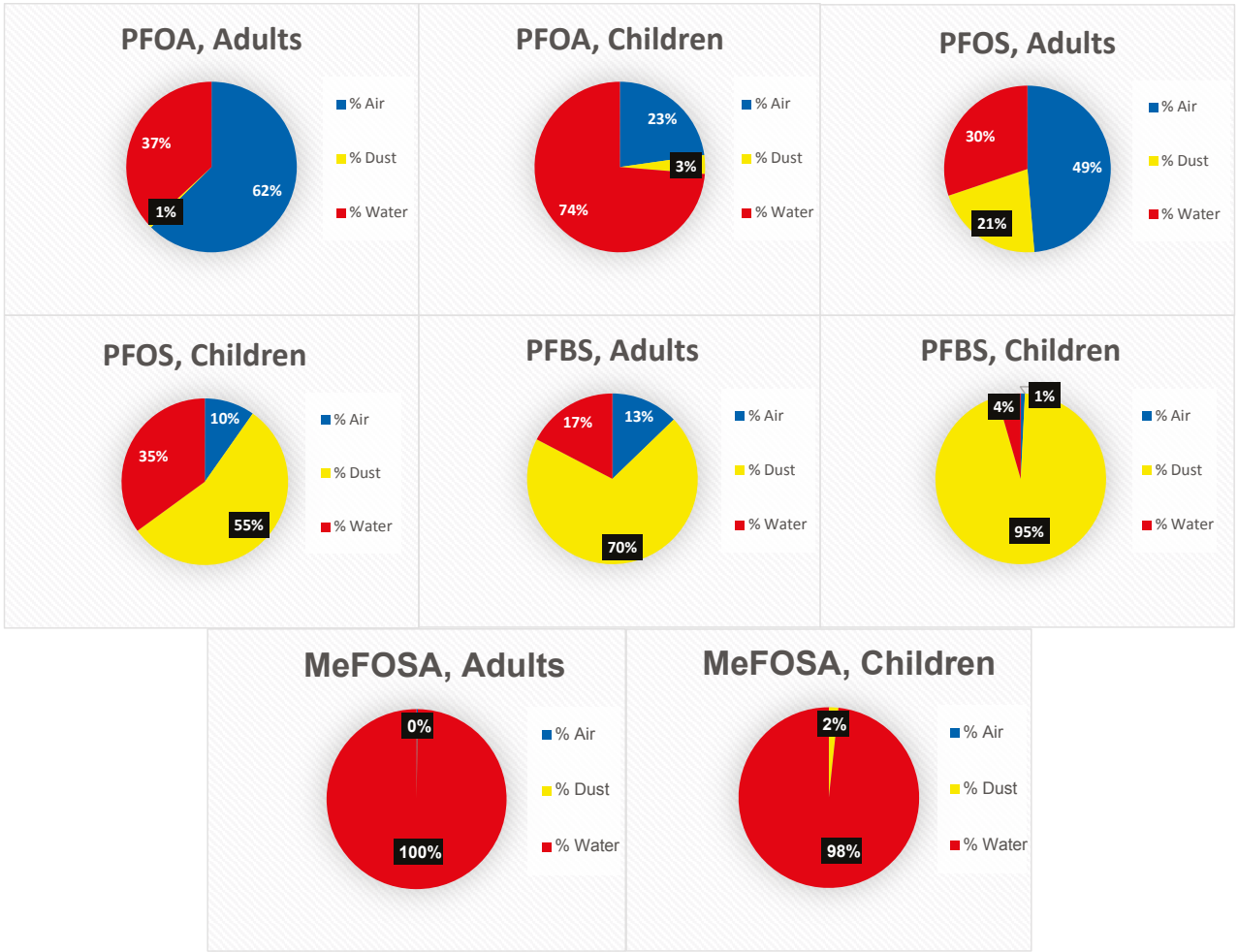

Figure 4.1. Relative contribution (expressed as %) of different target PFASs to the overall daily exposure (ng/day) of Irish toddlers and adults via drinking water, inhalation and dust ingestion under typical exposure scenarios. Source: Harrad *et al.* (2019). Reproduced with permission of the American Chemical Society.

currently of estimates of dietary exposure of the Irish population to PFOA and PFOS, we cannot – based on our indoor air, dust and drinking water data – definitively assess whether EFSA's 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 suggest 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 HBLV currently exists, and thus EFSA's ongoing assessment of the risks to human health from PFBS and other PFASs is welcome.

4.13 PBDEs and HBCDDs in Human Milk

The relative abundance of our target BFRs in this study contrasts with that observed in the previous Irish survey of BFRs in human milk collected in 2011 (Pratt *et al*., 2013) (Table 3.3). In the earlier study, although BDE-209 was still one of the most abundant congeners (21% $\sum PBDE_{28:209}$), it was less predominant than either BDE-47 (31% $\sum PBDE_{28:209}$) or BDE-153 (28% $\sum PBDE_{28:209}$). The concentrations of BDE-209 in our study (median 1.4 ng/g lw) are at the high end of those reported in previous European studies (Fromme *et al*., 2016). Specifically, they exceed those reported for UK human milk samples collected in 2010 (median 0.25 ng/g lw; Abdallah and Harrad, 2014) and between 2014 and 2015 (median < 0.22 ng/g lw; Tao *et al*., 2017), as well as those reported in samples collected in France and Finland between 2011 and 2014 (Antignac *et al*., 2016). The concentration of BDE-209 was noticeably elevated in pool 12, at 24 ng/g lw. While this pooled sample contained milk from both urban and rural dwellers, thus eliminating urbanisation as a likely cause of the elevated BDE-209 concentration, donors to this pool consumed at least two fish meals per week. This may imply that fish consumption is a source of elevated BDE-209 exposure. This is consistent with the observation of Garcia-Lopez *et al*. (2018), who reported BDE-209 to be – along with BDE-47 – the only PBDE to have a DF of 100% in Irish fish samples and that no other congener had a DF of 100% in any of the other food groups analysed by Garcia-Lopez *et al*. (2018). Against this, pool 12 contained a typical concentration of BDE-47 despite the 100% DF of this congener in Irish fish samples. An alternative explanation for the elevated BDE-209 in pool 12 is that some of the donors could have high exposures via dust ingestion. This is plausible, given our previous observations of BDE-209 concentrations in Irish house dust of up to 650,000 ng/g (Table 3.1).

$\sum PBDE_{28:183}$ concentrations in this study (median1.4 ng/g lw) are below those reported in the UK, Finland and Denmark (Abdallah and Harrad, 2014; Antignac *et al*., 2016) between 2010 and 2014, but exceed those reported in France for the same period (Antignac *et al*., 2016). BDE-153, followed by BDE-47, dominates our $\sum PBDE_{28:183}$ concentrations. A recent review revealed that BDE-153 and -47, along with BDE-99, tend to dominate the $\sum PBDE_{28:183}$ profiles for human milk in most studies (Fromme *et al*., 2016). Moreover, over the last decade, the proportion of BDE-153 compared with that of BDE-47 has risen, with similar proportions of BDE-153 to those found in this study having been reported in Germany (52% of $\sum PBDE_{28:183}$) for studies performed in 2007 (Hoopmann *et al*., 2009). This increasing predominance of BDE-153 in human milk has been attributed to it possessing a longer human half-life than other congeners and to possible *in vivo* metabolism of higher molecular weight PBDEs to BDE-153 (Abdallah and Harrad, 2014).

Concentrations of ∑HBCDDs (median 1.8 ng/g lw) in this study are slightly lower than those reported in samples collected in the UK in 2014–15 (2.9 ng/g lw; Tao *et al*., 2017) but exceed those reported in France, Denmark and Finland (Antignac *et al*., 2016) (for which only α-HBCDD was reported). The α-HBCDD diastereomer was dominant in our samples (on average 75% of ∑HBCDDs). This is similar to previous studies of HBCDDs in breast milk in which α-HBCDD typically contributes between 65% and 84% ∑HBCDDs (Fromme *et al*., 2016).

4.14 Temporal Trends in Concentrations of PBDEs and HBCDDs in Irish Human Milk

We compared concentrations of individual PBDEs and ∑HBCDDs in individual pools in our study with those reported for individual pools in the 2011 Irish study (Pratt *et al*., 2013). The detection limits in our study for BDE-154 and -183 exceeded concentrations detected in all samples collected in 2011, resulting in only BDE-183 being detected in one sample

in our study and precluding meaningful statistical analysis of any temporal trend for these congeners. In contrast, concentrations of BDE-47, -99, -100 and -153 in samples from our study are significantly lower ($p < 0.05$) than those recorded in samples taken in 2011 from mothers attending the same hospitals (Pratt *et al*., 2013). The decline in concentrations of these PBDEs in Irish human milk is likely to be a result of the impact of legislative bans on the use of the penta- and octa-BDE formulations, and is consistent with a recent report (Garcia Lopez *et al*., 2018) that concentrations of PBDEs in Irish foods collected in 2015 were lower than those collected in 2010 (Trudel *et al*., 2010). In addition, concentrations of ∑HBCDDs in Irish human milk are significantly lower ($p < 0.05$) in our study than in samples collected in 2011. This may also provide encouraging evidence that recent restrictions on the use of HBCDDs in Ireland have had a beneficial impact on human body burdens. The evidence provided here of declines in Irish human body burdens in concentrations of some PBDEs and HBCDDs that contrasts with recent findings for the UK (Tao *et al*., 2017) is welcome and suggests that future studies in the UK and other locations where these BFRs have been restricted will reveal similar declines in human body burdens. In contrast, and in line with our recent observations in the UK, BDE-209 concentrations in our study did not differ significantly from those reported previously (Pratt *et al*., 2013), suggesting no discernible response yet in Ireland to the restrictions on the manufacture and use of the deca-BDE formulation, which took effect later than those on HBCDDs, penta-BDE and octa-BDE.

4.15 Concentrations and Temporal Trend of DBDPE in Human Milk from Ireland

There have been few reported studies of DBDPE concentrations in human milk and so only limited comparisons can be made with our DBDPE data. Despite similar limits of detection in both studies (2.5 ng/g lw in this study and 2.1–2.5 ng/g lw in the Pratt *et al*., 2013 study), DBDPE was detected in 3 out of 16 samples in this study, compared with none in Irish samples collected in 2010. This increasing detection rate is in line with an increase in DF of DBDPE in UK human milk from 4% in samples collected in 2010 to 10% in those obtained in 2014–15 (Tao *et al*., 2017). This increased – albeit still low at 19% – DF for DBDPE in Irish human milk in our study is likely to be related to our recent report of elevated concentrations of DBDPE in Irish indoor air and dust (Tables 3.1 and 3.2). Concentrations of DBDPE measured in this study (range < 2.5–4.6 ng/g lw) compare with those reported in Canada for milk samples collected in 2008–09 (range 1.7–25 ng/g lw) (Zhou *et al*., 2014), in the UK in 2014–15 (range < 0.78–58 ng/g lw) (Tao *et al*., 2017), in China in samples collected in 2011 (range 2.45–21.8 ng/g lw) (Shi *et al*., 2016), and in New Zealand in 2008 (range 0.016–0.33 ng/g lw) (Mannetje *et al*., 2013).

4.16 Nursing Infants' Dietary Intake of BFRs via Breast Milk

Table 4.3 summarises estimated average and median intakes of our target BFRs via breast milk for a 1-month-old infant assuming C_{BFR} (see equation 2.9, section 2.5) are the average and

Table 4.3. Estimated exposure[a,b] (ng/kw bw/day) of a 1-month-old infant to target BFRs and DBDPE via ingestion of breast milk contaminated at the average and median levels in this study

BFR	Average	Median
BDE-28[c]	0.18	0.18
BDE-47	3.7	2.6
BDE-100[c]	0.59	0.59
BDE-99[c]	0.59	0.59
BDE-154[c]	0.35	0.35
BDE-153	4.6	4.4
BDE-183[c]	0.11	0.11
$\sum PBDE_{28:183}$	8.5	8.5
BDE-209	17	9.4
$\sum PBDE_{28:209}$	25	15
DBDPE[c]	2.8	2.8
α-HBCDD	8.8	7.7
β-HBCDD	1.3	1.1
γ-HBCDD	1.4	0.8
∑HBCDDs	12	10

[a]Values below LOQ were assumed to be ½ LOQ, where the DF was > 50%, DF < 50% the LOQ × DF.

[b]Based on a daily lipid intake of 24.4 lipid/day and an average body weight of 14.14 kg (US EPA, 2011).

[c]Substantial uncertainty as DF was < 50%.

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median concentrations in breast milk in this study. Infant exposures to BDE-47, -99 and -209 are all less than relevant US EPA RfD values (US EPA, 2008a,b,c). The estimate for BDE-209 (median 9.4 ng/kg bw/day) exceeds that based on UK human milk samples collected in 2014–15 (median 0.65 ng/kg bw/day; Tao *et al*., 2017) and reported estimates for Germany and the USA of 0.27 and 0.87 ng/kg bw/day, respectively, calculated for 3- to 6-month-old infants weighing 5 kg and consuming 30 mL of breast milk lipid per day (Fromme *et al*., 2016). Our exposure estimates for BDE-47 (median 2.6 ng/kg bw/day) accord with those for Germany (median 3.0 ng/kg bw/day), but are exceeded by those for the UK (median 13 ng/kg bw/day) and the USA (median 173 ng/kg bw/day) (Fromme *et al*., 2016; Tao *et al*., 2017). With respect to DBDPE, our exposure estimates (median 2.8 ng/kg bw/day) exceed slightly those reported for the UK (median 2.3 ng/kg bw/day; Tao *et al*., 2017) but are lower than estimates reported for China (Shi *et al*., 2016).

Exposure estimates in this study for ∑HBCDDs (median 10 ng/kg bw/day) are slightly higher than those reported for Germany and the USA (median 7.7 ng/kg bw/day and 6.6 ng/kg bw/day, respectively; Fromme *et al*., 2016) but lower than that reported for the UK (median 17 ng/kg bw/day; Tao *et al*., 2017).

4.17 Relationship between BFR Intakes and Human Body Burdens

The relationship between external dietary, inhalation and ingestion exposure and human body burdens (indicated by concentrations in breast milk) was examined for PBDEs, HBCDDs and DBDPE, using a first-order PK model (Table 4.4). Predicted body burdens were calculated using our median and average exposure estimates for Ireland for air and dust and dietary intake (Tlustos *et al.*, 2005, 2006; Garcia-Lopez *et al*., 2018). No estimates of dietary exposure to HBCDDs or DBDPE are currently available for Ireland and thus UK data were used as a proxy for Irish dietary exposure (FSA, 2006a; Tao *et al*., 2017).

Predicted body burdens for BDE-47 and BDE-154 agree well with observed values recorded in our study, with dietary exposure the predominant exposure pathway. In contrast, our observed body burdens of BDE-153 exceed predicted values; this is most likely to be a result of the long half-life of BDE-153 (Geyer *et al*., 2004; EFSA, 2011a), resulting in observed body burdens reflecting past as well as current exposure, as well as via metabolic stepwise meta–meta debromination of BDE-209 *in vivo* to yield BDE-153 (Roberts *et al*., 2011). Discrepancies between predicted and measured exposures to BDE-209, whereby observed values exceed predicted exposures, are more difficult to explain, given the short human half-life of this congener (Geyer *et al*., 2004). Possible reasons for the overestimation of the model are that the bioaccessibility and human half-life values used in the model for BDE-209 are underestimated, and that exposures such as dermal uptake from BDE-209-containing fabrics (Abdallah and Harrad, 2018) are not considered. While observed average DBDPE body burdens were below detection limits (< 2.5 ng/g lw) and thus hard to compare with predicted values, the fact that three samples in this study contained > 3 ng DBDPE/g lw suggests that the PK model used here similarly underestimates body burdens of DBDPE. Additional uncertainty associated with our predicted DBDPE body burdens arises from the lack of Irish dietary exposure data for this BFR, with the result that we have used the UK dietary exposure estimate of Tao *et al.* (2017).

Average and median predicted HBCDD body burdens exceed those observed in this study. This is likely to be a result of the dietary estimates used in the PK model, which in the absence of Irish dietary exposure data were based on UK estimates of dietary exposure (FSA, 2006a) that probably reflect higher dietary exposures in the past.

4.18 PFASs in Human Milk

Table 3.7 compares our data with those from selected other studies. Such a 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

Table 4.4. Estimated average and median daily intakes[a] of selected target PBDEs, DBDPE and HBCDDs and comparison of adult body burdens predicted using a simple PK model with those observed in human milk samples

	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	Σ5BDEs	BDE-209	DBDPE	α- HBCDD	β- HBCDD	γ- HBCDD	Σ HBCDDs
Average intake (ng/day)													
Dust[b]	0.45	0.79	0.14	0.060	0.065	0.11	1.5	880	610	5.2	2.0	5.9	9.5
Diet[c]	6.5	3.1	1.5	0.79	0.89	0.31	13	27	16	200	110	112	410
Air[b]	0.81	0.97	0.079	0.025	0.026	0.028	1.9	20	9.6	0.025	0.018	0.27	0.50
Median intake (ng/day)													
Dust[b]	0.16	0.25	0.0050	0.013	0.018	0.031	0.44	220	91	33	10	11	46
Diet[c]	3.8	2.7	0.64	0.68	0.49	0.26	8.5	24	9.5	200	110	110	410
Air[b]	0.073	0.16	0.024	0.023	0.024	0.028	0.33	9.4	1.1	0.60	0.28	2.6	3.4
Average predicted body burdens (ng/g lw)													
Dust	0.022	0.037	0.0050	0.0043	0.0042	0.0008	0.073	0.14	0.11	0.41	0.038	0.037	0.42
Diet	0.61	0.27	0.099	0.13	0.051	0.0021	1.2	0.0043	0.0027	2.5	0.38	0.37	3.8
Air	0.10	0.16	0.0080	0.0058	0.0052	0.0002	0.28	0.023	0.012	0.0082	0.0013	0.012	0.041
Sum	**0.74**	**0.46**	**0.11**	**0.14**	**0.060**	**0.0031**	**1.5**	**0.17**	**0.12**	**3.0**	**0.42**	**0.42**	**4.2**
Median predicted body burdens (ng/g lw)													
Dust	0.0077	0.012	0.0002	0.0009	0.0012	0.0002	0.022	0.035	0.016	0.066	0.0073	0.019	0.087
Diet	0.36	0.23	0.043	0.11	0.028	0.0018	0.77	0.0039	0.0017	2.5	0.38	0.37	3.8
Air	0.0092	0.025	0.0025	0.0054	0.0049	0.0002	0.047	0.011	0.0014	0.0003	0.0001	0.0012	0.0059
Sum	**0.37**	**0.27**	**0.046**	**0.12**	**0.034**	**0.0023**	**0.84**	**0.050**	**0.019**	**2.6**	**0.39**	**0.39**	**3.9**
Observed body burdens (ng/g lw)													
Average	0.64	<0.2	<0.2	0.78	<0.12	<0.3	1.4	2.8	<2.5	1.5	0.23	0.27	2.0
Median	0.50	<0.2	<0.2	0.71	<0.12	<0.3	1.4	1.4	<2.5	1.4	0.19	0.16	1.8

[a]Values below LOQ were assumed to be 1/2 LOQ; based on average adult dust ingestion rate of 20 mg/day and average inhalation rate of 20 m^3/day for adults.

[b]Data for Ireland (this study).

[c]Dietary exposures for PBDEs estimated from the average consumption rates calculated for each food group (Garcia Lopez *et al.*, 2018; Tlustos *et al.*, 2005, 2007), with upper bound values used. Those for DBDPE are taken from the UK (Tao *et al.*, 2017), with those for HBCDDs also taken from UK data (FSA, 2006a).

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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.

4.19 Nursing Infants' Intake of PFASs via Breast Milk

Table 4.5 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 average concentrations reported in this study. As noted earlier, EFSA has proposed provisional 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 breastfeeding 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.

Table 4.5. Estimated exposure[a] (ng/kg bw/day) of a 1-month-old nursing infant to PFASs in Irish human milk

PFAS	Mean	Median
PFOA	22	18
PFHxS	5.0	2.1
PFOS	6.4	3.5
PFNA	4.4	2.4

[a]Assuming a daily breast milk intake of 702 mL/day and a body weight of 4.14 kg (US EPA, 2011).

4.20 Pharmacokinetic Modelling of Daily Intakes of PFOS and PFOA Required to Support Observed Human Body Burdens in Ireland

Equation 2.8 was used to derive values of absorbed daily 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 (Table 4.2). Table 4.6 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 body burden is – at 728 pg/kg bw/day – well within the provisional EFSA TWI value that is equivalent to 1857 pg/kg bw/day. The situation is less reassuring for PFOA. As shown in Table 4.6, while average and median body burdens do not suggest additional exposures of concern, the maximum PFOA concentration in human milk in this study suggests additional exposure of 1478 pg/kg bw/day, which is approximately twice EFSA's provisional TWI for PFOA. It is important to stress at this point the uncertainties inherent in the PK model employed here. Specifically, more research is required to enhance our knowledge of the human half-lives, absorption efficiencies and partitioning ratios between breast milk and serum for PFASs. Based on current understanding of human exposure to PFOS and PFOA, the major contributor to our predicted additional exposures is likely to be the diet. However, we highlight that other exposure pathways, such as dermal uptake of PFASs from fabrics and cosmetics, may also contribute considerably to human exposure. Research to characterise the exposure of the Irish population to PFASs via the diet and dermal uptake is thus recommended.

Table 4.6. 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 covering inhalation of indoor air and ingestion of indoor dust and drinking water (this study).

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

[d]EFSA's provisional TWI (EFSA, 2018) converted here for the purposes of comparison with our TDI estimates.

Source: Abdallah *et al*. (2020). Reproduced with permission of Elsevier.

5 Conclusions and Recommendations

This project measured a range of BFRs and PFASs in samples of indoor air and dust collected from Irish homes, offices, cars and school classrooms ($n = 30$ per microenvironment category). The same contaminants were measured in 16 samples of human milk donated by Irish mothers, created from samples from 92 individual mothers. Median concentrations of DBDPE in air (88 pg/m^3) and dust (6500 ng/g) exceed significantly those previously reported internationally, with concentrations highest in offices and schools, suggesting that DBDPE is widely used in Ireland. Median concentrations of BDE-209 in air (340 pg/m^3) and dust (7100 ng/g) exceed or are within the range of concentrations reported recently for the same microenvironments in the UK and exceed those reported in many other countries. Concentrations of BDE-209 in cars exceeded significantly ($p < 0.05$) those in other microenvironments. HBCDDs were detected in all dust samples (median 580 ng/g) and in 81% of air samples (median 24 pg/m^3) at concentrations similar to those reported recently for the UK and elsewhere. Estimates of exposure to DBDPE of Irish adults (92 ng/day) and toddlers (210 ng/day), as well as to BDE-209 (220 ng/day and 650 ng/day for adults and toddlers, respectively), exceed substantially those reported for the UK population. Moreover, our estimates of exposure of the Irish population to ∑tri-deca-PBDEs exceed previous estimates for Ireland via dietary exposure.

Comparison of concentrations of BFRs in human milk detected in this study with those in a previous study conducted in Ireland in 2011 reveals that restrictions on the manufacture and use of HBCDDs and both the penta- and octa-BDE products appear to have been successful in reducing concentrations of these BFRs in Irish human milk. In contrast, probably as a consequence of the more recent ban on the manufacture and use of the deca-BDE product, concentrations in human milk in this study show no significant decline compared with the 2011 study. Moreover, while in 2011, DBDPE – a likely replacement for deca-BDE – was not detected in any human milk sample, it was detected in 3 out of 16 samples in this study. This is consistent with increasing use of DBDPE as a "drop-in" replacement for deca-BDE and is supported by the above findings that concentrations of DBDPE in both indoor air and dust in this study are the highest reported to date anywhere. This probably reflects the fact that this study is one of the few conducted since the listing of deca-BDE under the Stockholm Convention and it is considered that future studies elsewhere will probably reveal similarly elevated concentrations of DBDPE.

A single-compartment PK model suggests that, on average, dietary exposure may be the most prominent exposure pathway for our target BFRs, with the exception of BDE-209 and DBDPE, for which dust ingestion predominates. While there was reasonable agreement between predicted and observed body burdens for tri-octa-BDEs and HBCDDs, predicted body burdens were markedly lower than those observed for BDE-209 and DBDPE. This may be attributable to a combination of bioavailability and human half-lives being underestimated for these BFRs and to exposure from pathways such as dermal uptake from BFR-treated fabrics not being considered in the model.

In addition to indoor air and dust, PFASs were also measured in samples of tap ($n = 85$) and bottled water ($n = 31$). PFOA was the dominant PFAS in indoor air and drinking water, while PFBS was dominant in indoor dust. Concentrations of PFOS in classroom air exceeded significantly those in homes. Concentrations of PFOA, PFNA and MeFOSE in air were significantly higher in cars containing child car seats than in cars without. PFOS, PFOA, PFBS and PFHxS were all detected frequently in drinking water but at low concentrations and, although the concentration of ∑PFASs was 64 ng/L in one bottled water sample, this fell below a Swedish action level of 90 ng ∑PFASs/L. The Irish population's exposure to PFOS and PFOA via non-dietary sources was shown to fall well below estimates of dietary exposure elsewhere in Europe. Moreover, even under a high-end exposure scenario, it falls below EFSA's provisional TWIs for PFOS and PFOA.

PFOA, PFOS, PFHxS and PFNA were detected in Irish human milk at concentrations within the range of those reported elsewhere in the world. Moreover,

other PFASs were not detected in any sample. While concentrations of PFASs in the previous (2011) study of PFASs in Irish human milk were all below detection limits, those detection limits were higher than the concentrations reported in this study. This therefore precludes any meaningful evaluation of temporal trends in body burdens of PFASs in Irish mothers. Reassuringly, concentrations of PFOS and PFOA in Irish human milk in this study currently do not indicate a health concern, based on breastfeeding exposure scenarios carried out by EFSA. A one-compartment PK model was used to predict the intakes of PFOS and PFOA required to support the observed concentrations in human milk. This suggests that current adult exposure in Ireland to PFOS is below the provisional 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.

Based on our findings, the following recommendations are made.

- Further research into exposure to DBDPE and other emerging FR chemicals in Ireland and the consequent health effects are a priority.
- In view of the revised Drinking Water Directive and the specification of limit values for PFASs, further monitoring of Irish drinking water would appear prudent, alongside research to elucidate the sources of any such contamination. Related to this, research is recommended into identifying the fate of PFASs associated with AFFFs used in Ireland, given that such foams have been identified as potential sources of PFASs in drinking water in other jurisdictions.
- Research should be conducted to further scientific understanding of human bioavailability and half-lives for BFRs and PFASs and to generate better data via which dermal exposure may be quantified. While some data exist for the dermal uptake of BFRs from furniture fabrics, such information is currently lacking for PFASs, and research to provide such data is recommended.
- Further biomonitoring of PFASs and BFRs in Ireland every 5 years is recommended to facilitate evaluation of the impact of measures taken to reduce human exposure to such chemicals. In addition to continued monitoring of human milk, which has proven effective in this study, it is recommended that measurements are made in blood and in urine. This will permit assessment of body burdens across a wider range of the population and facilitate monitoring of exposure to less persistent organic contaminants, which are most effectively biomonitored via their urinary metabolites.
- Given that the health effects of PFASs other than PFOS and PFOA are currently under review by EFSA and that this study found non-dietary exposure of children to PFBS to exceed that of the other PFASs targeted in this study, it is recommended that as well as continuing to measure PFOS and PFOA, future research should also monitor exposure of the Irish population via all relevant exposure pathways to other PFASs, such as PFBS, as well as PFHxS and PFNA, that were detected in human milk. As a first priority, detailed study of dietary exposure to PFOS, PFOA and other PFASs in Ireland is recommended. Furthermore, it is recommended that the findings of such a study of human exposure to PFASs be evaluated against the new EFSA opinion once adopted.

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Abbreviations

AFFF	Aqueous fire-fighting foam
BDE	Bromodiphenyl ether
BFR	Brominated flame retardant
CIMH	Coombe Infant and Maternity Hospital
DBDPE	Decabromodiphenyl ethane
DF	Detection frequency
DI	Daily intake
EFSA	European Food Safety Authority
EPA	Environmental Protection Agency
EPS	Expanded polystyrene
EtFOSA	Ethyl perfluorooctane sulfonamide
EtFOSE	Ethyl perfluorooctane sulfonamido ethanol
EU	European Union
FOSA	Perfluorooctane sulfonamide
FR	Flame retardant
FSAI	Food Safety Authority of Ireland
HBCDD	Hexabromocyclododecane
HBLV	Health-based limit value
LOQ	Limit of quantitation
MeFOSA	Methyl perfluorooctane sulfonamide
MeFOSE	Methyl perfluorooctane sulfonamidoethanol
PBDE	Polybrominated diphenyl ether
PFAS	Perfluoroalkyl substance
PFBS	Perfluorobutane sulfonate
PFHxS	Perfluorohexane sulfonate
PFNA	Perfluorononanoic acid
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonate
PK	Pharmacokinetic
POP	Persistent organic pollutant
POSF	Perfluorooctane sulfonyl fluoride
PUF	Polyurethane foam
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
RfD	Reference dose
TDI	Tolerable daily intake
TWI	Tolerable weekly intake
UHG	University Hospital Galway
UNEP	United Nations Environment Programme
WHO	World Health Organization
XPS	Extruded polystyrene

Appendix 1 List of Peer-reviewed Publications Emerging from the Research Described in this Report

Harrad, S., Wemken, N., Drage, D.S., Abdallah, M.A.E. and Coggins, M.A., 2019. Perfluoroalkyl substances in drinking water, indoor air and dust from Ireland: implications for human exposure. *Environmental Science and Technology* 53: 13449−13457.

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AN GHNÍOMHAIREACHT UM CHAOMHNÚ COMHSHAOIL
Tá an Ghníomhaireacht um Chaomhnú Comhshaoil (GCC) freagrach as an gcomhshaol a chaomhnú agus a fheabhsú mar shócmhainn luachmhar do mhuintir na hÉireann. Táimid tiomanta do dhaoine agus don chomhshaol a chosaint ó éifeachtaí díobhálacha na radaíochta agus an truaillithe.

Is féidir obair na Gníomhaireachta a roinnt ina trí phríomhréimse:

Rialú: *Déanaimid córais éifeachtacha rialaithe agus comhlíonta comhshaoil a chur i bhfeidhm chun torthaí maithe comhshaoil a sholáthar agus chun díriú orthu siúd nach gcloíonn leis na córais sin.*

Eolas: *Soláthraímid sonraí, faisnéis agus measúnú comhshaoil atá ar ardchaighdeán, spriocdhírithe agus tráthúil chun bonn eolais a chur faoin gcinnteoireacht ar gach leibhéal.*

Tacaíocht: *Bímid ag saothrú i gcomhar le grúpaí eile chun tacú le comhshaol atá glan, táirgiúil agus cosanta go maith, agus le hiompar a chuirfidh le comhshaol inbhuanaithe.*

Ár bhFreagrachtaí

Ceadúnú
Déanaimid na gníomhaíochtaí seo a leanas a rialú ionas nach ndéanann siad dochar do shláinte an phobail ná don chomhshaol:
- saoráidí dramhaíola *(m.sh. láithreáin líonta talún, loisceoirí, stáisiúin aistrithe dramhaíola)*;
- gníomhaíochtaí tionsclaíocha ar scála mór *(m.sh. déantúsaíocht cógaisíochta, déantúsaíocht stroighne, stáisiúin chumhachta)*;
- an diantalmhaíocht *(m.sh. muca, éanlaith)*;
- úsáid shrianta agus scaoileadh rialaithe Orgánach Géinmhodhnaithe *(OGM)*;
- foinsí radaíochta ianúcháin *(m.sh. trealamh x-gha agus radaiteiripe, foinsí tionsclaíocha)*;
- áiseanna móra stórála peitril;
- scardadh dramhuisce;
- gníomhaíochtaí dumpála ar farraige.

Forfheidhmiú Náisiúnta i leith Cúrsaí Comhshaoil
- Clár náisiúnta iniúchtaí agus cigireachtaí a dhéanamh gach bliain ar shaoráidí a bhfuil ceadúnas ón nGníomhaireacht acu.
- Maoirseacht a dhéanamh ar fhreagrachtaí cosanta comhshaoil na n-údarás áitiúil.
- Caighdeán an uisce óil, arna sholáthar ag soláthraithe uisce phoiblí, a mhaoirsiú.
- Obair le húdaráis áitiúla agus le gníomhaireachtaí eile chun dul i ngleic le coireanna comhshaoil trí chomhordú a dhéanamh ar líonra forfheidhmiúcháin náisiúnta, trí dhíriú ar chiontóirí, agus trí mhaoirsiú a dhéanamh ar leasúchán.
- Cur i bhfeidhm rialachán ar nós na Rialachán um Dhramhthrealamh Leictreach agus Leictreonach (DTLL), um Shrian ar Shubstaintí Guaiseacha agus na Rialachán um rialú ar shubstaintí a ídíonn an ciseal ózóin.
- An dlí a chur orthu siúd a bhriseann dlí an chomhshaoil agus a dhéanann dochar don chomhshaol.

Bainistíocht Uisce
- Monatóireacht agus tuairisciú a dhéanamh ar cháilíocht aibhneacha, lochanna, uiscí idirchriosacha agus cósta na hÉireann, agus screamhuiscí; leibhéil uisce agus sruthanna aibhneacha a thomhas.
- Comhordú náisiúnta agus maoirsiú a dhéanamh ar an gCreat-Treoir Uisce.
- Monatóireacht agus tuairisciú a dhéanamh ar Cháilíocht an Uisce Snámha.

Monatóireacht, Anailís agus Tuairisciú ar an gComhshaol
- Monatóireacht a dhéanamh ar cháilíocht an aeir agus Treoir an AE maidir le hAer Glan don Eoraip (CAFÉ) a chur chun feidhme.
- Tuairisciú neamhspleách le cabhrú le cinnteoireacht an rialtais náisiúnta agus na n-údarás áitiúil *(m.sh. tuairisciú tréimhsiúil ar staid Chomhshaol na hÉireann agus Tuarascálacha ar Tháscairí)*.

Rialú Astaíochtaí na nGás Ceaptha Teasa in Éirinn
- Fardail agus réamh-mheastacháin na hÉireann maidir le gáis cheaptha teasa a ullmhú.
- An Treoir maidir le Trádáil Astaíochtaí a chur chun feidhme i gcomhair breis agus 100 de na táirgeoirí dé-ocsaíde carbóin is mó in Éirinn.

Taighde agus Forbairt Comhshaoil
- Taighde comhshaoil a chistiú chun brúnna a shainaithint, bonn eolais a chur faoi bheartais, agus réitigh a sholáthar i réimsí na haeráide, an uisce agus na hinbhuanaitheachta.

Measúnacht Straitéiseach Timpeallachta
- Measúnacht a dhéanamh ar thionchar pleananna agus clár beartaithe ar an gcomhshaol in Éirinn *(m.sh. mórphleananna forbartha)*.

Cosaint Raideolaíoch
- Monatóireacht a dhéanamh ar leibhéil radaíochta, measúnacht a dhéanamh ar nochtadh mhuintir na hÉireann don radaíocht ianúcháin.
- Cabhrú le pleananna náisiúnta a fhorbairt le haghaidh éigeandálaí ag eascairt as taismí núicléacha.
- Monatóireacht a dhéanamh ar fhorbairtí thar lear a bhaineann le saoráidí núicléacha agus leis an tsábháilteacht raideolaíochta.
- Sainseirbhísí cosanta ar an radaíocht a sholáthar, nó maoirsiú a dhéanamh ar sholáthar na seirbhísí sin.

Treoir, Faisnéis Inrochtana agus Oideachas
- Comhairle agus treoir a chur ar fáil d'earnáil na tionsclaíochta agus don phobal maidir le hábhair a bhaineann le caomhnú an chomhshaoil agus leis an gcosaint raideolaíoch.
- Faisnéis thráthúil ar an gcomhshaol ar a bhfuil fáil éasca a chur ar fáil chun rannpháirtíocht an phobail a spreagadh sa chinnteoireacht i ndáil leis an gcomhshaol *(m.sh. Timpeall an Tí, léarscáileanna radóin)*.
- Comhairle a chur ar fáil don Rialtas maidir le hábhair a bhaineann leis an tsábháilteacht raideolaíoch agus le cúrsaí práinnfhreagartha.
- Plean Náisiúnta Bainistíochta Dramhaíola Guaisí a fhorbairt chun dramhaíl ghuaiseach a chosc agus a bhainistiú.

Múscailt Feasachta agus Athrú Iompraíochta
- Feasacht chomhshaoil níos fearr a ghiniúint agus dul i bhfeidhm ar athrú iompraíochta dearfach trí thacú le gnóthais, le pobail agus le teaghlaigh a bheith níos éifeachtúla ar acmhainní.
- Tástáil le haghaidh radóin a chur chun cinn i dtithe agus in ionaid oibre, agus gníomhartha leasúcháin a spreagadh nuair is gá.

Bainistíocht agus struchtúr na Gníomhaireachta um Chaomhnú Comhshaoil
Tá an ghníomhaíocht á bainistiú ag Bord lánaimseartha, ar a bhfuil Ard-Stiúrthóir agus cúigear Stiúrthóirí. Déantar an obair ar fud cúig cinn d'Oifigí:
- An Oifig um Inmharthanacht Comhshaoil
- An Oifig Forfheidhmithe i leith cúrsaí Comhshaoil
- An Oifig um Fianaise is Measúnú
- Oifig um Chosaint Radaíochta agus Monatóireachta Comhshaoil
- An Oifig Cumarsáide agus Seirbhísí Corparáideacha

Tá Coiste Comhairleach ag an nGníomhaireacht le cabhrú léi. Tá dáréag comhaltaí air agus tagann siad le chéile go rialta le plé a dhéanamh ar ábhair imní agus le comhairle a chur ar an mBord.

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Elucidating Levels and Pathways of Human Exposure in Ireland to Brominated Flame Retardants and Perfluoroalkyl Substances

Authors: Stuart Harrad, Daniel Drage, Mohamed Abdallah, Nina Wemken and Marie Coggins

Identifying Pressures

Brominated flame retardants (BFRs) and perfluoroalkyl substances (PFASs) have been used extensively in applications such as electrical goods, soft furnishings and building insulation foam. Given concerns about their environmental persistence, ability to bioaccumulate and adverse health effects, some BFRs and PFASs are listed under the Stockholm Convention on Persistent Organic Pollutants (POPs), an international treaty designed to eliminate POPs and to which Ireland is a party. Previous studies revealed low levels of BFRs and PFASs in Irish foodstuffs and human milk; however, no data existed for Ireland on concentrations of BFRs and PFASs in indoor air and dust, and of PFASs in drinking water. This project generated these data to facilitate the assessment of human exposure to these chemicals, allowing comparison with guidelines on human exposure developed by the European Food Safety Authority (EFSA), the revised European Union Drinking Water Directive, and limits on concentrations of PFASs in drinking water. The project also measured BFRs and PFASs in Irish human milk to facilitate assessment of the success of actions designed to reduce human exposure since the last measurements in 2011.

Informing Policy

Comparison of the concentrations of BFRs in human milk determined in this study and those determined in a 2011 Irish study suggests that restrictions on penta- and octabromodiphenyl ether products and hexabromocyclododecane have successfully reduced human exposure. Probably because of the more recent restriction on decabromodiphenyl ether, concentrations of this BFR in human milk show no significant decline. Moreover, although decabromodiphenyl ethane (DBDPE) was not detected in human milk in 2011, it was detected in this study. Along with elevated concentrations of DBDPE in indoor air and dust, this implies increased use of DBDPE to replace decabromodiphenyl ether. The Irish population's exposure to PFASs through non-dietary sources falls below the EFSA's provisional tolerable weekly intake (TWI). Concentrations of PFASs in Irish human milk do not indicate a health concern for nursing infants based on EFSA breastfeeding exposure scenarios. However, pharmacokinetic modelling of the level of adult exposure to PFASs required to support levels in human milk suggests that overall exposure for some individuals may approach or exceed EFSA's TWI. Detailed study of dietary exposure to PFASs is therefore recommended.

Developing Solutions

The declines in concentrations of some BFRs in human milk from Irish mothers since the previous equivalent study in 2011 suggest that past policies aimed at reducing the exposure of the Irish population to these BFRs have been successful. Such biomonitoring also reveals that measures taken to restrict the use of decabromodiphenyl ether have not yet reduced exposure in Ireland and also suggests increased exposure to DBDPE, a likely replacement for decabromodiphenyl ether. To facilitate ongoing evaluation of the impact of measures taken to reduce human exposure to such chemicals, biomonitoring of BFRs and PFASs in the Irish population is recommended every 5 years. In addition to continued monitoring of human milk, which in this study has proven an effective biomonitoring tool, measurements should also be made in blood and urine. This will permit assessment of levels of BFRs and PFASs across a wider range of the population and facilitate monitoring of human exposure to less persistent contaminants through their urinary metabolites.

EPA Research: McCumiskey House, Richiew, Clonskeagh, Dublin 14.

Phone: 01 268 0100
Twitter: @EPAResearchNews
Email: research@epa.ie

www.epa.ie

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