Environment International 108 (2017) 51–62

Contents lists available at ScienceDirect

Environment International

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

Review article

Global distribution of perfluorochemicals (PFCs) in potential human exposure source–A review

Jun-Meng Jian[a,b], Ying Guo[a], Lixi Zeng[a], Liu Liang-Ying[a], Xingwen Lu[c], Fei Wang[a,b,*], Eddy Y. Zeng[a]

[a] School of Environment, Guangzhou Key Laboratory of Environmental Exposure and Health, and Guangdong Key Laboratory of Environmental Pollution and Health, Jinan University, Guangzhou 510632, China
[b] State Key Laboratory of Organic Geochemistry, Guangzhou Institute of Geochemistry, Chinese Academy of Sciences, Guangzhou 510640, China
[c] School of Environmental Science and Engineering, Guangdong University of Technology, Guangzhou 510006, China

ARTICLE INFO

Keywords:
Perfluorochemical
Indoor air
Food
Drinking water
Exposure

ABSTRACT

Human exposure to perfluorochemicals (PFCs) has attracted mounting attention due to their potential harmful effects. Breathing, dietary intake, and drinking are believed to be the main routes for PFC entering into human body. Thus, we profiled PFC compositions and concentrations in indoor air and dust, food, and drinking water with detailed analysis of literature data published after 2010. Concentrations of PFCs in air and dust samples collected from home, office, and vehicle were outlined. The results showed that neutral PFCs (e.g., fluorotelomer alcohols (FTOHs) and perfluorooctane sulfonamide ethanols (FOSEs)) should be given attention in addition to PFOS and PFOA. We summarized PFC concentrations in various food items, including vegetables, dairy products, beverages, eggs, meat products, fish, and shellfish. We showed that humans are subject to the dietary PFC exposure mostly through fish and shellfish consumption. Concentrations of PFCs in different drinking water samples collected from various countries were analyzed. Well water and tap water contained relatively higher PFC concentrations than other types of drinking water. Furthermore, PFC contamination in drinking water was influenced by the techniques for drinking water treatment and bottle-originating pollution.

1. Introduction

Perfluorochemicals (PFCs) are a family of man-made compounds with strong C—F bonds. Due to their unique properties, they are commonly used in consumer products and industrial processes, such as protective coatings of carpets and furniture, paper and cloth coatings, Polytetrafluoroethylene products, and fire-fighting foams (Ahrens and Bundschuh 2014; Clara et al. 2008; Paul et al. 2009; Route et al. 2014). They have been widely detected in drinking water (Hoffman et al. 2011; Post et al. 2009; Thompson et al. 2011), air (Fromme et al. 2015; Goosey and Harrad 2012; Karaskova et al. 2016; Piekarz et al. 2007), and human blood (Bjerregaard-Olesen et al. 2016; Ehresman et al. 2007; Karrman et al. 2007; Wu et al. 2017; Yeung et al. 2006), urine (Genuis et al. 2013; Jurado-Sanchez et al. 2014), breast milk (Barbarossa et al. 2013; So et al. 2006; Tao et al. 2008a; Tao et al. 2008b; Thomsen et al. 2010), nails(Li et al. 2012; Li et al. 2013; Liu et al. 2011) and hairs (Alves et al. 2015; Krol et al. 2013; Martin et al. 2016; Rodriguez-Gomez et al. 2017). Many studies also have reported that PFCs may be associated with human diseases, such as urine acid, thyroid diseases, peroxisome proliferation, asthmatic, liver tumor, hyperuricemia, pediatric atopy, chronic kidney disease, behavioral disorders, and immune toxicity (Bloom et al. 2010; Dong et al. 2013; Gump et al. 2011; Lopez-Espinosa et al. 2011; Wang et al. 2011a). As a result, the production and regulation of PFCs have attracted public attention. In 2000, 3M first announced a global phase-out of its products containing C6, C8, and C10 PFCs and replaced them with shorter C4 PFC (e.g., perfluorobutane sulfonic acid or PFBS) products. Eight major PFC manufacturers joined the United States Environmental Protection Agency (USEPA) 2010/15 PFOA Stewardship Program in 2006 to work towards the elimination of long-chain perfluoroalkyl carboxylic acids (PFCAs) and their potential precursors by 2015. In addition, perfluorooctane sulfonate (PFOS) and perfluorooctane sulfonyl fluoride (POSF) related compounds were listed under Annex B of the Stockholm Convention in 2009 (Wang et al. 2009). C11–C14 PFCAs, perfluorooctanoic acid (PFOA) and ammonium perfluorooctanoate (APFO) were recognized as vPvB chemicals (very persistent and very bioaccumulative), and included in the Candidate List of Substances of Very High Concern under the European chemicals regulation in 2012–2013.

---

[*] Corresponding author at: School of Environment, Guangzhou Key Laboratory of Environmental Exposure and Health, and Guangdong Key Laboratory of Environmental Pollution and Health, Jinan University, Guangzhou 510632, China.
E-mail address: wf1984@jnu.edu.cn (F. Wang).

http://dx.doi.org/10.1016/j.envint.2017.07.024
Received 25 May 2017; Received in revised form 20 July 2017; Accepted 29 July 2017

The different regulations of PFCs have led to varieties in production and time or region-dependent environmental distributions of PFCs.

Since PFCs are extremely persistent and associated with some human diseases (Costa et al. 2009; Dallaire et al. 2009; Gallo et al. 2012; Sakr et al. 2007; Stein et al. 2009), the biomonitoring of human exposure to PFCs has become increasingly important. It has been suggested that food sources, drinking water, and airborne sources are main PFC exposure routes for humans (D'Eon and Mabury 2011; D'Hollander et al. 2010a; Enault et al. 2015; Harada et al. 2005; Tittlemier et al. 2007), although the contribution from each source remains unclear. Thus, it is critical to profile PFC concentrations in these potential exposure sources from time to time, which will be useful for evaluation of health effects induced by PFCs. Although numerous studies have been published on the monitoring and exposure of PFCs (Butenhoff et al. 2006; De Felip et al. 2015; Hoelzer et al. 2008; Landsteiner et al. 2014), only a limited number of reviews have been done to outline these research data, especially within the recent five years. Earlier reviews included Houde et al. (2006) and Lau et al. (2007), which summarized the biological monitoring of PFCs in wildlife and humans, discussed possible sources, and identified knowledge gaps. Trudel et al. (2008) assessed and modeled consumer exposure to PFOS and PFOA from a variety of environmental and product-related sources. Fromme et al. (2009) published a review on PFC monitoring data in environmental media relevant to human exposure. They outlined PFC concentrations in indoor and ambient air, house dust, drinking water, and food, as well as human biomonitoring data in blood, breast milk, and human tissues, and proposed that the consumption of highly contaminated fish products may substantially increase PFC body burdens. Kantiani et al. (2010) reviewed PFCs and other emerging contaminants in food, but focused more on methods used for detecting and quantifying PFCs. The review by D'Hollander et al. (2010a) suggested that PFC data in human diet and indoor dust were relatively scarce compared to those published in fish and drinking water. Domingo (2012) summarized PFC concentrations in foodstuffs and human dietary exposure to these compounds, as well as human biomarkers from different countries. However, PFC concentrations in different foodstuffs were only added in the discussion without detailed analysis. Wang et al. (2015) reviewed the sources, multimedia distribution and health risks of PFCs in China and suggested that terrestrial food (meat) contributed 93.2% of PFOA to human exposure, while seafood contributed 78.9% of PFOS. Several articles reviewed human exposure to PFOS, especially PFOS isomers, and provided definitive insights into the role of "precursor exposure" (Chen et al. 2009; Houde et al. 2006; Kovarova and Svobodova 2008; Lau et al. 2007; Lindstrom et al. 2011; Mercier et al. 2011; Miralles-Marco and Harrad 2015). All these reviews are valuable for the establishment of biomonitoring and human exposure profiles for PFCs. As mentioned previously, the regulations on PFC production may lead to spatial and temporal changes of PFC concentrations and compositions in environmental media. Furthermore, journal articles related to human exposure to PFCs and health effects have increasingly emerged in recent six years. Thus, a systematic analysis of PFC concentrations in food, drinking water, and indoor air and dust is critically needed.

In this study, we reviewed recent studies on PFCs in potential sources (e.g. air, food and drinking water) related to human exposure. We outlined the occurrences of different PFC congeners/isomers in indoor air and dust, foodstuffs (e.g., vegetables, dairy products, beverages, eggs, meat and meat products, fish, and shellfish), and drinking water. Based on these data, we aimed to profile spatial distributions of PFCs in our surrounding environment and establish a good baseline for human exposure risk assessment.

2. Environmental monitoring

2.1. Indoor air

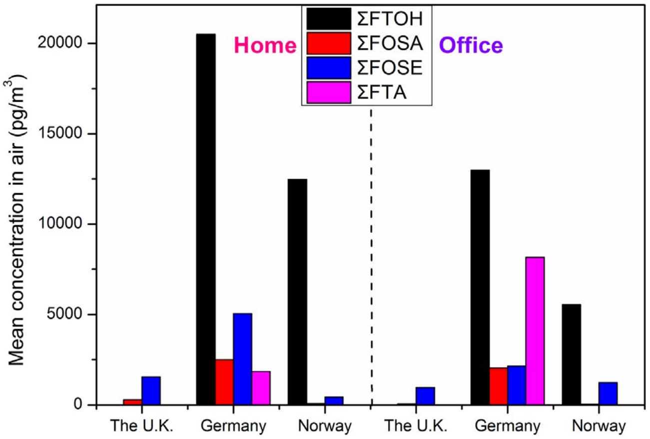

Fig. 1. The total mean concentrations of neutral PFCs in air collected from the U.K., Germany, and Norway.

Concentrations of PFCs in indoor air in different countries are summarized in the Supporting Information (Table S1). According to the sampling locations, data are generally divided into two groups: homes and offices. The results reveal that neutral PFCs, mainly FTOHs, FOSA, and FOSEs, were the dominant compounds in indoor air due to their low water solubility and high vapor pressure. We calculated total mean concentrations of ΣFTOH, ΣFOSA, ΣFOSE, and ΣFTA in indoor air in the U.K., Germany, and Norway (Fig. 1). It is clear that FOSE compounds were the most frequently detected PFCs in indoor air. FTOHs were frequently used for impregnation treatment of furniture and floor and as intermediates in manufacturing various household consumer products (e.g., paints, carpet, and cleaning agents). FTOHs were also found with much higher concentrations than other neutral PFCs, although they were not analyzed in the U.K. indoor air (Fig. 1). For example, data from Vestergren et al. (2015) indicated that concentrations of FTOHs were 2–3 orders of magnitude higher than those of PFCAs. Fluorotelomer acrylates (FTAs) were also an important group of PFCs with high concentrations in indoor air, although only one article analyzed their concentrations (Vestergren et al. 2015). Overall, concentrations of FTOHs, FOSAs, and FOSEs in indoor air were substantially lower in offices than in homes.

Haug et al. (2011) reported that the median concentrations of 8:2 FTOHs, 10:2 FTOH, and 6:2 FTOH in indoor air ($n = 40$) from houses in Oslo (Norway) were 5200, 2800, and 930 pg/m^3, respectively (Table S1). By contrast, 4:2 FTOH was only detected in 7 samples with a lower median concentration (4.8 pg/m^3), while 12:2 FTOH was not detected at all (Haug et al. 2011). Huber et al. (2011) reported greater concentrations of 8:2 FTOH and 10:2 FTOH than other substances in indoor air ($n = 6$) from Tromsø (another city in Norway) and the concentrations were generally higher than those from Oslo. For example, the median concentration of 8:2 FTOH in Tromsø was 10,000 pg/m^3, which was approximately twice higher than the Olso data (Table S1). However, a much lower median concentration (42 pg/m^3) of 6:2 FTOH was observed in Tromsø (Haug et al. 2011; Huber et al. 2011). Even greater concentrations of FTOHs and FOSA/Es in indoor air were reported in Hamburg (German) (Langer et al. 2010). For example, the concentrations of MeFOSA, EtFOSA, MeFOSE, and EtFOSE in Hamburg homes were 1100, 1400, 2900, and 2150 pg/m^3, respectively, much higher than the concentrations (0.5–760 pg/m^3) in Olso and Birmingham (the U.K.) homes (Table S1) (Goosey and Harrad 2012; Haug et al. 2011; Huber et al. 2011; Langer et al. 2010). Particularly, three types of FTAs (6:2 FTA, 8:2FTA, and 10:2FTA) were detected in samples from Hamburg homes. FTAs were generally used in the production of FTOH-based polymers. Except for neutral PFCs, several PFSAs and PFCAs, including PFOS, PFHxS, and PFOA, were also detected in 20 air samples from Birmingham (the U.K.) homes, with median concentrations of 11, 23, and 24 pg/m^3, respectively (Table S1) (Goosey and Harrad 2012).

In addition to indoor air in homes, office air was also dominated by FTOHs and FOSA/Es (Fig. 1), among which 8:2 FTOH appeared to be the most abundant PFC (Table S1). Goosey and Harrad (2012) reported that the average concentration of PFHxS in offices was 2–3 times greater than that in homes from Birmingham (the U.K.), while FTAs in Hamburg exhibited higher concentrations in offices than in homes (Langer et al. 2010). The reason for high concentrations of FTAs in Humburg offices may be attributed to paint additives, as air samples were collected from only two offices and one of them was freshly painted prior to sampling.

2.2. Indoor dust

Detailed information on sampling background and PFC concentrations in dust from different countries are summarized in Table S2. Overall, PFC concentrations in indoor dust were generally at the levels of ng/g. Indoor dust can be grouped into three types: household, office, and vehicle dust. Most available studies have focused on household dust.

PFOS, PFOA, PFHxS, FOSAs, and FOSEs exhibited elevated concentrations and detection frequencies in household dust among all PFCs (Table S2). Thus, we focused on these compounds and analyzed their spatial distribution patterns (Fig. 2). Relatively higher concentrations of these five PFCs were detected in indoor dust collected from the U.K. (Birmingham), Germany (Augsberg and Michelstadt), Australia (Brisbane, Newcastle, and Sydney), France (Annecy), Canada (Toronto), and the U.S. (Boulder), compared to other studied countries (Fig. 2). This may indicate that PFC concentrations in indoor dust are associated with the economic level of a country, as PFCs are widely used in household products. Furthermore, different cities within the same country (e.g., Bavaria and Augsberg in Germany and Boston and Boulder in the U.S.) also differed greatly in PFC concentrations, indicating that PFCs were not evenly distributed even in the same country. PFOS and PFOA were the dominant and most frequently detected PFCs in home dust. D'Hollander et al. (2010b) also frequently detected PFHxA at relatively high levels in addition to PFOS and PFOA. Haug et al. (2011) explored for the first time the relationship between neutral PFCs and ionic PFCs in house dust sampled from the same residence in Norway. Ellis et al. (2004) reported the degradation of FTOHs to PFCAs in the atmosphere, while Tomy et al. (2004) observed biodegradation of EtFOSA to PFOS and predicted the transformation of FOSA/FOSEs to PFSAs in the atmosphere. Such findings may indicate a positive association between FTOHs (or FOSA/Es) in indoor air and PFCAs (or PFSAs) in indoor dust. Significant associations between FOSA/Es in indoor air and PFOS in house dust (Haug et al. 2011) and between 8:2/10:2 FTOH and PFOA in dust (Xu et al. 2013) may strongly support this hypothesis. Haug et al. (2011) and Jogsten et al. (2012) detected FTUCAs and FTOHs in the same home dust. Since FTUCAs were intermediate degradation products of FTOHs (Butt et al. 2010), this is also a proof of FTOH degradation in dust. Jogsten et al. (2012) reported that concentrations of ionic PFCs in home dust from Catalonia (Spain) were comparable to or lower than those in Belgium (D'Hollander et al. 2010b) and Norway (Haug et al. 2011; Huber et al. 2011), and evidently lower than those in Germany (Xu et al. 2013), Central America (Knobeloch et al. 2012), and Sweden (Bjorklund et al. 2009). The reason was that Catalan homes generally did not use carpets, while it has been demonstrated that the percentage of home carpeting was positively related to indoor PFC contamination (Gewurtz et al. 2009; Haug et al. 2011; Kubwabo et al. 2005).

Four studies have examined PFCs in office dust from Belgium ($n = 10$) (D'Hollander et al. 2010b), Boston (U.S., $n = 31$) (Fraser et al. 2013), Sweden ($n = 10$) (Bjorklund et al. 2009), and Norway ($n = 1$) (Huber et al. 2011). In all four studies except for Fraser et al. (2013), PFOA and PFOS were the dominant chemicals and were detected with the highest frequencies in office dust (Bjorklund et al. 2009; D'Hollander et al. 2010b; Huber et al. 2011). In addition, positive correlations were determined between PFOS and PFOA (Bjorklund et al. 2009; D'Hollander et al. 2010b; Fraser et al. 2013), between PFOS and PFHxS (D'Hollander et al. 2010b), and between 8:2 FTOH and 10:2 FTOH (Huber et al. 2011). The significant associations between these PFCs may indicate a common source or the same precursors. The median concentrations of PFOA (2.9 ng/g) and PFOS (2.2 ng/g) in Belgian offices (Fig. S1) were at least one order of magnitude lower than those in Boston, Sweden, and Norway (D'Hollander et al. 2010b). Fraser et al. (2013) investigated PFCs in North American office dust and 8:2 FTOH was detected with the highest geometric mean concentration (309 ng/g) followed by 10:2 FTOH (210 ng/g) (Fig. S1). The high concentrations of FTOHs in indoor air may be a potential source of carboxylate PFCs to indoor dust, as FTOHs are their most important precursors (Fromme et al. 2015; Haug et al. 2011; Huber et al. 2011; Langer et al. 2010). FTOHs in office dust were proposed as a significant indicator of PFOA in workers' serum (Fraser et al. 2012). However, Fraser et al. (2013) demonstrated that FTOHs were no longer a significant predictor of PFOA in workers' serum when assessed individually. Such findings may suggest that dust is a minor pathway for human exposure to PFCs. Particularly, FTSs were reported in office dust for the first time, where 8:2 FTS and 6:2 FTS were detected at concentrations of 177 and 114 ng/g, respectively (Fig. S1) (Huber et al.

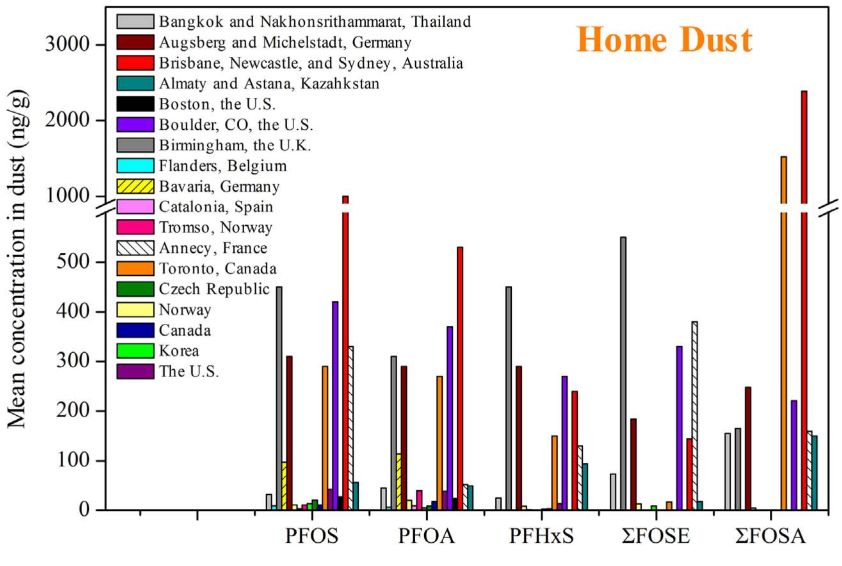

Fig. 2. The mean concentrations of typical PFCs in house dust from different locations.

2011).

Only two articles reported PFCs in vehicle dust. Bjorklund et al. (2009) detected PFOS (geometric mean: 15.8 ng/g) and PFOA (11.4 ng/g) in car dust ($n = 5$) from Sweden, where the concentrations were comparably low with those (12 and 33 ng/g, respectively) in Boston (Fraser et al. 2013). The source of PFOS and PFOA in vehicle dust was proposed to be the accessories and interiors in cars, as these chemicals are widely used in surface treatment for protection. PFOS and PFOA were detected with a significant correlation ($r = 0.90$) in vehicle dust from Boston, whereas no correlation was found in car dust from Sweden, probably due to a small sample size (Table S2). The strong correlation between PFOS and PFOA in vehicle dust may suggest the same source of these compounds. Fraser et al. (2013) found that the length of time period people spent in vehicle was negatively associated with PFOA and PFOS concentrations in dust, while the age of vehicle was positively correlated with PFOA and PFOS in vehicle dust.

Overall, the variability of PFC concentrations in different countries may be attributed to diversified lifestyles in different cultures (e.g., using carpets or not), building and decorating characteristics, residents' habits of buying clothes or consumer products, sample quantity and sampling sites, sample collection and analysis procedures, and the economic status of a region. Although various PFCs in dust were observed in offices, homes, and vehicles, indoor dust ingestion may not be a significant pathway for human exposure when compared with other pathways (i.e., food and drinking water).

2.3. Vegetables

Vegetables are rich in vitamins, minerals, and crude fibers essential for the digestive system, thus irreplaceable in human diet. Because PFCs have been found in soil, irrigation water, and air, vegetables may assimilate/absorb PFCs from these media. Thus, vegetables are potential PFC sources to humans. Noorlander et al. (2011) reported that vegetables/fruits were important food contributors to PFOA intake (19%) following drinking water. However, they showed that PFOS was not detected in vegetables and fruits. Detected PFCs varied in vegetables and the concentrations were not high. Four out of 14 PFCs were detected in vegetable samples from the Netherlands (Noorlander et al. 2011), while 7 out of 18 and 9 out of 16 PFCs were found in samples of Spain and Korea, respectively (Domingo et al. 2012b; Heo et al. 2014). As shown in Table 1, PFHxA, PFHpA, PFOA and PFOS were the most frequently detected compounds in vegetables (Domingo et al. 2012b; Haug et al. 2010; Vestergren et al. 2012). It was reported that the sorption of PFCs to organic matter was positively correlated with the chain length of PFCs, stronger for sulfonates than carboxylates (Higgins and Luthy 2006). Thus, PFCs of short chain lengths with carboxylic groups (PFHxA, PFHpA, and PFOA) are easily assimilated by vegetable roots, leading to widespread occurrence in vegetables (Table 1). Although PFOS was usually the dominant compound in the environment, its concentrations in vegetables were generally lower than PFOA. This also indicates that PFOS may be strongly associated with soil instead of being assimilated by vegetables.

It was reported that short-chain PFCAs were prone to retain in plant leaves rather than roots, while long-chain PFCAs were likely to accumulate in soil-contacting roots (Blaine et al. 2013; Felizeter et al. 2012; Felizeter et al. 2014; Lechner and Knapp 2011; Stahl et al. 2009; Yoo et al. 2011; Zhang et al. 2011a). Heo et al. (2014) reported that high concentrations of short-chain PFCAs (e.g., PFBA, PFPeA, and PFHxA) were frequently detected in vegetables and fruits, which is in accordance with higher uptake and bioaccumulation factors of short-chain PFCs in plant leaves. In addition, potatoes and potato fast food were related to human PFAS and PFOA serum concentrations (Fraser et al. 2012; Ji et al. 2012). Cornelis et al. (2012) reported the contributions of potatoes to dietary exposure of children and adults to PFOS were 48% and 28%, respectively. Herzke et al. (2013) also showed that potatoes contributed to dietary exposure to PFCs more than any other vegetables. However, the PFC concentrations in potatoes were generally low (Eriksson et al. 2013; Haug et al. 2010; Vestergren et al. 2012). For example, no PFAS was found in any potato sample from Faroe Islands (Eriksson et al. 2013), and only one PFC (PFBS) was detected in potatoes from the U.K. (Clarke et al. 2010). These findings may indicate that potato itself is not the significant source; rather, the food packaging materials of potato products (e.g., chips) may be an important source (Begley et al. 2008; Domingo et al. 2012b; Fromme et al. 2009; Lorber and Egeghy 2011; Trier et al. 2011). Herzke et al. (2013) detected a variety of PFCs in vegetables from four European countries (Norway, Czech Republic, Belgium, and Italy). Broad contamination was found in vegetables from Norway, including brassica, beans, leafs, roots, and stem. This may be because vegetables in Norway are mainly imported from other countries, which may increase the likelihood for vegetables to contact with various contaminant sources, including food packages.

2.4. Dairy products and beverages

Dairy products and beverages are also potential PFC sources to humans, especially in western countries due to high consumption of these products. However, only selected types of PFCs have been detected in dairy products and beverages, generally with low concentrations and low detection frequencies. The distribution profiles of PFCs in milk, yogurt, butter/cheese, and beverage indicate that milk contained more PFCs than yogurt, butter/cheese, and beverage (Fig. 3). This was contrary to the traditional view that PFC contamination would increase along with industrial processes. It indicates that the loading of different regions and different packaging materials may be the key factors affecting PFC concentrations in milk products and beverages.

Food packaging has been reported as a possible PFC source to foodstuffs (Begley et al. 2008; Fromme et al. 2009). Wang et al. (2010a) found that significant differences in PFHpA, PFNA, and total PFC concentrations were observed in three types of milk packaging. Eriksson et al. (2013) measured PFCs in raw milk collected from manufacturing procedures and packaged milk and found that all samples contained PFCs in the approximately same concentration range. The variation between dairy categories was smaller than that between samples. Wang et al. (2010a) analyzed milk, milk powder, and yogurt samples from three cities in China (Beijing, Tianjin, and Wuhan). The concentrations of PFNA and PFHpA (67 and 54 pg/g, respectively) were higher than those of other substances and had the highest contributions (37% and 29%, respectively) to the total PFC contamination in milk samples (Table S3). The proportion of PFOA was higher in milk powder (46 pg/g) than in milk (26 pg/g) and yogurt (32 pg/g). By contrast, PFNA, PFBA, PFUnDA, and PFDoDA were rarely detected in milk and dairy products. Eriksson et al. (2013) found abundant PFUnDA in milk samples from Faroe Islands, but PFDoDA was detected with low concentrations and frequencies. As PFUnDA was reported to be a degradation end-product of volatile fluorotelomer alcohols (FTOHs) (Ellis et al. 2004), this may indicate that long-range atmospheric transfer is an important pathway for the distribution of long-chain PFCs (Eriksson et al. 2013). Milk and dairy products from Catalonia (Spain) had low PFUnDA concentrations (46 and 22 pg/g), but elevated concentrations of PFOA (390 and 190 pg/g) and PFHpA (240 and 170 pg/g) (Table S3) (Domingo et al. 2012b). PFBA and PFOS were reported at the highest concentrations (43 and 10 pg/g) in milk from Netherlands, while PFOS, PFHxA, PFOA, and PFHxS were most abundant in butter samples (33, 20, 16, and16 pg/g) (Noorlander et al. 2011). Small amounts of PFBA, PFOA, PFNA, PFDA, PFOS, PFNA, and PFDA were reported in milk and yogurt samples, with concentrations lower than those in cheese (Still et al. 2013). PFOA, PFOS and PFDA were also found at low concentrations in milk and cheese samples collected from a grocery store in Norway (Haug et al. 2010).

Data on PFC contamination in beverages have remained limited. Vestergren et al. (2012) determined PFC concentrations in lemonade

Table 1
Mean concentrations (pg/g ww) of PFCs in vegetables.

Sampling	Region	PFHxA	PFHpA	PFHxS	PFOA	PFNA	PFOS	PFDA	PFUnDA	PFDS	PFDoDA	PFDcA	PFDOA	Year	Reference
Vegetables	Catalonia, Spain	< 47	360	4.5	370	< 370	100	< 37	140	< 2.2	< 41	-	-	2011	Domingo et al. (2012b)
Vegetables	Sweden	5.2	2.2	1.2	35	nd	4.6	3.1	nd	-	1.6	-	-	1999	Vestergren et al. (2012)
Vegetables	Sweden	3.0	1.7	1.0	52	nd	24.0	nd	nd	-	nd	-	-	2005	Vestergren et al. (2012)
Vegetables	Sweden	3.2	1.8	1.2	22	nd	4.1	2.5	nd	-	nd	-	-	2010	Vestergren et al. (2012)
Vegetables and fruits	Busan, Korea	39	2	-	1	nd	nd	2	9	7	2	-	-	2011–2012	Heo et al. (2014)
Vegetables	Norway	9.9	-	nd	19.9	2.26	nd	-	-	-	-	nd	nd	2011	Herzke et al. (2013)
Lettuce	Oslo, Norway	0.98	-	< 0.06	1.8	< 1.0	0.17	-	-	-	-	-	nd	2008–2009	Haug et al. (2010)
Carrots	Oslo, Norway	< 1.3	-	< 0.11	2.0	< 2.1	0.67	-	-	-	-	-	nd	2008–2009	Haug et al. (2010)
Vegetables and fruits	Netherlands	< 4	-	< 12	5	1	< 47	-	-	-	-	-	nd	2009	Noorlander et al. (2011)
Vegetables	Belgium	2.89	-	0.32	10.3	nd	3.24	-	-	-	-	nd	nd	2011	Herzke et al. (2013)
Vegetables	Belgium	-	-	-	650	-	600	-	-	-	-	-	-		Cornelis et al. (2012)
Vegetables	Czech Republic	3.41	-	nd	1.90	2.08	0.66	-	-	-	-	0.27	nd	2011	Herzke et al. (2013)
Vegetables	Italy	3.36	-	nd	25.0	nd	5.66	-	-	-	-	0.64	2.68	2011	Herzke et al. (2013)
Vegetables	The U.K.	-	-	-	< 1000	-	< 1000	-	-	-	-	-	-		Clarke et al. (2010)
Vegetables	Tianjin, China	-	-	-	4.6–36.8	-	2180-7260	-	-	-	-	-	-	2014	Shan et al. (2016)
Fruit and vegetables	Siena, Italy	-	-	-	< 500		< 500	-	-	-	-	-	-		Guerranti et al. (2013)
Lettuce	Siena, Italy	-	-	-	< 500		< 500	-	-	-	-	-	-		Guerranti et al. (2013)
Carrot	Siena, Italy	-	-	-	< 500		< 500	-	-	-	-	-	-		Guerranti et al. (2013)
Potatoes	Sandi, Faroe Islands	nq	-	-	< 140	< 1.2	< 0.5	< 0.9	nq	nd	nq	-	-	2012	Eriksson et al. (2013)
Potatoes	Kollafirði, Faroe Islands	nq	-	-	nq	nq	< 0.4	nq	nq	nd	nq	-	-	2012	Eriksson et al. (2013)
Potatoes	Sweden	nd	nd	nd	4.9	nd	1.9	1.7	nd	-	nd	-	-	1999	Vestergren et al. (2012)
Potatoes	Sweden	2.0	2.0	nd	12	nd	5.8	nd	nd	-	nd	-	-	2005	Vestergren et al. (2012)
Potatoes	Sweden	2.6	1.5	nd	57	nd	6.9	2.6	nd	-	nd	-	-	2010	Vestergren et al. (2012)
Potatoes	Oslo, Norway	3.1	-	< 0.22	5.3	< 4.1	1.0	-	-	-	-	-	nd	2008–2009	Haug et al. (2010)
Potatoes	Belgium	-	-	-	670	-	6180	-	-	-	-	-	-		Cornelis et al. (2012)

$^{-}$ Not analyzed; nd not detected; ^{nq} not quantified.

PFBA: below the limit of detection of 140 pg/g ww in vegetables collected in Catalonia, Spain (Domingo et al. 2012b); 30 pg/g ww in vegetables and fruits collected in Busan, Korea (Heo et al. 2014); not analyzed in other countries.

PFPeA: below the limit of detection of 19 pg/g ww in vegetables collected in Catalonia, Spain (Domingo et al. 2012b); 19 pg/g ww in vegetables and fruits collected in Busan, Korea (Heo et al. 2014); not analyzed in other countries.

PFBS: 13 pg/g ww in in vegetables collected in Catalonia, Spain (Domingo et al. 2012b); not detected in vegetables and fruits collected in Busan, Korea (Heo et al. 2014); below the limit of detection of 0.1 pg/g ww in potatoes collected in Faroe Islands (Eriksson et al. 2013) not analyzed in other countries.

PFHpS: not detected in vegetables and fruits collected in Busan, Korea (Heo et al. 2014); not analyzed in other countries.

THPFOS: 1100 pg/g ww in vegetables collected in Catalonia, Spain (Domingo et al. 2012b); not analyzed in other countries.

PFTrDA and PFTeDA: below the limit of detection of 60 pg/g ww in vegetables collected in Catalonia, Spain (Domingo et al. 2012b); not detected in vegetables and potatoes collected in Sweden (Vestergren et al. 2012) and vegetables and fruits collected in Busan, Korea (Heo et al. 2014); not analyzed in other countries.

PFUnA: below the limit of detection of 2 pg/g ww in vegetables and fruits collected from Netherlands (Noorlander et al. 2011); not detected in vegetables collected from Norway and Belgium, potatoes collected in Czech Republic (Herzke et al. 2013); not analyzed in other countries.

PFHxDA and PFOcDA: below the limit of detection of 400 pg/g ww in vegetables collected in Catalonia, Spain (Domingo et al. 2012b); not analyzed in other countries.

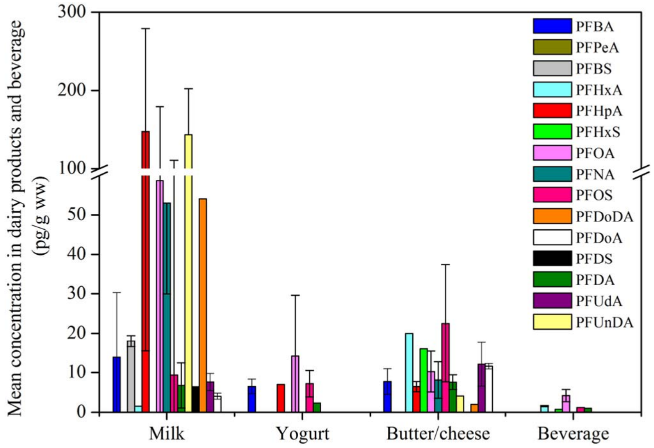

Fig. 3. The mean concentrations of PFCs in dairy products and beverages.

collected in different years (1999, 2005, and 2010). The results showed that PFHxA and PFOA were the main PFCs observed in all samples, and their concentrations ranged from 1.4 to 6.0 pg/g (Table S3) (Vestergren et al. 2012). Heo et al. (2014) reported that beverages were dominated by short-chain PFCs, such as PFBA and PFPeA. Compared with long-chain PFCs, short-chain PFCs were more likely to be detected in foodstuffs with high moisture contents (Felizeter et al. 2014; Yoo et al. 2011). This may be due to higher solubility of short-chain PFCs in foodstuffs with high moisture contents or their higher plant uptake potential from soil and lower sorption abilities in roots when compared with long-chain PFCs.

2.5. Eggs, meat and meat products

Only a few studies have published PFC data in egg samples. Vestergren et al. (2012) measured PFCs concentrations in diverse food basket samples collected in 1999, 2005, and 2010 from Sweden. Eggs and meat had relatively larger inter-year variations for some compounds. For example, several PFCAs (PFOS, PFNA, PFDA, and PFUnDA) and PFASs (PFHxS and PFOS) were determined at higher levels in egg samples collected in 1999 than in 2005 and 2010. Concentration of PFOS in egg samples from 1999 (1280 pg/g) was 30 to 100 times greater than that from 2005 (13 pg/g) and 2010 (39 pg/g) (Table 2). A similar trend was also reported by Gebbink et al. (2015) that PFOS concentrations in Norwegian egg samples decreased from 1999 to 2010. The temporal pattern should be attributed to the limitation of PFOS use and sales in the US and European countries (e.g., the phase-out of PFOS by 3M in 2002). However, some other countries (e.g., China) have not ceased the production and use of PFC-containing products, although the production volume has been declining (Li et al. 2015; Wang et al. 2014). Zhang et al. (2010) reported that PFOS, PFOA, PFDA, PFUnDA, and PFDS occurred in tens to hundreds of pg/g levels in eggs from China. The concentrations were generally greater than the contemporary data from other countries and suggested that the production of some PFC products has been continuing in China.

In general, PFOA and PFOS have been reported to be the dominant PFCs in meat and meat products, although some other common PFCs were also found (Haug et al. 2010; Noorlander et al. 2011; Vestergren et al. 2012; Zhang et al. 2010). However, the decrease of PFOS concentrations (190 pg/g to 25 pg/g) in meat samples was also observed from 1999 to 2010 (Table 2) (Vestergren et al. 2012). Similar to the egg study, PFOA, PFNA, PFDA, PFUnDA, PFHxS, and PFOS were detected in meat and meat products in Sweden. Particularly, small amounts of PFHpA and PFDoDA were found in meat samples (Vestergren et al. 2012). Domingo et al. (2012b) reported that PFHpA had the highest concentration (280 pg/g) among PFCs in meat samples from Catalonia, Spain (Table 2). In meat and meat products from Korea (Heo et al. 2014), PFHxA had a higher detection frequency (51%) than that of PFOS (13%). It also had the highest concentration (515 pg/g), followed by PFOS (353 pg/g) and PFDoDA (325 pg/g) (Table 2). This was different from some other studies, where PFOS was usually found with high concentrations and detection frequencies (Vestergren et al. 2012; Zhang et al. 2010). Some PFCs (e.g., PFHpA and PFHxA) with uncommonly high concentrations in meat samples indicated different regions may have different types of PFC-containing products in use. Zhang et al. (2010) collected 20 meat samples (pork, beef, chicken, and goat meat) from 17 Chinese cities, in which PFOA concentrations were generally > 1 ng/g (up to 12.5 ng/g) and much higher than those reported in other countries mentioned above (Table 2). However, concentrations of PFOS were much lower than those of PFOA in the same Chinese study, similar to the pattern reported by other studies.

2.6. Fish and shellfish

Fish and shellfish generally exhibited the highest PFC concentrations and detection frequencies among all types of foodstuffs (Domingo et al. 2012a; Domingo et al. 2012b; Guerranti et al. 2013; Haug et al. 2010; Heo et al. 2014). A number of studies investigated PFC concentrations and human dietary exposure via fish and shellfish consumption around the world. The results suggested PFOS was the predominant PFC in fish and shellfish (Table S4). Other PFCs, such as PFOA, PFNA, PFNA, PFUnDA and PFHxS, were also frequently found at considerable concentrations in fish and shellfish (Fig. 4). PFOS was detected in all fish and shellfish except mussels from Catalonia, Spain at concentrations up to 2.7 ng/g ww (Table S4) (Domingo et al. 2012b). PFOA had a mean concentration of 2.6 ng/g ww in the same samples (Table S4) (Domingo et al. 2012a). Fish samples collected in Norwegian grocery stores contained lower PFC concentrations than those reported in other countries (Haug et al. 2010), such as Greece (Vassiliadou et al. 2015), Korea (Heo et al. 2014; Naile et al. 2013), China (Wu et al. 2012; Zhang et al. 2011b), and Italy (Guerranti et al. 2013). Zhang et al. (2011b) analyzed 72 fish and seafood samples collected from markets and supermarkets in 13 Chinese provinces and municipalities, where PFOS had the highest mean concentration (0.92 ng/g ww) (Table S4). PFOS was also detected as the predominant PFC chemical in all fish samples collected from the coastal areas of north Bohai Sea, China (Wang et al. 2011b), consistent with the finding from other previous studies that PFOS has the higher bioaccumulation potential than other PFCs (Domingo et al. 2012a; Guerranti et al. 2013; Heo et al. 2014; Naile et al. 2013; Zhang et al. 2011b). However, PFOA appeared to be most abundant in mollusks collected from the Bohai Sea in another study (Pan et al. 2010), likely suggesting additional PFOA discharges from manufacturing industries in that area. Nania et al. (2009) observed high PFOS and PFOA concentrations (3.0–53 ng/g ww) in edible fish from the Mediterranean Sea (Table S4), although it is located in a semi-closed basin with scare water exchange. Similarly, several PFCs (e.g., PFOS, PFDA, and PFUnDA) were found in fish samples collected from Qinghai-Tibetan Plateau in China, although this region is far away from modern industrial or commercial activities (Shi et al. 2010). A few studies also evaluated the correlations between different types of PFCs in shellfish (Pan et al. 2010; Wang et al. 2011b; Wu et al. 2012). For example, concentrations of PFHpA, PFOA and PFOS in mollusks from coastal water in Bohai Sea were significantly correlated between each other (Pan et al. 2010). In addition, PFOA, PFNA, PFNA and PFUnDA in shellfish and PFOS and PFUnDA in fatty fish samples from Chinese coastal areas also showed significant correlations ($p < 0.01$) (Wu et al. 2012). These findings may indicate similar sources and accumulation pathways of these PFC compounds in the same region.

A cross-study comparison revealed several characteristics of PFC contamination in fish and shellfish. Firstly, the types and concentrations of PFCs differed between fish (or shellfish) species or regions (Fig. 4). Naile et al. (2013) reported that the bioconcentration factors (BCFs) of PFCs varied significantly in species even from the same region (i.e.,

Table 2
Mean concentration (pg/g, Fw) of PFCs in eggs, meat and meat products.

Sampling	Region	PFBA	PFPeA	PFHxA	PFHpA	PFHxS	PFOA	PFNA	PFOS	PFDA	PFUnDA	PFDS	PFDoDA	PFTrDA	PFTeDA	PFBuS	Year	Reference
Eggs	Sweden	-	-	3.6	nd	2.5	39	nd	39	3.3	nd	-	nd	nd	nd	-	2010	Vestergren et al. (2012)
Eggs	Sweden	-	-	5.1	nd	nd	7.2	5.6	13	4.9	3.3	-	nd	nd	nd	-	2005	Vestergren et al. (2012)
Eggs	Sweden	-	-	5.1	nd	39	31	22	1280	15	38	-	10	14	nd	-	1999	Vestergren et al. (2012)
Eggs	Catalonia, Spain	< 120	< 16	< 39	200	< 2.0	< 390	< 100	< 5.3	< 10	< 38	< 0.60	< 11	< 17	< 17	-	2011	Domingo et al. (2012b)
Eggs	Catalonia, Spain	-	-	-	< 5	-	< 55		82	-	-	-	-	-	-	-	2006	Ericson et al. (2008)
Eggs	Oslo, Norway	-	-	13	< 16	3.5	30	< 7.4	39	12	9.9	-	< 8.1	-	-	2.0	2008–2009	Haug et al. (2010)
Eggs	Netherlands	< 4000	< 512	< 54	< 2	< 6	< 32	6	29	11	-	-	< 13	< 107	< 5	-	2009	Noorlander et al. (2011)
Eggs (chicken)	China	-	-	-	< 130	< 110	260	< 120	80	20	< 620	20	< 520	-	-	-	2009	Zhang et al. (2010)
Eggs (duck)	China	-	-	-	< 130	< 110	120	240	340	140	300	80	< 520	-	-	-	2009	Zhang et al. (2010)
Meat and meat products	Busan, Korea	37	131	515	nd	nd	nd	nd	353	132	105	nd	325	nd	7	-	2011–2012	Heo et al. (2014)
Meat products	Sweden	-	-	nd	2.3	4.5	12	5.8	25	6.3	2.5	-	1.1	nd	nd	-	2010	Vestergren et al. (2012)
Meat products	Sweden	-	-	nd	nd	5.1	16	9.2	86	6.4	7.8	-	2.1	3.8	nd	-	2005	Vestergren et al. (2012)
Meat products	Sweden	-	-	nd	3.2	8.5	24	7.1	190	5.2	4.8	-	1.9	nd	nd	-	1999	Vestergren et al. (2012)
Meat and meat products	Catalonia, Spain	< 90	< 12	< 30	280	3.2	< 300	< 79	34	< 7.9	< 29	< 0.46	< 8.7	< 13	< 13	-	2011	Domingo et al. (2012a, b)
Meat (pork)	Catalonia, Spain	-	-	-	< 6	-	< 53	-	45	-	-	-	-	-	-	-	2006	Ericson et al. (2008)
Meat (chicken)	Catalonia, Spain	-	-	-	< 4	-	< 67	-	21	-	-	-	-	-	-	-	2006	Ericson et al. (2008)
Meat (pork)	Oslo, Norway	-	-	< 4.3	2.8	1.2	15	5.5	17	16	< 8.2	-	< 8.0	-	-	< 0.81	2008–2009	Haug et al. (2010)
Meat (chicken)	Oslo, Norway	-	-	< 13	20	< 2.3	52	6.8	21	< 23	13	-	< 9.2	-	-	3.2	2008–2009	Haug et al. (2010)
Meat (beef)	Oslo, Norway	-	-	< 3.3	7.6	< 0.28	12	15	60	23	< 6.4	-	< 6.2	-	-	< 0.63	2008–2009	Haug et al. (2010)
Meat (pork)	Netherlands	< 112	< 104	< 11	6	< 5	15	2	14	2	-	-	< 3	< 23	< 1	-	2009	Noorlander et al. (2011)
Meat (beef)	Netherlands	< 48	< 44	< 5	< 0.2	< 4	< 5	4	82	6	-	-	< 2	< 14	< 0.7	-	2009	Noorlander et al. (2011)
Meat (pork)	China	-	-	-	< 220	< 120	6170	< 100	60	< 130	< 600	< 120	< 1120	-	-	-	2009	Zhang et al. (2010)
Meat (beef)	China	-	-	-	150	< 120	4260	< 100	50	< 130	< 600	< 120	< 1120	-	-	-	2009	Zhang et al. (2010)
Meat (chicken)	China	-	-	-	< 220	< 120	12,500	< 100	80	< 130	< 600	< 120	< 1120	-	-	-	2009	Zhang et al. (2010)
Meat (goat)	China	-	-	-	< 220	< 120	1560	200	80	< 130	< 600	< 120	< 1120	-	-	-	2009	Zhang et al. (2010)
Meat	Siena, Italy	-	-	-	-	-	< 500	-	1430	-	-	-	-	-	-	-		Guerranti et al. (2013)
Meat (pork)	Siena, Italy	-	-	-	-	-	< 500	-	740	-	-	-	-	-	-	-		Guerranti et al. (2013)
Meat (beef)	Siena, Italy	-	-	-	-	-	< 500	-	2110	-	-	-	-	-	-	-		Guerranti et al. (2013)

[-] Not analyzed; [nd] not detected.

THPFOS: 43 pg/g ww in meat and its products, below the limit of detection of 47 pg/g ww in eggs that collected in Catalonia, Spain (Domingo et al. 2012b); not analyzed in other countries.

PFHxDA: below the limit detection of 130 and 99 pg/g in eggs and meat (and its products) respectively that collected in Catalonia, Spain (Domingo et al. 2012b); not analyzed in other countries.

PFOcDA: below the limit detection of 110 and 86 pg/g in eggs and meat (and its products) respectively that collected in Catalonia, Spain (Domingo et al. 2012b); not analyzed in other countries.

PFBuS: 2.0 and 3.2 pg/g in eggs and chicken, below the limit of detection of 0.81 and 0.63 in pork and beef respectively that collected in Oslo, Norway (Haug et al. 2010); not analyzed in other countries.

PFUDA: 2 pg/g ww in beef, below the limit detection of 19 and 4 pg/g in eggs and pork respectively that collected in Netherlands (Noorlander et al. 2011); not analyzed in other countries.

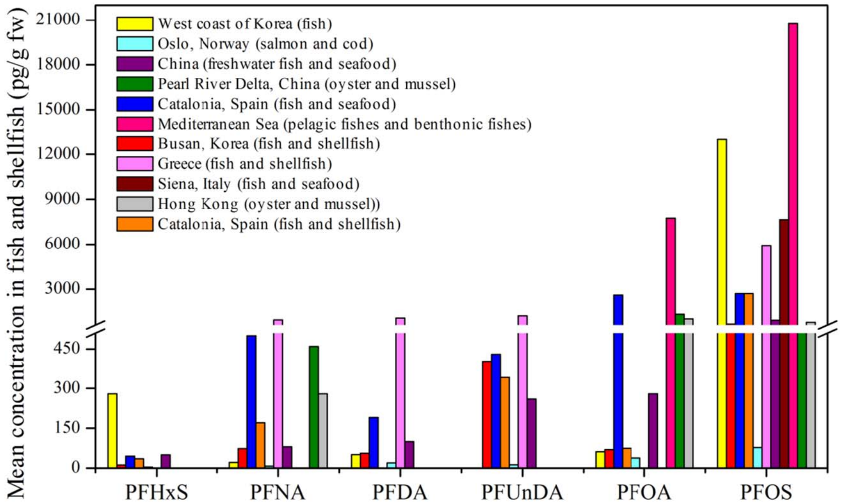

Fig. 4. The mean concentrations of typical PFCs in fish and shelfish from different regions.

west coast of Korea). PFBS and PFOS had the highest BCFs in crab, whereas the BCFs were the greatest for PFOS and PFDA in fish and for PFHxS in shellfish (Naile et al. 2013). Wu et al. (2012) reported that concentrations PFOA, PFNA, and PFOS differed significantly between fatty fish and shellfish in six coastal provinces of China. The detection frequency of PFOA also differed significantly between these two types of fish (i.e., 55% vs. 96%). Furthermore, the factors of age, weight and size of fish and shellfish may also lead to cross-study differences in PFC concentrations (Haug et al. 2010). It has been suggested that higher trophic level species generally contained greater PFC concentrations than lower trophic level organisms (Kannan et al. 2005; Li et al. 2008). Zhang et al. (2011b) found levels of PFOS, PFOA, PFDA and PFUnDA in carnivorous fish such as crucian carp (*Carassius carassius*) were considerably higher than in herbivorous fish (e.g., silver carp (*Hypophthalmichthys molitrix*)). These results suggest that PFC concentrations increased along with trophic levels through food chain transfer (Sinclair et al. 2006).

In addition, environmental media (such as water and sediment) would be another important factor influencing PFC concentrations in fish and shellfish. So et al. (2007) reported that PFOA or PFOS was the most abundant PFC in water samples from Yangtze River or Pearl River (China), respectively. Wu et al. (2012) revealed that PFOA was the dominant PFC substance in shellfish collected from the coast areas of Jiangsu and Zhejiang Provinces which receive water from Yangtze River, whereas PFOS dominated in shellfish collected from Guangdong located in the Pearl River Delta. This clearly indicated that PFC contamination profiles in aquatic organisms were substantially affected by their living environment (e.g., water and sediment).

Finally, PFC contamination in fish or shellfish was greatly affected by industrial emission, the most important discharge source. Vassiliadou et al. (2015) reported that high concentrations of PFOS in picarel samples were attributed to the dense human population and prosperous industries nearby the sampling site. As Wuhan is the main PFOS production region in China, where several PFOS and related chemical factories were located, high concentrations of PFOS were observed in wastewater from that area (Wang et al. 2010b). Fish and river foodstuffs in Wuhan also showed the highest mean concentration of PFOS (3.13 ng/g ww) among Chinese studies (Zhang et al. 2011b). Substantial PFOS contamination was also reported in aquatic food from Shenyang (0.46 ng/g ww), where the largest PFCA manufacturing facility in China was located (Bao et al. 2011). In the study by Wu et al. (2012), the maximum concentration of PFOA in shellfish (i.e., briny clam at 7.5 ng/g ww) and the highest concentrations of PFNA and PFOA in fatty fish (i.e. blue scad at 0.31 and 0.26 pg/g ww, respectively) were both detected in Jiangsu compared with other five provinces (Table S4), which may be a result of highly active industrial activities in the Yangtze River Delta where Jiangsu is located.

2.7. Drinking water

Drinking water is another common source of PFCs to humans. A variety of PFCs have been detected in different drinking water samples collected from various countries (Table S5) (Boiteux et al. 2012; Domingo et al. 2012a; Haug et al. 2010; Heo et al. 2014; Llorca et al. 2012). PFOS, PFHxA, PFHpA, PFNA, PFDA, and PFOA appeared to be the main PFCs with the highest concentrations and detection frequencies in drinking water. Their spatial distribution profiles are shown in Fig. 5. The top two PFOA concentrations were found in tap and well water samples collected from Japan and Ghana respectively, and their concentrations were approximately 100 ng/L and 800 ng/L (Essumang et al. 2017; Shiwaku et al. 2016). The concentrations of PFOA ranged from 0 to 20 ng/L in other countries, which may represent the general range of PFOA contamination in drinking water (Table S5). The top two PFOS concentrations were found in tap water collected from Ghana and Spain, and their concentrations were approximately 100 ng/L and 40 ng/L, respectively (Table S5) (Essumang et al. 2017; Schwanz et al. 2016).

Furthermore, we grouped drinking water samples into bottled water, tap water, raw water, drinking water, and well water (Fig. S2). It is clear that PFC levels varied in an order of well water > tap water > bottled water > drinking water > raw water. The highest PFC contamination in well water indicated that point sources could be the main cause. The greater PFC concentrations in tap and drinking water than in raw water indicated the role of drinking water treatment processes in PFC contamination. Domingo et al. (2012a) reported concentrations of PFOA and PFOS in 30 water samples from Spanish water purification plants were 2.40 and 1.81 ng/g, respectively (Table S5). It is noteworthy that samples collected in Barcelona area (Spain) were detected with the highest PFC concentration, likely due to active industries in this area. Nakayama et al. (2010) also attributed high PFBA concentrations in the Upper Mississippi River Basin to the presence of short-chain PFCs manufacturing industry. PFBA was detected in all untreated and treated water samples from three out of four lakes in Faroe Islands, where the highest concentration was 1.2 ng/g in untreated water from Kornvatn (Eriksson et al. 2013). Relatively high PFBA concentrations were also found in tap water from Korea (2.02 ng/L) and Spain (10 ng/L) (Table S5) (Heo et al. 2014; Llorca et al. 2012). Domingo et al. (2012a) reported that drinking water treatment processes decreased the levels of all PFCs in water except for PFBS. Eschauzier et al. (2012) demonstrated that granular activated carbon (GAC) could efficiently reduce long-chain PFCs, but have little effect on shorter chain PFCs such as PFBA and PFBS. Taniyasu et al. (2013)

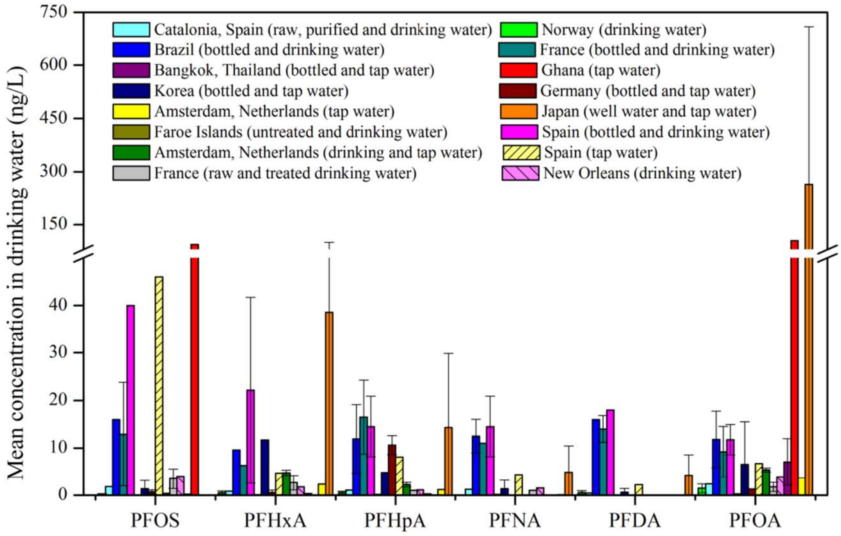

Fig. 5. The mean concentration of typical PFCs in drinking water from different regions.

reported that shorter chain PFCs have greater resistance to photolysis than long-chain PFCs and degradation of long-chain PFCs may become a potentially significant source for shorter chain PFCs. In the water treatment plants of Kornvatn and Havnardal, PFAS concentrations were found to decrease and increase after purification, respectively. It was proposed that the degradation of precursor compounds and long-chain chemicals could lead to PFAS increase in Havnardal (Eriksson et al. 2013). Boiteux et al. (2012) also observed both the elimination and degradation of PFCs in the water treatment processes. Thus, purification processes greatly influence PFC levels in post-treatment water, while the carbon chain lengths of PFCs in untreated water also have significant influence on the purification efficiency. Schwanz et al. (2016) determined that PFAS concentrations in tap and bottled water from Brazil, France and Spain were in the order of Brazil > France > Spain. It should be noticed that PFHxA was detected at elevated frequencies and concentrations in both tap and bottled water from three countries. PFOS was not found in bottled water in Brazil and Spain, but was the most abundant in tap water in these two countries with the detection frequency of 100% and 38%, respectively. Llorca et al. (2012) also reported the non-detection of PFASs in four Spanish mineral bottled water samples. However, a mean PFHpA concentration of 12 ng/L was reported in mineral bottled water from Germany and the plastic bottle itself was suspected as the contamination source (Table S5). Schwanz et al. (2016) also proposed that water bottles and label ink were potential PFHpA sources to bottled water, although no direct evidence has been obtained to support this hypothesis.

3. Conclusions

In this review, PFC concentrations in indoor air and dust, food, and drinking water were profiled for human exposure risk assessment. FTOHs, FOSAs, FOSEs, and FTAs were found to be the main PFC substances in indoor air and the total FTOH concentrations can reach up to 20,000 pg/m^3. In contrast with indoor air, indoor dust was more abundant in PFOS, PFOA, PFHxS, FOSAs, and FOSEs. The sources of these PFCs were mostly from direct release of PFC-containing dust (such as: fibers) while a few were from their partition on dust in air. PFC concentrations in indoor air or dust exhibited spatial variations, contributed by diversified lifestyles in different cultures, building characteristics and decorating characteristics, resident habits in buying clothes or consumer products, the quantities and locations of samples, sample collection and analysis methods, and a region's economic status. PFOS and PFOA were the dominant PFCs in vegetables with concentrations generally < 100 pg/g. Milk contained more PFCs than yogurt, butter/cheese, and beverages, which was contrast with the traditional understanding that PFC contamination would increase along with industrial processing. On the other hand, the loading of different regions and different packaging materials may critically affect PFC concentrations in milk products and beverages. PFC concentrations in eggs and meat products generally declined with time, likely resulting from the phase-out of and strict regulation on PFCs. However, high concentrations of PFCs were still detected in meat products from some countries, due to continuous productions of PFC products in these countries. Fish and shellfish generally exhibited the highest PFC concentrations and detection frequencies among all foodstuffs. PFC concentrations in fish and shellfish were affected by species, age, weight and size, as well as environmental media (e.g., water and sediment), and industrial emission. PFC levels in food items generally followed a trend as: fish and shellfish > eggs and meat products > milk products and beverages > vegetables. The high bioaccumulation potential and bio-magnification effect of PFC is the main reason for higher PFC contents in fish, shellfish, and meat products than other type of foods. PFCs can be accumulated more by food chain in higher trophic level species than lower trophic level ones. Drinking water is another main PFC source to humans. PFOS, PFHxA, PFHpA, PFNA, PFDA, and PFOA appeared to be the main PFCs in drinking water. The contamination by PFCs in different types of water were in the order of well water > tap water > bottled water > drinking water > raw water. Overall, besides traditional ones (such as: fish and drinking water), dust, eggs, and meat products are also needed to be attention for high PFC exposure risks.

Acknowledgments

This research was supported by the National Natural Science Foundation of China (Project No. 41503087, Project No. 21637001, and No. 21577050) and State Key Laboratory of Organic Geochemistry, GIGCAS (Project No. OGL-201507).

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.envint.2017.07.024.

References

Ahrens, L., Bundschuh, M., 2014. Fate and effects of poly- and perfluoroalkyl substances in the aquatic environment: a review. Environ. Toxicol. Chem. 33, 1921–1929.
Alves, A., Jacobs, G., Vanermen, G., Covaci, A., Voorspoels, S., 2015. New approach for assessing human perfluoroalkyl exposure via hair. Talanta 144, 574–583.
Bao, J., Liu, W., Liu, L., Jin, Y., Dai, J., Ran, X., Zhang, Z., Tsuda, S., 2011. Perfluorinated

compounds in the environment and the blood of residents living near fluorochemical plants in Fuxin, China. Environ. Sci. Technol. 45, 8075–8080.

Barbarossa, A., Masetti, R., Gazzotti, T., Zama, D., Astolfi, A., Veyrand, B., Pession, A., Pagliuca, G., 2013. Perfluoroalkyl substances in human milk: a first survey in Italy. Environ. Int. 51, 27–30.

Begley, T.H., Hsu, W., Noonan, G., Diachenko, G., 2008. Migration of fluorochemical paper additives from food-contact paper into foods and food simulants. Food Addit. Contam. 25, 384–390.

Bjerregaard-Olesen, C., Bach, C.C., Long, M., Ghisari, M., Bossi, R., Bech, B.H., Nohr, E.A., Henriksen, T.B., Olsen, J., Bonefeld-Jorgensen, E.C., 2016. Time trends of perfluorinated alkyl acids in serum from Danish pregnant women 2008-2013. Environ. Int. 91, 14–21.

Bjorklund, J.A., Thuresson, K., De Wit, C.A., 2009. Perfluoroalkyl compounds (PFCs) in indoor dust: concentrations, human exposure estimates, and sources. Environ. Sci. Technol. 43, 2276–2281.

Blaine, A.C., Rich, C.D., Hundal, L.S., Lau, C., Mills, M.A., Harris, K.M., Higgins, C.P., 2013. Uptake of perfluoroalkyl acids into edible crops via land applied biosolids: field and greenhouse studies. Environ. Sci. Technol. 47, 14062–14069.

Bloom, M.S., Kannan, K., Spliethoff, H.M., Tao, L., Aldous, K.M., Vena, J.E., 2010. Exploratory assessment of perfluorinated compounds and human thyroid function. Physiol. Behav. 99, 240–245.

Boiteux, V., Dauchy, X., Rosin, C., Munoz, J.F., 2012. National screening study on 10 perfluorinated compounds in raw and treated tap water in France. Arch. Environ. Contam. Toxicol. 63, 1–12.

Butenhoff, J.L., Olsen, G.W., Pfahles-Hutchens, A., 2006. The applicability of biomonitoring data for perfluorooctanesulfonate to the environmental public health continuum. Environ. Health Perspect. 114, 1776–1782.

Butt, C.M., Muir, D.C.G., Mabury, S.A., 2010. Elucidating the pathways of poly- and perfluorinated acid formation in rainbow trout. Environ. Sci. Technol. 44, 4973–4980.

Chen, C., Lu, Y., Zhang, X., Geng, J., Wang, T., Shi, Y., Hu, W., Li, J., 2009. A review of spatial and temporal assessment of PFOS and PFOA contamination in China. Chem. Ecol. 25, 163–177.

Clara, M., Scheffknecht, C., Scharf, S., Weiss, S., Gans, O., 2008. Emissions of perfluorinated alkylated substances (PFAS) from point sources - identification of relevant branches. Water Sci. Technol. 58, 59–66.

Clarke, D.B., Bailey, V.A., Routledge, A., Lloyd, A.S., Hird, S., Mortimer, D.N., Gem, M., 2010. Dietary intake estimate for perfluorooctanesulphonic acid (PFOS) and other perfluorocompounds (PFCs) in UK retail foods following determination using standard addition LC-MS/MS. Food Addit. Contam. 27, 530–545.

Cornelis, C., D'Hollander, W., Roosens, L., Covaci, A., Smolders, R., Van Den Heuvel, R., Govarts, E., Van Campenhout, K., Reynders, H., Bervoets, L., 2012. First assessment of population exposure to perfluorinated compounds in Flanders, Belgium. Chemosphere 86, 308–314.

Costa, G., Sartori, S., Consonni, D., 2009. Thirty years of medical surveillance in perfluooctanoic acid production workers. J. Occup. Environ. Med. 51, 364–372.

Dallaire, R., Dewailly, E., Pereg, D., Dery, S., Ayotte, P., 2009. Thyroid function and plasma concentrations of polyhalogenated compounds in inuit adults. Environ. Health Perspect. 117, 1380–1386.

De Felip, E., Abballe, A., Albano, F.L., Battista, T., Carraro, V., Conversano, M., Franchini, S., Giambanco, L., Iacovella, N., Ingelido, A.M., Maiorana, A., Maneschi, F., Marra, V., Mercurio, A., Nale, R., Nucci, B., Panella, V., Pirola, F., Porpora, M.G., Procopio, E., Suma, N., Valentini, S., Valsenti, L., Vecchie, V., 2015. Current exposure of Italian women of reproductive age to PFOS and PFOA: a human biomonitoring study. Chemosphere 137, 1–8.

D'Eon, J.C., Mabury, S.A., 2011. Is indirect exposure a significant contributor to the burden of perfluorinated acids observed in humans? Environ. Sci. Technol. 45, 7974–7984.

D'Hollander, W., de Voogt, P., De Coen, W., Bervoets, L., 2010a. Perfluorinated substances in human food and other sources of human exposure. In: Whitacre, D.M., De Voogt, P. (Eds.), Reviews of Environmental Contamination and Toxicology, Vol 208: Perfluorinated Alkylated Substances.

D'Hollander, W., Roosens, L., Covaci, A., Cornelis, C., Reynders, H., Campenhout, K.V., Voogt, P., Bervoets, L., 2010b. Brominated flame retardants and perfluorinated compounds in indoor dust from homes and offices in Flanders, Belgium. Chemosphere 81, 478–487.

Domingo, J.L., 2012. Health risks of dietary exposure to perfluorinated compounds. Environ. Int. 40, 187–195.

Domingo, J.L., Ericson-Jogsten, I., Perello, G., Nadal, M., Van Bavel, B., Karrman, A., 2012a. Human exposure to perfluorinated compounds in Catalonia, Spain: contribution of drinking water and fish and shellfish. J. Agric. Food Chem. 60, 4408–4415.

Domingo, J.L., Jogsten, I.E., Eriksson, U., Martorell, I., Perello, G., Nadal, M., Bavel, B., 2012b. Human dietary exposure to perfluoroalkyl substances in Catalonia, Spain, temporal trend. Food Chem. 135, 1575–1582.

Dong, G.-H., Tung, K.-Y., Tsai, C.-H., Liu, M.-M., Wang, D., Liu, W., Jin, Y.-H., Hsieh, W.-S., Lee, Y.L., Chen, P.-C., 2013. Serum polyfluoroalkyl concentrations, asthma outcomes, and immunological markers in a case-control study of Taiwanese children. Environ. Health Perspect. 121, 507–513.

Ehresman, D.J., Froehlich, J.W., Olsen, G.W., Chang, S.-C., Butenhoff, J.L., 2007. Comparison of human whole blood, plasma, and serum matrices for the determination of perfluorooctanesulfonate (PFOS), perfluorooctanoate (PFOA), and other fluorochemicals. Environ. Res. 103, 176–184.

Ellis, D.A., Martin, J.W., De Silva, A.O., Mabury, S.A., Hurley, M.D., Andersen, M.P.S., Wallington, T.J., 2004. Degradation of fluorotelomer alcohols: a likely atmospheric source of perfluorinated carboxylic acids. Environ. Sci. Technol. 38, 3316–3321.

Enault, J., Robert, S., Schlosser, O., de The, C., Loret, J.-F., 2015. Drinking water, diet, indoor air: comparison of the contribution to environmental micropollutants exposure. Int. J. Hyg. Environ. Health 218, 723–730.

Ericson, I., Marti-Cid, R., Nadal, M., Van Bavel, B., Lindstrom, G., Domingo, J.L., 2008. Human exposure to perfluorinated chemicals through the diet: Intake of perfluorinated compounds in foods from the Catalan (Spain) Market. J. Agric. Food. Chem. 56, 1787–1794.

Eriksson, U., Karrman, A., Rotander, A., Mikkelsen, B., Dam, M., 2013. Perfluoroalkyl substances (PFASs) in food and water from Faroe Islands. Environ. Sci. Pollut. Res. Int. 20, 7940–7948.

Eschauzier, C., Beerendonk, E., Scholte-Veenendaal, P., De Voogt, P., 2012. Impact of treatment processes on the removal of perfluoroalkyl acids from the drinking water production chain. Environ. Sci. Technol. 46, 1708–1715.

Essumang, D.K., Eshun, A., Hogarh, J.N., Bentum, J.K., Adjei, J.K., Negishi, J., Nakamichi, S., Habibullah-Al-Mamun, M., Masunaga, S., 2017. Perfluoroalkyl acids (PFAAs) in the Pra and Kakum River basins and associated tap water in Ghana. Sci. Total Environ. 579, 729–735.

Felizeter, S., McLachlan, M.S., de Voogt, P., 2012. Uptake of perfluorinated alkyl acids by hydroponically grown lettuce (Lactuca sativa). Environ. Sci. Technol. 46, 11735–11743.

Felizeter, S., McLachlan, M.S., De Voogt, P., 2014. Root uptake and trans location of perfluorinated alkyl acids by three hydroponically grown crops. J. Agric. Food Chem. 62, 3334–3342.

Fraser, A.J., Webster, T.F., Watkins, D.J., Nelson, J.W., Stapleton, H.M., Calafat, A.M., Kato, K., Shoeib, M., Vieira, V.M., McClean, M.D., 2012. Polyfluorinated compounds in serum linked to indoor air in office environments. Environ. Sci. Technol. 46, 1209–1215.

Fraser, A.J., Webster, T.F., Watkins, D.J., Strynar, M.J., Kato, K., Calafat, A.M., Vieira, V.M., McClean, M.D., 2013. Polyfluorinated compounds in dust from homes, offices, and vehicles as predictors of concentrations in office workers' serum. Environ. Int. 60, 128–136.

Fromme, H., Tittlemier, S.A., Volkel, W., Wilhelm, M., Twardella, D., 2009. Perfluorinated compounds–exposure assessment for the general population in western countries. Int. J. Hyg. Environ. Health 212, 239–270.

Fromme, H., Dreyer, A., Dietrich, S., Fembacher, L., Lahrz, T., Volkel, W., 2015. Neutral polyfluorinated compounds in indoor air in Germany–the LUPE 4 study. Chemosphere 139, 572–578.

Gallo, V., Leonardi, G., Genser, B., Lopez-Espinosa, M.-J., Frisbee, S.J., Karlsson, L., Ducatman, A.M., Fletcher, T., 2012. Serum perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS) concentrations and liver function biomarkers in a population with elevated PFOA exposure. Environ. Health Perspect. 120, 655–660.

Gebbink, W.A., Glynn, A., Darnerud, P.O., Berger, U., 2015. Perfluoroalkyl acids and their precursors in Swedish food: the relative importance of direct and indirect dietary exposure. Environ. Pollut. 198, 108–115.

Genuis, S.J., Beesoon, S., Birkholz, D., 2013. Biomonitoring and elimination of perfluorinated compounds and polychlorinated biphenyls through perspiration: blood, urine, and sweat study. ISRN Toxicol. 2013, 483832.

Gewurtz, S.B., Bhavsar, S.P., Crozier, P.W., Diamond, M.L., Helm, P.A., Marvin, C.H., Reiner, E.J., 2009. Perfluoroalkyl contaminants in window film: indoor/outdoor, urban/rural, and winter/summer contamination and assessment of carpet as a possible source. Environ. Sci. Technol. 43, 7317–7323.

Goosey, E., Harrad, S., 2012. Perfluoroalkyl substances in UK indoor and outdoor air: spatial and seasonal variation, and implications for human exposure. Environ. Int. 45, 86–90.

Guerranti, C., Perra, G., Corsolini, S., Focardi, S.E., 2013. Pilot study on levels of perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) in selected foodstuffs and human milk from Italy. Food Chem. 140, 197–203.

Gump, B.B., Wu, Q., Dumas, A.K., Kannan, K., 2011. Perfluorochemical (PFC) exposure in children: associations with impaired response inhibition. Environ. Sci. Technol. 45, 8151–8159.

Harada, K., Nakanishi, S., Saito, N., Tsutsui, T., Koizumi, A., 2005. Airborne perfluorooctanoate may be a substantial source contamination in Kyoto area, Japan. Bull. Environ. Contam. Toxicol. 74, 64–69.

Haug, L.S., Salihovic, S., Jogsten, I.E., Thomsen, C., van Bavel, B., Lindstrom, G., Becher, G., 2010. Levels in food and beverages and daily intake of perfluorinated compounds in Norway. Chemosphere 80, 1137–1143.

Haug, L.S., Huber, S., Schlabach, M., Becher, G., Thomsen, C., 2011. Investigation on per- and polyfluorinated compounds in paired samples of house dust and indoor air from Norwegian homes. Environ. Sci. Technol. 45, 7991–7998.

Heo, J.J., Lee, J.W., Kim, S.K., Oh, J.E., 2014. Foodstuff analyses show that seafood and water are major perfluoroalkyl acids (PFAAs) sources to humans in Korea. J. Hazard. Mater. 279, 402–409.

Herzke, D., Huber, S., Bervoets, L., D'Hollander, W., Hajslova, J., Pulkrabova, J., Brambilla, G., De Filippis, S.P., Klenow, S., Heinemeyer, G., de Voogt, P., 2013. Perfluorinated alkylated substances in vegetables collected in four European countries; occurrence and human exposure estimations. Environ. Sci. Pollut. Res. Int. 20, 7930–7939.

Higgins, C.P., Luthy, R.G., 2006. Sorption of perfluorinated surfactants on sediments. Environ. Sci. Technol. 40, 7251–7256.

Hoelzer, J., Midasch, O., Rauchfuss, K., Kraft, M., Reupert, R., Angerer, J., Kleeschulte, P., Marschall, N., Wilhelm, M., 2008. Biomonitoring of perfluorinated compounds in children and adults exposed to perfluorooctanoate-contaminated drinking water. Environ. Health Perspect. 116, 651–657.

Hoffman, K., Webster, T.F., Bartell, S.M., Weisskopf, M.G., Fletcher, T., Vieira, V.M., 2011. Private drinking water wells as a source of exposure to perfluorooctanoic acid (PFOA) in communities surrounding a fluoropolymer production facility. Environ.

Health Perspect. 119, 92–97.

Houde, M., Martin, J.W., Letcher, R.J., Solomon, K.R., Muir, D.C.G., 2006. Biological monitoring of polyfluoroalkyl substances: a review. Environ. Sci. Technol. 40, 3463–3473.

Huber, S., Haug, L.S., Schlabach, M., 2011. Per- and polyfluorinated compounds in house dust and indoor air from northern Norway - a pilot study. Chemosphere 84, 1686–1693.

Ji, K., Kim, S., Kho, Y., Sakong, J., Paek, D., Choi, K., 2012. Major perfluoroalkyl acid (PFAA) concentrations and influence of food consumption among the general population of Daegu, Korea. Sci. Total Environ. 438, 42–48.

Jogsten, I.E., Nadal, M., van Bavel, B., Lindstrom, G., Domingo, J.L., 2012. Per- and polyfluorinated compounds (PFCs) in house dust and indoor air in Catalonia, Spain: implications for human exposure. Environ. Int. 39, 172–180.

Jurado-Sanchez, B., Ballesteros, E., Gallego, M., 2014. Analytical method for biomonitoring of perfluoroalkyl acids in human urine. Talanta 128, 141–146.

Kannan, K., Tao, L., Sinclair, E., Pastva, S.D., Jude, D.J., Giesy, J.P., 2005. Perfluorinated compounds in aquatic organisms at various trophic levels in a Great Lakes food chain. Arch. Environ. Contam. Toxicol. 48, 559–566.

Kantiani, L., Llorca, M., Sanchis, J., Farre, M., Barcelo, D., 2010. Emerging food contaminants: a review. Anal. Bioanal. Chem. 398, 2413–2427.

Karaskova, P., Venier, M., Melymuk, L., Becanova, J., Vojta, S., Prokes, R., Diamond, M.L., Klanova, J., 2016. Perfluorinated alkyl substances (PFASs) in household dust in Central Europe and North America. Environ. Int. 94, 315–324.

Karrman, A., Langlois, I., van Bavel, B., Lindstrom, G., Oehme, M., 2007. Identification and pattern of perfluorooctane sulfonate (PFOS) isomers in human serum and plasma. Environ. Int. 33, 782–788.

Knobeloch, L., Imm, P., Anderson, H., 2012. Perfluoroalkyl chemicals in vacuum cleaner dust from 39 Wisconsin homes. Chemosphere 88, 779–783.

Kovarova, J., Svobodova, Z., 2008. Perfluorinated compounds: occurrence and risk profile. Neuroendocrinol. Lett. 29, 599–608.

Krol, S., Zabiegala, B., Namiesnik, J., 2013. Human hair as a biomarker of human exposure to persistent organic pollutants (POPs). TrAC Trends Anal. Chem. 47, 84–98.

Kubwabo, C., Stewart, B., Zhu, J.P., Marro, L., 2005. Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada. J. Environ. Monit. 7, 1074–1078.

Landsteiner, A., Huset, C., Williams, A., Johnson, J., 2014. Biomonitoring for perfluorochemicals in a Minnesota community with known drinking water contamination. J. Environ. Health 77, 14–19.

Langer, V., Dreyer, A., Ebinghaus, R., 2010. Polyfluorinated compounds in residential and nonresidential indoor air. Environ. Sci. Technol. 44, 8075–8081.

Lau, C., Anitole, K., Hodes, C., Lai, D., Pfahles-Hutchens, A., Seed, J., 2007. Perfluoroalkyl acids: a review of monitoring and toxicological findings. Toxicol. Sci. 99, 366–394.

Lechner, M., Knapp, H., 2011. Carryover of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) from soil to plant and distribution to the different plant compartments studied in cultures of carrots (Daucus carota ssp. Sativus), potatoes (Solanum tuberosum), and cucumbers (Cucumis Sativus). J. Agric. Food Chem. 59, 11011–11018.

Li, X., Yin Yeung, L.W., Xu, M., Taniyasu, S., Lam, P.K., Yamashita, N., Dai, J., 2008. Perfluorooctane sulfonate (PFOS) and other fluorochemicals in fish blood collected near the outfall of wastewater treatment plant (WWTP) in Beijing. Environ. Pollut. 156, 1298–1303.

Li, J., Guo, F., Wang, Y., Liu, J., Cai, Z., Zhang, J., Zhao, Y., Wu, Y., 2012. Development of extraction methods for the analysis of perfluorinated compounds in human hair and nail by high performance liquid chromatography tandem mass spectrometry. J. Chromatogr. A 1219, 54–60.

Li, J., Guo, F., Wang, Y., Zhang, J., Zhong, Y., Zhao, Y., Wu, Y., 2013. Can nail, hair and urine be used for biomonitoring of human exposure to perfluorooctane sulfonate and perfluorooctanoic acid? Environ. Int. 53, 47–52.

Li, L., Zhai, Z., Liu, J., Hu, J., 2015. Estimating industrial and domestic environmental releases of perfluorooctanoic acid and its salts in China from 2004 to 2012. Chemosphere 129, 100–109.

Lindstrom, A.B., Strynar, M.J., Libelo, E.L., 2011. Polyfluorinated compounds: past, present, and future. Environ. Sci. Technol. 45, 7954–7961.

Liu, W., Xu, L., Li, X., Jin, Y.H., Sasaki, K., Saito, N., Sato, I., Tsuda, S., 2011. Human nails analysis as biomarker of exposure to perfluoroalkyl compounds. Environ. Sci. Technol. 45, 8144–8150.

Llorca, M., Farre, M., Pico, Y., Muller, J., Knepper, T.P., Barcelo, D., 2012. Analysis of perfluoroalkyl substances in waters from Germany and Spain. Sci. Total Environ. 431, 139–150.

Lopez-Espinosa, M.-J., Fletcher, T., Armstrong, B., Genser, B., Dhatariya, K., Mondal, D., Ducatman, A., Leonardi, G., 2011. Association of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) with age of puberty among children living near a chemical plant. Environ. Sci. Technol. 45, 8160–8166.

Lorber, M., Egeghy, P.P., 2011. Simple intake and pharmacokinetic modeling to characterize exposure of Americans to perfluoroctanoic acid, PFOA. Environ. Sci. Technol. 45, 8006–8014.

Martin, J., Luis Santos, J., Aparicio, I., Alonso, E., 2016. Analytical method for biomonitoring of endocrine-disrupting compounds (bisphenol A, parabens, perfluoroalkyl compounds and a brominated flame retardant) in human hair by liquid chromatography-tandem mass spectrometry. Anal. Chim. Acta 945, 95–101.

Mercier, F., Glorennec, P., Thomas, O., Le Bot, B., 2011. Organic contamination of settled house dust, a review for exposure assessment purposes. Environ. Sci. Technol. 45, 6716–6727.

MiraIles-Marco, A., Harrad, S., 2015. Perfluorooctane sulfonate: a review of human exposure, biomonitoring and the environmental forensics utility of its chirality and isomer distribution. Environ. Int. 77, 148–159.

Naile, J.E., Khim, J.S., Hong, S., Park, J., Kwon, B.O., Ryu, J.S., Hwang, J.H., Jones, P.D., Giesy, J.P., 2013. Distributions and bioconcentration characteristics of perfluorinated compounds in environmental samples collected from the west coast of Korea. Chemosphere 90, 387–394.

Nakayama, S.F., Strynar, M.J., Reiner, J.L., Delinsky, A.D., Lindstrom, A.B., 2010. Determination of perfluorinated compounds in the upper Mississippi River Basin. Environ. Sci. Technol. 44, 4103–4109.

Nania, V., Pellegrini, G.E., Fabrizi, L., Sesta, G., Sanctis, P.D., Lucchetti, D., Pasquale, M.D., Coni, E., 2009. Monitoring of perfluorinated compounds in edible fish from the Mediterranean Sea. Food Chem. 115, 951–957.

Noorlander, C.W., van Leeuwen, S.P., Te Biesebeek, J.D., Mengelers, M.J., Zeilmaker, M.J., 2011. Levels of perfluorinated compounds in food and dietary intake of PFOS and PFOA in the Netherlands. J. Agric. Food Chem. 59, 7496–7505.

Pan, Y., Shi, Y., Wang, Y., Cai, Y., Jiang, G., 2010. Investigation of perfluorinated compounds (PFCs) in mollusks from coastal waters in the Bohai Sea of China. J. Environ. Monit. 12, 508–513.

Paul, A.G., Jones, K.C., Sweetman, A.J., 2009. A first global production, emission, and environmental inventory for perfluorooctane sulfonate. Environ. Sci. Technol. 43, 386–392.

Piekarz, A.M., Primbs, T., Field, J.A., Barofsky, D.F., Simonich, S., 2007. Semivolatile fluorinated organic compounds in Asian and western U.S air masses. Environ. Sci. Technol. 41, 8248–8255.

Post, G.B., Louis, J.B., Cooper, K.R., Boros-Russo, B.J., Lippincott, R.L., 2009. Occurrence and potential significance of perfluorooctanoic acid (PFOA) detected in New Jersey public drinking water systems. Environ. Sci. Technol. 43, 4547–4554.

Rodriguez-Gomez, R., Martin, J., Zafra-Gomez, A., Alonso, E., Vilchez, J.L., Navalon, A., 2017. Biomonitoring of 21 endocrine disrupting chemicals in human hair samples using ultra-high performance liquid chromatography-tandem mass spectrometry. Chemosphere 168, 676–684.

Route, W.T., Russell, R.E., Lindstrom, A.B., Strynar, M.J., Key, R.L., 2014. Correction to spatial and temporal patterns in concentrations of perfluorinated compounds in bald eagle nestlings in the upper Midwestern United States. Environ. Sci. Technol. 48, 9957.

Sakr, C.J., Leonard, R.C., Kreckmann, K.H., Slade, M.D., Cullen, M.R., 2007. Longitudinal study of serum lipids and liver enzymes in workers with occupational exposure to ammonium perfluorooctanoate. J. Occup. Environ. Med. 49, 872–879.

Schwanz, T.G., Llorca, M., Farre, M., Barcelo, D., 2016. Perfluoroalkyl substances assessment in drinking waters from Brazil, France and Spain. Sci. Total Environ. 539, 143–152.

Shan, G., Wang, Z., Zhou, L., Du, P., Luo, X., Wu, Q., Zhu, L., 2016. Impacts of daily intakes on the isomeric profiles of perfluoroalkyl substances (PFASs) in human serum. Environ. Int. 89-90, 62–70.

Shi, Y., Pan, Y., Yang, R., Wang, Y., Cai, Y., 2010. Occurrence of perfluorinated compounds in fish from Qinghai-Tibetan Plateau. Environ. Int. 36, 46–50.

Shiwaku, Y., Lee, P., Thepaksorn, P., Zheng, B., Koizumi, A., Harada, K.H., 2016. Spatial and temporal trends in perfluorooctanoic and perfluorohexanoic acid in well, surface, and tap water around a fluoropolymer plant in Osaka, Japan. Chemosphere 164, 603–610.

Sinclair, E., Mayack, D.T., Roblee, K., Yamashita, N., Kannan, K., 2006. Occurrence of perfluoroalkyl surfactants in water, fish, and birds from New York State. Arch. Environ. Contam. Toxicol. 50, 398–410.

So, M.K., Yamashita, N., Taniyasu, S., Jiang, Q.T., Giesy, J.P., Chen, K., Lam, P.K.S., 2006. Health risks in infants associated with exposure to perfluorinated compounds in human breast milk from Zhoushan, China. Environ. Sci. Technol. 40, 2924–2929.

So, M.K., Miyake, Y., Yeung, W.Y., Ho, Y.M., Taniyasu, S., Rostkowski, P., Yamashita, N., Zhou, B.S., Shi, X.J., Wang, J.X., Giesy, J.P., Yu, H., Lam, P.K., 2007. Perfluorinated compounds in the Pearl River and Yangtze River of China. Chemosphere 68, 2085–2095.

Stahl, T., Heyn, J., Thiele, H., Huther, J., Failing, K., Georgii, S., Brunn, H., 2009. Carryover of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) from soil to plants. Arch. Environ. Contam. Toxicol. 57, 289–298.

Stein, C.R., Savitz, D.A., Dougan, M., 2009. Serum levels of perfluorooctanoic acid and perfluorooctane sulfonate and pregnancy outcome. Am. J. Epidemiol. 170, 837–846.

Still, M., Schlummer, M., Gruber, L., Fiedler, D., Wolz, G., 2013. Impact of industrial production and packaging processes on the concentration of per- and polyfluorinated compounds in milk and dairy products. J. Agric. Food Chem. 61, 9052–9062.

Taniyasu, S., Yamashita, N., Yamazaki, E., Petrick, G., Kannan, K., 2013. The environmental photolysis of perfluorooctanesulfonate, perfluorooctanoate, and related fluorochemicals. Chemosphere 90, 1686–1692.

Tao, L., Kannan, K., Wong, C.M., Arcaro, K.F., Butenhoff, J.L., 2008a. Perfluorinated compounds in human milk from Massachusetts, USA. Environ. Sci. Technol. 42, 3096–3101.

Tao, L., Ma, J., Kunisue, T., Libelo, E.L., Tanabe, S., Kannan, K., 2008b. Perfluorinated compounds in human breast milk from several Asian countries, and in infant formula and dairy milk from the United States. Environ. Sci. Technol. 42, 8597–8602.

Thompson, J., Eaglesham, G., Mueller, J., 2011. Concentrations of PFOS, PFOA and other perfluorinated alkyl acids in Australian drinking water. Chemosphere 83, 1320–1325.

Thomsen, C., Haug, L.S., Stigum, H., Froshaug, M., Broadwell, S.L., Becher, G., 2010. Changes in concentrations of perfluorinated compounds, polybrominated biphenyl ethers, and polychlorinated biphenyls in Norwegian breast-milk during twelve months of lactation. Environ. Sci. Technol. 44, 9550–9556.

Tittlemier, S.A., Pepper, K., Seymour, C., Moisey, J., Bronson, R., Cao, X.-L., Dabeka, R.W., 2007. Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. J. Agric. Food Chem. 55, 3203–3210.

Tomy, G.T., Tittlemier, S.A., Palace, V.P., Budakowski, W.R., Braekevelt, E., Brinkworth, L., Friesen, K., 2004. Biotransformation of N-ethyl perfluorooctanesulfonamide by rainbow trout (Onchorhynchus mykiss) liver microsomes. Environ. Sci. Technol. 38, 758–762.

Trier, X., Granby, K., Christensen, J.H., 2011. Polyfluorinated surfactants (PFS) in paper and board coatings for food packaging. Environ. Sci. Pollut. Res. Int. 18, 1108–1120.

Trudel, D., Horowitz, L., Wormuth, M., Scheringer, M., Cousins, I.T., Hungerbuhler, K., 2008. Estimating consumer exposure to PFOS and PFOA. Risk Anal. 28, 251–269.

Vassiliadou, I., Costopoulou, D., Kalogeropoulos, N., Karavoltsos, S., Sakellari, A., Zafeiraki, E., Dassenakis, M., Leondiadis, L., 2015. Levels of perfluorinated compounds in raw and cooked Mediterranean finfish and shellfish. Chemosphere 127, 117–126.

Vestergren, R., Berger, U., Glynn, A., Cousins, I.T., 2012. Dietary exposure to perfluoroalkyl acids for the Swedish population in 1999, 2005 and 2010. Environ. Int. 49, 120–127.

Vestergren, R., Herzke, D., Wang, T., Cousins, I.T., 2015. Are imported consumer products an important diffuse source of PFASs to the Norwegian environment? Environ. Pollut. 198, 223–230.

Wang, T., Wang, Y., Liao, C., Cai, Y., Jiang, G., 2009. Perspectives on the inclusion of perfluorooctane sulfonate into the Stockholm convention on persistent organic pollutants. Environ. Sci. Technol. 43, 5171–5175.

Wang, J., Shi, Y., Pan, Y., Cai, Y., 2010a. Perfluorinated compounds in milk, milk powder and yoghurt purchased from markets in China. Chin. Sci. Bull. 55, 1020–1025.

Wang, Y., Fu, J., Wang, T., Liang, Y., Pan, Y., Cai, Y., Jiang, G., 2010b. Distribution of perfluorooctane sulfonate and other perfluorochemicals in the ambient environment around a manufacturing facility in China. Environ. Sci. Technol. 44, 8062–8067.

Wang, I.J., Hsieh, W.-S., Chen, C.-Y., Fletcher, T., Lien, G.-W., Chiang, H.-L., Chiang, C.-F., Wu, T.-N., Chen, P.-C., 2011a. The effect of prenatal perfluorinated chemicals exposures on pediatric atopy. Environ. Res. 111, 785–791.

Wang, T., Lu, Y., Chen, C., Naile, J.E., Khim, J.S., Park, J., Luo, W., Jiao, W., Hu, W., Giesy, J.P., 2011b. Perfluorinated compounds in estuarine and coastal areas of north Bohai Sea, China. Mar. Pollut. Bull. 62, 1905–1914.

Wang, P., Lu, Y., Wang, T., Fu, Y., Zhu, Z., Liu, S., Xie, S., Xiao, Y., Giesy, J.P., 2014. Occurrence and transport of 17 perfluoroalkyl acids in 12 coastal rivers in south Bohai coastal region of China with concentrated fluoropolymer facilities. Environ. Pollut. 190, 115–122.

Wang, T., Wang, P., Meng, J., Liu, S., Lu, Y., Khim, J.S., Giesy, J.P., 2015. A review of sources, multimedia distribution and health risks of perfluoroalkyl acids (PFAAs) in China. Chemosphere 129, 87–99.

Wu, Y., Wang, Y., Li, J., Zhao, Y., Guo, F., Liu, J., Cai, Z., 2012. Perfluorinated compounds in seafood from coastal areas in China. Environ. Int. 42, 67–71.

Wu, M., Sun, R., Wang, M., Liang, H., Ma, S., Han, T., Xia, X., Ma, J., Tang, L., Sun, Y., Xu, G., 2017. Analysis of perfluorinated compounds in human serum from the general population in Shanghai by liquid chromatography-tandem mass spectrometry (LC-MS/MS). Chemosphere 168, 100–105.

Xu, Z., Fiedler, S., Pfister, G., Henkelmann, B., Mosch, C., Volkel, W., Fromme, H., Schramm, K.W., 2013. Human exposure to fluorotelomer alcohols, perfluorooctane sulfonate and perfluorooctanoate via house dust in Bavaria, Germany. Sci. Total Environ. 443, 485–490.

Yeung, L.W.Y., So, M.K., Jiang, G.B., Taniyasu, S., Yamashita, N., Song, M.Y., Wu, Y.N., Li, J.G., Giesy, J.P., Guruge, K.S., Lam, P.K.S., 2006. Perfluorooctanesulfonate and related fluorochemicals in human blood samples from China. Environ. Sci. Technol. 40, 715–720.

Yoo, H., Washington, J.W., Jenkins, T.M., Ellington, J.J., 2011. Quantitative determination of perfluorochemicals and fluorotelomer alcohols in plants from biosolid-amended fields using LC/MS/MS and GC/MS. Environ. Sci. Technol. 45, 7985–7990.

Zhang, T., Sun, H.W., Wu, Q., Zhang, X.Z., Yun, S.H., Kannan, K., 2010. Perfluorochemicals in meat, eggs and indoor dust in China: assessment of sources and pathways of human exposure to perfluorochemicals. Environ. Sci. Technol. 44, 3572–3579.

Zhang, D.-Y., Shen, X.-Y., Xu, X.-L., 2011a. The effects of perfluorooctane sulfonate (PFOS) on Chinese cabbage (Brassica rapa pekinensis) germination and development. In: Zhu, R. (Ed.), Second Sree Conference on Chemical Engineering.

Zhang, T., Sun, H., Lin, Y., Wang, L., Zhang, X., Liu, Y., Geng, X., Zhao, L., Li, F., Kannan, K., 2011b. Perfluorinated compounds in human blood, water, edible freshwater fish, and seafood in China: daily intake and regional differences in human exposures. J. Agric. Food Chem. 59, 11168–11176.