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Review

Per- and polyfluoroalkyl substances in food and their contribution to human exposure

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Exposure to per- and polyfluoroalkyl substances (PFAS) and how to reduce exposure is worldwide debated, leads to new regulations and advice for food consumption. PFAS are a complex class of chemicals. These so-called 'forever chemicals' are ubiquitous in the environment and consumer products, enter the food chain, and are detected in human bodies worldwide. Human PFAS serum concentrations are associated with dietary intake of certain foods. Seafood and offal consumption, packaged and wrapped foods, and food grown in contaminated areas are often associated with higher serum PFAS levels, while the reverse holds for more plant-based and more fibers containing diets. In contaminated areas, consumption of some local foods should be discouraged. However, for effective reduction of human exposure through food, more information is needed on sources and transfer of PFAS into agricultural products and processed foods, not only for legacy PFAS but also for short-chain PFAS and their precursors.

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Introduction

Per- and polyfluoroalkyl substances (PFAS) are synthetic compounds containing at least one fully fluorinated methyl or methylene carbon (C) atom [1]. Various chemical groups may be attached to the carbon skeleton, which may be of variable length and linear or branched. PFAS are water-, oil-, and dust-repellent. They have been produced since the 1950s and are used in textiles, food packaging, cookware, electroplating, firefighting foams, pesticides, cosmetics, and many other applications [2]. The strong chemical bond between the carbon and fluorine atoms makes PFAS highly resistant to environmental degradation. Thousands of PFAS molecules with different molecular structures and physico-chemical properties have been identified. The length of the C-chain and the functional groups are important drivers of environmental fate. The shorter-chain (i.e. C4–C6) carboxylates such as perfluorobutanoic acid (PFBA), perfluoropentanoic acid, and perfluorohexanoic acid (PFHxA) are more water-soluble and mobile in the environment, whereas the longer-chain sulfonates such as perfluorohexane sulfonic acid (PFHxS) and perfluorooctane sulfonic acid (PFOS) are more hydrophobic and more readily absorbed to soil organic matter. The branched isomers appear more mobile. Short-chain sulfonate perfluorobutane sulfonic acid (PFBS) and long-chain carboxylate perfluorooctanoic acid (PFOA) have intermediate behavior. Only since the early 2000s have analytical methods become widely available, mainly for long-chain PFAS (i.e. C7–14 compounds) such as PFOS and PFOA. They are ubiquitous in the environment and in biota including humans [3]. PFAS are emitted into the air and wastewater and contaminate soils near industries that produce or use fluorochemicals, near waste disposal sites, at firefighting sites, or where contaminated sludge is used as fertilizer. PFAS are always present as mixtures with different compositions depending on their source and environmental behavior. Environmental concentrations, exposure pathways, and toxicological effects are largely unknown for most of the compounds [4]. Robust analytical methods that allow accurate quantification are available for only about 50 of the thousands of PFAS molecules. The toxicology of PFOS and PFOA is the most well-studied. PFAS exposure is associated with immune effects, reduced birth weight and preterm birth, endocrine effects on thyroid function, osteoporosis, overweight, obesity, and dyslipidemia [5]. The International Agency for Research on

Cancer has classified PFOA as a known carcinogen (testicular and kidney cancer) [6].

The European Food Safety Agency (EFSA) has identified decreased serum antibody concentrations after vaccination in one-year-old children as the most sensitive non-cancer health outcome associated with PFAS exposure. Based on this critical effect, EFSA derived a maximum tolerable weekly Intake (TWI) of 4.4 ng/kg body weight (bw) for the sum of PFOS, PFOA, perfluorononanoic acid (PFNA), and PFHxS [7]. This manuscript focuses on information from recent human biomonitoring (HBM) studies that contribute to and complement existing knowledge on PFAS contamination of food and PFAS intake through food. We discuss the main trends published and reviewed in recent literature [8], as well as the results of our own European [9] and Flemish HBM studies [10] that allowed us to quantify and interpret the strength of the association between serum PFAS levels and dietary information obtained from questionnaires. We also address the limitations of HBM studies and the need for additional data.

Human exposure assessment

Estimated intake from food

Food is considered the predominant source of long-chain PFAS exposures for the general population. Measured concentrations of PFAS in 11,528 different foods sampled in the EU between 2000 and 2016 were combined with food consumption data from 38 different dietary surveys to estimate dietary intake of four PFAS (PFOS, PFOA, PFNA, and PFHxS). The estimated mean dietary intake for adolescents and adults ranged from 3 to 22 ng/kg bw/week, with the 95th percentile ranging from 9 to 70 ng/kg bw/week, which is above the TWI of 4.4 ng/kg bw/week. Toddlers and 'other children' showed a twofold higher intake. The four PFAS accounted for about half of the dietary exposure to those PFAS for which occurrence data were available, with the remaining contribution coming primarily from PFBA and PFHxA, two PFAS with short half-lives [7]. Based on occurrence data and the amount of food consumed, EFSA concluded that for the general population, fish and other seafood were the most important contributors to the mean PFOS and PFOA exposure, followed by eggs and egg products, meat and meat products, and, for PFOA, also fruit and fruit products, vegetables and vegetable products, and drinking water. It is a challenge to convert external exposure to internal serum or blood concentrations due to the amphoteric characteristics of PFAS. Biokinetic parameters such as chemical half-lives in the human body, absorption efficiency, binding affinities, and distribution volumes in the body are lacking for most PFAS and introduce large uncertainties to estimate the actual body burden from intake data [11].

Human biomonitoring

Alternatively, HBM measures the concentrations of PFAS in blood or serum and reflects the actual body burden. PFOS, PFOA, PFNA, and PFHxS are widely detected in human blood samples, including those of susceptible populations such as pregnant women [9,12]. Despite decreasing serum concentrations of regulated long-chain PFOS and PFOA over the last two decades, a fraction of the population exceeds the health-based guidance values for serum concentrations. This is in line with the EFSA intake estimates, that a fraction of the population exceeds the TWI. Recently, the European HBM4EU project (2014–2021) has obtained harmonized HBM PFAS data from 1957 teenagers (12–18 years) from nine European countries [13]. Serum PFAS levels exceeded the critical value of 6.9 μg/L in 14.3% of teenagers, rising to 24% in teenagers from Northern and Western Europe [9].

HBM studies often collect individual information from participants on diet, lifestyle, environment, and socio-demographic characteristics. By regressing biomarker concentrations against potential exposure sources and analyzing the strength of the association, the importance of different exposure sources can be assessed, as reviewed recently [14]. This adds complementary information to estimates based on intake values. The results can be stratified according to age, sex, and residence area. The statistical models can be adjusted for covariates such as body mass index and socio-economic status that may also influence the serum PFAS concentrations. However, there are some limitations. The studies are heterogeneous. They differ in methods to evaluate dietary intake [15]. Most often, food frequency questionnaires or dietary recall studies are used, and recall bias cannot be excluded. They span different time periods. Recent consumption data is more suitable for short-lived compounds such as short-chain PFAS, whereas consumption data from longer periods is better suited for long-chain PFAS, which bioaccumulate in the body over a lifetime. The number and selection of PFAS serum biomarkers S vary between studies. Most biomarker data are available for legacy PFAS, rather than short-lived PFAS and their precursors. The quantification limits for many non-legacy compounds in serum differ between laboratories and remain too high for routine analysis. Quality assurance methods for linear forms of legacy compounds are widely used in analytical laboratories, but no standards are yet available for branched compounds [16]. We observe that their contribution varies among study populations, depending on the source of contamination. Further, most HBM studies are cross-sectional, and it should be emphasized that association studies do not prove causality.

Consumption of animal-based foods

In European teenagers (HBM4EU) consumption of *seafood and fish* at least twice a week was significantly

Table 1

Studies reporting significant associations ($p < 0.05$) between serum concentrations of individual PFAS and dietary consumption of specific food categories.

Food items	Compounds	Study population (N)	Reference
Fish and seafood	↑PFOS, PFOA, PFHxS, PFNA	EU Pregnant women (5897)	[12]
	↑PFOS, PFNA	HBM4EU adolescents (1957)	[9]
	↑PFOS, PFOA, PFNA, PFDA, PFUnDA, PFHxS, PFDoA, ∑PFCA, ∑PFSA	Shanghai birth cohort pregnant women (2545)	[38]
	↑PFOS, PFNA, PFDA, ∑PFAS	US NHANES 2003–2014, > 12 yrs (2788)	[39]
	↑PFNA,PFDA, PFUnDA	Swedish adolescents (1098)	[40]
Offal	↑PFOS, PFHxS,PFNA	EU Pregnant women (5897)	[12]
	↑PFOS	HBM4EU adolescents (1957)	[9]
	↑PFOS, PFOA	Shanghai birth cohort (2545)	[38]
	↑PFOS	HBM4EU adolescents (1957)	[9]
Meat	↑PFOS, PFOA, PFHxS	Pregnant women (5897)	[12]
	↑PFNA	Swedish adolescents (1098)	[40]
Meat other than poultry	↑PFOS	US NHANES 2013–2014, 3–11 yrs (639)	[41]
Eggs	↑PFOS, PFNA	Pregnant women (5897)	[12]
	↑PFOS, PFNA	HBM4EU adolescents (1957)	[9]
	↑PFHxS	Shanghai birth cohort (2545)	[38]
	↑PFOS, PFHxS	Swedish adolescents (1098)	[40]
Local food	↑PFOS	HBM4EU adolescents (1957)	[9]
Fruit and juices	↑PFHxS	US NHANES 2013–2014 3–11 yrs (639)	[41]
Sugary drinks	↑PFNA	Norwegian adolescents 15–19 yrs (940)	[42]
Canned food	↑PFHxS	Norwegian adolescents 15–19 yrs (940)	[42]
Pizza, hamburgers, and sausages	↑PFNA	Norwegian adolescents 15–19 yrs (940)	[42]
Packaged food	↑MeFOSAA, PFOS	Boston-area children (548)	[43]
Popcorn	↑PFOA, PFNA, PFDA, PFOS, ∑PFAS	US NHANES 2003–2014 > 12 yrs(2788)	[39]
Ultra-processed foods	↑PFHxS, ↓PFDA,↓PFUnDA	US NHANES, 2003–2015 ≥12-yrs (11,530)	[44]
Diary products	↓PFOS, PFOA,PFHxS,PFNA	Pregnant women (5897)	[12]
	↓PFOA, PFOS, PFNA, PFDA, PFHxS, PFUnDA	Swedish adolescents (1098)	[40]
	↑PFNA, ↑PFDA	US pregnancy cohort (509)	[45]
More vegetables	↓PFNA, PFDA, PFUnDA	Shanghai birth cohort (2545)	[38]
	↓PFOS, PFNA	36 vegans versus 39 omnivores (Germany)	[46]
	↑PFNA, PFUnDA	Swedish adolescents (1098)	[40]
More fruit	↓ PFDoA	Shanghai birth cohort (2545)	[38]
	↓PFOA, PFOS, PFHxS, PFNA	Cincinnati, adolescents (193)	[47]
Dietary fiber intake	↓ PFOS,PFOA,PFNA,PFHxS	Cincinnati, adolescents(193)	[47]
	↓ PFOS,PFOA,PFNA	Adults NHANES, 2005–2016 (6482)	[27]

N: number of participants in the study. ↑: Positive associations, ↓: Negative associations.

associated with 21% (95% CI: 12–31%) increase in PFOS concentrations and 20% (95% CI: 10–31%) increase in PFNA concentrations compared to less frequent consumption of seafood and fish. A similar trend for PFOA and PFHxS was not statistically significant. Consumption of *eggs* at least twice a week was associated with an 11% (95% CI: 2–22%) and 14% (95% CI: 2–27%) increase in PFOS and PFNA concentrations, respectively, compared to less frequent consumption of eggs. Significantly higher PFOS concentrations were observed in participants who consumed *offal* 14% (95% CI: 3–26%) [9]. Table 1 provides examples of recent studies with large sample sizes that have correlated serum concentrations of PFAS with food consumption data. This contributes to recent systematic evidence mapping of 103 European, American, and Asian studies that reported significant associations in covariate-adjusted models between measured concentrations in biomatrices, dietary information, and product use [8]. Some general patterns emerge. Consumption of fish, seafood, offal, and eggs was, in most studies, associated with higher serum concentrations of PFAS. This is consistent with median PFOS and PFOA levels in fish, meat, and eggs, which are higher than in other food categories when expressed per kg of weight (Table 2, extracted from the EFSA 2021 report). Fish and seafood contain

Table 2

Range of mean LB PFAS concentrations in samples of specific food categories.

Food category	PFOS	PFOA	PFNA	PFHxS
Fish and seafood	0.16–14.12	0.000–4.10	0.000–0.98	0.00–0.066
Offal (farmed animals)	0.87	0.092	0.087	0.014
Meat livestock	0.03	0.028	0.000	0.000
Eggs	0.27	0.106	0.000	0.000
Dairy (Milk, Cheese)	0.001	0.001	0.000	0.000
Fruits	0.027	0.009	0.011	0.022
Vegetables	0.003	0.006	0.001	0.000

Source: EFSA CONTAM Panel (EFSA Panel on Contaminants in the Food Chain) Schrenk et al. (2020) [7].

LB (lower bound): samples with no detected levels are set equal to zero.

the highest levels. In the aquatic food chain, long-chain PFAS are more bio-amplified and enriched than short-chain PFAS, such as PFBS and PFHxS. The latter are rapidly metabolized, and concentrations in fish are generally lower than in water. Significant differences in concentrations are observed between species, which may relate to their trophic level, the protein content of blood and tissues, and their habitat. In general, higher concentrations are measured in carnivorous than in omnivorous fish, in benthic than in pelagic fish, and in lean compared to fatty fish [17,18]. Edible aquatic products such as shrimps, crabs, and fish can bioconcentrate PFAS by one to two orders of magnitude above dissolved concentrations in water [18].

Terrestrial animals are mainly exposed through contaminated water and feed grown on polluted soil. Fluorinated precursors in sediments can be a source of long-chain PFAS in biota [19]. In livestock and poultry, long-chain PFAS concentrations are higher in liver and blood than in muscle and other tissues [14]. This has implications for the consumption of offal products, which may pose a risk. Poultry eliminates PFAS more rapidly than mammals; its meat contains lower average levels. PFAS migrate into eggs, particularly the yolk, which may therefore contain high levels [17]. This may explain why consumption of eggs is often associated with increased serum levels of legacy PFAS in HBM studies.

Lower serum PFAS concentrations may also be associated with specific food categories.

Higher consumption of milk and dairy products is generally associated with lower serum PFAS concentrations. As shown in Table 2, median levels of long-chain PFAS are lower in milk products than in meat, offal, or eggs. However, PFAS is transferred to milk in cows that consume contaminated feed or drinking water. The carry-over rate was higher for PFOS than for PFOA and PFBS [17]. This is due to the greater hydrophobicity of long-chain PFAS and the greater protein-binding affinity of sulfonic acids compared to sulfonamides and carboxylic acids. Linear PFAS accumulate more than their branched isomers [14]. Some studies found a positive association between milk and dairy consumption and serum levels. This may be due to local environmental contamination of the food chain.

Consumption of plant-based foods

Consuming more plant-based foods and fruit is generally associated with lower levels of long-chain PFAS in the blood. As shown in Table 2, median levels of these compounds in plants are very low, with many samples falling below the level of detection. This results in significant uncertainty regarding estimated intake levels from vegetables and fruits. However, people can consume large amounts of fruit and vegetables. Passive uptake and uptake via transporter proteins in plants have been described. Short-chain PFAS such as PFBA have the highest potential for transfer from soils and can be quantified in plant-based foods [20,21]. They are found in all parts, including the above-ground parts, with higher concentrations in vegetables than in fruits [22]. Often, PFBA has the highest bioaccumulation factor (concentration in plant/concentration in soil) as shown in greenhouse and field experiments [22,23]. Long-chain PFAS such as PFOS tend to accumulate in roots (e.g. radish, carrot, and potato). Branched isomers seem to have higher potential for environmental transport and bioaccumulation than their linear counterparts [24]. Protein-rich vegetables appear to have higher bioaccumulation factors [20]. Annual crops (fruits, leafy vegetables, and legumes) that grow rapidly have higher concentrations of summed PFAS than perennial crops [22]. Foliar uptake from airborne PFAS and precursors can also occur.

Agricultural crops can also be contaminated with PFAS through pesticide application. A recent US study reported that 30% of pesticide active ingredients registered in the last 10 years contain chemicals that meet the definition of PFAS [25]. Active ingredients are often fluorinated to contribute to molecular stability in the target organism, while fluorination of inert ingredients or adjuvants can ensure efficient spraying or better uptake by target organisms.

Several HBM studies (Table 1) and experimental animal studies [26] have associated fiber-rich food intake with lower serum PFOS levels. It has been hypothesized that the transit of PFOS through the gastrointestinal tract is accelerated by binding to soluble dietary fibers, shielding it from reabsorption [27].

Local food consumption

While consumption of locally produced and home-grown food is generally well regarded as more environmentally friendly and part of sustainability, HBM studies have shown that it is ultimately necessary to grow food or raise livestock in a clean environment.

European teenagers of the HBM4EU aligned studies which reported *local food consumption* at least twice a week had 40% (95% CI: 19–64%) higher serum PFOS levels compared to teenagers who consumed local food less frequently [9]. Mothers who resided in Flanders and consumed locally produced vegetables more than five times a week had 24% (95% CI: 5–46%) higher PFOS levels compared to mothers who did not consume locally produced vegetables. This signals the general contamination of the environment with PFAS. Recent mapping of tens of thousands of PFAS-contaminated sites in Europe[1] and industrial sites with suspected PFAS emissions in the USA[2] has revealed an alarming number of communities at increased risk of PFAS exposure through environmental contamination of food grown in the area. A study in Belgium near a fluorochemical plant found high levels of contamination in homegrown eggs from free-range laying hens, with PFOS levels declining steeply with distance from the fluorochemical plant [28]. A similar observation was made in China, suggesting that eggs are an important route of human exposure in contaminated areas [29]. Bioaccumulation of mainly short-chain PFAS in wheat and maize grain was observed with decreasing concentrations as the distance to the fluorochemical industrial park increased [30]. In both freshwater and marine environments, wild-caught fish consistently show higher concentrations of PFOS and PFOA than farmed fish [17]. Offal of game animals may contain PFOS levels up to 250 times higher than those of farmed animals [7]. These studies form the basis of the guidance issued by various local authorities to restrict the consumption of locally produced food, such as eggs, or the consumption of locally caught fish in areas with high levels of contamination [25].

Contamination of packaged and processed foods

HBM studies have also associated c*onsumption of packaged foods and junk foods* with elevated serum PFAS concentrations. This is expected as we know that PFAS have been detected in a wide range of *food contact materials* (FCM). Trays, bowls, straws, cups, and take-away boxes are coated with PFAS that act as a water/oil barrier [31]. Plastic FCM can also contain PFAS that are unintentionally added or formed during polymerization. Another source of PFAS in food is the use of coatings in non-stick cookware. Most of the measured PFAS released from FCM are perfluorocarboxylic acids and fluorotelomer-based compounds [32]. Migration into food and food simulants has been observed from paper-based materials and from polytetrafluoroethylene-coated pans [33]. Also, the home environment may be a source of food contamination as PFAS are measured in dust, indoor air, on surfaces, in tap water, and clothing [34]. Transfer factors to food are however not known.

Association of serum PFAS concentrations with consumption of *processed foods* is not clear and not well understood. Processed food is often packaged as discussed above. But food preparation and cooking practices may also lower the PFAS content of food. PFAS levels in vegetables decreased by 6.40–89.8% after steaming, mainly due to changes in short-chain PFAS (especially PFBA). Loss of protein content and volatilization of short-chain PFAS may explain this. Concentrations of PFOS and PFOA increased, which may be formed from degraded precursors during steaming [24]. Cooking fish and seafood reduced PFAS concentrations by an average of 29% [35]. In most cases, raw food items contained higher levels of PFAS than the processed or cooked foods, possibly due to ingredients added to the composite product. A recent study as part of the US total diet study analyzed 172 nationally collected samples of processed foods. PFAS were detected in canned tuna (PFDA), fish sticks (PFOS and PFNA), and protein powder (PFOS), all at levels below 0.15 μg/kg [36]. Little information is available on the effects of PFAS contamination in water used for food production. A study from the Netherlands reported that PFAS contamination in coffee and cola production was mainly due to water contamination [37]. Specific transfer factors for PFAS from water to food are lacking. In addition, contaminated water used to prepare food may become a fraction of the prepared food. Examples include canned soups and vegetables, pickles, jams, fruit jellies, and juices.

Conclusion

The HBM studies offer a complementary view to what we learn from food occurrence data and from intake estimates. The serum PFAS levels of a fraction of the population are currently too high. Interpretation of the associations between serum levels and consumed foods may give indications on how to lower internal exposure by changes in dietary patterns and food consumption. Although the designs and characteristics of the populations and measured PFAS differ between studies, general trends emerge. They emphasize the need to further control environmental emissions and pathways of PFAS into agricultural products such as fish and seafood, meat, and eggs. Sources and behavior of PFAS during food processing are largely unknown. Improved monitoring of PFAS in food is required for better

[1] 'Forever pollution': Explore the map of Europe's PFAS contamination.
[2] Interactive Map: Suspected industrial discharges of PFAS.

protection of the population, particularly in hotspot areas where advice may be needed on the consumption of local foods such as wild-caught fish, seafood, and locally produced eggs. Better data collection is needed, also in HBM studies, not only for long-chain legacy PFAS, but also for precursors and most short-chain PFAS, for which little or no data are available.

Data Availability

No data were used for the research described in the article.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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