PERFLUOROALKYL ACIDS AND THEIR PRECURSORS IN SWEDISH FOOD: THE RELATIVE IMPORTANCE OF DIRECT AND INDIRECT DIETARY EXPOSURE

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Introduction

Diet, drinking water, air, and dust have all been identified as human exposure pathways for perfluoroalkyl acids (PFAAs), although food has been suggested as the major direct exposure pathway for most PFAAs in the general population from Sweden and Norway[1,2]. Fish, meat, egg, and dairy products have been shown to be dominant food groups with respect to human exposure to PFAAs[1,2,3]. Whether diet, drinking water, air, and/or dust are important pathways for human exposure to PFAA precursors is not as well characterized. Polyfluoroalkyl phosphate esters (PAPs) have been detected in drinking water[4], while PAPs, fluorotelomer alcohols (FTOHs), perfluorooctane sulfonamides (FOSAs), and perfluorooctane sulfonamido ethanols (FOSEs) have been detected in dust and air (indoor and/or outdoor air)[5,6,7]. With respect to dietary exposure to precursors, up to ten diPAPs were found in target food samples packed in material containing PAPs obtained from the Swedish market[8], and FOSA, MeFOSAA and EtFOSAA were detected in herring from the Swedish west coast[9]. Both these studies provided information on precursors in specific food items, however, information on precursors in general diet is lacking.

The first aim of this study was to identify and quantify PFAAs and selected precursors in Swedish market basket samples. Year pools from 1999, 2005, and 2010 representing the general diet in Sweden as well as 12 individual food groups from 1999 were analyzed. The second aim was to estimate dietary intakes for the precursors and PFAAs, and assess the potential importance of indirect (i.e., precursor based) dietary human exposure to PFAAs.

Materials and methods

Food items were purchased at major grocery store chains in four large Swedish cities in 1999, 2005, and 2010. Each year, the food items were divided into 12 groups (see Table 1) and homogenates for each food group were prepared by mixing food items proportionally according to food consumption statistics. For 1999, all the 12 food groups were analyzed individually. Additionally, for all three sampling years, a homogenate was prepared by mixing proportional amounts of each food group according to consumption data for the respective year, and each year pool was analyzed in duplicate.

The extraction and clean-up of the samples was based on published methods[8]. Briefly, homogenized food basket samples (2.5-5 g) were spiked with labeled internal standards and extracted twice with 6 mL acetonitrile by sonication. The organic phase was concentrated and cleaned up on a SPE WAX cartridge (150 mg, 6 mL, Waters). Neutral compounds were eluted with 3 mL methanol (fraction 1), and ionic compounds were subsequently eluted with 4 mL of a solution of 1 % ammonium hydroxide in methanol (fraction 2). Both fractions were concentrated and filtered using centrifugal filters and $^{13}C_8$-PFOA and $^{13}C_8$-PFOS were added as recovery internal standards.

For all instrumental analyses separation was carried out on an Acquity UPLC system (Waters) equipped with a BEH C18 (50×2.1 mm, 1.7 μm particle size, Waters) analytical column. Mobile phases were (A) 95 % water and 5 % methanol and (B) 75 % methanol, 20 % acetonitrile, and 5 % water. Both mobile phases contained 2 mM ammonium acetate and 5 mM 1-methyl piperidine (1-MP). Connected to the UPLC system was a Xevo TQ-S triple quadrupole mass spectrometer (Waters) operated in negative ion electrospray ionization (ESI−) mode. The capillary voltage was set at 3.0 kV, and the source and desolvation temperatures were 150 and 350 °C, respectively. The desolvation and cone gas flow (nitrogen) were set at 650 and 150 L/h, respectively.

Food samples were analyzed for PFSAs ($C_{4, 6, 8, 10}$), PFOS precursors (FOSAs and FOSAAs), PFCAs (C_4-C_{14}), monoPAPs (4:2, 6:2, 8:2, 10:2), and diPAPs (4:2/4:2, 6:2/6:2, 8:2/8:2, 10:2/10:2). The samples were also analyzed for additional diPAPs for which no pure authentic standards were available. All analytical results are given on a wet weight basis.

Results and discussion

PFOS precursors in food samples

Of the monitored PFOS precursors, FOSA (linear and/or branched isomers), FOSAA, MeFOSAA and EtFOSAA were detected in the meat product, pastries, fish product, and/or egg food groups from 1999 (Table 1, Figure 1). FOSA concentrations were 10 pg/g in meat and egg food groups, while the concentration in fish was 495 pg/g. Concentrations of FOSAA, MeFOSAA, and EtFOSAA in the individual food groups ranged between 2 and 79 pg/g. The highest concentrations of the precursors were generally found in the fish products and eggs, which was also the case for PFHxS and PFOS. The PFOS + precursor patterns in meat, fish, and egg food groups from 1999 were all dominated by PFOS (58-89% of ΣPFOS + precursors on a molar basis), while in pastries the pattern was dominated by EtFOSAA (Figure 1). In the fish pattern, FOSA was the dominant precursor comprising 34%, while in meat and eggs EtFOSAA and FOSAA, respectively, were the major precursors.

In the year pools, a declining trend in the FOSA concentrations was observed, with a concentration of 7.7 pg/g in 1999, 2.6 pg/g in 2005, and 0.8 pg/g in 2010 (Table 1). MeFOSAA and EtFOSAA were detected in the 1999 year pool at 2.2 and 4.4 pg/g, respectively, and were below the detection limit in 2005 and 2010. The decline of PFOS precursors, and also PFOS itself, is probably a direct result of the phase out of these chemicals by the 3M Company in 2002. Since the PFOS concentration declined less rapidly over time compared to the precursors, the relative contribution of PFOS to ΣPFOS + precursors increased in the year pools between 1999 (82% on a molar basis) and 2010 (97%) (Figure 1). In all the year pools FOSA was the dominant precursor (and the only detected precursor in 2005 and 2010), however, with declining abundance in the pattern from 1999 (10%) to 2010 (3%).

Table 1. PFOS, ∑PFOS precursors, ∑PFCA, and ∑diPAP concentrations (pg/g) in12 individual food groups from 1999 and year pools from 1999, 2005, and 2010.

Food group	Year	PFOS	∑PFOS precursors	∑PFCA (C_6-C_{14})	∑diPAP
Dairy products	1999	5.9	<0.1	0.6	<0.01
Meat products	1999	156	49	46	7.0
Fats	1999	1.1	<0.1	4.9	0.4
Pastries	1999	2.2	13	6.5	5.0
Fish products	1999	842	620	437	659
Egg	1999	1078	137	133	16
Cereal products	1999	0.4	<0.1	0.3	5.3
Vegetables	1999	<0.1[1]	<0.1	16	2.4
Fruit	1999	0.1	<0.1	2.7	0.7
Potatoes	1999	0.3	<0.1	5.4	0.9
Sugar and sweets	1999	<0.1	<0.1	1.3	0.3
Soft drinks	1999	0.3	<0.1	4.0	2.1
Year pool	1999	64	14	20	16
Year pool	2005	32	2.6	27	16
Year pool	2010	26	0.8	32	7.8

[1] Value indicating the method limit of quantification (MLOQ).

DiPAPs in food samples

Up to 10 diPAPs were detected in the individual food groups with 6:2/6:2 diPAP being the dominant diPAP (monoPAPs were not detected in all the food samples) (Figure 1). With the exception of the dairy products, 6:2/6:2 diPAP was detected in all food groups with concentrations ranging between 0.2 (potatoes) and 428 pg/g (fish products). 6:2/8:2 and 8:2/8:2 diPAPs were also frequently detected, in 8 and 6 food groups, respectively, with concentrations ranging between 0.04 and 59 pg/g. The fish products contained not only the highest detection frequency amongst all food groups, but also the highest ∑diPAP concentration, i.e., 659 pg/g. The diPAP patterns in the individual food market basket groups were dominated by 6:2/6:2 diPAP in 8 food groups, while 8:2/8:2 diPAP dominated the pattern in the three remaining food groups (no diPAPs were detected in dairy products) (Figure 1).

In the three year pools, 6 diPAPs were consistently detected (4:2/6:2, 6:2/6:2, 6:2/8:2, 8:2/8:2, 6:2/10:2, and 10:2/10:2 diPAPs), with the highest concentrations of 6:2/6:2 diPAP (6 – 12 pg/g). In 1999 and 2005 the ∑diPAP concentrations were comparable (~16 pg/g), whereas in 2010 it was 8 pg/g. Concentrations of 6:2/6:2 diPAP showed a decline in the year pools from 1999 to 2010, whereas for 4:2/6:2, 6:2/8:2, 8:2/8:2, 6:2/10:2, and 10:2/10:2 diPAPs the highest levels were quantified in the 2005 year pool. For all year pools, the diPAP pattern was dominated by 6:2/6:2 diPAP (54-79%), followed by 8:2/8:2 (5-27%) and 6:2/8:2 diPAPs (8-9%) (Figure 1).

PFCAs were detected in all food groups and year pools (Table 1) with PFOA, PFNA, and PFUnDA as the dominant compounds. Among the individual food groups the highest ∑PFCA concentrations were found in fish and eggs, while in the three year pools the ∑PFCA concentrations were comparable (Table 1).

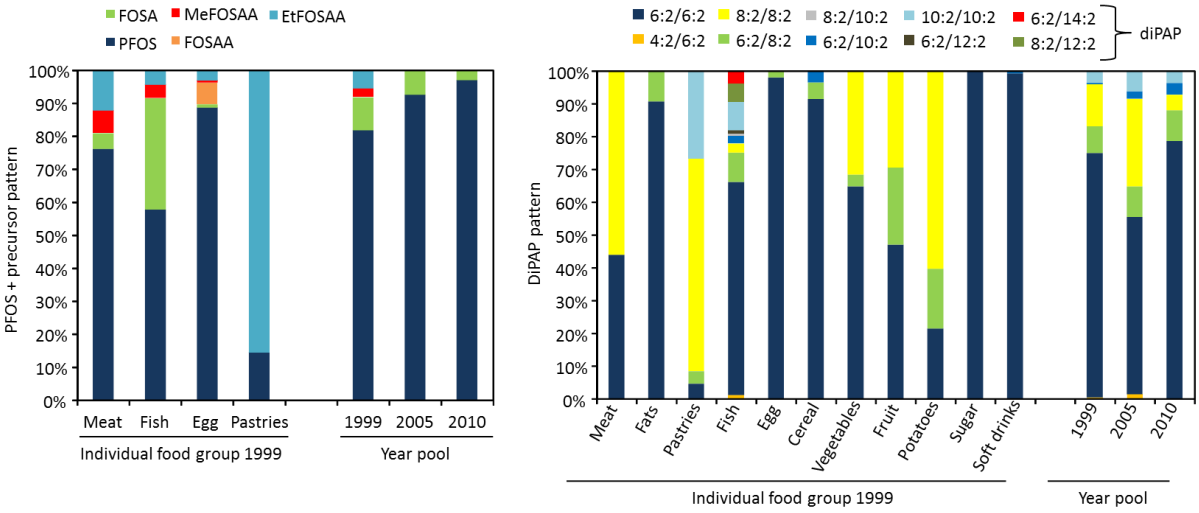

Figure 1. Relative abundance (%) of PFOS and its precursors (on a molar basis) (left plot) and diPAP concentrations (pg/g) (right plot) in individual food groups collected in 1999 and year food pools from 1999, 2005, and 2010.

Precursor dietary intakes in relation to PFAAs

The dietary intakes of total PFOS + precursors (precursor intakes converted to PFOS on a molar basis) were 1900, 930, and 750 pg/kg/d for 1999, 2005, and 2010, respectively (Figure 2A). Of these total intakes, the indirect PFOS intakes (i.e. potential maximum PFOS intake from analyzed precursors assuming 100% transformation to PFOS) contributed 18, 8, and 3% in 1999, 2005, and 2010, respectively. The relative importance of PFOS precursors in food as an indirect exposure source of PFOS has thus decreased over the studied time period.

Metabolism of diPAPs could be an indirect human exposure pathway for PFCAs after dietary intake of the diPAPs. In order to estimate the potential maximum contribution (assuming 100% biotransformation) of diPAPs

to the total dietary exposure to PFCAs, diPAP intakes were converted to their major PFCA metabolites on a molar basis. For example, dietary intake of one mole of 8:2/8:2 diPAP was converted to two moles of PFOA. Figure 2 shows the potential contribution of diPAP precursors to total (direct + indirect) dietary intakes of PFCAs with different chain lengths. Dietary intake of various diPAPs resulted in a maximum indirect dietary exposure to PFHxA of 245, 200, and 143 pg/kg/d in 1999, 2005, and 2010, respectively (Figure 2B). PFHxA itself was below detection limit in all three food pools. For PFOA, the potential contribution of diPAPs was variable among the studied years (Figure 2C). In 2005, up to 70% of the total PFOA dietary intake could have resulted from indirect exposure (diPAPs), whereas in 1999 and 2010 direct exposure was the major dietary source of PFOA. The total dietary intakes of PFOA (direct + indirect) were comparable among the years, ranging between 144 and 173 pg/kg/d. For PFDA, the majority of the total dietary intakes came from direct exposure to this PFCA (69-88% depending on the year). The total PFDA dietary intake (direct + indirect) based on the year pools ranged between 56 and 84 pg/kg/d.

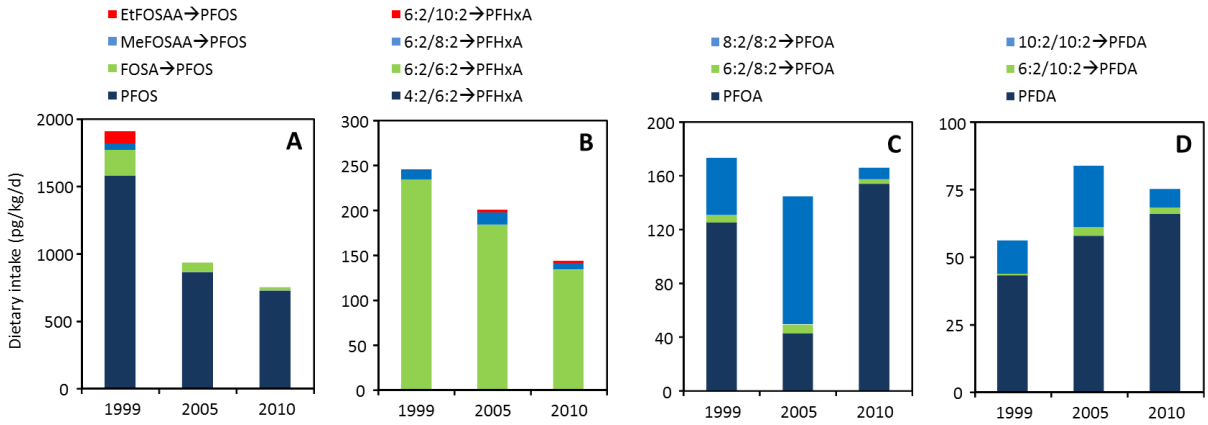

Figure 2. Contribution of precursors to dietary exposure intakes (pg/kg/d) of PFOS, PFHxA, PFOA, and PFDA in food pools from 1999, 2005, and 2010. Concentrations of precursors were converted to PFAA concentrations on a molar basis. PFHxA was below detection limit in the year food pools.

With the present study we show that several precursors to PFOS and PFCAs have been present in food samples representing the general diet of the average Swedish population in 1999, 2005, and 2010. With respect to dietary PFOS exposure, the analyzed precursors play a minor role compared to direct dietary PFOS intake, especially in more recent years. We further demonstrate that diet is a direct and an indirect pathway for human exposure to PFCAs. The relative importance of dietary diPAP exposure as an indirect source to PFCAs is chain length dependent.

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