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Time trends between 1987 and 2007 for perfluoroalkyl acids in plasma from Swedish women

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HIGHLIGHTS

- We investigated time trends between 1987 and 2007 in perfluoroalkyl acids.
- PFDA, PFNA, and PFUnDA increased during the period, but most markedly after 2000.
- PFOS, PFOA, and PFHxS peaked during the period 1990–2000.

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ABSTRACT

Perfluoroalkyl acids (PFAAs) are a large group of chemicals which are highly persistent in both nature and humans. The use of the most prominent ones, perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), was reduced in the early 21st century, and since then levels in human matrices have decreased. However, these two compounds have been exchanged by other PFAAs, for which time trends have not been as extensively investigated. By the use of 80 plasma samples collected between 1987 and 2007 from healthy women (n = 1–9 yearly for 1987–2001, n = 15 from 2006, and n = 10 from 2007), possible time trends of six PFAAs were assessed. Time trends were evaluated for the entire study period, as well as for three sub-periods. As seen in previous studies, levels of perfluorohexane sulfonate (PFHxS), PFOS, and PFOA peaked during the middle time period (1990–2000), with medians of 0.98 ng mL^{-1}, 18.06 ng mL^{-1}, and 3.73 ng mL^{-1}, respectively. However, levels of perfluorononanic acid (PFNA), perfluorodecanic acid (PFDA), and perfluoroundecanoic acid (PFUnDA) increased over the whole study period and most markedly so after year 2000, with medians of 0.73 ng mL^{-1}, 0.28 ng mL^{-1}, and 0.24 ng mL^{-1}, respectively, during the last study period.

1. Introduction

Perfluoroalkyl acids (PFAAs), of which perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are the most studied, constitute a large group of chemicals with an extensive use. These substances have been used for many years e.g. for impregnation of textiles, clothes, footwear, furniture and carpets. Other uses are in lubricants, floor and car waxes, paints and fire-fighting foam for oil fires.

The main exposure route for PFAAs for humans exposed to background levels only, is diet (Fromme et al., 2009; Vestergren and Cousins, 2009; Egeghy and Lorber, 2011; Haug et al., 2011). However, levels in indoor air and dust are also significant (Gewurtz et al., 2009; Harrad et al., 2010; Goosey and Harrad, 2012) making indoor environment an important contributor to exposure (Goosey and Harrad, 2011; Haug et al., 2011; Fraser et al., 2012).

Levels of PFAAs in human biological samples have been described in many studies. The exposure to humans tends to be higher in the Arctic, Europe, and Asia than in areas in the southern hemisphere (Kannan et al., 2004; Houde et al., 2011; Lindh et al., 2012). PFAAs have been found to be persistent in humans; The half-life for PFOA has been found to range between 2.3 and 8.5 years (Olsen et al., 2007; Bartell et al., 2010; Brede et al., 2010; Seals et al., 2011), whereas the half-life for PFOS has been estimated to approximately 5 years (Olsen et al., 2007).

Animal studies have found that exposure to PFAAs negatively impacts e.g. the immune and reproductive systems in several

Abbreviations: ANOVA, analysis of variance; PFAAs, perfluoroalkyl acids; PFBS, perfluorobutane sulfonate; PFBA, perfluorobutanoic acid; PFDS, perfluorodecane sulfonate; PFDA, perfluorodecanic acid; PFHpS, perfluoroheptane sulfonate; PFHpA, perfluoroheptanoic acid; PFHxS, perfluorohexane sulfonate; PFNA, perfluorononanoic acid; FOSA, perfluorooctane sulfonamide; PFOSA, perfluorooctane sulfonamide; PFOS, perfluorooctane sulfonate; PFOA, perfluorooctanoic acid; PFUnDA, perfluoroundecanoic acid.

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species (Lau et al., 2007; Jensen and Leffers, 2008; DeWitt et al., 2009; Sonne, 2010). Epidemiological studies on health effects in humans are limited and not consistent. However, there are suggestions that exposure to PFAAs may increase cholesterol, liver enzymes, diabetes mortality, and affect a range of reproductive parameters and indicators of fetal development (Steenland et al., 2010; Specht et al., 2012; Toft et al., 2012).

Several time trend studies have found that levels of PFOS, PFOA and perfluorohexane sulfonate (PFHxS) in human matrices increased until somewhere in the 1990ies when they reached a plateau and then started to decrease in the early 21st century (Harada et al., 2004, 2007; Olsen et al., 2005, 2007, 2008, 2011, 2012; Calafat et al., 2007; Jin et al., 2007; Haug et al., 2009; Wilhelm et al., 2009; Kato et al., 2011; Sundstrom et al., 2011; Glynn et al., 2012). This is consistent with the manufacturing phase-out of a PFOS and PFOA that was undertaken at the beginning of the 21st century by the 3M Company, a major producer of PFAAs.

The possible changes in levels of other PFAAs have not been investigated to the same extent as those in PFOS, PFOA and PFHxS. Studies that have been performed to date suggest that during the 21st century, perfluorobutanoic acid (PFBA), perfluoroheptane sulfonate (PFHpS), perfluorooctane sulfonamide (PFOSA), and perfluorodecane sulfonate (PFDS) perfluorooctane sulfonamide (FOSA) and perfluoroheptanoic acid (PFHpA) concentrations have decreased, whereas there has been an increase of perfluorobutane sulfonate (PFBS), perfluorononanoic acid (PFNA), perfluorodecanic acid (PFDA) and perfluoroundecanoic acid (PFUnDA) concentrations (Calafat et al., 2007; Haug et al., 2009; Kato et al., 2011; Olsen et al., 2011, 2012; Glynn et al., 2012).

Although the changes in levels of some PFAAs are well known today, there are others for which time trends are still not fully determined. In the present study, we use biobanked plasma samples to investigate possible time trends of PFHxS, PFOS, and PFOA, as well as some of the PFAAs for which only sparse previous literature exists, i.e. PFNA, PFDA, and PFUnDA.

2. Materials and methods

A total of 80 plasma samples were collected during 1987 through 2007 from healthy women born between 1934 and 1967. These women were sampled in connection with breast reduction surgery (1987–1991), at screening for hereditary genetic disease (but found to be without the disease-promoting gene; 1993–2001), or as wives of men with cancer (2006–2007). The samples were collected to be used as healthy controls. The women's mean age at the time of sampling was 48 years (min 36 and max 56). At least one sample per year, with the exception of 1992 and 2002–2005, was used (see Table 1). The numbers of samples collected during the winter (October–March; $n = 43$) and the summer (April–September; $n = 37$) were similar. All samples were stored in a freezer until analyses, which were done for levels of PFHxS, PFOS, PFOA, PFNA, PFDA and PFUnDA.

The chemical analyses of the PFAAs were performed by LC/MS/MS following precipitation of the proteins with acetonitrile according to a modified method by Midasch et al. (2007) (Lindh et al., 2012). ^{13}C-labeled internal standards were used. The limit of detection was set at 0.1 ng mL^{-1} plasma. The precision for the same sample analyzed nine times was for PFHxS 4% at 1 ng mL^{-1}, for PFOS 4% at 12 ng mL^{-1}, for PFOA 5% at 4 ng mL^{-1}, and for PFNA 5% at 0.8 ng mL^{-1}. The quality of the determinations was controlled by analyzing chemical blanks and in-house quality control samples. The analyses of PFOS and PFOA are part of the Round Robin inter-comparison program (Professor Dr. med. Hans Drexler, Institute and Out-Patient Clinic for Occupational, Social and Environmental Medicine, University of Erlangen-Nuremberg, Germany) with results within the tolerance limits.

Statistical analyses of possible time trends for the PFAAs were performed using analysis of variance (ANOVA). The different PFAAs were used as continuous variables, i.e. a linear trend was assumed. However, based on the results from previous studies, separate analyses were also performed for the time periods before 1990 (mean

Table 1
Median serum levels of perfluoroalkyl acids among 80 healthy Swedish women sampled in 1987 through 2007.

	N	PFHxS (ng mL^{-1})	PFOS (ng mL^{-1})	PFOA (ng mL^{-1})	PFNA (ng mL^{-1})	PFDA (ng mL^{-1})	PFUnDA (ng mL^{-1})
Calendar year of sampling							
1987	3	0.30	13.2	2.60	0.31	0.10	0.17
1988	8	0.65	21.3	3.72	0.32	0.12	0.19
1989	9	0.64	16.8	2.63	0.26	0.11	0.14
1990	4	0.81	16.8	1.78	0.41	0.14	0.20
1991	1	0.36	11.4	1.70	0.22	0.10	0.13
1993	1	0.99	18.5	4.90	0.28	0.05	0.11
1994	2	1.52	20.9	3.89	0.35	0.20	0.22
1995	4	0.68	22.1	4.64	0.31	0.14	0.20
1996	8	0.98	19.5	3.81	0.37	0.15	0.13
1997	3	0.88	17.4	2.97	0.39	0.17	0.15
1998	1	1.93	35.5	5.51	0.89	0.42	0.28
1999	2	0.80	17.3	2.50	0.22	0.14	0.10
2000	8	1.29	16.7	3.32	0.36	0.17	0.14
2001	1	0.71	12.1	2.22	0.36	0.11	0.05
2006	15	0.89	10.4	2.51	0.65	0.24	0.27
2007	10	0.93	10.2	2.93	0.87	0.35	0.24
Period of sampling							
Period 1: before 1990	24	0.65	17.40	2.80	0.30	0.11	0.17
Period 2: 1990–2000	30	0.98	18.06	3.73	0.36	0.15	0.15
Period 3: after 2000	26	0.82	10.84	2.54	0.73	0.28	0.24
Age at sampling (years)							
Less than 45	23	0.73	16.75	2.96	0.38	0.15	0.17
45–50	33	0.71	16.50	3.11	0.35	0.15	0.17
More than 50	24	1.11	17.11	3.09	0.50	0.19	0.19
Season at sampling							
Winter (October–March)	43	0.84	17.93	3.14	0.39	0.18	0.20
Summer (April–September)	37	0.71	15.76	2.60	0.36	0.15	0.15
Total	80	0.77	16.71	3.03	0.38	0.16	0.18