tra l'esposizione all'inquinante (nella fattispecie, l'esposizione all'inquinante attraverso l'acqua potabile) e l'insorgenza di patologie, ma anche i termini quantitativi attraverso cui questa esposizione debba essere valutata.

I dati sino ad ora in nostro possesso evidenziano dei possibili nessi di causalità tra l'esposizione a PFAS e vari tipi di patologie, come discusso in dettaglio nel paragrafo precedente, tra cui principalmente alcuni tipi di tumore, disordini del sistema endocrino, problemi cardiovascolari e disturbi della fertilità. I dati in letteratura non sono concordi né nell'elenco di queste patologie, né nei limiti quantitativi di esposizione con i quali l'insorgenza di queste patologie sarebbe correlata. In molti casi gli studi epidemiologici si concludono affermando che, sebbene vi siano sospette correlazioni, non si possono trarre conclusioni causa-effetto certe, e vi sono numerosi esempi in cui gli studi si contraddicono tra di loro, giungendo a conclusioni opposte.

Complessivamente, tuttavia, le ricerche e le indagini tossicologiche forniscono indicazioni sufficienti a suggerire la necessità di adottare misure di massima precauzione consistenti nel ridurre o annullare l'esposizione dei cittadini a questi inquinanti, anche in considerazione della loro spiccata tendenza ad accumularsi nell'ambiente e nell'organismo e dei lunghissimi tempi necessari per l'espulsione delle sostanze dall'organismo stesso una volta accumulate.

I limiti di presenza di PFAS nelle acque, come discusso nel paragrafo 2, sono stati definiti dalla normativa solo per alcuni di

questi inquinanti, mentre per altri sono suggeriti dei parametri di qualità ambientali, calcolati sulla base delle attuali conoscenze.

Sebbene non sia noto, a causa della frammentarietà dei dati, se questi limiti siano efficaci, sottostimati o sovrastimati, essi rappresentano al momento un importante parametro quantitativo a cui far riferimento per l'adizione di quelle misure precauzionali che le informazioni oggi in nostro possesso ci impongono di adottare.

Va sottolineato che la persistenza ambientale e la tendenza ad accumularsi nell'organismo per esposizioni prolungate, in combinazione con la sospetta associazione con l'insorgenza di alcune patologie, rappresentano i maggiori fattori di preoccupazione riguardo la presenza di queste sostanze nelle acque potabili e negli alimenti, anche in basse concentrazioni.

ALLEGATI

La relazione tecnica contiene tre allegati, che si ritiene utile inserire perché rappresentano fonti di approfondimento scientifico di quanto riportato in questo documento. Si tratta di rassegne di letteratura scientifica pubblicate su riviste internazionali, la cui consultazione consente di abbracciare in maniera abbastanza completa, ma al tempo stesso sintetica, lo stato dell'arte scientifico rilevante sull'argomento. Inoltre, le review contengono tutti i riferimenti bibliografici necessari per accedere alle letteratura scientifica specifica per tutti gli argomenti trattati.

Allegato 1 è una rassegna sugli studi epidemiologici riugardanti l'esposizione a composti perfluorurati di popolazioni nei paesi occidentali: "Perfluorinated compounds — exposure assessment for the general population in western countries" da H. Fromme, S.A. Tittlemier, W. Volkel, M. Wilhelm, D. Twardella Int. J. Hyg. Environ. Health (2009), 212:239–270.

Allegato 2 è una rassegna sulla tossicologia dei composti perfluorurati, "Toxicology of perfluorinated compounds" pubblicata da T. Sthal, D. Mattern and H.Brunn su Environmental Science Europe 2011 , 23:38.

Allegato 3 è un documento prodotto da ricercatori italiani che illustra il lavoro metodologico volto a definire gli *standard* di qualità ambientali per l'acqua potabile per PFOA e per gli altri acidi carbossilici perfluorurati a corta catena, per i quali la legislazione europea non fornisce indicazioni (S. Valsecchi, D. Conti, R. Crebelli, S. Polesello, M. Rusconi, M. Mazzoni, E. Preziosi, M. Carere, L.

Lucentini, E. Ferretti, S. Balzamo, M.G. Simeone, F. Aste “Deriving environmental quality standards for perfluorooctanoic acid (PFOA) and related short chain perfluorinated alkyl acids” *Journal of Hazardous Materials*, **2016**).

Available online at www.sciencedirect.com
ScienceDirect
Int. J. Hyg. Environ. Health 212 (2009) 239–270
International Journal of Hygiene and Environmental Health
www.elsevier.de/ijheh

REVIEW

Perfluorinated compounds – Exposure assessment for the general population in western countries

Hermann Fromme[a,*], Sheryl A. Tittlemier[b], Wolfgang Völkel[a], Michael Wilhelm[c], Dorothee Twardella[a]

[a]*Department of Environmental Health, Bavarian Health and Food Safety Authority, Veterinärstrasse 2, 85764 Oberschleissheim, Germany*
[b]*Food Research Division, Banting Research Centre 2203D, Health Canada, Ottawa, Ontario, Canada K1A 0L2*
[c]*Department of Hygiene, Social and Environmental Medicine, Ruhr-University Bochum, Universitätsstraße 150, 44801 Bochum, Germany*

Received 27 January 2008; received in revised form 19 March 2008; accepted 7 April 2008

Abstract

Perfluorinated compounds (PFCs) can currently be detected in many environmental media and biota, as well as in humans. Because of their persistence and their potential to accumulate they are of toxicological concern. The present review presents the current knowledge of PFC monitoring data in environmental media relevant for human exposure. In this context, PFC concentrations in indoor and ambient air, house dust, drinking water and food are outlined. Furthermore, we summarize human biomonitoring data of PFC levels in blood, breast milk, and human tissues. An estimate of the overall exposure of the general adult population is provided and compared with tolerable intake values.

Using a simplified model, the average (and upper) level of daily exposure including all potential routes amounts to 1.6 $ng/kg_{body\ weight}$ (8.8 $ng/kg_{body\ weight}$) for PFOS and 2.9 $ng/kg_{body\ weight}$ (12.6 $ng/kg_{body\ weight}$) for PFOA in adults in the general population. The majority of exposure can be attributed to the oral route, mainly to diet. Overall, the contribution of PFOS and PFOA precursors to total exposure seems to be limited.

Besides this background exposure of the general population, a specific additional exposure may occur which causes an increased PFC body burden. This has been observed in populations living near PFC production facilities or in areas with environmental contamination of PFCs. The consumption of highly contaminated fish products may also cause an increase in PFC body burdens.

Keywords: PFOS; PFOA; PFC; Biomarkers; Human biomonitoring; Indoor air; House dust

Abbreviation: PFBS, perfluorobutane sulfonate; PFBA, perfluorobutanoate; PFC, perfluorinated chemical; PFDA, perfluorodecanoate; PFDoDA, perfluorododecanoate; PFDS, perfluorodecane sulfonate; PFDA, perfluorodecanoate; PFHpS, perfluoroheptane sulfonate; PFHpA, perfluoroheptanoate; PFHxA, perfluorohexanoate; PFHxS, perfluorohexane sulfonate; PFNA, perfluorononanoate; PFOA, perfluorooctanoate; PFOS, perfluorooctane sulfonate; PFOSA, perfluorooctane sulfonamide; PFOSF, perfluorooctanesulfonyl fluoride; PFUnDA, perfluoroundecanoate; *N*-EtFOSE, *N*-ethyl perfluorooctane sulfonamidoethanol; *N*-MeFOSE, *N*-methyl perfluorooctane sulfonamidoethanol; *N*-EtFOSA, *N*-ethyl perfluorooctane sulfonamide; *N*-MeFOSA, *N*-methyl perfluorooctane sulfonamide; *N*,*N*-Et_2FOSA, *N*,*N*-diethyl perfluorooctane sulfonamide; *N*,*N*-Me_2FOSA, *N*,*N*-dimethyl perfluorooctane sulfonamide; 4:2 FTOH, 1H,1H,2H,2H-perfluoro-1-hexanol; 6:2 FTOH, 1H,1H,2H,2H-perfluoro-1-octanol; 8:2 FTOH, 1H,1H,2H,2H-perfluoro-1-decanol; 10:2 FTOH, 1H,1H,2H,2H-perfluoro-1-dodecanol.

*Corresponding author. Tel.: +49 8931 560231; fax: +49 8931 560835.
E-mail address: hermann.fromme@lgl.bayern.de (H. Fromme).

1438-4639/$ - see front matter © 2008 Elsevier GmbH. All rights reserved.
doi:10.1016/j.ijheh.2008.04.007

Contents

Introduction

Perfluorinated compounds (PFCs) represent a large group of chemicals which are characterized by a fully fluorinated hydrophobic linear carbon chain attached to various hydrophilic heads. The chemical structures of some important PFCs are given in Fig. 1. PFCs have been produced since the 1950s and are widely used for many industrial purposes and consumer-related applications. This is due to their unique physico-chemical characteristics such as chemical and thermal stability, low surface free energy and surface active properties (Hekster et al., 2003; Lehmler, 2005). The C–F bond is particularly strong, and is resistant to various modes of degradation, including reaction with acids and bases, oxidation, and reduction (Kissa, 2001). This resistance contributes to the extraordinary stability of PFCs. While some PFCs undergo chemical transformations, these reactions occur mainly at the hydrophilic portions of the molecule, as opposed to the perfluorinated alkyl chains. The most commonly studied PFC substances are the perfluorinated sulfonates and the perfluorinated carboxylates. Among these, perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) are of greatest concern. Both persist in humans and the environment.

PFOS, its precursors, and related compounds are used in many applications ranging from oil and water repellant coatings for carpets, textiles, leather, paper, cardboard, and food packing materials; electronic and photographic devices; and surfactants in diverse cleaning agents, cosmetics, and fire-fighting foams (OECD, 2002; Kissa 2001). PFOA, as its ammonium salt, is mainly used as an essential processing aid in the manufacture of certain fluoropolymers such as polytetrafluoroethylene (PTFE) and to a lesser extent in industrial applications as an antistatic additive and in the electronic industry (OECD, 2005).

There are two main processes used to commercially synthesize PFCs. PFOS, along with some other PFCs, are commercially synthesized by a process known as electrochemical fluorination (ECF), which uses an electric current to fully fluorinate organic feedstock dispersed in liquid hydrogen fluoride. During this non-selective process, the predominant perfluorinated alkyl chain

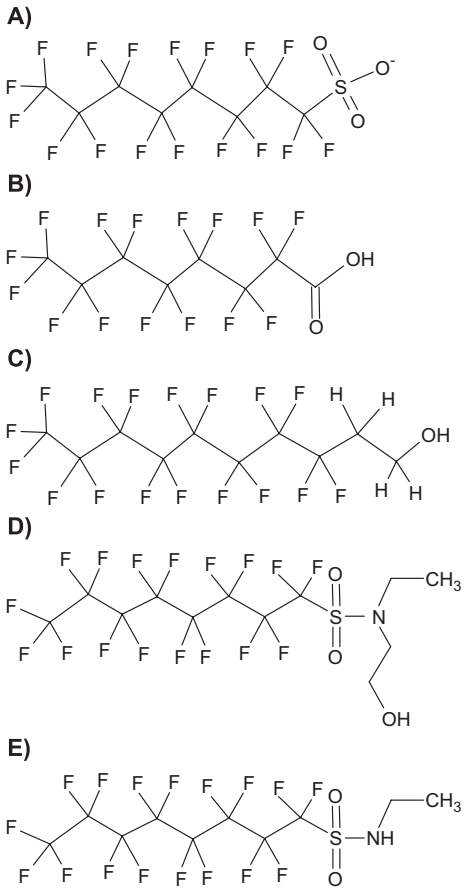

Fig. 1. Chemical structure of some typical perfluorinated substances. (A) Perfluorooctane sulfonate (PFOS), (B) perfluorooctanoate (PFOA), (C) 1-hydroxyethane-2-perfluorooctanol (8:2 FTOH), (D) *N*-ethyl perfluorooctane sulfonamidothanol (NEtFOSE), and (E) *N*-ethyl perfluorooctane sulfonamide (NEtFOSA).

length produced corresponds to the alkyl chain length of the organic feedstock used. However, other perfluoroalkyl homologues are also formed during ECF. For example, ECF of octanesulfonyl fluoride produces perfluorooctanesulfonyl fluoride (PFOSF) plus homologous sulfonyl fluorides and related fluorocarbons containing between 4 and 13 carbon atoms. Reaction by-products also include branched chain isomers. The resulting substances derived from various reactions with PFOSF, mainly perfluorooctane sulfonamides and perfluorooctane sulfonamide derivatives, are building blocks for different commercial perfluoroalkyl substances.

The other major commercially important process for PFC synthesis is telomerization. In this process, tetrafluoroethylene reacts with intermediate perfluoroalkyl iodides to form key compounds like fluoroalkyl silanes, carboxylates, acrylates and methacrylate polymers (Schultz et al., 2003). Branched chain isomers are not observed in the products formed by telomerization (Kissa, 2001).

The more persistent PFCs, such as PFOS and PFOA, can also be formed in the environment from abiotic and biotic transformation of commercially synthesized precursors. During ECF and subsequent commercial reactions, numerous substances such as perfluoroalkylsulfonamide alcohols were unintentionally produced, or remained as by-products in commercial products. Most of these substances can be converted in the ecosystem and in living organisms to persistent PFCs. For example, it has been demonstrated that perfluorooctane sulfonamides can be metabolized to PFOS (Xu et al., 2004; Tomy et al., 2004). It has to be noted that PFOS may therefore be the final degradation or metabolic product of many perfluorooctylsulfonyl substances (Hekster et al., 2003).

In addition, some precursors like fluorotelomer alcohols (FTOH) will be subsequently transformed into PFOA under environmental degradation processes (Ellis et al., 2004; Dinglasan et al., 2004; Wang et al., 2005). Furthermore, there is growing evidence from some studies that 8:2 FTOH is converted to PFOA after oral uptake in mice (Kudo et al., 2005; Henderson and Smith, 2007) and rats (Fasano et al., 2006; D'eon and Mabury, 2007). These findings were confirmed by in vitro studies using rat hepatocytes (Martin et al., 2005) and hepatocytes and microsomes from various species (Nabb et al., 2007) to study the metabolism of 8:2 FTOH.

From a regulatory point of view, PFOS is classified as very persistent, very bioaccumulative and toxic, thus fulfilling the criteria for being considered as a persistent organic pollutant under the Stockholm Convention (EU, 2006). In the European Union, the use of PFOS has been restricted and the PFOS Directive aims to end the use of all PFOS as soon as practical (EU, 2006). In particular, fire-fighting foams that have been placed on the market before 27 December 2006 can be used until 27 June 2011. Similar regulatory action has been taken in North America. In Canada, PFOS, its precursors, and salts are are being considered for addition to the list of Toxic Substances under the Canadian Environmental Protection Act 1999 (Government of Canada, 2006). This action would prohibit the manufacture, use, sale, offer for sale and import of PFOS, as well as manufactured items containing the perfluorooctylsulfonyl moiety. The United States Environmental Protection Agency (US EPA) has adopted federal Significant New Use Rules for PFOS and related substances for new manufacturers and new uses of these substances. These rules will allow the US EPA to evaluate any intended new uses, and subsequently restrict or prohibit these new uses.

In addition, one of the primary manufacturers of fluorinated chemicals in North America announced a

cease in production of perfluorooctanesulfonyl compounds in 2000. It was projected that from 2000 to 2002, the production of $C_8F_{17}SO_2$-containing compounds for US Food and Drug Administration-approved uses would decrease from 1,520,000 to 0 kg (US EPA 2002).

The toxicity of PFOS and PFOA has been studied extensively, mainly in rodents. Several reviews are available that discuss results from these studies (OECD, 2002; Kennedy et al., 2004; US EPA, 2005; Harada et al., 2005b; Andersen et al., 2008; Lau et al., 2007). Hepatotoxicity, developmental toxicity, immunotoxicity, hormonal effects and a carcinogenic potency are the effects of main concern. In contrast, epidemiologic data related to PFC exposure are limited. The data were collected mainly among PFC production plant workers and have not found consistent effects on morbidity and mortality in humans.

The persistence of PFCs in the environment, plus their potential to accumulate in organisms and to biomagnificate in the food chain is of particular toxicological concern. Several PFCs have been detected in nearly all environmental media and biota reflecting the widespread global pollution in all parts of the ecosystem (Giesy and Kannan, 2001). PFCs have also been detected in human blood and tissue samples from occupationally and non-occupationally exposed humans throughout the world. The persistence of certain PFCs may be a more relevant issue for humans versus other species. In contrast to investigations carried out in laboratory animals in which short half-lives of PFCs were observed, studies in retirees from PFC production facilities showed a mean elimination half-life of 3.8 years (PFOA) and 5.4 years (PFOS) (Olsen et al., 2007b). A widespread distribution of various PFCs and their corresponding degradation and metabolism products results in a very complex exposure situation. The contribution of single sources and pathways to the total exposure is currently not well defined.

The aim of this review was to compile in detail the current data available to define the environmental media responsible for human exposure to PFCs. For this purpose we used the results of different Medline inquiries to get an overwiew of the current scientific literature. We also included papers presented at conferences, reports from governmental, scientific and other institutions, and where possible, unpublished reports and other gray literature. In this context PFC concentrations in indoor and ambient air, house dust, drinking water, and food are outlined. Furthermore, we will summarize human biomonitoring data in blood, breast milk and human tissues. Current estimates of the overall exposure of the adult general population will also be addressed. All these data will be discussed in relation to present benchmark values used for risk assessment.

Environmental monitoring

For the assessment of human exposure to PFCs, different pathways have to be considered. Exposure via inhalation may result from outdoor air and indoor air PFC pollution, and from PFC in house dust. Oral exposure is mainly determined by contamination of food and drinking water. Ingestion of dust and soil due to hand-to-mouth activities may also contribute to the internal exposure for children. However, this paper will focus mainly on exposure pathways of adults. Data from PFC monitoring in environmental samples are discussed in the following sections.

Outdoor air

The neutral and more volatile PFCs [e.g. fluorotelomer alcohols (FTOHs), perfluorooctane sulfonamidoethanols (FOSEs), and perfluorooctane sulfonamides (FOSAs)] have been measured in outdoor air from various locations. Results from these analyses are given in Table 1.

In rural areas of Canada concentrations of 34 and 36 pg/m^3 (*N*-MeFOSE) and 68 and 85 pg/m^3 (*N*-EtFOSE) were found, respectively, while in urban sites concentrations were higher (101 pg *N*-MeFOSE/m^3 and 205 pg *N*-EtFOSE/m^3) (Martin et al., 2002). An urban–rural gradient was found in Germany too. Mean total concentrations of FOSEs/FOSAs (*N*-MeFOSE, *N*-EtFOSE, *N*-MeFOSA, and *N*-EtFOSA) of 50 pg/m^3 (9–142 pg/m^3) from an urban location (Hamburg) and 26 pg/m^3 (6–48 pg/m^3) from a rural site (Waldhof) were observed in 2005 (Jahnke et al., 2007b). Similar concentrations were found for FOSEs and FOSAs in northwestern Europe (Ireland, UK, Norway) (Barber et al., 2007). At 7 measuring sites in Ottawa concentrations of 76–99 pg *N*-MeFOSE/m^3 and 80–106 pg *N*-EtFOSE/m^3, were detected (Shoeib et al., 2005a). In the vicinity of a carpet processing factory in Griffin, GA, USA, total polyfluorinated sulfonamide (*N*-MeFOSE, *N*-EtFOSE, and *N*-EtFOSA) concentrations were found to be higher during one sampling event (1549 pg/m^3) probably due to specific meteorologic conditions and/or episodic point source release (Stock et al., 2004).

Measurements of single fluorotelomer alcohols in the North American troposphere ranged from 7 to 196 pg/m^3 (Martin et al., 2002), whilst mean total concentrations of FTOHs ranged from 11 to 165 pg/m^3 (Stock et al., 2004). In both studies higher concentrations were found in urban than rural settings. Similarly, in a German study, $\sum$FTOH concentrations in Hamburg were found to be 1.6 times higher compared to a rural area, reaching significance only for 4:2 FTOH and *N*-MeFOSE (Jahnke et al., 2007b). Furthermore, at four northwest European sampling sites comparable

Table 1. Concentration (range) of fluorinated organic compounds in gas and particulate phase of ambient air

Substance	Mean concentration (pg/m^3)	Sampling location	Sampling year	Number of samples (sampling sites)	Reference
N-MeFOSE	101 (86–123)	Canada, urban	2001	4	Martin et al. (2002)
	34, 36	Canada, rural		2	Martin et al. (2002)
	32, 16	Canada	2001–2003	2	Shoeib et al. (2004)
	83 (76–99)	Canada	2002–2003	7	Shoeib et al. (2005a)
	24–49[a]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	41 (15–95)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	8.6 (3.8–12)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
	(<1–11)	Oregon, USA, remote	2006	34	Piekarz et al. (2007)
N-EtFOSE	205 (51–393)	Canada, urban	2001	4	Martin et al. (2002)
	68, 85	Canada, rural		2	Martin et al. (2002)
	0.5 (0.3–1.0)	USA, Great Lakes	2003	8	Boulanger et al. (2005)
	9.8 and 8.5	Canada	2001–2003	2	Shoeib et al. (2004)
	88 (80–106)	Canada	2002–2003	7	Shoeib et al. (2005a)
	6.4–66[a]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	13 (6.0–30)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	16 (9.9–26)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
	(<1–3.7)	Oregon, USA, remote	2006	34	Piekarz et al. (2007)
N-MeFOSA	<5.3–6.1[a]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	9.0 (3.4–20)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	7.0 (3.8–11)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
N-EtFOSA	1.1 (0.4–2.2)	USA, Great Lakes	2003	8	Boulanger et al. (2005)
	<1.6–9.6	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	3.1 (1.3–5.9)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	2.6 (1.5–3.4)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
	(<0.4–3.2)	Oregon, USA, remote	2006	34	Piekarz et al. (2007)
∑FOSE/ FOSA	22–403 (<2–1549)[b]	Canada + USA	2001	26 (6)	Stock et al. (2004)
	39–89[b]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	50 (9–142)[d]	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	26 (6–48)[d]	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
4:2 FTOH	1.4–114	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	54 (22–117)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	19 (3.3–45)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
6:2 FTOH	87 (30–196)	Canada, urban	2001	4	Martin et al. (2002)
	41, 16	Canada, rural		2	Martin et al. (2002)
	5–187[a]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	66 (33–149)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	64 (17–125)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
	4.6	Oregon, USA, remote	2006	34	Piekarz et al. (2007)
	14, 35, 55	Japan, three areas		24	Oono et al. (2008)
8:2 FTOH	55 (9–123)	Canada, urban	2001	4	Martin et al. (2002)
	40, 25	Canada, rural		2	Martin et al. (2002)
	(<LOD–20)	Canada, Arctic	2004	10	Stock et al. (2007)
	11.3–237 [a]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	119 (62–275)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	75 (33–112)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
	24	Oregon, USA, remote	2006	34	Piekarz et al. (2007)
	550, 698, 2031	Japan, three areas		24	Oono et al. (2008)

Table 1. (*continued*)

Substance	Mean concentration (pg/m^3)	Sampling location	Sampling year	Number of samples (sampling sites)	Reference
10:2 FTOH	29 (7–46)	Canada, urban	2001	4	Martin et al. (2002)
	20 and 15	Canada, rural		2	Martin et al. (2002)
	7.8–75[a]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	35 (16–93)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	23 (10–32)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
	15	Oregon, USA, remote	2006	34	Piekarz et al. (2007)
	64, 88, 229	Japan, three areas		24	Oono et al. (2008)
∑FTOH	11–165[c]	Canada + USA	2001	26 (6)	Stock et al. (2004)
	28[c]	Canada, Arctic	2004	10	Stock et al. (2007)
	19.3–527 [a,c]	UK, Norway, Ireland	2005–2006	(6)	Barber et al. (2007)
	288 (150–546)	Germany, Hamburg	2005	7	Jahnke et al. (2007a)
	181 (64–311)	Germany, Waldhof	2005	4	Jahnke et al. (2007a)
	41 (16.0–83)[c]	USA, urban	2006	8	Kim and Kannan (2007)

[a]Only gas phase analyzed.
[b]Sum of *N*-MeFOSE, *N*-EtFOSE, and *N*-EtFOSA.
[c]Sum of 6:2 FTOH, 8:2 FTOH, and 10:2 FTOH.
[d]Only gas phase.

concentrations from 19 to 527 pg/m^3 (∑FTOH) were observed (Barber et al., 2007).

As yet, only Jahnke et al. (2007b) analyzed for a correlation between the outdoor air temperature and the sum concentration of FOSEs/FOSAs and FTOHs observing a significant association ($r = 0.95$ and 0.97).

Air samples collected on board the German research vessel Polarstern during a cruise between Bremerhaven (Germany) and Capetown (Republic of South Africa) indicate a strong decreasing gradient from the European continent towards the southern hemisphere (Jahnke et al., 2007c). The study confirms that volatile PFCs are mainly restricted to the northern hemisphere, with maximum 8:2 FTOH concentrations of 190 pg/m^3 compared to 14 pg/m^3 (southern hemisphere). During a cruise crossing the North Atlantic and the Canadian Arctic Archipelago in July 2005 Shoeib et al. (2006) measured maximum concentrations of 22.7 pg/m^3 (8:2 FTOH) and 23.6 pg/m^3 (*N*-MeFOSE) in the gas and particulate phase. Air concentrations of these two PFCs were somewhat higher, but on the same order of magnitude as reported in urban regions like Toronto.

Results on outdoor air concentrations of the much less volatile PFOS, PFOA and similar compounds in the particulate phase are given in Table 2. In Japan, in an exposed urban setting (high traffic load) the mean PFOA concentration was 372 pg/m^3 and the mean PFOS concentration 5.6 pg/m^3, while in a rural area concentrations were 2.0 pg PFOA/m^3 and 0.6 pg PFOS/m^3 (Harada et al., 2005a). Other investigations in Japan confirmed the regional differences in PFOS concentrations between rural (0.6 pg/m^3) and urban (5.3 pg/m^3) settings (Sasaki et al., 2003).

Concentration differences were also noted in samples collected from urban and rural sites in Europe. Higher concentrations of PFOS and PFOA in the particulate phase from specific areas also indicate influence from fluoropolymer production facilities. While very low concentrations of PFOA were found in rural areas of Ireland and Norway, the PFOA concentration in Manchester (UK) was found to be 341 pg/m^3 in February/March and 16 pg/m^3 in November/December. It is probable that the very high concentrations of 552 and 101 pg/m^3 observed at the fourth site (Hazelrigg/UK), and potentially in Manchester could be explained by a nearby fluorpolymer production plant. In addition, considerably high concentrations were observed along the fence line of an industrial area in the USA where a fluoropolymer processing factory is situated. Depending on the wind direction, in a 10-week period PFOA concentrations ranged between 120,000 and 900,000 pg/m^3 (Barton et al., 2006).

Sugita et al. (2007) analyzed dust samples collected on quartz fiber filters using a sampler placed on the rooftop of a building located in Wako City, Japan, in 2006. The concentrations were lower in December compared to July and also on weekends compared to weekdays with means of 4.3 and 7.3 pg/m^3, respectively. According to the few data available, particulate phase concentrations in North America are similar for PFOS but lower for PFOA (Boulanger et al., 2005; Stock et al., 2007), than concentrations reported for Japan. However, this could be due to the preselection of low exposure areas

Table 2. Mean concentration (range) of PFOS and PFOA in particulate phase of ambient air

Mean concentration (pg/m^3)	Sampling location	Sampling year	Number of samples (sampling sites)	Reference
PFOS				
5.3 (2.3–22)	Urban, Japan	2001–2002	12	Sasaki et al. (2003)
0.6 (0.1–2.1)	Rural, Japan		12	
5.6 (2.5–9.8)	Urban, Japan	2001–2003	12	Harada et al. (2005a)
0.7 (0.5–1.2)	Rural, Japan		8	
2.9	Urban, Japan	2005	1	Harada et al. (2006)
2.2	Rural, Japan		1	
6.8	High traffic road, Japan		1	
5.9[a]	Canada, Arctic	2004	10	Stock et al. (2007)
6.4 (<LOD–8.0)	USA, Lakes Erie and Ontario	2003	8	Boulanger et al. (2005)
<45	Hazelrigg, UK	2005–2006	2 (spring)	Barber et al. (2007)
1.6			10 (winter)	
46	Manchester	2005–2006	2 (spring)	
7.1			1 (winter)	
1,0	Kjeller, Norway	2005	2	
<1.8	Mace Head, Ireland	2006	4	
7.3 (3.6–15.7)	Wako City, Japan	2006	26 (July)	Sugita et al. (2007)
4.3 (0.9–8.9)			27 (December)	
0.6 (0.4–1.2) [b]	Albany, New York, USA	2006	8 (summer)	Kim and Kannan (2007)
1.7 (0.9–3.0) [c]				
PFOA				
372 (72–919)	Urban, Japan	2001–2003	12	Harada et al. (2005a)
2.0 (1.6–2.6)	Rural, Japan		8	
15.2	Urban, Japan	2005	1	Harada et al. (2006)
205	Rural, Japan		1	
320	High traffic road, Japan		1	
1.4 [a]	Canada, Arctic	2004	10	Stock et al. (2007)
552	Hazelrigg, UK	2005–2006	2 (spring)	Barber et al. (2007)
101			10 (winter)	
341	Manchester, UK	2005–2006	2 (spring)	
16			1 (winter)	
1.5	Kjeller, Norway	2005	2	
8.9	Mace Head, Ireland	2006	4	
2.0 (0.8–4.2) [b]	Albany, New York, USA	2006	8 (summer)	Kim and Kannan (2007)
3.2 (1.9–6.5) [c]				

[a]Gas and particulate phase.
[b]Particulate phase.
[c]Gas phase.

for measurements in North America. Stock et al. (2007) described data from a remote Arctic site with mean concentrations of 5.9 pg/m^3 (PFOS), 0.2 pg/m^3 (PFHxS), 0.2 pg/m^3 (PFDS), 1.4 pg/m^3 (PFOA), 0.4 pg/m^3 (PFNA) and 0.4 pg/m^3 (PFDA), respectively.

Barber et al. (2007) found that PFOA was the prevailing analyte observed mainly in the particulate phase. Up to now, this point is not well understood. It can be hypothesized that source strength and different degradation processes on particulate matter were responsible for this observation.

Currently only very few studies on outdoor air PFC concentrations are available; these are mainly characterized by very small sample sizes or short sampling time periods. Overall, the studies indicate that a concentration gradient exist between urban, rural, and remote areas for FOSEs/FOSAs as well as for PFOS and PFOA. Substantially higher concentrations observed in specific locations also highlight the influence of possible point in addition to diffuse sources for these compounds. For some of the more volatile PFCs, a temperature dependency was found in one study; in a similar fashion, another study observed seasonal fluctuations of PFOS and PFOA concentrations. Beyond the effects of these seasonal and localized geographical factors no marked differences were found between PFC outdoor air concentrations from the western countries.

Indoor air

Findings on indoor air concentrations are given in Table 3. The most comprehensive data are available from Canada, where samples were taken from four private homes in the city of Ottawa between 2001 and 2003 (Shoeib et al., 2004) and an additional 59 randomly selected homes in 2002/2003 with a different sampling technique (Shoeib et al., 2005a). While in the first study analytes were actively sampled on polyurethane foam (PUF) and glass fiber filters, in the second investigation a passive sampling method using PUF-disks (21 days sampling time) was employed. Despite this methodological difference both studies found comparable concentrations with mean values of 1110 and 770 pg/m^3 (*N*-EtFOSE), 2590 and 1970 pg/m^3 (*N*-MeFOSE), as well as 73 and 35 pg/m^3 (*N*-MeFOSEA), for the two studies, respectively.

Considerably higher concentrations of 14,900 pg/m^3 FOSEs/FOSAs were observed in the gas phase of four indoor locations in Tromsø, Norway in 2005 (Barber et al., 2007). In this study fluorotelomer alcohols were determined in the gas phase for the first time at a geometric mean sum concentration of 11,075 pg/m^3. In the particulate phase only negligible amounts of the investigated PFCs could be found. The first measurements in a Norwegian office resulted in concentrations below those in private homes, probably due to the absence of typical sources such as carpets and upholstery for these compounds in offices (Jahnke et al., 2007a).

In Canada indoor to outdoor ratios reached 18 for *N*-MeFOSE and 8 for *N*-EtFOSE (Shoeib et al., 2005a), whilst in an earlier study indoor air levels exceeded outdoor air concentrations by about a factor of 100 (Shoeib et al., 2004). In the study of Barber et al. (2007) no outdoor concentrations from the vicinity of the measured indoor places are available. In comparison to other outdoor levels, however, an indoor to outdoor ratio of 30–570 ($\sum$FTOH) and 170–380 ($\sum$FOSAs/FOSEs), respectively, can be deduced.

In addition to neutral PFCs, Barber et al. (2007) analyzed various perflorocarboxylates (PFCAs) and perfluorosulfonates (PFAS) in the particulate phase of the four aforementioned sites in Tromsø. The highest concentrations were found for PFHxA and PFOA (17.1 and 4.4 pg/m^3, respectively), while among the sulfonates, only perfluorodecane sulfonate (2.6 pg/m^3) exceeded the limit of quantification.

Up to now, there are only very few data on indoor air concentrations of PFCs available. It can be concluded, that the indoor PFCA and PFAS levels were not significantly elevated above outdoor air, whilst concentrations of volatile polyfluorinated compounds appear to be considerably higher in indoor than in outdoor air. Because humans spend a lot of their time in indoor spaces much more data are needed to better characterize the exposure in the different indoor environments, such as residences and work places. Studies on seasonal variation and the influence of different furnishings will also provide important data to help examine exposure to PFCs.

Household dust

In the winter of 2002/2003, 66 randomly selected households in Ottawa, Canada were investigated (Shoeib et al., 2005a) for PFCs in dust. Dust samples were collected with a vacuum cleaner and 0.001–75.4 μg *N*-EtFOSE/g (geometric mean: 0.14 μg/g) and 0.003–8.8 μg *N*-MeFOSE/g (geometric mean: 0.11 μg/g) were found. The investigators observed a good correlation between the dust concentrations of FOSEs and the corresponding values in indoor air.

All other studies that have examined PFCs in dust have focused on the less volatile PFCAs and PFAS. In 16 Japanese houses concentrations between 0.011 and 2.5 μg PFOS/g dust (unsieved, only large particles removed) and between 0.070 and 3.7 μg PFOA/g dust were determined in dust collected from vacuum cleaner bags (Moriwaki et al., 2003). Median concentrations were 0.025 μg PFOS/g dust and 0.165 μg PFOA/g dust. A strong correlation was found between PFOS and PFOA ($r^2 = 0.99$), however the association dropped to $r^2 = 0.35$ when one outlier was removed. In another Japanese study, PFOS and PFOA were detected in all 7 collected dust samples (particle size of 75 μm to 1 mm) from 0.007 to 0.041 μg/g and 0.018 to 0.089 μg/g, respectively (Nakata et al., 2007).

In two North American studies, a wider variety of PFCAs and PFAS were studied. Dust from vacuum cleaner bags was collected in winter 2002/2003 from 67 Canadian homes and was sieved to a size of <150 μm (Kubwabo et al., 2005). The most frequently detected PFCs were PFOS at <0.002–5.065 μg/g (median: 0.038 μg/g; 33% of measurements below the limit of detection, 0.005 μg/g), PFOA at <0.002–1.231 μg/g (median: 0.020 μg/g; 37% <0.002 μg/g), and PFHxS at <0.002–4.305 μg/g (median: 0.023 μg/g; 15% < 0.005 μg/g). PFC concentrations in the dust were statistically significantly correlated with the age of the houses and the floor covering. Older houses were characterized by lower concentrations of PFOS and PFOA, but not of PFHxS. All three compounds were positively correlated with each other and with the fraction of the floor covered with carpets.

Additionally, 112 dust samples were collected in 2000–2001 in Ohio and North Carolina and stored at room temperature in dark glass bottles (Strynar and Lindstrom, 2008). After sieving to <150 μm, samples were analyzed for a number of PFCAs and PFAS.

Table 3. Mean concentration (range) of volatile polyfluorinated compounds in indoor air

Substance	Concentration (pg/m^3)	Sampling location	Sampling year	Number of samples	Phase analyzed	Reference
N-MeFOSE	1970 (366–8190)	Canada	2002–2003	59[a]		Shoeib et al. (2005a)
	2590 (667–8315)	Canada	2001–2003	4	Particulate	Shoeib et al. (2004)
	6018	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	363	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
	727, 798	Norway	2006	2 office	Vapor and particulate	Jahnke et al. (2007b)
N-EtFOSE	1100 (227–7740)	Canada	2002–2003	59[a]		Shoeib et al. (2005a)
	770 (364–1799)	Canada	2001–2003	4	Particulate	Shoeib et al. (2004)
	5755	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	76	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
	305, 815	Norway	2006	2 office	Vapor and particulate	Jahnke et al. (2007b)
N-MeFOSEA	35 (12–109)	Canada	2002–2003	59[a]		Shoeib et al. (2005a)
	≈73 (LOD[b]–283)	Canada	2001–2003	4	Particulate	Shoeib et al. (2004)
N-MeFOSA	6608	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	6	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
N-EtFOSA	59 (5.9–646)	Canada	2002–2003	59[a]		Shoeib et al. (2005a)
	6626	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	7	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
	188, 158	Norway	2006	2 office	Vapor and particulate	Jahnke et al. (2007b)
4:2 FTOH	114	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	<20	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
6:2 FTOH	2990	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	<40	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
	177, 248	Norway	2006	2 office	Vapor and particulate	Jahnke et al. (2007b)
8:2 FTOH	2070 (261–28900)	Canada	2002–2003	52[a]		Shoeib et al. (2007)
	3424	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	<10	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
	853, 421	Norway	2006	2 office	Vapor and particulate	Jahnke et al. (2007b)
10:2 FTOH	891 (104–9210)	Canada	2002–2003	52[a]		Shoeib et al. (2007)
	3559	Norway	April–June 2005	4	Vapor	Barber et al. (2007)
	13	Norway	April–June 2005	4	Particulate	Barber et al. (2007)
	898, 1.660	Norway	2006	2 office	Vapor and particulate	Jahnke et al. (2007b)

[a]Passive sampling over 21 days.
[b]LOD: limit of detection.

PFOS concentrations of <0.009–12.1 μg/g (mean: 0.76 μg/g; 5% <limit of quantification, 0.009 μg/g) and PFOA concentrations of 0.01–1.96 μg/g (mean: 0.29 μg/g; 4% <0.01 μg/g) were found. For PFHxS the mean concentration was 0.87 μg/g, for PFHxA 0.12 μg/g and for PFHpA 0.11 μg/g. No differences were observed between the two sampling regions, but a significant correlation was found between PFOS and PFOA ($r = 0.87$).

In Germany, 12 dust samples were collected with a vacuum cleaner (Fromme et al., 2008) in a pilot study. Median (range) PFOS and PFOA concentrations in

the sieved fraction (<63 μm) were 0.016 μg/g (0.003–0.342 μg/g) and 0.011 μg/g (0.002–0.141 μg/g), respectively. Significantly lower median concentrations were observed in the unsieved samples (PFOS: 0.010 μg/g; PFOA: 0.007 μg/g) indicated that PFAS were mainly associated with smaller particles.

While only little information has been collected on the contamination of household dust at this point in time, the results indicate a large variability in the concentrations of the perfluorinated substances measured. Whilst the mean PFOS concentration in samples collected from Canadian and Japanese homes appear to be very similar, the mean PFOA concentration in Canada was 9 times lower than in Japan. On the other hand, very high concentrations were reported in the US study (Strynar and Lindstrom, 2008), where mean values exceed concentrations observed in the other countries by factors of 200 for PFOS and 150 for PFOA. The reasons for these differences, which may be partly due to methodological differences, are yet unknown.

Contamination of food and drinking water

Although dietary intake is assumed to be a major route of exposure for the general population, only few systematic data on PFC levels in foods are available. Often ecological or ecotoxicological questions are the focus of investigations on animals, so that information on the contamination of edible parts cannot be deduced. More detailed data are only available for PFC levels in fish, mainly in the context of surveys of fish caught in PFC-contaminated waters.

Commercially available food items

Only a limited number of studies have examined the presence of PFCs in commercially available food items. Details of these are provided in Table 4. These studies have analyzed only food items purchased from locations in North America and Western Europe; their main focus has been the analysis of PFCAs and PFAS.

Concentrations observed in all the studies conducted to date were in the sub- to low ng/g range. In 2000, the so-called "Multi-City-Study" conducted in 6 US cities observed PFOS was detected in 5 samples (milk and ground beef) and PFOA in 5 samples (green beans, apple, bread, and ground beef) at concentrations up to 0.85 $ng/g_{fresh\ weight}$ and 2.35 ng/g, respectively (US EPA, 2001). However, in only one of these instances (PFOS in ground beef) were the results from the duplicate consistent; for the remaining positive detections, PFCs were not detected in the duplicate analyzed. The UK Total Diet Study (TDS) found concentrations of PFOS, PFUA, PFDA, and PFTeDA up to 10 $ng/g_{fresh\ weight}$. These higher concentrations were all reported in the "potatoes" composite (which included potato chips, french fries, and other potato products) (FSA, 2006). The Canadian TDS observed concentrations up to 4.5 ng/g (PFNA in beef steak) (Tittlemier et al., 2007). The maximum concentration observed in a total of 36 composite samples purchased from local stores in Tarragona County, Spain was 0.84 $ng/g_{fresh\ weight}$ PFOS (Ericson et al., 2007b).

The German duplicate diet study was conducted in a slightly different fashion. As with the UK and Canadian TDSs, PFCs were analyzed in prepared and otherwise cooked food. However, the duplicate diet study did not analyze food items (or composites of similar food items) separately. Samples analyzed in this study were comprised of homogenized liquid or solid portions of whole meals. The maximum concentration observed in this study was 118 $ng/g_{fresh\ weight}$ PFOA; although most concentrations observed were less than 0.1 $ng/g_{fresh\ weight}$ (Fromme et al., 2007c).

Some of the studies have also analyzed for precursors to PFOS. These studies have mainly focused on PFOSA (US EPA, 2001; FSA, 2006; Fromme et al., 2007c); only the 2006 Canadian study has examined a wider range of perfluorooctanesulfonyl compounds (PFOSAs) in food items (Tittlemier et al., 2006).

The majority of food samples analyzed did not contain detectable PFCA or PFAS residues above the various limits of detection for the analytical methods employed. Generally, less than 50% of samples analyzed did not contain detectable levels of PFCAs or PFAS and in a study from Germany using 28 samples of packed and frozen French fries none reached the limit of detection of 1 ng/g for PFOS or PFOA (Stahl, 2007). A higher percentage of samples from the Canadian TDS contained PFOSAs (Tittlemier et al., 2006), even though PFOSA itself was not detected in samples from the other studies aside from one sample in the UK TDS (FSA, 2006). This may be due in part to the lower detection limit of the gas chromatographic–mass spectrometric method used in the Canadian study (Tittlemier et al., 2005).

Contamination of fish

PFOS and PFOA have been demonstrated to bioaccumulate in fish (Martin et al., 2003a, b; Gruber et al., 2007). Thus, fish is potentially an important dietary source of these PFCs for consumers. Freshwater and marine fish, and seafood, have been analyzed for PFCs in many studies.

Generally, PFOS has been found at higher levels in fish than PFOA. High PFOS concentrations of 59–297 $ng/g_{fresh\ weight}$ were found in muscle from fish caught in 1999 and 2000 in the American Great Lakes

(Giesy and Kannan, 2001, Kannan et al., 2005). By contrast, PFOA values did not reach the detection limit (LOD) of 36 ng/g (Kannan et al., 2005). Moreover, in China in 2004 freshly bought seafood (fish and shellfish edible portions) was analyzed for PFCs (Gulkowska et al., 2006). The PFOS concentrations ranged from 0.33 to 4.6 $ng/g_{wet\ weight}$; in one sample of shrimps a concentration of 13.9 ng/g was observed. In this study, the PFOA concentrations were between <0.25 and 1.7 ng/g and 45% of the samples contain levels below the LOD. In a Bavarian monitoring program that analyzed fish sampled from 15 bodies of water in 2005/2006, PFOS was found from 3.9 to 16.3 ng/g (19 eel samples), 7.1–14.7 ng/g (5 carp or perch), <1.0–1.3 ng/g (4 barbel), and 1.7–17.8 ng/g (5 pike) (LfU, 2007). In contrast, the PFOA concentrations in muscle of all measured fish ($n = 35$) ranged between <0.1 and 7.2 ng/g. In all 15 fish sampled in the Federal State of Hessen, Germany, PFOS and PFOA were below 1 ng/g; only in one carp a PFOS concentration of 1.8 ng/g was found (Stahl, 2007).

The difference between the observed PFOS and PFOA fish concentrations could suggest a lower potential of PFOA to bioaccumulate in fish than PFOS. Differences in bioconcentration and dietary accumulation of PFOS and PFOA have been demonstrated in laboratory experiments (Martin et al., 2003a, b; Gruber et al., 2007).

Fish sampled from areas that contain known point sources of PFCs, such as fluoropolymer or fluorochemical production plants, often contain higher PFC concentrations. For example, 3.0–52.5 ng/g PFOA were observed in fish (LfU, 2007) sampled from a waterbody with a known source of PFOA from a production plant in Bavaria. In addition, a survey on PFOS and PFOA levels in more than 200 fish was undertaken in the Federal State of North Rhine-Westphalia, Germany, in which a remarkable case of a contamination with PFCs became evident in 2006 (Wilhelm et al., 2008a). The highest level of PFOS (1100 $ng/g_{wet\ weight}$) was detected in a trout filet from a fish farm pond in the affected area. Fish (e.g. trout, chub, perch, zander) caught from contaminated rivers and lakes in 2006 and 2007 contained PFOS at levels between 6 and 425 $ng/g_{wet\ weight}$. PFOS in trout caught from non-contaminated creeks in North Rhine-Westphalia were <4 $ng/g_{wet\ weight}$. PFOA levels of fish were mostly below 2 $ng/g_{wet\ weight}$. The highest PFOA concentration (34 $ng/g_{wet\ weight}$) was measured in an eel sample. It should be noted that in the affected area only PFOA levels in surface waters were increased.

Similarly, elevated PFC concentrations were found in fish sampled from an area near a point source of PFCs. A second Bavarian Monitoring program analyzed 39 fish samples for PFOS and PFOA (LGL, 2007). The concentrations ranged from between LOQ (0.5 ng/g) and 80.3 ng/g for PFOS and between LOQ (1 ng/g) and 20.9 ng/g for PFOA. The highest concentrations of PFOA were found in eels and perches caught in rivers containing effluent from the point source. For both compounds, concentrations in fish living in fish ponds were lower compared to fish living in contaminated river water.

Some studies have found positive correlations between PFC body burdens and self-reported fish consumption. In Poland, blood samples from 45 donors living near the Baltic Sea were analyzed in 2004 (Falandysz et al., 2006). Subjects with a high consumption of regionally captured fish ($n = 15$) showed statistically higher PFC blood levels than the comparison groups. The authors concluded that the consumption of seafood was an important determinant for internal PFC exposure. The human biomonitoring study that examined residents in the affected North Rhine-Westphalia area also found a positive association between PFOS concentrations in plasma and consumption of locally caught fish, indicating that fish intake can be an important pathway for internal PFC exposure (Hölzer et al., 2008).

Contamination of drinking water

Current studies have shown that drinking water PFC concentrations are in the low ng/l range if there is no large point source of PFCs to the drinking water source. The analysis of potable water in Japan observed PFOS concentrations between 0.1 and 51 ng/l; the majority of results (8 of 9 waterworks) did not exceed 4 ng/l (Harada et al., 2003). Only in one waterworks concentrations of 43.7 and 51 ng/l were observed. The authors explain the high values by the fact that the waterworks draws water from the river Tama, which is contaminated upstream with PFOS by a wastewater treatment plant. In other investigations, the presence of potential sources of PFCs, such as an airport, has been observed to correlate with higher PFC surface water concentrations as well (Saito et al., 2004). In this study, concentrations of PFCs in drinking water from exposed areas ranged between 5.4 and 40.0 ng PFOS/l and 1.1 and 1.6 ng PFOA/l, while in areas with no known sources concentrations were only <0.1–0.2 ng PFOS/l and 0.1–0.7 ng PFOA/l.

Results from North America are generally similar. During the American "Multi-City-Study", PFOA was found at concentrations of 26 and 27 ng/l and PFOS at concentrations of 57 and 63 ng/l in tapwater from Columbus (US EPA, 2001). In the remaining 5 cities concentrations generally did not exceed the detection limit for PFOS (2.5 ng/l) and PFOA (7.5 ng/l). Only in one sample of potable water from Pensacola PFOS concentrations of 42 and 47 ng/l were found.

In Europe, Skutlarek et al. (2006) observed PFOA concentrations of <2–4 ng/l (13 of 16 were below the limit of detection) in 14 German, one French and one English drinking water samples. The PFOS and PFBS concentrations in these samples ranged between <2 and 6 ng/l (14 of 16 < LOD) and <2–20 ng/l (13 of 16 < LOD). In the vicinity of Lake Maggiore in Italy, concentrations of 8.1 ng/l (PFOS) and 2.4 ng/l (PFOA) were found in 6 samples of drinking water. These concentrations were very similar to the concentrations detected in the lake (Loos et al., 2007). The authors report that PFOS could not be detected in water samples from waterworks, which do not draw water from Lake Maggiore.

Contamination of drinking water by known sources

Worldwide two cases of PFOA contaminated drinking water have been studied in detail (Little Hocking, Ohio, USA and Sauerland, North Rhine-Westphalia, Germany).

Since 2004, drinking water wells in the Little Hocking Water Association, Ohio, a water catchment area in the vicinity of a localized PFOA source have been investigated. In this work, PFOA concentrations of 1900–10,100 ng/l (2004), 3900–18,600 ng/l (January 2005) and 1900–6600 ng/l (March 2005) were observed in four wells of the central water supply, as well as at the transit station to the distribution system 7200 ng/l (January 2005) (LHWA, 2005). A population-based study observed the highest PFOA concentrations in serum (median 374 ng/l) among those subjects which exclusively used water from the Little Hocking central drinking water supply (Emmett et al., 2006a). The private use of carbon water filters was associated with significant lower median blood levels, while subjects, who mostly drank water that originated from outside of the Little Hocking area, showed considerably lower serum PFOA concentrations.

The PFOA contamination in the Sauerland region was first discovered by Skutlarek et al. (2006). They reported levels of the sum of 7 PFCs in drinking water between 26 and 598 ng/l. The most abundant compound observed was PFOA; values in drinking water ranged from 22 to 519 ng/l. In 6 cities in this area concentrations above 100 ng/l were found. The proportion of PFOA in total PFCs detected was 50–80%. Industrial waste with high concentrations of PFCs was manufactured into soil improver by a recycling company and disseminated by framers on agricultural land in the rural area Sauerland. The use of the contaminated soil improver led to this substantial environmental pollution (details of this case are summarized in Wilhelm et al., 2008a). PFCs were washed from the highly contaminated area into small creeks and surface waters (Ruhr river, Möhne river, Möhne Lake) from which drinking water is drawn for several million residents of the Ruhr District. A survey performed between July 2006 and August 2007 showed that the sum of PFOS and PFOA levels in drinking water from the 17 waterworks along the Ruhr river were below 300 ng/l, mean levels were mostly between 50 and 100 ng/l (Wilhelm et al., 2008a). At the most affected waterworks of Möhnebogen, treatment with charcoal filtration effectively removed PFOA from drinking water. The initial PFOA concentrations of >500 ng/l observed in May 2006 rapidly declined to values mostly well below 100 ng/l after using charcoal filters. This concentration was set as a long-term minimum quality goal derived from a health-based precautionary value (DWC, 2006).

Dietary intake estimated from diet studies

At this point, only four studies which attempted to quantify the intake of PFCs via the diet have been published. Three of them used a market basket approach combining the measured concentrations in food composite samples with consumption patterns (FSA, 2006; Tittlemier et al., 2007; Ericson et al., 2008). The third study used a duplicate diet approach measuring PFC in duplicate portions of food prepared as for consumption (Fromme et al., 2007c). A summary of the results of these surveys is given in Table 4.

The first study analyzed PFCs in 20 composite food group samples from the 2004 UK TDS (FSA, 2006). The yearly composites were assembled by collecting retail food samples every fortnight from 24 locations in the UK and preparing as for consumption before compositing. PFOS was detected as the main analyte above LOD in potatoes (including chips, crisps, potato salad, hash browns, and croquettes), canned vegetables, eggs, and sugar and preserves. PFOA was detected only in the potato group. Based on the average and high (97.5th percentile) food consumption scenarios as derived from the nutritional surveys of British adults, the dietary intake of PFOS and PFOA was estimated. Concentrations below LOD were either substituted by the reporting limit (upper bound) or substituted with zero (lower bound). The estimated average daily intake was 100 $ng/kg_{body\ weight}$ (PFOS) and 70 $ng/kg_{body\ weight}$ (PFOA) (upper bound) or 10 $ng/kg_{body\ weight}$ (PFOS) and 1 $ng/kg_{body\ weight}$ (PFOA) (lower bound). The upper and lower bound intakes estimated using a high food consumption level were 200 $ng/kg_{body\ weigh}$ (PFOS) and 100 $ng/kg_{body\ weight}$ (PFOA) and 30 $ng/kg_{body\ weight}$ (PFOS) and 3 $ng/kg_{body\ weight}$ (PFOA), respectively.

Tittlemier et al. (2007) estimated the dietary exposure of Canadian teenagers and adults based on 25 composite samples collected in the 2004 TDS. Various food items