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Polyfluoroalkyl Compounds in the Marine Environment – Investigations on their Distribution in Surface Water and Temporal Trends in Harbor Seals (*Phoca vitulina*)

(Von der Fakultät III Umwelt und Technik der Leuphana Universität Lüneburg als Dissertation angenommene Arbeit)

Author:
L. Ahrens

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Lutz Ahrens

176 pages with 39 figures and 40 tables

Abstract

Recently polyfluoroalkyl compounds (PFCs) were discovered as emerging persistent organic pollutants. Because of their unique physicochemical properties due to their combination of lipophilic and hydrophilic characteristics, PFCs have been widely used in many consumer products, such as polymerisation aids, stain repellents on carpets, textiles, and paper products for over 50 years. From the production and use of these products, PFCs can be released into the environment. Scientific concern about PFCs increased due to their global distribution and ubiquitous detection in the environment, especially in marine mammals.
An analytical protocol was developed for the analysis of PFCs in water samples and various biological matrices. The samples were analysed for 40 PFCs plus 20 isotope-labelled internal standards using high performance liquid chromatography/negative electrospray ionisation-tandem mass spectrometry (HPLC/(-)ESI-MS/MS). Furthermore, the analytical quality of the laboratory has been approved in inter-laboratory studies.
In the first part of this Ph.D. thesis was investigated the occurrence, distribution pattern and transportation mechanisms of PFCs in seawater. The rivers had a high influence on the distribution of PFCs in offshore surface water in the German Bight, with decreasing concentrations with increasing distance from the coast (see **publication I**). The research on the spatial distribution of PFCs in coastal area is very important for the understanding of the transportation and fate of PFCs in the marine environment. Furthermore, the longitudinal and latitudinal distribution of PFCs in surface water of the Atlantic Ocean was investigated (see **publication II**). The results indicate that trans-Atlantic Ocean currents caused the decreasing concentration gradient from the Bay of Biscay to the South Atlantic Ocean and the concentration drop-off close to the Labrador Sea. These data are very useful for global transportation models, in which industrial areas are considered as sources, and ocean waters as sinks of PFCs.
The second part of this Ph.D. thesis examined the mechanisms and pathways of PFCs in harbor seals (*Phoca vitulina*) and their temporal trends in the German Bight. Firstly, the whole body burden of PFCs and their tissue distribution (i.e., liver, kidney, lung, heart, blood, brain, muscle, thyroid, thymus, and blubber) was investigated in harbor seals (see **publication III**). This study is relevant for calculation of the bioaccumulation potential of these compounds in marine mammals. Secondly, the temporal trends over the last decade and associations between PFC concentration and the evidence of diseases, spatial distribution, age and sex were evaluated in archived harbor seal livers (see **publication IV**). The results show significant declining concentrations of many PFCs indicating the replacement of these PFCs by shorter chained and less bioaccumulative compounds.
Several studies were performed besides the main issue of the Ph.D. work. Firstly, water samples were collected along the river Elbe into the North Sea to examine the distribution of PFCs in the dissolved and particulate phase, their discharge into the North Sea, and the influence of waste water treatment plant effluents to the riverine mass flow. Furthermore, surface water samples were collected in the North Sea, Baltic Sea and Norwegian Sea, where the occurrence and spatial distribution between river estuaries, coastal waters, in brackish as well as salt water, and open sea water were compared. Finally, within the frame of a research stay at the National Institute of Advanced Industrial Science and Technology (AIST) in Japan, the partitioning behaviour of PFCs between pore water and sediment in two sediment cores from Tokyo Bay was investigated.

This Ph.D. thesis has improved our knowledge of the occurrence and distribution of PFCs in water and biota highlighting association between PFCs and pathological conditions, potential sources and sinks, spatial distribution, and changes in their pattern and long-term perspective trends.

Polyfluorierte Alkylverbindungen in der marinen Umwelt – Untersuchung zur Verteilung im Wasser und zeitlichen Trends in Seehunden (*Phoca vitulina*)

Zusammenfassung

Polyfluorierte organische Substanzen (PFCs) gehören zu den neuartigen Problemstoffen in der Umwelt, die sich durch ihre Persistenz, Toxizität und ihr Potential zur Bioakkumulation auszeichnen. Aufgrund ihrer einzigartigen Eigenschaften finden sie seit ca. 50 Jahren vielfältige Anwendung in industriellen und kommerziellen Produkten wie u.a. Beschichtungen für Lebensmittelverpackungen, Imprägniermitteln für Textilien, Hilfsmittel in der Polymerchemie oder Bestandteil von Feuerlöschschäumen. PFCs wurden bereits ubiquitär in der Umwelt gefunden, mit den höchsten Konzentrationen in marinen Säugetieren.
Es konnte erfolgreich eine Methode zur Messung von PFCs in der Wasserphase und unterschiedlichen Gewebematerialien etabliert werden. Die instrumentelle Analyse umfasst insgesamt 40 Zielsubstanzen plus 20 isotopenmarkierte Standards die mittels Flüssigkeits-chromatographie – negativ Elektrospray Ionisation – Tandem Massenspektrometrie (HPLC/(-)ESI-MS/MS) gemessen werden.
In dem ersten Teil dieser Arbeit wird das Vorkommen, Verteilung und der Transportmechanismus von PFCs in Seewasser untersucht. Hierbei zeigte sich, dass die Flusseinträge einen großen Einfluss auf die Verteilung von PFCs in küstennahem Wasser in der Deutschen Bucht haben, wobei die Konzentrationen mit zunehmender Entfernung zur Küste abfallen (siehe **Publikation I**). Des Weiteren wurde die Verteilung von PFCs im Atlantischen Ozean untersucht (siehe **Publikation II**). Die Ergebnisse zeigen, dass die Strömungen des Atlantischen Ozeans im Wesentlichen für den fallenden Konzentrationsgradienten vom Golf von Biscaya zum südatlantischen Ozean und den Konzentrationsabfall in der Labrador-See verantwortlich sind. Die beiden Studien liefern wichtige Daten für u.a. globale Schadstoff-Transportmodelle, wobei die industriellen Gebiete als Quellen und der Ozean als Senke identifiziert werden konnten.
Der zweite Teil der Arbeit beschäftigt sich mit dem Aufnahmewegen und der Verteilung von PFCs in Seehunden (*Phoca vitulina*) und der Rekonstruktion ihrer zeitlichen Belastung in der deutschen Bucht. Es konnte eine gewebeabhängige Verteilung von PFCs in Seehunden gefunden werden, wobei auf Blut und Leber etwa drei Viertel der PFCs entfielen und der Rest in den anderen Organen (i.E., Muskel, Lunge, Niere, Fett, Herz, Gehirn, Thymus, Schilddrüse) verteilt waren (siehe **Publikation III**). Hiermit lassen sich das Bioakkumulationspotential und die Gesamtkörperbelastung für die einzelnen PFCs in marinen Säugetieren abschätzen. Des Weiteren wurde der Zusammenhang zwischen den PFC Konzentrationen und dem Auftreten von Krankheiten, räumlicher Verteilung, Alter und Geschlecht der Tiere untersucht und die zeitliche Belastung in Seehunden über die letzten zehn Jahre rekonstruiert (siehe **Publikation IV**). Die Ergebnisse zeigen signifikant fallende Konzentrationen für zahlreiche PFCs, was auf die Verwendung von kürzerkettigen PFCs und weniger bioakkumulierenden Substanzen zurückgeführt werden kann.
Zusätzlich wurden Wasserproben entlang der Elbe bis in die Deutsche Bucht genommen um die Verteilung zwischen der gelösten und partikelgebundenen Phase, ihren Masseneintrag in die Deutsche Bucht und den Einfluss von Kläranlagenausläufen zu untersuchen. Außerdem wurden weitere Oberflächenwasserproben in der Nordsee, Ostsee und Norwegischen See genommen, um das Vorkommen und die Verteilung von PFCs in den unterschiedlichen Gebieten zu vergleichen. Abschließend wurde im Rahmen eines Forschungsaufenthaltes in Japan die Verteilung von PFCs in Porenwasser und Sediment in zwei Sedimentkernen aus der Bucht von Tokio untersucht.
Die Promotionsarbeit verbessert das Verständnis des Verteilungsverhaltens von PFCs in der marinen Umwelt. Der Nachweis der Belastung der marinen Umwelt mit PFCs leistet einen wissenschaftlichen Beitrag zur Begründung und Erfolgskontrolle von politischen Maßnahmen und könnte einen Anstoß in Deutschland und auch auf europäischer Ebene geben, einen Handlungsplan für PFCs zu erstellen.

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List of publications

The cumulative Ph.D. thesis "Polyfluoroalkyl Compounds in the Marine Environment – Investigation on their Distribution in Surface Water and Temporal Trends in Harbor Seals (*Phoca vitulina*)" is based on the following scientific publications listed below. All of them are published.

Publication I

Lutz Ahrens, Sebastian Felizeter, Ralf Ebinghaus. Spatial distribution of polyfluoroalkyl compounds in seawater of the German Bight. *Chemosphere* 2009, 76, 179-184.

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Publication II

Lutz Ahrens, Jonathan L. Barber, Zhiyong Xie, Ralf Ebinghaus. Longitudinal and latitudinal distribution of perfluoroalkyl compounds in the surface water of the Atlantic Ocean. *Environ. Sci. Technol.* 2009, 43, 3122-3127.

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Publication III

Lutz Ahrens, Ursula Siebert, Ralf Ebinghaus. Total body burden and tissue distribution of polyfluorinated compounds in harbor seals (*Phoca vitulina*) from the German Bight. *Marine Poll. Bull.* 2009, 58, 520-525.

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Publication IV

Lutz Ahrens, Ursula Siebert, Ralf Ebinghaus. Temporal trends of polyfluoroalkyl compounds in harbor seals (*Phoca vitulina*) from the German Bight, 1999-2008. *Chemosphere* 2009, 76, 151-158.

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List of abbreviations

AFFFs	aqueous fire fighting foams
APCI	atmospheric pressure chemical ionisation
APPI	atmospheric pressure photo ionisation
BAFs	bioaccumulation factors
BCFs	bioconcentration factors
BMFs	biomagnification factors
BSH	Federal Maritime and Hydrographic Agency (Bundesamt für Seeschifffahrt and Hydrographie)
CFCs	chlorofluorocarbons
CI	chemical ionisation
CIC	combustion ion chromatography
dw	dry weight
DOC	dissolved organic carbon
ECD	electron capture detector
EI	electron impact
FASAAs	perfluoroalkyl sulfonamidoacetic acids
FASAs	perfluoroalkyl sulfonamides
FASEs	perfluoroalkyl sulfonamidoethanols
FBs	field blanks
FID	flame ionisation detection
f_{OC}	fraction organic carbon
FTALs	fluorotelomer aldehydes
FTAs	fluorotelomer acrylates
FTCAs	fluorotelomer carboxylates
FTOHs	fluorotelomer alcohols
FTolefins	fluorotelomer olefins
FTS	fluorotelomer sulfonates
FTUCAs	fluorotelomer unsaturated carboxylates
GC-MS	gas chromatography coupled with mass spectrometry
GFFs	glass-fibre filters
GJIC	gap junctional intercellular communication
HPLC/(-)ESI-MS/MS	high performance liquid chromatography coupled with a tandem mass spectrometry operated in an electrospray negative mode
IC_{50}	inhibition concentration at which 50% of subjects will inhibited
ICES	International Council for the Exploration of the Sea
IDLs	instrument detection limits
IF	inorganic fluorine
InjS	injection standard (spiked before analysis)
IPE	ion pairing extraction
IS	isotope-labelled internal standards (spiked before extraction)
ITCZ	inner tropical convergence zone

LD_{50}	lethal dose at which 50% of subjects will die
LLE	liquid-liquid extraction
LODs	limit of detection
LOQs	limits of quantitation
m/z	ion mass to charge ratio
MCWG	Marine Chemistry Working Group
MDLs	method detection limits
MQLs	method quantitation limits
NCI	negative chemical ionisation
NMR	nuclear magnetic resonance
NOAEL	no-observed-adverse effect levels
$NOEC_{community}$	community no-observable-effect concentration
OF	organic fluorine
ORP	oxygen reaction potential
PBSF	perfluorobutanesulfonyl fluoride
PCI	positive chemical ionisation
PFCAs	perfluoroalkyl carboxylates
PFCs	polyfluoroalkyl compounds
PFPAs	perfluoroalkyl phosphonates
PFSAs	perfluroalkyl sulfonates
PFSiAs	perfluroalkyl sulfinates
PLE	pressurised liquid extraction
POCF	perfluorooctanecarbonyl fluoride
POPs	persistent organic pollutants
POSF	perfluorooctanesulfonyl fluoride
PP	polypropylene
PTFE	polytetrafluoroethylene
PUF	polyurethane foam disk
RSD	relative standard deviation
S/N	signal to noise
SAX	strong anion exchange
SLE	solid-liquid extraction
SPE	solid-phase extraction
SPM	suspended particulate matter
TBA	tetrabutylammonium
TF	total fluorine
TN	total nitrogen
TOC	total organic carbon
TOF	time-of-flight
U.S. EPA	U.S. Environmental Prodection Agency
ww	wet weight
WAX	weak anion exchange
WWTPs	waste water treatment plants

The names and acronyms of the 40 target analytes, 20 IS and 1 InS are listed in **Table 14**.

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1. Introduction

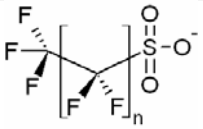
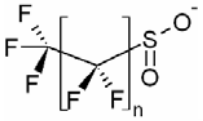
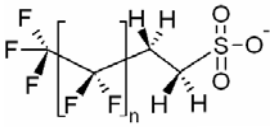
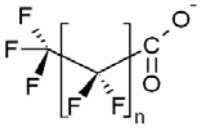
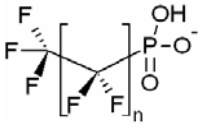
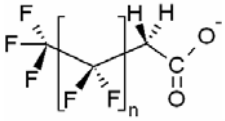
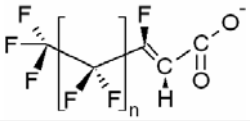
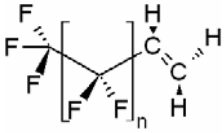
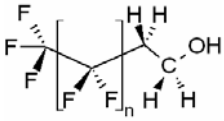
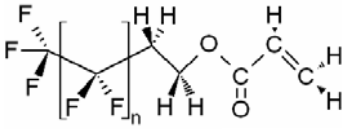
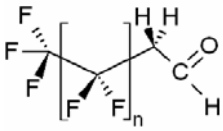
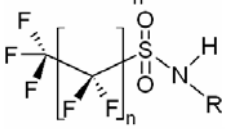
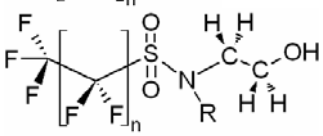
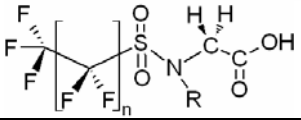
In this chapter is given an overview of polyfluoroalkyl compounds (PFCs). Firstly, the background (**1.1**), production and usage (**1.2**) and properties and environmental behaviour (**1.3**) of PFCs are described. Furthermore, an overview of the analytical methods for PFC analysis is given (**1.4**). Special emphasis is placed on the environmental concentrations (**1.5**) and temporal trends in biota (**1.6**), which represents the focus of this Ph.D. thesis. Finally, the bioaccumulation and biomagnification potential, and the toxicology of PFCs are described (**1.7**).

1.1 Background

The occurrence of PFCs has been reported the first time in human blood in 1968 by Taves 1968. Improvements of the analytical techniques during the 1990s, resulting in a characterisation of the groups of PFCs in environmental samples, and made it possible, that those PFCs were found around the globe in animals and humans (Giesy and Kannan 2001; Olsen et al. 1999). These findings had the consequence that, in 2000, the 3M Company voluntarily phased out the production of perfluorooctanesulfonyl fluoride (POSF), which is a major precursor in the synthesis of several PFCs. Due to their high persistence (P), bioaccumulation (B) and toxicity (T), perfluorooctanesulfonate (PFOS), can be classified as PBT compounds (Brooke et al. 2004). In addition, PFOS is a candidate for persistent organic pollutants (POPs) under the Stockholm Convention, caused by fulfilling the criteria persistent, toxic, bioaccumulative and their potential for long-range transportation (UNEP 2009).

PFCs comprise a wide range of different substances, consisting of a hydrophilic functional group and a hydrophobic fluorinated chain which can vary in chain length. The hydrophobic part is fully or partially fluorinated and can be linear or branched. The most investigated compounds are PFOS and perfluorooctanoic acid (PFOA). But there are several hundreds of PFCs, which can divide into the ionic and neutral PFCs (**Table 1**). The ionic PFCs include the perfluoroalkyl sulfonates (PFSAs), perfluroalkyl sulfinates (PFSiAs), fluorotelomer sulfonates (FTS), perfluoroalkyl carboxylates (PFCAs), perfluoroalkyl phosphonates (PFPAs), fluorotelomer carboxylates (FTCAs) and fluorotelomer unsaturated carboxylates (FTUCAs).

Table 1. Environmentally relevant groups of the ionic and neutral PFCs

Compound groups	Acronym	Chemical structure	Typical PFCs
Ionic PFCs			
Perfluroalkyl sulfonates	PFSAs		n = 3-9
Perfluroalkyl sulfinates	PFSiAs		n = 5, 7, 9
x:2 Fluorotelomer sulfonates	x:2 FTS		n = 5, 7, 9
Perfluoroalkyl carboxylates	PFCAs		n = 1-17
Perfluoroalkyl phosphonates	PFPAs		n = 5, 7, 9
Fluorotelomer carboxylates	x:2 FTCA		n = 5, 7, 9
Fluorotelomer unsaturated carboxylates	x:2 FTUCA		n = 4, 6, 8
Neutral PFCs			
x:2 Fluorotelomer olefins	x:2 FTolefin		n = 5, 7, 9, 11
x:2 Fluorotelomer alkohols	x:2 FTOH		n = 3, 5, 7, 9, 11
x:2 Fluorotelomer acrylates	x:2 FTA		n = 5, 7, 9
x:2 Fluorotelomer aldehydes	x:2 FTAL		n = 7
Perfluoroalkyl sulfonamides	FASAs		n = 7, R = H n = 7, R = CH_3 n = 7, R = C_2H_5 n = 3, R = CH_3
Perfluoroalkyl sulfonamidoethanols	FASEs		n = 7, R = CH_3 n = 7, R = C_2H_5 n = 3, R = CH_3
Perfluoroalkyl sulfonamidoacetic acids	FASAAs		n = 7, R = H n = 7, R = CH_3 n = 7, R = C_2H_5

Furthermore, the neutral PFCs comprise the fluorotelomer olefins (FTolefins), fluorotelomer alcohols (FTOHs), fluorotelomer acrylates (FTAs), fluorotelomer aldehydes (FTALs), perfluoroalkyl sulfonamides (FASAs), perfluoroalkyl sulfonamidoethanols (FASEs) and perfluoroalkyl sulfonamidoacetic acids (FASAAs), which are currently discussed as precursors of the ionic PFSAs and PFCAs (D'eon et al. 2006; Ellis et al. 2004; Martin et al. 2006).

1.2 Production and usage

PFCs are manufactured basically by two synthesis routes, which are illustrated in **Figure 1**. The first production process, the electrochemical fluorination (ECF), was invented in the early 1940s by Joseph Simons of the 3M Company (Simons 1950). The ECF is a free-radical process and yields a product mixture of linear and up to 30% branched isomers with even and odd numbers of carbon atoms in the chain (Giesy and Kannan 2002). The basic units of this process, perfluorooctanecarbonyl fluoride (POCF) and POSF, can convert in various of polymeric sale products (Kissa 2001). The second manufacturing process, the telomerisation, is used since the 1950s and produced exclusively linear and even-chained homologues like the FTOHs (Schultz et al. 2003). The longer-chained PFCs ($C \geq 3$) are exclusively man-made chemicals, whereas trifluoroacetic acid (TFA) has also natural sources (Frank et al. 2002).

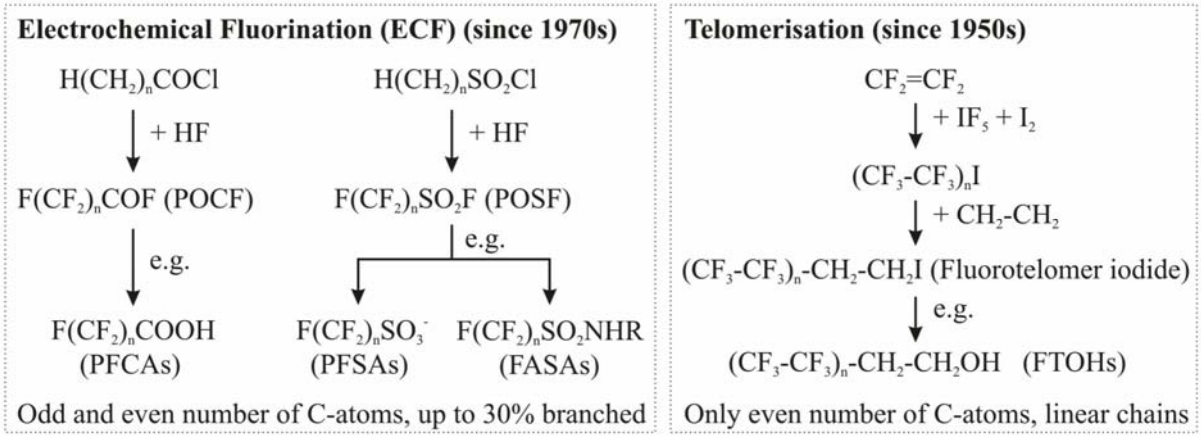


Figure 1. Electrochemical fluorination (ECF) and telomerisation manufacturing processes for PFCs

It exist only a few data about the production volume of PFCs. The 3M Company, the major producer of POSF, increased their production volume from around 300 tonnes in 1970 to over 3600 tonnes in 2000. In 2000, the 3M Company started to phase out the production of POSF. Because of the production stop by the 3M Company, Prevedouros et al. 2006 and Smithwick et al. 2006 suggested that the total global POSF production volume will decrease to zero after 2003. Conversely, Paul et al. 2009 assumed a production volume of 1000 tonnes

after 2003, because of the continuously production of POSF in Southeast Asia (**Figure 2**; Paul et al. 2009; Prevedouros et al. 2006; Smithwick et al. 2006). In 2003, the 3M Company substituted their POSF-based products by perfluorobutanesulfonyl fluoride (PBSF) (Newsted et al. 2008), because of the lower bioaccumulation potential (Martin et al. 2003b). But the production volume of the short chain PFBS is unknown. The European Union (EU) formed a directive in October 2006, which prohibits the general use of PFOS and their derivates from June 2008 (European Parliament and Council 2006).

The global historical production of PFCAs were estimated to be 4400-8000 tonnes (1951-2004) (Prevedouros et al. 2006), which was lower than the POSF-based production. The annual production of FTOHs, using the telomerisation manufacturing process, was estimated to be 5000 tonnes between 2000-2002 (Betts 2003), and increased to 11000-14000 tonnes per year after 2002 (Dinglasan-Panlilio and Mabury 2006). In 2006, the U.S. Environmental Protection Agency (U.S. EPA) launched a voluntary stewardship program to reduce PFOA and related chemicals from facility emissions and product content by 95% by 2010, and to work toward elimination of emissions and content by 2015 (U.S. EPA 2006).

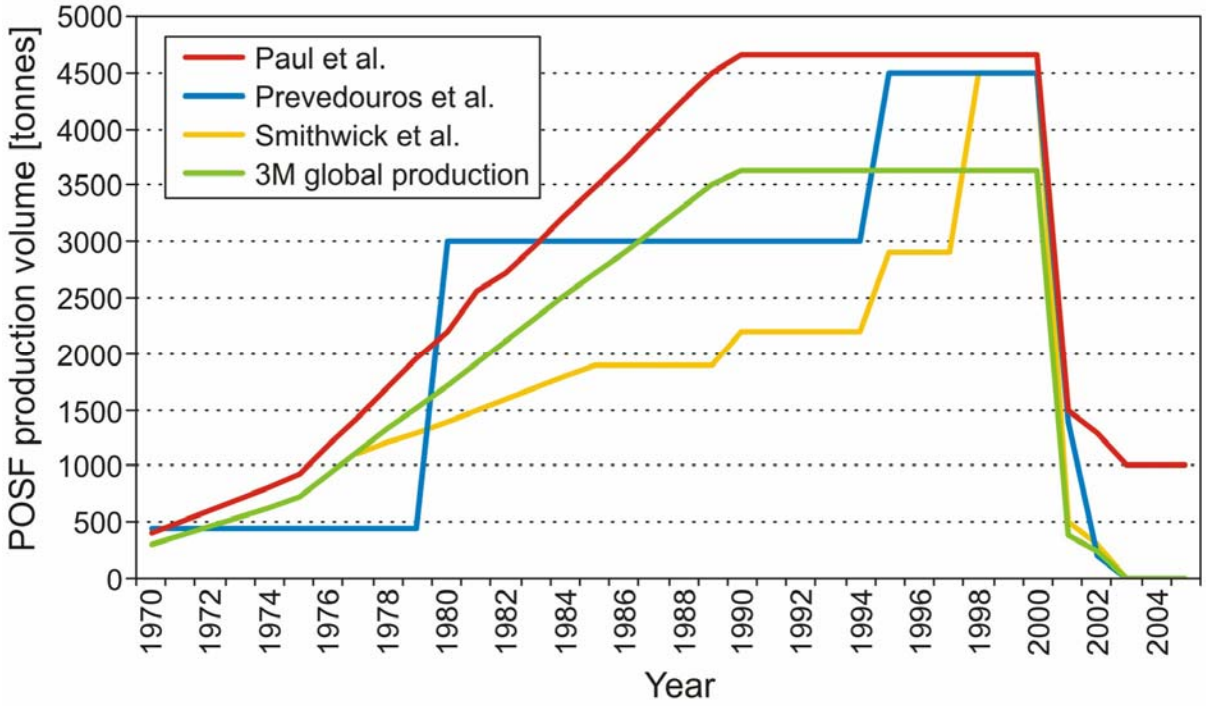


Figure 2. The total global POSF production volume of the 3M Company (1970-2002) (green line) was compared to estimates from Paul et al. 2009 (red line), Prevedouros et al. 2006 (blue line), and Smithwick et al. 2006 (yellow line). Reproduced from Paul et al. 2009

Because of their unique physicochemical properties due to their combination of lipophilic and hydrophilic characteristics, PFCs have been widely used in lots of consumer products for

over 50 years (Kissa 2001). Their surfactant properties make them suitable for aqueous fire fighting foams (AFFFs) while their lipid-and-water-repellent properties serve as stain repellents on carpets, textiles, leather, home furnishing, paper products, non-stick cookware and any kind of cleaning products. In addition, PFCs are also applied by metal plating, photographic, lubricants, varnishers, gasoline, and hydraulic fluids (Paul et al. 2009). In 2000, 3M reported that POSF-based products were used for coatings on paper and packing products (USA: 41%, EU: 33%), impregnation of textiles, leather and carpets (USA: 37%, EU: 49%), ingredients in industrial surfactants, additives and coatings (USA: 10%, EU: 15%), and AFFFs (USA: 3%, EU: 3%) (Schultz et al. 2003).

1.3 Properties and environmental behaviour

The physicochemical properties of PFCs are very limited, which makes the prediction of their environmental behaviour by modelling difficult. PFCs are very persistent, because of the strong bonding between the carbon and fluorine atom (> 450 kJ/mol) and the shielding of the carbon by the fluorine atoms. As a result PFSAs and PFCAs are resisted against degradations by acids, bases, oxidants, reductants, photolytic processes, microbes, and metabolic processes (Kissa 2001). In addition, PFCs are both lipophilic and hydrophilic, whereby they are surface active and can reduce the surface tension. These properties are very useful for the industry, but the persistency is also the reason for their global distribution in the environment (Key et al. 1997).

PFCAs and PFSAs have high water solubility, low pK_a values and therefore dissociated at environmental relevant pH values. Because of the low vapour pressure of the ions, they will be primarily found in water or bound to particles, sediment and soil (Brooke et al. 2004; Kissa 2001; Prevedouros et al. 2006). The physicochemical properties of the potassium salt of PFOS and PFOA are listed in **Table 2**.

Table 2. Physicochemical properties of PFOS potassium salt (K^+) and PFOA (free acid)

Property	PFOS K^+	PFOA	Reference
Molecular weight	538 g/mol	414.1 g/mol	
Vapour pressure	3.31 x 10^{-4} Pa (20 °C)	4.2 Pa (25 °C)	Brooke et al. 2004; Kaiser et al. 2005
Water solubility	519 mg/L (20 °C)	4.1 g/L (22 °C)	Brooke et al. 2004; U.S. EPA 2005
Melting point	> 400 °C	45-50 °C	Brooke et al. 2004; U.S. EPA 2005
Boiling point	n.a.	189-192 °C	U.S. EPA 2005
Log K_{OC}	2.57	2.06	Higgins and Luthy 2006
pK_a	-3.27	2.8	Kissa 2001; Brooke et al. 2004

n.a. = not available.

The neutral PFCs are less persistent than the PFSAs and PFCAs and can be transformed by hydrolysis, photolysis and biodegradation (Smart 2001). A biotransformation of FASAs and FASEs to PFOS was observed in rainbow trout (*Onchorhynchus mykiss*) liver microsomes (Tomy et al. 2004b) and activated sludge experiments (Boulanger et al. 2005b; Rhoads et al. 2008). In addition, the neutral PFCs have a higher vapour pressure and lower water solubility in comparison to the PFSAs and PFCAs, which make a long-range atmospheric transport for volatile neutral PFCs possible (Lei et al. 2004; Stock et al. 2004a). Smog chamber experiments have shown that FTOHs can degrade by OH-initiated oxidation pathway, with the intermediates FTALs, FTCAs and FTUCAs, to PFCAs in the atmosphere (Ellis et al. 2004), while a lifetime of approximately 20 days for the FTOHs was estimated (Ellis et al. 2003). Furthermore, in laboratory studies was found that FASEs and FASAs can degrade to PFSAs and PFCAs in the atmosphere (D'eon et al. 2006; Martin et al. 2006). A lifetime of approximately 2 days for n-methylperfluorobutane sulfonamidoethanol (MeFBSE) and > 20 days for n-methylperfluorobutane sulfonamide (MeFBSA) was supposed (D'eon et al. 2006).

Different pathways of PFCs in the environment are possible. Volatile PFCs can be transported by the atmosphere (see above), while ionic PFCs can be enter the environment directly. From the product manufacturing processes, supply chains, product use, and disposal, PFCs can be released into the aquatic environment. Sources are supposed to be dry and wet deposition, industrial and domestic waste water treatment plants (WWTPs), landfill sites, and runoff from contaminated sites (Boulanger et al. 2005a; Ellis et al. 2004; Kallenborn et al. 2004; Kim and Kannan 2007; Loewen et al. 2005; Moody and Field 1999; Stock et al. 2007). The proposed pathway of PFCs from the production and usage to the aquatic and land animals are shown in **Figure 3**.

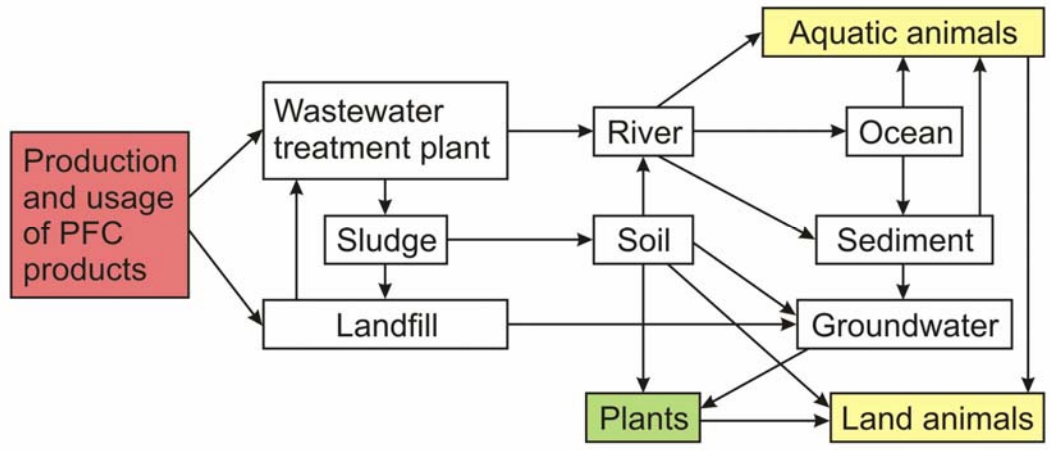


Figure 3. Environmental behaviour of PFCs from production/usage (red) to the plants (green) and organism (yellow). Note: The atmospheric pathway is not shown

The transportation pathways of individual PFCs to remote regions have not been conclusively characterised to date. Two main hypothesis were proposed for the global transportation of PFCs. Firstly, neutral, volatile precursor compounds could undergo long-range atmospheric transport and be degraded in remote regions (Ellis et al. 2004) or secondly ionic PFCs could be transported directly by oceanic currents or by means of sea-spray (McMurdo et al. 2008; Prevedouros et al. 2006).

1.4 Analytical methods

Historical analytical methods for the determination of PFCs are based on combustion and mineralisation methods. One of the first method is the "Wickbold method" which convert organic fluorine (OF) to soluble fluorine by combustion (Wickbold 1954). This method is non-specific and OF can not be detected separately. Another non-specific method for human serum determined the presence of CF_2 and CF_3 using the ^{19}F nuclear magnetic resonance (NMR) method (Taves 1968). Ellis et al. 2000 developed a ^{19}F NMR method including a gas chromatography coupled with mass spectrometry (GC-MS) confirmation for the analyses of short-chain PFCs in precipitation. The advantages of the ^{19}F NMR method are well resolved peaks, low maintenance expense and lack of matrix interferences (Kissa 2001).

The GC method is suitable for volatile PFCs using GC-MS with chemical ionisation (CI) (Martin et al. 2002). In addition, ionic PFCs can be analysed via GC by derivatisation of the compounds before measuring (Belisle and Hagen 1980; Henderson et al. 2007). Belisle and Hagen 1980 analysed PFOA in biological samples after derivatisation with diazomethane using GC coupled to an electron capture detector (ECD). Moody and Field 1999 determined PFCAs in groundwater after derivatisation with methyl iodide using GC/MS with an electron impact (EI) ionisation.

Hansen et al. 2001 developed a compound-specific method for PFCs in biological matrices using high performance liquid chromatography coupled with a tandem mass spectrometry operated in a negative electrospray mode (HPLC/(-)ESI-MS/MS). In addition, a confirmation of the exact mass was achieved by a high resolution time-of-flight (TOF)-MS. Berger et al. 2004 compared three different detection possibilities, i.e., ion trap MS, TOF-MS and triple-quadrupole MS. The TOF-MS had the best selectivity and sensitivity, but it lacked the possibilities of a triple-quadrupole MS. Alternatively, an atmospheric pressure photo ionisation (APPI) source was used by Takino et al. 2003. The application of an atmospheric pressure chemical ionisation (APCI) is not described for PFCs.

Recently a method for the determination of the total fluorine (TF), followed by fractionation of the samples to determine inorganic fluorine (IF) and OF separately, was

developed (Miyake et al. 2007a; Miyake et al. 2007b). The method based on the combustion ion chromatography (CIC) with the further development to analysed TF in low µg/L levels in aqueous and biological matrices by reducing the high background levels. This studies showed that the major contribution of the OF fraction is unknown (60-90%) indicating the presence of other PFCs in addition to the known PFCs.

In general, the most frequently instrumental method for volatile PFCs is the GC/CI-MS, while for ionic and neutral PFCs the HPLC/(-)ESI-MS/MS is mostly used (Jahnke and Berger 2009; Villagrasa et al. 2006). Over the years the detection limits, trueness, precision, and robustness for the analysis of PFCs in different matrices have been improved (Van Leeuwen and De Boer 2007; Van Leeuwen et al. 2006; Van Leeuwen et al. 2009). However, isotope-labelled internal standards (IS) should be used to check any signal enhancement/suppression or losses during sample preparation and analysis. The current sample preparation techniques for air, biological, aqueous and solid matrices are described in the following.

Air

The original method for the determination of FTOHs, FASAs and FASEs were described by Martin et al. 2002 using GC/CI-MS and HPLC/(-)ESI-MS/MS. The air samples were collected by high-volume air samplers enriched on glass-fibre filters (GFFs, particle phase) and polyurethane foam disk (PUF)/XAD-2 cartridges (gas phase). The extraction was done by shaking of the GFFs for the particle phase and by elution of the cartridges at room temperature for the gas phase. The analytical method was further optimised and validated including a larger number of target analytes and IS (Dreyer et al. 2008; Jahnke et al. 2007a). Moreover, Sasaki et al. 2003 described firstly the analysis of PFOS in airborne particulate matter.

Shoeib et al. 2005 determined neutral PFCs in indoor and outdoor air using passive air samplers with PUF disks. The method was further optimised for more volatile PFCs like FTOHs using sorbent-impregnated PUF disks (Shoeib et al. 2008). The advantage of passive air samplers in comparison to active air samplers is that they are inexpensive, simple and do not require electricity or skilled labour for operation. However, the results of passive air samplers should be checked against active air sampler.

Biological matrices

Ylinen et al. 1985 developed an ion pairing extraction (IPE) method with tetrabutylammonium (TBA) for plasma and urine samples using GC/flame ionisation detection (FID) or GC/EI-MS. This method was further optimised for environmental concentrations in biological matrices by Hansen et al. 2001 using HPLC/(-)ESI-MS/MS.

Since then, several extraction and clean-up techniques were developed for biological matrices which are described in the following.

Taniyasu et al. 2005 applied a solid-phase extraction (SPE) method using weak anion exchange (WAX) cartridges. Prior SPE the tissues were digested in potassium hydroxide and diluted in water. Flaherty et al. 2005 developed a protein precipitation sample preparation in acetonitrile using 96-well plates. Powley et al. 2005 developed a matrix-effect free extraction method for PFCAs using solid-liquid extraction (SLE) with methanol and a clean-up of the extract using bulk Envi-Carb® sorbent (purchased from Supelco). De Silva and Mabury 2006 described a method to analyse volatile derivates of PFCAs in human blood using GC/MS via negative chemical ionisation (NCI).

Berger and Haukas 2005 described a screening method for PFCs in liver samples. Basically, the compounds were extracted from homogenised samples by SLE and were injected directly into a HPLC/(-)ESI-TOF-MS system. The limit of detection (LODs) are similar to the IPE method but longer-chained PFCs and perfluorooctane sulfonamide (FOSA) can not covered by the screening method.

Comparable results for the IS of PFOS, PFOA and perfluorononanoic acid (PFNA) were obtained from three different extraction techniques (i.e., IPE, SPE, and protein precipitation sample preparation) in blood samples when matrix-matched calibration was used in quantification (Reagen et al. 2008). In contrast, quantification of IPE data using solvent based calibration curve resulted in significant analytical errors for all target analytes.

Aqueous matrices

Moody and Field 1999 described the first method for PFCAs in contaminated groundwater samples using SPE with strong anion exchange (SAX) disks. After the SPE the extracts were derivatised and analysed by GC/EI-MS. The sensitivity was further improved for the determination of PFCAs and PFSAs in surface water by using HPLC/(-)ESI-TOF-MS and ^{19}F-NMR (Moody et al. 2001).

Yamashita et al. 2004 described the problematic of background levels due to procedural and instrumental blank contaminations. The sources of contamination were identified and eliminated which made it possible to detect low pg/L levels in ocean waters. Taniyasu et al. 2005 further optimised the SPE method using Oasis® WAX cartridges and determined a wide range of PFCs including short and long-chained PFCs.

González-Barreiro et al. 2006 developed a liquid-liquid extraction (LLE) method using methyl *tert*-butyl ether (MTBE) for aqueous samples. But the LLE is limited for longer-chained PFCs ($C \geq 8$), but usually the shorter-chained PFC dominated in water samples. However, the extraction is generally done by SPE using C_{18} or anion exchange materials for

the enrichment. Scott et al. 2006a analysed PFOA by high-volume extraction on XAD-7 resin using 20-50 L water samples and subsequent derivatisation and GC/MS analysis. Another high-volume extraction method was described by Theobald et al. 2007a using HR-P resin and HPLC/(-)ESI-MS/MS analysis. On the other hand, Schultz et al. 2006a analysed waste water samples by large-volume injection (500 µL) using HPLC/(-)ESI-MS/MS. In general, the MDLs for the direct injection method are higher than for SPE, but further improvements of the instrument sensitivity will make this method applicable for routine analysis.

Solid matrices

Different methods have been developed for solid matrices like sediment, soil, sludge, dust, food and consumer products using mostly HPLC/(-)ESI-MS/MS. Schröder 2003 described three different extraction methods (i.e., soxhlet extraction, hot steam extraction, and pressurised liquid extraction (PLE)) for PFCs in sewage sludge. The PLE method resulted in the most efficient extraction method, however, no PFCs were detected in the 80 analysed sewage samples. Powley et al. 2005 developed a matrix-effect free extraction method for PFCAs in sediment, soil and sludge (see above), resulting in limits of quantitation (LOQ) of 1 ng/g dry weight. The method is characterised by their simplicity, robustness and selectivity, and therefore the method is widely used. Higgins et al. 2005 determined PFCs in sediment and sludge samples by sonication with a solvent mixture of 90:10 (v/v) methanol and 1% acetic acid in water and subsequently clean-up with C_{18} SPE cartridges. Washington et al. 2007, 2008 compared eight combinations of sample extraction pretreatments, extractions and clean-up steps on three test soils. The final method included alkaline pre-treatment, extraction with acetonitrile and water, and an IPE cleanup step.

Moriwaki et al. 2003 published an analytical method for PFOS and PFOA in vacuum cleaner dust. The extraction was done by ultrasonic extraction with methanol. Another method for vacuum cleaner dust was described by Shoeib et al. 2005 using soxhlet extraction with dichloromethane.

Tittlemier et al. 2005 described a SLE method for different food samples using hexane and acetone (2:1, v/v), followed by a silica gel column clean-up and analyses by GC/MS via positive chemical ionisation (PCI). Gulkowska et al. 2006 determined PFCs in food by using a modified IPE method from Hansen et al. 2001. Fromme et al. 2007b used for the analysis of PFCs in several food samples a combination of an ultrasonic extraction method and subsequent SPE clean-up described by Taniyasu et al. 2005.

Several methods are described for the determination of PFCs in different consumer products (i.e., textile, carpets, cookware, food packaging, and other polymeric and surfactant materials) (Larsen et al. 2006; Mawn et al. 2005; Sinclair et al. 2007; Stadalius et al. 2006).

An inventive method determined FTOHs and n-methyl perfluorooctane sulfonamidoethanol (MeFOSE) in industrially applied polymeric and surfactant materials by purging of these materials using a constant flow and trapping them on XAD resin (Dinglasan-Panlilio and Mabury 2006). The XAD resin was extracted with ethyl acetate and analysed using GC/PCI-MS.

1.5 Environmental concentrations

1.5.1 PFC levels in air

The first concentration levels of neutral PFCs in air were reported from Martin et al. 2002 in Canada using high-volume air samplers. ∑FTOHs and ∑FASAs/FASEs concentrations were higher at an urbanised site (Toronto, 171 and 320 pg/m^3, respectively), in comparison to a rural site (Long Point, 78 and 111 pg/m^3, respectively). Another study in six North American cities found a specific distribution pattern of the detected PFCs depending on the sampling sites, which indicates the importance of point sources for the spatial distribution of these compounds (Stock et al. 2004b). The concentrations of FTOHs and FASAs/FASEs in air around the globe is summarised in **Table 3**.

Shoeib et al. 2004a investigated PFCs in indoor and outdoor air. Concentrations of FASEs in indoor air were 10 to 100 times higher in comparison to outdoor air, which indicates the indoor air as a source to the outside environment. Passive air samplers consisting of PUF were effective to conduct indoor and outdoor surveys (Shoeib et al. 2005).

In the particulate phase both ionic and neutral PFCs were detected (Boulanger et al. 2005a; Jahnke et al. 2007c). Jahnke et al. 2007b described a significant correlation between the ambient temperature and the partitioning of FASEs in the gaseous and particulate phase, while FTOHs and FASAs were almost exclusively found in the gaseous phase. Ionic PFCs were found at several locations in European (Barber et al. 2007) and Atlantic Ocean air (Jahnke et al. 2007c). These findings suppose a direct atmospheric transportation of ionic PFCs on particles.

Jahnke et al. 2007c published airborne PFC concentrations in a latitudinal transect between Bremerhaven, Germany (53° N) and Cape Town, Republic of South Africa (33° S). The maximum concentration was found for 8:2 FTOH (290 pg/m^3) in the channel between France and UK, while towards South Africa the concentration of 8:2 FTOH decreased to 2.0 and 2.8 pg/m^3, respectively. These decreasing latitudinal gradient from the European continent towards South Africa indicate the industrial regions as potential sources for PFCs and transportation of PFCs through the inner tropical convergence zone (ITCZ). An altitudinal transect from 1300 m to 2740 m above sea level was reported from Loewen et al.

2008. The increasing concentrations of FTOHs and FASAs with the altitude were explained with the increasing uptake capacity of the resign at lower temperatures.

Table 3. Overview of ∑FTOHs and ∑FASAs/FASEs concentrations in air (pg/m^3) [a]

Location	Inhabitants	∑FTOHs	∑FASAs/FASEs	Reference
Toronto, ON (n=4)	2 480 000	171 [b]	320 [b]	Martin et al. 2002
Long Point, ON (n=2)	500	78 [b]	111 [b]	
Griffin, GA (n=5)	23 500	148 (49-224)	403 (57-1549)	Stock et al. 2004b
Cleves, GA (n=3)	2 200	132 (103-181)	69 (<MDL-134)	
Long Point (n=3)	500	26 (<MDL-52)	48 (29-65)	
Toronto, ON (n=3)	2 480 000	165 (113-213)	95 (31-211)	
Reno, NV (n=3)	180 500	76 (51-93)	291 (157-491)	
Winnipeg, MB (n=3)	685 900	11 (<MDL-18)	22 (15-32)	
Toronto, ON (n=2)	2 480 000	n.a.	33 (24-41)	Shoeib et al. 2004b
Ottawa, ON (n=7)	780 000	n.a.	171 (156-205)	Shoeib et al. 2005
Lake Erie (n=5)	-	n.a.	2.0 (n.d.-3.2)	Boulanger et al.
Lake Ontario (n=3)	-	n.a.	1.3 (n.d.-1.9)	2005a
Arctic atmosphere (n=20)	-	25 [b]	15 [b]	Shoeib et al. 2006
Toronto, ON (n=3)	2 480 000	81 [b]	15 [b]	
Hamburg, Germany (n=7)	1 740 000	288 (150-546)	68 (29-151)	Jahnke et al. 2007b
Waldhof, Germany (n=4)	20	181 (64-311)	34 (12-54)	
North Sea (n=1)	-	379 [c]	34 [c]	Jahnke et al. 2007c
North Atlantic Ocean (n=4)	-	46 (28-49) [c]	6.4 (2.7-11) [c]	
South Atlantic Ocean (n=3)	-	7.8 (3.3-16) [c]	0.8 (0.5-1.2) [c]	
Hazelrigg, UK (n=2+10)	-	269, 110	57 [b], 73 [b]	Barber et al. 2007
Manchester, UK (n=2+2)	458 000	535 [b], 381	69, <MQL	
Kjeller (Oslo), Norway (n=1)	573 000	63	89	
Mace Head, Ireland (n=4)	-	19	<MQL	
Sakyo (Kyoto), Japan (n=10)	1 465 000	644 (68-1959)	n.a.	Oono et al. 2008
Higashiyodogawa (Osaka) (n=10)	2 636 000	818 (270-1183)	n.a.	
Morinomiya (Osaka) (n=4)	2 636 000	2316 (364-5006)	n.a.	
German Bight (n=5)	-	38 (53-17)	30 (16-60)	Dreyer and
Hamburg, harbour, Germany (n=1)	1 740 000	180	18	Ebinghaus 2009
Hamburg, Barsbüttel, Germany (n=3)	1 740 000	117 (81-204)	16 (12-22)	
Geesthacht, Germany (n=5)	29 000	116 (32-192)	18 (4.9-32)	

[a] Sum of gas phase and particle air concentrations; minimum and maximum concentrations are given in brackets; n.d. = not detected; n.a. = not analysed; <MDL = below method detection limit; <MQL = below method quantitation limit; [b] sum of mean values; [c] mean values of duplicate samples.

1.5.2 PFC levels in solid samples

An overview of PFCs in solid samples including consumer products, food, dust, sediment and WWTP sludge is given in **Table 4** and is described in the following.

Consumer products

PFCs are used for various consumer products but only a little is known about their content and composition profile. The Danish Product Registry identified 92 different PFCs in consumer product, of which 11 were registered with a amount of over 100 kg in use in Denmark (Poulsen et al. 2008). Washburn et al. 2005 investigated the exposure of PFOA in selected consumer articles, including upholstery, textiles, sealants, garments, waxes, paints and cleaners. The maximum concentration of PFOA was found in treated apparel and home textiles (1.4 mg/kg article). Dinglasan-Panlilio and Mabury 2006 found that 0.04-3.8% (dry mass basis) of residual FASEs can left the manufacturing process of fluorinated polymers and can be potentially released into the environment.

Food

The analysis of PFCs in various food samples from Canada was carried out from Tittlemier et al. 2005, 2006. FASAs were detected in pg/g wet weight (ww) to low ng/g ww range, whereas highest concentration was found in fish, fast food and food prepared in packaging. These results were considered to indicate food as a potential exposure route of PFCs to humans (see **chapter 1.7.3**). Other studies investigated PFCAs and PFSAs in food samples and found PFOS and PFOA most frequently (Ericson et al. 2008; Tittlemier et al. 2007).

PFCs in seafood was investigated in different species in Europe (Hoff et al. 2003a; Kallenborn et al. 2004; Van de Vijver et al. 2003b), Asia (Nakata et al. 2006; Taniyasu et al. 2003) and North America (Furdui et al. 2007; Martin et al. 2004b) (for details see **chapter 1.5.4**). Del Gobbo et al. 2008 analysed PFCAs and PFSAs in raw and cooked fish samples. Interestingly, they found reduced PFC concentrations after cooking, however, it is possible that the extraction efficiency is lower for cooked fish and/or PFCs were lost by cooking residues.

Dust

Moriwaki et al. 2003 determined PFOS and PFOA in vacuum cleaner dust samples from homes in Japan in a range of 11 to 2500 ng/g dry weight (dw) and 69 to 3700 ng/g dw, respectively. Kubwabo et al. 2005 observed a positive correlation between the PFC concentration levels in dust and the using of carpeting from homes in Canada. Concentrations

of neutral PFCs (i.e., MeFOSE and n-ethyl perfluorooctane sulfonamidoethanol (EtFOSE)) were found in high concentrations of up to 8860 ng/g dw for MeFOSE and 75440 ng/g dw for EtFOSE in indoor dust samples from homes in Canada (Shoeib et al. 2005). Another study investigated FTOHs, PFCAs and PFSAs in indoor dust samples from homes in USA (Strynar and Lindstrom 2008). Maximum concentrations were found from perfluorohexane sulfonate (PFHxS) (35700 ng/g dw) and PFOS (12100 ng/g dw), while FTOHs and PFCAs were determined in a similar concentration range of tens to hundreds ng/g dw. The high concentrations of PFCs in indoor dust suggest that dust could be an important pathway for human exposure (see **chapter 1.7.3**).

Murakami and Takada 2008 investigated PFCs in fine (<63 μm) and coarse (63-2000 μm) street dust in residential areas and heavily trafficked areas in Tokyo, Japan. Significantly higher concentrations were observed in heavily trafficked areas in comparison to residential areas in the fine fraction. In addition, in heavily trafficked areas the PFC concentrations were significantly higher in the fine fraction in comparison to the coarse fraction. The street dust could be possibly the origin of the contamination of the street runoff, which could enter the water.

Sediment

Higgins et al. 2005 reported PFCs in sediment from the San Francisco Bay, USA. The PFCs showed a widespread occurrence at low ng/g to sub ng/g dw levels. In addition, a correlation of the PFC content in the sediment to total organic carbon (TOC) as well as iron oxide content was found from Higgins and Luthy 2006, indicating the importance of hydrophobic interactions.

Becker et al. 2008b reported PFOS and PFOA concentrations in sediment from the river Roter Main, Germany. A WWTP could identified as a local input source for PFCs in sediment, in which PFOS accumulated by a factor of 40 and PFOA by a factor of 3 relative to the concentration in the water. These results show that PFOS has a stronger sorption to sediment in comparison to PFOA. Furthermore, PFOS was investigated in sediment, benthic organism and higher trophic levels in the Ariake Sea, Japan. The results indicate a high bioaccumulation potential of PFOS through the coastal food chain (Nakata et al. 2006) (see also **chapter 1.7.1**).

Table 4. Overview of PFOS and PFOA concentrations in solid samples (ng/g) [a]

Country	PFOS	PFOA	Other PFCs	Remarks	Reference
Consumer products					
USA	n.a.	n.d.-1.4	n.a.	mg/kg article, various products	Washburn et al. 2005
Food					
China	0.33-14	n.d.-1.67	PFHxS; C_6, C_7, C_9-C_{11} PFCA	ww, seafood	Gulkowska et al. 2006
Canada	<MQL-2.7	<MQL-2.6	PFNA	ww, beef, fish	Tittlemier et al. 2007
	<MQL	3.6	PFHpA	ww, popcorn	
Germany	0.03-1.0	0.03-118	PFHxS; PFHxA	ww, total diet	Fromme et al. 2007b
Spain	<MQL-0.65	<MQL-0.06	PFHpA	ww, various food	Ericson et al. 2008
Canada	<MQL-1.68	<MQL-1.59	C_9-C_{12}, C_{14} PFCA	ww, raw/ cooked seafood	Del Gobbo et al. 2008
Dust					
Japan	11-2500	69-3700	n.a.	dw, vacuum cleaner dust	Moriwaki et al. 2003
Canada	n.d.-5065	n.d.-1234	PFHxS	dw, vacuum cleaner dust	Kubwabo et al. 2005
Japan	<MQL-8.1	1.2-11	PFNA; PFDA; PFUnDA	dw, fine/ coarse street dust	Murakami and Takada 2008
USA	<MQL-12100	<MQL-1960	6:2, 8:2, 10:2 FTOH; PFBS; PFHxS; C_6, C_7, C_9-C_{12} PFCA	dw, vacuum cleaner dust	Strynar and Lindstrom 2008
Sediment					
USA	n.d.-3.76	n.d.-0.25	PFHxS; PFDS; C_9-C_{12}, C_{14} PFCA; FOSAA; Me-, EtFOSAA	dw	Higgins et al. 2005
Japan	0.09-0.14	0.84-1.1	PFHxS	ww	Nakata et al. 2006
Japan	<MQL-11	<MQL-3.9	FOSA; PFDoA	dw	Senthilkumar et al. 2007
Germany	<MQL-175	<MQL-506	n.a.	dw, WWTP site	Becker et al. 2008b
WWTP sludge					
USA	14.4-2610	<MQL-29.4	PFHxS; PFDS; C_9-C_{12}, C_{14} PFCA; FOSAA; Me-, EtFOSAA	dw	Higgins et al. 2005
USA	18-160	<MQL-12	PFHxS; PFDS; C_9-C_{12}, C_{14} PFCA; Me-, EtFOSAA	dw	Schultz et al. 2006b
USA	<MQL-65	18-241	PFDA; PFUnDA	dw	Sinclair and Kannan 2006
USA	8.2-993	8.3-178	C_9-C_{12} PFCA; FOSA	dw	Loganathan et al. 2007
Germany	n.d.-120	n.d.-23	n.a.	dw	Becker et al. 2008a

n.d. = not detected; n.a. = not analysed; <MQL = below method quantitation limit.

WWTP sludge

Higgins et al. 2005 found approximately 3 orders higher concentration in WWTP sludge as in sediment, which indicates the high contamination of the sewage with PFCs. Furthermore, the two PFOS precursor compounds 2-n-methylperfluorooctanesulfonamido acetic acid (MeFOSAA) and n-ethylperfluorooctanesulfonamido acetic acid (EtFOSAA) were observed at high concentration levels, which can further biodegrade to PFOS (Rhoads et al. 2008). In addition, concentrations of PFCAs were 1 to 2 orders lower than PFSAs, showing the stronger adsorption of the PFSAs to the particles (Higgins et al. 2005). Another study found a preferred partitioning of perfluorodecanoic acid (PFDA) and perfluoroundecanoic acid (PFUnDA) to sludge in comparison to the shorter-chain PFOA (Sinclair and Kannan 2006).

Becker et al. 2008a reported that in WWTPs about a tenth of PFOA and the half of PFOS was removed by the sludge. However, similar or higher concentrations were observed in the effluent in comparison to the influent, indicating that the conventional WWTPs are not effective for removal of PFCs from the waste water (Schultz et al. 2006b). The increasing mass flow in WWTPs could be due to biodegradation of precursor compounds such as FASAs, FASEs and FTOHs (Schultz et al. 2006b; Sinclair and Kannan 2006).

1.5.3 PFC levels in aqueous samples

PFCs were ubiquitously found in aqueous samples. An overview of the PFC levels in snow, precipitation, tap water, groundwater, surface runoff, river water, lake water and waste water effluent is given in **Table 5**. In addition, the PFOA and PFOS concentrations in seawater in the open-ocean and the coastal area are illustrated in **Figure 4**.

Snow

Kim and Kannan 2007 determined PFCs in snow from Albany, USA. Snowfall was identified as a significant pathway into the lakes. PFCs was investigated in ice caps from the Canadian Arctic to study seasonal cycles, temporal trends and atmospheric fluxes (Young et al. 2007). The concentrations ranged from low to mid pg/L, with maximum concentrations in spring to summer. The concentration of PFOS decreased significantly between 1996 and 2005, while no trend was observed for the PFCAs. The presence of ionic PFCs in Arctic snow suggests atmospheric oxidation of volatile precursors as a source.

Precipitation

TFA was investigated in precipitation in several studies (Römpp et al. 2001; Wujcik et al. 1999). The sources of this short-chain PFC are photochemical degradation of chlorofluorocarbons (CFCs) and direct anthropogenic and natural emissions.

Taniyasu et al. 2008 described the analysis of 29 PFCs (including PFSAs, PFCAs, FASAs, FASAAs, FTCAs and PFUCAs) in precipitation at two locations in Japan. Dominated compounds were the short-chain TFA and perfluoropropanoic acid (PFPrA) with maximum concentrations of 75.9 ng/L (TFA) and 10.3 ng/L (PFPrA). Scott et al. 2006b determined PFCs in precipitation from nine sampling sites in North America. The short to long-chained PFCAs (C_2 to C_{12}) were detected as well as their potential precursor compounds FTCAs and FTUCAs. Interestingly, high PFOA concentration correlates with air masses coming from urban areas. Potential precursor compounds of PFCAs were detected in precipitation from Kyoto, Japan (Mahmoud et al. 2009) and Winnipeg, Canada (Loewen et al. 2005). These results suggest that neutral PFCs can remove through oxidation and wet deposition from the atmosphere.

Tap water

PFOS and PFOA was investigated in drinking water from Japan (Harada et al. 2003; Saito et al. 2004). Harada et al. 2003 detected PFOS concentrations usually less than 4 ng/L, except of one sampling site with maximum concentrations of 50.9 ng/L. The origin of the contamination was possibly the Tama River, which was contaminated with PFOS. Loos et al. 2007 found that the concentration in drinking water correlate with concentrations in the lake Maggiore, Italy, indicating an insufficient performance of the waterworks for PFCs in this region. Skutlarek et al. 2006 determined PFCs in drinking water in the Ruhr area, Germany. The maximum concentration was 598 ng/L for the ∑PFCs, with PFOA as the dominant compound. The high concentration originated from with soil improver contaminated agriculture land, which PFC contamination reached the drinking water.

Groundwater

Moody and Field 1999 determined PFCAs at two fire-training locations in USA, which is the first study of PFCAs in aqueous samples. C_6 to C_8 PFCA were detected ranging from 125 to 7090 µg/L. These extremely high concentrations can be explained by the using of AFFFs, which contains high levels of PFCs. But the still high concentrations even after 7 to 10 years inactivity indicate the high persistency of PFCs in the aqueous environment. Furthermore, Moody et al. 2003 investigated PFCs in groundwater at Wurtsmith Air Force Base, Canada.

In this respect, AFFFs were also identified as a source for PFCs, resulting in concentrations of low to high µg/L for PFHxS, PFOS and PFOA, respectively.

Plumlee et al. 2008 described PFCs in groundwater in California, USA. The ∑PFC concentration ranged from 20 to 150 ng/L with PFOS and PFOA as the dominated compounds. The overlying urban stream possibly contaminated the groundwater by infiltration.

Surface runoff

Kim and Kannan 2007 investigated PFCs in surface runoff in Albany, USA, to identify potential sources into lake water. ∑PFCs ranged from 1.11 to 81.8 ng/L in surface runoff, but the mass balance analysis in an urban lake suggests that the surface runoff is not the dominant input pathway into the lake and an unknown source must exist. Another study identified street runoff as a potential source of PFCAs into the aqueous environment (Murakami et al. 2009).

River water

PFC concentrations in river surface water were reported in several studies. Saito et al. 2004 investigated PFOS and PFOA in river samples all over Japan. The concentration of PFOA was higher than of PFOS, especially at contaminated sampling sites. McLachlan et al. 2007 determined PFCAs in 14 major rivers in Europe. Highest concentration was detected for PFOA in the river Po (200 ng/L), which is in agreement with the detected concentrations in another survey in the river Po watershed (Loos et al. 2008). The total discharge of PFOA from the European rivers was estimated to be 14 tonnes/year (McLachlan et al. 2007). Another study analysed PFCAs and PFOS in over 100 European rivers (Loos et al. 2009). Highest concentrations of PFOA was detected in the rivers Danube in Austria (25 ng/L), Scheldt in Belgium (88 ng/L) and Netherlands (73 ng/L), Rhone in France (116 ng/L), and Wyre in the UK (100 ng/L), while PFOS showed the highest concentration in the rivers Scheldt in Belgium (154 ng/L) and the Netherlands (110 ng/L), Seine in France (97 ng/L), Krka in Slovenia (1371 ng/L), Severn in the UK (238 ng/L), and Rhine in Germany (32 ng/L). In general, these studies show the widespread occurrence of PFCs in Japanese and European rivers and the large geographical differences in their levels.

Moody et al. 2001 found very high ∑PFCs concentration (11 to 2,270,000 ng/L). PFOS was the predominant compound in surface water collected from Toronto in Canada released from an AFFF spill. Hansen et al. 2002 determined PFOS and PFOA in the Tennessee River, USA. The effluent of a fluorochemical manufacturing facility could identify as a source for PFCs into the river. Skutlarek et al. 2006 found maximum ∑PFCs concentrations of 4385 ng/L in the Ruhr area, Germany. The origin of this contamination was contaminated soil

improver which was applied on agriculture land and released into the river by surface and subsurface runoff. So et al. 2007 investigated 14 PFCs in the Pearl and Yangtze River in China. Both rivers had a different composition profile which indicates dissimilar origins of the sources. WWTPs were identified as local sources for PFCs in the Cape Fear River Basin in North Carolina, USA (Nakayama et al. 2007), in the Glatt Valley Watershed in Switzerland (Huset et al. 2008), and in several rivers in Japan (Murakami et al. 2008). Ahrens et al. 2009c determined 20 PFCs in the river Elbe in Germany. Highest concentrations were found in the urban area in Hamburg, which indicates domestic and industrial waste water as a potential source for PFCs (for details see **chapter 7.1**)

In summary, different sources were responsible for the ubiquitous distribution of PFCs in surface water in rivers. Sources for PFCs into the rivers could be AFFF spill, fluorochemical manufacturing effluents, WWTP effluents and runoff, while the spatial distribution and the composition profile of individual PFCs can be used to identify the origin of the contamination.

Lake water

Boulanger et al. 2004 investigated PFCs in Great Lakes water. The dominated compounds were PFOS (21-70 ng/L) and PFOA (27-50 ng/L). In addition, some precursor compounds like EtFOSAA, FOSA and perfluorooctane sulfinate (PFOSi) were detected. Stock et al. 2007 determined PFCs in lakes in the Canadian Arctic, where airport waste water was identified as a local source into the lakes. PFC concentrations in lake water from two other studies ranged from mid pg/L to mid ng/L in Sri Lanka (Guruge et al. 2007) and Lake Victoria, Kenya, respectively (Orata et al. 2009). In general, the presence of PFCs in remote lakes indicates the ubiquitous distribution of PFCs in the aqueous environment.

Table 5. Overview of PFOS and PFOA concentrations in aqueous samples and potential sources (ng/L)

Country	PFOS	PFOA	Other PFCs	Source of PFCs	Reference
Snow					
USA	<MQL-1.93	<MQL-20	PFHxS; C_7, C_9-C_{12} PFCA; FOSA; 6:2, 8:2 FTS	atmospheric deposition	Kim and Kannan 2007
Canada	0.002-0.09 [a]	0.01-0.15 [a]	C_9-C_{11} PFCA	atmospheric deposition	Young et al. 2007
Precipitation					
Canada	0.59 [a]	n.d.	PFHxS; 8:2, 10:2 FTCA; 8:2, 10:2 FTUCA	degradation from volatile precursors	Loewen et al. 2005
North America	n.a.	<MQL-89	C_2-C_7, C_9-C_{12} PFCA; 8:2, 10:2 FTCA; 8:2, 10:2 FTUCA	degradation from volatile FTOHs	Scott et al. 2006b
Japan	0.13-1.0	1.0-3.8	C_2-C_7, C_9-C_{12} PFCA; 8:2 FTCA; 6:2, 8:2, 10:2 FTUCA; FOSA, EtFOSAA	degradation from volatile precursors	Taniyasu et al. 2008
Tap water					
Japan	0.1-50.9	n.a.	n.a.	Tama river	Harada et al. 2003
Japan	n.d.-12[a]	0.12-40	n.a.	river water	Saito et al. 2004
Germany	n.d.-22	n.d.-519	PFBS; C_4-C_7 PFCA	runoff from contaminated soil	Skutlarek et al. 2006
Italy	6.2-9.7	1.0-2.9	C_7, C_9-C_{12} PFCA	Lake Maggiore	Loos et al. 2007
Groundwater					
USA	n.a.	n.d.-6570000	PFHxA; PFHpA	AFFFs	Moody and Field 1999
USA	19-87	n.d.-18	PFHxS; PFDS; C_6, C_7, C_{10} PFCA; FOSA; EtFOSAA	infiltration from overlying urban stream	Plumlee et al. 2008
Surface runoff					
USA	<MQL-15	0.51-29	PFHxS; C_7, C_9-C_{12} PFCA; FOSA; 6:2, 8:2 FTS	surface, rain	Kim and Kannan, 2007
Japan	2.9-12	n.d.-174	C_7, C_9-C_{12}, C_{14} PFCA; FOSA	atmospheric deposition, dust	Murakami et al. 2009
River water					
USA	17-144	<MQL-598	n.a.	fluorochemical manufacturing facility	Hansen et al. 2002
Japan	0.24-37	0.1-456	n.a.	various sources	Saito et al. 2004
Germany	n.d.-193	n.d.-3640	PFBS; C_4-C_7 PFCA	runoff from contaminated soil	Skutlarek et al., 2006
China	0.15-99	0.85-260	PFBS; PFHxS; C_6, C_7, C_9-C_{11} PFCA; FOSA	industrial/ municipal wastewater effluent	So et al. 2007
Europe	n.a.	<MQL-200	C_6, C_7, C_9 PFCA	various sources	McLachlan et al. 2007
Germany	0.18-8.2	2.9-12.5	18 other PFCs	various sources	Ahrens et al. 2009c
Lake water					
Canadian Arctic	0.9-57	0.5-16	PFHxS; PFDS; C_7, C_9-C_{12} PFCA; 8:2, 10:2 FTUCA	atmosphere, airport waste water	Stock et al. 2007
Lake Victoria	<MQL-2.5	0.4-12	n.a.	industrial/ municipal wastewater effluent	Orata et al. 2009
Waste water effluent					
USA	3-68	58-1050	PFHxS, C_9-C_{11} PFCA, 8:2 FTCA, 8:2 FTUCA	waste water	Sinclair and Kannan 2006
Landfill effluent					
Finland, Norway	30-187	91-516	PFBS; PFHxS; C_6, C_9 PFCA; FOSA	landfill	Kallenborn et al. 2004
Denmark	<MQL-3.8	<MQL-5.8	PFHxS	landfill	Bossi et al. 2008

n.d. = not detected; n.a. = not analysed; <MQL = below method quantitation limit; [a] mean values.

Waste water influent and effluent

Several studies investigated the mass flow of PFCs in WWTPs (Becker et al. 2008a; Loganathan et al. 2007; Schultz et al. 2006b; Sinclair and Kannan 2006). All studies found similar or higher concentrations of PFCs in the effluent in comparison to the influent concentration. These results indicate that conventional WWTPs are not effective for removal of PFCs and biodegradation of precursor compounds could lead to increasing concentrations of PFCAs and PFSAs.

Schultz et al. 2006b observed increasing concentrations of PFOS and perfluorodecane sulfonate (PFDS) in ten WWTPs in the USA occurred from trickling filtration and activated sludge treatment. Similarly, Sinclair and Kannan 2006 and Loganathan et al. 2007 determined an increase in the mass flow of PFCs in effluent waters, relative to the influent concentration in the USA. In addition, Loganathan et al. 2007 found no significant seasonal variation in the mass flow of PFCs in a WWTP. Furthermore, Becker et al. 2008a determined a 20-fold higher PFOA concentration and a 3-fold higher PFOS concentration in WWTP effluents as in the influents of these plants.

In general, effluents of WWTPs are potential point sources of PFCs into the aqueous environment. The per capita discharge was estimated to be 57 µg/day/person for PFOS and 12 µg/day/person for PFOA (Huset et al. 2008).

Landfill effluent

Only a few data exist about PFCs in effluent water from landfill sites (Bossi et al. 2008; Kallenborn et al. 2004). Bossi et al. 2008 found low concentrations of PFCs in two landfill effluents in Denmark. The individual PFC concentration was in general lower than 5.8 ng/L (PFOA), which indicate them not as an important source into the aqueous environment. Conversely, Kallenborn et al. 2004 found very high PFC contamination in landfill effluents in Finland and Norway with ∑PFC concentrations of 199 to 1537 ng/L. However, consumer product can contain high levels of PFCs (Dinglasan-Panlilio and Mabury 2006; Washburn et al. 2005) and landfill were indicated as a significant sink for PFCs (Paul et al. 2009). Further systematic studies of different landfill sites are necessary to characterise landfill sites as a significant or insignificant source for PFCs into the environment.

Sea water

Only a few data exist about seawater measurements. An overview about open-ocean and coastal seawater concentrations is shown in **Figure 4**. Detected concentrations are usually around some tens pg/L to few ng/L, depending on the location and the compound.

Approximately two orders higher concentrations were found in the coastal area in comparison to the open-ocean.

Taniyasu et al. 2003 investigated PFSAs in coastal seawater around Japan. Maximum PFOS concentration was found in the Tokyo Bay with 59 ng/L, while the other PFSAs were below the method detection limit (MDL). So et al. 2004 determined PFCs in seawater in the Pearl River Delta (China), coastal area of Hong Kong and Korea. The concentrations ranged in low ng/L range except of one sampling location close to the urbanised and industrial city Seoul with a maximum concentration of 730 ng/L for PFOS. Similar concentration levels were observed for PFOS and PFOA in the coastal area of Dalian (China) (Ju et al. 2008). Caliebe et al. 2004 determined 8 PFCs in the North Sea, with highest concentrations of PFOA (~13 ng/L). The occurrence and composition profile of 15 PFCs was investigated in surface water in the North Sea, Baltic Sea and Norwegian Sea. The composition profile was influenced from local sources caused by human activities, whereas atmospheric depositions were negligible, but it could have possibly an influence on low contaminated sites like the open North Sea or Norwegian Sea. (for further details see **chapter 7.3**). Furthermore, the spatial distribution of 18 PFCs was investigated in surface water in the German Bight. The ∑PFC concentration decreased with increasing distance from the coast, indicating the rivers and coastal area as a potential source for PFCs (for further details see **publication I**).

The global occurrence of PFCs in open-ocean water was described firstly from Yamashita et al. 2005. Yamashita et al. 2005 collected samples from the North and Mid Atlantic Ocean in 2002 to 2004, and found concentration levels of several tens pg/L for PFHxS, PFOS and PFNA to a few hundreds pg/L for PFOA. A similar study from Theobald et al. 2007b was carried out from 53° N to 30° S in the Atlantic Ocean in 2005. The concentration of PFOA and PFOS were in a range of a few tens pg/L with a maximum concentration of 170 pg/L for PFOS. Overall, the ocean currents have a high influence on the occurrence of PFCs in the Atlantic Ocean (for further details see **publication II**). Concentrations of PFOS and PFOA reported in the Mid to South Pacific Ocean and the Indian Ocean were about one magnitude lower than in the North Atlantic Ocean (Wei et al. 2008). Furthermore, Yamashita et al. 2008 have studied vertical profiles of several PFCs in the Labrador Sea, Mid Atlantic Ocean, South Pacific Ocean and Japan Sea. It was hypothesised that PFCs could be transported globally with the thermohaline circulation system, and the open-ocean water is acting as a final sink for PFOS and PFOA.

In general, the concentration of PFOA is usually higher than of PFOS, which suggest that similar sources come from the urbanised/industrial coastal area. In addition, the higher level of PFOA in seawater could be explained with its higher water solubility (Brooke et al. 2004;

U.S. EPA 2005), lower bioaccumulation potential (Martin et al. 2003a) and lower sorption potential to sediment (Higgins and Luthy 2006). On the other hand, the PFOS concentration in the Antarctic coastal water was higher than of PFOA. These findings suggest that PFOS and its precursor compounds are mainly transported via the atmosphere or sea-spray, while PFOA is mainly transported by the ocean currents.

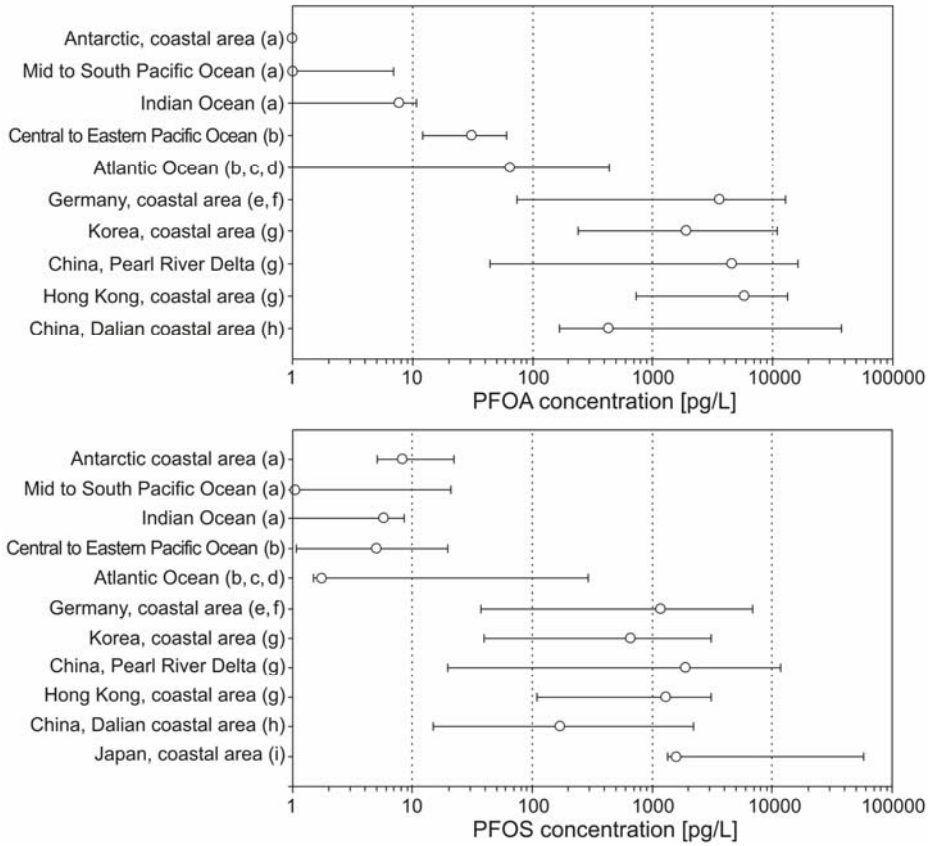


Figure 4. Concentrations (minimum, maximum, median (circles)) of PFOA and PFOS in seawater in the open-ocean and the coastal area in pg/L ((a) Wei et al. 2008, (b) Yamashita et al. 2005, (c) Theobald et al. 2007b, (d) **publication II**, (e) Caliebe et al. 2004, (f) **publication I**, (g) So et al. 2004, (h) Ju et al. 2008 and (i) Taniyasu et al. 2003). Note: Concentrations below the method quantitation limit are given as 0.5 of the method detection limit

1.5.4 PFC levels in wildlife

Global distribution

The global distribution of PFOS in wildlife was described firstly from Giesy and Kannan 2001. In general, concentrations of PFOS in animals from industrialised regions like North America and Europe are greater than from remote regions such as the Arctic. In addition, fish-eating animals have higher concentration levels than their diets, indicating the bioaccumulation of PFCs to higher trophic levels. Numerous studies showed the ubiquitous occurrence of PFCs along the food chain, whereas highest concentrations were found in mammals. An overview of the global distribution of PFOS in mammal is given in **Figure 5**.

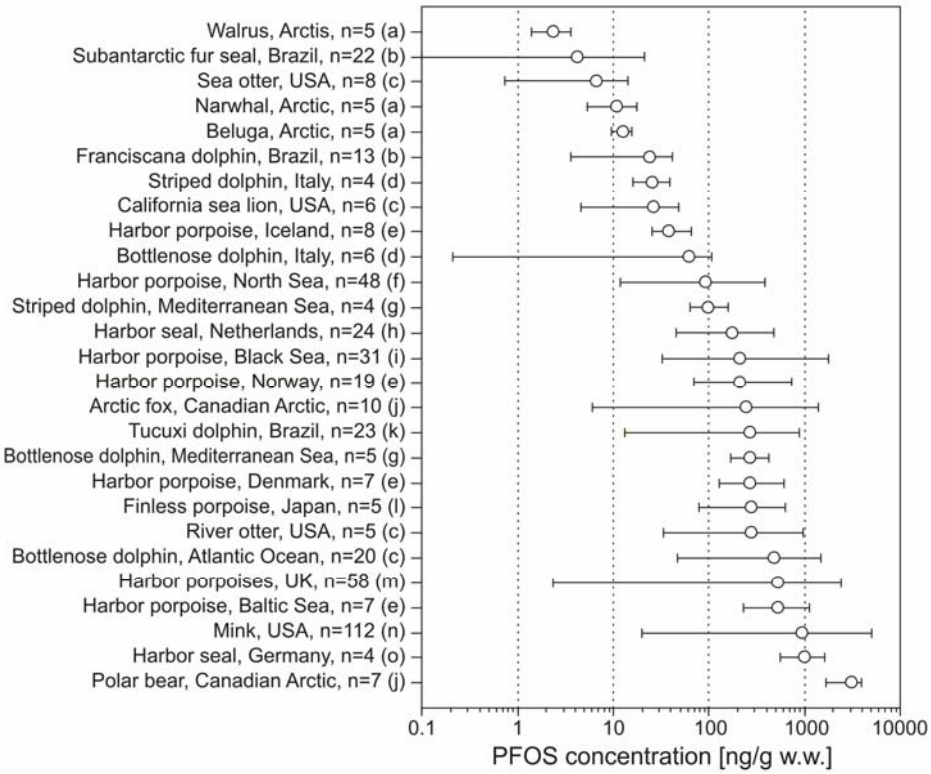


Figure 5. Hepatic concentrations (minimum, maximum, mean (circles)) of PFOS in mammals from (a) Tomy et al. 2004a, (b) Leonel et al. 2008, (c) Kannan et al. 2001b, (d) Kannan et al. 2002b, (e) Van de Vijver et al. 2004, (f) Van de Vijver et al. 2003b, (g) Giesy and Kannan 2001, (h) Van de Vijver et al. 2005, (i) Van de Vijver et al. 2007, (j) Martin et al. 2004a, (k) Dorneles et al. 2008, (l) Nakata et al. 2006, (m) Law et al. 2008, (n) Kannan et al. 2002c, and (o) **publication III** (ng/g ww). Note: Concentrations below the method quantitation limit are given as 0.5 of the method detection limit

Kannan et al. 2001a investigated PFOS in water birds from USA and central Nord Pacific Ocean. High PFOS concentrations were found in bald eagle (*Haliaeetus leucocephalus*) blood plasma with 13 to 2200 ng/mL from the USA, while concentrations in black-footed and laysan albatross sera (i.e., *Diomedea nigripes*, *Diomedea immutabilis*, respectively) ranging from 3 to 34 ng/mL. Furthermore, PFHxS, PFOS, PFOA and FOSA were analysed in bird liver samples from several species from Japan and Korea with highest PFOS concentrations in common cormorants (*Phalacrocorax carbo*) (650 ng/g ww) (Kannan et al. 2002a). In addition, extremely high PFOS concentrations were found in American mink (*Mustela vison*) livers from the USA (maximum concentration of 5140 ng/g ww) (Kannan et al. 2002c).

PFC concentrations were investigated in various species in the Arctic region. Martin et al. 2004a determined PFCs in the food web (mammals, birds, and fishes) in the Canadian Arctic. PFOS was the major contaminant with over 4000 ng/g ww in polar bears (*Ursus maritimus*). For the first time longer-chained PFCAs (C_8-C_{15}) were determined in biota, whereas PFNA

was the dominated compound. Furthermore, Tomy et al. 2004a analysed PFOS, PFOA, and the precursors EtFOSA, FOSA in the eastern Arctic marine food web (marine mammals, birds, fishes, shrimps, clams, and zooplankton). PFOS was detected in all analysed species ranging from 0.08 ng/g ww (two different clam species) to 33.2 ng/g ww (glaucous gulls (*Larus hyperboreus*)). Bossi et al. 2005b reported PFC concentrations in fish, birds and marine mammals from Greenland and the Faroe Islands. PFOS was the predominant compound in all species. Finally, Smithwick et al. 2005a determined PFCs in liver tissues and blood of polar bears from five locations in the North American Arctic. In this respect, the South Hudson Bay and East Greenland had significantly higher PFOS concentrations than western populations such as the Chukchi Sea, suggesting sources from Europe and Eastern North America.

Further data from remote regions are given by Tao et al. 2006. It was determined PFC concentrations in nine albatross species, elephant seals (*Mirounga leonine*), Adelie penguins (*Pygoscelis adeliae*) and polar skuas (*Stercorarius maccormicki*) from the Southern Ocean and North Pacific Ocean. In Adelie penguins no PFCs were detected, while in the other animals PFOS was the major contaminant. This study shows detectable levels in the Southern Hemisphere fauna, however, the concentration in this area are 10 to 100-fold lower than e.g. in seals and birds from the Arctic region.

Recently, an increasing number of publications of PFCs in wildlife from China were published. Dai et al. 2006 reported concentrations of PFOS and PFOA in red panda (*Ailurus fulgens*) and giant panda (*Ailuropoda melanoleuca*) blood samples from 7 different locations in China. The concentrations ranged from 0.76 to 73.80 ng/mL for PFOS and 0.32 to 8.20 ng/mL for PFOA for both species. Concentrations of PFCs in serum samples of Amur tigers (*Panthera tigris altaica*) was nearly one order of magnitude lower than of red and giant pandas (Li et al. 2008a). Another study from China investigated PFCs in eggs of three bird species from south China (Wang et al. 2008). 11 PFCs were detected with PFOS as the predominant compound (14.4-343 ng/g ww). PFOS concentrations were in the same range as in guillemot (*Uria aalge*) eggs from the Norwegian coast, Iceland and the Faroe Islands, whereas the concentrations in the Baltic Sea were higher (Löfstrand et al. 2008).

Van de Vijver et al. 2003b analysed PFOS in invertebrates (starfishes (*Asterias rubens*), crabs (*Carcinus maenas*), and shrimps (*Crangon crangon*)) from the Western Scheldt estuary and the southern North Sea. An increasing concentration gradient of PFOS was found along the Western Scheldt estuary to the harbour of Antwerp, indicating the industrial area of Antwerp as a source of PFCs. Another study from invertebrates were published from Cunha et

al. 2005, who reported PFOS concentrations in mussels (*Mytilus galloprovincialis*) from 10 Portuguese estuaries ranging from 36.8 to 117.8 ng/g ww.

Hepatic concentrations of PFCs were investigated in harbor porpoises (*Phocoena phocoena*) from different locations in Europe and Black Sea coast (Van de Vijver et al. 2007; Van de Vijver et al. 2004). A decreasing geographical trend from south to north was observed. Highest concentrations of PFOS were found in the Black Sea, German Baltic Sea and coastal areas near Denmark, while lower concentrations were observed in animals from remote areas like Iceland and Norway.

Keller et al. 2005 described PFC concentrations in plasma of loggerhead sea turtles (*Caretta caretta*) and kemp's ridley sea turtles (*Lepidochelys kempii*) from southeastern coast of the USA. PFOS and PFOA were the dominant compounds with mean concentrations of 11.0 ng/mL and 3.20 ng/mL for loggerhead turtles and 39.4 ng/mL and 3.57 ng/mL for Kemp's ridley turtles, respectively. The ∑PFC concentration was significantly higher in Kemp's ridley turtles than loggerhead turtles, higher in larger turtles and higher in turtles captured toward the north, which suggest an influence of the bioaccumulation by species, age, and habitat.

Hart et al. 2008b determined PFCs in livers of skipjack tuna (*Katsuwonus pelamis*) collected from Pacific offshore waters and the open-ocean along the Sea of Japan, East China Sea, Indian Ocean, and Western North Pacific Ocean. PFOS and PFUnDA were the predominant compounds in tuna at concentrations of <1-58.9 and <1-31.6 ng/g ww, respectively. 10 to 20-fold higher mean concentrations of PFOS was found in skipjack tuna from offshore sites (i.e., Japan, Taiwan, and Indonesia) as from open-ocean waters (i.e., the mid Pacific Ocean and the Indian Ocean), which reflected the concentrations previously reported in seawater samples from these areas (Wei et al. 2008; Yamashita et al. 2005). These findings suggest that tuna are good bioindicators of the contamination by PFOS in the marine environment (Hart et al. 2008b). The concentration of PFOS and PFOA in Mediterranean swordfish (*Xiphias gladius*) liver were below the LOD of 1.5 and 3 ng/g ww, respectively (Corsolini et al. 2008).

Leonel et al. 2008 investigated PFC concentrations in Franciscana dolphins (*Pontoporia blainvillei*) and Subantarctic fur seals (*Arctocephalus tropicalis*) from southern Brazil. Maximum concentrations were found for PFOS with 42 ng/g ww and 21.6 ng/g ww for Franciscana dolphins and Subantarctic fur seal, respectively. Moreover, higher PFOS concentrations were observed in marine tucuxi dolphins (*Sotalia guianensis*) from the Brazilian coast ranging between 43 and 2431 ng/g ww (Dorneles et al. 2008).

Tissue distribution

Relatively little is known about the tissue distribution of PFCs in organisms. For the calculation of the total body burden the concentration in the liver and plasma are often used. These estimations are often potential sources of errors because little is known about the distribution of PFCs in the whole body (Houde et al. 2006c). In addition, bioaccumulation evaluations may be overestimated when using liver and plasma concentrations. In **Table 6** is given an overview of the tissue distribution of PFOS and PFOA in wildlife.

Martin et al. 2003a determined the compound-specific tissue distribution of rainbow trout (*Oncorhynchus mykiss*) in a laboratory flow-through system exposed with PFCs. Highest PFC concentrations were found in blood > kidney > liver > gall bladder, while lowest in gonads > adipose > muscle tissue. Similarly, van de Vijver and co-workers observed decreasing PFOS concentrations in the order kidney > liver > blubber > skeletal muscle for harbor seals (*Phoca vitulina*) from the Dutch Wadden Sea (Van de Vijver et al. 2005) and liver > kidney > muscle > brain ≈ blubber for harbor porpoises from the Black Sea, respectively (Van de Vijver et al. 2007). Verreault et al. 2005 investigated the occurrence of PFCs in different tissues in glaucous gulls (*Larus hyperboreus*) from the Norwegian Arctic. PFOS was the predominant compound with highest concentrations in plasma (48.1-349 ng/g ww), followed by liver ≈ egg > brain. Olivero-Verbel et al. 2006 analysed different tissues of pelicans (*Pelecanus occidentalis*) from North Columbia. Interestingly, highest PFOS concentrations were detected in spleen, followed by liver > lung > kidney > brain > heart > muscle. This could be important in term of the effects, because the immune system and physiological functions were controlled by the spleen. Holmström and Berger 2008 showed different tissue distribution of PFOS and the ∑PFCAs in guillemots from the Baltic Sea. The highest concentration of PFOS was observed in egg tissue followed by liver tissue, while highest concentrations of ∑PFCAs were found in the liver, suggesting a compound specific accumulation mechanism.

The total body burden and tissue distribution was investigated in harbor seals from the German Bight. PFOS was the predominant compound in all measured seal tissues with a composition of over 90% compared to the ∑PFCs. The dominant PFCAs were PFNA and PFDA. The mean whole body burden in harbor seals of all detected PFCs was estimated to be 2665 ± 1207 µg absolute. The major amount of the total PFCs burden in the bodies was in blood (38%) and liver (36%), followed by muscle (13%), lung (8%), kidney (2%), blubber (2%), heart (1%), brain (1%), thymus (<0.01%) and thyroid (<0.01%) (for further details see **publication III**).

Table 6. Tissue distribution of PFOS and PFOA in wildlife (ng/g tissues ww and ng/mL for blood and bile)

Species	Location	PFOS	PFOA	Other PFCs	Reference
Egg					
Glaucous gull	Norwegian Arctic	52-196	<0.7	C_{10}-C_{13} PFCA	Verreault et al. 2005
Guillemot	Baltic Sea	243-432	n.d.	PFHxS; PFDS; C_9-C_{15} PFCA; FOSA	Holmström and Berger 2008
Blood					
Glaucous gull	Norwegian Arctic	48-349	<0.7-0.74	PFHxS; C_8-C_{15} PFCA	Verreault et al. 2005
Harbor seal	German Bight	48-887	n.d.-1.1	16 other PFCs	**publication III**
Liver					
Harbor porpoise	Black Sea	33-1790	n.d.	C_9-C_{12} PFCA	Van de Vijver et al. 2007
Harbor seal	Dutch Wadden Sea	46-488	<MQL	C_9-C_{11} PFCA	Van de Vijver et al. 2005
Guillemot	Baltic Sea	91-322	n.d.	PFDS; C_9-C_{15} PFCA; FOSA	Holmström and Berger, 2008
Pelican	Columbia	4.0-56	n.d.	PFHxS	Olivero-Verbel et al., 2006
Harbor seal	German Bight	559-1665	n.d.-1.4	16 other PFCs	**publication III**
Kidney					
Harbor seal	Dutch Wadden Sea	47-1036	<MQL-12	C_9-C_{12} PFCA	Van de Vijver et al. 2005
Pelican	Columbia	1.2-17	n.d.	FOSA	Olivero-Verbel et al., 2006
Harbor porpoise	Black Sea	2.6-1371	n.d.	n.d.	Van de Vijver et al. 2007
Guillemot	Baltic Sea	92-183	n.d.	PFHxS; PFDS; C_9-C_{15} PFCA; FOSA	Holmström and Berger, 2008
Harbor seal	German Bight	118-383	n.d.-0.93	16 other PFCs	**publication III**
Heart					
Pelican	Columbia	1.7-6.9	n.d.	FOSA	Olivero-Verbel et al., 2006
Harbor seal	German Bight	87-181	n.d.-0.99	16 other PFCs	**publication III**
Lung					
Pelican	Columbia	2.9-11	n.d.	PFHxS; FOSA	Olivero-Verbel et al., 2006
Harbor seal	German Bight	228-755	0.28-1.2	16 other PFCs	**publication III**
Brain					
Pelican	Columbia	1.3-11	n.d.	FOSA	Olivero-Verbel et al., 2006
Harbor porpoise	Black Sea	3.5-100	n.d.	n.d.	Van de Vijver et al. 2007
Harbor seal	German Bight	38-153	n.d.-0.20	16 other PFCs	**publication III**
Thymus					
Harbor seal	German Bight	159-416	0.43-0.93	16 other PFCs	**publication III**
Thyroid					
Harbor seal	German Bight	n.d.-121	n.d.-0.22	16 other PFCs	**publication III**
Spleen					
Harbor seal	Dutch Wadden Sea	152-439	<MQL	PFBS; C_9-C_{11} PFCA	Van de Vijver et al. 2005
Pelican	Columbia	6.2-132	<MQL-182	FOSA	Olivero-Verbel et al., 2006
Bile					
Mugil incilis (Fish)	Columbia	3673[a]	<50-1116	PFHxS; FOSA	Olivero-Verbel et al. 2006
Pelican	Columbia	17-100	n.d.	n.d.	
Blubber					
Harbor seal	Dutch Wadden Sea	19-297	<MQL	n.d.	Van de Vijver et al. 2005
Harbor porpoise	Black Sea	18[a]	n.d.	n.d.	Van de Vijver et al. 2007
Harbor seal	German Bight	n.d.-23	n.d.-0.08	16 other PFCs	**publication III**
Muscle					
Harbor seal	Dutch Wadden Sea	8.9-2725	<MQL	C_9-C_{11} PFCA	Van de Vijver et al. 2005
Pelican	Columbia	0.7-2.7	n.d.	FOSA	Olivero-Verbel et al., 2006
Harbor porpoise	Black Sea	41[a]	n.d.	n.d.	Van de Vijver et al. 2007
Guillemot	Baltic Sea	9.8-17	n.d.	C_9-C_{14} PFCA	Holmström and Berger, 2008
Harbor seal	German Bight	7.7-132	n.d.-0.24	16 other PFCs	**publication III**

n.d. = not detected; <MQL = below method quantitation limit; [a] mean values.

In general, highest concentrations were found in blood, kidney and liver which confirms the findings that PFCs bind to blood proteins instead of fatty tissue (Jones et al. 2003). But the tissue distribution is compound-specific and varies between the different species. Further investigations on the accumulation potential and whole body burden in marine wildlife are necessary to assess potential adverse effects of PFCs.

1.5.5 PFC levels in humans

The presence of OF has been reported the first time in human blood by Taves (1968) in 1968. But until the end of 1990s little attention was paid to the occurrence of these compounds. The public attention increased due to the observation of high PFOS concentrations in serum in occupationally exposed workers of 1750 to 2190 ng/mL, whereas serum levels of the general population were about 100 times lower (Hansen et al. 2001; Olsen et al. 1999). Since then PFCs were detected worldwide in human blood, milk and liver. An overview of the PFOS and PFOA concentrations in humans is given in **Table 7**.

Olsen et al. 2003a analysed PFCs in a total of 645 adult donor serum samples from American Red Cross blood, USA. The mean concentration of PFOS was higher in males (37.8 ng/mL) than in females (31.3 ng/mL). The other detected PFCs were approximately an order of magnitude lower than PFOS. In another study from the USA, the mean PFOS concentration in serum of elderly humans (age of 65–96) was 31.0 ng/mL, whereas the PFOS concentration in most elderly was slightly lower (Olsen et al. 2004).

Kannan et al. 2004 investigated PFOS, PFHxS, PFOA and FOSA in 473 human blood, serum and plasma samples collected from several countries (i.e., USA, Colombia, Brazil, Belgium, Italy, Poland, India, Malaysia, and Korea). PFOS was the predominant compound with highest concentrations in the samples collected from the USA and Poland (> 30 ng/mL), while the concentration was moderate in Korea, Belgium, Malaysia, Brazil, Italy, and Colombia (3 to 29 ng/mL) and the lowest concentration was found in India (<3 ng/mL). Kubwabo et al. 2004 reported concentrations of PFOS in 56 human serum samples from Canada in the same range as from the USA and Poland. Kärrman et al. 2006a determined 12 PFCs in 40 pooled serum samples from 3802 Australian residents. Interestingly, it was observed increasing concentrations of PFOS with increasing age. The PFOS concentration in Australia was similar to the concentration in Korea, Belgium, Malaysia, Brazil, Italy, and Colombia which suggests that the exposure from local sources is more important than emissions from the Northern Hemisphere. Yeung et al. 2006 observed PFOS concentrations in whole blood from nine cities in China which was comparable with the concentration level in USA and Poland. Fromme et al. 2007a found a lower PFC exposure in adult German people than in USA and Canadian people. Ultimately, different composition profiles of PFCs indicate

specific exposure sources and pathways of PFCs to humans in different countries (Yeung et al. 2008).

Calafat et al. 2006 investigated the PFC concentration level in three different ethnicities (non-Hispanic blacks, non-Hispanic whites, and Mexican Americans) in the USA. The results indicate different patterns of human exposure to PFCs between the ethnic groups. Interestingly, higher education was associated with higher contamination with PFOS and PFOA. A similar study showed, that serum samples from children from USA showed lower PFC concentrations for Mexican Americans than for the other two ethnic groups (Kato et al. 2009)

Tao et al. 2008a investigated PFCs in 45 human breast milk samples collected from the USA. PFOS and PFOA were the predominant compounds. It was found a higher partitioning of PFOA to milk than for PFOS. In addition, PFOA was significantly higher in milk of mothers nursing for the first time than in the milk of mothers who have previously nursed. These results suggest that PFOA can be excreted in breast milk and therefore the concentrations of PFCs in women decreased with increasing period of breastfeeding. In another study, Tao et al. 2008b determined PFCs in human breast milk from several Asian countries. The concentration of PFOS varied from different Asian countries significantly, whereas the lowest median concentration was found in India (39.4 pg/mL), and the highest in Japan (196 pg/mL). Another possible pathway of PFCs in the human foetus could be the maternal transfer via the umbilical cord (Inoue et al. 2004).

In summary, PFCs were detected in whole blood, serum, plasma, liver and milk all over the world. In the USA and Canada were detected generally higher concentrations than in other parts of the world. But no differences between urban and rural regions were observed (Kärrman et al. 2006a). However, the comparison of PFC levels in different tissues should be made with caution because of the compound specific ratio between whole blood, serum, plasma and liver (Kärrman et al. 2006b; Olsen et al. 2003b). Furthermore, no clearly trend between the concentration level and age was found. In addition, different studies showed that males are higher contaminated with PFCs than females (Harada et al. 2004; Midasch et al. 2006; Olsen et al. 2003a). The reasons for the gender differences are possibly different dietary habits or different elimination rates after the uptake. However, the extractable total OF in blood samples from China ranged between > 70% for Beijing, to 30% for Jintan, indicating that it exist a substantial amount of unidentified PFCs in human blood samples (Yeung et al. 2008).

Table 7. Overview of PFOS and PFOA concentrations in human blood, milk (ng/mL) and liver (ng/g ww)

Country	n	Year	Age	Sample type	PFOS	PFOA	Other PFCs	Reference
USA	65	-	-	serum	6.7-82	<5-35	PFHxS; FOSA	Hansen et al. 2001
USA	645	2000-2001	20-69	serum	<4.3-1656	<1.9-52	PFHxS; FOSAA; MeFOSAA	Olsen et al. 2003a
USA	24	-	5-74	serum	<6.1-58	<3.0-7.0	PFHxS; FOSA	Olsen et al. 2003b
	30		5-74	liver	<4.5-57	<MQL-47		
USA	238	-	65-96	serum	3.4-175	1.4-17	PFHxS; FOSAA; MeFOSAA	Olsen et al. 2004
USA	175		17-72	serum, whole blood, plasma	<1.3-164	<3.0-88		Kannan et al. 2004
Columbia	56		20-29	whole blood	4.6-14	3.7-12		
Brazil	29		18-74	whole blood	4.3-35	<20		
Italy	50		20-59	serum	<1-10	<3		
Poland	25	1998-2004	35-58	whole blood	16-116	9.7-40	PFHxS; FOSA	
Belgium	20		19-63	plasma	4.5-27	<1-13		
India	45		17-48	serum	<1-3.1	<3-3.5		
Malaysia	23		21-26	whole blood	6.2-19	<10		
Korea	50		15-95	whole blood	3.0-92	<15-256		
Japan	38		23-66	serum	4.1-40	<6.8-12		
Japan	15	2003	17-37	maternal blood	4.9-18	<0.5-2.3	n.a.	Inoue et al. 2004
	15			cord blood	1.6-5.3	<0.5		
Canada	56	2002	>20	serum	3.7-65	<1.2-7.2	n.a.	Kubwabo et al. 2004
Sweden	66	1997-2000	19-75	whole blood	1.7-37	0.5-12	PFHxS; PFDS; C_6, C_9-C_{11} PFCA; FOSA	Kärrman et al. 2006b
Australia	40	2002-2003	5 age groups	pooled serum samples	13-30	5.0-9.9	PFHxS; FOSA; PFNA	Kärrman et al. 2006a
Germany	105	2003-2004	5-84	plasma	6.2-131	1.7-39	n.a.	Midasch et al. 2006
USA	54	2001-2002	4 age groups	pooled serum samples	10-40 [a]	2.1-6.0 [a]	PFHxS; PFNA; MeFOSAA; EtFOSAA; FOSA	Calafat et al. 2006
China	85	2004	7-66	whole blood	1.7-155	0.1-3.5	PFHxS; C_9-C_{11} PFCA; FOSA	Yeung et al. 2006
Germany	356	2005	14-67	plasma	2.1-56	0.5-19	n.a.	Fromme et al. 2007a
USA	1562	1999-2000	4 age groups	pooled serum samples	30 [a]	5.2 [a]	PFHxS; C_9-C_{12} PFCA; MeFOSAA; EtFOSAA; FOSA;	Calafat et al. 2007
Sweden	12	2004	22-33	serum	8.2-48	2.4-5.3	PFHxS; PFDS; C_9-C_{11} PFCA; FOSA	Kärrman et al. 2007
	21	1996-2004	19-40	pooled milk samples	0.06-0.47	<0.21-0.49		
Germany	691	2006	5-69	plasma	1.0-93	0.7-100	PFBS; PFHxS;	Hölzer et al. 2008
Germany, Hungary	70	1996-2006	-	milk	0.10-0.64	<0.20-0.46	n.a.	Völkel et al. 2008
USA	45	2004	22-43	milk	<0.03-0.62	<0.03-0.16	PFBS; PFHxS; C_7, C_9-C_{12} PFCA	Tao et al. 2008a
Asia, USA	184	1999-2005	17-40	milk	<0.01-0.52	<0.04-0.34	PFBS; PFHxS; C_7, C_9 PFCA	Tao et al. 2008b

n.a. = not analysed; <MQL = below method quantitation limit; [a] mean values.

1.6 Temporal trends

The first temporal trend study was reported from Kannan et al. 2002b in white-tailed sea eagle (*Haliaeetus albicilla*) livers from Germany and Poland. The PFOS concentration increased significantly over the time, however, no statistical method was described. Since then several temporal trend studies were performed in different tissues in wildlife (i.e., eggs, livers, and whole fish homogenates) and humans (i.e., whole blood, plasma, serum, and milk). An overview of temporal trends of PFOS and PFOA in wildlife and humans is given in **Figure 6** and **Figure 7**, respectively.

Temporal trends in wildlife

Martin et al. 2004b described significant increasing PFOS concentrations by a factor of 4.2 in lake trout (*Salvelinus namaycush*) whole body homogenates from the Lake Ontario between 1980 and 2001. But the increasing trend was not linear due to an influence of the food web by the invasion of zebra mussels (*Dreissena polymorpha*). Another study in lake trout whole body homogenates observed significant increasing PFOS concentrations between 1979 and 1993 (Furdui et al. 2008). But the PFDS and FOSA concentration decreased from 1993 to 2004 while the PFCA concentration firstly increased (1979 to 1988), and thereafter decreased.

A long-term assessment (1968-2003) in guillemot eggs from the Baltic Sea was published from Holmström et al. 2005. The results have shown an almost 30-fold increase of the PFOS concentration with a decrease after 2002. Interestingly, even in the oldest samples could detect PFOS which indicate an input of PFOS in the marine environment before 1968.

In the following several temporal trend studies of different species from the Arctic region were described. Bossi et al. 2005a found for PFOS, PFDA, and PFUnDA significant increasing concentrations in ringed seal (*Phoca hispida*) livers between 1986 and 2003 in East Greenland and between 1982 and 2003 in West Greenland, respectively. In addition, significantly higher PFOS concentrations were observed in ringed seals from East Greenland. Smithwick et al. 2006 described exponential increasing concentrations for PFOS and PFCAs (C_9-C_{11}) in polar bear livers from the North American Arctic between 1972 and 2002. In contrast, the FOSA concentration decreased over this period. Two publications from Butt et al. 2007a,b showed temporal trends in ringed seals from the Arviat and Resolute Bay, and two seabird species (i.e., thick-billed murres (*Uria lomvia*) and northern fulmars (*Fulmaris glacialis*)) from Prince Leopold Island in the Canadian Arctic. The PFCA (C_9-C_{15}) concentration in ringed seal livers increased significantly between 1972 and 2005, whereas the concentrations of PFOS and FOSA showed a maximum between 1998 and 2000, and

afterwards significant decreasing concentrations from 2000 to 2005 (Butt et al. 2007b). In the two seabird species the PFC concentration increased overall between 1975 and 2003/2004, while the PFCA concentration appeared to remain steady between 1993 and 2004 (Butt et al. 2007a). Finally, Dietz et al. 2008 reported significant increasing concentrations of PFOS and PFCAs (C_8-C_{13}) in polar bear livers from East Greenland between 1984 and 2006.

Kannan et al. 2006 described significant increasing concentrations of PFOA in sea otter (*Enhydra lutris*) livers between 1992 and 2002 collected from the California coast, USA. The PFOS concentration increased from 1992 to 1998 and then decreased after 2000. Verreault et al. 2007 observed overall increasing concentrations of PFSAs (C_6, C_8, and C_{10}) and PFCAs (C_8-C_{11}) in herring gull (*Larus argentatus*) eggs from two geographically isolated colonies in northern Norway between 1983 and 2003. However, the concentration of PFHxS and PFOS were relatively constant between 1993 and 2003. Hart et al. 2008a investigated the temporal trends in melon-headed whale (*Peponocephala electra*) livers collected along the coast of Japan from 1982 to 2006. The ΣPFC concentrations increased by a factor of ~10 from 1982 to 2001/2002, while after 2001/2002 the ΣPFC concentrations did not decline further. Ishibashi et al. 2008a found significant increasing concentrations of PFOS, PFNA and PFDA in Baikal seal (*Pusa sibirica*) livers collected in 1992 and 2005. Decreasing concentrations were reported for C_5-C_7 PFSAs, PFOSi, FOSA and PFOA in harbor seal livers collected from the German Bight between 1999 and 2008 (**publication IV**). This study showed for the first time generally decreasing concentrations for these compounds in an industrial area.

In general, high variations in the concentration levels were observed depending on the species and sampling locations. Previous temporal trend studies indicated mostly increasing concentrations of PFCs before around the year 2000 (see **Figure 6**). Thereafter a few studies have shown significant decreasing concentrations, which indicate that source inputs of PFCs to the environment have changed or have been reduced.

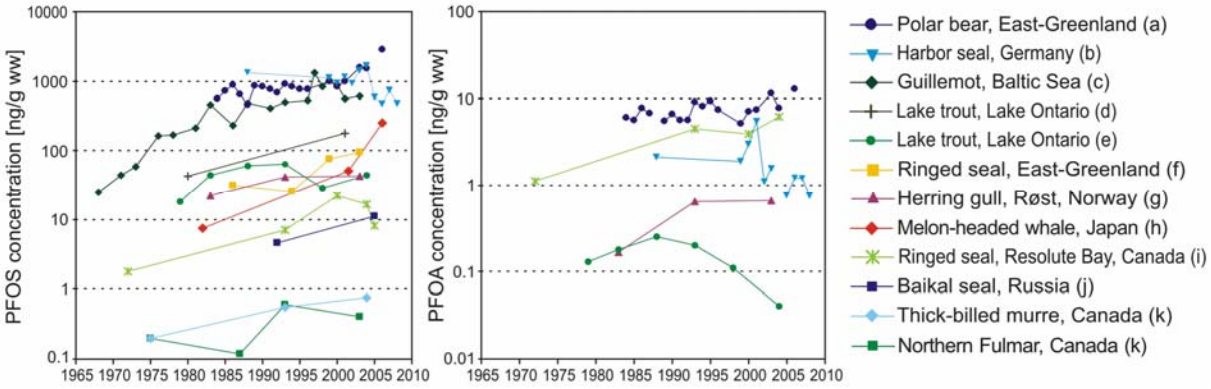


Figure 6. Overview of temporal trends of PFOS and PFOA in wildlife from (a) Dietz et al. 2008, (b) **publication IV**, (c) Holmström et al. 2005, (d) Martin et al. 2004b, (e) Furdui et al. 2008, (f) Bossi et al. 2005a, (g) Verreault et al. 2007, (h) Hart et al. 2008a, (i) Butt et al. 2007b, (j) Ishibashi et al. 2008a, and (k) Butt et al. 2007a (ng/g ww)

Temporal trends in humans

Harada et al. 2004 found increasing concentrations of PFOS and PFOA in human serum samples by a factor 3 and 14, respectively, at two locations in Japan between 1977 and 2003. However, at one location only the PFOA concentration increased significantly between 1991 and 2003. Olsen et al. 2005, 2008 assessed the temporal trend of PFCs in human serum and plasma from the USA between 1974 and 2006. The PFC concentrations increased from 1974 to 1989 significantly, and thereafter declined from 2000/2001 to 2006. Kärrman et al. 2007 found no significant temporal trends of PFOS and PFHxS in milk samples from Sweden between 1996 and 2004. However, some samples were collected from different regions, whereby regional differences in the concentration level could be important. Harada et al. 2007 analysed human serum from Japan between 1983 and 1999. While the PFOA concentration increased over the sixteen years, the PFOS concentration reached a plateau in the late 1980s. Spliethoff et al. 2008 investigated the temporal trends of PFCs in whole blood samples from the New York State, USA. The concentration level of PFOS, FOSA, PFHxS, and PFOA declined exponential significantly after the year 2000. Recently, Haug et al. 2009 described the temporal trends of PFCs in pooled serum samples from Norway between 1976 and 2007. The concentrations of PFSAs (C_6-C_8) and PFCAs (C_8-C_{11}) increased from 1976 to the mid 1990s where the concentrations reached a plateau. After around the year 2000 the concentrations of the compounds PFHpS, PFOS and PFOA started to decrease.

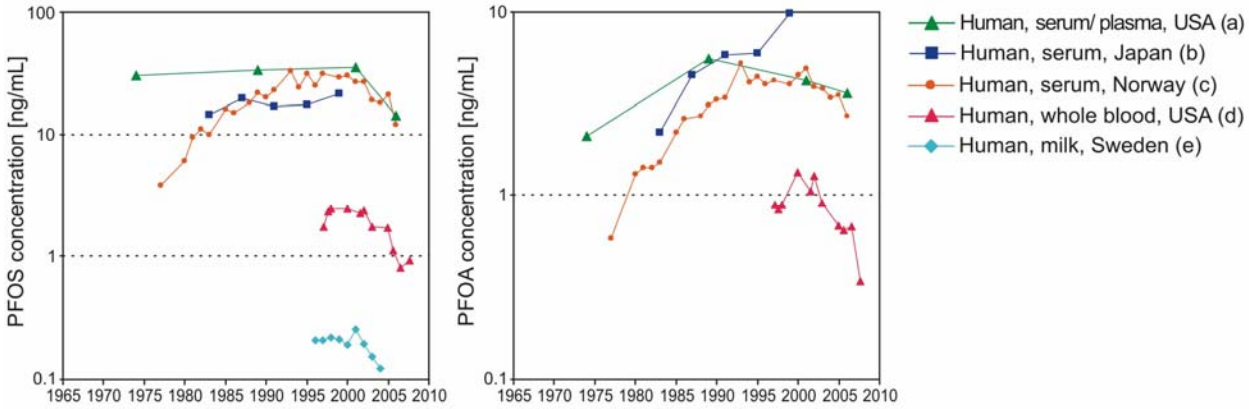


Figure 7. Overview of temporal trends of PFOS and PFOA in humans from (a) USA (Olsen et al. 2005; Olsen et al. 2008), (b) Japan (Harada et al. 2007), (c) Norway (Haug et al. 2009), (d) USA (Spliethoff et al. 2008) and (e) Sweden (Kärrman et al. 2007) (ng/mL)

In summary, the temporal trend studies in wildlife and humans showed overall increasing concentration in the past (the oldest samples come from guillemot eggs from 1968), where after a plateau was reached in the mid 1990s to 2000, and finally the concentration levels decreased after around 2000 (see **Figure 6** and **Figure 7**). The reason could be, probably, that this is the effect of the phase-out in POSF production by the 3M Company (see **Figure 2**), the reduction of PFOA emissions by the stewardship program from the U.S. EPA and replacement of PFOS and their derivates by less bioaccumulative compounds because of the formed directive from the EU (Prevedouros et al. 2006; European Parliament and Council 2006; U.S. EPA 2006). However, the still high contamination of PFCs in wildlife and humans indicates that further works on the reduction of environmental emissions of PFCs are necessary.

1.7 Toxicology

PFCs are very persistent and have the potential to bioaccumulate in the food web. The human and ecotoxicological effects were investigated in various studies. However, most studies focussed only on the compounds PFOS and PFOA. Additional toxicity information are needed for other PFCs and more exposed species. This chapter gives an overview on the bioaccumulation and biomagnification potential of PFCs (**1.7.1**), their human and ecotoxicology (**1.7.2**) and their current assumed risk to human health (**1.7.3**).

1.7.1 Bioaccumulation and biomagnification potential

The term “bioaccumulation” describes the potential of contaminants to travel through the various trophic levels of the ecosystem, whereby it will takes into account that PFCs accumulate through multiple exposure routes and that the total accumulation depends upon

the rate of intake versus elimination. A difference can be made between bioconcentration factors (BCFs), biomagnification factors (BMFs) and bioaccumulation factors (BAFs). The BCFs describe the direct uptake of a substance by living organisms from the medium (e.g., water), whereas the BMFs result from dietary uptake. The BAFs are the sum of these two processes. An overview of the three different bioaccumulation factors is given in **Table** 8.

Table 8. Overview of the BCFs, BMFs and BAFs of PFOS and PFOA

Species	PFOS	PFOA	Other PFCs	Remarks	Reference
Bioconcentration factors (BCFs)					
Rainbow trout	1100-5400	4.0-27	C_{10}-C_{12}, C_{14} PFCA, PFHxS	laboratory-based	Martin et al. 2003a
Benthic invertebrates	1000	n.a.	n.a.	Great Lakes	Kannan et al. 2005
Biomagnification factors (BMFs)					
Mink	11-23	n.a.	n.a.	laboratory-based	Kannan et al. 2002c
Food web	2.9	0.41	C_9-C_{14} PFCA, FOSA	Lake Ontario	Martin et al. 2004b
Food web	0.4-9	0.04-2.7	FOSA, EtFOSA	Eastern Arctic	Tomy et al. 2004a
Food web	5-20	n.a.	n.a.	Great Lakes	Kannan et al. 2005
Bottlenose dolphins food web	0.8-35	1.8-13	C_9-C_{12} PFCA, PFHxS, FOSA,	South Carolina, and Florida, USA	Houde et al. 2006b
Food web	0.32-38.7	n.a.	PFHxS, PFNA	Barent Sea	Haukas et al. 2007
Food web	7.0-8.7	n.a.	C_{10}-C_{12} PFCA	Western Arctic	Powley et al. 2008
Bioaccumulation factors (BAFs)					
Rainbow trout	6300-125000	n.a.	n.a.	field-based, contaminated site	Moody et al. 2002
23 fish species	274-41600	n.a.	n.a.	field-based, Japan	Taniyasu et al. 2003

n.a. = not analysed

Martin et al. 2003a investigated the accumulation potential of PFCs in a laboratory flow-through system, where rainbow trouts were exposed with PFCs. The BCFs ranged between 4 (PFOA) and 23000 (PFTeDA) in rainbow trout carcasses. The PFSAs had higher BCFs than the PFCAs relatively to their perfluoro carbon chain length. In addition, the BCFs of PFCAs increased by a factor of 8 for each additional perfluoro carbon chain between C_8 and C_{12} PFCAs. However, PFOA and shorter-chain PFCAs can not be classified as "bioaccumulative" (Conder et al. 2008). Kannan et al. 2005 calculated a BCF of approximately 1000 in various organisms in the benthic food web.

Martin et al. 2003b found no dietary accumulation for PFCs in rainbow trouts in a laboratory study. Another study investigated the BMFs in the food web of the Lake Ontario (Martin et al. 2004b). It was reported a high contamination of the benthic organisms possibly caused by contaminated sediment, which led to a underestimation of the BMFs. Tomy et al.

2004a found that PFOS biomagnify in the Arctic marine food web. The BMFs of the other PFCs (i.e., EtFOSA, FOSA, and PFOA) were often above 1, however no significant relationship with the trophic level was observed. Houde et al. 2006b described the biomagnification of PFCs in the food web of bottlenose dolphin at two locations in the USA (i.e., South Carolina, and Florida). The BMFs ranged between <1 and 156 based on the PFC concentration in the bottlenose dolphin plasma and liver. It was suggested that the calculation of the BMFs are possibly overestimated because only the plasma and liver concentration was used for the calculation of the whole body burden, while the tissue distribution of PFCs in this marine top predator was not known. Haukas et al. 2007 reported the biomagnification potential of PFCs in the Barent Sea food web. The BAFs were over 1 for PFHxS, PFNA and PFOS. Several further dietary studies exist in the food web, which show all that PFCs can potential biomagnify in the marine food web (Powley et al. 2008; Bossi et al. 2005b; Li et al. 2008b).

Moody et al. 2002 described the bioaccumulation of PFOS at an airport in Toronto, Canada, which was contaminated with AFFF spill. The BAF for PFOS ranged between 6300 and 125000 based on the concentration in the rainbow trout liver and surface water. A similar study found BAFs of 274-41600 for PFOS in surface water and liver of 23 fish species in Japan (Taniyasu et al. 2003).

1.7.2 Human and ecotoxicology

PFCs do not accumulate in fatty tissues due to their combination of lipophilic and hydrophilic characteristics. Instead they bind to the blood protein serum albumin (Austin et al. 2003; Han et al. 2003) and are therefore primarily distributed in blood, liver, kidney and other organs (Seacat et al. 2003; Seacat et al. 2002; **publication III**). Several studies investigated the half-life of PFCs in different species (Lau et al. 2007; Brooke et al. 2004). The half-life of PFOA ranged between a few days to 100 days in rats (Johnson et al. 1984). Similar half-lifes were calculated in chickens (*Gallus gallus*) with 4.6 days for PFOA and 125 days for PFOS (Yoo et al. 2009). Longest half-lifes were estimated in humans with 8.7 (2.3-21.3) years for PFOS and 4.4 (1.5-13.5) years for PFOA (Burris et al. 2002).

The mechanism of toxicity of individual PFCs is not well understood. The exposure of animals to PFOS leads to reduced body weight, increased liver weight, hepatoxicity, peroxisome proliferation, reduction of serum cholesterol, fatty acid transport and metabolism, inhibit of the gap junctional intercellular communication (GJIC), mitochondrial biogenesis, alteration of hepatic lipid metabolism, and neuroendocrine effects (Austin et al. 2003; Berthiaume and Wallace 2002; Hu et al. 2002; Ikeda et al. 1985; Luebker et al. 2002a). The toxicity of PFOA is based on peroxisome proliferation, and influences on mitochondrial,

microsomal, cytosolic enzymes, and the fatty acid transport and metabolism (Luebker et al. 2002; Berthiaume and Wallace 2002; Goecke-Flora and Reo 1996; Intrasuksri et al. 1998; Stevenson et al. 2006). Several other PFCs are expected to be peroxisome proliferators (Goecke-Flora and Reo 1996; Luebker et al. 2002). In addition, Maras et al. 2006 found up-regulation of the estrogen receptor as a consequence of the exposure with 6:2 FTOH or 8:2 FTOH in in vitro assays. Recently, Joensen et al. 2009 investigated the influence of the serum PFC levels on the semen quality. Interestingly, they found a positive correlation with high PFOS and PFOA concentrations and low semen quality.

The acute, subchronic and chronic toxicity of PFCs were investigated in several studies (Hekster et al. 2003; Nakayama et al. 2005). The acute toxicity of single injections was evaluated in rats (Olson and Andersen 1983). The LD_{50} (a lethal dose at which 50% of subjects will die) was 189 mg/kg for PFOA and 41 mg/kg for PFDA. Similar LD_{50} was found for PFOS elsewhere (Nakayama et al. 2005). In general, a greater toxicity of FTCAs and FTUCAs were described than for the PFCAs (Phillips et al. 2007). In addition, a dependence of the toxicity with the chain length was observed for PFCAs, however, the PFCAs have a relatively low acute toxicity (Mulkiewicz et al. 2007). The subchronic toxicity was investigated in a 6 month study of male cynomolgus monkeys (*Macaca fascicularis*) (Butenhoff et al. 2002). Mortalities were observed in monkeys treated with 20-30 mg PFOA/kg/day. Seacat et al. 2002 reported decreasing body and liver weights, and lowered serum total cholesterol, triiodothyronine, and estradiol levels in a 6 month study of cynomolgus monkeys feeding with 0.75 mg PFOS/kg/day. Chronic toxicity studies are very limited. Biegel et al. 2001 reported a carcinogenic potential to liver and pancreas of PFOA in rats.

The toxicity of PFCs in the aquatic environment was tested in laboratory microcosms and in the natural environment. Sanderson et al. 2002 investigated the community no-observable-effect concentration ($NOEC_{community}$) for freshwater zooplanktons. A reduction of 90-100% after an exposure of 30 mg/L PFOS over one week or 10 mg/L PFOS after two weeks was observed. In another study was found that an increasing concentration of PFOS and PFOA have an influence on the species contribution in the zooplankton community (Sanderson et al. 2004). Boudreau et al. 2003 investigated for PFOS the $NOEC_{community}$ for zooplankton in a 35 day study and the inhibition concentration (IC_{50}) for *Lemna gibba* in outdoor microcosms. The $NOEC_{community}$ and IC_{50} were determined to be 3.0 mg/L and 19.1 mg/L, respectively. The investigation of the toxicity of PFCs in different fish species indicates hepatic damage in bib (*Trisopterus luscus*) (Hoff et al. 2003a), influence on the membrane structure in common carp (*Cyprinus carpio*) (Hoff et al. 2003b), and impact on the reproductivity of fathead minnow

(*Pimephales promelas*) (Oakes et al. 2004).

Kannan et al. 2006 found a significant association between infectious diseases and PFOS and PFOA concentrations in sea otter livers. Another study observed physiological changes in Baikal seals depending on the PFCA concentration levels (Ishibashi et al. 2008b). However, no correlation was found between the PFC concentration and health status of harbor seals (**publication IV**), indicating the dependence of toxic effects on the species. Further research is necessary to evaluate long-term consequences of exposure for individual and ∑PFCs.

1.7.3 Human health risk assessment

Human exposure to PFCs based on several environmental and product-related sources. In addition, age and gender-specific pathways to humans are possible. The exposure of PFCs could cause by food, drinking water, hand-to-mouth intake, food contact, dust, and inhalation. In addition, the maternal transfer via the umbilical cord and milk is possible. The precursor compounds of PFCAs and PFSAs should be included in the risk assessment, because of their degradation potential. The contribution of PFOS and PFOA precursor compounds on the intake dosis was estimated to be 2–8% in an intermediate scenario and 28–80% in a high-exposure scenario (Vestergren et al. 2008). Trudel et al. 2008 expected a mainly intake by contaminated food and drinking water in a modelling based assessment, while consumer products had a minor contribution. However, further studies on the pathways of exposure are necessary.

Kärrman et al. 2007 investigated the PFC concentrations in human milk and serum in 12 primiparous women in Sweden. The PFOS milk concentration was on average 1% of the corresponding serum level which indicates an elimination of PFCs through lactation. The maternal transfer to the infant was calculated to be 200 ng PFCs per day. Tao et al. 2008b estimated the daily intake of PFOS by infants via breastfeeding from seven Asian countries. The daily intake was calculated to be 11.8 ng/kg body weight per day, which was 7-12 times higher than the estimated adult dietary intakes previously reported from Germany, Canada, and Spain.

The placental transfer via the cord blood was investigated by Inoue et al. 2004. The concentrations of PFOS in maternal samples ranged from 4.9 to 17.6 ng/mL, whereas those in foetal samples ranged from 1.6 to 5.3 ng/mL. Furthermore, a high correlation between PFOS concentrations in maternal and cord blood was found, suggesting that PFOS may be able to cross the placental barrier to enter foetal circulation.

Falandysz et al. 2006 described the exposure of PFCs by fish consumption. A positive correlation was found between the PFC concentration in fish and the concentration in Polish people, who have a high fish intake in their diet. This indicates that fish is a possible source

for PFCs in humans. Moreover, paper food packing material (e.g., microwave popcorn bag) could be an exposure source for food (D'eon and Mabury 2007; Sinclair et al. 2007; Washburn et al. 2005).

Harada et al. 2003 reported the influence of the tap water on the human serum concentration. The tap water was contaminated with 0.1 to 50.9 ng/L PFOS estimating a 25-50% rise in the serum PFOS concentration in those people who drink the tap water from the contaminated waterworks. Another study examined the human exposure with PFCs in drinking water in Arnsberg, Germany (Hölzer et al. 2008). The drinking water was highly contaminated with PFCs from contaminated agriculture sites (Skutlarek et al. 2006). An increase by a factor of 4 to 8 was found in blood plasma of children and adults exposed to PFC contaminated drinking water compared to control samples from another area. These findings indicate that drinking water is a potential source for human exposure.

Moriwaki et al. 2003 investigated PFOS and PFOA in dust from Japanese homes. Because of the high contamination of the dust with 11–2500 ng/g for PFOS and 69–3700 ng/g for PFOA, it was suggested that dust could be an important exposure pathways to humans. Shoeib et al. 2005 estimated a human exposure through inhalation and dust ingestion of ~40 and ~20 ng per day, respectively. However, higher exposure intakes were assumed for children because they spend more time on floors and carpets than adults.

2. Aim and outline of the work

Recently PFCs were discovered as emerging POPs. Because of their unique physicochemical properties due to their combination of lipophilic and hydrophilic characteristics, PFCs have been widely used in lots of consumer products such as polymerization aids, stain repellents on carpets, textiles, leather, and paper products for over 50 years (Kissa 2001). From the production and use of these products, PFCs can be released into the environment. Scientific concern about PFCs increased due to their persistence, high bioaccumulation potential for longer-chained PFCs in the marine food web (Martin et al. 2003a; Giesy and Kannan 2001) and possible adverse effects on humans and wildlife (Austin et al. 2003; Goecke-Flora and Reo 1996). As a result the general use of some PFCs are restricted, however, a variety of related PFCs are still being produced by other manufacturers (U.S. EPA 2006; European Parliament and Council 2006; Prevedouros et al. 2006).

The transportation pathways of individual PFCs to remote regions have not been conclusively characterised to date. Two main hypothesis were proposed for the global transportation of PFCs. Firstly, neutral, volatile precursor compounds could undergo long-range atmospheric transport and be degraded in remote regions (Ellis et al. 2004) or secondly ionic PFCs could be transported directly by oceanic currents or by means of sea-spray (McMurdo et al. 2008; Yamashita et al. 2005). However, ocean measurements are very limited and essential for the validation of models as well as quantifying fluxes of PFCs to remote regions like the Arctic. In addition, seawater measurements of PFCs are very useful for the identification of the dominant transportation pathway, either oceanic currents or atmospheric transport of precursors.

Previous temporal trend studies indicated mostly increasing concentrations of PFCs in biota from the Arctic (Bossi et al. 2005a; Dietz et al. 2008; Smithwick et al. 2006). Recently, a few temporal trend studies observed a significant decreasing trend of FOSA (Butt et al. 2007b; Furdui et al. 2008; Hart et al. 2008a), and one study found additionally a significant decreasing trend of PFOS in Arctic ringed seals (Butt et al. 2007b). These decreasing levels could be caused by restrictions and bans of production and/or use of POSF. However, recent temporal trend data on PFCs in biota tissue close to urbanised/industrialised regions with potential high PFC emissions are lacking in the published literature. This information is needed to examine effects of the reductions in overall emissions of PFCs on the concentration levels in marine mammals. Furthermore, such information is useful for any future strategies for the marine ecosystem to reduce PFC contaminations.

In this study, the analytical protocols from Taniyasu et al. 2005 and Powley et al. 2005 for water and biota samples, respectively, were further optimised and validated. The water samples were filtrated and the dissolved and particulate phases were extracted separately using SPE with Oasis® WAX or Strata® XAW cartridges for the dissolved phase and sonication with methanol for the particulate phase (see **publication I**; Ahrens et al. 2009c). The biota samples were extracted using sonication with acetonitrile and subsequent clean-up using ENVI-Carb® cartridges. This method was applied on a wide range of biological matrices (i.e., liver, kidney, lung, heart, blood, brain, muscle, thyroid, thymus, and blubber) (see **publication III**). All samples were analysed simultaneously for 40 target compounds (i.e., PFCAs, PFSAs, PFSiAs, FTCAs, FTUCAs, FASAs, FASEs, and 6:2 FTS) plus 20 IS using HPLC/(-)ESI-MS/MS. The optimised analytical protocol for the analysis of PFCs in biological matrices was integrated in the report of the Marine Chemistry Working Group (MCWG) (ICES 2008). In addition, it was participated in an international interlaboratory study to verify the accuracy, precision, robustness and matrix effects of the analysis of PFCs in water and biota samples (Van Leeuwen et al. 2009).

The first aim of this Ph.D. thesis was to investigate the transportation mechanisms of PFCs in offshore surface water. Samples were taken in the German Bight, where the occurrence and distribution pattern of PFCs was investigated to identify potential sources in the sampling area (see **publication I**). In addition, the distribution of PFCs in surface water in the Atlantic Ocean along the longitudinal gradient from Las Palmas (Spain) to St. Johns (Canada) (15° W to 52° W) and the latitudinal gradient from the Bay of Biscay to the South Atlantic Ocean (46° N to 26° S) was discovered. The observed spatial distribution, characterised by increasing and decreasing concentration gradients of PFCs, can be explained by the pattern of ocean water currents (see **publication II**).

The second aim of this Ph.D. thesis was to examine the mechanisms and pathways of PFCs in harbor seals and their temporal trend in the German Bight. Firstly, concentrations and whole body burden of PFCs in various tissues of harbor seals were determined. Furthermore, the compound-specific distribution in liver, kidney, lung, heart, blood, brain, muscle, thyroid, thymus and blubber of harbor seals was investigated (see **publication III**). Secondly, the temporal trends and composition profiles of PFCs in archived harbor seal livers collected from the German Bight were examined over the last decade. Finally, the association between PFC concentrations in livers of harbor seals and the evidence of diseases, spatial distribution, age and sex were evaluated (see **publication IV**).

3. Publication I

Spatial Distribution of Polyfluoroalkyl Compounds in Seawater of the German Bight

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Abstract

The spatial distribution of polyfluoroalkyl compounds (PFCs) and their composition profile was investigated in 48 water samples collected from the German Bight. All samples were prepared by solid-phase extraction with Strata® XAW cartridges and analysed using high performance liquid chromatography/negative electrospray ionisation-tandem mass spectrometry (HPLC/(-)ESI-MS/MS). Concentrations of various PFCs, including perfluorinated sulfonates (PFSAs), perfluorinated carboxylic acids (PFCAs), unsaturated fluorotelomercarboxylic acids, perfluoroalkyl sulfonamide and sulfonamidoethanol, were quantified. The ∑PFC concentration ranges from 9.36 ng/L to 31.2 ng/L, while perfluorobutane sulfonate (PFBS, 3.38-17.7 ng/L) and perfluorooctanoic acid (PFOA, 2.67-7.83 ng/L) dominated. The rivers Elbe, Weser and Ems had a high influence on the distribution of most PFCs in the German Bight, with maximum PFC concentrations found in their estuaries, and concentrations decreasing with increasing distance from the coast. Conversely, PFBS had its maximum concentration not in the estuaries but in the western German Bight, which suggest an additional source, where PFBS was transported into the German Bight with the westerly current.

3.1. Introduction

Polyfluoroalkyl compounds (PFCs) are persistent against typical environmental degradation processes and are ubiquitous distributed in the environment, having been found in water (Yamashita et al. 2005), air (Jahnke et al. 2007c) and organisms (Giesy and Kannan 2001) around the globe. Because of their unique physicochemical properties due to their combination of lipophilic and hydrophilic characteristics, PFCs have been widely used in many consumer products, such as polymerisation aids, stain repellents on carpets, textiles, leather, and paper products for over 50 years (Kissa 2001). From the production and use of these products, PFCs can be released into the environment. In general, neutral PFCs like perfluoroalkyl sulfonamides and fluorotelomer alcohols are less water-soluble and more volatile than perfluorinated acids. In the atmosphere as well as under aerobic conditions, e.g. in activated sludge, they can be degraded to perfluorinated carboxylic acids (PFCAs) and perfluorinated sulfonates (PFSAs) (Ellis et al. 2004; Martin et al. 2006; Rhoads et al. 2008).

Previous studies examined the release of PFCs into the aqueous environment by runoff from contaminated soil or waste water treatment plants (WWTPs) (Schultz et al. 2006b; Skutlarek et al. 2006) and their riverine transportation (McLachlan et al. 2007). The longer-chained PFCs are known to be bioaccumulative (Martin et al. 2003a) and have toxic effects in biota (Austin et al. 2003; Oakes et al. 2004). As a result, the 3M Company, major producer of perfluorooctyl sulfonyl fluoride (POSF, which is a major precursor for several PFCs), voluntarily phased out the production in 2002, but a variety of related PFCs are still being produced by other manufacturers (Prevedouros et al. 2006). In addition the European Union (EU) formed a directive in October 2006, which prohibits the general use of perfluorooctane sulfonate (PFOS) and their derivates from June 2008 (European Parliament and Council 2006). The former POSF-based products are now substituted by perfluorobutyl sulfonyl fluoride (PBSF)-based products. Highest PFC concentrations are found in top predators (Kannan et al. 2001b). Only a little is known about how PFCs reach the marine environment and about their spatial distribution in the coastal area.

In this study 48 surface water samples were collected in coastal water from the German Bight for the determination of PFCs in the water phase. We investigated the spatial distribution of 18 PFCs in coastal water and their composition profiles to identify sources in the urbanised/industrial sampling area. Furthermore, the relationship between concentrations of PFCs, and dissolved organic carbon (DOC) as well as suspended particulate matter (SPM) were examined.

3.2. Material and methods

3.2.1 Chemicals and standards

The standards used in this study are described elsewhere (Ahrens et al. 2009e). Methanol (SupraSolv), acetonitrile (LiChrosolv), ammonium hydroxide (25% for analysis), formic acid (98-100% suprapure) and ammonium acetate were purchased from Merck (Darmstadt, Germany).

3.2.2 Sample collection and sample pretreatment

Surface water samples were taken onboard the research vessel *Ludwig Prandtl* at 48 sampling stations in the German Bight in August 2007 (see **Figure 8**). Details of the sampling and the physicochemical parameters of the water samples are presented in **Table S1** in the Supplementary material. Sampling sites were chosen to show the influence of the rivers Elbe, Weser and Ems in comparison to the westerly current along the coast of the German Bight. Five litre water samples were collected in brown glass bottles via a metal ships' intake system at approximately one metre below the surface. In addition, at sampling stations 7, 29, 35 and 43 duplicate samples were collected for quality control.

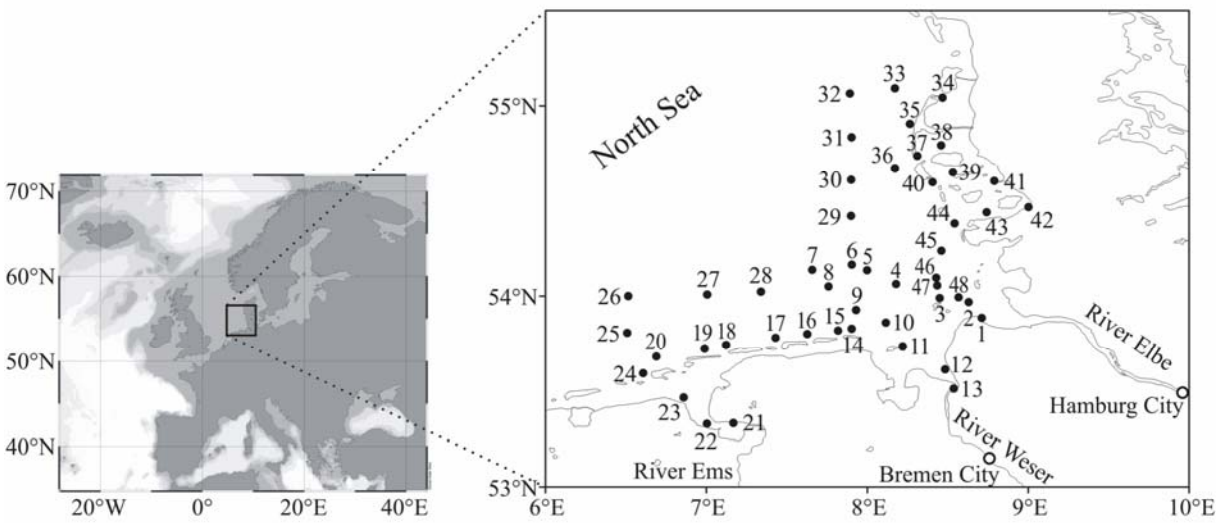

Figure 8. Map showing the sampling locations in the German Bight

The samples were filtered directly after sampling onboard using glass fibre filters (GFFs, GC/C, Whatman, ø 47 mm, > 1.2 µm). The filtrated samples were stored at 4 °C prior to solid-phase extraction (SPE) on board ship on the same or following days. Five field blanks (FBs) were taken to test for possible blank contamination. For the FB, 100 mL Millipore water (Millipore, Elix 5 and Millipore Milli Q Plus) was added to a 5 L brown glass bottle and then

put through SPE extraction. The FBs were then stored and extracted in the same manner as "real" samples.

3.2.3 Solid-phase extraction

The filtrate was extracted by SPE with Strata® XAW cartridges (Phenomenex, 500 mg, 12 cc, 33 µ), similar as described elsewhere (Taniyasu et al. 2005). Prior to the extraction, the samples were spiked with 10 ng absolute of an internal standard (IS) mix (i.e., [$^{13}C_2$]-PFHxA, [$^{13}C_4$]-PFOA, [$^{13}C_4$]-PFNA, [$^{13}C_4$]-PFDA, [$^{13}C_2$]-PFUnDA, [$^{13}C_2$]-PFDoDA, [$^{18}O_2$]-PFHxS, [^{13}C4]-PFOS, [$^{13}C_4$]-PFOSi, d_3-MeFOSA, d_5-EtFOSA, d_7-MeFOSE, d_9-EtFOSE, [$^{13}C_2$]-FHEA, [$^{13}C_2$]-FOEA, [$^{13}C_2$]-FDEA, [$^{13}C_2$]-FHUEA, [$^{13}C_2$]-FOUEA, [$^{13}C_2$]-FDUEA, 100 µL of a 0.1 µg/mL solution). Briefly, after preconditioning with 10 mL methanol and Millipore water, the cartridge was loaded with the five litres sample at approximately 5 drops per sec. The cartridge was then washed with 10 mL 0.1% formic acid in Millipore water and dried for 30 min under vacuum. The elution was divided into two parts: The sulfonamides were eluted with 20 mL acetonitrile; thereafter the acids were eluted with 15 mL 0.1% ammonium hydroxide in methanol. Both extracts were collected separately in brown glass vials and closed with a phenolic resin/ aluminium caps. The samples were stored in a freezer at -20 °C after the elution steps on board ship. After the end of the sampling cruise, both extracts were concentrated in a clean lab (class 10,000) to ~2 mL using rotary evaporator within a few days of arrival at the lab. Finally, both extracts were combined and reduced to 150 µL under a nitrogen stream and spiked with 20 ng absolute of the injection standard d_5-EtFOSAA (InjS, 50 µL of a 0.4 µg/mL solution).

3.2.4 Instrument analysis

An HP 1100 HPLC-system (Agilent Technologies) was used with a Synergi Hydro RP 80A column (150 x 2 mm, 4 micron) by Phenomenex, combined with a suitable guard column: Synergi 2 µ Hydro RP Mercury (20 x 2 mm, 2 micron). Modifications of the HPLC system were made as described elsewhere (Yamashita et al. 2004) to eliminate instrumental blank contamination. The triple-quadrupole mass spectrometer, supplied by Applied Biosystems/MDS SCIEX (API 3000), used an electrospray ionisation (ESI) interface in negative ionisation mode (for details see Ahrens et al. 2009e).

3.2.5 Data analysis

Quantification was performed by the internal standard method with an external calibration. A ten-point calibration curve (1, 5, 10, 25, 50, 100, 500, 1000, 2000 and 3000 pg injected) was used for calculation. For the compounds PFPS, PFNS, PFPeDA and PFHpDA no standards were available, thus these PFCs were calculated from the calibrations of

corresponding substances with plus and minus one carbon atom in the carbon chain. For peak integration only the main peak of a compound was used. The isomers were not included in the peak integration, because of the lack of standards.

3.2.6 Quality assurance

The analytical quality of the laboratory has been approved in interlaboratory studies (Van Leeuwen et al. 2009). As standard procedure, instrument detection limits (IDLs), method quantification limits (MQLs), FBs (see **Table 9**), recoveries and duplicate samples were examined.

Table 9. Instrument detection limits (IDLs), method quantification limits (MQLs) and field blank concentrations for the German Bight survey in August 2007 [a]

	IDL [pg absolute] [b]	MQL [ng/L] [c]	Field blanks [ng/L]
PFBS	0.50	0.367	n.d.
PFPS [d]	–	0.080	n.d.
PFHxS	0.31	0.097	n.d.
PFOS	0.48	0.120	n.d.
PFNS	0.17	0.072	n.d.
6:2 FTS	1.36	0.120	n.d.
PFPA	0.51	0.158	n.d.
PFHxA	0.27	0.084	n.d.
PFHpA	0.36	0.077	n.d.
PFOA	0.36	0.067	n.d.-(0.022)
PFNA	0.35	0.039	n.d.
PFDA	0.40	0.047	n.d.
PFUnDA	0.29	0.019	n.d.-(0.006)
PFDoDA	0.37	0.008	n.d.-(0.004)
FOSA	0.33	0.004	n.d.
MeFBSA	1.25	0.180	n.d.
MeFBSE	1.09	0.241	n.d.
FDUEA	0.58	0.016	n.d.

[a] n.d. = not detected; blank levels were calculated from a sample volume of 100 mL Millipore water; details are given in the text; values in brackets were below the respective MQL; [b] IDL [ng absolute] at 3 times of the signal to noise in the calibration standards (n = 4); [c] MQL [ng/L] at 10 times of the signal to noise in natural samples (n = 4); [d] have to be considered as estimates, because no standards were available for this compound.

After the removal of all Teflon parts from the HPLC system, no instrument blank was detected. In some FBs were found contamination levels of PFOA, PFUnDA and PFDoDA, but all the concentrations were below the MQL. IDLs and MQLs were calculated for substances that were found in real samples using the signal to noise ratios of 3 and 10, respectively. The IDLs were usually lower than 1 pg absolute, while the MQLs were in low

ppq level for the five litre water samples. These MQLs were approximately 3 times lower in comparison of using only one litre sample volume. Conversely to a previous study (Yamashita et al. 2004), for this method the background noise increase was negligible in comparison to the increasing target peak response. The mean recoveries of the IS ranged from 23% (d_3-MeFOSA) to 102% ([$^{13}C_2$]-FHUEA). The low recoveries of the perfluoroalkyl sulfonamides could possibly due to a breakthrough because of the high water volume of five litres. However, 19 IS were used to correct matrix effects as well as losses during sampling, sample extraction, concentration, and analysis. Duplicate samples showed a good agreement with a relative standard deviation of lower than 20% for each compound.

3.3. Results and discussion

3.3.1 Concentrations of PFCs in the German Bight

Overall 18 of the 39 examined analytes were found at the 48 sampling stations. The PFCs quantified included C_4-C_6, C_8 and C_9 PFSAs, 6:2 FTS, C_5-C_{12} PFCAs, MeFBSA, FOSA, MeFBSE and FDUEA (**Table S2**, **Table S3**, and **Table S4** in the Supplementary material). The spatial distribution of ∑PFC and individual PFC concentrations in the German Bight is shown in **Figure 9**. The ∑PFC concentration ranges from 9.36 ng/L (sampling station 26) to 31.2 ng/L (sampling station 13). The highest ∑PFC concentrations were found in the western sampling station 24 and the estuary mouths of the rivers Elbe, Weser and Ems. The ∑PFC concentrations decreased by a factor of 2-3 towards the offshore stations.

The dominant compound of the PFSAs was PFBS with concentrations ranging from 3.38 to 17.7 ng/L, while PFOS was detected in concentrations ranging from 0.69 to 3.95 ng/L. The PFCA concentrations were dominated by PFOA (2.67 to 7.83 ng/L) and PFHxA (0.47 to 9.56 ng/L), whereas the longer-chained PFCAs (C_9-C_{12}) had usually a contribution of under 3% of the ∑PFCs. Four precursor compounds of the PFCAs and PFSAs were detected (i.e., MeFBSA, FOSA, MeFBSE and FDUEA), with a concentration level of lower than 0.75 ng/L. Overall, PFBS was the predominated PFC in the German Bight with a composition of ~40%, followed by PFOA (~26%), PFOS (~9%) and PFHxA (~8%). Interestingly, the compounds PFOS and FOSA contained a contribution of ~60% branched isomers, while the PFCAs had a contribution of <10% and for the other PFCs no branched isomers were observed. It was previously hypothesized that the presence of branched isomers may indicate exposure from historical releases of electrochemical fluorination (ECF) manufacturing process (De Silva and Mabury 2006) or may be a sign of local ECF production. However, the specific distribution and composition profile of PFCs indicates an input from the rivers and western current and additionally an atmospheric deposition is possible.

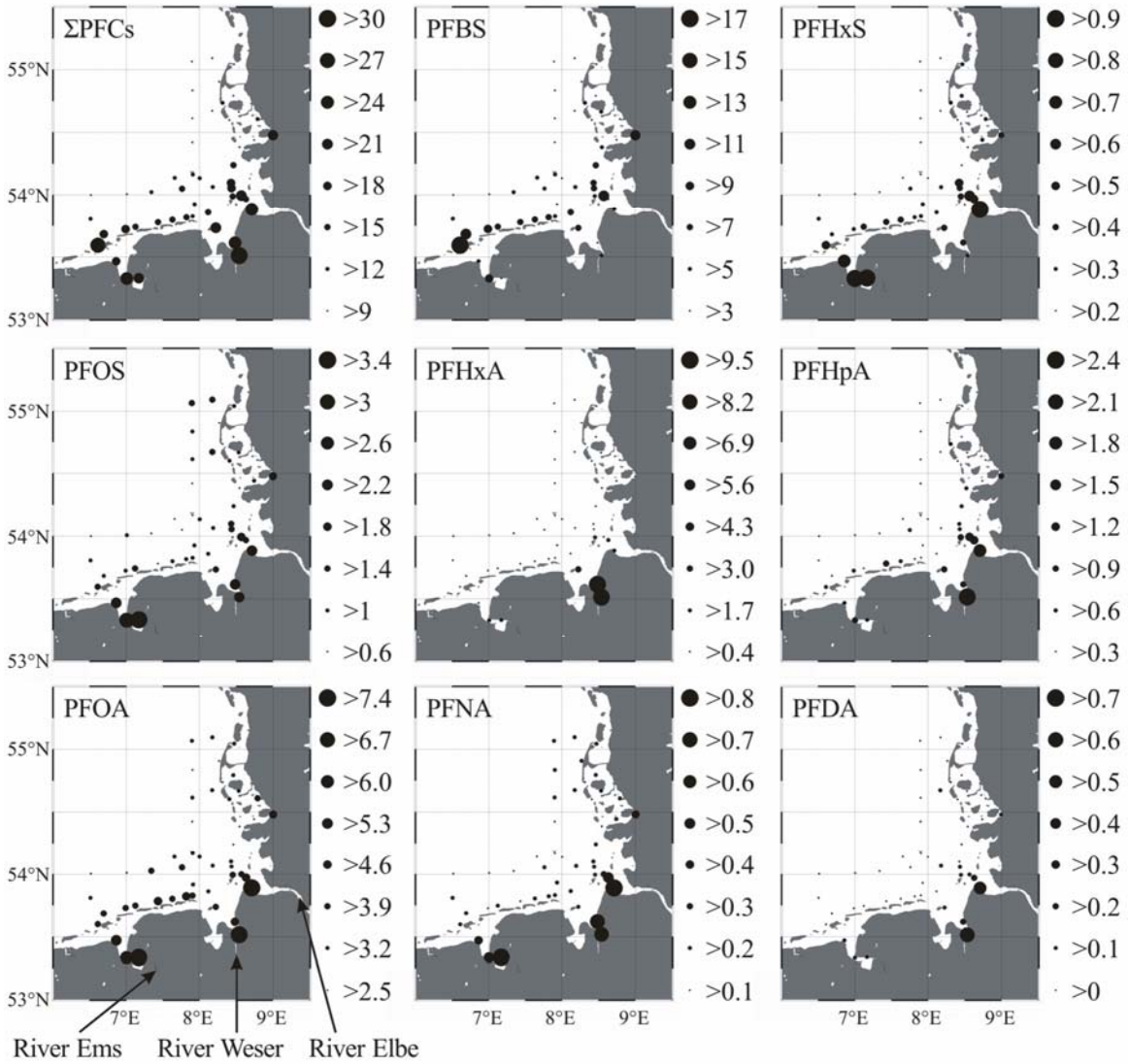

Figure 9. Spatial distribution of ΣPFCs and individual PFC concentrations in the German Bight in ng/L. Note: The different circle sizes at the sampling stations are in proportion to the concentration which is shown on the right side of each map

Significant correlation between DOC and ∑PFC, PFOS and PFOA concentration ($p < 0.0001$, see **Figure 10**) and PFHxS, C_6-C_{10} PFCA and FOSA concentration ($p < 0.0001$, see **Figure S1** in the Supplementary material), respectively, was found, while no correlation had the DOC with PFBS, PFPA and PFUnDA. This correlation corresponds with the positive relationship between sorption of PFCs to sediment and total organic carbon (TOC) amount (Higgins and Luthy 2006). Furthermore, significant correlation between SPM and PFPS, PFHxS, PFHpA, PFOA, PFNA ($p < 0.0001$), PFOS ($p < 0.001$), PFHxA ($p < 0.012$), PFDA ($p < 0.007$) and FOSA ($p < 0.024$) was observed (see **Figure S2** in the Supplementary material). This indicates that sedimentation could be an effective removal mechanism for PFCs in the water phase. However, the DOC and SPM concentrations decreased with increasing distance from the coast, which suggests that the distance from the coast has a high

influence on the distribution and concentration level of PFCs in the aquatic environment. Further investigations of their physical state are necessary.

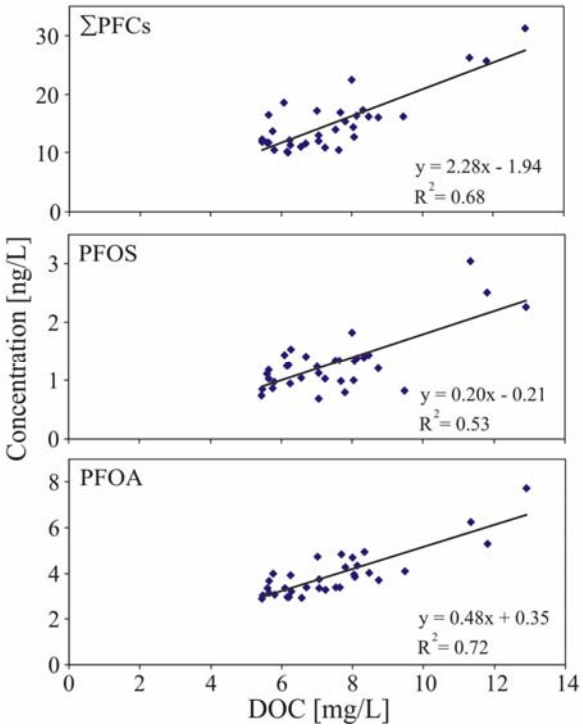

Figure 10. Relationship between concentrations of ΣPFCs, PFOS, PFOA and dissolved organic carbon (DOC) in surface water in the German Bight

3.3.2 Identification of sources of individual PFCs

Most PFCs had their highest concentrations in the river estuary and decreased with increasing distance from the coast. But the composition pattern in the three estuaries Ems, Weser and Elbe were not the same (**Figure 9**). PFOA and PFNA had a similar distribution in all estuaries, whereas PFHpA and PFDA were mainly distributed in the rivers Weser and Elbe. Interestingly, PFHxA was mainly found in the river Weser and not in the other both rivers. PFHxS and PFOS were distributed in all river estuaries with highest concentrations in the estuary of the river Ems. Investigations by Caliebe et al. 2004 determined mean concentrations of PFCs in the river Elbe in 2003 of about 20 ng/L for PFOA and PFOS, and 1 ng/L to 3 ng/L for other PFCs like PFHxA, PFNA, PFDA, PFHxS, and FOSA. Another study in the river Elbe reported PFHxA, PFHpA, PFOA and PFNA concentrations of 15.4 ng/L, 2.7 ng/L, 7.6 ng/L and 0.27 ng/L, respectively, in 2005 (McLachlan et al. 2007). These concentrations were 2 to 5 times higher than measured in the estuaries in this study. Potential sources for PFCs into the rivers Ems, Weser, and Elbe could be caused by effluents from domestic and industrial WWTPs and/or diffuse sources. The two big cities Bremen and Hamburg, located at the rivers Weser and Elbe, respectively, could be an additionally source

for PFCs because of their several industries like petroleum, textile, paper and polymer industries. Furthermore, the rivers transported the PFCs from the source regions into the German Bight, while their discharge (the mean discharge for the rivers Ems, Weser and Elbe was 80 m^3/s, 327 m^3/s, and 711 m^3/s, respectively, for the year 2007) could have a high influence on the distribution pattern of PFCs in the German Bight. Finally, McLachlan et al. 2007 estimated a total flux of 0.26 (PFNA) to 14.3 (PFOA) tonnes per year for 14 major rivers in Europe indicating the rivers as the major input pathway into the marine environment.

The occurrence of PFPS and PFNS at low concentration levels (0.06-0.86 ng/L and 0.11-028 ng/L, respectively) could be due to the POSF-based and PBSF-based production, where uneven carbon chained PFSAs can be produced as by-products (Giesy and Kannan 2002). In contrast to the other PFCs, PFBS composition decreased from 40-60% in the offshore area to 15-20% in the river estuary and its highest composition was found at the western sampling stations 20 (58%) and 24 (61%). This is probably the result of an additional source, where PFBS was transported into the German Bight with the westerly current. It is possible that this contamination of PFBS was originating from the river Rhine, where concentrations of up to 46 ng/L were found (Skutlarek et al. 2006).

Positive correlations between C_6-C_{10} PFCAs and C_5, C_6 and C_8 PFSAs (**Table S5** in the Supplementary material) suggest a common pollution source of these compounds into the marine environment. Possible sources could be the effluents of WWTPs (Schultz et al. 2006b) and rain or surface runoff (Kim and Kannan 2007). In addition, the usage of aqueous film-forming foams (AFFF) could be a source of PFCs, which correspond with the detection of 6:2 FTS in this study (Schultz et al. 2004). On the other hand, the detection of the perfluoroalkyl sulfonamide (i.e., MeFBSA, FOSA, and MeFBSE) and FDUEA indicates atmospheric deposition and/or incomplete biodegradation (Loewen et al. 2005; Martin et al. 2006; Rhoads et al. 2008).

So et al. 2004 found differences in the distribution patterns due to a seasonal shift of the water currents. The circulation in the North Sea is stable throughout the year in contrast to the situation at the Pearl River Delta, China, and thus it is unlikely that the distribution pattern in the North Sea will change dramatically. Nevertheless, unusual weather conditions like east wind might change the pattern on a smaller scale.

3.3.3 Comparison of PFC concentrations in the German Bight with other coastal water studies

Minimum and maximum PFOS and PFOA concentrations in coastal water are shown in **Table 10**. The concentration of PFOS and PFOA in previous studies in the German Bight, Pearl River Delta (China), coastal area of Hong Kong, coastal area of Korea, coastal area of

Dalian (China) were in the same range as in this study (Caliebe et al. 2004; Ju et al. 2008; So et al. 2004; Theobald et al. 2007a; Yamashita et al. 2005). Lower concentrations of PFOS and PFOA were only found in the South China Sea and West Baltic Sea (Theobald et al. 2007a; Yamashita et al. 2005). Highest concentrations of PFOA (320 ng/L) and PFOS (720 ng/L) were found at a contaminated coastal area of South Korea (So et al. 2004). In general, except for the coastal area of South Korea, the concentration of PFOA was higher than of PFOS, which suggests that similar sources exist in the urbanised/industrial coastal areas at the different locations. This corresponds with decreased contamination levels of PFOS and PFOA with increasing distance from the coast.

Table 10. Global comparison of PFOA and PFOS concentrations in seawater from the German Bight with coastal water studies from other areas

Location	Concentration [ng/L]		References
	PFOS	PFOA	
Coastal area of Japan	<2.5-59	n.a.	Taniyasu et al. 2003
German Bight, Germany	0.25-7.0	3-13	Caliebe et al. 2004
Coastal area of South Korea	0.04-730	0.24-320	So et al. 2004
Perl River Delta, China	0.02-12	0.24-16	
Hong Kong, China	0.09–3.1	0.73-5.5	
Tokyo Bay, Japan	0.34-58	1.8-192	Yamashita et al. 2005
Coastal area of Hong Kong	0.07-2.6	0.67-5.5	
Coastal area of Korea	0.04-2.5	0.24-11	
South China Sea	0.008-0.11	0.16-0.42	
German Bight, Germany	0.28 - 3.1	0.54 - 5.9	Theobald et al. 2007a
West Baltic Sea	0.33-0.90	0.47-1.1	
Coastal area of Dalian, China	<0.10-2.3	0.17-38	Ju et al. 2008
German Bight, Germany	0.69 - 3.95	2.67 - 7.83	This study

n.a. = not available.

3.4. Conclusion

Only a few studies exist about the spatial distribution of PFCs in coastal waters. It is very important to identify the sources for individual PFCs and their distribution mechanism. High concentrations (> 1 ng/L) of shorter-chained PFCAs (C_5-C_8), PFBS, PFHxS and PFOS in coastal water of the German Bight suggests that these compounds are entering the marine environment from rivers and can potentially undergo long-range transportation via the ocean currents (Yamashita et al. 2005). Dilution processes and/or adsorption to suspended particle matter could be responsible for the decreasing concentrations, however, PFCs are very

persistent and only the longer-chained PFCs (≥ C_8) have a high affinity to the sediment (Higgins and Luthy 2006; Theobald et al. 2007a). In addition, long-range transportation could also be possible by the atmosphere, which is in agreement with the detection of the precursor compounds MeFBSA, FOSA, MeFBSE and FDUEA.

Most PFCs had their highest concentrations in the river estuaries, but in contrast, PFBS had its maximum concentration in the western German Bight. This study suggests that PFBS has a significant source outside the study region, which makes research on the short-chained PFCs even more important. However, most studies have usually focused on PFOA and PFOS and future studies should be expanded to include the shorter-chained PFCAs and PFSAs.

The occurrence of high concentrations of PFCs in coastal water could possibly be problematic, because they are bioavailable and can accumulate in the marine food chain. Chemical 'fingerprints' may help to identify specific sources of PFC contamination into the aqueous environment. This research, the spatial distribution of PFCs in coastal area, is very important for the understanding of the transportation and fate of PFCs in the marine environment.

Acknowledgement

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4. Publication II

Longitudinal and Latitudinal Distribution of Perfluoroalkyl Compounds in the Surface Water of the Atlantic Ocean

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Abstract

Perfluoroalkyl compounds (PFCs) were determined in 2 L surface water samples collected in the Atlantic Ocean onboard the research vessels *Maria S. Merian* along the longitudinal gradient from Las Palmas (Spain) to St. Johns (Canada) (15° W to 52° W) and *Polarstern* along the latitudinal gradient from the Bay of Biscay to the South Atlantic Ocean (46° N to 26° S) in spring and fall 2007, respectively. After filtration the dissolved and particulate phases were extracted separately, and PFC concentrations were determined using high-performance liquid chromatography interfaced to tandem mass spectrometry. No PFCs were detected in the particulate phase. This study provides the first concentration data of perfluorooctanesulfonamide (FOSA), perfluorohexanoic acid, and perfluoroheptanoic acid from the Atlantic Ocean. Results indicate that trans-Atlantic Ocean currents caused the decreasing concentration gradient from the Bay of Biscay to the South Atlantic Ocean and the concentration drop-off close to the Labrador Sea. Maximum concentrations were found for FOSA, perfluorooctanesulfonate, and perfluorooctanoic acid at 302, 291, and 229 pg/L, respectively. However, the concentration of each single compound was usually in the tens of picograms per litre range. South of the equator only FOSA and below 4° S no PFCs could be detected.

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Introduction

Perfluoroalkyl compounds (PFCs) are persistent against the typical environmental degradation processes and have been found in water, wildlife, and human tissues around the globe (Giesy and Kannan 2001; Hansen et al. 2001; Yamashita et al. 2005). Because of their unique physicochemical properties due to their combination of lipophilic and hydrophilic characteristics, PFCs have been widely used in a lot of consumer products such as polymerization aids and stain repellents on carpets, textiles, leather, and paper products for over 50 years (Kissa 2001). From the production and use of these products, PFCs can be released into the environment. PFCs could be bioaccumulative (Martin et al. 2003a) and have toxic effects in biota (Austin et al. 2003; Oakes et al. 2004). The transportation pathways of PFCs to remote regions have not been conclusively characterized to date. Two main hypotheses were proposed for the global transportation of PFCs. First, neutral, volatile precursor compounds could undergo long-range atmospheric transport and be degraded in remote regions (Ellis et al. 2004), or second, ionic PFCs could be transported directly by oceanic currents or by means of sea spray (Armitage et al. 2006; McMurdo et al. 2008).

The first hypothesis is supported by the determination of precursor compounds, such as fluorotelomer alcohols (FTOHs), perfluoalkanesulfonamidoethanols, in the Arctic atmosphere (Shoeib et al. 2006). The second hypothesis is supported by the fact that ionic PFCs such as the perfluorinated carboxylic acids (PFCAs) and perfluorinated sulfonates (PFSAs) have high water solubilities and low pK_a values and are therefore dissociated at environmentally relevant pH values (Kissa 2001). The ocean currents were calculated by Prevedouros and co-workers to be the major transportation pathway for PFCAs in comparison to atmospheric transportation (Prevedouros et al. 2006). However, irrespective of the transportation pathway involved, high concentrations of PFCs have been found in biota from the Canadian Arctic, especially in marine mammals which are top predators in the marine ecosystem (Martin et al. 2004a).

The global occurrence of PFCs in open-ocean water was described first by Yamashita et al. 2005. Further investigations of PFCs in the Indian Ocean and close to Antarctica were described subsequently (Wei et al. 2008). Detected concentrations are usually around some tens to hundreds of picograms per litre, depending on the location and the compound. It was hypothesized that PFCs could be transported globally with the thermohaline circulation system (Yamashita et al. 2008), with the open-ocean water acting as a final sink for perfluorooctanesulfonate (PFOS) and perfluorooctanoic acid (PFOA) (Yamashita et al. 2005). However, ocean measurements are very limited and essential for the validation of models as well as quantifying inputs of PFCs to remote environments such as the Arctic. Further,

seawater measurements of PFCs are very useful for determining the dominant transportation pathway, either oceanic currents or atmospheric transport of precursors.

The aim of this study was to investigate the longitudinal and latitudinal gradient of PFCs in surface water in the Atlantic Ocean. Sixty water samples were collected during cruises on the research vessels *Maria S. Merian*, from Las Palmas (Spain) to St. Johns (Canada) (15° W to 52° W), and *Polarstern*, from the Bay of Biscay to the South Atlantic Ocean (46° N to 26° S), in spring and fall 2007, respectively. Concentrations of various PFCs, including perfluorobutanesulfonate (PFBS), PFOS, perfluorooctanesulfonamide (FOSA), perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), PFOA, and perfluorononanoic acid (PFNA), were quantified in open-ocean water samples. The observed distribution, characterized by increasing and decreasing concentration gradients of PFCs, can be explained by the pattern of ocean water currents. A comparison with open-ocean water PFC data from Yamashita et al. 2005, 2008 and Wei et al. 2008 is given. This study provides the first evidence for the presence of FOSA, PFHxA, and PFHpA in the Atlantic Ocean.

Experimental Section

Chemicals. The target analytes include 33 ionic PFCs (PFCAs, PFSAs, perfluorinated sulfinates (PFSiAs), fluorotelomer carboxylic acids (FTCAs), and unsaturated fluorotelomer carboxylic acids (FTUCAs)) as well as 7 neutral PFC precursor compounds (perfluoroalkanesulfonamides, perfluoroalkanesulfonamidoethanols) (for details, see Ahrens et al. 2009e). Methanol (SupraSolv), acetonitrile (LiChrosolv), ammoniumhydroxide (25% for analysis), formic acid (98-100% suprapure), and ammonium acetate were purchased from Merck (Darmstadt, Germany).

Sampling Campaign. Surface water samples were collected with the research vessels *Maria S. Merian* (Leibniz Institute for Baltic Sea Research (IOW), Warnemünde) and *Polarstern* (Alfred-Wegener-Institute (AWI), Bremerhaven) from April 14 to April 30 (cruise "MSM05") and Oct 29 to Nov 22 (cruise "ANT XXIV-1"), 2007, respectively. The first cruise with the R/V *Maria S. Merian* was performed along the longitudinal gradient from 15° W to 52° W, and the second cruise with the R/V *Polarstern* was performed along the latitudinal gradient from 46° N to 26° S (**Figure 11**, **Table S6** in the Supplementary material). 60 water samples (2 L) were collected in brown glass bottles via the ships' intake systems at approximately 11 m below the surface at sampling stations 1-42 (46° N to 26° S) and A-R (15° W to 52° W). In addition, at sampling stations J, K, L, O, and R water samples were collected at 2 m depth and directly at the water surface by an external sampler in a brown glass bottle, and at sampling station L two deep-water samples were taken at depths of 200 and 3800 m with a rosette-type sampler (Seabird SBE-32 carousel water sampler equipped

with 24 10 L Hydrobios-Freeflow bottles) to examine concentration differences between different water layers. The different sampling techniques, ship intake systems, rosette-type sampler, and outboard sampler using 2 L brown glass bottles were tested to evaluate for possible background contamination during sampling.

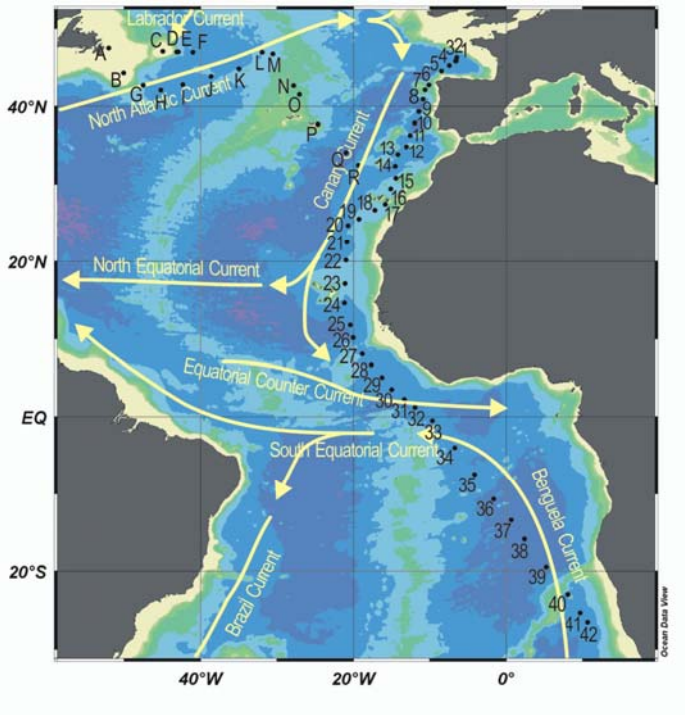

Figure 11. Map showing the sampling stations of the *Maria S. Merian* (15° W-52° W, A-R) and *Polarstern* (46° N-26° S, 1-42) cruise in 2007. The yellow arrows display the main surface currents in the Atlantic Ocean

The samples were filtered directly after sampling onboard using glass fibre filters (GFFs, GC/C, Whatman, ø 47 mm, > 1.2 µm). The dissolved-phase samples were stored at 4 °C prior to solid-phase extraction (SPE) onboard ship on the same or following days, whereas the GFFs were stored in sealed test tubes in a freezer at -20 °C and extracted after the end of the sampling cruise in a clean laboratory (class 10,000) within a few days of arrival at the laboratory.

Field blanks (FBs) were taken every 10th sample for the filtrate and GFFs to test for possible blank contamination. For the dissolved-phase FB, 100 mL of Millipore water (Millipore Elix 5 and Millipore Milli Q Plus) was added to a 2 L brown glass bottle and then put through SPE extraction. The sources of blank contamination are mostly caused by sampling, the extraction process, and instrument analysis (Yamashita et al. 2005), which indicates that the amount of contamination is independent of the volume of Millipore water extracted. FB GFFs were prepared by placing them on the filtration equipment for 1 min.

Both types of field blanks were then stored and extracted in the same manner as "real" samples.

Sample Extraction and Instrumental Analysis. The filtrate and the GFFs were separately spiked with 10 ng absolute of an internal standard (IS) mix (i.e., [$^{13}C_2$]PFHxA, [$^{13}C_4$]PFOA, [$^{13}C_4$]PFNA, [$^{13}C_4$]PFDA, [$^{13}C_2$]PFUnDA, [$^{13}C_2$]PFDoDA, [$^{18}O_2$]PFHxS, [$^{13}C_4$]PFOS, [$^{13}C_4$]PFOSi, d_3-MeFOSA, d_5-EtFOSA, d_7-MeFOSE, d_9-EtFOSE, [$^{13}C_2$]FHEA, [$^{13}C_2$]FOEA, [$^{13}C_2$]FDEA, [$^{13}C_2$]FHUEA, [$^{13}C_2$]FOUEA, [$^{13}C_2$]FDUEA, 100 μL of a 0.1 μg/mL solution). The filtrate was spiked with the IS mix and extracted by SPE with Oasis® WAX cartridges (Waters, 150 mg, 6 cm^3, 30 μm), as described elsewhere (Taniyasu et al. 2005) with some modifications. Briefly, after being preconditioned with 5 mL of methanol and Millipore water, the cartridge was loaded with the 2 L sample at approximately 4 drops/s (~0.1 mL/min). The cartridge was then washed with 5 mL of 0.1% formic acid in Millipore water and dried for 30 min under vacuum. After the loading and drying steps onboard the ships, the cartridges were stored in a freezer at -20 °C. The cartridges were eluted after the end of the sampling cruise in a clean laboratory (class 10,000) within a few days of arrival at the laboratory. The elution was divided into two parts: The sulfonamides were eluted with 14 mL of acetonitrile; thereafter the acids were eluted with 5 mL of 0.1% ammonium hydroxide in methanol. The combined extract was reduced to 150 μL under a nitrogen stream and spiked with 20 ng absolute of the injection standard d_5-EtFOSAA (InjS, 50 μL of a 0.4 μg/mL solution).

The particulate matter (> 1.2 μm) was analyzed by sonication as described elsewhere (Higgins and Luthy 2006) with some modifications. The GFF was spiked with the same IS mix as the filtrate and sonicated with 100 mL of methanol for 1 h. This extraction was performed twice, and the two fractions were combined, evaporated by rotary evaporation, and filtered. The extract was reduced to 150 μL under a nitrogen stream and spiked with 20 ng of the InjS (see above). Finally, the extracts from the dissolved- and particulate-phase samples were analyzed using high-performance liquid chromatography coupled with tandem mass spectrometry (HPLC-MS/MS). An HP 1100 HPLC system (Agilent Technologies) was used with a Synergi Hydro RP 80A column (150 × 2 mm, 4 μm) by Phenomenex, combined with a suitable guard column: Synergi 2 μ Hydro RP Mercury (20 × 2 mm, 2 μm). The triple-quadrupole mass spectrometer, supplied by Applied Biosystems/MDS SCIEX (API 3000), used an electrospray ionization (ESI) interface in negative ionization mode (for details see Ahrens et al. 2009e).

Results and Discussion

Quality Assurance. The analytical quality of the laboratory has been approved in interlaboratory studies (Van Leeuwen and De Boer 2007). As standard procedure, FBs, method detection limits (MDLs), method quantification limits (MQLs) (see **Table 11**), and recoveries of spiked samples were examined (see **Table S7** in the Supplementary material). Matrix spike recoveries of the target analytes ranged from 77% to 131% for the dissolved phase and from 72% to 113% for the particulate phase.

Table 11. MDLs and MQLs for the dissolved phase, and field blank concentrations (dissolved and particulate phase) for cruises onboard the research vessels *Maria S. Merian* (FBs 15° W-52° W, n = 4 + 4) and *Polarstern* (FBs 46° N-26° S, n = 6 + 6) in the Atlantic Ocean (pg/L) [a]

Analytes	MDL[b]	MQL[b]	Dissolved phase FB		Particulate phase FB	
			RV *Maria S. Merian*	RV *Polarstern*	RV *Maria S. Merian*	RV *Polarstern*
PFBS	0.49	1.6	<1.6	<1.6	n.d.	n.d.
PFOS	3.1	10	<10	<10-25	n.d.	n.d.
FOSA	5.1	17	<17	<17	n.d.	n.d.
PFHxA	1.7	5.7	<5.7	<5.7-9.2	n.d.	n.d.
PFHpA	1.8	5.9	<5.9	<5.9-9.7	n.d.	n.d.
PFOA	1.2	4.0	<4.0-15	<4.0-28	n.d.	n.d.
PFNA	1.5	5.1	<5.1	<5.1	n.d.	n.d.

[a] Blank levels were calculated from a sample volume of 100 mL Millipore water. One FB sample from the RV *Maria S. Merian* cruise and two FB samples from the *Polarstern* cruise showed a blank contamination; details are given in the text; n.d. = not detected; <x below the respective MQL; [b] MDL and MQL (ng/L) at 3 and 10 times of the signal to noise in natural samples (n = 4), respectively.

A variety of laboratory products contain fluoropolymers such as polytetrafluoroethylene (PTFE) (Yamashita et al. 2004). All fluorinated materials which could come in contact with the sample during the sampling (including SPE block), sample preparation, and instrumental analysis were removed (for details see Yamashita et al. 2005). After the removal of all PTFE parts from the HPLC system, no instrument blank was detected. All procedure blanks, using 1 L of Millipore water, which were extracted in the same manner as the samples, were below the MQL. No background contamination was detected in the FBs for the particulate phase. For the dissolved phase, the FBs from both sampling cruises were usually below the MQL, but in three FBs contamination levels of a few picograms per litre up to 28 pg/L (PFOA) were quantified (see **Figure S3** in the Supplementary material). For control of the repeatability and blank contamination of the ship inlet system at stations J, K, L, O, and R, five samples were taken in parallel using the outboard sampler and the ship inlet system; for all detected PFCs

no significant differences were observed (Mann-Whitney U-test [$p < 0.01$]). MDLs and MQLs were calculated for substances that were found in real samples using signal-to-noise ratios of 3 and 10, respectively. The MDLs were in the low pictogram per litre range for the 2 L water samples. Matrix spike recoveries of the IS at two different spike levels (5 and 20 ng/L) ranged from 23% (d_3-MeFOSA) to 90% ([$^{13}C_2$]FHUEA) for the dissolved phase and from 50% (d_3-MeFOSA) to 124% ([$^{13}C_2$]PFHxA) for the particulate phase.

Concentrations of PFCs in the Atlantic Ocean. In this study, 40 PFCs were measured in the water samples. PFBS, PFOS, FOSA, PFHxA, PFHpA, PFOA, and PFNA could be quantified in the dissolved phase of the marine water samples in a concentration range of <MQL to 1115 pg/L; all other PFCs were below the corresponding MDLs. PFCs were not detectable in the particulate phase. The low particle mass in the 2 L water samples could be responsible for the not detectable PFC concentration in the particulate phase in the Atlantic Ocean; however, the partitioning behaviour of PFCs is an important future research field to evaluate their physical state and bioavailability. To the authors' knowledge, this is the first report of quantifiable concentrations of PFHxA, PFHpA, and FOSA in surface water in the Atlantic Ocean. At five sampling locations, water samples were taken in parallel at 11 and 2 m depths and directly at the surface. No correlation between sampling depth and concentration levels was observed, which indicates that there is a well-mixed zone between the surface and 11 m water depth in open-ocean waters. Ju et al. 2008 found at three near-shore sites that the surface microlayer (50 μL thickness) had by a factor of 24-109 higher PFOS concentration than the subsurface water (> 30 cm depth); however, the results are not comparable because of the different sampling techniques. Concentrations of PFCs at location L in the two deep-water samples at 200 and 3800 m were below the MDL. Yamashita et al. investigated vertical profiles in the Labrador Sea, Middle Atlantic Ocean, South Pacific Ocean, and Japan Sea, where they detected relatively constant concentration levels of PFOS, PFOA, and PFBS down to 2000 m in the Labrador Sea and a decreasing concentration gradient with water depth in the other areas. They suggested that the global circulation system has an influence on the occurrence of PFCs in deep water (Yamashita et al. 2008). Our deep-water samples were taken far away from downwelling currents, which explains why no PFCs were detected.

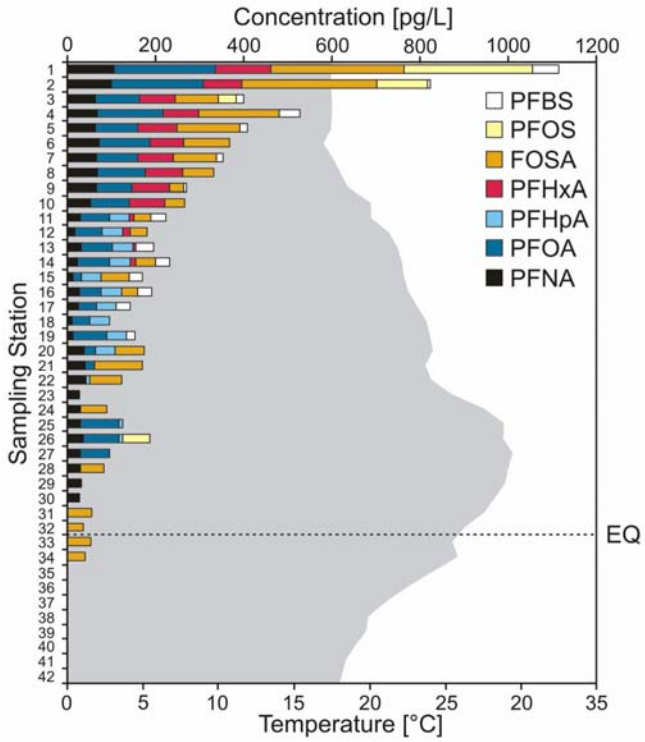

Figure 12. Individual PFC concentrations (pg/L) and ambient temperature are given from the *Polarstern* cruise (46° N-26° S, 1-42) in 2007

The longitudinal and latitudinal distribution of ΣPFC concentrations in the Atlantic Ocean is shown in **Figure 12** and **Figure 13**, and **Table S8** in the Supplementary material. The highest ΣPFC concentration (1115 pg/L) was found in the Bay of Biscay close to the European source area. A decreasing north to south latitudinal gradient was observed toward the Canary Islands where the mean ΣPFC concentration declined by a factor of 6 to 142-191 pg/L (sampling stations 16 and 17). The ΣPFC concentrations remained relatively constant toward the south from the Canary Islands down to 10° N. In the equator area, the ΣPFC concentrations decreased by a factor of 4, relative to those of the Canary Islands down to 10° N, with only FOSA quantifiable at 37-53 pg/L (sampling stations 31-34). South of 4° S, no PFCs were detected. The west to east transect in the North Atlantic showed a different pattern of concentrations. The ΣPFC concentrations east of 25° W (sampling stations G-R) and at sampling points A and B at the coast from Canada ranged from 52 to 117 pg/L and were on average a factor of 2 higher than those of samples C-F, clustered north of the main transect close to the Labrador Sea (n.d. to 40 pg/L). The increasing concentrations, observed from sampling station L to sampling station R, could be influenced by latitudinal as well as longitudinal trends. Two-thirds of the water samples contained quantifiable concentrations of PFOA, which was the most abundant compound in the water samples from the Atlantic Ocean, with a mean contribution of 37% to the ΣPFCs and a concentration range from <4.0 to 229 pg/L. PFNA was detected in 52% of the water samples at concentrations

greater than the LOQ, but the concentrations from <5.1 to 107 pg/L were less than those of PFOA. Concentrations of the other PFCs ranged from <17 to 307 pg/L for FOSA, from <10 to 291 pg/L for PFOS, from <5.7 to 127 pg/L for PFHxA, from <5.9 to 104 pg/L for PFHpA, and from <1.6 to 60 pg/L for PFBS; however, the concentrations were below the MQL in more than half of the samples.

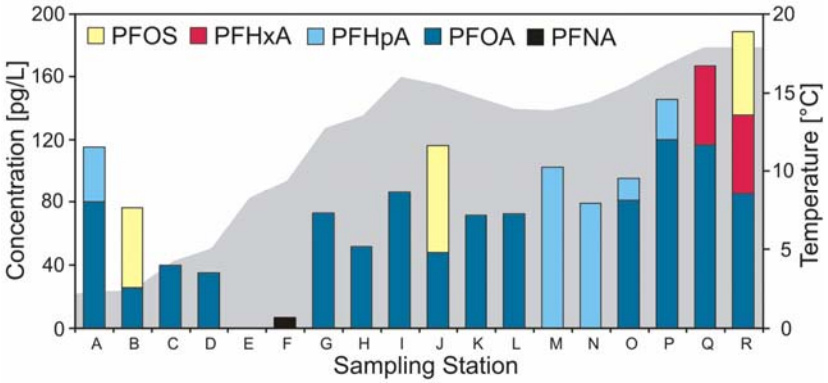

Figure 13. Individual PFC concentrations (pg/L) and ambient temperature are given from the *Maria S. Merian* cruise (15° W-52° W, A-R) in 2007

Comparison with Other Ocean Water PFC Measurements. Minimum and maximum PFC concentrations in surface open-ocean water from the Atlantic, Pacific, and Indian Oceans are shown in **Table 12**. In previous studies, the highest concentrations of PFOA and PFOS were found in the North and Mid Atlantic Ocean and the Western Pacific Ocean, while the lowest concentrations were observed in the Central and Southern Pacific and the Indian Ocean (Theobald et al. 2007b; Wei et al. 2008; Yamashita et al. 2005; Yamashita et al. 2008). The results presented here can be compared with open-ocean water samples presented from Yamashita et al. 2005. They collected samples from the North and Middle Atlantic Ocean in 2002-2004 and found concentration levels of several tens of picograms per litre for perfluorohexanesulfonate (PFHxS), PFOS, and PFNA to a few hundreds of picograms per litre for PFOA. A similar study from Theobald et al. 2007b was carried out from 53° N to 30° S in the Atlantic Ocean in 2005. The concentrations of PFOA and PFOS were in a range of a few tens of picograms per litre with a maximum concentration of 170 pg/L for PFOS. Both studies (Theobald et al. 2007b; Yamashita et al. 2005) reported concentrations in the same range as in this study, except for PFHxS, which was found in the first study, but could not be detected by us.

Concentrations of PFOS and PFOA reported in the West Pacific Ocean are in the same range as found in the Atlantic in this study, with concentrations of PFOS and PFOA in the Central and South Pacific and Indian Oceans about 1 magnitude lower than in the North

Atlantic Ocean. PFBS and PFNA were also found in the Central to East Pacific Ocean and the Antarctic region, respectively, but the concentrations were less than those found in the Atlantic Ocean. The global distribution of PFBS might originate from the increasing production of n-methyl perfluorobutane sulfonamidoethanol (MeFBSE) and related products with four perfluorinated carbons, which was introduced after the voluntary phase-out of perfluorooctanesulfonyl fluoride (POSF) by the 3M Co. in 2000 (3M 2000; D'eon et al. 2006). In comparison to this study, FOSA, PFHxA, and PFHpA could not be detected or were not analyzed in the Pacific and Indian Oceans, respectively. Conversely, PFHxS and PFDoDA were detected in the Indian Ocean and Arctic region in the few picograms per litre range, whereas both compounds could not be detected in this study.

Table 12. Comparison of minimum and maximum PFC concentrations in surface open-ocean water with literature data (pg/L) [a]

	Location	PFBS	PFHxS	PFOS	FOSA	PFHxA	PFHpA	PFOA	PFNA	PFDoDA
Yamashita et al. 2005	North Atlantic Ocean (n=9)	n.a.	4.1-6.1	8.6-36	n.a.	n.a.	n.a.	160-338	15-36	n.a.
	Mid Atlantic Ocean (n=7)	n.a.	2.6-12	37-73	n.a.	n.a.	n.a.	100-439	n.a.	n.a.
	Western Pacific Ocean (n=2)	n.a.	2.2-2.8	54-78	n.a.	n.a.	n.a.	136-142	n.a.	n.a.
	Central to Eastern Pacific Ocean (n=12)	n.a.	0.1-1.6	1.1-20	n.a.	n.a.	n.a.	15-62	1.0-16	n.a.
Theobald et al. 2007b	North to South Atlantic Ocean (n=22)	n.a.	n.a.	<14-170	n.a.	n.a.	n.a.	<17-90	n.a.	n.a.
Yamashita et al. 2008	South Pacific Ocean (n=5)	n.a.	n.a.	<5-11	n.a.	n.a.	n.a.	<5-11	n.a.	n.a.
Wei et al. 2008	Central and Southern Pacific Ocean (n=9)	<25	<5	<5-21	<5	<5	<5	<5-7.0	<5	<1
	Indian Ocean (n=7)	<5	<5	<5-8.6	n.a.	<5	<5	<5-11	<5	<1-1.4
	Antarctic region (n=5)	<1(5)-2.9	<1(5)	5.1-22.6	n.a.	<5	<5	<5	<5	<1-1.1
This study	North Atlantic Ocean (n=40)	<1.6-60	n.d.	<10-291	<17-307	<5.7-127	<5.9-104	<4.0-229	<5.1-107	n.d.
	Mid Atlantic Ocean (n=10)	<1.6	n.d.	<10-60	<17-60	<5.7	<5.9-9.7	<4.0-87	<5.1-35	n.d.
	South Atlantic Ocean (n=10)	<1.6	n.d.	<10	<17-53	<5.7	<5.9	<4.0	<5.1	n.d.

[a] n.d. = not detected; n.a. = not analysed; <x below the respective MQL.

Yamashita et al. have studied vertical profiles of several PFCs in the Labrador Sea, Middle Atlantic Ocean, South Pacific Ocean, and Japan Sea in 2004 and 2005 (Yamashita et al. 2008). The surface water concentrations of PFOA and PFOS in the Northwest Atlantic

Ocean were comparable with those from this study, but in addition in this study, PFHpA and PFNA were detected and PFBS was not detected in this area.

Impact of the Ocean Currents on the PFC Pattern and Concentration Level. The occurrence of elevated PFC levels in the Arctic ecosystem (Dietz et al. 2008) raises the question about the global transportation and fate of PFCs. In addition to the atmosphere, the ocean currents could be an important global transport pathway for transport of PFCs from industrial to remote areas (Yamashita et al. 2005). This study examined the impact of environmental factors, such as ocean currents, on the distribution of PFCs in the Atlantic Ocean. The PFC concentration distribution along the latitudinal gradient will be influenced by the Canary, Equatorial Counter, and Benguela Currents (see **Figure 11** and **Figure 12**). The Canary Current comes from the north from the European Continent source region and crosses the Equatorial Counter Current and the Benguela Current in the equator area. The Benguela Current has for its origin Antarctic water with low PFC loading (Wei et al. 2008), and the influence of this water body resulted in a rapid decrease of ΣPFC concentrations to below the MDLs. The presence of cold surface water from the Benguela Current was confirmed by the drop-off of the water temperature from the equator region to the south. Furthermore, the West African coast seemed to have no impact on the concentration level, which suggests lack of sources (e.g., river discharge) from this area in contrast to the industrial European area. It is probable that PFC-laden water from the Canary Current was transported to the west and northwest by the North and South Equatorial Currents and possibly further south along the coast of Brazil by the Brazil Current. The decreasing latitudinal gradient is consistent with the decreasing gradient of PFOA and PFOS on airborne particles described by Jahnke et al. 2007c. What it is not known, however, is whether the airborne particle-bound fraction originated from sea spray or atmospheric degradation of volatile precursor compounds, i.e., whether the ocean was the source or sink for this airborne contamination. It is noteworthy that the pattern of importance of individual PFCs changed depending on the sampling area. In the northeast of the Atlantic Ocean in the Bay of Biscay, all PFCs, except for PFHpA, were detected, with the latter detected for the first time at 37° N. The concentration ranged from several tens of picograms per litre (PFBS, PFHxA, and PFNA) to a few hundreds of picograms per litre (PFOS, PFOA, and FOSA). The concentration of PFSAs, PFOS, and PFBS dropped below the MDLs south of 32° N and 25° N, respectively. The occurrence of PFCAs toward the south depended on their chain length, with the longer the chain length of the PFCAs (C_6 to C_9), the further southward they were detected. The reason for this behaviour could be different physicochemical characteristics (e.g., vapour pressure, partition coefficient) and/or input from atmospheric sources (Ellis et al. 2004; Higgins and Luthy 2006). The

increasing ΣPFC concentration at sampling stations 24-28, attended by an increasing water temperature, could be caused by higher rainfall in this area, leading to increased deposition of PFCAs from the atmosphere (Scott et al. 2006b). Of all detected PFCs, FOSA was found furthest south, down to 4° S, possibly as a result of its higher vapour pressure increasing the importance of atmospheric transport.

The location of the Labrador, North Atlantic, and Canary Currents will similarly affect the distribution of PFCs along the longitudinal transect (see **Figure 11** and **Figure 13**). PFC concentrations dropped off in sampling stations C-F. These results imply that the northeastern samples were influenced by the Labrador Current, whose origin is the Arctic Ocean and is relatively "clean" (Yamashita et al. 2008), and the low ocean temperatures in this area support this hypothesis. In contrast, sampling stations A and B had elevated concentrations and were probably influenced by inputs from the Canadian coast and North Atlantic Current, respectively. In the area of the North Atlantic Current, PFOS, PFHpA, and PFOA dominated, with sum concentrations of 52-117 pg/L (sampling stations G-O). This is twice the concentration found at sampling stations C-F influenced by the Labrador Current. The warm temperature of the North Atlantic Current became noticeable at sampling stations L and M, which were at latitudes similar to those of stations C-F, but with surface water temperatures much higher than those close to the Labrador Sea. The highest concentrations where found at sampling stations P-R, which could be induced by the Canary Current carrying PFCs from the European Continent source region.

The concentrations of PFNA and PFOA were positively correlated (r^2 = 0.52; see **Figure 14**), which indicates that the sources of both compounds are related (Young et al. 2007). Young et al. 2007 found a positive correlation with a gradient of ~1 in snow on remote ice caps that were contaminated atmospherically by precursors, in this case 8:2 FTOH. The gradient in the Atlantic Ocean is ~0.4, and it is possible that the degradation of perfluoalkanesulfonamides could lead to a higher amount of PFOA relative to PFNA (Martin et al. 2006). Plots of PFHxA, PFOA, and PFNA concentrations versus PFOS concentrations are also correlated for the Atlantic Ocean, but the significance is lower because the calculations are based on only a few data points (**Figure S4** in the Supplementary material). Young et al. found no correlation among PFHxA, PFNA, and PFOS on the ice cap, which further supported the source there being the atmospheric pathway (Young et al. 2007). Concentrations of PFHpA and PFOA were negatively correlated in the Atlantic Ocean (r^2 = 0.32, see **Figure S4**), probably as a consequence of the patchy distribution of PFHpA. Simcik and Dorweiler 2005 found that the ratio of PFHpA to PFOA increased with increasing distance from nonatmospheric sources and suggested that a high ratio would be a good tracer

of atmospheric deposition. In this study, only a few stations (15, 20, M, and N) had PFHpA/PFOA ratios greater than 1, which suggests that direct releases are important determinants of open Atlantic Ocean surface water concentrations.

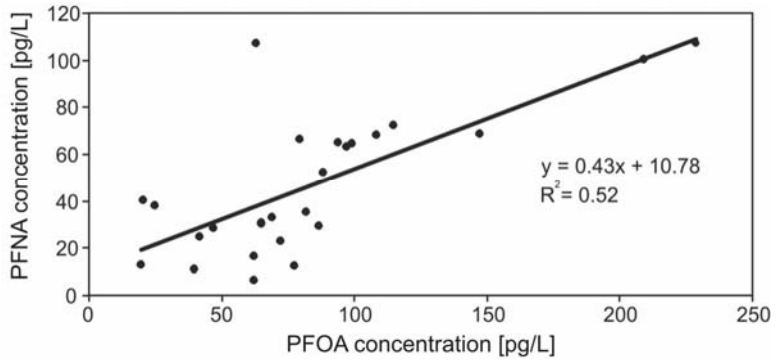

Figure 14. Correlations between PFNA and PFOA concentrations in surface water in the Atlantic Ocean

Several reasons for the distribution pattern of PFCs in the Atlantic Ocean have been suggested, but for each location several factors could be responsible for the occurrence of the PFCs. Ocean currents and related dilution effects have a crucial influence on PFC distribution (Yamashita et al. 2008). The spatial distribution data obtained in this study are useful for global transportation models (Wania and Mackay 1995), in which industrial areas are considered as source of PFCs, and ocean waters and the atmosphere are important as sinks and for transportation of these compounds. This transportation to remote regions could have adverse effects in top predators here, because of the high bioaccumulation potential for PFOS and longer-chained PFCAs in the marine food web (Houde et al. 2006c). Further investigations of the biochemical cycle of PFCs in ocean waters are necessary for understanding the transportation and fate of PFCs in the marine environment.

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5. Publication III

Total Body Burden and Tissue Distribution of Polyfluorinated Compounds in Harbor Seals (*Phoca vitulina*) from the German Bight

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Abstract

Total body burden and tissue distribution of polyfluorinated compounds (PFCs) were investigated in harbor seals (*Phoca vitulina*) from the German Bight in 2007. A total number of 18 individual PFCs from the following groups could be quantified in the different tissues: perfluorinated carboxylic acids (PFCAs) and perfluorinated sulfonates (PFSAs) and their precursors perfluorinated sulfinates (PFSiAs), perfluorinated sulfonamides, and sulfonamido ethanols. Perfluorooctanesulfonate (PFOS) was the predominant compound in all measured seal tissues (up to 1665 ng/g wet weight in liver tissue). The dominant PFCAs were perfluorononanoic acid (PFNA) and perfluorodecanoic acid (PFDA), but their concentrations were much lower compared to PFOS. The mean whole body burden in harbor seals of all detected PFCs was estimated to be 2665 ± 1207 µg absolute. The major amount of the total PFCs burden in the bodies was in blood (38%) and liver (36%), followed by muscle (13%), lung (8%), kidney (2%), blubber (2%), heart (1%), brain (1%), thymus (<0.01%) and thyroid (<0.01%). These data suggest large differences in body burden and accumulation pattern of PFCs in marine mammals.

Keywords: Total body burden; tissue distribution; harbor seal; PFCs; PFOS; PFOA

5.1. Introduction

Recently polyfluorinated compounds (PFCs) were discovered as emerging persistent organic pollutants. PFCs are widely used as processing additives during fluoropolymer production and as surfactants in consumer applications, including surface coatings for carpets, furniture and paper products over the past 50 years (Kissa 2001). From the production and use of these products, PFCs can be released into the environment. Scientific concern about PFCs increased due to their global distribution and ubiquitous detection in the environment, especially in marine mammals (Giesy and Kannan 2001). PFCs in general bind to blood proteins (Jones et al. 2003) and the longer-chained PFCs are known to bioaccumulate (Martin et al. 2004a). Toxic effects in biota like neuroendocrine effects (Austin et al. 2003) and peroxisome proliferation (Goecke-Flora and Reo 1996) were demonstrated. In addition, positive correlation between infection diseases of river otters and diet of high concentration of PFCs was observed (Kannan et al. 2006).

Relatively little is known about the total body burden of PFCs in organisms. For the calculation of the total body burden the concentration in liver tissue and plasma are often used. These estimations are often potential sources of errors because little is known about the distribution of PFCs in the whole body. In addition, bioaccumulation evaluations may be overestimated when using liver and plasma concentrations.

The bioconcentration factors (BCF), half-lives and uptake rates increased with increasing perfluoroalkyl chain length in all tissues of rainbow trouts (*Oncorhynchus mykiss*) exposed with perfluorinated carboxylic acids (PFCAs) and perfluorinated sulfonates (PFSAs) in a flow-through system (Martin et al. 2003a). Longer-chained PFCAs, perfluorobutanesulfonate (PFBS) and perfluorooctanesulfonate (PFOS) were quantified in kidney, liver, blubber, muscle, tracheo-branchial muscle and spleen in harbor seals (*Phoca vitulina*) from the Dutch Wadden Sea (Van de Vijver et al. 2005). Another study determined PFCs in different tissues from ringed seals (*Phoca hispida*), where the highest whole body distribution was observed in blood, muscle and liver with PFOS as the predominant compound (Sturman et al. 2007).

The object of this study was to determine concentrations and burden of PFCs in various tissues of harbor seals (*P. vitulina*) from the German Bight. To better understand the mechanisms and pathways of PFCs in marine wildlife, we examined the compound-specific distribution in liver, kidney, lung, heart, blood, brain, muscle, thyroid, thymus and blubber of harbor seals.

5.2. Materials and methods

5.2.1 Sample collection

The harbor seals (n = 4) were collected at the German Bight in 2007. All harbor seals were stranded and shot by trained personal due to severe illness such as bronchopneumonia and septicemia, the carcass were then post-mortem at the Research and Technology Centre Westcoast (FTZ) according to the protocol described by Siebert et al. 2007 (**Table 13**). Between finding and dissection the carcass was frozen in a plastic bag at -20 °C to minimize any degradation of PFC precursors. The age was determined based on the length of the animal, filling of tooth, growth layers in the tooth, date of birth of harbor seals in the sampling area and date of finding (Siebert et al. 2007). Length and weight of the seals were measured and organs were examined macroscopically. Organs were weighed before post-mortem subsampling. All tissue samples for PFC analysis were taken with stainless steel instruments, placed into polypropylene (PP) bags and stored in a -20 °C freezer until analysis.

Table 13. General information of the four analysed harbor seals from the German Bight including the organ and tissue weights (g)

	Harbor seals (*Phoca vitulina*)			
Sex	♂	♀	♂	♂
Age (years)	<1	<2	<2	<1
Date of finding	26/11/2007	05/12/2007	05/12/2007	24/11/2007
Date of dissection	24/01/2008	24/01/2008	24/01/2008	24/01/2008
Place of finding	Büsum, Germany	Helgoland, Germany	Helgoland, Germany	Sylt, Germany
Standard length (cm)	85	88	97	84
Blubber thickness breast/ sternum (mm)	15	18	22	20
Blubber thickness dorsal/ neck (mm)	11	18	15	15
Blubber in %	18.3 [a]	27.2 [a]	24.6 [a]	21.4 [a]
Tissue and organ weight (g)				
Liver	725	856	974	1196
Kidney	160 [b]	213 [b]	181 [b]	223 [b]
Lung	435 [c]	445 [c]	435 [c]	535 [c]
Heart	171	199	157	230
Blood	2610 [d]	2670 [d]	2610 [d]	3210 [d]
Brain	283 [e]	265 [e]	283 [e]	223
Muscle	5011 [f]	5126 [f]	5011 [f]	6163 [f]
Thyroid	0.60 [b]	0.78 [b]	1.15 [b]	0.69 [b]
Thymus	- [g]	1.40	0.52	0.57
Blubber	3181 [h]	4846 [h]	4281 [h]	4571 [h]
∑ tissue and organ weight	12593	14644	14662	16372
Whole body mass (g)	17400	17800	17400	21400

[a] The percent blubber content of the body mass was calculated by the formula B% = 4.44 + 5693 * (√ (standard length (m) / body mass (kg)) * dorsal blubber thickness (Ryg et al. 1990); [b] sum of the right and left organ; [c] calculation based on a relative lung weight of 2.5% of the of the body mass (Stewardson et al. 1999); [d] calculation by 150 mL per kg body mass (Burns et al. 2005); [e] it were uses the brain size of 283 g for males and 265 g for females (Bininda-Emonds 2000); [f] calculation based on a relative muscle weight of 28.8% of the body mass (Burns et al. 2005); [g] thymus macroscopically not detectable; [h] calculation based on the percent blubber content of the body mass (Ryg et al. 1990).

5.2.2 PFC analysis

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A list of the native and mass-labelled standards including their acronyms, formula, supplier and purity are presented in **Table 14**. Methanol (SupraSolv), acetonitrile (LiChrosolv) and acetic acid (glacial, > 99%) were purchased from Merck.

PFCs in liver, kidney, lung, heart, blood, brain, muscle, thyroid, thymus and blubber of harbor seals were analysed described by Powley et al. 2005 with some modifications. Shortly, tissue subsample were homogenised in a ice bath using an Ultraturrax® disperser (T 25 basic Ultraturrax, IKA, Germany) with plastic dispersing (made of polycarbonate and polysulfone). 1 to 2 g tissue and 2 mL blood sample respectively were weighed in a PP tube and spiked with 10 ng of an internal standard (IS) mix (i.e., [$^{13}C_4$]-PFBA, [$^{13}C_2$]-PFHxA, [$^{13}C_4$]-PFOA, [$^{13}C_4$]-PFNA, [$^{13}C_4$]-PFDA, [$^{13}C_2$]-PFUnDA, [$^{13}C_2$]-PFDoA, [$^{18}O_2$]-PFHxS, [$^{13}C_4$]-PFOS, [$^{13}C_4$]-PFOSi, [$^{13}C_2$]-FHEA, [$^{13}C_2$]-FOEA, [$^{13}C_2$]-FDEA, [$^{13}C_2$]-FHUEA, [$^{13}C_2$]-FOUEA, [$^{13}C_2$]-FDUEA, d_3-MeFOSA, d_5-EtFOSA, d_7-MeFOSE, d_9-EtFOSE, 100 µL of a 0.1 µg/mL solution, see **Table 14**) to correct matrix effects as well as for losses sample extraction, concentration, and analysis. Tissues were extracted with 5 mL acetonitrile three times for 30 min in an ultrasonic bath at 30 °C. The combined extract was reduced to 2 mL using rotary evaporation and acidulated with 50 µL acetic acid. For clean-up Supelclean ENVI-Carb® cartridges (100 mg, 1 mL, 100-400 mesh, Supelco, USA) were used. The conditioning of the cartridge was carried out with 2 mL acetonitrile and 1 mL 20% acetic acid in acetonitrile. Afterwards, the sample extract and three times 1 mL methanol was given onto the cartridge and directly collected into another vial. The extracts were reduced to 150 µL under a nitrogen stream and 20 ng of an injection standard (InjS, d_5-EtFOSAA, 50 µL of a 0.4 µg/mL solution, see **Table 14**) was spiked to the final extract for corrections of instrumental drift and differences of the injection volume for instrumental analysis.

Concentrations of PFCs in samples were determined by high performance liquid chromatography with tandem mass spectrometer interfaced with an electrospray ionisation source in a negative-ion mode (HPLC-(-)ESI-MS/MS) as previously described (Yamashita et al. 2005). A detailed list of the precursor and product ions for the MS/MS can be found in **Table 14**. Quantification was done using response factors calculated by a ten-point calibration curve from 0.1 to 300 ng/mL. For quantification the linear range of 0.1 to 50 ng/mL and 50 to 300 ng/mL was used. Some PFSAs and sulfonamides showed more than one peak in the chromatogram, which is due to the presence of branched isomers resulting from the production process (Giesy and Kannan 2002). These branched isomers could not be quantified precisely because of the lack of calibration standards.

Table 14. Analytes, acronyms, formula, supplier, purity, precursor and product ions for the MS/MS detection

Analyte	Acronym	Formula	Supplier (purity)	Precursor/ product ion [m/z]
Perfluorobutane sulfonate	PFBS	$C_4F_9SO_2O^-$	Fluka (97%)	298.877/ 79.8
Perfluoropentane sulfonate	PFPS	$C_5F_{11}SO_2O^-$	n.a.	348.939/ 79.8
Perfluorohexane sulfonate	PFHxS	$C_6F_{13}SO_2O^-$	Fluka (98%)	398.894/ 79.8
Perfluoroheptane sulfonate	PFHpS	$C_7F_{15}SO_2O^-$	Well. Lab.[a] (>98%)	449.034/ 79.3
Perfluorooctane sulfonate	PFOS	$C_8F_{17}SO_2O^-$	Well. Lab.[a] (>98%)	498.971/ 79.7
Perfluorononane sulfonate	PFNS	$C_9F_{19}SO_2O^-$	n.a.	548.926/ 79.8
Perfluorodecane sulfonate	PFDS	$C_{10}F_{21}SO_2O^-$	Well. Lab.[a] (>98%)	598.896/ 79.5
6:2 fluorotelomer sulfonate	6:2 FTS	$C_6F_{13}C_2H_4SO_3^-$	ABCR (98%)	426.925/ 406.7
Perfluoro-1-hexane sulfinate	PFHxSi	$C_6F_{13}SO_2^-$	Well. Lab.[a] (>98%)	382.865/ 319.0
Perfluoro-1-octane sulfinate	PFOSi	$C_8F_{17}SO_2^-$	Well. Lab.[a] (>98%)	482.824/ 418.9
Perfluoro-1-decane sulfinate	PFDSi	$C_{10}F_{21}SO_2^-$	Well. Lab.[a] (>98%)	582.934/ 518.9
Perfluorobutanoic acid	PFBA	C_3F_7COOH	ABCR (99%)	112.900/ 168.7
Perfluoropentanoic acid	PFPA	C_4F_9COOH	Alfa Aesar (98%)	262.825/ 218.9
Perfluorohexanoic acid	PFHxA	$C_5F_{11}COOH$	Fluka (97%)	312.934/ 268.8
Perfluoroheptanoic acid	PFHpA	$C_6F_{13}COOH$	Lanc. Syn.[b] (98%)	362.950/ 318.9
Perfluorooctanoic acid	PFOA	$C_7F_{15}COOH$	Lanc. Syn.[b] (95%)	412.987/ 368.9
Perfluorononanoic acid	PFNA	$C_8F_{17}COOH$	Lanc. Syn.[b] (97%)	462.908/ 418.9
Perfluorodecanoic acid	PFDA	$C_9F_{19}COOH$	Lanc. Syn.[b] (97%)	512.876/ 469.0
Perfluoroundecanoic acid	PFUnDA	$C_{10}F_{21}COOH$	ABCR (96%)	562.865/ 519.0
Perfluorododecanoic acid	PFDoDA	$C_{11}F_{23}COOH$	Alfa Aesar (96%)	612.991/ 568.9
Perfluorotridecanoic acid	PFTriDA	$C_{12}F_{25}COOH$	Well. Lab.[a] (>98%)	663.094/ 618.9
Perfluorotetradecanoic acid	PFTeDA	$C_{13}F_{27}COOH$	Alfa Aesar (96%)	713.036/ 669.0
Perfluorotridecanoic acid	PFPDA	$C_{14}F_{29}COOH$	n.a.	762.980/ 718.9
Perfluorohexadecanoic acid	PFHxDA	$C_{15}F_{31}COOH$	Alfa Aesar (95%)	812.840/ 769.1
Perfluoroheptadecanoic acid	PFHpDA	$C_{16}F_{33}COOH$	n.a.	862.980/ 818.9
Perfluorooctadecanoic acid	PFOcDA	$C_{17}F_{35}COOH$	Alfa Aesar (97%)	912.870/ 869.0
3,7-dimethylperfluorooctanoic acid	3,7m$_2$-PFOA	$C_9F_{19}COOH$	Alfa Aesar (97%)	512.885/ 468.9
N-methylperfluorobutane sulfonamide	MeFBSA	$C_4F_9SO_2NH(CH_3)$	3M (n.a.)	311.914/ 218.8
Perfluorooctane sulfonamide	FOSA	$C_8F_{17}SO_2NH_2$	ABCR (97%)	497.896/ 77.9
N-methyl perfluorooctane sulfonamide	MeFOSA	$C_8F_{17}SO_2NH(CH_3)$	3M (n.a.)	511.849/ 168.9
N-ethyl perfluorooctane sulfonamide	EtFOSA	$C_8F_{17}SO_2NH(C_2H_5)$	ABCR (95%)	526.008/ 169.0
N-methylperfluorobutane sulfonamidoethanol	MeFBSE	$C_4F_9SO_2N(CH_3)C_2H_4OH$	3M (n.a.)	416.047/ 59.0
N-methyl perfluorooctane sulfonamidoethanol	MeFOSE	$C_8F_{17}SO_2N(CH_3)C_2H_4OH$	3M (n.a.)	616.004/ 58.9
N-ethyl perfluroctane sulfonamidoethanol	EtFOSE	$C_8F_{17}SO_2N(C_2H_5)C_2H_4OH$	3M (n.a.)	630.109/ 58.8
2-Perfluorohexyl ethanoic acid	FHEA	$C_6F_{13}CH_2COOH$	Well. Lab.[a] (>98%)	376.945/ 292.8
2-Perfluorooctyl ethanoic acid	FOEA	$C_8F_{17}CH_2COOH$	Well. Lab.[a] (>98%)	476.909/ 392.9
2-Perfluorodecyl ethanoic acid	FDEA	$C_{10}F_{21}CH_2COOH$	Well. Lab.[a] (>98%)	577.011/ 493.0
2H-Perfluoro-2-octenoic acid	FHUEA	$C_6F_{12}CHCOOH$	Well. Lab.[a] (>98%)	356.885/ 293.0
2H-Perfluoro-2-decenoic acid	FOUEA	$C_8F_{16}CHCOOH$	Well. Lab.[a] (>98%)	456.803/ 292.8
2H-Perfluoro-2-dodecenoic acid	FDUEA	$C_{10}F_{20}CHCOOH$	Well. Lab.[a] (>98%)	556.937/ 493.1
Perfluoro-1-hexane[$^{18}O_2$]sulfonate	[$^{18}O_2$]-PFHxS	$C_6F_{13}S[^{18}O_2]O^-$	Well. Lab.[a] (>98%)	402.981/ 83.9
Perfluoro-1-[1,2,3,4-13C]octanesulfonate	[$^{13}C_4$]-PFOS	$C_4F_9[1,2,3,4\text{-}^{13}C_4]F_8SO_2O^-$	Well. Lab.[a] (>98%)	502.899/ 79.5
Perfluoro-1-[1,2,3,4-13C]octanesulfinate	[$^{13}C_4$]-PFOSi	$C_4F_9[1,2,3,4\text{-}^{13}C_4]F_8SO_2^-$	Well. Lab.[a] (>90%)	486.859/ 422.9
Perfluoro-n-(1,2,3,4-$^{13}C_4$)butanoic acid	[$^{13}C_4$]-PFBA	$2,3,4\text{-}^{13}C_3F_7{}^{13}COOH$	Well. Lab.[a] (>98%)	216.823/ 171.8
Perfluoro-n-(1,2-$^{13}C_2$)hexanoic acid	[$^{13}C_2$]-PFHxA	$C_4F_9[2\text{-}^{13}C]F_2{}^{13}COOH$	Well. Lab.[a] (>98%)	314.891/ 269.9
Perfluoro-n-[1,2,3,4-$^{13}C_4$]octanoic acid	[$^{13}C_4$]-PFOA	$C_4F_9[2,3,4\text{-}^{13}C_3]F_6{}^{13}COOH$	Well. Lab.[a] (>98%)	416.978/ 371.8
Perfluoro-n-[1,2,3,4,5-$^{13}C_5$]nonanoic acid	[$^{13}C_5$]-PFNA	$C_4F_9[2,3,4,5\text{-}^{3}C_4]F_8{}^{13}COOH$	Well. Lab.[a] (>98%)	467.907/ 423.0
Perfluoro-n-[1,2-$^{13}C_2$]decanoic acid	[$^{13}C_2$]-PFDA	$C_8F_{17}{}^{13}CF_2{}^{13}COOH$	Well. Lab.[a] (>98%)	514.944/ 469.8
Perfluoro-n-[1,2-$^{13}C_2$]undecanoic acid	[$^{13}C_2$]-PFUnDA	$C_9F_{19}{}^{13}CF_2{}^{13}COOH$	Well. Lab.[a] (>98%)	564.959/ 519.8
Perfluoro-n-[1,2-$^{13}C_2$]dodecanoic acid	[$^{13}C_2$]-PFDoDA	$C_{10}F_{21}{}^{13}CF_2{}^{13}COOH$	Well. Lab.[a] (>98%)	614.913/ 569.9
N-methyl-d$_3$-perfluoro-1-octanesulfonamide	d$_3$-N-MeFOSA	$C_9D_3HF_{17}NO_2S$	Well. Lab.[a] (>98%)	514.920/ 168.8
N-ethyl-d$_5$-perfluoro-1-octanesulfonamide	d$_5$-N-EtFOSA	$C_{10}D_5HF_{17}NO_2S$	Well. Lab.[a] (>98%)	530.984/ 168.8
2-(N-deuteriomethylperfluoro-1-octane-sulfoneamido)-1,1,2,2-tetradeuterioethanol	d$_7$-N-MeFOSE	$C_8F_{17}SO_2N(CD_3)C_2D_4OH$	Well. Lab.[a] (>98%)	623.058/ 58.9
2-(N-deuterioethylperfluoro-1-octane-sulfoneamido)-1,1,2,2-tetradeuterioethanol	d$_9$-N-EtFOSE	$C_8F_{17}SO_2N(C_2D_5)C_2D_4OH$	Well. Lab.[a] (>98%)	639.054/ 58.9
2-Perfluorohexyl-[1,2-$^{13}C_2$]ethanoic acid	[$^{13}C_2$]-FHEA	$C_6F_{13}{}^{13}CH_2{}^{13}COOH$	Well. Lab.[a] (>98%)	378.912/ 294.0
2-Perfluorooctyl-[1,2-$^{13}C_2$]ethanoic acid	[$^{13}C_2$]-FOEA	$C_8F_{17}{}^{13}CH_2{}^{13}COOH$	Well. Lab.[a] (>98%)	478.911/ 393.8
2-Perfluorodecyl-[1,2-$^{13}C_2$]ethanoic acid	[$^{13}C_2$]-FDEA	$C_{10}F_{21}{}^{13}CH_2{}^{13}COOH$	Well. Lab.[a] (>98%)	579.017/ 494.1
2H-Perfluoro-[1,2-$^{13}C_2$]-2-octenoic acid	[$^{13}C_2$]-FHUEA	$C_6F_{12}{}^{13}CH{}^{13}COOH$	Well. Lab.[a] (>98%)	358.907/ 294.0
2H-Perfluoro-[1,2-$^{13}C_2$]-2-decenoic acid	[$^{13}C_2$]-FOUEA	$C_8F_{16}{}^{13}CH{}^{13}COOH$	Well. Lab.[a] (>98%)	458.903/ 393.8
2H-Perfluoro-[1,2-$^{13}C_2$]-2-dodecenoic acid	[$^{13}C_2$]-FDUEA	$C_{10}F_{20}{}^{13}CH{}^{13}COOH$	Well. Lab.[a] (>98%)	558.955/ 494.0
N-deuterioethylperfluoro-1-octanesulfonamidoacetic acid	d$_5$-EtFOSAA	$C_8F_{17}SO_2N(C_2D_2C_2D_3)C_2H_2CO_2H$	Well. Lab.[a] (>98%)	589.015/ 418.7

[a] Well. Lab. = Wellington Laboratories; [b] Lanc. Syn. = Lancaster Synthesis; n.a. = not available.

As the analytical standards are not available for perfluorinated pentane- and nonanesulfonate (PFPS, PFNS) and perfluorinated pentadecanoic and heptadecanoic acid (PFPDA, PFHpDA), they were integrated into the method taking the MS/MS parameters of the compound having one carbon atom less in the carbon chain and their calibration was used for the quantification. Hence, the results given for PFPS, PFNS, PFPDA and PFHpDA should be considered only as an estimation.

5.2.3 Quality control

Data quality assurance and quality control included method blanks, method detection limits (MDLs), method quantification limits (MQLs), matrix spike recovery rates, matrix effect and continuing calibration verification. For the method blank one mL of acetonitrile was extracted in the same way as the natural samples. The MDLs and MQLs were calculated for substances which were found in real samples at a signal to noise (S/N) of 3 and 10, respectively. PFC recoveries were tested for liver tissues based on triplicate analysis of matrix spiked and extracted with the same analytical procedure.

All method blanks were under the MQL. The MQLs ranged from a few pg/g ww (e.g., perfluorooctanoic acid (PFOA)) to a few ng/g ww (PFOS), depending on the extracted tissue (**Table S9** in the Supplementary material). Relative recoveries of the 36 analytes, which were corrected for IS recovery and background concentration, ranged between 56% (perfluorohexadecanoic acid (PFHxDA)) and 135% (n-ethylperfluorooctane sulfonamidoethanol (EtFOSE)) (**Table S10** in the Supplementary material). The matrix effects of individual PFCs were determined in liver, kidney, lung, heart, blood, brain and muscle by analysis of a fortified extract (100 µL of a 0.1 µg/mL PFC standard solution), non-fortified extract and solvent based standard solution (matrix effect = ($response_{fortified\ extract}$ − $response_{non\text{-}fortified\ extract}$)/$response_{solvent\ based\ standard}$) (**Table S11** in the Supplementary material). Most PFCs showed similar matrix effects in different tissues and a low mean signal suppression of 0.88 to 0.98 except of 3,7-dimethylperfluorooctanoic acid (3,7m_2-PFOA) with a low signal enhancement of 1.01. Only PFHxDA and perfluorooctadecanoic acid (PFOcDA) have a stronger signal suppression in some tissues with a maximum of 0.24 (kidney) and 0.11 (lung), respectively.

5.3. Results and discussion

5.3.1 Tissue distribution

Levels of PFCs in liver, kidney, lung, heart, blood, brain, muscle, thyroid, thymus and blubber in harbor seals are shown in **Table 15**. 18 of 40 target analytes were found in the tissues (i.e., C_4 to C_{10} PFSAs, perfluorooctanesulfinate (PFOSi), C_8 to C_{15} PFCAs,

perfluorosulfonamide (FOSA) and n methylperfluorobutane sulfonamidoethanol (MeFBSE)). To our knowledge, this is the first report of PFPS, PFNS, PFOSi and MeFBSE in detectable concentrations in biota samples from the German Bight. Among to all detected PFCs the predominated compound in all tissues was PFOS with an average of over 90%, followed by perfluorohexanesulfonate (PFHxS) (2.7%), perfluorononanoic acid (PFNA) (1.8%) and perfluorodecanoic acid (PFDA) (1.6%). The highest PFC sum concentrations were detected in liver (1071 ng/g ww), lung (462 ng/g ww) and blood (381 ng/g ww). The lowest PFC sum concentration was found in blubber with an average of 11.4 ng/g ww. This confirms with the findings that PFCs bind to blood proteins instead of fatty tissue (Jones et al. 2003).

All PFSAs with a chain length of C_4-C_{10} could be detected in the analysed tissues. Although 3M, the major producer of perfluorooctyl sulfonyl fluoride (POSF), which is a major precursor for several PFCs, voluntarily phased out the production in 2002, PFOS was still the dominated compound in biota samples. The former POSF-based products are now substituted by perfluorobutyl sulfonyl fluoride (PBSF)-based products (U.S. EPA 2000). In the present study PFBS could be detected with a maximum concentration of 17 ng/g ww in blood, but usually the concentration was less than 0.5 ng/g ww. The production shift to the shorter-chained PFBS could not be observed because of the lower accumulation potential of the C_4 in comparison to the C_8 PFSA (Martin et al. 2003b). In contrast to the PFSAs the PFCAs could only be detected from a chain length grater than C_8, this suggest a higher accumulation potential of PFSAs compared to PFCAs (Martin et al. 2003b). PFNA and PFDA were the dominated PFCAs, with increasing chain length up to C_{15} concentration levels decreased. Two sulfonamides, which are precursors of PFSAs and PFCAs (Martin et al. 2006), were also detected. FOSA was detected in all tissues with a maximum of 6.9 ng/g ww in blood and MeFBSE was only observed in thyroid tissue and blubber with up to 2.0 ng/g ww.

Table 15. Average concentration (ng/g wet weight), standard deviation (SD) and ranges of PFCs in different organs and tissues of harbor seals from the German Bight (n = 4)

		Liver	Kidney	Lung	Heart	Blood	Brain	Muscle	Thyroid	Thymus	Blubber
PFBS	mean ± SD	0.32 ± 0.34	0.10 ± 0.15	0.17 ± 0.16	0.13 ± 0.18	4.32 ± 8.45	n.d.	0.02 ± 0.05	0.11 ± 0.22	0.07 ± 0.12	n.d.
	range	0-0.78	0-0.32	0.06-0.41	0-0.39	0.03-17.0	n.d.	0-0.10	0-0.43	0-0.21	n.d.
PFPS [a]	mean ± SD	1.75 ± 2.46	0.09 ± 0.11	0.09 ± 0.05	0.04 ± 0.07	5.90 ± 11.66	n.d.	n.d.	n.d.	0.07 ± 0.06	n.d.
	range	0.13-5.38	0-0.24	0.04-0.15	0-0.15	0-23.4	n.d.	n.d.	n.d.	0-0.11	n.d.
PFHxS	mean ± SD	6.90 ± 4.03	5.68 ± 3.83	8.14 ± 4.82	4.32 ± 3.33	3.16 ± 1.08	1.58 ± 1.00	1.94 ± 1.41	4.11 ± 2.64	10.49 ± 6.19	0.66 ± 0.48
	range	1.11-10.4	1.05-9.84	1.91-13.3	0.60-7.64	1.67-4.13	0.27-2.48	0.34-3.74	0.68-6.87	3.38-14.6	0.14-1.30
PFHpS	mean ± SD	2.27 ± 2.29	1.24 ± 0.87	3.67 ± 2.67	1.32 ± 1.15	0.66 ± 0.66	0.66 ± 0.45	0.41 ± 0.32	1.22 ± 1.17	3.43 ± 2.51	0.10 ± 0.15
	range	0-5.43	0.15-2.09	0.83-7.12	0.08-2.82	0-1.58	0.23-1.15	0.02-0.71	0-2.78	0.84-5.86	0-0.32
PFOS	mean ± SD	1017 ± 536	288 ± 117	433 ± 227	143 ± 40	349 ± 370	99 ± 49	59 ± 52	62 ± 58	312 ± 136	8.91 ± 9.93
	range	559-1665	118-383	228-755	87-181	48-887	38-153	7.65-132	0-121	159-416	0-23
PFNS [a]	mean ± SD	0.74 ± 0.74	0.06 ± 0.09	0.11 ± 0.12	n.d.	0.10 ± 0.11	n.d.	n.d.	0.08 ± 0.15	0.11 ± 0.04	n.d.
	range	0.12-1.80	0-0.19	0-0.27	n.d.	0-0.25	n.d.	n.d.	0-0.30	0.06-0.14	n.d.
PFDS	mean ± SD	0.53 ± 0.38	0.14 ± 0.16	0.18 ± 0.16	0.06 ± 0.08	0.12 ± 0.13	0.04 ± 0.08	n.d.	0.12 ± 0.15	0.20 ± 0.08	0.03 ± 0.03
	range	0.11-1.02	0-0.37	0-0.38	0-0.16	0-0.31	0-0.16	n.d.	0-0.33	0.11-0.27	0-0.06
PFOSi	mean ± SD	0.05 ± 0.08	n.d.	0.02 ± 0.04	n.d.	0.03 ± 0.05	n.d.	n.d.	n.d.	n.d.	n.d.
	range	0-0.16	n.d.	0-0.07	n.d.	0-0.09	n.d.	n.d.	n.d.	n.d.	n.d.
∑PFSAs/∑PFSiAs		1030	295	445	149	364	101	61	67	327	9.7
PFOA	mean ± SD	0.70 ± 0.59	0.40 ± 0.41	0.75 ± 0.46	0.42 ± 0.42	0.62 ± 0.58	0.06 ± 0.10	0.07 ± 0.11	0.09 ± 0.11	0.70 ± 0.25	0.03 ± 0.04
	range	0-1.42	0-0.93	0.28-1.21	0-0.99	0-1.14	0-0.20	0-0.24	0-0.22	0.43-0.93	0-0.08
PFNA	mean ± SD	15.3 ± 5.75	3.64 ± 0.62	4.84 ± 2.06	2.07 ± 0.84	3.93 ± 2.08	1.20 ± 0.50	0.96 ± 0.35	1.90 ± 1.19	4.99 ± 1.22	0.61 ± 0.27
	range	8.27-22.3	2.97-4.22	2.06-6.55	1.13-2.86	0.88-5.54	0.77-1.91	0.45-1.25	0.80-3.58	3.60-5.91	0.30-0.89
PFDA	mean ± SD	15.2 ± 4.49	4.11 ± 2.09	5.18 ± 1.63	2.44 ± 1.06	4.38 ± 2.35	1.55 ± 0.47	1.09 ± 0.46	1.23 ± 0.59	5.65 ± 2.04	0.29 ± 0.22
	range	8.83-19.0	1.99-6.97	2.90-6.70	1.37-3.88	0.86-5.68	0.95-2.00	0.56-1.67	0.52-1.74	3.31-7.05	0.01-0.51
PFUnDA	mean ± SD	5.26 ± 1.59	1.92 ± 0.69	2.80 ± 0.88	1.36 ± 0.50	1.71 ± 0.84	1.06 ± 0.16	0.26 ± 0.16	0.31 ± 0.34	2.30 ± 0.57	0.088 ± 0.10
	range	3.29-6.62	0.95-2.54	1.71-3.74	0.75-1.92	0.46-2.26	0.90-1.20	0.10-0.42	0-0.61	1.65-2.70	0-0.19
PFDoDA	mean ± SD	1.47 ± 0.49	0.75 ± 0.37	1.10 ± 0.43	0.54 ± 0.25	0.47 ± 0.24	0.51 ± 0.36	0.06 ± 0.11	0.18 ± 0.35	0.42 ± 0.13	0.042 ± 0.067
	range	1.04-2.17	0.27-1.16	0.74-1.63	0.24-0.84	0.25-0.74	0-0.84	0-0.22	0-0.70	0.31-0.57	0-0.14
PFTriDA	mean ± SD	1.53 ± 0.55	1.01 ± 0.54	1.27 ± 0.63	0.77 ± 0.47	0.76 ± 0.34	0.73 ± 0.55	0.12 ± 0.83	0.51 ± 0.43	1.00 ± 0.16	0.12 ± 0.090
	range	0.74-1.96	0.58-1.74	0.54-2.02	0.33-1.31	0.29-1.03	0-1.31	0-0.19	0.04-1.07	0.86-1.18	0-0.20
PFTeDA	mean ± SD	0.22 ± 0.16	0.05 ± 0.06	0.16 ± 0.22	0.06 ± 0.12	0.08 ± 0.06	0.10 ± 0.12	n.d.	0.05 ± 0.07	0.21 ± 0.17	n.d.
	range	0.08-0.44	0-0.11	0-0.46	0-0.23	0-0.15	0-0.23	n.d.	0-0.15	0.07-0.40	n.d.
PFPeDA [a]	mean ± SD	n.d.	n.d.	0.13 ± 0.26	n.d.	0.04 ± 0.05	n.d.	n.d.	n.d.	n.d.	n.d.
	range	n.d.	n.d.	0-0.53	n.d.	0-0.11	n.d.	n.d.	n.d.	n.d.	n.d.
∑PFCAs		39.7	11.9	16.2	7.65	12.0	5.20	2.55	4.27	15.3	1.17
FOSA	mean ± SD	1.55 ± 0.69	0.62 ± 0.44	0.40 ± 0.027	0.27 ± 0.18	5.06 ± 1.23	0.14 ± 0.14	0.07 ± 0.08	0.02 ± 0.02	0.46 ± 0.38	0.03 ± 0.04
	range	0.78-2.32	0-1.02	0.37-0.44	0.06-0.48	4.08-6.85	0-0.33	0-0.15	0-0.05	0.03-0.74	0-0.08
MeFBSE	mean ± SD	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.14 ± 0.27	n.d.	0.50 ± 1.00
	range	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0-0.55	n.d.	0-2.01
∑FOSA/FOSE		1.55	0.62	0.40	0.27	5.06	0.14	0.07	0.16	0.46	0.53
∑PFCs		1071	308	462	157	381	106	64	72	343	11

[a] Have to be considered as estimates, because no standards were available for this compound; n.d. = not detected.

PFCs are distributed on a global scale, with highest concentrations found close to urbanised/industrialised regions like Europe and USA (e.g., 970-3680 ng/g ww PFOS in Mink livers from Midwestern U.S.), while the Southern Hemisphere was less contaminated with PFCs (e.g., <0.08-3.52 ng/g ww for PFOS in elephant seal livers from the Antarctic) (Giesy and Kannan 2001; Tao et al. 2006). In most studies PFOS was the dominating PFC in marine animals (Houde et al. 2006c). PFCAs were detected in seal liver in the Canadian Arctic in the same range than those in this study, except of PFOS, which was found one magnitude lower (Butt et al. 2007b; Sturman et al. 2007). A similar trend was observed in pelicans (*Pelecanus occidentalis*) from Columbia where the concentration of PFOS in liver, kidney, lung, heart, brain and muscle from harbor seals were, on average, one to two magnitudes lower than in this study (Olivero-Verbel et al. 2006). This may be the result of the higher pollutant area around the North Sea in comparison to the Arctic and Columbia. The tendency of increasing PFOS concentration in the different tissues was comparable to this study in harbor seals (Phoca vitulina) (kidney > liver > blubber), ringed seals (*Phoca hispida*) (liver > lung > heart and liver > spleen > kidney, respectively) (Sturman et al. 2007) and rainbow trouts (*Oncorhynchus mykiss*) (blood > kidney > liver) (Martin et al. 2003a).

5.3.2 Total body burden

The calculation of the whole body burden distribution based on the individual tissue masses of the whole organs (concentration in the sub-sample x tissue weight) (see **Table 13**). The organs from thymus, thyroid, liver, kidney, heart and one brain were tared directly. The content of blood, not directly tared brains, lung and muscle was calculated based on the mass information found in the literature (Bininda-Emonds 2000; Burns et al. 2005; Stewardson et al. 1999). The blubber content was estimated with the dorsal blubber thickness, standard length and body mass as described by Ryg et al. 1990. The calculation included all examined tissues, which was around 75% of the whole body weight. Among others the skeleton and the pelt was unaccounted for the calculation because the extraction would be too difficult and the expected PFC concentrations very low. The mean whole body burden in harbor seals of all detected ∑PFCs was estimated to be 2665 ± 1207 µg absolute (**Table 16**, n = 4). The greatest proportion had PFOS (2477 ± 1122 µg absolute) which could be have potential developmental, reproductive, systemic and neuroendocrine effects to mammals (Austin et al. 2003). The high PFCs body burden which was found in this study could also have effects on the immune system and physiological functions (Kannan et al. 2006). The four seals investigated in the present study were all in moderate nutritional status. The main pathological findings were bronchopneumonia caused by parasitic and bacterial infection partly with final

septicemia (personal communication with Ursula Siebert). Therefore an effect of PFCs on the health status can not be routed out.

Table 16. Mean whole body burden and standard deviation for individual PFC in harbor seals from the German Bight in µg absolute (n = 4)

Analyte	Whole body burden [µg]
PFBS	12 ± 22
PFPS	17 ± 35
PFHxS	33 ± 9.1
PFHpS	8.6 ± 3.2
PFOS	2477 ± 1122
PFNS	1.0 ± 0.71
PFDS	1.0 ± 0.49
PFOSi	0.12 ± 0.17
PFOA	3.3 ± 1.6
PFNA	35 ± 7.8
PFDA	36 ± 7.8
PFUnDA	13 ± 2.8
PFDoDA	3.9 ± 1.1
PFTriDA	5.6 ± 1.1
PFTeDA	0.52 ± 0.21
PFPeDA	0.15 ± 0.14
FOSA	16 ± 3.3
MeFBSE	1.9 ± 0.01
∑PFCs	2665 ± 1207

Distribution of individual PFC in different tissues of harbor seals is presented in **Figure 16**. The whole body burden was related as follows: Blood ≈ liver > muscle > lung > kidney ≈ blubber > Heart ≈ brain >> thymus > thyroid (**Figure 15**).

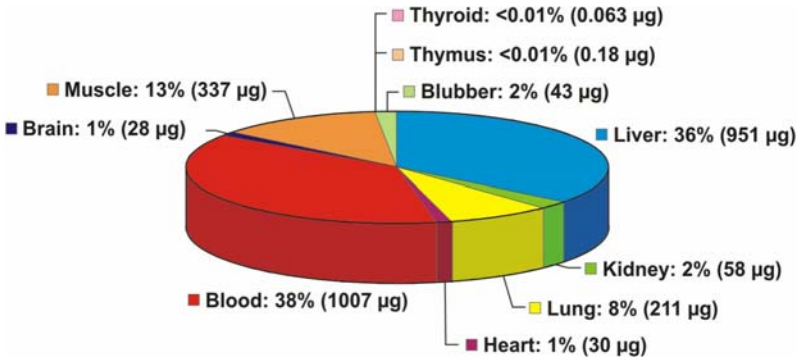

Figure 15. PFC whole body burden distribution in percent and µg per tissue in brackets for harbor seals from the German Bight

Blood and liver contributed three-fourths of the whole body burden for PFCs, but the

composition differed from compound to compound. For monitoring of PFCs in marine mammals in the North Sea it would be meaningful to collect liver or blood samples, because there were found an effectively accumulation of these compounds resulting in the highest concentrations of all examined tissues. Muscle and blubber tissue corresponded approximately to two-thirds of sum weight of all analysed tissues, but the PFC body burden of this both tissues was only 13% and 2%, respectively. However, the pattern of PFCs varied depending on the functional group and fluorinated chain length. It is noticeable that the proportion of the PFSAs in liver increased with increasing chain length, whereas the short-chained PFBS and PFPS were found with over 90% in blood. Otherwise, the PFCAs showed no obvious differences in the pattern between the different tissues. FOSA was mostly distributed in blood and MeFOSE in blubber. These different patterns indicated a compound-specific persistence of PFCs in different tissues of harbor seals.

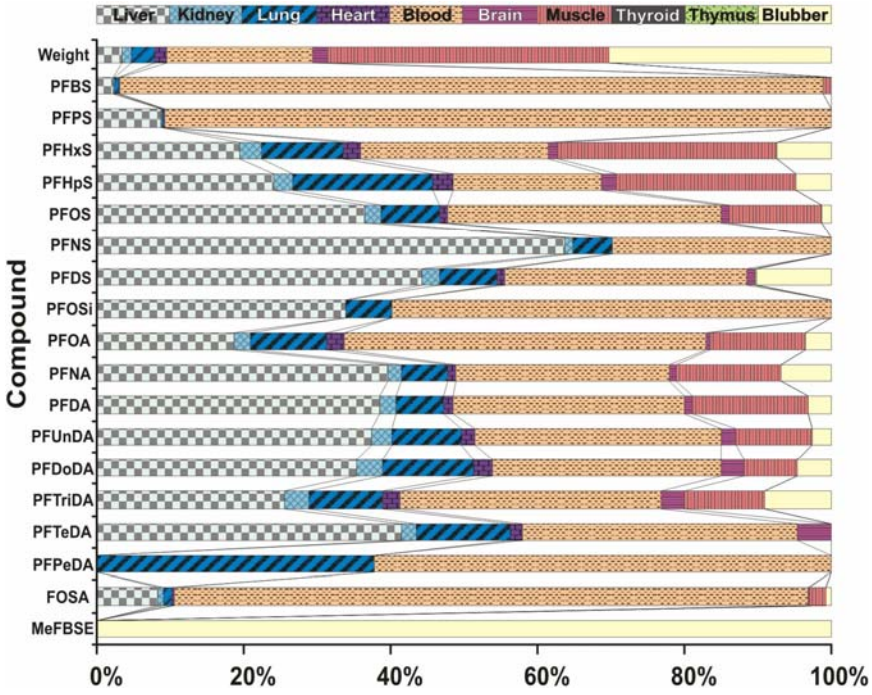

Figure 16. Tissue distribution of PFC burdens in harbor seals from the German Bight

5.4. Conclusion

In comparison to this study, PFC concentrations in ringed seals from the Canadian Arctic and elephant seals from the Antarctic were by a factor of ~100 and ~1000-10000 lower, respectively (Giesy and Kannan 2001; Tao et al. 2006). The occurrence of high concentrations of PFCs in harbor seals in the German Bight suggests that these compounds should also be found at several levels in the marine food chain. This could possibly be problematic, because the North Sea is an important source for the fishery and shrimp industry of the countries

bordering to the North Sea. A recent study has shown high concentrations of PFCs were found in plasma and whole blood of the Swedish population, which were usually much higher than other detected persistent organic pollutants (Kärrman et al. 2006b). Little information is available on the exposure of PFCs in the marine environment, further investigations about the accumulation potential and whole body burden in marine wildlife are necessary to assess potential adverse effects of PFCs. This study provides advice on the analysis of the whole body burden in harbor seals for individual PFCs, which is relevant for calculation of the bioaccumulation potential of these compounds in marine mammals.

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6. Publication IV

Temporal Trends of Polyfluoroalkyl Compounds in Harbor Seals (Phoca vitulina) from the German Bight, 1999-2008

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Abstract

Temporal trends of polyfluoroalkyl compounds (PFCs) were examined in liver samples from harbor seals (*Phoca vitulina*) collected from the German Bight (1999-2008). Concentrations of various PFCs, including C_4-C_{10} perfluoroalkyl sulfonates (PFSAs), perfluorooctane sulfinate (PFOSi), perfluorooctane sulfonamide (FOSA) and C_8-C_{15} perfluoroalkyl carboxylic acids (PFCAs) were quantified. PFOS was the predominant compound with a maximum concentration of 3676 ng/g ww (1996), making up on average 94% of the measured PFCs. Significantly higher concentrations were found in <7 month old in comparison to ≥ 7 month old harbor seals for C_6-C_8 PFSAs, perfluorododecanoic acid (PFDoDA) and FOSA, whereas perfluorodecanoic acid (PFDA) showed significantly lower concentrations in the younger harbor seals ($p < 0.05$). These results suggest a transplacental transfer of PFCs to the foetus and/or consumption of different contaminated food. Regression analysis of logarithmic transformed PFC mean concentrations indicated a significant temporal trend with decreasing concentrations for C_5-C_7 PFSAs ($p < 0.001$), PFOSi ($p = 0.028$), FOSA ($p < 0.001$) and perfluorooctanoic acid (PFOA) ($p = 0.031$) between 1999 and 2008. Furthermore, perfluorooctane sulfonate (PFOS) decreased by 49% between 1999 and 2008, which correspond with decreasing concentration levels of its metabolic precursors PFOSi and FOSA of 83% and 95% in the same time period. However, the decreasing trend of PFOS is not significant ($p = 0.067$). The reason for the decline during the past 10 years could be an effect of the replacement of these PFCs by shorter-chained and less bioaccumulative

compounds. But the observations of increasing perfluorodecane sulfonate (PFDS) levels ($p = 0.070$), the high concentrations of PFOS and constant levels of C_9-C_{13} PFCAs indicates that further work on the reduction of environmental emissions of PFCs are necessary.

6.1. Introduction

Polyfluoroalkyl compounds (PFCs) received increasing public attention due to their persistence, bioaccumulative potential (Martin et al. 2003a) and possible adverse effects on human and wildlife (Austin et al. 2003; Oakes et al. 2004). PFCs are widely used as processing additives during fluoropolymer production and as surfactants in consumer applications, including surface coatings for carpets, furniture and paper products over the past 50 years (Kissa 2001). From the production and use of these products, PFCs can be potentially released into the environment. PFCs were found ubiquitously in water (Yamashita et al. 2005), sediment (Higgins and Luthy 2006), wildlife (Giesy and Kannan 2001) and humans (Yeung et al. 2006), highest concentrations were found in marine top predators (Houde et al. 2006c). As a result, the 3M Company, the major producer of perfluorooctyl sulfonyl fluoride (POSF, which is a major precursor for several PFCs) voluntarily phased out the production in 2000, but a variety of related PFCs are still being produced by other manufacturers (Prevedouros et al. 2006). Furthermore, in 2006, the U.S. Environmental Protection Agency (U.S. EPA) launched a voluntary stewardship program to reduce perfluorooctanoic acid (PFOA) and related chemicals from facility emissions and product content by 95% by 2010, and to work toward elimination of emissions and content by 2015 (U.S. EPA 2006). In addition the European Union (EU) formed a directive in October 2006, which prohibits the general use of perfluorooctane sulfonate (PFOS) and their derivates from June 2008 (European Parliament and Council 2006).

Previous temporal trend studies indicated mostly increasing concentrations of PFCs in biota from the Arctic (Bossi et al. 2005a; Butt et al. 2007a; Dietz et al. 2008; Smithwick et al. 2006). A temporal trend study on polar bears (*Ursus maritimus*) showed increasing concentrations of perfluorooctane sulfonate (PFOS) and longer-chained perfluoroalkyl carboxylic acids (PFCAs) between 1972 and 2002, and between 1984 and 2006, respectively (Smithwick et al. 2006; Dietz et al. 2008). An increase in PFOS and PFCAs was also observed in two seabird species (i.e., thick-billed murres (*Uria lomvia*) and northern fulmars (*Fulmaris glacialis*)) between 1987 and 1993, whereas the concentration in northern fulmars were relatively constant between 1993 and 2003 (Butt et al. 2007a). Results of a long-term assessment (1968-2003) in guillemot (*Uria aalge*) eggs from the Baltic Sea have shown an almost 30-fold increase in PFOS concentrations with a decrease after 2002 (Holmström et al. 2005). Recently, a few temporal trend studies observed a significant decreasing trend of

perfluorooctane sulfonamide (FOSA) (Butt et al. 2007b; Furdui et al. 2008; Hart et al. 2008a), and one study found additionally a significant decreasing trend of PFOS in Arctic ringed seals (*Phoca hispida*) (Butt et al. 2007b). These decreasing levels could be caused by restrictions and bans of production and/or use of POSF. However, recent temporal trend data on PFCs in biota tissue close to urbanised/industrialised regions with potential high PFC emissions are lacking in the published literature. This information is needed to examine effects of the reductions in overall emissions of PFCs on the contamination levels in marine mammals. Such information is useful for any future strategies for the marine ecosystem to reduce PFC contaminations.

The aim of this study was to examine temporal trends (1999-2008) and composition profiles of archived harbor seal livers (*Phoca vitulina*) collected from the German Bight. In addition, the association between PFC concentrations in livers of harbor seals and the evidence of diseases, spatial distribution, age and sex were evaluated. Seals were selected because they are top predators in the marine ecosystem and accumulate various pollutants (Braune et al. 2005). Another advantage is that harbor seals are relatively sedentary in their habitat of around 400 km^2 so that local contaminations can be identified (Reijnders et al. 2005). Concentrations of various PFCs, including C_4-C_{10} perfluoroalkyl sulfonates (PFSAs), perfluorooctane sulfinate (PFOSi), FOSA and C_8-C_{15} PFCAs were quantified. These results show for the first time significant decreasing concentration of C_5-C_7 PFSAs, PFOSi and PFOA in marine mammals.

6.2. Materials and methods

6.2.1. Sample collection

Harbor seal liver samples were collected in the German Bight during 1988 to 2008 (**Table S12** in the Supplementary material). All harbor seals were stranded or shot by trained personnel due to severe illness. Necropsies were conducted on the carcasses at the Research and Technology Centre Westcoast (FTZ) according to the protocol described by Siebert et al. 2007. Sex and weight of the animals and their livers were measured (see **Table S12** in the Supplementary material). Based on the date when the individuals were found and the length of the animals the seals were grouped into <7 month (category 1) and ≥ 7 month (category 2) age classes. 19 of category 1 and 44 harbor seals of category 2 were examined, whereas the number of samples varied from 1 (2004) to 13 (2007), except for 1989 to 1995 and 1997 to 1998 when no samples were collected. The nutritional status of the animals was judged based on the weight, blubber thickness and state of muscles and categorized in ‘good’, ‘moderate’ and ‘emaciated’. The general health status was evaluated based on macroscopical,

histological, microbiological and virological findings and divided into the three categories ‘good’, ‘moderate’ and ‘poor’. Histological lesions of the liver were judged based on the severeness into mild, moderate and severe. All liver samples for PFC analysis were taken with stainless steel instruments, placed into polypropylene (PP) bags and stored in a -20 °C freezer until analysis.

6.2.2. Extraction and analysis

The target analytes include 33 ionic PFCs (PFCAs, PFSAs, perfluoroalkyl sulfinates (PFSiAs), fluorotelomercarboxylic acids (FTCAs) and unsaturated fluorotelomercarboxylic acids (FTUCAs)) as well as 7 neutral PFC precursor compounds (perfluoroalkyl sulfonamides, perfluoroalkyl sulfonamidoethanols). A list of the native and mass-labelled standards including their acronyms, formula, supplier and purity are presented in **Table 14**. Methanol (SupraSolv), acetonitrile (LiChrosolv) and acetic acid (glacial, > 99%) were purchased from Merck.

Liver samples were extracted based on the solid-liquid extraction method described by Powley et al. 2005 with a modified cleanup step (Ahrens et al. 2009e). The separation and detection of PFCs were performed by liquid chromatography with tandem mass spectrometer interfaced with an electrospray ionisation source in a negative-ion mode (LC-(-)ESI-MS/MS) as previously described (Ahrens et al. 2009e; Yamashita et al. 2005).

6.2.3. Quality assurance

The analytical quality of the laboratory has been approved in interlaboratory studies (Van Leeuwen et al. 2009). As standard procedure, blanks, instrument detection limits (IDLs), method quantification limits (MQLs), matrix effects and recoveries of spiked samples were examined (see **Table S13** in the Supplementary material). For the method blank 1 mL of acetonitrile was extracted in the same way as the real samples. The IDLs were determined using the calibration standards at a signal to noise (S/N) of 3, while MQLs were calculated for substances which were found in real samples at a S/N of 10. The matrix effects of individual PFCs were determined in liver tissue by analysis of a fortified extract (100 µL of a 0.1 µg/mL PFC standard solution), non-fortified extract and solvent based standard solution (matrix effect = $(\text{response}_{\text{fortified extract}} - \text{response}_{\text{non-fortified extract}})/\text{response}_{\text{solvent based standard}}$). PFC recoveries were tested for liver tissues based on triplicate analysis of matrix spiked samples and extracted with the same analytical procedure.

All method blanks were under the MQL. The MQLs ranged from a few tens of pg/g wet weight (ww) (e.g. perfluorooctanoic acid (PFOA)) to a few ng/g ww (PFOS), depending on the compound. Only PFHxDA and PFOcDA showed relevant signal suppression in liver

tissue of 0.50 and 0.38, respectively. The matrix effect of the other PFCs ranged from 0.83 (PFOSi) to 1.05 (FOSA). Matrix spike recoveries for all analytes ranged from 56% (PFHxDA) to 135% (EtFOSE) (mean ± standard deviation; 95% ± 22).

6.2.4. Statistical methods

Statistical analyses was performed using SPSS for Windows (version 16) and Microsoft Excel at a significance level of $\alpha = 0.05$. PFCs, which were detected in over 50% of the analysed samples, were used for the statistical comparison of means and temporal trends. Data were natural-logarithm transformed prior to statistical analysis to meet assumption of normality and homogeneity of variances. Significant differences were observed between the two age classes (<7 month old and ≥ 7 month old, respectively) within 2006 and 2007 (Mann-Whitney U test), for this reason the remaining statistical analyses were performed for only the harbor seals ≥ 7 month old, because of their greater sample number (n = 44). Pearson analysis was used for correlations between individual compounds concentrations. The t-test was used to assess differences between sexes and the varied nutritional and general health status. Temporal trends were depicted by linear regression analysis of logarithmic transformed mean concentrations using ANOVA tests for each analyte separately. Any measured sample reported lower as the MDL was calculated to be 0.5 of the MDL for statistical analysis. The year 1988 was not included in the ANOVA tests, because of the small sample number (n = 2). Doubling times were calculated with $t_{1/2} = \ln(2)/m$, where m represents the slope of the natural logarithm transformed liver concentration versus time.

6.3. Results and discussion

6.3.1. Contaminant concentrations and composition profiles

Concentrations of individual PFC in livers of harbor seals stranded along the German Bight in 1988, 1996 and 1999 to 2008 are shown in **Table 17**. In this study, 17 of 40 target analytes were found (i.e., C_4-C_{10} PFSAs, PFOSi, FOSA and C_8-C_{15} PFCAs). The geometric mean ∑PFSA concentrations were 1988 ng/g ww (207-3743 ng/g ww) and 907 ng/g ww (7.8-2451 ng/g ww) for <7 month old and ≥ 7 month old harbor seals, respectively. PFOS was the predominant compound with a maximum concentration of 3676 for <7 month old harbor seals (1996), making up on average 94% of the measured PFCs. The high mean concentrations of PFPS, PFOS and PFDS in 2003 and 2004 could be due to the small sample size in these years.

Table 17. Average concentrations (ranges) of PFCs in liver tissue of harbor seals from the German Bight in ng/g ww

Analyte	Age (month)	1988 (n=2/0) [a]	1996 (n=0/2) [a]	1999 (n=4/1) [a]	2000 (n=5/1) [a]	2001 (n=5/2) [a]	2002 (n=5/1) [a]	2003 (n=2/3) [a]	2004 (n=1/0)[a]	2005 (n=4/1) [a]	2006 (n=3/5) [a]	2007 (n=10/3) [a]	2008 (n=3/0) [a]
PFBS	≥ 7	0.1 (n.d.-0.3)	-	0.2 (n.d.-0.4)	0.4 (n.d.-1.9)	0.8 (n.d.-3.1)	n.d.	n.d.	0.5	0.3 (0.1-0.4)	0.2 (n.d.-0.5)	0.2 (0.1-0.4)	0.2 (0.2-0.3)
	<7	-	1.2 (0.8-1.6)	0.44	0.2	0.5 (0.3-0.7)	0.5	0.6 (0.2-1.2)	-	n.d.	0.6 (n.d.-1.4)	0.3 (0.3-0.4)	-
PFPS [b]	≥ 7	2.6 (0.9-4.2)	-	2.6 (2.1-3.7)	2.1 (1.0-3.4)	2.1 (0.7-6.2)	2.3 (1.3-3.4)	4.2 (2.0-6.4)	5.2	1.6 (1.0-2.4)	1.2 (0.6-2.2)	0.6 (n.d.-1.3)	0.5 (0.3-0.6)
	<7	-	2.3 (1.2-3.4)	3.8	1.8	4.1 (3.2-5.1)	5.0	2.5 (1.0-3.4)	-	2.7	0.4 (n.d.-1.1)	0.3 (n.d.-0.7)	-
PFHxS	≥ 7	13 (8.9-17)	-	10 (5.0-21)	11 (2.6-32)	11 (3.0-21)	10 (3.0-20)	6.9 (3.9-10)	9.8	5.8 (2.2-10)	2.4 (n.d.-4.7)	4.0 (1.3-7.9)	1.2 (0.6-1.7)
	<7	-	16 (13-19)	31	26	27 (19-35)	32	24 (21-29)	-	3.3	16 (2.7-33)	7.8 (1.4-17)	-
PFHpS	≥ 7	2.6 (1.7-3.4)	-	4.6 (1.3-9.5)	7.8 (1.9-22)	7.2 (1.7-16)	5.9 (1.2-13)	6.4 (3.7-9.1)	6.8	3.1 (1.0-5.1)	2.1 (n.d.-4.2)	3.8 (0.8-10)	0.2 (0.1-0.3)
	<7	-	47 (41-53)	28	49	16 (7.8-24)	13	25 (13-40)	-	3.8	5.4 (n.d.-23)	14 (1.3-39)	-
PFOS	≥ 7	1327 (1224-1429)	-	1111 (576-1470)	932 (292-2080)	1130 (313-2407)	902 (455-1396)	1436 (645-2227)	1632	785 (402-1023)	472 (7.2-806)	597 (277-951)	480 (367-577)
	<7	-	3520 (3363-3676)	3067	3662	2415 (1564-3265)	1143	2408 (1541-3451)	-	772	1198 (439-1983)	1077 (204-2723)	-
PFNS [b]	≥ 7	n.d.	-	0.3 (n.d.-0.5)	0.2 (n.d.-0.5)	0.6 (0.1-1.8)	0.7 (n.d.-1.5)	1.8 (0.8-2.8)	2.0	0.9 (0.5-1.2)	0.8 (n.d.-1.2)	1.4 (0.6-2.6)	0.1 (n.d.-0.2)
	<7	-	2.7 (1.0-4.4)	1.9	2.9	1.8 (0.8-2.8)	0.59	2.6 (1.3-4.4)	-	0.49	0.6 (n.d.-1.3)	2.0 (0.5-4.5)	-
PFDS	≥ 7	n.d.	-	0.1 (n.d.-0.3)	0.2 (n.d.-0.4)	0.2 (n.d.-0.8)	0.3 (n.d.-0.6)	0.5 (n.d.-1.1)	1.5	0.3 (n.d.-0.5)	0.3 (n.d.-0.5)	0.6 (n.d.-1.1)	0.1 (n.d.-0.2)
	<7	-	1.2 (1.0-1.4)	1.4	1.8	1.0 (0.4-1.6)	0.5	2.1 (1.3-3.0)	-	0.38	0.3 (0.1-0.9)	1.4 (n.d.-4.1)	-
PFOSi	≥ 7	n.d.	-	0.6 (0.1-1.7)	0.2 (n.d.-0.8)	0.1 (n.d.-0.3)	n.d.	0.2 (0.1-0.2)	n.d.	0.3 (0.2-0.4)	0.1 (0.1-0.2)	0.2 (n.d.-0.5)	0.04 (n.d.-0.1)
	<7	-	0.1 (n.d.-0.1)	0.5	0.4	0.6 (0.5-0.6)	0.1	0.4 (0.1-1.0)	-	0.18	0.1 (n.d.-0.2)	0.9 (0.2-2.3)	-
PFOA	≥ 7	1.0 (n.d.-2.1)	-	1.4 (n.d.-2.6)	2.4 (n.d.-8.4)	3.3 (n.d.-7.1)	0.6 (n.d.-1.8)	0.8 (n.d.-1.6)	n.d.	0.9 (0.3-1.2)	0.8 (n.d.-1.3)	1.0 (n.d.-1.7)	0.3 (n.d.-0.8)
	<7	-	3.9 (2.2-5.7)	n.d.	13	1.8 (1.1-2.4)	3.6	2.6 (1.5-4.6)	-	0.30	1.3 (n.d.-3.9)	2.0 (0.4-3.9)	-
PFNA	≥ 7	4.7 (4.1-5.2)	-	11 (5.2-23)	13 (2.5-27)	16 (1.0-27)	9.3 (3.2-17)	13 (11-15)	9.3	15 (7.8-18)	11 (0.3-19)	14 (9.2-21)	8.0 (6.2-9.7)
	<7	-	8.6 (4.8-12)	1.0	19	4.6 (3.9-5.3)	6.3	11 (1.6-19)	-	2.6	7.4 (0.9-17)	6.1 (5.1-8.1)	-
PFDA	≥ 7	4.2 (4.0-4.4)	-	9.4 (4.7-15)	14 (2.5-30)	12 (2.5-25)	17 (6.5-36)	16 (12-20)	13	14 (7.6-18)	14 (0.2-23)	18 (9.7-23)	8.0 (5.9-10)
	<7	-	4.5 (2.9-6.0)	3.4	14	4.4 (3.6-5.2)	4.4	10 (2.8-15)	-	1.8	7.0 (0.4-16)	11 (7.5-15)	-
PFUnDA	≥ 7	0.8 (0.5-1.2)	-	3.9 (2.2-5.4)	4.3 (1.0-8.3)	3.8 (1.3-7.9)	5.6 (1.5-12)	5.6 (4.0-7.3)	7.3	5.8 (3.7-7.2)	5.2 (0.7-7.5)	5.5 (3.8-7.0)	3.0 (2.5-3.8)
	<7	-	2.6 (2.3-3.0)	4.8	7.4	3.6 (3.2-3.9)	3.2	7.0 (3.8-11)	-	3.7	3.5 (3.0-4.5)	4.7 (2.7-7.4)	-
PFDoDA	≥ 7	n.d.	-	0.8 (0.6-1.1)	1.0 (0.3-2.0)	0.8 (0.3-1.7)	0.9 (n.d.-1.8)	0.9 (0.5-1.2)	2.3	1.7 (1.4-2.1)	1.1 (0.2-1.7)	1.2 (0.9-1.6)	0.9 (0.8-1.0)
	<7	-	1.4 (1.2-1.6)	2.7	2.9	1.9 (1.5-2.3)	1.1	2.4 (1.7-3.6)	-	1.1	1.8 (0.9-3.2)	2.6 (0.5-4.9)	-
PFTriDA	≥ 7	n.d.	-	1.3 (0.8-1.7)	1.4 (0.4-3.1)	1.3 (0.5-2.2)	1.5 (n.d.-2.3)	1.8 (1.5-2.1)	4.8	3.0 (2.4-3.6)	2.0 (1.4-2.6)	1.9 (1.5-2.7)	1.2 (1.0-1.4)
	<7	-	2.3 (2.1-1.6)	4.5	2.8	4.1 (3.7-4.5)	1.9	3.6 (1.0-6.0)	-	3.4	2.6 (1.2-5.9)	3.7 (1.1-6.1)	-
PFTeDA	≥ 7	n.d.	-	n.d.	n.d.	n.d.	0.1 (n.d.-0.2)	n.d.	n.d.	n.d.	n.d.	0.1 (n.d.-0.2)	n.d.
	<7	-	n.d.	0.56	0.39	0.5 (0.5-0.6)	n.d.	0.2 (n.d.-0.4)	-	0.24	0.2 (n.d.-0.4)	0.24 (n.d.-0.4)	-
PFPeDA [b]	≥ 7	n.d.	-	0.02 (n.d.-0.1)	n.d.	0.1 (n.d.-0.6)	0.04 (n.d.-0.2)	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
	<7	-	n.d.	0.4	0.2	0.8 (0.4-1.2)	0.13	n.d.	-	n.d.	0.1 (n.d.-0.4)	n.d.	-
FOSA	≥ 7	4.8 (3.3-6.3)	-	33 (3.4-81)	7.9 (2.5-20)	8.0 (1.3-16)	6.7 (n.d.-17)	8.2 (6.1-10)	6.1	6.2 (5.0-8.0)	7.0 (2.2-16)	2.3 (0.9-4.6)	0.9 (0.5-1.2)
	<7	-	16 (11-20)	202	7.9	45 (30-61)	9.1	41 (15-72)	-	11	3.9 (1.0-6.6)	7.0 (1.3-18)	-
∑PFCs	≥ 7	1361 (1264-1458)	-	1190 (614-536)	997 (319-2226)	1198 (332-2537)	962 (476-1501)	1502 (701-2303)	1701	844 (437-1090)	520 (13-873)	653 (325-1018)	504 (387-606)
	<7	-	3629 (3498-3761)	3353	3810	2533 (1649-3417)	1225	2542 (1669-3591)	-	807	1249 (474-2069)	1140 (237-2844)	-

[a] n.d. = not detected; n = number of ≥ 7 month old harbor seals/ <7 month old harbor seals; [b] To be considered as estimated, because no standard was available.

In general, the concentrations in all investigated years were much higher than measured in Arctic ringed seal samples. Hence, mean PFOS concentrations were by about a factor of 10 to 50 lower from the two locations (Grise Fjord and Holman) in the Canadian Arctic (1998 and 2001, respectively) and the two locations (Ittoqqortoormiit and Qeqertarsuaq) in Greenland (1986-2003 and 1982-2003, respectively) (Bossi et al. 2005a; Martin et al. 2004a). Van de Vijver et al. 2005 found mean PFOS concentrations of 175 ± 105 ng/g ww in liver of harbor seals from the Dutch Wadden Sea, which was still about a factor of 5 lower than in this study. However, in most studies PFOS was the dominating PFC in marine mammals (Houde et al. 2006c). FOSA was found in almost all measured samples in a range of 1.0 to 202 and 0.5 to 80.7 ng/g ww in <7 month old and ≥ 7 month old harbor seals, respectively. Lower concentrations were found for PFOSi with up to 2.3 and 1.7 ng/g ww in <7 month old and ≥ 7 month old harbor seals, respectively. The presence of FOSA and PFOSi at high concentrations may indicate incomplete biotransformation of EtFOSE to PFOS, since FOSA and PFOSi were proposed to be intermediates of this biotransformation pathway (Rhoads et

al. 2008). This is consistent with the high positive correlation of FOSA and PFOSi with each other ($p < 0.01$, see **Table S14** in the Supplementary material). The other PFSAs made only a small contribution to the ∑PFSAs (see **Figure 17**), but they were detected frequently in 55-100% of all analysed samples. The dominance of PFOS and the presence of the longer- and shorter-chained PFSA in biota tissue suggests that the source were POSF–derived products from electrochemical fluorination (ECF) manufacturing process, which contain homologous series of even- and odd-number PFSAs (Giesy and Kannan 2002).

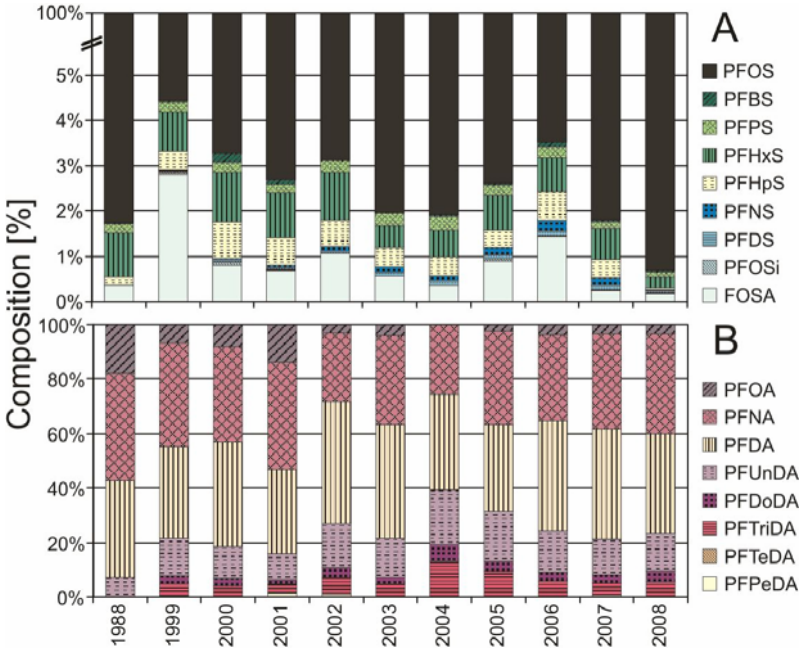

Figure 17. Composition profile of individual PFSAs, PFOSi and FOSA (A) and PFCAs (B) in habor seals (≥ 7 month old, n = 44)

In contrast to the PFSAs, only the longer-chained PFCAs (C_8-C_{15}) could be detected in harbor seals, which suggest a higher accumulation potential of PFSAs compared to PFCAs (Martin et al. 2003b). ∑PFCAs concentrations ranged between 13-58 ng/g ww and 2.4-79 ng/g ww for <7 month old and ≥ 7 month old harbor seals, respectively. PFNA, PFDA and PFUnDA dominated with an average contribution of 27%, 26% and 16% in <7 month old and 34%, 37% and 14% in ≥ 7 month old harbor seals to ∑PFCAs, respectively (see **Figure 17**). These results correspond with the observations of Smithwick et al. 2005a and Martin et al. 2004a in polar bears from the Arctic, whereas C_8-C_{15} PFCAs were detected with C_9-C_{11} PFCA as the dominant PFCAs. In the present study, concentrations of PFOA were relatively low (<0.087-13 and <0.087-8.4 ng/g ww in <7 month old and ≥ 7 month old harbor seals), which confirms the relatively low bioaccumulation potential of shorter-chained PFCAs (≤ C_8) (Martin et al. 2003a). On the other hand, with increasing PFCA chain length (≥ C_{11}) the

concentration decreased, with concentrations of PFTeDA and PFPeDA below of 1.3 ng/g ww. Biomagnification of PFCs were reported in the marine food web from Lake Ontario and in the Eastern Arctic (Martin et al. 2004b; Tomy et al. 2004a). Interestingly, in Arctic ringed seals the PFCA concentrations were about a factor of 2 lower (Butt et al. 2007b), while the PFOS concentrations were about a factor of 10-50 lower than in this study (see discussion above). These results suggest that PFOS is mainly found near source regions, against what the distribution of PFCAs are more uniform due to their long-range transportation via volatile precursors by the atmosphere and/or directly by the ocean currents.

Fluorinated telomer acids were suggested as atmospheric and/or microbial degradation products of fluorinated telomer alcohols (FTOHs), which could finally degrade to PFCAs (Ellis et al. 2004; Wang et al. 2005). In contrast to Arctic ringed seals (Butt et al. 2007b), FTCAs and FTUCA were not detected in any samples of this study. Therefore, these degradation pathways may play a minor role of the PFCAs contamination in harbor seals from the German Bight, which confirm the hypothesis that the main transport pathways in this urbanised areas is directly via the water phase (Prevedouros et al. 2006). However, the pathways of PFCs from production and/or products to the marine food web are not fully understood.

C_9-C_{13} PFCAs were significantly correlated with each other and with PFOS and PFNS (see in **Table S14** the Supplementary material). This corresponds with reported positive correlation between the C_6-C_{10} PFCAs in water samples (So et al. 2007). In addition, C_4 and C_6-C_8 PFSAs were significantly correlated with each other and also with the shorter-chained PFCAs (C_8-C_{10}). These results suggest that these PFCs had a common source as reported by Smithwick et al. 2005b. PFC concentrations in harbor seal livers collected in the same year showed a high mean standard deviation of 33% (PFTeDA) to 73% (PFHpS), which indicated influences from other variables such as differences in diet or different metabolisms may also play an important role. However, the composition profile is relatively constant over the time period (see **Figure 17**). In addition, no interannual variations in the relative compounds ratio were found in harbor seals, which is in agreement with the observation of an equal exposure of PFCs in bottlenose dolphins over the whole year (Houde et al. 2006a). Overall, the pattern and generally high PFC contamination suggest distinctive sources from high urbanised/industrialised regions at the German Bight.

6.3.2. Comparison of PFC concentrations in harbor seals with age, sex, location and health status

Significantly higher concentrations were found in <7 month old in comparison to ≥ 7 month old harbor seals for PFHxS ($p < 0.001$), PFHpS ($p < 0.001$), PFOS ($p < 0.01$), PFDoDA ($p < 0.01$) and FOSA ($p < 0.05$) (**Figure 18**), whereas PFDA showed significantly lower concentrations in <7 month old harbor seals ($p < 0.05$). This is consistent with the observation that higher concentrations of PFOS were found in juvenile harbor porpoises (Van de Vijver et al. 2003a). Conversely, significant increase in PFOS and PFCAs concentrations with age has been previously reported by Smithwick et al. 2005b for juvenile male polar bears from East Greenland. One explanation could be transplacental transfer of PFC contaminants to the foetus (Hart et al. 2008a). On the other hand, younger harbor seals have a different diet and they are going foraging close to the coast in comparison to the older harbor seals (Reijnders et al. 2005), so that they are possibly exposed to higher contaminated food. However, information on the influence of the age on the PFC concentrations in marine mammals is very sparse (Houde et al. 2006c; Kannan et al. 2002c).

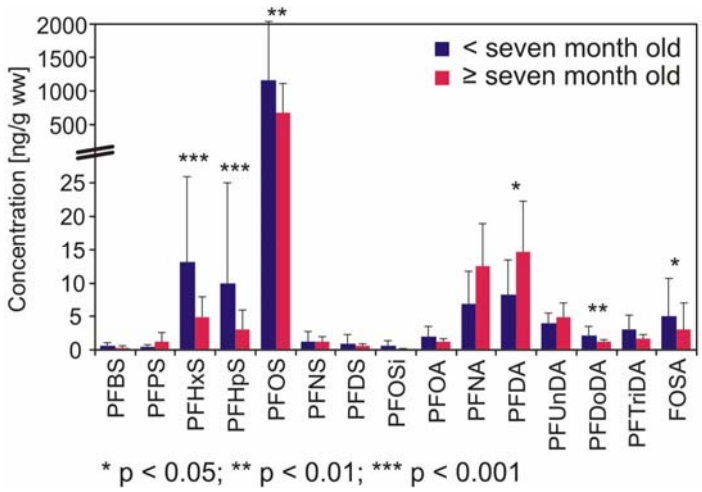

Figure 18. Comparison of individual PFCs in <7 month old (blue, n = 8) and ≥ 7 month old (red, n = 13) habor seals (*Phoca vitulina*) from 2006 and 2007

No significant correlation was identified between PFC concentrations and spatial distribution and sex, respectively for any analyte ($p > 0.05$). Mean concentrations of 'good' 'moderate' and 'poor' general health status (1204 ± 772, 969 ± 532 and 890 ± 505 ng/g ww, respectively) and 'good', 'moderate' and 'emaciated' nutritional status (992 ± 718, 940 ± 588 and 916 ± 366 ng/g ww, respectively) were not statistical significant different ($p > 0.05$; **Figure 19**). However, PFOS and PFOA have several possible adverse effects in biota, such as neuroendocrine effects (Austin et al. 2003) and peroxisome proliferation, have been shown to

occur (Oakes et al. 2004). Significantly positive correlations between high concentrations of PFOA and PFOS and infectious diseases in sea otters (*Enhydra lutris nereis*) were found from Kannan et al. 2006. In this study, the lack of significance could be caused by the low contamination level and the dependence on the species of the toxic effects. The lowest no-observed-adverse effect levels (NOAEL) of PFOS in the liver of rats (358 to 370 µg/g ww), and cynomolgus monkeys (*Macaca fasicularis*) (59 to 70 µg/g ww) were much higher than the concentration level measured in harbor seals from this study (Seacat et al. 2003; Seacat et al. 2002). However, physiological changes were observed in Baikal seals (*Pusa sibirica*) with similar concentrations of PFCA and lower concentrations of PFOS compared to this study (Ishibashi et al. 2008b).

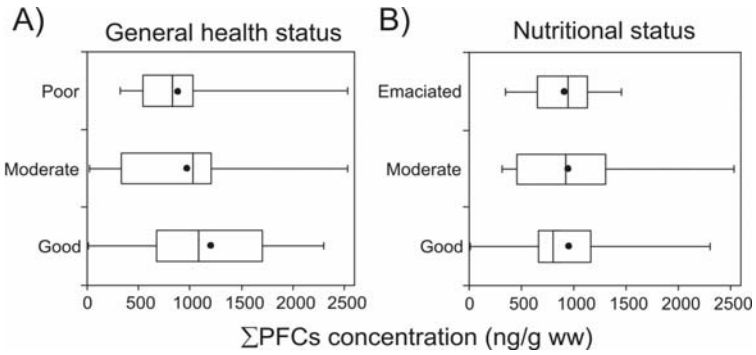

Figure 19. Box-whisker plots showing ∑PFCs concentration (ng/g ww) in livers of ≥ 7 month old harbor seals classified in (A) 'poor' (n = 22), 'moderate' (n = 8) and 'good' (n = 9) general health status and (B) 'emaciated' (n = 8), 'moderate' (n = 23) and 'good' (n = 13) nutritional status. Mean concentrations are indicated as a black circle, while the boxes show 25% and 75% percentiles and medians

6.3.3. Temporal Trends of PFSAs, PFOSi and FOSA

Harbor seal liver samples from the German Bight showed significantly decreasing temporal trends of C_5-C_7 PFSAs, PFOSi and FOSA between 1999 and 2008 (**Figure 20** and **Figure S6** in the Supplementary material). Overall, the C_5-C_7 PFSAs have decreased by 58% for PFPS, 80% for PFHxS and 79% for PFHpS between 1999 and 2008, conversely, concentrations of PFDS increased by 47%. However, the increasing trend of PFDS is not significant ($p = 0.070$). PFOS decreased by 49% between 1999 and 2008 ($p = 0.067$), which corresponds with decreasing concentration levels of its metabolic precursors PFOSi and FOSA of 83% and 95% in the same time period. However, the results of PFOSi must be considered carefully, because this compound can be decomposed very fast in the presence of oxygen to PFSAs and PFCAs. PFBS and PFNS concentrations were detected in 55% and 76% of the samples, respectively, but no significant temporal trend could observed ($p = 0.185$ and

p = 0.561, respectively). But if the year 1999 was excluded in the ANOVA tests, the PFBS concentration decreased statistically significant by 59% between 2000 and 2008 (p = 0.017, r^2 = 0.762) (**Figure 20**). The decreasing concentration of PFBS is in contradiction to the conversion of the production from POSF to the shorter-chained PFBS after the production termination of POSF in 2000 (Prevedouros et al. 2006). An explanation for this declining trend could be that the PFBS contamination, detected in this area, is the result of the POSF production, which yields a product mixture of linear and up to 30% branched isomers (Giesy and Kannan 2002). This possibility is supported by similar decreases in PFPS, PFHxS and PFHpS.

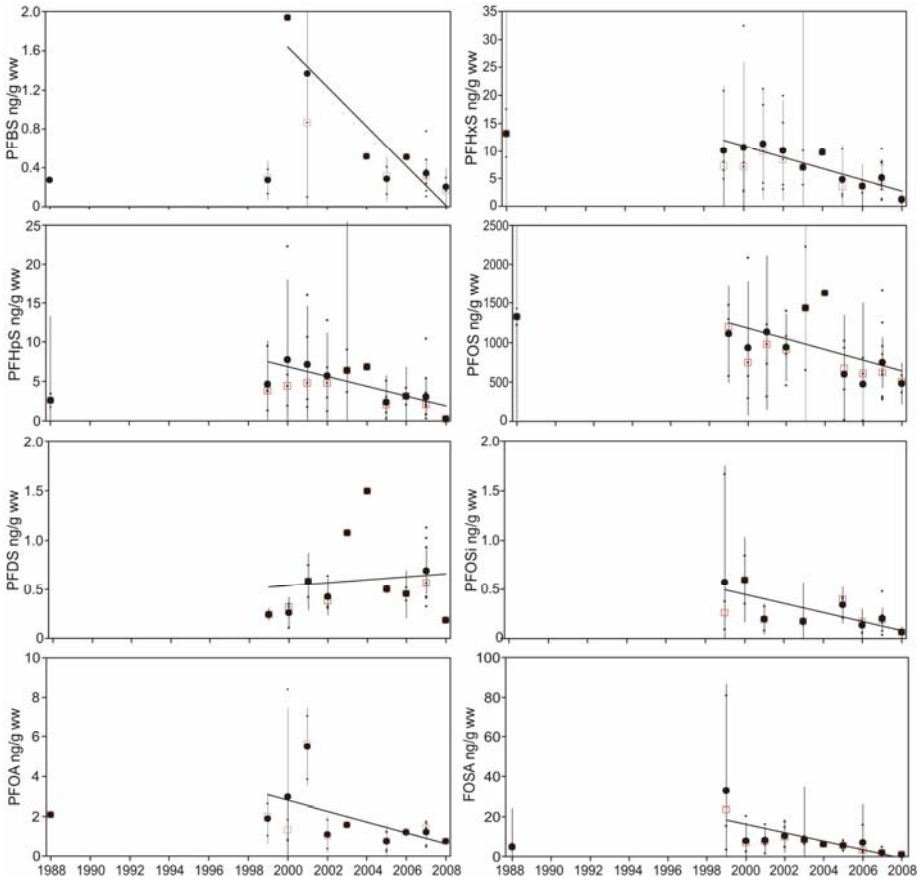

Figure 20. Temporal trends of eight PFCs in habor seals from the German Bight from 1999 to 2008. The plots display the geometric means (circles) and the median (squares) together with the individual analysis (small dots) and the 95% confidence intervals of the geometric means (≥ 7 month old, n = 44). Note: The linear regression of PFBS was calculated from 2000 to 2008

Opposite to other reports (Butt et al. 2007b; Smithwick et al. 2006) the concentrations of PFPS, PFHxS, PFHpS and PFOSi showed a statistically significant decrease in this study resulting in half-lives of 4.2 ± 0.9, 4.0 ± 0.9, 2.9 ± 0.5 and 4.7 ± 3.6 years, respectively (**Table 18**). The decreasing temporal trend of FOSA were confirmed in other reports for

example in melon-headed whales (2001/2002 to 2006), lake trouts (1993-2004) and Arctic ringed seals (1998-2005) (Butt et al. 2007b; Furdui et al. 2008; Hart et al. 2008a). The half-life (1999-2008) for FOSA was calculated here with 2.8 ± 0.9 years, which is comparable with the half-life (1998 to 2005) in Arctic ringed seals from Arviat (2.0 ± 0.6 years) (Butt et al. 2007b), but lower than reported in polar bears from eastern and western sampling sites in the North American Arctic (10.5 ± 7.5 and 13.8 ± 17.0 years for 1972 to 2002, respectively) (Smithwick et al. 2006). PFOS had a half-life of 5.6 ± 18.9 years, the uncertainty was higher than for the other calculated half-lives in this study, because of the lower significance ($p = 0.067$). However, most studies found a increasing temporal trend of PFOS like in polar bear and ringed seal livers from the Arctic (Bossi et al. 2005a; Dietz et al. 2008; Smithwick et al. 2006), whereas Smithwick et al. 2006 calculated half-lives of 9.8 ± 5.1 and 13.1 ± 4.0 years, respectively. Holmström et al. 2005 investigated the temporal trend of PFOS and PFOA in guillemot eggs from the Baltic Sea from 1968-2003. They found an increasing PFOS concentration, but after 2002 the concentration declined. Furthermore, Butt et al. 2007b were the first who found a statistically significant decreasing trend of PFOS in Arctic ringed seals with a half-life of 3.2 ± 0.9 years (Arviat, 1998-2005) and 4.6 ± 9.2 years (Resolute Bay, 2000-2005), respectively (see **Figure 21**).

Table 18. Doubling times, half-lives (years ± 95% confidence interval) and statistical parameters based on the linear regressions of PFCs in liver tissue of harbor seals (*Phoca vitulina*) from the German Bight, 1999-2008 (≥ 7 month old, n = 44)

Analyte	**r^2**	**P**	***Doubling times*/ half-lives (yr)**
Perfluorobutane sulfonate (PFBS) [b]	0.263	0.185	- [a]
Perfluoropentane sulfonate (PFPS)	0.425	<0.001	4.2 ± 0.9
Perfluorohexane sulfonate (PFHxS)	0.616	<0.001	4.0 ± 0.9
Perfluoroheptane sulfonate (PFHpS)	0.472	<0.001	2.9 ± 0.5
Perfluorooctane sulfonate (PFOS)	0.280	0.067	5.6 ± 18.9
Perfluorononane sulfonate (PFNS)	0.008	0.561	- [a]
Perfluorodecane sulfonate (PFDS) [c]	*0.014*	*0.070*	*26.2 ± 2.4*
Perfluorooctane sulfinate (PFOSi)	0.546	0.028	4.7 ± 3.6
Perfluorooctanoic acid (PFOA)	0.367	0.031	5.6 ± 4.2
Perfluorononanoic acid (PFNA)	0.063	0.993	- [a]
Perfluorodecanoic acid (PFDA)	0.069	0.912	- [a]
Perfluoroundecanoic acid (PFUnDA)	0.007	0.571	- [a]
Perfluorododecanoic acid (PFDoDA)	0.055	0.255	- [a]
Perfluorotridecanoic acid (PFTriDA)	0.066	0.189	- [a]
Perfluorooctane sulfonamide (FOSA)	0.710	<0.001	2.8 ± 0.9

[a] Not determined, because of insignificant slope; [b] if the linear regressions was carried out from 2000 to 2008, the PFBS concentration decreased statistically significant ($r^2 = 0.762$, $p = 0.017$); [c] value for PFDS in italics indicating doubling time per year.

It is noteworthy, that PFDS is the only compound in this study whose concentration increases resulting in a doubling time of 26.2 ± 2.4 years. This increase could possibly be explained by increasing usage of PFDS for example in photographic materials or for electrolysis (Kissa 2001). These applications are also explicitly accepted in the EU directive for the restriction of PFOS (European Parliament and Council 2006). However, the general decreasing PFC concentrations could be caused by the phase-out of POSF by the 3M Company, reduction of PFC emissions by optimization of the production process and/or production shift to shorter-chained PFCs, which are less bioaccumulative (Prevedouros et al. 2006). Fast depuration rates of PFCs could be responsible for the short half-lives of C_5-C_8 PFSAs, PFOSi and FOSA (2.8 to 5.6 years) in harbor seals after the reduction of PFC emissions (Butt et al. 2007b). However, the body burden of individual PFCs in harbor seals should react different to environmental changes depending on the depuration rate.

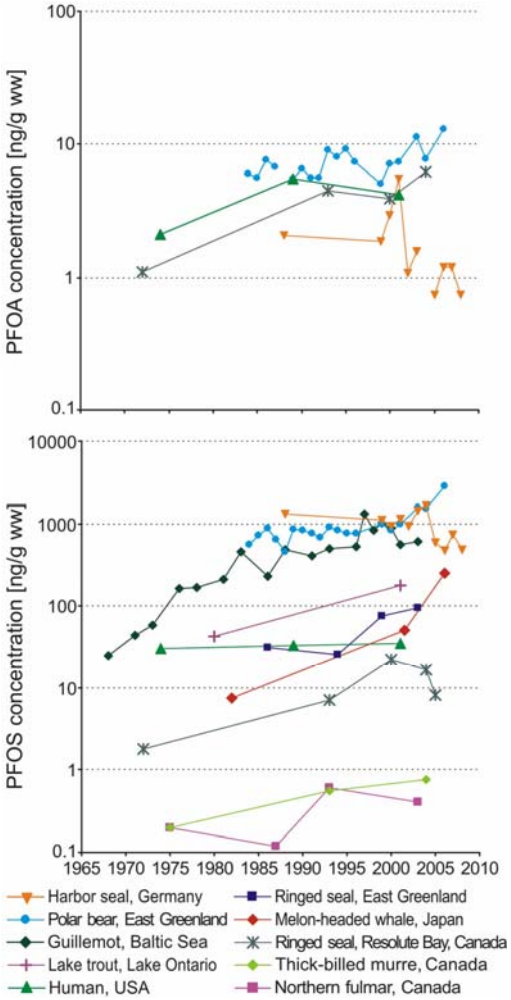

Figure 21. Temporal trends of PFOA and PFOS in harbor seal livers (this study), polar bear livers (Dietz et al. 2008), guillemot eggs (Holmström et al. 2005), lake trout homogenates (Martin et al. 2004b), melon-headed whale livers (Hart et al. 2008a), human serum (Olsen et al. 2005), thick-billed murre and northern fulmar livers (Butt et al. 2007a), and ringed seal livers from East Greenland (Bossi et al. 2005a) and Resolute Bay, Canada (Butt et al. 2007b)

6.3.4. Temporal Trends of PFCAs

Statistically significant temporal trends of PFCAs could only be found for PFOA ($p = 0.031$, **Figure 20**), representing a decrease of 86% between 1999 and 2008. C_9-C_{13} PFCA concentrations were detected in 95% to 100% of the samples, but no significant temporal trend could be observed ($p = 0.188$ to $p = 0.993$). PFTeDA and PFPDA were found in 17 and 7% of the samples, respectively and were, therefore, not used for the statistical temporal trend analysis. The half-life of PFOA was 5.6 ± 4.2 years (**Table 18**), conversely, other studies found increasing PFOA concentrations (Dietz et al. 2008; Smithwick et al. 2006) (see **Figure 21**), whereas Smithwick et al. 2006 calculated doubling times of 7.3 ± 2.8 and 13.9 ± 14.2 years in polar bears from east and west North American Arctic, respectively. The phase-out of POSF-based products in 2001 and the reduction of global PFOA emissions for the period 1999-2008 could be responsible for the clearly decreasing temporal trend of PFOA, but PFOA is still produced by other manufactures and used in fluoropolymer production (Prevedouros et al. 2006). Furthermore, the elimination rate of PFOA in organisms by urinary or faecal excretion, and possibly by milk by females is faster than, for example, PFOS, which could lead to a fast elimination after the uptake and low concentration level in the liver of the animals (Hart et al. 2008a; Olsen et al. 2007).

6.4. Conclusion

Previous temporal trend studies demonstrated decreasing concentrations of PFOS and FOSA (Butt et al. 2007b; Hart et al. 2008a), this study shows for the first time significant decreasing concentrations of C_5-C_7 PFSAs, PFOSi and PFOA in marine mammals during the past 10 years. The reason could be, probably, that this is the effect of the phase-out of POSF by 3M Company, the reduction of PFOA emissions by a stewardship program from the U.S. EPA and replacement of PFOS and their derivates by less bioaccumulative compounds because of the formed directive from the EU (Prevedouros et al. 2006; European Parliament and Council 2006). But the observations of increasing PFDS levels and the still high concentrations of PFOS in 2008 and constant levels of C_9-C_{13} PFCAs indicates that further work on the reduction of sources of PFCs are necessary. Ultimately, these high PFCs body burden could possibly have an effect on the immune system and physiological functions of the animals (Guruge et al. 2006; Ishibashi et al. 2008b; Yang et al. 2002).

The intake of water by harbor seals is being done completely via the food, hence the water could not be directly responsible for the contamination of the harbor seals with PFCs. However, the harbor seals were collected in an urbanised/industrialised area where PFCs could be released directly (e.g., manufacture and consumer products) or indirectly (e.g. PFC

precursors) into the aqueous environment and can be bioaccumulated in the marine food web. Other reports found increasing concentrations in polar bears and Arctic ringed seals from Greenland, which were explained by the degradation of neutral precursor compounds to ionic PFCs (Bossi et al. 2005a; Dietz et al. 2008). Hereby, the composition profile and temporal trends for PFCs in the German Bight could reflect the regional usage pattern of different PFC products. This study highlights the importance of further monitoring studies in seals and other marine mammals from highly populated sites in order to evaluate association between PFCs and pathological conditions, and changes in pattern and long-term perspective trends.

Acknowledgements

We kindly acknowledge the German Federal Environmental Foundation for sponsoring the project. The pathological investigations on the seals were partly funded by the Ministry of Agriculture, Environment and Rural Development of Schleswig-Holstein, Germany.

7. Additional studies

Several studies were performed besides the main issue of the Ph.D. work, which are described in the following. Firstly, water samples were collected along the river Elbe into the North Sea to examine the distribution of PFCs in the dissolved and particulate phase and their discharge into the North Sea (**7.1**) (Ahrens et al. 2009c). In addition, a second cruise along the river Elbe was carried out in the river Elbe to investigate the influence of WWTP effluents to the riverine mass flow (**7.2**) (Ahrens et al. 2009f). Furthermore, surface water samples were collected in the North Sea, Baltic Sea and Norwegian Sea, where the occurrence and spatial distribution between river estuaries, coastal water, in brackish as well as salt water, and open sea water was compared (**7.3**) (Ahrens et al. submitted). Finally, within the frame of a research stay at the National Institute of Advanced Industrial Science and Technology (AIST) in Japan, the partitioning behaviour of PFCs between pore water and sediment in two sediment cores from Tokyo Bay, Japan was investigated (**7.4.**) (Ahrens et al. 2009g).

7.1 PFCs in water and suspended particulate matter in the river Elbe and North Sea

Surface water samples were taken at 24 locations from the river Elbe and the North Sea (Germany) in August 2006 (**Figure 22**). Details of the sampling and the physicochemical parameters of the water samples are presented in **Table S15** in the Supplementary material.

Figure 22. Map showing the sampling locations in the river Elbe and the North Sea (a dam is located between sampling stations 21 and 22)

One to two litre of water samples were obtained via a metal ship inlet system at 1-m water depth into brown glass bottles. A depth profile was taken at sampling station 23 at 0.5, 1.0,

2.0, 2.5, and 2.8-m water depth for the estimation of the flux from the river Elbe into the North Sea. The samples were filtered using GFFs (> 1.2 µm) on the same or following day. The sample analysis was performed as described in **publication II**.

Concentration of PFCs in the particulate and dissolved phase

Overall 20 of the 40 examined analytes were found at the 24 sampling locations. The PFCs quantified included C_4-C_8 PFSAs, 6:2 FTS, C_6 and C_8 PFSiAs, C_4-C_{12} PFCAs, 3,7m$_2$-PFOA, FOSA, and EtFOSE. The uneven carbon chained analytes PFPS and PFHpS may be by-products of the POSF-based and PBSF-based production, respectively. 3,7m$_2$-PFOA could be a by-product of the production of PFCAs, while EtFOSE is a precursor of PFSAs.

In the particulate phase 10 PFCs were detected, however, only PFOSi and FOSA were found in all samples. PFOS was found in three-quarters of the samples and the occurrence of C_8 to C_{11} PFCAs decreased from 63% to 21% in the samples taken. A maximal ∑PFC concentration was observed with 6.0 ng/L (location 23). The average ∑PFC concentrations decreased by a factor of 3.6 and 16 from Hamburg towards the Elbe Estuary and the North Sea, respectively. The large decrease in ∑PFC concentration towards the North Sea could be a result of sedimentation processes and/or dilution with sea-borne particles in the estuary. However, the concentrations in the particulate phase of the single compounds were usually lower than 1 ng/L except FOSA and PFOS with a few ng/L, while towards the North Sea the concentrations decreased below the MQL.

In the dissolved phase the compounds, PFOS, FOSA, and C_5 to C_9 of PFCAs were detected in all water samples. In contrast to the particulate phase, the occurrence of PFCs in the dissolved phase decreased with increasing chain length. A maximal ∑PFC concentration of 50.7 ng/L was observed at location 16 in Hamburg City. This indicates the major influence of the urban area at Hamburg City as a source of PFC contamination of the river Elbe. The average ∑PFC concentration dropped by a factor of 3 towards the North Sea. Just PFOS and both short-chained PFBA and PFBS showed a different behaviour along the river Elbe towards the North Sea. PFOS had a maximum concentration of 7.5 ng/L in Hamburg, and in addition a second maximum in the North Sea of 4.2 ng/L. Over the entire course down to the North Sea the concentration of PFBS and PFBA was relatively constant at 1.3 and 2.3 ng/L, respectively, but towards the North Sea (locations 1 to 4) the concentration increased to 2.5 ng/L and 3.0 ng/L, respectively.

Investigations by Caliebe et al. 2004 determined mean concentrations of PFCs in the river Elbe in 2003 of about 20 ng/L for PFOA and PFOS, and 1 ng/L to 3 ng/L for other PFCs like PFHxA, PFNA, PFDA, PFHxS, and FOSA. The concentrations of PFOA and PFOS in 2003

were twice as high as in this study from 2006, which may be a result of the phased out of the production of POSF from 3M.

Particulate related PFC fraction ($\varphi = [PFC]_{SPM}/([PFC]_{dissolved}+[PFC]_{SPM}) \times 100$) was calculated from the concentrations in the dissolved and particulate phase, which varied from compound to compound and depended on the location from where the samples were taken. The PFCs were mostly distributed in the dissolved phase, only EtFOSE was exclusively found in the particulate phase. PFOSi and FOSA were detected in every particulate phase with a proportion of 7% to 100% and 9% to 45%, respectively. The mean percentages of the particulate associated fraction were also relatively high for PFOS (14%), PFUnDA (12%), and PFDA (10%). PFSAs and longer-chained PFCAs, and perfluorinated sulfonamides and sulfonamido ethanols were more associated to particles than the shorter-chained PFCAs. This indicates that these compounds could rather settle down and accumulate in the sediment depending on their solid-water distribution coefficients (Higgins and Luthy 2006). PFCs which exist predominantly in the dissolved phase such as the shorter-chained PFCAs, will be rapidly dispersed in the aquatic environment and can be transported over long distances (Yamashita et al. 2005).

Total PFC flux from the river Elbe into the North Sea

The riverine discharge calculations are based on data from the ARGE Elbe (personal communication), a consortium of German states for the prevention of pollution in the river Elbe. At the sampling day riverine discharge showed only a 7% higher amount compared to the mean annual riverine discharge. Because of this good agreement, or similarity, the total estimated flux per year was calculated with the riverine discharge of 736 m^3/s for the sampling day, which represents a riverine discharge of 2.32E+10 m^3 water per year. For this estimation the mean concentration of the water samples at 0.5, 1.0, 2.0, 2.5, and 2.8-m depth at location 23 was used, which is a rough estimation assuming a constant load over the whole year. The depth profile sampling was carried out at location 23 because this sampling point was behind the water dam at Geesthacht and was not influenced by the tides. The total estimated flux per year for the dissolved and the particulate phase is presented in **Table 19**. In the particulate phase the total riverine PFC flux was 152 kg/year, where the greatest proportions were observed for FOSA, PFOS, PFOSi, and PFOA with 63 kg, 35 kg, 27 kg, and 10 kg per year, respectively. A much higher riverine PFC flux was estimated for the dissolved phase with a total flux of 802 kg/year. The calculation of the flux of FOSA may be too high, because the downstream concentrations were lower and it can be degraded to PFOS.

Table 19. Total estimated flux of individual PFCs in the dissolved

and particulate phases in the river Elbe towards the North Sea [a]

Analytes	Total flux/(kg/year)	
	Dissolved phase	Particulate phase
PFBS	18	0
PFPS[b]	21	0
PFHxS	8	0
PFOS	106	35
PFOSi	13	27
PFBA	35	0
PFPA	50	0
PFHxA	88	0
PFHpA	54	0
PFOA	169	10
PFNA	36	3
PFDA	66	5
PFUnDA	0	3
PFDoDA	0	0
3,7m$_2$-PFOA	0	0
FOSA	139	63
EtFOSE	0	6
∑PFCs	802	152

[a] The mean concentration of the five water samples collected at 0.5-, 1.0-, 2.0-, 2.5-, and 2.8-m depth at location 23 (for details see text) was used for the calculation; [b] have to be considered as estimates, because no standard was available for this compound.

Conclusion

In summary, this is the first study to determine PFCs in the particulate phase in surface water. In addition, PFPS, PFHpS, PFHxSi and 3,7m$_2$-PFOA were determined for the first time in the dissolved phase in surface water. The PFCs were mostly distributed in the dissolved phase (~93%), only EtFOSE was exclusively found in the particulate phase. Particles are subject to sedimentation and can therefore be important for bioavailability to benthic organisms. Further investigations are necessary to clarify if this leads to adverse effects such as a reduction of biodiversity. The total riverine PFC flux was 802 kg/year for the dissolved phase and 152 kg/year for the particulate phase. Discharge from the river Elbe contributed to a contamination of the North Sea with PFCs. This study found that PFBS and PFBA had other unknown sources in addition to the river Elbe, making research on the short-chained PFCs even more important. Further studies should therefore include a separate analysis of the dissolved and particulate phases. The particle mass and its content of organic matter should be determined to obtain a better understanding of the exchange processes between the dissolved and particulate phases, and to calculate partition coefficients between them.

7.2 PFCs in effluents of waste water treatment plants and surface water along the river Elbe

In June 2007 one litre water samples were taken midstream at 15 locations of the river Elbe (1 to 15 in **Figure 23**) on board of the research vessel *Ludwig Prandtl* using the water intake system of the ship (~1 m depth). Water samples were filtered subsequently (> 1.2 µm, GFFs) on board (detailed information see **Table S16** in the in the Supplementary material).

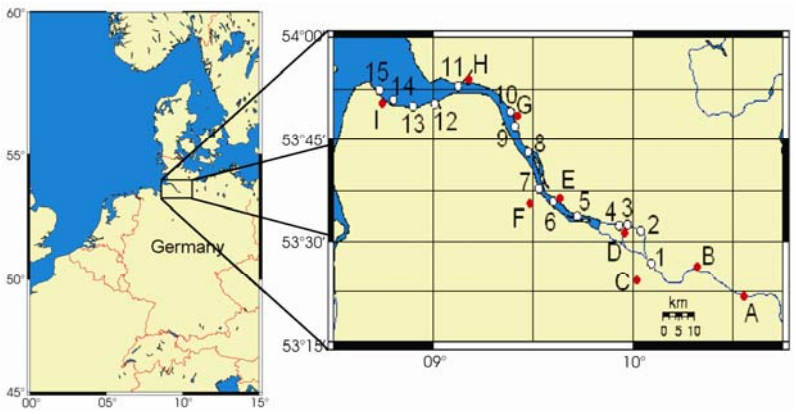

Figure 23. Sampling locations for surface water samples (white dots with numbers) and at the effluents of waste water treatment plants (red dots with letters) along the river Elbe, Germany

Effluent samples from 9 WWTPs along of the river Elbe from Lauenburg to Cuxhaven, Germany, were taken in May 2007 (A to I in **Figure 23**). The major WWTP (D) is treating waste water of about 2.2 million population equivalents (PEs), while the smallest one covers about 14,000 PEs (detailed information see **Table 20**).

Table 20. Sampling parameters of the waste water treatment plants

	A	B	C	D	E	F	G	H	I
Population equivalents	25 000	45 000	90 000	2 200 000	650 000	150 000	14 000	20 000	400 000
Effluent volume [m³/day]	1 900	6 000	17 000	432 000	72 000	13 200	1 285	1 000	18 000
Domestic waste water [%]	~60-70%	~60-70%	~100%	~80%	~65-70%	~100%	~100%	~100%	~100%
Industrial/commercial waste water [%]	~30-40%	~30-40%	~0%	~20%	~30-35%	~0%	~0%	~0%	~0%
Dissolved organic carbon [mg/L]	10.9	7.1	8.1	13.7	14.1	17.3	14.5	14.4	14.8
pH	7.0	7.3	6.9	7.4	7.0	7.4	8.0	7.1	7.7
Temperature [°C]	20.6	19.3	19.0	n.a.	18.9	20.1	15.8	16.6	24.7
Sampling date	20/06/07	29/05/07	31/05/07	31/05/07	29/05/07	07/06/07	29/05/07	29/05/07	07/06/07

n.a. = not available.

One litre water samples in duplicate were taken in glass bottles, filtered over 1.2 µm GFFs before the analysis. The sample analysis is described in **publication II**.

PFC concentrations and compositions in effluents of WWTPs

Overall, 21 substances of 39 analysed compounds were detected in WWTP effluents. The ∑PFC discharge of each WWTP and their composition profiles are shown in **Figure 24**. The ∑PFC concentrations in WWTP effluents ranged from 30 ng/L (WWTP I) to 266 ng/L (WWTP B). The composition profile in the WWTP B to D and F to I were relatively similar, whereas PFCAs were the major group with approximately 70% of the ∑PFC amount. WWTP A and E had a different composition, where 6:2 FTS and PFOS dominated, respectively. In 6 of the 9 WWTPs the concentrations of the PFSAs were 2-7 times lower than the concentrations of the PFCAs, in WWTP B they were even lower by a factor of 25. Conversely, in WWTP A and E the concentrations of PFSAs were about two times higher than the concentrations of PFCAs. Three potential precursors, FOSA, MeFBSA and MeFBSE, were found in all WWTPs, whereas FOSA can degrade to PFOS (Rhoads et al. 2008) and the C_4 compounds MeFBSA and MeFBSE could possibly degrade to the C_4 homologues of PFCAs and PFSAs (D'eon et al. 2006; Martin et al. 2006).

WWTP C, F, G, H and I were not influenced by industrial sewage water, on the other hand WWTP A, B, D and E have rather high industrial influences with about 20% to 40% industrial/commercial waste water. In average, ∑PFC concentrations of these four WWTP effluents were more than 4 times higher (171 ng/L) than the effluents of WWTPs without industrial waste water parts (36 ng/L). Thus it is presumed that industrial or commercial waste water had an influence on the PFC contamination level and profile, according to a previous study (Sinclair and Kannan 2006). The waste water of a big textile-service company, located close to WWTP A, might be responsible for the high concentrations of 6:2 FTS and MeFBSA in the effluent. Whereas a carpet factory located close to WWTP B might be responsible for the increased PFCA concentrations, because PFCs are used as coatings on textiles and carpets (Kissa 2001). The mean ∑PFC concentration in WWTP effluents (~99 ng/L) were about 5 times higher, compared to the mean concentrations of river water samples (~19 ng/L). Therefore, WWTPs are potential sources of PFCs to the marine environment.

PFCs discharges from the WWTPs into the river Elbe were roughly estimated from the PFC concentration and mean water discharge per day of the effluent and river flow, respectively (PFCs discharge = PFC concentration * water discharge) (see **Figure 24**). Although the effluent of WWTP B had the highest PFC concentration per litre, its contribution to the environment was rather low (1.6 g/day) due to its low effluent volume. WWTP D and E had the highest portion of PFC contamination of the river Elbe with 55.6 and 11.6 g/day, respectively. The remaining 6 WWTPs had a combined discharge of 3.8 g/day into the river Elbe. The total discharges per year were estimated assuming that the

concentrations were stable throughout the whole year. The samples were taken in a dry period with almost no precipitation, so the samples were not influenced by rain water. The total discharge of ∑PFCs from all nine WWTPs was approximately 26 kg per year based on a rough estimation, PFOA having the highest percentage (~47%), followed by 6:2 FTS (~20%), PFOS (~12%) and PFBS (~5.5%). WWTP D and E were responsible for approximately 95% of the total discharge.

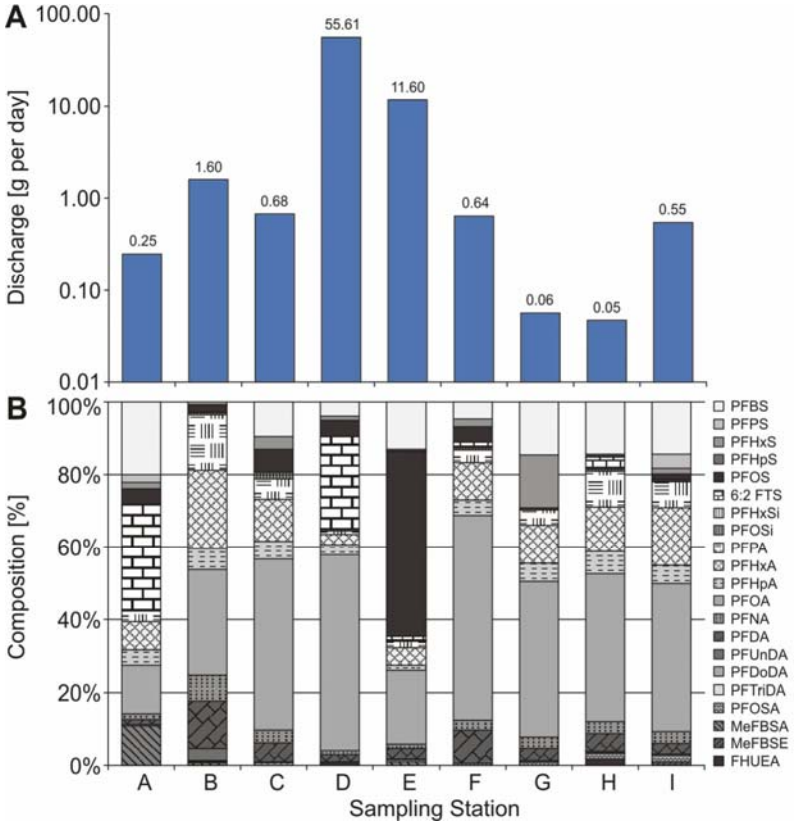

Figure 24. A: Discharge of ∑PFCs in g per day by the waste water treatment plant effluents (note: Logarithmic scale). B: Percentage composition of individual PFCs in the waste water treatment plant effluents

PFC concentrations and compositions in surface water of the river Elbe

The water samples of the river Elbe showed ∑PFC concentrations ranging from 7.6 ng/L at the estuary mouth (location 15) to 26.4 ng/L at Hamburg City (location 4). At all 15 sampling locations 17 PFCs were found, ten of them at each location (i.e., PFBS, PFHxS and PFOS of the PFSAs, C_5 to C_{10} of the PFCAs and FOSA). The PFCAs were the major group with approximately 70% of the ∑PFC amount. In all samples PFOA was the predominant substance with concentrations ranging from 2.8 ng/L (location 15) to 9.6 ng/L (location 6).

In 2003, Caliebe et al. 2004 found PFOS and PFOA concentrations of about 20 ng/L in the

river Elbe. In addition, they noticed a decrease of PFOS concentrations by approximately a factor of 10 from May to July. In 2006, a sampling campaign along the river Elbe resulted in two times higher PFC concentrations than during this sampling campaign in 2007 (Ahrens et al. 2009c). In 2006, especially PFOS and FOSA were detected in significantly higher concentrations (1.3 to 8.3 ng/L and 0.9 to 8.9 ng/L, respectively) than in 2007 (0.5 to 2.9 and 0.1 to 1.0 ng/L, respectively). The sampling campaign in the present study was done during a dry weather period. The river Elbe estuary stream flow in 2007 (474 m^3/s) was only the half of the mean flow in 2006. One possible explanation for the higher concentration levels in 2006 could be a runoff of PFCs from contaminated soil, as shown for the river Möhne and the river Ruhr, where PFCs were washed out by precipitation from contaminated soils applied to agricultural areas (Skutlarek et al. 2006). However, no contaminated soil has been observed along the river Elbe up to now. A second explanation could be that rain water was contaminated with PFCs and led directly to an increase PFC concentration in the river Elbe. Scott et al. 2006a found a total PFCA fluxes between 540 and 12471 ng/m^2 in precipitation, which supported the source there being the precipitation pathway. This could explain the higher concentrations of PFCAs in 2006 because the samples were taken during a wet weather period with an estuary stream flow of 916 m^3/s. The lower concentration levels of PFOS and FOSA cannot be explained by the higher amount of rain water, because PFCAs were the dominating PFCs in precipitation (Taniyasu et al. 2008). The most likely and straightforward explanation for the low PFOS and FOSA concentrations in 2007 could be the impact of the new EU directive, which regulates the general use of PFOS and their derivates like FOSA. Even though the use was not prohibited before July 2008, it might be possible that companies stopped using PFOS or switched to substitute compounds like the shorter-chained PFBS or their precursors already before the directive came into effect (D'eon et al. 2006). The reasons for using shorter-chained substitutes are their lower toxicity and accumulation potential compared to longer-chained PFCs (Lau et al. 2007).

Conclusion

No significant relationship between ∑PFC concentrations and DOC in WWTPs and in surface water was found, respectively. A positive relationship between sorption to sediment and TOC amount was found from Higgins and Luthy 2006, whereas the DOC may have a less influence on the distribution and concentration level of PFCs in the aquatic environment. The mass flow of ∑PFC from the effluents of the WWTPs and the surface water along the river Elbe is shown in **Figure 25**. Upstream Hamburg City (location 1) the ∑PFC mass flow was 729 g/day, whereas the mass flow increased to 964 g/day (location 4). This increase could be caused by WWTPs C and D with a combined discharge of 56.3 g/day, which is about 24% of

the total increase (235 g/day). The lacking 76% could originate from other point sources like industrial waste water effluents or diffuse sources from surface runoff, which were not covered in this study. An increasing ∑PFC mass flow was also observed between locations 5 and 6, whereas ~10% could stem from WWTP E with a discharge of 11.6 g/day. The other WWTPs showed no influence on the mass flow along the river Elbe. However, the PFC composition profile in surface water in the river Elbe is relatively similar to the compositions of WWTP B to D and F to I. These results suggest that the PFC contamination in these WWTPs and the river Elbe were caused by similar sources, whereas WWTP A and E were influenced by other sources.

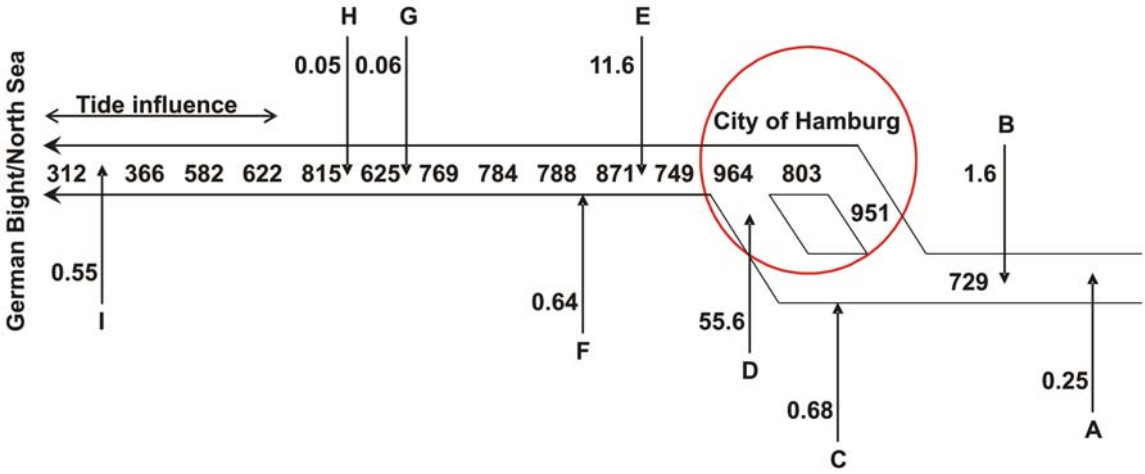

Figure 25. Estimated mass flow of ∑PFCs in g per day in surface water and waste water treatment plant effluents (A to I) along the river Elbe, Germany

Assuming that the concentrations found in this study were constant throughout the whole year and assuming that the concentrations measured at the surface were representative over the whole profile of the river, a rough estimation of the PFC mass flow reaching the German Bight through the river Elbe could be done. The long term mean water discharges of the river Elbe at the locations 1 and 11 were 752 and 812 m^3/s, respectively (personal communication ARGE Elbe). Using these data the calculation of the annual ∑PFC discharge results in 480 kg/year (location 1) to 540 kg/year (location 11). In further studies chemical substitutes of PFOS and PFOA should be investigated to see any production shifts to shorter-chained or less fluorinated PFCs. In addition to WWTPs, further sources are likely to exist which have not been identified yet. How to reduce the riverine discharge of PFCs into the marine ecosystem will be an important challenge for the future.

7.3 Sources of PFCs in the North Sea, Baltic Sea and Norwegian Sea: Evidence from their spatial distribution in surface water

Surface water samples were taken at 6 locations in the North Sea/Norwegian Sea (sampling locations I-VI), 22 locations in the North Sea/German coast (sampling locations 1-22) and 18 locations in the Baltic Sea (sampling locations A-R) in 2007 (**Figure 26**). Details of the sampling and the water temperature and salinity are presented in **Table S17** in the Supplementary material. Two litre water samples were collected in brown glass bottles via the ships' intake systems at approximately 11 m below the surface. The two litre water samples were filtered directly after sampling onboard or the following days using GFFs (> 1.2 µm). The samples were extracted and analysed as described in **publication II**.

In addition, at 19 sampling locations in the North Sea (i.e., location 1-8, 10, 11 and 13-21) water samples were collected in ten litre glass bowls at the same water depth as the two litre water samples. The ten litre water samples were taken from the Federal Maritime and Hydrographic Agency (BSH), in order to compare the two different sampling and analysis techniques. The water samples were extracted with an half-automated extraction system (APOS, Automated Extraction System for Organic Substances) using a 12 mL PP cartridge filled with 1.7 g glass fibre cotton for the separation of the particles and 1.7 g of Chromabond HR-P resin (Macherey-Nagel, Düren, Germany) for the enrichment of the target compounds (for details see Theobald et al. 2007a).

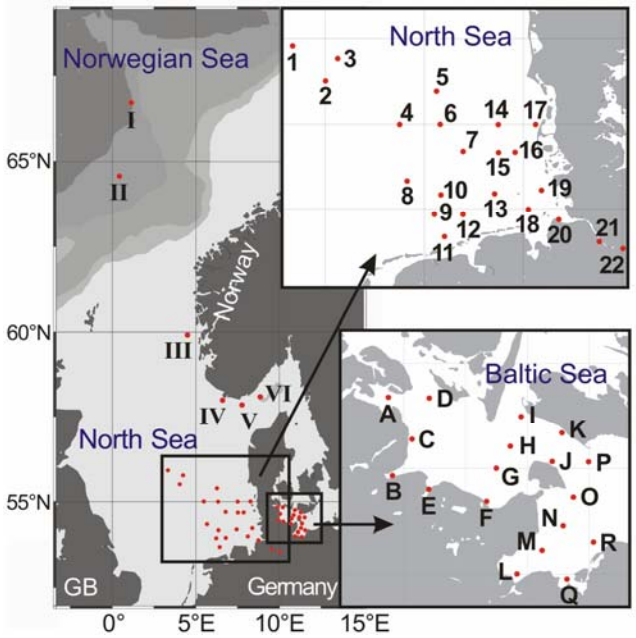

Figure 26. Geographic locations of the water sampling sites in the North Sea, Norwegian Sea and Baltic Sea

Interlaboratory comparison

Both laboratories used SPE and HPLC-(-)ESI-MS/MS approaches, but different sample volume, SPE method and instrumentations. Good agreement of the concentrations, determined by GKSS and BSH, was found for the PFCAs and PFOS (r^2 = 0.92, see **Figure 27**). Higher differences of PFHxS (mean difference = 185%) and FOSA (mean difference = 92%) could be explained by their concentration levels close or under the MQLs. The same discrepancy was also observed in some low concentration samples for PFOA (i.e., 0.040 ng/L (GKSS) and 0.190 ng/L (BSH) at sampling location 4) and PFOS (i.e., 0.073 ng/L (GKSS) and 0.240 ng/L (BSH) at sampling location 6). The results of the interlaboratory study show that the method improvement is an important challenge for the future work.

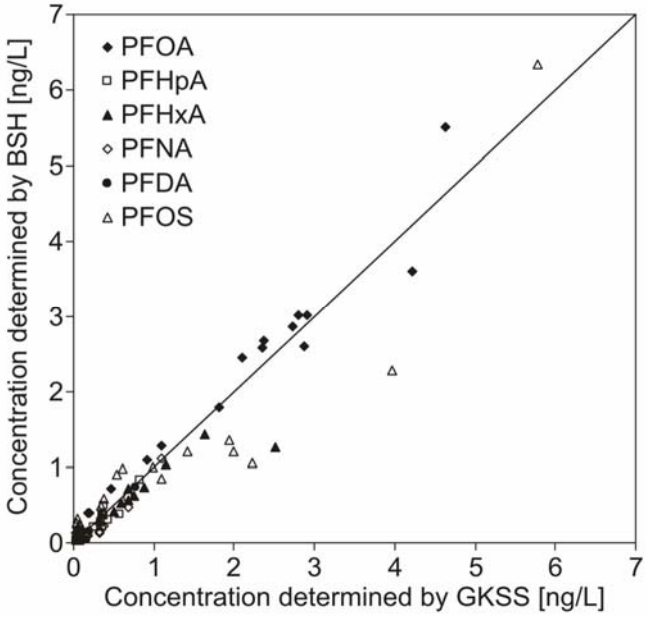

Figure 27. Comparison of PFCA and PFOS concentrations determined by GKSS, Geesthacht and BSH, Hamburg in the North Sea using two different PFCs analysis techniques in the dissolved water phase (details are given in the text)

Concentration levels in the North Sea, Norwegian Sea, and Baltic Sea

The concentration ranges, which were found in the dissolved and particulate phase, are shown for six areas (Norwegian Sea, Norwegian coast, open North Sea, German coast, river Elbe and Baltic Sea) in **Table 21**. Overall, 15 of 40 examined PFCs could be quantified, which included C_4-C_6 and C_8 PFSAs, C_4-C_{10} and C_{14} PFCAs, FOSA and C_4 and C_8 FASEs. PFCs were found in average to over 99% in the dissolved phase, whereas only PFOA, PFOA, PFTeDA and NEtFOSE could quantify in the particulate phase. The predominate compounds

in the dissolved phase were PFOA and PFBS and in the particulate phase PFOS.

Table 21. Concentration range for individual PFCs and ∑PFCs in ng/L (dissolved phase/particulate phases) for the Norwegian Sea, Norwegian coast, open North Sea, German coast, river Elbe and Baltic Sea

Area	**Norwegian Sea**	**Norwegian coast**	**Open North Sea**	**German coast**	**River Elbe**	**Baltic Sea**
Location number [a]	**I-II**	**III-VI**	**1-3**	**4-19**	**20-22**	**A-R**
	Concentration (dissolved phase/particulate phase) [ng/L]					
PFBA	n.d.	n.d.	n.d.	n.d.-4.73/-	n.d.-0.40/-	n.d.-0.44/-
PFPA	n.d.	n.d.	n.d.	n.d.-0.38/-	0.37-0.47/-	n.d.-0.12/-
PFHxA	n.d.	0.20-0.31/-	n.d.	n.d.-1.18/-	1.66-2.56/-	0.12-0.27/-
PFHpA	n.d.	n.d.-0.21/-	n.d.	n.d.-0.58/-	0.70-0.94/-	0.06-0.26/-
PFOA	0.01/-	0.07-0.35/-	0.02-0.07/-	0.08-3.02/-	4.36-4.81/-	0.25-4.55/-
PFNA	n.d.	0.01-0.04/-	0.04-0.05/-	0.05-0.37/-	0.69-1.16/-	0.10-0.42/-
PFDA	n.d.	n.d.	n.d.	n.d.-0.17/-	0.24-0.85/-	n.d.
PFTeDA	n.d.	-/n.d.-0.18	n.d.	n.d.	n.d.	n.d.
PFBS	n.d.	n.d.-0.09/-	n.d.-0.07/-	0.01-6.51/-	3.49-5.27/-	0.26-0.88/-
PFPS [b]	n.d.	n.d.	n.d.	n.d.-0.10/-	0.13-0.20/-	n.d.
PFHxS	n.d.	n.d.-0.03/-	n.d.	n.d.-0.28/-	0.32-0.50/-	n.d.-0.61/-
PFOS	n.d.	n.d.	n.d.-0.07/-	n.d.-2.26/ n.d.-0.16	4.09-6.16/ n.d.-1.07	n.d.-0.35/ n.d.-0.03
FOSA	n.d.	0.12-0.28/-	n.d.-0.07/-	n.d.-0.38/-	0.50-0.78/-	n.d.-0.46/-
NEtFOSE	n.d.	n.d.	n.d.	-/n.d.-0.74	n.d.	n.d.
MeFBSE	n.d.	n.d.	0.04-0.43/-	n.d.-0.93/-	0.41-0.69/-	n.d.
∑PFCs	0.01/-	0.55-1.10/ n.d.-0.18	0.15-0.57/-	0.15-16.15/ n.d.-0.94	19.41-22.08/ n.d.-1.07	0.92-6.24/ n.d.-0.03

n.d. = not detected; [a] the locations of the numbers can be found in the map displayed in **Figure** 26; [b] have to be considered as estimates, because no standard was available for this compound.

Highest ∑PFC concentrations of all sampling stations were found in the river Elbe with 22 ng/L (sampling station 21). Towards the offshore sea the ∑PFC concentration decreased rapidly by, in average, a factor of ~50 (sampling stations 1 to 3), which indicates a strong influence of the river on the concentration level at the German coast. This was confirmed with the high annual discharge for ∑PFCs of 506 kg/year for the dissolved phase and 4 kg/year for the particulate phase. The annual discharge based on the observed Elbe concentrations and mean annual discharge of 745 m^3/s at sampling station 22 (personal communication ARGE Elbe), which is a roughly estimation assuming a constant load over the whole year. The river Elbe flows through a high industrial area, where PFBS, PFOS and PFOA dominated (sampling stations 20 to 22), which suggests that there may be a specific source of these compounds in the river Elbe watershed. Conversely to the river Elbe, PFBA was detected at the German coast and also the concentration of PFBS increased (sampling stations 4-19). This may be the result of an additional source, where PFBS and PFBA were transported into the

North Sea by the westerly current. It is suggested that this contamination was originating from the river Rhine, where Skutlarek et al. 2006 found a high PFC contamination caused by contaminated sewage sludge applied to neighbouring agricultural fields. In contrast to the increasing PFBS and PFBA concentrations, decreased the concentration of PFOS by a factor of ~5 from the river Elbe towards the North Sea, which may be the result of dilution effects and/or sedimentation processes. However, it was found that the sorption of PFSAs is by a factor of 1.7 stronger than of PFCAs (Higgins and Luthy 2006). In the particulate phase only PFOS, PFOA and NEtFOSA could observed sporadically in low concentration range. Highest particle-bound concentration could find for PFOS with 0.9 ng/L (4.8% of the ∑PFCs) at location 21 in the river Elbe, whereas in the North Sea particle-bound PFOS was only detected at two sampling stations under 0.12 ng/L (<1.3% of the ∑PFCs), these results confirm the hypothesis that the sediment is a potential sink of PFOS.

∑PFC concentrations along the Norwegian coast ranged from 0.55 to 1.1 ng/L (sampling stations III to VI), which is by a factor of ~10 times lower than at the German coast (sampling stations 4 to 19). The low contamination corresponds with the sparse populated Norwegian coast and therefore it existed less potential sources like from domestic and industrial WWTP effluents. The dominated PFCs were PFHxA, PFOA and FOSA, whereas PFBA and PFBS were not detected. The water can further transport to the Arctic region, whereas the PFC concentration decreased rapidly and only PFOA could detect with 0.01 ng/L (sampling stations I and II) in the Norwegian Sea. The transport of PFCAs by the water phase is supposed as the main transportation pathway in the Arctic region, whereas the long range atmospheric transport was considered as negligible (Prevedouros et al. 2006).

In the Baltic Sea the ∑PFC concentration ranged between 0.92 to 6.24 ng/L and was therefore by a factor over 3 lower than in coastal water from the German coast (sampling locations 4 to 19), but much higher in comparison to open North Sea water (0.15 to 0.57 ng/L, sampling locations 1 to 3). The higher contamination of the Baltic Sea can be explained by the relatively closed ecosystem, where the water can only flows out into the North Sea through the Danish strait, and additionally by the influence from over 250 streams, which drain into the basin from mostly industrial areas. The Baltic Sea is a brackish inland sea, which may explain the relative uniform distribution of PFCs in contrast to the North Sea, where no primary contamination source such as fluorochemical production could identify, but diffuse sources dominated. PFOA and PFBS were the predominated compounds with a concentration range of 0.16 to 4.5 ng/L and 0.26 to 0.88 ng/L, respectively. Interestingly, a positive relationship was found between PFBS concentration and the salinity (r^2 = 0.74, **Figure 28**), which suggest that PFBS originated from the North Sea with their high salinity

content. Only at one sampling location could quantify a PFC in the particulate phase with 0.03 ng/L PFOS (sampling location L).

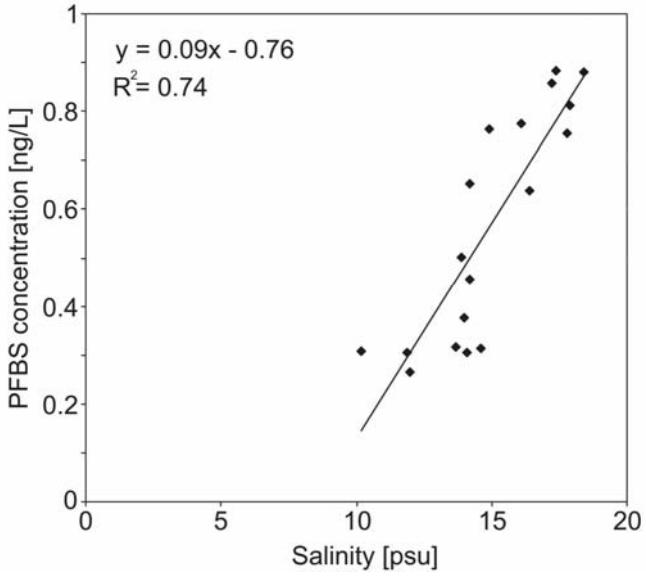

Figure 28. Relationship between concentrations of PFBS and salinity in surface water in the Baltic Sea

Composition and partitioning behaviour of PFC Congeners

The composition profile of individual PFCs of selected sampling locations is shown in **Figure 29**. The distribution pattern in the Norwegian Sea, North Sea and Baltic Sea depended on the sampling locations, where different kind of sources could identify. In the Norwegian Sea only PFOA could quantify, which can further transport to the Arctic region (sampling locations I). The composition in the open North Sea was dominated from the precursor compounds FOSA (0-13%) and MeFBSE (26-64%) (sampling locations 1 to 3), which can degrade to PFSAs and PFCAs (Martin et al. 2006; Rhoads et al. 2008). The source of these compounds could be the river Elbe (see sampling locations 20 to 22) or atmospheric transport and subsequent deposition (Stock et al. 2007). The composition from PFBA at the German coast increased from east to west, while the maximum of 35% was found at sampling location 10. Conversely, no PFBA was detected in the river Elbe (sampling location 21). These results suppose a transport from an unknown source with the westerly current. In the river Elbe the composition profile was similar to the profile at the German coast, but the composition of PFBS and PFBA was lower and equal to zero, respectively, and the composition of PFOS, PFHxA, PFNA and FOSA was higher, which indicates that the river Elbe was influenced from specific sources. Towards the Norwegian coast the composition of PFHxA, PFOA and FOSA increased, whereas the composition of PFOS decreased (sampling

stations V). Compared to the North Sea, in the Baltic Sea were PFBS and PFOA the predominated compounds with a proportion of over two-thirds of all quantified PFCs, otherwise the composition of PFOS and PFBA was lower. It was observed a gradient from west to east (sampling locations C to O), where the composition of PFOA increased from 46 to 68% and the composition of PFBS decreased from 19 to 13%.

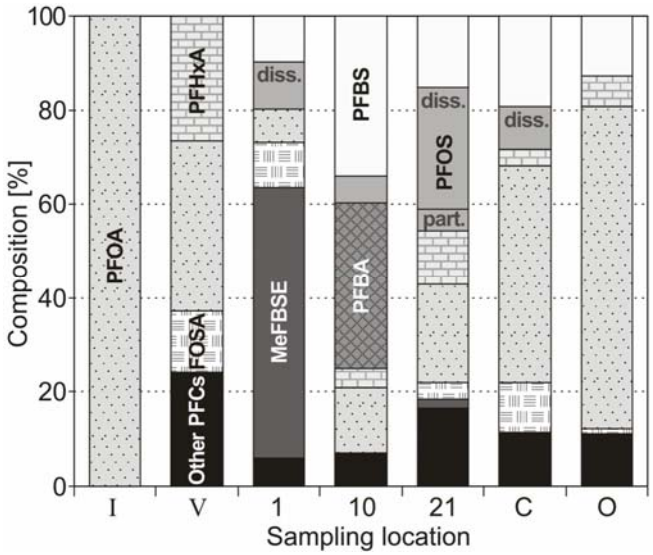

Figure 29. Relative composition of individual PFCs in surface water from selected sampling locations in the Norwegian Sea (I), North Sea (V, 1, 10), river Elbe (21) and Baltic Sea (C, O). Note: PFCs were distributed in the dissolved phase in the selected sampling locations, only PFOS was found in the dissolved (diss.) and particulate phase (part.). 'Other PFCs' include PFPS, PFHxS, PFPA, PFHxA, PFHpA, PFNA, PFDA, PFTeDA, and NEtFOSE

Conclusion

Each location in the North, Baltic and Norwegian Sea showed a specific composition profile depending on the distance to potential emission sources and transport pathways in the aqueous environment. Close to industrial or high populated areas PFC concentrations were higher, whereas in open sea water the concentration decreased rapidly. The reason could be high contaminated commercial and domestic waste water (Sinclair and Kannan 2006) resulting in local PFC hot spots and subsequent dilution during their aqueous transport. Close to contaminated sites the atmospheric deposition was negligible, but it could be relevant in open sea water (e.g., sampling locations 1 to 3). PFCs were found in average to over 99% in the dissolved phase, but in some samples with high SPM content the proportion in the particulate associate fraction increased for example in the river Elbe (sampling locations 21 and 22). Because of their persistence, PFCs can be transported over long distances (Yamashita et al. 2005), whereas sedimentation processes and the deep ocean water are

possible sinks (Prevedouros et al. 2006). Neutral precursor compounds like FASAs and FASEs were only detected sporadically, but it is possible that these precursors were already degraded in WWTPs (Schultz et al. 2006b; Sinclair and Kannan 2006). The occurrence of high concentrations of PFCs in coastal water could possibly be problematic, because they are bioavailable and can accumulate in the marine food chain (Martin et al. 2003a). Especially, the Baltic Sea has an unique flora and fauna, which is adapted to brackish water. Chemical 'fingerprints' may help to identify specific sources of PFC contamination into the aqueous environment.

7.4 Partitioning behaviour of PFCs between pore water and sediment in two sediment cores from Tokyo Bay

Two sediment cores were collected from Tokyo Bay using an acrylic tube (120 cm long and 12 cm i.d.) in May 2008 (core A and B, see **Figure 30**). These cores were sliced at 3-cm intervals for the first 9 cm for core A (6 cm for core B) and then at 2-cm intervals for up to 79 and 70 cm, respectively, using a clean stainless steel slicer and then stored in PP tubes. The sampling conditions, including total organic carbon (TOC), total nitrogen (TN), pH, oxygen reaction potential (ORP), moisture and dry density, are shown in **Table S18** and **Table S19** in the Supplementary material.

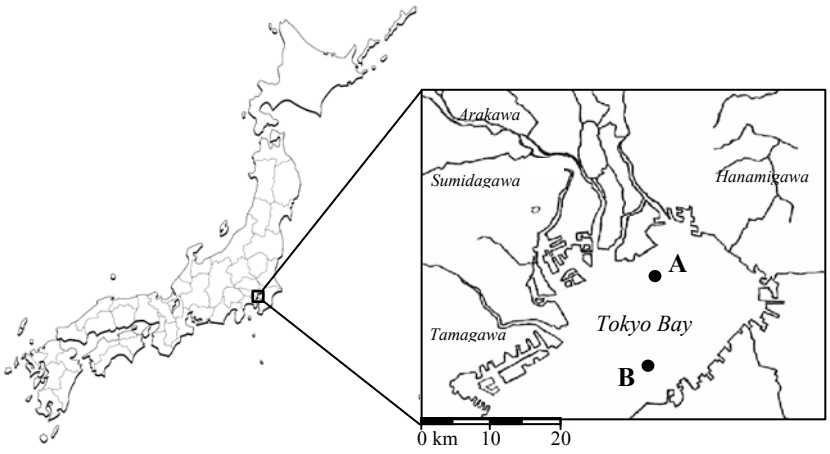

Figure 30. Map showing the sampling locations A and B in Tokyo Bay, Japan

The sedimentation rate was estimated from the excess ^{210}Pb (dpm) in each layer and the cumulative weight (g/cm) in core A. The average sedimentation rates of the dry matter were calculated to be 0.76 g/cm^2 per year for the 1958 to 2008 time period, which is approximately 1.5 cm/year for the core sections. The pore water was extracted from the wet sediment within

72 h by centrifugation at 10,000 rpm for 10 min at a constant temperature of 10 °C (AvantiTM J-25 Centrifuge, Beckman, USA).

The pore water was then filtered through 0.45 µm nylon syringe filters (Iwaki, Fukushima, Japan) into a PP bottle and extracted by SPE with Oasis® WAX cartridges as described by Taniyasu et al. 2005. The sediment was extracted using the method described by Powley et al. 2005 with a few modifications. Briefly, a 5 g sediment sample was weighed into a PP tube; 2 mL of 100 mM sodium hydroxide in 20% Millipore water and 80% methanol was added and then soaked for 30 min. The extraction was carried out with 20 mL methanol, and 1 ng absolute IS mix was spiked. The sample was then shaken in a wrist-action shaker at 250 rpm for 30 min. After shaking, the tube was centrifuged at 3000 rpm for 15 min, and the supernatant was decanted into another PP tube. The extraction was repeated with 1 mL 100 mM sodium hydroxide in 20% Millipore water and 80% methanol, soaked for 30 min, and 10 ml methanol, shaken at 250 rpm for 30 min and centrifuged at 3000 rpm for 15 min. Both extracts were combined and acidulated with 0.1 mL 4 M hydrochloric acid. This extract was centrifuged again at 3000 rpm for 5 min, and an aliquot of one-eighth (4.15 mL) of the supernatant was used for the cleanup with Supelclean ENVI-Carb® cartridges (100 mg, 1 mL, 100-400 mesh, Supelco, USA). The conditioning of the cartridges was carried out three times with 1 ml methanol. Afterwards, the sample extract and three times 1 mL methanol were added to the cartridge and directly collected in another vial. Finally, the extract was reduced to 1 mL under a nitrogen stream. Details of the instrumental conditions and quantification for PFC analysis have been described elsewhere (Taniyasu et al. 2008).

Vertical profiles

In the following, the concentrations in the pore water and dried sediment are expressed in pg/cm^3 to enable a direct comparison of their concentration levels. In this study, 11 PFCs were found in pore water (i.e., PFHxS, PFOS, 6:2 FTS, N-EtFOSAA, and C_4-C_{10} PFCA). In contrast to those in the pore water, the PFCs in the dried sediment showed a different vertical profile. No 6:2 FTS or shorter-chain PFCAs ($C \leq 7$) were detected in the sediment, although PFDS, FOSA and longer-chain PFCAs ($C \geq 11$) were. These results correspond with the findings of that each CF_2 moiety increases the distribution coefficient by 0.5 to 0.6 log units (Higgins and Luthy 2006) and ~0.87 log units (Liu and Lee 2007), respectively. In total 10 PFCs were quantified in the sediment (i.e., PFHxS, PFOS, PFDS, FOSA, N-EtFOSAA, and C_8-C_{12} and C_{14} PFCA).

Both cores had a similar vertical profile, with a maximum ∑PFC concentration at depths of 9-11 and 6-8 cm of 182 pg/cm^3 (sediment core A) and 132 pg/cm^3 (sediment core B), respectively (**Figure 31**). Close to the surface, these ∑PFC concentrations were slightly

lower, and, after reaching their maximum, with increasing sampling depth they rapidly decreased by a factor of 6 to 9. In general, PFHxS, PFOS, FOSA, N-EtFOSAA and, in a lower proportion, PFUnDA accounted for the highest proportion in the sediment, whilst in the deeper layers only PFOS was found. FOSA and N-EtFOSAA were found only in the upper layers (up to 35 cm in depth in core A to 46 cm in depth in core B), whereas the composition of PFHxS and PFOS increased with increasing depth. Interestingly, the proportion of ∑PFCs increased in the pore water with increasing sampling depth. In the pore water, PFBA, PFOA and PFNA dominated in the upper layers, but the composition changed in the deeper layers, in which, in addition to PFOA, PFHxA became the predominant compound, whereas PFBA was only predominant in core B.

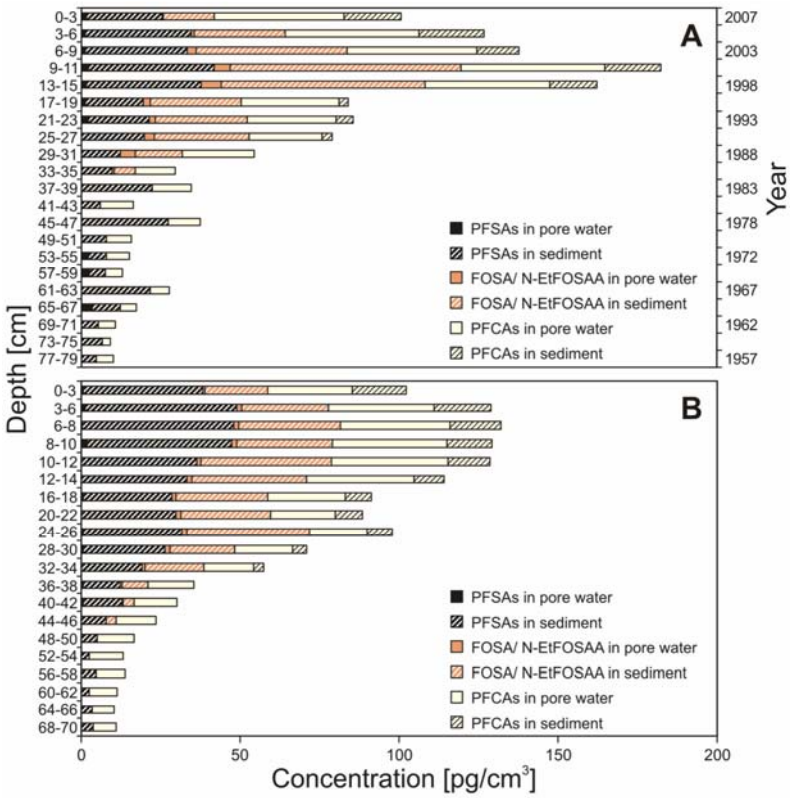

Figure 31. Vertical profile of PFSAs, FOSA/ N-EtFOSAA and PFCAs in pore water and sediment in pg/cm^3 from two sediment cores (A and B) collected from Tokyo Bay

Fluxes and temporal trends

The fluxes were calculated for core A using the dry sediment concentration multiplied by the density-corrected yearly sedimentation rate. The highest flux was observed for the ∑PFCs, with 197 pg/cm^2 per year in 2001-2002, respectively. Before and after these time periods the ∑PFCs decreased down to 7.1 pg/cm^2 per year (1956-1958) and 87.7 pg/cm^2 per year (2006-2008), respectively. The greatest proportion of the flux was attributable to PFOS and

EtFOSAA, whereas PFOS was largely responsible for the increasing flux and EtFOSAA for the reduction of the flux before and after 2001-2002. The calculated flux in sediment core A was used for a rough estimation of the total flux of PFCs in Tokyo Bay sediments from 1956 to 2008 assuming that the sediment surface amount to 1000 km^2. Based on this assumption the total flux for the 52 year period was estimated to be 28.5 kg total for ∑PFCs in Tokyo Bay sediments, a partition in 13.6 kg total for PFSAs, 11.6 kg total for FOSA/N-EtFOSAA and 3.2 kg total for PFCAs.

ANOVA tests (SPSS 16.0 for Windows, 2007) were used to determine the statistically significant differences (significance level $\alpha = 0.05$) between 1956 and 2008 for each analyte (**Figure 32**). Data were natural-logarithm transformed prior to statistical analysis to meet assumptions of normality and the homogeneity of variances. No significant trend was observed for PFHxS ($p = 0.200$), PFDA ($p = 0.413$) and PFDoDA ($p = 0.073$), whereas concentrations of PFOS ($p < 0.0001$), PFNA ($p < 0.0001$) and PFUnDA ($p < 0.001$) increased from 1956 to 2008, 1990 to 2008 and 1990 to 2008, respectively. Doubling times were calculated as described elsewhere (Butt et al. 2007b), with 16.1, 4.0 and 5.1 years for PFOS, PFNA and PFUnDA. In this study, however, the increasing trend of PFOS slowed down between 2001 and 2008 and possibly reached currently a steady state level. The concentration of FOSA and N-EtFOSAA increased from 1985 to 2001 with doubling times of 6.3 and 4.5 years, respectively ($p = 0.046$ and $p = 0.002$, respectively), but after 2001 the concentration decreased significantly with half-lives of 13.5 and 2.8 years, respectively ($p = 0.013$ and $p = 0.002$, respectively). This may reflect increased production and emissions since the 1950s in the Tokyo Bay area and the phased out of POSF-based compounds by 3M in 2000, of which at least 4 companies in Japan were affected (Paul et al. 2009). Nearly the same temporal trend was observed for FOSA in melon-headed whales stranded along the Japanese coast with increasing concentration between 1982 and 2001/2002, and following decreasing concentration until 2006 (Hart et al. 2008a).

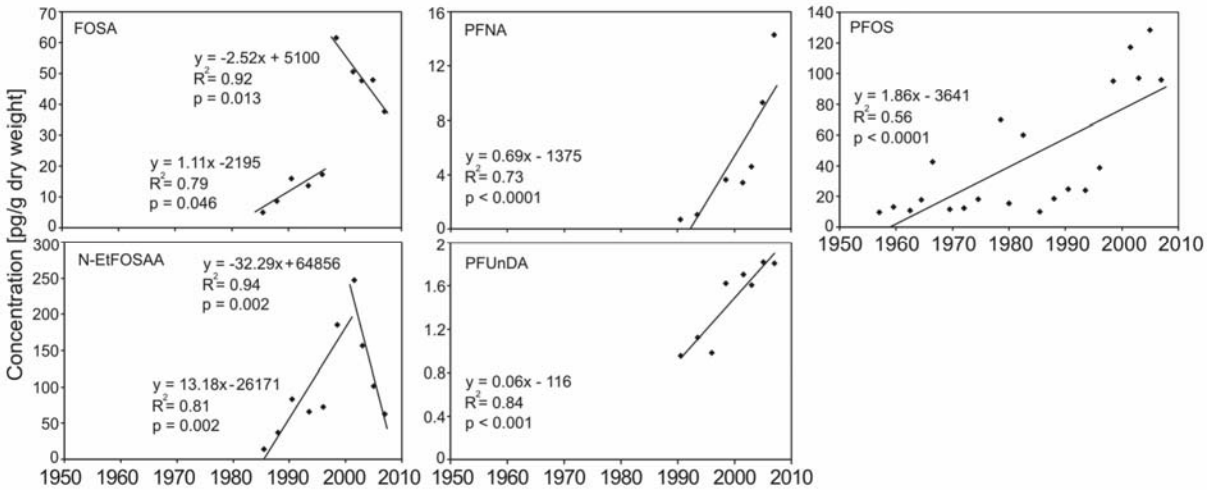

Figure 32. Temporal trends of PFOS, FOSA, N-EtFOSAA, PFNA and PFUnDA in sediment core A from Tokyo Bay

Influence of organic carbon and pH on sediment concentrations

The physical and geochemical characteristics of the vertical sediment profile were spatially variable. While the TOC decreased from 0.7 to 1.7%, the pH increased from 7.3 to 7.7 with the depth. In this study, a positive correlation was found between organic matter and concentrations of PFOS, FOSA and PFUnDA ($p < 0.0001$, **Figure 33**), and, with lower significance, N-EtFOSAA, PFNA and PFDoDA ($p = 0.010$, 0.015 and 0.022, respectively. This shows that the organic matter may have a high influence on the vertical distribution of PFCs in the studied sediment cores, which was also observed experimental for PFOS, N-EtFOSAA and PFDA (Higgins and Luthy 2006), and also 8:2 FTOH (Liu and Lee 2005) in previous studies. The concentration of PFOS, PFNA and PFDoDA increased with decreasing pH ($p = 0.009$, 0.022 and 0.015, respectively). This corresponds with the results of Higgins and Luthy 2006, showing increasing sorption of PFCs with decreasing pH of approximately 0.37 log units per unit pH.

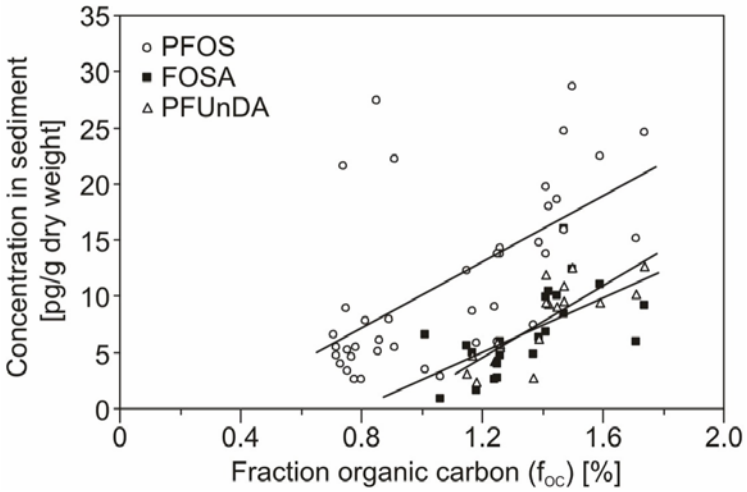

Figure 33. Dependence of PFOS, FOSA and PFUnDA concentrations in sediment on sediment fraction organic carbon (f_{OC})

Conclusion

The study showed the partition behaviour of PFCs between pore water and sediment in two sediment cores. Pore water concentrations of PFCs were determined for the first time. PFSAs, N-EtFOSAA and FOSA seemed to bind more strongly to sediment than PFCAs, whereas only the shorter-chain PFCAs ($C \leq 7$) could be found exclusively in the pore water. These results corroborate the laboratory findings of Higgins and Luthy 2006 and thus show that perfluorocarbon chain length and functional group both influence partitioning behaviour of PFCs in the real environment. In addition, an increasing sorption was found with increasing organic matter and decreasing pH, which correspond with experimental data (Higgins and Luthy 2006; Liu and Lee 2007). However, other factors like geochemical parameters (e.g., metal cations, etc.) (Higgins and Luthy 2006) or benthic organisms (e.g., degradation, bioturbated mixing, etc.) could have an influence on the partitioning behaviour of PFCs in sediment. The presence of longer-chain PFCAs ($C \geq 8$), PFSAs, FOSA and N-EtFOSAA in the sediment suggest a their bioavailability to benthic organisms (Higgins et al. 2007) and that the aquatic sediment act as a sink for these compounds. Future work could link the pore water and sediment concentrations of PFCs to bioconcentration factors, uptake routes and possible adverse effects. The temporal trend data presented here provide a basis for future trend studies of PFCs in sediment cores.

8. Summary and outlook

In this study the determination of PFCs in surface water and different biological matrices was developed. The water samples were filtrated and the dissolved and particulate phases were extracted separately using SPE with Oasis® WAX or Strata® XAW cartridges for the dissolved phase and sonication with methanol for the particulate phase (see **publication I**; Ahrens et al. 2009c). The biota samples were extracted using sonication with acetonitrile and subsequent clean-up using ENVI-Carb® cartridges (see **publication III**). All samples were analysed simultaneously for 40 target compounds plus 20 IS using HPLC/(-)ESI-MS/MS. The target compounds include 16 PFCAs, 7 PFSAs, 3 PFSiAs, 3 FTCAs, 3 FTUCAs, 4 FASAs, 3 FASEs and 6:2 FTS. In most studies, the concentrations of PFOS and PFOA were only reported, whereas in this study, over 20 PFCs could detect in the surface water and biota, respectively. In the dissolved phase in water dominated the short-chained PFCAs (C_4-C_{10}), PFBS, PFOS, PFOSi and FOSA, while in the particulate phases in water and in biota dominated the longer-chained PFCAs (C_8-C_{15}), PFSAs, PFOSi and FOSA. However, several of the detected compounds were found for the first time in surface water and biota.

The optimised analytical protocol for the analysis of PFCs in biota was integrated in the report of the MCWG for the International Council for the Exploration of the Sea (ICES) (ICES 2008). Furthermore, an interlaboratory comparison of the sampling techniques and analysis was conducted between the GKSS and BSH for water samples in the North Sea (see **chapter 7.3**). Both laboratories used SPE and HPLC-(-)ESI-MS/MS approaches, but different sample volume, SPE method and instrumentations. Overall, the results indicate a good agreement of the concentrations determined by the GKSS and BSH. In addition, the accuracy, precision, robustness and matrix effects of the analysis of PFCs in water and biota samples was verified in an interlaboratory method evaluation study (Van Leeuwen et al. 2009). In total 21 North American and European laboratories attended the interlaboratory study. The within laboratory precision of individual laboratories was good with 12% for water samples and 6.8% for fish tissue for all PFCs. These results improved considerably in comparison to an previous interlaboratory study (Van Leeuwen et al. 2006). The using of well-defined native and IS for quantification and the minimisation of matrix effects by an effective clean-up were identified as the most important steps for a good analytical performance. However, the method improvement is an important challenge for the future work.

A variety of laboratory products contain fluoropolymers such as PTFE, which can contaminate the samples during the sampling, sample preparation, and instrumental analysis. The sources of contamination were identified and eliminated which made it possible to detect

low pg/L levels in oceans waters and pg/g ww levels in biota samples. But instrumental signal suppression was observed for some compounds depending on the extraction volume for the water samples and the tissue-type for the biota samples. It is recommended for future studies to optimise further the extraction method, to use additional clean-up steps and/or, if possible, to reduce the sample volume. However, 20 IS were used for correction of matrix effects and possible losses during the sample preparation.

A water volume of 1-5 L was used for SPE in this study. For further studies at low contaminated sampling sites (e.g., Arctic, Antarctica, etc.) it could be useful to enrich a higher volume of water on SPE cartridges to reduce the MDL. On the other hand, for higher contaminated water samples (e.g., WWTP effluents, landfill sites, river water, etc.) a large-volume injection for the direct analysis of PFCs using HPLC/(-)ESI-MS/MS could be useful for routine analysis.

The riverine transportation of PFCs into the German Bight was investigated (see **chapter 7.1** and **7.2**). The municipal WWTP effluents had only a low influence on the mass flow along the river Elbe. Further investigations are necessary to identify the origin of the contamination like diffuse sources from runoff into the river or other point sources like landfill effluents and/or industrial WWTPs. Furthermore, for a mass balance, besides the sources, additionally the sinks (e.g., sediment, biota) should be investigated. In addition, a monitoring over a longer period using a flow-proportional composite sampler would be useful to determine temporal trends or seasonal changes. Ultimately, the particle mass and its content of organic matter should be determined to obtain a better understanding of the exchange processes between the dissolved and particulate phase, and to calculate partition coefficients between them.

The occurrence and distribution pattern of PFCs was investigated in the German Bight to identify potential sources in the sampling area (see **publication I**). The rivers Elbe, Weser and Ems had a high influence on the distribution of individual PFCs in the German Bight, with maximum PFC concentrations found in their estuaries, and decreasing concentrations with increasing distance from the coast. The compounds PFBS and PFBA are not originated from the rivers Elbe, Weser and Ems, but the river Rheine might be the source for the contamination of these compounds in the German Bight. However, chemical ‘fingerprints’ may help to identify specific sources of PFC contamination into the aqueous environment. Further studies on the spatial distribution of PFCs in coastal area are necessary to understand the transportation mechanism and fate of PFCs in the marine environment.

The distribution of PFCs in surface water in the Atlantic Ocean was investigated along the longitudinal gradient from Las Palmas (Spain) to St. Johns (Canada) (15° W to 52° W) and

the latitudinal gradient from the Bay of Biscay to the South Atlantic Ocean (46° N to 26° S) (see **publication II**). The increasing and decreasing concentration gradients of PFCs in the Atlantic Ocean can be explained by the pattern of ocean water currents, in which industrial areas are considered as a source for PFCs, and ocean waters and the atmosphere are important as sinks and for transportation of these compounds. These data of the spatial distribution of PFCs in ocean waters are very useful for global transportation models (Wania and Mackay 1995). In this study, only at one station were collected deep water samples. It was hypothesised that PFCs could be transported globally with the thermohaline circulation system (Yamashita et al. 2008), further investigations on the vertical concentration profile of PFCs are necessary to support this assumption. In addition, further studies on the distribution of PFCs in the surface microlayer (50 µL thickness) and subsurface water (> 30 cm depth) (Ju et al. 2008) are necessary to understand the importance of the seaspray-mediated transport (McMurdo et al. 2008). Overall, further investigations of the biochemical cycle of PFCs in ocean waters are necessary for understanding of the transportation and fate of individual PFCs in the marine environment.

In previous studies the concentration in liver tissue and plasma are often used to estimate whole body burden in animals. In this study, the distribution of PFCs in harbor seals for a wide range of biological matrices (i.e., liver, kidney, lung, heart, blood, brain, muscle, thyroid, thymus, and blubber) was investigated (see **publication III**). This provides advice on the analysis of the whole body burden in harbor seals for individual PFCs, which is relevant for calculation of the bioaccumulation potential of these compounds in marine mammals. Blood and liver contributed three-fourths of the whole body burden for PFCs in harbour seals. It is recommended for monitoring of PFCs in marine mammals to collect liver or blood samples, because there were found an effectively accumulation in these tissues. However, it is suggested that the distribution of individual PFCs depends on the species, which make further studies on the tissue distribution and whole body burden in other organisms necessary. Furthermore, the bioaccumulation potential could be studied by the determination of PFCs along the marine food web (e.g., zooplankton, benthic organisms, fish, etc.).

Higher concentrations were found in younger harbor seals (<7 month old) in comparison to older harbor seals (≥ 7 month old) for several PFCs suggesting transplacental transfer of PFC contaminants to the foetus and/or consumption of different contaminated food (see **publication IV**). However, information on the influence of the age on the PFC concentrations in marine mammals is very sparse. The harbor seals had a relatively high whole body burden for PFCs, but no significant correlation between PFC concentration levels and health status was found. But, physiological changes were observed in Baikal seals (Ishibashi et al. 2008b)

which required further studies in order to evaluate association between PFCs and pathological conditions.

Significant declining concentrations of C_5-C_7 PFSAs, PFOSi, FOSA and PFOA were observed in archived harbor seal livers from the German Bight over the last decade (see **publication IV**). The reason could be, probably, that this is the effect of the phase-out of POSF by the 3M Company, the reduction of PFOA emissions by the stewardship program from the U.S. EPA and replacement of PFOS and their derivates by less bioaccumulative compounds because of the formed directive from the EU (Prevedouros et al. 2006; European Parliament and Concil 2006; U.S. EPA 2006). But the high concentrations of PFOS and constant levels of C_9-C_{13} PFCAs indicates that further work on the reduction of environmental emissions of PFCs are necessary. In addition, it is suggested that in biota exist a high contribution of unidentified PFCs (Miyake et al. 2007b) which needs a non-target analysis to identify and quantify these compounds. Ultimately, this study highlights the importance of further monitoring studies in seals and other marine mammals from highly populated sites in order to evaluate changes in pattern and long-term perspective trends.

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10. Supplementary material

Table S1. Locations, sampling time and standard parameters of sampling for the German Bight survey from 1st to 9th August in 2007 [a]

Station number	Sampling date	UTC time [b]	Latitude	Longitude	Water temperature [°C]	Suspended particulate matter [mg/L]	Dissolved organic carbon [mg/L]	Particulate organic carbon [mg/L]
1	01/08/2007	11:45	N53° 53.40'	E08° 42.60'	18.0	53.68	n.a.	1.58
2	01/08/2007	12:33	N53° 58.20'	E08° 37.80'	17.6	178.38	8.32	4.87
3	01/08/2007	13:25	N53° 59.40'	E08° 26.10'	17.9	45.44	9.47	0.87
4	01/08/2007	14:45	N54° 03.83'	E08° 10.71'	n.a.	63.00	7.53	0.98
5	01/08/2007	15:30	N54° 08.15'	E07° 59.91'	n.a.	29.64	n.a.	0.45
6	01/08/2007	15:56	N54° 09.91'	E07° 54.22'	n.a.	27.13	7.06	0.37
7	02/08/2007	06:25	N54° 08.40'	E07° 39.60'	18.1	0.89	n.a.	0.23
8	02/08/2007	07:06	N54° 02.10'	E07° 45.60'	18.1	2.29	7.80	0.35
9	02/08/2007	08:11	N53° 55.80'	E07° 55.80'	18.0	3.77	8.07	0.32
10	02/08/2007	09:01	N53° 51.60'	E08° 07.20'	18.0	10.22	8.74	0.58
11	02/08/2007	10:02	N53° 44.40'	E08° 13.20'	17.9	26.80	n.a.	1.43
12	02/08/2007	11:20	N53° 37.20'	E08° 28.80'	19.1	41.02	11.81	1.72
13	02/08/2007	12:07	N53° 30.60'	E08° 32.40'	20.0	58.73	12.91	2.50
14	04/08/2007	09:37	N53° 49.80'	E07° 54.00'	18.3	29.82	8.04	1.42
15	04/08/2007	09:55	N53° 49.20'	E07° 49.20'	18.4	11.66	7.02	0.75
16	04/08/2007	10:36	N53° 47.10'	E07° 37.80'	18.6	57.02	8.12	1.09
17	04/08/2007	11:18	N53° 46.80'	E07° 25.80'	18.7	13.60	7.68	0.79
18	04/08/2007	12:33	N53° 44.40'	E07° 07.20'	18.7	5.98	8.48	0.66
19	04/08/2007	13:06	N53° 43.20'	E06° 59.40'	18.2	6.68	n.a.	0.54
20	04/08/2007	14:17	N53° 40.80'	E06° 41.40'	18.3	11.73	n.a.	0.79
21	05/08/2007	06:59	N53° 19.80'	E07° 10.20'	18.8	87.03	n.a.	2.97
22	05/08/2007	07:29	N53° 19.80'	E07° 00.00'	18.8	37.83	11.33	1.60
23	05/08/2007	08:15	N53° 28.20'	E06° 51.60'	18.7	81.54	n.a.	3.20
24	05/08/2007	09:13	N53° 36.00'	E06° 36.60'	18.6	27.32	n.a.	1.27
25	05/08/2007	10:36	N53° 48.60'	E06° 30.60'	18.7	2.37	7.06	0.42
26	05/08/2007	11:46	N54° 00.00'	E06° 30.60'	18.7	3.55	n.a.	0.38
27	05/08/2007	13:27	N54° 00.60'	E07° 00.60'	19.5	1.36	6.16	0.38
28	05/08/2007	14:32	N54° 01.20'	E07° 20.40'	19.5	1.05	5.75	0.31
29	06/08/2007	11:12	N54° 25.28'	E07° 53.94'	n.a.	45.96	n.a.	0.42
30	06/08/2007	12:23	N54° 36.71'	E07° 53.99'	n.a.	1.24	7.63	0.35
31	06/08/2007	13:47	N54° 50.07	E07° 54.14'	n.a.	1.07	6.20	0.30
32	06/08/2007	15:12	N55° 03.88'	E07° 53.52'	n.a.	1.39	n.a.	0.45
33	06/08/2007	16:11	N55° 05.55'	E08° 10.32'	n.a.	4.48	6.69	1.40
34	06/08/2007	17:11	N55° 02.60'	E08° 28.07'	n.a.	1.93	7.24	0.22
35	07/08/2007	09:17	N54° 54.32'	E08° 15.92'	n.a.	2.28	5.81	0.46
36	07/08/2007	10:46	N54° 40.30'	E08° 10.32'	n.a.	2.09	6.26	0.31
37	07/08/2007	11:33	N54° 44.16'	E08° 18.63'	n.a.	2.54	5.45	0.33
38	07/08/2007	12:18	N54° 47.56	E08° 27.55'	n.a.	3.77	n.a.	0.36
39	07/08/2007	14:30	N54° 40.20'	E08° 31.80'	n.a.	2.63	5.65	0.29
40	07/08/2007	15:00	N54° 35.97	E08° 24.35'	n.a.	1.62	5.59	0.33
41	07/08/2007	16:30	N54° 36.35'	E08° 47.36'	n.a.	10.67	6.24	0.80
42	09/08/2007	08:10	N54° 28.80'	E09° 00.11'	n.a.	22.68	7.99	1.22
43	09/08/2007	09:20	N54° 26.44'	E08° 44.53'	n.a.	4.41	6.56	0.37
44	09/08/2007	10:13	N54° 22.93'	E08° 32.53'	n.a.	3.30	5.47	0.31
45	09/08/2007	11:07	N54° 14.30'	E08° 27.63'	n.a.	2.82	5.64	0.20
46	09/08/2007	11:56	N54° 05.78'	E08° 25.70'	n.a.	5.57	6.08	0.78
47	09/08/2007	12:11	N54° 03.38'	E08° 26.07'	n.a.	8.06	n.a.	0.58
48	09/08/2007	12:51	N53° 59.63'	E08° 34.04'	n.a.	14.73	n.a.	0.86

[a] n.a. = not available; [b] UTC = universal time coordinated.

Table S2. Concentrations and standard deviation of PFCs in ng per litre surface water from sampling station 1 to 16 in the German Bight [a]

Analyte	1	2	3	4	5	6	7 [b]	8	9	10	11	12	13	14	15	16
PFBS	5.05 ± 0.56	4.75 ± 0.42	5.38 ± 0.57	6.52 ± 0.19	4.81 ± 0.26	5.00 ± 0.77	5.80 ± 0.51	6.32 ± 0.13	4.75 ± 0.29	7.68 ± 0.02	8.19 ± 1.10	3.84 ± 0.11	5.45 ± 0.42	6.69 ± 0.71	7.97 ± 0.37	8.09 ± 0.05
PFPS [c]	0.39 ± 0.03	n.d.	0.20 ± 0.02	n.d.	0.13 ± 0.02	n.d.	0.11 ± 0.04	0.12 ± 0.003	0.13 ± 0.01	0.24 ± 0.03	n.d.	0.44 ± 0.01	0.70 ± 0.04	0.18 ± 0.01	0.20 ± 0.02	0.20 ± 0.03
PFHxS	0.91 ± 0.03	0.56 ± 0.03	0.46 ± 0.01	0.30 ± 0.02	0.27 ± 0.003	0.26 ± 0.03	0.29 ± 0.07	0.34 ± 0.05	0.31 ± 0.02	0.30 ± 0.05	0.47 ± 0.10	0.44 ± 0.05	0.38 ± 0.005	0.35 ± 0.003	0.39 ± 0.001	0.40 ± 0.02
PFOS	2.42 ± 0.18	1.40 ± 0.07	0.83 ± 0.04	1.35 ± 0.10	1.15 ± 0.17	0.69 ± 0.03	0.96 ± 0.29	0.80 ± 0.04	1.33 ± 0.001	1.21 ± 0.02	1.48 ± 0.17	2.51 ± 0.08	2.27 ± 0.58	1.00 ± 0.15	1.24 ± 0.05	1.39 ± 0.09
PFNS [c]	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.29± 0.06	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
6:2 FTS	0.44± 0.04	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.49± 0.12	n.d.	n.d.	n.d.	n.d.	n.d.
PFPA	0.65 ± 0.09	0.78 ± 0.03	1.60 ± 0.17	0.20 ± 0.04	0.96 ± 0.14	1.25 ± 0.14	1.07 ± 0.54	0.31 ± 0.03	0.76 ± 0.03	0.22 ± 0.001	2.50 ± 0.12	1.53 ± 0.10	0.94 ± 0.02	0.58 ± 0.02	0.94 ± 0.08	(0.13) ± 0.21
PFHxA	2.79 ± 0.14	2.30 ± 0.06	1.83 ± 0.07	1.15 ± 0.06	0.59 ± 0.02	0.67 ± 0.10	0.67 ± 0.07	1.19 ± 0.03	0.77 ± 0.01	1.64 ± 0.06	3.37 ± 0.12	9.51 ± 0.58	9.56 ± 0.06	0.72 ± 0.01	0.77 ± 0.0002	0.97 ± 0.07
PFHpA	1.87 ± 0.02	1.42 ± 0.09	1.07 ± 0.04	0.56 ± 0.03	0.40 ± 0.001	0.60 ± 0.04	0.51 ± 0.13	0.88 ± 0.03	0.49 ± 0.002	0.60 ± 0.02	0.90 ± 0.05	1.04 ± 0.01	2.52 ± 0.06	0.50 ± 0.01	0.60 ± 0.03	0.62 ± 0.06
PFOA	7.83 ± 0.11	4.94 ± 0.11	4.08 ± 0.32	3.39 ± 0.14	3.22 ± 0.09	3.34 ± 0.13	3.66 ± 0.13	4.28 ± 0.06	3.83 ± 0.11	3.71 ± 0.06	4.16 ± 0.03	5.29 ± 0.61	7.74 ± 0.76	3.96 ± 0.04	4.75 ± 0.32	4.35 ± 0.08
PFNA	0.92 ± 0.02	0.53 ± 0.04	0.20 ± 0.002	0.27 ± 0.03	0.19 ± 0.005	0.12 ± 0.004	0.17 ± 0.07	0.21 ± 0.03	0.20 ± 0.002	0.24 ± 0.005	0.26 ± 0.005	0.72 ± 0.07	0.76 ± 0.08	0.22 ± 0.02	0.20 ± 0.04	0.25 ± 0.001
PFDA	0.50 ± 0.01	0.20 ± 0.03	0.10 ± 0.001	0.11 ± 0.01	0.09 ± 0.004	0.07 ± 0.01	0.08 ± 0.01	0.10 ± 0.01	0.07 ± 0.01	0.08 ± 0.002	0.12 ± 0.0003	0.24 ± 0.03	0.62 ± 0.01	0.08 ± 0.002	0.06 ± 0.01	0.09 ± 0.01
PFUnDA	n.d.	0.02 ± 0.01	(0.01) ± 0.01	n.d.	0.03 ± 0.001	(0.01) ± 0.004	0.02 ± 0.02	n.d.	(0.01) ± 0.001	0.02 ± 0.003	n.d.	n.d.	0.05 ± 0.003	0.03 ± 0.005	0.02 ± 0.003	0.03 ± 0.001
PFDoDA	n.d.	(0.005) ± 0.002	n.d.	n.d.	0.03 ± 0.001	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	(0.003) ± 0.001	0.02 ± 0.002	n.d.	n.d.
FOSA	0.11 ± 0.0004	0.14 ± 0.0004	0.06 ± 0.002	0.03 ± 0.01	0.02 ± 0.003	n.d.	n.d.	0.07 ± 0.01	n.d.	0.04 ± 0.003	n.d.	0.06 ± 0.004	0.20 ± 0.01	0.04 ± 0.01	0.03 ± 0.01	n.d.
MeFBSA	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
MeFBSE	0.54 ± 006	0.29 ± 0.06	0.37 ± 0.01	n.d.	(0.18) ± 0.03	n.d.	n.d.	0.74 ± 0.17	n.d.	n.d.	n.d.	(0.13) ± 0.003	n.d.	(0.12) ± 0.004	n.d.	n.d.
FDUEA	(0.01) ± 0.001	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.04 ± 0.002	n.d.	0.04 ± 0.005	n.d.	0.06 ± 0.01	n.d.	0.02 ± 0.0004	n.d.	n.d.
∑PFCs	**24.43 ± 1.38**	**17.34 ± 0.94**	**16.18 ± 1.25**	**13.88 ± 0.60**	**12.06 ± 0.75**	**12.00 ± 1.26**	**13.63 ± 2.03**	**15.39 ± 0.59**	**12.65 ± 0.49**	**16.03 ± 0.29**	**21.94 ± 1.82**	**25.82 ± 1.68**	**31.18 ± 2.05**	**14.51 ± 0.99**	**17.17 ± 0.93**	**16.50 ± 0.61**

[a] Values in brackets are between MDL and MQL and are not included in the sum concentrations. n.d. = not detected; [b] mean and standard deviation of a duplicate sample; [c] have to be considered as estimates, because no standards were available for this compound.

Table S3. Concentrations and standard deviation of PFCs in ng per litre surface water from sampling station 17 to 32 in the German Bight [a]

Analyte	17	18	19	20	21	22	23	24	25	26	27	28	29 [b]	30	31	32
PFBS	7.32 ± 0.76	7.76 ± 0.16	9.21 ± 0.37	11.43 ± 0.06	4.78 ± 0.22	10.56 ± 1.05	6.18 ± 0.20	17.67 ± 1.58	5.14 ± 0.18	4.20 ± 0.05	4.27 ± 0.21	5.72 ± 0.23	4.48 ± 1.55	3.38 ± 0.13	3.72 ± 0.07	3.48 ± 0.07
PFPS [c]	0.15 ± 0.01	0.30 ± 0.04	0.16 ± 0.02	0.21 ± 0.02	0.79 ± 0.04	0.86 ± 0.01	0.51 ± 0.01	n.d.	0.15 ± 0.004	0.10 ± 0.01	0.08 ± 0.01	0.10 ± 0.0004	0.17 ± 0.04	n.d.	n.d.	0.13 ± 0.03
PFHxS	0.46 ± 0.02	0.45 ± 0.02	0.38 ± 0.001	0.38 ± 0.02	0.98 ± 0.04	1.13 ± 0.04	0.75 ± 0.04	0.59 ± 0.06	0.30 ± 0.005	0.22 ± 0.0003	0.27 ± 0.02	0.29 ± 0.02	0.24 ± 0.02	0.26 ± 0.2	0.26 ± 0.02	0.27 ± 0.04
PFOS	0.99 ± 0.19	1.44 ± 0.02	1.33 ± 0.04	1.29 ± 0.11	3.95 ± 0.51	3.04 ± 0.20	2.44 ± 0.18	1.75 ± 0.03	1.13 ± 0.04	0.88 ± 0.02	1.25 ± 0.01	0.86 ± 0.08	0.74 ± 0.01	1.34 ± 0.07	1.27 ± 0.04	1.40 ± 0.10
PFNS [c]	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
6:2 FTS	n.d.	n.d.	0.73 ± 0.05	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.32 ± 0.01	n.d.	0.32 ± 0.01	n.d.	n.d.	n.d.	n.d.
PFPA	0.98 ± 0.02	0.45 ± 0.04	0.53 ± 0.01	0.49 ± 0.11	0.33 ± 0.01	n.d.	0.36 ± 0.02	2.20 ± 0.31	0.57 ± 0.02	n.d.	0.34 ± 0.0005	0.96 ± 0.03	0.54 ± 0.23	0.70 ± 0.03	0.46 ± 0.05	0.52 ± 0.01
PFHxA	0.91 ± 0.10	0.83 ± 0.02	0.84 ± 0.01	0.94 ± 0.02	2.47 ± 0.08	2.28 ± 0.09	1.58 ± 0.03	1.29 ± 0.25	0.72 ± 0.01	0.47 ± 0.04	0.53 ± 0.05	0.61 ± 0.01	0.80 ± 0.04	0.71 ± 0.001	0.74 ± 0.04	0.66 ± 0.02
PFHpA	0.90 ± 0.07	0.54 ± 0.02	0.66 ± 0.03	0.58 ± 0.04	0.83 ± 0.02	1.02 ± 0.03	0.69 ± 0.01	0.80 ± 0.08	0.36 ± 0.03	0.36 ± 0.005	0.31 ± 0.004	0.59 ± 0.04	0.40 ± 0.07	0.41 ± 0.04	0.35 ± 0.01	0.40 ± 0.01
PFOA	4.85 ± 0.08	4.03 ± 0.16	4.11 ± 0.03	3.91 ± 0.09	7.43 ± 0.55	6.24 ± 0.31	5.79 ± 0.23	4.31 ± 0.63	3.74 ± 0.02	2.67 ± 0.01	2.96 ± 0.07	3.99 ± 0.39	3.02 ± 0.23	3.37 ± 0.08	2.95 ± 0.04	3.21 ± 0.02
PFNA	0.14 ± 0.0004	0.22 ± 0.01	0.19 ± 0.01	0.22 ± 0.01	0.82 ± 0.08	0.57 ± 0.01	0.49 ± 0.02	0.21 ± 0.001	0.21 ± 0.01	0.11 ± 0.003	0.17 ± 0.01	0.10 ± 0.02	0.14 ± 0.02	0.25 ± 0.02	0.24 ± 0.03	0.28 ± 0.07
PFDA	0.09 ± 0.01	0.08 ± 0.01	0.05 ± 0.01	0.10 ± 0.01	0.19 ± 0.002	0.11 ± 0.001	0.11 ± 0.003	n.d.	0.09 ± 0.01	(0.02) ± 0.01	(0.04) ± 0.005	0.06 ± 0.01	0.05 ± 0.03	0.08 ± 0.01	0.08 ± 0.002	0.09 ± 0.002
PFUnDA	0.02 ± 0.01	0.02 ± 0.01	0.02 ± 0.0001	0.03 ± 0.01	0.03 ± 0.01	0.02 ± 0.001	0.02 ± 0.002	n.d.	0.03 ± 0.002	n.d.	n.d.	n.d.	0.02 ± 0.01	0.02 ± 0.01	n.d.	0.02 ± 0.004
PFDoDA	0.01 ± 0.003	0.01 ± 0.002	n.d.	n.d.	0.01 ± 0.002	n.d.	n.d.	n.d.	0.03 ± 0.01	n.d.	n.d.	n.d.	n.d.	n.d.	0.01 ± 0.002	n.d.
FOSA	0.04 ± 0.003	0.02 ± 0.002	n.d.	n.d.	0.04 ± 0.001	0.03 ± 0.01	n.d.	n.d.	0.02 ± 0.001	n.d.	0.01 ± 0.03	0.04 ± 0.004	n.d.	n.d.	n.d.	n.d.
MeFBSA	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.46 ± 0.17	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
MeFBSE	n.d.	(0.15) ± 0.001	n.d.	n.d.	(0.14) ± 0.01	0.36 ± 0.01	(0.22) ± 0.01	n.d.	(0.14) ± 0.01	n.d.	(0.09) ± 0.01	n.d.	n.d.	n.d.	n.d.	(0.10) ± 0.01
FDUEA	(0.01) ± 0.01	n.d.	n.d.	n.d.	0.05 ± 0.003	0.05 ± 0.0002	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
∑PFCs	**16.87 ± 1.29**	**16.30 ± 0.52**	**18.23 ± 0.58**	**19.56 ± 0.49**	**22.83 ± 1.56**	**26.27 ± 1.75**	**19.14 ± 0.76**	**28.82 ± 2.95**	**13.08 ± 0.52**	**9.36 ± 0.16**	**10.33 ± 0.41**	**13.65 ± 0.84**	**10.68 ± 0.88**	**10.51 ± 0.42**	**10.08 ± 0.30**	**10.57 ± 0.39**

[a] Values in brackets are between MDL and MQL and are not included in the sum concentrations; n.d. = not detected; [b] mean and standard deviation of a duplicate sample; [c] have to be considered as estimates, because no standards were available for this compound.

Table S4. Concentrations and standard deviation of PFCs in ng per litre surface water from sampling station 33 to 48 in the German Bight [a]

Analyte	33	34	35 [b]	36	37	38	39	40	41	42	43 [b]	44	45	46	47	48
PFBS	4.11 ± 0.21	4.22 ± 0.23	4.01 ± 0.07	3.97 ± 0.03	5.49 ± 0.56	4.45 ± 0.15	4.67 ± 0.19	5.38 ± 0.39	4.79 ± 0.41	12.44 ± 2.60	3.60 ± 0.43	5.00 ± 1.69	7.85 ± 1.75	8.78 ± 1.37	8.00 ± 1.6	11.93 ± 0.39
PFPS [c]	n.d.	n.d.	n.d.	0.16 ± 0.02	0.12 ± 0.01	(0.06) ± 0.09	0.16 ± 0.01	n.d.	0.14 ± 0.002	n.d.	n.d.	0.11 ± 0.07	n.d.	n.d.	n.d.	n.d.
PFHxS	0.26 ± 0.04	0.31 ± 0.01	0.29 ± 0.01	0.26 ± 0.06	0.33 ± 0.01	0.30 ± 0.01	0.26 ± 0.04	0.29 ± 0.002	0.30 ± 0.02	0.47 ± 0.15	0.37 ± 0.04	0.24 ± 0.02	0.24 ± 0.03	0.50 ± 0.01	0.43 ± 0.02	0.67 ± 0.01
PFOS	1.42 ± 0.07	1.04 ± 0.06	0.98 ± 0.12	1.53 ± 0.35	0.74 ± 0.04	0.80 ± 0.07	1.02 ± 0.02	1.12 ± 0.06	0.95 ± 0.06	1.82 ± 0.09	1.04 ± 0.11	0.86 ± 0.23	1.18 ± 0.17	1.44 ± 0.003	1.63 ± 0.14	1.83 ± 0.13
PFNS [c]	n.d.	n.d.	0.11 ± 0.01	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
6:2 FTS	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
PFPA	0.72 ± 0.08	0.46 ± 0.11	0.39 ± 0.03	0.43 ± 0.07	0.91 ± 0.05	0.33 ± 0.02	0.33 ± 0.005	(0.13) ± 0.03	0.33 ± 0.01		1.21 ± 0.17	0.70 ± 0.07	2.02 ± 0.06	2.23 ± 0.27	2.04 ± 0.28	1.85 ± 0.01
PFHxA	0.81 ± 0.02	0.86 ± 0.01	0.80 ± 0.01	0.78 ± 0.04	0.91 ± 0.03	0.83 ± 0.04	0.83 ± 0.03	0.85 ± 0.03	0.90 ± 0.03	1.16 ± 0.14	1.06 ± 0.04	0.96 ± 0.12	0.96 ± 0.08	1.09 ± 0.01	1.21 ± 0.03	1.51 ± 0.05
PFHpA	0.42 ± 0.01	0.38 ± 0.02	0.37 ± 0.02	0.37 ± 0.01	0.70 ± 0.07	0.41 ± 0.01	0.40 ± 0.01	0.49 ± 0.05	0.42 ± 0.02	1.19 ± 0.05	0.58 ± 0.04	0.76 ± 0.10	0.74 ± 0.11	0.74 ± 0.002	0.89 ± 0.06	1.27 ± 0.01
PFOA	3.39 ± 0.12	3.29 ± 0.06	3.05 ± 0.16	3.20 ± 0.17	2.91 ± 0.09	3.42 ± 0.10	3.67 ± 0.12	3.36 ± 0.15	3.93 ± 0.002	4.71 ± 0.31	2.92 ± 0.06	3.04 ± 0.05	3.14 ± 0.31	3.34 ± 0.06	3.64 ± 0.12	4.26 ± 0.29
PFNA	0.27 ± 0.02	0.23 ± 0.01	0.20 ± 0.02	0.29 ± 0.02	0.13 ± 0.02	0.23 ± 0.004	0.28 ± 0.04	0.17 ± 0.001	0.26 ± 0.01	0.50 ± 0.02	0.20 ± 0.03	0.17 ± 0.07	0.26 ± 0.02	0.26 ± 0.02	0.24 ± 0.001	0.30 ± 0.02
PFDA	0.09 ± 0.02	0.07 ± 0.01	0.08 ± 0.03	0.14 ± 0.003	(0.03) ± 0.003	0.06 ± 0.002	0.09 ± 0.003	(0.03) ± 0.002	0.08 ± 0.003	0.15 ± 0.03	n.d.	(0.03) ± 0.01	0.06 ± 0.003	0.08 ± 0.01	0.10 ± 0.005	0.13 ± 0.03
PFUnDA	0.03 ± 0.001	0.03 ± 0.004	0.04 ± 0.02	0.07 ± 0.01	n.d.	(0.01) ± 0.0002	0.02 ± 0.01	n.d.	(0.01) ± 0.002	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
PFDoDA	n.d.	n.d.	n.d.	0.05 ± 0.002	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
FOSA	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.02 ± 0.001	0.07 ± 0.01	n.d.	0.02 ± 0.005	n.d.	0.03 ± 0.02	n.d.	n.d.
MeFBSA	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
MeFBSE	(0.16) ± 0.001	(0.10) ± 0.004	(0.10) ± 0.02	(0.22) ± 0.03	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
FDUEA	n.d.	n.d.	n.d.	0.03 ± 0.04	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
∑PFCs	**11.67 ± 0.60**	**10.98 ± 0.51**	**10.42 ± 0.45**	**11.50 ± 0.85**	**12.28 ± 0.88**	**10.92 ± 0.58**	**11.73 ± 0.47**	**11.80 ± 0.72**	**12.14 ± 0.57**	**22.50 ± 3.41**	**10.98 ± 0.93**	**11.89 ± 2.19**	**16.46 ± 2.53**	**18.49 ± 1.76**	**18.19 ± 2.25**	**23.74 ± 0.94**

[a] Values in brackets are between MDL and MQL and are not included in the sum concentrations; n.d. = not detected; [b] mean and standard deviation of a duplicate sample; [c] have to be considered as estimates, because no standards were available for this compound.

Table S5. Correlation coefficient of Pearson analysis of PFCs in the German Bight

Analyte		PFBS	PFPS	PFHxS	PFOS	PFPA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFUnDA	FOSA
PFBS	Pearson correlation	1.000	.219	.388**	.212	.495**	-.042	.247	.186	-.005	-.088	-.262	-.044
	N	48	30	48	48	43	48	48	48	48	41	24	23
PFPS	Pearson correlation	.219	1.000	.829**	.911**	.001	.591**	.594**	.839**	.827**	.536**	.091	.437
	N	30	30	30	30	27	30	30	30	30	26	19	18
PFHxS	Pearson correlation	.388**	.829**	1.000	.817**	.184	.260	.512**	.773**	.692**	.364*	-.186	.191
	N	48	30	48	48	43	48	48	48	48	41	24	23
PFOS	Pearson correlation	.212	.911**	.817**	1.000	.062	.509**	.474**	.788**	.849**	.509**	.100	.276
	N	48	30	48	48	43	48	48	48	48	41	24	23
PFPA	Pearson correlation	.495**	.001	.184	.062	1.000	.219	.304*	-.015	-.047	.030	-.064	.068
	N	43	27	43	43	43	43	43	43	43	38	22	21
PFHxA	Pearson correlation	-.042	.591**	.260	.509**	.219	1.000	.701**	.612**	.684**	.751**	.336	.655**
	N	48	30	48	48	43	48	48	48	48	41	24	23
PFHpA	Pearson correlation	.247	.594**	.512**	.474**	.304*	.701**	1.000	.768**	.703**	.866**	.181	.924**
	N	48	30	48	48	43	48	48	48	48	41	24	23
PFOA	Pearson correlation	.186	.839**	.773**	.788**	-.015	.612**	.768**	1.000	.868**	.774**	.046	.672**
	N	48	30	48	48	43	48	48	48	48	41	24	23
PFNA	Pearson correlation	-.005	.827**	.692**	.849**	-.047	.684**	.703**	.868**	1.000	.800**	.200	.625**
	N	48	30	48	48	43	48	48	48	48	41	24	23
PFDA	Pearson correlation	-.088	.536**	.364*	.509**	.030	.751**	.866**	.774**	.800**	1.000	.457*	.850**
	N	41	26	41	41	38	41	41	41	41	41	24	20
PFUnDA	Pearson correlation	-.262	.091	-.186	.100	-.064	.336	.181	.046	.200	.457*	1.000	.603*
	N	24	19	24	24	22	24	24	24	24	24	24	11
FOSA	Pearson correlation	-.044	.437	.191	.276	.068	.655**	.924**	.672**	.625**	.850**	.603*	1.000
	N	23	18	23	23	21	23	23	23	23	20	11	23

* Correlation is significant at the 0.05 level; ** correlation is significant at the 0.01 level.

Table S6. Locations, sampling time and water temperature of water sampling from cruises of the research vessels *Maria S. Merian* (15° W-52° W) and *Polarstern* (46° N-26° S) in the Atlantic Ocean

Station number	Sampling date	UTC time[a]	Latitude	Longitude	Water temperature [°C]
1	29.10.2007	11:45	N46° 17.257'	W06° 29.386'	17.3
2	29.10.2007	18:09	N45° 50.129'	W06° 34.129'	17.3
3	30.10.2007	9:48	N45° 50.786'	W06° 38.372'	17.4
4	30.10.2007	15:07	N45° 15.217'	W07° 28.793'	17.3
5	30.10.2007	19:54	N44° 32.711'	W08° 29.306'	17.3
6	31.10.2007	6:02	N42° 45.431'	W10° 09.153'	16.8
7	31.10.2007	13:58	N42° 09.180'	W10° 39.598'	17.5
8	31.10.2007	21:30	N40° 56.412'	W10° 53.255'	17.9
9	01.11.2007	6:05	N39° 21.663'	W11° 27.465'	18.5
10	01.11.2007	14:00	N37° 53.236'	W12° 00.314	19.9
11	01.11.2007	22:03	N36° 18.403'	W12° 34.709'	19.9
12	02.11.2007	6:04	N34° 51.085'	W13° 05.819'	21.1
13	02.11.2007	13:58	N33° 49.468'	W14° 11.388'	21.7
14	02.11.2007	21:57	N32° 21.020'	W14° 33.162'	21.9
15	03.11.2007	6:00	N30° 48.669'	W14° 28.321'	22.1
16	03.11.2007	20:41	N29° 26.399'	W15° 08.289'	22.4
17	04.11.2007	14:27	N27° 26.048'	W15° 51.313'	22.9
18	04.11.2007	21:05	N26° 40.036'	W17° 13.217'	23.6
19	05.11.2007	7:26	N25° 29.587'	W19° 17.573'	23.8
20	05.11.2007	18:38	N24° 41.732'	W20° 44.640'	24.0
21	06.11.2007	7:18	N22° 30.657'	W20° 51.172'	23.5
22	06.11.2007	17:31	N20° 09.986'	W20° 58.390'	23.9
23	07.11.2007	7:10	N17° 09.137'	W21° 07.505'	25.2
24	07.11.2007	18:30	N14° 37.361'	W21° 11.231'	27.4
25	08.11.2007	7:44	N11° 47.590'	W20° 26.967'	28.7
26	09.11.2007	6:45	N10° 11.867'	W20° 03.115'	28.7
27	09.11.2007	20:10	N08° 06.418'	W18° 50.586'	29.2
28	10.11.2007	6:07	N06° 41.328'	W17° 39.397'	29.0
29	10.11.2007	18:13	N05° 02.993'	W16° 17.380'	28.8
30	11.11.2007	6:14	N03° 30.652'	W15° 00.554'	28.1
31	12.11.2007	8:02	N02° 15.446'	W13° 20.527'	27.4
32	12.11.2007	18:13	N01° 13.523'	W11° 57.961'	26.2
33	13.11.2007	10:45	S00° 29.615'	W09° 40.469'	25.3
34	14.11.2007	14:30	S03° 59.562'	W06° 44.382'	25.7
35	15.11.2007	17:10	S07° 26.390'	W04° 08.697'	24.0
36	16.11.2007	13:36	S10° 38.548'	W01° 39.086'	22.4
37	17.11.2007	17:28	S13° 21.221'	E00° 37.067'	21.0
38	18.11.2007	12:38	S15° 49.494'	E02° 22.162'	19.8
39	19.11.2007	12:41	S19° 27.539'	E05° 12.842'	19.7
40	20.11.2007	11:28	S22° 59.804'	E08° 02.746'	18.9
41	21.11.2007	10:15	S25° 16.359'	E09° 38.236'	18.3
42	22.11.2007	6:36	S26° 29.940'	E10° 37.656'	17.9
A	26.04.2007	19:38	N47° 31.530'	W52° 01.455'	0.2
B	25.04.2007	15:40	N44° 18.728'	W50° 05.773'	3.7
C	27.04.2007	17:00	N47° 06.103'	W44° 57.691'	4.1
D	28.04.2007	16:15	N47° 01.698'	W43° 09.601'	4.9
E	29.04.2007	15:20	N46° 59.893'	W42° 54.675'	6.1
F	30.04.2007	15:00	N46° 58.500'	W41° 01.986'	13.8
G	24.04.2007	14:06	N42° 42.012'	W47° 30.021'	8.2
H	23.04.2007	20:00	N42° 06.548'	W45° 11.957'	16.3
I	22.04.2007	22:40	N42° 47.637'	W42° 19.648'	16.1
J	21.04.2007	17:10	N43° 49.982'	W38° 37.856'	15.6
K	20.04.2007	14:00	N44° 51.484'	W34° 59.425'	15.0
L	19.04.2007	12:35	N46° 59.028'	W31° 56.440'	13.6
M	18.04.2007	19:29	N46° 45.984'	W30° 32.736'	13.3
N	17.04.2007	23:30	N42° 41.516'	W27° 49.606'	14.8
O	17.04.2007	16:55	N41° 32.530'	W27° 05.618'	15.1
P	16.04.2007	21:15	N37° 42.467'	W24° 38.880'	16.5
Q	15.04.2007	21:45	N34° 06.428'	W20° 59.002'	18.8
R	15.04.2007	09:50	N32° 28.675'	W19° 22.527'	18.4

[a] UTC = universal time coordinated.

Table S7. Matrix spike recovery values of the IS and relative standard deviation (RSD) (%) for the dissolved and particulate phase determined at two different spiking levels (5 ng/L and 20 ng/L)

	Recovery [%]							
	Dissolved phase				Particulate phase			
	5 ng/L, n = 4		20 ng/L , n = 4		5 ng/L , n = 4		20 ng/L , n = 4	
IS	**Mean**	**RSD**	**Mean**	**RSD**	**Mean**	**RSD**	**Mean**	**RSD**
[$^{18}O_2$]-PFHxS	66	4.5	64	3.4	87	15.2	83	4.8
[$^{13}C_4$]-PFOS	57	9.9	55	5.7	98	8.1	102	7.9
[$^{13}C_4$]-PFOSi	67	4.9	61	3.5	79	11.7	72	0.7
[$^{13}C_2$]-PFHxA	54	4.5	53	6.1	124	9.2	110	6.7
[$^{13}C_4$]-PFOA	54	3.9	57	7.7	105	7.4	111	7.9
[$^{13}C_4$]-PFNA	67	14.5	57	4.9	109	12.8	103	17.0
[$^{13}C_4$]-PFDA	80	9.3	87	2.4	77	6.7	70	3.2
[$^{13}C_2$]-PFUnDA	59	14.5	60	2.6	110	1.0	99	8.2
[$^{13}C_2$]-PFDoDA	52	12.2	53	4.6	110	4.0	101	7.9
d_3-MeFOSA	24	21.3	27	6.7	52	2.9	50	3.5
d_5-EtFOSA	34	20.9	36	5.1	56	1.0	56	0.7
d_7-MeFOSE	35	17.6	37	4.3	68	5.5	69	7.2
d_9-EtFOSE	32	17.7	33	2.0	67	2.3	63	4.4
[$^{13}C_2$]-FHEA	69	10.9	76	13.0	95	9.7	83	9.2
[$^{13}C_2$]-FOEA	50	14.9	53	7.5	74	11.5	65	1.6
[$^{13}C_2$]-FDEA	56	13.9	55	4.1	116	9.5	99	4.6
[$^{13}C_2$]-FHUEA	90	4.0	87	4.3	105	13.0	92	9.8
[$^{13}C_2$]-FOUEA	65	8.1	62	2.9	76	11.7	64	4.1
[$^{13}C_2$]-FDUEA	54	17.5	54	4.0	115	5.3	98	8.0

Table S8. Individual PFC concentrations (pg/L) for cruises of the research vessels *Maria S. Merian* (15° W-52° W) and *Polarstern* (46° N-26° S) in the Atlantic Ocean [a]

Station number	PFBS	PFOS	FOSA	PFHxA	PFHpA	PFOA	PFNA	∑PFC
1	60	291	302	127	<5.9	229	107	1115
2	7.1	114	307	87	<5.9	209	100	824
3	16	40	97	82	<5.9	99	65	399
4	45	<10	183	83	<5.9	147	69	527
5	17	<10	143	88	<5.9	97	63	409
6	<1.6	<10	104	77	<5.9	115	73	368
7	14	<10	97	83	<5.9	94	65	352
8	<1.6	<10	71	84	<5.9	108	68	332
9	7.2	<10	32	86	<5.9	80	66	271
10	<1.6	<10	44	81	<5.9	88	52	266
11	34	<10	37	11	45	65	31	223
12	<1.6	<10	39	17	46	62	16	181
13	40	<10	<17	7	47	69	33	196
14	32	<10	45	12	49	72	23	232
15	29	<10	64	<5.7	43	20	13	170
16	31	<10	37	<5.7	47	47	29	191
17	30	<10	<17	<5.7	45	42	25	142
18	<1.6	<10	<17	<5.7	45	40	11	96
19	20	<10	<17	<5.7	43	77	13	153
20	<1.6	<10	67	<5.7	45	25	38	175
21	<1.6	<10	110	<5.7	<5.9	20	40	171
22	<1.6	<10	72	<5.7	8.5	<4.0	42	123
23	<1.6	<10	<17	<5.7	<5.9	<4.0	28	28
24	<1.6	<10	60	<5.7	<5.9	<4.0	29	89
25	<1.6	<10	<17	<5.7	9.7	87	29	126
26	<1.6	60	<17	<5.7	8.9	82	35	187
27	<1.6	<10	<17	<5.7	<5.9	65	30	95
28	<1.6	<10	52	<5.7	<5.9	<4.0	31	82
29	<1.6	<10	<17	<5.7	<5.9	<4.0	33	33
30	<1.6	<10	<17	<5.7	<5.9	<4.0	27	27
31	<1.6	<10	56	<5.7	<5.9	<4.0	<5.1	56
32	<1.6	<10	37	<5.7	<5.9	<4.0	<5.1	37
33	<1.6	<10	53	<5.7	<5.9	<4.0	<5.1	53
34	<1.6	<10	41	<5.7	<5.9	<4.0	<5.1	41
35	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
36	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
37	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
38	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
39	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
40	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
41	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
42	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
A	<1.6	<10	<17	<5.7	36	81	<5.1	116
B	<1.6	51	<17	<5.7	<5.9	26	<5.1	77
C	<1.6	<10	<17	<5.7	<5.9	40	<5.1	40
D	<1.6	<10	<17	<5.7	<5.9	35	<5.1	36
E	<1.6	<10	<17	<5.7	<5.9	<4.0	<5.1	n.a.
F	<1.6	<10	<17	<5.7	<5.9	<4.0	6.6	7
G	<1.6	<10	<17	<5.7	<5.9	73	<5.1	73
H	<1.6	<10	<17	<5.7	<5.9	52	<5.1	52
I	<1.6	<10	<17	<5.7	<5.9	87	<5.1	87
J	<1.6	69	<17	<5.7	<5.9	48	<5.1	117
K	<1.6	<10	<17	<5.7	<5.9	72	<5.1	72
L	<1.6	<10	<17	<5.7	<5.9	73	<5.1	73
M	<1.6	<10	<17	<5.7	104	<4.0	<5.1	104
N	<1.6	<10	<17	<5.7	80	<4.0	<5.1	80
O	<1.6	<10	<17	<5.7	14	82	<5.1	96
P	<1.6	<10	<17	<5.7	26	121	<5.1	147
Q	<1.6	<10	<17	51	<5.9	118	<5.1	169
R	<1.6	54	<17	51	<5.9	86	<5.1	191

[a] <x below the respective method quantification limit (MQL); n.a. = not available.

Table S9. Method blanks (blanks, n = 6), method detection limit (MDL, n = 3) and method quantification limit (MQL, n = 3) in ng/g wet weight

		Liver	Kidney	Lung	Heart	Blood	Brain	Muscle	Thyroid	Thymus	Blubber
PFBS	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0481	0.0107	0.0084	0.0139	0.0030	n.d.	0.0081	0.0319	0.0079	n.d.
	MQL	0.1602	0.0357	0.0279	0.0465	0.0101	n.d.	0.0271	0.1064	0.0263	n.d.
PFPS	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0766	0.0086	0.0101	0.0030	0.0152	n.d.	n.d.	n.d.	0.0100	n.d.
	MQL	0.2553	0.0288	0.0337	0.0101	0.0505	n.d.	n.d.	n.d.	0.0335	n.d.
PFHxS	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0549	0.0152	0.0410	0.0160	0.0616	0.0098	0.0104	0.0259	0.0280	0.0150
	MQL	0.1830	0.0507	0.1366	0.0534	0.2054	0.0326	0.0348	0.0865	0.0933	0.0498
PFHpS	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.1253	0.0325	0.1492	0.0265	0.0174	0.0283	0.0136	0.0452	0.0433	0.0056
	MQL	0.4177	0.1084	0.4973	0.0882	0.0580	0.0944	0.0452	0.1506	0.1445	0.0187
PFOS	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	2.5301	0.5026	1.2277	1.0051	1.9227	0.1519	0.3031	0.0440	1.1397	1.9240
	MQL	8.4337	1.6754	4.0923	3.3504	6.4090	0.5063	1.0102	0.1466	3.7990	6.4133
PFNS	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0359	0.0075	0.0094	n.d.	0.0081	n.d.	n.d.	0.0901	0.0088	n.d.
	MQL	0.1195	0.0250	0.0314	n.d.	0.0271	n.d.	n.d.	0.3003	0.0293	n.d.
PFDS	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0336	0.0256	0.0385	0.0122	0.0141	0.0042	n.d.	0.0173	0.0081	0.0149
	MQL	0.1119	0.0855	0.1282	0.0407	0.0468	0.0139	n.d.	0.0575	0.0269	0.0498
PFOSi	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0131	n.d.	0.0051	n.d.	0.0012	n.d.	n.d.	n.d.	n.d.	n.d.
	MQL	0.0437	n.d.	0.0171	n.d.	0.0039	n.d.	n.d.	n.d.	n.d.	n.d.
PFOA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0260	0.0094	0.0245	0.0067	0.0144	0.0021	0.0023	0.0040	0.0102	0.0007
	MQL	0.0866	0.0315	0.0818	0.0223	0.0479	0.0070	0.0076	0.0134	0.0339	0.0024
PFNA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0424	0.0151	0.0099	0.0094	0.0284	0.0127	0.0114	0.0265	0.0254	0.0050
	MQL	0.1414	0.0505	0.0329	0.0313	0.0946	0.0423	0.0381	0.0884	0.0847	0.0167
PFDA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0477	0.0096	0.0211	0.0104	0.0151	0.0332	0.0250	0.0177	0.0206	0.0054
	MQL	0.1590	0.0321	0.0704	0.0346	0.0503	0.1108	0.0833	0.0590	0.0685	0.0181
PFUnDA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0369	0.0120	0.0288	0.0143	0.0203	0.0254	0.0065	0.0062	0.0230	0.0032
	MQL	0.1231	0.0401	0.0960	0.0476	0.0675	0.0848	0.0216	0.0208	0.0767	0.0107
PFDoDA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0460	0.0315	0.1353	0.0165	0.0100	0.0495	0.0012	0.0725	0.0117	0.0066
	MQL	0.1534	0.1051	0.4511	0.0550	0.0333	0.1650	0.0040	0.2416	0.0390	0.0220
PFTriDA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0721	0.0300	0.1588	0.0792	0.0336	0.0602	0.0206	0.0105	0.0161	0.0107
	MQL	0.2405	0.1002	0.5293	0.2639	0.1121	0.2007	0.0685	0.0351	0.0536	0.0357
PFTeDA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.0437	0.0304	0.0335	0.0149	0.0190	0.0173	n.d.	0.0159	0.0181	n.d.
	MQL	0.1457	0.1013	0.1115	0.0497	0.0633	0.0578	n.d.	0.0530	0.0604	n.d.
PFPeDA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	n.d.	n.d.	0.1577	n.d.	0.0129	n.d.	n.d.	n.d.	n.d.	n.d.
	MQL	n.d.	n.d.	0.5256	n.d.	0.0431	n.d.	n.d.	n.d.	n.d.	n.d.
FOSA	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	0.1327	0.0557	0.0487	0.0256	0.1188	0.0182	0.0173	0.0033	0.0391	0.0033
	MQL	0.4424	0.1857	0.1624	0.0854	0.3960	0.0606	0.0576	0.0111	0.1303	0.0109
MeFBSE	blanks	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL	<MQL
	MDL	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.0191	n.d.	0.4010
	MQL	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	0.0638	n.d.	1.3368

Table S10. Absolute (subtraction of background concentration) as well as relative (IS and subtraction of background concentration) recovery values (%) of a seal liver spiked with 10 ng absolute (100 µL of a 0.1 µg/mL solution, n = 3) and allocation of IS for individual PFCs

	Absolute recovery [%] [a]		Relative recovery [%] [a]		Allocation of the IS
Analyte	**mean**	**RSD**	**mean**	**RSD**	
PFBS	48	3.7	70	3.7	
PFHxS	51	7.7	75	7.7	$[^{18}O_2]$-PFHxS
PFHpS	78	6.3	115	6.3	
PFOS	52	5.0	74	5.0	
PFDS	46	19.9	65	19.9	$[^{13}C_4]$-PFOS
6:2 FTS	83	13.3	116	13.3	
PFHxSi	90	12.5	109	12.5	
PFOSi	67	4.1	81	4.1	$[^{13}C_4]$-PFOSi
PFDSi	61	6.8	74	6.8	
PFBA	88	4.0	88	4.0	$[^{13}C_4]$-PFBA
PFPA	64	3.5	92	3.5	
PFHxA	42	3.6	60	3.6	$[^{13}C_2]$-PFHxA
PFHpA	80	11.7	114	11.7	
PFOA	77	2.5	87	2.5	$[^{13}C_4]$-PFOA
PFNA	54	8.3	120	8.3	$[^{13}C_5]$-PFNA
PFDA	94	8.1	122	8.1	$[^{13}C_2]$-PFDA
PFUnDA	111	21.8	125	21.8	$[^{13}C_2]$-PFUnDA
PFDoDA	129	5.8	127	5.8	
PFTriDA	93	4.0	92	4.0	
PFTeDA	122	8.2	120	8.2	$[^{13}C_2]$-PFDoDA
PFHxDA	57	3.0	56	3.0	
PFOcDA	65	2.5	64	2.5	
3,7m$_2$-PFOA	108	9.1	121	9.1	$[^{13}C_4]$-PFOA
MeFBSA	52	10.5	109	10.5	d_3-N-MeFOSA
FOSA	44	18.8	62	18.8	$[^{13}C_4]$-PFOS
MeFOSA	53	7.2	112	7.2	d_3-N-MeFOSA
EtFOSA	48	5.4	92	5.4	d_5-N-EtFOSA
MeFBSE	83	3.1	98	3.1	d_7-N-MeFOSE
MeFOSE	84	8.0	99	8.0	
EtFOSE	72	11.5	135	11.5	d_9-N-EtFOSE
FHEA	60	7.2	73	7.2	$[^{13}C_2]$-FHEA
FOEA	64	7.2	82	7.2	$[^{13}C_2]$-FOEA
FDEA	82	0.8	85	0.8	$[^{13}C_2]$-FDEA
FHUEA	98	4.7	110	4.7	$[^{13}C_2]$-FHUEA
FOUEA	58	3.1	108	3.1	$[^{13}C_2]$-FOUEA
FDUEA	111	2.5	105	2.5	$[^{13}C_2]$-FDUEA

[a] Mean recoveries are given with relative standard deviations (RSD).

Table S11. Matrix effect of PFCs in liver, kidney, lung, heart, blood, brain and muscle of harbor seals determined by analysis of a fortified extract with a 10 ng absulute PFC-mix (100 µL of a 0.1 µg/mL standard solution) [a]

	Liver	Kidney	Lung	Heart	Blood	Brain	Muscle	Mean ± RSD
PFBS	0.99	0.97	0.95	0.99	0.95	0.97	0.96	0.97 ± 0.01
PFHxS	1.01	0.94	0.81	1.02	0.90	0.82	1.01	0.93 ± 0.09
PFHpS	0.97	0.75	0.85	1.04	0.75	0.91	0.90	0.88 ± 0.11
PFOS	1.03	0.84	1.03	1.09	0.74	0.87	1.02	0.95 ± 0.13
PFDS	1.02	0.94	0.67	1.14	0.83	0.88	1.03	0.93 ± 0.15
6:2 FTS	0.92	1.06	0.82	0.96	0.90	1.06	1.10	0.97 ± 0.10
PFHxSi	1.03	1.00	0.80	0.90	0.91	0.86	1.06	0.94 ± 0.10
PFOSi	0.83	0.81	0.95	0.99	0.91	0.89	1.04	0.92 ± 0.08
PFDSi	0.92	0.94	0.87	1.04	0.90	0.95	1.08	0.96 ± 0.08
PFBA	1.03	0.95	0.95	1.08	0.88	0.98	1.00	0.98 ± 0.06
PFPA	0.94	0.98	0.85	1.22	0.82	0.93	1.08	0.98 ± 0.14
PFHxA	0.89	0.99	0.98	1.00	0.88	0.90	1.08	0.96 ± 0.07
PFHpA	0.86	0.98	0.90	0.98	1.04	0.96	1.01	0.96 ± 0.06
PFOA	0.92	0.91	0.87	0.99	0.86	0.90	1.03	0.93 ± 0.06
PFNA	0.97	0.91	0.83	0.91	1.03	0.82	1.04	0.93 ± 0.09
PFDA	1.03	1.00	0.86	1.13	0.88	0.81	1.06	0.97 ± 0.12
PFUnDA	0.97	0.98	0.85	1.03	0.91	1.02	1.03	0.97 ± 0.07
PFDoDA	0.94	0.96	0.89	1.00	1.02	1.06	1.02	0.98 ± 0.06
PFTriDA	0.85	0.90	0.89	1.00	0.97	0.95	1.05	0.94 ± 0.07
PFTeDA	0.97	0.83	0.86	1.01	1.01	0.81	0.96	0.92 ± 0.09
PFHxDA	0.50	0.24	0.43	0.37	0.90	0.44	0.26	0.45 ± 0.22
PFOcDA	0.38	0.15	0.11	0.12	1.01	0.12	0.22	0.30 ± 0.33
3,7m_2-PFOA	1.04	0.99	0.99	0.97	0.93	0.97	1.14	1.01 ± 0.07
MeFBSA	0.96	0.96	0.91	0.99	0.94	0.94	1.00	0.96 ± 0.03
FOSA	1.05	0.92	0.94	1.00	1.03	1.02	1.02	1.00 ± 0.05
MeFOSA	0.95	0.94	0.94	0.96	0.97	0.94	0.99	0.96 ± 0.02
EtFOSA	0.90	0.87	0.95	0.94	0.90	0.91	1.05	0.93 ± 0.06
MeFBSE	1.01	0.93	0.92	1.03	0.93	0.93	1.02	0.96 ± 0.05
MeFOSE	0.92	0.98	0.91	0.94	0.96	0.94	1.04	0.95 ± 0.05
EtFOSE	0.94	0.67	0.85	0.97	0.89	0.86	0.97	0.88 ± 0.10
FHEA	0.91	1.01	0.96	1.04	0.90	0.95	1.01	0.97 ± 0.05
FOEA	0.98	0.90	0.90	0.93	0.90	0.97	1.12	0.96 ± 0.08
FDEA	0.92	0.98	0.90	1.05	0.95	1.03	1.00	0.98 ± 0.05
FHUEA	0.89	0.97	0.94	0.98	0.93	0.93	1.02	0.95 ± 0.04
FOUEA	0.94	0.96	0.94	0.96	0.87	0.98	1.06	0.96 ± 0.05
FDUEA	0.91	0.92	0.89	1.04	0.96	0.93	0.96	0.94 ± 0.05

[a] Calculation of the matrix effect: Matrix effect = (response $_{\text{fortified extract}}$ – response $_{\text{non-fortified extract}}$) / response $_{\text{solvent based standard}}$; value > 1: Signal enhancement; value <1: Signal suppression;
RSD = relative standard deviations.

Table S12. Sample collection year, identification number (ID), age, total weight, liver weight, sex, nutritional status, general health status and lesions in the liver of harbor seals collected from the German Bight

Year	ID	Age [month]	Total weight [kg]	Liver weight [g]	Sex [a]	Nutritional status	Gerneral Health status	Lesions in the liver
1988	15985	7-19	13.3	n.d.	m	emaciated	moderate	none
	15978	7-19	16.5	n.d.	m	emaciated	moderate	none
1996	40	<7	8.5	329	f	emaciated	poor	none
	61	<7	10.0	507	m	good	good	none
1999	1309	7-19	21.6	726	f	emaciated	poor	moderate fatty liver
	1311	>19	76.6	2429	f	good	good	none
	1312	>19	80.6	2299	m	moderate	moderate	none
	1400	>19	17.2	681	m	moderate	poor	none
	1295	<7	4.2	n.d.	f	emaciated	poor	none
2000	1490	>19	70.6	n.d.	m	moderate	good	none
	1543	7-19	16.0	879	f	emaciated	poor	mild hepatitis
	1571	>19	57.2	2967	m	good	good	none
	1655	7-19	22.2	n.d.	f	good	good	severe fatty liver
	1668	7-19	25.2	n.d.	m	moderate	moderate	none
	1588	<7	7.4	276	m	emaciated	poor	none
2001	1672	7-19	15.8	512	m	emaciated	poor	none
	1675	7-19	19.0	1008	m	moderate	poor	mild hepatitis
	1737	7-19	20.2	781	m	moderate	poor	mild appearance of parasitic structures
	1741	>19	55.2	2100	f	moderate	moderate	mild fatty liver
	1783	>19	46.2	1446	f	moderate	poor	none
	1742	<7	11.2	430	m	moderate	good	moderate fatty liver
	1759	<7	11.4	n.d.	f	moderate	poor	none
2002	2076	7-19	32.5	1856	m	moderate	poor	mild hepatitis
	2078	>19	41.6	2255	m	good	moderate	none
	2149	>19	45.2	2050	f	moderate	poor	none
	2150	>19	47.0	1898	m	moderate	poor	none
	2169	>19	53.4	2252	m	good	moderate	none
	2071	<7	9.6	594	f	moderate	poor	none
2003	2357	7-19	17.2	891	f	good	poor	none
	2435	7-19	30.6	865	m	good	good	none
	2166	<7	9.8	500	f	moderate	poor	moderate fatty liver
	2168	<7	10.2	325	f	moderate	poor	none
	2355	<7	7.0	282	f	emaciated	moderate	mild hepatitis
2004	2686	7-19	18.2	507	m	moderate	good	moderate periportal fibrosis
2005	2932	7-19	17.2	669	f	emaciated	good	none
	2937	7-19	20.8	801	m	moderate	poor	none
	3087	7-19	26.8	1381	m	good	poor	moderate hepatitis
	3212	>19	47.0	1539	f	good	moderate	none
	3090	<7	25.5	906	f	good	good	mild bile duct proliferation

(Continued)

Year	ID	Age [month]	Total weight [kg]	Liver weight [g]	Sex [a]	Nutritional status	Gerneral Health status	Lesions in the liver
2006	3242	7-19	19.4	842	m	emaciated	poor	none
	3452	>19	102.6	2774	f	good	good	none
	3510	>19	49.6	1346	m	good	good	none
	3405	<7	9.4	308	f	good	good	none
	3416	<7	7.0	303	m	emaciated	poor	none
	3420	<7	7.2	343	m	moderate	poor	none
	3461	<7	7.4	268	m	emaciated	poor	none
	3463	<7	7.8	251	m	emaciated	poor	mild haemosiderose
2007	3615	7-19	17.3	400	m	moderate	poor	severe hepatitis
	3655	>19	69.5	2507	f	good	poor	none
	3671	7-19	15.0	441	m	moderate	poor	none
	3675	7-19	18.8	711	m	moderate	poor	severe hepatitis
	3723	7-19	17.4	726	m	emaciated	poor	none
	4143	7-19	17.4	725	m	moderate	n.d.	n.d.
	4144	7-19	17.8	856	f	good	n.d.	n.d.
	4145	7-19	17.4	974	m	moderate	n.d.	n.d.
	4183	7-19	21.4	1196	m	moderate	n.d.	n.d.
	4184	7-19	16.0	n.d.	m	emaciated	poor	none
	3744	<7	11.4	750	m	good	moderate	none
	3757	<7	7.6	176	f	emaciated	poor	severe haemosiderose
	3821	<7	7.9	236	f	emaciated	poor	none
2008	4190	7-19	15.8	673	f	moderate	poor	severe hepatitis
	4191	7-19	22.9	1028	m	moderate	n.d.	n.d.
	4192	7-19	21.2	1419	m	moderate	poor	severe hepatitis

n.d. = not determined; [a] m= male, f = female.

Table S13. Quality assurance of the extraction of harbor seal livers: Instrument detection limits (IDL), method quantification limits (MQL), recoveries of spiked liver extracts, matrix effect and allocation of the IS

Analyte	IDL [pg absolute]	MQL [ng/g ww]	Recovery [%] [a]	Matrix effect [b]	Allocation of the IS
PFBS	0.191	0.1602	70 ± 3.7	0.99	[$^{18}O_2$]-PFHxS
PFPS [c]	- [c]	0.255	- [c]	- [c]	
PFHxS	0.220	0.183	75 ± 7.7	1.01	
PFHpS	0.199	0.418	115 ± 6.3	0.97	
PFOS	0.264	8.434	74 ± 5.0	1.03	[$^{13}C_4$]-PFOS
PFNS [c]	- [c]	0.120	- [c]	- [c]	
PFDS	0.156	0.112	65 ± 19.9	1.02	
6:2 FTS	3.789	n.d.	116 ± 13.3	0.92	
PFHxSi	0.319	n.d.	109 ± 12.5	1.03	[$^{13}C_4$]-PFOSi
PFOSi	0.221	0.044	81 ± 4.1	0.83	
PFDSi	0.307	n.d.	74 ± 6.8	0.92	
PFBA	1.545	n.d.	88 ± 4.0	1.03	[$^{13}C_4$]-PFBA
PFPA	0.420	n.d.	92 ± 3.5	0.94	[$^{13}C_2$]-PFHxA
PFHxA	0.270	n.d.	60 ± 3.6	0.89	
PFHpA	0.258	n.d.	114 ± 11.7	0.86	
PFOA	0.278	0.087	87 ± 2.5	0.92	[$^{13}C_4$]-PFOA
PFNA	0.296	0.141	120 ± 8.3	0.97	[$^{13}C_5$]-PFNA
PFDA	0.444	0.159	122 ± 8.1	1.03	[$^{13}C_2$]-PFDA
PFUnDA	0.644	0.123	125 ± 21.8	0.97	[$^{13}C_2$]-PFUnDA
PFDoDA	0.381	0.153	127 ± 5.8	0.94	[$^{13}C_2$]-PFDoDA
PFTriDA	0.510	0.240	92 ± 4.0	0.85	
PFTeDA	0.752	0.146	120 ± 8.2	0.97	
PFPDA [c]	- [c]	0.043	- [c]	- [c]	
PFHxDA	1.561	n.d.	56 ± 3.0	0.50	
PFHpDA [c]	- [c]	n.d.	- [c]	- [c]	
PFOcDA	1.160	n.d.	64 ± 2.5	0.38	
3,7m$_2$-PFOA	0.801	n.d.	121 ± 9.1	1.04	[$^{13}C_4$]-PFOA
MeFBSA	3.263	n.d.	109 ± 10.5	0.96	d_3-N-MeFOSA
FOSA	0.941	0.442	62 ± 18.8	1.05	[$^{13}C_4$]-PFOS
MeFOSA	1.239	n.d.	112 ± 7.2	0.95	d_3-N-MeFOSA
EtFOSA	1.205	n.d.	92 ± 5.4	0.90	d_5-N-EtFOSA
MeFBSE	0.598	n.d.	98 ± 3.1	1.01	d_7-N-MeFOSE
MeFOSE	0.803	n.d.	99 ± 8.0	0.92	
EtFOSE	0.646	n.d.	135 ± 11.5	0.94	d_9-N-EtFOSE
FHEA	3.918	n.d.	73 ± 7.2	0.91	[$^{13}C_2$]-FHEA
FOEA	2.520	n.d.	82 ± 7.2	0.98	[$^{13}C_2$]-FOEA
FDEA	5.901	n.d.	85 ± 0.8	0.92	[$^{13}C_2$]-FDEA
FHUEA	1.252	n.d.	110 ± 4.7	0.89	[$^{13}C_2$]-FHUEA
FOUEA	0.709	n.d.	108 ± 3.1	0.94	[$^{13}C_2$]-FOUEA
FDUEA	1.031	n.d.	105 ± 2.5	0.91	[$^{13}C_2$]-FDUEA

n.d. = not detected; [a] recovery values (%) of spiked seal liver extracts with 10 ng absolute (100 µL of a 0.1 µg/mL solution, n = 3) are given with relative standard deviations (RSD); [b] calculation of the matrix effect by analysis of a fortified extract with a 10 ng absulute PFC-mix (100 µL of a 0.1 µg/mL standard solution): Matrix effect = (response $_{\text{fortified extract}}$ − response $_{\text{non-fortified extract}}$) / response $_{\text{solvent based standard}}$; value >1: Signal enhancement; value <1: Signal suppression; [c] can not be calculated, because no standards were available for this compound.

Table S14. Correlation coefficient of Pearson analysis of all detected PFCs in ≥ 7 month old harbor seals from 1988 to 2008

		PFBS	PFPS	PFHxS	PFHpS	PFOS	PFNS	PFDS	PFOSi	PFOA	PFNA	PFDA	PFUnDA	PFDoDA	PFTriDA	FOSA
PFBS	Pearson correlation	1.000	.030	.647**	.716**	.721**	.171	.080	-.038	.797**	.580**	.523**	.428*	.320	.189	.048
	N	24	23	24	23	24	21	17	17	17	24	24	24	23	23	24
PFPS	Pearson correlation	.030	1.000	.119	.087	.213	-.077	.097	.019	.161	-.001	-.053	-.048	.044	.208	.121
	N	23	43	42	41	43	31	26	25	28	43	43	42	40	39	42
PFHxS	Pearson correlation	.647**	.119	1.000	.810**	.585**	-.058	-.184	.143	.679**	.405**	.371*	.262	.227	.189	.150
	N	24	42	43	42	43	32	26	24	28	43	43	42	40	39	42
PFHpS	Pearson correlation	.716**	.087	.810**	1.000	.692**	.140	-.058	.138	.748**	.580**	.637**	.566**	.396*	.262	.119
	N	23	41	42	42	42	31	25	23	28	42	42	41	39	38	41
PFOS	Pearson correlation	.721**	.213	.585**	.692**	1.000	.509**	.374	.120	.607**	.633**	.571**	.549**	.582**	.425**	.209
	N	24	43	43	42	44	32	26	25	28	44	44	43	41	40	43
PFNS	Pearson correlation	.171	-.077	-.058	.140	.509**	1.000	.887**	-.166	-.086	.417*	.579**	.666**	.599**	.552**	-.126
	N	21	31	32	31	32	32	25	22	23	32	32	32	32	32	32
PFDS	Pearson correlation	.080	.097	-.184	-.058	.374	.887**	1.000	-.318	-.137	.251	.314	.426*	.542**	.560**	-.251
	N	17	26	26	25	26	25	26	17	20	26	26	26	26	26	26
PFOSi	Pearson correlation	-.038	.019	.143	.138	.120	-.166	-.318	1.000	.047	-.219	-.099	.014	.030	-.113	.883**
	N	17	25	24	23	25	22	17	25	18	25	25	25	25	25	25
PFOA	Pearson correlation	.797**	.161	.679**	.748**	.607**	-.086	-.137	.047	1.000	.621**	.367	.159	.190	.116	.091
	N	17	28	28	28	28	23	20	18	28	28	28	27	27	26	28
PFNA	Pearson correlation	.580**	-.001	.405**	.580**	.633**	.417*	.251	-.219	.621**	1.000	.815**	.701**	.606**	.353*	-.164
	N	24	43	43	42	44	32	26	25	28	44	44	43	41	40	43
PFDA	Pearson correlation	.523**	-.053	.371*	.637**	.571**	.579**	.314	-.099	.367	.815**	1.000	.922**	.725**	.445**	-.079
	N	24	43	43	42	44	32	26	25	28	44	44	43	41	40	43
PFUnDA	Pearson correlation	.428*	-.048	.262	.566**	.549**	.666**	.426*	.014	.159	.701**	.922**	1.000	.858**	.652**	.000
	N	24	42	42	41	43	32	26	25	27	43	43	43	40	40	42
PFDoDA	Pearson correlation	.320	.044	.227	.396*	.582**	.599**	.542**	.030	.190	.606**	.725**	.858**	1.000	.815**	-.048
	N	23	40	40	39	41	32	26	25	27	41	41	40	41	40	41
PFTriDA	Pearson correlation	.189	.208	.189	.262	.425**	.552**	.560**	-.113	.116	.353*	.445**	.652**	.815**	1.000	-.128
	N	23	39	39	38	40	32	26	25	26	40	40	40	40	40	40
FOSA	Pearson correlation	.048	.121	.150	.119	.209	-.126	-.251	.883**	.091	-.164	-.079	.000	-.048	-.128	1.000
	N	24	42	42	41	43	32	26	25	28	43	43	42	41	40	43

* Correlation is significant at the 0.05 level; ** correlation is significant at the 0.01 level.

Table S15. Sampling locations, time, turbidity, oxygen content, pH-value, conductibility, water temperature, salinity, and river discharge at the sampling day

Location	Sampling time [UTC][a]	Latitude/ longitude	Turbidity [FNU][b]	Oxygen content [mg/L]	pH value	Conductivity [mS/cm]	Water temperature [°C]	Salinity [psu][c]	River discharge [m^3/s]
1	16.08.2006 11:50	N54.157/ E7.938	0.18	12.2	8.1	43.40	19.4	31.77	n.a.
2	16.08.2006 12:16	N54.113/ E8.050	0.18	12.2	8.1	43.12	19.6	31.37	n.a.
3	16.08.2006 13:05	N54.030/ E8.266	0.18	14.0	8.1	41.72	19.2	30.55	n.a.
4	16.08.2006 13:34	N53.982/ E8.396	0.18	14.2	8.1	41.91	19.1	30.75	n.a.
5	16.08.2006 14:04	N53.962/ E8.578	0.21	13.7	8.0	37.08	19.2	26.80	n.a.
6	16.08.2006 14:24	N53.914/ E8.673	0.53	13.1	7.9	31.61	19.1	22.49	n.a.
7	17.08.2006 05:36	N53.859/ E8.737	0.33	9.8	7.9	29.68	19.3	20.93	916
8	17.08.2006 05:48	N53.839/ E8.787	0.47	9.8	7.7	25.05	19.1	17.45	894
9	17.08.2006 06:24	N53.847/ E8.969	0.36	10.8	7.8	19.00	19.7	12.72	879
10	17.08.2006 06:50	N53.873/ E9.094	0.36	11.0	7.8	14.97	20.2	9.689	866
11	17.08.2006 07:00	N53.878/ E9.142	0.33	11.0	7.7	10.45	20.6	6.52	866
12	17.08.2006 07:53	N53.823/ E9.374	0.42	11.3	7.6	3.17	21.3	1.79	843
13	17.08.2006 08:05	N53.795/ E9.396	0.47	11.4	7.6	2.33	21.3	1.30	819
14	17.08.2006 08:21	N53.756/ E9.421	0.47	11.1	7.5	1.61	21.2	0.88	819
15	17.08.2006 09:14	N53.631/ E9.528	0.33	9.5	7.3	1.19	21.4	0.640	818
16	17.08.2006 10:34	N53.551/ E9.818	0.24	6.1	7.1	0.96	20.6	0.52	817
17	17.08.2006 11:01	N53.541/ E9.922	0.33	9.3	7.3	0.82	20.0	0.45	817
18	17.08.2006 11:21	N53.537/ E9.994	0.27	12.9	7.7	0.71	19.8	0.39	810
19	17.08.2006 12:39	N53.462/ E10.065	0.50	16.7	7.9	0.50	19.8	0.27	803
20	17.08.2006 13:54	N53.397/ E10.170	0.50	17.5	7.9	0.54	19.8	0.29	796
21	17.08.2006 14:15	N53.395/ E10.234	0.50	17.8	8.0	0.53	20.0	0.28	788
22	18.08.2006 08:55	N53.399/ E10.429	0.39	16.2	7.9	0.54	19.8	0.29	737
23	18.08.2006 08:05	N53.377/ E10.491	0.36	15.7	7.9	0.53	19.8	0.29	736
24	18.08.2006 07:10	N53.370/ E10.554	0.36	15.0	7.9	0.52	19.7	0.28	735

[a] UTC = universal time coordinated; [b] FNU = formazin nephelometric unit; [c] psu = practical salinity unit; n.a. = not available.

Table S16. Locations, time and standard parameters of sampling for the river Elbe survey on 10th June 2007

Sampling number	Latitude/ Longitude	Time	pH value	Temperature [°C]	Conductivity [mS/cm]	Discharge volume [m³/s]	Suspended particulate matter [mg/L]	Dissolved organic carbon [mg/L]
15	53°51.78/ 8°43.80	10:50	8.3	20.6	31.6	474	78.9	6.1
14	53°50.83/ 8°45.65	11:00	8.3	19.9	32.7	463	79.9	4.4
13	53°50.32/ 8°53.20	11:33	8.2	20.9	23.0	459	63.1	8.1
12	53°50.77/ 8°57.85	11:53	8.1	21.2	17.5	455	37.1	6.9
11	53°53.23/ 9°11.13	12:55	7.9	21.3	8.7	448	27.4	7.8
10	53°49.44/ 9°22.16	14:00	7.9	21.9	1.8	436	120.5	6.9
9	53°47.42/ 9°23.96	14:16	7.8	21.8	1.4	423	84.0	5.7
8	53°43.65/ 9°27.80	14:51	7.8	22.6	1.2	423	65.7	8.4
7	53°38.02/ 9°31.57	15:37	7.7	22.8	1.1	423	43.0	6.3
6	53°35.89/ 9°35.59	15:59	7.6	23.1	1.0	423	43.0	6.7
5	53°33.91/ 9°41.40	16:28	7.7	23.1	0.9	423	41.2	7.9
4	53°32.44/ 9°55.07	17:27	8.0	23.8	0.9	423	21.4	7.0
3	53°32.62/ 9°57.94	17:39	8.1	24.0	0.9	419	26.3	8.2
2	53°31.77/ 10°01.00	17:57	8.6	25.2	0.9	419	51.1	8.8
1	53°26.93/ 10°05.28	18:44	9.0	25.9	0.9	416	41.2	8.0

Table S17. Locations, sampling time, water temperature and salinity of water sampling in the German Bight (1-22), Baltic Sea (A-R) and North and Norwegian Sea (I-VI) in 2007

Station number	Sampling date	UTC time [a]	Latitude	Longitude	Water temperature [°C]	Salinity [psu]
1	02.11.2007	08:40	55 55.08 N	03 20.82 E	12.3	34.9
2	02.11.2007	14:40	55 30.07 N	04 00.56 E	12.4	34.9
3	02.11.2007	12:20	55 45.98 N	04 15.09 E	12.4	34.9
4	02.11.2007	20:25	54 59.99 N	05 29.96 E	13.3	34.9
5	03.11.2007	06:55	55 22.98 N	06 14.96 E	13.4	34.6
6	01.11.2007	19:20	55 00.02 N	06 19.42 E	13.5	34.7
7	01.11.2007	16:20	54 40.92 N	06 46.98 E	13.5	34.2
8	04.11.2007	15:10	54 20.07 N	05 39.93 E	13.6	34.4
9	04.11.2007	11:20	53 56.43 N	06 11.99 E	12.8	33.2
10	04.11.2007	17:55	54 10.02 N	06 20.50 E	13.1	33.6
11	04.11.2007	08:45	53 40.46 N	06 24.90 E	11.9	31.8
12	04.11.2007	21:05	53 56.54 N	06 46.99 E	12.4	32.3
13	31.10.2007	16:15	54 10.78 N	07 26.00 E	13.4	33.4
14	03.11.2007	12:10	54 59.91 N	07 30.01 E	12.6	32.1
15	01.11.2007	12:40	54 40.01 N	07 30.16 E	12.6	31.4
16	01.11.2007	10:15	54 39 87 N	07 49.94 E	12.1	30.5
17	03.11.2007	17:05	54 59.93 N	08 15.07 E	11.0	30.2
18	31.10.2007	09:25	53 59.85 N	08 06.32 E	11.8	29.3
19	31.10.2007	11:30	54 13.31 N	08 22.55 E	10.4	26.3
20	30.10.2007	17:55	53 52.68 N	08 43.28 E	10.3	n.a.
21	30.10.2007	13:05	53 37.01 N	09 32.84 E	9.5	n.a.
22	30.10.2007	10:45	53 32.17 N	10 00.82 E	n.a.	n.a.
A	10.12.2007	15:52	54 49.64 N	09 52.17 E	7.1	18.4
B	10.12.2007	11:17	54 27.28 N	09 54.37 E	6.3	17.4
C	10.12.2007	13:54	54 38.10 N	10 02.70 E	6.7	17.8
D	11.12.2007	13:24	54 49.56 N	10 10.51 E	6.6	17.9
E	10.12.2007	8:50	54 24.24 N	10 12.61 E	6.6	16.4
F	15.12.2007	9:07	54 20.21 N	10 40.43 E	5.6	16.1
G	15.12.2007	10:34	54 29.66 N	10 43.67 E	5.8	17.2
H	14.12.2007	14:45	54 35.53 N	10 49.88 E	5.8	14.9
I	14.12.2007	13:26	54 44.48 N	10 56.24 E	5.9	14.2
J	16.12.2007	14:13	54 32.13 N	11 11.14 E	5.4	13.7
K	16.12.2007	15:29	54 40.29 N	11 17.28 E	5.2	12.0
L	19.12.2007	8:22	53 59.40 N	10 54.60 E	4.9	14.2
M	19.12.2007	11:50	54 06.67 N	11 05.98 E	5.5	14.6
N	19.12.2007	13:53	54 13.33 N	11 16.57 E	5.9	11.9
O	17.12.2007	11:58	54 21.06 N	11 21.04 E	5.5	14.1
P	17.12.2007	10:10	54 31.74 N	11 29.75 E	5.8	10.2
Q	17.12.2007	15:46	53 58.37 N	11 18.74 E	4.8	14.0
R	17.12.2007	13:44	54 09.15 N	11 31.56 E	5.6	13.9
I	14.08.2007	15:10	66 43.82 N	01 08.56 E	12.2	n.a.
II	15.08.2007	06:45	64 34.62 N	00 25.22 E	12.2	n.a.
III	16.08.2007	15:10	59 54.85 N	04 29.41 E	13.8	n.a.
IV	17.08.2007	07:10	57 59.08 N	06 35.97 E	17.0	n.a.
V	17.08.2007	10:40	57 50.60 N	07 46.00 E	16.7	n.a.
VI	17.08.2007	14:10	58 04.91 N	08 51.54 E	16.6	n.a.

[a] UTC = universal time coordinated; psu = practical salinity unit; n.a. = not available.

Table S18. Sampling location, sampling date and basic parameters of sediment core A from Tokyo Bay, Japan

Sampling date	**10. May 2008**					
GPS position	**N 35°34'60''/ E 139°55'01''**					
Water depth (m)	**12**					
Bottom water temperature	**15.2**					
Depth (cm)	**pH**	**ORP [mV] [a]**	**TN [mg/g] [b]**	**TOC [%] [c]**	**Moisture [%]**	**Dry density [g/cm]**
0-3	7.3	-170	2.3	1.7	85	0.16
3-6	7.4	-190	2.2	1.7	82	0.19
6-9			2.0	1.6	79	0.23
9-11			1.8	1.5	78	0.24
13-15	7.6	-198	1.7	1.5	77	0.26
17-19			1.3	1.2	72	0.32
21-23			1.3	1.2	68	0.36
25-27	7.4	-185	1.5	1.4	74	0.30
29-31			1.3	1.3	72	0.32
33-35	7.5	-201	1.1	1.0	70	0.35
37-39			1.0	0.9	69	0.37
41-43	7.6	-198	0.9	0.9	68	0.39
45-47			0.9	0.9	67	0.39
49-51			0.9	0.8	66	0.43
53-55	7.7	-194	0.9	0.8	65	0.43
57-59			0.9	0.8	64	0.44
61-63	7.7	-199	0.8	0.7	61	0.50
65-67			0.8	0.7	59	0.50
69-71			0.7	0.7	61	0.50
73-75	7.7	-201	0.7	0.7	61	0.50
77-79			0.8	0.7	60	0.49

[a] Oxygen reaction potential; [b] total nitrogen; [c] total organic carbon.

Table S19. Sampling location, sampling date and basic parameters of sediment core B from Tokyo Bay, Japan

Sampling date	**10. May 2008**					
GPS position	**N 35°29'18"/ E 139°54'24"**					
Water depth (m)	**21**					
Bottom water temperature	**15.2**					
Depth (cm)	**pH**	**ORP [mV] [a]**	**TN [mg/g] [b]**	**TOC [%] [c]**	**Moisture [%]**	**Dry density [g/cm^3]**
0-3	7.3	-163	1.9	1.5	79	0.23
3-6			1.7	1.4	76	0.27
6-8	7.4	-150	1.8	1.4	75	0.27
8-10			1.8	1.5	75	0.28
10-12			1.7	1.4	73	0.29
12-14	7.6	-160	1.6	1.4	74	0.30
16-18			1.5	1.3	72	0.31
20-22			1.5	1.3	72	0.32
24-26	7.7	-162	1.5	1.3	70	0.34
28-30			1.4	1.2	71	0.33
32-34	7.6	-165	1.4	1.2	70	0.35
36-38			1.3	1.1	67	0.39
40-42			1.1	0.9	65	0.42
44-46	7.6	-150	1.0	0.9	65	0.42
48-50			1.0	0.9	64	0.44
52-54	7.7	-159	1.0	0.8	64	0.43
56-58			0.9	0.8	62	0.45
60-62			0.9	0.8	61	0.47
64-66	7.7	-153	0.9	0.8	63	0.46
68-70			0.9	0.7	62	0.47

[a] Oxygen reaction potential; [b] total nitrogen; [c] total organic carbon.

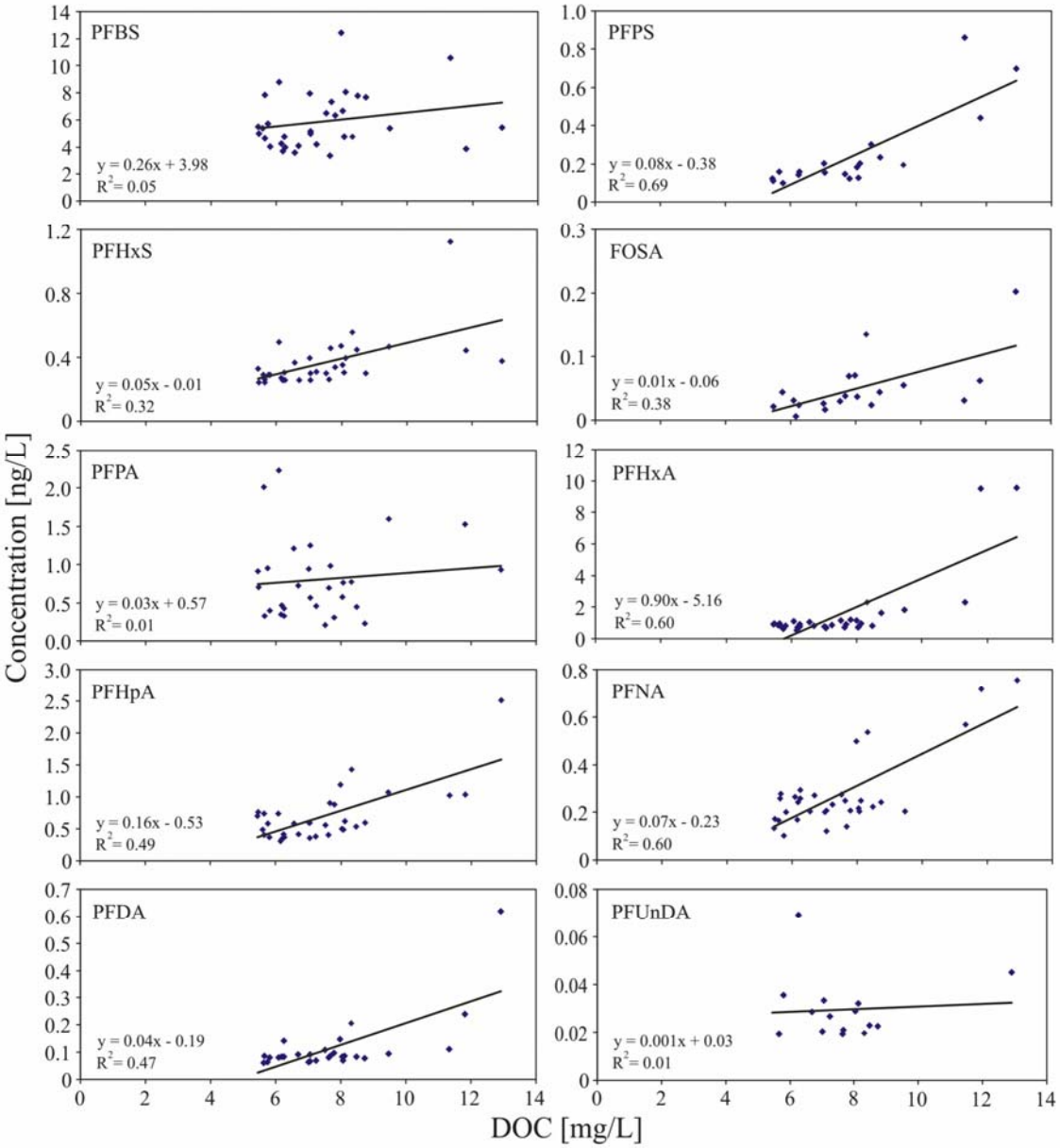

Figure S1. Relationship between concentrations of individual PFCs and dissolved organic carbon (DOC) in surface water in the German Bight

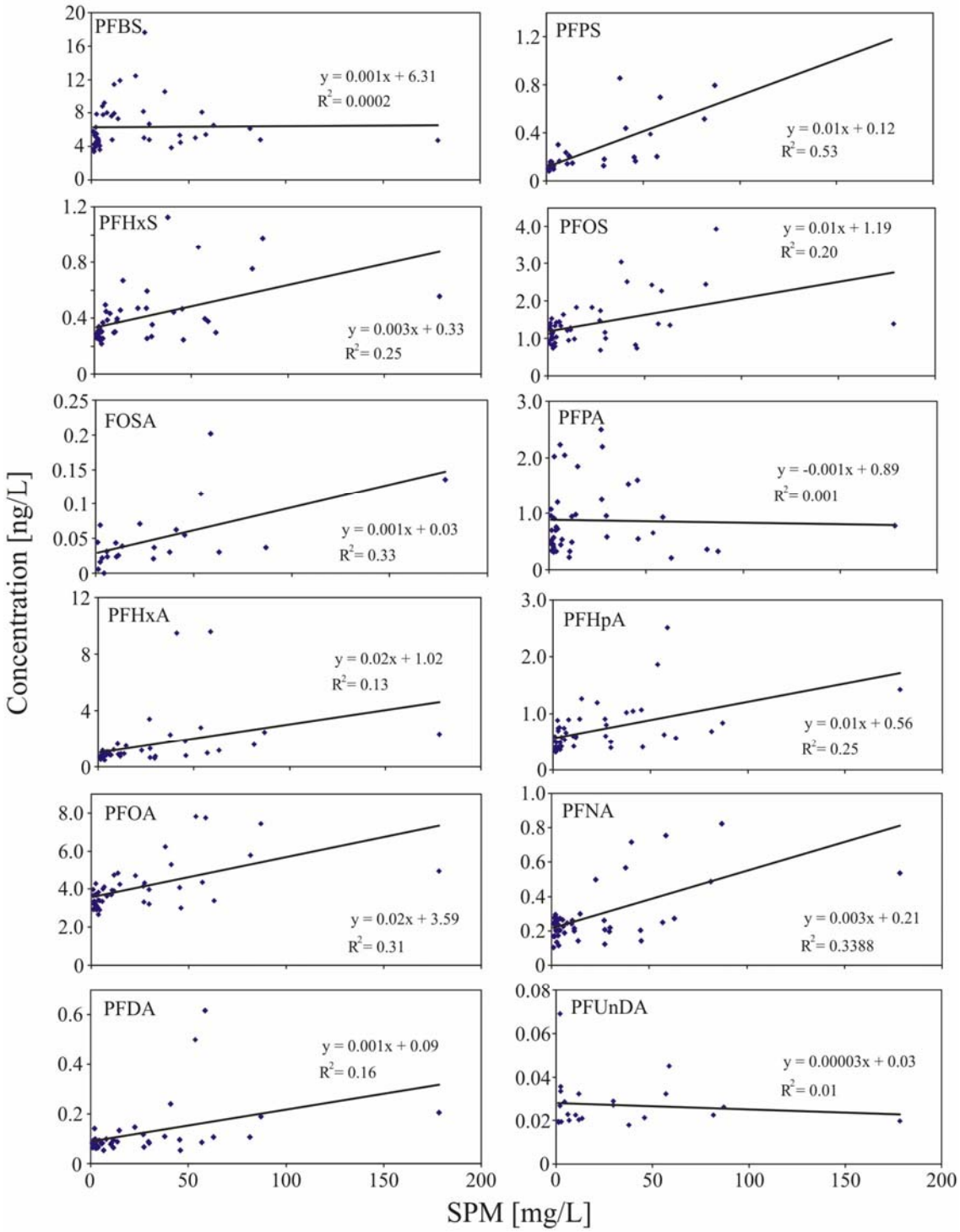

Figure S2. Relationship between concentrations of individual PFCs and suspended particulate matter (SPM) in surface water in the German Bight

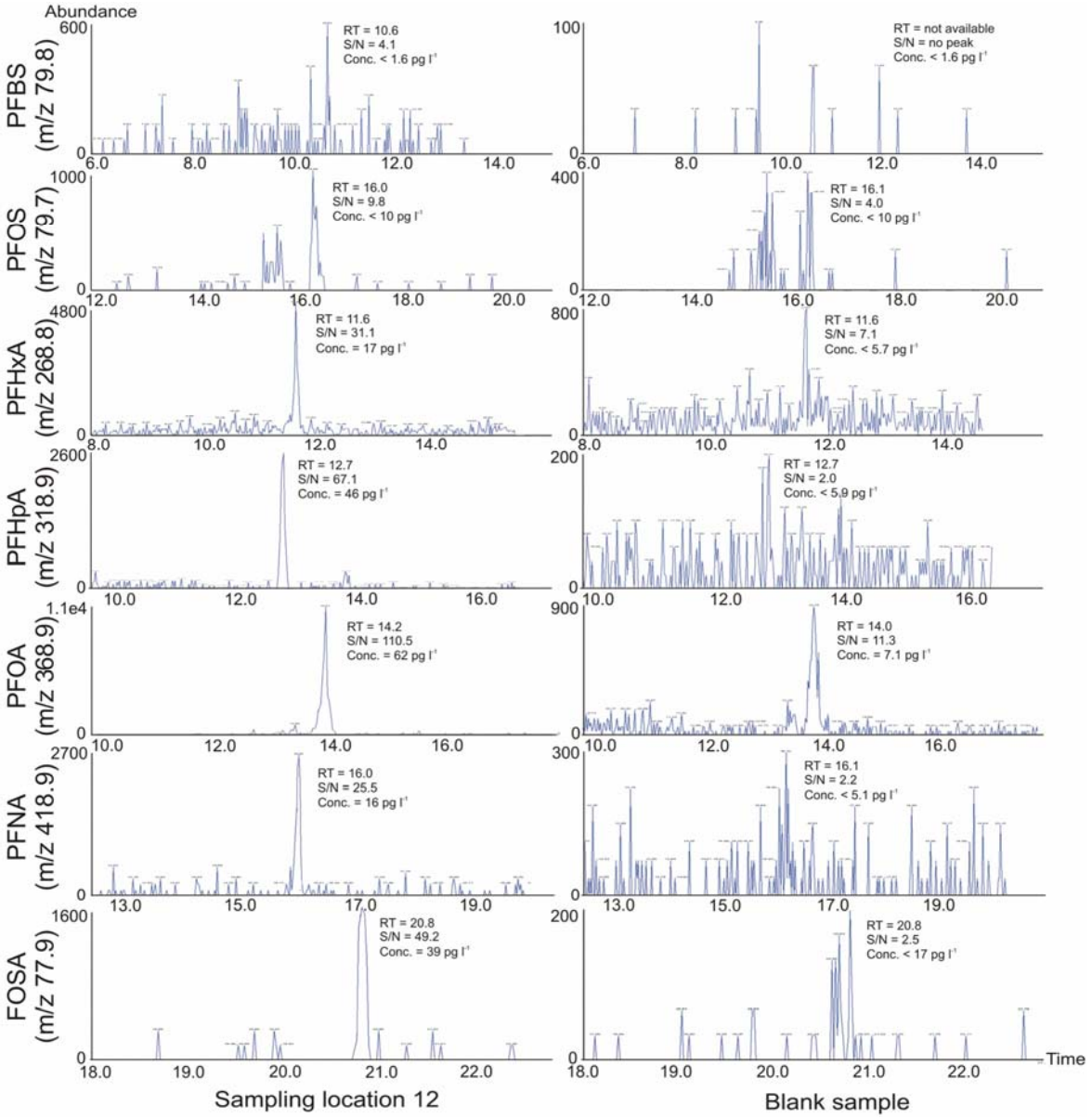

Figure S3. Chromatograms of PFBS, PFOS, PFHxA, PFHpA, PFOA, PFNA and FOSA in the dissolved phase for sampling location 12 and a typical blank sample from the cruise of the research vessel *Polarstern* (46° N-26° S) in the Atlantic Ocean

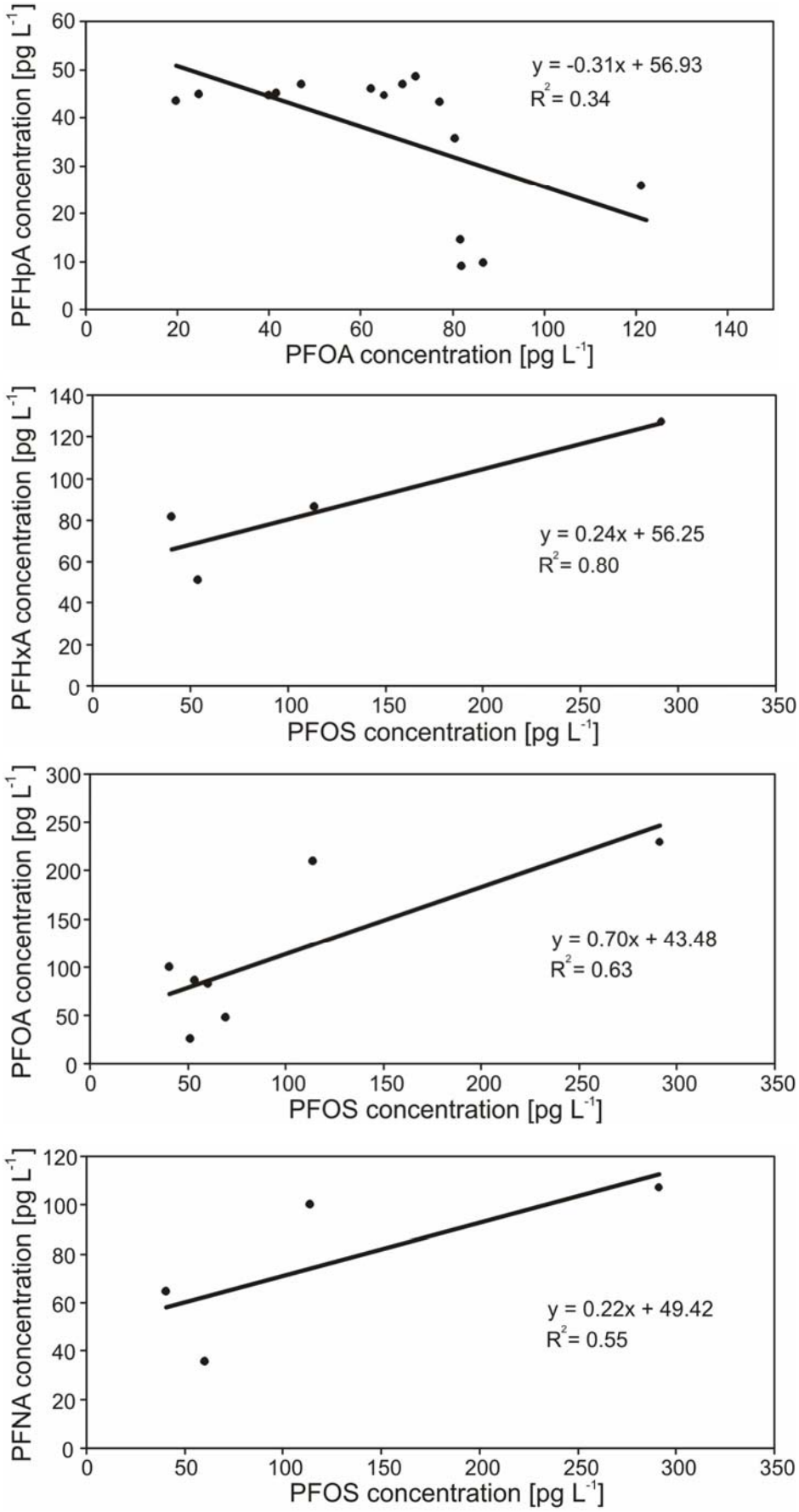

Figure S4. Correlations between PFC concentrations in surface water in the Atlantic Ocean

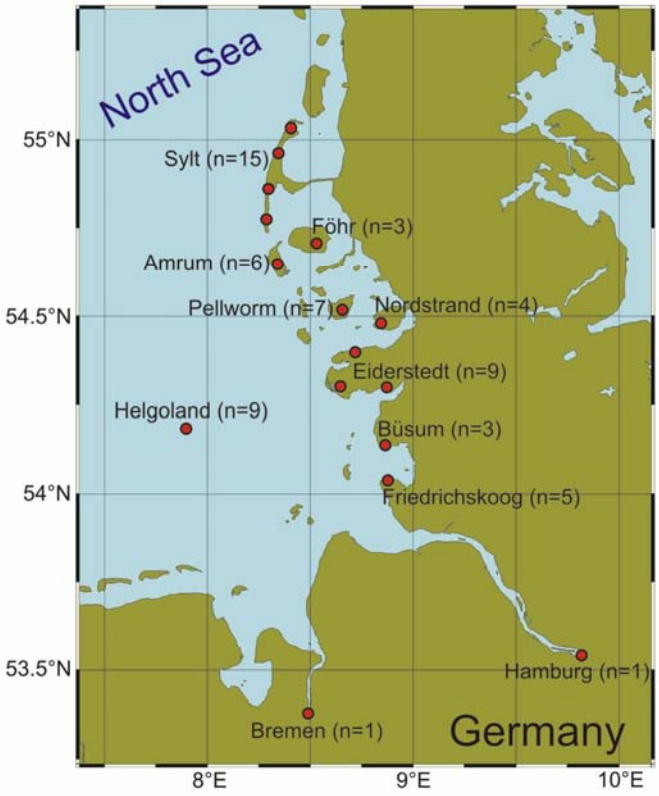

Figure S5. Sample locations of the collected habor seals in the German Bight, n = 63

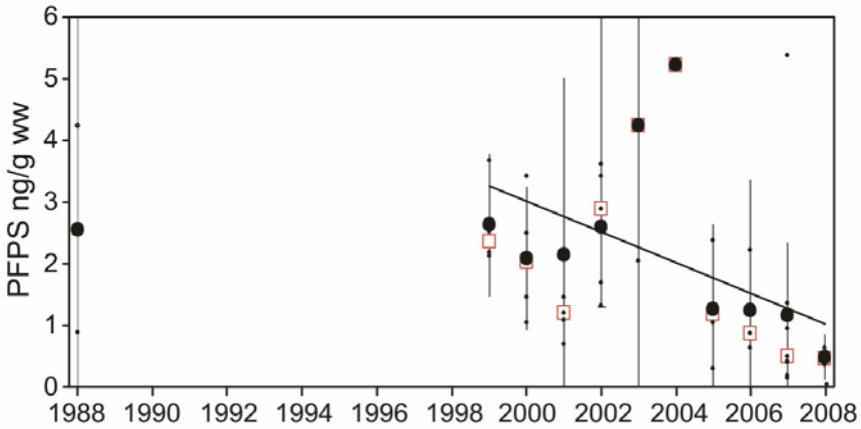

Figure S6. Temporal trends of PFPS in ≥ 7 month old habor seals from the German Bight from 1999 to 2008. The plots display the geometric means (circles) and the median (squares) together with the individual analysis (small dots) and the 95% confidence intervals of the geometric means