Annex XV dossier

PROPOSAL FOR IDENTIFICATION OF A SUBSTANCE AS A CMR 1A OR 1B, PBT, vPvB OR A SUBSTANCE OF AN EQUIVALENT LEVEL OF CONCERN

Substance Name(s): Pentadecafluorooctanoic Acid (PFOA)

EC Number(s): 206-397-9

CAS Number(s): 335-67-1

Submitted by: **BAuA**

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EC Number(s): 206-397-9

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- The substance is proposed to be identified as a substance meeting the criteria of Article 57 (c) of Regulation (EC) 1907/2006 (REACH) owing to the recent RAC opinion which concludes that PFOA should be classified as toxic for reproduction category 1B in accordance with the CLP Regulation (Regulation (EC) 1272/2008)1. This corresponds to classification as toxic to reproduction category 2 in accordance with Directive 67/548/EEC.
- It is proposed to identify the substance as PBT according to Article 57 (d).

Summary of how the substance meets the criteria set out in Article 57(c) and 57(d) of REACH

The free perfluoroocanoic acid (PFOA) stays in equilibrium with perfluorooctanoate (PFO), the conjugate base, in aqueous media in the environment as well as in the laboratory. The ammonium salt (APFO), which is often used in animal experiments, is very soluble in water. In aqueous solution it is present as anion PFO and the ammonium cation. The dissolved anion PFO will stay in equilibrium with the corresponding acid in aqueous media. In the following PFOA refers to the acid (PFOA) as well as to its conjugate base PFO. Therefore conclusions on PFOA/APFO are considered to be valid for APFO/PFOA as well.

Toxic for reproduction:

In its opinion of 2 December 2011 on the proposal for harmonised classification and labelling at EU level of Perfluorooctanoic acid (PFOA) ECHA's Risk Assessment Committee (RAC) concluded that the evidence is sufficiently convincing to classify PFOA for developmental effects as Repr. 1B (H360D - May damage the unborn child) according to CLP criteria (Regulation (EC) 1272/2008) and Repr. Cat. 2 (R61 - May cause harm to the unborn child) according to DSD (Council Directive 67/548/EEC).

Therefore, even though the substance is not yet listed in Annex VI of CLP (Regulation (EC) 1272/2008) there is evidence based on the RAC opinion on PFOA that seems to indicate that the substance meets the criteria for classification as toxic for reproduction in accordance with Article 57 (c) of REACH.

[1] http://echa.europa.eu/about/organisation/committees/rac/committee_opinions_en.asp

PBT:

A weight of evidence determination according to the provisions of Annex XIII of REACH is used to identify the substance as P and B. The available results are assembled together in a single weight of evidence determination.

Persistence:

Based on degradation experiments PFOA is not degradable in the environment and therefore fulfils the P- and vP-criterion of REACH Annex XIII.

Bioaccumulation:

All available information, i.e. bioaccumulation in terrestrial species and in humans was considered together in a weight of evidence approach. The individual results have been considered in the assessment with differing weights depending on their nature, adequacy and relevance. The bioaccumulative property is proved by studies from terrestrial food webs, which clearly indicate accumulation of PFOA in these food webs. Furthermore human data strongly indicate that PFOA bioaccumulates in humans. PFOA is present in human blood of the general population and elevated concentrations are seen following specific exposure to PFOA, either environmentally (e.g. contaminated drinking water) or occupationally. Time trend studies show that PFOA levels are significantly associated with the time exposed and some studies strongly indicate that PFOA levels increase with age. Additionally, in humans gestational and lactational exposure are of special concern as the foetus and newborn babies are highly vulnerable to exposure to toxic substances.

Furthermore it is of special concern that PFOA biomagnifies in endangered species as shown for the polar bear and in animals which are likely to become endangered in the near future (narwhale and beluga whale).

Based on weight of evidence, it is considered that the data from environmental species and humans show that the B criterion of REACH Annex XIII is fulfilled.

Toxicity

There is evidence based on the RAC opinion on PFOA that seems to indicate that the substance meets the criteria for classification as toxic for reproduction in accordance with Article 57 (c) of REACH. As a consequence the toxicity criteria of REACH Annex XIII is fulfilled.

Conclusion:

In conclusion PFOA meets the criteria for a PBT substance according to Article 57 (d)

PFOA has not been registered under REACH.

PART I

JUSTIFICATION

1 IDENTITY OF THE SUBSTANCE AND PHYSICAL AND CHEMICAL PROPERTIES

The free perfluoroocanoic acid (PFOA) stays in equilibrium with perfluorooctanoate (PFO), the conjugate base, in aqueous media in the environment as well as in the laboratory. The physico-chemical properties of PFOA and PFO are different. Therefore, the expected environmental fate will depend on the environmental conditions, which influence the equilibrium between base and acid (pH and pKa).

The ammonium salt (APFO), which is often used in animal experiments, is very soluble in water. In aqueous solution it is present as anion PFO and the ammonium cation. The dissolved anion PFO will stay in equilibrium with the corresponding acid in aqueous media.

With currently available analytical methods it is not possible to distinguish between PFO and PFOA in samples. In the literature reporting human and environmental monitoring studies the concentrations are referred to as PFOA or APFO, but always both species (PFO and PFOA) are included in the given concentration.

In the following PFOA refers to the acid (PFOA) as well as to its conjugate base PFO. Only in cases where it is important to distinguish between both species and where species specific knowledge is available it is clearly indicated that either the acid PFOA or the conjugate base PFO is meant.

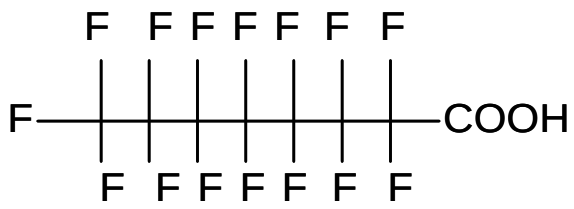
This Annex XV-Report covers both PFOA and APFO. For simplicity, in the discussions and conclusions in this document PFOA is usually referred to. Based on the reasoning above, the conclusions are considered to be valid for APFO as well.

1.1 Name and other identifiers of the substance

Table 1: Substance identity

EC number:	206-397-9
EC name:	Pentadecafluorooctanoic acid
CAS number (in the EC inventory):	335-67-1
CAS number:	335-67-1
CAS name:	Octanoic acid, 2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-pentadecafluoro-
IUPAC name:	Pentadecafluorooctanoic acid
Index number in Annex VI of the CLP Regulation	-
Molecular formula:	C8HF15O2
Molecular weight range:	414.07 g/mol
Synonyms:	Perfluorooctanoic Acid; PFOA; Pentadecafluoro-1-octanoic acid; Perfluorocaprylic acid; Perfluoroheptanecarboxylic acid; Perfluoro-n-octanoic acid; Pentadecafluoro-n-octanoic acid; Pentadecafluorooctanoic acid; n-Perfluorooctanoic acid 1-Octanoic acid, 2,2,3,3,4,4,5,5,6,6, 7,7,8,8,8-pentadecafluoro

Structural formula:



1.2 Composition of the substance

Name: Pentadecafluorooctanoic acid (PFOA)

Description: mono constituent substance

Degree of purity: > 99%

The detailed composition of the substance is confidential and provided in the technical dossier.

Pentadecafluorooctanoic acid is a mono constituent substance. The identification of Pentadecafluorooctanoic acid as SVHC is based on the properties of the main constituent only i.e. only the (hypothetical) ideal substance (i.e. purity of 100%) will be included in the Candidate List. However, by definition all mono constituent substances (real substances) with Pentadecafluorooctanoic acid as main constituent will be covered.

Therefore, in this case of a mono-constituent substance other constituents as well as the impurity profile are not relevant for the identification as SVHC.

1.3 Physico-chemical properties

Table 2: Overview of physicochemical properties

Property	Value	Remarks
Physical state at 20°C and 101.3 kPa	solid	(Kirk-Othmer, 1994)
Melting/freezing point	54.3 °C 44 - 56.5 °C	(Lide, 2003) (Beilstein, 2005)
Boiling point	188 °C (1013.25 hPa) 189 °C (981 hPa)	(Lide, 2003) (Kauck and Diesslin, 1951)
Vapour pressure	4.2 Pa (25 °C) for PFOA extrapolated from measured data 2.3Pa (20 °C) for PFOA extrapolated from measured data 128 Pa (59.3 °C) for PFOA measured	(Kaiser et al., 2005); (Washburn et al., 2005) (Washburn et al., 2005) (Washburn et al., 2005)
Water solubility	9.5 g/L (25° C) 4.14 g/L (22°C)	(Kauck and Diesslin, 1951) (Prokop et al., 1989)
Partition coefficient n-octanol/water (log value)	2.69 at pH7 and 25°C 6.3	Calculated using Advanced Chemistry Development (ACD/Labs) Software V11.02 (© 1994-2012 ACD/Labs). EPI suite [*Syracuse_Research_Corporation*, 2000-2008]
Dissociation constant pKa	2.5 2.8 in 50% aqueous ethanol 1.5 - 2.8	(Ylinen et al., 1990) (reliability not assignable) (Brace, 1962) (Kissa, 2001)
pH-value	2.6 (1 g / L at 20 °C)	(Merck, 2005) (reliability not assignable)

2 HARMONISED CLASSIFICATION AND LABELLING

In March 2010 Norway submitted a CLH dossier for harmonized classification and labeling of PFOA. In December 2011 the Risk Assessment Committee (RAC) concluded that PFOA should be classified as Carc. 2 H351, Repr 1B H360D, Lact H362, STOT RE 1 (liver) H372, Acute tox 4H332, Acute tox 4 H302 and Eye dam 1 H318.

The conclusions included in the RAC opinion presented in Table 3 and Table 4. The RAC opinion has been forwarded to the European Commission for inclusion in Annex VI to the CLP Regulation. On 11 January 2013 the Commission notified the WTO Committee on technical Barriers to Trade of its intention to classify PFOA accordingly.

Table 3: Harmonized classification according to the RAC opinion[2], in accordance with the CLP Regulation (Regulation (EC) 1272/2008)

International Chemical Identification	EC No	CAS No	Classification	
			Hazard Class and Category Code(s)	Hazard statement Code(s)
Pentadecafluorooctanoic acid (PFOA)	206-379-9	335-67-1	Carc. 2, Repr. 1B Lact STOT RE 1 (liver) Acute Tox. 4 Acute Tox. 4 Eye dam. 1	H351 H360D H362 H372 H332 H3012 H318

Table 4: Harmonized classification according to the RAC opinion[2], in accordance with the criteria of Directive 67/548/EEC

International Chemical Identification	EC No	CAS No	Classification
Pentadecafluorooctanoic acid (PFOA)	206-379-9	335-67-1	Carc. Cat 3; R40 Repr. Cat. 2: R61: R64 T; R48/23, Xn; R48/21/22, R20/22 Xi; R41

Thirty-three notifications (5 aggregated notifications) have been submitted for PFOA to the C&L Inventory. This information is publicly available via the ECHA website at the following link:

http://echa.europa.eu/web/guest/information-on-chemicals/cl-inventory-database.

[2] The RAC opinion on PFOA is available at the following link:
http://echa.europa.eu/documents/10162/13579/rac_pfoa_adopted_opinion_en.pdf

3 ENVIRONMENTAL FATE PROPERTIES

3.1 Degradation

3.1.1 Abiotic degradation

3.1.1.1 Hydrolysis

PFOA is hydrolytically stable under relevant environmental conditions. One study has been discussed in the OECD SIDS Initial Assessment Report for PFOA, which has been copied here in italic letters (OECD, 2006):

The 3M Environmental Laboratory performed a study of the hydrolysis of APFO (3M Co., 2001a). The study procedures were based on USEPA's OPPTS Guideline Document 835.2110; although the procedures do not fulfil all the requirements of the guideline, they were more than adequate for these studies. Results were based on the observed concentrations of APFO in buffered aqueous solutions as a function of time. The chosen analytical technique was high performance liquid chromatography with mass spectrometry detection (HPLC/MS).

During the study, samples were prepared and examined at six different pH levels from 1.5 to 11.0 over a period of 109 days. Experiments were performed at 50 °C and the results extrapolated to 25 °C. Data from two of the pH levels (3.0 and 11) failed to meet the data quality objective and were rejected. Also rejected were the data obtained for pH 1.5 because ion pairing led to artificially low concentrations for all the incubation periods. The results for the remaining pH levels (5.0, 7.0, and 9.0) indicated no clear dependence of the degradation rate of PFOA on pH. From the data pooled over the three pH levels, it was estimated that the hydrolytic half-life of PFOA at 25°C is greater than 92 years, with the most likely value of 235 years. From the mean value and precision of PFOA concentrations, it was estimated the hydrolytic half-life of PFOA to be greater than 97 years.

A newer study showed no decomposition of perfluorocarboxylic acids (PFCAs) in hot water in absence of $S_2O_8^{2-}$. After the addition of $S_2O_8^{2-}$ to the reaction system efficient decomposition of PFCAs has been observed at 80 °C. After a reaction time of 6 hours PFOA was decomposed completely. The reaction products were mainly F^- and CO_2 at a yield of 77.5 % and 70.2 %, respectively. Short chain PFCAs were a minor reaction product. However, at higher temperatures (150°C) 12.3% of the initial PFOA remained and the yields of F^- and CO_2 were 24.6 % and 37.0 %, respectively (Hori et al., 2008) (Reliabilty = 2).

In summary, PFOA is hydrolytically stable under environmental conditions.

3.1.1.2 Phototransformation/photolysis

Direct photolysis of a carbon fluorine chain is expected to be very slow, with stability expected to be sustained for more than 1000 years (Environment Canada, 2012).

3.1.1.2.1 Phototransformation in air

A slow indirect photodegradation in air with an atmospheric lifetime of 130 days has been reported (OECD, 2006). This value is predicted from shorter-chain perfluorinated acids (conclusion by analogy).

The following information was copied from the OECD SIDS Initial Assessment Report for PFOA (OECD, 2006):

Hurley et al. determined the rate constants of the reactions of OH radicals with a homologous series of perfluorinated acids (from trifluoroacetic acid to nonafluoropentanoic acid) in 700 Torr of air at 296 K (Hurley et al., 2004). For C_3 to C_5 chain length had no discernible impact on the reactivity of the molecule. The rate constant $k(OH + F(CF_2)_nCOOH) = (1.69±0.22)\times10^{-13}$ cm^3 $molecule^{-1}$ s^{-1} for n = 2, 3, 4, respectively. Atmospheric lifetimes of $F(CF_2)_nCOOH$ with respect to reaction with OH radicals are estimated to be approximately 230 days for n = 1 and 130 days for n > 1. (Calculation of lifetime by comparison with CH_3CCl3 (half-life 5.99 years, $k = 1.0\times10^{-14}$ cm^3 $molecule^{-1}$ s^{-1}).) The authors conclude, that the major atmospheric loss mechanism of perfluorinated carboxylic acids is dry and wet (particle mediated) deposition which occur on a time scale which is probably of the order of 10 days. Reaction with OH is a minor atmospheric loss mechanism for perfluorinated carboxylic acids.

In summary half-lives of 130 days have been reported for phototransformation in air.

3.1.1.2.2 Phototransformation in water

Studies on the phototransformation of PFOA in water are summarized in Table 5.

Table 5: Summary of photodegradation studies for APFO and PFOA

Test Substance	Result	Remarks	Reliability	Reference
APFO	No photodegradation	Direct photolysis	2	(OECD, 2006);(3M Co., 1979)
APFO	No photodegradation	Direct and indirect (H_2O_2; synthethic humic water, Fe_2O_3) photolysis	1	(OECD, 2006);(3M Co., 2001b)
	Estimated half-life > 349 days	Indirect photolysis (Fe_2O_3)		
PFOA		Short wave length (<300 nm) used for irradiation → limited relevance for an aqueous environment	2	(Hori et al., 2004)
	44.9% of the initial PFOA was decomposed after 24 hours	Direct photolysis; 0.48 MPa O_2		
	35.5% of the initial PFOA was decomposed after 24 hours	Indirect photolysis (H_2O_2); 0.48 MPa O_2		
	100% of the initial PFOA was decomposed after 24 hours	Indirect photolysis (tungstic heteropolyacid photocatalyst); 0.48 MPa O_2		
PFOA		Short wave length (<300 nm) used for irradiation → limited relevance for an aqueous environment	2	(Hori et al., 2005)
	16.8% of the initial PFOA was decomposed after 4 hours	Direct photolysis; 0.48 MPa O_2		
	100% of the initial PFOA was decomposed after 4 hours	Indirect photolysis ($S_2O_8^{2-}$); 0.48 MPa O_2		

The following information was copied from the OECD SIDS Initial Assessment Report for PFOA (OECD, 2006):

Direct photolysis of APFO was examined in two separate studies (3M Co., 1979; 3M Co., 2001b) and photodegradation was not observed in either study. In the 3M (1979) study, a solution of 50 mg/l APFO in 2.8 litres of distilled water was exposed to simulated sunlight at 22±2 ºC. Spectral energy was characterized from 290-600 nm with a max output at ~360 nm. Direct photolysis of the test substance was not detected.

In the 3M (3M Co., 2001b) study, both direct and indirect photolysis were examined utilizing techniques based on USEPA and OECD guidance documents. To determine the potential for direct photolysis, APFO was dissolved in pH 7 buffered water and exposed to simulated sunlight. For indirect photolysis, APFO was dissolved in 3 separate matrices and exposed to simulated sunlight for periods of time from 69.5 to 164 hours. These exposures tested how each matrix would affect the photodegradation of APFO. One matrix was a pH 7 buffered aqueous solution containing H_2O_2 as a well-characterized source of OH radicals. This tested the propensity of APFO to undergo indirect photolysis. The second matrix contained Fe_2O_3 in water that has been shown to generate hydroxyl radicals via a Fenton-type reaction in the presence of natural and artificial sunlight. The third

matrix contained a standard solution of humic material. Neither direct nor indirect photolysis of APFO was observed based on loss of starting material. Predicted degradation products were not detected above their limits of quantitation. There was no conclusive evidence of direct or indirect photolysis whose rates of degradation are highly dependent on the experimental conditions. Using the iron oxide (Fe_2O_3) photoinitiator matrix model, the APFO half-life was estimated to be greater than 349 days.

According to Hori et al., aqueous solutions of PFOA absorb light strongly from the deep UV-region to 220 nm (Hori et al., 2004). A weak, broad absorption band reaches from 220 to 270 nm (no absorption coefficient stated). The irradiation of a 1.35 mM PFOA solution (29.6 µmol) in water (under 0.48 MPa of oxygen) with light from a xenon-mercury lamp (no radiant flux stated) for 24 hours resulted in a ca. 44.9 % reduction (13.3 µmol) of PFOA concentration. Concentrations of CO_2 and fluoride increased simultaneously. Small amounts (0.1-5 µmol) of short chain perfluorinated hydrocarbon acids (C_2-C_7) were detected. The addition of the photocatalyst tungsten heteropolyacid ($[PW_{12}O_{40}]^-$) or persulfate ($S_2O_8^{2-}$) (Hori et al., 2005) accelerates the reaction rate. Due to the short wave length used for irradiation (< 300 nm) the photodegradation described may be of limited relevance for an aqueous environment but may be used as a technical process.

In summary no phototransformation of PFOA has been observed under environmental relevant conditions.

3.1.1.2.3 Phototransformation in soil

No data available

3.1.1.3 Summary on abiotic degradation

On the basis of the available data, abiotic degradation of PFOA in the atmosphere is expected to be slow. The atmospheric lifetime of PFOA has been predicted to be 130 days (conclusion by analogy from short-chain perfluorinated acids). In the aqueous phase PFOA is hydrolytically stable (DT_{50} > 92 years) under environmentally relevant conditions and does not undergo direct photodegradation in natural waters. The estimated half-life for indirect photolysis (addition of Fe_2O_3) is > 349 days.

3.1.2 Biodegradation

3.1.2.1 Biodegradation in water

3.1.2.1.1 Estimated data

3.1.2.1.2 Screening tests

Screening tests for the biodegradation of PFOA are summarized in Table 6.

Table 6: Summary of screening tests for PFOA/APFO

Test substance	Method	Result	Reliability	Reference
PFOA	OECD 301 C	5 % in 28 days	2	(MITI-List, 2002)
APFO	OECD 301 C	7 % in 28 days	2	(MITI-List, 2002)
PFOA	OECD 301 F	No biodegradation in 28 days	2	(Stasinakis et al., 2008)
APFO	Shake culture test modelled after the Soap and Detergent Association´s presumptive test for degradation	No biodegradation after 2.5 months	2	(OECD, 2006), (3M Co., 1978a)

A number of studies were already discussed in the OECD SIDS Assessment Report (OECD, 2006). The following text was copied from there:

Using an acclimated sludge inoculum, the biodegradation of APFO was investigated using a shake culture study modeled after the Soap and Detergent Association's presumptive test for degradation (3M Co., 1978a). Both thin-layer and liquid chromatography did not detect the presence of any metabolic products over the course of 2 1/2 months indicating that PFOA does not readily undergo biodegradation. In a related study, 2.645 mg/l APFO was not measurably degraded in activated sludge inoculum (Pace Analytical, 2001). Test flasks were prepared using a mineral salts medium, 1 ml methanol, and 50 ml settled sludge. Analysis was conducted with a HPLC/MSD system. Although the results were deemed unreliable due to a lack of description of experimental protocols or indications of a high degree of experimental error, several other studies conducted between 1977-1987 also did not observe APFO biodegradation (Pace Analytical, 1987; 3M Co., 1985; 3M Co., 1980c; 3M Co., 1979). In addition, a study conducted by Oakes et al.) indicated little biotic or abiotic degradation of PFOA on a time scale of 35 days, i.e., the PFOA exposure concentrations were stable over time and ranged from 84.5 % to 114.5 % of the initial concentrations (Oakes et al., 2004).

In a 28 day ready biodegradability test (OECD 301 C) using 100 mg/l PFOA and APFO, respectively, and 30 mg/l activated sludge non-biodegradability was demonstrated. Only 5 % (PFOA) and 7% (APFO) degradation was observed by BOD (MITI-List, 2002).

In a further test of ready biodegradability (OECD 301 F) no biodegradation of PFOA was observed in 28 days (Stasinakis et al., 2008).

In summary, on the basis of the available screening tests, PFOA is not readily biodegradable.

3.1.2.1.3 Simulation tests

No environmental half-lives for PFOA have been reported, even in the cases where corresponding tests have been performed (see table 7 below).

Table 7: Summary of simulations tests of PFOA/APFO

Test substance	Method	Result	Reliability	Reference
PFOA	Closed-loop systems in laboratory scale; Aerobic and anaerobic conditions	No elimination	3	(Meesters and Schroeder, 2004; Schröder, 2003)
APFO	Biodegradation in mixed bacterial culture and activated sludge Aerobic conditions	< 0.6 % of $^{14}CO_2$ was detected after 28 days	4	(Wang et al., 2005)
Sodium pentadeca-fluoro-octanoate	Microcosm study Aerobic conditions	No significant dissipation from water column after 35 days (initial concentration 0.3 mg/L; 1mg/L; 30 mg/L) 32% dissipation in 35 days (initial concentration 100 mg/L)	3	(Hanson et al., 2005)
PFOA/APFO	1.Preliminary screening: PFOA serves as an electron acceptor under anaerobic conditions (in combination with different inoculum) 2. Hypothesis refinement: ^{14}C APFO serves as an electron acceptor under anaerobic conditions	No significant consumption of the initial PFOA during 110 – 259 days No loss of APFO No production of $^{14}CO_2$ No detection of radiolabel transformation products	2	(Liou et al., 2010)

In the OECD SIDS Initial Assessment Report it was concluded that PFOA is not expected to undergo biodegradation (OECD, 2006). The following text in italic letters was copied from there:

Schroeder (2003), and Meesters and Schroeder (2004) investigated the biochemical degradation of PFOA in sewage sludge in laboratory scale reactors. After 25 days under aerobic conditions PFOA (initial concentration 5 mg/l) was not eliminated by metabolic processes, mineralization processes or by adsorption (Meesters and Schroeder, 2004; Schröder, 2003). This study is assessed with reliability 3 due to significant methodological deficiencies.

Wang et al. studied the biodegradation of fluorotelomer alcohols. However, ^{14}C-labelled APFO was used as starting material in this study, too. The authors analyzed the headspace of sealed vessels containing mixed bacterial cultures and vessels containing activated sludge from a domestic sewage treatment plant under continuous air flow. The mixed bacterial culture from industrial wastewater treatment sludge was enriched using 8:2 telomere alcohol and ^{14}C-labelled APFO, respectively. However, for using APFO as a starting material no detailed information are available from the report. The authors describe that potential biodegradation products were separated and quantified by LC/ARC (on-line liquid chromatography/accurate radioisotope counting). Transformation products were identified by quadrupole time of flight mass spectrometry. Only <0.6 % of $^{14}CO_2$ was detected after 28 days. The report contains no graphs or further data to re-evaluate this statement. Although the study seems to be very well documented for ^{14}C labelled 8:2 FTOH, we can only flag the study with a reliability of 4, since details on APFO are not available. The documentation for the results

obtained with APFO is missing in the report. However the result indicates that APFO is not biodegradable within 28 days (Wang et al., 2005).

Hanson et al. performed a microcosm study. Microcosms were approximately 1.2 m deep with a water depth of 1 m, a diameter of 3.9 m, and a surface area of 11.95 m². Each microcosm had a capacity of approximately 12 m³ of water. Sediment consisted of a 1:1:1 mixture of sand, loam and organic matter (mainly composted manure). The total carbon content of the sediment was 16.3%. Microcosms were circulated for 2 weeks from a well-fed irrigation pond prior to the experiments. Nominal concentrations of 0.3, 1, 30, and 100 mg/l PFOA, as the sodium salt, plus controls were added to the microcosms. Each exposure was randomly assigned to three separate microcosms from a total of 15 microcosms. Immediately prior to treatment, water flow into each microcosm from the main irrigation pond ceased, creating a closed system relative to the other microcosms and the irrigation pond.

Water chemistry and PFOA analysis were taken at the same time on a regularly basis. Temperature and dissolved oxygen content were measured daily. Water samples were collected with a metal integrated water column sampler. Integrated subsamples from at least 4 randomly selected locations in each microcosm were collected to a total volume of 4 L. Samples were stored at 4 °C until analysis. Water samples were analyzed by ion chromatography. The mobile phase was 0.5 mM NaOH, 5 % methanol, and 5% acetonitrile with a flow rate of 0.4 mL/min. Injection volumes varied from 5,10,75, and 200 µl for the 100, 30, 1 and 0.3 mg/L microcosms, respectively. For each set of samples analyzed five standards and one quality control sample were included at the beginning of each run and again at the end. Radioactive labelling was not performed. Over a 35-day field study PFOA showed no significant dissipation from the water column. However, at the highest concentration (100 mg/L) a partitioning from the water column into other compartments is suspected (32% dissipation in 35 days) (Hanson et al., 2005). Since the documentation of the procedure was insufficient in our opinion the study is not reliable (reliability 3).

Liou et al. investigated the anaerobic biodegradability of PFOA respectively APFO. In a two-phase experiment (preliminary screening, hypothesis refinement) the use of PFOA as a physiological electron acceptor (electron donator: acetate, lactate, ethanol or hydrogen gas) was studied. Additionally, the possibility of co-metabolism of PFOA during reductive dechlorination of trichloroethene and during various physiological conditions (aerobic, nitrate-reducing, iron-reducing, sulfate reducing, and methanogenic) was analyzed. Five different inoculums were used (from a municipal waste-water treatment plant, industrial site sediment, an agricultural soil, and soils from two fire training areas). Environmental samples used as inoculum sources in the biodegradation experiments were aseptically gathered (sterile spatula) placed in 0.5 L sterilized canning jars (filled to the brim), stored on ice in the field, and maintained at 4 °C before being transferred to an anaerobic hood where samples were degassed and dispensed as slurries in biodegradation assays. Soils and sludges were gathered from: the Ithaca sewagetreatment plant; a water-saturated drainage ditch adjacent to the DuPont Chambers Works waste treatment facility in Salem County, New Jersey, previously shown to carry out reductive dechlorination (Fung et al., 2009); the Cornell agricultural field station (Collamer silt loam, Ithaca, NY), the Ithaca fire training facility, and the Rochester, NY fire training facility (the latter two sites were chosen due to potential contamination with fluorinated fire retardant chemicals) (Liou et al., 2010).

For the serum bottle-based biodegradation assays treatments occurred in triplicats (160 ml serum bottles with 100 mL of media; live ± PFOA and abiotic controls, autoclaved for 1 h). For the ^{14}C-PFOA experiments, 15-mL serum bottles were utilized (50% O_2-free N_2 headspace, 50% inoculated anaerobic test medium) with non-radioactive PFOA and ^{14}C- PFOA (4.5 lCi/mL test medium) to give a final concentration of 100 mg/L PFOA. For establishing the various terminal electron-accepting processes, a standard anaerobic procedure was used. The anaerobic mineral salts buffer

(plus vitamins and trace minerals) was used as diluents for the various inoculums sources (5% wt/volume) with addition of electron donors (10 mM sodium acetate ± 40 mM sodium lactate or 0.6 mM ethanol or 2 atm H_2) or electron acceptors [O_2 as air headspace or O_2- free N_2 headspace in each serum bottle with additions of 30 mM nitrate or 4 mg mL_1 FeOOH or 10 mM sulfate or 0.4 mM trichloroethene (TCE) or no addition (for the methanogenic treatment)]. Samples (1.0 mL) were periodically removed from each serum bottle, placed in 4-mL glass vials sealed with Al-backed caps, immediately mixed with an equal volume of methanol and stored at _20 °C until analyzed. Accumulated batches of samples from serum vials were analyzed for concentrations of PFOA, ^{14}C-PFOA, fluoride, nitrate, sulfate, and potential PFOA transformation products. Headspace gases were sampled with a gas-tight syringe (250 mL) and analyzed for TCE, vinyl chloride and methane. In the radiotracer study, dissolved ^{14}C activity in the anaerobic medium and in the 0.4 N KOH solution retrieved from the internal reservoir to trap $^{14}CO_2$ were determined by scintillation counting. To assay potential microbial inhibition by PFOA, triplicate serum- bottle assays inoculated with 5% Ithaca sewage were prepared, as above. Anaerobic preparations (±100 ppm PFOA) were assayed for methanogenesis. Aerobic preparations containing 15 ppm naphthalene were sampled as above and analyzed by high-performance liquid chromatography (HPLC). After filtration through nylon acrodisc filters, naphthalene was separated at room temperature. Methanol–water (1:1) was the mobile phase at a flow rate of 1.5 mL/ min. The eluent was monitored by UV VIS at 340 nm. Quantification was done by comparison to authentic standards (Liou et al., 2010). PFOA quantification was performed by LC/MS/MS following a standard procedure. Potential PFOA metabolites were screened by applying LC/MS (Liou et al., 2010).

In no combination of the inoculum source, electron donator or physiological conditions a significant percentage of the initial PFOA (100 ppm and 100 ppb) was consumed (110-259 days). In a test with ^{14}C labelled APFO (inoculum = sewage), no loss of APFO was detected, no $^{14}CO_2$ was produced and no radiolabelled APFO transformation product was indicated. Co-metabolism of PFOA during reductive dechlorination of trichlorethene was suggested by a drop in PFOA concentration in the 100 ppb treatment after a 65-d incubation. However, extensive analysis failed to determine corroborating transformation products (Liou et al., 2010).

In summary, under conditions which were examined in this study, PFOA is environmentally persistent (Liou et al., 2010).

Although for aerobic conditions no reliable study is available, it can be concluded that the above-mentioned studies support that PFOA respectively APFO is not biodegradable under aerobic conditions. In the environment aerobic as well as anaerobic conditions occur. Hence, simulations tests under both conditions are necessary for assessing the persistence. In conclusion, degradation simulation studies on PFOA demonstrate the high persistence of the compound in various media, like sludge, sediment and water.

3.1.2.2 Biodegradation in sediments

The anaerobic biodegradability of PFOA respectively APFO in industrial site sediment was investigated by Liou et al. (see above 3.1.2.1.3 Simulation tests). No significant amount of the initial PFOA was dissipated after 259 days.

3.1.2.3 Biodegradation in soil

A number of studies were already discussed in the OECD SIDS Initial Assessment Report. The following text was copied from there (OECD, 2006):

Moody and Field (1999) conducted sampling and analysis of samples taken from groundwater 1 to 3 meters below the soil surface in close proximity to two fire-training areas with a history of aqueous film forming foam use. Perfluorooctanoate was detected at maximum concentrations ranging from 116 to 6750 µg/l at the two sites many years after its use at those sites had been discontinued. These results suggest that PFOA can leach to groundwater (Moody and Field, 1999).

Extensive site specific monitoring of soil and ground water concentrations of PFOA and related substances was conducted by 3M, DuPont Daikin and others. PFOA in soil has been shown to persist for decades and to be a long term source of groundwater and surface water contamination (see for example (DuPont Co., 2003; 3M Co., 2005)).

At the DuPont Washington Works site soil contaminated by perfluorochemical waste has been shown to contain ppm levels of PFOA 3 decades after application ceased. The underlying groundwater also contains ppm levels of PFOA (DuPont Co., 1999a).

Extensive field monitoring data generated by 3M at the Decatur, AL site have also shown that PFOA is persistent in soils. Soil samples were collected from a former sludge application area of the 3M Decatur, AL facility also show soil contamination and underlying groundwater contamination up to ppm levels decades after application ceased.

Moody et al. investigated groundwater at a former fire-training area at Wurtsmith Air Force Base which was used between 1950s and 1993. Groundwater samples were collected from two types of monitoring wells. All samples were collected in high density polypropylene bottles. Samples were shipped on ice without preservation and stored at 4 °C prior to analysis. Perfluorocarboxylate concentrations were measured as described in the following: Strong anion exchange disks were used to extract perfluorocarboxylates (6 to 8 carbons) from groundwater. The perfluorocarboxylates were simultaneously eluted from the disks and derivatized to their methyl esters by treatment with iodomethane for direct analysis by electron impact gas chromatography-mass spectrometry (GC-MS). A single analysis was conducted for each groundwater sample. The detection limit (defined as a signal-to-noise ratio greater than 3) and quantification limit (defined as a signal-to-noise ratio greater than 10) for perfluorocarboxylates were 3 mg/L and 13 mg/L, respectively, using 2-chlorolepidine as the internal standard. Additionally, electron capture negative chemical ionization GC-MS was employed to confirm the identity of PFOA, in groundwater samples (Moody et al., 2003). Depending on the location of sampling, the concentrations of PFOA were between 8 µg/L and 105 µg/L in groundwater. The authors estimated that perfluorinated surfactants had been in the groundwater for at least five years and possibly for as long as 15 years. This showed that degradation of PFOA was negligible under the environmental conditions at this site (for both soil and groundwater) (Reliability = 2) (Moody et al., 2003).

The anaerobic biodegradability of PFOA and APFO, respectively, in soil from two fire training areas was investigated by Liou et al. (see above 3.1.2.1.3 Simulation tests). No significant amount of the initial PFOA was dissipated after 259 days.

3.1.2.4 Summary and discussion on biodegradation

PFOA is not ready biodegradable using standard test methods. The results of one non-standard aerobic biodegradation simulation test, one non-standard anaerobic biodegradation simulation test and field monitoring data on PFOA from contaminated sites provide evidence that no biodegradation in water, soil and sediment occurs. The monitoring data show that PFOA in soil leaches over time and can be a long term source to underlying groundwater.

3.1.3 Summary and discussion on degradation

Abiotic degradation

Abiotic degradation of PFOA in the atmosphere is expected to be slow (atmospheric lifetime = 130 days; conclusion by analogy from short-chain perfluorinated acids). The hydrolytic half-life of PFOA at 25°C is greater than 92 years, with the most likely value of 235 years under relevant environmental conditions. No photodegradation of PFOA has been observed in studies conducted under relevant environmental conditions. The estimated DT_{50} for indirect photolysis is 349 days.

Biotic degradation

Standard screening studies indicate that PFOA is not ready biodegradable. The results of simulation tests and field monitoring data give additional support that no biodegradation in water, soil and sediment did occur.

Conclusion

The stability of organic fluorine compounds has been described in detail by Siegemund et al., 2000: When all valences of a carbon chain are satisfied by fluorine, the zig-zag-shaped carbon skeleton is twisted out of its plane in the form of a helix. This situation allows the electronegative fluorine substituents to envelope the carbon skeleton completely and shield it from chemical attack. Several other properties of the carbon-fluorine bond contribute to the fact that highly fluorinated alkanes are the most stable organic compounds. These include polarizability and high bond energies, which increase with increasing substitution by fluorine. The influence of fluorine is greatest in highly fluorinated and perfluorinated compounds. Properties that are exploited commercially include high thermal and chemical stability (Siegemund et al., 2000).

Based on their molecular properties it is, thus, not surprising, that researchers could not measure degradation of the intensively studied PFOA or its salts.

In summary, PFOA is very persistent and does not undergo any further abiotic or biotic degradation under relevant environmental conditions.

3.2 Environmental distribution

3.2.1 Adsorption/desorption

Not relevant for this dossier

3.2.2 Volatilisation

Not relevant for this dossier

3.2.3 Distribution modelling

Not relevant for this dossier

3.3 Bioaccumulation

3.3.1 General remarks

A commonly agreed descriptor to estimate the bioaccumulation potential of a substance is its partition coefficient log K_{OW} between water and *n*-octanol. When evaluating lipophilic substances this partition model sufficiently mimics the extent of uptake by aquatic organisms. For substances which tend to dissociate or are prone to form ionic structures the affinity to *n*-octanol is diminished resulting in low experimentally observed log K_{OW} values. In contrast to this assumption, it has been demonstrated from field studies that ionic compounds can be efficiently taken up by aquatic organisms and exhibit bioconcentration potential (e.g. perfluorooctanesulfonate). Similar problems emerge when assessing K_{OW} for surface active compounds. In biphasic test systems these surfactants will aggregate in multi-layers or micellar structures yielding colloidal dispersed solutions rather than a partition equilibrium. In such cases an experimental determination of log K_{OW} is hardly feasible.

Nevertheless, in account of the notable water solubility of PFOA, the high degree of dissociation (low pK_a value) as well as the inherent lipid repellence, caused by the perfluorinated alkyl chain, the coefficient K_{OW} is hypothesized to be low.

With this approach no preliminary estimation of possible bioconcentration can be gained. Nevertheless, results from studies which do not focus on K_{OW} show that PFOA bioaccumulates.

This issue has been discussed in detail in the OECD SIDS Initial Assessment Report for PFOA. For consistency, the following text was copied here in italic letters (OECD, 2006):

PFOA does not behave like lipophilic compounds that accumulate in fat tissues. For lipophilic substances, accumulation is expected preferentially in the fat tissues. Due to the perfluorination, the hydrocarbon chains are oleophilic and hydrophobic and the perfluorinated chains are both oleophobic and hydrophobic. In addition, functional groups attached to the perfluorinated chain (e.g., a charged moiety such as a hydroxyl group or sulfonic acid) can impart hydrophilicity to part of the molecule. Hydrophobicity is unlikely to be the sole driving force for the partitioning of perfluorinated substances to tissues because the oleophobic repellency opposes this partitioning process. Perfluorinated substances are also intrinsically polar chemicals because fluorine, a highly electronegative element, imparts polarity. Thus, perfluorinated substances have combined properties of oleophobicity, hydrophobicity, and hydrophilicity over portions of a particular molecule. Due to these properties, the assumption that the traditional hydrophobic and lipophilic interactions between compound and substrate are the main mechanisms governing partitioning may not be applicable for PFOA.

According to the revised Annex XIII not only the numerical bioaccumulation (B) criterion based on bioconcentration factors can be used to assess the bioaccumulation potential of a substance but also other information can be used. These information on the bioaccumulation potential are measured elevated levels in biota, information on the ability of the substance to biomagnify in the food chain, data from analysis of human body fluids or tissues and assessment of toxicokinetic behaviour of the substance should also be considered for the assessment using a weight-of-evidence approach. New sections 3.3.4 and 3.3.5 are added to include such data on PFOA.

3.3.2 Bioaccumulation in aquatic organisms

3.3.2.1 Bioconcentration factor BCF

Bioconcentration is the process by which a chemical enters an organism and/or is adsorbed on to it as a result of exposure to the chemical in water – it often refers to a condition usually achieved under laboratory and steady state conditions. The BCF is typically measured as the ratio of the chemical concentrations in the organism and the water once a steady state has been achieved:

$$BCF = \frac{c_{Biota}}{c_{Water}}$$

or alternatively be determined kinetically by using the uptake rate k_1 and the depuration rate k_2:

$$BCF = \frac{k_1}{k_2}$$

The bioconcentration of PFOA has been discussed in detail in the OECD SIDS Initial Assessment Report for PFOA. For consistency, the following text was copied here in italic letters (OECD, 2006):

To determine bioconcentration of PFOA, rainbow trout were exposed in a flow-through system for 12 days followed by a depuration time of 33 days in fresh water to determine tissue distribution and bioconcentration (Martin et al., 2003a). For determination of bioconcentration, juvenile fish (5-10 g) were exposed to a concentration of 1.5 µg/l in a flow-through system. At 7 occasions during uptake period and 9 occasions during depuration phase, fish were removed to determine the kinetics of uptake and depuration. Additionally, for the tissue distribution study, four immature trout (30-48 g) were exposed in separate tanks but under the same uptake conditions.

PFOA concentration was highest in blood, kidney, liver and gall bladder and low in the gonads, adipose and muscle tissue. Within the blood, the plasma contained between 94 – 99% of PFOA, with only a minor fraction detectable in the cellular fraction. Recovery from hearts and spleen was low (<10%).

A steady state was reached during uptake time. Visual observation of depuration data indicated possible biphasic depuration in blood, liver and carcass. However, this could not be verified statistically because of the small sample size. The following BCFs are calculated:

$BCF_{carcass}$ = 4.0 (+- 0.6); depuration half-life: 5.2 d (±0.67)
BCF_{blood} = 27 (+- 9.7); depuration half-life: 4.5 d (±1.6)
BCF_{liver} = 8.0 (+- 0.59); depuration half-life: 3.9 d (±0.28)

PFOA occurs mainly in muscle, blood and organs (liver, kidney) but not in lipid tissue and is reported for other species such as birds and mammals by several authors.

Fathead minnows (Pimephales promelas) were exposed to PFOA in a static system to a concentration of 25 mg/L for 13 days, followed by a depuration phase of 15 days. A BCF of 1.8 was calculated (3M Co., 1995).

Daikin performed a bioaccumulation test according to OECD Guideline 305, with the carp Cyprinus carpio (Daikin, 2000). The fish were exposed to PFOA concentrations of 5 and 50 µg/l for

28 days. For the higher concentration of 50 µg/l, the steady state was reached after 16 days and a BCF of 3.2 was calculated. For the lower concentration of 5 µg/l, a BCF of 9.4 was determined after 16 days; this level was reduced to ≤ 5.1 after 28 days. No steady state was reached until end of exposition. Although experiments with fish and other aquatic species provide evidence that PFOA is not highly bioaccumulative, these results should not be extrapolated to other animals. Fish gills may provide an additional mode of elimination and uptake which birds, terrestrial organisms, and marine mammals do not possess (Kelly et al., 2004).

The BCFs reported from laboratory experiments are summarized in Table 8.

Table 8: Examples of measured bioconcentration factors (BCF) of PFOA

Location	Species (tissue)	BCF	Reliability	Reference
Laboratory	Fathead minnow	1.8	2	*(*3M *Co., 1995)*
Laboratory	Rainbow trout (Carcass)	4.0 ± 0.6	2	(Martin et al., 2003a)
Laboratory	Rainbow trout (Blood)	27 ± 9.7		
Laboratory	Rainbow trout (Liver)	8.0 ± 0.59		
Laboratory	Carp	3.2-9.4	4	(Daikin, 2000)

Conclusion: BCFs for PFOA are below 2000, indicating no bioaccumulation in aquatic organisms due to uptake from the aqueous phase by diffusion via the gills. The high water solubility of PFOA may enable fish to quickly excrete this substance via gill permeation, facilitated by the high water throughput (Kelly et al., 2004; Martin et al., 2003a; Martin et al., 2003b). However, bioconcentration values in fish may not be the most relevant endpoint to consider, because other mechanisms of accumulation might be of relevance.

3.3.2.2 Bioaccumulation factors (BAFs)

In field studies on bioaccumulation of chemicals bioaccumulation factors (BAF) are measured. The BAF is typically measured in the field as the ratio of the chemical concentrations in the organism and the surrounding medium (e.g. water in natural ecosystems). In contrast to the BCF, the uptake is not only limited to exposure via water but all routes including diet contributes to the concentration in organisms:

$$BAF = \frac{c_{Biota}}{c_{Water}}$$

where chemicals concentration in the organism (c_{biota}) is usually expressed in units of gram of chemical per kilogram of organism. The weight of the organism can be expressed on a wet weight basis or appropriately normalized, if needed, (e.g. lipid- or protein-normalized) (Conder et al., 2011). BCFs are measured under controlled laboratory conditions, whereas the BAF is a field measurement and therefore different from BCF. Once up taken into the body, perfluorinated substances tend to partition to liver and blood. However, most field measurements for these substances have been performed on those individual organs and tissues. This is especially true for organisms at the higher trophic levels (e.g., polar bear), where whole-body analysis is not feasible for ethical reasons and due to the challenging logistics with respect to sampling and laboratory constraints. While it is feasible to measure whole-body BAFs on smaller species at lower trophic levels, the lower trophic status of the organism means that the estimated overall BAFs for

perfluorinated substances may be underestimated. Thus, from a toxicological perspective, BCFs, BAFs and BMFs based on concentrations in individual organs, such as the liver, may be more relevant when the potential for direct organ-specific toxicity (i.e. liver toxicity) is being predicted. On the other hand BCFs and particularly BMFs based on concentrations in whole organisms may provide a useful measure of overall potential for transfer up the food chain.

Although some authors describe BCF values in their field studies, BAFs would be more appropriate, because it cannot be excluded that the tested organisms did not take up PFOA via the diet. BAFs are given in Table 9. The following text in italic letters was copied from the OECD SIDS Initial Assessment Report for PFOA (OECD, 2006).

Martin et al. (Martin et al., 2003b) exposed juvenile rainbow trout (Oncorhynchus mykiss) for 34 d to PFOA in the diet, followed by a 41 day depuration period. During the uptake period, animals were daily fed with spiked food (0.42 mg PFOA/kg food) at a rate of 1.5 % food per fish. Assimilation efficiency (% of PFOA absorbed relative to the amount fed) was 59 %, indicating efficient absorption from food. At 6 occasions during uptake period and during depuration period, fish were removed to determine the kinetics of uptake and depuration. Carcass and liver concentrations were determined by using liquid chromatography-tandem mass spectrometry, and kinetic rates were calculated to determine bioaccumulation parameters.

The carcass uptake curves clearly showed by visual inspection, that the slope of the curve levels off by the end of the uptake period. According to the authors the steady state was reached after 10 days. A depuration half-life time of 3d (± 0.42) and a BAF (Bioaccumulation factor) of 0.038 (± 0.0062) were determined.

The bioaccumulation of PFOA in the wild turtles Trachemys scripta elegans and Cinemy reevesii was reported by Morikawa et al. 2005. Serum concentrations of PFOA from 94 turtles were compared to surface water samples from the site of the turtle capture for several rivers in Japan. In Ai River water concentrations up to 87,100 ng/l were reported. Serum concentrations in turtles collected in Ai River ranged from 47.1 to 115.6 ng/l, the corresponding BCF_{serum} values ranged from 0.9 to 2.9. In Taisyo River water concentrations of 42.3 and 63.4 ng/l (two samples) and 9800 ng/l (one sample) were detected. Serum concentrations of 0.4 and 1.0 ng/l were reported for the turtles collected in low water concentration sides, and 7.6 ng/l were reported for turtles collected in high water concentration sides; corresponding BCF_{serum} of 10-15.8 and 0.8 to 15.8 were reported with surface water concentrations ranging from 21.8 to 87,100 ng/l. However, as the wild turtles' exposure to PFOA was probably not limited to surface water only, the BCFs reported by Morikawa et al (Morikawa et al., 2005) may actually be BAFs.

Quinete et al. investigated the accumulation of PFOA in mussels (n=3-4), fish (7-15), and dolphins (n=10) at different sampling sites in south eastern Brazil. BCFs (BAFs) were calculated based on PFOA concentrations measured in water and fish collected from the sample area. Up to 3.3 ng L^{-1} PFOA were found in water. BCFs (BAFs) for different species ranged from 0.9 (croaker) to 266 (mussel) (Quinete et al., 2009).

Loi et al. investigated a subtropical pelagic food web in a nature reserve including phytoplankton (n=1), zooplankton (n=2), gastropod (n=3), worm (n=2-3), shrimp (n=2-3), fish (n=2-6), and water bird (n=3). Samples were collected between 2008 and 2010. Surface water (n=12) and sediment samples (n=6) were collected concurrently with the biota samples. Livers samples from water birds were all collected in 2003. A BAF for the phytoplankton for PFOA of 292 was derived (Loi et al., 2011).

Table 9: Examples of measured bioaccumulation factors (BAF) of PFOA

Location	Species (tissue)	BAF	Reliability	Reference
Laboratory	juvenile rainbow trout (Carcass)	0.038	2	(Martin et al., 2003b)
Brazil, Paraiba do Sul River	Scabbardfish	2.2 - 11	2	(Quinete et al., 2009)
Brazil, Paraiba do Sul River	Croaker	18 - 96		
Brazil, Guanabara Bay	Scabbardfish	1.8 – 4.4		
Brazil, Guanabara Bay	Croaker	0.9 – 2.8		
Brazil, Guanabara Bay	Mullet	8.1 - 14		
Brazil, Guanabara Bay	Mussels	63.5 - 266		
Japan, Ai River	Turtles	0.9 – 2.9	2	(Morikawa et al., 2005)
Japan, Taisyo River	Turtles	0.8 – 15.8		
Mai Po Marshes Nature Reserve	Phytoplankton	292	2	(Loi et al., 2011)

Conclusion: Most BAFs for PFOA are below 2000, indicating no bioaccumulation in aquatic organisms. Again, the notable water solubility of PFOA may enable fish and mussels to quickly excrete this substance via gill permeation, facilitated by the high water throughput (Kelly et al., 2004; Martin et al., 2003a; Martin et al., 2003b).

3.3.2.3 Biota-sediment accumulation factors (BSAFs)

For evaluation the bioaccumulation potential of chemicals also biota-sediment accumulation factors (BSAFs) can be used. BSAFs are field-based measurements for the chemical concentration in the organism and the sediments calculated according to the following equation:

$$BSAF = \frac{C_{Biota}}{C_{Sediment}}$$

Whereas C_{Biota} is the chemical concentration in the organism at steady-state, and $C_{Sediment}$ is the sediment chemical concentration at steady-state (Conder et al., 2011).

For assessing the bioaccumulation from fresh water sediments (n=3) a study using oligochaete *Lumbriculus variegatus* was commenced (Higgins et al., 2007). This benthic-dwelling worm species is a deposit feeder and serves as an entry point for sediment-bound contaminants into food webs. During the screening one uncontaminated field sediment, laboratory-spiked with PFOA, and two contaminated field sediments were applied, respectively. After attaining steady state (56 days) in all cases the calculated BSAFs ranged from 0.95 to 0.52 and from 94 to 95 in a lipid-normalized approach. These results indicate an uptake of PFOA during worm´s sediment ingestion.

Table 10: Biota-sediment accumulation factors (BSAF) analyzed with *Lumbriculus variegatus*

Location	Species (tissue)	BSAF		Reliability	Reference
		Lipid normalized	non lipid-normalized	2	
Downstream from two WWTP, California	*Sediment 1 (CA1 (56 days*	95 ± 20	0.74 ± 0.12		(Higgins et al., 2007)
	Sediment 2 (CA2 (56 days	94 ± 14	0.52 ± 0.07		
Laboratory	estimated steady-state values	33 ± 12	0.95 ± 0.13		

Conclusion: Only one study is available for BSAFs for PFOA. The lipid normalized steady state BSAFs are above 1 and support that PFOA bioaccumulates in *Lumbriculus variegates*. The available non lipid normalized data are, however, below 1.

3.3.2.4 Biomagnification factors (BMFs)

Besides bioconcentration also biomagnification describes the potential of a chemical to bioaccumulate. Biomagnification factors (BMFs) can be measured in the laboratory in a fashion similar to that used in the OECD and US-EPA bioconcentration test protocols. Organisms are exposed to a chemical preliminary via diet. The BMF test typically includes an uptake phase, where

levels of chemicals are followed over time, ideally until the chemical concentration in the organism no longer changes with time (i.e., reaching the steady-state). If a steady-state cannot be reached in the experiment, the uptake phase is followed by a depuration phase where organisms are exposed to uncontaminated food. The rate of decline in chemical concentration over time measured in the depuration phase can then be used to derive the chemical uptake rate from which a hypothetical steady-state concentration can be estimated (Conder et al., 2011).

The laboratory-derived BMF is calculated using the ratio of the chemical concentrations in the test animals at steady-state and their diet:

$$BMF = \frac{C_{biota}}{C_{diet}}$$

where chemical concentration in the organism (C_{biota}) and its diet (C_{diet}) are appropriately normalized, if needed, (e.g., lipid- or protein-normalized) (Conder et al., 2011).

BMF values based on field studies are based on the ratio of the concentration in the predator and the prey:

$$BMF_{(field)} = \frac{C_{predator}}{C_{prey}}$$

There are several uncertainties concerning field based BMFs similar to field based trophical magnifacation factors which regard food webs. There are biological, ecological factors which can influence the outcome of a BMF. Additionally as there is no standard procedure so far how to conduct such filed studies and therefore different study designs may too have an influence. The uncertainties of field studies have been addressed and discussed by Borga et al. (2011). As the authors actually refer to field based trophical magnifacation factors a summary of the discussion has been included in chapter 3.3.2.5 trophic magnification factors.

Problems arise with increasing body size of predators because analysis is based on tissue or serum samples. Whole-body analysis is not feasible for ethical reasons, i.e. a whole whale would be needed, and due to the challenging logistics with respect to sampling and laboratory constraints. Therefore, some of the derived BMF-values are restricted to certain tissue samples rather than whole body samples. BMF values based on liver samples may be over estimative. From a toxicological perspective concentrations in individual organs, such as the liver, may be more relevant when the potential for direct organ-specific toxicity (i.e., liver toxicity) is being predicted. Whole body values may be estimated if the tissue mass fraction is known for the organism regarded. There may however be some uncertainties due to inter individual and geographical differences (Houde et al., 2006).

At present no internationally accepted trigger value for BMF exists. The question whether only enrichment of a substance in predator proofs biomagnification or whether transfer from prey to predator already may be sufficient still is up for discussion. Additionally, experiences with revision or development of test guidelines show that even substances known to be bioaccumulative may show only BMF < 1 in laboratory test systems. However, keeping this in mind a BMF ≥ 1 will be used here as trigger value for the sake of decision making. BMFs for PFOA are summarized in Table 11.

Transfer of PFOA was elucidated in Lake Ontario (Martin et al., 2004b) including one 4-membered pelagic food chain. Whole body samples were collected. Two macroinvertebrates (*Diporeia* and *Mysis*) were considered as primary prey whereas rainbow trout inhabited the top predator´s position. Lake trout samples were taken at various locations and years (1980-2001) in Lake Ontario. Seven samples were selected every three years (i.e. 7 individual fish samples per year). Forage fish species, including sculpin, smelt, and alewife, were collected on October 9th 2002 at an offshore site near Niagara-on-the-Lake, Lake Ontario. In both exemplary food chains no stepwise as well as overall biomagnification could be proven. Due to the inherent uncertainties correlated with constitution of diet 4 individual combinations of rainbow trout and its prey were regarded. In all examples BMF ranged between 0.02 and 0.63 (Table 11). As this study was conducted with fish uptake of PFOA may not have occurred exclusively over diet but also over the gills. Thus the factors may be more accurately addressed as BAF. A striking finding of this study was the unexpectedly high content of PFOA in both macro invertebrates occupying the lowest trophic level. Proportions in *Diporeia* were as high as 90 ng/g and the mechanism leading to this exceptional accumulation still needs to be unravelled. As a consequence sculpin as *Diporeia´*s consecutive predator still shows significant levels of PFOA (44 ng/g). Although no biomagnification can be

proven, accounting for this elevated levels in *Diporeia* PFOA is still arousing suspicion of bioaccumulation.

Tomy et al. analysed an East Arctic food chain also including marine mammalians (n=5-7). Again, as outlined in the previous investigation, out of all examined organisms zooplankton (n=5) as the initial part of a food web exhibited the highest level of PFOA (2.6 ng/g). For consecutive segments of food chains, based on zooplankton, BMF values were calculated far below 1 (Table 11). Samples were taken from different years. This may influence the interpretation of the food web transfer due to temporal changes of the PFC concentration. On the other hand the Arctic as a remote area may be less prone to temporal changes and the existence of point sources there is unlikely. Problems arise with increasing body size of predators because analysis is based on tissue or serum samples. Whole-body analysis is not feasible for ethical reasons and due to the challenging logistics with respect to sampling and laboratory constraints. Therefore, for walrus, narwhale and beluga whale only liver concentrations were assessable. From a toxicological perspective concentrations in individual organs, such as the liver, may be more relevant when the potential for direct organ-specific toxicity (i.e., liver toxicity) is being predicted. However, in order to gain comparable factors recalculation or extrapolation from liver or serum concentrations to whole body burdens is necessary though the required estimation may imply uncertainties. Such an estimation was, however, not conducted in this study. Therefore, the resulting BMFs will probably be overestimated and the three stated BMFs exceeding one have to be regarded with precaution (Table 11) (Tomy et al., 2004).

Tomy et al. also investigated beluga whale, ringed seal, fish pelagic amphipod and arctic copepod of the Western Canadian Arctic. The animals selected were from the sample archived repository at Fisheries and Oceans, Canada. Blubber and liver of beluga (n = 10, all males,) from Hendrickson Island and ringed seal (n = 10, all males) from Holman Island were collected in 2007 and 2004, respectively. Fish species collected in 2004 and 2005 included the marine pelagic Arctic cod (n = 10) from the Amundsen Gulf, the marine coastal Pacific herring (n = 10) from the Mackenzie Shelf and the anadromous Arctic Cisco (n = 9) from the Mackenzie estuary. The marine pelagic amphipod Themisto libellula (pooled samples, n = 2) and the marine Arctic copepod Calanus hyperboreus (pooled samples, n = 5) were collected in 2004 from the eastern Beaufort Sea and Amundsen Gulf region. As the authors state themselves differences in sampling years may influence the interpretation of the food web transfer. Again some of the derived BMF-values are restricted to the liver and the resulting BMF may be over estimative. The BMF-values reported range from 0.1 for ringed seal liver/arctic cod liver and 2.2 for arctic cod liver/marine arctic copepod (Tomy et al., 2009).

Also Houde et al., assume an overestimation of the BMF if it is not based on whole body. Thus, they claim that utilization of serum or liver concentrations of dolphins will overestimate the BMF by a factor of 10-30. In the course of the study PFOA serum concentrations in bottlenose dolphins were examined at two different habitats. Charleston Harbor and its tributaries (i.e., the Cooper, Ashley, and Wando rivers) and the Stono River estuary, South Carolina, and in Sarasota Bay, Florida. Marine water (n=18), surface sediment (n=17), Atlantic croaker(n=3), pinfish(n=4), red drum (n=8), spotfish (n=10), spotted seatrout (n=11), striped mullet (n=8), and bottlenose dolphin samples (n=24) were collected around the Charleston Harbor area. Marine water (n=10), surface sediment (n=8), zooplankton 8n03), sheephead (n= 3), pigfish (n= 10), pinfish (n=10), striped mullet (n=9), spotted seatrout (n=8), and bottlenose dolphin samples 8n=12) were collected at Sarasota Bay. Dolphin plasma, skin, and teeth were collected from both locations. Additionally, dolphin tissue samples (i.e., liver, kidney, muscle, lungs, heart, thyroid, and thymus) were collected of recently deceased bottlenose dolphins from Sarasota Bay (2002, n = 1, male, 233.5 kg) and Charleston (2003, n = 1, female, 708.4 kg). Additional liver (n = 6) and kidney (n = 6) samples collected from stranded bottlenose dolphins were available at Sarasota Bay. Samples were collected

between 2002 and 2004. In a more industrialized location showing averaged PFOA concentrations of 9.5 ng/L in water serum concentrations of 43 ng/g were measured. Unfortunately, concentrations in other representative fish species originated from different years, thus, entailing additional uncertainty when assessing BMF through the food chain. It may be assumed that media and biota were continuously exposed to PFOA in this area throughout the years. Regardless of this, BMFs ranging from 1.8 to 13 for seven individual dolphin/prey relationships were stated using recalculated PFOA whole body burdens for dolphin. But it has to be pointed out that averaged PFOA concentration in all other fish were generally below 2 ng/g and exhibited high standard deviation. At the other less contaminated location (3.6 ng/g PFOA in water) serum concentration in dolphin was analyzed for 3.4 ng/g and whole body burden in all other fish were below 0.5 ng/g (Houde et al., 2006a).

Butt et al. conducted a study in the Canadian Arctic. Ringed seal liver samples (n=10 per site) were provided by local hunters from 11 different locations in the Canadian Arctic. The age of the animals was determined via tooth aging and for a few samples the age was estimated using length-age correlations. Stable isotope analysis was done with ^{15}N to ^{14}N and ^{13}C to ^{12}C. Based on liver samples from polar bears obtained from another study and ringed seal data measured in this study BMFs were calculated. The polar bear sample sites were associated with ringed seal populations. In four different regions these factors ranged from 45 to 125 with a mean of 79. However, the sample collection year for ringed seal populations varied from 2002 to 2005, and it is possible that interpretation of spatial trends may be confounded by temporal variations of PFC concentration within seal populations (Butt et al., 2008).

Various predator prey relationships in the Westerschelde (Netherlands) were investigated by van Heuvel-Greve and co-workers. Samples (n=3-4) were collected in 2007 and 2008. The trophic level was estimated based on stable isotope (15N) analysis. BMFs were considerable for harbor seal as well as for the sediment dwelling flounder (Environment Canada, 2012; van den Heuvel-Greve et al., 2009)

Table 11: Biomagnification factors (BMF) for PFOA

Location	Species (tissue)	BMF	Reliability	Reference
Lake Ontario	Lake trout/alewife	0.63	2	(Martin et al., 2004b)
Lake Ontario	Lake trout/smelt	0.50		
Lake Ontario	Lake trout/sculpin	0.02		
Lake Ontario	Lake trout/prey (weighted)	0.41		
US, South Carolina	Seatrout/pinfish	7.2	2	(Houde et al., 2006a)
US, South Carolina	Dolphin (whole, estimated)/striped mullet	13		
US, South Carolina	Dolphin (whole, estimated)/pinfish	13		
US, South Carolina	Dolphin (whole, estimated)/red drum	2.7		
US, South Carolina	Dolphin (whole, estimated)/atlantic croaker	2.3		
US, South Carolina	Dolphin (whole, estimated)/spotfish	6.4		
US, South Carolina	Dolphin (whole, estimated)/seatrout	1.8		
Eastern Arctic	Walrus (liver)/clam	1.8	2	(Tomy et al., 2004)
Eastern Arctic	Narwhal (liver)/arctic cod	1.6		
Eastern Arctic	Beluga whale (liver)/arctic cod	2.7		
Eastern Arctic	Beluga whale (liver)/ redfish	0.8		
Eastern Arctic	Black-legged kittiwakes (liver)/arcitc cod	0.3		
Eastern Arctic	Glaucous gulls (liver)/arctic cod	0.6		
Eastern Arctic	Arcitc cod / zooplankton	0.04		
Canadian Arctic	Polar bear (liver)/ ringed seal (liver)	45-125	2	(Butt et al., 2008)
Western Canadian Arctic	Ringed seal (liver)/ arctic cod (liver)	0.1	2	(Tomy et al., 2009)
Western Canadian Arctic	Beluga whale (liver)/ arctic cod (liver)	0.9		
Western Canadian Arctic	Beluga whale (liver)/ Pacific herring (liver)	1.3		
Western Canadian Arctic	Beluga whale (liver)/ arctic cisco (liver)	0.7		
Western Canadian Arctic	Arctic cod (liver)/ marine arctic copepod (whole body)	2.2		
Western Canadian Arctic	Arctic cod (liver)/ marine pelagic amphipod (whole body)	0.8		
Westerschelde, Netherland	Zooplankton/ herring	1.6	2	(Environment Canada, 2012; van den Heuvel-Greve et al., 2009)
Westerschelde, Netherland	Herring/ sea bass	0.6		
Westerschelde, Netherland	Herring/ harbour seal	14		

Westerschelde, Netherland	Sea bass/ harbour seal (benthic food web for harbour seal	23		
Westerschelde, Netherland	Peppery furrow shell/ flounder	31		
Westerschelde, Netherland	Lugworm/ flounder	0.03		
Westerschelde, Netherland	Flounder/ harbour seal (pelagic food web for harbour seal)	3.8		
Brazil, Paraiba do Sul River	Croaker (liver) or scabbardfish (liver)//tucuxi dolphin (liver)	1.3-2.6	2	Quinete et al., 2009

Conclusion: The biomagnification potential of PFOA was investigated in several field studies. Especially for dolphin, walrus, narwhal, polar bear, arctic cod and harbour seal, BMFs greater than one have been reported, indicating biomagnification within the food webs.

3.3.2.5 Trophic magnification factors (TMFs)

The trophic magnification factor (TMF) is a measure to evaluate biomagnification occurring in food webs. In the Guidance Document on Information Requirements, Chapter R.7.10.1.1, TMF is defined as the concentration increase in organisms with an increase of one trophic level. According to Conder et al., TMFs represent some of the most conclusive evidence of the biomagnification behaviour of a chemical substance in food webs (Conder et al., 2011). Again, a TMF greater than one indicates accumulation within the food chain.

There are several uncertainties concerning TMFs. These have been addressed and summarized by Borga et al. (2011). There are biological factors such as the differences between poikilotherms and homeotherms, sex, different energy requirements, different abilities to metabolize chemicals and slow or fast growing organisms.

Steady state between a consumer and its diet is assumed. However, as opportunistic feeders wild animals vary their diet over seasons or with life stage and point sources may influence observed TMFs. Additionally, apart from the diet there is always the possibility of a direct uptake of the substance under scrutiny and the relative importance of food versus e.g. water exposure can influence the magnitude of the TMF. The position in the food web is quantified using relative abundances of naturally occurring stable isotopes of N ($^{15}N/^{14}N$, referred to as $\delta^{15}N$). However the relative abundance of these isotopes and thus the determination of the trophical level and TMF is influenced by the physiology of the organism and its life trait history. Rapid growth with a higher protein demand for new tissue leads to lower enrichment factors than those with slower growth rates. Insufficient food supply and fasting and starvation leads to catabolism of body proteins and an increase of ^{15}N in organisms relative to those organisms with adequate food supply. There is no standard procedure for the conductance of TMF field studies. Hence, the conductance and sampling may vary between different studies. Disproportionate sampling of the food web or unbalanced replication of samples may significantly influence the TMF.

Additionally, as already discussed in the BMF chapter sample collection is often restricted to tissue or serum samples with increasing body size of predators due to ethical reasons and due to the challenging logistics with respect to sampling and laboratory constraints.

The following text in italic letters was copied from the OECD SIDS Initial Assessment Report for PFOA (OECD, 2006):

Martin et al. (Martin et al., 2004b) examined PFOA contents in the food web from Lake Ontario (Canada). Adult lake trouts (top predator) were collected at various years and locations in Lake Ontario. Samples of prey fish (sculpins, smelts and alewifes) and macroinvertebrates (Mysis sp., Diporeia sp.) were collected at one location in October 2002. Lake trout samples analyzed represented individual whole fish homogenates. The other species were processed as composites of whole individuals. The mean PFOA content in Diporeia sp. and sculpin was 90 ng/g and 44 ng/g, respectively. In the other fish samples contents of 1.0 to 2.0 ng/l and in Mysis sp. of 2.5 ng/g could be detected. The authors note that Diporeia sp. is a benthic invertebrate species, and sculpins feed mainly in the benthic environment. Benthic contamination may therefore be the source of contamination of this food web. As PFOA content in predators is lower than in prey species trophic biomagnification of PFOA in the food web of Lake Ontario is unlikely to occur.

Trophic transfer of PFOA and other related perfluorinated compounds was examined in a Great Lakes benthic foodweb including water – algae – zebra mussel – round goby – smallmouth bass. In addition, perfluorinated compounds were measured in livers and eggs of Chinook salmon and lake whitefish, in muscle tissue of carp, and in eggs of brown trout. Similarly, green frog livers, snapping turtle plasma, mink livers, and bald eagle tissues were analyzed to determine concentrations in higher trophic-level organisms in the food chain. Biotic samples were collected from several rivers in Michigan and in the Calumet River in Indiana, USA. PFOA-concentrations in two of the sampling sites, Raisin River and St. Clair River, were 14.7 and 4.5 ng/l, respectively. The concentrations of PFOA in all tissue samples were above detection limit but below the LOQ. Therefore, biomagnification of PFOA in the Great Lakes benthic foodweb is unlikely occur (Kannan et al., 2005).

Houde et al. investigated the food web of bottlenose dolphins. The results are summarized in Table 12. The authors sampled different biota, i.e. Atlantic croaker (n=3), pinfish (n=4), spotfish (n=10), spotted seatrout (n=11), striped mullet (n=8) and samples from bottlenose dolphins (n=24), as well as water (n=18, samples analyzed in duplicate) and surface sediment (n=17, samples analyzed in triplicate). Sample collection was conducted between 2002 and 2004. Based on stable isotope (^{15}N) analysis the trophic level of each biota sample was determined. PFOA was analysed in plasma and liver of dolphins and afterwards a whole body burden was calculated. For prey whole body homogenates were analysed for PFOA. The TMF for Arctic beluga whale was calculated on the basis of liver samples of beluga whale (n=5) and narwhal (n=5) from another study. For estimating the trophic magnification on the basis of the whole body, the weight of the animals tested in the former study was estimated, as well as the weight of their organs and plasma volume. It was assumed that the anatomy of dolphin and beluga is similar. The available dolphin anatomy data such as organ proportion compared to the entire body were extrapolated to beluga and narwhal. The authors conclude that the TMF for PFOA is >1 when using liver measurements and <1 when using whole marine mammal body burdens. The authors conclude further, that TMFs based on liver samples overestimate biomagnification. However, the calculated TMFs are due to above described estimations not reliable for the Arctic and should therefore be used with caution (Houde et al., 2006a).

Kelly et al. measured PFOA in the Canadian Arctic marine food web. Concentrations in sediment (n=9) and in different organisms (lichens, macroalgae (n=6), bivalves, fish (n=3-6)) and tissues and organs (stomach contents, liver, muscle, blubber and/or milk) of common eider ducks (n=5), seaducks (n=4), and marine mammals beluga whales and ringed seals to calculate TMFs (Table 12). Sample collection was conducted between 1999 and 2003 along the eastern Hudson Bay coastline in close proximity to the Inuit village Umiujaq. PFOA was measured in different tissues/fluids of the beluga whale including blood (n=18), muscle (n=18), liver (n=22), milk (n=6) and also in foetuses (n=2). The authors showed that PFOA especially accumulates in protein rich compartments

such as blood and liver and that the TMF of perfluorinated compounds such as PFOA correlates with the partitioning behaviour between protein and water and protein and air. Comparisons of different food webs show that the TMF is below one in the case of piscivorous food webs if air breathing organisms are excluded but becomes larger than one if air breathing organisms are taken into account (Kelly et al. 2009).

TMFs for PFOA are summarized in Table 12.

Table 12: Trophic Magnification Factors (TMF) of PFOA

Location	Species (tissue)	TMF	Reliability	Reference
Lake Ontario	Diporeia/slimy sculpin	0.37	2	(Martin et al., 2004b)
Lake Ontario	Mysis/alewife/rainbow smelt/lake trout	0.58		(Martin et al., 2004b)
US, South Carolina	Dolphin plasma croaker, pinfish, spotfish, spotted seatrout	13 ± 22	2	(Houde et al., 2006a)
US, South Carolina,	Whole dolphin burden	6.3 ± 6.7		(Houde et al., 2006a)
Arctic	Beluga Whale/narwhale liver	1.6 ± 3	3	(Houde et al., 2006a)
Arctic	Whole beluga whale/narwhale burden	0.3 ± 0.3	3	(Houde et al., 2006a)
Hudson Bay (north-eastern Canada	Sediment/ macroalgae/ bivalves/ fish/ seaduck/ beluga whale	2.33-4.61 1.4-2.64 (protein corrected)	2	(Kelly et al., 2009)
Hudson Bay (north-eastern Canada	Sediment/ macroalgae/ bivalves/ fish	0.3-0.53 (protein corrected)		
Westerschelde, Netherland	Sea bass/ harbour seal (benthic food web for harbour seal	1.2	2	(Environment Canada, 2012; van den Heuvel-Greve et al., 2009)
Westerschelde, Netherland	Flounder/ harbour seal (pelagic food web for harbour seal)	1.2		(Environment Canada, 2012; van den Heuvel-Greve et al., 2009)
Mai Po Marshes Nature Reserve	Phytoplankton/zooplankton/gastropod/worm/shrimp/fish/waterbird liver	0.93-1.07	2	(Loi et al., 2011)

Conclusion: A number of field studies are available which analyzed the trophic magnification potential of PFOA. For food chains of dolphin, beluga whale, and harbour seal, TMFs greater than one have been reported, indicating trophic biomagnification.

3.3.3 Terrestrial bioaccumulation

Food web analyses covering also terrestrial mammals and birds have been performed. Martin et al. examined PFOA proportions in biota from Canadian Arctic. Only liver samples from polar bear exhibited significant PFOA levels (3-13 ng/g) whilst in 4 other terrestrial mammals and all of the 3 investigated bird species levels remained below the limit of detection (< 2 ng/g) (Martin et al., 2004a).

An analogue result was stated by Kannan et al. (Kannan et al., 2005) indicating absence of PFOA in liver samples of predatory birds and presence only in 1 out of 8 piscivorous mammals (mink). In general, PFOA is occasionally detected in high trophic level avian predators, whereas it is frequently found in piscivorous mammals. In particular predatory birds and mammals at higher trophic levels usually inhabit a large geographic home range and their flexible migratory patterns impede a collection of collocated samples of prey and predator. Despite this, piscivorous mammals show a more residential behaviour and the proximate local association to their prey allows for proposing a more realistic trophic correlation of samples.

In a study undertaken by the German Environmental Specimen Bank (ESB), eggs from herring gull and from cormorants were analysed according their contamination with per- and polyfluorinated compounds. Herring gulls are omnivorous and opportunistic top predators of the North and Baltic Sea marine ecosystem, and eggs are routinely collected for the German ESB in the same regions where mussels and/or fish are sampled. PFOA values in herring gull eggs ranged from 6.5 to 118 ng/g ww at the North Sea), and from below the level of quantification up to 2.8 ng/g ww at the Baltic Sea). The cormorants from the Baltic Sea site Heuwiese are nesting on the ground in the neighbourhood of herring gull nest. PFOA was one of the chemicals frequently detected above the limit of quantification. The PFOA levels ranged from 0.9 to 1.8 ng/g ww. The levels in samples from the North Sea were higher than those from the Baltic Sea. Additionally, eggs of rook and feral pigeon from terrestrial ecosystems were analyzed regarding their burden of per- and polyfluorinated compounds. The values where very low compared to the ones from the coast. It was hypothesized that differences in per- and polyfluorinated compounds levels between aquatic and terrestrial birds are caused by different exposure pathways (Rüdel et al., 2011)

Swedish peregrine falcon eggs collected between 1974 and 2007 were also analyzed according to their PFC load. In contrast to the study of Rüdel et al. (2011), PFOA could not be detected above limit of quantification (Holmström et al., 2010). Ahrens and co-workers investigated PFCs in eggs from tawny owl from Norway collected from 1986 to 2009. PFOA was detected in 8% of the samples (Ahrens et al., 2011).

Müller et al. conducted a terrestrial food web study consisting of lichen and plants, caribou, and wolves from two remote northern areas in Canada. Liver, muscle, and kidney samples (n=7 Porcupine herd food web and n=10 for the Bathurst food web) from two caribou herds were collected; from the Porcupine herd in northern Yukon Territory and the Bathurst herd in the Northwest Territories (NWT)/western Nunavut . Wolf (n=6 Porcupine herd food web and n=10 for the Bathurst food web), lichen, and plant samples were collected in the same region as the caribou. Plant samples included cottongrass, aquatic sedge, willow, moss, and mushrooms. Liver and muscle samples were collected from the sampled wolves. Lichen, moss and mushrooms were collected as a whole grass and willow without roots. Plant samples are from the same season (summer 2008 in Porcupine or summer 2009 in Bathurst) whereas wolf and caribou samples are from different years (2007 and 2010 in Porcupine and 2008 and 2007 in Bathurst). Some samples are not from the same season. This food web is considered as relatively well documented example (Kelly and Gobas, 2003). The study illustrates a considerable carry over between plants and caribou. Caribou are a major human food source in numerous arctic communities. This food-chain may also be considered comparable to the pasture-cow food-chain in temperate regions. The results of the study, BMFs as well as TMFs are shown in Table 13 and Table 14. Tissue concentrations and whole body concentrations were used for calculations. Tissue based BMFs differ considerably. Therefore it is concluded that BMFs based on whole body concentrations are more appropriate (Müller et al., 2011).

Table 13: BMFs for PFOA in a remote terrestrial food chain (from two different locations)

Species (tissue)	BMF	Reliability	Reference
Caribou (muscle)/lichen	0.9 ± 0.4	2	(Müller et al., 2011)
Caribou (liver)/lichen	11 ± 1.2		
Wolf (muscle)/caribou muscle	3.8 ± 1.5, 2.6 ± 0.8		
Wolf (liver)/caribou liver	0.9 ± 0.3		
Caribou (whole)/lichen	1.4 ± 0.4, 2.6 ± 0.5		
Caribou (whole)/vegetation	1.8 ± 0.7, 0.3 ± 0.1		
Wolf (whole)/caribou (whole)	2.4 ± 0.6, 2.1 ± 0.5		

Table 14: TMFs for PFOA in a remote terrestrial food chain (from two different locations)

Species (tissue)	TMF	Reliability	Reference
Wolf (liver) /caribou (liver)/lichen	2.4 ± 0.1, 2.2 ± 0.1	2	(Müller et al., 2011)
Wolf (whole)/caribou (whole)/lichen	1.3 ± 0.1, 1.3 ± 0.1		
Wolf (whole)/caribou (whole)/vegetation	1.1 ± 0.1, 1.3 ± 0.1		

Conclusion: The terrestrial BMF and TMF of PFOA is greater than one for the remote Arctic food chain lichen – caribou – wolf, indicating trophic biomagnification.

3.3.4 Summary and discussion of bioaccumulation

The estimation of bioaccumulation based on partition coefficient K_{OW} appears to be inappropriate for PFOA, because the experimental determination of K_{OW} is impeded by strong surface activity of PFOA and calculation of K_{OW} using QSAR methods rely on physico-chemical parameters which are not completely validated for PFOA. As shown from binding assays and analyzing distribution pattern in aquatic animals PFOA preferentially binds to proteins in blood and liver (Ishibashi et al., 2008) (Ahrens et al., 2009b).

Reported BCFs for fish for PFOA are in the range from 1.8 for fathead minnow to 27 for carp. Bioaccumulation factors (BAFs) have been shown to be in the range from 0.038 for rainbow trout to 266 for mussels. Both of the factors describe the accumulation for aquatic species. The BCF is typically measured in the laboratory, whereas the BAF is measured in field studies.

The numeric criterion as suggested in REACH Annex XIII as a bioaccumulative substance is not fulfilled. It is not clear if fish in fact takes up PFOA or if the notable water solubility of PFOA may enable fish to quickly excrete this substance via gill permeation, facilitated by the high water throughput. However, this possible excretion pathway does not exist for air breathing animals (Kelly et al., 2004; Kelly et al., 2007). Hence, bioconcentration values in fish may not be the most relevant endpoint to consider. Therefore, the numerical bioaccumulation (B) criterion defined in the REACH regulation Annex XIII is not suitable for PFOA.

PFOA is frequently analyzed in environmental monitoring studies. PFOA has been found in piscivorous mammals, and occasionally detected in high trophic level avian predators (Kannan et al., 2005). In herring gull eggs, e.g. PFOA concentrations were measured in the range from 6.5 to 118 ng/g (ww) (Rüdel et al., 2011). Values in polar bear liver ranged from 3-13 ng/g (Martin et al., 2004b). Although, the focus of these studies was not to measure the bioaccumulation potential the fact that PFOA is ubiquitously present in terrestrial species especially in top predators and even in remote areas is of great concern.

It has been shown, that air-breathing organisms are more likely to biomagnify PFOA than water breathing organisms such as fish (Kelly et al., 2009). Piscivorous mammals (mink, seal, and dolphin) exhibited significant amounts of PFOA mainly accumulated in serum and liver. There are studies which report trophical magnification factors (TMFs) or biomagnification factors (BMFs) greater than one, indicating bioaccumulation of PFOA:

- For the food chains walrus (liver) / clam, narwhal (liver) /Arctic cod, and beluga (liver)/Arctic cod the BMFs are 1.8, 1.6, and 2.7, respectively, indicating bioaccumulation (Tomy et al., 2009).
- BMFs ranging from 1.8 to 13 for seven individual dolphin/prey relationships were stated using recalculated PFOA whole body burdens for dolphin (Houde et al., 2006b).
- Kelly et al. 2009 measured PFOA in the Canadian Arctic marine food web (sediment and in different organisms (macroalgae, bivalves, fish, seaducks, and marine mammals). A TMF of 3.28 was one results of the study. The protein-normalized value is 1.93.
- Bioaccumulation was also studied in lichen, caribou, and wolf, living in the remote Canadian environment. Measured BMFs were in the range from 0.9 to 11 and indicate bioaccumulation. Calculated TMFs were in the range from 1.1 to 2.4, indicating trophic magnification, too (Müller et al., 2011).

In the literature it was discussed that the BCF is less accurate in quantifying bioaccumulation than TMFs and BMFs in terms of dietary accumulation (Borga et al., 2011; Gobas et al., 2009; Weisbrod et al., 2009). According to Conder et al. (2011), TMFs represent some of the most conclusive evidence of the biomagnification behaviour of a chemical substance in food webs (Conder et al., 2011). BCFs reflect a chemical equilibrium between water and organism. In addition, BCFs apply only to aquatic organisms in a laboratory context. For air breathing organisms it has been shown that BCFs and K_{OW}-predicted BCFs are inadequate for assessing bioaccumulation (Conder et al., 2011; Czub and McLachlan, 2004; Kelly and Gobas, 2001; Kelly and Gobas, 2003; Kelly et al., 2007; Kitano, 2007). BMFs present only a single trophic transfer, since they describe enrichment of chemicals between predator and prey. TMFs, however, provide a characterization of the average degree of biomagnification that occurs in an entire food web by incorporating multiple food web interactions (Borga et al., 2011; Hop et al., 2002; Jardine et al., 2006).

Field studies are complex and therefore difficult to judge concerning their reliability. Each of the field studies presented here has its drawbacks due to sample collection in different years, the sampling of body tissues and fluids instead of whole body or uncertainty of prey constitution etc. and may not be considered as a standalone proof for the bioaccumulation potential of PFOA. Overall, these studies suggest that PFOA can biomagnify in the food chain as indicated by biomagnifications factors and trophic magnification factors larger than one. Additionally, it is of special concern that PFOA biomagnifies in endangered species as shown for the polar bear and in animals which are likely to become endangered in the near future (narwhale and beluga whale).

Taken together in a weight of evidence approach the data presented can be considered overall conclusive. Environmental studies suggest that PFOA can biomagnify in the food chain. It is of special concern that PFOA biomagnifies in endangered species as shown for the polar bear and in animals which are likely to become endangered in the near future (narwhale and beluga whale).

4 HUMAN HEALTH HAZARD ASSESSMENT

4.1 Toxicokinetics (absorption, metabolism, distribution and elimination)

4.1.1 Non-human information

Absorption
Absorption in male rats was studied following administration of a single oral dose of ^{14}C- PFOA (11mg/kg), and at least 93% of the total ^{14}C was absorbed at 24 hours (Gibson and Johnson, 1979).

In another study, male and female rats were exposed via nose-only to aerosol atmospheres of PFOA (Hinderliter et al., 2006). The study was comprised of two separate experiments, a single inhalation exposure and repeated inhalation exposures for 3 weeks. The results demonstrated that the pharmacokinetic properties of inhaled PFOA in male and female rats are similar to those observed in male and female rats following oral dosing with PFOA.

Penetration of APFO through rat and human skin was tested in an in vitro study and by the end of the 48-h exposure period, only a negligible amount of the total APFO applied (0.048 ± 0.01%) had penetrated through human skin (Fasano et al., 2005). The steady-state penetration of APFO was approximately 34-fold faster through rat skin than human skin.

In conclusion, PFOA/APFO is well absorbed in laboratory animals following oral and inhalation exposure, and to a lesser extent following dermal exposure.

Metabolism
Carbon-fluorine bonds are among the strongest in organic chemistry, and PFOA has not been found to be metabolised (Lau et al., 2007).

In conclusion, PFOA has not been found to be metabolised.

Distribution and elimination
In a study on male and female mice, rats, hamsters, and rabbits the absorption, distribution and excretion of APFO was studied (Hundley et al., 2006). The laboratory animals were treated with a single oral dose of ^{14}C-APFO, and the excretion and tissue distributions were followed for 120 h (168 h in the rabbit). Substantial sex and species differences in the excretion and disposition of ^{14}C-radioactivity derived from ^{14}C-labeled APFO were observed. The female rat and the male hamster excreted more than 99% of the original ^{14}C-radio activity by 120 h after dosing; conversely, the male rat and the female hamster excreted only 39% and 60% of the original ^{14}C-radio activity, respectively, by 120 h postdosing. The male and female rabbits excreted the ^{14}C-radio activity as rapidly and completely as the female rat and the male hamster, whereas male and female mice excreted only 21% of the original ^{14}C-radio activity by 120 h postdosing. The rapid excretors (female rat, male hamster, and male and female rabbits) contained negligible amounts of ^{14}C in organs and tissues at sacrifice. The slow excretors exhibited the highest ^{14}C- concentrations in the blood and liver followed by the kidneys, lungs, and skin. Preferential sequestering of ^{14}C-labeled APFO in the fat was not observed in any of the species studied.

In a study on rats, ^{14}C-PFOA was administered orally and binding to plasma proteins was studied (Han et al., 2003). Most PFOA was found to be in protein-bound form in male and female rat plasma, and the primary PFOA binding protein in plasma was serum albumin. In the same study no significant difference was found between PFOA binding to rat serum albumin and PFOA binding to human serum albumin. PFOA has been demonstrated to undergo enterohepatic circulation in rats (Johnson et al., 1984).

The pharmacokinetics of PFOA in cynomolgus monkeys was studied in a six-month oral capsule dosing study of APFO and in a single dose intravenous study (Butenhoff et al., 2004b). During the repeated oral dosing, PFOA reached a steady concentration in the serum, urine, and feces within four weeks with concentrations increasing with dose in a nonlinear manner. Serum PFOA followed first-order elimination kinetics after the last dose. Urine was the primary elimination route. The PFOA elimination half life following either oral or intravenous dosing was approximately 20–30 days.

To develop understanding of the potential for gestational and lactational transfer of PFOA, female rats were dosed by oral gavage once daily with APFO starting on gestation day 4 and continuing until sacrifice (Hinderliter et al., 2005). Concentrations of PFOA in all biological samples were proportional to maternal dose. PFOA was detected in the embryo/foetus and placenta, and nursing pup and milk confirming placental and lactational transfer. Steady-state concentrations in milk were approximately 10 times less than those in maternal plasma. The concentration of PFOA in fetal plasma was approximately half the steady-state concentration in maternal plasma. The milk concentrations appeared to be generally comparable to the concentrations in pup plasma.

In conclusion, the highest concentrations of PFOA are found in blood, liver, kidney and lung. Urine is the primary route of excretion. There are large sex and species differences in the excretion of PFOA. PFOA is transferred to the foetus through the placenta and the offspring is exposed to PFOA from breast milk.

4.1.2 Human information

Levels of PFOA in human body fluids

PFOA has been found in human blood samples all around the world (Lau et al., 2007). In European populations, serum and plasma concentrations of PFOA in the range from <0.5 to 40 ng/mL have been reported (Vestergren and Cousins 2009, Fromme et al., 2009). For instance, the results of a Bavarian human biomonitoring study (n=365) with background exposed young adults showed PFOA concentrations of 0.5 to 19 ng/mL in blood plasma (Fromme et al., 2007).

Considerably higher levels have been found at two locations, in USA and in Germany, where the population had been exposed to PFOA contaminated drinking water (Emmet et al., 2006; Wilhelm et al., 2008). For the people living in the vicinity of a fluoropolymer production facility in Ohio, a median serum PFOA concentration of 354 ng/mL has been reported (Emmet et al., 2006). From the dependence of serum levels on the person's use of water, it was concluded that drinking water was the major route of exposure. In the same study group, markedly higher serum levels of PFOA were associated with working at the chemical plant that was the source of the contamination (Steenland et al., 2009). Workers who no longer worked at the plant had much higher PFOA levels than did non-workers but lower levels than those who continued working there. These findings are consistent with a gradual excretion of PFOA from the body after ending high exposure. Age showed a J-shaped relationship with serum PFOA, with higher levels in the young and the old subjects. In Germany, PFC contaminated material had been applied on a large agricultural area leading to the contamination of drinking water sources. Drinking water concentrations of PFOA ranged from 500 ng/L to 640 ng/L. Plasma PFOA levels were around 24 ng/mL in adult residents from the

contaminated area which was 4.4 (males) and 8.3 (females) times higher than PFOA levels from a control region (Wilhelm et al., 2008; Hölzer et al., 2009).

Very high serum concentrations have been reported in fluorochemical production workers with mean concentrations of PFOA in the range of 500 to 7,000 ng/mL depending on the type of work (Fromme et al., 2009). The highest serum level reported for PFOA was 114,100 ng/mL in 1995 (Fromme et al., 2009).

A recent Swedish study reported significantly elevated PFOA levels in humans after using fluorinated ski wax. Monthly blood samples were collected before the ski season, i.e., pre-season, then at four FIS World Cup competitions in cross country skiing, and finally during an unexposed 5-month post-season period (Nilsson et al., 2010a). The PFOA levels in three technicians with "low" initial levels of PFOA (<100 ng/mL in pre-season whole blood) increased from pre-season to post-season by 254, 134, and 120 %, whereas no increases in the blood levels were observed for the five technicians with "high" initial levels (>100 ng/mL in pre-season sample).

In a Norwegian study, serum samples from 13 professional male ski waxers were collected at three occasions (Freberg et al., 2010). The first blood sample was drawn at the end of season I (spring), the second at the beginning of season II (autumn) and the third at the end of season II (spring). The median concentration of PFOA was 50 ng/mL by the end of season I (range; 15-174 ng/mL), which is around 25 times higher than the background level. The median concentrations of PFOA sampled in the aerosol fractions were 15 mg/g dust (range: 5.6-38 mg/g). Precursor substances were not evaluated. A statistically significant positive association between years exposed as a ski waxer and concentration of PFOA in serum was observed. The reduction in the concentrations measured at the start of season II (autumn) compared to the end of season I (spring) was of statistical significance ($p < 0.05$), but was below 10%. This indicates long elimination half-lives of PFOA in humans.

Several factors could potentially affect the human blood levels of PFOA. In some publications addressing human blood levels of PFOA with life time, no correlation between PFOA concentration and age was reported (Calafat et al., 2007; Olsen et al., 2003; Olsen et al., 2004), while in other studies the concentration of PFOA in blood increased significantly with increasing age (Haug et al., 2010b; Haug et al., 2011a). In the US NHANES study, Calafat and co-workers (2007) found higher levels of PFOA in males at age 26 and 39 (fertile age), but not at age 55, compared to females. Similar findings have been observed in a Japanese study (Harada et al., 2004). In a study by Thomsen and co-workers relatively high levels of PFOA were found in breast milk. After breast-feeding for a year, the concentration of PFOA in the breast milk was reduced by more than 90%. This demonstrates a significant transfer of PFOA to breast-fed children and a significantly reduced PFOA level in the mothers (Thomsen et al., 2010). A highly reduced PFOA level in breast-feeding women may at least partly explain the lower levels of PFOA in females compared to males at fertile age (26 and 39 year) shown in the NHANES study.

Also, PFOA in diet is an important exposure source. It has been shown that people eating more shrimps have statistically significant higher levels of PFOA than people eating a smaller amount (Haug et al., 2010b). Other sources such as ski-waxing, prolonged use of proofing agents, indoor carpets and food contact materials may also be of importance. In a previous study, levels of PFOA in dust samples were highly correlated to serum levels in humans and the study indicated that inhalation of PFOA in the indoor environment may be a significant contributing source to total PFOA exposure (Haug et al., 2011a). As a result of different activities and age of fabrics and furniture, exposure via indoor environment may also vary between age groups. Taken together, breastfeeding, differences in diet, life style and indoor environment are important exposure factors not addressed in the studies by Calafat et al., and Olsen et al. and are factors that most likely will hide the measurable accumulation increase of PFOA with age (Calafat et al., 2007; Olsen et al.,

2003; Olsen et al., 2004). This is further supported by two Norwegian studies using multiple linear regression analyses to adjust for different contributing factors. In the Norwegian Fish and Game Study (n=175) levels of PFOA in serum from men and women increase statistically significant with age (Haug et al., 2010b). Also in a study with 41 women in the age of 25-45 years a statistically significant increase in the PFOA levels with age was found (Haug et al., 2011a). These two studies strongly indicate that PFOA levels increase with age, but that breast feeding, diet and indoor environment are important factors for PFOA exposure that need to be addressed in the evaluation of human exposure and accumulation of PFOA.

In a Norwegian time trend study, PFOA concentrations in serum were measured in samples collected in the period from 1977 to 2006. A nine-fold increase in the serum concentrations was measured for from 1977 to the mid 1990s where the concentrations reached a plateau before starting to decrease around year 2000 (Haug et al., 2009). This is in line with a decrease of PFOA blood concentrations reported by several studies from the USA (Vestergren and Cousins, 2009). Time trend of PFOA levels in archived human blood specimen from Germany has also been analysed (Wiesmüller and Gies 2011). In 1982, mean blood levels (standard deviation) of PFOA, were 4(2) ng/mL, concentrations were highest in 1986 (7(4) ng/mL) and fluctuated more or less around 5 ng/mL until 2007. The decrease found in Norwegian and American studies could not be confirmed for Germany.

In conclusion, PFOA is present in human blood in the general population and elevated concentrations are seen following specific exposure to PFOA, either via the environment (e.g contaminated drinking water) or occupationally. Further, breastfeeding, diet, life style and indoor environment influences the human blood levels and are important to take into consideration.

Gestational and lactational transfer

Several studies have reported detectable concentrations of PFOA in cord blood (Apelberg et al., 2007a; Fei et al., 2007; Gützkow et al., 2011; Hanssen et al., 2010; Midasch et al., 2007; Monroy et al., 2008). The concentrations of PFOA in cord blood have been shown to be highly correlated with the corresponding concentration in maternal serum at the time of delivery (Gützkow et al., 2011; Monroy et al., 2008). The transport across the placental barrier seems to be dependent on the compound structure. In a study from Norway including 123 pairs of maternal and cord plasma samples, the median PFOA concentration in cord plasma was 78% of the corresponding concentration in maternal plasma (Gützkow et al., 2011).

PFOA has also been found to be transferred to infants through breast-feeding (Fromme et al., 2009; Kärrman et al., 2007; Tao et al., 2008; Völkel et al., 2008). The average breast milk concentration of PFOA was 3.8% of the corresponding serum concentrations in a recent Norwegian study (Haug et al., 2011a), and similar numbers were also found in a study from Korea (Kim et al., 2011). Although levels of PFOA in breast milk are low compared to those in blood (Fromme et al., 2010; Kuklenyik et al., 2004; Llorca et al., 2010; So et al., 2006; Tao et al., 2008; Wilhelm et al., 2009), a breast-fed infant will be exposed to considerable amounts of PFOA during the first months of life. A median daily intake of 4.1 ng PFOA/kg bw/day was calculated in a recent Norwegian study, and consumption of breast milk was found to be the major source of exposure for exclusively or predominantly breast-fed infants (Haug et al., 2011a). The total exposure to PFOA for infants was around 15 times higher than the corresponding estimates for adults. The considerable exposure of infants through breast feeding is also supported by the decreasing concentrations of PFOA in breast milk during the course of lactation, seen in an elimination rate study (Thomsen et al. 2010). In a study from Germany, median PFOA levels in cord blood were reported to be 1.7 ng/mL and in blood of 6 month old infants the corresponding level was 6.9 ng/mL (Fromme et al., 2010). PFOA concentrations in infant serum at 6 months of age were 4.6 times higher than in maternal serum at delivery. Further, for all subjects, increasing PFOA concentrations were seen during the first 6

months of life, and most subjects showed a clear decrease in the following months likely due to ended breast feeding.

In conclusion, PFOA has been shown to be readily transferred to the foetus through the placenta in humans. Further, breast milk is an important source of exposure to breast-fed infants and the PFOA exposure for infants is considerably higher than for adults.

Distribution in the human body
In an Italian study, the concentrations of PFOA were examined in various tissues (liver, kidney, adipose tissue, brain, basal ganglia, hypophysis, thyroid, gonads, pancreas, lung, skeletal muscle and blood) from post-mortem examinations of seven subjects whose cause of death had not been related to intoxication (Maestri et al., 2006). PFOA was observed in all tissues, and in line with findings in animal studies the highest concentrations were found in lung, kidney, liver and blood.

In a study from the US, the concentrations of PFOA in 23 paired samples of blood and liver were examined and the mean liver to serum ratio was found to be 1.3 (Olsen et al., 2003). In contrast, higher concentrations were found in blood than liver in a study from Spain, but the samples of liver and blood were not from the same subjects thus drawing conclusions is more difficult (Kärrman et al., 2010).

In conclusion, a similar distribution pattern was seen in humans as in laboratory animals for PFOA, with the highest concentrations found in lung, kidney, liver and blood.

Elimination
The half-life of PFOA has been studied in 26 retired fluorochemical production workers who had high initial serum concentrations (mean = 691 ng/mL) (Olsen et al., 2007). Elimination followed a first-order kinetic model, and the geometric mean half-life for PFOA was 3.5 years. In a study from West Virginia where people had been exposed to PFOA contaminated drinking water, filtration through granular activated carbon was started (Bartell et al., 2010). Up to six blood samples were collected from each of 200 participants the first year after filtration. The observed data are consistent with first-order elimination and a median serum PFOA half-life of 2.3 years was found. The authors found no evidence of age- or sex-dependence of the postfiltration elimination rates. In a following study of the same authors, differences in serum clearance rates between low- and high-exposure water districts were seen, and it was suggested a possible concentration-dependent or time-dependent clearance process or inadequate adjustment for background exposures to being the reason for this observation (Seals et al., 2011). In examinations of people from Germany having consumed contaminated drinking water, a geometric mean plasma PFOA half-life of 3.3 years (range: 1.0 – 14.7 years) was calculated (Brede et al., 2010). Two recent studies on exposures of professional ski waxing technicians indicated a long half-life of PFOA as well (Freberg et al., 2010; Nilsson et al., 2010a).

The long half-life in humans is in contrast to mice and rats with a half-life of PFOA of around 30 to 60 days in mouse and from 1 to 30 days in rat (Tatum-Gibbs et al., 2011). A study by Harada et al. (Harada et al., 2005) showed that the renal clearances of PFOA were almost negligible in both sexes in humans, in clear contrast to the large active excretion in the female rat.

In conclusion, an elimination half-life around 2-4 years for PFOA has been reported in humans, and in contrast to certain laboratory animals no sex differences have been observed with respect to the elimination rates.

4.1.3 Bioaccumulation in humans

As described above, PFOA is a very persistent contaminant that does not undergo metabolism and has a long elimination half-life in humans. When the elimination rate is lower than the uptake and there is no metabolism of the substance, the body burden will increase with age. This is well described for other persistent organic compounds such as PCBs and dioxins.

However, scientific papers on the effect of age on concentrations of PFCs in serum are not consistent. In some studies addressing human blood levels of PFOA with life time, no correlation between PFOA concentration and age was reported (Calafat et al., 2007; Olsen et al., 2003; Olsen et al., 2004). In contrast, two Norwegian studies reported significant positive associations between age and serum PFOA concentrations (Haug et al., 2010b; Haug et al., 2011a).

As described in section 4.1.2, breast feeding history, diet, life style and indoor environment are important exposure factors and are factors that most likely will hide the measurable accumulation of PFOA with age. This is further supported by two Norwegian studies using multiple linear regression analyses to adjust for different contributing factors. In the Norwegian Fish and Game Study (n=175) levels of PFOA in serum from men and women increase statistically significant with age (Haug et al., 2010b). Also in a study with 41 women aged 25-45 years a statistically significant increase in the PFOA levels with age was found (Haug et al., 2011a). These two studies strongly indicate that PFOA levels increase with age, but that other important factors of PFOA exposure also need to be addressed in the evaluation of human exposure and accumulation of PFOA. The studies above that did not observe any correlation between PFOA levels and age did not take these factors into consideration.

As already mentioned, two recent studies from Norway and Sweden reported significantly elevated PFOA levels in blood serum samples and whole blood samples of professional ski waxers compared to the general populations, after using fluorinated ski wax (Freberg et al., 2010; Nilsson et al., 2010a). In the Swedish study, the PFOA levels in three technicians with “low” initial levels of PFOA (<100 ng/mL in pre-season blood) increased from pre-season to post-season by 254, 134, and 120% each, whereas no increases in the serum levels were observed for the five technicians with “high” initial levels (>100 ng/mL in pre-season sample). In the Norwegian study, a statistically significant positive association between the number of years exposed as a ski waxer and the PFOA concentrations in blood serum was observed.

In other words, there are strong indications that PFOA bioaccumulates in humans. This is also as expected based on the toxicokinetic properties of PFOA.

4.1.4 Conclusion on toxicokinetics and bioaccumulation in humans

PFOA is well absorbed following oral and inhalation exposure, and to a lesser extent following dermal exposure in laboratory animals. PFOA is present in human blood of the general population and elevated concentrations are seen following specific exposure to PFOA, either environmentally (e.g. contaminated drinking water) or occupationally. PFOA has not been found to be metabolised. The highest concentrations of PFOA are found in blood, liver, kidney and lung. Urine is the primary route of excretion. Humans have a very slow elimination of PFOA compared with other species, with a half-life around 2-4 years. PFOA has been shown to be readily transferred to the foetus through the placenta both in laboratory animals and humans. Further, breast milk is an important source of exposure to breast-fed infants and the PFOA exposure for these infants is considerably higher than for adults. Gestational and lactational exposure is of special concern as the foetus and newborn babies are highly vulnerable to exposure to toxic substances. The time trend studies show that PFOA levels are significantly associated with the time working as a ski waxer (Freberg et al.,

2010; Nilsson et al., 2010a; Nilsson et al., 2010b) and some recent studies strongly indicate that PFOA levels increase with age (Haug et al., 2011a; Haug et al., 2010b). Based on a weight of evidence approach, this demonstrates that PFOA bioaccumulates in humans.

5 ENVIRONMENTAL HAZARD ASSESSMENT

The acute and chronic toxicity of PFOA and APFO to environmental species has already been assessed in the OECD SIDS Initial Assessment Report (OECD, 2006). Low toxicity to the organisms in aquatic and terrestrial compartment was observed. As no newer data are available the toxicity of PFOA and APFO to environmental species is considered to be low.

6 CONCLUSIONS ON THE SVHC PROPERTIES

The free perfluoroocanoic acid (PFOA) stays in equilibrium with perfluorooctanoate (PFO), the conjugate base, in aqueous media in the environment as well as in the laboratory. The ammonium salt (APFO), which is often used in animal experiments, is very soluble in water. In aqueous solution it is present as anion PFO and the ammonium cation. The dissolved anion PFO will stay in equilibrium with the corresponding acid in aqueous media. In the following PFOA refers to the acid (PFOA) as well as to its conjugate base PFO. Therefore conclusions on PFOA/APFO are considered to be valid for APFO/PFOA as well.

6.1 PBT, vPvB assessment

6.1.1 Assessment of PBT/vPvB properties – comparison with the criteria of Annex XIII

A weight of evidence determination according to the provisions of Annex XIII of REACH is used to identify the substance as P and B. The available results are assembled together in a single weight of evidence determination.

6.1.1.1 Persistence

The stability of organic fluorine compounds has been described in detail by Siegemund et al., 2000: When all valences of a carbon chain are satisfied by fluorine, the zig-zag-shaped carbon skeleton is twisted out of its plane in the form of a helix. This situation allows the electronegative fluorine substituents to envelope the carbon skeleton completely and shield it from chemical attack. Several other properties of the carbon-fluorine bond contribute to the fact that highly fluorinated alkanes are the most stable organic compounds. These include polarizability and high bond energies, which increase with increasing substitution by fluorine. The influence of fluorine is greatest in highly fluorinated and perfluorinated compounds. Properties that are exploited commercially include high thermal and chemical stability (Siegemund et al., 2000).

Abiotic degradation

Under relevant environmental conditions in aqueous media PFOA is hydrolytically stable (DT_{50} > 92 days) and does not undergo direct photodegradation in natural waters. The estimated DT_{50} for indirect photolysis is 349 days.

Biotic degradation

Screening studies indicate that PFOA is not ready biodegradable. The results of biodegradation tests demonstrate that no biodegradation in water, soil and sediment occurs. Due to the high persistency and missing degradation, no half-lives could be calculated.

Conclusion on Persistence

All results show, that PFOA is persistent and does not undergo any further abiotic or biotic degradation under relevant environmental conditions. According to Annex XIII, APFO and PFOA meet the criteria for being persistent and very persistent.

6.1.1.2 Bioaccumulation

According to Annex XIII a number of different data can be used to assess the bioaccumulation potential of a compound. In the following, all available information, i.e. bioaccumulation in terrestrial species and in humans, was considered together in a weight of evidence approach. The individual results have been considered in the assessment with differing weights depending on their nature, adequacy and relevance.

(a) Bioconcentration or bioaccumulation in aquatic species:

The reported BCFs and BAFs for PFOA and APFO are in the range from 0.9 to 266. Therefore, the numerical criterium of Annex XIII is not met.

However, bioconcentration values in fish may not be the most relevant endpoint because other mechanisms for bioaccumulation might be of relevance, i.e. the bioaccumulation potential in air breathing and terrestrial species. Therefore, the numerical bioaccumulation (B) criterion defined in the REACH regulation Annex XIII is not suitable for PFOA.

(b) Other information on the bioaccumulation potential of the substance:

— Bioaccumulation in terrestrial species;

PFOA is frequently analyzed in environmental monitoring studies. PFOA has been found in piscivorous mammals, and occasionally detected in high trophic level avian predators (Kannan et al., 2005). In herring gull eggs, e.g. PFOA concentrations were measured in the range from 6.5 to 118 ng/g (ww) (Rüdel et al., 2011). Values in polar bear liver ranged from 3-13 ng/g (Martin et al., 2004b). Although, the focus of these studies was not to measure the bioaccumulation potential values the fact that PFOA is ubiquitously present in terrestrial species, even in remote areas is of special concern.

Bioaccumulation of PFOA was studied in lichen, caribou, and wolf, living in the remote Canadian environment. The measured biomagnification factors (BMF) were in the range from 0.9 to 11 (Müller et al., 2011). Values greater than 1 indicate bioaccumulation.

— Toxicokinetics and bioaccumulation in humans

PFOA is well absorbed following oral and inhalation exposure, and to a lesser extent following dermal exposure in laboratory animals. PFOA is present in human blood of the general population and elevated concentrations are seen following specific exposure to PFOA, either environmentally (e.g. contaminated drinking water) or occupationally. PFOA has not been found to be metabolised. The highest concentrations of PFOA are found in blood, liver, kidney and lung. Urine is the primary route of excretion. Humans have a very slow elimination of PFOA compared with other species, with a half-life around 2-4 years. PFOA has been shown to be readily transferred to the foetus through the placenta both in laboratory animals and humans. Further, breast milk is an important source of exposure to breast-fed infants and the PFOA exposure for these infants is considerably higher than for adults. Gestational and lactational exposure is of special concern as the foetus and newborn babies are highly vulnerable to exposure to toxic substances. The time trend studies show that PFOA levels are significantly associated with the time working as a ski waxer (Freberg et al., 2010; Nilsson et al., 2010a; Nilsson et al., 2010b) and some recent studies strongly indicate that PFOA levels increase with age (Haug et al., 2011a; Haug et al., 2010b). This demonstrates that PFOA bioaccumulates in humans.

— Detection of elevated levels in biota, in particular in endangered species or in vulnerable populations, compared to levels in their surrounding environment;

Values in polar bear liver ranged from 3 ng/g to 13 ng/g (Martin et al., 2004b). Butt et al. report concentrations of PFOA in polar bears up to 3.4 ng/g ww. Polar bears life in a remote region where PFOA concentrations in the surrounding water are in the pg/l range. Hence, the levels of PFOA analyzed in polar bear tissues and blood indicate uptake and accumulation of PFOA from the surrounding environment and food (Butt et al., 2010).

(c) Ability of the substance to biomagnify in the food chain,

Piscivorous mammals (mink, seal, dolphin) exhibited significant amounts of PFOA mainly accumulated in serum and liver. Looking at predator-prey relationships or whole food chains there are studies available which report trophical magnification factors (TMFs) or biomagnification factors (BMFs) greater than one, indicating bioaccumulation of PFOA. The studies on dolphins, caribou, or turtles clearly show that bioaccumulation of PFOA is taking place.

For the food chains Walrus (liver) / Clam, Narwhal (liver) /Arctic Cod, and Beluga (liver)/Arctic Cod the BMFs are 1.8, 1.6, and 2.7, respectively, indicating bioaccumulation (Tomy et al. 2009).

BMFs ranging from 1.8 to 13 for seven individual dolphin/prey relationships were stated using recalculated PFOA whole body burdens for dolphin (Houde et al., 2006b).

Kelly et al. 2009 measured PFOA in the Canadian Arctic marine food web (sediment and in different organisms: macroalgae, bivalves, fish, seaducks, and marine mammals). A TMF of 3.28 for PFOA was one result of the study. The protein-normalized value is reported to be 1.93.

Bioaccumulation was also studied in lichen, caribou, and wolf, living in the remote Canadian environment. Measured BMFs were in the range from 0.9 to 11 and indicate bioaccumulation. Calculated TMFs were in the range from 1.1 to 2.4, indicating trophic magnification, too (Müller et al., 2011).

Field studies are complex and therefore difficult to judge concerning their reliability. Each of the presented field studies has its drawbacks due to sample collection in different years, the sampling of body tissues and fluids instead of whole body or uncertainty of prey constitution etc. and may not be considered as a standalone proof for the bioaccumulation potential of PFOA. Nevertheless, when reviewing all studies together their results can be considered overall conclusive. The weight of evidence of these studies suggests that PFOA can biomagnify in the food chain as indicated by biomagnifications factors and trophic magnification factors larger than one.

Conclusion on bioaccumulation

In summary, taken together all data presented can be considered overall conclusive. The weight of evidence of these studies in environmental species and human data suggests that PFOA and APFO can biomagnify in the food chain and bioaccumulates in humans. It is of special concern that PFOA and APFO biomagnify in endangered species as shown for the polar bear and in animals which are likely to become endangered in the near future (narwhale and beluga whale).

Additionally, in humans gestational and lactational exposure are of special concern as the foetus and newborn babies are highly vulnerable to exposure to toxic substances.

6.1.1.3 Toxicity

The acute and chronic toxicity of APFO and PFOA to environmental species is considered to be low.

However, the Risk Assessment Committee (RAC) has concluded that PFOA and APFO fulfil the criteria for classification as toxic for reproduction category 1B and the criteria for classification with STOT RE 1. This classification is of relevance for the assessment of PFOA and APFO as a substances of very high concern according to Article 57 d), i.e. under the T-criterion of PBT; see REACH Annex XIII; Section 1.1.3 c).

6.1.2 Summary and overall conclusions on the PBT, vPvB properties

Based on all available information degradation experiments PFOA and APFO are not degraded in the environment and therefore fulfil the P- and vP-criterion.

Furthermore, it is concluded that PFOA and APFO are bioaccumulative compounds.

The bioaccumulative property is proven by studies from terrestrial food webs, which clearly indicate accumulation of PFOA and APFO. Human data strongly indicate that PFOA and APFO bioaccumulates in humans.

It is of special concern that PFOA and APFO biomagnify in endangered species as shown for the polar bear and in animals which are likely to become endangered in the near future (narwhale and beluga whale). Additionally, in human gestational and lactational exposure are of special concern as the foetus and newborn babies are highly vulnerable to exposure to toxic substances.

Based on a weight of evidence approach, it is considered that the data from environmental species and humans shows that the B criterion is fulfilled.

According to the recent RAC-opinion PFOA and APFO fulfil the criteria for classification as Repr 1B and STOT RE 1, each of which proves that PFOA and APFO fulfil the T-criterion.

Overall, PFOA and APFO are identified as a PBT-substances according to Art. 57 (d) of REACH by comparing all relevant and available information listed in Annex XIII of REACH with the criteria set out in the same Annex, partly a weight of evidence determination using expert judgement was applied.

6.2 CMR assessment

The substance is not yet listed in Annex VI of CLP (Regulation (EC) 1272/2008) however there is evidence based on the RAC opinion on PFOA that seems to indicate that the substance meets the criteria for classification as toxic for reproduction in accordance with Article 57 (c) of REACH.

The classification of PFOA/APFO is currently included in draft proposal for the 5th ATP to CLP.

6.3 Substances of equivalent level of concern assessment.

Not relevant for this dossier.

PART II

INFORMATION ON USE, EXPOSURE, ALTERNATIVES AND RISKS

INFORMATION ON MANUFACTURE, IMPORT/EXPORT AND USES –CONCLUSIONS ON EXPOSURE

Manufacture and Import

From 1951 until 2004 the estimated total global production was 3,600 - 5,700 t PFOA and APFO (Prevedouros et al., 2006). APFO is mainly used as a processing aid in the production of fluoropolymers and fluoroelastomers. In 2002, its world-wide production was about 200-300 t (Prevedouros et al., 2006). According to a recent market analysis on behalf of the European Commission, only one company manufacturing APFO and related substances was active in Europe 27 in 2010. This company announced cessation of production of APFO as per August 2010 and cessation of its commercialisation as per November 2010. Imports of APFO are expected to partly replace this production and to most probably remain stable at <50 tonnes per year until 2015 (van der Putte et al., 2010).

Direct sources

According to the above mentioned market analysis, total direct source of APFO/PFOA in the EU-27 will be 50-100 tonnes per annum for industrial use only (van der Putte et al., 2010).

Fluoropolymers are high performance plastic materials and fluoroelastomers are high performance synthetic rubbers. The main fluoropolymers produced with PFOA as a processing aid are polytetrafluoroethylene (PTFE) and polyvinylidenefluoride (PVDF). PVDF is also produced with a mix of perfluorocarboxylic acids (carboxylic acids, $C_{7\text{-}13}$, perfluoro, ammonium salts; CAS no. 72968-38-8), and the mix contains mainly other perfluorocarboxylic acids than PFOA and is listed as containing less than 1% PFOA (van der Putte et al., 2010). Entry into the environment occurs during manufacture of PFOA/APFO and during the production of fluoropolymers and fluoroelastomeres. Residues from production, processing and use of fluorinated polymers are suspected in several industries (for example textile finishing, electroplating and paper industry).

The use volumes of PFOA and APFO in the photographic industry and in the semiconductor industry are estimated at about 2.6 tonnes per year and 25 kg per year respectively (van der Putte et al., 2010).

Indirect sources

There are a number of products containing PFOA such as textiles, carpets, upholstery, paper, leather, toner, cleaning agents and carpet care solutions, sealants, floor waxes, paints, impregnating agents, etc. PFOA might also be present as impurity, i.e. in perfluorooctylsulfonyl fluoride (POSF) based products (Begley et al., 2005; Berger and Herzke, 2006; Danish Ministry of the Environment, 2005; Kissa, 1994; Prevedouros et al., 2006; Swedish Chemicals Agency, 2006; Trier et al., 2011; van der Putte et al., 2010; Walters and Santillo, 2006; Washburn et al., 2005)

PFOA might be a residue in PTFE based applications, such as (van der Putte et al., 2010; Walters and Santillo, 2006; Washburn et al. 2005):

- Electrical wire insulation
- Specialist circuit boards
- Plumbers tape (thread seal tape (TEFLON-Tape))
- Waterproof membranes for garments (such a Gore-Tex)
- Surgical implants
- Dental floss
- Engine protector additives
- Non-stick coatings

Other indirect PFOA sources are fluorotelomers, which are not produced using PFOA, but which may contain low levels of PFOA as an unintended by-product. Fluorotelomers are used in a number of products, among others, in fire fighting foam and for surface coating of carpeting, textiles, paper, leather, and ski wax.

The importance of imported products as a source of PFOA is highlighted by a report from KEMI, the Swedish chemicals Agency (Report 07/06): 25 kg of PFOA and approximately 22 tons of fluorotelomers were imported to Sweden in 2005. The main use of these compounds (~75 %) was textile industry. However, the textile industry in Sweden is rather small nowadays and more than 9,000 tonnes of outdoor clothes were imported to Sweden in 2005. The proportion of fluorinated substances is unknown (Swedish Chemicals Agency, 2006).

CURRENT KNOWLEDGE ON ALTERNATIVES

In general, PFCs with eight carbon atoms can be replaced with shorter chain fluorinated chemicals containing six or less carbon atoms.

Non-fluorinated alternatives are available as well, i.e. propylated aromatics (naphthalene or biphenyls) and aliphatic alcohols (sulphosuccinate and fatty alcohol ethoxylates) (Danish Ministry of the Environment, 2005; van der Putte et al., 2010; Walters and Santillo, 2006).

In the following table known PFOA alternatives are summarized.

Table 15: Alternative compounds, their product names, company and use for PFOA and its salts**.**

Alternative compound	Product name	Company	Used for /Used in	Ref.
PFBS or based on different C_4- perfluoro-compounds	Novec®	3M	Paint and coatings industry. Electronic coating, industrial and commercial cleaning, cleaner for solder flux residue, degreasing applications	(Walters and Santillo, 2006; van der Putte et al., 2010; Poulsen and Jensen, 2005)
Dodecafluoro-2-methylpentan-3-one(CF_3-CF_2-C(O)-$CF(CF_3)_2$)	Novec®	3M	Fire-fighting fluid	(Poulsen and Jensen, 2005; Walters and Santillo, 2006)
C6-fluorocompounds	Forafac®	DuPont	Fire-fighting foam	(Poulsen and Jensen, 2005)
CF_3 or C_2F_5 pendant fluoroalkyl polyethers	PolyFox®	OMNOVA Solutions Inc.	Surfactant and flow, level and wetting additive for coating formulations. Also used in floor polish	(Poulsen and Jensen, 2005)
Propylated aromatics (naphthalenes or biphenyls)	Ruetasolv®	Rütgers Kurehe Solvents GmbH	Water repelling agents for rust protection systems, marine paints, coatings, etc.	(Walters and Santillo, 2006) (Poulsen and Jensen, 2005)
Aliphatic alcohols (sulphosuccinate and fatty alcohol ethoxylates)	Emulphor®, Lutensit®	BASF	Levelling and wetting agents	(Poulsen and Jensen, 2005)
Sulfosuccinates	EDAPLAN ® LA451	Münzing Chemie	Paint and coatings industry: Wetting agents for water based applications, e.g. wood primers	(Poulsen and Jensen, 2005)
Sulfosuccinate	Hydropalat® 875	Cognis	Paint and coating industry: Weting and dispersing agents	(Poulsen and Jensen, 2005)
Silicone Polymers	WorléeAdd ®	Welrée-Chemie	Wetting agents in paint and ink industry	(Poulsen and Jensen, 2005)
Branched fluoro ethers			Can be applied for all products	(van der Putte et al., 2010)
short-chain fluorinated technologies (six or less carbons)	Capstone	DuPont	commercially available in home furnishings, fire fighting foam, fluorosurfactants, paper packaging, textiles, stone and tile, and leather end uses	[3]
Ammonium 4,8-dioxa-3H-perfluorononanoate	ADONA	3M	emulsifier used in the aqueous emulsion polymerization of fluoropolymers made from tetrafluoroethylene (TFE)	(Gordon, 2011)

[3] http://www.oecd.org/document/34/0,3746,en_21571361_44787844_44799586_1_1_1_1,00.html

Exposure pathways for humans

Exposure of the general public

The large historical production volumes and widespread applications of PFOA also in consumer products represent a potential for contamination of the indoor as well as the outdoor environment and thereby also of food and drinking water. Dietary exposure has been suggested to be the main exposure route of PFOA in adult general populations (Fromme et al., 2009; Trudel et al., 2008; Vestergren and Cousins, 2009), and there has previously been reported significant associations between estimated dietary intakes of PFOA and the corresponding serum concentrations (Haug et al.). Contaminated food, like popcorn or fries, meat, fish, sea food, cereals and eggs was reported to be a source of PFOA in the human body and carryover from soil to food vegetables has also been shown (D'Hollander et al., 2010; Ericson et al., 2008a; European Food Safety Authority, 2011; Fromme et al., 2009; Haug et al., 2011a; Haug et al., 2010b; Haug et al., 2010a; Llorca et al., 2010; Lechner et al., 2011, Rylander et al., 2010; Rylander et al., 2009; Tittlemier et al., 2007; Trudel et al., 2008; Zhang et al., 2010). Significant correlations between estimated dietary intake and measured serum concentrations of PFOA have been found (Haug et al., 2010a; Haug et al., 2010b; Zhang et al., 2010).

Indirect PFOA contamination of food from paper packaging and cookware has been proven (Begley et al., 2005; D'Hollander et al., 2010; Llorca et al., 2010; Powley et al., 2005; Tittlemier et al., 2007; Trier et al., 2011). Additionally, PFOAAPFO and other perfluorinated compounds are used as emulsifiers in the production of non-stick coating of cookware and have been evaluated for this use by the European Food Safety Authority (EFSA, 2005): Residual PFOAwas never detected in the fluoropolymeric sample. Based on the detection limit of 0.022 mg/kg polymer, the calculated worst case migration was 0.017 mg/kg food, (sample thickness 0.6 cm, 6dm^2 /kg food, first use data) (EFSA, 2005).

Several studies reported PFOA contaminations in drinking water (Loos et al., 2007; Ericson et al., 2008b; Haug et al., 2010a; Saito et al., 2004; Wilhelm et al., 2010; Emmet et al., 2006), and in certain cases contaminated drinking water has been shown to be a major source of human exposure. (Egeghy and Lorber 2010; Emmett et al. 2006; Vestergren and Cousins, 2009).

A review by Harrad et al. (2010) also emphasized the importance of evaluating exposure from ingestion of house dust and inhalation of indoor air. The PFOA concentrations reported in house dust range from <LOD to 4100 ng/g and the median concentrations from <LOD to 300 ng/g (Costner et al. 2005; D´Hollander et al. 2010; Fromme et al., 2009; Bjorklund et al., 2009; Goosey and Harrad, 2011; Haug et al 2011b; Kato et al. 2009; Kubwabo et al. 2005; Moriwaki et al. 2003;; Strynar et al. 2008; Wang et al., 2010). Furthermore, precursor substances are present in indoor air and house dust as well. For example, three times higher 8:2 FTOH concentrations were found in house dust compared to those of PFOA (Shoeib et al., 2011).

An additional source of PFOA may result from the biotransformation of precursor substances, e.g. polyfluoroalkyl phosphate esters (PAPs) (D´eon and Mabury, 2011) and fluorotelomer alcohols (FTOHs) (Fasano et al., 2006). PAPs and FTOHs have been confirmed as migrants from food-contact paper products into food. PAP diesters (diPAPs) at concentrations in the range of microgram per liter haven been detected in human serum (D´eon and Malbury, 2011). Considering the long serum half-life of PFOA and the bioavailability of 8:2 diPAP it is expected that the biotransformation of 8:2 diPAP may contribute significantly to the PFOA concentration in human serum.

In a recent Norwegian study, the relative importance of different exposure pathways of PFOA was assessed on an individual basis using measured PFOA concentrations in indoor air and house dust as well as information from food frequency questionnaires and concentrations in food (Haug et al., 2011a). Food was generally the major exposure source, representing 67 - 84% of the median total intake for PFOA using different dust ingestion rates and biotransformation factors of 'precursor' compounds. However, on an individual basis, the indoor environment accounted for up to around 50% of the total intake for several women. Furthermore, significant positive associations between concentrations of PFOA in house dust and the corresponding serum concentrations underline the importance of the indoor environment as an exposure pathway for PFOA. Breast milk was calculated to be the single most important source to PFOA for breast-fed infants (Haug et al., 2011a). So far no other studies have compared exposure pathways for infants based on individual measurements of PFOA concentrations in breast milk, house dust and indoor air. The median total intakes of PFOA were in the range 0.26 - 0.33 ng/kg bw/day. This is in the same range as has been modelled for PFOA in studies on populations exposed to background contamination levels (Egeghy and Lorber, 2010; Fromme et al., 2009; Vestergren and Cousins, 2009). In the Norwegian study, the median total intake for infants of around six months of age ranged from 4.3 to 4.9 ng/kg bw/day for PFOA, depending on the dust ingestion rates and biotransformation factors used. This is around 15 times higher than the corresponding estimates for adults.

Workplace exposure

Very high serum concentrations have been reported in fluorochemical production workers with mean concentrations of PFOA in the range of 500 to 7,000 ng/mL depending on the type of work. The highest serum level reported for PFOA was 114,100 ng/mL in 1995 (Fromme et al., 2009).

Manufacture

The worker exposure during the manufacture of PFOA/APFO has a long history of surveillance (Costa *et al*., 2009, Sakr *et al*., 2009, Olsen *et al*., 2003 and references therein). According to Costa *et al*., in 2007 for 37 workers at a manufacturing plant in Italy blood serum levels were 0.20 - 47.04 μg/mL with a geometric mean of 4.02 μg/mL compared to typical average values <10 ng/mL for the general population given in the OECD SIDS Initial Assessment Report (OECD, 2006). This example shows additional exposure to, and uptake of, the substances by workers.

Typical situations with potential regular exposure include the production process (in particular sampling, cleaning and maintenance operations), drying, shipping and packaging of the substance(s). In addition, the solid substance(s) readily sublime (Kaiser, 2010) making handling more difficult and increasing the risk of airborne workplace exposure which can be reduced by improved industrial hygiene and the use of aqueous solutions.

Manufacturing and use of fluoropolymers

According to information from the Plastics Europe Fluoropolymer Committee reported by van der Putte (van der Putte et al., 2010) in fluoropolymer synthesis PFOA/APFO are used in low concentrations of < 1%. In an analysis of community exposure in the USA published by Emmet et al. (Emmet et al., 2006) a group of workers with "substantial exposure" to PFOA/APFO in a fluoropolymer production facility had increased median serum blood levels of PFOA (775 ng/mL) compared to the studied group with no occupational exposure (329 ng/mL). Human monitoring data by DuPont, likely from the same production site, show similar levels in 2004 (OECD, 2006).

A quantitative investigation of worker exposure in any of the numerous professional applications for fluoropolymer preparations is not known, it is however expected to be low due to the generally small amounts of PFOA/APFO in the respective preparations.

Photographic applications

PFOA/APFO are used for specific coating applications with potential worker exposure *via* inhalation or dermal contact. Exposure level data (blood levels, workplace concentration measurements) are not available. In general, the frequency of exposure is low and diluted aqueous solutions are used and handled with protective gloves (van der Putte et al., 2010, Michiels 2010).

Ski-waxers

In a Swedish study of the inhalation exposure to FTOH and PFOA and levels in blood of ski wax technicians was examined. Air was collected in the breathing zone of ski wax technicians during work. The results show concentrations of 8:2 FTOH and PFOA in air in the range of 0.830 to 250 µg/m3 and 0.080 to 4.900 µg/m3, respectively. Estimation range (average) of daily inhalation exposure based on four samplings presented in the study is 0.011 - 3.4 (1.2) g for 8:2 FTOH and 0.0011 - 0.065 (0.015) g for PFOA, respectively. The PFOA concentrations in the blood of the technicians rose even until May after the end of the World Cup in March. Therefore, the authors conclude also an indirect PFOA exposure via precursor substances and suggest that metabolic biological systems are active for some time after the exposure (Nilsson et al., 2010a).

Conclusion

Exposure of workers to PFOA/APFO can occur in several workplace situations in various industrial and professional applications and in particular if production of the substance(s) is resumed at historical levels. Compared to the general population, increased blood levels in workers involved with PFOA/APFO manufacture are evident. Even in the situations where occupational exposure is low the additional uptake of PFOA/APFO at the workplace puts workers at an increased risk to potential adverse health effects caused by the substance(s).

Disposal

PFOA and its precursors are widely present in consumer products which are disposed via municipal landfill or incineration plants. There is no specific disposal practice for PFOA, because it is disposed together with the corresponding product. Therefore PFOA is present in landfills as shown by detections of PFOA in landfill leachates (mean PFOA concentrations 2.9 to 537 ng/L or in emissions from landfills into air (0.2 – 1.1. pg/m^3) (Busch et al., 2010a; Weinberg et al., 2010).

PFOA is often reported as the investigated PFCA with the highest concentrations in WWTP´s effluents (Ahrens and Ebinghaus, 2010). Municipal and industrial sewage and degradation of precursors are supposed to be the source (Murakami et al., 2008; Loganathan et al., 2007). If the sewage sludge is used as fertilizer in agriculture, PFOA and related substances may contaminate soil, crops, surface water and ground water. Additionally, in some countries, municipal sewage sludge and industrial wastewater sludge is dumped into the ocean, which is another important source of PFOA in surface water (Guo et al., 2010).

Different methods for the decomposition of PFOA were examined. During photolysis (245 nm) <5% of PFOA was decomposed after 120 h, decarboxylation was observed at 307 °C and during sonochemical irradiation a half-live of 120 min was reported. But reaction times are still too long

for industrial application and short chain PFCAs were observed as reaction product for every single method (Rayne and Forest, 2009).

The behaviour of PFOA during recycling of materials containing PFOA is to the best of our knowledge not yet investigated. But due to the properties of PFOA no degradation is expected.

ANNEX I - RISK-RELATED INFORMATION

Environmental distribution

PFOA is ubiquitously present in oceans (Yamashita et al., 2004; Yamashita et al., 2005; Ahrens, 2011; Busch et al., 2010b). In the Atlantic Ocean and the North Sea up to 223 pg/L PFOA were detected whereas concentrations decreased from North to South (Ahrens et al., 2010). In general, in coastal regions with industrial areas the PFOA concentrations are two orders of magnitude higher than in open ocean waters (Ahrens, 2011).

Rivers are a potential source for PFOA detected in the oceans. A flux of 14 t PFOA per year from rivers into oceans was estimated. PFOA concentrations in European rivers range between <0.65 – 23 ng/L (McLachlan et al., 2007). In the vicinity of fluoropolymer manufacturing facilities the values are usually higher, for example 337 ng/L in the river Po in Italy (Loos et al., 2008). Effluents from wastewater treatment plants (WWTPs) are a known source for PFOA in rivers (up to 1,050 ng/L PFOA (Ahrens et al., 2009a)). The daily releases of PFOA into rivers were calculated to be in the range of 5.9 µg/person to 220 µg/person (Sinclair and Kannan, 2006; Schultz et al., 2006b; Schultz et al., 2006a; Becker et al., 2010). Further sources are landfill leachates and nonpoint sources, as dry and wet atmospheric deposition and surface runoff, which contribute to the occurrence of PFOA in surface water.

In a European survey PFOA was detected in 66% of analysed ground water samples with average concentrations of 3 ng/L (Loos et al., 2010). Highest concentrations reported in ground water of up to 1,050,000 ng/L were tracked back to contamination with aqueous fire fighting foams (Moody et al., 2003). Ground water near fluoropolymer manufacturers might generally be contaminated with PFOA and other PFCs. In Gendorf, Bavaria, for example, high PFOA concentrations of up to 4300 ng/L were measured (Bayerisches Landesamt für Umwelt, 2010).

PFOA can be measured in the atmosphere (Jahnke et al., 2007; Butt et al., 2010; Fromme et al., 2009; Barber et al., 2007; Dreyer et al., 2009). Concentrations up to 0.8 ng/m^3 and 0.006 ng/m^3 were reported for the particulate and gaseous phase, respectively (Barber et al., 2007; Kim and Kannan, 2007). The dry deposition of PFOA nearby a fluoropolymer manufacturer was three magnitudes higher than in urban areas (Bayerisches Landesamt für Umwelt, 2010).

PFOA has also been detected in precipitation**.** The concentrations are one order of magnitude higher than PFOA levels in air (Kim and Kannan, 2007; Young et al., 2007; Liu et al., 2009; Dreyer et al., 2010; Kwok et al., 2010; Scott et al., 2006; Ahrens, 2011).

Sediment has been regarded as an important sink and reservoir of PFOA (Prevedouros et al., 2006; Ahrens, 2011). PFOA concentrations in sediment have been reported in the pg range (Higgins and Luthy, 2006; Nakata et al., 2006; Bao et al., 2009; Bao et al., 2010; Becker et al., 2008).

Soils receive PFOA via atmospheric wet and dry deposition or via the application of sewage sludge. Nearby fluoropolymer manufacturers higher concentrations were found compared to other regions (Bayerisches Landesamt für Umwelt, 2010; Wang et al., 2010). Carryover of PFOA from soil to plants has been observed even at low concentrations with grass soil accumulation factors of 0.09 to 0.65 (Stahl et al., 2009; Yoo et al., 2011).

Adsorption/desorption

The following studies were already discussed in the OECD SIDS Initial report and were copied here in italic letters:

The adsorption-desorption of APFO was studied in 25 ml solutions of ^{14}C-labeled APFO in distilled water with 5 g Brill sandy loam soil for 24 hours at a temperature of 16-19 ºC. The study reported a K_d of 0.21 and a K_{oc} of 14 indicating that PFOA has high mobility in Brill sandy loam soil (3M Co., 1978b). The K_{OC} value, however, is questionable due to the lack of accurate information on the purity of the ^{14}C-labeled test substance (Boyd, 1993a; Boyd, 1993b).

An adsorption-desorption test according to OECD guideline 106 was made by Association of Plastic Manufactures in Europe (APME) at DuPont, Newark sponsored by Plastics Europe. APFO was tested with four soil and one activated sludge samples (equilibration time 24 h). Quantification (analytics: LC-MS/MS) was made using a calibration curve. The K_{OM} values ranged from 28 l/kg to 133 l/kg (Association of Plastic Manufactures in Europe (APME), 2003).

Yu et al. performed a study to measure concentrations of PFOA in the biological units of various municipal sewage treatment plants. The K_d was estimated by dividing PFOA concentration in primary sludge or activated sludge by their aqueous concentration in primary effluent or secondary effluent (various full-scale municipal sewage treatments plants). The K_d values for PFOA were observed at 201 – 513 L/kg (activated sludge;) and 188 – 597 L/kg (primary sludge). The authors did not observe differences between K_d values in primary sludge and activated sludge. Log K_{OC} values were in the range from 2.43 to 2.83 for PFOA (Yu et al., 2009).

In the study of Zhou et al., activated sludge was used to test the adsorption behaviour of sodium pentadecafluorooctanoate in aqueous solution. Batch experiments including sorption kinetics, sorption isotherms, and the effect of solution pH and temperature were carried out. The sorption equilibrium of PFOA was reached within about 11 h, indicating that the normal hydraulic residence time in actual wastewater treatment plants (WWTPs) was enough for PFOA to be adsorbed on activated sludge. However, at pH 5-7 only 50 % of the initial PFOA was sorped to the aerobic activated sludge. The sorption of PFOA on sludge decreased with increasing pH. At pH 3 85% of the initial PFOA was sorped to the sludge in comparison to 40 % at pH 9.5. At 25 °C the removal of sodium pentadecafluorooctanoate was a little higher than at 15° or 45°C. In the sorption isotherm experiments K_d values ranging from 150 to 350 L/kg were observed (Zhou et al., 2010).

The relevant data are summarized in Table 16. It has to be kept in mind, that calculations of K_{OC} are in most studies based on total concentrations of PFOA and its conjugate base PFO in water whereas only the neutral acid PFOA is expected to be sorped on organic carbon.

Table 16: Adsorption coefficients for PFOA and its salts

Test substance	Media	Type of adsorption coefficient	Value (L/kg)	Reliability	Reference
APFO	Soil	K_d	0.41 - 8.86	1	(OECD, 2006), (Association of Plastic Manufactures in Europe (APME), 2003)
		K_{oc}	48.9 - 229		
	Activated sludge	K_d	12.6 - 36.8		
		K_{oc}	20.5 – 59.6		

	Soil	K_d	0.21	4	(OECD, 2006), (3M Co., 1978b)
		K_{oc}	14		
Sodium pentadeca-fluoro-octanoate	Activated sludge	K_d	150 - 330	2	(Zhou et al., 2010)
PFOA	Primary sludge	K_d	188 - 597	3	(Yu et al., 2009)
	Activated sludge	K_d	201 – 513		
		K_{oc}	269 - 676		

Conclusion:

PFOA has a low to moderate potential to adsorb on soil and sludge, whereas sorption in sludge is stronger compared to soil. Therefore a high mobility of PFOA in soils can be assumed and soil can be a long-term source of PFOA to underlying groundwater.

Volatilisation

The Henry´s Law constant (K_H) of PFOA was determined at 298 K by an inert-gas stripping method. A helical plate was used to increase the residence time of the gas bubbles in the solutions (aqueous sulphuric acid solution, aqueous sodium chloride and sulphuric acid mixture). The partial pressures of PFOA (p_{PFOA}) in the purge gas were determined by means of Fourier-transform infrared spectroscopy. Kutsuna and Hori derived overall gas-to-water partition coefficients by simulating the time-courses of p_{PFOA} and c_{PFOA} (concentrations of PFOA in the test solutions) simultaneously to optimize parameters of the model relating to the partitioning, the aggregation, and the adsorption. The K_H values of PFOA at 298 K were $1.01 \cdot 10^{-4}$ atm·m³·mol^{-1} for pK_a = 2.8 and $2 \cdot 10^{-4}$ atm·m³·mol^{-1} for pK_a = 1.3. The pKa value of 1.3 seems to be the most probable. At this pK_a most PFOA is present as it´s conjugate base PFO, which is not expected to partition into the gas phase at all, at typical environmental pH of 5-8. However, since K_H of PFOA was relatively small at 298 K the partitioning in air is possible (Kutsuna and Hori, 2008).

Li et al. (2007) developed a novel system for the determination of the air-water coefficient (K_{AW}) for substances that have low K_{AW} and may aggregate in solution, ionize and display surface activity. PFOA is evaporated isothermally from solution through an undisturbed air-water interface at a known gas flow rate, and its concentrations in the water and gas phases are measured. The experimentally determined K_{AW} of PFOA was $1.02 \cdot 10^{-3}$. This K_{AW} corresponds to an K_H of $2.45 \cdot 10^{-5}$ atm·m³·mol^{-1} (calculated from K_{AW}, gas constant and T=293K) (Li et al., 2007).

The following table shows measured and calculated Henry´s law constants from the values for vapour pressure and solubility (Henry´s law constant = vapour pressure/solubility).

Table 17: Henry´s Law constant of PFOA and its salts

Test substance	Vapour pressure [Pa]	Solubility [g/L]	Henry´s Law constant [atm· m³· mol^{-1}]	Reliability	Reference
PFOA (measured)			$1.01 \cdot 10^{-4}$ (pK_a=2.8)	2	(Kutsuna and Hori, 2008)
			$2 \cdot 10^{-4}$ (pK_a=1.3)		
			$2.45 \cdot 10^{-5}$	2	(Li et al., 2007)
APFO	$<1.3 \cdot 10^{-3}$	> 500	$<1.1 \cdot 10^{-11}$	2	(Hekster et al., 2002)
	$9.2 \cdot 10^{-3}$		$7.8 \cdot 10^{-11}$		
PFOA	70	9.5	$4.6 \cdot 10^{-6}$ *	3*	

*Recalculation yields a value for Henry´s Law = $3.008 \cdot 10^{-5}$ atm· m³· mol^{-1}

Conclusion: The protonated form of PFOA has sufficient volatility to leave surface and atmospheric water and/or soil, and generating a slow release of PFOA into the atmosphere. The environmental relevance of this release is unknown. While perfluorooctanoate (PFO), the conjugate base, is not volatile, pure PFOA (protonated) is moderately volatile. When dissolved in water the strong acid PFOA dissociates. The degree is dependent on the pH. Consequently partitioning between environmental media depends on environmental conditions.

Distribution modelling

Distribution modeling is challenging because of the dependence on distribution coefficients. Determination of these coefficients by experimental set ups is difficult especially for the conjugate base of PFOA and PFO. Reasons for these difficulties are surface active properties and micelle building of PFO during the experiments. Therefore there is a lack of reliable distribution coefficients under controlled conditions in the laboratory. Nevertheless, a recent study shows that sediment-water distribution coefficients and bioconcentration factors (biota-water distribution) are proportional for PFOA and other perfluoroalkyl acids (Webster and Ellis, 2011). The authors used a measured bioconcentration factor to predict a sediment-water distribution coefficient. The comparison of the predicted versus the measured values showed good agreement (within one order of magnitude). Therefore, the applicability of equilibrium models for PFOA and other perfluoroalkyl acids is validated (Webster and Ellis, 2011). Also, other studies, i.e. focusing on the transport of PFOA, used equilibrium models, too (Armitage et al., 2009).

For distribution modeling is has to be considered that the conjugate base PFO and the acid PFOA are in equilibrium. This equilibrium needs to be included in the models because of the different properties of the PFOA species, i.e. vapor pressure. Therefore, a pK_a and pH are needed. Some measured as well as estimated pK_a values for PFOA are reported in the literature and are summarized in the following table. There is a high variance in reported pK_a values (up to four log units), whereas highest reported data based on measurements and lower pK_a values are estimations from models. Under environmental conditions at pH 5 – 8 assuming pK_a of 3.8 99.9 % of PFOA is present as conjugate base, whereas with a pK_a of 0 > 99.999 % is present as conjugate base. Because of the dominance of the conjugate base in combination with its high solubility and negligible vapour pressure aqueous phases are expected to be of importance.

Table 18: pK_a-values of PFOA reported in the literature

pK_a	Method	Reliability	Reference
3.8	Experimental, potentiometrically	2	(Burns et al., 2008)
2.8	Experimental, measured in 50/50 v/v ethanol/water	2	(Brace, 1962; Kissa, 2001)
1.01	Experimental, potentiometric titration	2	(Igarashi and Yotsuyanagi, 1992)
1.3	Experimental, pH measurements	2	(López-Fontán et al., 2005)
2.5	No details provided	3	(Ylinen et al., 1990)
2.3	Experimental data cited from others studies	3	(Rayne and Forest, 2009)
3.4			
-0.1	Modeled, PM6	2	
0.90	Modeled, COSMOTHERM	2	(Wang et al., 2011)
-0.11	Modeled, SPARC	2	(Goss, 2008)
0.7	Modeled, COSMO-RS	2	
0	Estimation	2	
-0.2	Modelled, SPARC	2	(Steinle-Darling and Reinhard, 2008)

Long range transport

The following information was copied from the OECD SIDS Initial Assessment Report for PFOA (OECD, 2006):

PFOA, as the anion perfluorooctanoate, PFO, has been detected in remote areas of the world in monitoring programs involving various abiotic and biotic samples (Butt et al., 2010). For example, PFOA has been measured in biota such as polar bears and seals in the Canadian Arctic.

Some examples for PFOA concentrations in remote areas are summarized inTable 19.

Table 19: Concentration of PFOA in remote areas and biota

Sample	Value	Remarks	Reference
Surface water			
Canadian Arctic lakes (Armituk Lake, Char Lake, Resolute Lake)	0.5 – 16 ng/L		(Stock et al., 2007)
Seawater / ice			
Baydaratskaya Bay (Russian Federation)	130.7 (±77.2) pg/L		(Saez et al., 2008)
Greenland Sea	20 – 111 pg/L		(Theobald et al., 2007)
Sediment			
Canadian Arctic lakes (Char Lake and Resolute Lake)	1.7 and 7.5 ng/g dw <1.1 and 2.3 ng/g dw 1.2 and <1.8 ng/g dw	0-1 cm 1-2 cm 2-3 cm	(Stock et al., 2007)
Biota			
Polar bear (liver) (East Greenland)	0.6 – 14 ng/g ww 6.8 – 15.8 ng/g ww 11.8 – 17.6 ng/g ww	1990 1995 2006	(Dietz et al., 2008)
Polar bear (liver) (North American Arctic, European Arctic)	2.4 – 36 ng/g ww		(Smithwick et al., 2005)
Ringed seal (liver) (Arviat - Canadian Arctic)	0.96 – 1.01 ng/g ww		(Butt et al., 2007)

No information is available about current or historical use of PFOA or related substances in the Arctic. A possible explanation for this finding is the long-range transport of either PFOA or potential precursors. Two possible transportation pathways include atmospheric and aquatic transport.

Atmospheric Transport

Due to the relative vapour pressures of APFO, PFOA, and PFO, the chemical form potentially most subject to gas-phase atmospheric transport is PFOA. Franklin suggested that in the presence of water in air (humidity), gaseous PFOA condenses to aerosol particles and dissociates to the corresponding perfluorooctanoate, resulting in a low vapour pressure (Franklin, 2002). The atmospheric lifetime of PFOA (respectively its salts) was calculated in the order of days when emitted from a ground source.

Additional sources of PFOA to the atmosphere are the degradation or transformation of precursors, which could lead to indirect environmental releases. Potential precursors include related fluorinated chemicals which are detectable in the atmosphere (e.g., fluorotelomer alcohols, olefins, and perfluoroalkyl sulfonamido substances) which can degrade in the atmosphere or after deposition to the surface to PFOA. Calculations using a three-dimensional global atmospheric chemistry model (IMPACT) indicate that 8:2 fluorotelomer alcohol (widely used in industrial and consumer products) degrades in the atmosphere to give PFOA (Wallington et al., 2006). FTOHs

have sufficient vapour pressure to be present in air (Prevedouros et al., 2006). Smog chamber studies prove the potential for FTOHs to react in the atmosphere with ubiquitous OH radicals to yield PFOA (Ellis et al., 2004). Ellis et al. showed that the atmospheric lifetime of short chain FTOHs, as determined by reaction with OH radicals wais approximately 20 days. Piekarz et al. estimated that atmospheric residence times of 6:2 FTOH, 8:2 FTOH and 10:2 FTOH were 50, 80 and 70 days, respectively (Piekarz et al., 2007).

However, there is not enough data available to estimate how much the different sources contribute to the PFOA detected in the Arctic and in biota of remote areas. While there is evidence for the possible role of precursors for the long-range atmospheric transport of PFOA, the extent to which these precursors and their transformation may explain the concentrations of PFOA found in remote areas such as the Canadian Arctic is uncertain.

Aquatic Transport and Marine Aerosols

Another possible mechanism for the transport of PFOA to the Canadian Arctic is aquatic transport (Prevedouros et al., 2006). Given PFOA's environmental persistence, high water-solubility and the fact that PFOA and related substances have been emitted to air and water for approximately 50 years and may have accumulated in the oceans, a hypothesis has been presented to suggest ocean water transport as a possible pathway explaining the presence of PFOA in the Canadian Arctic. Currently there is insufficient data to evaluate the significance of this potential pathway.

Several researchers have indicated that the timelines involved with transport via ocean currents could not account for what appears to be rapidly increasing levels of perfluorinated substances in certain Arctic biota (Smithwick et al., 2006). While PFOA has been detected in coastal water and seawater even in remote areas (Yamashita et al., 2005), the extent to which this may be due to ocean or atmospheric transport is uncertain. Ocean water transport of perfluorocarboxycyclic compounds is a combination of :a) discharges of PFCAs to surface waters and transport to oceans; b) atmospheric loadings of PFCAs to surface waters and transport to oceans; and c) discharge of precursors to surface waters, transformation to PFCAs and transport to oceans (Prevedouros et al., 2006).

In addition to the possible role of aquatic transport via oceans to the Arctic, the possibility of atmospheric transport of PFOA on marine aerosols has been proposed (Prevedouros et al., 2006). Due to its nature as surfactant, PFOA is expected to be enriched on the water surface. As hypothesized, marine aerosols may be generated from this PFOA enriched surface through gas-bubble production and collapse through breaking waves and rough sea conditions. The sea surface micro-layer may thus, supply the atmosphere with PFOA-rich particles which undergo atmospheric transport over, at least, short distances. Studies are needed to determine whether and to what extent marine aerosols contain PFOA and contribute to their global transport. The determination of whether perfluorocarboxylic acids are present, and to what extent, in marine aerosols, and whether this contributes to their global transport, is the subject of ongoing scientific investigations (Prevedouros et al., 2006).

<u>Conclusion</u>

Pure PFOA at room temperature has moderate vapour pressure (2.3 Pa). The vapour pressure of APFO is much lower with 0.008 Pa. APFO or PFOA dissolved in water dissociate to ions. Although the dissociated fraction is not subject to volatilization, depending on the pH, pure PFOA is expected to volatize from water to a certain degree.

Due to emissions for more than 50 years, PFOA is distributed worldwide in the marine environment, and hence may be transported to remote areas via the aqueous phase and the atmospheric phase. However, the significance of these sources are not currently known. Both atmospheric and aquatic transport mechanisms are actively being investigated.

PFOA and PFOA precursors including fluorotelomer alcohols, olefins and perfluoroalkyl sulfonyl derivates are subject to long range transport. The relative environmental significance of these sources are not known currently.

Distribution of PFOA via sewage sludge and effluents from Waste Water Treatment Plants (WWTP)

A lot of studies estimated an increase of PFOA between the influent and the effluent of a WWTP. The most reliable ones are discussed below:

In one study six WWTP (domestic and commercial wastewater as well as domestic and industrial wastewater) were tested (Sinclair and Kannan, 2006). The concentrations in the effluents ranged from 58 – 1050 ng/L. The highest concentrations of PFOA were detected in two WWTP which had no industrial influence. The authors assumed that high PFOA concentrations result from the commercial wastewater, primarily from the cleaning of fluorochemical-treated products. Furthermore, Sinclair and Kannan studied the mass loading and fate of PFOA in two of this WWTP (identical treatment processes). They identified no change of the mass flows after primary treatment. But after secondary treatment the mass flows significantly increased (Plant A: influent 6.0-8.9 g/day, primary-treated 5.6-10 g/day, effluent 11-21 g/day; Plant B: influent 2.9-6.0 g/day, primary-treated 2.3-6.0 g/day, effluent 6.0-7.8 g/day). This increase could follow from biodegradation of precursors to PFOA during the activated sludge treatment.

Another study compared the PFOA content in wastewater in two different WWTP (Yu et al., 2009). Plant A received 95 % domestic wastewater and plant B 60 % industrial and 40 % domestic wastewater. The waste water treatment was different in both WWTP. Whereas plant A was based on a conventional activated sludge process line (CAS), a liquid treatment module (LTM) and a membrane biological reactor (MBR), plant B was only based on a conventional activated process line. Mean mass flow of PFOA increased 41.6 % in CAS of plant A and 67.0 % in CAS of plant B and 76.6 % in MBR, while remained unchanged after the treatment of LTM. These findings suggest that change in mass flow of PFOA in secondary sludge treatment may be determined by the presence of precursors and operating sludge retention time of the activated sludge system. In contrast to the study from Sinclair and Kannan (Sinclair and Kannan, 2006), PFOA concentrations of the WWTP with industrial influence were much higher than the WWTP with mainly domestic wastewater, although there were no known source of exposure of fluorochemicals.

Boulanger et al. investigated a WWTP that receives domestic and industrial wastewater (Boulanger et al., 2005). Also in this study PFOA concentrations increased from influent (>4 ng/l; exact quantitative determination could not be made due to low recoveries of the compound in field spike samples) to effluent (22±2.1 ng/L). Boulanger et al. reported that the transformation of precursors within WWTP in not an important source of these compounds compared to direct use and disposal of products containing residual amounts.

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