**Format for submitting pursuant to Article 8 of the Stockholm Convention the information specified in Annex E of the Convention**

# Introductory information

### Name of the submitting Party/observer
NGO Observer: International POPs Elimination Network (IPEN)

### Contact details
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### Chemical name
Perfluorooctane Sulfonate (PFOS) and 96 PFOS-related substances

### Date of submission
27 January 2006

# (a) Sources, including as appropriate **(provide summary information and relevant references)**

## (i) Production data:

See PERFLUOROOCTANE SULFONATE (PFOS), Dossier prepared in support for a nomination of PFOS to the UN-ECE LRTAP Protocol and the Stockholm Convention The dossier is prepared by the Swedish Chemicals Inspectorate (KemI) and the Swedish EPA, Sweden, August 2004

## Quantity

## Location

## Other

## (ii) Uses

See PERFLUOROOCTANE SULFONATE (PFOS), Dossier prepared in support for a nomination of PFOS to the UN-ECE LRTAP Protocol and the Stockholm Convention The dossier is prepared by the Swedish Chemicals Inspectorate (KemI) and the Swedish EPA, Sweden, August 2004

## (iii) Releases:

*"Generation of FCs in wastes during use of spray can products by residential consumers … 34% of product expelled from the can is initially lost as waste to the air, with the potential for deposition. Spray cans used to apply treatments in the home are expected to retain a small fraction of the product material at the time of disposal. Based on information for non-food spray cans in general, up to 12.5% of the original contents may remain in spray cans at the time of disposal.*

*…it is expected that 50% of the FC treatment will be removed over the nine-year life of the carpet due to walking and vacuuming, while an additional 45% of the FC treatment will be removed in steam cleaning throughout the carpet life...*

*Garments treated with home-applied products are assumed to wear in a similar manner to textile mill applied treatments. Thus, 73% of these treatments are expected to wash off during garment cleaning over a two-year life span…*

*Any remaining FCs on coated products at the end of their usefil life are assumed to be disposed of at the national averages for landfill disposal (83%) and incineration* (17%)"

Sulfonated Perfluorochemicals: U.S. Release Estimation - 1997, Part 1: Life-Cycle Waste Stream Estimates. Final Report for b 3M Specialty Materials. Prepared by Battelle Memorial Institute, Ohio, April 21,2000

## Discharges

[1] Cheryl A. Moody, Jonathan W. Martin, Wai Chi Kwan, Derek C. G. Muir, and Scott A. Mabury, **Monitoring Perfluorinated Surfactants in Biota and Surface Water Samples Following an Accidental Release of Fire-Fighting Foam into Etobicoke Creek**. *Environ. Sci. Technol.,* 36 (4), 545 -551, 2002

In June 2000, 22000 L of fire retardant foam containing perfluorinated surfactants was accidentally released at L. B. Pearson International Airport, Toronto, ON, and subsequently entered into Etobicoke Creek, a tributary to Lake Ontario. Analytical tools that include liquid chromatography/tandem mass spectrometry (LC/MS/MS) and $^{19}$F NMR were employed to characterize fish (common shiner, *Notropus cornutus*) and surface water samples collected following the discharge of the perfluorinated material. Total perfluoroalkanesulfonate (4, 6, and 8 carbons) concentrations in fish liver samples ranged from 2.00 to 72.9 μg/g, and total perfluorocarboxylate (5-14 carbons) concentrations ranged from 0.07 to 1.02 μg/g. In addition to fish samples, total perfluoroalkanesulfonate (6 and 8 carbons) concentrations were detected in creek water samples by LC/MS/MS over a 153 day sampling period with concentrations ranging from <0.017 to 2260 μg/L; perfluorooctanoate concentrations (<0.009-11.3 μg/L) were lower than those observed for the perfluoroalkanesulfonates. By $^{19}$F NMR, the total perfluorinated surfactant concentrations in surface water samples ranged from <10 to 17000 μg/L.

*A bioaccumulation factor range of 6300-125000 was calculated for perfluorooctanesulfonate, based on concentrations in fish liver and surface water.*

## Losses

**SEDIMENTS AND DOMESTIC SLUDGE**:

[2] Higgins CP, Field JA, Criddle CS, & Luthy RG., **Quantitative determination of perfluorochemicals in sediments and domestic sludge.** *Environ Sci Technol*. 2005 Jun 1;39(11):3946-56

Limited available data indicate that some PFCs such as perfluorooctane sulfonate (PFOS) may strongly sorb to solids, and sewage sludge is widely suspected as a major sink of PFCs entering municipal waste streams. A quantitative analytical method was developed that consists of liquid solvent extraction of the analytes from sediments and sludge, cleanup via solid-phase extraction, and injection of the extracts with internal standards into a high-performance liquid chromatography (HPLC) system coupled to a tandem mass spectrometer (LC/MS/MS). The limits of detections of the method were analyte and matrix dependent, but ranged from 0.7 to 2.2 ng/g and 0.041 to 0.246 ng/g (dry weight) for sludge and sediment, respectively.

*The concentration of PFCs in domestic sludge ranged from 5 to 152 ng/g for total perfluorocarboxylates and 55 to 3370 ng/g for total perfluoroalkyl sulfonyl-based chemicals. Data from a survey of San Francisco Bay Area sediments suggest widespread occurrence of PFCs in sediments at the low ng/g to sub-ng/g level. Substances that may be transformed to PFOS, such as 2-(-(N-ethylperfluorooctanesulfonamido) acetic acid (N-EtFOSAA) and 2-(N-methylperfluorooctanesulfonamido) acetic acid (N-MeFOSAA), are present in both sediments and sludge at levels often exceeding PFOS.*

[3] Schultz, Melissa, Higgins, Christopher, Luthy, Richard , Barofsky, Douglas, Field, Jennifer, **Behavior and fate of fluorochemicals during wastewater treatment.** SETAC 2005. Available at <http://abstracts.co.allenpress.com/pweb/setac2005 >

Untreated fluorochemicals may enter the environment via wastewater effluent, septic discharge or land application of biosolids. A field study was conducted at a full-scale municipal wastewater treatment plant to determine the mass flows of selected perfluoroalkyl sulfonates, perfluoroalkyl carboxylates, fluorotelomer sulfonates, and perfluoroalkyl sulfonamides in wastewater and sludge. 24 h composite samples of wastewater (raw influent, primary effluent, trickling filter effluent, secondary effluent, and final effluent) and grab samples of sludge (primary, thickened, activated, anaerobically digested, and storage lagoon) were collected over a duration of 10 days and were analyzed by liquid chromatography (LC), electrospray ionization (ESI) tandem mass spectrometry (MS/MS). To gain further understanding of fluorochemical behavior throughout wastewater treatment, additional studies were performed to look at the solid-water partitioning of fluorochemicals and their daily and diurnal variations. Overall removals and increases of fluorochemical concentrations in wastewater treatment plants were observed when the effluent concentrations were compared to the influent levels.

Individual fluorochemical concentrations in the wastewater and sludge ranged from 1.1 ng/L to 32.5 ng/L and 1 ng/g (dry wt) to 737 ng/g (dry wt), respectively. Perfluoroalkyl sulfonates were found to increase significantly (≈200%) in the plant mass balance (30 days). Fluoroalkyl sulfonamide acetic acids were also found to increase by at least 300% throughout the sludge treatment process with a residence time of a year. Perfluoroalkyl carboxylates were overall removed by the wastewater treatment plant.

*From this plant, significant quantities of fluorochemicals are discharged with treated wastewater and biosolids, indicating that wastewater treatment plants are point sources of fluorochemicals.*

## Emissions

**SURFACE WATER :**

[4] Bryan Boulanger, John Vargo, Jerald L. Schnoor, and Keri C. Hornbuckle, **Detection of Perfluorooctane Surfactants in Great Lakes Water.** *Environ. Sci. Technol.,* **38** (15), 4064 -4070, 2004

Sixteen Great Lakes water samples were analyzed for eight perfluorooctane surfactants. The monitored perfluorooctane surfactants were quantitatively determined using single quadrupole HPLC/MS and qualitatively confirmed using ion trap MS/MS. Additionally, PFOS was quantitatively confirmed using triple quadrupole LC/MS/MS. Concentrations of PFOS and PFOA in the two lakes ranged from 21-70 and 27-50 ng/L, respectively.

*Analysis also showed the presence of PFOS precursors, N-EtFOSAA (range of 4.2-11 ng/L) and FOSA (range of 0.6-1.3 ng/L), in all samples above the LOQ. PFOSulfinate, another precursor, was identified at six of eight locations with a concentration range, when present, of <2.2-17 ng/L. Other PFOS precursors, N-EtFOSE, PFOSAA, and N-EtFOSA were not observed at any of the sampling locations. These are the first reported concentrations of perfluorooctane surfactants in Great Lakes water and the first report of PFOS precursors in any water body.*

**RAINWATER:**

[5] Mark Loewen, Thor Halldorson, Feiyue Wang, and Gregg Tomy, Fluorotelomer **Carboxylic Acids and PFOS in Rainwater from an Urban Center in Canada.** *Environ. Sci. Technol.,* **39** (9), 2944 -2951, 2005

Perfluorocarboxylic acids (PFCAs) and perfluorooctane sulfonate (PFOS) were analyzed in the rainwater samples from Winnipeg, Manitoba, Canada using established LC/MS/MS methods.

*PFOS was deposited in rainwater with a concentration of 0.59 ng/L while PFCAs were not detected above their respective method detection limits. Low parts per trillion levels of the $C_{10}$- and $C_{12}$- fluorotelomer carboxylic acids (FTCAs) and fluorotelomer unsaturated*

*carboxylic acids (FTUCAs) were also detected, suggesting that one possible pathway of removing FTOHs from the atmosphere is through oxidation and wet deposition.*

**OCEANS** :

[6] Yamashita N, Kannan K, Taniyasu S, Horii Y, Petrick G, Gamo T., **A global survey of perfluorinated acids in oceans. (**National Institute of Advanced Industrial Science and Technology, Japan ) *Mar Pollut Bull*. 2005 May 20; [Epub ahead of print]

This study demonstrated a highly sensitive analytical method to monitor perfluorinated compounds in oceanic waters, capable of detecting PFOS, perfluorohexanesulfonate (PFHS), perfluorobutanesulfonate (PFBS), perfluorooctanoate (PFOA), perfluorononanoate (PFNA), and perfluorooctanesulfonamide (PFOSA) at a few pg/L in oceanic waters.

*Seawater samples collected during 2002-2004 in the central to eastern Pacific Ocean, South China Sea and Sulu Seas, north and mid Atlantic Ocean, and the Labrador Sea plus an additional 50 samples of coastal seawater from several Asian countries (Japan, China, Korea) were analyzed. PFOA was found at levels ranging from several thousands of pg/L in water samples collected from coastal areas in Japan to a few tens of pg/L in the central Pacific Ocean. PFOA was the major contaminant detected in oceanic waters, followed by PFOS.*

**AIR :**

[7] Barber, J, Berger, U, Jones, K, **A study of fluorinated alkyl compounds in European air samples**. SEATAC 2005. Available at <http://abstracts.co.allenpress.com/pweb/setac2005 >

While perfluorooctane sulfonate (PFOS) and perfluorinated carboxylic acids (PFCAs) have been detected in a large variety of environmental samples from places as remote as the Arctic, these compounds are ionic and possess very low volatility, and therefore not expected to undergo long-range atmospheric transport (LRAT). In this study, it was proposed that neutral volatile precursor compounds, such as fluorotelomer alcohols (FTOHs) and fluorooctane sulfonamides/sulfonamide ethanols (FOSAs/FOSEs), might undergo LRAT and ultimately be degraded to PFCAs and PFOS at these remote locations. The study provided evidence of volatile fluorinated compounds in European air samples.

High-volume samplers were used to collect gas-phase and particle-bound fluorinated compounds on polyurethane foam (PUF)/XAD-2 resin/PUF sandwiches and glass fiber filters (GFFs), respectively. Field blanks and samples of $\approx$1400 $m^3$ air were collected at Manchester (UK, urban site) and Hazelrigg (40 miles north-west of Manchester, semi-rural site) in February and March 2005. Neutral compounds were extracted from the PUFs/XAD or filter with ethyl acetate and analyzed by GC/NCI- and GC/PCI-MS. Additionally, GFF samples were analyzed for PFCAs and perfluorinated sulfonates. These ionic compounds were extracted from the filter with methanol and analyzed by LC/TOF-MS.

*All 9 volatile target compounds were detected in the gas-phase at both locations, and in the particulate-phase in the Manchester samples, with concentrations higher in urban samples than rural samples. The volatile compounds with the highest concentrations were 8:2 FTOH, followed by 6:2 FTOH, both found in the hundreds of pg/m$^3$ range, with N-methyl-FOSE the most abundant of the FOSAs/FOSEs. A number of ionic compounds were present in the particulate-phase, including PFOA, PFOS, PFHpA, PFDcA, and TH-PFOS, with PFOS at ≈50 pg/m$^3$, and PFOA at levels >800 pg/m$^3$, making PFOA the most abundant fluorinated analyte in these air samples.*

## Other

**HOUSE DUST :**

[8] Kubwabo C, Stewart B, Zhu J, Marro L., **Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada.** *J Environ Monit*. 2005;7(11):1074-1078.

In order to better understand the human exposure routes of these chemicals, levels of PFOS, PFOA, perfluorobutane sulfonate (PFBS), perfluorohexane sulfonate (PFHS) and perfluorooctane sulfonamide (PFOSA) in house dust samples were investigated.

*The data revealed a correlation between the concentrations of PFCs and the percentage of carpeting in the house; older houses tended to have less carpeting, hence lower levels of these perfluorinated compounds in their dust.*

# (b) Hazard assessment for endpoints of concern, including consideration of toxicological interactions involving multiple chemicals **(provide summary information and relevant references)**

*"The toxicity profile of PFOS is similar among rats and monkeys. Repeated exposure of results in hepatotoxicity and mortality; the dose-response curve is very steep for mortality. This occurs in animals of all ages, although the neonate may be more sensitive. In addition, a 2-year bioassay in rats has shown that exposure to PFOS results in hepatocellular adenomas and thyroid follicular cell adenomas… Epidemiologic studies have shown an association of PFOS exposure and the incidence of bladder cancer; … Adverse signs of toxicity observed in Rhesus monkey studies included anorexia, emesis, diarrhea, hypoactivity, prostration, convulsions, atrophy of the salivary glands and the pancreas, marked decreases in serum cholesterol, and lipid depletion in the adrenals. The dose range for these effects was reported between 1.5-300 mg/kg/day. No monkeys survived beyond 3 weeks into treatment at 10 mg/kg/day or beyond 7 weeks into treatment at doses as low as 4.5 mg/kg/day."*

November 21, 2002 report: Hazard Assessment of Perfluorooctane sulfonate (PFOS) and its salts. Organisation for Economic Co-operation and Development. ENV/JM/RD(2002)17/FINAL.

## REPRODUCTIVE AND DEVELOPMENTAL EFFECTS

[9] Deanna J. Luebker, Raymond G. York, Kristen J. Hansenc, John A. Moore, and John L. Butenhoff ., **Neonatal mortality from in utero exposure to perfluorooctanesulfonate (PFOS) in Sprague–Dawley rats: Dose–response, and biochemical and pharamacokinetic parameters.** *Toxicology* V 215, Issues 1-2, 5 Nov. 2005, 149-169

Neonatal mortality has been observed following PFOS exposure in a two-generation reproduction study in rats and after dosing pregnant rats and mice during gestation. In the current study, additional doses of 0.8, 1.0, 1.2, and 2.0 mg/kg/day were included with original doses used in the two-generation study of 0.4 and 1.6 mg/kg/day in order to obtain data in the critical range of the dose–response curve. Dosing of dams occurred for 6 weeks prior to mating with untreated breeder males, through confirmed mating, gestation, and day four of lactation. Dose levels for the dose–response and etiological investigation were 0.0, 0.4, 0.8, 1.0, 1.2, 1.6, and 2.0 mg/kg/day PFOS.

*Statistically significant decreases in gestation length were observed in the 0.8 mg/kg and higher dose groups. Decreases in viability through lactation day 5 were observed in the 0.8 mg/kg and higher dose groups, becoming statistically significant in the 1.6 and 2.0 mg/kg dose groups. Reduced neonatal survival did not appear to be the result of reductions in lipids, glucose utilization, or thyroid hormones. The endpoints of gestation length and decreased viability were positively correlated, suggesting that late-stage fetal development may be affected in pups exposed to PFOS in utero and may contribute to the observed mortality.*

*Significant transfer of PFOS from dam to fetus in utero was confirmed, and results suggest that dam and corresponding fetal body burdens, as indicated by serum and liver PFOS levels, correlate with neonatal survival.*

[10] Deanna J. Luebker., Marvin T. Case, Raymond G. York, John A. Moore, Kristen J. Hansen, and John L. Butenhoff, **Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats.** *Toxicology*. V 215, Issues 1-2 , 5 November 2005, 126-148

To investigate the potential reproductive and developmental effects of PFOS, a two-generation reproduction study was conducted in rats. Male and female rats were dosed via oral gavage at dose levels of 0, 0.1, 0.4, 1.6, and 3.2 mg/(kg day) for 6 weeks prior to mating, during mating, and, for females, through gestation and lactation, across two generations. Due to substantial F1 neonatal toxicity observed in the 1.6 and 3.2 mg/(kg day) groups, continuation into the second generation was limited to F1 pups from the 0, 0.1, and 0.4 mg/(kg day) groups. No adverse effects were observed in F0 females or their fetuses upon caesarean sectioning at gestation day 10.

*Statistically significant reductions in body-weight gain and feed consumption were observed in F0 generation males and females at dose levels of 0.4 mg/(kg day) and higher, but not in F1 adults. PFOS did not affect reproductive performance (mating, estrous cycling, and fertility); however, reproductive outcome, as demonstrated by decreased*

*length of gestation, number of implantation sites, and increased numbers of dams with stillborn pups or with all pups dying on lactation days 1–4, was affected at 3.2 mg/(kg day) in F0 dams.*

Neonatal toxicity in F1 pups, as demonstrated by reduced survival and body-weight gain through the end of lactation, occurred at a maternal dose of 1.6 mg/(kg day) and higher while not at dose levels of 0.1 or 0.4 mg/(kg day) or in F2 pups at the 0.1 or 0.4 mg/(kg day) dose levels tested. In addition to these adverse effects, slight yet statistically significant developmental delays occurred at 0.4 (eye opening) and 1.6 mg/(kg day) (eye opening, air righting, surface righting**,** and pinna unfolding) in F1 pups. Based on these data, the NOAELs were as follows: reproductive function: F0 ≥ 3.2 and F1 ≥ 0.4 mg/(kg day); reproductive outcome: F0 = 1.6 and F1 ≥ 0.4 mg/(kg day); overall parental effects: F0 = 0.1 and F1 ≥ 0.4 mg/(kg day); offspring effects: F0 = 0.4 and F1 ≥ 0.4 mg/(kg day).

*Results of this study indicated that in utero exposure to PFOS causally contributed to post-natal pup mortality, and that pre-natal and post-natal exposure to PFOS was additive with respect to the toxic effects observed in pups.*

[11] Grasty RC, Bjork JA, Wallace KB, Lau CS, Rogers JM., **Effects of prenatal perfluorooctane sulfonate (PFOS) exposure on lung maturation in the perinatal rat.** *Birth Defects Res B Dev Reprod Toxicol*. 2005 Oct 25;74(5):405-416

Environmental PFOS may be from its use as a surfactant, hydrolysis of perfluorooctanesulfonyl fluoride, and degradation of N-alkyl-perfluorooctanesulfonamide compounds formerly used in numerous applications. Prenatal exposure to PFOS in rodents causes neonatal mortality; treatment on gestation days (GD) 19-20 is sufficient to induce neonatal death in rats. Affected pups are born alive but present with labored breathing. Their lungs are pale and often do not expand fully on perfusion. In this study, pregnant Sprague-Dawley rats received 0, 25, or 50 mg/kg/day PFOS/K(+) orally on GD 19-20. Lungs from GD 21 fetuses and neonates were prepared for histology and morphometry. Rescue experiments included co-administration of dexamethasone or retinyl palmitate with PFOS. Pulmonary surfactant was investigated with mass spectrometry in GD 21 amniotic fluid and neonatal lungs. Microarray analysis was carried out on PND 0 lungs.

*Histologically, alveolar walls were thicker in lungs of PFOS-exposed newborns compared to controls. The ratio of solid tissue:small airway was increased, suggesting immaturity. Morphometric changes in lungs of PFOS exposed neonates were suggestive of immaturity, but the failure of rescue agents and normal pulmonary surfactant profile indicate that the labored respiration and mortality observed in PFOS-treated neonates was not due to lung immaturity.*

[12] Ankley GT, Kuehl DW, Kahl MD, Jensen KM, Linnum A, Leino RL, Villeneuvet DA., **Reproductive and developmental toxicity and bioconcentration of perfluorooctanesulfonate in a partial life-cycle test with the fathead minnow (Pimephales promelas).** *Environ Toxicol Chem*. 2005 Sep;24(9):2316-24.

This study assessed the reproductive and developmental toxicity and bioconcentration of PFOS in the fathead minnow (Pimephales promelas). Sexually mature fish were exposed via the water for 21 d to 0 (control), 0.03, 0.1, 0.3, or 1 mg PFOS/L, and effects on reproductive capacity and endocrinology were assessed. To determine possible developmental effects, a subset of embryos from parental exposures at each test concentration was held for an additional 24 d in the same PFOS treatments. A concentration of I mg PFOS/L was lethal to adults within two weeks. The 21-d 50% effect concentration (95% confidence interval) for effects on fecundity of the fish was 0.23 (0.19-0.25) mg PFOS/L.

*Exposure to PFOS caused various histopathological alterations, most prominently in ovaries of adult females. Adult males exposed to 0.3 mg PFOS/L for 21 d exhibited decreased aromatase activity and elevated concentrations of plasma 11-ketotestosterone and testosterone. No significant adverse effects on survival or growth were observed in developing fathead minnows held for 24 d at PFOS concentrations up to 0.3 mg/L. Adult fathead minnows readily accumulated PFOS from the water. The largest concentrations of PFOS were in blood, followed by liver and then gonad; for all tissues, females accumulated higher concentrations than males.*

[13] Oakes KD, Sibley PK, Martin JW, MacLean DD, Solomon KR, Mabury SA, Van Der Kraak GJ., **Short-term exposures of fish to perfluorooctane sulfonate: acute effects on fatty acyl-coa oxidase activity, oxidative stress, and circulating sex steroids.** *Environ Toxicol Chem*. 2005 May;24(5):1172-81

This study investigated the effects of exposure to waterborne perfluorooctane sulfonate (PFOS) on oxidative stress and reproductive endpoints in fish, including the fathead minnow (Pimephales promelas) and rainbow trout (Oncorhynchus mykiss), as well as relatively insensitive taxa such as creek chub (Semotilus atromaculatus), spottail shiner (Notropis hudsonius), and white sucker (Catostomus commersoni). In all fish species, short-term (14-28 d) exposure to PFOS produced only modest mortality at concentrations consistent with environmental spill scenarios.

*However, PFOS consistently increased hepatic fatty acyl-CoA oxidase activity and increased oxidative damage, as quantified using the 2-thiobarbituric acid-reactive substances assay. Plasma testosterone, 11-ketotestosterone, and 17beta-estradiol titers were often elevated with PFOS exposure. Vitellogenin, the egg yolk precursor protein, was occasionally altered in the plasma with PFOS exposure, but responses varied with maturity. Short-term PFOS exposure produced significant impacts on biochemical and reproductive endpoints in fish at concentrations consistent with environmental spills.*

[14] Fan YO, Jin YH, Ma YX, Zhang YH**., Effects of perfluorooctane sulfonate on spermiogenesis function of male rats.** *Wei Sheng Yan Jiu*. 2005 Jan;34(1):37-9

This study evaluated the effects of perfluorooctane sulfonate (PFOS) on spermiogenesis function of male rats. 36 male rats were randomly divided into 4 groups, which received 0, 0.5, 1.5, 4.5 mg x kg(-1) PFOS by food intake per day for 65 days. The testicular and epididymal viscera coefficients, the number, motility and deformity of sperm were

examined. The activities of lactate dehydrogenase isoenzyme-x (LDHx), sorbitol dehydrogenase (SDH) and the generation of maglonydiadehyde (MDA) in the testes were also measured. The viscera coefficients did not show any significant change ( $P > 0.05$) while the body weight and weight of testis decreased ( $P < 0.05$) in treated rats compared with the corresponding control group animals.

*In 1.5, 4.5 mg x kg(-1) PFOS treated rats, there were significant decreases in the sperm count ($P < 0.05$) and the mean activities of LDHx and SDH whereas obvious increases in the rate of sperm deformity ( $P < 0.05$). In 4.5 mg x kg(-1) PFOS group the generation of MDA increased ($P < 0.05$) while the motility of sperm reduced ($P < 0.05$) with respect to the control value. This suggested that PFOS could elicit the impairment of sperm production and maturation of male rats.*

[15] Thibodeaux JR, Hanson RG, Rogers JM, Grey BE, Barbee BD, Richards JH, Butenhoff JL, Stevenson LA, Lau C., **Exposure to Perfluorooctane Sulfonate During Pregnancy in Rat and Mouse. I. Maternal and Prenatal Evaluations.** *Toxicol Sci* 2003 May 28;

The maternal and developmental toxicities of perfluorooctane sulfonate (PFOS) were evaluated in the rat and mouse. Pregnant Sprague-Dawley rats were given 1, 2, 3, 5, or 10 mg/kg PFOS daily by gavage from gestational day (GD) 2 to GD 20; CD-1 mice were similarly treated with 1, 5, 10, 15 and 20 mg/kg PFOS from GD 1 to GD 17. Controls received 0.5% Tween-20 vehicle (1 ml/kg for rats and 10 ml/kg for mice). Maternal weight gain, food and water consumption, and serum chemistry were monitored. Rats were killed on GD 21, and mice on GD 18. PFOS levels in maternal serum, maternal and fetal livers were determined.

*Maternal weight gains in both species were suppressed by PFOS in a dose-dependent manner, likely attributed to reduced food and water intake. Serum PFOS levels increased with dosage, and liver levels were approximately 4-fold higher than serum.*

Serum thyroxine (T4) and triiodothyronine (T3) in the PFOS-treated rat dams were significantly reduced as early as one week after chemical exposure, although no feedback response of thyroid-stimulating hormone (TSH) was observed. A similar pattern of reduction in T4 was also seen in the pregnant mice. Maternal serum triglycerides were significantly reduced, particularly in the high dose groups, although cholesterol levels were not affected.

In the mouse dams, PFOS produced a marked enlargement of the liver at 10 mg/kg and higher dosages. In the rat fetuses, PFOS was detected in the liver, but at levels nearly half of those in the maternal counterparts, regardless of administered doses. In both rodent species, PFOS did not alter the numbers of implantations or live fetuses at term, although small deficits in fetal weight were noted in the rat.

*A host of birth defects including cleft palate, anasarca, ventricular septal defect, and enlargement of the right atrium were seen in both rats and mice, primarily in the 10 and 20 mg/kg dosage groups, respectively. Our results demonstrate both maternal and developmental toxicity of PFOS in the rat and mouse.*

## HORMONE EFFECTS

[16] Wenyue Hu, Paul D. Jones, Trine Celius and John P. Giesy, **Identification of genes responsive to PFOS using gene expression profiling.** *Environmental Toxicology and Pharmacology* - Volume 19, Issue 1, January 2005, 57-70

In this study, the Affymetrix rat genome U34A genechip was used to identify alterations in gene expression due to PFOS exposure. Rat hepatoma cells were treated with PFOS at 2–50 mg/L for 96 h. Sprague-Dawley rats were orally dosed with PFOS at 5 mg/kg/day for 3 days or 3 weeks.

*Genes that were significantly (P <0.0025) induced were primarily genes for fatty acid metabolizing enzymes, cytochrome P450s, or genes involved in hormone regulation. Consistent expression profiles were obtained for replicate exposures, for short-term and long-term in vivo exposures, and for acute and chronic exposures. One major pathway affected by PFOS was peroxisomal fatty acid* B-*oxidation, which could be explained by the structural similarity between PFOS and endogenous fatty acids.*

[17] Austin ME, Kasturi BS, Barber M, Kannan K, MohanKumar PS, MohanKumar SM., **Neuroendocrine effects of perfluorooctane sulfonate in rats**; *Environ Health Perspect.* 2003 Sep;111(12):1485-9.

To investigate the effect of PFOS on the neuroendocrine system, adult female rats were injected intraperitoneally with 0, 1, or 10 mg PFOS/kg body weight (BW) for 2 weeks. Food and water intake, BW, and estrous cycles were monitored daily. At the end of treatment, PFOS levels in tissues were measured by high-performance liquid chromatography (HPLC) interfaced with electrospray mass spectrometry. Changes in brain monoamines were measured by HPLC with electrochemical detection, and serum corticosterone and leptin were monitored using radioimmunoassay.

*Treatment with PFOS produced a dose-dependent accumulation of this chemical in various body tissues, including the brain; decreased food intake and BW in a dose-dependent manner; affected estrous cyclicity and increased serum corticosterone levels while decreasing serum leptin concentrations; and increased norepinephrine concentrations in the paraventricular nucleus of the hypothalamus. These results indicate that exposure to PFOS can affect the neuroendocrine system in rats.*

[18] Deanna J. Luebker, Kris J. Hansen, Nathan M. Bass, John L. Butenhoff, and Andrew M. Seacat., **Interactions of flurochemicals with rat liver fatty acid-binding protein,** *Toxicology* V 176, Issue 3, 15 July 2002, pp175-185

Liver-fatty acid binding protein (L-FABP) is an abundant intracellular lipid-carrier protein. The hypothesis that perfluorooctanesulfonate (PFOS), perfluorooctanoate (PFOA), and certain related perfluorooctanesulfonamide-based fluorochemicals (PFOSAs) can interfere with the binding affinity of L-FABP for fatty acids was tested. This study determined the relative effectiveness of PFOA, PFOS, N-ethylperfluorooctanesulfonamide (N-EtFOSA),

N-ethylperfluorooctanesulfonamido ethanol (N-EtFOSE), and of the strong peroxisome proliferator Wyeth-14 643 (WY) to inhibit 11-(5-dimethylaminonapthalenesulphonyl)-undecanoic acid (DAUDA) binding to-L-FABP. The dissociation constant (Kd) of the DAUDA-L-FABP complex was 0.47 nM.

*PFOS exhibited the highest level of inhibition of DAUDA-L-FABP binding in the competitive binding assays, followed by N-EtFOSA, WY, and, with equal IC50s, N-EtFOSE and PFOA. The in vitro data presented in this study support the hypothesis that PFOS and other fluorochemicals interfere with the binding of fatty acids or other endogenous ligands to L-FABP, contributing to the toxicity in rodents fed fluorochemicals.*

## LIVER TOXICITY

[19] Philippe Tony Hoff, Jan Scheirs,Kristin Van de Vijver, Walter Van Dongen, Eddy Louis Esmans, Ronny Blust, and Wim De Coen, **Biochemical Effect Evaluation of Perfluorooctane Sulfonic Acid-Contaminated Wood Mice (*Apodemus sylvaticus*).** *Environ Health Perspect* 112:681-686 (2004)

Wood mice (*Apodemus sylvaticus*) were captured at Blokkersdijk, a nature reserve in the immediate vicinity of a fluorochemical plant in Antwerp, Belgium, and at Galgenweel, 3 kilometers farther away.

The liver perfluorooctane sulfonic acid (PFOS) concentrations in the Blokkersdijk mice were extremely high (0.47-178.55 µg/g wet weight). Perfluorononanoic, perfluorodecanoic, perfluoroundecanoic, and perfluorododecanoic acids were found sporadically in the liver tissue of the Blokkersdijk mice. The liver PFOS concentrations at Galgenweel were significantly lower than those at Blokkersdijk (0.14-1.11 µg/g wet weight).

Several liver end points were significantly elevated in the Blokkersdijk mice: liver weight, relative liver weight, peroxisomal ß-oxidation activity, microsomal lipid peroxidation level, and mitochondrial fraction protein content.

*The liver weight, relative liver weight, and liver microsomal lipid peroxidation level (indicating oxidative stress) increased significantly with the liver PFOS concentration. The study also suggests that hepatic PFOS bioaccumulation is age dependent and that maternal PFOS transfer to the young during pregnancy and/or lactation might occur.*

[20] Hoff PT, Van de Vijver K, Dauwe T, Covaci A, Maervoet J, Eens M, Blust R, De Coen W., **Evaluation of biochemical effects related to perfluorooctane sulfonic acid exposure in organohalogen-contaminated great tit (Parus major) and blue tit (Parus caeruleus) nestlings.** *Chemosphere*. 2005 Jun 24.

A perfluorooctane sulfonic acid (PFOS) biomonitoring survey was conducted on great tit (Parus major) and blue tit (Parus caeruleus) nestlings from Blokkersdijk, a bird reserve in the proximity of a fluorochemical plant in Antwerp (Belgium) and Fort IV, a control area.

PFOS, together with 11 organochlorine pesticides, 20 polychlorinated biphenyl congeners and 7 polybrominated diphenyl ethers were measured in liver tissue.

*The hepatic PFOS concentrations at Blokkersdijk (86-2788 and 317-3322ng/g wet weight (ww) for great and blue tit, respectively) were among the highest ever measured and were significantly higher than at the control area (17-206 and 69-514ng/g ww for great and blue tit, respectively).*

The hepatic PFOS concentration was species- and sex-independent and correlated significantly and positively with the serum alanine aminotransferase activity and negatively with the serum cholesterol and triglyceride levels in both species but did not correlate with condition or serum protein concentration.

*In the great tit, a significant positive correlation was observed between the liver PFOS concentration and the relative liver weight. In the blue tit, the hepatic PFOS concentration correlated positively and significantly with hematocrite values.*

## INTERCELLULAR COMMUNICATION

[21] Wenyue Hu, Paul D. Jones, Brad L. Upham, James E. Trosko, Christopher Lau and John P. Giesy., **Inhibition of Gap Junctional Intercellular Communication by Perfluorinated Compounds in Rat Liver and Dolphin Kidney Epithelial Cell Lines *in Vitro* and Sprague-Dawley Rats *in Vivo*.** *Toxicological Sciences* 68, 429-436 (2002)

Gap junctional intercellular communication (GJIC) is the major pathway of intercellular signal transduction, and is thus important for normal cell growth and function. Because other perfluoroalkanes had been shown to inhibit GJIC, the effects of PFOS and related sulfonated fluorochemicals on GJIC were studied using a rat liver epithelial cell line (WB-F344) and a dolphin kidney epithelial cell line (CDK). *In vivo* effects on GJIC were studied in Sprague-Dawley rats orally exposed to PFOS for 3 days or 3 weeks. Effects on GJIC were measured using the scrape loading dye technique. PFOS, perfluorooctane sulfonamide (PFOSA), and perfluorohexane sulfonic acid (PFHA) were found to inhibit GJIC in a dose-dependent fashion, and this inhibition occurred rapidly and was reversible. A structure activity relationship was established among all 4 tested compounds, indicating that the inhibitory effect was determined by the length of fluorinated tail and not by the nature of the functional group.

*The results of the studies of the 2 cell lines and the in vivo exposure were comparable, suggesting that the inhibitory effects of the selected perfluorinated compounds on GJIC were neither species- nor tissue-specific and can occur both in vitro and in vivo.*

## OTHER

[22] Kouji Harada., Feng Xu, Kyoichi Ono, Toshihiko Iijima, and Akio Koizumia. **Effects of PFOS and PFOA on L-type Ca2+ currents in guinea-pig ventricular myocytes.** *Biochemical and Biophysical Research Communications* - Volume 329, Issue 2 , 8 April 2005, 487-494

This study investigated the effects of PFOS and PFOA on action potentials and L-type Ca2+ currents, ICaL, in isolated guinea-pig ventricular myocytes using whole-cell patch-clamp recording. In current-clamp experiments, PFOS significantly decreased the rate of spike, action potential duration, and peak potential at doses over 10 μM. In voltage-clamp experiments, PFOS increased the voltage-activated peak amplitude of ICaL, and shifted the half-activation and inactivation voltages of ICaL to hyperpolarization. PFOA had similar effects PFOS, but showed significantly lower potency.

*These findings suggest that PFOS may change membrane surface potential, thereby eliciting general effects on calcium channels.*

# (c) Environmental fate (provide summary information and relevant references)

## Chemical/physical properties

## Persistence

*"Perfluorooctane sulfonic acid (PFOS) results from the chemical or metabolic hydrolysis of POSF-derived FCs. Current information indicates that PFOS or its salts cannot be broken down further chemically under normally occurring environmental conditions. Therefore PFOS is the ultimate degradation product from POSF derived fluorochemicals and will generally persist in that form."*

3M Fluorochemical Use, Distribution and Release Overview (Unpub) Prepared by 3M Company, May 26, 1999.

## How are chemical/physical properties and persistence linked to environmental transport, transfer within and between environmental compartments, degradation and transformation to other chemicals?

**PFOS PRECURSORS**

[23] J. W. Martin, D. A. Ellis, S. A. Mabury, M. D. Hurley and T. J. Wallington, **Atmospheric Chemistry of Perfluoroalkanesulfonamides: Kinetic and Product Studies of the OH Radical and Cl Atom Initiated Oxidation of *N*-Ethyl Perfluorobutanesulfonamide**. *Environ. Sci. Technol.,* (Epub; December 24, 2005)

Perfluorooctanesulfonamides [$C_8F_{17}SO_2N(R^1)(R^2)$] present in the atmosphere may, via atmospheric transport and oxidation, contribute to perfluorocarboxylates (PFCA) and perfluorooctanesulfonate (PFOS) pollution in remote locations. Smog chamber experiments with the perfluorobutanesulfonyl analogue *N*-ethyl

perfluorobutanesulfonamide [NEtFBSA; $C_4F_9SO_2N(H)CH_2CH_3$] were performed to assess this possibility. By use of relative rate methods, rate constants for reactions of NEtFBSA with chlorine atoms (296 K) and OH radicals (301 K) were determined to be $k_{Cl} = (8.37 \pm 1.44) \times 10^{-12}$ and $k_{OH} = (3.74 \pm 0.77) \times 10^{-13}$ $cm^3$ $molecule^{-1}$ $s^{-1}$, indicating OH reactions will be dominant in the troposphere.

Modeling suggest that reaction with OH radicals will dominate removal of perfluoroalkanesulfonamides from the gas phase (wet and dry deposition will not be important) and that the atmospheric lifetime of NEtFBSA in the gas phase will be 20-50 days, thus allowing substantial long-range atmospheric transport. Liquid chromatography /tandem mass spectrometry (LC/MS/MS) analysis showed that the primary products of chlorine atom initiated oxidation were the ketone $C_4F_9SO_2N(H)COCH_3$; aldehyde 1, $C_4F_9SO_2N(H)CH_2CHO$; and a product identified as $C_4F_9SO_2N(C_2H_5O)^-$ by high-resolution MS but whose structure remains tentative.

Another reaction product, aldehyde 2, $C_4F_9SO_2N(H)CHO$, was also observed and was presumed to be a secondary oxidation product of aldehyde 1. Perfluorobutanesulfonate was not detected above the level of the blank in any sample; however, three perfluoroalkanecarboxylates ($C_3F_7CO_2^-$, $C_2F_5CO_2^-$, and $CF_3CO_2^-$) were detected in all samples.

*Taken together, results suggest a plausible route by which perfluorooctanesulfonamides may serve as atmospheric sources of PFCAs, including perfluorooctanoic acid.*

[24] Mahiba Shoeib, Tom Harner, Michael Ikonomou and Kurunthachalam Kannan, **Indoor and Outdoor Air Concentrations and Phase Partitioning of Perfluoroalkyl Sulfonamides and Polybrominated Diphenyl Ethers.** *Environ. Sci. Technol.,* **38** (5), 1313 -1320, 2004.

This study made connections between indoor sources of Perfluoroalkyls (PFAs) and polybrominated diphenyl ethers (PBDEs) and the potential and mode for their transport in air. In the case of the PFAs, three perfluoralkyl sulfonamides (PFASs) were investigated- *N*-methyl perfluorooctane sulfonamidoethanol (MeFOSE), *N*-ethyl perfluorooctane sulfonamidoethanol (EtFOSE), and *N*-methyl perfluorooctane sulfonamidethylacrylate (MeFOSEA). These are believed to act as precursors that eventually degrade to perfluorooctane sulfonate (PFOS), which is detected in samples from remote regions.

High-volume samples were collected for indoor and outdoor air to investigate the source signature and strength. Mean indoor air concentrations ($pg/m^3$) were 2590 (MeFOSE), 770 (EtFOSE), and 630 ($\sum$PBDE). The ratios of concentration between indoor and outdoor air were 110 for MeFOSE, 85 for EtFOSE, and 15 for $\sum$PBDE. The gas and particle phases were collected separately to investigate the partitioning characteristics of these chemicals. Measured particulate percentages were compared to predicted values determined using models based on the octanol-air partition coefficient ($K_{OA}$) and supercooled liquid vapor pressure ($P_L^0$); these models were previously developed for nonpolar, hydrophobic chemicals. To make this comparison for the three PFASs, it was necessary to measure their

$K_{OA}$ and vapor pressure. $K_{OA}$ values were measured as a function of temperature (0 to +20 °C).

*Values of log $K_{OA}$ at 20 °C were 7.70, 7.78, and 7.87 for MeFOSE, EtFOSE, and MeFOSEA, respectively. Partitioning to octanol increased at colder temperatures, and the enthalpies associated with octanol-air transfer ($\Delta H_{OA}$, kJ/mol) were 68-73 and consistent with previous measurements for nonpolar hydrophobic chemicals.*

Solid-phase vapor pressures ($P_S$) were measured at room temperature (23 °C) by the gas saturation method. Values of $P_S$(Pa) were $4.0 \times 10^{-4}$, $1.7 \times 10^{-3}$, and $4.1 \times 10^{-4}$, respectively. These were converted to $P_L$ for describing particle-gas exchange. Both the $P_L$-based model and the $K_{OA}$ model worked well for the PBDEs but were not valid for the PFASs, greatly underpredicting particulate percentages.

*These results suggest that existing $K_{OA}$- and $P_L$-based models of partitioning will need to be recalibrated for PFASs.*

## BIOTRANSFORMATION

[25] Gregg T. Tomy, Sheryl A. Tittlemier, Vince P. Palace, Wes R. Budakowski, Eric Braekevelt, Lyndon Brinkworth, and Ken Friesen, **Biotransformation of N-Ethyl Perfluorooctanesulfonamide by Rainbow Trout (Onchorhynchus mykiss) Liver Microsomes.** *Environ. Sci. Technol*., **38** (3), 758 -762, 2004.

In this study Rainbow trout (Onchorhynchus mykiss) liver microsomes were incubated with N-ethyl perfluorooctanesulfonamide [N-EtPFOSA, $C_8F_{17}SO_2NH(C_2H_5)$], to examine the possibility of in vitro biotransformation to perfluorooctane sulfonate (PFOS, $C_8F_{17}SO_3$) and perfluorooctanoate (PFOA, $C_7F_{15}COO^-$). Incubations were performed by exposing trout liver microsomes to N-EtPFOSA at 8 °C in the dark. Reaction mixtures were analyzed after incubation periods of 0, 2, 4, 8, 16, and 30 h for N-EtPFOSA, PFOS, PFOA, and perfluorooctanesulfonamide (PFOSA, $C_8F_{17}SO_2NH_2$), a suspected intermediate.

*Amounts of PFOS and PFOSA were found to increase with incubation time, but only background levels of PFOA were detected. Three possible reaction pathways are proposed for the conversion of N-EtPFOSA to PFOS:*

*(i) direct conversion of N-EtPFOSA to PFOS by deethylamination accompanied by conversion of the sulfone group to sulfonate,*

*(ii) deethylation of N-EtPFOSA to PFOSA, followed by deamination to form PFOS; and*

*(iii) direct hydrolysis of N-EtPFOSA.*

*These findings represent the first report indicating a possible biotransformation of a perfluorosulfonamide to PFOS in fish and may help to explain the detection of PFOS, which is relatively involatile, and thus not likely to undergo atmospheric transport, in biota from remote regions*.

[26] Lin Xu, Daria M. Krenitsky, Andrew M. Seacat, John L. Butenhoff, and M. W. Anders, **Biotransformation of *N*-Ethyl-*N*-(2-hydroxyethyl)perfluorooctanesulfonamide by Rat Liver Microsomes, Cytosol, and Slices and by Expressed Rat and Human Cytochromes P450.** *Chem. Res. Toxicol.,* **17** (6), 767 -775, 2004

*N*-Substituted perfluorooctanesulfonamides are believed to be degraded to Perfluorooctanesulfonic acid (PFOS) and, therefore, contribute to the accumulation of PFOS in the environment. *N*-Ethyl-*N*-(2-hydroxyethyl)perfluorooctanesulfonamide (*N*-EtFOSE) is converted to PFOS in experimental animals. The objective of this study was to elucidate the pathways for the biotransformation of *N*-EtFOSE, which is a major precursor and component of PFOS-based compounds. *N*-EtFOSE and several putative metabolites were incubated with liver microsomes and cytosol and with liver slices from male Sprague-Dawley rats.

Microsomal fractions fortified with NADPH catalyzed the *N*-deethylation of *N*-EtFOSE to give *N*-(2-hydroxyethyl)perfluorooctanesulfonamide (FOSE alcohol) and of FOSE alcohol to give perfluorooctanesulfonamide (FOSA). These *N*-dealkylation reactions were catalyzed mainly by male rat P450 2C11 and P450 3A2 and by human P450 2C19 and 3A4/5. Rat liver microsomal fractions incubated with UDP-glucuronic acid catalyzed the *O*-glucuronidation of *N*-EtFOSE and FOSE alcohol and the *N*-glucuronidation of FOSA. Cytosolic fractions incubated with $NAD^+$ catalyzed the oxidation of FOSE alcohol to perfluooctanesulfonamidoacetate (FOSAA).

*The oxidation of N-EtFOSE to N-ethylperfluorooctanesulfonamidoacetate (N-EtFOSAA) was observed in liver slices but not in cytosolic fractions. FOSA was biotransformed in liver slices to PFOS, albeit at a low rate. These results show that the major pathway for the biotransformation of N-EtFOSE is N-dealkylation to give FOSA. The biotransformation of FOSA to PFOS explains the observation that PFOS is found in animals given N-EtFOSE.*

## Bio-concentration or bioaccumulation factor, based on measured values (unless monitoring data are judged to meet this need)

[27] Sachi Taniyasu, Kurunthachalam Kannan, Yuichi Horii, Nobuyasu Hanari, and Nobuyoshi Yamashita., **A Survey of Perfluorooctane Sulfonate and Related Perfluorinated Organic Compounds in Water, Fish, Birds, and Humans from Japan.** *Environ. Sci. Technol.,* **37** (12), 2634 -2639

In this study, concentrations and distribution of PFOS, perfluorohexane sulfonate (PFHS), and perfluorobutane sulfonate (PFBS) were determined in samples of surface water, fish and bird blood and livers, and human blood collected in Japan. Notable concentrations of PFOS were found in surface water and fish from Tokyo Bay. PFOS was found in all of the 78 samples of fish blood and liver analyzed. Concentrations of PFOS in the blood of Japanese human volunteers ranged from 2.4 to14 ng/mL. PFHS was detected in 33% of the fishes analyzed, at concentrations several fold less than those of PFOS.

*Based on the concentrations of PFOS in water and in fish livers, bioconcentration factors were calculated to range from 274 to 41 600.*

[28] Morikawa A, Kamei N, Harada K, Inoue K, Yoshinaga T, Saito N, Koizumi A., **The bioconcentration factor of perfluorooctane sulfonate is significantly larger than that of perfluorooctanoate in wild turtles (Trachemys scripta elegans and Chinemys reevesii): An Ai river ecological study in Japan.** *Ecotoxicol Environ Saf.* 2005 Jul 22; [Epub ahead of print]

Turtles, high in the river food chain, were suitable for predicting the bioconcentrations of chemicals through the food chain. Trachemys scripta elegans (N=46) and Chinemys reevesii (N=51) were captured in a river in Japan, from September to October 2003 and April to June 2004. Surface water samples were collected simultaneously from the same sites at which the turtles were caught. Serum perfluorooctane sulfonate (PFOS) ranged from 2.4 to 486mug/L, while water PFOS levels ranged from 2.9 to 37ng/L.

*The geometric mean (GM) (geometric standard deviation, GSD) of the bioconcentration factor (BCF) of PFOS was 10,964 (2.5). PFOS could be preferentially bioconcentrated in biota.*

## (d) Monitoring data

**HUMANS**

[29] Health Canada Screening Assessment Report, March 5, 2004, **Perfluorooctane Sulfonate, Its Salts and Its Precursors that Contain the C8F17SO2 or C8F17SO3 Moiety.**

*In one study of 599 children (aged 2–12 years) in the United States conducted between 1994 and 1995, the geometric mean concentration of PFOS in the serum was 37.5 ppb (i.e., 0.0375 µg/ml); the 95th percentile was 97 ppb (0.097 µg/ml), with a small number of samples (<20) exceeding this value. Individual values ranging widely from 7 to 515 ppb (i.e., 0.0067– 0.515 µg/ml)*

[30] Leo W. Y. Yeung, M. K. So, Guibin Jiang, Taniyasu, N. Yamashita, Maoyong Song, Yongning Wu, Jingguang Li, J. P. Giesy, K. S. Guruge, and Paul K. S. Lam, **Perfluorooctanesulfonate and Related Fluorochemicals in Human Blood Samples from China.** *Environ. Sci. Technol.,* Web Release Date**:** December 24, 2005

In this study, concentrations of perfluorohexanesulfonate (PFHxS), perfluorobutanesulfonate (PFBS), perfluorooctanesulfonate (PFOS), perfluorohexanoic acid (PFHxA), perfluorooctanoate (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnDA), perfluorododecanoic acid (PFDoDA), and PFOSA were measured in 85 samples of whole human blood collected from nine cities (eight provinces) in China.

*Among the 10 perfluorinated compounds (PFCs) measured, PFOS was the predominant compound. The mean concentration of PFOS was greatest in samples collected from Shenyang (79.2 ng/mL) and least in samples from Jintan (3.72 ng/mL). PFHxS was the next most abundant perfluorochemical in the samples. No age-related differences in the concentrations of PFOA, PFOS, PFOSA, and PFHxS were observed. Gender-related differences were found, with males higher for PFOS and PFHxS, and females higher in PFUnDA. Concentrations of PFHxS were positively correlated with those of PFOS, while concentrations of PFNA, PFDA, and PFUnDA were positively correlated with those of PFOA. There were differences in the concentration profiles (percentage composition) of various PFCs in the samples among the nine cities*.

[31] Calafat AM, Needham LL, Kuklenyik Z, Reidy JA, Tully JS, Aguilar-Villalobos M, Naeher LP., **Perfluorinated chemicals in selected residents of the American continent.** *Chemosphere.* 2005 Oct 4;

This study measured 11 perfluorinated chemicals (PFCs) in 23 pooled serum samples collected in the United States from 1990 through 2002, and in serum samples collected in 2003 from 44 residents from Trujillo, Peru. PFOS and PFOA were detected in all the pooled samples; perfluorohexane sulfonic acid (PFHxS) was detected in 21. Median concentrations were 31.1 micrograms per liter (mug/l, PFOS), 11.6mug/l (PFOA), and 2mug/l (PFHxS). The 90th percentile concentrations of PFCs in the 44 Peruvian residents were 0.7mug/l (PFOS), 0.1mug/l (PFOA), and <0.3mug/l (PFHxS). The frequencies of detection were 20% (PFOS), 25% (PFOA), and 9% (PFHxS).

*The frequent detection of selected PFCs in the pooled samples from the United States and the lack of clear concentration trends based on a year of collection suggest a sustained widespread exposure to these compounds among US residents, at least since the 1990s. By contrast, the much lower frequency of detection and concentration ranges of PFCs in Peru suggest a lower exposure of Peruvians to PFCs compared with North Americans. Genetic variability, diet, lifestyle, or a combination of all these may contribute to the different patterns of human exposure to PFCs in the United States and Peru.*

[32] Harada K, Inoue K, Morikawa A, Yoshinaga T, Saito N, Koizumi A.., **Renal clearance of perfluorooctane sulfonate and perfluorooctanoate in humans and their species-specific excretion.** *Environ Res.* 2005 Oct;99(2):253-261

In this study, concentrations of PFOA and PFOS were measured in subjects who had lived in Kyoto city for more than 10 years. The serum concentrations of PFOA and PFOS were higher in females who menstruated than those who did not ($P<0.01$), but in males this did not change by age; the levels in females reached those in males at an age of 60 years. The study then determined the renal clearances of PFOA and PFOS in young (20-40 years old, N=5 for each sex) and old (60 years old, N=5 for each sex) subjects of both sexes. All young females were menstruating, while all old females were not.

*The renal clearances were 10(-5)-fold smaller than the glomerular filtration rate in humans, suggesting the absence of active excretion in human kidneys. The renal clearances of PFOA and PFOS were approximately one-fifth of the total clearance based*

*on their serum half-lives, assuming a one-compartment model. The sex differences in renal clearance that have been reported in rats and Japanese macaques were not found in our human subjects.*

[33] Geary W. Olsen, Han-Yao Huang, Kathy J. Helzlsouer, Kristen J. Hansen, John L. Butenhoff and Jeffrey H. Mandel., **Historical Comparison of Perfluorooctanesulfonate, Perfluorooctanoate, and Other Fluorochemicals in Human Blood.** ***Environ Health Perspect*** **113:539–545 (2005)**

This investigation was to determine the change in the human blood concentration of perfluorooctanesulfonate (PFOS), perfluorooctanoate (PFOA), and five other fluorochemicals since 1974. Blood samples were collected in 1974 (serum) and 1989 (plasma) from volunteer participants of a large community health study. The 365 samples (178 from each time period) were analyzed by high-pressure liquid chromatography/ tandem mass spectrometry methods.

The median 1974 and 1989 fluorochemical concentrations, respectively, were PFOS, 29.5 ng/mL vs. 34.7 ng/mL; PFOA, 2.3 ng/mL vs. 5.6 ng/mL; perfluorohexanesulfonate (PFHS), 1.6 ng/mL vs. 2.4 ng/mL; and *N*-ethyl perfluorooctanesulfonamidoacetate (PFOSAA), less than the lower limit of quantitation (LLOQ; 1.6 ng/mL, vs. 3.4 ng/mL). For *N*-methyl perfluorooctanesulfonamidoacetate (M570), perfluorooctanesulfonamide, and perfluorooctanesulfonamidoacetate, median serum concentrations in both years were less than the LLOQ values (1.0, 1.0, and 2.5 ng/mL, respectively).

*Statistical analysis of 58 paired samples indicated that serum concentrations of PFOS, PFOSAA, PFOA, PFHS, and M570 were significantly ($p < 0.001$) higher in 1989 than in 1974. However, a comparison with other regional American Red Cross data collected in 2001 did not suggest a continued increase in concentrations since 1989.*

[34] Koichi Inoue, Fumio Okada, Rie Ito, Shizue Kato, Seiko Sasaki, Sonomi Nakajima, Akiko Uno, Yasuaki Saijo, Fumihiro Sata, Yoshihiro Yoshimura, Reiko Kishi, and Hiroyuki Nakazawa, **Perfluorooctane Sulfonate (PFOS) and Related Perfluorinated Compounds in Human Maternal and Cord Blood Samples: Assessment of PFOS Exposure in a Susceptible Population during Pregnancy.** ***Health Perspect*** 112:1204–1207 (2004)

In this study, concentrations of fluorinated organic compounds (FOCs), such as perfluorooctane sulfonate (PFOS), perfluorooctanoate (PFOA), and perfluorooctane sulfonylamide (PFOSA), were determined in maternal and cord blood samples. Pregnant women 17–37 years of age were enrolled as subjects. FOCs in 15 pairs of maternal and cord blood samples were analyzed by liquid chromatography–electrospray mass spectrometry coupled with online extraction. The limits of quantification of PFOS, PFOA, and PFOSA in human plasma or serum were 0.5, 0.5, and 1.0 ng/mL, respectively. The method enables the precise determination of FOCs and can be applied to the detection of FOCs in human blood samples for monitoring human exposure.

*PFOS concentrations in maternal samples ranged from 4.9 to 17.6 ng/mL, whereas those in fetal samples ranged from 1.6 to 5.3 ng/mL. The results revealed a high correlation between PFOS concentrations in maternal and cord blood (r2 = 0.876). The study revealed that human fetuses in Japan may be exposed to relatively high levels of FOCs.*

[35] Cariton Kubwabo, Natalia Vais and Frank M. Benoit, **A pilot study on the determination of perfluorooctanesulfonate and other perfluorinated compounds in blood of Canadians.** ***Journal of Environmental Monitoring***, 2004, **6**(6), 540 - 545

In this study, 56 human serum samples were collected from non-occupationally exposed Canadians and analyzed by microbore HPLC-negative ion electrospray tandem mass spectrometry.

*PFOS was the main component of perfluorinated organic compounds (PFCs) and was detected in all 56 blood specimens at an average concentration of 28.8 ng $mL^{-1}$ and a range from 3.7 to 65.1 ng $mL^{-1}$.*

The concentration of PFOA was an order of magnitude lower than that of PFOS and was found only in 16 samples (29%) at concentrations above the limit of quantification (LOQ).Two distinct PFOS isomers in human serum were identified by accurate mass determination.

[36] Geary W. Olsen, Timothy R. Church, John P. Miller, Jean M. Burris, Kristen J. Hansen4 James K. Lundberg, John B. Armitage, Ross M. Herron, Zahra Medhdizadehkashi, John B. Nobiletti, E. Mary O'Neill, Jeffrey H. Mandel, and Larry R. Zobel , **Perfluorooctanesulfonate and Other Fluorochemicals in the Serum of American Red Cross Adult Blood Donors.** *Environ Health Perspect* 111:1892–1901 (2003).

In this investigation, 645 adult donor serum samples from six American Red Cross blood collection centers were analyzed for PFOS and six other fluorochemicals using HPLC-electrospray tandem mass spectrometry.

*PFOS concentrations ranged from the lower limit of quantitation of 4.1 ppb to 1656.0 ppb with a geometric mean of 34.9 ppb [95% confidence interval (CI), 33.3–36.5]. The geometric mean was higher among males (37.8 ppb; 95% CI, 35.5–40.3) than among females (31.3 ppb; 95% CI, 30.0–34.3). No substantial difference was observed with age.*

**FOODCHAIN**

[37] Jonathan W. Martin, D. Michael Whittle, Derek C. G. Muir, and Scott A. Mabury, **Perfluoroalkyl Contaminants in a Food Web from Lake Ontario,** *Environ. Sci. Technol.,* **38** (20), 5379 -5385, 2004

This study analyzed for PFOS, the homologous series of PFCAs ranging from 8 to 15 carbons in chain length, and the PFOS-precursor heptadecafluorooctane sulfonamide (FOSA) in various organisms from a food web of Lake Ontario. The sampled organisms

included a top predator fish, lake trout (*Salvelinus namaycush*), three forage fish species including rainbow smelt (*Osmerus mordax*), slimy sculpin (*Cottus cognatus*), and alewife (*Alosa pseudoharengus*), and two invertebrates *Diporeia* (*Diporeia hoyi*) and *Mysis* (*Mysis relicta*).

*PFOS was the dominant acid in all samples, but long-chain PFCAs, ranging in length from 8 to 15 carbons, were also detected in most samples between <0.5 and 90 ng/g. The highest mean concentration for each fluorinated contaminant was detected in the benthic macroinvertebrate Diporeia, which occupies the lowest trophic level of all organisms analyzed. Perfluorinated acid concentrations in Diporeia were often 10-fold higher than in Mysis, a predominantly pelagic feeder, suggesting that a major source of perfluoroalkyl contaminants to this food web was the sediment, not the water.*

Among *Mysis* and the more pelagic fish species (e.g. excluding *Diporeia* and sculpin) there was evidence for biomagnification, but the influence of foraging on highly contaminated *Diporeia* and sculpin by these fish may have overestimated trophic magnification factors (TMFs), which ranged from 0.51 for FOSA to 5.88 for PFOS. By accounting for the known diet composition of lake trout, it was shown that bioaccumulation was indeed occurring at the top of the food web for all perfluoroalkyl compounds except PFOA.

*Archived lake trout samples collected between 1980 and 2001 showed that mean whole body PFOS concentrations increased from 43 to 180 ng/g over this period, but not linearly, and may have been indirectly influenced by the invasion and proliferation of zebra mussels (Dreissena polymorpha) through effects on the population and ecology of forage fishes.*

**BIRDS**

[38] Toschik PC, Rattner BA, McGowan PC, Christman MC, Carter DB, Hale RC, Matson CW, Ottinger MA., **Effects of contaminant exposure on reproductive success of ospreys (Pandion haliaetus) nesting in Delaware River and Bay, USA.** *Environ Toxicol Chem*. 2005 Mar;24(3):617-28.

In 2002, this study conducted a comprehensive evaluation of contaminant exposure and reproduction of ospreys (Pandion haliaetus) breeding in Delaware River and Bay. Sample eggs were collected from 39 nests and analyzed for organochlorine pesticides, polychlorinated biphenyls (PCBs), and mercury; a subset of 15 eggs was analyzed for perfluorinated compounds and polybrominated diphenyl ethers (PBDEs).

*Several perfluorinated compounds and PBDEs were detected in eggs at concentrations approaching 1 microg/g wet weight.*

[39] Holmstrom KE, Jarnberg U, Bignert A., **Temporal trends of PFOS and PFOA in guillemot eggs from the Baltic Sea, 1968--2003.** *Environ Sci Technol.* 2005 Jan 1;39(1):80-4

Temporal trends in the concentrations of PFOS and PFOA in the Baltic Sea marine environment were measured using archived guillemot eggs. Samples collected from Stora Karlso (Sweden) between 1968 and 2003 were received from an environmental specimen bank and concentrations of PFOS and PFOA were analyzed using HPLC coupled to ESI-MS/MS. PFOA was not detected in any of the samples (LOD 3 ng/g), but there was an almost 30-fold increase in PFOS concentrations in the guillemot eggs during the time period, from 25 ng/g in 1968 to 614 ng/g in 2003 (wet weight). Regression analysis indicated a significant trend, increasing on average between 7 and 11% per year.

*A sharp peak in PFOS concentrations was observed in 1997 followed by decreasing levels up to 2002, but this cannot be linked to the PFOS phase-out, which occurred at the end of this period*.

**MARINE MAMMALS**

[40] Van de Vijver KI, Hoff P, Das K, Brasseur S, Van Dongen W, Esmans E, Reijnders P, Blust R, De Coen W., **Tissue distribution of perfluorinated chemicals in harbor seals (Phoca vitulina) from the Dutch Wadden Sea.** *Environ Sci Technol.* 2005 Sep 15;39(18):6978-84.

This study reports for the first time on levels of longer chain PFCAs, together with some short chain PFAs, perfluorobutane sulfonate (PFBS) and perfluorobutanoate (PFBA), in liver, kidney, blubber, muscle, and spleen tissues of harbor seals (Phoca vitulina) from the Dutch Wadden Sea.

*PFOS was the predominant compound in all seal samples measured (ranging from 89 to 2724 ng/g wet weight); however, there were large variations between tissues that were monitored and large differences in tissue distribution and accumulation patterns of perfluorinated compounds in marine organisms*.

Although these are preliminary results, it is the first time that PFBS could be found at detectable concentrations (2.3 +/- 0.7 ng/g w wt) in environmental samples. PFBS was only detected in spleen tissue. PFCA levels were much lower than PFOS concentrations.

[41] Houde M, Wells RS, Fair PA, Bossart GD, Hohn AA, Rowles TK, Sweeney JC, Solomon KR, Muir DC., **Polyfluoroalkyl compounds in free-ranging bottlenose dolphins (Tursiops truncatus) from the Gulf of Mexico and the Atlantic Ocean.** *Environ Sci Technol*. 2005 Sep 1;39(17):6591-8.

Concentrations of two major PFA groups, carboxylic acids (PFCAs) and sulfonic acids (PFSAs), were assessed in plasma of bottlenose dolphins from the Gulf of Mexico (Sarasota Bay, FL) and the Atlantic Ocean (Delaware Bay, NJ, Charleston, SC, Indian River Lagoon (IRL), FL, and Bermuda). Eight PFAs were detected in the plasma of all dolphins. An increase in PFA concentrations was associated with a decrease of blubber thickness in animals from Sarasota Bay and IRL. Fluorotelomer 8:2 and 10:2 unsaturated carboxylic acids (FTUCAs), known degradation products of fluorotelomer alcohols and

suspected precursors to PFCAs, were detected for the first time at low concentrations in plasma of dolphins

*Perfluorooctane sulfonate (PFOS) was the predominant compound at all locations and ranged from 49 ng/g wet weight (w.w.) in dolphins from Bermuda, to 1171 ng/g w.w. in plasma of animals from Charleston.* .

[42] Jennifer M. Keller, Kurunthachalam Kannan, Sachi Taniyasu, Nobuyoshi Yamashita, Rusty D. Day, Michael D. Arendt, Al L. Segars and John R. Kucklick, **Perfluorinated Compounds in the Plasma of Loggerhead and Kemp's Ridley Sea Turtles from the Southeastern Coast of the United States**. *Environ. Sci. Technol.,* 39 (23), 9101 -9108, 2005

This study examined 12 perfluorinated compounds (PFCs) in the plasma of 73 loggerhead sea turtles (*Caretta caretta*) and 6 Kemp's ridley sea turtles (*Lepidochelys kempii*) captured from inshore waters of Core Sound, North Carolina (NC), and offshore waters of South Carolina, Georgia, and Florida (SC-FL).

*Perfluorooctanesulfonate (PFOS) and perfluorooctanoic acid (PFOA) were the dominant compounds, with respective mean concentrations of 11.0 ng/mL and 3.20 ng/mL for loggerhead turtles and 39.4 ng/mL and 3.57 ng/mL for Kemp's ridley turtles.*

Mean PFOS concentrations were 2- to 12-fold higher than typical mean ΣPCB concentrations (~5 ng/g wet mass) measured previously in sea turtle blood. In loggerhead turtles, ΣPFC concentrations were not influenced by sex ($p > 0.05$), but were higher in turtles captured from inshore waters of NC than in turtles from offshore waters of SC-FL ($p = 0.009$). A backward stepwise multiple regression model showed that ΣPFC concentrations were (1) significantly higher in Kemp's ridley turtles than loggerhead turtles ($p < 0.0001$), (2) higher in larger turtles ($p = 0.018$; carapace length used as a proxy for age), and (3) higher in turtles captured toward the north ($p = 0.006$).

*These findings suggest that bioaccumulation of PFCs in sea turtles is influenced by species, age, and habitat.*

**FISH**

[43] Hoff PT, Van Campenhout K, Van de Vijver K, Covaci A, Bervoets L, Moens L, Huyskens G, Goemans G, Belpaire C, Blust R, De Coen W., **Perfluorooctane sulfonic acid and organohalogen pollutants in liver of three freshwater fish species in Flanders (Belgium): relationships with biochemical and organismal effects.** *Environ Pollut.* 2005 Sep;137(2):324-33.

Gibel carp (Carassius auratus gibelio), carp (Cyprinus carpio), and eel (Anguilla anguilla) in Flanders (Belgium) were sampled for perfluorooctane sulfonic acid (PFOS). Eel from the Oude Maas pond (Dilsen-Stokkem) and Watersportbaan basin (Ghent) had PFOS concentrations ranging between 212 and 857 ng/g wet weight.

*The liver PFOS concentrations in fish from the Ieperlee canal (Boezinge, 250-9031 ng/g wet weight, respectively) and the Blokkersdijk pond (Antwerp, 633-1822 ng/g wet weight) were higher than at the Zuun basin (Sint-Pieters-Leeuw, 11.2-162 ng/g wet weight) and among the highest in feral fish worldwide.*

**SHELLFISH**

[44] So MK, Taniyasu S, Lam PK, Zheng GJ, Giesy JP, Yamashita N., **Alkaline Digestion and Solid Phase Extraction Method for Perfluorinated Compounds in Mussels and Oysters from South China and Japan.** *Arch Environ Contam Toxicol*. 2005 Sep 16; [Epub ahead of print]

Perfluorinated compounds (PFCs), such as perfluorooctane sulfonate (PFOS) have been identified in the coastal waters of China and Japan. An alkaline digestion method, coupled with solid-phase extraction (SPE), and high-performance liquid chromatography interfaced with high-resolution electrospray tandem mass spectrometry was developed to determine PFCs in mussel and oyster samples from coastal waters of south China and Japan.

*Concentrations of individual PFCs in mussels and oysters from south China and Japan ranged from 113.6 to 586.0 pg/g, wet weight (ww) for PFOS, 63.1 to 511.6 pg/g, ww for perfluorohexane sulfonate, 9.3 to 30.1 pg/g, ww for perfluorobutane sulfonate and 37.8 to 2957.0 pg/g, ww for perfluorooctane sulfonamide*.

[45] Jesus Olivero-Verbel, Lin Tao, Boris Johnson-Restrepo, Jorge Guette-Fernández, Rosa Baldiris-Avila, Indira O'byrne-Hoyos and Kurunthachalam Kannan., **Perfluorooctanesulfonate and related fluorochemicals in biological samples from the north coast of Colombia.** *Environmental Pollution*, Article in Press.

In this study, the distribution of perfluorooctanesulfonate (PFOS), perfluorooctanoic acid (PFOA), perfluorohexanesulfonate (PFHxS), and perfluorooctanesulfonamide (PFOSA) was determined in the bile of mullet, Mugil incilis, and in tissues of pelicans (Pelecanus occidentalis) collected from North Colombia. Analysis was performed by HPLC mass spectrometry after ion-pair extraction. PFOS was found in all bile samples and PFOA and PFHxS were detected at lower frequency.

*Average concentrations of PFOS, PFOA, and PFHxS in bile of fish from Cartagena Bay, an industrialized site, and Totumo marsh, a reference site, were 3673, 370, 489 and 713, 47.4, 1.27 ng/mL, respectively. PFOS concentrations in pelican organs decreased in the order of spleen > liver > lung > kidney > brain > heart > muscle. These results show for the first time that perfluorinated compounds are also found in wildlife from Latin American countries.*

## (e) Exposure in local areas **(provide summary information and relevant references)**

## - general

## - as a result of long-range environmental transport

### HUMANS

[46] Guruge KS, Taniyasu S, Yamashita N, Wijeratna S, Mohotti KM, Seneviratne HR, Kannan K, Yamanaka N, Miyazaki S., **Perfluorinated organic compounds in human blood serum and seminal plasma: a study of urban and rural tea worker populations in Sri Lanka.** *J Environ Monit*. 2005 Apr;7(4):371-7

The concentrations and accumulation of 13 fluorinated organic compounds (FOCs) in human sera and seminal plasma were measured in an Asian developing country, Sri Lanka. Six of the FOCs, PFOS (perfluorooctanesulfonate), PFHS (perfluorohexanesulfonate), PFUnA (perfluoroundecanoic acid), PFDA (perfluorodecanoic acid), PFNA (perfluorononanoic acid) and PFOA (perfluorooctanoic acid), were detected in all of the sera samples.

*Measurable quantities of two main perfluorosulfonates, PFOS and PFHS, were found in all seminal plasma samples. Accumulation of PFOS in sera was significantly positively correlated with PFOA, PFHS and PFNA. Positive linear regressions were also found between PFNA and PFUnA and PFNA and PFDA suggesting that these compounds may have a similar origin of exposure and accumulation.*

*Concentrations of FOCs in Sri Lanka were similar to those reported for industrialized countries suggesting that human exposure to such chemicals is widespread even in developing countries. The novel finding of FOCs in human seminal plasma implies that further studies are needed to determine whether long-term exposure in humans can result in reproductive impairments.*

### MAMMALS

[47] Kurunthachalam Kannan, Se Hun Yun and Thomas J. Evans, **Chlorinated, Brominated, and Perfluorinated Contaminants in Livers of Polar Bears from Alaska**. *Environ. Sci. Technol.,* 39 (23), 9057 -9063, 2005.

In this study, differences in concentrations and profiles of organochlorine pesticides, polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), and perfluorinated acids were examined in livers of polar bears from the two subpopulations (the Beaufort Sea and the Chukchi Sea) in Alaska. Concentrations of chlordanes, PCBs, and perfluorooctanesulfonate (PFOS) were significantly different between the two subpopulations with Chlordane was the predominant contaminant in the Beaufort Sea population, and PFOS was the major contaminant in the Chukchi Sea population.

*The concentrations and profiles of organohalogens analyzed in the two subpopulations of polar bears suggest differences in the sources of exposures between the two regions of Alaska.*

[48] Smithwick M, Mabury SA, Solomon KR, Sonne C, Martin JW, Born EW, Dietz R, Derocher AE, Letcher RJ, Evans TJ, Gabrielsen GW, Nagy J, Stirling I, Taylor MK, Muir DC., **Circumpolar study of perfluoroalkyl contaminants in polar bears (Ursus maritimus).** *Environ Sci Technol.* 2005 Aug 1;39(15):5517-23.

Perfluoroalkyl substances were determined in liver tissues and blood of polar bears (Ursus maritimus) from five locations in the North American Arctic and two locations in the European Arctic. Concentrations of perfluorooctane sulfonate (PFOS), perfluorohexane sulfonate, heptadecafluorooctane sulfonamide, and perfluoroalkyl carboxylates with C(8)-C(15) perfluorinated carbon chains were determined using liquid chromatography tandem mass spectrometry.

*PFOS concentrations were significantly correlated with age at four of seven sampling locations, while gender was not correlated to concentration for any compound measured. Populations in South Hudson Bay (2000-2730 ng/g wet wt), East Greenland (911-2140 ng/g wet wt), and Svalbard (756-1290 ng/g wet wt) had significantly (P < 0.05) higher PFOS concentrations than western populations such as the Chukchi Sea (435-729 ng/g wet wt). Concentrations of PFOS in liver tissue at five locations were correlated with concentrations of four polychlorinated biphenyl congeners (180, 153, 138, and 99) in adipose tissue of bears in the same populations, suggesting similar transport pathways and source regions of PFOS or precursors.*

[49] Smithwick M, Muir DC, Mabury SA, Solomon KR, Martin JW, Sonne C, Born EW, Letcher RJ, Dietz R., **Perflouroalkyl contaminants in liver tissue from East Greenland polar bears (Ursus maritimus).** *Environ Toxicol Chem*. 2005 Apr; 24(4):981-6.

Perfluoroalkyl substances were determined in polar bears (Ursus maritimus) collected in East Greenland (69 degrees 00'N to 74 degrees 00"N) to compare with other populations and to examine effects of age and gender on concentrations of these contaminants. Hepatic tissue (n = 29) was analyzed for perfluorooctane sulfonate (PFOS), perfluorooctanoate (PFOA), perfluorohexane sulfonate, heptadecafluorooctane sulfonamide (PFOSA), and perfluoroalkyl carboxylates (PFCAs) with C9-C15 perfluorinated carbon chains by liquid chromatography tandem mass spectrometry.

*Concentrations of PFOS found in samples from East Greenland (mean = 2,470+/-1,320 ng/g wet weight) were similar to Hudson Bay, Canada, and both populations had significantly greater concentrations than those reported for Alaska, suggesting a spatial trend.*

Male bears showed a significant increase in concentration up to age six for PFCAs with C10-C14 carbon chains (r2 > or = 0.50, p < or = 0.05). Significant correlations were found between adjacent chain length PFCAs, (e.g., PFNA to PFDA: p < 0.05; r2 = 0.90). This may indicate a common source for these chemicals, although the specifics of source and mode of transport are unknown.

**MARINE MAMMALS**

[50] Bossi R, Riget FF, Dietz R., **Temporal and spatial trends of perfluorinated compounds in ringed seal (Phoca hispida) from Greenland.** *Environ Sci Technol*. 2005 Oct 1;39(19):7416-22.

Spatial and temporal trends in the concentrations of selected PFCs were measured using archived liver samples of ringed seal (Phoca hispida) from East and West Greenland. The samples were collected in four different years at each location, between 1986 and 2003 in East Greenland and between 1982 and 2003 in West Greenland.

*PFOS was the major contributor to the burden of PFCs in samples, followed by perfluoroundecanoic acid (PFUnA).*

Perfluorononanoic acid (PFNA) and perfluorodecanoic acid (PFDA) were also detected in most samples. Perfluorohexane sulfonate (PFHxS) and perfluorooctane sulfonamide (PFOSA) were only found sporadically. Perfluorooctanoic acid was not found in detectable concentrations in any sample. Regression analysis of logarithmic transformed PFOS, PFDA, and PFUnA median concentrations indicated a significant temporal trend with increasing concentrations at both locations.

*A spatial trend in PFOS concentrations (ANOVA, $p < 0.0001$) was observed between the two sampling locations, with significantly higher concentrations in seals from East Greenland.*

**BIRDS**

[51] Verreault J, Houde M, Gabrielsen GW, Berger U, Haukas M, Letcher RJ, Muir DC., **Perfluorinated alkyl substances in plasma, liver, brain, and eggs of glaucous gulls (Larus hyperboreus) from the Norwegian arctic.** *Environ Sci Technol.* 2005 Oct 1;39(19):7439-45.

This study investigated the distribution of a suite of PFAS in plasma, liver, brain, and egg samples from adult glaucous gulls (Larus hyperboreus), an apex scavenger-predator seabird breeding in the Norwegian Arctic. Among the body compartment/ tissue samples analyzed, PFOS was highest in plasma (48.1-349 ng/g wet weight (ww)), followed by liver approximately equal to egg > brain.

*Perfluorooctane sulfonate (PFOS) was the predominant PFAS in all samples and was present at concentrations that are the highest reported thus far in any arctic seabird species and populations.*

**FOODCHAIN**

[52] Rossana Bossia, Frank F. Riget, Rune Dietz, Christian Sonne, Patrik Fauser, Maria Dam and Katrin Vorkamp., **Preliminary screening of perfluorooctane sulfonate (PFOS) and**

**other fluorochemicals in fish, birds and marine mammals from Greenland and the Faroe Islands.** *Environmental Pollution* Volume 136, Issue 2 , July 2005, 323-329

In this study a preliminary screening of PFOS and related compounds was performed in liver samples of fish, birds and marine mammals from Greenland and the Faroe Islands. PFOS was the predominant fluorochemical in the biota analyzed, followed by perfluorooctane sulfonamide (PFOSA). PFOS was found at concentrations above LOQ (10 ng/g wet weight) in 13 out of 16 samples from Greenland and in all samples from the Faroe Islands.

*The results from Greenland showed a biomagnification of PFOS along the marine food chain (shorthorn sculpin<ringed seal<polar bear). The greatest concentration of PFOS was found in liver of polar bear from east Greenland (mean: 1285 ng/g wet weight, n=2). The geographical distribution of perfluorinated compounds in Greenland was similar to that of persistent organohalogenated compounds (OHCs), with the highest concentrations in east Greenland, indicating a similar geographical distribution to that of OHCs, with higher concentrations in east Greenland than in west Greenland. Perfluorinated acids were detected in livers of fish, birds and marine mammals from Greenland and the Faroe Islands.*

[53] Gregg T. Tomy, Wes Budakowski, Thor Halldorson, Paul A. Helm, Gary A. Stern, Ken Friesen, Karen Pepper, Sheryl A. Tittlemier and Aaron T. Fisk, **Fluorinated Organic Compounds in an Eastern Arctic Marine Food Web**, *Environ. Sci. Technol.,* 38 (24), 6475 -6481, 2004.

In this study, an eastern Arctic marine food web was analyzed for perfluorooctanesulfonate (PFOS, $C_8F_{17}SO_3^-$), perfluorooctanoate (PFOA, $C_7F_{15}COO^-$), perfluorooctane sulfonamide (PFOSA, $C_8F_{17}SO_2NH_2$), and *N*-ethylperfluorooctane sulfonamide (*N*-EtPFOSA, $C_8F_{17}SO_2NHCH_2CH_3$) to examine the extent of bioaccumulation.

*PFOS was detected in all species analyzed, and mean concentrations ranged from 0.28 ± 0.09 ng/g (arithmetic mean ± 1 standard error, wet wt, whole body) in clams (Mya truncata) to 20.2 ± 3.9 ng/g (wet wt, liver) in glaucous gulls (Larus hyperboreus).*

PFOA was detected in approximately 40% of the samples analyzed at concentrations generally smaller than those found for PFOS; the greatest concentrations were observed in zooplankton (2.6 ± 0.3 ng/g, wet wt). *N*-EtPFOSA was detected in all species except redfish with mean concentrations ranging from 0.39 ± 0.07 ng/g (wet wt) in mixed zooplankton to 92.8 ± 41.9 ng/g (wet wt) in Arctic cod (*Boreogadus saida*). This is the first report of *N*-EtPFOSA in Arctic biota. PFOSA was only detected in livers of beluga (*Delphinapterus leucas*) (20.9 ± 7.9 ng/g, wet wt) and narwhal (*Monodon monoceros*) (6.2 ± 2.3 ng/g, wet wt), suggesting that *N*-EtPFOSA and other PFOSA-type precursors are likely present but are being biotransformed to PFOSA.

A positive linear relationship was found between PFOS concentrations (wet wt) and trophic level (TL), based on $\delta^{15}N$ values, ($r^2 = 0.51$, $p < 0.0001$) resulting in a trophic magnification factor of 3.1. TL-corrected biomagnification factor estimates for PFOS ranged from 0.4 to 9.

*The results indicate that PFOS biomagnifies in the Arctic marine food web when liver concentrations of PFOS are used for seabirds and marine mammals. However, transformation of N-EtPFOSA and PFOSA and potential other perfluorinated compounds to PFOS may contribute to PFOS levels in marine mammals and may inflate estimated biomagnification values. The presence of perfluorinated compounds in seabirds and mammals provides evidence that trophic transfer is an important exposure route of these chemicals to Arctic biota.*

### - information regarding bio-availability

## (f) National and international risk evaluations, assessments or profiles and labelling information and hazard classifications, as available **(provide summary information and relevant references)**

## (g) Status of the chemical under international conventions

Please see UNEP/POPs/POPRC.1/INF/10 Status of chemicals under consideration in other international forums.