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

Introductory information	
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Chemical name (as used by the POPS Review Committee (POPRC))	**Perfluorooctane sulfonate and its salts** **Acid 1763-23-1** **Potassium 2795-39-3** **Lithium 29457-72-5** **Ammonium 29081-56-9** **Diethanolamine salt 70225-14-8**
Date of submission	**27.1.2006**

(a) Sources, including as appropriate (provide summary information and relevant references)	
(i) Production data: **Quantity** **Location**	**No production**
Other	
(ii) Uses	**Information on PFAS:** The substances are imported, either as chemical products or constituents in manufactured products. The largest quantities are used in fire extinguishers (Aqueous Film Forming Foams, AFFFs). AFFFs are used for fighting fires that involves petroleum products (oil, petrol etc.) and flammable water soluble liquids (acetone, alcohols etc.), and are used on offshore installations, airports, The Norwegian Defence, oil refineries and tankers. PFAS for textile protection are imported mainly as constituents in manufactured textile products. Protection agents that contain PFAS are mainly used for waterproof and breathing garments, for instance in garments with Gore-Tex membrane. The estimated amount is uncertain. Amounts used in other product types are considerably smaller. Yearly amounts of PFAS used in Norway in 2002: **Use Yearly amount used (2002) — Tons of PFAS** Fire extinguishers — 15 Textile protection — 7 – 10 Carpet protection — 0,4 Leather protection — < 0,1 Protection products for private use — 0,2 Floor polishes — 0,2 Detergents — < 0,1 Paintings etc. — < 0,1 Paper treatment — Not known Electronics — < 0,1 **Total — 23,2 – 26,2** The amounts are mainly based on estimates of contents in imported fabricated

	products, and are uncertain. This applies in particular to the estimate for textile protection, where information about both imported amounts of textiles and the type of protection agent is uncertain. The estimate for fire extinguishers is based on information directly from producer and importers, and may be regarded as reliable. Information has not been available for estimating amounts used for paper treatment. A survey on the uses of PFAS (incl. PFOS and PFOS-related compounds) in textiles and clothing in Norway was started in 2005. Results are expected in the beginning of 2006. <u>Information on PFOS</u> An investigation on uses in 2005 show that fire extinguishers are the main use of PFOS related compounds in Norway. Fire extinguishers was estimated to amount to 90 % of all uses of PFOS and PFOS-related compunds in Norway to day. A national survey on uses in fire extinguishers revealed that the estimated releases of PFOS since 1980 to 2003 has been at least 57 tonn. The estimates of historic emissions are uncertain. The estimates for offshore platforms and mobile rigs are most reliable. Historic emissions could not be estimated for airports, fire fighting training sites, fire and rescue brigades and The Norwegian Defence. Remaining quantities of foam with PFOS is estimated at approx. 1.4 million liters, as a minimum. The largest quantities are held on offshore oil platforms and at refineries and onshore gas terminals. The Norwegian Defence also has relatively large remaining quantities of foam with PFOS. The quantity of PFOS in the remaining foam is estimated at approx. 22 tons, as a minimum. Norway aims on phasing out the largest remaining quantities of PFOS-containing foam through dialog and voluntary agreements. Chromium-plating in the galvanic industry is another source of relevance. This use is related to health- and security provisions for workers. PFOS related compounds limit the extent of foam and the evaporation of 6-valued chrome from the process of chromium-plating. The releases are small since used PFOS is collected and treated as hazardous waste.
(iii) Releases: **Discharges** **Losses** **Emissions** **Other**	**Releases from industry, difuse sources and oil- and gas production are small and not yet estimated.** **Releases of PFAS from products (fire fighting extinguishers) and from the municipal sector in 2002 were 13 – 15 tonns and 5-7 tonns, respectively. The releases is assumed to bee somewhat reduced today, because of the focus on PFAS, and especially for PFOS. Sum releases in 2002 were 18 – 22 tonns.** **The yearly releases from use of fire fighting extinguishers have not yet been estimated.**

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

Studies on reproductive and developmental effects of PFOS

To understand the potential reproductive and developmental effects of PFOS, a two-generation reproduction study was conducted in rats (Luebker et al. 2005 I). 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. These effects were not observed in F1 dams at the highest dose tested, 0.4 mg/(kg day). 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> or =3.2 and F1> or =0.4 mg/(kg day); reproductive outcome: F0=1.6 and F1> or =0.4 mg/(kg day); overall parental effects: F0=0.1 and F1> or =0.4 mg/(kg day); offspring effects: F0=0.4 and F1> or =0.4 mg/(kg day). To distinguish between maternal and pup influences contributing to the perinatal mortality observed in the two-generation study, a follow-up cross-foster study was performed. 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.

Objectives of another study (Luebker at al. 2005 II) were to better define the dose-response curve for neonatal mortality in rat pups born to PFOS-exposed dams and to investigate biochemical and pharmacokinetic parameters potentially related to the etiology of effects observed in neonatal rat pups. 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. Biochemical parameters investigated in dams and litters included: (1) serum lipids, glucose, mevalonic acid, and thyroid hormones; (2) milk cholesterol; and (3) liver lipids. Pharmacokinetic parameters investigated included the interrelationship of administered oral dose of PFOS to maternal body burden of PFOS and the transfer of maternal body burden to the fetus in utero and pup during lactation, as these factors may affect neonatal toxicity. 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. Benchmark dose (BMD) estimates for decreased gestation length, birth weight, pup weight on lactation day 5, pup weight gain through lactation day 5, and viability resulted in values ranging from 0.27 to 0.89mg/kg/day for the lower 95% confidence limit of the BMD5 (BMDL5). Results of analyses for PFOS in biological matrices indicate a linear proportionality of mean serum PFOS concentration to maternal administered dose prior to mating and through the first two trimesters of gestation. However, at 21 days of gestation, mean serum PFOS concentrations were notably reduced from values measured earlier in gestation. Urinary and fecal elimination was low as expected from prior observations in adult rats. 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.

(c) Environmental fate (provide summary information and relevant references)	
Chemical/physical properties	Vapour pressure = 3,31 x 10-4 Pa Atmospheric half life > 2 days (estimated value based on photolytic half life > 3,7 years)
Persistence	Hydrolysis of perfluorooctane sulfonate at a range of temperatures and pH values with no observable degradation; the half-life of perfluorooctane sulfonate is determined to be over 41 years. No apparent biodegradation under aerobic and anaerobic conditions.
How are chemical/physical properties and persistence linked to environmental transport, transfer within and between environmental compartments, degradation and transformation to other chemicals?	In a study from Japan (Yamashita 2004) perfluorinated acids was detected in ocean water. PFOA was the major perfluorinated compound detected, followed by PFOS.
Bio-concentration or bio-accumulation factor, based on measured values (unless monitoring data are judged to meet this need)	Perfluorooctane sulfonate does *not* accumulate in fatty tissue, as is typical of many persistent organic pollutants. This is because perfluorooctane sulfonate is both hydrophobic and lipophobic. Rather, perfluorooctane sulfonate binds to proteins in the blood and the liver. The octanol-water partition coefficient (logKow) is not measurable for perfluorooctane sulfonate. The kinetic bioconcentration factor in bluegill sunfish (*Lepomis macrochirus*) for whole fish is determined to be 2,796. For rainbow trout (*Oncorhynchus mykiss*), bioconcentration factors in liver and plasma is estimated at 2,900 and 3,100 respectively.
(d) Monitoring data (provide summary information and relevant references)	

Levels in organisms in different trophic positions

In a study performed at Toronto University (Martin et al. 2004) it was 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). A striking finding was that the highest mean concentration for each fluorinated contaminantwas 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. 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. 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. Future monitoring at other locations in Lake Ontario, and in other aquatic environments, is necessary to determine if these food web dynamics are widespread. 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.

Trophic transfer of perfluorooctanesulfonate (PFOS) and other related perfluorinated compounds was examined in a Great Lakes benthic foodweb including water-algae-zebra mussel-round goby-smallmouth bass (Kannan et al. 2005). 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 collected from Michigan. 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. PFOS was the most widely detected compound in benthic organisms at various trophic levels. Concentrations of PFOS in benthic invertebrates such as amphipods and zebra mussels were approximately 1000-fold greater than those in surrounding water, which suggested a bioconcentration factor (BCF; concentration in biota/concentration in water) of 1000 in benthic invertebrates. Concentrations of PFOS in round gobies were two- to fourfold greater than those in their prey organisms such as zebra mussels and amphipods. Concentrations of PFOS in predatory fishes (Chinook salmon and lake whitefish) were 10 to 20-fold greater than those in their prey species. Concentrations of PFOS in mink and bald eagles were, on average, 5- to 10-fold greater than those in Chinook salmon, carp, or snapping turtles. Because of the accumulation of PFOS in liver and blood, the biomagnification factor (BMF) of perfluorinated compounds in higher trophic-level organisms such as salmonid fishes, mink, and eagles were based on the concentrations in livers or plasma. Overall, these results suggest a BCF of PFOS of approximately 1000 (whole-body based) in benthic invertebrates, and a BMF of 10 to 20 in mink or bald eagles, relative to their prey items. Eggs of fish contained notable concentrations of PFOS, suggesting oviparous transfer of this compound. PFOA was found in water, but its biomagnification potential was lower than that of PFOS.

In a preliminary screening of PFOS and related compounds 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) (Bossi et al. 2005). 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.

To provide a preliminary assessment of fluorinated contaminants, including PFCAs, in the Canadian Arctic, polar bears, ringed seals, arctic fox, mink, common loons, northern fulmars, black guillemots, and fish were collected at various locations in the circumpolar region (Martin et al. 2004). PFOS was the major contaminant detected in most samples and in polar bear liver was the most prominent organohalogen (mean PFOS = 3.1 microg/g wet weight) compared to individual polychlorinated biphenyl congeners, chlordane, or hexachlorocyclohexane-related chemicals in fat. Using two independent mass spectral techniques, it was confirmed that all samples also contained ng/g concentrations of a homologous series of PFCAs, ranging in length from 9 to 15 carbons. Sum concentrations of PFCAs (sum(PFCAs)) were lower than total PFOS equivalents (sum(PFOS)) in all samples except for mink. In mink, perfluorononanoate (PFNA) concentrations exceeded PFOS concentrations, indicating that PFNA and other PFCAs should be considered in future risk assessments. Mammals feeding at higher trophic levels had greater concentrations of PFOS and PFCAs than mammals feeding at lower trophic positions. In general, odd-length PFCAs exceeded the concentration of even-length PFCAs, and concentrations decreased with increasing chain length in mammals. PFOS and PFCA concentrations were much lower for animals living in the Canadian Arctic than for the same species living in mid-latitude regions of the United States. Future studies should continue to monitor all fluorinated contaminants and examine the absolute and relative toxicities for this novel suite of PFCAs.

An eastern marine Arctic food web was analyzed for PFOS and other related perfluorinated compounds to examine the extent of bioaccumulation (Tomy et al. 2004). 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, liver) in glacous gulls (Larus hyperboreus). 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. Both 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.

Global distribution

The global distribution of PFOS has been investigated by researchers at the Michigan University in US (Giesy and Kannan 2001). PFOS was measured in the tissues of wildlife, including, fish, birds , and marine mammals . Some of the species studied include bald eagles, polar bears, albatrosses, and various species of seals. Samples were collected from urbanized areas in North America, especially the Great Lakes region and coastal marine areas and rivers, and Europe. Samples was also collected from a number of more remote, less urbanized locations such the Arctic and the North Pacific Oceans. The results demonstrated that PFOS is widespread in the environment. Concentrations of PFOS in animals from relatively more populated and industrialized regions, such as the North American Great Lakes, the Baltic Sean, and the Mediterranean Sea, were greater than those in animals from remote marine locations. Fish-eating, predatory animals such as mink and bald eagles contained concentrations of PFOS that were greater than the concentrations in their diets. This suggests that PFOS can bioaccumulate to higher trophic levels of the food chain. Currently available data indicates that the concentrations of PFOS in wildlife are less than those required to cause adverse effects in laboratory animals.

In another study (Kannan et al. 2001), 247 tissue samples from 15 species of marine mammals collected from Florida, California, and Alaskan coastal waters; the northern Baltic Sea; the Arctic (Spitsbergen); and Sable Island in Canada were analysed for PFOS. PFOS was detected in liver and blood of marine mammals from most the locations including those from Arctic waters. The greatest concentrations of PFOS found in liver and blood were 1520 ng/g wet wt in a bottlenose dolphin from Sarasota Bay in Florida, and 475 ng/mL in a ringed seal from the northern Baltic Sea (Bothnian Sea), respectively. No age-dependent increase in PFOS concentrations in marine mammals was observed in the samples analyzed. The occurrence of PFOS in marine mammals from the Arctic waters suggests widespread global distribution of PFOS including remote regions.

Levels in biota in the Arctic

A recent study (Verrault et al. 2005) supports the conclusion for bioaccumulation made in the background document provided by Sweden. The study investigated the distribution of a suite of PFAS in plasma, liver, brain, and egg samples from adult glaucous gulls (Larus hyperboreus), an apex scavengerpredator seabird breeding in the Norwegian Arctic. 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. Among the body compartment/ tissue samples analyzed, PFOS was highest in plasma (48.1-349 ng/g wet weight (ww)), followed by liver ≈ egg >brain.
PFAS concentrations in the study suggest a bioaccumulation potential in Norwegian arctic glaucous gulls that needs to be assessed as part of a broad organohalogen contaminant cocktail with potential for mediating biological processes in this vulnerable top-predator marine species.

A report from 2004 (Verrault et al. 2004) examined the congener patterns and the current levels of new and established organohalogen contaminants and their metabolites in plasma and egg homogenate samples of glaucous gulls breeding at Bear Island (Bjørnøya). Perfluorooctane sulfonate (PFOS), a compound of PFCs, was quantified in plasma samples at levels higher than those of some legacy OCs, being comparable to e.g. levels of DDTs. Hence, PFOS appears to be a chemical of relatively high importance in glaucous gull plasma.

A Norwegian screening screening study (Gabrielsen at al. 2004) detected levels of PFOS in polar bear (Ursus maritimus) adipose tissue and blood samples from Svalbard. The plasma levels was higher than those reported in ringed seals in the same area from 1996 and 1998 suggesting that PFOS have a biomagnification potential. PFOS plasma concentrations is reported to be one of the most prominent contaminants in polar bears. According to this study the plasma concentrations in polar bears in Svalbard appear to be 2-3 times higher than those in Alaska. The PFOS levels in polar bears reported in this study (converted to liver concentrations by using conversion factors; 1290 ng/g w.w) are well below the estimated NOALS and LOAELS (15 000 and 58 000 ng/g w.w. in liver, respectively) for second generations effects in rats.

Perfluoroalkyl substances were determined in liver tissues and blood of polar bears from five locations in the North American Arctic and two locations in the European Arctic (Smithwick et al. 2005). Concentrations of perfluorooctane sulfonate (PFOS), perfluorohexane sulfonate, heptadecafluorooctane sulphonamide, and perfluoroalkyl carboxylates with C8-C156 perfluorinated carbon chains were determined us8ing 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 Chucki Sea (453-729 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.

In a study performed on Glacous gulls, an apex scavenger-predator seabird breeding breeding in the Norwegian Arctic (Verrault et al. 2005), the distribution of a suite of PFAS in plasma, liver, brain and egg samples from adults were investigated. PFOS was the predominant PFAS in all samples and was present Ar concentrations that are the highest reported thus far in any arctic seabird species and populations. Among the body compartement/tissue samples analyzed, PFOS was highest in plasma (48.1- 349 ng/g wet wt), followed by liver ≈ egg > brain. Current PFAS concentrations suggest a bioaccumulation potential in Norwegian Arctic glacous gulls that need to be assessed as part of a broad organohalogen contaminant cocktail with potential for mediating biological

processes in this vulnerable top-predator marine species.

Levels in human blood serum and milk

A recent study (Olsen et al. 2005) explored the changes in levels of perfluorooctanesulfonate (PFOS), perfluorooctanoate (PFOA), and five other fluorochemicals in human blood serum from 1974 - 2001. Blood samples were collected in 1974 (serum) and 1989 (plasma) from volunteer participants of a large community health study. The study included a total of 356 samples (178 from each time period). These samples were analyzed by high-pressure liquid chromatography/tandem mass spectrometry methods. The median 1974 and 1989 fluorochemical concentrations, respectively, were as follows: 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. The data from 1989 were then compared with geometric mean fluorochemical concentrations of serum samples collected in 2001 from 108 American Red Cross adult blood donors from the same region. Except for M570, there were no statistically significant ($p < 0.05$) geometric mean fluorochemical concentration differences between the 1989 and 2001 samples. In conclusion, based on this study population, PFOS and other serum fluorochemical concentrations have increased between 1974 and 1989. Comparison with other regional data collectedin 2001 did not suggest a continued increase in concentrations since 1989.

In another study (Inoue et al. 2004), the concentrations of FOCs in maternal and cord blood samples was determined. 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. In contrast, PFOSA was not detected in fetal or maternal samples, whereas PFOA was detected only in maternal samples (range, < 0.5 to 2.3 ng/mL, 4 of 15). The results revealed a high correlation between PFOS concentrations in maternal and cord blood ($r2 = 0.876$). However, there were no findings of significant correlations between PFOS concentration in maternal and cord blood samples and age bracket, birth weight, or levels of thyroid-stimulating hormone or free thyroxine. The study revealed that human fetuses in Japan may be exposed to relatively high levels of FOCs. Further investigation is required to determine the postnatal effects of fetal exposure to FOCs.

Toronto University in Canada (Kuklenyik et al. 2004) have developed a high-throughput method for measuring trace levels of 13 PFCs (2 perfluorosulfonates, 8 perfluorocarboxylates, and 3 perfluorosulfonamides) in serum and milk using an automated solid-phase extraction (SPE) cleanup followed by high-performance liquid chromatography-tandem mass spectrometry. The method is sensitive, with limits of detection between 0.1 and 1 ng in 1 mL of serum or milk, is not labor intensive, involves minimal manual sample preparation, and uses a commercially available automated SPE system. The method is suitable for large epidemiologic studies to assess exposure to PFCs. The serum levels of these 13 PFCs were measured in 20 adults nonoccupationally exposed to these compounds. Nine of the PFCs were detected in at least 75% of the subjects. Perfluorooctanesulfonate (PFOS), perfluorohexanesulfonate (PFHxS), 2-(N-methylperfluorooctane-sulfonamido)acetate (Me-PFOSA-AcOH), perfluorooctanoate (PFOA), and perfluorononanoate (PFNA) were found in all of the samples. The concentration order and measured levels of PFOS, PFOA, Me-PFOSA-AcOH, and PFHxS compared well with human serum levels previously reported. Although no human data are available for the perfluorocarboxylates (except PFOA), the high frequency of detection of PFNA and other carboxylates in this study suggests that human exposure to long-alkyl-chain perfluorocarboxylates may be widespread. In the same study their were findings of PFOS in the serum and milk of rats administered PFOS by gavage, but not in the milk of rats not dosed with PFOS. Furthermore, we did not detect most PFCs in two human milk samples. These findings suggest that PFCs may not be as prevalent in human milk as they are in serum.

Because of its persistence, an important question has been whether elderly humans might have higher PFOS concentrations. From a prospective study (Olsen et al. 2004) designed to examine cognitive function in the Seattle (WA) metropolitan area, blood samples were collected from 238 dementia-free subjects (ages 65-96). High-pressure liquid chromatography-electrospray tandem mass spectrometry determined seven fluorochemicals: PFOS; N-ethyl perfluorooctanesulfonamidoacetate; N-methyl perfluorooctanesulfonamidoacetate; perfluorooctanesulfonamidoacetate; perfluorooctanesulfonamide; perfluorooctanoate; and perfluorohexanesulfonate. Serum PFOS concentrations ranged from less than the lower limit of quantitation (3.4 ppb) to 175.0 ppb (geometric mean 31.0 ppb; 95% CI 28.8-33.4). An estimate of the 95% tolerance limit was 84.1 ppb (upper 95% confidence limit 104.0 ppb). Serum PFOS concentrations were slightly lower among the most elderly. There were no significant differences by sex or years residence in Seattle. The distributions of the other fluorochemicals were approximately an order of magnitude lower. Similar to other reported findings of younger adults, the geometric mean serum PFOS concentration in non-occupational adult populations likely approximates 30-40 ppb with 95% of the population's serum PFOS concentrations below 100 ppb.

11 PFCs in 23 pooled serum samples has been measured collected in the United States from 1990 through 2002, and in serum samples collected in 2003 from 44 residents from Trujillo, Peru (Calafat et al. 2005). 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.

A pilot study has been conducted to provide preliminary data on the concentrations of perfluorooctanesulfonate (PFOS), perfluorooctanoic acid (PFOA) and perfluorooctanesulfonamide (PFOSA) in the blood of Canadians (Kubwabo et al. 2204). A set of 56 human serum samples was 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). PFOSA was not detected at levels above the method detection limit (MDL) in any of the samples. The levels of PFCs observed in the sample group of non-occupationally exposed humans in Canada were similar to the levels reported in a previous US study with a similar sample pool size. Two distinct PFOS isomers in human serum were identified by accurate mass determination.

To investigate the impacts of time, geographical location and sex on the levels of these chemicals, PFOS and PFOA concentrations in human sera samples has been measured collected both historically and recently in Miyagi, Akita and Kyoto Prefectures in Japan (Harada et al. 2004). The PFOS and PFOA levels in sera [Geometric Mean (Geometric Standard Deviation)] (microg/L) in 2003 ranged from 3.5 (2.9) in Miyagi to 28.1 (1.5) in Kyoto for PFOS and from 2.8 (1.5) to 12.4 (1.4) for PFOA. Historical samples collected from females demonstrated that PFOS and PFOA concentrations have increased by factors of 3 and 14, respectively, over the past 25 yr. There are large sex differences in PFOS and PFOA concentrations in serum at all locations. Furthermore, there are predominant regional differences for both PFOS and PFOA concentrations. In Kyoto the concentrations of PFOA in dwellers who had lived in the Kinki area for more than 2 yr were significantly higher than in people who had recently moved into the area, in both sexes. This finding suggests that there are sources of PFOA in the Kinki area that have raised the PFOA serum levels of its inhabitants. Further studies are needed to elucidate these sources in the Kinki area of Japan.

PFOS and PFOS related compounds, PFHxS, PFOA and PFOSA, have been measured in human blood from Several countries in a joint study performed by research institutes in different countries (Kannan et al. 2004). Samples were collected from the United States, Colombia, Brazil, Belgium, Italy, Poland, India, Malaysia, and Korea. Among the four perfluorochemicals measured, PFOS was the predominant compound found in blood sera. The PFOS related compounds degrade to PFOS. Concentrations of PFOS was highest in the samples collected from the United States and Poland (>30 ng/mL); moderate in Korea, Belgium, Malaysia, Brazil, Italy and Colombia (3 to 29 ng/mL); and lowest in India (<3 ng/mL). No age- or gender-related differences in the concentrations of PFOS were found in serum samples. The degree of assoctation between the concentrations of the four perfluorochemicals varied, depending on the origin of the samples. These results suggested the existence of sources with varying levels and compositions of perfluorochemicals, and differences in exposure patterns to these chemicals, in various countries.

Levels in indoor and outdoor air

High-volume samples of Perfluoroalkyls (PFAs) was collected for indoor and outdoor air to (Shoeib et al. 2004). Precursors to PFOS was measured, *N*-methyl perfluorooctane sulfonamideoethanol (MeFOSE), *N*-ethyl perfluorooctane sulfonamidoethanol (EtFOSE) and *N*-ethyl perfluorooctane sulfonamidethylacrylate (MeFOSEA). Mean indoor air concentrations (pg/m3) were 2590 (MeFOSE), and 770 (EtFOSE). The ratios of concentrations between indoor and outdoor air were 110 for MeFOSE and 85 for EtFOSE.

Levels in biota in Latin America

In a study performed at the north coast of Colombia (Olivero et al. 2005), 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, 1.27ng/mL, respectively. PFOS concentrations in pelican organs decreased in the order of spleen>liver>lung>kidney>brain>heart>muscle. These results suggest, for the first time, that perfluorinated compounds are also found in wildlife from Latin American countries.

Levels in surface water in Japan

The concentrations of PFOA in surface water samples have been analysed collected from all over Japan by LC/MS with a solid phase extraction method (Saito et al. 2004). The lowest limits of detection (LOD) (ng/L) were 0.06 for PFOA and 0.04 for PFOS. The lowest limits of quantification (LOQ) (ng/L) were 0.1 for both analytes. The levels [geometric mean (GM); geometric standard deviation (GS)] (ng/L) of PFOA and PFOS in the surface waters were GM (GS): 0.97 (3.06) and 1.19 (2.44) for Hokkaido-Tohoku (n=16); 2.84(3.56) and 3.69 (3.93) for Kanto (n=14); 2.50 (2.23) and 1.07 (2.36) for Chubu (n=17); 21.5 (2.28) and 5.73 (3.61) for Kinki (n=8); 1.51 (2.28) and 1.00 (3.42) for Chugoku (n=9); 1.93 (2.40) and 0.89 (3.09) for Kyushu-Shikoku (n=15). The GM of PFOA in Kinki was significantly higher than in other areas (ANOVA $p<0.01$). Systematic searches of Yodo and Kanzaki Rivers revealed two highly contaminated sites, a public-water-disposal site for PFOA and an airport for PFOS. The former was estimated to release 18 kg of PFOA/d. PFOA in drinking water in Osaka city [40 (1.07) ng/L] was significantly higher than in other areas. The present study confirms that recognizable amounts of PFOA are released in the Osaka area and that people are exposed to PFOA through drinking water ingestion.

Time trends in biota

In a study performed at archived guillemot eggs from the Baltic Sea concentrations of PFOS and PFOA were measured (Holmström et al. 2005). PFOA was not detected in any of the samples, but there was an 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, butthis cannot be linked to the PFOS phase-out, which ocuured a the end of this period.

(e) Exposure in local areas (provide summary information and relevant references)	
- general	
- as a result of long-range environmental transport - 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)

International:

OECD risk assessment report by UK November 2002

EU Risk assessment report by UK from 2004

Report on PFOS by SCHER in EU from 2005

Report on PFOS in TC-NES PBT subgroup in EU from 2005

EU is now considering a proposal on prohibition of PFOS and PFOS-related compounds in products and chemical mixtures, with an exception for fire fighting foam.
UK and Sweden classification proposal for PFOS in EU 2005:
T Toxic
R48/25 Toxic;danger of serious damage to health by prolonged exposure if swallowed
R61 May cause harm to the unborn child
R51/53 Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment

National:

Survey on uses and releases in Norway from 2004

Survey on uses in fire fighting foam from 2005

A national action plan from april 2005;

Norway is now considering a proposal to prohibit use of fire fighting foam containing PFOS and PFOS-related compounds. This is today the major use in Norway.

PFOS and PFOS related compounds are on the list of national priority substances, with aim of substantially reductions of releases no later than 2010.

(g) Status of the chemical under international conventions

Nominated to the Stockholm Convention under UNEP and the POP-protocol under the Long-range air transport Convention under UNEP-ECE.

Considered a POP under the LRTAP Convention, Stockholm Convention and in the EU TC-NES PBT-subgroup.

PFOS was added to the OSPAR List of Chemicals for Priority Action in June 2003. HSC 2003 agreed that the UK should act as a lead country for this chemical.

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