9 FLUOROS

INTERNATIONAL SYMPOSIUM ON
FLUORINATED ALKYL ORGANICS
IN THE ENVIRONMENT

AUGUST 18,19, 20, 2005
TORONTO, CANADA

UNIVERSITY OF TORONTO

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Department of Chemistry, University of Toronto

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Derek Muir; NWRI, Environment Canada, Canada

Welcome to Fluoros

Greetings and welcome to Toronto and the first *Fluoros Symposium*.

I am extremely pleased at the overwhelming turnout, with attendance from every sector and from all over the world. It was with significant disappointment that we had to close registration early and turn away many who wanted to attend. This meeting was borne in the deliverables promised in a strategic grant written to the Canadian funding agency, NSERC, in the summer of 2000. To sell the impact I hoped for our project on fluorotelomer alcohols, I proposed a small meeting to explore progress and the outstanding research questions. I figured "surely a meeting would attract 50 and perhaps even a 100 participants." At this writing *Fluoros* has over 300 registrants, 12 invited talks, and 100 poster presentations. I'm looking forward to writing the final report for this grant!

The mandate of *Fluoros* was to be broad with respect to fluorinated alkyl organic chemicals and highlight research into fluorinated pesticides, pharmaceuticals, and industrial and consumer materials. Given the significant interest in PFOS and PFOA by regulators and the public at large, it is not surprising that almost the entire suite of submitted presentations revolves around these and associated materials. The questions regarding the widespread nature, high biological concentrations, sources, mechanisms of movement, environmental processing and toxic modes of action of these compounds are at the forefront of research and regulatory agendas worldwide. It is my hope and conviction that *Fluoros* will make a significant contribution to enhancing our understanding of these issues and promote the activities necessary to solve the environmental problems linked with the use of these materials.

This is a volunteer run and organized meeting. My graduate students who work on 'chemicals fluorinated' all pitched in to make this meeting happen. The international scientific advisory committee designed the program format and selcted the oral presenters. This effort, along with significant financial support from our sponsors, made possible a meeting that was financially modest in cost and hopefully memorable and valuable. I encourage all attendees to approach the meeting in this spirit and work to make this a highly successful exchange of ideas.

Scott Mabury

Fluoros Chair

Symposium Program

Thursday August 18, 2005

6:00 – 7:00	Registration	Royal York Hotel
7:00 – 9:00	Registration	Great Hall, Hart House, University of Toronto
7:00 – 10:00	Welcome Reception	Great Hall, Hart House, University of Toronto

Friday August 19, 2005

Morning Session	Environmental Fate and Transport	Royal York Hotel, Ontario Room
8:30	Opening Remarks, Scott Mabury, Fluoros Chair	
8:40 – 8:50	Moderator, Bob Buck, Dupont	
8:50 – 9:20	Scott Mabury, University of Toronto *Chemical Personality of Fluorinated Organics*	
9:20 – 9:50	Jennifer Field, Oregon State University *Fluorochemical fate in natural and engineered systems*	
9:50 – 10:20	Tim Wallington, Ford Motor Company *Atmospheric chemistry of long chain fluorinated organic compounds*	
10:20 – 10:30	Poster Trailers – Preview of Session Posters	
10:30 – 12:30	Poster Session 1 – Coffee and Tea, **Wellington Labs**	
12:30 – 2:00	Break for Lunch	

Afternoon Session	Analytical Chemistry & Monitoring	Royal York Hotel, Ontario Room
2:00 – 2:10	Moderator, Jon Martin, University of Alberta	
2:10 – 2:40	Bogdan Szostek, Dupont *Analytical methodology for monitoring of perfluoroalkyl substances (PFAS) in the environment: past, present and future*	
2:40 – 3:10	Nobuyoshi Yamashita, AIST, Japan *Global monitoring and trace analysis of perfluorinated chemicals in the environment*	
3:10 – 3:40	Derek Muir, NWRI, Environment Canada *Biomonitoring of perfluoroalkyl acids: An overview of the global and temporal trend data*	
3:40 – 3:50	Poster Trailers – Preview of Session Posters	
3:50 – 6:00	Poster Session 2 – Coffee and Tea	
7:00 – 9:00	**Fluoros Banquet**	Dim Sum in Chinatown Yui Wah, 421 Dundas St. W., Second Floor

Symposium Program

Saturday, August 20, 2005

Morning Session	Toxicology	Royal York Hotel, Ontario Room
8:30	Open	
8:40 – 8:50	Moderator, Keith Solomon, University of Guelph	
8:50 – 9:20	Wim deCoen, University of Antwerp *Wildlife toxicology of perfluorooctane sulfonic acid: effects and mechanisms*	
9:20 – 9:50	Joseph dePierre, Stockholm University *Nature of the molecular mechanism underlying the biological effects of perfluorooctane sulfonate and perfluorooctanoic acid*	
9:50 – 10:20	John Butenhoff, 3M *The descriptive toxicology of perfluorooctane sulfonate and perfluorooctanoate*	
10:20 – 10:30	Poster Trailers – Preview of Session Posters	
10:30 – 12:30	Poster Session 3 – Coffee and Tea	
12:30 – 2:00	Break for Lunch	

Afternoon Session	Risk Assessment and Regulatory Policy	Royal York Hotel, Ontario Room
2:00 – 2:10	Moderator, Charlie Auer, U.S. EPA	
2:10 – 2:40	John Giesy, Michigan State University *Toxicological evaluation of perfluorooctane sulfonate (PFOS) in the environment*	
2:40 – 3:10	Jennifer Seed, U.S. EPA *Overview of US EPA's assessment activities on PFOA and PFOS*	
3:10 – 3:40	John Arseneau, Environment Canada *Perfluorinated substances and Environment Canada's regulatory framework*	
3:40 – 3:50	Poster Trailers – Preview of Session Posters	
3:50 – 5:00	Poster Session 4 – Ice Cream Social, **3M**	
5:00 – 6:00	Fluoros Summary and Wrap-up	Royal York Hotel, Ontario Room

Environmental Fate and Transport

8:50 – 9:20
Scott Mabury, University of Toronto
Chemical personality fluorinated organics

9:20 – 9:50
Tim Wallington, Ford Motor Company
Atmospheric chemistry of long chain fluorinated organic compounds

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9:50 – 10:20
Jennifer Field, Oregon State University
Fluorochemical fate in natural and engineered systems

Analytical Chemistry and Monitoring

2:10 – 2:40
Bogdan Szostek, Dupont
Analytical methodology for monitoring of perfluoroalkyl substances (PFAS) in the environment: past, present and future

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2:40 – 3:10
Nobuyoshi Yamashita, AIST, Japan
Global monitoring and trace analysis of perfluorinated chemicals in the environment

3:10 – 3:40
Derek Muir, National Water Research Institute, Environment Canada
Biomonitoring of perfluoroalkyl acids: An overview of the global and temporal trend data

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Toxicology

8:50 – 9:20
Wim de Coen, University of Antwerp
Wildlife toxicology of perfluorooctane sulfonic acid: effects and mechanisms

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2:40 – 3:10
Jennifer Seed, U.S. Environmental Protection Agency
Overview of US EPA's assessment activities on PFOA and PFOS

3:10 – 3:40
John Arseneau , Risk Assessment Directorate, Environment Canada
Perfluorinated substances and Environment Canada's regulatory framework

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Environmental Fate and Transport

ENV001 Goss	Contributing to a better understanding of the partition behavior of fluorinated alcohols and olefins
ENV002 Katsuna	Tropospheric sink of $C_2F_5OC(O)H$ through dissolution in clouds and the ocean, estimated from the experimentally determined Henry's law constants of the related fluorinated esters
ENV003 Mellouki	The gas phase tropospheric removal of two fluoroaldehydes: C_3F_7CHO and C_4F_9CHO
ENV004 Koch	Biodegradation potential of a Clariant fluorotelomer-based acrylate polymer – Results from a test on inherent biodegradability (OECD 302B)
ENV005 Sulbaek Andersen	The atmospheric chemistry of perfluorinated aldehyde hydrates: A source of perfluorinated carboxylic acids
ENV006 Higgins	Potential role of sediments in determining perfluorochemical surfactant fate
ENV007 Liu	Solubility and sorption of 8:2 fluorotelomer alcohol by surface soils
ENV008 Klasmeier	Evaluation of the environmental fate and exposure for perfluorinated telomer B alcohols
ENV009 Buck	Determining the fate of fluorotelomer alcohol and PFOA in the textile finishing process
ENV010 Korzeniowski	Sources of perfluorocarboxylic acids in the environment
ENV011 Wang	8-2 Fluorotelomer alcohol microbial biotransformation pathways. Time for a paradigm shift?
ENV012 van Roon	A mechanistic study of hydrogen bonding in fluorotelomer alcohol liquids
ENV013 van Roon	Vapour pressures of fluorotelomer alcohols
ENV014 Schultz	Behavior of fluorochemicals during wastewater treatment

ENV015 Rhoads	Microbial transformation of N-Ethyl-N-(2-Hydroxyethyl)Perfluorooctanesulfonamide
ENV016 Waterland	Global transport of biogenic and anthropogenic surfactants on marine aerosols
ENV017 Martin	Atmospheric lifetime and oxidation products of N-Ethyl perfluorobutylsulfonamide ($C_4F_9SO_2N(H)CH_2CH_3$)
ENV018 Berti	Biodegradation studies of fluorotelomer-based polymers to assess their potential to contribute to perfluorinated carboxylic acids in the environment
ENV019 Panko	Retrospective modeling of potential residential exposure to perfluorooctanic acid (PFOA) releases from a manufacturing facility
ENV020 Ellis	The degradation of fluorotelomer alcohols in the troposphere
ENV021 D'eon	Atmospheric Fate of N-Methyl Perfluorobutane sulfonamidoethanol (N-MeFBSE, $C_4F_9SO_2N(CH_3)(CH_2CH_2OH)$)
ENV022 Dinglasan	Biodegradation of 8:2 telomer alcohol and 8:2 telomer acids under aerobic conditions
ENV023 Dinglasan	Evidence of 8:2 FTOH production from the biodegradation of 8:2 telomer methacrylate under aerobic conditions
ENV024 Furdui	Perfluoroalkyl contaminants in lake trout from the Great Lakes
ENV025 Young	Atmospheric lifetime and global warming potential of a perfluoropolyether
ENV026 Young	Atmospheric flux of perfluorinated acids into the High Arctic
ENV027 Butt	Spatial and temporal trends of perfluoronated alkyl substances in ringed seals and seabirds from the Canadian Arctic

Contributing to a better understanding of the partition behavior of fluorinated alcohols and olefins

Kai-Uwe Goss[1], Guido Bronner[1], Hans Peter Arp[1], Christian Niederer[1], Torsten Schmidt[2], Monika Hertel[2]

[1] Dep. of Environmental Sciences, Swiss Federal Inst. of Technology, Zurich,Switzerland [2] University of Tübingen, Germany

The transport and accumulation of organic chemicals in the environment is governed by their equilibrium partitioning between various media such as water, air, soil, plants, etc. Data for saturated air/water partitioning and vapor pressure have been published in the literature. From the latter data it has been concluded that the ability for hydrogen bonding is strongly reduced for the fluorinated alcohols. This would have wide ranging consequences for their environmental partitioning. We have performed various checks on consistency and plausibility of these data and conclude that they are not correct. In our own experiments we have measured data for air/water, air/quartz surface and air/humic acid partitioning as well the partitioning between air and various organic polymers for fluorinated alcohols and olefins. All our data pass the plausibility and consistency checks that we applied before on the literature data. Furthermore, all our data indicate that the fluorinated alcohols engage in strong hydrogen bonds. From our partition data for the organic polymers we determined Abraham's LSER descriptors of the fluorinated compounds. These LSER descriptors comprehensively describe the partition behavior of a compound in any kind of partition system and have been shown to be much superior to descriptors such as octanol/water or octanol/air that have been used traditionally for the prediction of environmental partitioning. Here we find a good agreement between LSER predictions based on our descriptors and our experimental data for air/water and air/quartz partitioning. However, our experimental sorption coefficients from air to humic acid are systematically higher by up to an order of magnitude and more compared to those predicted with the respective LSER equation. This came as a surprise because the LSER equation worked well for 170 other organic compounds covering a range of 7 orders of magnitude in the partition constants.

Tropospheric sink of $C_2F_5OC(O)H$ through dissolution in clouds and the ocean, estimated from the experimentally determined Henry's law constants of the related fluorinated esters

S. Kutsuna, L. Chen, T. Abe, J. Mizukado, T. Uchimaru, K. Tokuhashi, and A. Sekiya

National Institute of Advanced Industrial Science and Technology (AIST)

$C_2F_5OC(O)H$ is an atmospheric oxidation product of $C_2F_5OCH_3$, an HFC substitute, and $C_2F_5OC(O)H$ is expected to have a relatively long lifetime (3.6 year) through only a gaseous reaction with OH. Since $C_2F_5OC(O)H$ is known to hydrolyze easily, dissolution in cloud water and/or seawater is expected to act as sinks for tropospheric $C_2F_5OC(O)H$ and it should be estimated. However, $C_2F_5OC(O)H$ is difficult to make and its solubility data such as Henry's law constants are unknown.In this study, we determined the Henry's law constants of four fluorinated esters [$CF_3CH_2OC(O)H$, $CF_3CH_2OC(O)CH_3$, $C_2H_5OC(O)CF_3$, $CH_3OC(O)CF_3$] and four related non-fluorinated esters [n-$C_3H_7OC(O)H$, $C_2H_5OC(O)H$, $CH_3OC(O)H$, $C_2H_5OC(O)CH_3$] at 278 – 298 K by a column-stripping method, and used the solubility and reactivity of the esters determined herein to estimate tropospheric lifetime of $C_2F_5OC(O)H$ through dissolution in clouds and the ocean.

$$H = H_{298} \exp\left[-\frac{\Delta H_{sol}}{R}\left(\frac{1}{T}-\frac{1}{298}\right)\right],$$

From the van't Hoff equation of the H_{298} (M atm^{-1}) and ΔH_{sol} (kJ mol^{-1}) values obtained were 0.55 ± 0.04, _39 ± 3 ($CF_3CH_2OC(O)H$); 0.58 ± 0.05, _ 44 ± 3 ($CF_3CH_2OC(O)CH_3$); 0.09 ± 0.01, _ 41 ± 5 ($C_2H_5OC(O)CF_3$); 0.09 ± 0.01, _ 45 ± 3 ($CH_3OC(O)CF_3$); 4.2 ± 0.2, _ 33 ± 2 ($CH_3OC(O)H$); 3.4 ± 0.2, _ 38 ± 2 ($C_2H_5OC(O)H$); 2.6 ± 0.1, _ 42 ± 1 (n-$C_3H_7OC(O)H$); and 6.0 ± 0.5, _ 49 ± 3 ($C_2H_5OC(O)CH_3$), respectively. Errors reported are at 95% confidence level and represent precision only. Hydrolysis was observed for $C_2H_5OC(O)CF_3$ and $CH_3OC(O)CF_3$. Replacement of CH_3 by CF_3 decreased H_{298} values by 6, 11, and 67 times, respectively, for $CF_3CH_2OC(O)H$, $CF_3CH_2OC(O)CH_3$, and $C_2H_5OC(O)CF_3$, while the differences in the ΔH_{sol} values were small with these substitutions. Sodium chloride salting-out effects were also examined.

On the basis of the solubility data determined herein and the hydrolysis rate constant reported for $CF_3CH_2OC(O)H$ with the effects of fluorination on Henry's law constants of the esters studied, the ocean is expected to serve as a tropospheric sink of $C_2F_5OC(O)H$, even if the solubility of $C_2F_5OC(O)H$ is one-hundredth that of $CF_3CH_2OC(O)H$. Dissolution into clouds may also be a significant sink of $C_2F_5OC(O)H$, only if $C_2F_5OC(O)H$ has a large hydrolysis rate constant such as 10^{-1} s^{-1}.

The gas phase tropospheric removal of two fluoroaldehydes: C_3F_7CHO and C_4F_9CHO

G. Solignac[1], A. Mellouki[1], G. Le Bras[1], Mu Yujing[2]

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Fluorotelomer alcohols (FTOHs, $C_nF_{2n+1}CH_2CH_2OH$), used in a variety of industrial products, have been suggested as a potential source of persistent perfluorinated carboxylic acids (PFCAs, $C_nF_{2n+1}C(O)OH$) which have been detected in the environment. PFCAs may be produced in the gas phase from the aldehydes (PFAs, $C_nF_{2n+1}CHO$), which are likely to be secondary atmospheric oxidation products of FTOHs.

The contribution of perfluorinated aldehydes to the atmospheric production of PFCAs depends largely on their degradation mechanisms. In the gas phase, the principal degradation processes of PFAs are controlled by reaction with OH and photolysis. While the OH-initiated oxidation of PFAs has been suggested to lead ultimately to PFCAs, the atmospheric photolysis may not produce these acids. Hence, it is of importance to assess the contribution of each process to the atmospheric degradation of the perfluorinated aldehydes in order to evaluate the gas phase atmospheric production of PFCAs from PFAs.

In this context, we have conducted a study of the photolysis and the OH reaction of two fluoroaldehydes (C_3F_7CHO and C_4F_9CHO). The UV absorption spectra have been determined in a 100 cm long double-jacketed Pyrex cell using a D_2 lamp as a light source and a detection system based on diode array detector. The absorption cross sections were measured in the wavelength range 250-350 nm. The rate constants for the reactions of OH with C_3F_7CHO and C_4F_9CHO have been determined using the pulsed laser photolysis-laser induced fluorescence (PLP-LIF) system in the temperature range 253-373 K. In addition, the European outdoor photoreactor EUPHORE will be used to study the photolysis of the two aldehydes under sunlight conditions in June 2005.The data obtained in different systems will be presented and discussed in terms of their atmospheric impact.

Biodegradation potential of a Clariant fluorotelomer-based acrylate polymer – Results from a test on inherent biodegradability (OECD 302B)

Willibald Boese[1], Catherine Ho[2], Wolfgang Knaup[3] and Volker Koch[4*]

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Fluorotelomer-based acrylic polymers (FBAPs) are speciality chemicals being used for coating of textiles, paper and carpet to achieve oil, stain and water repellency properties. The major building block of these high molecular polyacrylates is the Fluorotelomer alcohol 2-Perfluorooctylethanol (8-2 OH) which may undergo e.g. biotransformation to oxidation products like 2H,2H-Perfluorodecanoic acid (8-2 COOH), 2H-Perfluoro-2-decenoic acid (8-2 U COOH) and PFOA (8 COOH). Such biotransformation processes are slow under conditions of 'ready biodegradability' and therefore a test on'inherent biodegradability' was carried out.

It is known from the scientific literature that unpolar high molecular weight (MW) polymers e.g. alkyl polyacrylates are not biodegraded in a short time frame (Alexander, M., Biodegradation and Bioremediation, 2nd Ed., p. 400, Academic Press, San Diego, 1999). In addition Clariant investigations have shown that FBAPs applied to textiles are not hydrolyzed when heated in water at 50 degree C at pH 4, 7 and 9 for one year. Based on these facts the following Working hypothesis was established: 1) High MW FBAPs will not be cleaved hydrolytically in a 28d Test on Inherent Biodegradability (Zahn-Wellens Test, OECD 302B). As Hydrolase enzymes are polar an enzymatic ester cleavage is also not likely as the perfluoro moiety will effectively prevent enzyme docking at the ester function. This is related to observed and desired effects on water, oil and stain repellency and 2) If the Clariant FBAP is tested for Inherent Biodegradability in a laboratory test any formation of transformation products should be attributable to the residuals (low molecular starting materials) only whereas the polymer itself will not be transformed. The Clariant FBAP tested has to > 99.9% the 8-2 OH bound as ester in the polymer and << 0.1% is low MW residuals.

The 28d Zahn-Wellens Test was carried out in a Sturm test setting to allow trapping of volatiles in a reversed phase air cartridge as well. Great care was taken to develop specific analytical methods for the analytes 8-2 OH, 8-2 Acrylate, 8-2 COOH, 8-2 U COOH and 8 COOH in air and sludge matrix. Sampling 8-2 OH and 8-2 Acrylate in sludge was carried out by SPME, whereas in the air cartridge methanol was used for extraction. Analysis itself was done by GC MS (8-2 OH: SLUDGE LOQ 10µg/L, LOD 3.3 µg/L, AIR LOQ 1µg/trap, LOD 0.3 µg/trap; 8-2 Acrylate: SLUDGE LOQ 5µg/L, LOD 1.7 µg/L, AIR: LOQ 1µg/trap, LOD 0.3 µg/trap) . The acids were extracted using acetonitrile and analyzed using LC MS/MS (8xx acids: SLUDGE LOQ 3 µg/L, LOD 1 µg/L, AIR LOQ 0.2 µg/trap, LOD 0.07 µg/trap).

A detailed and rigorous sampling and analysis scheme was applied to the test (multiple samplings and analysis per time point). The statistical evaluation of the test results supports the working hypothesis given above which means that the amount of 8xx Acids formed after 28d can be explained by the amount of residuals in the test substance. Ester cleavage of the FBAP was not observed under the test conditions using the analytical methods described above.

The atmospheric chemistry of perfluorinated aldehyde hydrates: A source of perfluorinated carboxylic acids

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Perfluorinated aldehydes, $C_xF_{2x+1}CHO$, are formed during the atmospheric oxidation of HFCs, HCFCs, fluorinated alcohols and fluorotelomer alcohols (FTOHs). Atmospheric oxidation of perfluorinated aldehydes has previously been investigated by this group and offers a possible route to the formation of perfluorinated carboxylic acids (PFCAs). It is also well established that $C_xF_{2x+1}CHO$ can form stable hydrates of the formula $C_xF_{2x+1}CH(OH)_2$:

$$C_xF_{2x+1}CHO_{(g)} + H_2O_{(l)} \rightleftarrows C_xF_{2x+1}CH(OH)_{2\,(l)} \rightleftarrows C_xF_{2x+1}CH(OH)_{2\,(g)}$$

Smog chamber/FTIR techniques were used to study the Cl atom and OH radical initiated oxidation of perfluorinated aldehyde hydrates, $C_xF_{2x+1}CH(OH)_2$ (x=1,3,4) in 700 Torr of Air/N_2/O_2 diluent. It was established that reaction of the hydrate with OH radicals gives $C_xF_{2x+1}C(\bullet)(OH)_2$ radical which reacts with O_2 to give $C_xF_{2x+1}COOH$ (and HO_2) in100% molar yield.

Rate constants of $k(C_xF_{2x+1}CH(OH)_2 + Cl) = (5.92 \pm 0.88) \times 10^{-13}$ and $k(C_xF_{2x+1}CH(OH)_2 + OH) = (1.26 \pm 0.16) \times 10^{-13}$ cm^3 $molecule^{-1}$ s^{-1} were also determined and the IR spectra of $C_xF_{2x+1}CH(OH)_2$ (x=1,3,4) were recorded.To investigate the rate of hydration of perfluorinated aldehydes, gas phase trifluoroacetaldehyde, CF_3CHO, was bubbled though liquid water and introduced to the smog chamber. During a period of 2 hours the aldehyde showed a 35% conversion to gas phase hydrate through the addition of water. Experimental evidence (infrared and NMR spectroscopy) suggests that the process of hydrate formation occurs via a multi-step heterogeneous mechanism involving a) uptake of the aldehyde on the water surface, b) chemical formation of the hydrate and c) significant evaporation to the gas phase.The results are discussed with regard to the potential for the atmospheric degradation of perfluorinated aldehyde hydrates to contribute to the observed environmental burden of PFCAs.

Potential role of sediments in determining perfluorochemical surfactant fate

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While perfluorochemicals (PFCs) have been detected in many different environmental media such as air, water, and biota, data on the presence of PFCs in sediments are scarce. Quantifying PFCs in sediments may provide insight into their sources, distribution, and mobility in the environment. However, a major limitation to obtaining sediment data has been the lack of a sufficiently sensitive and accurate analytical method that accounts for the matrix effects typically observed in complex environmental media. Furthermore, while sorption of PFC surfactants onto sediments has been suspected, it remains unclear whether the organic-carbon partitioning paradigm for hydrophobic organic contaminants is applicable to PFC surfactants. To investigate the potential role of sediments in determining the environmental fate of PFC surfactants, a novel analytical method was developed that is capable of detecting PFCs at the sub-ng/g level in urban sediments. The method includes hot methanol extraction of the analytes from the sediment, a reversed phase solid phase extraction (SPE) clean-up procedure, and analysis via liquid chromatography tandem mass spectrometry. Once validated, the method was then applied to sediments collected from various locations in the San Francisco Bay Area. Aqueous concentrations of nutrients such as ammonium and ortho-phosphate, as well as the ∂^{15}N of particulate organic matter were also measured at these sites. Lastly, batch sorption isotherm experiments were conducted using gamma-irradiated sediments with varying geochemical compositions and anionic PFC surfactants of varying chain length.

Data from the survey of San Francisco Bay Area sediments suggests widespread occurrence of PFCs at the low ng/g to sub-ng/g level. Furthermore, substances that may be transformed to perfluorooctanesulfonate (PFOS), such as 2-(*N*-ethylperfluorooctanesulfonamido) acetic acid (*N*-EtFOSAA) are present in sediments at levels comparable to PFOS. Strong correlations were found between $\sum$ PFOS levels (sum of PFOS and all quantified PFOS precursors) and other indicators of sewage pollution such as ∂^{15}N, ortho-phosphate and ammonium (R^2 values of 0.67, 0.90, and 0.87, respectively), suggesting sewage may be a source of PFCs such as PFOS in urban sediments. For the batch sorption studies, perfluorocarbon chain length was the dominant structural feature influencing sorption, with significant sorption only occurring for PFCs with chain lengths of eight or greater (i.e., PFOS and perfluorononanoate, PFNA). *N*-EtFOSAA exhibited substantially stronger sorption than PFOS, suggesting that differences in the relative abundances of PFOS and these analytes in aqueous systems when compared to sediments may be due to differences in sediment-water partition coefficients. These interactions are the subject of continuing studies.

Solubility and sorption of 8:2 fluorotelomer alcohol by surface soils

Jinxia Liu and Linda S. Lee

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Fluorotelomer alcohols are widely used as raw intermediates in the synthesis of fluorinated polymers and surfactants, which serve as surface modifiers and protective chemicals. These polymers and surfactants are suspected to contain or degrade into fluorotelomer alcohols, and subsequently into perfluorinated acids, thus contributing to the global contamination by alky perfluorinated acids. Quantifying sorption and elucidating the sorption mechanisms of fluorotelomer alcohols in soils are important towards understanding the fate of these alcohols and related polymers particularly in landfills where products such as polymer-coated carpets and paper utensils are disposed.

The 8:2 fluorotelomer alcohol (CF_3-$(CF_2)_7$-$(CH_2)_2$-OH), one of the confirmed precursors of perfluorooctanoic acid and the most abundant species among fluorotelomer alcohols of various carbon chain lengths, was selected as the probe compound. Sorption from aqueous solutions by five soils representing a wide range of properties was determined directly and estimated by extrapolation from data measured in acetone/water solutions using a log-linear cosolvency model. The log-linear cosolvency model assumes a log-linear relationship between solubility or sorption and volume fraction cosolvent (f_c). The use of cosolvents minimizes volatilization losses, degradation, sorption to glassware, and effects of dissolved organic carbon (DOC), which can bias direct aqueous measurements. LC-ESI-MS/MS was employed to directly analyze the 8:2 fluorotelomer alcohol in soil solutions and soil extracts.

Sorption isotherms of 8:2 fluorotelomer alcohols with all five surface soils were generally linear regardless of f_c or soil organic carbon (OC) content. Aqueous sorption coefficients extrapolated from data measured in acetone/water solutions were in good agreement but consistently higher than those measured in aqueous solutions. Sorption appeared to be primarily driven by hydrophobic partitioning with a log K_{oc} value (OC-normalized soil-water distribution coefficient, L/kg OC) of 4.13 ± 0.16. Sorption by a high clay soil was relatively small suggesting that hydrogen bonding and electron donor/acceptor interactions do not play significant role in the sorption process.

Experimental variables such as temperature, soil:water ratio and equilibration time were further studied. Sorption of 8:2 fluorotelomer alcohol was exothermic, similar to many hydrophobic compounds, as indicated by a 60% increase in sorption when temperature was decreased from 22 ºC to 4 ºC. Sorption measured from a 1:1 soil:water slurry was five times smaller than that determined at a 1:40 soil:water ratio. Differences in sorption was hypothesized to be due association of the 8:2 fluorotelomer alcohol with DOC. DOC concentrations were 12 and 273 mg/L for the 1:1 and 1:40 soil:water ratios, respectively, which yielded a DOC binding constant of ≈ 10^4 L/kg DOC, similar to the estimated K_{oc}. Increasing equilibration time from 3 h to 72 h reduced soil extraction efficiency from 85% to 45% for the high OC soil even when including a NaOH-acetonitrile extraction step suggesting that there is a time-dependent irreversible sorption mechanism related to soil OC. Sorption coefficients comparable to a 3-ring polyaromatic hydrocarbon, irreversible sorption, and the potential for DOC-facilitated transport given the elevated DOC levels common to landfills are important processes to consider in assessing the likely environmental fate of fluorotelomer alcohols.

Evaluation of the environmental fate and exposure for perfluorinated telomer B alcohols

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Substance specific information to describe environmental partitioning and degradation behavior of four perfluorinated Telomer B alcohols (TBA) were collected and adapted for use with two steady-state multimedia fate models, namely SimpleBox2.0 and ELPOS1.0. The compounds' peculiar sorption behavior to environmental media was explicitly considered by using empirical correlations of substance properties with sorption constants from existing linear free energy relationships (LFER) and actual measurements of surface adsorption constants. Model simulations suggest that atmospheric deposition is not significantly affected by adsorption onto rain droplets or aerosol particles. On the other hand, total distribution constants for partitioning into soil and sediment are much higher than predicted by estimated K_{oc}-values, especially for the 8:2-TBA and 10:2-TBA congeners. With the best available data set the steady-state mass distribution calculated with SimpleBox shifts from 80% in air (for 4:2-TBA) to approximately 80% in soil (for 10:2-TBA).

Predicted regional environmental concentrations for Germany were simulated based on a realistic release scenario for textile application of perfluorinated polyacrylates containing residual perfluorinated Telomer B alcohols. The actual emission scenario was derived from measurements in a pilot textile mill under real application conditions. Surface water concentrations were below 1 pg/l for all four congeners and air concentrations ranged from 0.2 pg/m³ (4:2-TBA) to 15 – 20 pg/m³ (8:2-TBA). The latter is assumed to account for approximately 55% of total Telomer B alcohol emissions. Soil and sediment concentrations were shown to be highly dependent on the model assumptions with regard to the use of sewage sludge as agricultural fertilizer. The larger congeners tend to strongly partition to the sludge leading to higher concentrations in soil. A worst-case scenario assuming 100% transfer of sewage sludge to agricultural soil resulted in concentrations of 1.1-1.5 ng/kg in the soil for the 8:2 and 10:2-TBA congeners.

Characteristic travel distances in air (CTD) calculated with ELPOS were in the range of 2000– 4000 km, which suggests moderate long-range transport potential (LRTP) in the atmosphere. The CTDs for the perfluorinated Telomer B alcohols are close to the boundary range recently suggested for the LRTP classification of organic substances. Due to the limited knowledge about the environmental degradation behavior of the compounds, there is considerable uncertainty with the assumed half-lives in the environment. Atmospheric OH radical reactions seem to be the major removal pathway in the environment with an estimated half-life of 6– 14 days for all congeners depending on the actual OH radical concentration. Calculated overall persistence (Pov) increases with carbon number from 15 days (4:2-TBA) to half a year (10:2-TBA) as a consequence of the enhanced partitioning into soil and sediment, where the Telomer B alcohols are fairly persistent.

Determining the fate of fluorotelomer alcohol and PFOA in the textile finishing process

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Fluorotelomer-based acrylic polymer products are manufactured in batch processes from fluorotelomer alcohol raw materials such as 8-2 fluorotelomer alcohol (8-2 FTOH). While proceeding in high yields (>99%), small amounts, e.g. tenths of a percent, of unreacted raw materials remain. In addition, perfluorooctanoic acid may be present at trace levels in fluorotelomer-based products as an unintended reaction by-product. As part of an overall program to better understand the environmental fate of residual unreacted fluorotelomer alcohol and PFOA that may be present in products, a study was conducted to determine their disposition in textile finishing. Fluorotelomer-based polymeric products are aqueous dispersions which are diluted in water then applied via a foulard to fabrics followed by drying whereby the polymer is affixed to the treated fabric providing oil and water repellency and stain resistance. The study sought to achieve mass balance for 8-2 FTOH and PFOA through the textile finishing process using a standard textile finishing pilot plant facility which fully replicates industrial practice and is outfitted for sampling to capture and quantify air emissions through the dryer exhaust. How the study was conducted and the results of the study are the subject of this paper.

Two fluorotelomer-based acrylic polymeric products were prepared according to published literature procedures with 8-2 FTOH and PFOA added. The two test materials contained approximately 1,000 and 10,000 ppm 8-2 FTOH respectively and both approximately 15ppm PFOA. These levels are higher than what would be expected in commercial products yet served to allow for determinations well above limits of quantitation thereby facilitating mass balance throughout the process. Polyester fabric was treated and processed at a dryer temperature of 190°C. Samples of the application bath solution, wet fabric and dry fabric were taken as well as samples of exhaust air in a charcoal air sampling tube. Overall mass balance was achieved within experimental error. All 8-2 FTOH was accounted for in analyses of the air samples. The majority (>95%) of the PFOA was also accounted for in analyses of the air samples. $C_7F_{15}H$, a degradation product of PFOA, was observed and quantified in the air samples. No formation of additional 8-2 FTOH or PFOA through the textile finishing process was observed.

Sources of perfluorocarboxylic acids in the environment

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Perfluorinated carboxylic acids (PFCAs) have been identified as an emerging class of persistent environmental contaminants. Perfluorosulfonates (e.g. PFOS) and their potential precursors have been reported generally in the largest amounts as well as the widest presence throughout the global environment. Perfluorocarboxylic acids have also been found in a wide array of environmental samples ranging from water to biota. PFCAs of carbon chain lengths from four to fifteen and varying chemical structure (e.g. branched and unbranched) have been identified in environmental samples. PFCAs are generally considered to be persistent substances and potentially bioaccumulative for chain lengths >C9. The sources of PFCAs in the environment are neither well understood nor well documented. This paper will describe direct sources of PFCAs produced as industrial chemicals as well as indirect sources arising from transformation of potential precursor substances such as fluorotelomer alcohols and olefins. Direct emission sources include the manufacture of Ammonium Perfluorooctanote (APFO) and Ammonium Perfluorononanoate (APFN), Fluoropolymer manufacture from the 1950's, Fluoropolymer Dispersion manufacture from the 1950's, and Consumer and Industrial products that directly used PFCAs in their formulation. Indirect sources include the PFCA impurities in the POSF-based products, AFFF formulations, and potential POSF chemicals degradation products as well as the PFCA impurities in Fluorotelomer-based products, AFFF agents, and the potential degradation of PFCA precursors. The chain-length composition, release sources, historical and current amounts, and routes of entry to the environment will be discussed. The presentation will detail the sources of the historical breakdown of the several thousand metric tones of PFCAs released to the environment, most notably APFO and APFN. Additionally, it has been calculated that the global emissions of APFO/PFOA in 2000 alone were on the order of 225 metric tones (MT) to air, water, and land dominated by direct sources. The paper will also describe the many proactive measures taken by producers to reduce environmental releases of PFCAs and fluorotelomers. Programs will be reviewed and data presented to show that the various industries will have reduced APFO/PFOA global emissions by over 75% in 2006 from 225 to approximately 50 MT.

8-2 Fluorotelomer alcohol microbial biotransformation pathways. Time for a paradigm shift?

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8-2 Fluorotelomer Alcohol [$CF_3(CF_2)_7CH_2CH_2OH$; 8-2 FTOH] is a principal raw material used to manufacture fluorotelomer-based products. If a fluorotelomer-based product were biodegradable in the environment, the 8-2 FTOH would be an expected initial degradation product, which may be transformed in the environment. Understanding of 8-2 FTOH biotransformation pathways is critical to be able to determine the environmental fate of fluorotelomer-based products. We have investigated the biotransformation of [3-^{14}C] 8-2 FTOH [$CF_3(CF_2)_6{}^{14}CF_2CH_2CH_2OH$] in soil, activated sludge, sediment, and mixed bacterial culture over a period up to 7-months. Numerous novel transformation products have been identified that indicate transformation pathways. Multiple fluorinated carbons have been transformed in all cases as indicated by significant formation of perfluorohexanoic acid (PFHA).

120-130mL volume sealed glass bottles were used for the studies. The test substance (in ethanol stock solution) was added at 0.2 – 1.0 mg L^{-1}. An elevated 8-2 FTOH concentration (~20 mg kg^{-1} soil) was also included to facilitate metabolite identification. Sealed bottles were incubated at room temperature. The headspace ^{14}C-volatiles were monitored over time and the sample bottles were extracted periodically with an organic solvent such as acetontrile to recover the parent compound and transformation products. After extraction, the acetonitrile-extracted solution was used to separate and quantify the ^{14}C-labeled 8-2 TBA parent and ^{14}C-labeled transformation products by LC/ARC (on-line liquid chromatography/accurate radioisotope counting). The ^{14}C-labeled transformation products were identified by Q-TOF-MS. We found that the 8-2 FTOH can be strongly adsorbed to activated sludge and soil and subsequently transformed to non-volatile fluorinated acids, significantly reducing any potential air emission of 8-2 FTOH. PFOA [$CF_3(CF_2)_6{}^{14}COOH$], 7-3 acid ($CF_3(CF_2)_6{}^{14}CH_2CH_2COOH$), and a fluorinated acid (MW < 416 and eluted before ^{14}C-PFOA by LC/ARC) are three stable metabolites observed. The 8-2 fluorotelomer aldehyde [$CF_3(CF_2)_6{}^{14}CF_2CH_2CHO$] was observed in small quantity (~1%) in all studies. No perfluorononanoic acid (PFNA) is formed, indicating α-oxidation does not take place. 8-2 acid [$CF_3(CF_2)_6{}^{14}CF_2CH_2COOH$], 8-2 u acid [$CF_3(CF_2)_5CF={}^{14}CFCH_2COOH$] both are converted to other metabolites after day 28 in soil and activated sludge. Over 12% of $^{14}CO_2$ was detected in the headspace of the bottles containing activated sludge medium and over 2% of PFHA [$CF_3(CF_2)_4COOH$] was observed in soil samples. The formation of fluoride ion in mixed bacteria culture represents >10% equivalent of 8-2 FTOH total mineralization. These results suggest that alternative metabolic pathways are available to degrade 8-2 FTOH beyond PFOA, forming metabolites less than 8-carbons long. In addirion, decarboxylation of ^{14}C-PFOA [$CF_3(CF_2)_6{}^{14}COOH$; 99.7% radiochemical purity] by activated sludge was also observed at a very slow rate (~0.5% over 63 days), suggesting that: 1) PFOA may be degraded very slowly by microorganisms and such degradation won't be detected by conventional LC/MS/MS analysis; and 2) the majority of PFHA observed did not originate from PFOA, rather from 8-2 u acid, 7-3 u acid, and/or 7-3 acid via still yet unknown mechanisms.

A mechanistic study of hydrogen bonding in fluorotelomer alcohol liquids

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Intramolecular hydrogen (H-) bonding has been proposed as an explanation for the relatively high vapour pressures (*P*) of fluorotelomer alcohols (FTOHs) [1, and references therein]. Other researchers have used both experimental and computational methods to illustrate that intramolecular H-bonding is not significant and that the published high *P* values overestimate the actual *P* [2].

The discussion on FTOH *P*s would benefit from further computational work. The aim of this computational study was to obtain mechanistic insight into H-bonding in FTOH liquids by molecular mechanics force field (FF) simulations.The compounds included are *n*-alcohols (C6, 7, 8 and 10), and X:1 (X = 6, 7, 8 or 9) and X:2 (X = 4, 6 or 8) FTOHs. [For example, 6:1 FTOH and 6:2 FTOH consists of a methanoic moiety and ethanoic moiety, respectively, connected to a perfluorinated hexane tail.] The *n*-alcohols and X:1 FTOHs were included because they are structurally similar to the X:2 FTOHs but cannot (*n*-alcohols) or are less likely (X:1 FTOHs) to form intramolecular H-bonds. Liquids of the compounds were constructed by filling (periodic) boxes with an appropriate number of molecules. The AMBER FF [3] with additional parameters from [4] was used. The liquids were subjected to 500 ps molecular dynamics (MD) simulations. For an intramolecular H-bond to be present the O-C$_{\square}$-C$_{\square}$-C torsional angle should be in the gauche conformation and the hydroxylic proton directed to the fluorine atoms. During the simulation these angles and distances were monitored.

The structural data obtained from the simulations are summarized in Table 1. Although *n*-alcohols cannot form intramolecular H-bonds, the gauche conformation is observed more frequently in the *n*-alcohol liquid simulations than in the FTOH liquid simulations. For the gauche conformations, the average distances for the *n*-alcohols and FTOHs are not significantly different. The simulation results suggest that intramolecular H-bonding is not significant in FTOH liquids. Therefore we conclude that intramolecular H-bonding cannot explain the relatively high *P* values of FTOHs. The current results will be improved by calibrating the FF using experimental heats of vapourization and by fine tuning the criteria for H-bonding.

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Vapour pressures of fluorotelomer alcohols

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The presence of perfluorocarboxylates in remote areas has been explained partly by the atmospheric transport and oxidation of fluorotelomer alcohols (FTOHs). The vapour pressure (*P*) is a key parameter for atmospheric transport of chemicals. The *P* values reported for the FTOHs differ by two orders of magnitude [2-4]. The higher *P* values have been explained by the presence of an intramolecular hydrogen (H-) bond in FTOH liquids. Other researchers have used both experimental and computational methods to illustrate that intramolecular H-bonding is not significant and that the higher *P* values published overestimate the actual *P* [4].

The discussion on FTOH *P*s would benefit from further experimental work. The aim of this experimental study was to measure *P* values for FTOHs and related compounds using a method (GC-VAP [1]) based on gas chromatography (GC), still different from the methods used by others. The GC-VAP method has been applied successfully to several classes of compounds, including volatile and polar ones [1, and references therein].

The compounds included are X:1 (X = 6, 7, 8 or 9) and X:2 (X = 4, 6 or 8) FTOHs. [For example, 6:1 FTOH and 6:2 FTOH consist of a methanoic moiety and ethanoic moiety, respectively, connected to a perfluorinated hexane tail.] The X:1 FTOHs were included because they are structurally similar to the X:2 FTOHs but cannot form the same intramolecular H-bond. The GC-VAP method is based on the equilibrium fugacity model and the use of *n*-alkanes as reference compounds. In addition to *n*-alkanes, we have used *n*-alcohols as reference compounds because they are structurally similar to the FTOHs.

GC-VAP and literature *P* values are summarised in Table 1. The GC-VAP *P* values of the X:2 FTOH are at the lower range of the reported *P* values. The *P* values for 6:2 and 8:2 FTOH are similar to the *P* values for 7:1 and 9:1 FTOH, respectively. Because intramolecular H-bonding is probably less favourable in X:1 FTOHs, these results indicate that intramolecular H-bonding does not significantly affect *P* for X:2 FTOHs. It should be emphasised that, although the GC-VAP *P* values are at the lower range of reported *P* values, these values are still relatively high and do not exclude long range atmospheric transport of FTOHs as an important fate process.

The results of GC based methods for *P* depend on the choice of reference compounds. The current results will be improved by using perfluorinated *n*-alkanes as reference compounds (in addition to *n*-alkanes and *n*-alcohols). In addition, perfluorinated olefines will be included in the study.

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Behavior of fluorochemicals during wastewater treatment

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Fluorochemicals have widespread applications, and as a result of extensive consumer use, fluorochemicals may be released to municipal wastewater treatment plants via domestic wastewater. Wastewater effluent is a potential environmental point source of fluorochemicals. A high-volume-injection liquid chromatography with electrospray ionization tandem mass spectrometry (LC ESI-MS/MS) quantitative method was developed for the determination of trace levels of fluorochemicals in municipal wastewater influents and effluents. Recoveries from field spiked standard addition experiments ranged from 77.0% - 95.1% (± 2.4%) and 85.3% - 95.5% (± 2.3%) in the raw influent and final effluent, respectively. The limit of quantitation for the fluorinated alkyl substances was 0.5 ng/L. The method was applied to 24 h composites of raw influent and final effluent samples collected from 10 wastewater treatment plants (WWTPs) nationwide. Fluorochemicals were observed in wastewater at all treatment plants sampled, and each plant exhibited a unique fingerprint of fluorochemicals, despite similar treatment processes. In 9 out of the 10 plants sampled, at least one class of fluorochemicals exhibited increased concentrations in the effluent as compared to the influent concentrations. Detection of these analytes in final effluents at the ng/L level indicates that treated wastewater is a point source of fluorochemicals.

To better understand the behavior of fluorochemicals through a wastewater treatment plant, 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. Samples of wastewater (raw influent, primary effluent, trickling filter effluent, secondary effluent, and final effluent) and sludge (primary, thickened, activated, anaerobically digested, and storage lagoon) were collected over a duration of 10 days and were analyzed by LC ESI-MS/MS. Both removals and increases of fluorochemical concentrations in wastewater treatment plants were observed. 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. In this study, perfluoroalkyl carboxylates were overall removed by the wastewater treatment plant. When the assumption is made that the monitored plant is representative of wastewater treatment plants nationwide, fluorochemicals are discharged in wastewater effluents at a rate of 3180 kg/year and are introduced to terrestrial environments via biosolids at a rate of 7080 kg/year. If this assumption is valid, wastewater treatment plants are point sources of fluorochemicals and cannot be overlooked when determining origins and fate of fluorochemicals in the environment.

Microbial transformation of N-Ethyl-N-(2-Hydroxyethyl)Perfluorooctanesulfonamide

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Phosphate esters of *N*-Ethyl-*N*-(2-hydroxyethyl)perfluorooctanesulfonamide (N-EtFOSE) have been used in oil- and water-repellent coatings for paper and packaging products [1]. Hydrolysis of these phosphate esters presumably yields N-EtFOSE, which likely ends up in wastewater treatment plants and landfills. A study by Lange[2] indicated that N-EtFOSE is degraded in the presence of aerobic activated sludge to products including perfluorooctanesulfonate (PFOS). PFOS causes liver damage in monkeys and has been found in marine mammals and human blood [3,4,5]. The goal of this study was to determine the microbial degradation pathway and product distribution of N-EtFOSE in conditions approximating a wastewater treatment plant. The identification of N-EtFOSE metabolites may partially explain their occurrence in the environment.

N-EtFOSE was incubated in sealed serum vials with dilute activated sludge (~600 mg/L VSS) at 30ºC along with abiotic and autoclaved sludge controls. To gain further understanding degradation pathway, several detected metabolites were later incubated in the same fashion as N-EtFOSE. All samples were processed using solid phase extraction and analyzed by liquid chromatography with tandem electrospray mass spectrometry (LC/MS/MS).

N-EtFOSE was degraded to 34% of its initial concentration after 28 days in active cultures. Of the initial N-EtFOSE added, 63% can be accounted for in unreacted parent compound and products at the termination of the experiment. The major products detected were 2-(N-ethylperfluorooctanesulfonamido) acetic acid (N-EtFOSAA) and perfluorooctanesulfinate (PFOSI), which account for 60% and 15% of the metabolites, respectively. The proposed end product, PFOS, accounted for 7% of the metabolites at 28 days. Another potential end product, perfluorooctanoic acid (PFOA), accounted for 2% of the metabolites. PFOS and PFOA have both been shown to be stable in microbial cultures [6, 7]. The other detected metabolites (and relative amounts at 28 days) were perfluooctanesulfonamidoacetate (FOSAA, 9%), perfluorooctanesulfonamide (FOSA, 3%), and N-Ethylperfluorooctanesulfonamide (N-EtFOSA, 4%).

The incubation of N-EtFOSA in separate experiments yielded the formation of FOSA, indicating possible oxygenase activity followed by loss of a two-carbon unit either as ethylene glycol or acetate. The incubation of FOSA yielded the deaminated product PFOSI, a reaction likely mediated by an amonialyase.

We propose a pathway for the aerobic biotransformation of N-EtFOSE involving initial oxidation to N-EtFOSAA. N-EtFOSA is formed by hydrolysis of N-EtFOSE or N-EtFOSAA. Further oxidation of N-EtFOSA yields FOSAA, which can hydrolyze to FOSA. The stable aerobic end products of N-EtFOSE transformation are PFOS and PFOA.

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Global transport of biogenic and anthropogenic surfactants on marine aerosols

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Direct mass spectrometric composition measurements of individual aerosol particles show that about 10% of the lower tropospheric marine aerosol is organic in nature. This organic fraction increases with altitude and reaches 50% or more in the upper troposphere[1]. Bulk solubility arguments cannot account for such high organic compositions. In addition, Tervahattu et al. have shown that individual aerosol particles from a variety of marine and continental sources have organic surface films[2]. Surfactants are expected to concentrate at the water-air interface and subsequent wave action can produce marine aerosols enriched with these surfactants. Ellison *et al*. [3] have proposed an "inverted micelle" structure for the organic marine aerosol in which a biological surfactant monolayer encapsulates an aqueous core. In this poster, we propose that such inverted micelles may represent an important environmental transport pathway for anthropogenic fluorinated surfactants such as perfluorocarboxylic acids over large distances on the time scales of synoptic meteorology. This pathway may contribute to the observed widespread distribution of these materials.

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Atmospheric lifetime and oxidation products of N-Ethyl perfluorobutylsulfonamide ($C_4F_9SO_2N(H)CH_2CH_3$)

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A novel class of perfluorooctylsulfonamides ($C_8F_{17}SO_2N(R^1)(R^2)$) was previously detected in the atmosphere, and we hypothesized that these may serve as "PFOS precursors" - which through atmospheric transport and oxidation would lead to the deposition of PFOS at remote global locations. Here we present results of relative rate and product studies performed in the Ford Motor Company Smog Chamber with the model compound *N*-ethyl perfluorobutylsulfonamide (NEtFBSA; $C_4F_9SO_2N(H)CH_2CH_3$), but results are discussed by extrapolation to the environmentally relevant species, *N*-ethyl perfluorooctylsulfonamide (NEtFOSA; $C_8F_{17}SO_2N(H)CH_2CH_3$). Kinetic experiments were analyzed using *in situ* FTIR while product studies were analyzed by both *in situ* FTIR and off-line LC/MS(/MS). The first-order rate constant for reaction of NEtFBSA with chlorine atoms was $k_{Cl} = (8.68\pm1.76)\times10^{-12}$ cm^3 molecule^{-1} s^{-1}. However, the fate of NEtFBSA in the gas-phase of the troposphere will be controlled by reaction with OH radical ($k_{OH} = (3.74\pm0.7)\times10^{-13}$ cm^3 molecule^{-1} s^{-1}), and by scaling to the empirically observed lifetime of CH_3CCl_3, NEtFBSA will have an estimated lifetime of 59 d in the atmosphere; thus allowing for substantial long-range atmospheric transport.

COF_2 and SO_2 were the sole products that could be confirmed in product studies analyzed by FTIR, but these two species shared very similar temporal profiles, such that they were formed only in small quantities until the majority of NEtFBSA was consumed, followed by a dramatic increase thereafter. The "residual products" were more persistent than NEtFBSA, and secondary reactions, including unzipping of the fluorinated chain to yield COF_2, proceeded only after NEtFBSA was largely consumed. LC/MS/MS analysis of smog chamber air, collected during the product studies using XAD cartridges, identified several products distinguishable by their retention times, product ion scans, and/or accurate mass determination. The primary products of chlorine atom initiated oxidation were a 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 the ketone and aldehyde #1. LC/MS/MS also indicated that a major product in all samples was the perfluorobutylsulfonamide (i.e. $C_4F_9SO_2NH_2$), however this product was later synthesized and the spectrum collected by FTIR did not confirm its formation *in situ* until very late in the experiments. Therefore, its detection by LC/MS/MS was perhaps an artefact of the analytical conditions. Perfluorobutanesulfonate was not detected above the level of the blank in any sample, however, three perfluoroalkyl carboxylates (PFCAs) were detected above the level of the blank in Sample 1, 2, and 3; perfluorobutanoic acid, perfluoropropionic acid, and trifluoroacetic acid. Taken together, results suggest a plausible route by which perfluorooctylsulfonamides may serve as atmospheric sources of PFCAs (including PFOA), but not as a direct source of perfluoroalkylsulfonates, such as PFOS. The observed intermediate products may, however, be deposited by wet or dry deposition to the biosphere where they may be further processed to PFOS and/or perfluoroocanesulfonamide, which are both ubiquitous environmental contaminants. To better estimate the environmental fate of perfluoroalkylsulfonamides, the wide range of existing physical property determinations must be reconciled and the gas-particle partitioning determined.

Biodegradation studies of fluorotelomer-based polymers to assess their potential to contribute to perfluorinated carboxylic acids in the environment

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Biodegradation of fluorotelomer-based polymers has been postulated as a potential source of perfluorinated carboxylic acids (PFCAs) to the environment. Studies are underway to determine if the fluorinated moieties covalently bonded to a urethane and an acrylate polymer can be liberated from the polymer and transformed to PFCAs. The studies include four soils and two sediments that represent a range of chemical, physical, and mineralogical characteristics. The studies are being conducted under both aerobic and anaerobic conditions.

Treatments for the soil studies include an untreated control soil, live (non-sterilize) soil containing the test substance (i.e., commercially-sold polymer product emulsion) added at 200 mg polymer kg^{-1} soil (soil dry wt.), sterilized soil containing the test substance, and sterilized soil containing 8-2 Telomer B Alcohol (8-2 TBA), two telomer acids, and 8, 9, 10, and 11 carbon-containing PFCAs to assess recoveries over the year-long experiment. The test vessels being used are glass serum bottles with aluminum foil-lined closures that are incubated statically at 20°C. Soil moisture content is assessed regularly by weighing each bottle and adding water as needed to maintain a level of 40 to 60% water-holding capacity. Anaerobic conditions are imposed by flushing the test vessels with anaerobic mixed gas and then sealing them. A headspace sample from each vessel is passed through a C18 cartridge. The entire contents of the test vessel are then extracted using acetonitrile and an aliquot of a 200 mM NaOH solution.

Treatments and methods for the sediment studies are similar to those of the soil studies. Sediment and water are added to each test vessel so the resulting water:sediment volume ratio is between 3:1 and 4:1 and the minimum sediment layer is approximately 2 cm. The headspace gases within each of the aerobic test vessels are continuously purged with air at a low flow rate and passed through a C18 cartridge. Anaerobic test vessel conditions are imposed as in the soil test. In addition to headspace and sediment extract samples, separate aqueous samples are collected and analyzed.

The analyte 8-2 TBA was not found in the head space of any samples, including sterile soils to which it had been fortified at 0.54 µmole kg^{-1} (250 µg kg^{-1}, LOQ < 0.0125 µg mL^{-1} headspace gas). Results from the soil studies after a maximum duration of four weeks indicate that the concentrations measured of 8-2 TBA, two telomer acids, and 8 to 11 carbon-containing PFCAs extracted from soils can be accounted for by the concentrations of fluorinated residuals in the polymer products. Fluorinated residuals are low molecular weight intermediates, which did not react to form a covalent bond with the polymer, and their degradation products. These residuals include, but are not limited to, the 6-2 TBA, 8-2 TBA, and 10-2 TBA that are found in the polymer products at low concentrations.

Retrospective modeling of potential residential exposure to perfluorooctanic acid (PFOA) releases from a manufacturing facility

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A retrospective exposure analysis using various environmental models was conducted to estimate potential intake of perfluorooctanoic acid (PFOA) over the past 53 years by persons residing in parts of Ohio and West Virginia. PFOA has been detected in the public water supplies of water districts near Washington, WV and has been attributed to emissions from a local fluoropolymer manufacturing facility which has operated since 1951. A processing aid, ammonium perfluorooctanoate (APFO), was used in the manufacture of the fluoropolymers and released to the environment in air, water and solid waste emissions. In the environment, APFO disassociates to its anion form which is referred to as PFOA. Following considerable analyses, it was concluded that particulate deposition from facility air emissions to soil and the subsequent transfer of the chemical through the soil was the most likely source of the PFOA detected in the groundwater. For some water districts, releases to the Ohio River also served as a partial source of PFOA to the groundwater. A mass balance analysis of APFO used and released by the facility for each year of operation from 1951-2003 was the foundation of this analysis. Air emissions and deposition rates were modeled using EPA's ISCST3 model. Air deposition rates were then used as continuous input into the PRZM-3 model to estimate the PFOA concentrations in surface soil and the movement of the chemical to the groundwater. Estimates of the intake of PFOA by residents were model estimated for each water district for all relevant routes of exposure. Exposures were modeled for a 185 square mile area surrounding the facility. The highest off-site environmental concentrations were predicted to occur about 1 mile away. For this 1 square mile area, during the time period 1951 -2003, the model estimated air concentration was 0.2 $\mu g/m^3$ (range =0.00007 to 0.4 $\mu g/m^3$), the estimated surface soil concentration was 11 µg/kg (range =0.001 to 30 µg/kg), and the estimated drinking water concentration was 3 µg/L (range =0 to 14 µg/L). Similar data were generated for each of the other 18 areas around the facility. Comparison of the data on the actual concentrations of PFOA in groundwater in the various water districts indicated that the models over predicted recent groundwater concentrations by factor of 3 to 5. The predicted historical lifetime and average daily estimates of PFOA intake by persons who lived within 5 miles of the plant over the past fifty years (since the plant began operation) were about 10,000 fold less than the doses that were considered acceptable by a science advisory panel which was convened to identify public health risk criteria. To our knowledge, this is the first case study where air deposition of an organic chemical from an industrial facility impacted groundwater which served as a source of public drinking water. The methodology for exposure assessment used here may well be applicable to other water soluble, persistent chemicals emitted to the air.

The degradation of fluorotelomer alcohols in the troposphere

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Oxidation of fluorotelomer alcohols in the atmosphere by OH leads quantitatively to the production of the corresponding polyfluorinated aldehyde. Both the alcohol and the aldehyde have atmospheric lifetimes on the order of 20 days due to this process, e.g. ($k(OH + F(CF_2CF_2)_nCH_2CH_2OH = 1.07 \pm 0.22 \times 10^{-12}$ cm^3 molecule^{-1} s^{-1}. Wet and dry depositions are expected to be negligible for these species in comparison to their OH chemistry. The predominant oxidative pathway of the first formed aldehyde with OH is the production of a further aldehyhde which is perfluorinated. In the absence of NO_x, this perfluorinated aldehyde undergoes further oxidation by OH to produce the corresponding perfluorocarboxylic acids (PFCA) via the hydrolysis of a first formed acid fluoride. A second pathway available to the perfluorinated aldehyde is the production shorter chain PFCAs. Production of PFCAs by this second route is minor in comparison to the production of carbonyl fluoride, although environmentally still deemed to be significant. Questions still remain to be answered concerning the degree to which wet deposition plays a role in the atmospheric fate of the perfluorinated aldehyde and how significant these pathways are to the environmental burden of PFACs which contains varying amounts of NO_x.

Atmospheric fate of N-Methyl perfluorobutane sulfonamidoethanol (N-MeFBSE, $C_4F_9SO_2N(CH_3)(CH_2CH_2OH)$)

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In 2000 3M announced the discontinuation of its perfluorooctane sulfonamido chemistries due to environmental concerns. Recently 3M returned to the market with a product containing only four perfluorinated carbons, *N*-methyl perfluorobutane sulfonamidoethanol (*N*-MeFBSE), under its standard trade name Scotchgard. The atmospheric fate of this newly introduced industrial compound is the subject of this investigation. Considering little monitoring data is available for the recently introduced *N*-MeFBSE, the results of this study will be compared with considerable data regarding the environmental distribution and prevalence of its perfluorooctane counterparts *N*-MeFOSE and *N*-EtFOSE and their degradation products.

Atmospheric degradation was simulated using a 120 L pyrex smog chamber. OH radicals were produced by photolysis of CH_3NO with UV irradiation in the presence of NO. The OH initiated reaction was monitored using the relative rate technique, with acetylene and ethylene as competitors. The first order rate constant for the reaction of *N*-MeFBSE with OH radicals was found to be $k_{OH} = 5.6 \times 10^{-12}$ cm^3 molecule^{-1} s^{-1}. Using the empirically observed lifetime of CH_3Cl_3, *N*-MeFBSE was calculated to have an atmospheric lifetime of approximately 4 days. Estimating an average global wind speed of 4 m s^{-1}, *N*-MeFBSE will travel 1400 Km downwind from a point source.

Product analysis was carried out by sampling the smog chamber at appropriate intervals. Major products include carbonyl products from the oxidation of the ethanol chain, perfluorobutanoic acid (PFBA), perfluoropropanoic acid (PFPA), trifluoroacetic acid (TFA), perfluorobutane sulfonic acid (PFBS), and *N*-methyl perfluorobutane sulfonamine (*N*-MeFBSA).

Biodegradation of 8:2 telomer alcohol and 8:2 telomer acids under aerobic conditions

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Telomer alcohols are polyfluorinated compounds primarily used as an intermediate in the production of fluorinated surfactants and polymers used widely in textile, paper and carpet industries. They have been recently identified as potential precursor compounds to the environmentally ubiquitous perfluorinated acids. The biodegradation of the 8:2 telomer alcohol under aerobic conditions in a mixed microbial culture known to degrade ethanol was investigated. The cultures were spiked with approximately 50ug/L of the 8:2 telomer alcohol (8:2 FTOH) and its degradation was monitored using solid phase microextraction (SPME) coupled with gas chromatograph equipped with an electron capture detector (GC/ECD) The initial measured half-life of the 8:2 FTOH was ~0.2 days mg^{-1} of initial biomass protein**.** Telomer acids (8:2 FTCA, $CF_3(CF_2)_7CH_2COOH$; 8:2 FTUCA, $CF_3(CF_2)_6CF{=}CHCOOH$) and PFOA were identified as metabolites during the degradation, the unsaturated telomer acid being the predominant metabolite measured. The overall mechanism involves the oxidation of the 8:2 FTOH to the telomer acid via the transient telomer aldehyde. The telomer acid was further transformed via a mechanism similar to ß-oxidation, leading to the unsaturated acid and ultimately producing the highly stable PFOA.

Degradation experiments of the observed intermediate compounds were also conducted. Both 8:2 telomer acids (8:2 FTCA and 8:2 FTUCA) were degraded under similar conditions providing further evidence to the degradation pathway. The production of the 8:2 FTUCA and PFOA was observed in vessels spiked only with the 8:2 FTCA, while low levels of PFOA were detected in vessels spiked with 8:2 FTUCA only. Complete mass balance was not achieved in the experiments; however this may be due to metabolites found associated with biomass that was routinely removed from samples prior to analysis. These studies demonstrate that telomer alcohols are potential sources of PFCAs as a consequence of biotic degradation. Biological transformation may be a significant degradation pathway for fluorinated telomer alcohols in aquatic systems.

Evidence of 8:2 FTOH production from the biodegradation of 8:2 telomer methacrylate under aerobic conditions

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Telomer alcohols have recently been identified as precursor compounds to poly- and perfluorinated acids through aerobic microbial degradation. These alcohols however are not typically marketed as industrial scale products but are used as intermediates in the production of telomer-based surfactants and polymers. The 8:2 methacrylate (3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-Heptadecafluorodecyl methacrylate), a fluorinated monomeric acrylate produced from a typical synthetic reaction involving the transesterification of the 8:2 FTOH along with ethyl pyruvate, is an example of an unpolymerized compound presumed to be similar to intermediates used in the manufacture of telomer based polymers. In this study, evidence of 8:2 FTOH production from the biodegradation of this monomeric compound using inoculum obtained from a local sewage treatment plant (Ashbridges Bay, Toronto, ON Canada) was observed. Biodegradation experiments were conducted in purge and trap vessels equipped with XAD cartridges designed to trap the volatile metabolites. Residual alcohols were first stripped prior to addition of inoculum. 8:2 FTOH production was measured using a gas chromatographic system coupled with a mass selective detector (GCMS). A closed degradation experiment was also performed due to the volatility of the compound. In a closed system, telomer acids such 6:2 FTCA, 6:2 FTUCA, 8:2 FTCA and 8:2 FTUCA were detected as metabolites using electrospray LCMSMS. The observed lower chain telomer acids may be attributed to the degradation of residual 6:2 FTOH known to also be present in the 8:2 methacrylate. The total measured telomer acids at day 48 of the experiment make up approximately 3% of the initial spike of 8:2 methacrylate. This amount is higher than what can be attributed to degradation of residual telomer alcohols known to be present. Telomer alcohols have already been shown to biodegrade to both poly and perfluorinated acids while this study presents evidence of a fluorinated monomer producing similar metabolites, the important question that remains is whether biodegradation of polymeric material will also lead to perfluorinated acids that are found widespread in the environment.

Perfluoroalkyl contaminants in lake trout from the Great Lakes

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The goal of this study was to determine the spatial distribution of some selected perfluorinated surfactants in fish from the Great Lakes. Results were determined for perfluorinated carboxylic acids (heptanoic PFHpA, octanoic PFOA, nonanoic PFNA, decanoic PFDA, undecanoic PFUnA, dodecanoic PFDoA and tetradecanoic PFTeA), sulfonates (octa PFOS, deca PFDS and octane sulfonamide PFOSA) and unsaturated fluorotelomer acids (6:2, 8:2 and 10:2 FTUA). Individual whole fish homogenates were analyzed from 46 identically aged lake trout sampled in 2001 from each of the Great Lakes. Duplicates were analyzed from 20% of the samples. The analytes of interest were extracted using an ion-pairing agent and analyzed by LC/MS/MS, using an Agilent 1100 Series liquid chromatograph coupled with a 4000 QTRAP triple quadrupole mass spectrometer. Quantitation was performed using the relative response of each analyte (triple injections) to ^{13}C mass-labeled internal standards ($^{13}C_2$-PFOA, $^{13}C_2$-PFDA and $^{13}C_2$-8:2 FTUA).

The major contaminant was PFOS. The highest level was determined in samples from Lake Erie (280 - 510 ng/g), followed by samples from Lake Ontario (80 - 200 ng/g), Lake Huron (20 - 240 ng/g), Lake Michigan (25 - 85 ng/g) and Lake Superior (7 - 22 ng/g). PFDS was determined in all samples from Lake Erie (5 - 18 ng/g), Lake Ontario (3 - 9 ng/g) and some samples from Lake Michigan (0 - 3 ng/g), Lake Huron (0 - 0.8 ng/g), Lake Superior (0.4 - 1.2 ng/g).

The perfluorinated carboxylic acids were detected at 0 - 10 ng/g. For PFOA the highest level was observed for samples from Lake Michigan (average of 3.1 ng/g), followed by samples from Lake Ontario (1 ng/g), Lake Erie (0.7 ng/g), Lake Huron (0.7 ng/g) and Lake Superior (0.6 ng/g). The other acids detected were PFNA (1 – 10 ng/g), PFDA (0.2 – 5.5 ng/g), PFUnA (0.2 – 4.8 ng/g), PFDoA (0.2 - 2 ng/g) and PFTeA (0 – 1.5 ng/g).

From the fluorotelomer acids series the 8:2 FTUA was detected in 52% of the samples from each of the Great Lakes, at concentrations ranging between 0.1 and 0.5 ng/g. The occurrence of the 8:2 FTUA varied between 17% for samples from Lake Erie and 90% for samples from Lake Michigan. The 10:2 FTUA was detected only in four samples from Lake Ontario and Superior at an average concentration of 40 pg/g.

The lowest level of perfluoroalkyl contaminants was determined in samples from Lake Superior. Samples from Lake Erie had the highest level of PFOS, PFDA, PFUnA and PFDoA, while samples from Lake Michigan had the highest level of PFOA and 8:2 FTUA. Although comparable the levels determined in this study for perfluorinated carboxylic acids and sulfonates are slightly lower than the levels determined in an earlier study for samples from Lake Ontario(1).

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Atmospheric lifetime and global warming potential of a perfluoropolyether

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Hydrofluoroethers (HFEs) and perfluoropolyethers (PFPEs) are commonly used industrial heat transfer fluids for which little is known about their environmental occurrence and fate. HFEs and PFPEs have been marketed as replacements for the stratospheric ozone-depleting chlorofluorocarbons, yet they may be equally hazardous to the environment. C-F bonds absorb IR radiation within the atmospheric window and disrupt the energy balance of the earth, in a phenomenon known as radiative forcing. Both PFPEs and HFEs contain an unprecedented number of C-F bonds, which suggests that they would have a high radiative forcing. Some HFEs have been shown to have extremely high instantaneous forcing, of up to 1.37$Wm^{-2}ppb^{-1}$. It has been demonstrated that HFEs degrade in the atmosphere via hydrogen abstraction by OH, while it is unlikely that PFPEs will be oxidized. Consequently, it is expected that they will have a much higher global warming potential (GWP), due to longer lifetimes and similar radiative forcing values.

A distilled fraction of a PFPE found in the commercial mixture Galden HT70 was added to a 140 L pyrex chamber interfaced to an FTIR spectrometer. Competition kinetics was used to determine an upper limit of reactivity with both OH and Cl. The IR spectrum was recorded in 700 Torr of diluent air at 296K. The UV/vis spectrum in the liquid phase was used to assess photolysis lifetime. The reactivity of the PFPE with Cl was greater than 2 x $10^{-17}cm^3molecule^{-1}s^{-1}$, while the reactivity of OH was greater than 2.2 x $10^{-16}cm^3molecule^{-1}s^{-1}$. Given the unreactive nature of this compound, the minimum lifetime was assumed to be 1000 years. Using IR measurements, instantaneous forcings of the PFPE and CFC-11 were calculated as 0.64 and 0.26, respectively. Assuming a lifetime of 1000 years for the PFPE, a GWP can be calculated. Over a 100 year horizon, the GWP of the PFPE was calculated as 8400. Relative to the GWP of CFC-11, this corresponds to a value of 1.84. The low radiative forcing of this PFPE compared to some HFEs, H-Galden 1040x in particular, is surprising. An examination of the IR spectra of these compounds will provide insight into this observation.

Atmospheric flux of perfluorinated acids into the High Arctic

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Perfluorinated acids have been found ubiquitously in water and biota. Of special interest is the contamination of biota in the High Arctic, as these compounds have low volatility and are poor candidates for long range transport. A possible source of contamination of perfluorocarboxylic acids (PFCAs) to remote regions is the atmospheric oxidation of fluorotelomer alcohols (FTOHs). PFCAs were shown to be an oxidation product of FTOHs under low NOx conditions. Thus, in low NOx environments, such as the Arctic, it is possible that FTOHs provide a source of PFCAs. By measuring fluxes of selected PFCAs into the Arctic, it is possible to assess whether this mechanism provides the primary source of these compounds. Analogously, it is possible that perfluorooctane sulfate (PFOS) and related chemicals could be delivered to the Arctic via oxidation of polyfluorosulfamido-alcohols. Calculating a flux of PFOS would provide insight into this suggestion.

Surface samples were collected in spring of 2005 on Melville Ice Cap (Melville Island, NWT) and Agassiz Ice Cap (Ellesmere Island, NU). Surface and depth samples were collected during the same time period on Devon Ice Cap (Devon Island, NU). These locations are far from water bodies and any contamination should be principally atmospherically derived. Samples were injected into a SciEx 4000 LC-MS-MS for analysis. Perfluorinated acids were observed in the snow. Quantification was done using isotopically labeled perfluorooctanoic acid, perfluorononanoic acid, perfluorodecanoic acid and PFOS. Standard additions were used to verify the results. Data was corrected for precipitation and area to give the flux into the Arctic.

Spatial and temporal trends of perfluorinated alkyl substances in ringed seals and seabirds (Northern Fulmar and Thick-Billed Murre) from the Canadian Arctic

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It is now widely accepted that perfluorinated alkyl substances (PFASs) are globally distributed contaminants in both humans and biota. Further, several recent studies have shown that Arctic foodwebs, including trophic levels from zooplankton to polar bears, contain concentrations of C8-C15 perfluorinated carboxylic acids (PFCAs), perfluorooctane sulfonate (PFOS) and perfluorooctane sulfonamide (PFOSA). However, the spatial variation of PFAS concentrations and profiles between populations of Arctic biota has not been well characterized. This study examined the geographic variation of PFASs in two species of biota from the Canadian Arctic; ringed seals (*Phoca hispida*) (5 locations x 10 individuals) and seabirds (northern fulmars, 2 locations x 10 individuals). Further, the main producer of PFOS-related chemistry voluntarily ceased production in 2001. However, the response of animal populations to this production termination is not known. Thus, this study also examined temporal trends (~1970s-20004) of PFASs in ringed seals and two species of seabirds (northern fulmars and thick-billed murres).

Liver samples were homogenized and extracted with methyl tert-butyl ether (MTBE) and the ion-pairing agent, tetrabutylammonium hydrogen sulphate (TBAS). In addition, a protein precipitation clean-up step using a fluorinated solvent was included. Target analytes included C7-C15 PFCAs, 4-, 6-, 8-, 10-carbon perfluorosulfonates, PFOSA. In addition, suspected degradation compounds of the fluorotelomer alcohols, 8:2 fluorotelomer acid (8:2 FTCA), and 8:2 and 10:2 fluorotelomer unsaturated acids (8:2 and 10:2 FTUCA) were monitored. Chemical analysis was performed using liquid chromatography coupled with tandem mass spectrometry. Analytes were quantified by normalizing analyte response to stable isotope labelled standards ($^{13}C_2$-PFOA, $^{13}C_2$-PFDA and $^{13}C_2$-8:2 and 10:2 FTUCA).

Results indicated that PFAC concentrations varied between ringed seal populations with the most southern site, Inukjuaq, showing the highest concentrations. Within seal populations, PFOS was the major PFAS analyzed. Among PFCAs, profiles were dominated by C8-C11 acids with PFUnA dominant at eastern Arctic sites and PFNA dominant at central-western Arctic sites. Differences in PFCA profiles may indicate different sources of perfluorinated compounds to these regions. Examining northern fulmars, PFAS concentrations and profiles were similar between Cape Vera and Prince Leopold Island. In general, the major PFAS detected was the long-chained C11-C15 PFACs (2.5-11 ng/g), as compared to lower concentrations of PFOS (mean=0.7 ng/g).

Temporal trends of ringed seal and seabirds showed an increase in PFCAs and perfluorosulfonates from ~1970s-2004. Examining ringed seals (1972, 1993, 2000, 2004), PFOS was the only PFAS detected in the 1972 samples. However, concentrations of C8-C14 PFCAs increased from 1993-2004 with doubling times of 4-10 years. Surprisingly, PFOS concentrations in ringed seals declined from 2000-2004. Within seabird samples, PFAS concentrations increased rapidly between 1993-2004 in thick-billed murre samples, however, only a modest increase was observed in northern fulmars over this time period.

Analytical Chemistry and Monitoring

ANA001 Hoppe	Development of a new NCI GCMS method for the determination of environmental exposure to perfluorooctanoic acid and perfluorononanoic acid
ANA002 Verrault	Perfluorinated alkyl substances in plasma, tissues and eggs of glaucous gulls (Larus hyperboreus) from the Norwegian Arctic
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Development of a new NCI GCMS method for the determination of environmental exposure to perfluorooctanoic acid and perfluorononanoic acid

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While occupational exposure to perfluorinated compounds (PFC) can easily be assessed by analyzing PFC in plasma by LC-MSMS, the respective determination of plasma PFC levels resulting from environmental exposure is often complicated by contaminations from various sources such as Teflon plastics.

To overcome this analytical problem, we developed a new method for the trace level determination of perfluorooctanoic acid (PFOA) and perfluorononanoic acid (PFNA). Analyses of these two PFC are most frequently requested at our lab. Moreover, we present tentative reference values for PFOA and PFNA in plasma derived from the analysis of samples obtained from randomly selected German children.

PFC were extracted from plasma as ion pairs in TBME and subsequently derivatized with benzylbromide. The derivatives were quantified by NCI-GCMS using $^{13}C_2$-PFOA as internal standard. The applicability of the GCMS method was examined by analyzing plasma samples of 174 children and young adults (age range 0-24 years). The specimens were randomly selected from patients's samples of the Medical Lab Bremen.

The GCMS method permitted the quantification of 1 µg PFOA/l and of 0.1 µg PFNA/l. The reagent blank values were usually below 0.4 and 0.03 µg/l, respectively. The method was validated for concentrations from 1 to 100 µg/l. Comparative measurements by ESI-LC-MSMS and NCI-GCMS yielded highly corresponding results . In children, PFOA and PFNA were found in 95 % and 85 % of the analyzed samples. PFOA plasma concentrations ranged from less than the LOQ to 18 µg/l (mean 5.2 µg/l; median 4.7 µg/l) and the respective PFNA concentrations from less than LOQ to 2,9 µg/l (mean 0.7 µg/l; median 0.6 µg/l). No gender- and age-dependend associations were observed. PFOA levels correlated only weakly with the concentrations of PFNA ($R^2 = 0.352$). Tentative reference values for PFOA and PFNA, derived from the 95th percentile, were 11 and 1.8 µg/l, respectively.

The new GCMS method for the analysis of PFC is highly selective and resistant to interferences. Moreover, the cross-validation results obtained from the PFOA analyses confirm the accuracy of the method. The finding that PFOA and PFNA can be quantified in almost all children plasma samples indicates that the method is suitable for the monitoring of environmental exposure to PFC. The tentative reference values derived for PFOA and PFNA give an idea to which extent German children are exposed to PFC.

Perfluorinated alkyl substances in plasma, tissues and eggs of glaucous gulls (Larus hyperboreus) from the Norwegian Arctic

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Recent environmental surveys have depicted the widespread occurrence of perfluorinated alkyl substances (PFASs) in tissues of wildlife inhabiting various regions of the Arctic. In the present study, we investigated the distribution of a suite of PFASs in plasma, liver, brain, and egg samples from a scavenger-predator seabird species breeding in Svalbard (Arctic Norway), the glaucous gull (*Larus hyperboreus*). Chemical analyses of glaucous gull samples were carried out by two laboratories using different extraction, cleanup, and instrumental quantification methods, i.e. using high-performance liquid chromatographs (HPLC) with electrospray ionization in the negative ion mode (ESI) tandem mass spectrometer (MS) (HPLC/MS/MS ESI) or time-of-flight MS (HPLC/ToF-MS ESI). An inter-laboratory test based on the determination of a suite of PFASs in glaucous gull liver samples demonstrated moderate analytical variation (range 6 - 77%), thus validating the quantitative comparisons of PFASs between matrices. Quality assurance and quality control included laboratory blanks, matrix spikes, and standard material injections for each block of 5 – 10 samples to monitor changes in instrument sensitivity and effects of matrix on ESI suppression/enhancement. The perfluorooctane sulfonate (PFOS) was consistently the most frequent PFAS detected in the samples, and at concentrations that are thus far the highest reported in any arctic seabird species. Among the body compartment/tissue samples analyzed, PFOS levels in plasma (range: 48.1 - 349 ng/g wet wt) was highest, followed by liver ≈ egg > brain. Other perfluorosulfonic acids, i.e. perfluorobutane and perfluorohexane sulfonate (PFBS and PFHxS, respectively), and perfluorooctane sulfonamide (PFOSA) and four fluorotelomer acids (8:2 FTA, 8:2 FTUA, 10:2 FTA, and 10:2 FTUA), were either very minor or not detected in samples. The perfluorocarboxylic acids (PFCAs) with 9 to 14 carbon-chain length were found in samples (sum (∑) PFCA range in plasma: 41.8 – 262 ng/g wet wt) whereas a minority or no samples contained detectable amounts of 5- to 8C-, and 15C-PFCAs. The accumulation profiles of PFCAs were characterized by high proportions of long- and odd-chain length compounds, namely perfluoroundecanoic (PFUnA) and perfluorotridecanoic (PFTriA) acids, although their individual contribution to ∑ PFCA differed largely among the body compartments/tissues. The ∑ PFCA-to-PFOS concentration ratios in samples revealed plasma, in comparison with tissues and eggs, retained substantially higher burdens of ∑ PFCA proportionally to PFOS, suggesting body compartment/tissue-specific carrier protein affinity may play a primary role in the bioaccumulation dynamics of PFCAs versus PFOS in glaucous gulls. Current concentrations of PFOS and other accumulated fluorochemicals in this vulnerable top-predator from the Norwegian arctic marine food web are predicted to behave as part of a broad organohalogen contaminant cocktail with potential for mediating biological processes. Further studies are warranted to assess the toxicological potential of these persistent substances in adult glaucous gulls and developing embryos.

Exposure to perfluorinated acids in 108 Swedish women in relation to methylmercury and fish consumption

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Perfluorinated acids have been found in human blood samples worldwide. They have a long half-life in humans, and have shown a potential for reproductory interference and carcinogenity in animal experiments.To reduce human exposure to potentially hazardous chemicals it is important to identify the main sources of exposure. But despite their ubiquitous presence little is known on exposure to perfluorinated acids in the general population. Food is one possible source of exposure and in wildlife the highest concentrations of perfluorinated acids can be found in piscivouros animals. This study aims at describing the exposure level of perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) in Swedish women in relation to their level of fish consumption.

Women with a moderate to high proportion of fish in their diet were recruited for the study. The volunteers answered a detailed questionnaire regarding their diet, in particular their fish intake, and provided blood samples for analysis. The samples were analyzed for PFOS and PFOA using HPLC coupled to ESI-MS/MS. The samples were also analysed for methylmercury (MeHg), using an automated multiple injection analysis system and CV-AFS. The concentrations found were evaluated in relation to the volunteers' stated fish intake. A total of 108 women participated in the study.

The mean concentration of PFOS in the blood samples was 18 ng/ml (sd=11), and mean PFOA concentration was 2.0 ng/ml (sd=0.8). No correlation between concentrations of PFOS or PFOA and total stated fish intake was found in the study. However PFOS (r_s =0.27; p=0.004), and PFOA (r_s =0.22; p=0.02) were significantly positively correlated to MeHg in blood, and to increasing intake of fish species that generally contain high levels of MeHg (r_s=0.24; p=0.01), such as pike, perch and pikeperch. PFOS was also correlated to intake of shellfish (r_s=0.25; p=0.01). Concentrations of PFOS and PFOA were not correlated to intake of fat fish, such as salmon and herring. No correlation was found to other types of food contained in the survey.

The results indicate that predatory fish species can be a source of exposure to perfluorinated acids. The weak correlations, however, suggest that other sources also are of importance for human exposure to perfluorinated acids. The lack of correlation between PFOS or PFOA and the intake of fat fish, which generally contain high concentrations of organic contaminants, also implies that available risk models developed for organic contaminants may not apply to perfluorinated acids.

Determination of airborne polyfluorinated organic compounds in northern Germany

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Polyfluorinated organic compounds (PFCs) represent a diverse class of chemicals which has been produced in large amounts since the 1950ies. Due to their unique properties to repel both water and oil, PFCs are used in various products like spray-on treatments for clothing, leather, carpets and upholstery, non-stick cooking pans, and food wraps. PFCs combine persistence, bioaccumulation and toxicity to an extraordinary degree.

The ultimate breakdown products of PFCs include perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). Both substances are weakly volatile and moderately water-soluble, making them poor candidates for long-range atmospheric transport (LRAT). Nevertheless, they have been determined in species from remote areas like Alaskan polar bears. Perfluorooctane sulfonamides and fluorotelomer alcohols (FTOHs) are possible precursors of PFOS and PFOA, being neutral and volatile, therefore having the potential for LRAT.

An analytical method for those volatile precursors using high-volume air samplers and determination by GC-CI-MS has been developed, based on [1,2], in order to investigate the atmospheric occurrence of PFCs. A full method validation has been performed covering the evaluation of the calibration procedure, within-day and between-day precision, solvent and column blanks, the application of adequate internal standards, the determination of limits of detection (LODs) and limits of quantification (LOQs) and several recovery experiments. Based on this, results from metropolitan Hamburg and Waldhof, a rural location in Northern Germany, will be presented.

The analytes determined in this study were 4:2 FTOH, 6:2 FTOH, 8:2 FTOH, 10:2 FTOH, 6:2 Perfluorooctane acrylate (6:2 PFOAc), NMeFOSA, NEtFOSA, NMeFOSE, NEtFOSE and PFOSA. Five mass-labelled IS have been applied: 6:2 FTOH M+4, 8:2 FTOH M+4, 10:2 FTOH M+4, NMeFOSA M+3 and NEtFOSA M+5. The recovery IS (RIS) added prior to GC-MS analyses were 7:1 and 11:1 fluorinated alcohol.

For most analytes, no blank problems were found. Solvent blanks were not detected at all, while column blanks could be detected for 10:2 FTOH and NEtFOSA (<LOQ). Only for 8:2 FTOH, a column blank could be determined: 2.6 ± 0.5 pg/µL.Within-day (100 pg/µL) and between-day precision (200 pg/µL) ranged from 4.2% (4:2 FTOH) to 7.4% (10:2 FTOH) and 5.5% (NMeFOSE) to 9.7% (PFOSA), respectively. Several recovery experiments at two concentration levels (100 and 400 pg/µL) showed IS-corrected solvent recoveries between 44% (PFOSA) and 164% (NMeFOSE). IS-corrected column recoveries ranged from 56% (4:2 FTOH) to 151% (NMeFOSA). Only for two analytes where mass-labelled analogues were not available (NMeFOSE, NEtFOSE), column recoveries were very high: 311-319%.Instrumental LODs and LOQs (PCI) were estimated from standard analyses at very low concentrations at a signal-to-noise-ratio (S/N) of 3:1 and 10:1, respectively. Typical LODs were 0.2 (NEtFOSA, NMeFOSA) to 1.1 pg (PFOSA) while LOQs were 0.6 (NEtFOSA) to 3.7 pg (PFOSA).

Air samples of 900-1500 m^3 have been taken in parallel in metropolitan Hamburg (n = 14) and a rural area (Waldhof, n = 8) in Northern Germany. Results will be shown and discussed.

References: [1] J.W. Martin et al. (2002): Anal. Chem., 74, 584-590, [2] N.L. Stock et al. (2004): Environ. Sci. Technol., 38 (4), 991-996.

Mass spectral study of linear perfluorooctanesulfonamide

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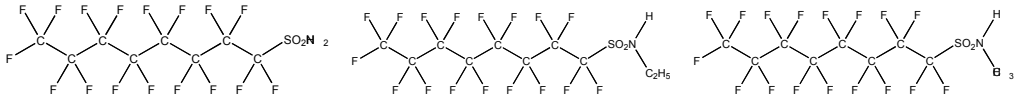
The objective of this work was to examine carefully the mass spectrum of linear FOSA in an attempt to elucidate the various possible fragmentation pathways accessible to this molecule under electron impact (EI) conditions. Perfluorooctane-1-sulfonamide (1) was prepared free of all branched isomers. The N-alkylated perfluorooctane-1-sulfonamides (2-3) were produced from 1 via alkylation at nitrogen.

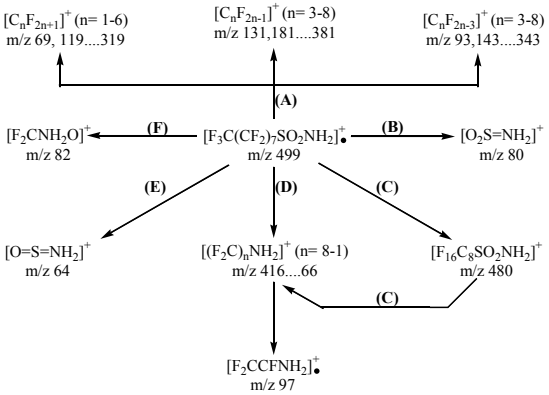


FOSA (1) NEtFOSA (2) NMeFOSA (3)

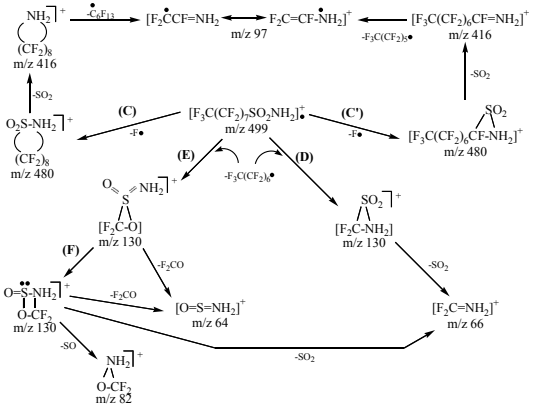
High Resolution Gas Chromatography/High Resolution Mass Spectrometry (HRGC/HRMS) experiments were conducted on an Agilent 6890N coupled to a Waters Autospec Ultima (HRMS). The GC column used was a J&W DB-FFAP (30 m x 0.25 mm id x 0.25 μm film). The injector, transfer line and source temperature were maintained at 230 °C.

Some of the fragments identified in the mass spectrum of linear FOSA, using 50 eV ionization, are summarized in Scheme 1. The molecular ion (m/z 499) is not detected (a weak signal, at m/z 500, corresponding to $[M+H]^+$, can be seen), while the observed fragments are of two types. After cleavage, the positive charge may reside either on a purely fluorocarbon residue (Scheme 1, pathway A) or on a nitrogen-containing fragment (Scheme 1, pathway B). The latter gives $[O_2SNH_2]^+$, m/z 80, the base peak in the spectrum. The results of our investigations on FOSA and its N-methyl and N-ethyl derivatives allow us to conclude that the fragments at m/z 416, 97 and 64 are mainly, if not exclusively, the nitrogen-containing species $[F_{16}C_8NH_2]^+$, $[F_2CCFNH_2]^+$ (Scheme 1, pathway C) and $[OSNH_2]^+$ (Scheme 1, pathway E), respectively. The spectrum in Figure 1 shows two additional relatively strong peaks at m/z 82 and 66. The first of these we ascribe to the fragment $[F_2CNH_2O]^+$ (Scheme 1, pathway F) since N-alkyl sulfonamides show a strong signal corresponding to the appropriate $[F_2C(NRR')O]^+$ ion. The second, at m/z 66, appears to belong to a series of fragments differing in mass by 50 amu (CF_2), and terminating with the ion m/z 416 mentioned above (Scheme 1, pathway D). In earlier work (D.W.Kuehl and B.Rozynov, Rapid Comm. Mass Spec., 2003. 17, 2364), a cyclic structure with a ten-membered ring was proposed for the ion m/z 480 (Scheme 2, pathway C). However, preferred formation of such a thermodynamically unfavorable ring seems highly unlikely. A possible alternative route to the m/z 97 fragment is outlined is Scheme 2, pathway C', involving a kinetically favorable three-membered ring species having a sultam structure. Three-membered ring intermediates may also be suggested (see Scheme 2) to rationalize the formation of three other major fragments at the low-mass end of the mass spectrum of linear FOSA. We have also considered other possible pathways to explain additional observed signals. Initial formation of 3-to-6-membered rings would be

expected to be more favourable than closure to larger ring sizes and would explain the formation of most fragments found in the mass spectrum of linear FOSA.



Scheme 1



Scheme 2

ANA005 Arsenault

Separation and fluorine nuclear magnetic resonance spectroscopy (^{19}F-NMR) analysis of the individual branched isomers present in technical perfluorooctanesulfonic acid (PFOS)

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The production of perfluoroalkylsulfonate derivatives via electrochemical fluorination is not a clean process but rather gives a complex mixture. The objective of this work was to isolate individual isomers present in technical perfluorooctanesulfonyl fluoride (PFOF) and characterize their structures by ^{19}F NMR. As a result, the quantification of the individual internal CF_3 branched isomers present in technical perfluorooctanesulfonic acid (PFOS) proved possible, an outcome apparently not feasible in earlier NMR studies.

A commercial sample of PFOF was derivatized to secondary sulfonamides (PFOSamide) using benzylamine and the resulting mixture separated by a combination of crystallisation and preparative-scale HPLC. A set of six fractions was obtained, each containing a different isomer as the major component accompanied by smaller amounts of other isomers. The ^{19}F-NMR spectra of these PFOSamide isomers were run on a 400 MHz Bruker instrument using d_4-methanol as solvent and hexafluorobenzene as an internal standard (set at -169ppm).

Structures of the various FOSA isomers analyzed by ^{19}F-NMR are shown in Scheme 1. Only isomers **1-7** were isolated and characterized by ^{19}F-NMR (see Table 1).

Scheme 1: Structures of the 7 major PFOSamide isomers (* signifies that the signal exist a clearly resolved an AB quartet; R=benzyl group)

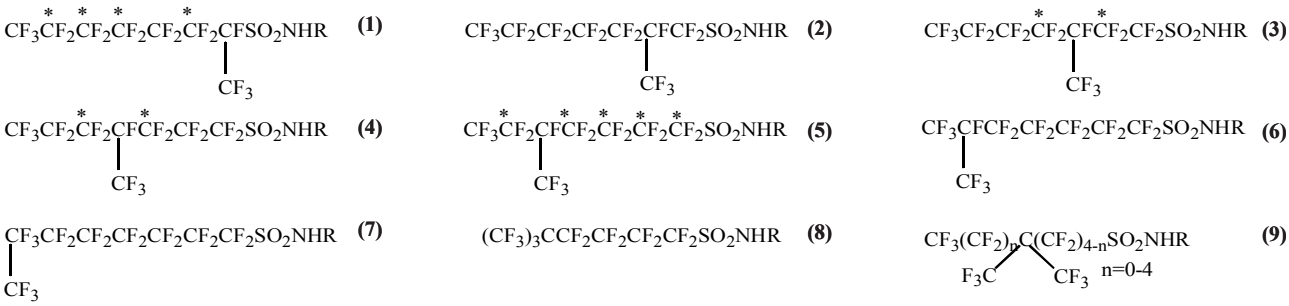

Table 1: ^{19}F-NMR[a] of the of the 7 major isomers separated from in a technical mixture of PFOS

Compound[b]	C-1	C-2	C-3	C-4	C-5	C-6	C-7	Branched CF_3
1	-172.31	-118.02[c] -117.27	-124.37	-126.38[c] -126.08	-127.52[c] -127.08	-131.12[c] -130.68	-85.99	-75.21
2	-107.98[c] -107.42	-186.81	-116.38	-124.30	-127.03	-130.81	-85.98	-74.75
3	-116.13	-115.30[c] -114.63	-189.68	-107.03[c] -116.10	-125.19	-130.62	-85.97	-75.13
4	-117.63	-123.02	-116.68[c] -116.07	-190.03	-117.57[c] -117.07	-128.87	-85.77	-75.12
5	-117.30[c] -117.53	-124.32[c] -124.51	-124.44[c] -124.12	-117.42[c] -117.11	-189.78	-121.26[c] -120.65	-85.13	-75.50
6	-117.85	-125.03	-125.90	-125.26	-119.54	-190.74	-76.90	-76.90
7 (linear)	-117.84	-125.08	-126.31	-126.41	-126.58	-127.40	-130.95	-86.02

a Hexafluorobenzene was used as an internal standard (set at -169ppm)

b The numbering of the carbon chain is as follows: C(7)-C(6)-C(5)-C(4)-C(3)-C(2)-C(1)-SO_2NH_2

c AB pattern observed due to chirality in the structure

^{19}F-NMR analysis of technical PFOS and integration of specific isolated signals (**1**: C-1; **2**: branched CF3, C-1, C-2; **3**: C-6, C-3; **4**: C-7, C-2, C-6; **5**: branched CF3, C-7, C-6; **6**: C-7, C-3, C-6; **7**: C-8; **8**: t-butyl group at -66.4 ppm: and **9**: gem-dimethyl groups at -70.0 ppm) has permitted quantification of the following isomers: **1**: 1.9%; **2**: 1.9%; **3**: 5.0%; **4**: 4.8%; **5**: 6.2%; **6**: 10.8%; **7**: 68.9%; **8**: 0.2%; and **9**: 0.4%. The sum of these individual values matches closely that reported earlier using NMR, except here, we are able to give the individual percentage of each internal CF_3 branched isomer.

Separation and detection of structural isomers of perfluorooctanoate (PFOA) in biological matrices: Solid phase extraction, volatile analog derivatization, and analysis using automated thermal desorption gas chromatography/mass spectrometry (ATD/GC/MS)

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DeSilva and Mabury[1], from the University of Toronto, recently reported the use of a chemical derivatization method for the gas chromatography/mass spectrometry (GC/MS) separation and detection of structural isomers of PFOA and other perfluorocarboxylate anions (PFCAs) in polar bear liver extracts. The derivatization procedure uses 1,3-dicyclohexylcarbodiimide (DCC), a well-known "zero-length" carbodiimide cross-linking agent used extensively in peptide chemistry to convert the terminal carboxylic acid functional group of PFCAs to corresponding volatile 2,4-difluoroaniline (2,4-DFA) analogs easily analyzed by gas chromatography. An anaytical method for the qualitative analysis of PFOA isomer distributions in biological extracts will be presented. This method incorporates a simplified preparatory procedure of the same derivatization reaction, coupled with protein precipitation and solid phase extraction (SPE) techniques. Derivatized sample extracts are introduced onto the analytical GC column using an automated thermal desorption (ATD) unit. The ATD provides a means for performing large volume injections which improves method sensitivity as several microliters (20 μL) of extract can be successfully analyzed in a single injection. By using a low-temperature purge cylce, the extract solvent can be selectively removed from the spiked TD tube while the analytes of interest are retained. The TD tube then undergoes a two-stage thermal desorption procedure to remove the analytes from the sorbent material where they are then cryofocused onto a Tenax-TA cold trap held at -30ºC. The cold trap is rapidly heated to sweep the analytes onto the head of the GC column for analysis. Besides concentrating the analytes, the ATD unit also controls the carrier gas flow for the GC analysis. Improved flows through longer GC columns required for isomer separation aids in peak resolution. Excellent separation of nine different PFOA structural isomers with retention times *less than 25 minutes* will be shown.

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Historical and geographic aspects of perfluorooctanoate and perfluorooctane sulfonate in human serum in Japan

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Perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) have recently received attention with their widespread contamination. Although some reports have shown that human exposure to PFOA had reached a steady-state in the 1990s, human exposure levels of PFOA in residents in Northern Japan have still continued to increase from 90s to the millennium. To investigate a long-term time trend of exposure levels, we measured PFOS and PFOA concentrations in human serum samples collected historically in Kyoto and at various locations throughout Japan.We employed the extraction process developed by Hansen et al. [1] as reported previously [2]. Each extracted solution was analyzed by liquid chromatography-mass spectrometry (LC/MS) as previously reported[3]. The lowest limits of quantification (LOQ) (mg/L) were 0.1 for both analytes in the serum samples collected.

Historical samples [10 samples per each time point per each sex and 5 time points in total (N=100)] collected from people during 1983-1999 in Kyoto demonstrated that PFOA concentrations have steadily increased by factors of 4.4 (Geometric mean (GM)) (Geometric standard deviation (GSD)): 2.2 ng/ml (1.7) in 1983 to 9.7 ng /ml (1.7) in 1999); levels of exposure to PFOS have reached a plateau in the late 1980s ;14.2 ng/ml (1.5) in 1983 to 20.7 ng /ml (1.6) in 1999.

For geographical survey, 200 samples (97 males and 103 females) were collected from 10 locations in 2003. PFOS concentrations ranged from 0.2 (ng/mL) to 92.2 (ng/mL), and PFOA from 0.4 (ng/mL) to 52.2 (ng/mL). In male participants, the GM of serum level was 13.4 ng/mL (GSD, 1.9) for PFOS, 4.2 ng/mL (1.9) for PFOA. In female participants, GM of serum level was 8.3 ng/mL (2.1) for PFOS, 3.5 ng/mL (2.0) for PFOA. There are predominant regional differences for both PFOS and PFOA concentrations (ANOVA: $p<0.001$). The PFOS and PFOA levels in sera [GM (GSD)](ng/mL) in 2003-2004 ranged from 3.5(2.9) in Taiwa of Miyagi pref. to 27.8(1.6) in Kyoto, for PFOS and from 2.4(1.7) in Yokote of Akita pref. to 14.5(1.3) in Osaka for PFOA.

Exposure to PFOA was increasing steadily in Kyoto in Japan, as well as rural areas[2], in sharp contrast to the survey conducted in the U.S.A.[4]. However, exposure to PFOS had reached plateau levels in the late 1980s, being consistent with a study in U.S.A. [4]. Relatively high concentrations of PFOA in the Kinki district (Osaka and Kyoto) compared with other districts in Japan or in the U.S.A is very likely which suggests potential sources of contaminations with PFOA and PFOS in the Kinki district as indicated by heavy contamination of drinking water and air-borne dust[3].

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Perfluorinated compounds in archived house dust samples

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Perfluorinated compounds have come under recent scrutiny for their persistence, global distribution (environmental media, wildlife, humans) and animal toxicity. The majority of the scientific literature on PFCs suggests routes of exposure are largely unknown. Data gaps appear to include both indoor and outdoor sources. A study was under taken to develop methods to detect PFCs in house dust, and reduce uncertainty in indoor exposure. Archived EPA house dust samples (n=112) were sieved to 100 μm and analyzed for select PFCs. Telomer alcohols (6:2-FTOH, 8:2-FTOH, 10:2-FTOH) were analyzed by GC/MS after sonic extraction and SPE cleanup. Perfluorinated carboxylic acids (C6-C12) and sulfonates (PFOS, PFHS, and PFBS) were analyzed by LC/MS/MS. Recovery of spiked PFCs in house dust ranged from 39.5% (cv 0.10) for PFNA to 107.3% (cv 0.08) for PFUA. Mean concentrations were calculated for values above the lower limit of quantitation (LOQ). Mean concentrations for PFCs ranged from 0.357 μg.g (PFDA) to 11.23 μg/g (PFHS). Maximum concentrations ranged from 2.42 μg/g (6:2 FTOH) to 357.0 μg/g (PFHS). Data appear to be log-normally distributed with many samples at or below the limit of quantitation (LOQ) (~ 0.1 μg/g dust). PFOS, PFOA and 8:2 FTOH were below the LOQ in 5.4% , 3.6% and 46.4% of the samples respectively. Correlation coefficients suggest strong correlations between certain PFCs in house dust (α = 0.01) and lack of correlation for others.

Occurrence of perfluorinated organic acids in the water of the North Sea and Arctic North Atlantic

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In recent years, perfluorinated organic acids (PFC) and their derivatives have attracted large attention as a new group of environmental pollutants which are industrially produced in large quantities and have been proved to be very persistent in the environment. An analytical method consisting of SPE extraction, subsequent enrichment and HPLC-neg.-ESI-MS-MS has been developed to determine a number of perfluorinated organic acids in river, coastal and marine water samples. Compared to our first report in 2004, the limits of determination (S/N= 10) could be improved by a factor of ca. 10 and now range between 5 and 30 pg/L in seawater matrix. Extreme care has been taken for blank control and prevention of contamination. In particular, the perfluorinated carboxylic acids are susceptible to contamination, e.g., by Teflon^{R}, and Viton materials, while for the sulfonic acids less contamination risk was observed.

The investigations started in the estuary of the river Elbe – one of the most important sources for pollutants entering the German Bight (southern North Sea) - and continued to the open sea. Seven PFCs were determined in the German Bight in 2003; the highest concentrations were observed for PFOA (perfluoro octanoic acid) and PFOS (perfluorooctylsulfonic acid). At the Elbe estuary values of about 20 ng/L were encountered, while the other compounds (C_6 to C_{10} acids) range from 1 to 3 ng/L. Along the Elbe plume towards the north concentrations of the major compounds decrease to 3 to 6 ng/L (at a salinity of 28 to 30), while to the open sea they drop to 0.5 to 1.2 ng/L (salinity 34.4). In the southern German Bight (salinity: 32) a different pattern with higher PFOA but low PFOS was observed, indicating a different source. In August 2003 the investigation were extended into the whole North Sea. The distribution of PFOA was very similar to that of the survey in May 2003. However, the PFOS concentrations were lower this time. At most positions of the open sea PFOA was detected at concentrations of ca. 0.5 ng/L, all other target compounds were below the detection limits of that time. At the Norwegian coast, however, slightly higher values of ca. 1 ng/L were found which could be attributed to the outflow of Baltic Sea water (salinity 28 to 30).

In 2004, the investigations were expanded to the arctic North Atlantic. After having improved the limits of determination it was possible to detect PFOS and PFOA in all water samples at levels between 20 and 120 pg/L. The C_6 acids ranged between 10 and 40 pg/L.

The results revealed that PFCs are widely distributed in marine waters. Both, low level background concentrations in a remote area and a distinct gradient from sources from the European continent could be observed. The concentrations of the major occurring PFOA and PFOS determined on the cruises are within a similar range as other polar pollutants such as phenylurea, triazine or phenoxyacetic acid herbicides. They were detected well above classical contaminants like chlorinated hydrocarbons (HCB, DDT group, PCB).

Blood levels of perfluorooctane sulfonate (PFOS) and its dietary and non-dietary determinants in middle-aged Japanese men from 10 regions nationwide

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Perfluorooctane sulfonate (PFOS) is used in industry as a surfactant. PFOS and its related fluorinated organic compounds have been used since the 1950s as stain-resistant coatings for fabrics, carpet, leather and paper products. Recent studies have indicated that human exposure to PFOS has increased on a global scale. Therefore, we conducted this study with the aim of determining human exposure to fluorinated organic compounds, such as PFOS, perfluorooctanoate (PFOA) and perfluorooctanesulfonylamide (PFOSA), using male human blood samples (n = 272) from validation study participants of a self-administered food frequency questionnaire (FFQ) from 1994 through 1997 in 10 regions in Japan. The serum samples were analyzed by column-switching liquid chromatography - electrospray mass spectrometry (CS-LC-MS). The concentrations of PFOS, PFOA and PFOSA were measured and related to age, residential area, BMI, smoking and diet.

An acetonitrile spiked internal standard solution was added to a blood sample. After removing protein by centrifugation (3000 rpm for 10 min), the supernatant was subjected to CS-LC-MS. A Waters OASIS-HLB (2.1 x 20 mm, 25 μm) was used as the extraction column, and an Agilent 1100 series LC/MSD-SL was used for the analysis. PFOS, PFOA and PFOSA were monitored in the selective ion mode (SIM), and the monitored ions were assumed to be m/z 499, 369 and 498, respectively, in the negative ion mode. The limits of quantitation (S/N > 10) of PFOS, PFOA and PFOSA in human blood were 0.5, 0.5 and 1.0 ng ml^{-1}, respectively. The method enables the precise determination of standards and can be applied to the detection of PFOS, PFOA and PFOSA in human blood samples.

PFOS was detected from all the samples at concentrations between 2.1 and 271.1 ng ml^{-1}, and PFOA was detected from 91 of the 272 samples at concentrations between < 0.5 and 10.2 ng ml^{-1}. By contrast, no PFOSA was detected in any of the samples. Their concentrations increased with age and differed according to residential area, particularly for PFOS. However, they were not associated with BMI or fish intake, in contrast to dioxin. Further investigation of the source of PFOS exposure is vital to determining human exposure to it.

Differentiation of important perfluorooctane sulfonate isomers by tandem mass spectrometry

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Perfluorooctane sulfonate (PFOS), one perfluorinated compound (PFCs) has become an important contaminant. PFCs are mainly used as coating reagent. PFC main production process yields the formation of specific impurities besides the main linear compound. Therefore, the origin of PFCs in biota should be identifiable by analysing the impurity pattern.

This study aimed the first mass spectrometric characterisation of some purified PFOS isomers expected to be present as by-products in commercial PFOS solutions. High performance liquid chromatography (HPLC) combined with a triple quadrupole (TQ) mass spectrometer (MS) was applied to analyse linear PFOS (L-PFOS), perfluoroisopropyl (*iso*-PFOS), 5-perfluoromethyl (5m-PFOS), 4-perfluoromethyl (4m-PFOS), 3-perfluoromethyl (3m-PFOS), 2-perfluoromethyl (2m-PFOS) and α-perfluoromethyl (α-PFOS) branched isomers.

Methanol (≥99.8%) was delivered by SDS (France) and ammonium acetate (98.0%) by Merck (Germany). The PFOS isomers were provided by Wellington Laboratories (Canada) after derivatisation and separation steps of a commercial sample of PFOS.

A phenyl perfluorinated (PFP) (Fluophase, Thermo Electron, 150 x 2.1 mm, 5 μm particles size, 100 Å pore size) and a X-Terra C18 phases (Waters, 100 x 3.0 mm i.d., 5 μm particles size, 125 Å pore size) were employed. A water/methanol gradient was applied with 4 mM ammonium acetate. The flow rate was 200 μl/min.

ESI in the negative ion mode was used. The TQ experiments were conducted with a 1200L mass spectrometer (Varian, USA) applying the following parameters: Drying gas flow 137 kPa, temperature 200 °C, spray voltage 4.0 kV, nebulizing gas flow 413 kPa, heated capillary voltage -45 V. Argon was the collision gas (collision cell pressure of 0.27 Pa). Tandem MS experiments were conducted on the molecular ions (*m/z* 499) and the spectra were recorded in full scan mode.

HPLC-ESI(-)-TQ-MS^2 experiments with a PFP and a C18 phases allowed to characterise the seven PFOS isomers. Figure 1 shows the spectra of the pure isomers. The 9-series (*m/z* 119 to *m/z* 419) and the 0-series (*m/z* 130 to *m/z* 430) observed were typical for PFOS (Figure 1). 50 mass units (CF_2) separated the ions of each series. Additionally, the fluorosulfonate (*m/z* 99) and the sulfonate (*m/z* 80) groups were observed. The MS^2 spectrum of L-PFOS (Figure 1a) mainly contained these both fragments. Suppression of one ion in the 0-series allowed the determination of the CF_3 position along the chain. An arrow in Figure 1 marked the missing masses. The MS^2 spectrum of α-PFOS (Figure 1g), did not show any 0-series fragmentation.

This study will allow the identification of these isomers in biota and demonstrates that systematic errors are made when PFOS is quantified with the usually fragmentation m/z 499 → 99^2. PFOS isomers as pure standards would be required for a proper PFOS quantification.

Dervitization of perflurooctane for analysis by gas chromatography coupled with mass spectrometry

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Perfluorooctane sulfonate contaminant (PFOS) has been proven to be important key in the distribution of the fluorinated chemicals in the environment. Due its ionic properties, PFOS is not suitable for the analysis by gas chromatography (GC). Thus, high performance liquid chromatography coupled with mass spectrometry (MS) is the method of choice for its quantification.The aim of this study was the development of the first derivatisation procedure for the PFOS analysis by EI-GC-MS. Structure of the derivatives were confirmed by employing a labelled derivatisation reagent. PFOS was purchased as potassium salt from ABCR (Germany). Cyclohexane (99.8 %) was delivered by Scharlau (Spain), sulfuric acid (95-98 %) by J.T Baker (The Netherlands) and iso-propanol by Biosolve Ltd. (The Netherlands). The iso-propyl-1,1,1,3,3,3-d6 alcohol (99.8 atom %) was purchased from CDN Isotops (Canada). Separation was performed on a gas chromatograph equipped with a split/splitless injector and with a capillary column HP-5MS (15 m length, 0.25 mm i.d, 0.25 mm film thickness). 1 µl of the sample was injected in the split mode (1:10). The injector temperature was 220 °C. Helium (99.999 %, Carbagas, Switzerland) was the carrier gas. The temperature program was 40 °C isothermal for 8 min, then 5 °C/min to 130 °C, 30 °C/min to 220 °C, and isothermal for 5 min. A MD800 quadrupole instrument (Fison instrument, United Kingdom) was employed in the electron ionisation (EI) mode (70 eV). Compounds were detected in the full scan mode (*m/z* 60-600).

The derivatisation procedure was qualitatively performed with 3 mg of PFOS. 1ml of iso-propanol and 20 µl of sulfuric acid were added and the mixture was shaken over night. 800 µl of cyclohexane and 200 µl of purified water were added. The mixture was shaken for 15 min. 1 µl of the cyclohexane layer containing the derivatised PFOS (deriv-PFOS) sample was analysed. The same procedure was performed with labelled iso-propanol.

The molecular ions ($(C_8F_{17}SO_3CH(CH_3)_2)$, *m/z* 542) were not detected in the EI-MS spectrum of deriv-PFOS. However, the typical fragments of the perfluorinated compounds were observed. These were the fragments *m/z* 69, 119, 169… and *m/z* 131, 181, 231… (50 mass units separated each fragment). The higher masses were of interest for structure elucidation. The fragments *m/z* 527 ($[M\text{-}CH_3]^+$), *m/z* 483 ($[M\text{-}OCH(CH_3)_2]^+$) and *m/z* 463 ($[M\text{-}CH_3\text{-}SO_2]^+$) were detected. The proposed formulas were confirmed by performing the derivatisation with labelled iso-propanol. The corresponding masses *m/z* 530 $[M\text{-}CD_3]^+$ and 466 $[M\text{-}CD_3\text{-}SO_2]^+$ were observed with shift of 3 mass units. The fragment *m/z* 483 corresponding to the loss of the iso-propyl group did not provide any mass shift. Additionally, the molecular ions *m/z* 549 ($[M+H]^+$) with a mass shift of 6 units were observed. Furthermore, this procedure could be also applied to the perfluorodecanoic acid.

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Tissue distribution of perfluorinated alkyl acids in bottlenose dolphins (Tursiops truncatus)

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Perfluorinated alkyl acids (PFAs) are ubiquitous contaminants that have been detected in abiotic compartments as well as in humans and wildlife. Recently, high PFA concentrations have been detected in plasma of free-ranging bottlenose dolphins from the southeastern US waters. The main objective of this study is to understand the kinetics of perfluorinated compounds and their elimination in small cetaceans. Plasma and urine of dolphins were collected in 2003 during live captures in Sarasota Bay, Florida, USA and Charleston, South Carolina, USA. Tissues (e.g. liver, kidney, thyroid, thymus, muscle, heart, lungs) were also collected from two dead dolphins from the same locations. An ion-pairing method was used to extract perfluorinated compounds from the matrices. Compounds of interest included perfluorinated carboxylic acids (molecules including 8 to 15 carbons), sulfonic acids (6 and 8 carbons) and the neutral precursor perfluorooctane sulfonamide (PFOSA) which can be degraded to perfluorooctane sulfonate (PFOS) under natural conditions. All analyses were conducted using LC-MS/MS. PFAs were detected in all samples analyzed from both locations. Higher PFA concentrations were detected in plasma and tissue of dolphins from the US east coast compared to the Gulf of Mexico. Concentrations were higher in liver and plasma (PFOS plasma concentration range: 470-3070 ng/g w.w. in Charleston dolphins and 140-1740 ng/g w.w. in Sarasota dolphins) compared to other tissues. High PFA concentrations were also detected in the thyroid, thymus and lungs of dolphins examined. Our results show that PFA distribution in tissues varied by geographical locations. In addition, the pattern of PFA contamination varied by tissue. These results suggest that dolphins from the two locations may be exposed, at different degrees, to different sources of PFAs. Finally, PFOS was detected in urine of dolphins (mean concentration at Charleston: 3.8 ng/g w.w., mean concentration at Sarasota Bay: 23.3 ng/g w.w.). Dolphins excrete large volume of urine daily (up to 4 L) suggesting that the urinary system might be an important route for PFA elimination in this species.

Analysis of fluorinated alkyl compounds in air samples from England

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In recent years, perfluorooctane sulfonate (PFOS) and perfluorinated carboxylic acids (PFCAs) have been detected in high-trophic biota from as remote places as the Arctic [1]. These compounds are ionic and possess very low volatility. The question arose, how they were transported from densely populated application areas to the Arctic. Ellis et al. [2] proposed that neutral precursor compounds could undergo atmospheric long-range transport and finally be degraded to persistent products in the remote location. Possible precursor compounds for PFCAs and PFOS are fluorotelomer alcohols (FTOHs) and fluorooctane sulfonamides/sulfonamido ethanols (FOSAs/FOSEs), respectively. The aim of this study was to provide first evidence for the presence of FTOHs, FOSAs and FOSEs in European air samples.

A sampling and analysis method developed by Martin et al. [3] was adapted. It is based on high-volume sampling of particles on a glass fiber filter (GFF) and airborne fluorinated compounds on polyurethane foams (PUFs) and XAD-2 resin. Ionic compounds were extracted from the filter with methanol and analyzed by liquid chromatography coupled to time-of-flight mass spectrometry (LC/TOF-MS) [4]. For neutral compound analysis, the filter or PUFs/XAD were extracted with ethyl acetate and the extract was analyzed by gas chromatography with negative and positive chemical ionization MS detection (GC/NCI- and PCI-MS). The instrumental GC-MS method was validated with standard solutions of unknown concentrations in an inter-laboratory comparison study. For all analyses, quantification was based on the internal standard (ISTD) method, applying 3,7-dimethyl-PFOA and 7:1 FTOH as ISTD for ionic and neutral compounds, respectively. Field blanks and samples of about 1400 m^3 air were taken in Manchester (England, urban site) and Hazelrigg (40 miles north-west of Manchester, semi-rural site).

More than 80 % of all reported values in the inter-lab comparison study of the GC-MS methods were within ±30 % of the theoretical values. However, some results revealed deviations from theoretical values of up to 180 % (95 % for the here presented method). In view of the analytical challenges posed by polyfluorinated compounds and the low concentrations in the test mixtures (20 – 900 pg/µL), these results were satisfactory.

All analyzed volatile compounds were detected in air phase samples from both locations as well as in the particle phase of the Manchester samples. The urban samples were higher contaminated than the semi-rural. The highest concentrated volatile compound was 8:2 FTOH with up to 326 pg/m^3 air in a Manchester sample (Hazelrigg 196 pg/m^3), followed by 6:2 FTOH (315 and 147 pg/m^3, respectively). Up to 6 pg/m^3 of 8:2 FTOH was also found in the particle phase. Of the FOSAs and FOSEs, N-methyl-FOSE showed the highest levels with up to 29 pg/m^3 in the air phase and 33 pg/m^3 in the particle phase. Among the ionic compounds in the particle phase, PFOA was highest concentrated with levels up to 828 pg/m^3. Surprisingly, this makes PFOA the highest concentrated of all fluorinated analytes in air samples. It was followed by PFOS (51 pg/m^3), PFHpA and PFDcA (both 14 pg/m^3) and TH-PFOS (10 pg/m^3). PFBS, PFHxS and PFDcS were additionally detected.

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Perfluorooctane sulfonate (PFOS) and related compounds in a Norwegian Arctic marine food chain

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In this study we report concentrations and species-specific biomagnification potential of perfluoroalkylated substances (PFAS) in a marine food chain from the Norwegian Arctic. The species investigated, i.e. glaucous gull (*Larus hyperboreus*), black guillemot (*Cepphus grylle*), polar cod (*Boreogadus saida*) and ice amphipod (*Gammarus wilkitzkii*), represented three trophic levels of the food chain and were collected from the western Barents Sea marginal ice zone. Liver samples from birds and fish, as well as whole body homogenates of amphipods, were analyzed for seven PFAS by application of a screening method based on liquid chromatography and high resolution mass spectrometry.

Long-chain and less polar PFAS were not included due to low levels in Arctic samples and high method detection limits. Of the PFAS determined in the samples, perfluorooctane sulfonate (PFOS) was found at highest levels, and was the only fluoroorganic compound detected in all four species. Mean concentrations of PFOS increased in the order polar cod (2.02 ng g^{-1} wet wt) < ice amphipod (3.85 ng g^{-1} wet wt) < black guillemot (13.5 ng g^{-1} wet wt) < glaucous gull (65.8 ng g^{-1} wet wt). Levels of PFOS in all species, except for glaucous gull, were comparable to those reported from the Canadian Arctic and Greenland. The levels of PFOS found in glaucous gull are the highest reported thus far in any Arctic seabird liver. Additionally, perfluorohexane sulfonate (PFHxS), perfluorohexanoic acid (PFHxA), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDcA) and tetrahydro perfluorooctane sulfonic acid (TH-PFOS) were detected in at least two of the species studied. Of these analytes, the highest concentration was found for PFOA in black guillemot (17.1 ng g^{-1} wet wt).

Trophic levels and food chain transfer of perfluorinated pollutants were investigated using the ratio of stable isotopes of nitrogen, ∂N^{15} ($^{15}N/^{14}N$). No correlation was found between PFOS concentrations and trophic level within species. Nevertheless, a significant nonlinear relationship was established when the entire food chain was analyzed.

PFAS levels were compared to concentrations of polychlorinated biphenyls (PCBs), dichloro-diphenyl-trichloroethanes (DDTs) and polybrominated diphenyl ethers (PBDEs) from the food chain. Multivariate analysis showed that 67.2% of total variance in concentrations of PFAS, PCBs, DDTs and PBDEs in the liver samples, could be explained by trophic level. There were positive significant correlations between these four contaminant groups and trophic levels.

Biomagnification factors (BMFs) based on predator-prey analyte concentrations showed that PFOS had the highest biomagnification propensity of the detected PFAS. BMFs for PFOS were comparable to lipid-normalized BMFs of PCB-28, PCB-52, PCB-101, *o,p*-DDT and PBDE-28.

$\sum PFAS_7$ concentrations in glaucous gull, black guillemot and polar cod, showed no or minor correlation with $\sum PCB_{13}$, $\sum DDT_5$ and $\sum PBDE_{10}$; most likely due to differences in compound-specific physiochemical properties such as lipophilicity and binding affinity to proteins. However, $\sum PCB_{13}$, $\sum DDT_5$ and $\sum PBDE_{10}$ concentrations were positively correlated with each other. Present results suggest that PFOS has a potential for biomagnification in species of the Norwegian Arctic marine food chain, and that trophic transfer of PFAS is comparable to that of PCBs, DDTs and PBDEs.

Determination of perfluorooctanesulfonic acid in human serum and semen samples by liquid chromatography-electrospray mass spectrometry

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There is increasing interest in the relationship between impaired human male reproductive health and environmental factors. As human male reproductive tract is a complex process, a variety of sites may be affected by exogenous noxious substances. Lifestyle factors as well as environmental and industrial agents may impair male reproductive health. Many studies have been published on reproductive dysfunction in male animals and humans. Thus, we focused on perfluorooctanesulfonic acid (PFOS), a new environmental pollutant, and evaluated semen quality in relation to PFOS concentrations in blood and semen samples from healthy Japanese male volunteers.

Blood and semen samples were collected from fifty male university students aged 18-24 after providing informed consent and approval of the local ethics committee. Semen samples were analyzed according to WHO 1992 guidelines. Liquid chromatography-electrospray mass spectrometry (LC/MS) of PFOS was performed using an Agilent 1100 MSD-SL system. Separation was achieved on an Inertsil C_8-3 column (2.1 x 100 mm, 5 μm, GL Sciences Inc., Tokyo, Japan) that was equipped with a Mightysil RP-18 GP pre-column (2.0 x 5 mm, 5 μm, Kanto Chemical Inc., Osaka, Japan).The fragmentor voltages were 220 V for PFOS, 130 V for PFOA (perfluorooctanoic acid) and 170 V for PFOSA (perfluorooctanesulfonamide). When working in the single ion monitoring (SIM) mode, the ions were determined as $[M-K]^-$, $[M-COOH]^-$ and $[M-H]^-$ for PFOS, PFOA and PFOSA, respectively. The column-switching LC/MS coupled with an on-line extraction system consisted of this LC/MS and another LC pump (Shimadzu LC-10ADvp pump, Shimadzu, Kyoto, Japan), and a Waters Oasis HLB extraction column (20 x 2.1 mm, 25 μm). After a 30 μl sample was injected by an auto sampler, it was loaded onto the extraction column by flowing water/methanol (90/10, v/v). The valve was switched 5 min after the sample injection. Then, the sample was eluted to the analytical column in the back-flashing mode from the extraction column, and the separated compounds were sequentially introduced into the MS. After 20 min, the switching valve was returned to its original position. The separation was carried out using a mobile phase of 1.0 mM ammonium acetate in water/acetonitrile (v/v). The calculated detection limits (LODs) of PFOS, PFOA and PFOSA were 0.05, 0.05 and 0.25 ng/ml, respectively. The calculated limits of quantification (LOQs) when S/N = 10 were 0.5 for PFOS, 0.5 for PFOA and 1.0 ng/ml for PFOSA. The average recoveries of PFOS, PFOA and PFOSA were above 80% (RSD<10%, n = 6) in the serum and semen samples.

Residual PFOS, PFOA and PFOSA in human serum and semen samples were determined by column-switching liquid chromatography-electrospray mass spectrometry. PFOS was detected in all the serum samples, whereas both PFOA and PFOSA were not always detected in the samples. When PFOS concentration was related to hormone concentration (LH, FSH and Inhibin B) in serum, no significant correlation was observed between them. Moreover, when PFOS concentration in semen was related to sperm motility and sperm concentration, no significant correlation was observed between them as well.

Separation and determination of perfluorinated carboxylic acids using liquid chromatography with fluorescence detection

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Perfluorinated carboxylic acids (PFCAs) constitute an important group of anthropogenic perfluoroalkyl substances that have been detected in the environment and in various organisms including humans. Widespread presence of PFCAs in the environment requires reliable and possibly inexpensive methods for their determinationThe most commonly used methods for determination of PFCAs are based on high-performance liquid-chromatography (HPLC) combined with mass spectrometry (MS) detection, employed both for environmental and biological samples. In spite of outstanding selectivity and sensitivity, these methods require expensive MS instrumentation with skilled operators, suffer from blank problems; often additional sample clean-up and/or preconcentration of analytes is necessary. Recently, we have developed methods based on capillary electrophoresis separations with either direct or indirect UV detection, exhibiting good selectivity for PFCA analysis ranging from C6 to C12 [1].

This work was focused on optimization of reversed-phase-HPLC method for determination of PFCAs ranging from C2 to C12 carbon atoms in the PFCA molecule, using fluorimetric detection. As the PFCAs do not exhibit native fluorescence, they have been derivatized with commercially available fluorogenic reagent 3-(bromoacetyl)cumarin (BAC). Several factors affecting yield of derivatization such as concentration of BAC, temperature, time of reaction, and effect of different solvents have been optimized. Derivatization, at optimized conditions, was carried using 1:1 mixture of sample solution containing PFCAs in acetonitrile (ACN) with 0.2 % BAC solution in acetone and such a mixture was incubated for 25 min at 70˚C. The optimized HPLC measurements utilized a gradient elution with linear change of ACN content in the eluent from 65 to 95%. The fluorimetric detection was carried out at wavelengths 366 nm for excitation and 419 nm from emission, which were also optimized in this work.

For these conditions detection limits evaluated for S/N = 3 were 0.13, 0.082 and 0.42 ppm for C2, C6 and C12 PFCA, respectively. Further improvement of detection limits, down to single ppb levels, is expected when larger injection sample volume (e.g. 400 μL) is utilized. Further improvement of method sensitivity that will address the analysis of PFCAs at environmental levels will be achieved by appropriate solid-phase extraction based analyte concentration. The method is also capable of satisfactory chromatographic resolution of C8 PFCA isomers at optimized condition.

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Perfluorochemicals in residents of the United States in 2001 through 2002

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Perfluorochemicals (PFC) have been used since the 1950s in a wide variety of industrial and consumer products, including protective coatings for fabrics and carpet, paper coatings, insecticide formulations, and surfactants. Several perfluorinated surfactants, a group of these PFCs, are persistent ubiquitous contaminants found in humans and wildlife. Animal studies also suggest potential developmental and other adverse effects associated with exposures to PFCs. In response to these findings, 3M, a primary manufacturer of PFCs in the United States, phased out the production of perfluorooctanesulfonate (PFOS) and related compounds starting in 2002. At present, the sources of exposure to PFCs in humans, and the potential health risks associated with exposure are still unclear; both are the subject of investigations worldwide. Demographic differences (e.g., age, sex, race) may exist in human exposure patterns to these compounds. We measured the serum levels of PFOS, perfluorooctanoate (PFOA), and 9 other PFCs in over 50 pooled serum samples collected from 1836 participants 12 years of age and older of the 2001-2002 National Health and Nutrition Examination Survey. The pools represented three major race/ethnicity groups (non-Hispanic blacks, non-Hispanic whites, and Mexican Americans), four age categories (12-19 years, 20-39 years, 40-59 years, and 60 years and older), and both sexes. PFCs were extracted from 100 μL of serum using an on-line solid-phase extraction method coupled to isotope dilution-high performance liquid chromatography-tandem mass spectrometry.

The limits of detection ranged from 0.05 ng/mL to 0.2 ng/mL. PFOS, PFOA, and perfluorohexanesulfonate (PFHxS) were detected in all of the pools analyzed at concentrations above 0.2 ng/mL (0.1 ng/mL for PFHxS); PFOS was found at the highest concentrations followed by PFOA and PFHxS in agreement with previous studies. Perfluorooctanesulfonamide (PFOSA), 2-(N-ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH), 2-(N-methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH), and perfluorononanoate were detected in more than 80% of the pools at concentrations lower than those of PFHxS; perfluorohexanoate, perfluorodecanoate, perfluoroundecanoate, and perfluorododecanoate were infrequently detected (limits of detection for these analytes are between 0.05 ng/mL and 0.4 ng/mL). Non-Hispanic whites had statistically significantly higher concentrations of PFOS, PFOA, PFHxS, Et-PFOSA-AcOH and Et-PFOSA-AcOH than both non-Hispanic blacks and Mexican Americans; Mexican Americans had statistically significant lower concentrations of these same PFCs than non-Hispanic blacks. Males had concentrations of PFOS, PFOA, Me-PFOSA-AcOH and PFNA significantly higher than females, but similar concentrations of PFHxS, PFOSA, and Et-PFOSA-AcOH. The concentrations of most PFCs were similar across the four age groups, unlike other persistent pollutants (e.g., dioxins) that are found at higher concentrations with increased age. However, adolescents (12 to 19 years of age) had higher concentrations of PFHxS than adults 20 years of age and older, although the differences were of borderline statistical significance ($p = 0.0779$) in agreement with others' findings suggesting that PFHxS concentrations in children are higher than in adults. Genetic variability, diet, lifestyle, or a combination of all these factors may contribute to the different patterns of human exposure to PFCs observed among the population groups represented in the pools.

Determination of 8-2 fluorotelomer alcohol, telomer-, and perfluorinated carboxylic acids. An analytical methodology for investigating biodegradation of fluorotelomer-based polymers in soils and sediment

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To elucidate potential sources of perfluorinated carboxylic acids (PFCAs) in the environment, the potential biodegradation of precursor substances is an important area of investigation. These studies require development and thorough validation of analytical methods to determine PFCAs and precursor compounds that could biodegrade to PFCAs, such as fluorotelomer alcohols or fluorotelomer acids. Although significant progress has been made in the analytical methodology for determination of PFCAs in various environmental matrices, soil or sediment matrices still present a challenge. This challenge is increased when telomer alcohols and telomer acids are to be quantified together with PFCAs.

We have developed and validated analytical methods that address these challenges in biodegradation studies conducted in soils and sediments. The methods utilize the same sample extraction and clean up procedure for determination of 8-2 Fluorotelomer Alcohol (8-2 FTOH), two telomer acids and C8-C11 PFCAs. The analytes are extracted with acetonitrile/water. Sodium hydroxide is added to aid in analyte partitioning into ACN and to minimize binding in soil matrices. Through direct addition of Envi Carb sorbent to the extract, cleanup is accomplished by preferential removal of dissolved soil/sediment matrix from the extract. The extract is analyzed by LC/MS/MS for determination of acid analytes and GC/MS for 8-2 FTOH. The methods were validated by testing recoveries on four different soils and a sediment, fortified with the fluorotelomer and perfluorinated acids at 5 and 50 ng/g and 8-2 FTOH at 300 and 2000 ng/g. The methods were also tested on "aged" samples, by analyzing fortified soil samples that were prepared as quality control samples at the start of biodegradation experiments and were extracted at predetermined time intervals. The recoveries generally ranged from 70-120%. It was also discovered that the presence of dissolved components of fluorotelomer-based polymer in the extract can bias the GC/MS analysis of 8-2 FTOH. Apparently, these components can thermally degrade to 8-2 FTOH in the GC inlet at typical inlet temperature of 250°C, but lowering the inlet temperature to 150°C alleviates the problem. The proposed methods were demonstrated to be of suitable sensitivity, selectivity, accuracy and precision to support the biodegradation studies with fluorotelomer-based polymers in soils and sediment.

Comparison of ion-pairing, solid phase extraction, and protein precipitation sample preparation techniques applied to human serum and plasma for the analysis of fluorinated alkyl compounds using high performance liquid chromatography/tandem mass spectrometry

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A study was undertaken to determine the variability associated with the analysis of fluorinated alkyl compounds in commercial lots of human serum and plasma using three different sample preparation methodologies. Specifically, isotopically labeled ammonium perfluorooctanesulfonate ([$^{18}O_2$] PFOS), perfluorooctanoic acid ([$^{13}C_2$] PFOA), and perfluorononanoic acid ([$^{13}C_2$] PFNA) were tested using ion pairing, solid phase extraction, and protein precipitation sample preparation techniques. Isotopically labeled material was used instead of non-labeled material due to the presence of endogenous levels of PFOS and PFOA in human serum and plasma. Additionally, perfluorobutane sulfonate (PFBS) and 1H,1H,2H,2H-perfluorooctane sulfonate (THPFOS) were tested using only the ion pairing method. The lowest spiking level was set at 1 ng/mL serum/plasma. A series of quality control samples were prepared to compare method performance. Initially, each sample preparation technique was quantified versus an extracted calibration curve prepared in human serum/plasma. In addition, samples produced using the ion pairing method were quantified on two different LC/MS/MS instruments versus solvent standards prepared in methanol to measure matrix effects.

Comparable results were obtained from all sample preparation techniques when each sample set was analyzed versus the corresponding extracted calibration curve. However, this study demonstrates an inherent variability with the ion pairing method when solvent standards are used for the calibration curve. Significant matrix enhancement was observed for [$^{18}O_2$] PFOS, [$^{13}C_2$] PFOA, and THPFOS in serum and plasma on both the Micromass Quattro II and Quattro Ultima. Matrix spike recoveries were variable and in some instances the individual matrix spike enhancement was greater than 200%. Matrix effects were not as prevalent for [$^{13}C_2$] PFNA, however, for PFBS the matrix effect was dependent upon which instrument was used. When PFBS was analyzed on a Micromass Quattro Ultima, matrix enhancement of the mid and high-level QC samples was as high as 163%. Conversely, matrix induced signal suppression resulted in recoveries as low as 37% for mid and high-level QC samples when the sample extracts were analyzed on a Micromass Quattro II. Matrix effects may dramatically influence analytical results (producing false high or false low results) if the ion pairing method is used with solvent standards. Matrix effects are minimized when samples are quantified versus extracted calibration standards.

Matrix-effect free analytical methods for the determination of perfluorinated carboxylic acids in biological matrices

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The sample matrix can have significant effects upon the performance of analytical methods for the quantitative determination of perfluorinated carboxylic acids in biological samples including signal suppression or enhancement in LC/MS/MS determinations. Methods for the determination of perfluorinated carboxylic acids in biological matrices (blood serum and plasma, liver and plant tissue) that address these issues, with limits of quantitation of 1-5 ppb were developed. Matrix enhancement and suppression are negligible using a novel dispersive solid-phase extraction procedure with a graphitized carbon sorbent. Quality control data have been developed that demonstrate (1) recovery of laboratory fortifications into the matrices of interest; (2) freedom from matrix enhancement or suppression; and (3) absence of significant background interference from both the sample and the fluorochemical components of the instrumentation.

Whole blood, blood serum and plasma samples. Add 1 mL acetonitrile and internal standard to 100 µL of serum or plasma. Vortex mix to precipitate the protein and cells (if present). Centrifuge 10 min at 10,000 rpm. Add 25 mg of Envi-Carb® bulk packing (Supelco Inc.) to 1.7-mL microcentrifuge tube. Add 50 µL glacial acetic acid. Transfer 1 mL of sample extract to microcentrifuge tube and vortex mix. Centrifuge 10 min at 10,000 rpm. Transfer 500 µL sample to autosampler vial and add 500 µL of water.

Liver samples. Extract 1 g sample with 10 mL of acetonitrile using probe homogenizer. Add internal standard and centrifuge. Perform same sample purification procedure and final dilution as blood method.

Plant tissue samples. Extract 1 g sample with 10 mL of methanol using probe homogenizer. Add internal standard and centrifuge. Perform same sample purification procedure and final dilution as blood method.

LC/MS/MS Analysis. C6 through C14 perfluorinated acids are analyzed by reversed-phase LC/MS/MS using a 15 cm x 2.1 mm i.d. C8 column and an acetonitrile/0.15% glacial acetic acid in water mobile phase gradient. A small HPLC column was inserted between the pump and autosampler to remove interfering contaminants introduced by components of the HPLC system. The MS/MS electrospray ionization interface is operated in the negative ion mode. One precursor to fragment transition is used for quantification, and a second transition is acquired for confirmation purposes.

Limits of quantitation (LOQs) of 2 ppb for blood and 5 ppb for liver and plant tissue were demonstrated by conducting fortification experiments at the target LOQ and 10x the target LOQ. Recovery levels were 80% to 110% for all matrices were obtained at both fortification levels. Controls samples showed negligible interference at the LOQ. The ratios of the two transitions collected matched those acquired from calibration standards within ± 30%. The use of the graphitized carbon sorbent removes co-eluting matrix components that would have caused any matrix enhancement or suppression. The methods are simple to perform, yield precise and accurate results and have been thoroughly validated on the matrices to which they apply.

Analytical methods for the determination of extractable perfluorooctanoic acid (PFOA) from consumer articles

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Perfluorooctanoic acid (PFOA) salts are used as processing aids in the manufacturing of some fluoropolymers. While not used or added in the manufacture of fluorotelomer-based products, PFOA may be present at trace levels as an unintended reaction by-product. Analytical methods were developed and validated to determine the amount of PFOA that could be extracted from various consumer articles (e.g. cookware, and paper) using extractants relevant to potential human exposure (e.g. water and food-simulating solvents). Extracts were analyzed via liquid chromatography tandem mass spectrometry (LC/MS/MS). The results were used to support a consumer exposure assessment and risk characterization.

Fry pans were purchased at retail. A FDA procedure [1] was modified for water extraction. The pans were first washed as recommended by the manufacturer, rinsed and dried. A custom-made glass lid fitted with a condenser was placed atop each pan filled with laboratory deionized water. The pans were heated to boil the water and refluxed for 30 minutes. The water was cooled, then removed for LC/MS/MS determination of PFOA. Ethanol/water mixture (1:9) was used to simulate watery and acidic foods and a mixture of 19:1 ethanol/water was used to simulate fatty or oily foods [2]. Each fry pan was cut into multiple rectangular pieces, placed in a pressurized fluid extraction apparatus and extracted at 125 ºC and 1000 psi. Dual ^{13}C labeled PFOA internal standard was added to each collection vial and mixed. Extract solutions were analyzed via LC/MS/MS. Recovery experiments corresponding to levels of 400 pg/cm^2 for the boiling water experiment gave 88% recovery. For the ethanol/water extractions, fortifications were made at the 100 and 1000 pg/cm^2 levels with recoveries from 87 to 100%. None of the fluoropolymer-treated pans showed any detectable levels of PFOA.

Solvent controls, untreated control paper samples and treated paper samples were prepared in triplicate and extracted in pressurized fluid extraction cells for 2-hours and 240 hours. The first 2 hours of extraction were conducted at a temperature of 100ºC with the remaining 238 hours at 40ºC. Stability samples were prepared in triplicate and subjected to the same thermal treatment as the samples. The extracts were analyzed by LC/MS/MS. Samples and calibration standards were prepared over the range of 0.1 ng/mL to 50 ng/mL using dual-isotope labeled ^{13}C-PFOA internal standard to correct for matrix effects. Recoveries at the 0.5x, 1.0x, and 2.0x fortified levels ranged from 84% to 98%.References: [1] US FDA, see 21 CFR 175.300(d), table 2 condition of use B., [2] US FDA, Guidance for Industry, "Preparation of Food Contact Petitions for Food Contact Substances: Chemistry Recommendations", Final Guidance, April 2002, Appendix I.

Temporal trends of polyfluorinated sulphonamides and telomer alcohols in Toronto: Results of a long-term sampling campaign

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Polyfluorinated sulphonamides and telomer alcohols have previously been detected in the North American troposphere, with average total airborne concentrations ranging from approximately 10 to 400 pg/m^3. Although these compounds have been detected in the troposphere, most studies have been of short duration and as such, little is understood about the temporal variability of these contaminants. A sampling campaign of airborne polyfluorinated sulphonamides and telomer alcohols has been conducted in downtown Toronto since February 2002. The primary objectives of this study were to investigate the temporal distribution and temperature dependence of the gas-particle partitioning of the selected polyfluorinated contaminants. In addition, this study will determine whether a decrease in tropospheric concentrations of the polyfluorinated sulfonamides and sulfonamideoethanols has been observed since the major manufacturer of these chemicals removed them from the market in 2000. Samples were collected using polyurethane foam - XAD sandwich and a high-volume sampler and analyzed using gas chromatography-mass spectrometry employing chemical ionization. Preliminary results indicate that polyfluorinated sulphonamides and telomer alcohols were detected throughout the study period at concentrations similar to those reported in earlier studies. In addition, preliminary results indicate that the concentrations of the polyfluorinated telomer alcohols may be increasing throughout the sampling period, relative to the concentrations of the polyfluorinated sulphonamides.

Development and validation of analytical methods for the low-level determination of perfluorooctanoic acid (PFOA) in paper, textile and carpet by LC/MS/MS

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Fluorotelomer-based acrylic polymers are applied to the surface of textiles, paper and carpet to impart oil, stain and water repellence properties. Concerns that fluorotelomer-based polymers are a possible source of low level exposure to humans coupled with their widespread use have prompted the need to develop methods to detect and measure PFOA in common articles of commerce, namely textile, paper and carpet.

Two separate methods, one method for the determination of low-levels of perfluorooctanoic acid (PFOA) in paper and textile and the other method for determination of PFOA in carpet were developed and validated. This effort was overseen by the members of the Telomer Research Program (TRP) in order to evaluate their products and determine whether they contribute to significant human or environmental levels of PFOA.

The LC/MS/MS method in paper and textiles utilized a dual labeled 13C-PFOA internal standard. Levels of PFOA were determined using an isocratic, reversed-phase HPLC method with an ammonium acetate/methanol buffer, separated on a 2.1x50 mm , 4 μm Genesis C8 column. The LC/MS/MS was an Agilent 1100 bench top liquid chromatograph coupled to a PE Sciex API 4000 triple quadrupole mass spectrometric detector with a Turbo Ion Spray Liquid Introduction Interface. MS/MS detection was performed in the turbospray negative ionization mode. Ions monitored were 413 (parent) and 369 (daughter) for PFOA and 415 (parent) and 370 (daughter) for dual labeled 13C-PFOA internal standard. Hypercarb™ cartridges (4 mm) were placed before the HPLC injector to capture any PFOA introduced from the mobile phase and instrument components. Any captured PFOA was removed from pre-columns before the injector by flushing the system with 100% methanol prior to equilibration with the isocratic mobile phase. The interday recoveries and standard deviations (three day validation) for samples (n = 54-55) fortified with PFOA at 5, 50, and 200 ng g-1 were 114% ± 4.9% for textile and 110% ± 7.6% for paper.

The LC/MS/MS method in carpet also utilized a dual labeled 13C-PFOA internal standard. Levels of PFOA were determined using a reversed-phase HPLC method with acetic acid-acidified water/methanol gradient, separated on a 2x50 mm Phenomenex Synergi Polar RP column. The LC/MS/MS was a Shimadzu solvent delivery system coupled to an Applied Biosystems triple quadrupole mass spectrometric detector with a Turbo Ion Spray Liquid Introduction Interface. MS/MS detection was performed in the turbospray negative ionization mode. Ions monitored in MRM mode included; 413 (parent) to 369 (daughter) for PFOA and 415 (parent) to 370 (daughter) for dual labeled 13C-PFOA internal standard. Overall recovery and standard deviation for samples (n = 30) fortified with PFOA at 5, 50, and 500 ng g-1 were 98.9% ± 8.1%. Specificity of the method was evaluated with two different lots of untreated carpet samples.

Isomer pattern of PFOS in human blood

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Highly fluorinated compounds are mainly produced in three ways; electrochemical fluorination, telomerisation and oligomerisation [1]. The electrochemical process is difficult to control and results in several byproducts, such as cleaved, branched and cyclic structures[1]. The market products of perfluorooctansulfonyl derivatives (including PFOS) normally contained 70% of the linear compound and 30% of branched impurities according to 3M[2]. Also PFOS standard materials can contain different isomers, which varies between different batches and suppliers[3].

A study of the isomer pattern of PFOS in human blood is presented here. A total of 40 serum samples from Sweden and Australia were analysed. Further, blood samples from five persons were analysed both as plasma and whole blood. All samples were extracted with a C18 SPE and analysed by HPLC-ESI-MS [4].

Eight peaks were detected in human serum in the retention time window around the main peak (16.5 min), presumably the linear PFOS, denoted PFOS#L. Six peaks were found in front and one peak after PFOS#L, with relative retention time (RRT) to PFOS#L between 0.86 and 1.03. Of the total area for m/z 499, 70% consisted of PFOS#L in the Swedish serum compared to 60% in the Australian. The two major isomers, denoted PFOS#5 (RRT 0.96) and PFOS#6 (RRT 0.97) were 18 and 13% in Swedish serum and 22 and 16% respectively in Australian serum. A batch of standard material (Fluka, K+, 98%) contained 79% PFOS#L and 12 and 9% of PFOS#5 and PFOS#6 respectively. MS/MS analysis using the daughter ions SO_3F^- (m/z 99), SO_3^- (m/z 80) and $CF_2SO_3^-$ (m/z 130) showed that the different isomers fragmented differently and therefore the area percentage can not be used to estimate the real amount in the serum sample.

Whole blood and plasma from the same individual contained approximately the same amount of PFOS#L and the two prominent isomers PFOS#5 and PFOS#6. The early eluting RRT 0.86 peak was however on average about 3 times larger in plasma compared to whole blood and the late RRT 1.03 peak was about 3 times larger in whole blood. This pattern was observed for all 5 persons.

The pattern of m/z 499 signals, presumably isomers, around the main PFOS peak varies somewhat in human plasma. One explanation to this can be that specific exposure causes deviating PFOS pattern. Branched isomers presumably behave differently compared to the straight chain. The observed difference between plasma and whole blood for two of the isomers might indicate different interaction abilities. Other factors still have to be elucidated and diet, contact with products containing different PFOS mixtures or the pharmacokinetic behaviour of each isomer could also be responsible for observed isomer patterns in human serum.

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Comparing modeled and monitored values: Characterizing perfluorooctanoate in ambient air near the fence line of a manufacturing facility

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A six-event, 24-hour monitoring series was performed around the fence line of a fluoropolymer manufacturing facility that uses ammonium perfluorooctanoate as a fluoropolymer processing aid. Both particulate and vapor phase analyte were differentiated using the OSHA versatile sampler (OVS) system. Perfluorooctanoate concentrations were determined as perfluorooctanoic acid (PFOA) via liquid chromatography and mass spectrometry (LC/MS) [1]. Those data indicate that the majority of the PFOA is present as a particulate. No vapor-phase PFOA was detected above a detection limit of approximately 0.07 $\mu g/m^3$. A follow-up study using a high-volume cascade impactor verified the range of concentrations observed in the OVS data.

The Industrial Source Complex Short Term Model (ISCST3) was used to conduct air dispersion modeling. ISCST3 is a steady state Gaussian model recommended by the U.S. EPA. It is included in the «Guideline on Air Quality Models» which is codified as Appendix W to 40 CFR Part 512. Both studies aligned with the major transport direction and range of concentrations predicted by an air dispersion model, demonstrating that model predictions agreed with monitoring results. Results from both monitoring methods and predictions from air dispersion modeling showed the primary direction of transport for PFOA was in the prevailing wind direction. The PFOA concentration measured at the site fence over the 10-week sampling period ranged from <0.12 and 0.9 $\mu g/m^3$. Modeled predictions for the same time period ranged from <0.12 to 3.84 $\mu g/m^3$. Less than 6% of the particles were larger than 4 μm, while almost 60% of the particles were below 0.3 μm. These studies are believed to be the first reported ambient air data for PFOA in the environment surrounding a manufacturing facility. Another follow-up study is planned for 2005 summer/fall.

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Physicochemical properties of fluorotelomer alcohols and perfluorocarboxylic acids

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Accurate knowledge of the physicochemical properties of fluorotelomer alcohols and perfluorocarboxylic acids is necessary to understand not only environmental transport potential but also human exposure potential. We have determined the vapor pressure of fluorotelomer alcohols using both direct measurement (headspace GC/AED) and gas chromatographic relative-retention time measurement [1]. The gas chromatographic measurement is more problematic since it assumes identical interaction between the stationary phase and the analyte and reference materials for similar functional groups of relatively nonpolar systems.

We have also measured the vapor pressure of perfluoro-octanoic, -nonanoic, decanoic, -dodecanoic, -undecanoic, and -dodecanoic acids using the Scott method [2]. We observed evidence of sublimation of some acids and their salts stored under ambient conditions.

The vapor pressure using the relative retention time method with perfluorohydrocarbons and perfluoroalcohols as reference standards is about one order of magnitude higher than the direct (GC/AED) measurement method. If hydrocarbons are used as references, then the vapor pressure is two orders of magnitude higher than the direct method. The GC/AED data agree with previously published data from our laboratory using NMR spectroscopy, Scott, and vapor saturation methods [3], but differ from other data determined from relative retention time measurements [4] and a boiling point method[5]. The vapor pressure was measured from 59 to 191ºC (C8), 100 to 203ºC (C9), 130 to 219 ºC (C10), 112 to 238 ºC (C11), and 128 to 247 ºC (C12).

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Discovery of semi-volatile fluoroalkyl substances in municipal wastewater treatment plant sludge

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The role of the municipal wastewater treatment plant in the overall fate and transport of fluoroalkyl substances in the environment is unknown. Reports of fluoroalkyl substances in municipal wastewater treatment plant sludge have recently been published. Previous work has demonstrated that select semi-volatile fluoroalkyl substances degrade to anionic fluoroalkyl substances, however to date, the occurrence of semi-volatile fluoroalkyl substances in municipal wastewater treatment plant sludge has not been reported.

In this paper we describe analytical methods developed for the extraction of semi-volatile fluoroalkyl substances from municipal wastewater treatment plant sludge. The semi-volatile fluoroalkyl analytes evaluated include fluoroalkyl sulfonamides, sulfonamide alcohols, sulfonamide acrylates, fluoroalkyl olefins and fluorotelomer alcohols. The recoveries of semi-volatile fluoroalkyl substances extracted with ethyl acetate from air dried sludge vs. wet sludge were determined. Selection of extraction solvent was based upon both extraction efficiency and analyte loss upon sample concentration. Sequential, exhaustive extractions of sludge using both accelerated solvent extraction (ASE) and hot sonication extraction with solvent are evaluated. Quantitation of analytes in sludge extracts was performed using GC/MS/MS with selected reaction monitoring. Both pentadecafluorooctanol was used as an internal standard and the stable isotope labeled 8:2 fluorotelomer alcohol was utilized as a surrogate standard in both quantitation and recovery determinations. All classes of semi-volatile fluoroalkyl substances under evaluation were detected in municipal wastewater treatment plant sludge.

Laboratory measurements of the infrared absorption cross sections of fluorotelomer alcohols

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Fluorotelomer alcohols (FTOHs, CF3(CF2)nCH2CH2OH where n = 0,3,5,7) are industrial chemicals widely used in a variety of industrial products, such as paints, coatings, polymers, adhesives, waxes, polishes, electronic materials, and caulks. Their concentration in the atmosphere over North America has been estimated at 17-135 pg/m^3 with a lifetime from 11 to 20 days. Although the environmental fate of FTOHs is not fully determined, it has been suggested that their atmospheric oxidation is a source of long-chain perfluoroalkyl carboxylic acids (PFCAs, $C_xF_{2x+1}COOH$, where x = 6 - 12) observed in remote locations. To facilitate laboratory studies of the atmospheric chemistry of FTOHs and the spectroscopic detection of FTOHs in the atmosphere, an experimental study of the IR spectra of $C_xF_{2x+1}CH_2CH_2OH$ (x=1,4,6,8) has been performed. We report here on the measurements made at the University of Toronto using samples of pure FTOHs in a 0.25-m-path cell at room temperature over the spectral region 500-4000 cm^{-1} using a Bomem DA8 Fourier transform spectrometer. We show that the FTOHs have absorption cross-sections in the spectral region 1000-1500 cm^{-1} indicating their potential importance as greenhouse gases. These results are shown to be consistent with the measurements done at Ford Research Laboratories using a Mattson Instruments Sirius 100 Fourier transform infrared spectrometer and with theoretical calculations performed at DuPont Engineering Research and Technology.

Direct measurement of perfluoroalkylated surfactants in Great Lakes water samples

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Considerable effort has been made in the last few years to determine the level of perfluorinated surfactants contamination in the environment. The unique physicochemical properties of the perfluorinated compounds contributed to their large industrial and household use over the last 50 years. These chemicals are part of a large number of surface treatment and surfactant formulations such as fire-fighting foams, special cleaners, mining surfactants and insecticides. A recent report[1] showed higher values of perflourooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) than the values determined earlier[2] for the Great Lakes and has created a scientific debate[3,4].

This paper presents the first report of a direct determination of perfluorinated surfactants from water samples at the parts-per-trillion (ng/L) level, eliminating the extraction/concentration steps used in the previous studies[1,5]. The goal of the investigation was to determine the level of selected perfluorinated surfactants in Great Lakes waters using a simple method, with minimal manipulation of the original sample. Results are reported for PFOS ($C_8F_{17}SO_3^-$) and PFOA ($C_7F_{15}CO_2^-$) in water samples from Lake Ontario, Lake Erie and Lake Huron, quantified with internal standard and standard addition methods. Samples from 11 different locations (Lake Ontario, Lake Erie and Lake Huron) were analyzed in triplicate.

Samples were collected from off shore and near shore locations in September 2004 and stored in polypropylene bottles at 4 °C. Aliquots of 300 ìL from the water samples were mixed with an equal volume of MeOH containing the internal standards $^{13}C_2$-PFOA and $^{13}C_2$-PFDA giving final concentrations of 20 fg/ìL and 50 fg/ìL, respectively. Analysis of target analytes was performed using a high performance liquid chromatograph-tandem mass spectrometer system (HPLC-MS/MS), consisting of an Agilent 1100 Series liquid chromatograph coupled with a 4000QTRAP triple quadrupole mass spectrometer.

Quantitation was performed using internal standards and standard additions. The analytes monitored in all samples were: perfluoroheptanoic acid (PFHpA), PFOA, perfluorononanoic acid (PFNA), perfluorodecanoic acid PFDA, perfluoroundecanoic acid (PFUnA), perfluorododecanoic acid (PFDoA), perfluorohexane sulfonate (PFHxS), PFOS and heptadecafluorooctane sulfonamide (PFOSA). PFOS (2-12 ng/L) and PFOA (2-6 ng/L)were detected in all lake samples tested with the exception of the Lake Huron sample. Matrix effects were observed in all real samples tested based on monitoring the peak area counts of the two internal standards. The analytes with less retention were more affected, including the $^{13}C_2$-PFOA internal standard. The effect was less evident for the second internal standard $^{13}C_2$-PFDA.

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Trace level analysis for per and poly- fluorinated compounds

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Solid phase extraction (SPE), using Sep-Pak C18 or Oasis HLB cartridges, has been followed in the analysis of perfluorinated acids in water. These SPE cartridges are not suitable for the analysis of short chain (C4 to C6) perfluorinated acids and fluorotelomers. In this study, we describe detailed parameters to optimize a new SPE method with HPLC tandem mass spectorometry (HPLC-MS/MS) for the analysis of C4 to C18 perfluorinated acids, some telomer alcohols and telomer acids.

Although there is evident that background contamination is one of the most difficult aspects of perfluorochemical analysis, instrumental blank is the first obstruction in the analysis. We successfully decreased blank levels of HPLC-MS/MS to measure part per quadrillion levels of perfluoros in deep sea water. However, different types of contamination issues were encountered when setting up a new HPLC-MS/MS. Blank levels were detected for chemicals including C6 to C14 PFCAs, 8:2 FTUCA and 8:2 FTCA. The source of contamination was found to be from fluoropolymer sealing inside of pump, which is commonly used in forward phase HPLC. After the modification of the above parts of the instrument, the blank levels were greatly reduced.

Another source of contamination in blank is standard chemicals themselves. Impurities were found in all target perfluorochemical standards commercially available. In most cases, the impurities in standards were negligible. However, 7% of PFBS was found in PFOS standard (3M). Six to 11% of PFBA and 6 to 9% of PFPeA were detected in ^{13}C-PFOA (Perkin Elmer), PFTeDA(SynQuest), PFHxDA(SynQuest), PFOcDA(SynQuest) and 10:1 FTOH (SynQuest), respectively. It is clear that inaccurate use of these standards can cause analytical error in the determination of short chain PFCs and telomers.Extraction using Oasis®HLB and Oasis®WAX cartridges was examined. Recoveries of target fluorinated compounds spiked into HLB cartridges were generally >80%, except for short-chain carboxylic acids such as, PFHxA, PFPeA, and PFBA, whose recoveries were less than 30%. The average recoveries of poly- and per-fluorinated acids, including short-chain carboxylates, through WAX cartridges were between 85 and 107% (n=5), except for two FTOHs, 10:1 FTOH and 7:1 FTOH, for which the recoveries were 59 and 54%, respectively. Nevertheless, the recoveries of short-chain perfluorocarboxylates, PFBA, PFPeA, and PFHxA, through WAX cartridge extraction were higher than those through HLB cartridge extraction.Estimated LOQs, calculated based on spiking known concentrations of standards, and passed through the entire analytical procedure involving the SPE extraction were 1 pg for PFOS, PFHS, PFBS, PFOSA, PFUnDA, PFDA, PFHpA, PFHxA, PFPeA, ^{13}C-PFOA, THPFOS, 8:2FTUCA, 5 pg for PFOcDA, PFHxDA, PFTeDA, PFDoDA, PFBA, 8 pg for N-EtFOSA, 25 pg for 8:2FTCA, 40 pg for 10:1FTOH, 800 pg for 7:1FTOH, and 4000 pg for 8:2FTOH and 10:2FTOH in 1L of water sample.Overall, the methods developed in this study can be applied in the trace level analysis in water, so that we can better understand the fate of per- and poly-fluorinated compounds in the environment.

Monitoring of perfluorinated compounds in human breast milk from Zhoushan, China

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Many recent studies have reported the ubiquitous distribution of perfluorinated compounds (PFCs), especially PFOS and PFOA, in wildlife species and human samples. An accurate assessment of the exposure of human to PFCs is important in risk assessment. Previous studies have focused on determining concentrations of PFCs in whole blood or blood serum. It was not until 2003 that a solid phase extraction (SPE) method was developed, which allowed measurement of PFCs in human breast milk. This method has not only provided an alternative method for human exposure assessment, but also improved our understanding of the partitioning of PFCs into breast milk and the subsequent impacts on lactating infants.

In the present study, PFCs in 20 human breast milk samples collected from Zhoushan, China, were analyzed by modifying a published method, using weak-anion exchange SPE extraction. The mean matrix spike recoveries were 102% for PFOS, 95% for PFHxS, 96% for PFBS, 41% for PFOcDA, 44% for PFHxDA, 51% for PFTeDA, 79% for PFDoDA, 101% for PFUnDA and PFOA, 99% for PFDA, PFHpA, PFHxA and PFBA, 91% for PFNA, 100% for PFPeA, 101% for 13C-PFOA, 130% for THPFOS, 74% for 8:2 FTCA and 87% for FTUCA. Comparing to the previous method allowing the detection of 11 chemicals (PFOSA, Et-PFOSA, PFHxS, PFOS, PFPeA, PFHxA, PFOA, PFNA, PFDA, PFUnA and PFDoDA), the present method enables the measurement of wider ranges of chemicals.

PFOS and PFOA were detected in all the samples. PFNA and PFHS were detected in more than 80% and 50% samples, respectively. Concentrations of PFOS ranged from 71.3 to 592.6 pg/mL, whereas those for PFOA ranged from 142.3 to 341 pg/mL. These concentrations were much less than those reported in the blood serum of the general population of the US (47 ng/mL for PFOS and 3 ng/mL for PFOA). Lesser concentrations were detected for PFHxS (10 – 67.2 pg/mL) and PFNA (13.3 – 83.8 pg/mL). Health risks of infants through the consumption of mothers' breast milk were assessed by estimating the health indexes based on the USEPA guidelines. Assuming an average milk consumption rate and body weight for one-year old infants were 600 g/d and 7.3 kg, respectively, the daily intake of PFOS to the child from the mother with the greatest exposure would be 0.049 μg/kg/d, which exceeds the very conservative reference dose of 0.025 μg/kg/d. Following the same principle, the daily intake for PFOA would be 0.028 μg/kg/d, which was less than the reference dose of 0.333 μg/kg/d. These results indicate that there may be a small potential risk of PFOS for the infants in Zhoushan via the consumption of breast milk. Further monitoring studies should be carried out to clarify this issue. In addition, it would be instructive to study the relationship between PFC exposure and diet habits for different age groups.

Concentration of perfluorinated carboxylic acids in river waters supply to the city of Gdansk, Poland

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The Radunia River, with 837 square kilometers of the catchment area, is the only source of surface water supply to the Straszyn Water Treatment/Purification Plant, which supplies potable water to the city of Gdansk and is located in the northern part of Poland. The Motlawa River is a river in the Eastern Pomerania. The source of water is from Szpegawski Lake, locating northeast from Stragard Gdanski. Radunia River has its outflow to Motlawa going through Gdansk with an outflow to Dead Vistula. In this study, surface water samples collected in 2004 at different sites along both rivers and including the lakes and small tributaries were examined for several perfluorinated chemicals (PFCs).

In spite of many publications for the last five years, there are very few information about short chain PFCs such as PFBA and PFBS. The most likely reason is the absence of reliable method for short chain PFC analysis. We have developed the new solid phase extraction method using a weak anion exchange cartridge, WAX (Waters) for trace level water analysis. Recovery and blank level of C4 to C12 carboxyl acids by this method is applicable to any kind of water sample at part per quadrillion level analysis. The method limit of quantification for individual PFCs was down to 0.1 ppt (0.1 ng/L). 400 mL of water samples were extracted using WAX and eluted with methanol and 0.1% ammonium methanol. Target compounds in above water samples included PFOS, PFHS, PFBS, PFOSA, PFNA, PFOA, PFHpA, PFHxA, PFPeA, PFDoDA, PFBA PFOcDA, PFHxDa, PFTeDA, PDUnDA, PFDA, 10:1 FTOH, 8:2 FTUCA. To our knowledge, this was the initial investigation to analyze C4 to C18 PFCs in water samples from Poland.

Regarding the result, most of target compounds were identified with varying frequency depending on the compound and sampling site, while PFOcDA, PFHxDa, PFTeDA, PDUnDA, PFDA, 10:1 FTOH, 8:2 FTUCA were undetected (< 0.1 ppt) at the all sites surveyed. PFOS remained undetected (< 0.5 ppt) at the upper-most segment of the Radunia River but was predominant (18 ppt) amongst all PFCs quantified when starting from the Zukowo site and down the river. Concentrations of PFCs in the Motlawa River were remarkably lower than those in Radunia River. This could probably be due to the dilution effect by larger volume of water flow in this river comparing to the Radunia River. Interestingly, higher concentrations of PFOS than PFOA were found in all water samples from Poland. It's clearly opposite to the finding in surface water samples from Japan and China and also those reported in US and some European countries. It suggested that different type of PFCs pollution from above countries may be expected from further investigation in eastern Europe.

Perfluorinated chemicals in blood of fishes and waterfowl from the Gulf of Gdansk, Baltic Sea

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After finding of world wide pollution by perfluorooctane sulfonate (PFOS) in wild animals in 1999, many monitoring effort have been carried out in US and several developed countries for the last five years. However, they reported very few about old eastern Europe except human exposure study. It also appeared that PFOS is only a member of related perfluorinated compounds (PFCs) instead of global concern about PFOS itself. In this study a preliminary screening of several PFCs has been performed in whole blood samples of fish and fish eating birds. Cod (Gadus morhua) from the Gulf of Gdansk and waterfowl were collected in Poland during wintering season of 2002 and 2003. A total of 65 samples from 5 species of waterfowl such as razorbill (Alca torda), red-throated loon (Gavia stellata), black scoter (Melanita nigra), long-tailed duck (Clangula hyemalis) and common eider (Somateria molissima), and 18 of cod have been examined.

C6 to C9 of PFCs in blood samples were analyzed by the ion pair extraction method as a quick screening tool. We have already developed solid phase extraction method for short chain carboxylic acids and applied to some samples. Concentrations of PFCs were determined by the use of high-performance liquid chromatography coupled with electrospray tandem mass spectrometry (HPLC-ESI MS/MS). In the present study, PFOS was the predominant fluorochemical found in blood of the biota examined. The fish-eating red-throated loon showed the highest body burden of PFOS with 40-198 ng/mL. The concentration profile of PFOS and four other fluorochemicals (PFOSA, PFHxS, PFNA and PFOA) was largely similar between all 5 bird species and their concentrations in descending order were as follow: PFOS > PFHS/PFOSA > PFNA > PFOA. Cod sample displayed a similar concentration profile of PFCs to that of waterfowl, however, PFHxS was significantly lower than PFOSA and somehow comparable with PFOA concentration.

PFOS, PFOSA, PFNA, PFOA and PFHxS concentrations in cod blood ranged from 6 to 52, 1.0 to 13, 0.1 to 2.0, <LOQ (0.05) to 0.73, and <LOQ (0.05) to 0.83 ng/mL, respectively. A striking finding was that only two of five bird species examined showed higher concentration of PFCs in blood than cod, which occupies the lowest trophic level of all organisms surveyed. We have tried to estimate bioconcentration factor (BCF) of PFCs in cod. It's not exactly same to BCF because we assumed same concentration in blood to liver but estimated average value was 34,000 (ranged 6,500 to 100,000). This is comparable to the highest BCF (41,600) observed from our previous study in Tokyo Bay, Japan in 2002. This result also showed interesting coincidence between residue level in biota and water in this region and seemed to provide some information about BCF and accumulation. To our knowledge, this is the first report about PFCs bioaccumulation including C4 to C9 carboxylic acids in wild animals in Poland.

Human exposure to perfluorinated compounds via food

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Recently perfluorooctane sulfonate (PFOS) and related perfluorinated compounds were identified as a new group of persistent environmental pollutants. They were detected in biological samples worldwide[1,2] , and have been shown to bioaccumulate in marine food webs[3]. In contrast, little is known about terrestrial food chain accumulation[4]. Both, persistence and bioaccumulation, support that diet is one of the main sources of human background exposure. In addition, application of perfluorinated compounds as surfactants in food packaging and cooking utensils might serve as other sources of food contamination[5].

Thus, PFOS and related compounds were included in a recent Bavarian monitoring study on the dietary exposure to a number of endocrine contaminants. 50 volunteers were recruited from the general population and requested to collect duplicates of their daily food and beverages over a 7-day period. Daily samples are mixed, homogenized and stored at -20°C for later analysis. The sampling period started in April 2005 and will proceed at least until August 2005. In parallel, an appropriate, robust and sensitive analytic method was developed and validated. However, only a limited number of samples has been analyzed yet.

The method bases on ion-pair extraction of the wet samples using tetrabutylammonium ions as ion pairing agent. Extracts are analyzed for PFOS, perfluorohexanoic acid (PFOA), perfluorooctanoic acid (PFHxA), and perfluorooctane sulfonamide (PFOSA) applying LC-MSMS. Chromatography is performed by gradient elution on a C18 phase (Hypersil gold, 100 x 2 mm, 5µm) using methanol and ammonia acetate buffer (pH 3.2) as eluents. Detection is executed by negative electrospray ionization followed by triple quadrupole mass spectrometry (Finnigan TSQ 7000). Quantitation is performed by internal calibration using 7-H-dodecafluoroheptanoic acid as internal standard.

An initial fortification study proved a 80-120% recovery range for PFOS, PFOA and PHxA, recoveries for PFOSA were of about 50%. Limits of determination (LOD), defined as method blank runs, were below 1-2 ng/g fresh weight. Analytical results obtained for three diet samples are given in Table 1.

Dietary concentrations of the observed perfluorinated compounds were measured in the range of 3.6 - 10.5 ng/g wet weight, whereas PFOS and PFOA exhibited the highest levels. In comparison, PFOS levels in 6 pooled samples of German blood banks were reported to be in the range between 32 and 45.6 ng/g . These experimental results are a strong indication of biomagnification of PFOS and related compounds in humans. Wet weights of consumed foodstuffs and beverages were in the range of 2500-3000 g. Based on the very first results of this study, dietary daily intakes of the investigated substances are expected to be in the range between 9 to 32 µg/day.

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A comparison of whole blood, plasma and serum evaluations for the determination of PFOA, PFOS, and PFHS in human subjects

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The purpose of this investigation was to determine whether "blood" specimens collected from human subjects using different anticoagulants for whole blood and plasma would affect the results of analytical measurements on perfluorooctanesulfonate (PFOS), perfluorooctanoate (PFOA), and perfluorohexanesulfonate (PFHS) when compared with the results from serum studies completed on the same individuals. The blood collection matrices evaluated included: whole blood collected in lithium heparin, whole blood collected in ethylenediamine tetraacetic acid (EDTA), plasma samples collected with lithium heparin, plasma samples collected with EDTA and whole blood samples where no anticoagulation agents were used allowing the blood to clot. The clotted blood was then processed by centrifugation to yield serum samples. Blood sample tubes were drawn sequentially from a single venipuncture site from each of twelve volunteer fluorochemical production workers during medical surveillance testing. The 3M Institutional Review Board approved this study. Fluorochemical analyses were completed using high-pressure liquid chromatography / tandem mass spectrometry methods. Solid Phase Extraction (SPE) methods were used for all analytical sample preparation. Significantly lower levels of all fluorochemicals measured were found in the whole blood specimens regardless of the anticoagulant used. There is good correlation to the lower levels of fluorochemicals observed in whole blood corresponding to the actual volume displacement of the red blood cells. Thus indicating that the fluorochemicals are not found in or attached to the red blood cells, but are in fact associated with serum proteins.

The mean serum concentration of PFOS was 177 ng/mL, the mean lithium heparin plasma concentration of PFOS was 176 ng/mL, and the mean EDTA plasma PFOS concentration was182 ng/mL. The mean lithium heparin whole blood concentration of PFOS was 86 ng/mL. The mean EDTA whole blood concentration of PFOS was 86 ng/mL.

The mean serum concentration of PFOA was 1,754 ng/mL, the mean lithium heparin plasma concentration of PFOA was 1,741 ng/mL, and the mean EDTA plasma concentration of PFOA was 1,743 ng/mL. The mean concentration of PFOA in lithium heparin whole blood was 898 ng/mL. The mean concentration of PFOA in EDTA whole blood was 898 ng/mL.

The mean serum concentration for PFHS was 64 ng/mL, the mean lithium heparin plasma concentration for PFHS was 59 ng/mL, and the mean EDTA plasma concentration of PFHS was 61ng/mL. The mean concentration of PFHS in lithium heparin whole blood was 25 ng/mL. The mean concentration of PFHS in EDTA whole blood was 25 ng/mL.

Plasma to serum values match very closely, on the case of PFOA and PFOS the mean difference between serum and plasma results is 3 percent. The mean differences between whole blood and serum results for PFOA and PFOS were 51 percent and 44 percent respectively.

Polyfluoro and perfluorinated carboxylates in North American precipitation

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The perfluorinated carboxylates (PFCAs) have been detected in a number of environmental matrices, including wildlife, water and sediments, but there is only limited information on their presence in atmospheric samples.. Recently, fluorinated telomer carboxylates (FTCAs) and fluorinated telomer unsaturated carboxylates (FTUCAs) have been shown to be the product of atmospheric decomposition of fluorinated telomer alcohols and have been detected in precipitation. We analysed precipitation samples from 7 sites across North America for perfluorocarboxylates by using a derivatization method to measure for the 2,4-difluoroanilide using GC-MS. Samples for the 4 American sites (New York, Delaware, Maryland and Vermont), situated in the urban north-east of the United States, were archived 1998 and 1999 samples. The 1999 American samples were investigated for the FTCAs and FTUCAs. The 3 Canadian sites were located in rural locations (Saturna Island, Algoma and Kejimkujik). Samples from the Canadian sites had low concentrations of PFOA and PFNA, with the central site having the highest concentration values compared with the other sites located on both coasts. The American sites exhibited significantly higher concentrations with greater frequency of occurrence of PFCAs, with the highest concentrations determined in the Delaware samples (85 ng/L PFOA; 77 ng/L PFNA). The other American sites had concentration values 10x greater than the Canadian sites. FTCAs and FTUCAs were detected at the American sites, most often at the Delaware site and less frequently at the Maryland and Vermont sites. No 10, 2- FTUCA was detected. Concentrations of these compounds were generally below 0.5 ng/L. The results confirm that there are significant precipitation fluxes of PFCAs (e.g. 50 -1200 ng/m2 PFOA at the Delaware site) with higher deposition near urban areas.

Detection of perfluoro-alkyl compounds (PFCs) in sewage treatment plant (STP) effluents and biosolids by liquid chromatography - tandem mass spectrometry

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The same chemical properties which contribute to the wide spread use of perfluoro-alkyl compounds (PFCs), stability under extreme heat and chemical stress and ability to reduce surface tension imparting oil and water repellency, also contribute to their environmental significance and persistence. PFCs, including perfluorooctane sulfonate (PFOS, $C_8F_{17}SO_3^-$) and perflurooctanoic acid (PFOA, $C_7F_{15}CO_2^-$), have been detected in human serum, various surface waters, sediments and dust in both industrialized and remote locations throughout the world. Even though the occurrence of these compounds in the environment has been established, there is limited information on the actual sources of these chemicals to the environment. Previous studies have identified sewage treatment plant (STP) effluents and biosolids as sources of various contaminants to the environment including PFCs. STP final effluents and biosolids were tested for the presence of perfluoro-alkyl compounds.

Biosolids samples were processed using a modified Hansen method. Final effluent samples were processed solely in Whatman Mini-UniPrep syringeless polypropylene filter devices to allow direct injection. Labelled internal standards ($^{13}C_2$-PFOA, $^{13}C_4$-PFOA and $^{13}C_2$-PFDA) were used as internal standards to monitor method performance and compensate for matrix effects / instrument variability. Reagent blanks and spikes were processed with each batch of samples. Instrument analysis was done using a Waters LC - Micromass Quattro Micro (biosolids) or LC - MDS Sciex 4000QTrap (effluents) triple quadrupole mass spectrometer operated in negative electrospray ionization (ESI) MRM mode. Chromatographic separations were performed on a 50mm x 2.1mm x 4um Jones Genesis C8 analytical column. The MRM transitions used for quantification were $[M\text{-}H]^- \rightarrow [SO_3]^-$ or $[FSO_3]^-$ for perfluorinated sulfonates and $[M\text{-}H]^- \rightarrow [M\text{-}COOH]^-$ for perfluorinated carboxylates.

A direct sample introduction LC-MS/MS method developed for part-per-trillion (ng/L) testing of natural waters was applied to the testing of STP final effluents. PFOS and PFOA were detected in all the STP final effluents tested at concentrations of 17 – 100 ng/L and 10 – 34 ng/L, respectively. Results indicate selected PFCs survive or are created in the STP treatment process and are subsequently discharged into ambient waters. PFCs were detected in all sewage treatment plant and paper fibre biosolids tested at parts-per-billion (ng/g) levels. PFCs profiles in the biosolids tested were dominated by PFOS but differences in PFCs patterns could be observed between sources. Paper fibre biosolids had much higher percentages of perfluorocarboxylic acids. No definitive statements with regard to the correlation between PFCs content of STP final effluents and STP biosolids could be made because samples were obtained from the same locations but at different dates. Biosolid samples were also analysed by GC-HRMS for other persistent organic pollutants (POPs) including polychlorinated dioxins and furans (PCDD/Fs), dioxin-like polychlorinated biphenyls (DLPCBs) and brominated diphenyl ethers (BDEs). No correlation between the POPs and PFCs results could be determined although PFCs appear to contribute significantly to the POPs content of the STP biosolids.

The 1st worldwide interlaboratory study on perfluorinated compounds in environmental and human samples

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The 1st PFC interlaboratory study reported here was carried out in 2005 to assess, and if needed to improve, the current performance of the analytical techniques applied by laboratories world wide[1]. The study covers environmental matrices (brackish water, fish liver extract and muscle tissue of pike perch), a standard solution, and human blood (plasma and whole blood). Laboratories were asked to use any method they preferred, and to analyze any number of PFCs mentioned below. Twenty seven laboratories submitted results for the environmental matrix part. Nineteen laboratories signed up for the human matrix part. The PFCs dealt with in this study were perfluorobutanesulfonate (PFBuS), perfluorohexanesulfonate (PFHxS), perfluorooctanesulfonate (PFOS), perfluorodecane-sulfonate (PFDS), perfluorohexanoate (PFHxA), perfluoroheptanoate (PFHpA), perfluoro-octanoate (PFOA), perfluorononanoate (PFNA), perfluorodecanoate (PFDA), perfluoro-undecanoate (PFUnDA), perfluorododecanoate (PFDoDA), perfluorotetradecanoate (PFTDA) and perfluorooctanesulfonamide (PFOSA).

Clean-up methods included ion-pair extraction, different types of solid-phase extractions or only protein precipitation. Analysis and detection were LC-ESI-MS/MS (triple quadrupole), LC-ESI-MS/MS (ion trap), LC-ESI-MS (single quadrupole), LC-ESI-TOF-MS and GC-MS. Quantification was performed by using extracted or non-extracted standard curves with or without matrix or internal standard present.

Results have been reported for PFOS and PFOA from most of the laboratories. Data on the other perfluorinated compounds have been reported to a lesser extent. Table 1 shows a summary of results of PFOS and PFOA in the study standard and the environmental samples. For both compounds the study standard showed the best results, followed by the cleaned fish extract and the fish tissue. In the latter case, the extraction and clean-up contributes approximately 2 times the variation caused by the LC-MS determination (the standard) only. For both compounds the study standard showed the best results, followed by the cleaned fish extract and the fish tissue. This shows clearly that laboratories need to improve their analysis, with special attention for the extraction and clean-up of tissue materials.

Almost all 17 laboratories reported values for PFOS and PFOA. The CV for PFOS was lower (CV~30%) for plasma and higher (CV~60%) for whole blood. Corresponding results for PFOA were ~50 and ~40%. A lower number of participants reported levels for PFNA and PFHxS and the variations were higher compared to PFOS and PFOA. For PFOSA, PFBS, PFDS, PFDA, PFHpA and PFUnDA, the very low levels hampered the detection of these compounds and a considerable number of data was <LOQ. Table 2 shows a summary of the results for PFOS and PFOA in the human plasma and whole blood samples. Mean, median and variability (CV%) are given if a sufficient number of participants (>50%) reported values > non-detect (ND). Considering the relatively new analytical area and the number of different methods used, the variation between participating laboratories is satisfactory. Concentrations of approximately 10-20 ng/ml for PFOS and 2 ng/ml for PFOA in whole blood and plasma were determined reasonably well by most laboratories.

Examination of dietary exposure to polyfluorinated compounds via consumption of traditional foods

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The routes of human exposure to perfluorinated compounds (PFCs) have not been well-characterized. One possible route is the consumption of foods containing PFCs. Past work on PFCs in Arctic biota[1;2] demonstrates that these compounds are present in the Arctic environment, and in some traditional foods consumed by northern populations. This initial study was performed to examine the dietary exposure of traditional food consumers to PFCs. Portions of liver consumed as traditional food were analyzed for five perfluorinated carboxylates (PFCAs), one perfluorinated sulfonate, three perfluorooctanesulfonamides (PFOSAs), three fluorinated telomer carboxylates (FTCAs), and three unsaturated fluorinated telomer carboxylates (FTUCAs).

Liver samples (20) from Arctic char, burbot, caribou, loche, ringed seal, and walrus were collected in Nunavut between 1997-99. Samples were stored at -20oC prior to analysis. The samples were extracted using a newly developed solvent extraction method and analyzed using LC-MS/MS[3]. A portion of liver was spiked with recovery internal standards, homogenized with methanol and centrifuged to obtain supernatant. The extraction was repeated twice more, and the supernatants were combined and reduced in volume using N_2. Aliquots of methanol taken through the procedure were used as method blanks. Instrument performance internal standards were added, and the final solution was centrifuged. A portion of the final solution was transferred to a polypropylene autosampler vial prior to injection on the LC-MS/MS. All PFCs were quantitated using external standard solutions made up in methanol. The ringed seal liver contained the greatest amount of PFCs (ΣPFC = 103 – 313 ng/g wet weight), followed by walrus (ΣPFC = 40 – 76 ng/g wet weight), caribou (ΣPFC = 13 – 86 ng/g wet weight), and fish livers (ΣPFC = 5 – 69 ng/g wet weight). This rank order in ΣPFA concentrations is driven by PFOS concentrations, which are approximately 2 to 10 times higher in ringed seal liver compared to samples from other species. PFCAs were also frequently detected, and were found at the highest levels in caribou and walrus (mean ΣPFCA = 39 ng/g wet weight). Perfluorooctanesulfonamide and N-ethylperfluorooctanesulfonamide were the two PFOSAs detected in the samples. They were only observed in walrus (mean 2 ng PFOSA/g wet weight), ringed seal (mean 1 ng PFOSA/g wet weight), and the one Arctic char liver sample (N-EtPFOSA, 153 ng/g wet weight). FTUCA/FTCAs were not detected in any of the 20 liver samples.

The results of this pilot study indicate that consumers of liver are exposed to PFCAs, PFOS, and some PFOSAs. Consumption of ringed seal liver will lead to the greatest dietary exposure to PFCs, followed by caribou and walrus liver. However, since liver is not one of the main traditional food items consumed, a wider variety of food items must be analyzed to obtain a better picture of the dietary exposure of traditional food consumers to PFCs.

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Polyfluorinated compounds in the home: levels in air and dust and human exposure

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Polyfluorinated compounds such as perfluorinated alkyl sulfonamides (PFASs) and polyfluorinated telomer alcohols (FTOHs) are used in a variety of consumer products for surface protection. These chemicals were investigated through a comprehensive survey of indoor air, house dust and outdoor air in the city of Ottawa, Canada during the winter of 2002/03. Homes were selected randomly to avoid biases. Passive air samplers consisting of polyurethane foam (PUF) disks were calibrated and used to conduct the indoor (n=59, 21 day exposure) and outdoor (n=7, 70 day exposure) survey. Samples were analyzed for several PFASs including N-methylperfluorooctane sulfonamidoethanol (MeFOSE), N-ethylperfluorooctane sulfonamidoethanol (EtFOSE), N- ethylperfluorooctane sulfonamide (EtFOSA) and N-methylperfluorooctane sulfonamidethylacrylate (MeFOSEA). Indoor air concentrations for MeFOSE and EtFOSE (1490 pg m-3 and 740 pg m-3, respectively) were about 10 to 20 times greater than outdoor concentrations, establishing indoor air as an important source to the outside environment. EtFOSA and MeFOSEA concentrations were lower in indoor air (40 pg m^{-3} and 29 pg m^{-3} respectively) and below detection in outdoor air samples. FTOHs were not analyzed in passive samplers due to methodology issues which are now being addressed. Indoor dust samples (n=59) were collected from individual vacuum cleaners from the same homes surveyed for air concentrations. Highest dust concentrations were recorded for MeFOSE and EtFOSE with geometric mean concentrations of 110 and 120 ng g^{-1} while concentrations for EtFOSA and MeFOSA were below detection and 7.9 ng g^{-1} respectively. Air and dust concentrations for MeFOSE and EtFOSE were correlated. FTOHs (6:2, 8:2 and 10:2) were detected in all dust samples with geometric mean values (ng g^{-1}) of 33, 55 and 35 respectively. Results for 4:2 FTOH are not reported due to poor method recoveries. A human exposure assessment for PFASs based on median air and dust concentrations revealed that human exposure through inhalation (100% absorption assumed) and dust ingestion were ~40ng d^{-1} and ~20ng d^{-1} respectively. However, for children the dust ingestion pathway was dominant and accounted for ~44ng d^{-1}. For FTOHs (6:2, 8:2 and 10:2) the ingestion pathway for children was 4.9, 8.0 and 4.6ng d^{-1} respectively. These findings confirm the important role of indoor environments (homes) as i.) sources of PFASs and FTOH to the outside and ii) key sources for human exposure to these chemicals.

Synthesis, purification and characterization of phosphate fluorosurfactants

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Paper food wrappers are commonly coated with nonpolymeric fluorosurfactants as protection from contact with both water and oil. A specific class of compounds used in this application are phosphate fluorosurfactants, with phosphate anionic head groups and telomer based fluorinated tails (fluorotelomer alcohols (FTOH), $F(CF_2)_nCH_2CH_2OH$). The phosphate linkage of these surfactants is available for hydrolysis and if broken would release FTOHs into the environment. FTOHs have been observed in the North American atmosphere at 10-100 pg m^{-3} levels. Considering nonpolymeric fluorosurfactants account for about 20% of the 12 million kg/year production of fluorinated compounds, the degradation of phosphate fluorosurfactants could represent a significant point source for release of FTOHs into the environment.

The degradation of a specific phosphate fluorosurfactant, Zonyl FSE, was quantified by monitoring FTOH production under environmentally relevant conditions. This investigation was complicated by residual FTOHs that remain in the product after manufacture. As per Dinglasan *et al,*, purified air was purged through an aqueous solution of Zonyl FSE to remove the residual FTOHs before introducing any stress on the molecule. The entire suite of FTOHs from the 4:2 to the 24:2 was observed as residuals in industrial product. Although those of chain lengths above 10 perfluorinated carbons could not be quantified using the reference compounds available, they have tentatively been identified using GC retention times and 3 mass transitions in both NCI-MS and PCI-MS.

Despite attempts to remove residual FTOHs, their presence severely hindered any quantification of product degradation. To overcome the complexities of working with the commercial product, fluorosurfactants akin to Zonyl FSE were synthesized and purified. Reported here is a novel synthetic scheme producing tri-, di- and mono-substituted 8:2 FTOH phosphate fluorosurfactants. The products were subsequently separated by liquid column chromatography and the structures confirmed using ^{31}P-NMR, ^{1}H-NMR and ESI-MS/MS.

Toxicology

TOX001 Hanson	Microcosm evaluation of the toxicity and risk to aquatic macrophytes from perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS)
TOX002 Harada	Effect of PFOS and PFOA on L-type Ca^{2+} current in guinea pig ventricular myocytes
TOX003 Yin Yueng	Gene expression profiles in rat liver treated with pentadecafluorooctanic acid (PFOA)
TOX004 Stevenson	Tissue concentrations of perfluorochemicals and their inhibitory effect on multi-drug transporters of the mussel, Mytilus californianus
TOX005 Endo	Mutagenicity of perfluorooctane sulfonate (PFOS) and related compounds
TOX006 Fasano	Pharmacokinetics of 8-2 fluorotelomer alcohol in the rat
TOX007 Van de Vijver	Toxicity identification of perfluorinated compounds in the Western Scheldt Estuary
TOX008 MacDonald	Aquatic toxicity of fluorinated telomer acids
TOX009 Yoo	Separation and characterization of structural isomers of perfluorinated compounds
TOX010 Martin	Metabolic products and pathways of fluorotelomer alcohols in isolated rat hepatocytes
TOX011 Maras	Estrogen-like properties of fluorotelomer alcohols as revealed by MCF-7 breast cancer cell proliferation
TOX012 McGregor	Microcosm assessment of the toxicity of 10:2 fluorotelomer saturated acid to aquatic macrophytes
TOX013 Loveless	Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO)
TOX014 Kennedy	Dermal penetration of APFO through rat and human skin

TOX015 Beach	Comparison of avian reproduction and tissue uptake with exposure to perfluorooctane sulfonate or perfluorobutane sulfonate
TOX016 Olsen	Urinary bladder endpoints in workers and rats exposed to perfluorooctanesulfonyl fluoride (POSF) and related compounds
TOX017 Olsen	Evaluation of the half-life ($t_{1/2}$) of elimination of perfluorooctanesulfonate (PFOS), perfluorohexanesulfonate (PFHS) and perfluorooctanoate (PFOA) from human serum
TOX018 Tanaka	Thyroid hormone status in adult rats given oral doses of perfluorooctanesulfonate
TOX019 Vanden Heuvel	Differential activation of nuclear receptors by normal and perfluorinated fatty acids: a comparison of human, mouse and rat PPARńńń LXRńand RXRń
TOX020 Lau	Evaluation of perfluorooctane sulfonate (PFOS) in the rat brain
TOX021 Frame	Pathology working group review of mammary glands from chronic feeding study in rats with APFO
TOX022 Abedi-Valugerdi	Deleterious effects of perfluorooctane sulfonate (PFOS) on the immune organs of mice
TOX023 Rahman Qazi	Local activation of the innate immune system of mice by perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA)
TOX024 Shabalina	Comparison of the effects of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) on wild-type mice and knock-out animals lacking uncoupling protein-1 (UCP-1)
TOX025 Young	Ecotoxicity assessment & remediation of firefighting training wastewater by air - sparged hydrocyclone (ASH) system

Microcosm evaluation of the toxicity and risk to aquatic macrophytes from perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS)

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Perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) are anthropogenic contaminants detected globally in aquatic ecosystems, but for which little ecotoxicological data is currently available. This paper will describe research characterizing effects and estimating risk of these compounds to aquatic macrophytes under field conditions. The toxicity of PFOA and PFOS to *Myriophyllum sibiricum*, *M. spicatum* and *Lemna gibba* were investigated in two separate studies using 12,000 L outdoor microcosms that are designed to mimic small ponds. Replicate microcosms (n=3) were treated with the salt forms of PFOA or PFOS at various concentrations, plus controls, and plants were assessed for a suite of endpoints at regular intervals over 35 and 42 days, respectively. The two species of *Myriophyllum* were similar in their sensitivity to PFOA, but *M. sibiricum* was more sensitive than *M. spicatum* to PFOS exposure, while *L. gibba* was less sensitive than both species to these compounds. PFOA toxicity was observed in the evaluated endpoints at >6 mg/L for EC_{10}s and >32 mg/L for EC_{50}s for *M. spicatum* and in *M. sibiricum* at >8 mg/L for EC_{10}s and >36 mg/L for EC_{50}s. *Lemna gibba* toxicity was >21 mg/L PFOA for EC_{50}s, but was undefined at lower EC values due to the lack of an unambiguous concentration-response. PFOS toxicity was observed in the evaluated endpoints at >3 mg/L for EC_{10}s and >12 mg/L for EC_{50}s for *M. spicatum* and in *M. sibiricum* at >0.1 mg/L for EC_{10}s and >1.6 mg/L for EC_{50}s. *Lemna gibba* toxicity was observed at >19.1 mg/L PFOS for EC_{50}s. Except at the highest concentrations tested (100 mg/L PFOA and 30 mg/L PFOS), plant growth and development continued throughout the study period, implying that recovery of populations impacted by these fluoro-compounds is possible if the toxicant is removed. A probabilistic risk assessment for these plants indicated negligible toxicity from PFOA or PFOS exposure at current environmental concentrations.

Effect of PFOS and PFOA on L-type Ca^{2+} current in guinea pig ventricular myocytes

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Perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) are amphiphiles found ubiquitously in the environment including in wildlife and in humans and are known to have toxic effects on various tissues and organs. Mechanisms of toxic effects, however, remains unknown. The amphiphilic nature of PFOS and PFOA may suggest that their effects could primarily be associated with the function of cell membranes.

We investigated the effect of PFOS and PFOA on the action potential and L-type Ca^{2+} current ICaL in isolated guinea-pig ventricular myocytes using whole cell patch-clamp recording. Ventricular cells were isolated from hearts of the guinea-pig (300 – 400 g body weights) using the enzymatic dissociation technique (Shinbo and Iijima, 1997).

In current clamp experiments, PFOS decreased the rate of spike and action potential duration and peak potential significantly at doses over 10 μM. Under conventional whole-cell voltage-clamp conditions using the standard internal solution and normal Tyrode solution, exposure of the cell to PFOS at 10μM markedly decreased the inward peak of the current at the test potentials positively more than –20 mV from the holding potential at –40 mV. We investigated the effects of PFOS on ICaL with a use of Na^+, K^+-free external solution and Cs^+-rich pipette solution. It was found that the effects of PFOS on ICa was voltage-dependent. PFOS decreased ICaL amplitude in response to a depolarizing pulse to 0 mV from –40 mV although increased it when the holding potential was set to –90 mV. The EC50 value of PFOS was calculated to be 6.3 ± 1.4 μM. To address the voltage-dependent nature of the effects of PFOS, the steady-state activation and inactivation of ICaL were examined. PFOS shifted the voltage showing maximum ICaL amplitude toward the negative potentials with EC50 value, 5.2 ± 2.2 μM, respectively. The effects of PFOS on inactivation were measured by a double-pulse protocol. The extent of inactivation induced by prepulse increased in presence of PFOS with EC50 value, 11.9 ± 7.2 μM. PFOA had effects similar to those of PFOS but significantly lower potency than PFOS.

These findings agreed with previously reported observation with non-fluorinated anionic n-alkyl surfactants (Post et al., 1991). PFOS and PFOA may change membrane surface potential, eliciting general effects on calcium channels. It is reported that the serum levels of PFOS and PFOA were about 10^{-2} μM and 0.5 x10^{-2} μM in the general population (Harada et al., 2004). In contrast, much higher levels (μM; geometric mean and range) were reported in workers: 1.82 (0.12-20.1) for PFOS and 2.73 (range 0.10-30.7) for PFOA, respectively (Olsen et al., 2003). Thus, the calcium channel is a realistic toxicological target of these chemicals in workers while it remains unknown in the general population. These findings provide a clue to elucidate mechanisms of toxicity of PFOA and PFOS.

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Gene expression profiles in rat liver treated with pentadecafluorooctanic acid (PFOA)

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Recently, gene expression profiles in rat responsive to perfluorooctane sulfonate (PFOS) exposure was reported by GeneChip analysis, however, no such data are available for perfluorooctanoic acid (PFOA). Therefore, we investigated gene regulation in rats dosed with different concentrations of PFOA by oral gavage to determine of the responses of animals were similar for PFOS and PFOA. Microarray techniques were used to compare gene expression profiles in liver of the PFOA fed rats with control rats in order to identify changes in specific genes and biochemical pathways in rats resulting from PFOA exposure.

7-week-old male Sprague-Dawley rats were obtained from Japan CLEA Inc (Tokyo, Japan) and housed at 20-24ºC in humidity-controlled (40-60%) facilities at the National Institute of Animal Health, Japan. After acclimatization to a standard diet for 1 week, rats (225 – 250 g) were given a single oral gavage of different concentration of PFOA (1mg/kg, 3mg/kg, 5mg/kg, 10mg/kg, 15mg/kg), or vehicle control (0.5% Tween-20) daily at a rate of 1ml/kg body weight for 21 days. At the end of the exposure, livers were removed and frozen in liquid nitrogen and stored at -80ºC until RNA isolation.Six chips of Rat Genome 230 2.0 array were purchased from Affymetrix and microarray analysis was carried out following manufacturer recommended procedures. Microarray Suite (MAS) ver. 5.0 and GeneChip Operating Software (GCOS) were used for gene expression analysis.

Over 500 genes were significantly ($P<0.0025$) altered by PFOA. The 10mg/kg PFOA treatment altered the expression pattern of the greatest numbers (over 800) of genes while 1mg/kg altered the least number (501). 106 genes and 38 genes were determined to be up- and down-regulated, respectively, and they showed consistent alternations of gene expression in all treatments. Specifically, KEGG pathways of fatty acid synthesis, fatty acid degradation, mitochondrial fatty acid beta-oxidation and cholesterol synthesis were found to be altered significantly ($P<0.0025$) by exposure to PFOA.

Genes associated with lipid or fatty acid metabolisms were greatly altered by in rats treated with all concentrations of PFOA, and some of the genes were linked with pathways of peroxisomal and mitochondrial fatty acid beta-oxidation. In mammals, the mitochondria oxidize short, medium and some long chain fatty acids, while peroxisomes oxidize some long chain and very long chain fatty acids. As PFOA has a similar structure as fatty acids except that the hydrogen atoms linked with carbon atoms in fatty acids were replaced by fluorine atoms in PFOA. Therefore, there might be a possibility that the PFOA could be mistaken by the fatty acid metabolism machinery as a substrate because of the structural similarity of PFOA to endogenous fatty acids. The induced enzymes for fatty acid oxidation might increase the normal oxidation of fatty acids and might disrupt the normal balance of fatty acid metabolism in mammals.

Tissue concentrations of perfluorochemicals and their inhibitory effect on multi-drug transporters of the mussel, Mytilus californianus

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Among the great number of emerging chemical pollutants, perfluorochemicals (PFCs) rank among the most stable and globally pervasive. The environmental fate for many PFCs is transport to the ocean and accumulation in marine sediments and ultimately marine organisms. In this study, we examined the inhibitory effect of PFCs on the cellular multidrug p-glycoprotein (p-gp) transporter. This transporter is part of the ATP Binding Cassette super-family of cellular membrane spanning proteins specified for transport. This transporter has been identified in human tissue as well as in many aquatic organisms, such as the marine mussel, Mytilus californianus; it has been suggested as aquatic organisms' first line of defense against xenobiotics.Transporter proteins bind a large range of small moderately hydrophobic compounds and export them from the cell using ATP. This low specificity binding is advantageous, as the transporter recognizes many toxins; however, low specificity also enables the transporters to be overwhelmed easily in the presence of multiple chemical substrates. This diversion of the transport activity by non-toxic substrates or by multiple substrates is called chemosensitization. In this way, even non-toxic substances can cause negative effects on a cell by diverting the transporters' activity away from toxic substrates.

We find that four of the eleven PFCs studied cause significant inhibition of the p-gp transporter in *Mytilus californianus* gill tissue: perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), and perfluorohexane sulfonate (PFHxS). Inhibition is maximal for PFNA and PFDA, which decrease the transporter activity to less than half the basal level. From dose-response curves, inhibitory concentration (IC10 and IC50) values were determined as a measure of inhibitory potency. The average IC50 values for PFNA and PFDA were 8.6 and 10.0 micromolar respectively.

Using Western-Blot analysis, we find that PFNA and PFDA cause a significant induction of the transporter protein over two days after a two-hour exposure. Induction of the transporter is considered a stress response and requires energy; therefore, independent of the toxicology of these PFCs, their simple presence creates a metabolic cost, which could cause long-term detrimental effects on marine organisms living in polluted areas.

Extraction of homogenized mussel gill tissue samples followed by analysis using high performance liquid chromatography coupled with tandem mass spectrometry (LC/MS/MS) has revealed that *Mytilus californianus* collected in Monterey Bay, CA have on average 2.0 (± 0.3) ng/g (wet weight) PFNA in the gill tissue. The detection of PFNA in mussel gill tissue, where the p-gp transporter is localized, along with its role as a transporter inhibitor, makes this specific PFC of particular concern.

Although generally aquatic PFC concentrations may not be acutely toxic, this study raises the concern of the metabolic costs of dealing continuously with a cellularly recognized xenobiotic, as well as the chemosensitization of cells. Research showing that PFCs bioaccumulate and biomagnify in the food web, in conjunction with the fact that all mammals including humans share cellular transporters as a first line of defense, adds a new dimension of concern for the toxicology of PFCs.

Mutagenicity of perfluorooctane sulfonate (PFOS) and related compounds

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Perfluorooctane sulfonate (PFOS) is one of perfluorinated compounds (PFCs) which have been manufactured for over 50 years and have been widely used as surfactants and surface protectors in carpets, leather, paper, food containers, fabric, and upholstery and as performance chemicals in products such as fire-fighting foams, floor polishes, and shampoos (1). These compounds have recently been detected in environment, in animals and in human serum. Even though the environmental fate of PFOS is not fully understood, the 3M Co., one of the largest producers of PFCs, ceased most of production of PFOS in 2000 due to the widespread occurrence and potential toxicities of PFCs. In the present study, Salmonella mutagenicity of PFOS and related compounds has been demonstrated.

Eight PFCs (PFOS potassium salt, perfluorobutyric acid, perfluoropentanoic acid, perfluorohexanoic acid, perfluoroheptanoic acid, perfluorooctanoic acid, perfluorononanoic acid and nonafluoro-1-butane sulfonic acid) has been investigated. Mutagenicity test was conducted by preincubation method which was a modification of Ames method using Salmonella typhimurium TA100 and TA98 under the conditions of both with and without a metabolic activation system (S9mix).

From the result of PFOS, increase of revertant colonies were observed only for TA98 without S9mix although not reach for twice of spontaneous plate at least up to 5000 micro gram dose per plate. Neither increasing revertant colonies nor killing response has been observed for TA100 strain (both with and without S9mix) and for TA98 with S9mix. Other 7 related compounds showed no mutagenicity for both tester strains with and without S9mix, some of which showed clear killing nearby maximum dose (5000 micro gram or 10 micro liter per plate) on this demonstration. It was suggested that 8 PFCs have no or very weak mutagenicity for Salmonella strains.

(1) OECD. Draft assessment of perfluorooctane sulfonate (PFOS) and its salts: Complete assessment, ENV/JM/RD (2002) 17; September 2002.

Pharmacokinetics of 8-2 fluorotelomer alcohol in the rat

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Pharmacokinetic studies were conducted to elucidate mammalian metabolism of [3-^{14}C] 8-2 Fluorotelomer Alcohol, $C_7F_{15}{}^{14}CF_2CH_2CH_2OH$. *In*-vivo oral absorption, distribution, metabolism and excretion (ADME) and dermal absorption in the rat followed by comparative human and rat *in-vitro* dermal absorption kinetics and metabolism in hepatocytes studies have been completed. The results of these studies will be discussed.

The majority of test substance was eliminated unchanged following a 5 and 125 mg/kg single oral dose to male and female rats. The major metabolites in bile were glutathiones and glucoronide. Minimal ^{14}C was eliminated in urine or present in tissues. Approximately one-percent of the administered ^{14}C was transformed to PFOA. Approximately 53% and 27% of the administered dose was absorbed, respectively. The plasma kinetics following oral dosing was low and short-lived. The peak concentration of 8-2 Fluorotelomer Alcohol in plasma following oral administration at 5 and 125 mg/kg was not proportional to the dose and the time to peak concentration was slightly longer at the high dose level indicating that absorption was saturated. For female rats, there was a proportional increase in the systemically-available dose of 8-2 Fluorotelomer Alcohol at the 125 mg/kg dose level, as indicated by the area under the curve (AUC). Following a 5 mg/kg single oral dose, 8-2 Fluorotelomer Alcohol was conjugated to glucuronic acid and metabolized to glutathione conjugates that were eliminated in the feces via the bile, and accounted for approximately 42% of the administered dose. At the 125-mg/kg-dose level, the percent of administered dose eliminated in the bile was lower (~20%). Independent of dose level, elimination via the urine was a minor pathway (<4%), which contained low levels of perfluorooctanoic acid that accounted for approximately 0.48-1.16% of the administered dose. At 7 days post-dose, residual radioactivity remaining in tissues in rats at the low dose (~7%) and high dose (~4%) was generally greater than whole blood, with the highest concentrations in fat, liver, thyroid, and adrenals.

Following a 6-hour dermal exposure, 8-2 Fluorotelomer Alcohol either volatized from the skin surface or was removed by washing and was not absorbed. Although a portion of the applied dose remained in the skin at 7 days post-dose (<1%), systemic absorption following dermal exposure to 8-2 Fluorotelomer Alcohol was negligible.

Clearance of 8-2 Fluorotelomer Alcohol *in vitro* was five times faster in rat hepatocytes than human hepatocytes, suggesting that *in vivo* hepatic first-pass clearance of 8-2 Fluorotelomer Alcohol may be more extensive for rats than for humans. Metabolism of 8-2 Fluorotelomer Alcohol in primary rat hepatocytes was reflective of that observed *in vivo*. Also, when compared to rat hepatocytes, the metabolic profile for human hepatocyte, though less extensive, suggests that metabolism of 8-2 Fluorotelomer Alcohol was comparable.

Toxicity identification of perfluorinated compounds in the Western Scheldt Estuary

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Estuarine and coastline ecosystems are of major economic and ecological importance, but due to large industrial and domestic waste water discharges, the structure and functions of these habitats are often disturbed. Like other estuarine areas, the Western Scheldt estuary, situated in the southwest of the Netherlands and the north of Belgium, is a highly polluted ecosystem. Until now attention was primarly focused on organochlorines, pesticides and heavy metals because of their persistent character and ability to accumulate in aquatic organisms.

As recent studies have indicated that fluorinated organic compounds (FOCs), and especially perfluorooctane sulfonate (PFOS), occur worldwide in the environment and show high persistence, there is an urgent need to characterize the distribution patterns of fluorinated organochemicals in the western part of Europe.

The first aim of this study was to further evaluate the magnitude of exposure in fish biota from the Western Scheldt estuary and North Sea. A pilot study, conducted two years ago, already gave an indication of a pollution gradient along the Western Scheldt (Van de Vijver et al., 2003). One of the largest fluorochemical production plants is located near Antwerp, Belgium, and is thought to be a potential source of PFOS in the estuary. The second aim of this study was to link data obtained trough field work to experimental work in the laboratory. For this purpose, a recently established technique for differential gene expression, Suppression Subtractive Hybridization-Polymerase Chain Reaction (SSH-PCR), has been used in combination with cDNA microarrays.

During sampling campaigns in the winter of 2004-2005, we collected the European sea bass (*Dicentrarchus labrax*) at seven different locations along the Western Scheldt using traps. Liver tissue was taken for PFC analysis and for toxicity identification using microarrays. Blood samples were investigated for some general parameters. Liver samples were extracted with methanol and tert-butyl methyl ether and concentrations of PFOS were determined using high performance liquid chromatography tandem mass spectrometry (HPLC/MS/MS).

All samples that were analysed, contained detectable concentrations of PFOS (>10 ng/g). Concentrations ranged from 269 ng/g wet wt in fish trapped close to the North Sea, to PFOS concentrations of 3800 ng/g wet wt in sea bass originating from close to Antwerp. Although sea bass is a fish species with quiet abroad living range, a clear pollution gradient could be observed along the estuary with the highest PFOS levels close to the city of Antwerp. Other PFCs, like several perfluorinated carboxylic acids with varying chain lengths (C9-C12), were detected sporadically, with the highest concentrations measured in fish with the highest PFOS levels.

A custom made microarray has been hybridised with RNA isolated from sea bass from the different locations. Several genes, e.g. genes of the CYP450 complex, were identified to be PFOS responsive in the European sea bass. The results of this study will be discussed in the context of the environmental hazard of FOCs for marine and estuarine ecosystems. Of course, it is necessary to take different confounding factors, like the presence of other (unknown) chemicals, salinity and temperature, into account when discussing field data.

Van de Vijver et al., 2003. Environm. Toxicol. Chem. 22, 2037-2041.

Aquatic toxicity of fluorinated telomer acids

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The recent identification of fluorotelomer alcohols (FTOHs) as a probable atmospheric source of the globally recalcitrant perfluorinated carboxylic acids (PFCAs) has spurred considerable interest in elucidating the chemical pathways of FTOH degradation to PFCAs. However, little attention has been paid to the resulting intermediate products themselves. Biotic and abiotic oxidation of the FTOHs leads to the production of alpha,ß-saturated and alpha,ß-unsaturated fluorotelomer carboxylic acids (FTCAs). The lower volatility and higher water solubility of the FTCAs compared with their FTOH counterparts suggest surface waters as a likely a repository for the FTCAs. While environmental fate and toxicity data exist for the PFCAs, no such information has been documented for the FTCAs.

The toxicity of the saturated (s) and unsaturated (u) forms of the 4:2, 6:2, 8:2 and 10:2 FTCAs was assessed in the laboratory on three common freshwater species: the pelagic microcrustacean *Daphnia magna*, the benthic macroinvertebrate *Chironomus tentans*, and the floating macrophyte *Lemna gibba*. Acute toxicity studies indicated that all three species were most sensitive to FTCAs with chain lengths ≥ 8 fluorocarbons (FCs). *L. gibba* was the most sensitive of the three to FTCAs of chain lengths ≤ 8 FCs, with EC_{50} values for growth ranging from 0.71 - 10.04 mg/L for the 8:2 and 6:2 u-FTCAs, respectively. *D. magna* was the most sensitive to FTCAs of chain lengths > 8 FCs with EC_{50} values for immobility of 0.025 and 0.279 mg/L for the 10:2 s-FTCA and u-FTCA, respectively. For all three species, toxicity increased with increasing chain length from 6 to 8 FCs. This trend continued for *D. magna* through FC chain lengths of 10, but not for *C. tentans* or *L. gibba*. In fact, the 8 FC chain length was the inflection point beyond which toxicity decreased for *C. tentans* and *L. gibba*. The s-FTCAs were generally more toxic than corresponding u-FTCAs, with the exception of the 8:2 FTCA for *L. gibba* and the 10:2 FTCA for *C. tentans* and *L. gibba*.
A subsequent 60-d chronic life cycle assay on *C. tentans* with the 8:2 s-FTCA resulted in toxicity thresholds for growth and mortality 5-6 times smaller than those measured in the acute study. Adult emergence was the most sensitive endpoint in the life cycle assay with an EC_{50} of 0.44 mg/L.

Although the chain-length trends observed in our acute studies agree with those previously reported for the closely related PFCAs, the toxicity thresholds generated here are 1-4 orders of magnitude smaller for the FTCAs.

Separation and characterization of structural isomers of perfluorinated compounds

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The occurrence of perfluorinated compounds (PFCs) in the environment has been the subject of much recent research and concern for potential environmental effects. The presence of complex mixtures of PFCs with different physical-chemical and toxicological properties provides a challenge for risk assessment. While current analytical methods are able to separate and quantify different PFC compounds e.g. sulfonates from carboxylates and PFCs with different carbon chain lengths little attention has been paid to the separation of branched from straight chain isomers and in general standards used for PFC quantification have not been characterized for their isomer distribution. Similarly, all toxicological studies to date have been conducted using relatively crude commercial available PFC preparations. Using modifications of current PFC analytical methods we have separated a commercial PFOS mixture into three separate isomer peaks. The largest peak (peak III), presumed to be the straight chain isomer constitutes greater than 63 % of the materials mass while the earlier eluting peaks, peaks I and II constitute 2.5% and 34% of the mass respectively. The mixture also contained traces of PFOA and shorter chain sulfonates that were not quantified. Using a preparative HPLC column (150mm x 21.2mm) the straight chain peak was separated from the branched chain to greater than 99.5% purity. To assess the epigenetic toxicity of the purified isomers, a gap junction intercellular communication (GJIC) assay was used. GJIC plays a central role in maintaining tissue homeostasis, and chronic closure of gap junction channels has been associated with many human diseases including cancer. Previous *in vitro* and *in vivo* studies with this assay indicated that chain length, albeit mixtures of straight and branched chains, determined the toxic-potential of the PFC., The NOEL and EC50 values for a commercially available PFOS mixture were 3.1 µg/ml and 14.98 µg/ml, respectively.. In the current study the straight chain PFOS isomer was approximately equipotent, on a mass basis, at inhibiting GJIC compared to the commercial mixture. This indicates the different PFOS isomers have similar potency in the inhibition of GJIC. The potential for differences in compound potency in other assay systems due to isomer branching are currently being investigated. The ability to separate and quantify structural isomers of PFCs will be important for further risk assessment of PFCs.

Metabolic products and pathways of fluorotelomer alcohols in isolated rat hepatocytes

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Fluorotelomer alcohols (FTOHs; $CF_3(CF_2)_xC_2H_4OH$; where x=3, 5, 7, 9) are a novel class of polyfluorinated contaminants, recently detected in the North American atmosphere, that are possible precursors to the series of perfluoroalkyl carboxylates (PFCAs) in human blood. Although the magnitude of human exposure to FTOHs has not been assessed, their widespread distribution in ambient air warranted a comprehensive examination of their metabolic fate. An *in vivo* rat study validated earlier independent work that poly- and per-fluoroalkyl carboxylates were metabolites of FTOHs, but our detection of several novel metabolites prompted us to examine their pathways in greater detail using isolated rat hepatocytes. Using 8:2 FTOH (i.e. where x=7) as a model compound, the metabolic products formed by isolated rat hepatocytes were identified, and three synthesized intermediates were incubated separately to elucidate the metabolic pathways. For 8:2 FTOH a major fate was direct conjugation to form the *O*-glucuronide and *O*-sulfate. Using 2,4-dinitrophenylhydrazine (DNPH) trapping, the immediate oxidation product of 8:2 FTOH was identified as 8:2 fluorotelomer aldehyde (8:2 FTAL; $CF_3(CF_2)_7CH_2C(H)O$). 8:2 FTAL was transient and eliminated HF non-enzymatically to yield 8:2 fluorotelomer α/β-unsaturated aldehyde (8:2 FTUAL; $CF_3(CF_2)_6CF{=}CHC(H)O$) which was also short-lived and reacted with GSH and perhaps other endogenous nucleophiles. Four polyfluorinated acid intermediates were also detected, including 8:2 fluorotelomer carboxylate (8:2 FTCA; $CF_3(CF_2)_7CH_2C(O)O^-$), 8:2 fluorotelomer α/β-unsaturated carboxylate (8:2 FTUCA; $CF_3(CF_2)_6CFCHC(O)O^-$), tetrahydroperfluorodecanoate ($CF_3(CF_2)_6(CH_2)_2CO_2^-$), and dihydroperfluorodecanoate ($CF_3(CF_2)_6CH{=}CHCO_2^-$). The pathways leading to 8:2 FTCA and FTUCA involve oxidation of 8:2 FTAL, however the pathways leading to the latter two polyfluorinated acids remain inconclusive. The fate of the unsaturated metabolites, 8:2 FTUAL and FTUCA, included conjugation with GSH and dehydrofluorination, to yield alpha/beta unsaturated GSH conjugates, and GS-8:2 FTUAL was reduced to the corresponding alcohol. Perfluorooctanoate (PFOA) and minor amounts of perfluorononanoate (PFNA) were confirmed as metabolites of 8:2 FTOH, and the respective roles of beta- and alpha-oxidation mechanisms are discussed. The analogous acids, aldehydes, and conjugated metabolites of 4:2, 6:2, and 10:2 FTOH (i.e. where x=3, 5, and 9, respectively) were also detected, and metabolite profiles among FTOHs generally differed only in the length of their perfluoroalkyl chains. Preincubation with aminobenzotriazole (1mM), but not pyrazole (100 μM or 1mM), inhibited the formation of metabolites from all FTOHs, suggesting that their oxidation was catalyzed by a P450 isozyme, not alcohol dehydrogenase.

Estrogen-like properties of fluorotelomer alcohols as revealed by MCF-7 breast cancer cell proliferation

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During past years, major research efforts in environmental health sciences have been devoted to the development of easy to perform and reliable *in vitro* bio-assays. The need for latter assays is very urgent, since the number of chemicals that needs toxicological screening is enormous. *In vitro* cell based assays have already shown their usefulness for studying, for instance, endocrine disrupting chemicals. MCF-7 breast cancer cells are used for the detection of estrogen-like compounds. This project started with the well known 'E-screen assay', in which chemicals are tested for their capacity to re-induce the proliferation of growth arrested breast cancer cells (in estrogen-free growth medium).

We analyzed diverse perfluorinated compounds, such as perfluorosulfonate (PFOS), perfluorononanoic acid (PFNA), perfluoroctanoic acid (PFOA), and the fluorotelomer alcohols 1H,1H,2H,2H-perfluorooctan-1-ol (6:2 FTOH) and 1H,1H,2H,2H-perfluoro-decan-1-ol (8:2 FTOH). Both fluorotelomer alcohols were able to re-induce MCF-7 cell proliferation. In order to confirm estrogen-like properties, two additional assays are performed. Cell cycle analysis by flow cytometry reveals whether exposures to chemicals during 24 hours leads to increased percentages of cells in the S(ynthesis) phase of the cell cycle. While an increased percentage is regarded as a measure of estrogenicity, this assay again confirmed the estrogen-like properties of the tested fluorotelomer alcohols. Finally, we analyzed gene expression of well known estradiol-responsive genes in the MCF-7 breast cancer cells. While the expression levels of genes such as TFF1 (Trefoil factor 1) or PGR (Progesterone receptor) were unchanged upon exposures to PFOS, PFNA or PFNA, significant inductions were observed by fluorotelomer alcohols. During all assays, estradiol and the xeno-estrogenic compound 4-nonylphenol were the positive controls, while 2,3,7,8-tetrachlorodibenzo-p-dioxin was used as a negative control. We also tested all perfluorinated compounds in combination with faslodex, which is a well known anti-estrogenic compound that interferes with binding to the estrogen receptor. Latter assay showed that faslodex was able to undo the estrogenic effects of 6:2 FTOH and 8:2 FTOH, from which can be concluded that the estrogen receptor is involved.

Discussion

Fluorotelomer alcohols were characterized as estrogen-like chemicals *in vitro*, by a combination of the E-screen assay, flow cytometric analysis of the cell cycle and gene expression analysis of estrogen-responsive genes. The structural similarities of these compounds and 4-nonylphenol, the reference xeno-estrogen during this study, offer a possible explanation why these new compounds may act as ligands for the estrogen receptor. The characterization of fluorotelomer alcohols as new xeno-estrogens during this study demonstrates the need to carefully monitor their environmental distribution and to investigate further the effects of perfluorinated compounds on biota.

Microcosm assessment of the toxicity of 10:2 fluorotelomer saturated acid to aquatic macrophytes

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Fluorinated telomer alcohols (FTOH) are potential sources of environmentally persistent perfluorinated carboxylicacids (PFCAs) to aquatic ecosystems. As part of a larger environmental fate and ecotoxicological study, the phytotoxicity of the 10:2 fluorotelomer saturated acid on rooted floating (*Lemna gibba)* and submersed rooted macrophytes (*Myriophyllum sibiricum*, *Elodea canadensi and Egeria densa*) were investigated utilizing 12,000 L outdoor microcosms. The microcosms were designed to mimic small ponds and were treated with 10:2 fluorotelomer saturated acid at 0 ug/L (n=3), 0.1, 0.5, 1, 10, 50, 100, 250, 750, 1000, 2000 ug/L (n=1) in a regression design. *L. gibba* were assessed for a range of endpoints after a 7 day exposure duration. Individual rooted plants were assessed for growth and pigment endpoints at regular intervals over an 84 day exposure. The submersed plants were also grown in pots in order to represent small populations and communities and evaluated over a 70 day period to assess possible interactions and modification of the toxicity response, specifically between *E. canadensis* and *M. sibiricum*. Preliminary analysis of the data for all plant species at the individual, population and community level does not indicate any significant phytotoxicity at environmentally relevant concentrations.

Comparative responses of rats and mice exposed to linear/branched, linear, or branched ammonium perfluorooctanoate (APFO)

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Ammonium perfluorooctanoate, as synthesized by the ECF process, exists as a mixture of linear and branched isomers in a ratio of ~approximately 80:20 (linear/branched). Much of the currently produced APFO is linear. To compare the forms rats and mice were given doses by oral gavage ranging from 0.3 to 30 mg/kg of either linear/branched (L/B), linear (L), or branched-only (B) APFO for 14 days and selected toxicologic end-points were determined. The L/B and L forms produced a more pronounced effect on body weights compared to the B form in both rats and mice. Liver weights increased in both species proportional to dose with all 3 forms responding similarly. Hepatic β-oxidation was elevated in both species; increased activity was dose related in the rat but not in the mouse. Total cholesterol, HDL and non-HDL cholesterol levels were decreased in both rats and mice, while triglyceride levels decreased in rats and tended to increase in mice at lower doses. All 3 forms produced a similar response. LOELs of 0.3 mg/kg based on liver weight and β-oxidation changes were similar for all 3 forms in mice. LOELs in rats were 1 mg/kg for L/B and B forms and 0.3 mg/kg for the L form based on changes in lipid parameters. Serum PFOA levels at the LOEL in mice were 10-15 ppm, in rats 22-55 ppm. In both rats and mice, the overall responses to the L/B and L forms of APFO were similar. The B form appeared somewhat less potent, particularly with regard to body weight. The toxicologic database developed primarily using the L/B form appears to be applicable to the L form.

Dermal penetration of APFO through rat and human skin

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The permeability of APFO through rat and human epidermal skin was measured *in vitro*. Epidermal membranes for both rat and human were mounted on *in vitro* diffusion cells with an exposure area of 0.64 cm^2. Skin integrity was confirmed using electrical impedence. A 20% aqueous solution of APFO was applied to the epidermal surface at 0.150 $\mu L/cm^2$ and serial receptor fluid samples were collected hourly for 6 hours and at 12, 24, 30, and 68 hr. Samples were analyzed for PFOA using LC/MS. For rat skin the time to steady-state penetration was 6500 ± 3000 $mg/cm^2/hr$ and occurred within 12 hrs. The permeability coefficient (K_p) for APFO was calculated to be $3.25 \pm 1.51 \times 10^{-5}$ cm/hr. At the end of the 48-hr exposure period, 1.13% had penetrated through rat skin. For human skin, steady-state penetration was 190 ± 57 $mg/cm^2/hr$ reached at 12 hr. The K_p for human skin was $9.49 \pm 2.86 \times 10^{-7}$ cm/hr. By the end of the 48-hr exposure period, only a negligible amount of the APFO applied ($0.048 \pm 0.01\%$) had penetrated through human skin as compared to that which penetrated rat skin. In the occupational setting, despite this low penetration rate, the amount in contact with the skin and the time of contact still allows some uptake through the skin; hence, steps need to be taken to minimize dermal contact with APFO.

Comparison of avian reproduction and tissue uptake with exposure to perfluorooctane sulfonate or perfluorobutane sulfonate

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Bobwhite Quail (*Colinus virginianus*) were exposed to varying concentrations of Perfluorooctane Sulfonate (PFOS) or Perfluorobutane Sulfonate (PFBS) in the diet for up to 21-weeks. The in-life phase test procedures followed FIFRA Guideline 71-4 and OECD Guideline 206. Survival, growth and feed consumption were monitored in adult birds. Reproductive parameters evaluated included egg production, fertility, hatchability and hatchling growth and survival. Adults and offspring were subjected to gross necropsy at study termination and selected tissues were examined histopathologically. Samples of egg, liver, and sera were collected and analyzed for PFOS and PFBS using liquid chromatography/mass spectrometry (LC/MS). For PFOS, the NOEC could not be determined. The LOEC was found to be 10 mg/kg feed based on a reduction in the number of 14-day old surviving offspring as a percentage of the number of eggs set. Additionally, a slight, but statistically significant, reduction in testes size was noted in some males receiving 10 mg/kg feed. However, the toxicological significance of this phenomenon is unclear since testicular regression is a normal post-reproductive process and no effects were noted on other reproductive endpoints related to egg production and fertility. The PFBS NOEC was reported as 900 mg/kg feed with no treatment-related effects observed on survival, growth, feed consumption, histopathology or any reproductive parameters. Concentrations of PFOS measured in liver and sera were higher in males than in females while those of PFBS were similar between the sexes. Results from the study with the shorter perfluorinated chain chemical (PFBS) demonstrated a striking reduction in toxicity and reproductive effects as well as differences in tissue distribution when compared to the longer-chained PFOS.

Urinary bladder endpoints in workers and rats exposed to perfluorooctanesulfonyl fluoride (POSF) and related compounds

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Workers with potential high exposure to POSF-related compounds, including perfluorooctanesulfonate (PFOS), had 3 bladder cancer deaths vs. 0.2 expected (SMR 12.8; 95% CI 2.6 – 37.4). These compounds have not shown genotoxicity, and toxicology studies (including cancer bioassays) with PFOS and related sulfonamides have not shown bladder effects, but repeat-exposure data were not available for POSF. To further study the association of exposure to bladder cancer, we investigated bladder cancer incidence in workers and potential bladder effects of POSF in rats. Epidemiology: Because of high survivorship, living members (N = 1895) of the study were contacted by mail to answer a questionnaire about bladder cancer, and 74% participated. Validation occurred by medical record or death certificate for those original members deceased (n = 188). Incidence of bladder cancer was compared to expected based on NCI SEER data. Results: 11 bladder cancers were identified. The Standardized Incidence Ratio (SIR) for the entire cohort was 1.3 995% CI 0.6 – 2.3). The SIR for ever working and working for more than one year in a high exposed job were 1.7 (95% CI 0.6 – 3.8) and 1.1 (95% CI 0.2 – 3.3), respectively. Toxicology: Rats were exposed by inhalation to 20, 30, 100 or 300 ppm POSF 6 hrs/day, 5 days/week for 4 (0 & 300 ppm) or 13 weeks. Complete urinalysis was done on overnight urines. SEM/Xray element analysis for crystals/calculi and immediate pH were conducted on fresh morning urines. Sagittal sections of urinary bladder were taken for SEM analysis and assessment of cell proliferation by proliferating cell nuclear antigen (3 sections, 103 cells/section). Results: There were not treatment related findings with respect to urinalysis parameters, including pH and solids, nor changes of the urothelium, including no exposure-related increase in cell proliferation. These studies do not support the hypothesis that the excess risk of bladder cancer initially reported was due to exposure of POSF-related materials or PFOS.

Evaluation of the half-life ($t_{1/2}$) of elimination of perfluorooctanesulfonate (PFOS), perfluorohexanesulfonate (PFHS) and perfluorooctanoate (PFOA) from human serum

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PFOS is well-absorbed orally and very slowly eliminated from the body, and these combined properties can result in the accumulation of PFOS body burden from various sources and pathways of exposure. Elimination half-lives in rats and monkeys are currently estimated to be approximately 100 days and 200 days, respectively. Enterohepatic circulation likely plays a predominant role in the long elimination half-life of PFOS. Serum elimination half-lives for PFHS in cynomolgus monkeys have been estimated at approximately 75 percent less than PFOS, and limited data in rats also suggests a shorter elimination. Unlike PFOS and PFHS, marked sex and species differences occur in the elimination of PFOA. Urine is the primary route of excretion for PFOA. The elimination $T_{1/2}$ in male rats is 4-6 days and 2-4 hours in females, and is approximately 21 and 30 days in male and female monkeys, respectively. Sex hormones may modulate differential expression of organic anion transporters involved in the urine elimination of PFOA in rats.

To investigate the $T_{1/2}$ of serum elimination in humans of PFOS, PFHS and PFOA, 27 retirees (25 males, 2 females) from two fluorochemical manufacturing plants were followed for up to 5.5 years with periodic blood collections. One retiree's samples were excluded due to the likelihood of occupational exposure during follow-up. The analysis used a primary extraction in combination with an alkaline back extraction technique. A 5 µL injection was introduced to the mass spectrometer through a high performance liquid chromatography system. All quantitative calculations for PFOS and PFHS were based on the ion ratios between PFOS or PFHS and the internal standard (dual substituted ^{18}C-PFOS). All quantitative calculations for PFOA were based on the ion ratios between PFOA and the internal standard (dual substituted ^{13}C-PFOA). Individual serum elimination rates were calculated with Pharsight WinNonlin® software.

Initial serum concentrations for the 26 subjects ranged between 0.15 – 3.49 µg/mL for PFOS, 0.02 – 1.30 µg/mL for PFHS and 0.07 – 5.10µg/mL for PFOA. The mean $T_{1/2}$ of serum elimination for PFOS, PFHS and PFOA were 5.4 years (95% CI 3.9 – 6.9), 8.7 years (95% CI 6.7 – 10.9) and 3.8 years (95% CI 3.1 – 4.4), respectively. The $T_{1/2}$ of serum elimination for each fluorochemical was not associated with initial concentration, age or sex of retiree, years worked at the manufacturing facility or the time between retirement and first blood collection.

Thyroid hormone status in adult rats given oral doses of perfluorooctanesulfonate

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Decreases in serum total thyroxine (TT4) and free thyroxine (FT4) without a compensatory rise in thyrotropin (TSH) or histological changes of the thyroid have been observed in studies with perfluorooctanesulfonate (PFOS) in rats. Hypothyroidism is defined by reduced FT4 and compensatory elevation of TSH. Prior observations do not fit the profile of a hypothyroid state. Serum free fatty acids increases are known to interfere with FT4 measurement using analog assays due to competition with thyroxine (T4) for albumin binding. PFOS is known to compete for binding with fatty acids on albumin. Therefore, we hypothesized that decreases in measured serum FT4 by analog methods in the presence of PFOS is due to binding interference. We employed a reference method (equilibrium dialysis/RIA) for FT4 in rat serum containing PFOS *in vitro* and *in vivo*. Results - PFOS was found to increase FT4 *in vitro* and have no effect on TT4 *in vitro* and FT4 and TSH *in vivo* after 24 hours. TT4 was decreased. Negative bias analog methods for FT4 in the presence of PFOS was confirmed. We also hypothesized that PFOS may transiently increase serum FT4 *in vivo*, and, as a result, the hepatic response to thyroid hormone. We measured malic enzyme (ME), FT4, TT4, and TSH at 2, 6, and 24 hours following a single oral dose of 15 mg PFOS/kg. Results - ME mRNA (2 hrs) and activity (24 hrs) and FT4 (2 & 6 hrs) were increased, and TSH (6 hrs) and TT4 (2,6, and 24 hrs) were decreased relative to controls. To investigate the reduction of TT4 *in vivo*, we hypothesized that, if FT4 is increased in the presence of PFOS, it may be taken up by tissues and eliminated at a greater rate than normal. Therefore, the effects of PFOS serum TT4 and on ^{125}I tissue distribution and elimination in urine and feces after a single intravenous (*iv*) injection of ~ 10 uCi ^{125}I-T4 followed by a subsequent single oral dose of 15 mg PFOS/kg were investigated over a 24-hour time period. Results - Serum TT4 and ^{125}I were decreased. Urine and fecal elimination of ^{125}I were increased. To rule out an effect of PFOS on pituitary response to hypothalamic thyrotropin-releasing hormone (TRH) and release of TSH from the pituitary, we gave propylthiouracil (PTU) to female rats in drinking water for 10 days to induce hypothyroidism and gave 3 mg PFOS/kg/d for 7 days and then measured the pituitary response to TRH in static culture *ex vivo* and serum TSH *in vivo*. Results - PFOS did not affect pituitary TSH release or serum TSH after PTU treatment. These findings suggest that PFOS does not induce a hypothyroid state or interfere with pituitary TSH release.

This abstract does not reflect EPA policy.

Differential activation of nuclear receptors by normal and perfluorinated fatty acids: a comparison of human, mouse and rat PPARα,β,γ, LXRβ and RXRα

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Administration of perfluorooctanoate (PFOA), a perfluorinated fatty acid, to rats results in peroxisome proliferation and benign liver tumors, events associated with activation of the nuclear receptor (NR) peroxisome proliferator-activated receptor-α PPAR α). There is evidence that PPARs exhibit significant species differences in response to ligand activation. In addition, based on the fatty acid structure of these agents, it is plausible that other NRs are targets such as PPARß, PPAR γ, liver X receptor (LXR) or retinoid X receptor (RXR).

In this study, the activation of mouse, rat and human PPAR α, PPARß, PPAR γ, LXRß and RXR α by PFOA and some of its linear and branched isomers, and perfluorooctanesulfonate (PFOS) were investigated and compared to several structural classes of natural fatty acids and appropriate positive control ligands. A NR ligand-binding domain/GAL4 DNA-binding domain chimeric reporter system in mouse 3T3-L1 cells was used. Activation in this system shows the ability of the chemicals to interact with the NR and produce a conformational change capable of regulating reporter gene transcription, but does not necessarily equate to their ability to produce a biological or toxicological response *in vivo*.

Human, rat and mouse PPARα was activated by PFOA and its isomers, PFOS, as well as by oleic acid (OA) and α-linolenic acid (ALA) while only the rat and human PPAR α responded to octanoic acid (OCT) and linoleic acid (LA). PPARß was less sensitive to the agents tested, with PFOA and OCT affecting the mouse receptor while OA, LA and ALA acted upon all three species. PFOA and PFOS also activated PPARγ in rat, mouse and human, as did ALA; LA affected mouse and rat PPAR γ whereas OCT was ineffective. Although the two perfluorinated fatty acids were capable of activating PPARγ, the maximum induction was much less than seen with Rosiglitazone (e.g. PFOA elicited a maximum induction of 2-fold vs.20-fold for Rosiglitazone against human PPARγ) suggesting that PFOA and PFOS are partial agonists of PPARγ. In this assay system, LXR was not affected by PFOA in any species although the mouse receptor was affected by OCT and ALA. The common heterodimerization partner RXR α was activated by OA and LA in the human and ALA in the human and mouse while PFOA inhibited RXR α in the mouse.

Taken together these data show that PPARα is the most likely target of PFOA and PFOS, although PPARγ is also activated to some extent. Compared to naturally occurring long-chain fatty acids, e.g., LA and ALA, these perfluorinated fatty acids were more selective and less potent in their activation of NRs.

Evaluation of perfluorooctane sulfonate (PFOS) in the rat brain

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This study examined whether there is a differential distribution of PFOS within the brain, and compares adult rats with neonatal rats at an age when formation of the blood-brain barrier is not yet complete (postnatal day 7). Male and female Sprague-Dawley rats (60-70 day old, 4/sex) were treated with 3 mg PFOS per kg per day by gavage for 14 days and sacrificed 24 h after the last administration. A blood sample was obtained from tail-bleed prior to sacrifice, and the brain was perfused with isotonic saline. Five brain regions, which included cerebral cortex (CX), hippocampus (HP), cerebellum (CB), hypothalamus (HT), and brainstem (BS), were dissected immediately and stored frozen at -80° C until analysis. For the developmental study, four timed-pregnant dams were given PFOS (3 mg/kg) from gestational day (GD) 2 through GD 21 and allowed to deliver litters. Male and female pups from each litter were sacrificed on postnatal day 7, and their brains were perfused and dissected similarly to the adults. Trunk blood was obtained from littermates after decapitation. Extraction of brain tissues homogenates employed a base digestion followed by a solid phase extraction at an acidic pH for optimal recovery. A 15-µL injection was introduced to the mass spectrometer through a high performance liquid chromatography system. All quantitative calculations for PFOS were based on the ion ratios between PFOS and the stable-isotope-labeled internal standard (dual substituted ^{18}O-PFOS). Mean serum concentrations were 123 µg/mL and 52 µg/mL for adults and pups, respectively. Mean corresponding concentrations (µg/g) in brain regions ranged from approximately 5 to 8 in adults and approximately 15 to 29 in neonates. Brain region mean PFOS concentrations expressed as percentage of mean serum PFOS concentrations ranged from approximately 4 to 6 in adults and 29 to 55 in neonates. Male and female values were similar. From these data, it is evident that PFOS can be detected in the adult rat brain after subchronic exposure, although the extent is limited (ca. 4-6% of the serum level) and there is no differential distribution or accumulation within the brain. In comparison, higher PFOS concentrations were found in the postnatal day 7 neonatal rat brain, most likely due to the fact that the blood-brain barrier is not fully functional in rat pups on postnatal day 7.

This abstract does not necessarily reflect EPA policy.

Pathology working group review of mammary glands from chronic feeding study in rats with APFO

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In a chronic feeding study in rats with APFO completed in 1983, incidences of some mammary gland tumor types were increased or decreased compared to controls. However, these changes were not considered to be treatment related since they were usually not statistically significant and did not occur in a dose-related manner. To better clarify the mammary tumor response in the two-year study, and to assess these mammary tumors based on contemporary diagnostic criteria, all sections of mammary gland from all female rats in the study were examined by a peer review pathologist. Mammary sections were then examined by a pathology working group (PWG) which consisted of the peer review pathologist and two board-certified veterinary pathologists with extensive experience in the microscopic evaluation and interpretation of lesions observed rodent bioassay. The results of the peer review and PWG assessments were compared to the original findings of the study pathologist.

The primary difference between the original reported findings by the study pathologist and the PWG results involved changes in the mammary gland that were initially reported as lobular hyperplasia by the study pathologist. The PWG felt these changes were more characteristic of mammary gland fibroadenoma. A majority of the other mammary gland neoplasms originally reported by the study pathologist was confirmed during the PWG review. Although the incidence of neoplasms varied among the control and treated groups, there were no statistically-significant increases in treated groups relative to controls for fibroadenoma, adenocarcinoma, total benign neoplasms or total malignant neoplasms. In addition, no increase in tumor multiplicity or incidence with dose was observed. The PWG also concluded that the morphologic appearance of the neoplasms present in treated groups was similar to those observed in the control groups, indicating that treatment did not affect the phenotype of the neoplasms in the study. The incidence of mammary gland neoplasms observed in this study was similar to historical control data for mammary gland neoplasms in untreated Crl:CD female Sprague-Dawley Rats in chronic studies.

The PWG concluded that the incidence of mammary gland neoplasms in this study was not affected by chronic dietary administration of APFO. The morphologic appearance, overall incidence and distribution of the neoplasms observed in treated and control groups were similar.

Reference:
[1] Hardisty, JF (2005). Pathology Peer Review and Pathology Working Group Review of Mammary Glands from a Chronic Feeding Study in Rats with APFO. Experimental Pathology Laboratories, Inc., Research Triangle Park, NC

Deleterious effects of perfluorooctane sulfonate (PFOS) on the immune organs of mice

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Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) exert a number of potent effects on rodents, including increases in the number and size of hepatic peroxisomes (peroxisome proliferation) and accompanying potent up-regulation of hepatic perxoisomal fatty acid β-oxidation, as well as extensive hypertrophy of the liver. Recently, we have reported that PFOA also exerts profound deleterious effects on the immune system of mice, including thymic and splenic atrophy (reflecting an intensive reduction in the numbers of thymocytes and splenocytes) and potent suppression of adaptive immune responses. The present investigation was designed to determine whether the related compound PFOS has similar effects on the immune system.

Male C57Bl/6 mice (approximately 6-weeks-old and 20 g) received diets containing 0.02% (w/w) PFOS or PFOA or standard laboratory chow for 10 days, following which the animals were killed and their livers, thymuses and spleens dissected out and weighed. Thereafter, single cell suspensions of these organs, as well as of the bone marrow were prepared, counted and stained with fluorescent-conjugated monoclonal antibodies (PE-conjugated anti-mouse CD3, FITC-conjugated anti-mouse CD4, PerCP-conjugated anti-mouse CD8 and FITC-conjugated anti-mouse CD19) in order to characterize their phenotypes. The stained cells were analyzed employing a laser FACScalibur flow cytometer.

As we reported earlier for PFOA-treated animals, mice receiving PFOS in their diet also exhibited considerable weight loss compared to the control animals, in addition to significant atrophy of the thymus and spleen and hypertrophy in the liver. Analysis of the numbers and phenotypes of thymocytes and splenocytes from PFOS-treated mice revealed the following: (i) the numbers of these cells were reduced by approximately 85% and 40%, respectively (compared to 95% and 50% reductions, respectively, in the case of treatment with PFOA); (ii) although all populations of thymocytes were decreased, the immature $CD4^+CD8^+$ population was decreased most dramatically; (iii) the numbers of T cells in the spleen and the numbers of B cells in the spleen and bone marrow were also reduced by PFOS treatment.

Our present findings indicate that PFOS, like its analog PFOA, exerts adverse effects on both primary and secondary immune organs. We are presently determining whether these effects of PFOS are reversible and whether, as expected, they reflect potent suppression of adaptive immune responses, both of which are the case for PFOA. Although the underlying mechanism(s) for the deleterious effects of PFOS and PFOA on immune organs of the mice remains to be elucidated, we suggest that the concomitant depletion of fat depots and/or associated activation of the innate immune system may be involved. This study was supported by an unrestricted grant from the 3M Company. The technical assistance of Jarl Olsson is gratefully acknowledged.

Local activation of the innate immune system of mice by perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA)

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Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) exert a number of potent effects on rodents, including increases in the number and size of hepatic peroxisomes (peroxisome proliferation) and accompanying potent up-regulation of hepatic perxoisomal fatty acid β-oxidation, as well as extensive hypertrophy of the liver. Recently, we have reported that these compounds also exert profound deleterious effects on the adaptive immune system of mice, including thymic and splenic atrophy (reflecting an intensive reduction in the numbers of thymocytes and splenocytes) and, at least in the case of PFOA, potent suppression of adaptive immune responses. In the present study, our aim was to elucidate possible effects of PFOA and PFOS on the innate immune system of mice as well, utilizing the production of tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6) as indicators of the activity of this system.

Male C57Bl/6 mice (approximately 6-weeks-old and 20 g) received diets containing 0.02% (w/w) PFOS or PFOA or standard laboratory chow for 10 days, following which blood samples were analyzed for total white blood cells (WBC) and differential count using hemocytometer and Wright staining, respectively. In addition, macrophages collected from the peritoneal cavity were cultured for 18 h in Delbecco´s Minimum Essential Medium supplemented with 10% fetal calf serum (FCS), at 37° C under a humid atmosphere containing 5% CO_2. Both the supernatants from these cultures and serum samples were assayed for the presence of TNF-α and IL-6 employing commercial ELISA kits.

The WBC counts in PFOS- and PFOA-treated animals were slightly lower than those for control mice, with no apparent change in the number of circulating erythrocytes. Both groups showed a slight reduction in the percentage of circulating lymphocytes (from 85% to 71% in the case of PFOS (statistically significant) and to 78% with PFOA (not significant)). Peritoneal cells from mice receiving either PFOS or PFOA, but not from control animals produced and released high levels of TNF-α *ex vivo* and in the case of PFOS, a significantly elevated level of IL-6 as well. Indeed, the production of TNF-α induced by PFOS and PFOA in these cells was comparable to that observed in response to bacterial lipopolysaccharide, a potent inducer of this cytokine. However, the serum levels of TNF-α and IL-6 in treated and untreated animals were similar.

Our findings to date indicate that PFOS and PFOA exert a unique influence on the immune system, locally activating innate immunity while extensively suppressing adaptive immune responses. The enhanced production of TNF-α and IL-6 by resident peritoneal macrophages ex vivo implies that these innate immune cells are activated directly or indirectly by PFOA and PFOS. However, the observation that treatment does not alter circulating levels of TNF-α and IL-6 implies that different macrophage populations respond differently to these perfluorinated organic compounds. Obviously, the cause and significance of local activation of cells of the innate immune system by PFOS and PFOA remain to be determined. This study was supported by an unrestricted grant from the 3M Company. The technical assistance of Jarl Olsson is gratefully acknowledged.

Comparison of the effects of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) on wild-type mice and knock-out animals lacking uncoupling protein-1 (UCP-1)

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Recently, we found that PFOS and PFOA dramatically reduce the size of the major adipose tissue depots in mice, accounting at least in part for the accompanying decrease in total body weight. Here, we examine the influence of uncoupling protein-1 (UCP-1, expressed primarily in brown adipose tissue) on murine responses to these compounds. Wild-type (+/+) and UCP-1-knock-out (-/-) male C57Bl/6 mice (7-8 weeks old and 24-25 g) were fed 0.02% (w/w) PFOS or PFOA or standard laboratory chow for 10 days. During this period, body weight, food and water consumption, and rectal temperature were monitored. After sacrifice, the liver, thymus, spleen, interscapular brown adipose tissue and retroperitoneal and epididymal fat depots were dissected out and weighed. Brown fat mitochondria were isolated and their levels of UCP-1, carnitine-palmitoyltransferase 1 and cytochrome oxidase-1 determined by Western blotting and their capacity to oxidize palmitate and pyruvate measured polarogaphically.

Whereas treated UCP-1 +/+ mice weighed approximately 15% less (PFOA 21%, PFOS 14%) than untreated animals after 10 days, the UCP-1 -/- animals lost less (PFOA 9%, PFOS 8%) of their body weight. At the same time, the food intake of the +/+ and -/- animals was decreased 25% and 2% by PFOS and 28% and 10% by PFOA, respectively. Water intake was elevated about 70% (78% and 64%) in both genotypes after about 5 days of treatment with either compound, when stabilization of body weight was observed. A transient decrease in body temperature (up to 2.5°C between days 3 to 7) occurred in the wild-type, but not the knock-out mice. Ablation of the UCP-1 gene had no effect on the atrophy of the thymus or spleen or on the liver hypertrophy caused by PFOS and PFOA. Interestingly, total body weight and the major fat depots were less reduced and brown adipose tissue weight more reduced in the knock-out mice. Brown fat mitochondria from treated wild-type animals demonstrated 50-100% increases in the levels of UCP-1 protein and in their ability to oxidize palmitate and pyruvate, with no change in their levels of cytochrome oxidase-1 or carnitine-palmityltransferase-1. PFOA and PFOS acted as mild uncoupling agents, similarly to C8 fatty acids, when tested in isolated mitochondria.

Clearly, UCP-1 is not involved in the atrophy of the thymus and spleen or the liver hypertrophy caused in mice by PFOS and PFOA. However, the presence of this protein correlated with a greater loss of total body weight and of the major fat depots in response to these compounds. The present findings raise a number of questions: is the loss of fat depots in response to treatment with PFOS or PFOA due partially to anorexia? Why do these compounds increase water consumption so dramatically? Why do the brown fat mitochondria of wild-type mice administered PFOS and PFOA display an elevated level of UCP-1 and enhanced oxidative capacity? Might these changes represent a response to the transient decrease in body temperature observed? And what causes this hypothermia?

This study was supported by an unrestricted grant from the 3M Company. The technical assistance of Jarl Olsson is gratefully acknowledged.

Ecotoxicity assessment & remediation of firefighting training wastewater by air - sparged hydrocyclone (ASH) system

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Firefighting training activities at Canadian Forces Base generates substantial volume of wastewater containing aqueous film forming foams (AFFF) and various other chemicals that have been proven toxic to aquatic species. Therefore, it was necessary to remediate the lagoon wastewater before release into the environment. DND / Quality Engineering Test Establishment (QETE) was tasked to evaluate the Air Sparged Hydrocyclone (ASH) technology as a potential pre-treatment for the previously assessed Reverse Osmosis (RO) system, or as a potential viable stand alone remediation technology. QETE study, together with the Canada – United States Test and Evaluation Program (CANUSTEP) field work, demonstrated that the eight to ten–stage ASH system (190 L/minute) can operate as a stand alone technology with an efficiency of 90%, when used in conjunction with a chemical pre-treatment, to eliminate or significantly reduce the toxicity of the lagoon wastewater to rainbow trout (*Oncorhynchus mykiss*), *Daphnia magna,* fathead minnows *(Pimephales promelas*), *Ceriodaphnia dubia,* and algae (*Selenastrum capricornutum*). The ASH system design removes free and emulsified hydrocarbons, oil, surfactants, metals and other contaminants. Without chemical pretreatment, the efficiency ratio of the ASH system is 80%. QETE also ascertained that Georemediation™ treatment of the toxic concentrate (i.e., 10% of the original volume) resulted in rendering the georemediated product non-hazardous with an efficiency ratio of 90%, by significantly reducing the leachability of organics and inorganics to levels below Ontario regulatory limits. Overall, only 1% (in volume) of the initial wastewater remains subject to high temperature incineration for acceptable waste disposal. The savings that is accrued from being able to discharge 99% of the treated non-toxic wastewater to the environment, as opposed to having to dispose of the total volume was substantial.

Risk Assessment and Regulatory Policy

RAP001 Beers	Short chain alternative to PFOS based fluorosurfactants
RAP002 Russell	Development of a global exposure model for DuPont fluorotelomer-based products
RAP003 Buck	Consumer Article Exposure and Risk Characterization
RAP004 Kurias	Progress on Environment Canada's ecological risk assessment activities for certain perfluoroalkyl compounds
RAP005 Endres	PFOA - A project on hazard assessment under the OECD existing substance programme

Short chain alternative to PFOS based fluorosurfactants

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With the decision of the major US manufacturer of PFOS based fluorosurfactants to withdraw its products from the market, a number of key product manufacturers were left with the decision to switch to the telomer-based, long-chain fluorosurfactants, reformulate their products without fluorosurfactants or search for a fluorosurfactant that did not use the C8 or longer perfluoroalkyl chain length materials in their synthesis. Many users had already qualified the telomer based products and were able to switch with little or no difficulty. Other users tried to eliminate fluorosurfactants totally with mixed results. Still other users were sufficiently concerned about the possibility that telomer-based, long-chain fluorosurfactants could present similar environmental concerns that they were interested in exploring short perfluoroalkyl-chain alternatives. One such short-chain alternative technology developed by OMNOVA Solutions Inc. was emerging as the long-chain fluorosurfactants came under extensive scrutiny from the US EPA, Environment Canada and other regulatory agencies across the globe. This poster will describe the success of this alternative technology to date.

In the late 1990's, OMNOVA Solutions Inc. was developing a new fluorochemical technology for surface modification coatings in its Decorative Products business unit. During the development it was observed that the fluorochemical, trade name PolyFox™, had flow, leveling and wetting properties that contributed significantly to the surface appearance of coatings in which it was formulated. The surfactant properties were surprising as the PolyFox™ material being developed was a polymer rather than a small molecule and it used C1 and C2 perfluoroalkyl starting materials rather than the C8 and above materials that were the industry standard. When the PFOS products were removed from the market, OMNOVA Solutions Inc. began a program to determine whether this technology could provide an environmentally preferable alternative. The first application to be explored was in commercial and industrial floor polishes, an application for which OMNOVA had extensive experience as a major manufacturer of floor polish polymers. The result of this development was a family of anionic surfactants that provided equal flow, leveling and wetting performance to the standard PFOS product at equivalent addition rates and offered the advantage of significantly less foaming.

Although the initial technical development appeared to be viable, OMNOVA chose to take its plan for alternative fluorosurfactant technology to the EPA in a pre-notice meeting to seek their advice on the environmental impact of these products. With extensive review by the EPA and eventually similar review of toxicology data with European regulators, OMNOVA has launched and has made a significant market penetration in the floor care market with its PolyFox™ TM (PF-136A) and PolyFox™ VM (PF-156A) products. The toxicological data on PolyFox™ VM is extensive and includes bioaccumulation data to address the concerns raised by traditional fluorosurfactants. Similar toxicological data is being developed on other members of the PolyFox™ family which are designed for use in other applications.This poster will also present comparisons of the performance in various applications of a fluorosurfactant synthesized from starting materials with a perfluoroalkyl chain length of C2 or less as alternatives for the traditional longer chain (C8 and above) fluorosurfactants.

Development of a global exposure model for DuPont fluorotelomer-based products

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As a part of its ongoing product stewardship program, DuPont is evaluating the potential consequences of release of perfluorooctanoic acid (PFOA) and related perfluorocarboxylic acids (PFCAs) into the environment that may be related to manufacturing, processing, use or disposal of DuPont fluorotelomers. To assess the significance of current and future environmental releases of fluorotelomer-related chemicals, a continental-scale to global-scale exposure model is currently being developed to allow simulation of chemical releases, environmental fate, transport and transformation and uptake by human and wildlife receptors, and comparison of selected endpoints such as blood sera and liver concentrations to monitoring data.

The model incorporates chemical kinetics and transformation pathways into one or more of the existing steady-state models that have been developed to simulate the fate of persistent chemicals in the environment. The model assesses the potential marginal contributions of current and proposed chemical releases to tissue concentrations in human and wildlife receptors in comparison to the concentrations resulting from the extant fluorinated materials in the environment. An additional application of the model will be to compare simulated endpoints such as blood sera and liver concentrations to existing monitoring data to determine the relative effects of geographic release patterns as well as chemical transformation, environmental distribution and route of exposure.

This poster will present schematic diagrams of the conceptual human and wildlife receptor exposure pathways including potential release points, environmental entry media, transport media, exposure media, and exposure route. The specific pathways that are likely to be most important based upon receptor behavior patterns will be described as well as a proposed methodology for quantitative exposure assessment of each pathway. Finally, key issues associated with multi-media modeling of fluorotoelomer chemistry will be summarized to highlight the need for additional research and to provide a basis for discussion and further model refinement.

Consumer Article Exposure and Risk Characterization

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An exposure and risk characterization was conducted to understand the potential human health significance of trace levels of perfluorooctanoate (PFO) detected in certain consumer articles. Concentrations of PFO were determined from extraction tests using simulants relevant to potential human exposure (e.g. water, sweat, saliva, food-simulating solvents). Potential human exposures during consumer use were quantified based on an assessment of behavior patterns and regulatory guidance. Health benchmarks were developed and then compared to exposure estimates to yield margins of exposure (MOEs).

This paper will present the results of the assessment for apparel textiles and food-contact paper. The conceptual exposure pathways and exposure media will be described for five receptor groups: infants, children, adolescents, adults and adult professionals. Exposure was quantified based upon extractions studies and compared to health benchmarks to develop a risk characterization. The margins of exposure under reasonable maximum exposure scenarios ranged from 50,000 to 70 million for apparel textiles and 300,000 to 3 million for food contact paper. These results indicate that exposures to PFO during consumer use are not expected to cause adverse human health effects.

Progress on Environment Canada's ecological risk assessment activities for certain perfluoroalkyl compounds

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The Existing Substances Branch at Environment Canada has undertaken ecological risk assessment activities since 2000 on certain perfluoroalkyl substances currently in commerce in Canada. Perfluorooctane sulfonate (PFOS), its salts and precursors were identified as the first assessment priority. Therefore, Environment Canada, in conjunction with Health Canada, initiated and completed a screening assessment on PFOS, its salts and precursors. The draft assessment report was released for a 60-day public comment period in October 2004.

Overall, priority for conducting assessments is based on analysis of results from categorization under the *Canadian Environmental Protection Act, 1999* (CEPA 1999), as well as additional information from: industry, emerging science, international assessment activities, new substances notifications, public nominations, and provincial or international decisions. Categorization identifies those substances that are persistent or bioaccumulative, and inherently toxic to human or non-human biota, as well as substances that may present the greatest potential for exposure to humans in Canada.

As PFOA and its salts were identified as the next assessment priority, a preliminary ecological review of PFOA within the Canadian environment has been initiated. Environment Canada is also identifying priorities for other perfluoroalkyl substances including long chain perfluorocarboxylic acids (PFCAs) which are emerging contaminants of concern due to their persistence, high bioaccumulation potential and presence in the Arctic. In partnership with researchers, data gathering activities are underway for PFCAs and related substances.

As part of broader data collection work to determine import and manufacturing activities for perfluorinated substances in Canada, Environment Canada published in January 2005 a *Notice with Respect to Certain Perfluoroalkyl and Fluoroalkyl Substances* in the *Canada Gazette*. This Notice requires companies who manufactured or imported greater than 100 kilograms of specific perfluoroalkyl and fluoroalkyl (PFA/FA) substances to provide information on activities. The information required by the PFA/FA Notice will be used, along with other data sources, to inform current and future risk assessment and risk management activities conducted under CEPA 1999. A separate Notice specific to PFOS, its salts and precursors, was also issued in January 2005, to support the development of potential risk management activities for PFOS.

PFOA - A project on hazard assessment under the OECD existing substance programme

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Perfluorinated compounds, especially carboxylic acids and sulfonates, are regularly measured in human blood (e.g. Kannan et al. 2004) and in the environment (e.g. Martin et al. 2004, Yamashita et al. 2004). In the water-phase, the predominant compound is PFOA (perfluorooctanoic acid). In an ongoing research project funded by UBA, PFOA was measured in water samples from the arctic North Atlantic and the German Bight, see also poster contribution of Theobald et al..A study was initiated recently to determine the level and temporal trend of perfluorinated compounds in humans. The study was conducted on human plasma samples archived by the German Environmental Specimen Bank (ESB). The ESB samples originated from the eight years between 1985 and 2004 and were analysed for perfluorooctanesulfonate (PFOS), perfluorohexanesulfonate (PFHxS), perfluorooctanoate (PFOA), perfluorohexanoate (PFHxA), and perfluorooctanesulfonamide (PFOSA). PFOS and PFOA were determined in all plasma samples. The predominant compound was PFOS with concentrations between 5.5 and 104 ng/ml plasma followed by PFOA with concentrations ranging from 1.4 to 58 ng/ml.

Based on these findings, UBA decided to prepare an environmental hazard assessment for PFOA and its ammonium salt APFO in cooperation with the BUA. The report summarises the available information about production, use, and releases into the environment. The geographic focus is Europe (EU 15). Data on environmental fate and behaviour are being compiled and ecotoxicological studies as well. The draft report is scheduled for submission to the OECD existing substances programme in April 2006 (SIAM 22). The focus of our work is to draft the environmental part of the hazard assessment, since human health effects of PFOA have been evaluated in detail by the US-EPA.

It is also our intention to evaluate the exposure situation in Europe in the legal context of the EU. However, information about production, releases, impurities and metabolism of possible precursors is still incomplete. We have agreed with the FAS group of PlasticsEurope to share information about production. To complete the data for environmental exposure, we will consider the results of the PERFORCE Project of the European Commission and several other ongoing studies about degradation and transport mechanisms.

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Thompson, J.T.	TOX019 Vanden Heuvel
Tittlemier, S.A.	ANA041 Tittlemier
Todoriki, H.	ANA008 Saito
Toft, A.	ENV005 Sulbaek Andersen
Tokuhashi, K.	ENV002 Katsuna
Tomy, G.T.	**ANA041 Tittlemier**
Trojanowicz, M.	ANA018 Trojanowicz
Trosko, J.E.	TOX009 Yoo
Tsugane, S.	ANA011 Kawaguchi
Tsunoda, M.	TOX005 Endo
Tuck, A.F.	ENV016 Waterland
Tully, J.S.	ANA019 Calafat
Uchimaru, T.	ENV002 Katsuna
Ungewiss, J.	ANA036 Schlummer
Unice, K.	ENV019 Panko
Upham, B.L.	TOX009 Yoo
Vaida, V.	ENV016 Waterland

van Bavel, B.	ANA026 Karrman, ANA040 Lindstrom
Van de Vijver, K.I.	**TOX007 Van de Vijver**
van der Veen, I.	ANA040 Lindstrom
Van Hoven, R.L.	ANA020 Szostek, ENV018 Berti
van Leeuwen, S.	ANA040 Lindstrom
van Roon, A.	**ENV012 van Roon, ENV013 van Roon**
Vanparys, C.	TOX011 Maras
Verrault, J.	**ANA002 Verrault**
Wada, Y.	ANA008 Saito
Wallington, T.J.	ANA030 Dumoulin, ENV005 Sulbaek Andersen, ENV017 Martin, ENV020 Ellis, ENV021 D'eon, ENV026 Young,
Wang, N.	**ENV011 Wang**, ENV018 Berti
Washburn, S.T.	RAP002 Russell, RAP003 Buck
Watanabe, I.	TOX005 Endo
Watanabe, T.	ANA008 Saito
Waterland, R.L.	ANA030 Dumoulin, **ENV016 Waterland**
Wells, R.S.	ANA014 Houde
Whittle, D.M.	ENV024 Furdui
Widmann, K.	RAP005 Endres
Wilford, B.H.	ANA042 Shoeib
Wojcik, L.	**ANA018 Trojanowicz**
Wooler, G.R.	ANA028 Kaiser
Xia, Z.	TOX022 Abedi-Valugerdi
Xu, F.	TOX002 Harada
Yamanaka, N.	**TOX003 Yin Yeung**, ANA032 Taniyasu, ANA033 Ka So, ANA034 Gulkowska, ANA035 Gulkowska, TOX003 Yin Yeung, TOX005 Endo,
Yeo, B.	ANA005 Arsenault
Yeung, W.Y.	TOX003 Yin Yeung
Yoo, H.	**TOX009 Yoo**
Yoshiike, M.	ANA017 Nakata
Yoshinaga, T.	ANA008 Saito
Young, C.J.	**ENV026 Young, ENV027 Young**
Young, I.J.	**TOX025 Young**
Yujing, M.	ENV003 Mellouiki
Zammit, A.	ANA040 Lindstrom
Zarzecki, C.J.	ANA027 Kaiser
Zhu, J.	ANA042 Shoeib
Zhu, Y.	ANA025 L'empereur

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