CONCENTRATIONS AND TEMPORAL TRENDS OF TWO PERFLUOROOCTYL SULFONAMIDES IN FAST FOOD COMPOSITES COLLECTED DURING THE CANADIAN TOTAL DIET STUDY

Sheryl A. Tittlemier, Laura Edwards, Karen Pepper

Food Research Division, Health Canada, Ottawa, Ontario, Canada, K1A 0L2

Introduction

Recent work has described levels of certain perfluorooctyl compounds, an emerging class of organohalogen contaminants, in human sera and liver[1-4]. The occurrence of these perfluorooctyl compounds in human tissues from both occupationally and non-occupationally exposed North Americans indicates a generalized mode of exposure. These compounds – perfluorooctanesulfonate ($C_8F_{17}SO_3^-$, PFOS), perfluorooctanoate ($C_8F_{17}CO_2^-$, PFOA), and perfluorooctanesulfonamide ($C_8F_{17}SO_2NH_2$, PFOSA) – were all used as surfactants in a variety of industrial and commercial applications, including fire fighting foams, cleaning products, and water and oil repellent coatings for fabrics and paper. Two of the perfluorooctyl compounds – PFOS and PFOSA – are also metabolites of N-ethylperfluorooctanesulfonamide (N-EtPFOSA)[5], a compound present as a by-product in some oil and water repellent coatings for paper and paperboard used in food packaging. N-EtPFOSA is also used as an insecticide in the US, but has not been registered for use in Canada.

Both *in vitro* and *in vivo* assays indicate that the perfluorooctyl compounds are bioactive. PFOA has been shown to act as a peroxisome proliferator[6] and induce atrophy of the thymus and spleen in mice[7]. An in vitro study demonstrated that PFOA, PFOS, and PFOSA all inhibit gap junctional intercellular communication[8]. However, in one recent study, serum PFOS and PFOA concentrations in occupationally exposed subjects were not correlated with any changes in lipid, hepatic, haematological, or thyroid parameters typically associated with exposure to perfluorooctyl compounds[3].

Due to its presence in coatings applied to food packaging, N-EtPFOSA and its deethylated metabolite (PFOSA) were measured in fast food composites collected for the Canadian Total Diet Study. A robust quantitative method was developed for the analysis of the two perfluorooctyl sulfonamides. Food samples obtained over the past decade (1992-2002) were analyzed to examine changes in concentrations of the two compounds prior to and during the cease in production of perfluorooctyl compounds by their manufacturer 3M.

Materials and Methods:

Samples. The Canadian Total Diet Study samples all foods that comprise greater than 1% of the average Canadian's diet. Over a five week period each year, various food items are purchased from four different grocery stores and fast food restaurants in a selected Canadian city. Foods are prepared as for consumption, and replicate food items from the various grocery stores or restaurants are combined and homogenized to form a composite sample. Seven fast food composites from the

Total Diet Studies undertaken from 1992 to 2002 (pizza, french fries, hamburger, hot dog, fish burger, chicken burger, chicken nuggets) were analyzed.

Analytical method. Since N-EtPFOSA and PFOSA are estimated to have relatively high log K_{ow}s (6.85 and 4.5, respectively)[9], an analytical method suitable for hydrophobic compounds was used. Approximately 10g of each composite was placed in a polypropylene centrifuge tube. Samples were spiked with methyl perfluorotetradecanoate ($C_{15}H_3F_{27}O_2$, MePFTeD) as a recovery standard and a mixture of $^{13}C_{12}$-labelled penta to octachlorinated PCBs (CB-118, CB-153, CB-180, CB-194) for comparative purposes. Solvent [2:1 (v/v) hexane/acetone] was added to the tube and homogenized using a Polytron mixer. Homogenates were heated in a water bath at 40°C for 30 minutes, and then centrifuged to separate the organic layer from solids. The organic layer was then removed and transferred into a round bottom flask through a bed of activated Na_2SO_4 to remove any residual water. The solvent extraction was repeated, and organic layers were combined, and reduced in volume on a rotary evaporator. Lipid content was determined via gravimetric analysis. Lipids were subsequently removed by washing with concentrated sulfuric acid until the organic layer was colourless. The organic layer was again reduced in volume, and passed through a silica gel column containing 8 g 40% acidified (with sulphuric acid) and 4 g neutral silica gel using dichloromethane as an eluant. The column eluate was reduced in volume to 500 μL and spiked with methylperfluorodecanoate ($C_{11}H_3F_{19}O_2$, MePFD) and $^{13}C_{12}$-CB138 as performance standards. A sample containing Milli-Q purified water was run through the method as a blank concomitantly with each set of fast food composites analyzed.

Samples were analyzed by GC-PCI-MS using an Agilent 5973N mass spectrometer coupled to a 6890 GC fitted with a DB-1701 (30m x 0.25mm i.d., 0.250 μm film thickness) column. The selected ion monitoring mode was used to monitor $[M+H]^+$ ions of all fluorinated compounds. The PCB recovery standards were analyzed using GC-ECNI-SIM-MS on a DB5-MS (30m x 0.25mm i.d., 0.250 μm film thickness) column.

To examine the extent to which N-EtPFOSA and PFOSA were recovered by the method, recent (2002) freshwater fish Total Diet Study composites were spiked with various amounts of each compound (50 ng each N-EtPFOSA, PFOSA; 26 ng each $^{13}C_{12}$-PCB) and run through the method.

Results and Discussion

Analytical method. Average recoveries (± standard deviation, n=5) of N-EtPFOSA, PFOSA, and $^{13}C_{12}$-PCBs in the fortified freshwater fish were 88 ± 9%, 106 ± 15%, and 76 ± 3%, respectively. Recoveries of MePFTeD average 66 ± 3% for the fast food composites analyzed. Method detection limits (MDLs) were calculated as the lowest concentration of analyte in a standard that would produce a signal three times greater than surrounding baseline noise. The MDLs were estimated to be 10 pg/g and 100 pg/g for N-EtPFOSA and PFOSA, respectively. Method quantitation limits (MQLs) were calculated as the concentration of analyte in a standard that would produce a signal ten times greater than surrounding baseline noise (estimated from MDLs as 33 pg/g and 333 pg/g for N-EtPFOSA and PFOSA, respectively).

Concentrations of N-EtPFOSA and PFOSA in Total Diet Study fast food composites. Wet weight concentrations of N-EtPFOSA measured in the fast food composites are given in Table 1. Concentrations shown are recovery corrected. Concentrations ranged from 23 500 pg/g in a pizza composite, to less than the method detection limit of 10 pg/g. Greater than 55% of composites contained N-EtPOFSA; all except one contained concentrations greater than the MQL. However, PFOSA was detected in only three of the composites – french fries 1999 (709 pg/g), pizza 1998 (547

pg/g), and hamburger 1998 (348 pg/g). In the hamburger composite, the concentration of PFOSA was only slightly above the MQL.

Temporal trends of N-EtPFOSA in fast food composites. A sharp decrease in N-EtPFOSA concentrations is observed in composites collected after 1999 for all of the fast foods. This decrease coincided with the commencement of 3M's phase-out of perfluorooctyl chemical production in 2000, which was anticipated to cease entirely at the end of 2002.[10] The presence of N-EtPFOSA in a few composites collected after 2000 may be attributed to use of stockpiled paper/paperboard coating or packaging materials purchased prior to the phase-out of perfluorooctyl compound production.

This decrease in N-EtPFOSA is most likely due to the cessation in production of perfluorooctyl compounds, and will contribute to a decrease in human dietary exposure to N-EtPFOSA. It is expected that a subsequent decrease in human body burdens of metabolites of N-EtPFOSA (PFOSA and PFOS) will also occur, if dietary intake and metabolism of N-EtPFOSA is a significant route of exposure.

Acknowledgement

This work was supported by funding through the Strategic Science Fund, Office of the Chief Scientist, Health Canada.

References

1. Hansen KJ, Clemen LA, Ellefson ME, Johnson HO. 2001. *Environ Sci Technol* 35:766-770.

2. Olsen GW, Hansen KJ, Stevenson LA, Burris JM, Mandel JH. 2003. *Environ Sci Technol* 37:888-891.

3. Olsen GW, Burris JM, Burlew MM, Mandel JH. 2003. *J Occup Environ Med* 45:260-270.

4. Vais N, Kubwabo C, Benoit FM. Pilot study on the determination of perfluorooctanesulfonate and other perfluorinated compounds in blood of Canadians. *Proceedings*: *Health Canada Research Forum*, November 18-20th, 2002, Ottawa, ON, Canada,

5. Tomy GT, Tittlemier SA, Palace VP, Braekevelt E, Budakowski WR, Lau BPY, Eales G, Plohman JC. In vitro biotransformation of perfluorooctane sulfonamide (PFOSA) and ethyl perfluorooctane sulfonamide (N-EtPFOSA) by rainbow trout (*Onchorhynchus mykiss*) liver microsomes. *Proceedings SETAC 23rd Annual Meeting*, November 16-20, 2002, Salt Lake City, Utah, USA.

6. Kudo N, Bandai N, Suzuki E, Katakura M, Kawashima Y. 2000. *Chem-Biol Int* 124:119-132.

7. Yang Q, Xie Y, DePierre JW. 2000. *Clin Exper Immunol* 122:219-226.

8. Hu W, Jones PD, Upham BL, Trosko JE, Lau C, Giesy JP. 2002. *Toxicol Sci* 68:429-436.

9. Grossman MR, Mispagel ME, Bowen JM. 1992.. *J Agric Food Chem* 40:2505-2509.

10. US Environmental Protection Agency. *Phase-out Plan for POSF-Based Products*. AR 226-0600, 3M Co., St. Paul, MN, USA, 2000.

Organohalogen Compounds, Volumes 60-65, Dioxin 2003 Boston, MA

Table 1. Concentrations of N-EtPFOSA (pg/g, wet weight) in fast food composites from the Canadian Total Diet Study, 1992 – 2002.

	Concentration of N-EtPFOSA (pg/g, wet weight)							
Composite	**1992**	**1993**	**1994**	**1998**	**1999**	**2000**	**2001**	**2002**
Chicken Burger				212	98.0	< 10[a]	< 10	< 10
Fish Burger	< 10	< 10	< 10	< 10	1240			
Hot Dog				3450	< 10	< 10	< 10	< 10
Chicken Nuggets				1660	6730	294	709	< 10
Hamburger	< 10	99.9	583	< 10	< 10	< 10	< 10	< 10
Pizza	< 10	3190	576	23500	466	49.8	65.8	< 10
French Fries	12400	6730	8330	1470	1490	213	932	15.1

[a]estimated method detection limit = 10 pg/g wet weight