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ARTICLE

Historic Dietary Exposure to Perfluorooctane Sulfonate, Perfluorinated Carboxylates, and Fluorotelomer Unsaturated Carboxylates from the Consumption of Store-Bought and Restaurant Foods for the Canadian Population

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Perfluorinated compounds (PFCs) have been detected in humans worldwide and are of health concern. This study measured the concentration of PFCs in composite samples collected for the 1998 Health Canada Total Diet Study and estimated dietary exposure for the Canadian population (older than 12 years of age) using previously collected dietary data (n=1721). PFCs were detected in 8 samples including processed meats, preprepared foods, and peppers with a range of concentrations from 0.48 to 5.01 ng g^{-1} (wet weight). 6:2 fluorotelomer unsaturated carboxylate (FTUCA) was detected in cold cuts at a concentration of 1.26 ng g^{-1}. Mean daily PFC exposure estimates ranged from 1.5 to 2.5 ng (kg of body weight)$^{-1}$. Perfluorinated carboxylates (PFCA C_7−C_{11}) contributed more to PFC exposure than either perfluorooctane sulfonate (PFOS) or FTUCA. Total PFCAs in cakes and cookies, lunchmeats, and green vegetables were the main contributors to dietary exposure, although these exposure levels were below the provisional tolerable daily intake provided by the German Drinking Water Commission. Dietary exposure to total PFCs has not changed over time, although the contribution of PFOS to total PFC exposure may have increased between 1998 and 2004. Further research on the sources of contamination of processed and preprepared foods is required. Dietary exposure to PFCs among Canadians poses minimal health risks based on current toxicological information.

KEYWORDS: Perfluorooctanesulfonate; perfluorooctanoate; fluorotelomer; food; diet; Canada

INTRODUCTION

Perfluorinated compounds (PFCs) used in such applications as cosmetics, fire-fighting foams, and water and grease repellent coatings for fabric and food packaging have been identified as global contaminants. PFCs are a group of highly persistent chemicals without known natural sources; their production and use since the 1950s has led to the contamination of wildlife and humans worldwide (see, e.g., refs *1*−*7*). Human exposure to perfluorooctane sulfonate (PFOS), perfluorooctanoate (PFOA), and other PFCs has been observed in nonoccupationally exposed men and women in North America (*1*, *8*−*14*), South America (*1*), Europe (*15*, *16*), Asia (*5*, *17*, *18*), and Australia (*19*). The human health effects associated with chronic exposure to PFOS, perfluorinated carboxylates (PFCAs), and other PFCs are not well understood but require attention on the basis of the known toxicities of PFOA and PFOS (*20*−*23*). Recent studies have documented that food and dust may be major contributors to human exposure to PFOA and PFOS (*24*, *25*); however, the lack of a comprehensive food survey and large variations in PFC concentrations measured in foods create challenges for estimating dietary exposure (*25*). Although the human health risks associated with chronic PFC exposure are not well understood, recent epidemiological studies suggest that developmental effects may arise at current levels of exposure (*26*−*29*).

Environmental and human health concerns regarding PFCs arose following the discovery that throughout the world, humans, wildlife, and abiotic compartments of the environment were contaminated with these persistent, bioaccumulative fluorinated compounds (*30*). The concern over PFOS and PFCA pollution led to the elimination of perfluorosulfonyl-based production in 2001 (*30*) and the Environmental Protection Agency (EPA) program to reduce PFOA emissions and content in products (*31*). Due to its persistence, toxicity, and atmospheric transport, perfluorooctane sulfonic acid, its salts, and perfluorooctane sulfonyl fluoride were recently listed under the Stockholm Convention (*32*).

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Table 1. Instrument Performance Standards Used To Account for Matrix Effects for Each Analyte and MS/MS Multiple Reaction Monitoring Parameters[a]

analyte	performance standard	transition	cone voltage (V)	collision energy (eV)
perfluoroheptanoate (PFHpA)	1,2-^{13}C perfluorooctanoate	362.9 → 318.8	10	15
perfluorooctanoate (PFOA)	1,2-^{13}C perfluorooctanoate	412.9 → 368.8	10	15
		412.9 → 168.8		
perfluorononanoate (PFNA)	1,2,3,4,5-C^{13} perfluorononanoate	462.8 → 418.8	12	18
		462.8 → 218.8	16	18
perfluorodecanoate (PFDA)	1,2-^{13}C perfluorodecanoate	512.8 → 468.9	15	18
		512.8 → 218.9	15	18
perfluoroundecanoate (PFUA)	1,2-^{13}C perfluorodecanoate	562.9 → 518.9	15	15
		562.9 → 268.8		
perfluorooctane sulfonate (PFOS)	L-$^{18}O_2$ perfluorooctane sulfonate	498.9 → 98.9	50	60
		498.9 → 79.9		
6:2 fluorotelomer unsaturated carboxylate (6:2 FTUCA)	2*H*-perfluoro-[1,2-$^{13}C_2$]-2-octenoate	356.9 → 292.9	15	17
8:2 fluorotelomer unsaturated carboxylate (8:2 FTUCA)	2*H*-perfluoro-[1,2-$^{13}C_2$]-2-decenoate	457 → 392.9	18	20
10:2 fluorotelomer unsaturated carboxylate (10:2 FTUCA)	2*H*-perfluoro-[1,2-$^{13}C_2$]-2-dodecenoate	557 → 493	20	25

[a] For analytes in which two transitions were monitored the first transition listed was used for quantitation.

Levels of PFCs in the Canadian population are similar to those in United States and Europe according to a recent pilot biomonitoring study (*10*). Several studies have been conducted worldwide to estimate dietary exposure to PFCs using various analytical methods and sampling techniques (*24*, *33*−*35*). Daily dietary exposure to total PFCs has been estimated to range from 0.89 ng (kg of bw)$^{-1}$(*33*) to 100 ng (kg bw)$^{-1}$ (*35*), although the elevated exposure estimate reported by Mortimer et al. (*35*) was associated with high concentrations of PFOS and PFOA in one composite sample analyzed. Many of these earlier studies measured PFCs only in selected food products, for example, fast foods and animal products (*24*), homogenized meals (*34*), and homogenized composite food samples (*33*, *35*). To better understand the sources of PFCs in food there is a need for a comprehensive food survey (*25*).

Nonoccupational exposure to elevated levels of PFOA were not associated with adverse health outcomes in adults (*36*); however, PFOS and POA are able to cross the placenta and therefore may pose a risk to the developing fetus (*17*, *37*). Recent epidemiological studies carried out in Denmark, Japan, and the Unites States have identified associations between exposure to PFCs in utero and low birth weight (*26*−*29*). Fecundity may also be adversely affected by PFOS and PFOA exposure; a recent study found that PFOA and PFOS exposure among Danish women was associated with increased time to pregnancy (*38*), and lower sperm counts were observed in men with higher PFOS and PFOA serum levels (*39*). Although these studies suggest that PFOS and PFOA may adversely affect reproduction in humans at current levels of exposure, past studies have not found that dietary exposure is occurring at levels that pose a risk to human health based on hazard indices, margins of exposure, or provisional reference doses derived from laboratory feeding trials (*34*, *33*, *24*).

In this paper, we present the results from the analysis of PFCs in 65 foods commonly consumed by Canadians. Nine fluorinated compounds [C_7−C_{11} PFCA, PFOS, 6:2 fluorotelomer unsaturated carboxylate (FTUCA), 8:2 FTUCA, 10:2 FTUCA] were analyzed in archived food samples collected in stores and restaurants in Whitehorse, Yukon Territory, Canada, in 1998 for the Total Diet Study (TDS) conducted by Health Canada (*40*). In 1998, a Canadian cross-sectional food-use survey was conducted that included 1721 individuals (1543 adults randomly selected across the country and 178 adolescents selected from participating households) (*41*). This provided an opportunity to estimate PFC exposure for the average Canadian population at that point in time by the combination of the two data sets.

The objective was to estimate total PFC exposure for the Canadian population and to identify key food items that contributed to PFC exposure. Furthermore, the reanalysis of current dietary exposure for Canadians using recent dietary intake information allowed us to compare dietary exposure from the late 1990s and 2004. These results will provide valuable data for reducing dietary exposure to PFCs and monitoring changes in dietary exposure over time.

MATERIALS AND METHODS

Standards and Reagents. Nine perfluorinated and fluorotelomer compounds (purity > 95%) were used as standards: perfluoroheptanoic acid (Aldrich, Oakville, ON, Canada); perfluorooctanoic acid (Wellington Laboratories, Guelph, ON, Canada); perfluorononanoic acid (Aldrich); perfluorodecanoic acid (Aldrich); perfluoroundecanoic acid (Aldrich); L-perfluorooctane sulfonate (Wellington); 2*H*-perfluoro-2-dodecenoic acid (Wellington); 2*H*-perfluoro-2-decenoic acid (Wellington); and 2*H*-perfluoro-2-octenoic acid (Wellington). Mass-labeled perfluorinated and fluorotelomer compounds were used as recovery and internal performance standards: 1,2-^{13}C-perfluorooctanoic acid (Perkin-Elmer, Boston, MA; 98% chemical purity, 99% isotopic purity); perfluoro-*n*-[1,2,3,4-$^{13}C_4$]octanoic acid (Wellington); 1,2-^{13}C-perfluorononanoic acid (3 M, 95% chemical purity, 99% isotopic purity); $^{13}C_5$-perfluorononanoic acid (Wellington); 1,2-^{13}C-perfluorodecanoic acid (Wellington Laboratories, 98% chemical purity, > 99% isotopic purity); sodium 1,2,3,4-^{13}C-perfluorooctane sulfonate (Wellington, 98% chemical purity, > 99% isotopic purity); L-$^{18}O_2$-PFOS (RTI International, Research Triangle Park, NC); 2*H*-perfluoro-[1,2-$^{13}C_2$]-2-octenoic acid (Wellington); 2*H*-perfluoro-[1,2-$^{13}C_2$]-2-decenoic acid (Wellington); and 2*H*-perfluoro-[1,2-$^{13}C_2$]-2-dodecenoic acid (Wellington).

Given that it was not feasible to prepare matrix-specific calibration curves for the 65 food samples analyzed, matrix effects on ionization were accounted for through the use of instrument performance standards (*42*). The instrument performance standards that were used to correct for matrix effects are presented in **Table 1**. All water used in the method was Milli-Q purified (Millipore, Billerica, MA) and passed through a glass column containing Amberlite XAD-7 resin (Aldrich) to remove any possible perfluorinated contaminants. Methanol (MeOH; Optima, FisherScientific), ammonium hydroxide (Baker analyzed, 29% purity), glacial acetic acid (FisherScientific HPLC grade), and anhydrous sodium acetate were used without extra purification. Weak anion exchange (WAX) cartridges (OasisWax, 6 cm^3, 150 mg, 30 μm) solid phase extraction (SPE) cartridges were purchased from Waters (Milford, MA).

Food Samples. Archived composite samples of store-bought and restaurant foods were analyzed. Food items were purchased in 1998 from four grocery stores and restaurants in Whitehorse, YT, Canada, and were prepared as for consumption. Individual replicates ($n = 4$) for each food were homogenized and then combined to produce composite food samples for various analyses. All samples were stored in polypropylene Nalgene containers at −20 °C until analysis. Previous studies have reported results

Table 2. Summary of Composite Food Samples Analyzed for PFCs and FTUCAs[a]

food group	composite samples analyzed for PFCs ($N = 65$)
meat, poultry, fish, eggs ($N = 9$)	cold cuts and luncheon meats, luncheon meats (canned), canned fish, eggs, beef (steak), beef (roast and stewing), beef (ground), pork, wieners
dairy ($N = 8$)	cheese (processed Cheddar), cheese, ice cream, cottage cheese, butter, milk (whole), milk (2%), evaporated milk (canned)
fast foods and preprepared foods ($N = 21$)	pizza, French fries, fish burger, chicken, cookies, frozen dinner, frozen dinner (microwave or oven), pasta (plain), pasta (mixed dishes), jams, danish and donuts, potato chips, candy and chocolate bars, baked beans, cooking fats and salad oils, soup (tomato, canned), soup (meat, canned), soup (cream of vegetable, canned), soup (dehydrated), apple pie, pancakes
fruits, vegetables, grains ($N = 20$)	bread (whole wheat), tomato sauce, cauliflower, mushrooms (canned), cabbage, peppers, corn, peas, rice, citrus fruit (raw), lettuce, potatoes (boiled), potatoes (baked), cereal (cooked wheat), cereal (wheat and bran), cereal (oatmeal), cereal (corn), crackers, wheat flour, white sugar
beverages ($N = 7$)	citrus juice (canned), alcoholic drinks (beer), citrus juice (frozen), apple juice (canned, unsweetened), soft drinks, coffee, tea

[a] All samples were purchased in Whitehorse, Yukon Territory, in 1998 for the Health Canada Total Diet Study.

from the analysis of PFOS, PFCAs, and FTUCAs in biological samples that were stored at −20 °C for > 10 years (*4*, *43*, *44*). Samples are frequently conserved by storage in a freezer (*45*); freeze−thaw cycles and storing samples at −20 or 4 °C did not change PFOS, PFOA, and PFNA concentrations in blood samples by > 5% (*46*). PFCAs were found to be stable in sewage sludge when frozen at −20 for > 6 months (*47*). Furthermore, there was no difference in moisture content in composite food samples (French fries) over 12 years in composite samples collected for the Canadian TDS (*48*), which suggests that the moisture content in the TDS samples from 1998 was not affected by storage at −20 °C. The food samples analyzed correspond to foods consumed by Canadians based on 24 h recalls carried out in the 1998 Food Habits of Canadians study (*41*). In total, 65 composite samples including drinks ($n = 4$), plant-based foods ($n = 24$), meat and fish ($n = 10$), milk products ($n = 7$), fats and oils ($n = 2$), fast foods and prepared meals ($n = 16$), and sugar ($n = 2$) were analyzed (**Table 2**).

Sample Preparation. The analytical method used has been previously described in detail (*49*). In brief, a methanol extraction modified from that of Tittlemier et al. (*50*) was used; samples (1 g of cooked sample or 2 g of uncooked sample) were spiked with 5000 pg of internal recovery standard (50 μL of 100 pg μL^{-1} solution made up of $^{13}C_4$ PFOA, $^{13}C_2$ PFNA, and $^{13}C_4$ PFOS). Four milliliters of methanol was added to samples, and the samples were shaken using an orbital shaker at 200 rpm and 25 °C for 4 h, followed by vortex-mixing and centrifugation at 667*g* and 10 °C for 10 min. The supernatant was transferred to a polypropylene centrifuge tube precleaned with methanol, and two more extractions were carried out under the following conditions: 2 mL of methanol was added to the sample, which was vortexed and then placed on the orbital shaker for 10 min and centrifuged (same conditions as above). Clean-up involved an SPE process in which samples were diluted to 50 mL with water and were passed through preconditioned WAX cartridges. Eluate was dried to 0.5 mL under a gentle stream of nitrogen and vortexed; internal performance standard (20 μL of a 100 pg/μL solution) and 230 μL water were added to a 250 μL aliquot of sample.

Instrumental Analysis. Samples were analyzed using liquid chromatography negative electrospray tandem mass spectrometry (LC-MS/MS) using the methods described previously (*49*). In brief, samples (10 μL injection) were chromatographed on a 2.1 × 50 mm Genesis C_{18} analytical column (Jones Chromatography Ltd., Hengoed, Mid Glamorgan, U.K.) and a C_{18} guard column (4 mm × 2.0 mm i.d.; Phenomenex, Torrance, CA) installed on an HP 1100 binary pump high-performance liquid chromatograph (Agilent, Palo Alto, CA). The liquid chromatograph was connected to a VG Quattro II triple-quadrupole mass spectrometer (Micromass, Manchester, U.K.). Analytes and the transitions monitored are provided in **Table 1**: two transitions were monitored for all native analytes (except PFHpA), and one mass transition was monitored for the mass-labeled standards (internal and recovery standards).

Quantitation. Details about the methods for quantification are provided elsewhere (*49*). Briefly, quantitation was based on response factors calculated for an external five-point calibration curve made up in methanol and water (1:1 ratio). Relative response factors were calculated for the analytes of interest in samples based on the ratio of the peak area of the target analyte and that of the corresponding mass-labeled internal performance standard (**Table 1**). Instrument detection limits (IDL) for analytes were calculated for each sample as the concentration for which the corresponding peak had a signal-to-noise ratio of 3. Method detection limits (MDL) were 3 times the standard deviation of the blanks plus the instrument detection limit (*51*) divided by sample weight. The limit of quantification (LOQ) was determined as 3 times the method detection limit. Concentrations below the LOD were entered as zero for the estimation of dietary exposure.

Quality Assurance and Quality Control. Quality assurance and control steps included methanol laboratory blanks, fortified matrix samples, and the addition of isotope-labeled internal recovery standards and internal performance standards to each sample. One methanol blank and one homogenized raw ground beef sample fortified with target analytes (50 μL of a 100 pg μL^{-1} standard solution containing PFHpA, PFOA, PFNA, PFDA, PFUA, PFOS, 6:2 FTUCA, 8:2 FTUCA, 10:2 FTUCA) were included in each batch of samples. Blank-subtraction of the samples was carried out to account for the presence of analytes in methanol blanks. Method validation steps involved the recovery analysis for seven spiked food samples representative of various matrices in the diet.

Dietary Exposure Estimate and Statistical Analysis. Dietary exposure to PFCs was estimated for Canadians on the basis of food intake data collected through 24 h dietary recalls in 1998 for the Canadian Food Habits study (*41*). Trained dieticians administered 24 h dietary recalls between August 1997 and July 1998 to 1542 adults (18−65 years old) and 178 adolescents (13−17 years old). The surveys were home-administered, and participants provided detailed information regarding their diet, including drinks, during the previous 24 h using food portion models to estimate food portion sizes. PFC dietary exposure estimates were generated for the 1990s using the concentrations of PFCs measured in TDS samples from 1998. To assess changes in PFC exposure over time, we re-evaluated 2004 dietary exposure for the Canadian population (*24*) using recent dietary intake information (*41*). We calculated 2004 exposure using either the concentrations of PFCs reported in the 2004 TDS samples (*24*) or a combination of data from the 1998 and 2004 TDS. For the exposure estimate in which we combined concentration data from 1998 and 2004, we included samples that had not been analyzed in 2004 that had been analyzed in 1998 (natural and processed cheese, green peppers) and we also included concentrations of FTUCAs measured in the 1998 TDS (6:2 FTUCA in lunchmeat), given that FTUCAs were not analyzed by Tittlemier et al. (*24*).

Food intake data from 24 h recalls were organized into 123 food groups based on similarities in nutrient content. To estimate dietary exposure, we matched the composite samples from the TDS with food groupings from the 24 h recalls, based on the contents of each food group. The concentrations of PFCs in each food group were multiplied by the quantities of these foods consumed by participants in the 24 h recalls ($n = 1721$). Concentrations below the LOD were entered as zero. Daily dietary exposure was calculated for each individual by summing the intake of PFCs from each food group. Population-level dietary exposure estimates for the late 1990s were generated for four age categories of men and women (13−19, 20−40, 41−60, > 60 years old). All statistical analyses were performed using SAS (Cary, NC, v. 9.1). Dietary exposure estimates at the population level for Canadian males and females were calculated for 1998 and 2004 and compared to exposure estimates reported previously (*24*).

Table 3. Frequency of Detection and Concentration of Analytes in Methanol Blanks Converted from Picograms per Microliter to Nanograms per Gram for a Hypothetical 2 g Sample[a]

analyte	no. detected/no. analyzed	mean concentration (ng g^{-1})	standard deviation	method detection limit (ng g^{-1})
6:2 FTUCA	1/7	nd	0	0.27 ± 0.16
PFHpA	4/7	0.10	0.05	0.35 ± 0.10
PFOA	1/7	0.02	0.03	0.23 ± 0.10
PFNA	4/7	0.32	0.23	0.97 ± 0.31
8:2 FTUCA	1/7	0.01	0.01	0.05 ± 0.02
PFOS	4/7	0.05	0.03	0.21 ± 0.10
PFDA	1/7	0.06	0.08	0.50 ± 0.15
PFUA	0/7	nd	0	0.44 ± 0.15
10:2 FTUCA	0/7	nd	0	0.54 ± 0.49

[a] Mean method detection limits (± standard deviation) for analytes (mass-labeled and native compounds).

Table 4. Blank Corrected Concentrations (Nanograms per Gram, Wet Weight) of PFCs Detected in Canadian Total Diet Study Composite Food Samples[a]

description	6:2 FTUCA	PFHpA	PFOA	PFNA	PFOS	PFDA	total
cold cuts	1.26 (3.72)	<0.41	<0.26	3.75 (5.65)	<0.68	<0.70	5.01
cookies	<<0.22	0.59 (1.05)	0.36 (0.76)	1.72 (2.74)	<0.15	<0.44	2.66
cheese (processed)	<0.21	0.55 (1.11)	0.43 (1.09)	<0.75	1.14	<0.42	2.12
peppers	<0.23	<0.40	0.77 (1.52)	<0.79	<0.15	1.02 (1.33)	1.79
lunchmeats (canned)	<0.78	<0.46	0.52 (1.45)	<1.43	<0.37	<0.77	0.52
pizza	<0.21	0.31 (0.90)	0.42 (0.65)	<0.74	<0.20	<0.41	0.73
cheese	<0.21	<0.25	<0.16	<0.74	0.71 (0.95)	<0.41	0.71
frozen dinner (beef)	<0.22	0.48 (1.30)	<0.16	<0.78	<0.17	<0.44	0.48

[a] Values below the LOQ are followed by the LOQ value in parentheses.

RESULTS

Quality Control and Quality Assurance. Concentrations of PFCs detected in methanol blanks are presented in **Table 3**. Levels in blanks frequently exceeded 10% of the concentration detected in the samples; therefore, sample concentrations were blank-subtracted. 10:2 FTUCA and PFDA were not detected in the blanks. Average percent recoveries of PFCs in the fortified beef reference material ($n = 6$, ± standard deviation) were 63 ± 6 (PFHpA), 76 ± 12 (PFOA), 81 ± 25 (PFNA), 40 ± 43 (PFDA), 6 ± 11 (PFUA), 54 ± 13 (PFOS), 53 ± 29 (6:2 FTUCA), 57 ± 36 (8:2 FTUCA), and 53 ± 11 (10:2 FTUCA). Average recoveries ($n = 73$, ± standard deviation) for the three internal recovery standards in samples were 84 ± 20%, 77 ± 29% ($n = 73$), and 81 ± 23% ($n = 73$) for $^{13}C_4$ PFOA, $^{13}C_2$ PFNA, and $^{13}C_4$ PFOS, respectively. Concentrations measured in samples were not recovery-corrected. The mean MDL (ng g^{-1}, ± standard deviation) were the following: 0.27 ± 0.16 (6:2 FTUCA), 0.05 ± 0.02 (8:2 FTUCA), 0.54 ± 0.49 (10:2 FTUCA), 0.35 ± 0.10 (PFHpA), 0.23 ± 0.10 (PFOA), 0.97 ± 0.31 (PFNA), 0.50 ± 0.15 (PFDA), 0.44 ± 0.15 (PFUA), and 0.21 ± 0.10 (PFOS) (**Table 3**).

Concentrations in 1998 TDS samples. PFCs were detected in 8 of the 65 composite samples analyzed (**Table 4**). PFCs were below the MDL in all unprocessed meats, breads, cereals, fruits, and beverages. The concentration of PFCs was below the LOQ in all composite samples with the exception of PFOS in processed cheese. PFHpA and PFOA were detected most frequently (4 and 5 samples, respectively). PFOS was detected in 2 samples, and the concentration exceeded the LOQ only in processed cheese (1.14 ng/g). PFOS was detected in processed cheese and regular cheese but not in the other dairy samples analyzed (i.e., milk, ice cream, cottage cheese, or butter).

Longer chained PFCAs (C_9−C_{10}) were detected in three composite samples. PFNA was detected in cold cuts and cookies, and PFDA was detected in peppers. PFUA was not detected in any samples analyzed. 8:2 and 10:2 FTUCA were not detected in any samples, and 6:2 FTUCA was measurable only in the cold cuts composite samples (1.26 ng g^{-1}). 6:2 FTUCA was above the instrument detection limit in wieners and chicken, but at concentrations below the MDL.

Dietary Exposure in the Late 1990s. For the purpose of this study, it was assumed that the detection of PFCs and FTUCAs in composite samples collected for the 1998 TDS from supermarkets and restaurants in Whitehorse, YT, would be similar to levels in foods from other Canadian cities. The majority of foods included in the 1998 TDS were from nationally available brands; therefore, they would be consumed elsewhere in Canada. Furthermore, the analysis of pesticides in the 1998 TDS samples from Whitehorse, YT, indicated that the concentration and frequency of pesticide detection were similar between food samples from Whitehorse, YT, and southern Canadian cities (*52*). It is likely that the PFC residue levels would follow a similar trend, although more studies are needed to improve our understanding of the sources of PFCs in the food system.

Estimated dietary exposure to PFCs based on the dietary intake of Canadians in the late 1990s (*44*) and the analytical data from the 1998 TDS ranged from 97 to 113 ng day^{-1} [1.5−1.9 ng (kg of bw)$^{-1}$ day^{-1}] for women and from 123 to 157 ng day^{-1} [1.6−2.5 ng (kg of bw)$^{-1}$ day^{-1}] for men, with the highest intake of PFCs estimated for males under 20 years of age (**Figure 1**). Dietary exposure estimates for PFOS, 6:2 FTUCA, and PFCA (C_7−C_{11}) are presented in **Figure 1**. The intake of ΣPFCAs [1.3−2.1 ng (kg of bw)$^{-1}$] exceeded that of 6:2 FTUCA [0.1 ng (kg of bw)$^{-1}$] and PFOS [0.1−0.2 ng (kg of bw)$^{-1}$]. Total PFCA exposure was consistent among age and gender groups; PFNA intake was greatest and accounted for 37−56% of ΣPFCA exposure [0.5−1.2 ng (kg of bw)$^{-1}$ day^{-1}]; PFOA and PFHpA contributed 17−26% [0.2−0.4 ng (kg of bw)$^{-1}$ day^{-1}] and 16−24% [0.2−0.5 ng (kg of bw)$^{-1}$ day^{-1}], respectively, and PFDA contributed the least to PFCA exposure [2−19%, 0.1−0.3 ng (kg of bw)$^{-1}$ day^{-1}].

Mean sources of exposure to PFCs for the Canadian population in 1998 are provided in **Figure 2**. Cakes and cookies contributed 41% to daily dietary exposure to PFCs. Lunchmeats and green vegetables contributed 23 and 16%, respectively, to daily PFC exposure. Natural cheese, mixed beef dishes, processed

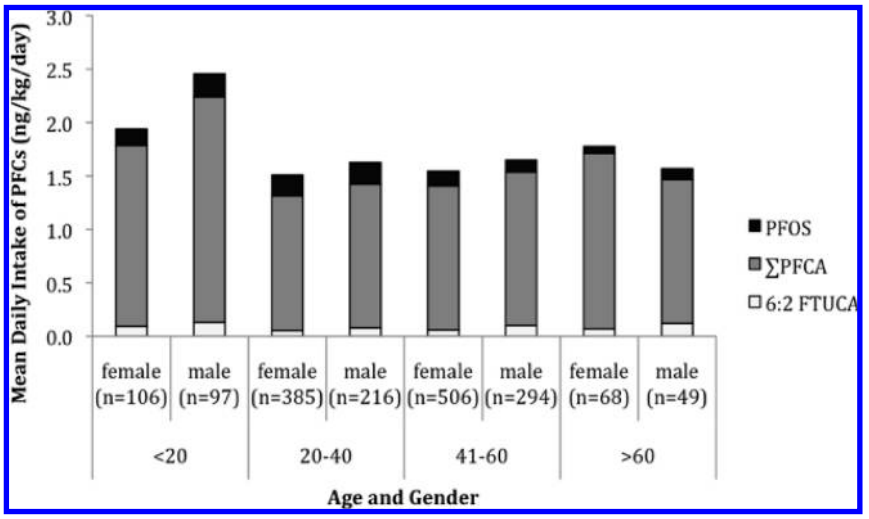

Figure 1. Estimated dietary exposure to PFOS, ΣPFCAs, and 6:2 FTUCA for the Canadian population in the late 1990s. Dietary intake data from the Food Habits of Canadians Study (*44*) were multiplied by PFC concentrations in 1998 TDS samples collected in Whitehorse, YT, in 1998.

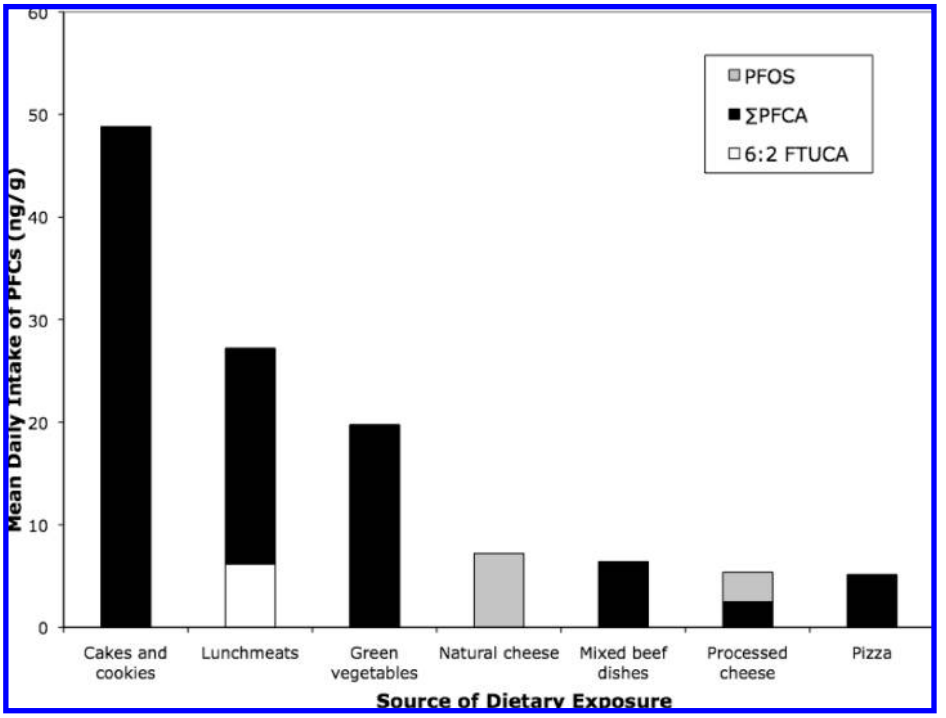

Figure 2. Sources of dietary exposure to PFOS, ΣPFCAs, and 6:2 FTUCA for Canadians in the late 1990s based on the analysis of 1998 TDS samples collected in Whitehorse, YT, and dietary intake data from the Food Habits of Canadians Study (*44*).

cheese, and pizza each contributed < 10% to daily PFC exposure. Lunchmeats were the only source of 6:2 FTUCA, and cheese (natural and processed) was the only source of PFOS identified in 1998.

Dietary Exposure among Canadians between 1998 and 2004. Dietary exposures to PFCs were very similar in 1998 and 2004, when both estimates were derived using 1998 dietary intake data (*41*) multiplied by analytical data from 1998 TDS samples (this study) or 2004 TDS samples (*24*). Dietary exposure to PFCs for the Canadian population was estimated to range from 1.5 to 2.5 ng (kg of bw)$^{-1}$ day^{-1} (97−157 ng day^{-1}) in 1998 and from 1.2 to 3.1 ng (kg of bw)$^{-1}$ day^{-1} (75−200 ng day^{-1}) in 2004. Mean exposure to PFCs was estimated to be 133 ng day^{-1} among males and 100 ng day^{-1} among females in 1998 and 180 ng day^{-1} among males and 101 ng day^{-1} among females in 2004 (**Figure 3**). Estimated intakes of PFOA and PFNA were similar in 1998 and 2004. PFOA intake ranged from 0.2 to 0.4 ng (kg of bw)$^{-1}$ day^{-1} in 1998 and from 0.1 to 0.4 ng (kg of bw)$^{-1}$ day^{-1} in 2004; PFNA exposure ranged from 0.5 to 1.2 ng (kg of bw)$^{-1}$ day^{-1} in 1998 and from 0.2 to 0.7 ng (kg of bw)$^{-1}$ day^{-1} in 2004. However, dietary exposure to PFOS was estimated to be an order of magnitude higher in the Canadian population in 2004 [0.8−2.0 ng (kg of bw)$^{-1}$ day^{-1}) than in 1998 [0.1−0.2 ng (kg of bw)$^{-1}$ day^{-1}].

Comparison of Methodologies. PFC exposure estimates calculated using analytical data from the 1998 and 2004 TDS and dietary intake data from the 1990s (*44*) and 1970s (*53*) are provided in **Figure 3**. The use of 1972 dietary intake information to estimate current dietary exposure for Canadians [as presented by Tittlemier et al. (*24*)] resulted in an exposure estimate that was

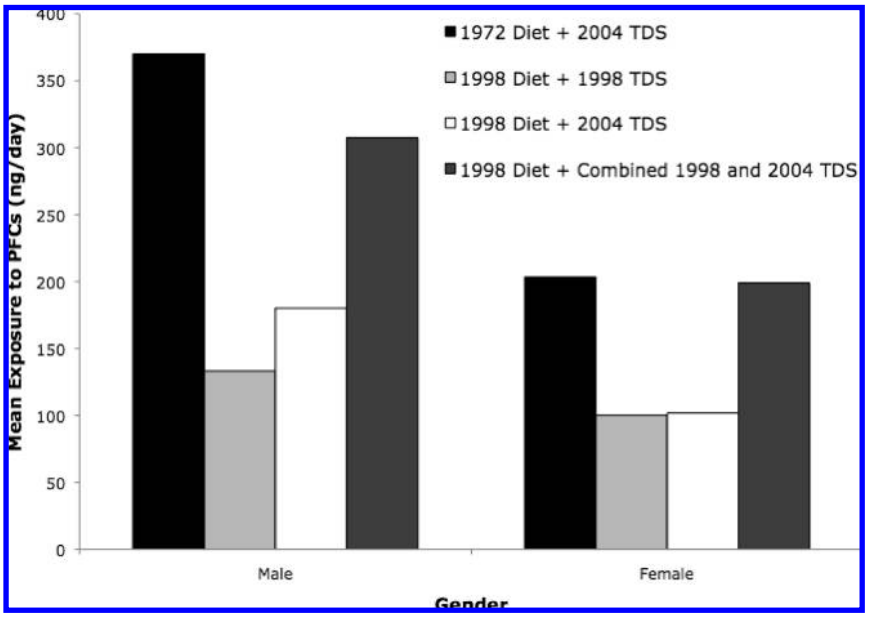

Figure 3. Estimated dietary exposure to ΣPFCs and FTUCAs for Canadians. The 1998 Diet refers to the dietary intake for Canadians from the Food Habits of Canadians study (*44*), and the 1972 Diet refers to the diet of Canadians based on the dietary intake data collected in 1972 (*53*). Analytical data from the 1998 TDS (this study), 2004 TDS (*24*), and a combination of the 1998 and 2004 TDS were multiplied by dietary intake data to estimate dietary exposure among Candian males and females.

double that of the estimate generated when 1998 dietary intake information was used (this study), although both estimates used the same analytical data (2004 TDS). When analytical data from the 1998 and 2004 TDS were combined to estimate dietary exposure in 2004 using current dietary intake information, the dietary exposure estimate was higher for both men and women than when 2004 data were used alone, although the estimates were lower than those calculated using 1972 dietary intake information (*24*). Combining the PFC concentrations from the 1998 and 2004 TDS resulted in exposure estimates ranging from 2.9 to 5.5 ng (kg of bw)$^{-1}$ (181−350 ng day^{-1}); mean exposure was estimated to be 307 ng day^{-1} among males and 198 ng day^{-1} among females in 2004 (**Figure 3**). Overall, ΣPFCA exposure was highest and ranged from 1.8 to 3.2 ng (kg of bw)$^{-1}$, followed by PFOS [0.8−2.1 ng (kg of bw)$^{-1}$] and 6:2 FTUCA [0.1 ng (kg of bw)$^{-1}$]. Overall, the contribution of individual analytes to total PFC exposure was similar between age and gender groups; PFNA and PFOS contributed the largest amounts to total PFC exposure (27−39 and 28−38% for PFOS and PFNA, respectively), followed by PFOA (14−18%) and PFHpA (6−10%). Low contributions to PFC exposure arose from both PFDA (1−9%) and 6:2 FTUCA (< 5%).

DISCUSSION

Few composite samples analyzed in this study contained PFCs, and unprocessed foods were generally free of PFCs. PFCs were most frequently detected in foods that had undergone a form of processing, including industrial preparation (e.g., cookies), or mixing and cooking (e.g., lunchmeats, processed cheese), which suggests that contamination occurs after the initial production or cultivation of various foods. PFCs were not detected in milk, unprocessed meat, beverages, and plant products (excluding peppers).

This is the first time that 6:2 FTUCA was found in the food chain. To our knowledge, PFCs have not been analyzed in fluorinated cleaning products or food grade lubricants, although 6:2 fluorotelomer alcohol was detected in Zonyl FSE and other fluorinated materials (*54*). Further study of the presence of PFCAs and FTUCAs in products used by the food industry is warranted given the presence of PFCs in a variety of processed foods. Some PFCs and PFC precursors have been shown to migrate from packaging into foods (*55*) and have been found in packaged foods (*51*), which has been linked to the use of fluorinated grease and water repellent coatings on food packaging. FTUCA, perfluorooctanesulfonamidoethanol (PFOSE), and polyfluoroalkyl phosphate surfactants (PAPs) have been shown to undergo transformation to persistent PFCs such as PFOA and PFOS in the atmosphere (*56*) or in vivo (*57*, *58*).

The detection of PFOA and PFDA in the pepper composite sample suggests that contamination occurred during the cultivation of the peppers, because there is no processing and little packaging of this food. Previous studies have detected PFCs in vegetables, including PFOS and PFOA in potatoes (10 ± 2 and 1 ± 0.2 ng g^{-1}, respectively) (*35*), PFOS in canned vegetables (2 ± 0.4 ng g^{-1}) (*35*), and PFOS in vegetables (0.022 ng g^{-1}) (*33*). PFCAs have been detected in agricultural areas from the application of contaminated sewage sludge (*59*), and this could be the source of contamination observed in the pepper sample. PFCAs have also been detected in rainwater (*60*) and surface water (*5*, *61*−*64*); therefore, these may also be potential sources of contamination during the growing season. The lack of broad contamination of fruits and vegetables with PFCAs suggests that there could be a point source of contamination of peppers in the composite sample.

The concentrations of PFCs detected in the U.K. and Canadian TDS samples (*24*, *35*) were within the range of levels detected in the 1998 Canadian TDS samples analyzed in this study. The concentration of PFCs measured in 18 food groups by Ericson et al. (*33*) was very low (< 1 ng g^{-1}), which is also consistent with our results. However, our analysis of various dairy products indicates that processed and regular cheese have higher levels of PFOS than other dairy products (ice cream, milk, and cottage cheese), and the concentrations that we measured in the composite cheese samples (1.14 and 0.71 ng g^{-1}) exceeded those measured by Ericson et al. in the dairy product composite sample (0.121 ng g^{-1}) (*33*). In their study, PFHpA was detected only in

whole milk (*33*), which differs from our study in which PFHpA was detected in cookies, processed cheese, pizza, and frozen beef dinner. It is possible that combining a variety of foods into single food groups will dilute PFCs present at low concentrations; our data suggest that food with similar compositions but different preparatory steps (e.g., lunchmeats vs ground beef) will have different concentrations of PFCs.

PFOS was below the MDL in meat composite samples measured in this study, which differed from results reported in previous studies. PFOS was detected in fish, meat, and egg samples from the Catalan market in Spain (*33*); however, the concentrations reported for meat samples (<0.1 ng g^{-1}) were below the limits of detection in our study (0.21 ng g^{-1}). PFOS was not detected in meat and fish samples from the 1998 TDS samples that we analyzed. However, PFOS was detected in the following 2004 TDS samples: lunchmeats, beef steak, roast beef, and freshwater fish (*24*). One freshwater fish sample from the 1998 TDS was analyzed by Tittlemier et al. (*24*) and had 1.5 ng g^{-1} PFOS. We did not reanalyze this sample, but the canned fish and fish burger composite samples did not have detectable levels of any PFCs. There has been concern about false-positive results for PFOS when the 499 to 80 mass transition was used for quantification, given that taurodeoxycholic acid, a compound that has been found to coelute with PFOS, is detected at the mass transition 498 to 80 (*65*). In both our study and the study by Tittlemier et al. (*24*), PFOS was quantified by monitoring the mass transition 499 to 99 instead of 499 to 80. Therefore, it is unlikely that differences in the detection of PFOS were due to the false-positive identification of PFOS in the study by Tittlemier et al. (*24*).

Differences in PFOS concentrations in 1998 TDS samples (this study) compared to levels reported in previous studies could be the result of inconsistent levels of PFOS in market foods, spatial and temporal variation in PFOS concentrations in biota, or analytical differences (sample extraction and cleanup, detection limits, mass transitions monitored for quantification, etc). PFOS has been detected inconsistently in fish and meat samples analyzed previously (*33*, *35*, *42*). For example, in the study of Canadian TDS samples by Tittlemier et al. (*24*), PFOS was detected in the beef steak and ground beef samples, but was below the LOD in roast beef, beef liver, wieners, and hamburger samples. PFOS was also below detection limits in other animal products including lamb, veal, and pork. The levels of PFOS detected in the two beef samples from the 2004 Canadian TDS were higher than those presented by Ericson et al. (*33*) for the veal composite sample purchased from the Catalan market in 2006 (2.7 and 2.1 ng g^{-1} ww compared to 0.028 ng g^{-1} ww). Given that taurodeoxycholic acid may interfere with identification and quantification of PFOS, future monitoring studies should consider using an appropriate ion exchange column that is capable of separating PFOS and taurodeoxycholic acid effectively (*65*).

The study by Reagen et al. (*66*) demonstrated that the accuracy of results varied when solvent calibration curves were used with ion-pairing extraction data. Regional variation in PFOS contamination is possible for livestock, given that PFOS and PFOA were transferred from contaminated soil to plants in a concentration-dependent manner (*67*). Livestock exposure to PFCs may vary depending on the source of their feed. PFOS concentration in oceans and rivers was found to vary with location in past studies, which could affect concentrations of PFC accumulating in fish. Coastal water close to urban centers and river water below wastewater treatment plants were more highly contaminated than deep seawater and river water upstream from wastewater treatment plants (*6*, *68*, *69*). The inconsistent detection of PFOS in meat and fish samples suggests that more studies are needed to elucidate the sources of PFOS contamination of meat products and that analytical methods should be standardized to enable comparison between studies.

Estimating dietary exposure to PFCs at the population level requires accurate dietary intake information; the 24 h dietary recall provides a snapshot of the diet of individuals, which is useful for estimating mean intake for a population (*70*) and provides more accurate intake assessments than food frequency questionnaires, because memory is less of a problem (*71*). Although the accuracy of exposure estimates at the individual level suffers from the use of 24 h dietary recalls, it is expected that at the population level, dietary exposure estimates derived from 24 h dietary recalls may be used to assess mean exposure for large group of individuals (*72*). The accuracy of estimates for chronic dietary exposure to contaminants would be improved by using longer term dietary intake data; however, short-term dietary surveys are expected to err on the side of safety by providing a relatively accurate estimate for high-end consumers (*73*). Twenty-four hour dietary recalls provide an estimate of dietary intake for large groups (*74*) and have been used previously for estimating dietary exposure to contaminants among Inuit and First Nations in Canada (*75*, *76*).

The dietary exposure estimates for Canadians in the late 1990s were lower than the range of exposure estimates derived from previous Canadian and U.K. dietary exposure estimates (*24*, *35*) but were similar to exposure estimates from Ericson et al. (*33*). The previously reported estimate of PFC intake for Canadians was based on the analysis of PFCs and perfluorooctane sulfonamides (PFOSAs) in a subset of Health Canada's TDS samples, primarily fast food and animal products (*24*, *51*). The analytical data were multiplied with dietary intake data from food surveys conducted in Canada from 1970 to 1972 (*53*). Dietary exposure to PFCs for Canadians in 2004 was estimated to range from 120 to 480 ng day^{-1} (*24*), which is slightly higher than our 1998 dietary exposure estimates (97−157 ng day^{-1}). However, recent dietary intake data collected via 24 h recalls indicate that changes in diet have occurred in Canada since 1970 (*77*).

Dietary exposure to PFOS and total PFCAs and FTUCAs were lower than provisional tolerable daily intake levels established for PFOS and PFOA [100 ng (kg of bw)$^{-1}$ day^{-1}] by the German Drinking Water Commission (*78*), which is consistent with previous dietary exposure estimates (*34*, *33*, *24*). Daily dietary exposures to PFOS and PFOA for Canadians in the late 1990s were estimated to range from 0.1 to 0.2 ng (kg of bw)$^{-1}$ and from 0.2 to 0.4 ng (kg of bw)$^{-1}$, respectively, which were lower than exposure estimates reported elsewhere (*24*, *33*, *34*). Previously reported dietary exposure estimates for PFOS were 1.06 ng (kg of bw)$^{-1}$ day^{-1} (*33*), 1.4 ng (kg of bw)$^{-1}$ day^{-1} (*24*), and 1.8 ng (kg of bw)$^{-1}$ day^{-1} (*24*). The observed differences in PFOS exposure between our study and others are due to the detection of PFOS in fish and meat in the studies by Ericson et al. (*33*) and Tittlemier et al. (*24*). Ericson et al. estimated that the consumption of fish and seafood by adult males in Catalonia accounts for an intake of 62.5 ng of PFOS day^{-1} (*33*). Furthermore, PFOS was detected in beef steak (2.7 ng g^{-1}), ground beef (2.1 ng g^{-1}), fish (2.0−2.6 ng g^{-1}), and lunchmeats (0.5 ng g^{-1}) in the 2004 TDS (*24*); meat and fish contributed 100% to daily dietary intake of PFCA and PFOS for Canadians in 2004 (*24*). However, PFOS was not detected in the meat and fish samples from the 1998 TDS. Dietary exposure estimates for PFOA were also higher in previous studies; estimated exposures to PFOA were 1.1 ng (kg of bw)$^{-1}$ day^{-1} for the Canadian population (*24*) and 2.9 ng (kg of bw)$^{-1}$ day^{-1} for the German population (*34*). The PFNA exposure estimates for Canadians in the late 1990s ranged from 0.5 to 1.2 ng (kg of bw)$^{-1}$ day^{-1}, which is similar to

the exposure estimated for the Canadian population in 2004 [1.1 ng (kg of bw)$^{-1}$ day^{-1}] (*24*). PFNA did not contribute to dietary exposure to PFCs in other estimates, because PFNA was not detected in the duplicate diet study in Germany (*34*) or in market foods collected in Catalonia, Spain (*33*).

Dietary exposure was estimated to range from 75 to 200 ng day^{-1} for the Canadian population in 2004 when recent dietary intake information was used (this study), which is lower than the exposure estimates reported by Tittlemier et al. (120−480 ng day^{-1}) (*24*). The reduction in estimated PFC exposure associated with the use of more recent dietary intake information for Canadians reflects changes that have occurred in the Canadian diet (*82*) and indicate the importance of using current dietary intake information to estimate exposure to contaminants. A comparison of dietary exposure estimates for 1998 and 2004 indicates that overall PFC exposure has not changed over time. However, PFOS exposure was estimated to have increased by an order of magnitude between 1998 and 2004 for the Canadian population. The increase in PFOS exposure was linked to the detection of PFOS in meat and fish samples from the 2004 TDS (*24*). Given that few TDS samples contained detectable levels of PFCs in 1998 (8 of 65) and 2004 (6 of 25), combining these data did not have a large effect on the exposure estimates. The use of a comprehensive analytical data set (65 samples analyzed rather than 25) for estimating dietary exposure changed the exposure estimate by a factor of <2.

However, temporal trends of PFOS in humans do not support an increase in PFOS exposure between 1998 and 2004; instead, studies have found a decrease in blood PFOS, PFOA, perfluorohexane sulfonate (PFHxS), and PFOSA concentrations between these years (no temporal trend was observed for PFNA) in infants in the New York State newborn screening program (*79*). Furthermore, the analysis of PFCs in serum samples from 2000 to 2001 ($n=600$) and plasma samples in 2006 ($n=600$), obtained from the Red Cross in six areas of the United States, suggests that the concentrations of PFOS, PFOA, and PFHxS declined during this time period (*80*). The increase that we estimated for dietary exposure to PFOS among Canadian between 1998 and 2004 must be confirmed via the analysis of serum or plasma samples from this time period.

Gender and age group differences in dietary exposure estimates were observed in our results for 1998 and 2004, when the dietary intake information from the 1990s was used. Differences in absolute dietary exposure between genders is likely due to differences in the quantity of food consumed and differences in food choices. Energy intake was calculated for participants of the Food Habits of Canadians study; total energy intakes were 3045 ± 1159 and 2494 ± 1018 kcal for adult men, which were higher than the intakes for adult women (1931 ± 885 and 1757 ± 693 kcal) (*81*). This gender difference in energy intake would likely lead to a difference in absolute exposure to contaminants, including PFCs. Furthermore, results from this study indicated that males between the ages of 18 and 34 consumed more cheese products, processed meats (sausages, lunchmeats, and bacon), and mixed meat dishes (hamburgers and pizza) than older men (35−65 years old) and adult women (18−65 years old) (*81*). Adolescent males consumed more processed meats (sausages, lunchmeats, and bacon) and cake-like products (cakes, cookies, pies, and granola bars) than adolescent females (*82*). Dietary differences between adolescents and adults in the Canadian population would explain differences in PFC exposure estimates generated in this study. Given that PFCs were detected in the cheese, pizza, and lunchmeat composite samples, it is likely that gender differences in the consumption of these contaminated foods resulted in higher exposure estimates for men than women in 1998 (see **Figure 3**). However, when exposure was corrected for the average mass of individuals in different age and gender groups, exposure differences were no longer apparent (see **Figure 1**).

A reduction in human exposure to PFCs is expected following the phase-out of perfluorosulfonyl-based production in 2001 (*30*) and the Environmental Protection Agency (EPA) program to reduce PFOA emissions and content in products (*31*). This study did not fully address possible exposure to PFOS and PFCA precursors, such as perfluorooctanesulfonamides (PFOSA) and PAPS; a recent study found detectable concentrations of PAP diesters in human serum from the United States collected between 2004 and 2005 (*83*). We anticipate that the phase-out of perfluorooctyl sulfonyl compounds will result in a reduction of sulfonyl-based perfluorinated compounds in store-bought foods; a decreasing trend in perfluorooctanesulfonamide concentrations in fast food in Canada was observed between 1992 and 2004 (*60*). Decreasing levels of PFOS and PFOA have been observed in biomonitoring studies in the United States, suggesting the phase-out of POSF-based materials and PFOA emissions has led to reduced exposure in the American population (*79*,*80*). Given that human exposure to PFCs continues via the consumption of contaminated food (*24*, *33*−*35*, *51*), an improvement in our understanding of the sources of PFCs in the food chain would be useful for further reducing human exposure. The presence of PFOS in food samples after the phase-out of POSF production may reflect the persistence and bioaccumulation of PFOS in the environment.

Given the long half-lives of PFCs and uncertainty in human health impacts of chronic PFC exposure, we recommend the continued monitoring of PFCs, FTUCAs, PAPs, and perfluorinated sulfonamides (e.g., N-EtPFOSA) in market foods to determine if current regulations have resulted in a reduction in dietary exposure to PFCs. This study highlighted the effect of using current dietary intake information on PFC exposure estimates for the Canadian population. Reductions in exposure estimates resulted from the use of current dietary intake data, indicating that appropriate dietary intake data must be used to accurately estimate dietary exposure to PFCs and other contaminants, in order to effectively assess human health risks, evaluate changes in exposure over time, and compare exposures between populations.

ABBREVIATIONS USED

SPE, solid phase extraction; WAX, weak anion exchange; IDL, instrument detection limit; MDL, method detection limit; LOQ, limit of quantitation; PFHpA, perfluoroheptanoate; PFOA, perfluorooctanoate; PFNA, perfluorononanoate; PFDA, perfluorodecanoate; PFUnDA, perfluoroundecanoate; PFOS, perfluorooctane sulfonate; PFHxS, perfluorohexane sulfonate; N-EtPFOSA, *N*-ethyl perfluorooctane sulfonamide; FTUCA, fluorotelomer unsaturated carboxylic acid; POSF, perfluorooctanesulfonyl fluoride; PAPS, polyfluoroalkyl phosphate surfactants.

SAFETY

Current data suggest PFOA is "likely to be carcinogenic for humans" according to the EPA Guidelines for Carcinogen Risk Assessment. PFOS and PFOA cause developmental toxicity.

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