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Dietary Exposure of Canadians to Perfluorinated Carboxylates and Perfluorooctane Sulfonate via Consumption of Meat, Fish, Fast Foods, and Food Items Prepared in Their Packaging

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Human exposure to perfluorinated compounds is a worldwide phenomenon; however, routes of human exposure to these compounds have not been well-characterized. Fifty-four solid food composite samples collected as part of the Canadian Total Diet Study (TDS) were analyzed for perfluorocarboxylates and perfluorooctanesulfonate (PFOS) using a methanol extraction liquid chromatography tandem mass spectrometry method. Foods analyzed included fish and seafood, meat, poultry, frozen entrées, fast food, and microwave popcorn collected from 1992 to 2004 and prepared as for consumption. Nine composites contained detectable levels of perfluorinated compounds—four meat-containing, three fish and shellfish, one fast food, and one microwave popcorn. PFOS and perfluorooctanoate (PFOA) were detected the most frequently; concentrations ranged from 0.5 to 4.5 ng/g. The average dietary intake of total perfluorocarboxylates and PFOS for Canadians was estimated to be 250 ng/day, using results from the 2004 TDS composites. A comparison with intakes of perfluorocarboxylates and PFOS via other routes (air, water, dust, treated carpeting, and apparel) suggested that diet is an important source of these compounds. There was a substantial margin of exposure between the toxicological points of reference and the magnitude of dietary intake of perfluorinated compounds for Canadians ≥ 12 years old.

KEYWORDS: PFOS; PFOA; diet; food; exposure estimate

INTRODUCTION

It is well-known that humans are exposed to perfluorinated compounds. This exposure is a worldwide phenomenon, since two persistent perfluorinated compounds [perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA)] have been found in serum and plasma collected from populations in North America (*1*, *2*), South America (*3*), Europe (*3*), Asia (*4*, *5*), and Australia (*6*).

The routes of human exposure to perfluorinated compounds have not been well-characterized. Because of their chemical resistance and surfactant properties, these compounds are used in a wide variety of industrial and consumer applications including adhesives, cosmetics, cleaners, coatings, and electronics (*7*). There is thus a broad opportunity for human exposure to perfluorinated compounds from this large number of different sources.

Dietary intake is one possible route of exposure. Perfluorinated compounds have already been observed in some foods (*8*, *9*). Dietary intake is the predominant route of exposure for some persistent hydrophobic halogenated contaminants, such as polychlorinated biphenyls (*10*). However, because some perfluorinated compounds, such as PFOS and PFOA, contain both hydrophobic and oleophobic moieties, it is possible that diet may not be as predominant a source of these perfluorinated compounds for humans.

Food can become contaminated with perfluorinated compounds directly from food packaging, since perfluorinated compounds are used in grease and water repellent coatings for food packaging. This scenario has been demonstrated by Begley et al. (*11*) and Tittlemier et al. (*8*). Food can also become contaminated at stages prior to food storage. Food items derived from animals may contain perfluorinated chemicals due to exposure of the animal to air, water, or feed containing perfluorinated compounds.

In addition to diet, the general population may be exposed to perfluorinated compounds by intake of contaminated air, water, dust, or direct physical contact with treated consumer products. A number of perfluorinated compounds have already been measured in air (*12*, *13*), household dust (*14*), and water (*15*, *16*).

This current study investigated the degree to which Canadians are exposed to the most abundant persistent perfluorinated

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Table 1. Canadian TDS Composite Samples Analyzed for Perfluorinated Carboxylates and Sulfonates

TDS composite	year	city	composition
			fast foods
chicken burger	1999	Calgary	plain chicken burger, breaded chicken, no condiments
chicken burger	2004	Winnipeg	plain chicken burger, breaded chicken and grilled chicken, no condiments
chicken nuggets	2004	Winnipeg	breaded chicken nuggets
egg breakfast sandwich	1998	Whitehorse	egg, cheese, and ham on a bun or English muffin
egg breakfast sandwich	1999	Calgary	egg, cheese, and bacon on a bun or English muffin
fish burger	1999	Calgary	plain fish burger, breaded or battered fish, no condiments
fish burger	2000	Ottawa	plain fish burger, breaded or battered fish, no condiments
french fries	1992	Toronto	
french fries	1993	Montreal	
french fries	1994	Winnipeg	
french fries	1998	Whitehorse	
french fries	1999	Calgary	
french fries	2001	St. John's	
french fries	2004	Winnipeg	
hamburger	1992	Toronto	plain hamburger, no condiments
hamburger	1994	Winnipeg	plain hamburger, no condiments
hamburger	2004	Winnipeg	hamburger and cheeseburger with ketchup, mustard, and pickle
hotdog	2004	Winnipeg	hotdog with ketchup or mustard
pizza	1993	Montreal	cheese, pepperoni, sausage, and vegetables topping
pizza	1994	Winnipeg	cheese, pepperoni, sausage, and vegetables topping
pizza	1998	Whitehorse	combination, with ground beef
pizza	1999	Calgary	cheese, pepperoni, mushrooms, and pepper topping
pizza	2001	St. John's	cheese, pepperoni, mushrooms, peppers, and sausage topping
pizza	2004	Winnipeg	cheese, pepperoni, mushroom, green pepper, ham, ground beef, and onion topping
			fish and seafood
fish, freshwater	1993	Montreal	trout
fish, freshwater	1998	Whitehorse	smelt, perch
fish, freshwater	2004	Winnipeg	trout, pickerel
fish, marine	2004	Winnipeg	haddock, cod, sole
fish, canned	1994	Winnipeg	salmon, tuna
fish, canned	2004	Winnipeg	salmon, tuna
shellfish	1998	Whitehorse	shrimp
shellfish	2004	Winnipeg	shrimp
			meat, poultry, and eggs
beef steak	2004	Winnipeg	
ground beef	2004	Winnipeg	
roast and stewing beef	2004	Winnipeg	
lamb	2004	Winnipeg	
pork, cured	2004	Winnipeg	
pork, fresh	2004	Winnipeg	
veal cutlets	2004	Winnipeg	
luncheon meat, cold cuts	2004	Winnipeg	beef bologna and pastrami
luncheon meat, canned	2004	Winnipeg	corned beef, pork luncheon meat
organ meat	2004	Winnipeg	beef liver
pâté	2004	Winnipeg	duck, goose, and chicken liver
wieners	2004	Winnipeg	beef wieners, pork and beef sausages
eggs	2004	Winnipeg	chicken eggs
			preprepared foods
frozen entrée	1998	Whitehorse	pepper steak
frozen entrée	2004	Winnipeg	chicken and pasta; pasta and cheese sauce
microwave popcorn	1999	Calgary	
microwave popcorn	2004	Winnipeg	

compounds observed in human tissues (PFOS and PFOA), plus structurally similar perfluorinated compounds, via food consumption. A suite of conjugate bases of perfluorinated acids (PFAs) including perfluorocarboxylates and PFOS were measured in selected archived food composites collected for the Canadian Total Diet Study (TDS).

MATERIALS AND METHODS

Samples. Forty-nine composite samples (**Table 1**) from the Canadian TDS were analyzed for PFAs. Just over half of the composite samples were from the 2004 TDS; the remaining composites were archived samples collected during TDSs organized from 1992 to 2001. These composites were selected for analysis since they consisted of meat or other animal-derived food items or could have been stored in packaging treated with grease-resistant coatings.

The Canadian TDS is a market basket survey that samples various food items from four different grocery stores and fast food restaurants in a selected Canadian city over a 5 week period each year (*17*). 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. Composites are stored in chemically cleaned polypropylene bottles and lids or glass jars with plastic lids and polytetrafluoroethylene (PTFE) lid liners at −20 °C until analysis. Six composites were available in both glass jars and plastic bottles and were analyzed to examine if the sample container type affected results.

Chemicals and Materials. The following perfluorinated compounds (purity >95%) were used as standards: perfluoroheptanoic acid (Aldrich, Oakville, ON, Canada), perfluorooctanoic acid (Aldrich), perfluorononanoic acid (Aldrich), perfluorodecanoic acid (Aldrich), perfluoroundecanoic acid (Aldrich), perfluorododecanoic acid (Aldrich),

Table 2. MS/MS Multiple Reaction Monitoring Parameters[a]

analyte	instrument performance standard used in quantitation	transition	cone voltage (V)	collision energy (eV)
perfluoroheptanoate (PFHpA)	$^{13}C_2$-PFOA	362.9 → 318.8	17	15
PFOA	$^{13}C_2$-PFOA	412.9 → 368.8	15	10
		412.9 → 168.8	20	19
perfluorononanoate (PFNA)	$^{13}C_2$-PFNA ($^{13}C_2$-PFOA)[b]	463 → 418.8	13	15
		463 → 218.9	20	15
PFOS	$^{13}C_4$-PFOS ($^{13}C_2$-PFOA)[b]	498.9 → 98.9	60	50
		498.9 → 79.9	60	50
$PFMe_2OA$[c]	$^{13}C_2$-PFDA	512.8 → 468.9	18	15
perfluorodecanoate (PFDA)	$^{13}C_2$-PFDA	512.8 → 468.9	18	15
		512.8 → 218.9	18	15
perfluoroundecanoate (PFUA)	$^{13}C_2$-PFDA	562.9 → 518.9	15	15
		562.9 → 268.8	20	15
perfluorododecanoate (PFDoA)	$^{13}C_2$-PFDA	612.8 → 568.8	30	12
		612.8 → 318.9	22	17
perfluorotetradecanoate (PFTeDA)	$^{13}C_2$-PFDA	712.9 → 668.9	22	17
		712.9 → 168.9	17	20
1,2-^{13}C-PFOA ($^{13}C_2$-PFOA)[d]		415 → 369.9	15	12
1,2-^{13}C-perfluorononanoate ($^{13}C_2$-PFNA)[d]		464.9 → 419.9	15	13
$^{13}C_4$-PFOS[d]		502.9 → 79.9	60	50
1,2-^{13}C-perfluorodecanoate ($^{13}C_2$-PFDA)[d]		515 → 470	18	15

[a] For analytes in which two transitions were monitored, the first transition listed was used for quantitation. [b] Initial performance standard used, prior to commercial availability of current performance standard. [c] Recovery internal standard. [d] Instrument performance internal standard.

perfluorotetradecanoic acid (Aldrich), and tetraethylammonium PFOS (Aldrich). Acronyms for these analytes are provided in **Table 2**. Perfluoro-3,7-dimethyloctanoic acid ($PFMe_2OA$; SynQuest Labs, Alachua, FL) was used as a recovery internal standard for all analytes. Mass-labeled 1,2-^{13}C perfluorooctanoic acid (Perkin-Elmer, Boston, MA; 98% chemical purity, 99% isotopic purity), 1,2-^{13}C perfluorononanoic acid (3 M, 95% chemical purity, 99% isotopic purity), 1,2-^{13}C perfluorodecanoic acid (Wellington Laboratories, 98% chemical purity, ≥99% isotopic purity), and sodium 1,2,3,4-^{13}C PFOS (Wellington Laboratories, 98% chemical purity, ≥99% isotopic purity) were used as instrument performance internal standards. The mass-labeled instrument performance standards were used to account for matrix effects on analyte ionization, since it was not feasible to prepare matrix-matched standard calibration curves for all different composite sample matrices. Standards were prepared in a 1:1 (v/v) methanol/water solution.

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; OmniSolv grade, EMD Chemicals, Darmstadt, Germany) was used without extra purification.

Analytical Method. Composite samples were thawed at room temperature and mixed. A 2 g aliquot was taken and placed in a polypropylene centrifuge tube that had been precleaned with MeOH. Recovery internal standard (20 μL of 1000 pg/μL $PFMe_2OA$) was added directly to the aliquot, immediately followed by 4.0 mL of MeOH. Blanks of MeOH were processed concurrently with food composite samples. Tubes were capped, and samples were mixed well on vortex and rotary mixers for 5 min. After mixing, samples were centrifuged for 10 min at 2200*g*. The supernatant was removed using a polypropylene transfer pipet and placed into a precleaned polypropylene centrifuge tube. The extraction was repeated with another 4.0 mL of MeOH. Supernatants were combined and reduced in volume to 2.5 mL in a 37 °C water bath using a gentle stream of N_2. After the supernatants were mixed on a vortex mixer, a 250 μL aliquot was transferred to a polypropylene microcentrifuge tube. Instrument performance standard solution (10 μL of a 200 pg/μL solution) was added to each sample, along with 230 μL of water, to improve the chromatography of analytes. Samples were mixed on a vortex mixer and then centrifuged at 14000*g* for 10 min in a microcentrifuge. Approximately 450 μL of sample was then transferred to a polypropylene autosampler vial, capped, and stored at 4 °C until analysis.

Analyses were performed using liquid chromatography negative electrospray tandem mass spectrometry (LC-MS/MS). Samples (10 μL injection) were chromatographed at ambient temperature on a 2.1 mm × 50 mm Genesis C_{18} analytical column (Jones Chromatography Ltd., Hengoed, Mid Glamorgan, United Kingdom) and 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). A 5 mM solution of ammonium formate in Milli-Q purified water and a 2:1 (v/v) solution of acetonitrile/methanol were used as mobile phase solutions A and B, respectively. The perfluorinated analytes were chromatographically resolved using the following gradient program: 45% B at 0.150 mL/min for 1 min, increasing to 70% B over 4 min, 75% B over 8 min, and 90% B over 3 min, and then held at 90% B for 7 min. The column was then flushed with 90% B at 0.200 mL/min for 1 min and 45% B for 6 min. The liquid chromatograph was connected to a Quattro II triple quadrupole mass spectrometer (Micromass, Manchester, United Kingdom).

Samples were analyzed for PFAs in the multiple reaction monitoring mode. Two transitions were monitored for each analyte (aside from PFHpA); one transition was monitored for $PFMe_2OA$ and each mass-labeled internal standard. Transitions were separated into three chromatographic windows in the multiple reaction monitoring program. **Table 2** lists the monitored transitions for all analytes. Mass spectrometer operational parameters used were as follows: capillary voltage, −3.0 kV; source temperature, 140 °C; and nebulizer and drying gas (N_2) flow rates, 20 and 400 L/h, respectively. Argon was used as the collision gas at 2.0×10^{-3} mbar. Mass resolution for both mass analyzers was set at 1.2 mass units at the base.

Composites from studies prior to the 2004 TDS were analyzed using a slightly different method. One chromatographic window, along with one transition for each analyte, was used. In addition, two mass-labeled compounds ($^{13}C_2$-PFNA and $^{13}C_4$-PFOS) were not available at the time of analysis and were thus not incorporated into the method until the later TDS composites were analyzed.

Peak areas were integrated using QuanLynx software (version 4.0) provided as part of the LC-MS/MS system. Analytes were considered to be positively identified if retention times were within 2.5% of the standard retention time, the peak area was greater than three times the adjacent baseline peak-to-peak noise, and the confirmation transition was present (for applicable analytes). Relative response factors were calculated as the ratio of analyte quantitation transition peak area to corresponding instrument performance internal standard peak area (indicated in **Table 2**). Concentrations of analytes were determined using an external calibration curve.

Limits of Detection (LODs) and Quantitation (LOQs). LODs were estimated on a per sample basis as the lowest analyte concentration that produced a peak with area at least three times greater than adjacent baseline noise. For most analytes and samples, LODs were in the 0.5−1 ng/g range. Two analytes (PFDA and PFTeDA) had LODs that ranged from 2 to 6 ng/g. LOQs were estimated as three times the LOD value.

Dietary Exposure Calculations. The dietary exposure of Canadians (≥12 years old) was estimated in order to approximate the magnitude of dietary exposure to PFAs and to determine the relative importance of dietary exposure to overall PFA exposure. TDS composites were used for this purpose since they were representative of foods actually being consumed. The food items used to prepare TDS composites were purchased in the appropriate commercial packaging, cleaned, otherwise prepared, and cooked prior to homogenization and compositing. Thus, the contaminant concentration data derived from these composites were more useful in estimating dietary exposure than data from unprepared and raw food items. In addition, national intake data for the Canadian TDS composites exist for various age/sex groups (*18*).

A deterministic estimate of dietary exposure to PFAs was made using the available national food intake data for both eaters and noneaters; thus, food intake values include incidences when survey respondents indicated that they did not consume a specific food item. Because the widest variety of TDS composites analyzed in this study were those prepared in 2004, only these composites ($n = 25$) were used for estimating dietary intakes. Concentrations of zero were assigned to composites that were not analyzed and when analyte was not detected above the LOD.

The concentration of individual PFAs in each TDS composite was multiplied by the daily intake of the composite for each age/sex group to calculate PFA dietary intakes. The dietary intake of an individual PFA for each age/sex group was the sum of intakes over all TDS composites. The dietary intakes of individual PFAs were then summed over all PFAs for each age/sex group. The dietary intakes on a body weight basis were calculated by dividing the daily intake of total PFAs for each age/sex group by the average weight of the individuals in the group.

RESULTS

Method Recoveries. Method recoveries were examined by fortifying composites previously determined to be free of detectable levels of PFAs. Replicates of chicken burger ($n = 3$), organ meat ($n = 3$), cured pork ($n = 3$), and meat, poultry, or egg-containing prepared infant food ($n = 3$) composites were fortified with perfluorocarboxylates, PFOS, and $PFMe_2OA$, extracted according to the method outlined above, and analyzed. The pork and infant food composites were fortified at 2 ng/g; the chicken nuggets and organ meat composites were fortified at 10 ng/g. Calculated percent recoveries of analytes and recovery internal standard are given in **Table 3**.

Mean recoveries were generally greater than 80% at both fortification levels, although the longer chain perfluorocarboxylates (i.e., PFUA and greater) could not be detected in the infant food-fortified composite at 2 ng/g since this level was below the LOD for these analytes. In some instances, the mean recoveries of the longer chain perfluorocarboxylates were between 64 and 74%. Recoveries of the individual analytes were not significantly different from the recovery of $PFMe_2OA$ (one-way analysis of variance and Kruskal−Wallis one-way analysis of variance on ranks), indicating that this compound would act as a suitable recovery internal standard for the analytes.

The acceptable recoveries also suggest that matrix effects do not influence quantitation in this method. It appears that the use of mass-labeled structural analogues or mass-labeled homologues (e.g., 1,2-^{13}C-PFOA for PFHpA) as instrument performance internal standards negated effects of matrix on quantitation. It is possible that other matrices aside from the four used during the recovery experiments could have different effects. However, this will mainly impact those compounds that did not have mass-labeled structural analogues.

Table 3. Mean (± Standard Deviation) Recovery of Perfluorinated Carboxylate and Sulfonate from Pre-extraction-Fortified Composites ($n = 3$ Replicates)[a]

compound	cured pork	infant food	chicken nuggets	organ meats
PFHpA	101 ± 5	102 ± 5	108 ± 15	103 ± 7
PFOA	91 ± 9	104 ± 11	116 ± 8	114 ± 1
PFNA	108 ± 7	110 ± 24	103 ± 18	120 ± 13
PFOS	108 ± 18	107 ± 18	85 ± 7	91 ± 5
$PFMe_2OA$	NA[b]	NA	94 ± 4	100 ± 23
PFDA	96 ± 10	97 ± 3[c]	106 ± 3	107 ± 8
PFUA	113 ± 13	–[d]	74 ± 14	81 ± 8
PFDoDA	81 ± 14	–	84 ± 9	71 ± 16
PFTeDA	76 ± 14	–	64 ± 46	73 ± 7

[a] Compounds were fortified at 2 ng/g (in cured pork and infant food composites) and 10 ng/g (in chicken nuggets and organ meats composites). [b] Not analyzed. [c] Analytes could be detected in only two samples above at the fortification level. [d] Analytes could not be detected at the fortification level.

Effect of Container on Analysis. Past studies and reports have alluded to the use of plastic labware to avoid contamination from PTFE and other fluoropolymeric materials (*19*) and to avoid sorption of perfluorinated analytes to glass (*1*, *20*). Because some of the archived TDS composites were only available in glass jars with PTFE lid liners, composites that were available in both polypropylene bottles and glass jars were analyzed to examine if the type of sample container used for storage affected results. Six composites were available in both polypropylene and glass containers (freshwater fish 1993 and 1998, canned fish 1994, shrimp 1998, and two different frozen entreés 1998). Only the freshwater fish sample from 1998 contained an analyte above the LOD or LOQ; PFOS was measured at 1.5 and 1.3 ng/g in the composite stored in polypropylene and glass containers, respectively. The correlation of results from sample stored in the different containers, plus the lack of perfluorinated compounds detected in composites stored in glass containers with PTFE lid liners, suggests that PFOS is not adsorbing to the glass and that the PTFE lid liner is not a source of contamination.

PFAs in TDS Composites. PFAs were detected in nine out of 54 composites analyzed (**Table 4**). Average recovery ± standard deviation of the recovery internal standard was 85 ± 24% ($n = 70$ samples and blanks); results presented in **Table 4** are not corrected for recovery. No perfluorinated analytes were detected in any of the method blanks. PFOS was detected the most frequently in the samples analyzed (seven out of 54 composites), followed by PFOA (three out of 54 composites). The only other PFAs positively detected were PFHpA and PFNA. About half of the PFA residues were observed at trace concentrations (i.e., at concentrations between the LOD and the LOQ). Concentrations of the quantitated PFAs were in the low ng/g range—PFOA (2.6−3.6 ng/g), PFNA (4.5 ng/g), and PFOS (2.0−2.7 ng/g). Average detection limits (estimated as the absolute amount of analyte producing a signal three times greater than the peak to peak noise divided by the sample weight) for the TDS composites ranged from 0.5 ng/g for PFOS to 6 ng/g for PFTeDA.

Correlation of PFA and PFOSAs Concentrations. Thirty-five of the composites analyzed in this study have associated data on perfluorooctanesulfonamides (PFOSAs) (*8*). These data sets were examined in order to determine whether a correlation existed between PFOS and PFOS precursor (i.e., PFOSAs) concentrations in the TDS composites. No statistically significant

Table 4. Concentrations (ng/g, Wet Weight) of Perfluorinated Carboxylates, PFOS, and Unsaturated Fluorotelomer Carboxylates Positively Detected in Canadian TDS Composites[a]

composite	year	PFHpA	PFOA	PFNA	PFOS	PFDA	PFUA	PFDoDA	PFTeDA
beef steak	2004	<0.6	<0.5	4.5	2.7	<2	<1	<1	<3
roast beef	2004	<0.6	2.6	<1	<0.6	<2	<2	<1	<3
ground beef	2004	<0.5	<0.4	<1	2.1	<4	<1	<1	<3
luncheon meats, cold cuts	2004	<0.4	<0.4	<1	(0.5)[b]	<2	<1	<1	<3
fish, marine	2004	<0.4	<0.5	<1	2.6	<2	<1	<0.8	<5
fish, freshwater	2004	<0.4	<0.5	<1	2.0	<2	<1	<0.9	<5
fish, freshwater	1998	<1	<2	<1	(1.5, 1.3[c])	<2	<2	<2	<2
pizza	1998	(2.0)	(0.74)	<1	<1	<1	<1	<1	<1
microwave popcorn	1999	(1.5)	3.6	<1	(0.98)	<1	<0.9	<1	<1

[a] The remaining 44 composites did not contain perfluorinated analytes above the limits of detection. [b] Values in parentheses indicate that the concentration measured was above the LOD but below the LOQ. [c] Results from analysis of an analogous sample that was stored in glass.

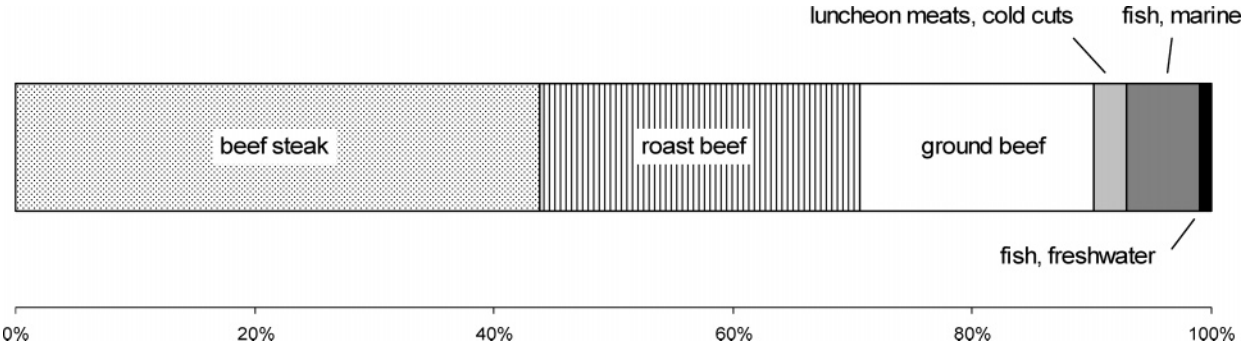

Figure 1. Percentage contribution of 2004 TDS composites to the average daily dietary intake of total perfluorocarboxylates and PFOS for Canadians >12 years old.

Table 5. Estimate of Dietary Exposure to Perfluorinated Carboxylates and PFOS According to Age and Sex of Canadians

group	ng ∑PFAs/person/day
female (12–19 years)	170
female (20–39 years)	200
female (40–64 years)	240
female (>65 years)	120
male (12–19 years)	290
male (20–39 years)	480
male (40–64 years)	340
male (>65 years)	200

correlation was observed for PFOS and total PFOSAs concentrations in the 35 composites or in the subset of 10 composites in which there was a detectable amount of PFOS. However, concentrations of PFOA were significantly positively correlated with PFHpA, PFNA, and PFTeDA ($p < 0.02$, Pearson product moment correlation), as were concentrations of PFNA and PFTeDA ($p = 0.009$).

Dietary Exposure Estimate. Dietary exposure estimates for various age and sex groupings are listed in **Table 5**. The average Canadian's (≥12 years old) dietary exposure to PFAs was 250 ng/day. This estimate was based upon results from the 25 composites collected in the 2004 TDS. Of these 25 composites, six were found to contain detectable levels of PFCs (**Table 4**). Concentrations of PFAs in the remaining 19 composites were assigned a value of zero for the dietary exposure estimate.

Just under half of the average dietary exposure to PFAs is accounted for by PFOS (110 ng/day), followed by PFOA and PFNA (70 ng/day each). The percentage contribution of 2004 TDS composites to the total average dietary intake of total PFAs is shown in **Figure 1**. These are conservative exposure estimates since only 25 of the 140 food composites that were collected as part of the 2004 TDS were analyzed, and values of zero rather than the method LOD were used in the dietary exposure calculations.

DISCUSSION

There are very few reports in the literature that focus on concentrations of PFAs in food. Most research has measured PFAs in liver and plasma sampled from wildlife (for example, see refs *21*−*24*). One study contracted by industry analyzed 10 different raw foods collected from six cities in the southern United States for PFOS and PFOA (*25*). Four whole milk and one ground beef sample contained quantifiable levels of PFOS (0.573−0.852 ng/g); two ground beef, two apple, one bread, and one green bean sample contained quantifiable levels of PFOA (0.504−2.35 ng/g). Only one of these positive results (PFOS in ground beef) was confirmed in its duplicate sample.

Another study performed by the UK Food Standards Agency analyzed 20 composites from the 2004 UK TDS for PFOS, PFOA, and other perfluorinated compounds (*9*). Four composite samples contained detectable levels of PFOS: potatoes (10 ng/g), canned vegetables (2 ng/g), eggs (1 ng/g), and sugars and preserves (1 ng/g). Only the potato composite (which included potato chips, french fries, and other potato products) contained PFOA at a detectable level (1 ng/g).

The concentrations reported in these two food studies were all in the similar range of concentrations observed in the current study. It must be noted that concentrations of PFAs in individual food items used to prepare the composite samples will be higher than those reported for the composite since PFA-free food items in the same composite can effectively act to dilute PFA concentrations in individual food items.

Sources of PFAs in TDS Composites. Environmental exposure of food-producing animals to air, water, or feed containing PFAs can result in PFAs in human food items.

Table 6. Estimate of Adult (Mass, 60 kg) Exposure to Perfluorinated Carboxylates and PFOS

source of PFAs	estimated daily intake (ng/day)	notes	ref
food	250	mean female and male (≥ 12 years old) dietary intake of SPFAs for 2004 Canadian TDS data	this study
water	0.3	calculated from PFOA tap water concentrations for Calgary and Vancouver, Canada	*34*
dust	28	calculated from mean PFOS and PFOA dust concentrations from homes in Ottawa, Canada	*14*
solution-treated carpeting	120	reasonable maximum aggregate adult exposure to PFOA	*35*
treated apparel	12	reasonable maximum aggregate adult exposure to PFOA	*35*
air		negligible due to low vapor pressures of perfluorinated carboxylates and PFOS	
total intake from all sources	410		

Laboratory studies have demonstrated that PFOS and perfluorocarboxylates can bioconcentrate (*26*) and bioaccumulate in fish (*27*); PFAs have also been detected in fish and other biota sampled from various locations (*21*, *22*, *24*). The exposure of food-producing animals to PFA precursors, such as PFOSAs, can also result in PFAs in human food items. Some PFOSAs have been detected in fish and other aquatic organisms (*22*, *23*) and have been shown to be metabolized to PFOS (*28*, *29*).

Food packaging can also serve as a source of PFAs in food items. Fluorotelomer compounds and PFOA have been shown to migrate from treated materials such as microwave popcorn bags into food oil (*11*). Concentrations of N-ethyl PFOSA in some fast food composites collected over the 1990s and early 2000s suggest that food packaging can also be a source of PFOS precursors (*8*). However, because PFOS and PFOSAs concentrations are not significantly correlated, it is not clear whether the PFOS detected in the TDS composites in this study entered the composites from the same source as the PFOSAs.

The positive detection of PFAs in the TDS composites listed in **Table 4** likely reflects both the environmental exposure and the food-packaging sources of entry into prepared foods, since both animal-derived and vegetable-based foods were found to contain PFAs. The fast food and microwave popcorn composites are foods that are often packaged in paper or paperboard that has been treated with fluorinated compounds to impart oil resistance (*7*).

Estimated Dietary Exposure to PFAs. It is clear from the results of this study that food is a source of PFAs for Canadians. Only one other study has estimated dietary exposure to PFAs. The average lower bound dietary intake of PFAs by adults was estimated to be 100 ng/kg body weight/day by the UK Food Standards Agency (*9*). This estimate used data from the 2004 UK TDS, assigned concentrations of zero when PFAs were not detected in samples, and was approximately 25 times more than the dietary intake estimated in the Canadian study (4.0 ng/kg body weight/day; i.e., 250 ng/g divided by an average body weight of 62 kg). The estimated Canadian PFAs dietary intake was also approximately six times greater than dietary intake of PFOSAs analyzed in the same 2004 TDS composites (*8*).

It is not clear at this point which factors contribute the most to the differences between the Canadian and the UK dietary intake estimates. One likely contributor is the difference between the number and the type of composites analyzed. The 20 UK composites analyzed represent a whole diet. The 25 Canadian composites analyzed do not represent the whole diet, only those foods that could have been environmentally exposed through bioaccumulation to perfluorinated compounds (animal-derived foods) or have come into contact with food packaging treated with perfluorinated compounds (fast foods and foods cooked in packaging). These composites analyzed were selected prior to completion of the UK study. Thus, Canadian samples analogous to two of the UK composites found to contain PFAs (canned vegetables and sugars and preserves) were not analyzed.

It is unlikely that differences in dietary intake estimates were heavily influenced by the number of PFAs analyzed in the UK and Canadian study. Six additional PFAs were analyzed during the UK study (*30*). Three of these were not detected in any composites; the remaining two additional PFA analytes were confidently detected in only one composite at levels equal to or less than 5 ng/g wet weight. These two additional PFAs accounted for approximately 10% of the total PFA content of this composite (*30*).

There also appears to be differences in food PFA concentrations contributing to differences in the UK and Canadian dietary intake estimates. The UK dietary intake estimate seems driven by a relatively high concentration (approximately 50 ng/g PFAs) in the potato composite, which contains potato chips, french fries, and other potato products, whereas the 2004 Canadian french fry composite did not contain PFAs. In addition, there may be differences in food intake values, specifically because these values were developed during different decades. The Canadian food intake values used to generate the dietary exposure estimate were the most recent available and were developed in 1972 (*31*). The food intake values used to prepare the UK dietary exposure estimate were generated at least 20 years after the Canadian values.

Dietary exposure to PFAs has also been indirectly examined by Falandysz et al. (*32*), in which they have described a correlation between PFA concentrations in blood sampled from adults in Poland and self-reported consumption of Baltic fish. However, no estimation of dietary exposure to PFAs could be made from this study.

Exposure to PFAs via nondietary routes was estimated using mean reported concentrations of PFAs in dust (*14*) combined with an estimated average adult intake rate of 50 mg/day (*33*), mean concentrations of PFAs in tap water sampled from Calgary and Vancouver (*34*) combined with an estimated average adult intake rate of 1.4 L/day (*33*), and reasonable maximum exposures to treated carpet and apparel (*35*). Exposure via nondietary routes was used in conjunction with the dietary intake estimate to assess overall exposure of adults to PFAs (**Table 6**). The dietary intake PFA exposure estimate of 250 ng/day was on the same order of magnitude as the estimated exposure to PFAs via water, dust, treated carpet, and apparel; it accounted for 61% of the total adult exposure to PFAs. This value is based, however, on a conservative estimate of dietary intake of PFAs; thus, the true contribution of the diet to total intakes could be higher.

Risk Assessment of Dietary Exposure to PFAs. The likelihood that the estimated dietary exposure to PFAs could cause adverse human health effects was investigated using toxicological points of reference for non-neoplastic effects derived during a literature review of toxicological studies. Because of differences in the pharmacokinetics and toxicity of PFOS and PFOA, the risks of dietary exposure to PFOS and perfluorocarboxylates were evaluated separately.

For PFOS, a conservative LOEL (lowest observable effect level) of 0.03 mg/kg body weight/day was identified based on results from a 26 week oral capsule dosing study involving cynomolgus monkeys (*36*) and is in agreement with the LOEL identified in the Health Canada Screening Assessment Report for PFOS (*37*). This LOEL was based on evidence of increased thymic atrophy in females and reduced serum high-density lipoprotein, cholesterol, bilirubin, and triiodothyronine levels in males. For PFOA, a $BMDL_{10}$ (i.e., lower 95% confidence limit of a benchmark dose for a 10% response level) of 0.6 mg/kg bw/day was estimated based on increased relative liver weights in F_0 and F_1 male rats observed in a two-generation reproduction study (*38*).

These toxicological points of reference were compared to the average Canadian's (≥ 12 years old) estimated daily exposure to PFOS and perfluorocarboxylates from food (assuming all perfluorocarboxylates analyzed have the same biological activity as PFOA) to derive margin of exposure (MOE) estimates. An MOE can be defined as a risk comparison value, which is the ratio between a defined toxicological point of departure or reference and a predicted exposure under a given scenario. The MOEs for PFOS and PFOA were greater than 1.6×10^4 and 2.7×10^5, respectively, indicating that a difference exists between the average Canadian's dietary exposure and the doses eliciting effects in feeding studies involving nonhuman primates and rodents. The European Food Safety Authority has recommended that an MOE of 1.0×10^4 or greater that is based on a $BMDL_{10}$ from an animal study would be of low concern from a public health point of view, even for a genotoxic carcinogen (*39*).

It is worth noting again that there are some limitations to this risk assessment. First, it used a conservative estimate of Canadians' PFA dietary exposure since selected food items that represent only a portion of the average Canadian diet were analyzed. In addition, dietary habits for infants and children can result in substantially different exposures as compared to adults; thus, separate exposure evaluations and analysis of a wider variety of composites are required for these age categories. The factors contributing to these limitations will be addressed in future studies.

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