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Cooking Decreases Observed Perfluorinated Compound Concentrations in Fish

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Dietary intake is a major route of exposure to perfluorinated compounds (PFCs). Although fish and seafood contribute significantly to total dietary exposure to these compounds, there is uncertainty with respect to the effect of cooking on PFC concentrations in these foods. Eighteen fish species purchased from markets in Toronto, Mississauga, and Ottawa, Canada were analyzed for perfluorooctanesulfonamide (PFOSAs)-based fluorochemicals and perfluorinated acids (PFAs) in raw and cooked (baked, boiled, fried) samples. Of 17 analytes, perfluorooctanesulfonic acid (PFOS) was detected most frequently; concentrations ranged from 0.21 to 1.68 ng/g ww in raw and cooked samples. PFOSAs were detected only in scallops at concentrations ranging from 0.20 ng/g ww to 0.76 ng/g ww. Total concentrations of PFAs in samples were 0.21 to 9.20 ng/g ww, respectively, consistent with previous studies. All cooking methods reduced PFA concentrations. Baking appeared to be the most effective cooking method; after baking samples for 15 min at 163 C (325 °F), PFAs were not detected in any of the samples. The margin of exposures (MOE) between the toxicological points of reference and the dietary intake of perfluorocarboxylates (PFCAs) and PFOS in fish and seafood muscle tissue were greater than 4 orders of magnitude. This indicates that reducing consumption of fish muscle tissue is not warranted on the basis of PFC exposure concerns at the reported levels of contamination, even for high fish consuming populations.

KEYWORDS: Cooking fish; fish contaminants; PFOS; PFOA; PFOSAs; perfluorinated compounds

INTRODUCTION

Perfluorinated compounds (PFCs) such as perfluorooctyl sulfonate (PFOS) and perfluorooctanoate (PFOA) have numerous industrial applications as surfactants, surface protectants (food packaging, textiles, carpets, upholstery), and lubricants (*1, 2*). After 50 years of production, 3M, the major PFOS manufacturer, phased out this compound and related fluorochemicals due to concerns of persistence, toxicity, bioaccumulation, and global distribution (*3*). By the end of 2006, DuPont, the major PFOA manufacturer, achieved a voluntary total reduction in PFOA manufacturing emissions of approximately 95% (*4*). Despite cessations and reductions in PFC use, temporal trends show the concentrations of some perfluorinated carboxylates (PFCAs) such as perfluorononanoate (PFNA) in Arctic ringed seals have increased or remained stable in recent years (*5, 6*).

Persistence and bioaccumulation of PFCs can be explained by their resistance to hydrolysis, photolysis, biodegradation and metabolism (*7*). Fluorine, possessing the greatest electronegativity (4.0), can induce electron withdrawal more readily than any of the halogens, forming a strong carbonfluorine bond (110 kcal/mol). The C−F bond confers thermal and chemical stability to PFCs. Longer carbon chained perfluorocarboxylates (PFCAs) and perfluorosulfonate moieties are more bioaccumulative and toxic than those with shorter carbon chains (*8, 9*) and in most aquatic food webs, the overall PFCA profile is dominated by long-chained PFCAs (*10*).

As diet is an important PFC exposure route for humans, focused monitoring studies of PFCs in foods, including fish and seafood, have emerged recently (*11, 12*). PFC concentrations have been measured in raw seafood samples (*13*), yet the effect of preparation and cooking methods may significantly modify PFC concentrations in seafood and consequently total intake, as has been observed for other contaminants such as polychlo-

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rinated biphenyls (*14*). Comparisons of PFC concentrations between raw and cooked seafood using preparation methods employed by high fish consuming populations have yet to be conducted. Moreover, since PFCs accumulate preferentially in liver tissue, biomonitoring studies tend to report levels in liver rather than muscle tissue (*15, 16*). Liver sampled in biomonitoring studies is, however, rarely relevant to human dietary exposure assessments. Therefore, we focused on concentrations of PFCs in muscle tissue.

It has been argued that exposure to PFCs via fish muscle tissue may be limited and that analytical methods are not adequately characterized for the analysis of muscle tissue as they are for liver tissue (*17*). However, fish muscle is the predominant tissue consumed in large quantities by high-consuming populations, and human exposure to PFCs via consumption of fish muscle tissue has yet to be estimated. Previous analysis of PFCs in biota typically involved limited cleanup for removing lipids during sample extraction (*10*), increasing the potential for matrix interference during analysis. We used a well-characterized extraction method, in addition to a large number of mass-labeled internal standards to account for matrix effects during analysis.

The purpose of this study is to determine the concentrations of PFCs in raw and cooked fillets (muscle) of fish species. We focused on those species consumed by subpopulations of Canadians that frequently consume fish, in addition to those that have been infrequently sampled in Health Canada surveillance activities, such as the Total Diet Study (TDS).

MATERIALS AND METHODS

Rationale for Species Selection. The TDS is a targeted, market basket survey designed to monitor contaminant concentrations in a variety of foods in order to estimate the average dietary exposure of Canadians to a variety of contaminants and nutrients (*18*). Approximately 200 food items are sampled annually and prepared as composite food samples. One limitation of the TDS is that data reported from the analysis of the composites cannot be dissociated into major contributing food items. In the case of seafood, contaminant concentrations for individual species cannot be disaggregated. In addition, the selection of food items for inclusion in the TDS based on consumption by the average Canadian may preclude selection of unique fish species from newly immigrated populations. These fish species may not be included in TDS sampling if survey respondents do not speak a heritage language. Consequently, contaminant concentrations in fish species consumed by subpopulations are not captured by the TDS.

There is abundant evidence that shows individuals of certain subpopulations, for example North Americans of Asian origin, consume fish much more frequently than those of non-Asian origins. Southeast Asian immigrants consume 3−5 times more seafood than the general American population (*19*). The mean fish consumption rate of 10 Asian and Pacific Islanders groups exceeds that of the mean Canadian consumption rate (eaters only) 5-fold [117.2 g/day (*20*); 22 g/day (*21*)]. In Southern Ontario, Asian-Canadians consumed more total fish meals annually (medians = 213.0 females, 223.0 males) than Euro-Canadians (medians = 131.0 females, 137.5 males) (*22*). Fish intake is higher among Canadian children of Chinese and southeast Asian origin (2.1 servings/wk) compared to children from First Nations (1.0 servings/wk), Hispanic (1.3 servings/wk), South Asian (1.2 servings/wk) and other ethnic origins (1.3 servings/wk) (*23*). Thus, species consumed by Canadians of southeast Asian origin and several markets catering to Asian-Canadian communities were selected for sampling.

Sampling of Fish. Sampled seafood items were selected on the basis of annual import mass (kg) to Canada, which acts as a proxy for consumption frequency. Import data were provided for items collected between 2001−2004 by the Fish, Seafood and Production Division of the Canadian Food Inspection Agency. The quantity of each seafood item (kg) was corrected for waste percentage (i.e., of inedible parts) in order to more accurately rank fish and seafood consumed versus purchased. Items were then ranked in decreasing order according to import quantity. Species regularly sampled in the TDS were removed. After this revision, highest ranked items available for purchase during February−April 2006 were selected. Ten Asian-Canadian supermarkets and fish markets in downtown Toronto, Mississauga, and Ottawa, Canada supplied the species. Eighteen fish and shellfish species were sampled: silver pomfret, milkfish, cuttlefish, sea squirt, grouper, red snapper, catfish, monkfish, cherrystone clams, conch, scallops, mackerel, yellow croaker, gray mullet, whiting, skate, octopus, squid.

Table 1. Recipes Used to Cook Finfish and Non-Finfish

	ratio liquid/fish (v/v)	type of liquid used in cooking method	preparation	time/temperature
		Finfish		
baked	1:40	rice wine	marinate fillet in wine	15 min/163 °C
			bake in oven	325 °F
boiled	30:1	water	boil until firm	n/a
fried	1:16	sesame oil	cut into 1 in. cubes	15 min/163 °C
			fry in wok	325 °F
		Non-Finfish		
baked	n/a	vegetable oil	place in pan coated in oil	15 min/163 °C
			bake in oven	(325 °F) or until tender
boiled	35:1	water	boil until firm	n/a

Postpurchase, samples were immediately packed in ice and shipped to Kemptville Campus, University of Guelph, Kemptville, Canada, for preparation and cooking. Detailed systematic protocols for preparation and cooking were devised; a summarized method is described here.

Sample Preparation and Cooking. Fish organs, heads, scales and bones were removed using clean, stainless steel utensils on non-PTFE cutting boards and discarded according to previously published methods (*24*). After filleting, the knife and cutting board were cleaned between each fish species. Instances of fillet tissue contamination by organ rupture during or before preparation were recorded. The fillet or edible tissue of all individuals was rinsed in distilled water and blotted dry. Skin is not often included in fish nutrient or contaminant analyses (*14, 25, 26*), yet high fish consuming populations eat finfish with skin more often than without (*20*). Thus, skin (when applicable) and edible flesh were retained for each seafood item. Cherrystone clams were soaked 4−5 h in a salt brine solution to remove sand and scrubbed with a nail-brush to remove embedded dirt. A screw-driver and hammer was used to open shells; a sharp knife was then used to sever the hinge muscles. Shells were discarded. Conch were frozen to ease removal. Conch eyes, mouth, foot, snout, nail, feet, and intestinal sac were discarded. Teflon, Viton, aluminum foil, and gloves with talcum powder were avoided during the entire preparation to avoid sample contamination (*24*).

Individuals of each species were combined to create a composite sample. A minimum of three individuals from three different sources or markets ($n = 9$) for each species were combined into composite samples. The composite samples for finfish (silver pomfret, milkfish, grouper, red snapper, catfish, monkfish, mackerel, yellow croaker, gray mullet, whiting) were each divided into four equal portions by mass. The composite samples for nonfinfish (cuttlefish, octopus, sea squirt, skate, squid, conch, and cherrystone clam, scallops) were each divided into three equal portions by mass. Individuals were divided and apportioned among the divisions to avoid bias due to interindividual variability in contaminant concentrations. For each species, one of these portions was left raw and allocated to a precleaned polypropylene wide mouthed bottle. As described in **Table 1**, the three remaining portions of each finfish species were baked, boiled or fried; the two remaining portions for nonfinfish species were baked or boiled. The recipes were devised from online recipes and suggestions from Toronto Public Health dietitians. Post-cooking, each portion was homogenized in a Cuisinart food processor, distributed into precleaned polypropylene wide mouthed bottles, and shipped to Health Canada for PFC analysis.

Table 2. List of Analytes, Their Acronyms, and Associated Instrument Performance Internal Standards for PFCs

analyte	acronym	instrument performance internal standard
Analyzed by GC-MS		
N-ethyl perfluorooctanesulfonamide	N-EtPFOSA	N-Me-d_3-PFOSA
perfluorooctanesulfonamide	PFOSA	N-Me-d_3-PFOSA
N,N-diethyl perfluorooctanesulfonamide	N,N-Et_2PFOSA	N-Me-d_3-PFOSA
N-methyl perfluorooctanesulfonamide	N-MePFOSA	N-Me-d_3-PFOSA
N,N-dimethyl perfluorooctanesulfoamide	N,N-Me_2PFOSA	N-Me-d_3-PFOSA
N-ethyl-d_5 perfluorooctanesulfonamide	N-Et-d_5-PFOSA[a]	N-Me-d_3-PFOSA
N-methyl-d_3- perfluorooctanesulfonamide	N-Me-d_3-PFOSA	
	Analyzed by LC-MS/MS	
perfluoroheptanoate	PFHpA	1,2,3,4-^{13}C PFOA
perfluorooctanoate	PFOA	1,2,3,4-^{13}C PFOA
perfluorononanoate	PFNA	1,2,3,4,5-^{13}C PFNA
perfluorodecanoate	PFDA	1,2-^{13}C PFDA
perfluoroundecanoate	PFUA	1,2-^{13}C PFDA
perfluorododecanoate	PFDoDA	1,2-^{13}C PFDoDA
perfluorotetradecanoate	PFTeDA	1,2-^{13}C PFDoDA
linear perfluorohexane sulfonate	PFHxS	1,2-^{18}O PFOS
linear perfluorooctane sulfonate	PFOS	1,2-^{18}O PFOS
linear perfluorodecane sulfonate	PFDS	1,2-^{18}O PFOS
1,2-^{13}C perfluorooctanoate	1,2-^{13}C PFOA[a]	1,2,3,4-^{13}C PFOA
1,2,3,4-^{13}C perfluorooctane sulfonate	1,2,3,4-^{13}C PFOS[a]	1,2-^{18}O PFOS
1,2,3,4-^{13}C perfluorooctanoate	1,2,3,4-^{13}C PFOA	
1,2,3,4,5-^{13}C perfluorononanoate	1,2,3,4,5-^{13}C PFNA	
1,2-^{13}C perfluorodecanoate	1,2-^{13}C PFDA	
1,2-^{13}C perfluorododecanoate	1,2-^{13}C PFDoDA	
1,2-^{18o} perfluorooctane sulfonate	1,2-^{18}O PFOS	

[a] Denotes compound was used as a recovery internal standard.

ANALYTICAL METHODS

Chemicals for Perfluorooctanesulfonamide-Related Compound (PFOSAs) Analysis. N-Ethyl perfluorooctanesulfonamide (96%) was purchased from Interchim (France). Perfluorooctanesulfonamide (>95%) was provided by Griffin LLC (Valdosta, GA, USA). N,N-Diethyl perfluorooctanesulfonamide was synthesized. N-Methyl perfluorooctanesulfonamide (>98%), N,N-dimethyl perfluorooctanesulfoamide (>98%), N-ethyl-d_5-perfluorooctanesulfonamide (>98% chemical and ≥98% isotopic purity), and N-methyl-d_3-perfluorooctanesulfonamide (>98% chemical and >98% isotopic purity) were obtained from Wellington Laboratories (Guelph, ON, Canada). Acronyms for all analytes are listed in **Table 2**.

Chemicals and Materials for Analysis of Perfluorocarboxylates and Sulfonates (PFAs). The following perfluorinated compounds (purity >95%) were obtained from Aldrich (Oakville, ON, Canada) unless otherwise indicated, and were used as standards for perfluorocarboxylates and perfluorosulfonates: perfluoroheptanoic acid, perfluoro-n-octanoic acid (Wellington), perfluorononanoic acid, perfluorodecanoic acid, perfluoroundecanoic acid, perfluorododecanoic acid, perfluorotetradecanoic acid, sodium perfluorohexanesulfonate (Wellington), sodium perfluorooctanesulfonate (Wellington), and sodium perfluorodecanesulfonate (Wellington). Mass-labeled 1,2-^{13}C perfluorooctanoic acid (Perkin-Elmer, Boston, MA, USA), sodium 1,2,3,4-^{13}C perfluorooctane sulfonate (Wellington), 1,2,3,4-^{13}C perfluoro-n-octanoic acid (Wellington), 1,2,3,4,5-^{13}C perfluoro-n-nonanoic acid (Wellington), 1,2-^{13}C perfluorodecanoic acid (Wellington), 1,2-^{13}C perfluoro-n-dodecanoic acid (Wellington), and ammonium 1,2-^{18}O perfluorooctane sulfonate (RTI International, Research Triangle Park, NC, USA) were used as internal standards (**Table 2**).

All water used during PFA analyses was Milli-Q purified (Millipore, Billerica, MA, USA) 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.

Analysis of PFOSAs. Samples were extracted and analyzed for PFOSAs according to previous methods (*27*). Briefly, samples were extracted with solvent [2:1 (v/v) hexane/acetone], lipids were removed by washing with concentrated sulfuric acid, and extracts were cleaned using silica gel column chromatography. A sample containing Milli-Q purified water was concurrently run through the method as a blank with each set of samples analyzed to monitor laboratory sources of the PFOSAs.

Samples were analyzed by gas chromatography-positive chemical ionization-mass spectrometry (GC-PCI-MS). The GC was fitted with a retention gap (1 m × 0.53 mm i.d., deactivated fused silica) and a DB-5MS (30 m × 0.25 mm i.d., 0.25 μm film thickness; Agilent) column. Samples were injected (2.0 μL) by an Agilent 7683 Automatic Liquid Sampler using cool on-column injection. The selected ion monitoring mode was used to monitor the quasimolecular ion $[M+H]^+$ of all PFOSAs.

Quantitation was performed using the quasimolecular ion $[M+H]^+$ as the target ion. Analyte areas were normalized to areas of the appropriate instrument performance internal standard prior to quantitation using a calibration curve constructed from four external standards prepared in isooctane.

Analysis of PFAs. Samples were analyzed for PFAs following previous methods (*12*), with minor changes to the compounds used as internal standards. Thawed samples were extracted with MeOH, and reduced in volume for analysis. Blanks of MeOH were processed concurrently with each set of samples. Tilapia previously analyzed and known to be blank was also fortified at 25 ng/g with all native PFA analytes and analyzed with each set of samples. An instrument performance standard solution was added to each sample prior to instrumental analysis.

Analyses were performed using liquid chromatography negative electrospray tandem mass spectrometry (LC-MS/MS). Samples were analyzed for PFAs in the multiple reaction monitoring mode. Peak areas were integrated using QuanLynx software (version 4.0) provided as part of the LC-MS/MS system. Relative response factors were calculated as the ratio of analyte quantitation transition peak area to corresponding instrument performance internal standard peak area. Concentrations of analytes were determined using an external calibration curve.

RESULTS

Analysis of PFAs and PFOSAs. Recoveries of PFAs from the fortified tilapia averaged 80 ± 25%. Average recoveries of individual PFAs did not differ significantly from each other, or from the two recovery internal standards ($p = 0.483$, Kruskal−Wallis

Table 3. Concentrations (ng/g ww) of PFCAs and PFOS in Raw and Cooked Seafood Samples Containing PFAs above Detection Limits

sample	prep	PFOA	PFNA	PFDA	PFUA	PFDoDA	PFTeDA	PFOS	ΣPFAs
catfish	raw	nd	nd	nd	nd	nd	nd	1.57	1.57
	fried	nd	nd	nd	nd	nd	nd	0.90	0.90
cuttlefish	raw	nd	1.44	nd	nd	nd	nd	nd	1.44
grey mullet	fried	nd	nd	nd	nd	nd	nd	1.14	1.14
grouper	raw	1.36	nd	nd	nd	nd	nd	nd	1.36
	fried	nd	nd	nd	nd	nd	nd	0.47	0.47
monkfish	raw	nd	1.34	nd	nd	nd	nd	1.34	2.69
	boiled	0.06	nd	nd	0.39	nd	nd	0.22	0.67
octopus	raw	0.78	1.29	1.55	1.88	nd	2.61	nd	9.20
	boiled	0.06	nd	nd	1.59	nd	nd	0.23	1.88
red snapper	raw	nd	nd	nd	nd	nd	nd	1.46	1.46
	boiled	nd	nd	nd	nd	nd	nd	0.21	0.21
	fried	nd	nd	nd	nd	nd	nd	0.78	0.78
sea squirt	raw	1.58	1.32	nd	nd	nd	nd	nd	2.89
	boiled	1.59	0.96	nd	nd	nd	nd	nd	2.65
skate	raw	nd	1.09	nd	1.55	1.33	0.67	1.51	6.14
	boiled	nd	nd	nd	1.35	nd	nd	0.88	2.24
whiting	raw	nd	1.46	nd	nd	nd	1.44	nd	2.91
yellow croaker	raw	nd	nd	nd	1.57	nd	nd	1.68	3.26
	boiled	nd	nd	nd	nd	nd	nd	0.89	0.89
	fried	nd	nd	nd	2.11	nd	nd	0.68	2.80

one-way analysis of variance on ranks). Average ± standard deviation percent recoveries of recovery internal standards added to samples were 80 ± 16% and 88 ± 13% for 1,2-^{13}C-PFOA and 1,2,3,4-^{13}C- PFOS, respectively. PFAs were not detected in any of the method blanks. Sample-specific LODs for individual PFAs ranged from 0.03−10 ng/g tissue ww. Limits of quantitation (LOQs) were set at three times the LODs for each compound, following ref *12*.

Average ± standard deviation percent recoveries of the recovery internal standard added to samples was 63 ± 12% for N-Et-d_5-PFOSA. N-EtPFOSA and N-MePFOSA were both detected in the method blanks. Blank concentrations were approximately 0.03 pg/g based on a theoretical 10 g sample. Due to the presence of N-EtPFOSA and N-MePFOSA in the blanks, limits of detection were calculated as previously described (*11*). Limits of detection (LOD) for individual PFOSAs ranged from 0.006−0.2 ng/g tissue ww. Concentrations presented are blank and recovery corrected.

PFA and PFOSA Concentrations and Contaminant Profiles in Raw Samples. PFAs were detected in 11 of 18 species analyzed; PFOSAs were detected solely in scallops. PFAs were detected most frequently in octopus (8/30 samples), skate (7/30 samples), scallops (3/21 samples), monkfish and yellow croaker (5/40 samples each).

PFOS was the most widely detected perfluorinated compound (24% of samples), consistent with other surveys of aquatic organisms (*5, 17, 28, 29*). The measured concentrations of PFAs are on the order of a few nanograms per gram (ppb) (**Table 3**), similar to other reports of muscle and soft tissue in fish and shellfish from Europe, Asia and America (**Table 4**), but orders of magnitude lower than those reported in areas of high contamination. After the release of fire-fighting foam into Etobicoke Creek, concentrations in fish reached 72.9 μg/g (*30*).

PFNA and PFUA were the main PFCA compounds detected in approximately 11% of samples, with concentrations ranging from 0.39 to 2.11 ng/g ww. PFNA, previously reported in mammals (*10, 28, 31*) and porpoises (*29, 32*) is among the most frequently detected PFCA in wildlife samples (*6*). Odd carbon numbered PFCAs were detected more frequently than even-length PFCAs, which is consistent with results in mammals (*10*). In addition, PFCA concentrations generally decreased with increasing chain length, a trend noted in invertebrates and reptiles (*33*). PFOA was present in 10% of samples, with concentrations ranging from 0.06 to 1.78 ng/g ww, values similar to those previously reported (**Table 4**).

Concentrations of total PFAs were highest in raw samples from carnivorous species: octopus (9.20 ng/g ww), skate (6.14 ng/g ww), yellow croaker (3.26 ng/g ww), whiting (2.91 ng/g ww) and monkfish (2.69 ng/g ww). Planktivorous species (scallop, silver pomfret), low trophic level fish (milkfish) and filter feeders (cherrystone clam, conch) did not have detectable concentrations of PFAs, with the exception of sea squirt (2.89 ng/g ww). Ratios of PFOS/PFOA exceeded 1 for most fish species in this study. PFOA dominated in grouper (1.36 ng/g ww) octopus (1.78 ng/g ww) and sea squirt (1.58 ng/g ww) while PFOS was not detected in raw samples of these species.

Effect of Cooking on PFC Concentrations and Comparison to Other Contaminants. Three positive detections of PFOSAs occurred in baked scallop (NMeFOSA: 75.7 pg/g ww, NEtFOSA: 33.6 pg/g ww) and boiled scallop (NMeFOSA: 19.6 pg/g ww). PFHS and PFDS were not detected.

Whereas all cooking methods reduced PFA concentrations, baking reduced PFA residues below the LOD in all species. Boiling reduced PFA concentrations by 79% on average; frying yielded a 54% average reduction. Boiling and frying eliminated PFA concentrations in whiting and boiling removed PFA in cuttlefish. Baking reduced sample mass by an average of 80%, broiling reduced mass by 82%, frying by 85% (data not shown). Mass loss due to cooking fish was much higher than previous reports (*14, 34*), but lower temperatures and shorter cooking times, respectively, were used in the latter two studies. There was no correlation between % mass loss due to cooking and % contaminant change for PFAs.

The decline in PFA concentrations from cooking was compared to the concentration change of other contaminants in fish due to cooking from previous studies (*14, 34–37*). The decline in PFA concentrations (54−100%) was greater than that for organochlorine and organobromine contaminants (11−44%).

Estimation of PFA Exposure. PFA exposure from diet was estimated for high and average consumption scenarios for Canadian adults <20 years. PFCA and PFOS exposures were estimated separately since these compounds differ in amount consumed and toxicity. Fish intake rate (g/day) was estimated

Table 4. Review of PFCAs and PFOS in Muscle Tissue of Selected Fish and Shellfish

species	*n*	location	year	tissue	range of contaminants	concentrations (ng/g ww)	reference
clam	n/a	Tidal flat, Ariake Sea, Japan	2003	soft tissue	PFOS	<0.3	Nakata et al., 2006
					PFOA	7.5	
					PFNA	<1.5	
					PFHA	<1.5	
					PFOSA	<1.5	
carp (*Cyprinus carpio*)	10	Saginaw Bay, Michigan	2001	muscle	PFOS	60−300	Giesy & Kannan, 2001
Chinook salmon (*Oncorhynchus ishawytscha*)	6	Michigan lakes	2001	muscle	PFOS	7−190	
lake whitefish (*Coregonus clupeaformis*)	5	Michigan lakes	2001	muscle	PFOS	97−170	
brown trout	10	Michigan lakes	2001	muscle	PFOS	<6−46	
yellow croaker	n/a	Guangzhou	2004	muscle	PFOS	2.93	Gulkowska et al., 2006
silver pomfret		Zhoushan	2004	muscle	PFOS	0.92	
		Guangzhou	2004	muscle	PFOS	0.67	
Japanese mackerel		Zhoushan			PFOS	0.38	
whitemouth croaker		Guangzhou	2004	muscle	PFOS	2.18	
					PFHpA	0.41	
cuttlefish		Zhoushan	2004	muscle	PFOS	0.86	
		Guangzhou			PFOS	0.87	
squid		Zhoushan	2004	soft tissue	PFOA	0.31	
		Guangzhou			PFOS	0.96	
Mantis shrimp		Zhoushan	2004	soft tissue	PFOA	0.31	
					PFOS	1.07	
					PFOA	0.43	
					PFOS	1.32	
		Guangzhou			PFHS	0.28	
					PFOA	0.35	
					PFOS	13.9	
					PFOA	0.45	
clams	5	Frobisher Bay, Eastern Arctic	2002	soft tissue	PFOS	0.28	Tomy et al., 2004
					PFOA	nd	
shrimp	5	Frobisher Bay, Eastern Arctic	2002	soft tissue	PFOS	0.35	
					PFOA	0.17	
plaice	4−7	Western Scheldt, Netherlands	2001	muscle	PFOS	<10−87	Hoff et al., 2003
bib	4−8				PFOS	<10−111	
haddock, cod, sole (composite)	n/a	Winnipeg (market)	2004	muscle	PFOA	<0.5	Tittlemier et al., 2007
					PFNA	<1	
trout, pickerel (composite)		Winnipeg (market)	2004	muscle	PFOS	2.6	
					PFOA	<0.5	
smelt, perch (composite)		Whitehorse (market)	1998	muscle	PFNA	<1	
					PFOS	2.0	
					PFOA	<0.5	
					PFNA	<1	
					PFOS	1.5	
carp	10	Saginaw Bay, Michigan	2005	muscle	PFOS	124	Kannan et al., 2005
					PFOSA	<19	
small mouth bass	n/a	Raisin River	1999	muscle	PFOA	<36	
					PFHS	<34	
		St. Clair River	1998	muscle	PFOS	17.8	
					PFOSA	<1	
		Calumet River	1998	muscle	PFOA	<2	
					PFHS	<1	
					PFOS	2.7	
					PFOSA	1.1	
					PFOA	<2	
					PFHS	<1	
					PFOS	2.6	
					PFOSA	<1	
					PFOA	<2	
					PFHS	<1	

from consumption frequency (meals/year) multiplied by portion size (g/meal). Consumption frequency data for adults of Asian and European origin consuming higher than average amounts of fish was obtained (*38*); ref *21* supplied data on average Canadian fish consumption. Portion size was estimated for high fish consumers (*39*). Dietary exposure to PFCAs (or PFOS) from fish was calculated as PFCA (or PFOS) concentration in ng/g averaged across all species and preparation methods (raw, baked, boiled, fried) multiplied by the fish intake rate based on refs *21* and *38*.

Total PFCA exposure from fish (ng/day) was estimated from this study as 87 ng/day for a high fish consumer of Asian origin,

Table 5. Assessment of PFCA and PFOS Dietary Exposure for Canadian Adults (>20 years of age).

population	Asian-Canadian high fish consumers		Euro-Canadian high fish consumers		average Canadian consumers (fish eaters only)		reference
consumption frequency (# meal/year)	325		174				Cole et al., 2004[a]
portion size (g/meal)	150		150				Bureau of Chemical Safety, 2004
fish intake rate (g/day)	134		72		22		calculated from above; Market Facts of Canada, 1991
analytes	ΣPFCAs	ΣPFOS	ΣPFCAs	ΣPFOS	ΣPFCAs	ΣPFOS	
total analyte exposure from fish (ng/day)[b]	87	14	47	7	14	2	this study
total analyte exposure from nonfish dietary sources, Canadians <12 years (ng/day)	140	90	140	90	140	90	Tittlemier et al., 2007
total analyte exposure from diet (ng/day)	227	104	187	97	154	92	calculated from above
MOE[c]	1.6×10^5	1.8×10^5	2.0×10^5	1.9×10^5	2.4×10^5	2.0×10^5	

[a] Community-based samples, Great Lakes areas of concern high-fish consumers. [b] Assumes each fish/preparation method is equally consumed. [c] MOE: toxicological point of reference (see text)/predicted exposure.

47 ng/day for a high fish consumer of European origin, and 14 ng/day for the average Canadian (fish eaters only). The latter value is identical to previous estimates (*12*). Total PFOS exposure from fish (ng/day) was estimated as 14 ng/day for a high fish consumer of Asian origin, 7 ng/day for a high fish consumer of European origin, and 2 ng/day for the average Canadian. This measure of dietary intake of PFCAs (or PFOS) from fish was added to nonfish dietary exposure of these compounds previously estimated (*12*). Thus, total PFCA exposure from diet is 227 ng/day for high fish consumers of Asian origin, 187 ng/day for high fish consumers of European origin, and 154 ng/day for the average Canadian (**Table 5**) of which 38%, 25%, and 9% are due to fish and seafood consumption, respectively. Total PFOS exposure from diet is 104 ng/day for high fish consumers of Asian origin, 97 ng/day for high fish consumers of European origin, and 92 ng/day for the average Canadian (**Table 5**) of which 13%, 7%, and 2% are due to fish and seafood consumption, respectively.

DISCUSSION

PFC Concentrations of Fish Muscle. Factors such as high volume production (pre-2002), high biomagnification potential, and persistence help to explain high relative amounts of PFOS in aquatic organisms and its prevalence as the dominant PFC observed in biota in this and other studies. PFOS is regarded as a degradation end-product of other PFCs such as PFOSA, N-EtFOSE, and N-EtFOSA (*40, 41*) and possibly NMeFOSE (*3*).

The reason for the detection of PFOS in fried grouper and boiled octopus, but not in raw samples of the respective species, is not readily apparent. PFOS was not detected in cooking oil prefrying or in blanks. Generation of PFOSAs via cooking, or heat-facilitated conversion of precursors to PFOS in fish, has not been reported previously. The presence of PFOSAs in scallop may reflect the inability of lower trophic species to convert PFOSAs to PFOS. However, the cause for detection of PFOSAs in baked and boiled scallops, but not in raw scallops, is uncertain. It is possible that high mass loss in fried grouper (87%), boiled octopus (87%) and baked and boiled scallops (83% each) may have increased analyte concentrations above the LODs.

The highest concentrations of total PFAs in octopus, skate, yellow croaker, whiting, and monkfish may be explained by their status as carnivores. Differential accumulation and retention abilities is an alternative explanation (*42*).

An odd−even carbon length pattern of PFCAs has been described in Arctic biota, whereby those carboxylates with an odd number of carbons were more prevalent than their even length counterparts with one fewer carbons, for example, PFNA > PFOA, PFUnA > PFDA (*10*). The pattern, reproduced in this study, suggests fluorotelomer alcohols as a possible source of the observed PFCAs (*43*). Although fluorotelomer alcohols, which are raw materials used in the production of perfluorinated surfactants, are manufactured only in even carbon lengths, both even and odd chain length PFCAs are observed upon degradation (*10*).

Effect of Cooking on PFA Concentrations and Comparison to Other Contaminants. The mechanisms involved in the transfer and/or degradation of legacy POPs during the cooking process are not clear (*34*). Moisture is lost due to cooking (*37, 44*) while total lipids in fish may decrease (*34, 45*), increase (*45*; this study, data not shown), or remain constant (*46*). If total lipid mass increases via the addition of cooking oil, the mass of the hydrophobic contaminant could remain the same, but the concentration will decline (diluted by an increase in total fish mass from the added oil). If total lipid mass decreases, the hydrophobic contaminant could be removed in the lipid, as previously speculated (*34*).

Though concentrations of mercury increase in samples after cooking (*35, 37*), both studies report that the actual mercury mass is not changing. MeHg (the form of mercury in which nearly 100% of total mercury is found in fish) binds strongly to sulfhydryl groups of cysteine-rich proteins and is thereby retained in muscle. The sulfhydryl bond is not destroyed by cooking, and thus mercury is retained in fish muscle post cooking (*47*). Thus, as moisture and/or lipid is lost through cooking, the corresponding concentration of MeHg increases as a function of mass loss.

By contrast, the lack of correlation between total mass loss and total PFA concentration changes may indicate that these contaminants are not removed with the water or lipid of fish during cooking. PFOS and the structurally analogous PFAs aggregate hydrophobically with serum albumin (*48, 49*). If aggregation between those PFAs that are major contributors to total PFA concentrations and serum albumins remains intact during cooking, an increase in PFA concentration as a function of mass loss would be expected. Instead, no relationship was found and the very high losses of PFAs from cooking suggest that aggregation to proteins such as albumin may be disrupted, releasing the PFA compound.

To determine potential loss pathways (volatilization, loss in cooking residues) for PFCAs, the vapor pressures of PFOA, PFNA, PFDA, PFUS and PFDoDA were plotted against % concentration loss from baking and boiling. There was no relationship between PFCA vapor pressure and % concentration loss from baking, indicating that volatilization is not the loss

pathway for PFCAs. Indeed, the very low vapor pressures of PFCAs (8.29−63.98 kPa at 172.02−177.8 °C) alone suggest their ability to partition into the gas phase is very limited (*50*).

There was no relationship between PFCA vapor pressures and % concentration loss from boiling, which is consistent with chemical loss to liquid. To confirm this hypothesis, the remaining PFA content would have to be detected in cooking residues.

Baking may be more effective at reducing concentrations than boiling due to higher heat, resulting in increased mass loss of the fillets. Fish were baked at 163 C (325 °F) and boiled at 100 C (212 °F). Although fish were baked and fried at the same temperature, total mass loss was substantially higher for frying than baking, thereby increasing PFA concentrations in fried samples relative to baked samples, and reducing % concentration loss of PFAs.

Dietary Exposure Assessment to PFCAs and PFOS. Estimated intake of PFCAs and PFOS from dietary exposure was compared to toxicological points of reference as previously described (*12*). Due to the paucity of toxicological data for longer chain PFCAs, the $BMDL_{10}$ (lower 95% confidence limit of a benchmark dose for a 10% response level) for PFOA of 0.6 mg/kg body weight/day (*51*) based on the end point of increased liver weights in rats, was used as the toxicological reference point for PFCAs. For PFOS, a conservative LOEL (lowest observable effect level) of 0.03 mg/kg body weight/day was used as a toxicological reference point based on evidence of increased thymic atrophy in female and altered blood markers in male cynomolgus monkeys (*52*). The $BDML_{10}$ and LOEL were compared to three exposure scenarios: that of the average Canadian's dietary exposure to PFCAs from food, and for that of high fish consumers of Asian and European origin. Margins of exposure (MOE) were then computed. MOE is defined as the ratio between a defined toxicological point of departure or reference and a predicted exposure under a given scenario. The MOEs for PFCAs were 2.4×10^5 for the average Canadian's dietary exposure scenario, 1.6×10^5 for Asian Canadian high fish consumers, and 2.0×10^5 for European Canadian high fish consumers. The MOEs for PFOS were 2.0×10^5 for the average Canadian's dietary exposure scenario, 1.8×10^5 for Asian Canadian high fish consumers, and 1.9×10^5 for European Canadian high fish consumers. The European Food Safety Authority (*53*) has recommended that an MOE of 1.0×10^4 or greater that is based on a $BMDL_{10}$ from an animal study is of low concern from a public health point of view.

Limitations to this exposure assessment are similar to those discussed previously (*12*). Notably, the nonfish food items included in this estimate represent only a portion of the average Canadian diet (*12*). One limitation not thoroughly discussed in ref *12* is the use of $BMDL_{10}$ for PFOA as the toxicological reference for all PFCAs. PFOA is a lower numbered carbon chained PFCA (C = 8); PFCAs with shorter carbon chains are more quickly excreted in urine and accumulate to lower concentrations in liver and serum than longer chained PFCAs (*8*). As the $BMDL_{10}$ for PFOA was applied for all PFCAs, MOEs may be overestimated, particularly if longer-chained PFCAs are more toxic than PFOA.

Further, as PFCs are relatively stable compounds and this study did not measure chemical degradation of PFCs by cooking or loss to cooking residues, it is possible that cooking rendered the compounds more difficult to extract by the analytical method. If the latter hypothesis is correct, observed decreases in PFC concentrations from cooking may not be correlated to reduced toxicity.

As the risk incurred under the high fish consumption scenarios is well below the toxicological reference points, a recommendation for high fish consumers to reduce consumption of fish muscle tissue is not warranted at this time on the basis of PFC exposure concerns, especially as exposure to PFCs from fish is reduced below detection limits by baking fish 15 min at 163 C (325 °F). Decisions regarding fish consumption should not be solely based on PFC exposure concerns, but rather the benefits of consumption (i.e., omega 3 fatty acids) and risks from other contaminants should be considered. Future studies should target issues of PFC extractability, chemical degradation, and loss to cooking residues to elucidate the mechanism behind the observed concentration declines.

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LITERATURE CITED

(1) Midasch, O.; Schettgen, T; Angerer, J. Pilot study on the perfluorooctanesulfonate and perfluorooctanoate exposure of the German general population. *Int. J. Hygiene Environ. Health* **2006**, *209*, 489–496.

(2) Kissa, E. A miniature closed vessel technique for testing textile chemicals. *AATCC Rev.* **2001**, *1*, 27–28.

(3) D'Eon, J. C.; Hurley, M. D.; Wallington, T. J.; Mabury, S. A. Atmospheric chemistry of N-methyl perfluorobutane sulfonamidoethanol, $C_4F_9SO_2N(CH_3)CH_2CH_2OH$: Kinetics and mechanism of reaction with OH. *Environ. Sci. Technol.* **2006**, *40*, 1862–1868.

(4) Dupont Re: DuPont progress towards meeting the Environmental Protection Agency (EPA) 2010115 PFOA Stewardship Program, 2007. Online available: http://www.epa.gov/oppt/pfoa/pubs/DuPont_AppF.pdf. Last Accessed: Oct 2007.

(5) Bossi, R.; Riget, F.; Dietz, R. Temporal and spatial trends of perfluorinated compounds in ringed seal (*Phoca hispida*) from Greenland. *Environ. Sci. Technol.* **2005**, *39*, 7416–7422.

(6) Butt, C. M.; Muir, D. C.; Stirling, I.; Kwan, M.; Mabury, S. A. Rapid response of arctic ringed seals to changes in perfluoroalkyl production. *Environ. Sci. Technol.* **2007**, *41*, 42–49.

(7) Zhao, X. L.; Li, J. D.; Shi, Y. L.; Cai, Y. Q.; Mou, S. F.; Jiang, G. B. Determination of perfluorinated compounds in wastewater and river water samples by mixed hemimicelle-based solid-phase extraction before liquid chromatography-electrospray tandem mass spectrometry detection. *J. Chromatogr., A* **2007**, *1154*, 52–59.

(8) Kudo, N; Suzuki, E.; Katakura, M.; Ohmori, K; Noshiro, R.; Kawashima, Y. Comparison of the elimination between perfluorinated fatty acids with different carbon chain length in rats. *Chem.-Biol. Interact.* **2001**, *134*, 203–216.

(9) Goeke-Flora, C. M.; Reo, N. V. Influence of carbon chain length on the hepatic effects of perfluorinated fatty acids, A ^{19}F- and ^{31}P-NMR Investigation. *Chem. Res. Toxicol.* **1996**, *9*, 689–695.

(10) Martin, J. W.; Whittle, D. M.; Muir, D. C. G.; Mabury, S. A. Perfluoroalkyl contaminants in a food web from lake Ontario. *Environ. Sci. Technol.* **2004**, *38*, 5379–5385.

(11) Tittlemier, S. A.; Pepper, K.; Edwards, L. Concentrations of perfluorooctanesulfonamides in Canadian total diet study composite food samples collected between 1992 and 2004. *J. Agric. Food Chem.* **2006**, *54*, 8385–8389.

(12) Tittlemier, S. A.; Pepper, K.; Seymour, C.; Moisey, J.; Bronson, R.; Cao, X. L.; Dabeka, R. W. Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. *J. Agric. Food Chem.* **2007**, *55*, 3203–3210.

(13) Gulkowska, A.; Jiang, Q. T.; So, M. T.; Taniyasu, S.; Lam, P. K. S.; Yamashita, N. Persistent perfluorinated acids in seafood

collected from two cities of China. Environ. Sci. Technol. **2006**, *40*, 3736–3741.
(14) Zabik, M. E.; Zabik, M. J. Influence of processing on environmental contaminants in foods. Food Technol. **1996**, *50*, 225–229.
(15) Giesy, J. P.; Kannan, K. Global distribution of perfluorooctane sulfonate in wildlife. Environ. Sci. Technol. **2001**, *35*, 1339–1342.
(16) Kannan, K.; Perrotta, E.; Thomas, N. J. Association between perfluorinated compounds and pathological conditions in southern sea otters. Environ. Sci. Technol. **2006**, *40*, 4943–4948.
(17) Sinclair, E.; Mayack, D. T.; Roblee, K.; Yamashita, N.; Kannan, K. Occurrence of perfluoroalkyl surfactants in water, fish, and birds from New York State. Arch. Environ. Contam. Toxicol. **2006**, *50*, 398–410.
(18) Conacher, H. B. S.; Graham, R. A.; Newsome, W. H.; Graham, G. F.; Verdier, P. The Health Protection Branch Total Diet Program: An Overview. *Can. Inst. Food Sci. Technol. J.* **1989**, *22*, 322–326.
(19) Judd, N.; O'Neill, S. M.; Kalman, D. A. Are seafood PCB data sufficient to assess health risk for high seafood consumption groups. Hum. Ecol. Risk Assess. **2003**, *9*, 691–707.
(20) Sechena, R.; Liao, S.; Lorenzana, R.; Nakano, C.; Polissar, N.; Fenske, R. Asian American and Pacific Islander seafood consumption - a community-based study in King County, Washington. J. Exposure Anal. Environ. Epidemiol. **2003**, *13*, 256–266.
(21) Market Facts of Canada. Research Report: National Seafood Consumption Study. Conducted for Health and Welfare Canada, 1991, #C388/JdeB.
(22) Cole, D. C.; Sheeshka, J.; Murkin, E. J.; Kearney, J.; Scott, F.; Ferron, L. A.; Weber, J. P. Dietary intakes and plasma organochlorine contaminant levels among Great Lakes fish eaters. Arch. Environ. Health **2002**, *57*, 496–509.
(23) Innis, S. M.; Palaty, J.; Vaghri, Z.; Lockitch, G. Increased levels of mercury associated with high fish intakes among children from Vancouver, Canada. J. Pediatr. **2006**, *148*, 759–763.
(24) USEPA. Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories.Volume 1. 2000, Fish Sampling and Analysis. EPA 823-B-00−007.
(25) Sioen, I.; Van Camp, J.; Verdonck, F. A. M.; Van Thuyne, N.; Willems, J. L.; De Henauw, S. W. J. How to use secondary data on seafood contamination for probabilistic exposure assessment purposes? Main problems and potential solutions. Hum. Ecol. Risk Assess. **2007**, *13*, 632–657.
(26) Gokoglu, N.; Yerlikaya, P.; Cengiz, E. Effects of cooking methods on the proximate composition and mineral contents of rainbow trout (*Oncorhynchus mykiss*). Food Chem. **2004**, *81*, 19–22.
(27) Tittlemier, S. A.; Pepper, K.; Edwards, L.; Tomy, G. Development and characterization of a solvent extraction-gas chromatographic/mass spectrometric method for the analysis, of perfluorooctanesulfonamide compounds in solid matrices. J. Chromatogr., A **2005**, *1066*, 189–195.
(28) Kannan, K.; Yun, S. H.; Evans, T. J. Chlorinated, brominated, and perfluorinated contaminants in livers of polar bears from Alaska. Environ. Sci. Technol. **2005**, *39*, 9057–9063.
(29) Nakata, H. K.; Kannan Tetsuya, N.; Cho, H.; Sinclair, E. Perfluorinated contaminants in sediments and aquatic organisms collected from shallow water and tidal flat areas of the Ariake Sea, Japan: Environmental fate of perfluorooctane sulfonate in aquatic ecosystems. Environ. Sci. Technol. **2006**, *40*, 4916–4921.
(30) Moody, C. A.; Martin, J. W.; Kwan, W. C.; Muir, D. C. G.; Mabury, S. C. Monitoring perfluorinated surfactants in biota and surface water samples following an accidental release of fire-fighting foam into Etohicoke Creek. Environ. Sci. Technol. **2002**, *36*, 545–551.
(31) Smithwick, M.; Muir, D. C. G.; Mabury, S. A.; Solomon, K. R.; Martin, J. W.; Sonne, C.; Born, E. W.; Letcher, R. J.; Dietz, R. Perflouroalkyl contaminants in liver tissue from East Greenland polar bears (Ursus maritimus). Environ. Toxicol. Chem. **2005**, *24*, 981–986.
(32) Van de Vijver, K. I.; Hoslbeek, L.; Das, K.; Blust, R.; Joiris, C.; De Coen, W. Occurrence of perfluorooctane sulfonate and other perfluorinated alkylated substances in harbor porpoises from the Black Sea. Environ. Sci. Technol. **2007**, *41*, 315–320.
(33) Houde, M.; Martin, J. W.; Letcher, R. J.; Solomon, K. R.; Muir, D. C. G. Biological monitoring of polyfluoroalkyl substances: A review. Environ. Sci. Technol. **2006**, *40*, 3463–3473.
(34) Bayen, S.; Barlow, P.; Lee, H. K.; Obbard, J. P. Effect of cooking on the loss of persistent organic pollutants from salmon. J. Toxicol. Environ. Health **2005**, *68*, 253–265.
(35) Burger, J.; Dixon, C.; Boring, C. S.; Gochfeld, M. Effect of deep-frying fish on risk from mercury. J. Toxicol. Environ. Health **2003**, *66*, 817–828.
(36) Burger, J.; Gaines, K. F.; Boring, C. S. Effects of cooking on radiocesium in fish from the Savannah River: Exposure differences for the public. *Arch. Environ. Contam. Toxicol.* **2004**, *24*, 231–235.
(37) Morgan, J. N.; Berry, M. R.; Graves, R. L. Effects of commonly used cooking practices on total mercury concentration in fish and their impact on exposure assessment. J. Exposure Anal. Environ. Epidemiol. **1997**, *7*, 119–133.
(38) Cole, D. C.; Kearney, J.; Sanin, L. H.; Leblanc, A.; Weber, J. P. Blood mercury levels among Ontario anglers and sport-fish eaters. Environ. Res. **2004**, *95*, 305–314.
(39) Bureau of Chemical Safety, Health Canada. Fish Consumption: Review and Recommendation of Current Intake Figures for Canadian Consumers, 2004, http://www.hc-sc.gc.ca/fn-an/pubs/mercur/merc_fish_poisson_e.htmlappd#.
(40) Young, C. J.; Furdui, V. I.; Franklin, J.; Koerner, R. M.; Muir, D. C. G.; Mabury, S. A. Perfluorinated acids in arctic snow: New evidence for atmospheric formation. Environ. Sci. Technol. **2007**, *41*, 3455–3461.
(41) Martin, J. W.; Ellis, D. A.; Mabury, S. A.; Hurley, M. D.; Wallington, T. J. Atmospheric chemistry of perfluoroalkanesulfonamides: Kinetic and product studies of the OH radical and Cl atom initiated oxidation of N-ethyl perfluorobutanesulfonamide. Environ. Sci. Technol. **2006**, *40*, 864–872.
(42) Haukas, M.; Berger, U.; Hop, H.; Gulliksen, B.; Gabrielsen, G. W. Bioaccumulation of per- and polyfluorinated alkyl substances (PFAS) in selected species from the Barents Sea food web. Environ. Pollut. **2007**, *148*, 360–371.
(43) De Silva, A. O.; Mabury, S. A. Isomer distribution of perfluorocarboxylates in human blood: Potential correlation to source. Environ. Sci. Technol. **2006**, *40*, 2903–2909.
(44) Kalogeropoulos, N.; Andrikopoulos, N. K.; Hassapidou, M. Dietary evaluation of Mediterranean fish and molluscs panfried in virgin olive oil. J. Sci. Food Agric. **2004**, *84*, 1750–1758.
(45) Gladyshev, M. I.; Sushchik, N. N.; Gubanenko, G. A.; Demirchieva, S. M.; Kalachova, G. S. Effect of boiling and frying on the content of essential polyunsaturated fatty acids in muscle tissue of four fish species. Food Chem. **2007**, *101*, 1694–1700.
(46) Gladyshev, M. I.; Sushchik, N. N.; Gubanenko, G. A.; Demirchieva, S. M.; Kalachova, G. S. Effect of way of cooking on content of essential polyunsaturated fatty acids in muscle tissue of humpback salmon (*Oncorhynchus gorbuscha*). Food Chem. **2006**, *96*, 446–451.
(47) Chicourel, E. L.; Sakuma, A. M.; Zenebon, O. Inefficacy of cooking methods on mercury reduction from shark. Arch. Latinoam. Nutr. **2001**, *51*, 288–292.
(48) Jones, P. D.; Hu, W. Y.; De Coen, W.; Newsted, J. L.; Giesy, J. P. Binding of perfluorinated fatty acids to serum proteins. Environ. Toxicol. Chem. **2003**, *22*, 2639–2649.
(49) Han, X.; Snow, T. A.; Kemper, R. A.; Jepson, G. W. Binding of perfluorooctanoic acid to rat and human plasma proteins. Chem. Res. Toxicol. **2003**, *16*, 775–781.
(50) Kaiser, M. A.; Larsen, B. S.; Kao, C.-P. C.; Buck, R. C. Vapor pressures of perfluorooctanoic, -nonanoic, -decanoic, -undecanoic, and -dodecanoic acids. J. Chem. Eng. Data **2005**, *50*, 1841–1843.

(51) Butenhoff, J. L.; Kennedy, G. L.; Frame, S. R.; O'Connor, J. C.; York, R. G. The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat. *Toxicology* **2004**, *196*, 95–116.
(52) Seacat, A. M.; Thomford, P. J.; Hansen, K. J. Subchronic toxicity studies on perfluorooctanesulfonate potassium salt in cynomolgus monkeys. *Toxicol. Sci.* **2002**, *68*, 249–264.
(53) European Food Safety Authority, 2005, EFSA J 282, 1
(54) Tomy, G. T.; Budakowski, W.; Halldorson, T; Helm, P. A.; Stern, G. A.; Friesen, K.; Pepper, K.; Tittlemier, S. A.; Fisk, A. T. Fluorinated organic compounds in an eastern Arctic marine food web. *Environ. Sci. Technol.* **2004**, *38*, 6475–6481.
(55) Hoff, P. T.; van de Vijver, K.; van Dongen, W.; Esmans, E. L.; Blust, R.; de Coen, W. *Environ. Toxicol.* **2003**, *3*, 608–614.

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