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Specific accumulation of perfluorochemicals in harbor seals (*Phoca vitulina concolor*) from the northwest Atlantic

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ABSTRACT

Concentrations of perfluorochemicals (PFCs) including perfluoroalkylsulfonates (PFSAs), and perfluoroalkylcarboxylates (PFCAs) were determined in liver of harbor seals ($n = 68$) collected from the northwest Atlantic between 2000 and 2007. Of ten PFCs measured, perfluorooctane sulfonate (PFOS) concentrations were the highest in liver (8–1388 ng/g, ww), followed by perfluoroundecanoic acid (PFUnDA) (<1–30.7 ng/g, ww). An unusual accumulation profile of long-chain (C7-C12) PFCAs, and the predominance of PFUnDA, followed by PFNA in seal liver suggested that fluorotelomer alcohols (FTOHs) may be a major source of PFCAs in the northwest Atlantic. No gender-related differences in the concentrations of individual PFCs or total PFCs were found. Concentrations of PFOS and PFDS were higher in tissues of the pups than the adults, whereas concentrations of the PFCAs were similar between pups and adults. PFOS concentrations in the pups were 2.6-fold higher than those in the adult females, suggesting the importance of maternal transfer of PFCs. Hepatic PFOS concentrations were strongly, positively correlated with PFOSA, PFDS and individual PFCAs, indicating that harbor seals are exposed simultaneously to these compounds. Temporal comparisons of hepatic PFC concentrations showed a marginal increase of PFOS and PFCAs in the adult seals from 2000 to 2007. Unlike the spatial trend observed for polychlorinated biphenyls (PCBs), no south to north (urban–rural–remote) decreasing trend was observed for PFCs, suggesting the presence of diffuse sources of PFC contamination throughout the northwest Atlantic.

1. Introduction

Perfluorochemicals (PFCs) are persistent contaminants of anthropogenic origin that are found distributed in the environment, in wildlife, and in humans worldwide (Giesy and Kannan, 2001; Kannan et al., 2001, 2002, 2004; Houde et al., 2006). For over 40 years, PFCs have been used in a variety of industrial and consumer products, including protective coatings for carpets and apparel, nonstick cookware, paper coatings, insecticide formulations, and surfactants in fire-fighting foams (Giesy and Kannan, 2002). PFCs are oleophobic and hydrophobic, thus their accumulation is not driven by lipophilicity (Kannan et al., 2001). Some PFCs have been shown to bioaccumulate and biomagnify in marine food webs (Tomy et al., 2004; Houde et al., 2006) and elevated concentrations are detected in apex predators such as marine mammals (Kannan et al., 2001; Van de Vijver et al., 2005; Houde et al., 2006).

Two major classes of PFCs are perfluoroalkyl sulfonic acids (PFSAs), and perfluoroalkyl carboxylic acids (PFCAs). The PFSAs (e.g., perfluorooctanesulfonate [PFOS] and perfluorooctane sulfonamide [PFOSA]), are degradation products of perfluoroalkyl sulfamido alcohols via biotransformation processes and abiotic oxidation (Xu et al., 2004; D'eon et al., 2006; Martin et al., 2006). Concerns about widespread global contamination by PFOS led to a phase-out of production of PFOS-based compounds by a major producer in 2001 (3M, 2000); however, PFCAs continue to be manufactured worldwide for use as emulsifiers and additives in the polymerization process (Houde et al., 2006). Recent reports have documented the occurrence of long-chain PFCAs (C8-12) including perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluoroundecanoic acid (PFUnDA), and perfluorododecanoic acid (PFDoDA) in biota (Keller et al., 2005; Butt et al., 2007a,b; Hart et al., 2008a,b; Yoo et al., 2008). It is likely that there are multiple sources of the compounds, both direct and indirect, including those related to the manufacture and use of commercial products, and biotic and abiotic degradation of perfluoroalkyl sulfamide alcohols (to PFSAs and PFCAs) and flourotelomer alcohols (FTOHs) (to PFCAs) (Ellis et al., 2004). Recent studies suggest that the global distribution of PFSAs and PFCAs may result from the airborne transport and degradation of volatile precursor molecules as well as atmospheric and oceanic transport of PFCAs

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themselves (Ellis et al., 2004; Yamashita et al., 2005; Prevedourous et al., 2006; Young et al., 2007).

PFCs are known to adversely affect both pre- and post-natal development and the neuroendocrine and immune systems in animals via at least five different pathways (Hu et al., 2002; Houde et al., 2006; Peden-Adams et al., 2008). Recent field studies suggest that PFC-mediated effects occur in marine mammals, including infectious disease in California sea otters (Kannan et al., 2006) and modulation of the peroxisome proliferator-activated receptor α-cytochrome P450 4A-signaling pathway associated with carcinogenesis in Baikal seals (Ishibashi et al., 2008a,b). There is no evidence for biodegradation of PFCs in the environment (Giesy and Kannan, 2002), thus the toxic potential of PFSAs and long-chain PFCAs in wildlife and humans is of concern.

Most studies to date have focused on Europe, the Arctic, and the US Pacific and southeast coasts. Little is known about the status of PFC contamination in marine mammals from the northwest Atlantic. This is one of the most industrialized, heavily populated regions in the world and environmental contamination has been a concern since the 1950s. At the top of the food chain, harbor seals (*Phoca vitulina concolor*) feed on teleost fishes in coastal and estuarine waters and are exposed to high levels of persistent and bioaccumulative contaminants (Shaw et al., 2005, 2007, 2008a,b). In 1991–1992, a morbillivirus outbreak resulted in a mass mortality of harbor seals in the southern, urbanized part of the northwest Atlantic harbor seal range (Duignan et al., 1995). The most recent event occurred in 2006–2007, resulting in the deaths of more than 1000 animals in the same area (Garron and McNulty, 2008). Immunosuppression resulting from chronic exposure to environmental contaminants could not be ruled out as a contributing factor in these events. Analyses of harbor seal tissues have shown that these seals are highly exposed to polychlorinated biphenyls (PCBs), 1,1,1-trichloro-2,2-bis (p-chlorophenyl) ethane (DDTs) and other chlorinated pesticides, dioxin-like compounds including polychlorinated dibenzo-p-dioxin (PCDDs) and dibenzofurans (PCDFs), and polybrominated diphenyl ethers (PBDEs) (Shaw et al., 2005, 2007, 2008a,b). However, no data are presently available on the current status of PFC contamination in these seals.

The aim of this study was to characterize exposure to PFCs including PFSAs and PFCAs in harbor seals inhabiting the northwest Atlantic. Herein we investigate concentrations, patterns, and trends of PFCs in liver of harbor seals collected from Maine to New York between 2000 and 2007. This is the first report of the occurrence of PFCs in marine mammals from the northwest Atlantic.

2. Materials and methods

2.1. Samples

Liver samples were collected from 68 harbor seals (8 adult males, 10 adult females, 25 male pups and 25 female pups) that stranded along the northwest Atlantic coast (from Maine to New York) between 2000 and 2007 (see map of stranding locations, Fig. SI-1). Seals were weighed, and standard length and axillary girth were measured. Age was estimated based on body size. Condition indices were calculated by dividing axillary girth/standard length and body weight/standard length. Liver samples were stored in hexane and acetone rinsed aluminum foil or I-Chem jars in a freezer at −40 °C until analysis.

2.2. Chemical analysis

Concentrations of PFCs in liver were determined by the ion pairing extraction method described elsewhere (Kannan et al., 2001; Tao et al., 2006). Briefly, liver samples (0.3 g) were homogenized in 3 mL of Milli-Q water. A 2-mL aliquot was spiked with 5 ng each of $^{13}C_4$-PFOS, $^{13}C_4$-PFOA, $^{13}C_2$-PFNA, and $^{13}C_2$-PFDA as internal standards (Wellington Laboratories, Guelph, Canada). One milliliter of 0.5 M tetrabutyl-ammonium hydrogen sulfate solution, 2 mL of sodium carbonate buffer (0.25 M, pH 10), and 5 mL methyl-*tert*-butyl ether (MTBE) were added to the sample. After shaking for 40 min, the organic layer was separated by centrifugation, and the extraction was repeated with 5 mL of MTBE. The extracts were combined and evaporated to dryness under a gentle flow of nitrogen, before being reconstituted in 1 mL of methanol, vortexed, and filtered into an autosampler vial.

Separation of perfluorinated acids was performed using an Agilent 1100 high performance liquid chromatograph (HPLC). Ten microliter of the extract were injected onto a 100 × 2 mm (5 μm) Keystone Betasil C_{18} column. A gradient mobile phase of methanol and 2 mM ammonium acetate was used. At a flow rate of 300 μL/min, the mobile phase gradient was ramped from 10% to 75% methanol in 7 min and then to 100% methanol at 10 min, held at 100% methanol for 2 min, and then ramped down to 10% methanol. For quantitative analysis, the HPLC was interfaced with an Applied Biosystems API 2000 tandem mass spectrometer (MS/MS) in negative electrospray ionization mode. Analyte ions were monitored using multiple reaction monitoring mode. Parent and daughter ion transitions were monitored for detection of PFOSA, PFDS, PFOS, PFHxS, $^{13}C_4$-PFOS, $^{13}C_4$-PFOA, $^{13}C_2$-PFNA, $^{13}C_2$-PFDA, PFHpA, PFOA, PFNA, PFDA, PFUnDA, and PFDoDA. Quantitation was performed using a seven-point external calibration curve produced from concentrations of 0.1–100 ng/mL. The coefficient of determination (r^2) for each calibration was >0.99. Quality-control standards were measured after every 10 samples. The coefficient of determination (r^2) for each calibration was >0.99. Quality-control standards were measured after every 10 samples. All procedural blank peak areas were less than half the determined limit of quantitation (LOQ) for each analyte. The LOQ was estimated as three times the lowest concentration point on the calibration curve, which is accurately measured within ±30% of its theoretical value. Matrix spikes were performed several times for liver samples by spiking 5–10 ng of each target analyte, and passing through the whole analytical procedure. The coefficient of variation was <20% for each of the analytes measured. Recoveries of target analytes from the matrix were between 65% and 99%. Mean recoveries of internal standards spiked to samples were between 68% and 81%. Concentrations are reported on a wet weight (ww) basis.

2.3. Statistics

Concentrations were log normalized prior to statistical analysis using SPSS 14.0. Concentrations below the level of detection were calculated by treating the result as if half the detection limit. Two-way analysis of variance was used to test for effects of age class (adult vs. pup) and gender on contaminant levels. Since age class consistently had a significant effect, regional comparisons were performed with Student's *t*-tests within each age class separately. Time trends were analyzed with linear regressions within each age class.

3. Results and discussion

3.1. PFC concentrations

Concentrations of total PFCs (sum of 10 PFCs: PFOS, PFDS, PFOSA, PFHxS, PFHpA, PFOA, PFNA, PFDA, PFUnDA, and PFDoDA) in harbor seal livers ranged from 18.8 to 1430 ng/g, wet weight (overall mean ± standard deviation: 247 ± 289 ng/g, ww; $n = 68$)

Table 1
Concentrations of PFCs (ng/g, wet weight) in liver of northwest Atlantic harbor seals.

	Adult males (N = 8)		Adult females (N = 10)		Pups (N = 50)		All (N = 68)
	Mean ± SD	(Min.–max.)	Mean ± SD	(Min.–max.)	Mean ± SD	(Min.–max.)	% Detect
Weight (kg)	58.4 ± 10.6	(36.3–70.0)	62.8 ± 11.2	(45.4–79.6)	10.4 ± 2.8	(6.4–16.5)	
Length (cm)	147 ± 9.4	(130–158)	144 ± 7.1	(129–154)	81.4 ± 6.8	(70–112)	
PFHpA	<0.77	(<0.77)	<0.77	(<0.77)	<0.77	(<0.77–1.6)	2.9
PFOA	<1.6	(<1.6)	<1.6	(<1.6)	1.9 ± 1.3	(<1.6–8.9)	5.9
PFNA	5.8 ± 5.4	(1.3–16)	5.4 ± 5.3	(<1.9–17)	6.6 ± 7.7	(<1.9–40)	68
PFDA	5.6 ± 4.9	(<1.9–17)	4.9 ± 3.9	(<1.9–12)	4.4 ± 4.8	(<1.9–23)	62
PFUnDA	11 ± 8.1	(<3.1–23)	9.0 ± 8.1	(<3.1–22)	9.9 ± 7.0	(<3.1–31)	75
PFDoDA	3.2 ± 2.2	(<1.9–7.1)	2.8 ± 3.1	(<1.9–12)	2.4 ± 1.8	(<1.9–11)	18
Sum PFCAs	28 ± 17	(11–59)	24 ± 15	(<8.1–58)	26 ± 18	(<8.1–87)	91
PFHxS	<0.63	(<0.63)	<0.63	(<0.63)	0.68 ± 0.38	(<0.63–2.4)	8.8
PFOS	98 ± 104	(30–349)	100 ± 56	(26–221)	258 ± 312	(8.0–1388)	100
PFDS	1.7 ± 1.3	(<1.6–4.9)	<1.6	(<1.6)	3.0 ± 4.2	(<1.6–26)	28
PFOSA	1.3 ± 0.86	(<0.77–2.9)	1.3 ± 0.95	(<0.77–2.9)	1.7 ± 1.9	(<0.77–8.8)	31
Sum PFSAs	102 ± 106	(32–357)	103 ± 56	(27–227)	264 ± 315	(10–1395)	100
Sum PFCs	129 ± 120	(46–416)	127 ± 64	(45–249)	290 ± 323	(19–1430)	100

(Table 1). PFOS was the dominant PFC found in seal liver at concentrations ranging from 8 to 1388 ng/g ww (mean ± SD: 216 ± 279 ng/g ww). Other perfluoroalkyl sulfonates (PFDS and PFOSA) were detected at much lower concentrations than PFOS, ranging from <1 to 25.8 ng/g ww. PFHxS was detected at trace concentrations in only 9% of the samples. Perfluoroalkyl carboxylates (PFCAs, C7-C12) were detected at concentrations ranging from <1 to 87.4 ng/g, ww (overall mean ΣPFCAs 25.9 ng/g, ww). Among the PFCAs, PFUnDA (C11) was dominant, followed by PFNA (C9) and PFDA (C10). PFHpA, PFOA, and PFDoDA were detected at low concentrations in only 3%, 6%, and 18% of the samples, respectively. PFC concentrations in northwest Atlantic harbor seals were within the range of concentrations previously reported in liver of marine mammals from various mid-latitude locations (Table SI-1). Mean hepatic PFOS concentrations in our samples (216 ng/g, ww) were similar to those reported in Baltic gray seals (214 ng/g, ww) (Kannan et al., 2002), harbor seals from the Dutch Wadden Sea (160 ng/g, ww) (Van de Vijver et al., 2005), and harbor porpoises from the Black Sea (327 ng/g, ww) (Van de Vijver et al., 2007), but higher than those reported in harbor seals from the southern North Sea (range: <10–532 ng/g, ww) (Van de Vijver et al., 2003), and the US Pacific coast (27 ng/g,ww) (Giesy and Kannan, 2001). Higher PFOS concentrations were reported in harbor seals from the Danish coast (794 ng/g, ww) (Kallenborn et al., 2004), Baltic ringed seals (454 ng/g, ww) (Kannan et al., 2002), harbor porpoises from UK waters (range 16–2420 ng/g, ww) (Law et al., 2008), and bottlenose dolphins from the Florida coast (489 ng/g, ww) (Kannan et al., 2001) and the South Carolina coast (914 ng/g, ww) (Houde et al., 2006). The highest PFOS concentrations were reported in polar bears from Greenland (2470 ng/g, ww) (Smithwick et al., 2005) and the Canadian Arctic (3100 ng/g, ww) (Martin et al., 2004a,b), reflecting the high trophic position of these animals in the marine food chain. Much lower hepatic PFOS concentrations were reported in Arctic seals comprising the polar bear diet ranging from a mean of 8.2 and 4.6 ng/g, ww in ringed and bearded seals from the Bering and Chukchi Seas, Alaska (Quakenbush and Citta, 2008) to 95.6 ng/g, ww in ringed seals from East Greenland (Bossi et al., 2005).

No gender-related differences were found in concentrations of ΣPFCs and individual PFCs in the adult seals (mean ΣPFCs 129 ± 120 and 127 ± 64 ng/g, ww in adult males and adult females, respectively). However, the pups had significantly higher concentrations of ΣPFCs (p = 0.02) and PFOS (p = 0.01) than the adult seals (Fig. 1). Concentrations of PFDS (p = 0.01) were also higher in the pups, whereas concentrations of PFCAs were similar in pups and adults. This suggests that PFCs do not increase with age in harbor seals. A similar pattern of decreasing PFOS concentrations with age was reported in Baikal seals (Ishibashi et al., 2008a,b), bottlenose dolphins from the Florida coast (Kannan et al., 2001), Baltic gray and ringed seals (Kannan et al., 2002), and harbor porpoises in the North Sea (Van de Vijver et al., 2005). In polar bears from Greenland, PFOS concentrations were increasing up to about age six (Smithwick et al., 2005). The general lack of correlation between concentrations and age implies that the elimination capacity of PFCs may be significant in adult animals and half-lives of the compounds may be relatively short (Houde et al., 2006). A $t_{1/2}$ of 21 weeks was estimated for PFOS in bottlenose dolphins, and urine was indicated as an important depuration pathway for PFSAs and PFCAs (Houde et al., 2006). This depuration also suggests more or less continuous exposure to and uptake of PFCs to maintain tissue concentrations. The accumulation pattern of PFCs in northwest Atlantic harbor seals differs from that previously reported for lipophilic organic compounds such as PCBs, DDTs, and PBDEs (Shaw et al., 2005, 2008a,b) in which concentrations increase with age in males and decrease with age in females (after sexual maturity) due to placental and lactational transfer of these compounds from females to pups. These observations suggest that the residence time for PFCs may be shorter in adult seals than that observed for chlorinated and brominated hydrocarbons.

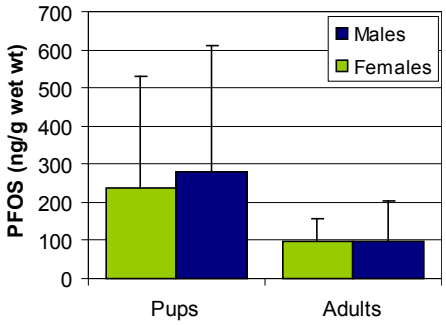

Fig. 1. PFOS concentrations (ng/g wet weight) in harbor seal livers (n = 68) by age and gender.

Although our seals were not mother–pup pairs, mean PFOS concentrations in the pups (258 ng/g) were 2.6-fold higher than those in the adult females (100 ng/g), indicating that maternal transfer is a significant exposure route for PFCs to pups. Placental transfer of PFOS has been demonstrated in laboratory animals, and was shown to affect the post-natal survival of rats (Lau et al., 2003). Although data on maternal transfer in pinnipeds are scarce, placental and/or lactational transfer of PFCs have been indicated by the results of paired studies of bottlenose dolphin mother–calves from Sarasota Bay, Florida (Houde et al., 2006), melon-headed whale

mother–fetuses from the Japanese coast (Hart et al., 2008a,b), and in a harbor porpoise mother–fetus from northern Europe (Van de Vijver et al., 2005). Compared to concentrations in the mothers, PFOS concentrations were up to 10 times higher in the dolphin calves and were detected in milk (Houde et al., 2006), implying lactational transfer of PFOS to the calves. In fetuses of harbor porpoise and melon-headed whales, PFOS concentrations were more than twofold higher than in their mothers. Transplacental rates of PFCs between whale mothers and fetuses were higher than those observed for PCBs and PBDEs, suggesting that significant placental transfer and fetal exposure to PFCs occur in cetaceans.

3.2. PFC profiles

Of the 10 PFCs detected in harbor seal liver samples, PFOS contributed 77–89% of the total PFC content, followed by perfluoroundecanoic acid (PFUnDA), accounting for 3–8% of the total (Fig. 2). Whereas PFOS is the predominant PFC found in most wildlife species, elevated ratios of PFOS to ΣPFCs (>0.7) have been reported in species from various locations including polar bears from the Canadian Arctic (Martin et al., 2004a,b) and Greenland (Bossi et al., 2005), bottlenose dolphins from the southeastern US coast (Houde et al., 2006), and humpback dolphins and finless porpoises from Hong Kong, China (Yeung et al., 2009).

The PFCA profile in harbor seal liver was dominated by PFUnDA (C11, mean 9.9 ng/g, ww), accounting for 47–63% of the ΣPFCA content in liver, followed by PFNA (C9, mean 6.3 ng/g, ww), PFDA (C10, mean 4.6 ng/g, ww), and PFDoDA (C12, mean 2.5 ng/g, ww) (Fig. 2). This profile is interesting because for most marine mammals from North American and European coastal waters, PFNA dominated the PFCA profile and is the second most prevalent PFC, after PFOS (Martin et al., 2004a,b; Kannan et al., 2005; Smithwick et al., 2005; Van de Vijver et al., 2005). PFUnDA was the second most abundant PFC in liver of humpback dolphins and finless porpoises from Hong Kong waters (Yeung et al., 2009), ringed seals from Greenland (Bossi et al., 2005), fish from Lake Ontario (Martin et al., 2004a,b), and birds and fish from the Canadian Arctic (Martin et al., 2004a,b). Concentrations of PFUnDA in skipjack tuna from several locations in the western North Pacific Ocean were greater than the concentrations of PFOS (Hart et al., 2008a,b). Although there are different exposure pathways and bioaccumulative potentials for individual PFCs in marine mammals, seabirds, and fish, it is clear that PFOS and PFUnDA are present in the northwest Atlantic marine environment and these compounds can accumulate in tissues of harbor seals.

Another interesting finding of the present study was the pattern of PFCA profiles in harbor seal liver: PFUnDA (C11) > PFNA (C9) > PFDA (C10) > PFDoDA (C12), which differs from the general odd/even pattern observed in biota. In addition, whereas concentrations of PFCAs generally decrease with increasing perfluoroalkyl chain length (Martin et al., 2004a,b), the profile in harbor seals peaked at PFUnDA, and there were decreasing concentrations of longer and shorter PFCA homologues alike. The reason for the different contamination profile in the harbor seals is unknown, but it may be indicative of common sources such as the FTOHs. The finding of higher concentrations of odd-chain-length PFCAs than even-chain-length PFCAs also implicates FTOHs as a source of exposure (Ellis et al., 2004; Kannan et al., 2005). Species-specific differences in elimination capacity for PFCAs can be ruled out, since a consistent odd/even pattern of PFCAs has been reported in harbor seals (Van de Vijver et al., 2005). FTOHs are manufactured in even-chain-lengths but are reported to degrade to even- and odd-chain-length PFCAs (Ellis et al., 2004). While 8:2 FTOH was shown to degrade to PFOA and PFNA, 10:2 FTOH degraded to PFDA and PFUnDA in wastewater treatment (Sinclair and Kannan, 2006). Thus, the predominance of PFUnDA in our samples is suggestive of 10:2 FTOH as a source of PFUnDA. Atmospheric oxidation of 10:2 FTOH produces equal amounts of PFDA and PFUnDA (Ellis et al., 2004), but because PFUnDA is more bioaccumulative, this odd-chain-length acid is predominant in biota. The finding of PFNA as the second most abundant PFCA in harbor seal liver suggests that 8:2 FTOH may also be a significant source, since atmospheric oxidation of 8:2 FTOH to PFOA and PFNA would lead to the predominance of PFNA. It is also possible that proximity to ambient sources such as wastewater effluents in the coastal habitat could obscure the odd- and even-chain-length pattern in these seals.

PFC homologue profiles in the harbor seals varied by age and gender (Fig. 2). The pups retained a higher proportion of PFOS and PFNA compared with the adult seals, and short-chain PFCAs (PFHpA and PFOA) were present in pup liver but were not detected in the adults. These differences reflect the various exposure pathways between pups and adults and suggest that pups may possess a limited metabolic/elimination capacity for PFCs. PFUnDA was more abundant in adult females than adult males or the pups, while PFDoDA was more abundant in adult males. These differences in profiles could reflect the effect of maternal transfer in the females, differences in elimination capacity for individual PFCs, and differences in habitat and prey selection between males and females.

Significant positive correlations were found between PFOS and PFCAs in the harbor seal liver samples. PFOS was significantly correlated with PFOSA ($p < 0.05$), as well as PFDS, PFNA, PFDA, and PFUnDA ($p < 0.01$), but not with PFDoDA, probably because of the low detection of this compound (Fig. SI-2). PFOSA, the precursor molecule for PFOS, was not correlated with PFDS or any of the PFCAs. Among the PFCAs, PFNA, PFDA, and PFUnDA were highly

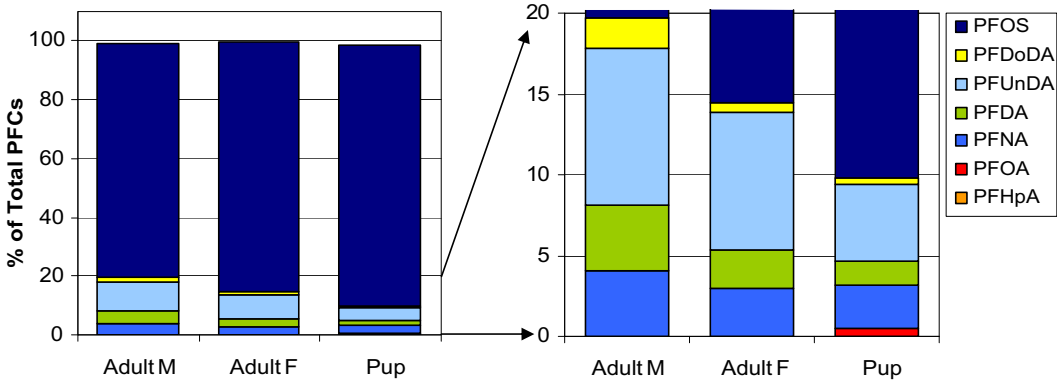

Fig. 2. Perfluorochemical profiles in liver of harbor seals from the northwest Atlantic.

intercorrelated in seal liver. Overall, these results suggest that the harbor seals were exposed to PFOS and PFCAs simultaneously, probably through the same pathways, and the compounds might have originated from similar sources (Ellis et al., 2004).

3.3. Temporal trends

Using linear regression statistics, we investigated temporal trends of PFCs in harbor seals collected between 2000 and 2007 (Fig. 3). A trend of increasing PFOS and ΣPFCA concentrations was observed in the adult seals between 2000 and 2007, although this was not statistically significant ($p = 0.18$ and 0.17 for PFOS and ΣPFCA, respectively). In the harbor seal pups, no temporal trend was observed for PFOS or PFCA concentrations during this seven-year period. Several studies have reported an increasing trend in PFOS and PFCA concentrations in marine mammals over the past 20–30 years (Bossi et al., 2005; Smithwick et al., 2006; Dietz et al., 2008; Ishibashi et al., 2008a,b). In some locations, PFOS concentrations appear to be declining following the phase-out of perfluorooctanesulfonylfluoride (POSF)-based compounds in 2001 (Butt et al., 2007a,b; Hart et al., 2008a,b), whereas this pattern is not observed for long-chain PFCAs (C9-11). To examine possible changes in homologue composition over time, we compared PFC profiles in harbor seal livers collected at two time points: 2000–2002 and 2003–2007. PFOS was the dominant PFC and its relative abundance was similar over time, indicating that the source composition of POSF-based compounds has not changed in the northwest Atlantic marine ecosystem. PFDA was relatively more abundant in adults and pups and PFUnDA was less abundant in the adult seals in 2003–2007 compared with 2000–2002, which suggests changes in uses/releases of PFCAs during this period. In recent studies, we found a similar lack of a time trend for PCBs, DDTs, and PBDEs in blubber of harbor seals from this region between 1991 and 2005 (Shaw et al., 2005, 2008a,b), suggesting continuous inputs and/or recycling of these persistent halogenated compounds in the northwest Atlantic.

3.4. Spatial trends

Considered relatively non-migratory, northwest Atlantic harbor seals nevertheless make seasonal movements along the coast from Maine southward to the coast of New Jersey (Fig. SI-1) (NMFS,

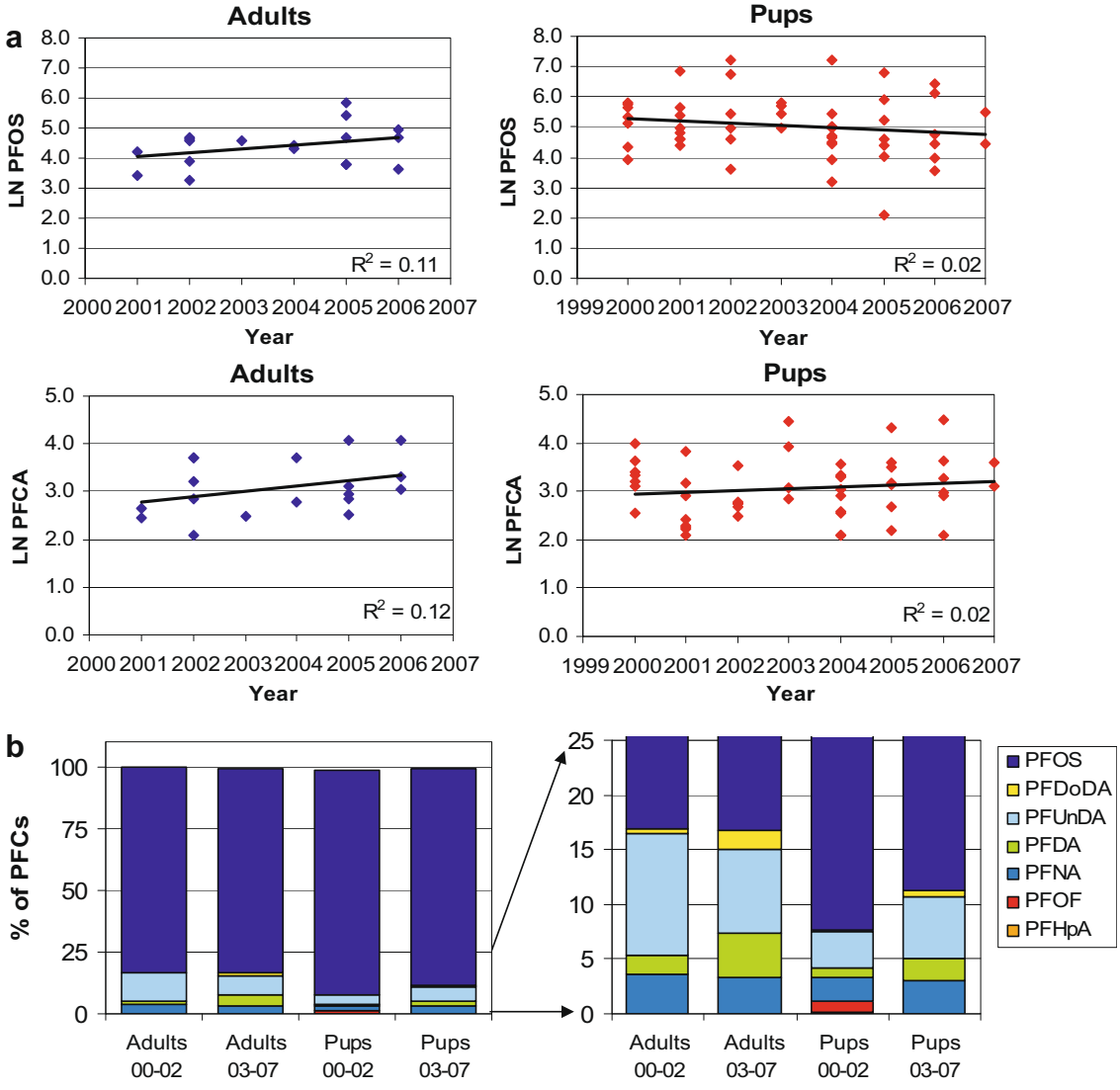

Fig. 3. (a) Perfluorochemical concentrations in harbor seals collected along the northwest Atlantic from 2000 to 2007; (b) composition profiles of PFCs in harbor seal liver at two time points: 2000–2002 and 2003–2007.

2007), and accumulate high concentrations of persistent organic chemicals through consumption of a variety of teleost fishes (Shaw et al., 2005, 2007, 2008a,b). To examine spatial trends, we compared PFC concentrations in liver of adults and pups from the industrialized southern area (Massachusetts to New York) with those from the lightly populated, rural northern area (central and eastern Maine). PFOS was the dominant PFC in liver at both locations; mean PFOS concentrations were higher in adult seals from the southern area (128 ng/g, ww) than the northern area (70.3 ng/g, ww), although this difference was marginally significant (Student's t-test $p = 0.07$). This finding is consistent with the spatial trend reported for loggerhead sea turtles along the US eastern coast (Keller et al., 2005). Taken together, the data suggest that the densely populated mid-Atlantic region may be a large source region for PFCs. Concentrations of PFCAs were not different by location in the adult seals, but higher PFDoDA concentrations were found in pups from the northern area ($p = 0.02$). This spatial distribution varies from that reported previously for PCBs (Shaw et al., 2005) and is consistent with the pattern observed for PBDEs in northwest Atlantic harbor seals (Shaw et al., 2008a,b). Unlike the trend for PCBs, which decreased with increasing latitude as a function of distance from sources near industrialized urban centers in the south, a south to north (urban–rural–remote) decreasing gradient was not observed for PBDEs or PFCs. It is believed that direct sources of PFCs including emissions/releases from fluorochemical plants and airports and military bases with fire-fighting operations can result in elevated concentrations in urbanized locations (Houde et al., 2006). However, unlike PCBs, PFCs and PBDEs are widely used in household and consumer products and therefore may originate from diffuse common sources within the region including landfill leachate and wastewater effluent from households and industries generally (Houde et al., 2006; Law et al., 2006). Moreover, the PFC contamination pattern in these seals suggests that volatile precursors (sulfonamido alcohols and FTOHs) are important sources of PFCs. Thus the lack of an urban–rural–remote decreasing spatial gradient in hepatic PFC concentrations implies that such diffuse sources are significant across the harbor seal range. These results underline the growing problem of PFC contamination of marine ecosystems and the importance of monitoring PFCs in the northwest Atlantic marine food web.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.chemosphere.2008.10.063.

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