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Contamination and Effects of Perfluorochemicals in Baikal Seal (*Pusa sibirica*). 1. Residue Level, Tissue Distribution, and Temporal Trend

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Concentrations of perfluorochemicals (PFCs) including perfluoroalkylsulfonates (PFSAs) and perfluoroalkylcarboxylates (PFCAs) were determined in liver and serum of Baikal seals (*Pusa sibirica*) collected from Lake Baikal, Russia in 2005. Among the 10 PFC compounds measured, perfluorononanoic acid (PFNA, 3.3–72 ng/g wet wt) concentrations were the highest in liver, followed by perfluorooctanesulfonate (PFOS, 2.6–38 ng/g). The accumulation profile of long-chain (C7-C12) PFCAs in particular, the predominance of PFNA, indicated that 8:2 fluorotelomer alcohol or commercially manufactured PFNA is a major local source of PFCs in Lake Baikal. No gender-related differences in the concentrations of individual PFCs or total PFCs were found. Tissues from pups and juveniles contained relatively higher concentrations of PFCs than tissues from subadults and adults, suggesting that maternal transfer of PFCs is of critical importance. Comparison of concentrations of PFCs in livers and sera collected from the same individuals of Baikal seals revealed that residue levels of PFOS, PFNA, perfluorodecanoic acid (PFDA), and perfluoroundecanoic acid (PFUnDA) were significantly higher in liver than in serum. The concentration ratios of PFNA and PFDA between liver and serum were calculated to be 14 and 15, respectively, whereas the ratio of PFOS was 2.4. This suggests preferential retention of both PFNA and PFDA in liver. Concentrations of PFOS, PFNA, and PFDA in liver were significantly correlated with those in serum, whereas concentrations of PFUnDA were not correlated in between the two tissues, suggesting differences in pharmacokinetics among these PFCs. Temporal comparisons of hepatic PFC concentrations in seals collected between 1992 and 2005 showed that the concentrations of PFOS ($p = 0.0006$), PFNA ($p = 0.061$) and PFDA ($p = 0.017$) were higher in animals collected in recent years, indicating ongoing sources of PFC contamination in Lake Baikal.

Introduction

Bioaccumulation and widespread distribution of perfluorochemicals (PFCs) have been reported in wildlife and humans (*1–3*). PFCs that are frequently detected in biological samples are perfluoroalkylsulfonates (PFSAs) and perfluoroalkylcarboxylates (PFCAs). PFSAs, represented by perfluorooctane sulfonate (PFOS) and perfluorooctane sulfonamide (PFOSA), are degradation products of perfluoroalkylsulfonamido alcohols via biotransformation processes and through abiotic oxidation (*4–6*). Recent surveys have reported the occurrence of long-chain PFCAs including perfluorooctanoic acid (PFOA, C8), perfluorononanoic acid (PFNA, C9), perfluorodecanoic acid (PFDA, C10), perfluoroundecanoic acid (PFUnDA, C11), and perfluorododecanoic acid (PFDoDA, C12) in the environment (*7–10*). There are both direct and indirect sources of PFCA emissions to the environment. Direct sources are the manufacture and use of the commercial products (*11*); indirect sources are impurities in perfluorooctyl sulfonyl-based and fluorotelomer-based products and degradation of fluorotelomer alcohols (FTOHs) and fluoropolymers (*12*). Recent studies have suggested global transport of PFCAs via atmospheric transport and degradation of FTOHs, and atmospheric and oceanic transport of the PFCAs themselves (*11, 13, 14*).

Owing to the high persistence and high bioaccumulation potential of PFCs such as PFOS and PFOA in the environment, their potential toxic effects in biota are of great concern. However, much of the information on the contamination by PFCs is from biological samples collected in North America and Western Europe (*2, 15*). Little is known about the current status and temporal trend of PFC contamination in Russia.

Since the 1990s, it has been shown that Lake Baikal, located in eastern Siberia, Russia, and retaining a fifth of the world's freshwater, is exposed to a variety of anthropogenic contaminants, including organochlorines (*16, 17*). Baikal seal (*Pusa sibirica*), an endemic species and a high trophic-level predator at the top of the food web in the lake, is vulnerable to exposure to persistent and bioaccumulative contaminants. In 1987–1988, an outbreak of morbillivirus infection resulted in mass mortality of Baikal seals. Immunosuppression resulting from chronic exposure to environmental contaminants was considered as a contributing factor for this epizootic, even though the direct cause for this outbreak was infection (*18*). Analyses of Baikal seal tissues demonstrated high contamination by polychlorinated biphenyls (PCBs), 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl) ethane and its metabolites (DDTs), and dioxin-like compounds including polychlorinated dibenzo-*p*-dioxin (PCDDs), dibenzofurans

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TABLE 1. Concentrations of PFCs in Liver (ng/g, wet wt) and Serum (ng/g, wet wt) of Baikal Seals Collected in 2005

	liver				serum			
	male (n = 20)		female (n = 24)		male (n = 19)		female (n = 24)	
	mean ± SD	min–max	mean ± SD	min–max	mean ± SD	min–max	mean ± SD	min–max
age (year)	10 ± 14	(0.3–42)	12 ± 14	(0.3–42)				
body weight (kg)	40 ± 22	(14–89)	44 ± 26	(13–91)				
body length (cm)	120 ± 22	(92–158)	120 ± 23	(86–153)				
sulfonates								
PFOS[a]	13 ± 9.4	(3.9–36)	9.7 ± 8.2	(2.6–38)	5.8 ± 4.9	(1.4–17)	4.5 ± 3.6	(1.3–14)
PFHS	<0.55		<0.55		0.14 ± 0.10	(<0.12–0.55)	0.13 ± 0.043	(<0.12–0.31)
PFDS	<0.57		<0.57		<0.17		<0.17	
PFOSA	0.62 ± 0.092	(<0.55–0.81)	0.69 ± 0.42	(<0.55–2.2)	<0.17		<0.17	
carboxylates								
PFHpA (C7)	<0.56		<0.56		0.85 ± 1.8	(<0.33–8.2)	0.47 ± 0.39	(<0.33–2.0)
PFOA (C8)	1.6 ± 0.55	(<1.5–3.9)	1.5 ± 0.24	(<1.5–2.5)	<0.33		0.40	(<0.33–1.9)
PFNA (C9)[a]	19 ± 19	(3.4–72)	16 ± 11	(3.3–42)	2.5 ± 3.6	(<0.33–16)	2.0 ± 3.6	(<0.33–14)
PFDA (C10)[a]	7.1 ± 7.2	(<0.56–26)	8.5 ± 7.6	(1.1–35)	0.85 ± 0.78	(<0.33–3.1)	0.58 ± 0.38	(<0.33–1.4)
PFUnDA (C11)[a]	7.2 ± 7.2	(<0.56–27)	8.0 ± 7.3	(<0.56–26)	2.3 ± 2.1	(<0.83–7.5)	1.9 ± 1.7	(<0.83–8.2)
PFDoDA (C12)	0.66 ± 0.31	(<0.56–1.8)	0.69 ± 0.32	(<0.56–1.5)	0.36 ± 0.073	(<0.33–0.56)	0.34 ± 0.034	(<0.33–0.48)
ΣPFCs	50 ± 37	(15–143)	47 ± 26	(14–124)	14 ± 10	(4.4–33)	11 ± 6.4	(4.3–27)

[a] Shows that the concentrations in liver were significantly higher than concentrations in serum ($p < 0.05$).

(PCDFs), and coplanar PCBs (Co-PCBs) (*16, 19, 20*). These results also suggested that Baikal seal is a suitable bioindicator with which to elucidate chemical contamination in Lake Baikal. Nevertheless, no data on the contamination by PFCs is available for this species.

Apart from the broader concern regarding contamination status of PFCs in Russia, the pharmacokinetics of individual PFCs in Baikal seal is also of particular interest. Although some literatures address the pharmacokinetics of PFCs, there appears to be a species difference in gender- and age-dependency on PFC concentration (*21, 22*).

The objective of our study was to assess contamination levels and effects of PFCs in Baikal seals. In this paper, we report the concentrations of PFCs in liver and serum of Baikal seals. Based on the PFC concentrations, gender- and age-related differences in tissue concentrations are addressed. The relative partitioning of PFCs between the liver and serum is also discussed. In addition, we compared the concentrations of PFCs in seals collected in 1992 and 2005, to investigate temporal trends in contamination by these compounds. The specific toxic effects of PFCs in Baikal seal are presented in the companion paper (*23*).

Materials and Methods

Standards and Reagents. Potassium salts of PFOS (>95%), PFOA (98%), perfluorohexanesulfonate (PFHS, 99.9%), perfluorooctane sulfonamide (PFOSA, 95%), and perfluorobutane sulfonate (PFBS, 95%) were provided by the 3M Company (St. Paul, MN). Perfluoroheptanoic acid (PFHpA), PFNA, PFDA, PFUnDA, and PFDoDA were from Fluorochem Ltd. (>95%; Old Glossop, Derbyshire, U.K.). Perfluorodecanesulfonate (PFDS), $^{13}C_4$-PFOS, and $^{13}C_4$-PFOA were from Wellington Laboratories (>98%; Guelph, ON, Canada). All solvents were HPLC grade, and all reagents were ACS grade (J. T. Baker, Phillipsburg, NJ).

Samples. A total of 44 Baikal seals were collected from Lake Baikal, Russia, in May–June of 2005, with the permission of the local government. For the PFC analysis, liver (n = 44) and serum (n = 43) samples were immediately removed after the recording of biometrics (body weight and length) on board, and were stored in a freezer. Details of the samples analyzed are shown in Table 1. For the analysis of temporal trend of PFCs, liver samples (n = 11) from Baikal seals collected in 1992 (*19, 20*), which were kept frozen at −20 °C in the Environmental Specimen Bank (*es*-BANK) at Ehime University, were used. The age of the animals collected in 2005 was determined from dentinal and cemental growth layers in a canine tooth. Ages and biometrics of seals collected in 1992 have been reported elsewhere (*19, 20, 24–26*).

Chemical Analysis. PFCs in liver and serum of Baikal seals were analyzed following the method described elsewhere (*27*). For the extraction of serum samples, 1 mL of serum, 5 ng of internal standards (PFBS, $^{13}C_4$-PFOS, and $^{13}C_4$-PFOA), 2 mL of 0.25 M sodium carbonate buffer, and 1 mL of 0.5 M tetrabutylammonium hydrogensulfate solution (adjusted to pH 10) were mixed in a 15-mL polypropylene (PP) tube. The sample was then extracted with 5 mL of methyl-*tert*-butyl ether (MTBE) by shaking vigorously for 45 min. The MTBE layer was separated by centrifugation at 3500 rpm for 5 min and then transferred into another PP tube. The MTBE extract was evaporated to near-dryness under a gentle stream of nitrogen and was then reconstituted with 1 mL of methanol. The sample was vortexed for 30 s and filtered through a 0.2-μm nylon filter into an autosampler vial. For the extraction of liver samples, a small amount of liver tissue (about 1 g) was homogenized with 5 g of Milli-Q water, and then 1 g of the homogenate was transferred into a PP tube and extracted following the procedure described previously (*27*). Matrix-matched calibration standards (seven points ranging from 0.5 to 75 ng/mL) were prepared by spiking of various amounts of calibration standards into a sample that contained no quantifiable amount of the target analytes; these standards were passed through the entire analytical procedure along with the samples.

Analytes were detected and quantified using an Agilent 1100 series high-performance liquid chromatograph (HPLC) coupled with an Applied Biosystems API 2000 electrospray triple-quadrupole mass spectrometer (ESI-MS/MS). Ten microliters of the extract were injected onto a 100 mm × 2.1 mm (5 μm) Keystone Betasil C18 column. The mobile phase was 2 mM ammonium acetate/methanol starting at 10% methanol at a flow rate of 300 μL/min. The gradient increased to 100% methanol over 10 min and was held for 2 min and then reversed back to 10% methanol. The MS/MS was operated in electrospray negative ion mode. Target compounds were determined by multiple reaction monitoring (MRM). The MRM transitions were 299 > 80 for PFBS, 399 > 80 for PFHS, 499 > 99 for PFOS, 503 > 99 for $^{13}C_4$-PFOS, 599 > 99 for PFDS, 498 > 78 for PFOSA, 363 > 169 for PFHpA, 369 > 169 for PFOA, 372 > 172 for $^{13}C_4$-PFOA, 463 > 219 for PFNA, 513 > 219 for PFDA, 563 > 169 for PFUnDA, and 613 > 169 for PFDoDA. Samples were injected twice, for

monitoring of sulfonates and carboxylates separately, and PFBS was monitored in both of the injections. A midpoint calibration standard was injected after every 10 samples, to check for the instrumental response and drift. Calibration standards were injected daily before and after the analytical runs.

The quantitation of liver and serum samples was performed using quadratic regression fit analysis weighted by $1/x$ of the extracted calibration curve. The limit of quantitation (LOQ) was determined as the lowest acceptable standard in the calibration curve that is defined as a standard within ± 30% of the theoretical value and that has a peak area twice as great as the analyte peak area in blanks. For all analytes, LOQs were from 0.56 to 1.46 ng/g wet wt in liver samples, and from 0.12 to 0.33 ng/mL in serum samples.

Matrix spikes (six liver and three serum samples) were performed for liver and serum samples. Known amounts of mixed PFC standards (10 ng each) were spiked into sample matrices before extraction and were passed through the entire analytical procedure. Recoveries of most of PFCs and the internal standards through the analytical procedure were 61–102% and 69–105%, respectively. Concentrations were not corrected for the recoveries of internal standards. The relative standard deviation (RSD) of repeated matrix spike analysis was <10%. Blanks were analyzed by passing water and reagents through the entire analytical procedure. Blanks contained trace levels of PFOA. Concentration values reported here were subtracted from the highest blank value.

Statistical Analysis. All the statistical analyses were performed using StatView J 5.0 (SAS Institute Inc., Cary, NC). Concentrations below the LOQ were assigned a value equal to half of the LOQ. Regression analysis was carried out to assess the relationships among biometric parameters (age, gender, body weight, and body length) and PFC concentrations, and the relationships between concentrations of PFCs in liver and in serum samples. The paired *t*-test was used to compare the concentrations of PFCs between liver and serum, and to compare PFC concentrations in the two collection years (1992 and 2005). Statistical significance was regarded as $p < 0.05$.

Results and Discussion

Residue Level and Contaminant Profiles. Concentrations of PFCs (ΣPFCs) in seal livers were in the ranges of 15–143 ng/g wet wt (mean ± standard deviation: 50 ± 37) for males and 14–124 ng/g wet wt (47 ± 26) for females (Table 1). ΣPFC concentrations in the serum of males (14 ± 10) and females (11 ± 6.4) were lower than the concentrations in liver (Table 1). No gender-related difference in the concentrations of ΣPFCs and individual PFCs was found in either sample type (Table 1 and Figure S1, Supporting Information). The concentrations of ΣPFCs and individual PFCs in liver and serum of Baikal seals were within the range of concentrations previously reported for pinnipeds and cetaceans from various areas (*7–9, 28*). Hepatic PFOS concentrations in Baikal seal were lower than concentrations reported for harbor seals from the Dutch Wadden Sea (46–488 ng/g), but similar to concentrations reported for ringed seals from the Canadian Arctic (8.6–37 ng/g). Since the age of animals from which the tissues were acquired ranged from 0.3 to 42 yr, relationships between age and individual PFC concentrations were examined. Concentrations of ΣPFCs and individual PFCs including PFOS, PFNA and PFDA in liver (Figure S2) and serum (Figure S3) showed no increase with age, in Baikal seals. These associations were consistent with results of several previous reports of PFOS concentration in bottlenose dolphin (*2*) and in gray and ringed seals (*29*). Interestingly, concentrations of ΣPFCs and individual PFCs in pups and juveniles were higher than concentrations in subadult/adult seals, although a high level of variability was observed in the pups/juveniles as compared to the subadults/adults (Figure S2 and S3). A significant difference in the concentrations of ΣPFCs between pups/juveniles (age: <0.3–2 yr, mean 73 ng/g wet wt) and subadults/adults (age: >2 yr, mean 33 ng/g wet wt) was found (Figure 1, $p < 0.05$). This indicates placental and/or lactational transfer of PFCs from mother to pups. It is known that lactation in phocid seals continues briefly but intensively, and phocid milk contains a higher proportion of fat than the milk of other aquatic and terrestrial mammals. Throughout pregnancy and lactation, large amounts of fatty acid are provided by the mother from the mobilization of fatty acid stored in adipose tissue (*30*). Considering that PFCs have a structural similarity to fatty acids, it is likely that efficient mobilization of PFCs from maternal blubber and blood resulted in the elevated levels of PFCs in the pups and juveniles. In addition, it may be due to the less metabolic/elimination capacity of PFCs in pups/juveniles compared with subadults/adults. The gender- and age-related concentration patterns of PFCs in Baikal seals were different from the patterns reported for persistent organic pollutants (POPs) such as PCBs. Concentrations of most of the POPs generally increased linearly with age in males and decreased with age in adult females (*19, 20*). Our results clearly indicate that PFCs have a shorter residence times than POPs in seal tissues (*9*).

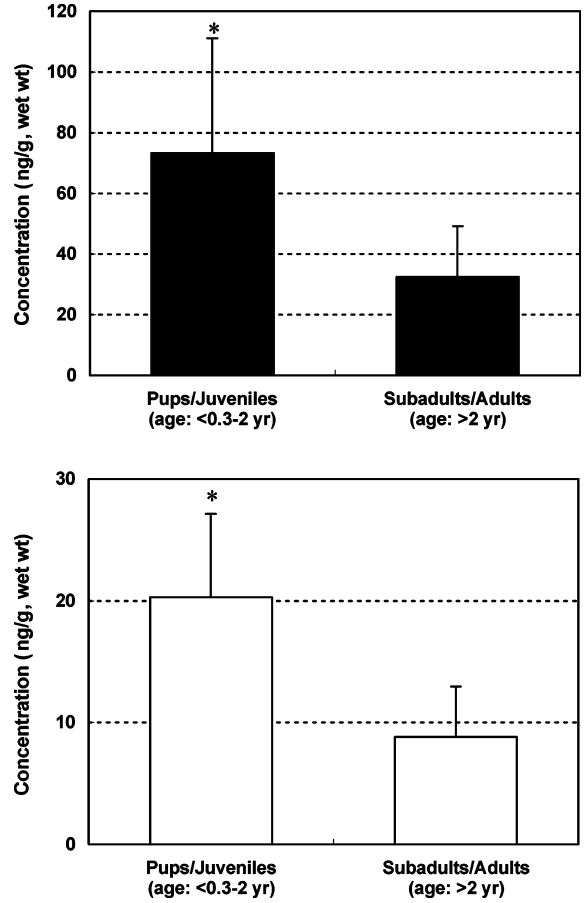

FIGURE 1. Concentrations of ΣPFCs in liver (■) and serum (□) of pup/juvenile (age: <0.3–2 yr) and subadult/adult (age: >2 yr) Baikal seals. Data are presented as the mean and standard deviation of ΣPFC concentrations. The * indicates that the concentrations in liver or serum of pups/juveniles were significantly higher than concentrations in those of subadults/adults ($p < 0.05$).

Among the 10 PFC compounds analyzed in liver of Baikal seals, concentrations of PFNA (male: 3.4–72 ng/g wet wt, female: 3.3–42 ng/g wet wt) were the highest, followed by PFOS (male: 3.9–36 ng/g wet wt, female: 2.6–38 ng/g wet wt)

(Table 1). Interestingly, the concentration profile of PFOS and PFNA in Baikal seal liver was unique; the mean (±SD) concentration ratio of PFNA to PFOS in individual seals was 1.7 ± 6.3. In contrast to the predominance of PFNA in Baikal seal, PFOS has been found previously to be the most abundant PFC in most wildlife species from various locations (*28*). A previous study showed that the concentrations of PFNA (mean 16 ng/g) were approximately 2-fold greater than those of PFOS (mean 8.7 ng/g) in the liver of mink (*Mustela vison*) collected from Watson Lake, Yukon (*7*). In polar bear and ringed seal, concentrations of PFOS were greater than PFNA concentrations by 3–20 fold, suggesting that PFOS was the dominant compound in the Arctic's top predators (*7*). The reasons for the differences in PFC contamination profiles between Baikal seal in our study and other wildlife species are unclear, but may be related to the difference in local source of PFCs and elimination capacity of PFCs in seals. However, species-specific elimination capacity of PFCs can be ruled out, because Canadian ringed seal, which are phylogenetically closer to Baikal seal, contain PFOS as the predominant contaminant in tissues (*7*). Therefore, the predominance of PFNA in Baikal seal may be due to local sources; 8:2 FTOH or commercially manufactured PFNA could be sources of PFCs in Lake Baikal. Metabolic degradation of 8:2 FTOH can result in the formation of PFNA in tissues (*31*), although the formation of PFNA from 8:2 FTOH has not been observed directly in aquatic mammals including seals.

Among PFCAs, PFNA (C9) was the predominant contaminant in liver, followed by PFDA (C10) and PFUnDA (C11) (Figure S1). Concentrations of PFHpA (C7) and PFOA (C8), and of PFDoDA (C12), were lower than concentrations of C9−C11 acids. A similar PFCA profiles was reported for polar bear (*Ursus maritimus*) and ringed seal (*Pusa hispida*) from southern Hudson Bay (*7*) and for polar bear from East Greenland (*32*). The odd- and even-chain-length pattern (PFNA, C9 vs PFOA, C8) observed in Baikal seal is consistent with the hypothesis that 8:2 FTOH is a major source of PFNA in the environment; since the atmospheric oxidation of 8:2 FTOH produces equal amounts of PFOA and PFNA (*33*), but PFNA is more bioaccumulative than PFOA (*34*), a preponderance of PFNA is expected in biota. The accumulation profile of PFOA and PFNA in the liver of Baikal seals further suggests the presence of 8:2 FTOH-related sources in Lake Baikal. The profile of C9-C12 acids in livers showed an overall trend of decreasing concentration with increasing chain length (C9 > C10 = C11 > C12) (Figure S1). This PFCA profile is similar to the profile reported for polar bear from Canada and Greenland and harbor seal from the Dutch Wadden Sea (*15, 28*). The importance of carbon chain length of C7-C11 PFCAs in hepatic kinetics has been shown (*35*). ^{19}F- NMR spectra of urine and bile samples showed no fluorometabolites of PFCAs examined and suggested that the distribution of PFCAs in urine and bile is dependent upon carbon chain length (*35*). The aqueous solubility of C7- and C8-PFCAs appears to facilitate rapid urinary excretion. The relative hydrophobicity of C9-, C10-, and C11-PFCAs appears to favor biliary enterohepatic recirculation.

The hepatic PFOS concentrations in Baikal seals were significantly, positively correlated with the concentrations of PFNA and PFDA, and the PFNA concentrations were also positively correlated with the concentrations of PFDA (Figure S4). This indicates that there is a common source for PFCs such as perfluorooctylsulfonamides, 8:2 FTOH, and 10:2 FTOH near Lake Baikal.

In serum, concentrations of PFOS (male: 1.4–17 ng/g, female: 1.3–14 ng/g) were significantly higher than those of PFNA (male: <0.33–16 ng/g, female, <0.33–14 ng/g) (Table 1 and Figure S1, $p < 0.05$). This result is different from what was found in liver (PFNA > PFOS). PFNA and PFDA concentrations were significantly lower in serum than in liver (Figure S1, $p < 0.05$), indicating differences in the partitioning of these compounds between tissues.

TABLE 2. Liver to Serum Concentration Ratios of PFCs in Baikal Seals Collected in 2005

	liver/serum concentration ratio		
	n^a	mean ± SD[b]	min−max
sulfonates			
PFOS	43	2.4 ± 0.80	(0.78–4.5)
PFHS	0	NA	
PFDS	0	NA	
PFOSA	4	2.1 ± 2.8	(0.10–6.2)
carboxylates			
PFHpA (C7)	0	NA	
PFOA (C8)	2	0.63	(0.48–0.78)
PFNA (C9)	39	14 ± 7.8	(1.6–39)
PFDA (C10)	29	15 ± 8.6	(1.2–32)

[a] The number of specimens for which PFCs was detected both in liver and serum. [b] NA: no data available.

PFC concentrations in serum samples exhibited significant positive correlations among PFOS and PFDA (Figure S5). Nevertheless, PFNA showed poor associations with PFOS and PFDA (Figure S5). This suggests that PFNA is more efficiently retained in liver and/or less stable in serum than are PFOS and PFDA.

Liver-to-Serum Distribution. Concentrations of PFCs in livers and sera collected from the same individuals of Baikal seals were compared. Concentrations of PFOS, PFNA, and PFDA were significantly higher in liver than concentrations in serum (Figure S1, $p < 0.05$). Interestingly, liver-to-serum concentration ratios (defined as ratios of concentrations of individual PFCs in the liver to those in the serum) for PFNA were 14 ± 7.8, and the ratios for PFDA were 15 ± 8.6 (Table 2). The liver-to-serum concentration ratios for PFOS were 2.4 ± 0.80 (Table 2). These results suggest compound-specific persistence and retention of PFCs in liver. An earlier study has shown that liver-to-serum concentration ratios of PFOS in ringed and gray seals were 2.7 and 5.5, respectively; lower ratios were found for fish (*29*). The ratios in Baikal seal were similar to ratios reported for other seals, but greater than the ratios reported for fish, suggesting that the mechanism underlying toxicokinetics of PFOS is conserved in pinnipeds.

Concentrations of PFOS, PFNA, and PFDA in livers were significantly correlated with concentrations in serum (Figure 2). On the basis of the strength of the correlation for PFOS ($r^2 = 0.82$, $p < 0.0001$), we suggest that PFOS does undergo biliary enterohepatic recirculation. It is known that cholestyramine, an anion-binding resin that binds bile acids in the gastrointestinal tract to prevent their reabsorption, has a considerable effect in enhancing the fecal elimination of PFOS (*36*). Therefore, PFOS elimination may depend on the interaction with bile acids. The different mode of interaction of PFCs with bile acids may influence the concentrations and tissue distribution of individual PFCs in the body. Little information is available on the relationship of concentrations of PFCs between different tissues in wildlife. A significant positive correlation was found for PFOS concentrations in the liver and blood of ringed and gray seals (*29*).

Temporal Trend. To investigate temporal trends of PFCs in Baikal seals, we compared PFC concentrations in liver of seals collected in 1992 (Table S1) with the liver samples collected in 2005. The mean concentrations of PFOS, PFNA and PFDA in livers of seals collected in 2005 were respectively 2.4-, 1.2- and 1.7-fold greater than in seals collected in 1992 (Figure 3). Among the PFCs analyzed, concentrations of PFOS and PFDA in the liver of seals collected in 2005 were

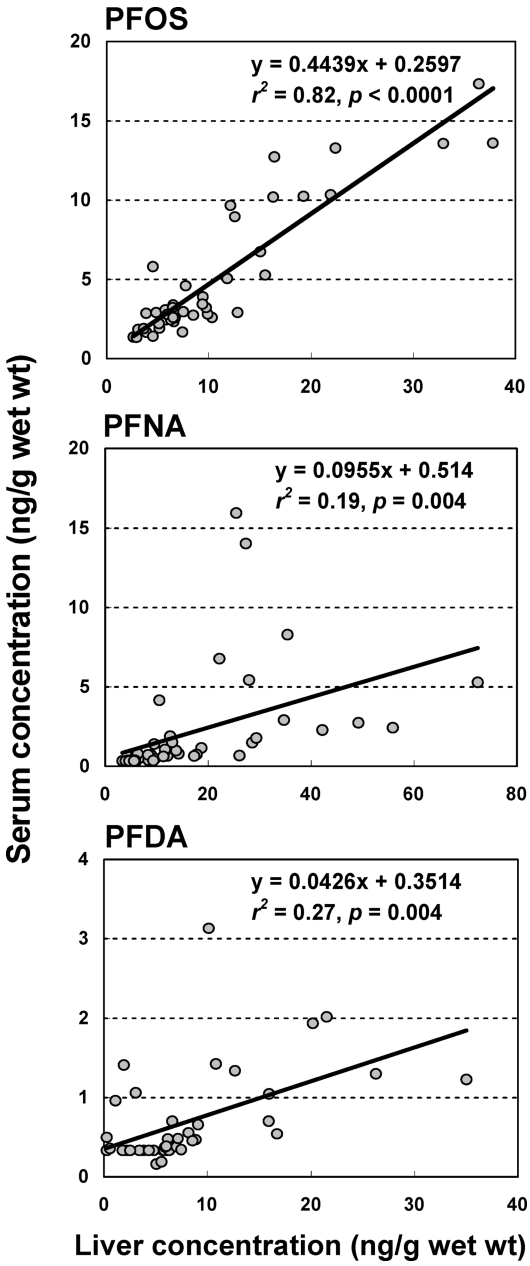

FIGURE 2. Relationship between liver (ng/g wet wt) and serum concentrations (ng/g wet wt), for PFOS, PFNA, and PFDA in Baikal seals collected in 2005.

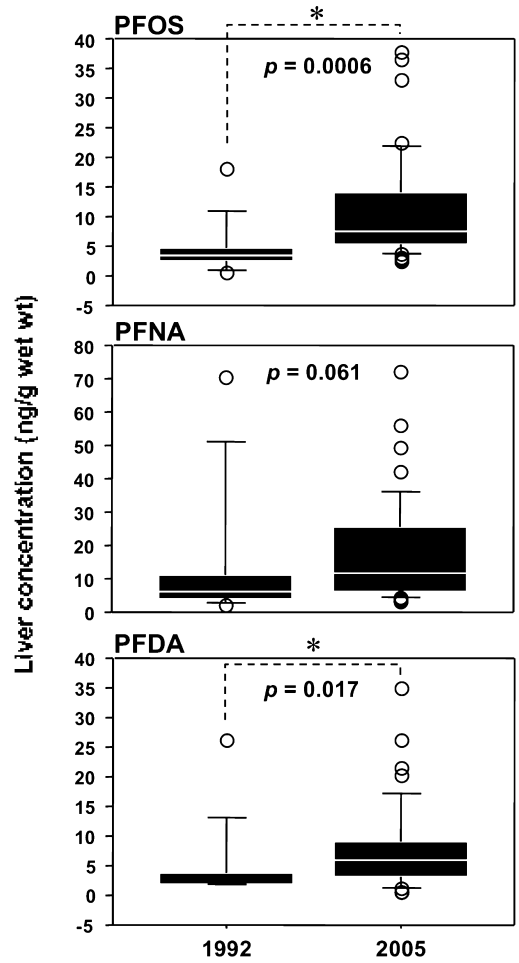

FIGURE 3. Concentrations (ng/g wet wt) of PFOS, PFNA, and PFDA in the livers of Baikal seals collected in 1992 (n = 11) and 2005 (n = 44). Box and whisker plots indicate percentiles (bottom whisker = 10th value, bottom of box = 25th, horizontal line inside box = 50th, top of box = 75th, top whisker = 90th. Open circles indicate outlier values). The asterisk (∗) denotes significant difference between the 1992 and 2005 livers (t-test, p < 0.05).

significantly higher than in seals collected in 1992 (PFOS: p = 0.0006, PFDA: p = 0.017). Furthermore, a trend of increasing PFNA concentration from 1992 to 2005 was found, although no significant differences were observed (p = 0.061). The relative composition of PFNA was significantly higher than that of individual PFCs in the 1992 and 2005 livers (p < 0.05); PFNA was the dominant PFC in both sample sets (Figure S6). This indicates that the source composition of PFCs in Lake Baikal has not been changed over the 13-year period. Several previous temporal trend studies on aquatic mammals have similarly shown increasing concentrations of PFCs. For example, ringed seals from Greenland showed significant increase in PFOS, PFDA, and PFUnDA concentrations from 1982 to 2003 (*8*). Another study demonstrated that PFCA (C9 to C11) concentrations in Canadian Arctic ringed seal livers showed a steady increase from the 1970s to 2000s; however, PFOS concentrations increased from 1992 to 1998, but decreased after 2003, indicating that the current PFOS concentrations in Canadian Arctic ringed seals are equivalent to 1992 levels (*9*). Similarly, sea otters from California coastal water showed increasing concentrations of several PFCs during 1992–2002, except for PFOS, which showed a trend of decrease after 2001 (*37*). These results suggest the geographical differences in temporal trends of PFCs contamination and/or the lack of temporal resolution in Lake Baikal. In our recent study, the concentrations of PCDDs and mono-ortho PCBs in livers of seal collected in 2005 showed no significant decrease, compared to the concentrations in seals collected in 1992, whereas significant decreases were observed for PCDFs and non-ortho PCBs (except PCB 77). This implies that sources of PCDDs and PCBs are still present around Lake Baikal (*38*). Given these results, continuous monitoring of PFCs as well as dioxin-like compounds in Baikal seals is necessary to assess potential biological effects of PFCs.

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Supporting Information Available

Additional tables and figures. This material is available free of charge via the Internet at http://pubs.acs.org.

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