Perfluorinated Compounds in the Environment and the Blood of Residents Living near Fluorochemical Plants in Fuxin, China

ARTICLE *in* ENVIRONMENTAL SCIENCE & TECHNOLOGY · OCTOBER 2010

Impact Factor: 5.33 · DOI: 10.1021/es102610x · Source: PubMed

8 AUTHORS, INCLUDING:

Jia Bao
United States Environmental Protection Age…
9 PUBLICATIONS **200** CITATIONS
SEE PROFILE

Wei Liu
Dalian University of Technology
50 PUBLICATIONS **822** CITATIONS
SEE PROFILE

Li Liu
Pfizer Inc.
59 PUBLICATIONS **456** CITATIONS
SEE PROFILE

Jiayin Dai
Chinese Academy of Sciences
94 PUBLICATIONS **1,902** CITATIONS
SEE PROFILE

Environmental Science & Technology

ARTICLE

pubs.acs.org/est

Perfluorinated Compounds in the Environment and the Blood of Residents Living near Fluorochemical Plants in Fuxin, China

Jia Bao,[†] Wei Liu,[†] Li Liu,[‡] Yihe Jin,[*,†] Jiayin Dai,[§] Xiaorong Ran,[⊥] Zhixu Zhang,[⊥] and Shuji Tsuda[∥]

[†]School of Environmental Science and Technology, Dalian University of Technology, Key Laboratory of Industrial Ecology and Environmental Engineering, Ministry of Education, Dalian 116024, China

[‡]Department of Social Medicine, School of Public Health, China Medical University, Shenyang 110001, China

[§]Key Laboratory of Animal Ecology and Conservation Biology, Institute of Zoology, Chinese Academy of Sciences, Beijing 100101, China

[⊥]Life Sciences and Chemical Analysis, Agilent Technologies Co., Ltd., Beijing 100102, China

[∥]Laboratory of Veterinary Public Health, Department of Veterinary Medicine, Faculty of Agriculture, Iwate University, Morioka 020-8550, Japan

Supporting Information

ABSTRACT: A fluorochemical industrial park was built in 2004 in Fuxin, China, for the production of polytetrafluoroethylene (PTFE) and perfluorobutane sulfonate (PFBS). Yet little is known about the distribution of fluorochemicals in the environment and in people living in and around the park. In this study, environmental samples were collected from 22 sites in Fuxin to investigate the extent of perfluorinated compound (PFC) contamination in the environment around the park, and in drinking water from the public water supply system and groundwater in shallow aquifers from private wells near the park. Serum samples were also collected from nonoccupationally exposed residents living in Fuxin to determine the PFC load of local residents. As the dominant contaminant of eight target PFCs, the maximum concentrations of perfluorooctanoic acid (PFOA) in sediment and river water of the River Xi along the industrial park were 48 ng/g dry weight and 668 ng/L, respectively; the highest PFOA concentration in groundwater beneath the park was 524 ng/L; and the PFOA levels in drinking water from the public water supply system ranged between 1.3 and 2.7 ng/L. In human serum, PFOA had the geometric mean at 4.3 ng/mL, ranging from 0.02 to 93 ng/mL. This study serves to document what should be the beginning of a long-term surveillance effort to minimize potential exposure of residents living in Fuxin.

INTRODUCTION

Perfluorinated compounds (PFCs) consisting of perfluorosulfonates (PFSAs) and perfluorocarboxylates (PFCAs) have been manufactured and employed as surfactants and surface protectors in carpets, leather, paper, packaging, fabric, and upholstery, and in or as aqueous film-forming foams (AFFFs), mining and oil well surfactants, alkaline cleaners, floor polishes, photographic film, denture cleaners, shampoos, and insecticide, since their initial commercialization over half a century ago.[1] It has become well-known that the extremely strong C−F bonds of PFCs contribute to their resistance to environmental and biological degradation, chemical and thermal stability, and both hydrophobicity and oleophobicity,[2] leading to their global distribution,[3] bioaccumulation, [4] various toxicities,[5] and potential for long-range transport.[6,42] As a result, one important PFC, perfluorooctane sulfonate (PFOS), was recently added to Annex B of the Stockholm Convention on Persistent Organic Pollutants (POPs), calling for restricted use worldwide.[7]

Research on the PFC load in nonoccupationally exposed populations in China has been carried out since 2006.[8−10,12] Jin et al.[9] found increasing trends in serum PFOS and perfluorooctanoic acid (PFOA) levels between the years of 1987 and 2002 in residents of Shenyang in Liaoning Province of northeast China, which is contrary to more recent trends in the U.S.,[11] likely due to the relatively short history of manufacture and application of PFCs within China when compared to other developed countries. Recently, another study on PFCs in human blood from seven cities of Liaoning Province showed that the highest level of blood PFOA in these cities with the geometric mean (GM) of 3.8 ng/mL was found in donors from Fuxin, which might relate to the fluorochemical plants located there.[10]

Fuxin is situated in northwest Liaoning Province (Figure 1) and has until recently been known for its coal mining industry. However, with declining coal reserves, the city has taken steps to develop the fluorochemical industry, due to the local abundance of the mineral fluorite (CaF_2). As a result, a 1.7 km^2 fluorochemical industrial park, containing several fluorochemical plants, has been built in a suburban area of Fuxin since 2004. This industrial park currently specializes in the manufacture of the potassium salt of perfluorobutane sulfonate (PFBS) and fluoropolymers, such as polytetrafluoroethylene (PTFE), with an annual production of 1700 tons of PTFE. Yet little is known about the impact of

Special Issue: Perfluoroalkyl Acid

Received: July 31, 2010
Accepted: October 8, 2010
Revised: October 6, 2010
Published: October 21, 2010

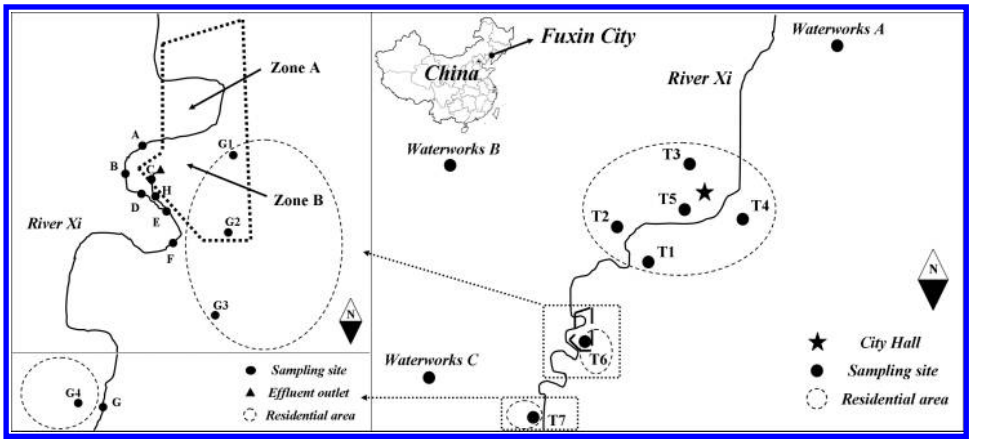

Figure 1. Sampling area and locations in Fuxin.

PFCs emissions on the environment and residents living near the park.

Previous studies have shown how PFOA emissions from PTFE production facilities can contaminate surrounding communities leading to significant human exposure issues.[13] PFOA contamination discharged or emitted from fluorochemical plants could pose potential threats to surrounding ecosystems due to contamination of food webs.[4] Human health risks that have been recently associated with PFOA exposure include elevated cholesterol, uric acid, and serum liver enzymes,[14−16] as well as thyroid disease.[17] Potential exposure pathways include a variety of routes including drinking water, house dust, and food.[18−20] The consumption of contaminated drinking water and groundwater has been determined to be an important human exposure route in PFCs-contaminated areas. In one well-known case in the U.S., severe PFOA contamination downwind and downstream from a fluoropolymer manufacturing facility in West Virginia led to PFOA levels in the blood of local residents that were 60−75 times higher than those in the general population.[21] As a result, in January 2009, the U.S. Environmental Protection Agency (EPA) issued provisional health advisories for PFOA and PFOS in drinking water at 400 and 200 ng/L, respectively, after several revisions.[22] In addition, health risk limits (HRLs) for groundwater from private wells were set at 300 ng/L for both PFOS and PFOA and 9000 ng/L for PFBS by the Minnesota Department of Health (MDH) in September 2008,[23] following the discovery of PFC contamination at fluorochemical manufacturing sites in the Minneapolis area.

The objectives of the present study were to (i) investigate the extent of PFC contamination in the environment around Fuxin fluorochemical industrial park, (ii) assess the safety of drinking water from the public water supply system of Fuxin and groundwater in shallow aquifers from private wells near the park, (iii) characterize the PFC load in nonoccupationally exposed residents living in this city, and (iv) determine to what extent consumption of drinking water from this region might be an important human exposure pathway. This is a baseline study to report concentrations of PFCs in environmental samples and humans near fluorochemical plants, and serves to document what should be the beginning of a long-term surveillance effort to minimize potential exposure of residents living in Fuxin.

EXPERIMENTAL SECTION

Water, sediment, and serum samples were prepared and extracted using methods described elsewhere,[24−26] and the extracts were analyzed via an Agilent 1100 high-performance liquid chromatograph (HPLC)-tandem an Agilent 6410 Triple Quadruple (QQQ) mass spectrometer (MS/MS) targeting eight PFCs analytes: PFBS, perfluorohexane sulfonate (PFHxS), PFOS, PFOA, perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA), perfluorododecanoic acid (PFDoA), and perfluorotetradecanoic acid (PFTA). Information about standards and reagents, environmental sample collection, sample preparation and extraction, instrumental analysis, quality assurance and quality control, and recovery experiments in the present study are detailed in the Supporting Information (SI).

Sample Collection. As Figure 1 shows, the sampling area for this study was focused on Fuxin (42°01′N, 121°39′E) of northeast China, which had an urban population of 0.78 million in the year of 2009. Geographically, Fuxin fluorochemical industrial park is situated in a suburban area of southwest Fuxin approximately 10 km from the city center. PFBS and PTFE manufacturing plants are located at Zones A and B in the park, respectively. In addition, the River Xi flows southwestward along the park, receiving wastewater discharged from two fluorochemical plants at Zones A and B.

In August 2009, sampling was conducted at 22 sites in Fuxin (Figure 1) including (i) river water and sediment samples from six sites (A−F) at sections of the River Xi that are close to the park as well as site G which is 5 km downstream, (ii) a sediment core sample from site H located in the effluent channel for Zone B which flows into the River Xi, (iii) groundwater samples from shallow aquifers at depths of 5−7 m from three private wells (G1−G3) in the vicinity of the park and in another private well (G4) that is 5 km away from site G1, (iv) raw and finished water samples from three waterworks (A−C) which serve the city, and (v) drinking water samples in the public water supply system from five locations in an urban area (T1−T5) and two suburban locations near the park (T6 and T7). Site T6 was located in the residential area that is closest to the park, while site T7 was situated in the one that is 5 km downstream from the park.

During November 2009, 120 serum samples were collected from nonoccupationally exposed Fuxin residents aged 0.1 through 87 years, consisting of 63 males and 57 females. All the serum samples were pooled into five groups according to ages of <15, 15−30, 30−45, 45−60, and >60 years, and stored in 2-mL methanol-rinsed PP vials at −20 °C until extraction. The sample collection was approved by the Medical Ethics Committee at China Medical University in China.

Modeling of Exposures Based upon Serum PFOA Concentrations. A simplified one-compartment pharmacokinetic

model has been successfully used to estimate the daily intake of PFC contaminants.[20,27,28] In the present study, the daily intake of PFOA by residents in Fuxin was estimated based upon serum PFOA concentrations via the equation employed by Zhang et al., Fromme et al., and Thompson et al.[20,27,28] The change in blood concentration (C_p) resulting from a given exposure dose (E) can be described by the following equation:

$$d(C_p)/dt = E - k \times V_d \times C_p$$

where V_d is the volume of distribution (mL/kg bw) and k is the first-order elimination per day = $0.693/t_{1/2}$, at steady-state conditions, where $d(C_p)/dt = 0$,

$$E = k \times V_d \times C_p$$

and

$$E = 0.693/t_{1/2} \times V_d \times C_p$$

In accordance with previous studies, median half-life of PFOA was 1257 days (3.4 years) with a range of 561−3334 days (1.5−9.1 years)[29] and volume of distribution for PFOA was 170 mL/kg bw.[28]

Statistical Analysis. Statistical analysis was performed with the software SPSS 16.0 (SPSS Inc., Chicago, IL). During the analysis, values of environmental and serum samples lower than the LOQ were set to one-half of the LOQ, and those of samples lower than the LOD were assigned as values of the LOD/$\sqrt{2}$.[11] The mean value of duplicate environmental samples was used in all analyses. Correlations between age and serum PFCs levels, differences of serum PFCs levels between genders and those among five age groups were assessed via Spearman rank correlation test and Mann−Whitney test.[30] Statistical correlations and differences were considered to be significant at $p < 0.05$.

RESULTS AND DISCUSSION

Results from the quality assurance and quality control procedures discussed above show no detectable concentrations of any PFCs in any of the travel, procedural, and solvent blanks prepared for this study. Recoveries of the target PFCs in environmental and serum samples were determined between 81% and 110%, and RSD for each target analyte in different matrixes was lower than 15% (Table S1, Supporting Information). Duplicate environmental field samples had RSDs of ≤15% for various matrices (Tables 1 and 2), indicating very acceptable precision for all steps in the analytical process.

In the present study, a total of eight target PFCs were monitored in all the environmental and serum samples. PFTA was lower than the LOD in all of the environmental samples and had a detection frequency of only 13% in the serum samples. As a result, concentrations of PFTA are not discussed in the following sections.

PFCs in the Environment around Fuxin Fluorochemical Industrial Park. Table 1 summarizes PFCs concentrations in the sediment, river water, and groundwater around Fuxin fluorochemical industrial park. In general, total concentrations of PFCs (ΣPFCs) in sediment from the River Xi ranged between 0.48 and 90 ng/g dry weight (dw). The highest sediment concentration of 90 ng/g dw ΣPFC appeared in site C, which is close to the effluent outlet for Zone B of the park. In addition, the second-ranked sediment ΣPFC with a concentration of 38 ng/g dw occurred at site E, which is situated in the river section connected to the effluent channel for Zone B (Figure 1). PFOA was the major PFC contaminant in sediment, contributing from 37% to 99% of the total. The other six PFCs were below the LOQs at most of sites in the river except site C, which had total long-chain PFCAs (C > 8) at 41 ng/g dw. Sites C and E had sediment PFOA concentrations of 48 and 38 ng/g dw, respectively, possibly due to PFOA discharged in the effluent from Zone B which is released into the river via an effluent channel. ΣPFCs in river water of the River Xi ranged from 370 to 713 ng/L. Similar to the sediment samples, the highest ΣPFC in river water came from sites C and E, with concentrations of 713 and 622 ng/L, respectively. Moreover, PFOA concentrations were 668 and 223 ng/L at sites C and E, respectively, from two to eight times higher than the other five sites, possibly stemming from PFOA in the effluents from Zone B as mentioned above. PFOA contributed 94% of all the target analytes at site C, which is close to the effluent outlet for Zone B. However, PFBS was the dominant PFC contaminant detected in river water at the other six sites,

Table 1. PFCs Concentrations in Sediment, River Water, and Groundwater Samples from Fuxin[a,b]

site	PFBS	PFHxS	PFOS	PFOA	PFNA	PFDA	PFDoA	ΣPFC
				sediment (ng/g dw)				
A	<0.20	n.d.	n.d.	0.29(3)	<0.20	<0.20	n.d.	0.59
B	<0.20	n.d.	n.d.	0.31(12)	<0.20	<0.20	n.d.	0.61
C	0.72(6)	n.d.	n.d.	48(6)	19(7)	12(15)	10(11)	90
D	<0.20	n.d.	n.d.	0.62(11)	<0.20	<0.20	n.d.	0.92
E	<0.20	n.d.	n.d.	38(3)	<0.20	<0.20	n.d.	38
F	<0.20	n.d.	n.d.	0.61(5)	<0.20	<0.20	n.d.	0.91
G	<0.20	n.d.	n.d.	0.18(8)	<0.20	<0.20	n.d.	0.48
				river water (ng/L)				
A	445(2)	0.58(1)	0.54(8)	27.2(6)	0.46(2)	<0.10	n.d.	474
B	426(1)	0.32(6)	0.33(7)	33.4(4)	0.43(1)	<0.10	n.d.	461
C	7.77(7)	0.22(2)	0.45(6)	668(5)	16(1)	21(1)	<0.20	713
D	353(6)	0.23(3)	0.33(5)	80.8(2)	0.48(4)	<0.10	n.d.	435
E	397(1)	0.18(6)	0.28(4)	223(7)	0.49(4)	<0.10	n.d.	622
F	313(5)	0.21(1)	0.53(7)	55.3(4)	0.59(9)	<0.10	n.d.	370
G	297(4)	0.15(8)	0.50(2)	95.6(2)	0.51(5)	<0.10	n.d.	394
				groundwater (ng/L)				
G1	872(5)	0.68(5)	0.73(7)	524(7)	0.47(7)	n.d.	n.d.	1400
G2	63.8(1)	<0.10	<0.10	434(5)	0.18(8)	n.d.	n.d.	498
G3	5.05(7)	<0.10	<0.10	31.6(5)	0.07(5)	n.d.	n.d.	36.8
G4	1.19(9)	n.d.	n.d.	4.85(6)	n.d.	n.d.	n.d.	6.03

[a] Values in parentheses were %RSD ($n = 2$). [b] n.d. = not detected.

Table 2. PFCs Concentrations in Raw Water, Finished Water, and Drinking Water Samples from Fuxin (ng/L)[a,b]

site	PFBS	PFHxS	PFOS	PFOA	PFNA	PFDA	PFDoA	ΣPFC
				raw water				
A	0.51(2)	0.10(2)	0.15(6)	5.0(4)	0.70(7)	<0.10	n.d.	6.5
B	0.22(9)	0.14(1)	0.14(4)	4.0(3)	0.14(3)	n.d.	n.d.	4.7
C	0.67(9)	<0.10	0.30(7)	16(6)	1.6(8)	<0.10	n.d.	18
				finished water				
A	0.57(1)	n.d.	0.10(1)	2.7(2)	0.49(3)	n.d.	n.d.	3.8
B	<0.10	n.d.	n.d.	0.48(5)	<0.10	n.d.	n.d.	0.58
C	0.50(2)	n.d.	0.15(6)	2.7(4)	0.53(7)	n.d.	n.d.	3.8
				drinking water				
T1	0.56(1)	n.d.	n.d.	1.6(4)	0.29(7)	n.d.	n.d.	2.4
T2	0.53(3)	n.d.	<0.10	1.6(1)	0.29(9)	n.d.	n.d.	2.5
T3	0.64(6)	n.d.	<0.10	1.6(2)	0.30(4)	n.d.	n.d.	2.6
T4	0.53(2)	n.d.	<0.10	1.7(5)	0.34(7)	n.d.	n.d.	2.7
T5	<0.10	n.d.	n.d.	0.30(4)	n.d.	n.d.	n.d.	0.35
T6	0.43(1)	n.d.	<0.10	1.2(7)	0.31(4)	n.d.	n.d.	2.0
T7	0.52(7)	n.d.	n.d.	0.56(6)	0.19(7)	n.d.	n.d.	1.3

[a] Values in parentheses were %RSD ($n = 2$). [b] n.d. = not detected.

contributing 64−94% of the total PFCs for analysis. Considering the flow of the river indicated in Figure 1, it appears that PFBS was discharged from Zone A leading to a decreasing trend in concentration as the river proceeds downstream from this area (Table 1). The sediment PFOA levels in the vicinity of the industrial park, which ranged between 0.18 and 48 ng/g dw, were higher than those have been determined in other regions within China (0.09−0.64 ng/g dw).[25,31] In addition, PFOA levels in the River Xi along the park, ranging from 27.2 to 668 ng/L, were also higher than measurements that have been made in other rivers in China (0.2−298 ng/L).[32]

In the analysis of the sectioned sediment core collected from site H, a decreasing trend in sediment ΣPFCs with depth was observed in this sample, collected from the effluent channel for Zone B of the park, which is consistent with what has been reported in previous studies.[25,31] PFOA was the only target contaminant measured in the sediment core at this site, with the PFOA concentration of 31 ng/g dw in the first slice of 0−2 cm being 100 times higher than 0.31 ng/g dw measured in the seventh slice at 12−14 cm (Figure S1, SI). This might suggest that PFOA contamination has been increasing in this area over the past few years.

ΣPFCs in groundwater beneath the park were determined to be between 6.03 and 1400 ng/L, with a notable decreasing trend in groundwater ΣPFCs from site G1 to site G4 (Table 1), which is consistent with the southwestward flow of groundwater in this area toward the River Xi. Site G1, which is located in Zone B, showed the highest concentrations with PFBS and PFOA at 872 and 524 ng/L, respectively. PFBS was the dominant PFC contaminant at this site, contributing 62% of the total PFCs. In contrast, PFOA was the main contaminant in groundwater underneath sites G2−G4, contributing 80−87% of all the target analytes. Geographically, private well G1 is located in the center of the park, and the other three private wells are 0.5, 1, and 5 km distant from site G1 (Figure 1). The concentrations of each PFC in groundwater from these four private wells could be used to model dispersion trends of PFCs in groundwater in the vicinity of the park. Based upon the HRLs for PFBS and PFOA from MDH of 9000 and 300 ng/L, respectively, it might be inferred that the high levels of PFBS up to 872 ng/L in groundwater beneath the park do not represent a significant hazard, however, considerable PFOA contamination with the maximum concentration of 524 ng/L in these same locations suggests that the groundwater is unsuitable for consumption until at least 0.7 km away from the center in the park.

PFCs in the Public Water Supply System of Fuxin. As indicated in Table 2, ΣPFCs in raw source water for the three waterworks serving Fuxin ranged from 4.7 to 18 ng/L, while the finished water samples all decreased to levels between 0.58 and 3.8 ng/L. PFOA was the major PFC contaminant in both of raw and finished water, contributing 77−86% and 69−83% of the total PFCs loading in the raw and finished water, respectively. Overall, reservoir water was the raw water source for waterworks A, while groundwater was the water source for waterworks B and C. Waterworks C had the highest PFOA concentration of raw water up to 16 ng/L, which was nearly four times higher than those in waterworks A and B. However, PFOA levels in finished water were reduced to the range of 0.48 and 2.7 ng/L following the treatment. Because drinking water in the public water supply system of Fuxin was a mixture of finished water from several waterworks, ΣPFCs in drinking water from the water supply system were diminished further to levels between 1.3 and 2.7 ng/L at six of the seven sampling locations. Interestingly, site T5, which is a hotel in city center that uses an additional activated carbon filtration system for drinking water, had the lowest ΣPFCs in drinking water with a low concentration of 0.35 ng/L. PFOA was the principal PFC contaminant in all locations with concentrations ranging between 0.56 and 1.7 ng/L everywhere but site T5, which was only 0.30 ng/L.

Remarkably, the distance between the center of the industrial park and the boreholes for waterworks C is only 10 km. Given that PFOA levels are nearly four times higher in the raw water at waterworks C compared to that at the other two waterworks, it suggests that PFOA contamination from the aquifer beneath the park might be contributing to low level contamination observed at waterworks C. However, it is interesting to note that PFOA in the drinking water of Fuxin had a median concentration of 1.6 ng/L, which is one tenth that of the raw water at waterworks C, suggesting that waterworks treatment and mixture with other water from the public water supply system leads to a substantial reduction in concentration. It therefore appears that the efficiency of PFOA removal in waterworks C was much higher than that determined in Osaka, Japan[33] but further studies would be required to confirm this situation in Fuxin.

According to previous research on PFOA concentrations in drinking water from various areas within China, PFOA levels in drinking water of Fuxin with a median of 1.6 ng/L are marginally higher than those in most of Chinese cities, but much lower than those in Guangzhou, Shenzhen, and Shanghai with concentrations of 12, 46, and 78 ng/L, respectively.[32,34] This might be attributable to the highly developed economies of these specific areas and the presence of more and different sources of PFOA compared to other regions of China. Clearly, the drinking water in Fuxin is considerably lower in PFOA than the Little Hocking region in the U.S. which had an average level of 3550 ng/L during 2002−2005.[21] At present, the PFOA level in drinking water from the public water supply system of Fuxin appears to be much lower than the provisional health advisories for PFOA in drinking water at 400 ng/L issued by the U.S. EPA mentioned earlier, although the groundwater of this region is threatened by PFOA contamination.

PFCs in Serum Samples from Fuxin Residents. Table S2 (SI) summarizes the PFC in human serum data, with the overall frequency of detection being as follows: PFBS (93%), PFHxS (93%), PFOS (94%), PFOA (96%), PFNA (100%), PFDA (94%), PFDoA (81%), and PFTA at only 13%. Serum concentrations are further broken down by age group category and gender in Table S2 (SI). PFOA had the highest GM at 4.3 ng/mL, ranging from 0.02 to 93 ng/mL. PFOS had the GM of 0.65 ng/mL in the range of 0.01 to 33 ng/mL. PFOA and PFOS contributed 61% and 20% of all the PFCs analytes, while the other five analytes each contributed less than 10%. Statistically, all the PFCs analytes showed good mutual correlations (Table S3, SI), suggesting a similar source of exposure in this region.

A significant positive correlation was observed between age and serum concentrations of PFHxS, PFOS, and PFOA with males, wheras with females only PFHxS was significantly correlated with age. Although there were marginal distinctions between GMs of each PFC in both genders, no statistical differences were found between serum concentrations of each compound in male and female populations. However, differences among serum concentrations of PFOA, PFBS, PFHxS, and PFOS in five age groups are evident as illustrated in Figure S2 (SI). For example, serum PFOA concentrations in the 15−30 year age group were much lower than those in adults >45 years (p = 0.016 and 0.035). Significantly higher

serum concentrations of PFBS were observed in children ages <15 years compared to those in adults ages 15−60 years (p = 0.009, 0.008, and 0.050). In addition, serum concentrations of PFHxS in the age groups between 15 and 45 years were significantly lower than those ages >60 years (p = 0.045 and 0.025). Similarly, considerably lower serum PFOS levels were found in the age classes between 15 and 45 years when compared to those >60 years (p = 0.008 and 0.008) as well as 45−60 years (p = 0.014 and 0.025). Although no statistical differences were determined between serum PFOA, PFHxS, and PFOS concentrations in children ages <15 years and adults ages 15−30 years, serum levels of these three PFCs showed J-shaped curves with growing age of total population. Furthermore, serum PFOA concentrations of females were found much higher than those of males in children ages <15 years (p = 0.015); on the contrary, serum levels of PFHxS in males ages 45−60 years were observed with significantly elevated concentrations compared to those in females with the same ages (p = 0.005). The J-shaped relationships between age and serum PFOA, PFHxS, and PFOS concentrations were similar to those found previously.[13,35] The differences between serum levels of these three PFCs in children and adults with ages <15 and 15−30 years, respectively, might be attributable to high exposures in infants and toddlers via breast milk and dust ingestion.[20,36] Moreover, the considerable variation between serum PFBS levels in children <15 years and adults 15−60 years, could result from faster serum elimination rate of PFBS in human bodies than those of long-chain PFCs.[37]

Following the conversion of PFC concentration in the whole blood into serum PFC concentration by multiplying by a factor of 2,[38] the median serum PFOA concentration of residents in Fuxin (5.5 ng/mL) was found to be comparable to medians measured in Colombia and Japan (6.2−6.4 ng/mL), higher than those of Italy, Belgium, and India (2.6−4.8 ng/mL), but much lower than those in the U.S., Poland, and Korea (19−62 ng/mL).[38] As economic conditions evolve in China, it will be interesting to observe whether concentrations increase and become more similar to those of the more developed regions of the world. In a previous study in Liaoning Province in 2008, Liu et al.[10] found the GM serum level for PFOA in six other cities to be 0.88−4.9 ng/mL, with the GM of Fuxin residents to be 7.6 ng/mL, which is higher than that of Fuxin residents from the present study during 2009 (4.3 ng/mL). Moreover, the GM level for serum PFOS in Fuxin in the 2008 study was 13 ng/mL, being approximately 20 times higher than that observed in the present study during 2009 (0.65 ng/mL). The reasons for these variations remain unknown but should be the subject for further investigation.

Modeling of Daily Intake of PFOA by Fuxin Residents. We calculate that the daily intake of PFOA by Fuxin residents through drinking water is 0.05 ng/kg bw/day, assuming an average body weight of 60 kg[39] and intake of 2 L of drinking water per day. Based upon the simplified one-compartment pharmacokinetic model, the total daily intake of PFOA from all sources of exposure ranged from 0.15 to 0.90 ng/kg bw/day, with a median of 0.40 ng/kg bw/day, for individuals living in Fuxin can be achieved from the GM of serum PFOA concentration observed in this study. Therefore, consumption of drinking water in Fuxin appears to contribute 6−33% of overall exposure, with a median of 13%, which is very similar to the value of 16% estimated for the common European population.[40] In a worst case scenario, if Fuxin residents consumed the most contaminated well water directly, we calculate resulting PFOA serum levels would be in a range of 81.0−481 ng/mL, with a median of 186 ng/mL. Although residents within the industrial park preferred to consume water from the municipal service rather than their own private wells, it is important to note that groundwater irrigation is commonly used for growing vegetables in this area. Uptake by plants and contamination of agricultural products has been hypothesized as being a potential source of human PFC exposure, but research in this field is still very preliminary.[35,41]

In summary, PFOA contamination has been observed in the groundwater beneath Fuxin fluorochemical industrial park, posing potential risks to the health of residents living in this area. PFOA levels in the public drinking water supply system of Fuxin were determined to considerably lower than previously published health advisory levels established elsewhere, with our model indicating that drinking water is likely to contribute approximately 13% of the daily exposure in Fuxin. However, in light of the growing domestic and overseas demands for perfluorinated materials and the industries that have developed locally to meet this need, further long-term monitoring in Fuxin should be conducted on the prospective impact of PFOA contamination on the residents living in this city.

■ ASSOCIATED CONTENT

Supporting Information. Additional information, tables, and figures as noted in text. This material is available free of charge via the Internet at http://pubs.acs.org.

■ AUTHOR INFORMATION

Corresponding Author
*Phone/fax: +86 411 84708084. E-mail: jinyihe@dlut.edu.cn.

■ ACKNOWLEDGMENT

We are grateful to Dr. Andrew B. Lindstrom, National Exposure Research Laboratory, U.S. Environmental Protection Agency, for his valuable comments. This research is funded by the National Nature Science Foundation of China (20837004) and also supported by the Program for Changjiang Scholars and Innovative Research Team in University (IRT0813).

■ REFERENCES

(1) OECD. *Hazard assessment of perfluorooctane sulfonate (PFOS) and its salts*, 2002.

(2) Kissa, E. *Fluorinated Surfactants and Repellents*, 2nd ed; Marcel Dekker: New York, 2001.

(3) Giesy, J. P.; Kannan, K. Global distribution of perfluorooctane sulfonate in wildlife. *Environ. Sci. Technol.* **2001**, *35* (7), 1339–1342.

(4) 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* (20), 5379–5385.

(5) Lau, C.; Butenhoff, J.; Rogers, J. M. The developmental toxicity of perfluoroalkyl acids and their derivatives. *Toxicol. Appl. Pharmacol.* **2004**, *198* (2), 231–241.

(6) Prevedouros, K.; Cousins, I. T.; Buck, R. C.; Korzeniowski, S. H. Sources, fate and transport of perfluorocarboxylates. *Environ. Sci. Technol.* **2006**, *40* (1), 32–44.

(7) UNEP. Stockholm Convention on Persistent Organic Pollutants (POPs), 2009; http://chm.pops.int/Portals/0/Repository/convention_text/UNEP-POPS-COP-CONVTEXT-FULL.English. PDF.

(8) Yeung, L. W. Y.; So, M. K.; Jiang, G.; Taniyasu, S.; Yamashita, N.; Song, M.; Wu, Y.; Li, J.; Giesy, J. P.; Guruge, K. S.; Lam, P. K. S. Perfluorooctanesulfonate and related fluorochemicals in human blood samples from China. *Environ. Sci. Technol.* **2006**, *40* (3), 715–720.

(9) Jin, Y.; Saito, N.; Harada, K.; Inoue, K.; Koizumi, A. Historical trends in human serum levels of perfluorooctanoate and perfluorooctane sulfonate in Shenyang, China. *Tohoku J. Exp. Med.* **2007**, *212* (1), 63–70.

(10) Liu, J.; Li, J.; Luan, Y.; Zhao, Y.; Wu, Y. Geographical distribution of perfluorinated compounds in human blood from Liaoning Province, China. *Environ. Sci. Technol.* **2009**, *43* (11), 4044–4048.

(11) Calafat, A. M.; Wong, L. Y.; Kuklenyik, Z.; Reidy, J. A.; Needham, L. L. Polyfluoroalkyl chemicals in the U.S. population: Data from the National Health and Nutrition Examination Survey (NHANES) 2003−2004 and comparisons with NHANES 1999−2000. *Environ. Health. Perspect.* **2007**, *115* (11), 1596–1602.

(12) Zhang, T.; Wu, Q.; Sun, H. W.; Zhang, X. Z.; Yun, S. H.; Kannan, K. Perfluorinated compounds in whole blood samples from infants, children, and adults in China. *Environ. Sci. Technol.* **2010**, *44* (11), 4341–4347.

(13) Emmett, E. A.; Zhang, H.; Shofer, F. S.; Freeman, D.; Rodway, N. V.; Desai, C.; Shaw, L. M. Community exposure to Perfluorooctanoate: Relationships between serum levels and certain health parameters. *J. Occup. Environ. Med.* **2006**, *48* (8), 771–779.

(14) Nelson, W.; Hatch, E. E.; Webster, T. F. Exposure to polyfluoroalkyl chemicals and cholesterol, body weight, and insulin resistance in the general U.S. population. *Environ. Health. Perspect.* **2010**, *118* (2), 197–202.

(15) Steenland, K.; Tinker, S.; Shankar, A.; Ducatman, A. Association of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) with uric acid among adults with elevated community exposure to PFOA. *Environ. Health. Perspect* **2010**, *118* (2), 229–233.

(16) Gilliland, F. D.; Mandel, J. S. Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins, cholesterol: A study of occupationally exposed men. *Am. J. Ind. Med.* **1996**, *29* (5), 560–568.

(17) Melzer, D.; Rice, N.; Depledge, M. H.; Henley, W. E.; Galloway, T. S. Association between serum perfluorooctanoic acid (PFOA) and thyroid disease in the NHANES study. *Environ. Health. Perspect.* **2010**No. doi: 10.1289/ehp.0901584.

(18) Hölzer, J.; Midasch, O.; Rauchfuss, K.; Kraft, M.; Reupert, R.; Angerer, J.; Kleeschulte, P.; Marschall, N.; Wilhelm, M. Biomonitoring of Perfluorinated Compounds in Children and Adults Exposed to Perfluorooctanoate-Contaminated Drinking Water. *Environ. Health. Perspect.* **2008**, *116* (5), 651–657.

(19) Strynar, M. J.; Lindstrom, A. B. Perfluorinated compounds in house dust from Ohio and North Carolina, USA. *Environ. Sci. Technol.* **2008**, *42* (10), 3751–3756.

(20) Zhang, T.; Sun, H. W.; Wu, Q.; Zhang, X. Z.; Yun, S. H.; Kannan, K. Perfluorochemicals in meat, eggs and indoor dust in China: Assessment of sources and pathways of human exposure to perfluorochemicals. *Environ. Sci. Technol.* **2010**, *44* (9), 3572–3579.

(21) Rumsby, P. C.; McLaughlin, C. L.; Hall, T. Perflurooctane sulphonate and perfluorooctanoic acid in drinking and environmental waters. *Philos. Trans., R. Soc. A* **2009**, *367* (1904), 4119–4136.

(22) U.S. Environmental Protection Agency. Provisional Health Advisories for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS), 2009; http://www.epa.gov/waterscience/criteria/drinking/pha-PFOA_PFOS.pdf.

(23) Minnesota Department of Health. Health Risk Limits for Groundwater 2008, 2009; http://www.health.state.mn.us/divs/eh/risk/guidance/gw/index.html.

(24) Saito, N.; Harada, K.; Inoue, K.; Sasaki, K.; Yoshinaga, T.; Koizumi, A. Perfluorooctanoate and perfluorooctane sulfonate concentrations in surface water in Japan. *J. Occup. Health* **2004**, *46* (1), 49–59.

(25) Bao, J.; Jin, Y.; Liu, W.; Ran, X.; Zhang, Z. Perfluorinated compounds in sediments from the Daliao River system of northeast China. *Chemosphere* **2009**, *77* (5), 652–657.

(26) Hansen, K. J.; Clemen, L. A.; Ellefson, M. E.; Johnson, H. O. Compound-specific, quantitative characterization of organic fluorochemicals in biological matrices. *Environ. Sci. Technol.* **2001**, *35* (4), 766–770.

(27) Fromme, H.; Schlummer, M.; Moller, A.; Gruber, L.; Wolz, G.; Ungewiss, J.; Bohmer, S.; Dekant, W.; Mayer, R.; Liebl, B.; Twardella, D. Exposure of an adult population to perfluorinated substances using duplicate diet portions and biomonitoring data. *Environ. Sci. Technol.* **2007**, *41* (22), 7928–7933.

(28) Thompson, J.; Lorber, M.; Toms, L. M. L.; Kato, K.; Calafat, A. M.; Mueller, J. F. Use of simple pharmacokinetic modelling to characterize exposure of Australians to perfluorooctanoic acid and perfluorooctane sulfonic acid. *Environ. Int.* **2010**, *36* (4), 390–397.

(29) Olsen, G. W.; Burris, J. M.; Ehresman, D. J.; Froehlich, J. W.; Seacat, A. M.; Butenhoff, J. L.; Zobel, L. R. Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. *Environ. Health. Perspect.* **2007**, *115* (9), 1298–1305.

(30) Kärrman, A.; Mueller, J. F.; Van Bavel, B.; Harden, F.; Toms, L. M. L.; Lindstrom, G. Levels of 12 perfluorinated chemicals in pooled Australian serum, collected 2002−2003, in relation to age, gender, and region. *Environ. Sci. Technol.* **2006**, *40* (12), 3742–3748.

(31) Bao, J.; Liu, W.; Liu, L.; Jin, Y.; Ran, X.; Zhang, Z. Perfluorinated compounds in urban river sediments from Guangzhou and Shanghai of China. *Chemosphere* **2010**, *80* (2), 123–130.

(32) Jin, Y.; Liu, W.; Sato, I.; Nakayama, S. F.; Sasaki, K.; Saito, N.; Tsuda, S. PFOS and PFOA in environmental and tap water in China. *Chemosphere* **2009**, *77* (5), 605–611.

(33) Takagi, S.; Adachi, F.; Miyano, K.; Koizumi, Y.; Tanaka, H.; Mimura, M.; Watanabe, I.; Tanabe, S.; Kannan, K. Perfluorooctanesulfonate and perfluorooctanoate in raw and treated tap water from Osaka, Japan. *Chemosphere* **2008**, *72* (10), 1409–1412.

(34) Mak, Y. L.; Taniyasu, S.; Yeung, L. W. Y.; Lu, G.; Jin, L.; Yang, Y.; Lam, P. K. S.; Kannan, K.; Yamashita, N. Perfluorinated compounds in tap water from China and several other countries. *Environ. Sci. Technol.* **2009**, *43* (13), 4824–4829.

(35) Steenland, K.; Jin, C.; MacNeil, J.; Lally, C.; Ducatman, A.; Vieira, V.; Fletcher, T. Predictors of PFOA levels in a community surrounding a chemical plant. *Environ. Health. Perspect.* **2009**, *117* (7), 1083–1088.

(36) Toms, L. M. L.; Calafat, A. M.; Kato, K.; Thompson, J.; Harden, F.; Hobson, P.; Sjödin, A.; Mueller, J. F. Polyfluoroalkyl chemicals in pooled blood serum from infants, children, and adults in Australia. *Environ. Sci. Technol.* **2009**, *43* (11), 4194–4199.

(37) Olsen, G. W.; Chang, S. C.; Noker, P. E.; Gorman, G. S.; Ehresman, D. J.; Lieder, P. H.; Butenhoff, J. L. A comparison of the pharmacokinetics of perfluorobutanesulfonate (PFBS) in rats, monkeys, and humans. *Toxicology* **2009**, *256* (1−2), 65–74.

(38) Kannan, K.; Corsolini, S.; Falandysz, J.; Fillman, G.; Kumar, K. S.; Loganathan, B. G.; Mohd, M. A.; Olivero, J.; Van Wouwe, N.; Yang, J. H.; Aldous, K. M. Perfluorooctanesulfonate and Related Fluorochemicals in Human Blood from Several Countries. *Environ. Sci. Technol.* **2004**, *38* (17), 4489–4495.

(39) General Administration of Sport of China. Bulletin of the Second National Fitness Survey of China, 2005; http://www.sport.gov.cn/n16/n1077/n1467/n1587/616932.html.

(40) Europe Food Safety Authority. Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and their salts: Scientific opinion of the panel on contaminants in the food chain. *EFSA J.* **2008**, *653*, 1–131.

(41) Stahl, T.; Heyn, J.; Thiele, H.; Huther, J.; Failing, K.; Georgii, S.; Brunn, H. Carryover of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) from soil to plants. *Arch. Environ. Contam. Toxicol.* **2009**, *57* (2), 289–298.

(42) Yamashita, N.; Taniyasu, S.; Petrick, G.; Wei, S.; Gamo, T.; Lam, P. K. S.; Kannan, K. Perfluorinated acids as novel chemical tracers of global circulation of ocean waters. *Chemosphere* **2008**, *70* (7), 1247–1255.