Serum levels of perfluorinated compounds in the general population in Shenzhen, China

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Perfluorinated compounds (PFCs) have been detected in many environmental matrices, biota, and nonoccupationally exposed populations in China recently. However, little is known about the distribution and levels of various PFCs in the general population living in areas where there is PFC exposure. In the present study, the levels and prevalence of ten target PFCs were determined in 227 serum samples from a population of nonoccupationally exposed individuals in Shenzhen, China. Results indicated that human exposure to PFCs was prevalent in Shenzhen. Perfluorooctanoate (PFOA) was the dominant PFC contaminant in the serum samples, with a median concentration of 6.72 ng/mL, followed by perfluorooctane sulfonate (PFOS) with a median concentration of 2.07 ng/mL. Other PFCs were detected at much lower concentrations, with median concentrations ranging from 0.02 to 0.87 ng/mL. Statistically, no significant (P>0.05) gender differences were observed for any of the PFCs. Significant (P<0.01) positive correlations were found between age and serum concentrations of the target PFCs, except for perfluorobutane sulfonate (R = -0.16, P = 0.01), perfluorohexanoic acid (R = 0.08, P = 0.22), and perfluoroheptanoic acid (R = -0.11, P = 0.10). Based on the one-compartment pharmacokinetic model, the total daily intakes of PFOA and PFOS for the general population in Shenzhen were calculated as 0.63 and 0.20 ng/kg body weight/day, respectively.

Since the 1950s, perfluorinated compounds (PFCs) have been produced and used in multiple industrial and consumer products, such as protective coatings for fabrics and carpet, paper coatings, paints, adhesives, cosmetics, pharmaceuticals, insecticide formulations, fire retardants and surfactants [1]. PFCs have unique physicochemical characteristics such as thermal and chemical stability, amphiphilicity, low surface free energy and surface active properties [2]. The most commonly studied PFCs are perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA), and these two compounds have been detected in various environmental matrices and biota, as well as occupationally and nonoccupationally exposed populations around the world. Because of their global distribution [3], bioaccumulation [4], toxicity [5], and the potential for transportation into remote regions, these compounds have received much attention internationally [6]. As a direct consequence, 3M (St. Paul, MN), which is the primary global manufacturer of fluorochemicals, announced in 2000 that they would phase out the production of PFOS and PFOS-based products by the end of 2002 [7]. Subsequently, the U.S. [8], the European Union [9] and Canada [10] established new rules and guidelines to regulate the production and use of PFOS and its related substances. Recently, PFOS was added to Annex B of the...
Previous studies on PFCs in China have mainly focused on water bodies [12,13], sediment [14], precipitation [15], biota [16] and nonoccupationally exposed populations [17,18]. Until now, there has been limited information available on PFCs in the general population in China to PFCs, and especially on the distribution and levels of various PFCs in the general population living in areas of China where there is exposure to PFCs.

The potential exposure sources and pathways of PFCs for the general population include through food [19], drinking water [20,21], dust, and indoor/outdoor air [22]. Dietary intake is regarded as the most important exposure pathway for the general population [23]. Drinking water is also an important source of exposure to PFCs contamination, and many studies have investigated PFCs levels in populations resulting from contaminated drinking water [20,21].

One study from Germany found that the concentrations of PFCs in plasma from children and adults exposed to PFCs increased 4- to 8-fold in comparison with the controls because of consumption of contaminated drinking water [20]. Similar results were also obtained in the U.S. [21]. In view of the safety of drinking water, the U.S. Environmental Protection Agency issued provisional health advisories for PFOA and PFOS in drinking water at 400 and 200 ng/L, respectively, in 2009 [24]. To our knowledge, no regulatory drinking water threshold values have been recommended for PFCs in China. A previous study found the drinking water in Shenzhen of China contained elevated levels of PFOS (mean 11 ng/L), which were at least 2- to 420-fold higher than those found in the other areas of China [25]. The median level of PFOA in the drinking water exceeded 40 ng/mL [26], which might have adverse effects on the health of consumers in this PFC-exposed area [27].

Shenzhen, the first Chinese Special Economic Zone, is located in the Pearl River Delta of South China, which has experienced extremely rapid economic development over the past few decades. In 2009, this area was ranked fourth in China for total GDP, and ranked first for GDP per capita. There are a wide range of industrial activities in this area that could use PFCs, such as production of electronic/electric equipment and machinery, petrochemicals, pharmaceuticals, plastic goods and textiles. These activities could potentially release of PFCs into the environment. Some studies of surface water, drinking water, and sediment in Shenzhen have found higher levels of PFCs than in other areas of China [12,14,25,26]. However, information on the PFCs load in the human and related health risks in this heavily contaminated area is limited.

In the present study, the levels and prevalence of ten PFCs were determined in serum samples from the nonoccupationally exposed population in Shenzhen of China. The aims of this study were to evaluate the PFCs background levels that the local general population are exposed to, assess gender- and age-related differences in the serum PFCs concentrations, and estimate if drinking water could be an important exposure pathway for PFCs in Shenzhen. This data could be used for a human health risk assessment for the general population living in PFC-contaminated areas in China.

1 Materials and methods

1.1 Sample collection

In collaboration with the Shenzhen Center for Disease Control, 227 serum samples were collected in 2009 by venipuncture from nonoccupationally exposed individuals in Shenzhen, China. The participants were 0.3 to 90 years old, and all except for the infants and toddlers had lived in Shenzhen for >5 years. There were 133 males (mean age 32±30 years) and 94 females (mean age 38±31 years), and there was no significant difference (P>0.05) between their ages. Samples were void of personal identifiers, and the known demographic factors such as age, gender, sampling location, and date of collection. Serum samples were stored in methanol-rinsed polypropylene containers at –20°C.

1.2 Reagents and chemicals

Potassium PFOS (purity 98%) was purchased from Fluka (Steinheim, Germany). Potassium perfluorobutane sulfonate (PFBS, purity 98%), undecafluorohexanoic acid (PFHxA, purity 98%), trihexfluorheptanoic acid (PFHpA, purity 98%), and heptadecafluoropelargonic acid (PFNA, purity 95%) were purchased from Tokyo Chemical Industry (Tokyo, Japan). Potassium perfluorohexane sulfonate (PFHxS, purity 98%) was acquired from Merck (Darmstadt, Germany). Pentadecafluorooctanoic acid (PFOA, purity 95%) was obtained from Wako Pure Chemical Industries (Osaka, Japan). Nonadecafluorodecanoic acid (PFDA, purity 96%) and perfluorododecanoic acid (PFDoA, purity 97%) were acquired from Acros Organics (Geel, Belgium). High-performance liquid chromatography (HPLC) grade tetraethylammonium hydrogen sulfate and anhydrous extra pure sodium carbonate (Na2CO3, purity 99.5%) were obtained from Acros Organics (Geel, Belgium). HPLC grade ammonium acetate was obtained from Dikma Technology (Richmond, VA, USA). HPLC grade methyl tert-butyl ether (MTBE), methanol, and acetonitrile were obtained from Tedia (Fairfield, OH). Milli-Q water was cleaned using Waters Oasis HLB Plus cartridges (Milford, MA, USA) to remove potential PFCs residues. A mixed stock standard solution of PFCs was prepared in methanol. All reagents were used as received. All the equipment used in the study was pre-cleaned with methanol and then Milli-Q water.
Teflon and glass equipment was used in the experiment.

1.3 Sample preparation and extraction

Serum samples were extracted using the method described by Hansen et al. [28]. Briefly, 0.5 mL of serum, 2 mL of Na2CO3 (0.25 mol/L), and 1 mL of tetrabutylammonium hydrogen sulfate (0.5 mol/L) were added to a 15 mL pre-washed polypropylene tube for extraction and mixed well. MTBE (5 mL) was then added to the solution, and the mixture was shaken for 20 min. The organic and aqueous layers were separated by centrifugation at 3000 t/min for 15 min, and the organic layer was transferred to a second polypropylene tube. The aqueous layer was extracted again with MTBE, and the organic layer was separated and combined with that from the first extraction. The combined MTBE extracts were evaporated to dryness under high purity nitrogen, and then the residue was reconstituted in 1 mL of a mixture of methanol and 10 mmol/L ammonium acetate (2:3, v/v). Finally, the sample was filtered through a 0.22 μm nylon filter.

1.4 Instrumental analysis

The PFCs concentrations in the serum samples were analyzed using HPLC-tandem mass spectrometry (HPLC-MS/MS). Chromatography was performed on an Agilent 1100 HPLC system (Agilent Technologies, Palo Alto, CA). A 25 μL aliquot of the extract was injected onto a Agilent Eclipse Plus C18 column (100 mm × 2.1 mm I.D., 3.5 μm particle size, Agilent Technologies, Palo Alto, CA). The column temperature was 40°C. The mobile phase was a mixture of 10 mmol/L ammonium acetate and acetonitrile at a flow rate of 0.25 mL/min. The mobile phase gradient started at 40% acetonitrile, increased to 90% acetonitrile at 9 min, and was held at this level for 2 min. The system was allowed to equilibrate for 8 min between injections. The HPLC system was interfaced to an Agilent 6410 Triple Quadrupole mass spectrometer (Agilent Technologies, Santa Clara, CA) operated in negative electrospray ionization (ESI) mode. The dry gas temperature and ion spray voltage were maintained at 350°C and 4000 V. The dry gas flow and nebulizer pressure were 7 L/min and 35 psi (1 psi = 6.895 kPa), respectively.

1.5 Quality assurance and quality control

During the analysis, procedural blanks were prepared and analyzed after every 10 samples to check if contamination occurred during the preparation of samples. Solvent blanks containing acetonitrile and Milli-Q water (2:3, v/v) were prepared and analyzed after every 20 samples to monitor instrumental background contamination. Duplicate injections and calibration check standards were run after every 20 samples to ensure the precision and accuracy of each run. The concentrations of serum extracts were quantified via nine-point matrix-matched calibration curves constructed from 0.01 to 100 ng/mL by adding the mixed PFC standard solution into newborn bovine serum (HyClone, Logan, Utah). The limit of detection (LOD) was defined as the analyte concentration required produce a signal-to-noise (S/N) ratio of 3:1, and the limit of quantification (LOQ) was defined as the lowest point on the standard curve, above the LOD, that had a relative standard deviation (RSD) <20%. The LODs for the ten target PFCs ranged from 0.01 to 0.02 ng/mL, while the LOQs ranged from 0.03 to 0.05 ng/mL. The recovery and reproducibility of the serum sample extraction were determined on six replicate analyses of 0.5 mL of newborn bovine serum containing 2 ng of each PFC standard. No PFC contamination was found above the LOD in newborn bovine serum. Recoveries of all the PFCs analytes were between (82±6)% and (101±6)%.

1.6 Modeling of exposure based on serum PFCs levels

In this study, the daily intake of PFOA and PFOS in the general population in Shenzhen was estimated based on serum PFC concentrations with a simple one-compartment pharmacokinetic model [19,29,30]. The change in blood concentration (Cp) resulting from a given exposure dose (E) can be described by the following equation:

\[
\frac{d(C_p)}{dt} = E - k \times V_d \times C_p,
\]

where \(V_d\) is the volume of distribution (mL/kg bw) and \(k\) is the first-order elimination per day, which is equal to \(0.693/t_{1/2}\) under steady-state conditions. In the case where \(d(C_p)/dt = 0\), this equation becomes

\[
E = k \times V_d \times C_p,
\]

and

\[
E = 0.693/t_{1/2} \times V_d \times C_p.
\]

In agreement with previous studies, the median half-life of PFOA and PFOS were 1257 and 1661 d [31], respectively. The volume of distribution of PFOA and PFOS were 170 and 230 mL/kg (bw) [29], respectively.

1.7 Statistical analysis

Statistical analyses were performed with SPSS 16.0 (SPSS Inc., Chicago, IL). Concentrations lower than the LOD and the LOQ were assigned a value of half of the LOD and LOQ, respectively. Differences among the serum PFCs levels in different gender and age groups were evaluated using the Mann-Whitney test. The Spearman’s rank correlation was applied to assess the relationships between age and the PFCs levels in serum, and among various PFCs in the samples. All the differences and correlations were considered to be significant at \(P<0.05\).
2 Results and discussion

The serum PFCs levels of all the participants, stratified by gender, are given in Table 1. PFOS and PFOA were detected in all of the samples analyzed. The frequency of detection of the other target PFCs was in the order PFNA > PFHpA > PFHxS > PFBS > PFHxA > PFDoA > PFTA. These results indicate that exposure to PFCs was widespread in the general population of Shenzhen. PFOA had the highest median concentration of 6.72 ng/mL (range 0.30 to 48.4 ng/mL), followed by PFOS (median 2.07 ng/mL, range 0.10 to 40.0 ng/mL). The other target PFCs were detected at much lower levels, with median concentrations ranging from 0.02 to 0.87 ng/mL.

The PFCs concentrations in the present study were comparable to those from previous studies (Table 2). For the purpose of comparison, the PFCs concentrations in whole blood were converted into serum PFCs concentrations by multiplying by a factor of 2 [32]. The median serum PFOA concentration in Shenzhen was slightly higher than those observed in North America [33,34], Belgium [32], Italy [35], Japan [36], and other areas of China [17,18,30,37,38], and similar to the median concentrations detected in Australia [39] and Germany [40]. However, it was much lower than those from PFC-contaminated regions in the U.S. [21] (374 ng/mL) and Germany [20] (>20 ng/mL). The median concentrations of PFOS (2.1 ng/mL) and PFHxS (0.46 ng/mL) in the current study were considerably lower than those reported in North America [33,34], Europe [32,41], Australia [39,42] and some other regions in China (14.8–35.8 ng/mL for PFOS, 1.3–6.2 ng/mL for PFHxS) [17,18]. The long-chain perfluorocarboxylate, PFNA, was detected in most of the serum samples, and its median concentration (0.87 ng/mL) was comparable to those found in Australia [39] (0.8 ng/mL) and the U.S. [33] (0.6 ng/mL).

2.1 Gender- and age-related differences in serum PFC levels

To evaluate possible gender and age influences on the serum PFCs concentrations, serum samples were stratified into four different groups according to the age of the individual (<5, 5–13, 14–60, and >60 years) and the gender. The age groups were established based on the enrollment age for children (5–6 years), the average age of menarche (13 years), and the maximum retirement age of the general population (60 years).

The concentrations for each PFC in the serum samples stratified by gender are given in Table 1. In general, no statistically significant differences were found for any of the PFCs between genders. However, the median concentrations of PFOA, PFOS, PFNA and PFHxS were slightly higher in males than in females. The geometric mean concentrations of PFOA (6.72 ng/mL) and PFHxS (0.46 ng/mL) in the present study were comparable to those from previously studies (Table 2). For the purpose of comparison, the PFCs concentrations in whole blood were converted into serum PFCs concentrations by multiplying by a factor of 2 [32]. The median serum PFOA concentration in Shenzhen was slightly higher than those observed in North America [33,34], Belgium [32], Italy [35], Japan [36], and other areas of China [17,18,30,37,38], and similar to the median concentrations detected in Australia [39] and Germany [40]. However, it was much lower than those from PFC-contaminated regions in the U.S. [21] (374 ng/mL) and Germany [20] (>20 ng/mL). The median concentrations of PFOS (2.1 ng/mL) and PFHxS (0.46 ng/mL) in the current study were considerably lower than those reported in North America [33,34], Europe [32,41], Australia [39,42] and some other regions in China (14.8–35.8 ng/mL for PFOS, 1.3–6.2 ng/mL for PFHxS) [17,18]. The long-chain perfluorocarboxylate, PFNA, was detected in most of the serum samples, and its median concentration (0.87 ng/mL) was comparable to those found in Australia [39] (0.8 ng/mL) and the U.S. [33] (0.6 ng/mL).

### Table 1 PFCs concentrations (ng/mL) in serum samples from Shenzhen residents stratified by gender

| Gender | No. | PFBS | PFHxS | PFOS | PFHxA | PFHpA | PFOA | PFNA | PFDA | PFDoA | PFTA |
|--------|-----|------|-------|------|-------|-------|------|------|------|-------|------|
| Male   | 133 | 37.6 | 80.5  | 100  | 26.3  | 87.2  | 100  | 97   | 45.1 | 24.1  | 22.6 |
| GM     | 0.05| 0.39 | 2.07  | 0.04 | 0.18  | 6.41  | 0.75 | 0.12 | 0.05 | 0.03  |
| Min    | ND  | ND   | 0.10  | ND   | ND    | ND    | ND   | ND   | ND   | ND    |
| 25th   | 0.02| 0.11 | 0.48  | 0.02 | 0.12  | 3.66  | 0.53 | 0.03 | 0.03 | 0.02  |
| Median | 0.02| 0.42 | 1.88  | 0.02 | 0.22  | 6.53  | 0.87 | 0.03 | 0.03 | 0.02  |
| 75th   | 0.08| 1.81 | 10.9  | 0.12 | 0.36  | 12.1  | 1.36 | 0.48 | 0.03 | 0.02  |
| Max    | 9.97| 13.2 | 39.5  | 1.64 | 1.78  | 42.9  | 7.05 | 3.58 | 1.57 | 0.54  |
| Female | 94  | 51.1 | 75.5  | 100  | 27.7  | 86.2  | 100  | 96.8 | 51.1 | 24.5  | 21.3 |
| GM     | 0.06| 0.32 | 2.28  | 0.04 | 0.16  | 6.33  | 0.63 | 0.13 | 0.05 | 0.03  |
| Min    | ND  | ND   | 0.13  | ND   | ND    | 0.30  | ND   | ND   | ND   | ND    |
| 25th   | 0.02| 0.04 | 0.45  | 0.02 | 0.10  | 3.24  | 0.42 | 0.03 | 0.03 | 0.02  |
| Median | 0.03| 0.49 | 2.39  | 0.02 | 0.19  | 7.64  | 0.88 | 0.15 | 0.03 | 0.02  |
| 75th   | 0.20| 1.35 | 11.4  | 0.11 | 0.36  | 13.0  | 1.29 | 0.52 | 0.05 | 0.02  |
| Max    | 7.72| 14.9 | 40.0  | 2.36 | 1.14  | 48.4  | 6.17 | 2.24 | 0.53 | 0.56  |
| P      | 0.04| 0.59 | 0.78  | 0.94 | 0.25  | 0.85  | 0.42 | 0.61 | 0.91 | 0.87  |

Total 227 Detection (%) 43.2 78.4 100 26.9 86.8 100 96.9 47.6 24.2 22.0

GM 0.05 0.36 2.15 0.00 0.17 6.38 0.70 0.12 0.05 0.00
Min ND ND 0.10 ND ND 0.30 ND ND ND ND
25th 0.02 0.08 0.45 0.02 0.11 3.54 0.49 0.03 0.03 0.02
Median 0.02 0.46 2.07 0.02 0.20 6.72 0.87 0.03 0.03 0.02
75th 0.13 1.58 11.2 0.11 0.36 12.5 1.31 0.52 0.03 0.02
Max 9.97 14.9 40.0 2.36 1.78 48.4 7.05 3.58 1.57 0.56

a) No., the number of participants; Detection, the frequency of detection; GM, geometric mean; ND, not detected; the significance of the difference (P) between genders was determined by the Mann-Whitney test.
Table 2  Comparison of median (range) concentrations (ng/mL) of selected perfluorinated compounds in human serum/plasma from several countries

| Area          | PFOA      | PFOS      | PFNA     | PFHxS    | No. | Year sampled | Reference |
|---------------|-----------|-----------|----------|----------|-----|--------------|-----------|
| Asia/Pacific  |           |           |          |          |     |              |           |
| China         | 6.72 (0.30–48.4) | 2.07 (0.10–40.0) | 0.87 (ND–7.05) | 0.46 (ND–14.9) | 227 | 2009         | Present study |
|               | 4.3 (0.20–60) | 22.4 (0.20–145) | –        | –        | 119 | 2002         | [18]      |
|               | 1.01 (0.05–76.26) | 5.58 (0.41–33.47) | –        | 1.47 (0.12–25.23) | 138 | 2008         | [37]      |
|               | 1.59       | 52.7       | –        | 1.88     | 85  | 2004         | [17]      |
| Japan         | 2.8–12.4   | 3.5–28.1   | –        | –        | 205 | 2003         | [36]      |
| Australia     | 6.4 (0.8–9.1) | 14.8 (5–28.5) | 0.8 (0.1–1.4) | 2.9 (<0.1–11.3) | 84  | 2006–2007    | [39]      |
|               | 7.6 (5.0–9.9) | 20.8 (12.7–29.5) | 1.1 (0.4–2.0) | 6.2 (2.7–19.0) | 40  | 2002–2003    | [42]      |
| North America |           |           |          |          |     |              |           |
| USA           | 5.1       | 30.2      | 0.6      | 2.1      | 1562 | 1999–2000   | [43]      |
|               | 4.7 (<1.9–52.3) | 35.8 (<4.3–1656) | –        | 1.5 (<1.4–66.3) | 645 | 2000–2001    | [34]      |
|               | (<3–88)a) | (<1.3–164)a) | –        | (<0.4–32)a) | 175 | 2000–2002    | [32]      |
| Europe        |           |           |          |          |     |              |           |
| Belgium       | 4.1 (1.1–12.8) | 17.2 (4.5–27) | –        | 1.3 (1.1–1.4) | 20  | 1998, 2000   | [32]      |
| Sweden        | 5.0 (1.0–24.8) | 34.2 (3.4–74) | –        | 3.0 (0.8–56.8) | 66  | 1997–2000    | [41]      |
| Germany       | 6.8 (1.7–39.3) | 22.3 (6.2–131) | –        | –        | 105 | 2005         | [40]      |
| Poland        | (9.7–40)a) | (16–116)a) | –        | (0.4–2.6)a) | 25  | 2003         | [32]      |

a) Calculated from the concentration in whole blood by multiplying by a factor of 2; b) geometric mean; c) range of the geometric means of different regions; d) forty pooled samples were created from 3802 individual samples; e) Eighty-four pooled samples were created from 2420 individual samples.

Table 3  Spearman’s rank correlation coefficients between age and serum PFCs concentrations

|       | PFBS | PFHxA | PFOS | PFHxS | PFOA | PFNA | PFDA | PFDoA | PFTA |
|-------|------|-------|------|-------|------|------|------|-------|------|
| Male  | −0.25a) | 0.41a) | 0.43a) | −0.02 | −0.18b) | 0.24a) | 0.16 | 0.55a) | 0.36a) | 0.33a) |
| Female| −0.08 | 0.49a) | 0.37a) | 0.20 | 0.01 | 0.35a) | 0.37a) | 0.42a) | 0.26a) | 0.16 |
| Total | −0.16a) | 0.44a) | 0.41a) | 0.08 | −0.11 | 0.29a) | 0.24a) | 0.50a) | 0.32a) | 0.26a) |

a) Correlation is significant at the 0.01 level (2-tailed); b) correlation is significant at the 0.05 level (2-tailed).
sized as important PFCs elimination routes for premenopausal adult females in some studies [46]. The decrease in serum PFBS levels with age could result from the faster serum elimination rate of PFBS compared to long-chain PFCs [47].

The correlation between the PFCs serum levels and age in the various age groups was not consistent (Figure 1). The serum concentrations of PFOA and PFHxS increased with age in the four groups. With PFOS, the concentration increased with age in the groups of individuals <60 years old, and then decreased at >60 years. For PFOA, PFOS, PFHxS and PFNA the concentrations in the groups aged <13 years were much lower than those in the group aged >60 years. Significantly higher serum concentrations of PFOS, PFHxS and PFNA were observed in the 14–60 years group, compared with those in the <13 years groups. Overall, the serum concentrations of PFCs in the 14–60 and >60 years groups were significantly greater than those in the <13 years groups, which suggests these compounds accumulate with age. These results were not consistent with some earlier studies, in which higher levels of PFCs were found in children than in adults [30,39]. Those findings suggested that contact with carpeted floors and furniture, hand-to-mouth activity, ingestion of dust [22], breast milk [45], and transplacental movement of PFCs from the mother to the child [48] might contribute to the higher PFC concentrations in children. Furthermore, the concentrations of PFOS decreased in the >60 years group compared with those in the 14–60 years group. Further studies needed to be carried out to determine the reason for this.

2.2 Correlations among serum levels of PFCs analogues

The Spearman’s rank correlation coefficients among the different PFCs were examined. Significant positive correlations (P<0.01) between PFOS and PFHxS, PFOA and PFHxS, PFNA and PFHxS, and PFOS and PFNA were found. In addition, significant positive correlations were found among PFOA, PFDA, PFDoA, PFTA and PFNA, which suggests there is a common exposure pathway for these long-chain PFCAs in Shenzhen. However, PFDoA and PFTA were negatively correlated to PFHpA and PFOA, respectively (P<0.01). In general, the correlations found in the present study showed similar sources of exposure for PFCs in this region.

2.3 Modeling of daily intake of PFCs by the general population in Shenzhen

The one-compartment toxicokinetic model and the measured

Figure 1  Serum concentrations of PFOA (a), PFOS (b), PFNA (c), and PFHxS (d) in four age groups of the general population in Shenzhen, China. The box plots show the 25th percentile, median, and 75th percentile, and the error bars indicate the minimum and maximum values. The results presented were calculated after excluding outliers.
serum PFC concentrations from the present study were used to estimate the daily intake of PFOA and PFOS for the general population in Shenzhen. The median concentrations of PFOA and PFOS in serum samples from Shenzhen were 6.7 and 2.1 ng/mL, respectively, and the total daily intakes of PFOA and PFOS for the general population in Shenzhen were calculated as 0.63 and 0.20 ng/kg(bw), respectively. The estimated total daily intake of PFOA in the present study was higher than that in Fuxin, China (0.40 ng/kg(bw)), where several fluorochemical plants exist [30]. However, the total daily intake of PFOS was lower than that in Nanchang, China (0.74 ng/kg(bw) for males, 1.2 ng/kg(bw) for females) [38]. In comparison with developed countries, the estimated total daily intakes of PFOA and PFOS in Shenzhen were much lower than those in Australia (PFOA, 1.3 ng/kg(bw); PFOS, 2.4 ng/kg(bw)) [29].

Median concentrations of PFOA and PFOS in drinking water from Shenzhen have been reported as 45.9 and 14.8 ng/mL, respectively [26]. Mean concentrations of 1 ng/mL (PFOA) and 11 ng/mL (PFOS) have also been reported [25]. These data were used to calculate daily intakes of PFOA and PFOS from drinking water by assuming a usual body weight of 60 kg [49] and a drinking water intake of 2 L. The daily intakes from drinking water for the general population in Shenzhen were calculated to be 1.53 and 0.03 ng/kg(bw) for PFOA, and 0.37 and 0.49 ng/kg(bw) for PFOS. From these values, it was estimated the consumption of drinking water in Shenzhen contributes 243% or 5% of the total PFOA, and 245% or 185% of the total PFOS. These estimated contributions of drinking water to PFCs exposure in Shenzhen were much higher than that estimated for the general European population (16%), which suggests drinking water might be a major PFCs source in this city.

However, there were some limitations to the estimation of the contribution of drinking water, because the calculation was based on data from studies which only single collection for each sample was taken for monitoring of water quality. Because the concentrations of PFOA and PFOS in drinking water could vary with time, these estimates might not provide a real-life reflection of the drinking water exposure over a long time. This could explain the differences in the estimated contributions for drinking water using the data from the two different studies. Therefore, continuous monitoring of drinking water is required to accurately evaluate the exposure to PFCs from this source. Further research is needed to investigate the potential sources of human exposure to PFCs in Shenzhen.

3 Conclusions

In summary, the results from this study indicate that human exposure to PFCs is prevalent in Shenzhen. Among the 10 target PFCs, PFOA was the major compound detected in the serum samples from the general population living in Shenzhen. The results were consistent with those for drinking water from this area. Estimated total daily intakes of PFOA and PFOS for the general population in Shenzhen were 0.63 and 0.20 ng/kg(bw), respectively, and were much lower than those found in an earlier study in Australia. However, the contribution from drinking water was estimated to be much higher than that estimated in an earlier study for the general European population (16%), which suggests that drinking water may be a major source of PFC exposure in Shenzhen. Because of the persistence of PFCs, legacy sources will continue to contribute a constant, low-level exposure to PFCs for many years. Therefore, further studies are required to understand the sources and pathways of PFC exposure for the general population in Shenzhen, and assess the potential human health risk for the local population.

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1 Organization for Economic Co-operation and Development. Co-operation on existing chemicals. Hazard assessment of perfluorooctane sulfonate (PFOS) and its salts, ENV/JM/RD17/FINAL, Paris, 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: 1339–1342
4 Martin J W, Whittle D M, Muir D C G, et al. Perfluoralkyl contaminants in a food web from Lake Ontario. Environ Sci Technol, 2004, 38: 5379–5385
5 Lau C, Anitole K, Hodes C, et al. Perfluoralkyl acids: A review of monitoring and toxicological findings. Toxicol Sci, 2007, 99: 366–394
6 Martin J W, Smithwick M M, Braune B M, et al. Identification of long-chain perfluorinated acids in biota from the Canadian Arctic. Environ Sci Technol, 2004, 38: 373–380
7 3M Company. Phase-out plan for POSF-based products, U.S. Environmental Protection Agency Public Docket AR-226-0588. U.S. EPA, St.Paul, MN, 2000
8 United States Environmental Protection Agency. Perfluorooctane sulfonate: proposed significant new use rule. Fed Reg, 2000, 65: 62319–62333
9 European Union. Directive 2006/122/ECOF of the European Parliament and of the Council of 12 December 2006. Official J Eur Union, L/372/32–34, 2006
10 Environment Canada. Order Adding Toxic Substances to Schedule I to the Canadian Environmental Protection Act, 1999. Can Gaz Part II. 2006,140: 2147–2153
11 Stockholm Convention on Persistent Organic Pollutants. Governments unite to step-up reduction on global DDT reliance and add nine new chemicals under international treaty. Geneva: Stockholm Convention Secretariat, 8 May 2009. http://chm.pops.int/Convention/Pressrelease/ COP4Geneva8May2009/tabid/542/language/en-US/Default.aspx
12 So M K, Taniyasu S, Yamashita N, et al. Perfluorinated compounds in coastal waters of Hong Kong, South China, and Korea. Environ Sci Technol, 2004, 38: 4056–4063
13 Ju X, Jin Y, Sasaki K, et al. Perfluorinated surfactants in surface, subsurface water and microlayer from Daliao coastal waters in China. Environ Sci Technol, 2008, 42: 3538–3542
14 Bao J, Liu W, Liu L, et al. Perfluorinated compounds in urban river sediments from Guangzhou and Shanghai of China. Chemosphere, 2010, 80: 123–130
15 Liu W, Jin Y, Quan X, et al. Perfluorosulfonates and perfluorocar-
boxylates in snow and rain in Dalian, China. Environ Int, 2009, 35: 737–742

16 Li X, Yeung L W Y, Taniyasu S, et al. Perfluorooctane sulfonate and related fluorochemicals in the Amur Tiger (Panthera tigris altaica) from China. Environ Sci Technol, 2008, 42: 7078–7083

17 Yeung L W Y, So M K, Jiang G, et al. Perfluorooctanesulfonate and related fluorochemicals in human blood samples from China. Environ Sci Technol, 2006, 40: 715–720

18 Jin Y, Saito N, Harada K, et al. Historical trends in human serum levels of perfluorooctanoate and perfluorooctane sulfonate in Shenyang, China. Tohoku J Exp Med, 2007, 212: 63–70

19 Fromme H, Schlummer M, Moller A, et al. Exposure of an adult population to perfluorinated substances using duplicate diet portions and biomonitoring data. Environ Sci Technol, 2007, 41: 7928–7933

20 Holzer J, Midasch O, Rauchfuss K, et al. Biomonitoring of perfluorinated compounds in children and adults exposed to perfluorooctanotocante-contaminated drinking water. Environ Health Perspect, 2008, 116: 651–657

21 Emmett E A, Shofer F S, Zhang H, et al. Community exposure to perfluorooctanoate: relationships between serum concentrations and exposure sources. J Occup Environ Med, 2006, 48: 759–770

22 Shoeb M, Harner T, Wilford B H, et al. Perfluorinated sulfonamides in indoor and outdoor air and indoor dust: Occurrence, partitioning, and human exposure. Environ Sci Technol, 2005, 39: 6599–6606

23 Fromme H, Tittlemier S A, Völkel W, et al. Perfluorinated compound-exposure assessment for the general population in western countries. Int J Hyg Environ Health, 2009, 212: 239–270

24 U.S. Environmental Protection Agency. Provisional Health Advisories for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS), 2004. http://www.epa.gov/waterscience/criteria/drinking/phac-PFOA_PFOS.pdf

25 Mak Y L, Taniyasu S, Yeung L W Y, et al. Perfluorinated compounds in tap water from China and several other countries. Environ Sci Technol, 2009, 43: 4824–4829

26 Jin Y, Liu W, Sato I, et al. PFOS and PFOA in environmental and tap water in China. Chemosphere, 2009, 77: 605–611

27 Post G B, Louis J B, Cooper K R, et al. Occurrence and potential significance of perfluorooctanoic acid (PFOA) detected in New Jersey public drinking water systems. Environ Sci Technol, 2009, 43: 4547–4554

28 Hansen K J, Clemen L A, Ellefson M E, et al. Compound-specific, quantitative characterization of organic fluorochemicals in biological matrices. Environ Sci Technol, 2001, 35: 766–770

29 Thompson J, Lorber M, Toms L M L, et al. Use of simple pharmacokinetic modeling to characterize exposure of Americans to perfluorooctanoic acid and perfluorooctane sulfonic acid. Environ Int, 2010, 36: 390–397

30 Bao J, Liu W, Liu L, et al. Perfluorinated compounds in the environment and the blood of residents living near fluorochemical plants in Fuzin of China. Environ Sci Technol, doi: 10.1021/es102610x

31 Olsen G W, Burris J M, Ehresman D J, et al. Half-life of serum elimination of perfluorooctanesulfonate, perfluorononanesulfonate, perfluorooctanoate in retired fluorochemical production workers. Environ Health Perspect, 2007, 115: 1298–1305

32 Kannan K, Corsolini S, Falandysz J, et al. Perfluorooctanesulfonate and related fluorochemicals in human blood from several countries.

Environ Sci Technol, 2004, 38: 4489–4495

Calafat A M, Kuiksten Y Z, Reidy J A, et al. Serum concentrations of 11 polyfluoroalkyl compounds in the US population: Data from the National Health and Nutrition Examination Survey (NHANES) 1999–2000. Environ Sci Technol, 2007, 41: 2237–2242

Olsen G W, Church T R, Miller J P, et al. Perfluorooctane sulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors. Environ Health Perspect, 2003, 111: 1892–1901

Corsonoli S, Kannan K. Perfluorooctanesulfonate and related fluorochemicals in several organisms including humans from Italy. Organohalogen Compd, 2004, 66: 4079–4085

Harada K, Saito N, Inoue K, et al. The influence of time, sex and geographical factors on levels of perfluorooctanoate sulfonate in human serum over the last 25 years. J Occup Health, 2004, 46: 141–147

Liu J Y, Li J G, Luan Y, et al. Geographical distribution of perfluorinated compounds in human blood from Liaoning Province, China. Environ Sci Technol, 2009, 43: 4044–4048

Zhang T, Wu Q, Sun H W, et al. Perfluorinated compounds in whole blood samples from infants, children, and adults in China. Environ Sci Technol, 2010, 44: 4341–4347

Toms L M L, Calafat A M, Kato K, et al. Polyfluorooalkyl chemicals in pooled blood serum from infants, children, and adults in Australia. Environ Sci Technol, 2009, 43: 4194–4199

Midasch O, Schettgen T, Angerer J. Pilot study on PFOA and PFOA of the German general population. Int J Hyg Environ Health, 2006, 209: 489–496

Kärman A, Van Bavel B, Järnberg U, et al. Levels of perfluorooalkylated compounds in whole blood from Sweden. Organohalogen Compd, 2004, 66: 4058–4062

Kärman A, Mueller J F, Van Bavel B, et al. Levels of 12 perfluorinated chemicals in pooled Australian serum, collected 2002–2003, in relation to age, gender, and region. Environ Sci Technol, 2006, 40: 3742–3748

Calafat A M, Wong L Y, Kuiksten Y Z, et al. Polyfluorooalkyl 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: 1596–1602

Fromme H, Midasch O, Twaddell D, et al. Occurrence of perfluorinated substances in an adult German population in southern Bavaria. Int Arch Occup Environ Health, 2007, 80: 313–319

Kärman A, Ericson I, Van Bavel B, et al. Exposure of perfluorinated chemicals through lactation: Levels of matched human milk and serum and a temporal trend, 1996–2004, in Sweden. Environ Health Perspect, 2007, 115: 226–230

Harada K, Inoue K, Morikawa A, et al. Renal clearance of perfluorooctanoic acid and perfluorooctanoate in humans and their species-specific excretion. Environ Res, 2005, 99: 253–261

Olsen G W, Chang S C, Noker P E, et al. A comparison of the pharmacokinetics of perfluorobutanesulfonate (PFBS) in rats, monkeys, and humans. Toxicology, 2009, 256: 65–74

Inoue K, Okada F, Ito R, et al. Perfluorooctane sulfonate (PFOA) and related perfluorinated compounds in human maternal and cord blood samples: assessment of PFOA exposure in a susceptible population during pregnancy. Environ Health Perspect, 2004, 112: 1204–1207

General Administration of Sport of China. Bulletin of the Second National Fitness Survey of China, 2005

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