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Long-term trends in dietary intake of perfluoroalkyl carboxylic acids in relation to their serum concentration in two regions in Japan from 1979 to 2011

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HIGHLIGHTS

- Perfluoroalkyl carboxylic acids (PFCAs) in diet and serum have been investigated.
- The analyzed samples obtained since the 1980s until the 2010s.
- A positive correlation was observed between diet and serum concentration of PFCAs.
- This indicates that dietary intake may be a source of C8–C11 PFCAs exposure.

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ABSTRACT

We investigated temporal changes of perfluoroalkyl carboxylic acids (PFCAs) with 8–14 carbon atoms (C8 to C14) in duplicate diet and serum samples in Japan. The sum dietary intakes of PFCAs (C8 to C13) in the Kansai and Tohoku region were highest in the 2010s (mean; 177 ng/day for Kansai, 107 ng/day for Tohoku) followed by the 2000s (77 ng/day for Kansai, 34 ng/day for Tohoku) and the 1990s (53 ng/day for Kansai, 58 ng/day for Tohoku), then the 1980s (19 ng/day for Kansai, 23 ng/day for Tohoku). The sum of the serum concentartions (C8 to C13) was also highest in the 2010s (mean; 17 ng/mL for Kansai, 7.4 ng/mL for Tohoku), followed by the 2000s (12 ng/mL for Kansai, 6.3 ng/mL for Tohoku), then the 1990s (6.8 ng/mL for Kansai, 5.5 mg/mL for Tohoku) and the 1980s (3.8 ng/mL for Kansai, 0.4 ng/mL for Tohoku). A positive correlation was observed between dietary intakes and serum concentration for C8 to C11 ($r = 0.94$, $p < 0.05$ for C8; $r = 0.80$, $p < 0.05$ for C9; $r = 0.98$, $p < 0.05$ for C10; and $r = 0.84$, $p < 0.05$ for C11). The levels of C8, C9 and C10 in serum and dietary intake in the 2010s were much higher in Kansai than those in Tohoku, although those of C11 did not show such differences. Kansai has a fluoropolymer manufacture known as a specific source of PFOA (C8), and is more urbanized than Tohoku, which may be attributed to the higher levels of PFCAs (C8 to C10). On the other hand, C11 is common to residents in Kansai and Tohoku.

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1. Introduction

Perfluoroalkyl carboxylic acids (PFCAs) are carboxylic acids with a perfluoroalkyl chain, where all the hydrogen atoms have been replaced by fluorine atoms (Kissa, 2001). PFCAs persist in the environment, as they cannot decompose in the ecosystem (ECHA). The PFCA with eight carbon atoms is called perfluorooctanoic acid (PFOA or C8), and has been used for the manufacture of fluoropolymer resin (Scheirs, 1997).

Previous animal studies have revealed the potential toxicities of PFCAs, particularly developmental toxicity (Case et al., 2001),

Abbreviations: PFCAs, perfluoroalkyl carboxylic acids; PFOA or C8, perfluorooctanoic acid; PFNA or C9, perfluorononanoic acid; PFDA or C10, perfluorodecanoic acid; PFUnDA or C11, perfluoroundecanoic acid; PFDoDA or C12, perfluorododecanoic acid; PFTrDA or C13, perfluorotridecanoic acid; PFTeDA or C14, perfluorotetradecanoic acid; IDLs, instrumental detection limits; MDLs, method detection limits; SD, standard deviation; GM, geometric mean.

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immune toxicity (Yang et al., 2002), and hepato-toxicity (Klaunig et al., 2003; Lau et al., 2007). Recently, a systematic review concluded that there was sufficient evidence that developmental exposure to PFOA reduces fetal growth in humans (Johnson et al., 2014). Epidemiological studies demonstrated that PFCAs can suppress antibody production after immunization against tetanus and diphtheria in children (Grandjean et al., 2012), and have been linked to miscarriage (Jensen et al., 2015). PFCAs therefore have potential health risks in the general population.

Major fluoropolymer manufacturers have pledged to try to reduce PFOA emissions under a stewardship program by the US Environmental protection agency (EPA, 2015). In Japan, Daikin Industry in Osaka, Kansai region, announced that PFOA emissions in 2012 were less than 1% of that in 2000 (Daikin, 2012). To evaluate the effect of those voluntary reductions, follow-up monitoring for PFOA and other PFCAs in diet and serum over long periods of time is needed.

Several previous studies around the world have reported temporal PFCA trends in human serum from around 1995 to the 2010s (Calafat et al., 2007; Harada et al., 2011; Kato et al., 2011; Glynn et al., 2012; Gebbink et al., 2015b). According to these reports, long-chain PFCAs (>C8) levels in human serum have been increasing since around the 2000s, although PFOA levels have decreased since 2002 (Calafat et al., 2007; Harada et al., 2011; Glynn et al., 2012). More recently, long-term PFCA trends (1982–2009) in human serum were evaluated in Germany (Yeung et al., 2013), and the C9 compound showed a transient elevation from 1982 to 1990, which was not observed in the other countries.

Previous reports have also described PFCA levels in food samples (D'Hollander et al., 2010; Haug et al., 2010; Noorlander et al., 2011; Vestergren et al., 2012; Klenow et al., 2013; D'Hollander et al., 2015). For PFOA, exposure modeling studies have shown that dietary exposure is the major exposure pathway in most of general population (Trudel et al., 2008; Fromme et al., 2009; Vestergren and Cousins, 2009; Niisoe et al., 2010). However, the importance of different exposure pathways of PFCAs longer than PFOA (C8) for humans is still ambiguous (D'Eon et al., 2011; Gebbink et al., 2015a).

In the present study, we evaluated the concentration of PFCAs (C8 to C14) in diet composite samples from 1979 to 2011 in two different regions in Japan. We used duplicate sampling methods; all food and drink items include tap water for drinking that was consumed over a 24-h period. This method evaluates the dietary exposures which are very close to actual levels. We also analyzed serum samples, which were collected at corresponding time points and regions. Ecological evaluation of both diet duplicate and serum samples over three decades allowed us to assess whether long-term trends of serum PFCA concentrations is associated with dietary PFCA intakes.

2. Materials and methods

2.1. Sample collection

We used archived samples from the Kyoto University Human Specimen Bank (Koizumi et al., 2005, 2009). Details of the target population and sampling regions are shown in Table S1 and Fig. S1. Kansai is located in the central of the main island of Japan, while Tohoku is in the north of the same island. The Kansai region is more urban than the Tohoku region. The Kansai region had around 20 million residents in 2014, while the Tohoku region had 9 million residents (MIC, 2016). Moreover, Osaka Prefecture in the Kansai region is known to have a specific source of PFOA contamination from fluoropolymer manufacture (i.e., Daikin). The PFOA emission flux from Daikin to the air was estimated as 2.3 t/year in 2003 (Niisoe et al., 2010). However, Daikin announced that PFOA emissions in 2012 were less than 1% of that in 2000 (Daikin, 2012). Diet duplicate samples were collected from healthy volunteers in 1981 ($n = 18$), 1992 ($n = 12$), 2004 ($n = 16$), and 2011 ($n = 26$) in the Tohoku region (Miyagi and Fukushima), and in 1979 ($n = 15$), 1993 ($n = 25$), 2003/2004 ($n = 18$), and 2011 ($n = 18$) in the Kansai region (Kyoto and Wakayama). At all collection times, all participants were requested to donate duplicate samples of all food and drink items, including tap water, which they consumed over a 24-h period. Thus, all samples in this study are referred to as duplicate 24-h diet samples (24-h food duplicate method). Individual serum samples were collected in 1981 ($n = 27$), 1997 ($n = 30$), 2003 ($n = 30$), and 2017 ($n = 30$) in the Tohoku region (Miyagi), and in 1983 ($n = 15$), 1993 ($n = 30$), 2004–2005 ($n = 30$), and 2011 ($n = 30$) in the Kansai region (Kyoto and Wakayama). The subjects in this study were all females, and residential areas of the serum donators matched those of the dietary sample donators. All samples were stored at −20 °C until analysis. We obtained approval for this research proposal from the Ethics Committee of the Kyoto University Graduate School of Medicine (E25).

2.2. Chemicals

Methanol, acetone, and methyl *tert*-butyl ether (MTBE) of pesticide residue analysis grade were obtained from Kanto Chemicals (Tokyo, Japan). Benzyl bromide, tetrabutylammonium hydrogen sulfate, sodium carbonate, sodium hydrogen carbonate, and 11H-perfluoroundecanoic acid (11H-PFUnDA) were obtained from Wako Pure Chemical Industries (Osaka, Japan). A mixture of $^{13}C_4$-labeled PFOA, $^{13}C_5$-labeled perfluorononanoic acid (PFNA or C9), $^{13}C_2$-labeled perfluorodecanoic acid (PFDA or C10), $^{13}C_2$-labeled perfluoroundecanoic acid (PFUnDA or C11) and $^{13}C_2$-labeled perfluorododecanoic acid (PFDoDA or C12) were purchased from Wellington Laboratories Inc. (Guelph, Canada) as internal standards.

2.3. Determination of PFCAs

2.3.1. Clean-up procedure

PFOA, PFNA, PFDA, PFUnDA, PFDoDA, perfluorotridecanoic acid (PFTrDA or C13) and perfluorotetradecanoic acid (PFTeDA or C14) were analyzed by gas chromatography/mass spectrometry (Agilent 6890GC/5973MSD, Agilent Technologies Japan, Ltd., Tokyo, Japan), following detailed instrumental methods described previously (Fujii et al., 2012, 2013). Briefly, approximately 1 g of diet composite sample, or 0.1 mL of serum sample, was placed in 15 mL polypropylene centrifugation tubes. Then, a internal standard mixture (500 pg of each $^{13}C_4$-labeled PFOA, $^{13}C_5$-labeled PFNA, $^{13}C_2$-labeled PFDA, $^{13}C_2$-labeled PFUnDA, and $^{13}C_2$-labeled PFDoDA in methanol) was added to the tubes. Next, 1 mL of 0.5 mol L^{-1} tetrabutylammonium/0.25 mol L^{-1} sodium carbonate buffer (pH adjusted to 10 using $NaHCO_3$) and 2 mL of MTBE were added to the sample tubes, and the tubes were vortex mixed for 60 s for serum and 60 m for food samples. The samples were centrifuged at 9840 × g for 5 min, then the upper organic layer was transferred to clean tubes. The extraction process was repeated again with the residue, and the upper organic layers were combined in the tube, then concentrated under a stream of nitrogen until 30 μL. The residue was dissolved in 0.1 mL of a 0.1 mol L^{-1} benzyl bromide/MTBE solution containing 10 ng of 11H-PFUnDA as an injection standard. The solution in a sample vial was then derivatized at 60 °C for 1 h, which was sufficient for the benzylation reaction to reach completion (Fujii et al., 2012).

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