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Decline in perfluorooctane sulfonate and perfluorooctanoate serum concentrations in an Australian population from 2002 to 2011

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ABSTRACT

Some perfluoroalkyl and polyfluoroalkyl substances (PFASs) have become widespread pollutants detected in human and wildlife samples worldwide. The main objective of this study was to assess temporal trends of PFAS concentrations in human blood in Australia over the last decade (2002–2011), taking into consideration age and sex trends.

Pooled human sera from 2002/03 (n = 26); 2008/09 (n = 24) and 2010/11 (n = 24) from South East Queensland, Australia were obtained from de-identified surplus pathology samples and compared with samples collected previously from 2006/07 (n = 84). A total of 9775 samples in 158 pools were available for an assessment of PFASs. Stratification criteria included sex and age: <16 years (2002/03 only); 0–4 (2006/07, 2008/09, 2010/11); 5–15 (2006/07, 2008/09, 2010/11); 16–30; 31–45; 46–60; and >60 years (all collection periods). Sera were analyzed using on-line solid-phase extraction coupled to high-performance liquid chromatography–isotope dilution-tandem mass spectrometry.

Perfluorooctane sulfonate (PFOS) was detected in the highest concentrations ranging from 5.3–19.2 ng/ml (2008/09) to 4.4–17.4 ng/ml (2010/11). Perfluorooctanoate (PFOA) was detected in the next highest concentration ranging from 2.8–7.3 ng/ml (2008/09) to 3.1–6.5 ng/ml (2010/11). All other measured PFASs were detected at concentrations <1 ng/ml with the exception of perfluorohexane sulfonate which ranged from 1.2–5.7 ng/ml (08/09) and 1.4–5.4 ng/ml (10/11). The mean concentrations of both PFOS and PFOA in the 2010/11 period compared to 2002/03 were lower for all adult age groups by 56%. For 5–15 year olds, the decrease was 66% (PFOS) and 63% (PFOA) from 2002/03 to 2010/11. For 0–4 year olds the decrease from 2006/07 (when data were first available for this age group) was 50% (PFOS) and 22% (PFOA).

This study provides strong evidence for decreasing serum PFOS and PFOA concentrations in an Australian population from 2002 through 2011. Age trends were variable and concentrations were higher in males than in females. Global use has been in decline since around 2002 and hence primary exposure levels are expected to be decreasing. Further biomonitoring will allow assessment of PFAS exposures to confirm trends in exposure as primary and eventually secondary sources are depleted.

1. Introduction

Perfluoroalkyl and polyfluoroalkyl substances (PFASs) have entered the environment since the 1950s from fluoropolymer manufacturing processes and disposal of products containing fluorochemicals, such as carpet and apparel, pharmaceuticals, fire fighting foams, lubricants, adhesives, cosmetics, paper coatings, leather, pesticides and insecticides (Key et al., 1997; Paul et al., 2009; Prevedouros et al., 2006). Directly emitted PFASs are globally distributed and transported long distances via oceanic transport (Armitage et al., 2009; Wania, 2007). Perfluorinated alkylated acids, one type of PFASs, including perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA), are also distributed through wet and dry deposition as a result of oxidative degradation processes in the atmosphere of volatile precursors, such as fluorotelomer alcohols, perfluorinated sulfonamide alcohols, fluorotelomer acrylates and fluorotelomer olefins (Ellis et al., 2003; Young and Mabury, 2010).

In recent years, PFOS and PFOA have been studied extensively due to their high resistance to both chemical and biological degradation as well as potential for bioaccumulation (Lau et al., 2007). As a consequence of

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their persistence and widespread usage, ubiquitous distribution in both environmental and human samples exists (e.g. Calafat et al., 2007a,b; Giesy and Kannan, 2001; Kannan et al., 2004; Kärrman et al., 2007). Because of its characteristics of toxicity, persistence, bioaccumulation and long range transport, PFOS was listed under the Stockholm Convention on Persistent Organic Pollutants in 2009 (Stockholm Convention on POPs, 2010). Definitive health risks associated with PFAS exposure in humans have not been reported, with studies of persons occupationally exposed to relatively high concentrations showing varying results (Olsen et al., 2003; Wang et al., 2012). Similarly, epidemiological studies of PFASs and various endpoints have also shown varying results. Several authors have reported associations between maternal PFAS concentrations and negative effects with regard to fetal development. Fei et al. (2007) reported PFOA levels were inversely associated with birth weight; Apelberg et al. (2007) found negative associations between both PFOS and PFOA concentrations and birth weight and size; and Darrow et al. (2013) also found a negative association with PFOS and birth weight. Some studies have suggested associations between PFOS and PFOA serum concentrations and thyroid disease (Melzer et al., 2010); and alterations to lipid metabolism (Steenland et al., 2009). Associations have been observed for perfluorohexane sulfonate (PFHxS) but not PFOA and PFOS with an elevated odds of high cholesterol, total cholesterol, low-density lipoprotein cholesterol, total cholesterol/high density lipoprotein (HDL) cholesterol ratio and non-HDL cholesterol (Fisher et al., 2013). An assessment of potentially exposed persons in West Virginia, USA found among other results, probable links between PFOA exposure and diseases such as kidney and testicular cancer (Barry et al., 2013), thyroid disease (Winquist and Steenland, 2014) high cholesterol, ulcerative colitis and pregnancy-induced hypertension (C8 Science Panel, 2014).

Human exposure to PFASs occurs via point sources such as manufacturing plants (Oliaei et al., 2013), food (Clarke et al., 2010; Egeghy and Lorber, 2011; Fromme et al., 2009) and its packaging (Begley et al., 2005), drinking water (Eriksson et al., 2013), and household dust (Fraser et al., 2013; Goosey and Harrad, 2011). Both direct and indirect exposures occur because PFOS and PFOA are stable degradation products/metabolites of other PFASs (Kato et al., 2011).

PFASs were first detected in Australian human blood serum collected in 2002–03 at concentrations equal to or higher than reported in Europe and Asia but lower than in the USA (Kärrman et al., 2006). This finding was unexpected as concentrations of "traditional" persistent organic pollutants such as dioxins and polychlorinated biphenyls have been relatively low in Australia (Harden et al., 2007).

The main objective of this study is to assess temporal trends of PFAS concentrations in Australia over the last decade (2002–2011). Assessment of any temporal trends allows a gauge of the success of the increased regulatory scrutiny in recent years of PFAS in Australia. It can also reflect changes in the pattern and extent of exposure in the Australian population following a global decrease in manufacture and emission of certain PFASs and potential increase in others due to shifts in production. In this study existing data on PFASs in blood from Australians in 2006/07 (Toms et al., 2009), archived samples from 2002/03, and newly collected samples from 2008/09 and 2010/11 will be used to evaluate whether global changes in PFAS usage have affected human exposure to these chemicals in Australia.

2. Materials and methods

2.1. Sample collection

We used archived pooled human sera from 2002/03 (n = 26) and samples collected in 2008/09 (n = 24 pools, 2400 individual samples) and 2010/11 (n = 24 pools, 2400 individual samples) from South East Queensland, Australia. PFAS pooled serum concentrations from 2006/07 (n = 84) were reported previously (Toms et al., 2009). All samples were obtained in collaboration with Sullivan Nicolaides Pathology from de-identified surplus pathology samples. That is, samples were collected from individuals in the community setting for assessment of biochemical parameters; the serum remaining after these assessments was surplus to requirement by the pathology clinic and made available for research purposes. Stratification criteria included age: <16 years (02/03 only); 0–4 (08/09, 10/11); 5–15 (08/09, 10/11); 16–30; 31–45; 46–60; and >60 years (all collection periods). As reported and discussed in detail in Toms et al. (2009), the 2006/07 samples were stratified as follows: 0–0.5; 0.6–1; 1.1–1.5; 1.6–2; 2.1–2.5; 2.6–3; 3.1–3.5; 3.6–4; 4.1–6; 6.1–9; 9.1–12; 12.1–15 years. For comparative purposes, in this study these age brackets will be combined into 0–4 years or 5–15 years as appropriate for comparison with age groups from other collection periods. Both males and females were included. Each pool consisted of up to 100 samples (see Harden et al., 2007 for details), with the exception of the 2006/07 pools that consisted of approximately 30 samples (see Toms et al., 2009 for details). It was not possible to determine if any one donor contributed to more than one collection period. Ethics approval for this study was granted by the University of Queensland Medical Research Ethics Committee. The involvement of investigators at the Centers for Disease Control and Prevention (CDC) was determined not to constitute engagement in human subjects research [45 CFR 46.101(d)].

2.2. Measurement of PFASs in serum

All samples were analyzed at the Division of Laboratory Sciences, National Center for Environmental Health, CDC, Atlanta, USA by a modification of the Kuklenyik et al. (2005) approach, involving on-line solid-phase extraction coupled to high-performance liquid chromatography–isotope dilution-tandem mass spectrometry (Calafat et al., 2007a,b). Limits of detection (LOD) were 0.2 ng/ml (2-(N-ethyl-perfluorooctanesulfonamido) acetate [Et-PFOSA-AcOH], 2-(N-methyl-perfluorooctanesulfonamido) acetate [Me-PFOSA-AcOH], perfluorodecanoate [PFDeA]) and 0.1 ng/ml (PFHxS, perfluorononanoate [PFNA], PFOA, PFOS, perfluorooctanesulfonamide [PFOSA]) (Calafat et al., 2007a,b). Quality control/quality assurance (QC/QA) included sampling replication of pools for a given strata and analysis of blank samples. CDC researchers received coded samples and were blind to the pools' characteristics. Analytical standards, low- and high-concentration QC samples (prepared from spiked calf serum) and reagent blank samples were included in each analytical batch along with the study samples (Kuklenyik et al., 2005).

Moreover, for the 2006/07, 2002/03 and 2008/09 pools, two, two and one samples of calf serum (Sigma Aldrich B8655), respectively, known to have concentrations of the target PFASs below the LOD, were aliquoted, pooled, stored, shipped and analyzed using identical procedures to human blood sera. No PFASs were detected in these blank samples. Sample replication between pools of the same strata (i.e., two pools which were obtained for the same age and sex) was assessed using the normalized difference ($([a–b]/([a + b]/2)) \times 100\%$) (where a is the value from Pool 1 and b is the value from Pool 2) and the average described as the mean normalized difference (MND) for all age groups and both sexes combined. In 2008/09, the MNDs for PFOS and PFOA were 13% and 10%, respectively and in 2010/11, the MNDs for PFOS and PFOA were 18% and 16%, respectively. This agreement between replicates and absence of PFASs in the blank serum suggests that the pooling procedures were uniform and contamination during sampling or analysis was unlikely.

2.3. Statistical analysis

Statistical analysis was mainly descriptive to estimate average concentrations (means or medians as appropriate) and standard deviations or ranges. ANOVA models with the Tukey's post test carried out using GraphPad Prism 5. The conventional 5% cut-off was used to report