

Polyfluoroalkyl chemicals (PFCs) in Australian human blood serum

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Introduction

Polyfluoroalkyl chemicals (PFCs) have been used in a wide variety of industrial and consumer products including refrigerants, surfactants and polymers. In addition, they are used as components of carpet and apparel, pharmaceuticals, fire retardants, lubricants, adhesives, cosmetics, paper coatings and insecticides¹. Several PFCs are environmentally persistent² and have been detected in environmental and human samples in many countries^{3,4}.

A previous study determined the concentrations of PFCs in human blood serum from the Australian population collected in 2002/03 based on age, gender and geographical region⁵. No difference was observed for PFC concentrations between residents from urban and rural regions. For perfluorooctane sulfonate (PFOS) there was an increase in concentration with age while for perfluorohexane sulfonate (PFHxS), perfluorooctane sulfonamide (PFOSA) and perfluorooctanoate (PFOA) there appeared to be a trend of higher concentrations in younger age (<16 years) relative to the groups between 16 and > 60 years. Moreover, PFC concentrations in the Australian population were similar or higher than concentrations in populations in Europe and Asia but lower than those reported from the USA⁵. There is limited information on the pathways of PFC exposure and unlike 'traditional' persistent organic pollutants, food consumption may or may not be the main route of exposure⁶. Other suggested pathways include drinking water, particularly for residents near fluoropolymer production facilities⁷.

The aim of the current study was to assess whether the observed age differences from the previous study⁵ were reproducible and to assess PFC exposure in younger age groups in the Australian population in an attempt to focus future exposure studies.

Materials and Methods

Human blood sera was collected and pooled for analysis in 2006/07. De-identified serum samples were obtained from Sullivan and Nicolaides Pathology from surplus stored sera that had been collected as part of routine pathology testing. The original ethics approval for blood collection was granted on 20 September 2002 by The University of Queensland Medical Research Ethics Committee with an amendment to collect samples from the below age brackets approved on 28 June 2006.

Overall, 2420 individual serum samples were collected from South-East Queensland, Australia and combined to make 84 pools. Prior to pooling, all samples were stratified according to age and gender. The age groups were: cord blood; 0-6; 6-12 months; 1-1 ½; 1 ½-2; 2-2 ½; 2 ½-3; 3-3 ½; 3 ½-4; 4-6; 6-9; 9-12; 12-15; 16-30; 31-45; 46-60 and >60 years.

The samples were analysed at the Division of Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, USA by an on-line solid-phase extraction method coupled to high-performance liquid chromatography-isotope dilution-tandem mass spectrometry using a modified method of Kuklenyik et al⁸.

Results and discussion

Here we report the results of four PFCs: perfluorooctane sulfonate (PFOS), perfluorooctanoate (PFOA), perfluorohexane sulfonate (PFHxS) and perfluorononanoate (PFNA). Results are reported in ng/ml of serum, excluding the limit of detection (LOD). PFOS was detected at the highest mean concentrations (15.2 ng/mL) followed by PFOA (6.4 ng/mL), PFHxS (3.1 ng/mL) and (PFNA, 0.8 ng/mL) (Table 1). These findings confirm that exposure to PFCs is widespread in Australia.

Table 1. Summary results of PFC concentrations (ng/mL) in 84 pools of human blood serum.

	Frequency of detection	Range	Mean	Standard deviation	Median
PFHxS	95%	<LOD - 11.3	3.1	2	2.9
PFNA	100%	0.1 - 1.4	0.8	0.25	0.8
PFOA	100%	0.8 - 9.1	6.4	1.5	6.4
PFOS	100%	5 - 28.5	15.2	4.9	14.8

PFHxS - perfluorohexane sulfonate; PFNA – perfluorononanoate; PFOA – perfluorooctanoate; PFOS - perfluorooctane sulfonate

Mean concentrations of most PFCs were generally higher in pools of males compared to females, although the differences in concentrations by gender were only statistically significant for PFOS which was weakly significant ($p=0.05$). Higher concentrations of PFCs in males compared to females have been reported previously with gender differences affecting exposure and/or pharmacokinetic reasons for differences⁹.

The concentrations of PFCs measured in this study appear to be influenced by age, although there is no consistent trend for all PFCs. For the PFCs detected at the highest concentrations - PFOS, PFHxS and PFOA, concentrations increase from birth (cord blood) to peak between 3-13 years then decrease until around 23 years when a gradual increase occurs for PFOS, while concentrations of PFOA and PFHxS plateau (Figure 1). The differences in PFC concentration by age were only significant for PFOS ($p<0.001$). Some previous studies have reported age trends in some PFC concentrations, with higher concentrations in younger age groups for PFOS⁵ and PFHxS & Me-PFOA-AcOH¹⁰. The varying pattern of PFC body burden by age differs from patterns of persistent organic pollutants or for example, polybrominated diphenyl ethers where congeners have different levels of concentration but follow the same trend by age. This indicates that exposure to these four PFCs is not uniform. Notably, due to different PFCs being used in different products and processes it may be expected that exposure sources will have varying impacts. The elevated concentrations in the younger age groups compared to the older age groups are consistent with the findings of the previous study on Australian pooled samples.

Overall, the PFOS concentrations found in this study were lower than those reported in the U.S.¹¹ and in Poland, higher than South America, Italy and India and similar to Belgium, Malaysia, Korea and Japan⁴. For PFHxS, the concentrations were higher than Colombia, Italy, Poland, Belgium, India and Malaysia⁴ while similar to the U.S.¹¹, Brazil, Korea and Japan⁴. For PFOA, the Australian concentrations were lower than found in Poland, Korea, Japan (females), higher than Brazil, Italy, Belgium, India and Malaysia⁴ and lower than or similar to the U.S.¹¹ and Colombia⁴.

When compared to previous data on PFCs in Australia, the results of the current study are similar or slightly lower which could indicate that exposure has changed little since 2002/03. However, the difference in the age brackets for the younger age groups which would be expected to be more 'reactive' to changes in recent exposure complicates the interpretation. Furthermore since different analytical methods for analyzing the pooled samples collected in 2002/03 and in 2006/07 were used, we cannot rule out that the observed trends may be related to methodological differences.

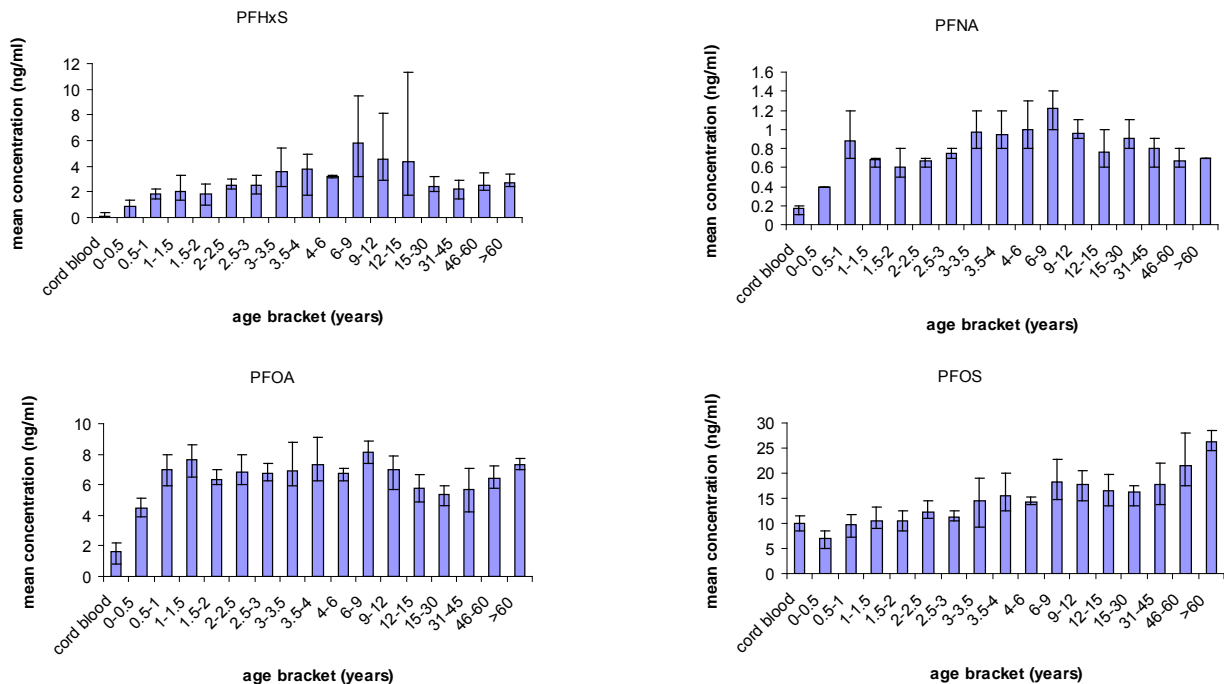


Figure 1. Mean \pm range (ng/mL serum) of PFHxS, PFNA, PFOA and PFOS by age bracket (years) for males and females combined (note change of scale on y-axis for different PFCs)

Since PFCs are not manufactured in Australia, exposure may occur from emissions during application of these chemicals and from use and disposal of products containing PFCs. Further investigation of the sources and exposure pathways of PFCs in the Australian population is required to elucidate the trends in concentrations by age.

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