POLYFLUOROALKYL COMPOUNDS (PFCS) IN TEXAS CHILDREN FROM BIRTH THROUGH 12 YEARS OF AGE

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Introduction

Polyfluoroalkyl compounds (PFCs) are chemicals that contain a carbon-fluorine chain attached to hydrophilic heads. Several PFCs are persistent, nonbiodegradable, and can be transported over long distances[1]. PFCs have many industrial and consumer applications, including use as surfactants and emulsifiers in food packaging, non-stick pan coatings, fire fighting foams, paper and textile coatings, and personal care products[2]. In 2002, 3M, a major North American producer of PFCs, discontinued production of PFOS related compounds, the ammonium salt of PFOA, and other perfluorooctanesulfonyl fluoride based products[3-4]. In humans, PFC exposure has been associated with reduced semen quality[5], thyroid disease[6], high serum lipids[7], reduced fecundity[8], and low birth weight[9]. In animals, PFC exposure has been associated with changes in thyroid hormone levels[10], immune system alterations[11], and neurotoxicity[12].

Assessing human exposure to PFCs provides useful information for understanding the potential adverse health effects from population exposure to PFCs. The Centers for Disease Control and Prevention (CDC), as part of the National Health and Nutrition Examination Survey (NHANES), provides an exposure assessment of the U.S. general population to polyfluoroalkyl compounds (PFCs) by measuring the concentrations of these compounds in serum[10]. Currently, NHANES only reports blood serum analysis for PFCs for individuals 12 years and older. To address this data gap, we investigated serum concentrations of perfluorooctane sulfonic acid (PFOS), perfluorooctane sulfonamide (PFOSA), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorohexane sulfonic acid (PFHxS), 2-(N-ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH), 2-(N-methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH), and perfluorodecanoic acid (PFDeA) in 300 infants and children ranging from ages 0 – 12 years from Dallas, Texas. To the best of our knowledge, we provide the first estimate of serum PFC levels in a sample of U.S. children since the 2002 changes in U.S. PFCs production[3-4].

Materials and methods

This study was approved by the Institutional Review Board (IRB) committees for the protection of human subjects at the University of Texas Southwestern Medical Center and the University of Texas Health Science Center at Houston. The protocol was reviewed by the CDC IRB and determined to fall under the exempt category because of the study design and lack of access to personal identifiers by researchers. Serum samples were collected from August 2009 to November 2009 at Children's Medical Center in Dallas, Texas for pathology tests and stored frozen at -80°C. Approximately 0.5 to 1.0 mL of residual serum from each child was collected at the Children's Medical Center Department of Pathology and Laboratory Medicine and sent on dry ice to CDC for chemical analysis. We collected 300 samples, 25 for each of the following age groups (in years): 0 – 1; 1 – 2; 2 – 3; 3 – 4; 4 – 5; 5 – 6; 6 – 7; 7 – 8; 8 – 9; 9 – 10; 10 – 11; and 11 – 12. We measured the serum concentrations of eight PFCs by on-line solid phase extraction–high performance liquid chromatography–isotope dilution–tandem mass spectrometry as previously described[11]. The limits of detection (LOD) in 0.1 mL of serum were: 0.1 ng/mL (PFOA, PFNA, PFHxS, PFOSA) and 0.2 ng/mL (PFOS, PFDeA, Et-PFOSA-AcOH, Me-PFOSA-AcOH).

Results and discussion:

Table 1 shows summary statistics for serum concentrations of PFCs measured in 300 serum samples collected in 2009 from Dallas, Texas children (157 girls and 143 boys) ranging from 0 to 12 years of age. The frequencies of

detection of the analytes ranged from < 1% to 98% and were ≥ 93% for PFHxS, PFOS, PFOA, and PFNA. The frequencies of detection of the analytes were not significantly different for girls and boys and increased with age. For all study subjects, median serum concentrations were 1.2 ng/mL (PFHxS, PFNA), 2.85 ng/mL (PFOA), and 4.1 ng/mL (PFOS). Infants less than one year old (n=25) had serum concentrations that ranged from non-detectable to 4.0 ng/mL (PFHxS; median = 0.5 ng/mL), from non-detectable to 2.2 ng/mL (PFNA; median = 0.6 ng/mL), from non-detectable to 8.8 ng/mL (PFOA; median = 1.9 ng/mL), and from non-detectable to 6 ng/mL (PFOS; median = 2.4 ng/mL). These serum concentrations are similar to those detected in composite blood spots from New York infants born between 1997 and 2007, which ranged from 1.22 – 2.46 ng/mL for PFHxS, 0.27 – 0.51 ng/mL for PFNA, 0.34 – 1.41 ng/mL for PFOA, and 0.81 – 2.41 ng/mL for PFOS[13]. These serum concentrations are also similar to median concentrations in dried blood spots among a group of 98 infants born in Texas in May 2007 (PFHxS and PFNA: 0.3 ng/mL, PFOA: 0.9 ng/mL , and PFOS: 2.2 ng/mL)[12]. We did not observe statistically significant differences in serum concentrations in any of the PFCs examined based on sex (Wilcoxon rank-sum test p>0.05).

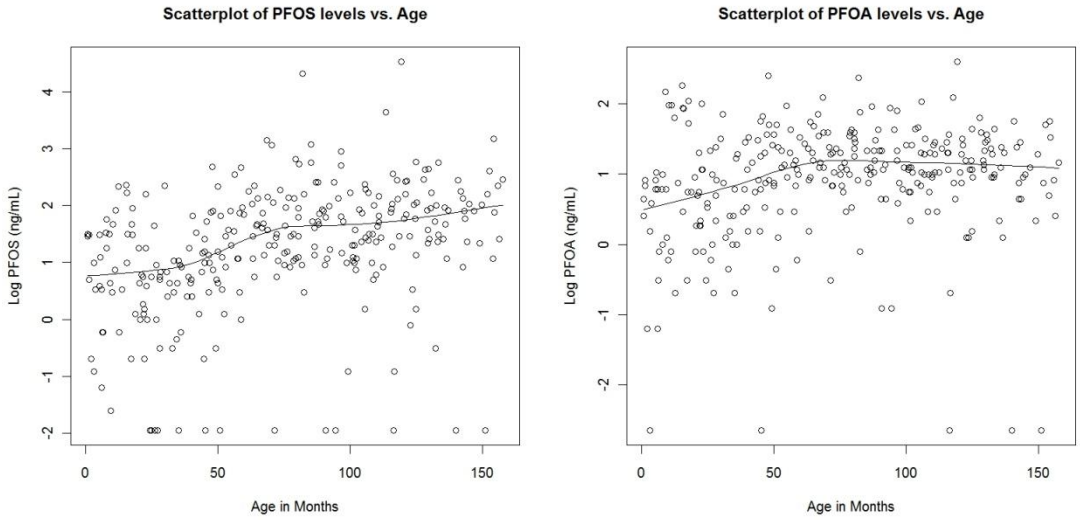
Table 1. Summary Statistics for Serum Concentrations in Children 0-12 years old from Dallas, Texas (n=300)

	Detection Frequency (%)			Limit of detection (ng/mL)	Arithmetic Mean [a] (ng/mL)			Median (ng/mL)			Maximum (ng/mL)	
	Boys (n=143)	Girls (n=157)	All		Boys	Girls	All	Boys	Girls	All	Boys	Girls
Et-PFOSA-AcOH	0	<1	<1	0.2	NA	NA	NA	<LOD	<LOD	<LOD	<LOD	0.7
Me-PFOSA-AcOH	36	36	36	0.2	NA	NA	NA	<LOD	<LOD	<LOD	28.9	13.4
PFDeA	12	15	14	0.2	NA	0.21	NA	<LOD	<LOD	<LOD	1	2.1
PFHxS	93	92	93	0.1	1.81	1.45	1.62	1.2	1.15	1.2	31.2	9.6
PFNA	96	98	97	0.1	1.94	1.35	1.63	1.2	1.25	1.2	55.8	4.2
PFOA	97	99	98	0.1	3.28	3.08	3.17	2.7	2.9	2.85	13.5	9.6
PFOS	94	97	96	0.2	6.14	5.32	5.71	4.15	4.1	4.1	93.3	38.3
PFOSA	1	1	1	0.1	NA	NA	NA	<LOD	<LOD	<LOD	0.6	0.3

[a] NA: Not applicable (mean calculated if detection frequency was > 60%).

Figure 1 shows scatterplots of serum concentration of PFOS and PFOA by age, with a LOESS curve overlaid. Spearman rank correlations were significant (p<0.001 for PFOS, PFNA, PFHxS, and PFOA), suggesting that an increase in age is associated with an increase in serum concentrations of these PFCs. The observed increase until age 5 years may be partially related to PFCs exposure from food and dust intake. Younger children in our study population may have experienced lower exposure to some PFCs than older children as PFOS production has changed and PFOA manufacturing release is lower than in the early 2000's. Stratifying the analysis to those children born before the 2002 PFOS phase-out compared to children born after revealed significantly higher concentrations only of PFOS and PFHxS in children born before 2002 (Wilcoxon rank-sum p-value<0.0001).

Figure 1. Scatterplot of Age vs. Serum PFOS/PFOA Concentrations (ng/mL) with LOESS Smoothing Curve



We detected five PFCs in at least one third of the tested serum samples. These results suggest that U.S. children continued to be exposed to several PFCs at least until 2009, several years after discontinued production of PFOS and of PFOA at some U.S. facilities occurred. Further research is necessary to determine if, at the measured concentrations, PFCs are associated with adverse health events in children, the extent to which children are exposed to PFCs, and the main exposure pathways.

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