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Authors

Zhu, Yachen
Ro, Annie
Bartell, Scott M

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Household low pile carpet usage was associated with increased serum PFAS concentrations in 2005-2006

Yachen Zhu¹, Annie Ro¹, Scott M. Bartell^{1,2,3}

¹Program in Public Health, University of California, Irvine, CA 92697-3957, USA

²Department of Environmental and Occupational Health, University of California, Irvine, CA 92697-1250, USA

³Department of Statistics, University of California, Irvine, CA 92697-1250, USA

Abstract

Background: Per- and polyfluoroalkyl substances (PFAS) are ubiquitous in the serum of the general US population. Food, drinking water, consumer products, dust, and air have been assessed as PFAS exposure sources for humans. The effects of various types of carpeting on serum PFAS concentrations have been less studied, despite the known use of PFAS in stain-resistant carpet treatments.

Objective: This study aimed to examine the associations between serum PFAS concentrations and type of residential flooring among the general US population aged 12 years and older using the 2005–2006 National Health and Nutrition Examination Survey (NHANES).

Methods: We used multiple linear regressions adjusted for complex survey design and relevant covariates to analyze the relations between serum PFAS concentrations and type of floor covering (smooth surface, low pile carpet, medium to high pile carpet, and combination of carpet and smooth surface), as well as other potential exposure factors. We used multiple imputation to address missing values.

Results: We found significantly higher serum concentrations of perfluorohexane sulfonic acid (PFHxS) and 2-(*N*-methyl-perfluorooctane sulfonamido) acetic acid (MeFOSAA) in US residents residing in homes with low pile carpeting compared with those residing in homes with smooth surface. We concluded that among US residents aged 12 years and older residing in homes with low pile carpeting in the home in 2005–2006, on average 24% and 19% of the PFHxS and MeFOSAA body burdens, respectively, could be attributed to carpeting. We found associations between other types of floor covering (medium to high pile carpet, combination of carpet and

Corresponding Author: Scott M. Bartell, Department of Environmental and Occupational Health, Program in Public Health, University of California, Irvine, Irvine, CA 92697-1250, USA, sbartell@uci.edu.

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Scott M. Bartell serves as a compensated expert witness for PFOA medical monitoring lawsuits in New Hampshire. The terms of this arrangement were reviewed and approved by the University of California, Irvine in accordance with its conflict of interest policies.

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smooth surface) and some PFAS concentrations compared with the smooth surface, but these results were less consistent and generally not statistically significant. Additionally, a group Wald Chi-squared test showed a significant result for PFOS, indicating different contributions of various types of flooring to PFOS serum concentration.

Significance: Our results are representative of the general US population at the time of the survey, and potentially informative regarding ongoing PFAS exposure from a variety of sources including carpeting.

Keywords

PFAS exposure; carpeting; serum; sampling survey

Introduction

Per- and polyfluoroalkyl substances (PFAS) are a group of human-made chemicals that widely exist in the environment, biota, and humans. Due to the strong single bond between carbon (C) and fluorine (F), which requires very high energy to dissociate, PFAS have unique physiochemical characteristics such as stability in extreme temperatures and water resistance (Faithfull and Weers, 1998). Strong carbon-fluorine bonds also make PFAS resistant to hydrolysis, microbial degradation, and metabolism by vertebrates (ATSDR, 2018). Therefore, they have been used in a variety of applications, including textiles, carpeting, food packaging, nonstick cookware, and firefighting foams (Kissa, 2001). They persist indefinitely in the environment and bioaccumulate in humans and organisms (Buck et al., 2011). For example, the average half-lives for serum or plasma elimination of PFAS in humans were reported as about 2–4 years for perfluorooctanoic acid (PFOA) (Bartell et al., 2010; Olsen et al., 2007; Worley et al., 2017), 3–27 years for perfluorooctane sulfonic acid (PFOS), 5–35 years for PFHxS (Olsen et al., 2007; Worley et al., 2017; Li et al., 2018; Zhang et al., 2013), and 2–4 years for perfluorononanoic acid (PFNA) (Zhang et al., 2013) in previous studies. Some studies have found associations between PFAS and a series of adverse health effects such as reduced fecundity (Vélez et al., 2015), low birthweight (Johnson et al., 2014), and cancers (Vieira et al., 2013).

Perfluoroalkyl carboxylic acids (PFCA, e.g., PFOA and PFNA) and perfluoroalkane sulfonic acid (PFSA, e.g., PFOS and PFHxS) both belong to perfluorinated acids (PFA) in the PFAS family (Buck et al., 2011; D'eon and Mabury, 2011). Long-chain PFAS (PFCA with 8 carbons and PFSA with 6 carbons) have been shown to be more bioaccumulative than their short-chain analogues (Martin et al., 2003; Olsen et al., 2009), and therefore have attracted more attentions from the global scientific and regulatory community (Buck et al., 2011; US EPA, 2009). There are two main methods to produce PFAS: electrochemical fluorination (ECF) and telomerization. The ECF method was used by the 3M Company from 1949 to 2001 to manufacture perfluorooctanesulfonyl fluoride (POSF)-based materials (including PFOS, MeFOSAA, EtFOSAA, and high-molecular-weight fluorinated polymers), PFCA (such as PFOA and PFNA) and their ammonium salts (Beesoon et al., 2011; Buck et al., 2011; D'eon and Mabury, 2011). The ECF technique resulted in a mixture of linear (70–80%) and branched (20–30%) isomers, while the telomerization method initially developed by the DuPont Company in the 1970s produced almost completely linear isomers. Starting

from 2002, the 3M Company, the major global manufacturer of PFOA, PFOS and related perfluorooctanesulfonyl fluoride compounds, ceased its production of these substances using the ECF technique due to health concerns, and has since introduced short-chain perfluoroalkane sulfonates and products such as perfluorobutane sulfonic acid (PFBS) (Zushi et al., 2012). In 2006, EPA initiated a global stewardship program to achieve a 95% reduction in PFOA and its precursors by 2010, and work towards the elimination of these chemicals by 2015. During this time the manufacture of PFOA, PFOS and their precursors was largely relocated to Asia, especially China (UNEP, 2015; Yue, 2008), where other manufacturers continued to use the ECF process to develop these substances (Buck et al., 2011). Unlike PFOS, which was almost solely produced using the ECF technique, PFOA could be manufactured through not only the ECF method, but also telomerization, which was still in use after 2002 to produce fluorotelomer-based chemicals including polyfluoroalkyl phosphate esters (PAPs) and PFOA linear isomers. Additionally, some fluorotelomer-based materials such as PAP diesters (diPAPs) can be enzymatically hydrolyzed to produce 8:2 fluorotelomer alcohol (8:2 FTOH), an intermediate metabolite that is subsequently transformed into perfluoroalkyl carboxylic acids (PFCA) such as PFNA, PFOA, and PFHpA (Buck et al., 2011; D'eon and Mabury, 2007; D'eon and Mabury, 2011). NHANES data have accordingly shown a significant downward trend in serum concentrations of PFOS and PFHxS in the general US population since 1999 (Calafat et al., 2007; Kato et al., 2011). However, PFOA did not decline as much as expected after the phase-out by 3M (Olsen et al., 2008; Beesoon et al., 2011) and remained essentially unchanged, and may have increased during 2003–2008 in the US (Kato et al., 2011) before declining slowly since 2008 (CDC, 2019). The percentage of linear isomer for PFOA was also found to be increasing during 1997–2012 in Sweden, indicating the ongoing production of fluorotelomer-based chemicals (Gebbink et al., 2015).

According to the Carpet and Rug Institute, most commercial and residential carpets and rugs in the US have been treated with perfluorooctane sulfonyl fluoride (POSF)-based materials in the manufacturing process to achieve stain resistance (DTSC, 2018). For example, sulfonamido-ethanol (MeFOSE) is a raw material used in textile and carpet products, and can be metabolized into MeFOSAA (aka M570), a precursor of PFOS; PFOS was a key ingredient in Scotchgard and had been extensively produced for use in carpet treatment until the phase-out. Perfluorohexane sulfonyl fluoride (PHxSF)-based derivatives such as PFHxS had also been produced by the 3M Company until the phase-out parallel to the phase-out of POSF-based products, and was used in specific postmarket carpet treatment products (Wang et al., 2014; Olsen et al., 2003). In 2009, PFOS and its precursors were added to Annex B of Stockholm Convention on Persistent Organic Pollutants (POPs). While this is an important step to restrict the production and use of PFOS and its precursors, a list of specific exemptions associated with this treaty still allows the mass production and almost all the historic uses of PFOS and its precursors, including the use in carpets, leather and apparel, textiles and upholstery, paper and packaging, coating and coating additives, and so forth (UNEP, 2010).

Dust ingestion has been associated with stain-resistant treatment of carpets or rugs, which is an important pathway for PFAS exposure (D'Hollander et al., 2010; Beesoon et al., 2012; Harris et al., 2017; Hu et al., 2018; Hurley et al., 2018; Karásková et al., 2016). Previous

studies have indicated the ubiquitous presence of diPAPs, 8:2 FTOH, PFCA, and some POSF-based materials in indoor dust or air, which could come from consumer products such as carpets, upholstery, and textiles (De Silva et al., 2012; Fraser et al., 2012; Kato et al., 2009; Strynar et al., 2008; Winkens et al., 2018; Kubwabo et al., 2005). In a Canadian study conducted by Shoeib et al. (2005), MeFOSE and N-ethylperfluorooctane sulfonamidoethanol (EtFOSE) were detected at the median concentrations of 110 ng/g and 120 ng/g in indoor air, respectively, 10–20 times higher than their outdoor concentrations. EtFOSE is a POSF-based raw material used in paper and packaging products, and it can be metabolized into EtFOSAA (Buck et al., 2011). Both EtFOSAA and MeFOSAA are precursors of perfluorooctane sulfonamidoacetic acid (FOSAA, aka M556), perfluorooctane sulfonamide (FOSA), and PFOS. A US study by Strynar and Lindstorm (2008) found ubiquitous existence of PFOS and PFOA in house dust, with median concentrations of 201 and 142 ng/g, respectively. 8:2 FTOH, an important precursor for PFOA, was also detected with a maximum concentration of 1660 ng/g in the house dust. These results from previous studies highlight the potential of house dust and air as important pathways for human PFAS exposure.

Despite efforts to remove PFOA, PFOS, and PFOS precursors (MeFOSAA and EtFOSAA) produced using the ECF method, telomerization continued to be used to manufacture PFOA and related chemicals after the 2002 phase-out. A recent review paper revealed the large uncertainty about the amount of PFAS manufactured and imported due to the fact that a large part of the amounts has been claimed as confidential business information (CBI) and only substances manufactured or imported at above 11.34 tonnage per year at a single site have been reported (Glüge et al., 2020); also, PFAS that can break down into PFOA and PFOS are still in use in the US (BloombergLaw, 2020). Additionally, commercial and residential carpets treated with POSF-based materials such as MeFOSAA, PFOS, and PFHxS may not be replaced as frequently as other products. Thus, the PFAS-treated carpets before the phase-out may continue to be used in households and commercial settings. Besides, some short-chain PFAS have been introduced as alternative chemicals to long-chain PFAS (Birnbbaum et al., 2015; Gomis et al., 2018). For example, PFBS has been extensively produced and used as a replacement of PFOS in the ScotchGard formulas, and perfluorobutanoic acid (PFBA) has also been introduced as an alternative to PFOA in recent years, which may also have adverse health effects despite their relatively shorter half-lives (Buck et al., 2011; Olsen et al., 2009; Eschauzier et al., 2010; Liu et al., 2020).

Some studies have reported positive associations between PFAS concentrations in serum or indoor dust and carpet (Beesoon et al., 2012; Harris et al., 2017; Hu et al., 2018; Hurley et al., 2018; Karásková et al., 2016). However, none of these studies distinguished between different types of carpets. Because different types of carpets were designed for different social settings and frequency of foot traffic, they may present distinct patterns of PFAS exposure contribution. For example, low pile carpets are designed for and more common in places with larger traffic than medium to high pile carpets, and may have different PFAS treatment. We here extend the previous work by investigating and quantifying the contribution of PFAS exposure from various types of floor coverings using the NHANES public-use database. In this study, we also accounted for other previously reported exposure pathways to PFAS, including dietary intake of fish and shellfish (Christensen et al., 2017),

fast food consumption (Susmann et al., 2019), water contamination by firefighting foams near military sites (Hu et al., 2016), and tap water sources (Shin et al., 2011a; Shin et al., 2011b). We also accounted for kidney function (Jain et al., 2019) and special PFAS excretion pathways for females, including menstrual blood loss (Wu et al., 2015), maternal transfer to offspring through pregnancy (Beesoon et al., 2011) and breastfeeding (Fromme et al., 2010). To our knowledge, this is the first analysis that distinguishes low pile carpet from medium/high pile carpet, and focuses specifically on the association between carpet type and PFAS, which can inform PFAS exposure assessment from carpet more comprehensively.

Methods

We obtained data from the 2005–2006 NHANES data files. NHANES uses a complex, multistage, stratified, clustered, probability sampling design to select participants representative of the civilian, non-institutionalized US population (NCHS, 2012). It is a cross-sectional survey designed to monitor the health and nutritional status of adults and children in the US. NHANES survey includes interviews, physical examinations for every participant and laboratory tests for a subsample (NCHS, 2013). Sampling weights were created in the sample and subsamples in each 2-year cycle to account for oversampling, non-response, and poststratification in the complex survey design (NCHS, 2012). Our analyses were restricted to the 2005–2006 cycle based on the availability of the key variable of interest: type of floor covering. This variable was not included in subsequent cycles of NHANES.

Serum PFAS Measurements.

In the 2005–2006 NHANES data, 12 PFAS were measured in a one-third subsample of eligible participants aged 12 years and older using tandem mass spectrometry, including PFOA, PFOS, PFHxS, 2-(*N*-ethyl-perfluorooctane sulfonamido) acetic acid (EtFOSAA), MeFOSAA, perfluorodecanoic acid (PFDA), PFBS, perfluoroheptanoic acid (PFHpA), PFNA, perfluorooctane sulfonamide (PFOSA), perfluoroundecanoic acid (PFUnDA), and perfluorododecanoic acid (PFDoA). Procedures for collecting, storing, and handling specimens, and quality control have been described elsewhere (CDC, 2013). PFAS serum concentrations below the limit of detection (LOD) were substituted with a value of LOD divided by $\sqrt{2}$ (CDC, 2013). We restricted our analyses to the six PFAS (i.e., PFOA, PFOS, PFHxS, MeFOSAA, PFDA, and PFNA) with detection rates of >70% in the sample to avoid bias from the substitutions. The LOD was 0.1 ng/ml for PFOA, PFHxS, and PFNA; and 0.2 ng/ml for PFOS, PFDA, and MeFOSAA (CDC, 2012).

Explanatory Variables.

We obtained NHANES variables relevant to known or suspected PFAS exposure pathways, including the type of floor covering (collected using household interview questionnaires) from the dust allergen subsample in the laboratory data; tap water source, self-reported fish and shellfish consumption in the past 30 days from the dietary interview component; the frequency of eating out per week from the dietary behavior questionnaire; serum creatinine (an indicator of kidney function) from the standard biochemistry profile; whether or not had at least one menstrual period in the past 12 months, number of pregnancies, and number of

children breastfed for at least one month from the reproductive health questionnaire data. We also obtained age, sex, race/ethnicity, education, family poverty income ratio (PIR), veteran/military status, country of birth, and body mass index (BMI) from the demographic component of the survey. These have been established as predictors for serum PFAS concentrations in previous studies (Calafat et al., 2007; Kato et al., 2011; Christensen et al., 2017; Susmann et al., 2019; Hu et al., 2016; Zhang et al., 2010; Jain, 2014). We recategorized race/ethnicity as Hispanic (including Mexican American and other Hispanic), non-Hispanic white, non-Hispanic black, and the others. We categorized education as less than college, some college, and above college.

The type of floor covering was categorized as smooth surface, low pile carpet, medium to high pile carpet, and combination of carpet and smooth surface. We recategorized the frequency of eating out per week as “Yes” and “No” based on the provided combination of numbers (1–21 times/week) and categories (never, more than 21 times per week, less than weekly) in the original variable, because this variable is not fully numeric. We checked the linearity of the relations between serum PFAS concentrations and age, family PIR, BMI, serum creatinine, number of pregnancies, and number of children breastfed for at least one month by examining both the scatter plots and plots of residuals against each of the predictor variables, and included these predictors as continuous variables in the regression models. We treated all “Refused” and “Don’t know” answers in the data as missing values. We checked data on sex, menstrual period, pregnancy, and breastfeeding against each other. For males, missing values in reproductive health information were substituted with “No” (menstrual period) or 0 (number of pregnancies and number of children breastfed). For females aged 55 and older, missing values in “at least menstrual period in the last 12 months” were substituted with “No”. For females who were never pregnant, missing values in the number of pregnancies and the number of children breastfed for at least one month were substituted with 0. For females who never breastfed, the number of children breastfed for at least one month were also substituted with 0. After these substitutions, most variables have missing rates less than 10% except for type of floor covering (26.9%) and veteran/military status (19.4%). We compared the missing percentage of these variables with respect to other variables, and found the type of floor covering is missing at random (MAR) with respect to race/ethnicity and education; and veteran/military status is MAR with respect to country of birth, race/ethnicity, and education.

Statistical Analysis.

We fit multiple linear regression models adjusted for covariates using log-transformed serum PFAS concentrations while accounting for complex survey design (R package *survey*). We adjusted for the sampling weights of the serum PFAS subsample, the smallest analysis subpopulation in this study, for parameter estimates in regression models to reflect the probability of selection, nonresponse, and post-stratification (NHANES, 2020a). We also accounted for the pseudo-stratum and pseudo-PSU variables in order to produce asymptotically unbiased variance estimation in our regression models (NHANES, 2020b). Because of the dispersed distribution of missing values in different variables, listwise deletion of missing data that is performed as default in most statistical software packages including R would substantially reduce the sample size by 55%. Although listwise deletion

is acceptable when some observations are “missing completely at random” (MCAR) without respect to any of the other variables, multiple imputation performs better when missingness is covariate-dependent (Little and Rubin, 2020). We used multivariate imputation by chained equations (MICE) to create k=100 imputed datasets (R package *mice*). We applied Rubin’s rules (Rubin, 1987) to pool the results from k=100 analyses accounting for complex survey design based on the imputed datasets, averaging the estimates and computing the total variance over the repeated analyses. We also compared the results from regression analyses with those of complete case analysis (analysis of data after listwise deletion). We exponentiated the regression coefficients, subtracted by one, and multiplied by 100% to estimate the percent difference in PFAS concentrations associated with each predictor. For variables with more than two categories/levels, Wald Chi-squared tests of equivalence on multiple parameters can be used to test their effects. For example, to test the hypothesis that multiple types of flooring have no effect on serum PFAS concentrations, we conducted a simultaneous Wald Chi-squared test of equivalence for all parameters related to type of flooring. Because the education level of adolescents are mostly determined by their age rather than family socioeconomic status, in sensitivity analyses, we ran the analysis for adults (aged > 19) and adolescents (aged 12–19) separately.

Because PFAS do not tend to accumulate in the fat tissues (lipophobic property) (Benford et al., 2008), we did not include BMI (an indicator of body fatness) in the primary multiple regression models. PFAS has been suggested as a potential “cause” of body weight change (Liu et al., 2018), not the other way around, in which case, BMI would not confound the associations between type of flooring and serum PFAS; thus adjustment for BMI would not be necessary and could even induce selection bias under certain conditions (Rothman, 2012). In sensitivity analyses, we also fit multiple regression models with additional adjustment for BMI, following the practice in some previous studies (Harris et al., 2017; Christensen et al., 2017; Susmann et al., 2019). We used R 4.0.0 for statistical analyses.

Results

Characteristics of study participants by type of residential floor covering are shown in Table 1. Accounting for the complex survey design, we estimated the geometric means of PFAS serum concentrations by type of floor covering in the general US population during 2005–2006 shown in Table 2. In general, we found higher geometric means of PFAS in people residing in homes with low pile carpets compared to those residing in homes with smooth surfaces and medium to high pile carpets. The pairwise Spearman correlation matrix among different serum PFAS concentrations is shown in Figure 1. The correlations among perfluoroalkyl carboxylic acids, including PFNA, PFDA, and PFOA are moderate to high, which is likely due to the fact that they have a common precursor 8:2 FTOH (Buck et al., 2011). The high correlation between PFOA and PFOS has been reported elsewhere previously (Haug et al., 2009), indicating the likelihood of common exposure sources for these two legacy PFAS, such as food, dust, and air. However, the correlations among PFOS and its precursors MeFOSAA, PFHxS were not as high as expected, indicating the likelihood of other commercial sources of PFOS (e.g., di-SAmPAP) (Yeung et al., 2013).

After imputation, the study participants ($n = 2,323$) represent 244 million general US population, half of whom resided in homes with low pile carpets. Adjusting for potential confounders, we found low pile carpets were associated with 32% (95% CI: 3%–70%) increase in serum PFHxS concentration and 25% (95% CI: 7%–45%) increase in serum MeFOSAA concentration compared to smooth surfaces (Table 3). Given the geometric means of 1.29 ng/ml and 0.31 ng/ml for PFHxS and MeFOSAA, respectively, for people residing in homes with smooth surfaces (Table 2), on average low pile carpets were associated with 0.41 ng/ml increase in PFHxS and 0.08 ng/ml increase in MeFOSAA, accounting for 24% and 19% of the geometric means of serum PFHxS and MeFOSAA concentrations, respectively, in people residing in homes with low pile carpets in the US.

The Wald Chi-squared test of equivalence for multiple parameters showed significant differences in serum PFOS and MeFOSAA concentrations among people who used different types of floor covering (p -value = 0.02 and 0.04, respectively; Table 3). Overall, multiple imputation and complete case analysis created similar results in regression analyses with the exception of PFHxS, for which the association with low pile carpets was not significant using complete case analysis (20%, 95% CI: [–14%, 67%], see Table S1 in the Supplementary material) but significant using multiple imputation (32%, 95% CI: [3%, 70%], Table 3). Complete case analysis only included 1,044 observations in the adjusted model, losing 55% of the information, which may bias the results (Little and Rubin, 2020). Separate analysis for adults ($n=1,593$) produced similar results to the analysis using all participants ($n=2,323$) (Table S2), while the results for adolescents ($n=730$) were different, i.e., the effects of low pile carpet on PFHxS (29%, 95% CI: [–14%, 92%], Table S3) and MeFOSAA (13%, 95% CI: [–9%, 41%], Table S3) were not significant, which is likely due to the smaller sample size of adolescents and the less statistical power. Additional adjustment for BMI in the sensitivity analyses also produced similar results to the primary analysis (see Table S4 in the Supplementary material).

Other important predictors for PFAS in this study include race/ethnicity, country of birth, family PIR, shellfish consumption, tap water sources, menstruation period, similar to previous findings reported elsewhere (Calafat et al., 2007; Christensen et al. 2017; Haug et al., 2010; Hurley et al., 2016; Kato et al. 2011; Suominen et al., 2011; Yamaguchi et al., 2013).

Discussion

In this study, we examined the associations between type of floor covering and serum PFAS concentrations. Descriptive statistics in Table 2 also showed higher geometric means of serum PFAS concentrations in people residing in homes with low pile carpets than people residing in homes with smooth surfaces and medium to high pile carpets. After adjusting for potential confounding variables in the multiple regression model, we found significantly higher serum PFHxS and MeFOSAA concentrations in people residing in homes with low pile carpets compared with those residing in homes with smooth surfaces, especially adults; while the effects of medium to high pile carpets and combination of carpet and smooth surface on serum PFAS concentrations were less clear, suggesting that low pile carpet is a more important source of exposure compared with other types of flooring. Because PFHxS

and MeFOSAA are both key ingredients in carpet treatment products that are intended for stain resistance, our results could be explained by the fact that low pile carpets frequently used in highly-trafficked spaces were more likely to have more extensive PFAS treatment. In addition, we concluded that among US residents using low pile carpeting in the home in 2005–2006, on average 24% and 19% of the PFHxS and MeFOSAA body burdens, respectively, could be attributed solely to the carpeting.

A previous study by Harris et al. (2017) found higher serum PFOS, PFHxS, and MeFOSAA concentrations in US children aged 6–10 years who slept in a bedroom with carpeting or a rug, indicating hand-to-mouth transfer from treated carpets or inhalation of volatile precursors as important exposure pathways for children. Our study does not include children less than 12 years old, who are more likely than adolescents and adults to crawl on the ground and have their hands contact the carpets. Our results derived from the NHANES data are more representative of the general US population aged 12 and older, and can potentially inform the PFAS exposure from various types of flooring, especially low pile carpeting. Previous studies have indicated carpets as both a source and sink of PFAS chemicals. Given that the dust ingestion of children is about two times that of adults (Shoeib et al., 2005; Strynar et al., 2008), the PFAS exposure coming from house carpets for children less than 12 years old is likely to be higher.

Similar to the previous studies (Beesoon et al., 2012; Hurley et al., 2018; Goosey and Harrad, 2011), we found that other long-chain PFAS such as PFHxS and MeFOSAA were elevated in individuals residing in homes with carpets. We also found somewhat different PFOS concentrations among people using various types of flooring, which may suggest distinct PFAS treatment or formulations for different types of carpets, and the potential for other PFAS to degrade or be metabolized to PFOS (ATSDR, 2018). However, the conclusion is tempered by the limitation of using $p\text{-value}<0.05$ to represent statistical significance in the Wald Chi-squared tests, especially with multiple testing (Wasserstein and Lazar, 2016). Our estimates with 95% confidence intervals in Table 3 more directly address effect sizes and their associated uncertainties.

This study has several noteworthy features. Among them is the use of multiple imputation to address missing values in the dataset, which appropriately accounted for the uncertainty of imputation. Additionally, we used statistical methods accounting for the complex survey design to obtain unbiased estimates of regression parameters and accurate variance estimation. Hu et al. (2018) have used the same data as us to assess the associations between PFAS and carpet. Based on the 2005–2006 NHANES data, they concluded that fully or partially carpet covered floors were associated with serum concentrations of PFOS, PFHxS, and MeFOSAA ($p\text{-value}<0.05$). However, they did not distinguish between the contributions of various types of carpeting. Although they may have applied sampling weights to obtain the regression parameter estimates that are representative of the general US population, their analysis did not appear to incorporate other attributes of the complex survey design (cluster and stratification) to obtain the correct variance estimation, given this limitation of the *gam* package in R that they used. Failure to account for the homogeneity of individuals within a cluster would lead to inaccurate lower variance estimates that bias the results of statistical hypothesis testing (NHANES, 2020b). Although Hu et al. (2018) applied statistical methods

to deal with left-censored data on serum PFAS concentrations, it is not clear how they addressed missing values in the explanatory variables.

Our study has several limitations. First, our analyses were restricted to the 2005–2006 cycle based on the unique availability of the key variable interest, type of floor covering in the NHANES datasets. Future research collecting and/or using more recent data from other sources would be a valuable addition to the present study. Second, although similar compositional patterns have been found in carpets and dust previously (Wu et al., 2020), there is a lack of linkage between PFAS in air and/or dust samples and PFAS in the house carpets in our study due to the limitation of the NHANES datasets. Also, lack of information on other furniture and upholstery, which may also be treated with PFAS, may also impact our inference due to the potential uncontrolled confounding. Third, although we tried to obtain a crude estimate of PFAS exposure through selection of several key dietary recall variables, there are no direct measurements of PFAS in the NHANES participants' individual diets and drinking water. Although dietary seafood intake has been found to be a strong predictor of serum PFAS concentrations (Christensen et al., 2007), our use of seafood consumption in the past 30 days is limited in its ability to predict long-term seafood consumption. Previous studies have implicated consumption of PFAS-contaminated tap water as a significant predictor for serum PFAS concentrations (Shin et al., 2011a; Shin et al., 2011b) and published exposure-pharmacokinetic models predict that tap water contributions can be dominant at fairly low PFAS water concentrations (Bartell, 2017; Lu and Bartell, 2019). We did not adjust for the amount of tap water consumed due to the spatial heterogeneity of PFAS contamination in US public water supplies, the removal of geographical identifiers from public use NHANES data sets, and the limitations of using short-term recall data to represent long-term water consumption. Future research linking individual-level data from NHANES to the nationwide PFAS detection data in public water supplies (UCMR 3) would help assess the contributions of drinking water to serum PFAS.

Conclusions

This study found that low pile carpeting was associated with increased serum concentrations of PFHxS and MeFOSAA in the general US population, while no clear association has been observed between the other types of carpeting and serum PFAS concentrations. Further studies would be needed to fully understand PFAS formulations in different types of carpeting.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

- ATSDR (U.S. Department of Health and Human Services Agency for Toxic Substances and Disease Registry). 2018. Toxicological Profile for Perfluoroalkyls, Atlanta, GA:Centers for Disease Control and Prevention. <https://www.atsdr.cdc.gov/toxprofiles/tp200.pdf>.
- Bartell SM. 2017. Online Serum PFOA Calculator for Adults. *Environmental Health Perspectives*. 125, 104502. [PubMed: 29068316]
- Bartell SM, Calafat AM, Lyu C, Kato K, Ryan PB, Steenland K, et al. 2010. Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. *Environ Health Perspect*. 118, 222–228. [PubMed: 20123620]
- Beesoon S, Genuis S, Benskin JP, Martin JW. 2012. Exceptionally high serum concentrations of perfluorohexanesulfonate in a Canadian family are linked to home carpet treatment applications. *Environ Sci Technol*. 46, 12960–7. [PubMed: 23102093]
- Beesoon S, Webster GM, Shoeib M, Harner T, Benskin JP, Martin JW. 2011. Isomer profiles of perfluorochemicals in matched maternal, cord, and house dust samples: manufacturing sources and transplacental transfer. *Environ Health Perspect*. 119, 1659–64. [PubMed: 21757419]
- Benford D, de Boer J, Carere A, di Domenico A, Johansson N, Schrenk D, et al. 2008. Perfluorooctane sulfonate, perfluorooctanoic acid and their salts. Scientific opinion of the panel on contaminants in the food chain. *European Food Safety Authority*. 653, 1–131.
- Birnbaum LS, Grandjean P. 2015. Alternatives to PFASs: perspectives on the science. *Environ Health Perspect*. 123(5), A104. [PubMed: 25932670]
- BloombergLaw. 2020. Older PFAS That EPA Thought Obsolete Still Used, Agency Told. <https://news.bloomberglaw.com/environment-and-energy/older-pfas-that-epa-thought-obsolete-still-used-agency-told>.
- Buck RC, Franklin J, Berger U, Conder JM, Cousins IT, Voogt P, et al. 2011. Perfluoroalkyl and polyfluoroalkyl substances in the environment: terminology, classification, and origins. *Integr Environ Assess Manag*. 7(4), 513–541. [PubMed: 21793199]
- Calafat AM, Wong LY, Kuklennyk Z, Reidy JA, Needham LL. 2007. Polyfluoroalkyl 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*. 115(11), 1596–1602. [PubMed: 18007991]
- CDC (The Centers for Disease Control and Prevention). 2012. National Health and Nutrition Examination Survey. 2005–2006 Data Documentation, Codebook, and Frequencies. Polyfluoroalkyl Chemicals (PFC_D), https://wwwn.cdc.gov/Nchs/Nhanes/2005-2006/PFC_D.htm
- CDC. 2013. Laboratory Procedure Manual for Per- and Polyfluoroalkyl Substances (PFAS). https://www.cdc.gov/nchs/data/nhanes/nhanes_11_12/PFC_G_met.pdf
- CDC. 2019. Fourth Report on Human Exposure to Environmental Chemicals, Updated Tables, January 2019. Atlanta, GA: US Centers for Disease Control and Prevention. <https://www.cdc.gov/exposurereport/index.html>.
- Christensen KY, Raymond M, Blackowicz M, Liu Y, Thompson BA, Anderson HA, et al. 2017. Perfluoroalkyl substances and fish consumption. *Environ Res*. 154, 145–151. [PubMed: 28073048]
- D’eon JC, Mabury SA. 2007. Production of perfluorinated carboxylic acids (PFCAs) from the biotransformation of polyfluoroalkyl phosphate surfactants (PAPS): exploring routes of human contamination. *Environ Sci Technol*. 41(13), 4799–4805. [PubMed: 17695932]
- D’eon JC, Mabury SA. 2011. Exploring indirect sources of human exposure to perfluoroalkyl carboxylates (PFCAs): evaluating uptake, elimination, and biotransformation of polyfluoroalkyl phosphate esters (PAPs) in the rat. *Environ Health Perspect*. 119(3), 344–350. [PubMed: 21059488]
- D’Hollander W, Roosens L, Covaci A, Cornelis C, Reynders H, Campenhout KV, et al. 2010. Brominated flame retardants and perfluorinated compounds in indoor dust from homes and offices in Flanders, Belgium. *Chemosphere*. 81(4), 478–87. [PubMed: 20709355]

- De Silva A, Allard CN, Spencer C, Webster GM, Shoeib M. 2012. Phosphorus-containing fluorinated organics: polyfluoroalkyl phosphoric acid diesters (diPAPs), perfluorophosphonates (PFPA), and perfluorophosphinates (PFPIAs) in residential indoor dust. *Environ Sci Technol.* 46, 12575–12582. [PubMed: 23102111]
- DTSC (Department of Toxic Substances Control). 2018. Product-Chemical Profile for Perfluoroalkyl and polyfluoroalkyl substances (PFASs) in carpets and rugs. <https://dtsc.ca.gov/wp-content/uploads/sites/31/2018/10/Product-Chemical-Profile-PFAS-Carpets-and-Rugs.pdf>
- Eschauzier C, Haftka J, Stuyfzand PJ, Voogt PD. 2010. Perfluorinated compounds in infiltrated river Rhine water and infiltrated rainwater in coastal dunes. *Environ Sci Technol.* 44, 7450–7455. [PubMed: 20831216]
- Faithfull NS, Weers JG. 1998. Perfluorocarbon compounds. *Vox sanguinis.* 74(S2), 243–8. [PubMed: 9704451]
- Fraser AJ, Webster TF, Watkins DJ, Nelson JW, Stapleton HM, Calafat AM, et al. 2012. Polyfluorinated compounds in serum linked to indoor air in office environments. *Environ Sci Technol.* 46(2), 1209–15. [PubMed: 22148395]
- Fromme H, Mosch C, Morovitz M, Alba-Alejandre I, Boehmer S, Kiranoglu M, et al. 2010. Pre- and postnatal exposure to perfluorinated compounds (PFCs). *Environ Sci Technol.* 44, 7123–9. [PubMed: 20722423]
- Gebbink WA, Glynn A, Berger U. 2015. Temporal changes (1997–2012) of perfluoroalkyl acids and selected precursors (including isomers) in Swedish human serum. *Environ Pollution* 199, 166–173.
- Glüge J, Scheringer M, Cousins IT, DeWitt JC, Goldenman G, Herzke D, Lohmann R, Ng CA, Trier X, Wang Z. 2020. An overview of the uses of per- and polyfluoroalkyl substances (PFAS). *Environ Sci Processes Impacts.*
- Gomis MI, Vestergren R, Borg D, Cousins IT. 2018. Comparing the toxic potency in vivo of long-chain perfluoroalkyl acids and fluorinated alternatives. *Environ Int.* 113, 1–9. [PubMed: 29421396]
- Goosey E, Harrad S. 2011. Perfluoroalkyl compounds in dust from Asian, Australian, European, and North American homes and UK cars, classrooms, and offices. *Environ Int.* 37, 86–92. [PubMed: 20810169]
- Harris MH, Rifas-Shiman SL, Calafat AM, Ye X, Mora AM, Webster TF, et al. 2017. Predictors of per- and polyfluoroalkyl substance (PFAS) plasma concentrations in 6–10 year old American children. *Environ Sci Technol.* 51(9), 5193–5204. [PubMed: 28325044]
- Hurley S, Goldberg D, Wang M, Park J, Petreas M, Bernstein L, et al. 2018. Time trends in per- and polyfluoroalkyl substances (PFASs) in California women: declining serum levels, 2011–2015. *Environ Sci Tech.* 52, 277–287.
- Hu XC, Andrews DQ, Lindstrom's AB, Bruton TA, Schaidler LA, Grandjean P, et al. 2016. Detection of poly- and perfluoroalkyl substances (PFASs) in U.S. drinking water linked to industrial sites, military fire training areas, and wastewater treatment plants. *Environ Sci Technol Lett.* 3, 344–350. [PubMed: 27752509]
- Hu XC, Dassuncao C, Zhang X, Grandjean P, Weihe P, Webster GM, et al. 2018. Can profiles of poly- and Perfluoroalkyl substances (PFASs) in human serum provide information on major exposure sources? *Environmental Health.* 17, 11. [PubMed: 29391068]
- Jain RB. 2014. Contribution of diet and other factors to the levels of selected polyfluorinated compounds: Data from NHANES 2003–2008. *Int J Hyg Environ Health.* 217, 52–61. [PubMed: 23601780]
- Jain RB, Ducatman A. 2019. Perfluoroalkyl substances follow inverted U-shaped distributions across various stages of glomerular function: Implications for future research. *Environ Res.* 169, 476–482. [PubMed: 30530087]
- Johnson PI, Sutton P, Atchley DS, Koustas E, Lam J, Sen S, et al. 2014. The navigation guide—evidence-based medicine meets environmental health: systematic review of human evidence for PFOA effects on fetal growth. *Environ Health Perspect.* 122(10), 1028–1039. [PubMed: 24968388]

- Karásková P, Venier M, Melymuk L, Beánová J, Vojta Š, Prokeš R, Diamond ML, Klánová J. 2016. Perfluorinated alkyl substances (PFASs) in household dust in Central Europe and North America. *Environ Int.* 94, 315–324. [PubMed: 27295048]
- Kato K, Calafat AM, Needham LL. 2009. Polyfluoroalkyl chemicals in house dust. *Environ Res.* 109(5), 518–23. [PubMed: 19261270]
- Kato K, Wong LY, Jia LT, Kuklennyik Z, and Calafat AM. 2011. Trends in exposure to polyfluoroalkyl chemicals in the U.S. population: 1999–2008. *Environ Sci Technol.* 45(19), 8037–8045. [PubMed: 21469664]
- Kissa E 2001. *Fluorinated Surfactants and Repellents*, 2nd edition (Vol. 97). Marcel Dekker, Inc.: New York. 97, 640.
- Kubwabo C, Stewart B, Zhu J, Marro L. 2005. Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada. *J Environ Monit.* 7, 1074–1078 [PubMed: 16252056]
- Li Y, Fletcher T, Mucs D, Scott K, Lindh CH, Tallving P, Jakobsson K. 2018. Half-lives of PFOS, PFHxS and PFOA after end of exposure to contaminated drinking water. *J Occup Environ Med.* 75, 46–51.
- Little RJA, Rubin DB. 2020. *Statistical Analysis with Missing Data*, 3rd edition. John Wiley & Sons, Inc.
- Liu G, Dhana K, Furtado J, Rood J, Zong G, Liang L, et al. 2018. Perfluoroalkyl substances and changes in body weight and resting metabolic rate in response to weight-loss diets: A prospective study. *PLOS Med.* 15(2), e1002502. [PubMed: 29438414]
- Liu S, Yang R, Yin N, Faiola F. 2020. The short-chain perfluorinated compounds PFBS, PFHxS, PFBA and PFHxA, disrupt human mesenchymal stem cell self-renewal and adipogenic differentiation. *J Environ Sci (China).* 88, 187–199. [PubMed: 31862060]
- Lu S, Bartell SM. 2019. Serum PFAS Calculator for Adults, Version 1.1, www.ics.uci.edu/~sbartell/pfascal.html.
- Martin JW, Mabury SA, Solomon KR, Muir DCG. 2003. Dietary accumulation of perfluorinated acids in juvenile rainbow trout (*Oncorhynchus mykiss*). *Environmental Toxicology and Chemistry.* 22(1), 189–195. [PubMed: 12503764]
- NCHS (National Center for Health Statistics). 2012. The National Health and Nutrition Examination Survey: Sample Design, 1999–2006. https://www.cdc.gov/nchs/data/series/sr_02/sr02_155.pdf
- NCHS. 2013. National Health and Nutrition Examination Survey: Plan and Operations, 1999–2010. https://www.cdc.gov/nchs/data/series/sr_01/sr01_056.pdf
- NHANES (National Health and Nutrition Examination Survey). 2020a. Tutorials, Module 3: Weighting, Selecting the Correct Weight in NHANES. <https://www.cdc.gov/nchs/nhanes/tutorials/module3.aspx>.
- NHANES. 2020b. Tutorials, Module 4: Variance Estimation, Variance Estimation Methods in NHANES. <https://www.cdc.gov/nchs/nhanes/tutorials/module4.aspx>.
- Olsen GW, Church TR, Miller JP, Burris JM, Hansen KJ, Lundberg JK, Armitage JB, Herron RM, Medhizadehkashi Z, Nobiletti JB, O'Neill EM, Mandel JH, Zobel LR. 2003. Perfluorooctanesulfonate and other fluorochemicals in the serum of American Red Cross adult blood donors. *Environ Health Perspect.* 111(16), 1892–1901. [PubMed: 14644663]
- Olsen GW, Burris JM, Ehresman DJ, Froehlich JW, Seacat AM, Butenhoff JL, et al. 2007. Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. *Environ Health Perspect.* 115(9), 1298–305. [PubMed: 17805419]
- Olsen GW, Mair DC, Church TR, Ellefson ME, Reagen WK, Boyd TM, Herron RM, Medhizadehkashi Z, Nobiletti JB, Rios JA, Butenhoff JL, Zobel LR. 2008. Decline in perfluorooctanesulfonate and other polyfluoroalkyl chemicals in American Red Cross adult blood donors, 2000–2006. *Environ Sci Technol.* 42(13), 4989–4995. [PubMed: 18678038]
- Olsen GW, Change S-C, Noker PE, Gorman GS, Ehresman DJ, Lieder PH, Butenhoff JL. 2009. A comparison of the pharmacokinetics of perfluorobutanesulfonate (PFBS) in rats, monkeys, and humans. *Toxicology.* 256, 65–74. [PubMed: 19059455]
- Rothman KJ. 1987. *Epidemiology: An Introduction*, 2nd edition. Oxford University Press, Inc, 2012.

- Rubin DB. 1987. Multiple Imputation for Nonresponse in Surveys. John Wiley & Sons, Inc.
- Shin HM, Vieira VM, Ryan PB, Detwiler R, Sanders B, Steenland K, Bartell SM. 2011a. Environmental fate and transport modeling for perfluorooctanoic acid emitted from the Washington Works Facility in West Virginia. *Environ Sci Technol.* 45(4), 1435–1442. [PubMed: 21226527]
- Shin HM, Vieira VM, Ryan PB, Steenland K, Bartell SM. 2011b. Retrospective exposure estimation and predicted versus observed serum perfluorooctanoic acid concentrations for participants in the C8 Health Project. *Environ Health Perspect.* 119(12), 1760–1765. [PubMed: 21813367]
- Shoeib M, Harner T, Wilford BH, Jones KC, Zhu J. 2005. Perfluorinated sulfonamides in indoor and outdoor air and indoor dust: occurrence, partitioning, and human exposure. *Environ Sci Technol.* 39:6599–6606. [PubMed: 16190217]
- Strynar MJ, Lindstrom AB. 2008. Perfluorinated compounds in house dust from Ohio and North Carolina, USA. *Environ Sci Technol.* 42(10), 3751–6. [PubMed: 18546718]
- Susmann HP, Schaidler LA, Rodgers KM, Rudel RA. 2019. Dietary Habits Related to Food Packaging and Population Exposure to PFASs. *Environ Health Perspect.* 127(10), 107003. [PubMed: 31596611]
- UNEP (United Nations Environment Programme). 2010. The 9 new pops; an introduction to the nine chemicals added to the stockholm convention by the conference of the parties at its fourth meeting, 2009. <http://chm.pops.int/programmes/newpops/publications/tabid/695/language/en-us/default.aspx>.
- UNEP. 2015. Working towards a global emission inventory of PFASs; environment directorate, Organization for Economic Cooperation and Development: Paris. <http://www.oecd.org/chemicalsafety/Working%20Towards%20a%20Global%20Emission%20Inventory%20of%20PFASS.pdf>
- US EPA (US Environmental Protection Agency). 2009. Long-chain perfluorinated chemicals (PFCs) action plan. Available from: http://www.epa.gov/opptintr/existingchemicals/pubs/pfcs_action_plan1230_09.pdf
- Vélez MP, Arbuckle TE, Fraser WD. 2015. Maternal exposure to perfluorinated chemicals and reduced fecundity: the MIREC study. *Human Reproduction.* 30(3), 701–709. [PubMed: 25567616]
- Vieira VM, Hoffman K, Shin HM, Weinberg JM, Webster TF, Fletcher T. 2013. Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: a geographic analysis. *Environ Health Perspect.* 121(3), 318–323. [PubMed: 23308854]
- Wang Z, Cousins IT, Scheringer M, Buck RC, Hungerbühler K. 2014. Global emission inventories for C4-C14 perfluoroalkyl carboxylic acid (PFCA) homologues from 1951 to 2030, part II: The remaining pieces of the puzzle. *Environ Int.* 69, 166–176. [PubMed: 24861268]
- Wasserstein R, Lazar NA. 2016. The ASA’s statement on p-Values: context, process, and purpose. *The American Statistician.* 70(2), 129–133.
- Winkens K, Giovanoulis G, Koponen J, Vestergren R, Berger U, Karvonen AM, et al. 2018. Perfluoroalkyl acids and their precursors in floor dust of children’s bedrooms - Implications for indoor exposure. *Environ Int.* 119, 493–502. [PubMed: 30053737]
- Worley RR, Moore SM, Tierney BC, Ye X, Calafat AM, Campbell S, et al. 2017. Per- and polyfluoroalkyl substances in human serum and urine samples from a residentially exposed community. *Environ Int.* 106, 135–143. [PubMed: 28645013]
- Wu Y, Romanak K, Bruton T, Blum A, Venier M. 2020. Per- and polyfluoroalkyl substances in paired dust and carpets from childcare centers. *Chemosphere.*
- Wu H, Yoon M, Verner M, Xue J, Luo M, Anderson ME, et al. 2015. Can the observed association between serum perfluoroalkyl substances and delayed menarche be explained on the basis of puberty-related changes in physiology and pharmacokinetics? *Environ Int.* 82, 61–68. [PubMed: 26043300]
- Yue R, Ministry of Environmental Protection of China. 2008. Additional information on production and use of PFOS, http://chm.pops.int/portals/0/repository/comments_draftRME2008/UNEP-POPS-POPRC-DRME-08-CHI-SCCP.english.pdf.

- Zhang T, Wu Q, Sun HW, Zhang XZ, Yun SH, Kannan K. 2010. Perfluorinated compounds in whole blood samples from infants, children, and adults in China. *Environ Sci Technol.* 44(11), 4341–7. [PubMed: 20441147]
- Zhang Y, Beesoon S, Zhu L, Martin JW. 2013. Biomonitoring of perfluoroalkyl acids in human urine and estimates of biological half-life. *Environ Sci Technol.* 47(18), 10619–10627. [PubMed: 23980546]
- Zushi Y, Hogarh J, Masunaga S. Progress and perspective of perfluorinated compound risk assessment and management in various countries and institutes. *Clean Technol Environ Policy* 2012; 14:9–20.

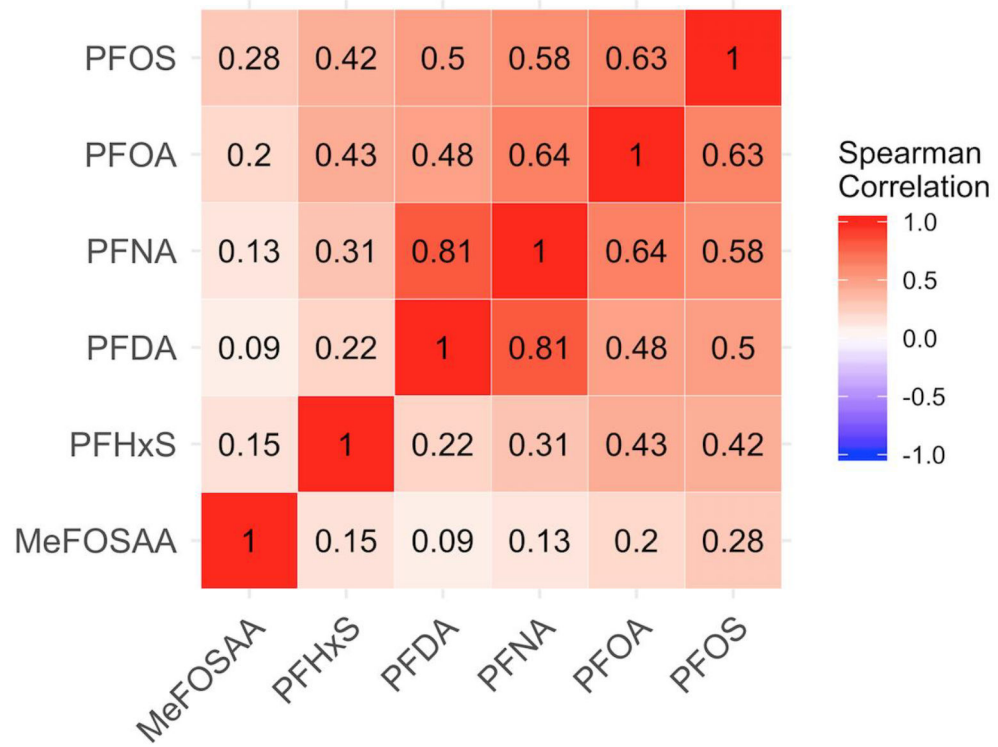


Figure 1.
Pairwise Spearman Correlation Matrix among Different PFAS in Serum

Table 1.

Characteristics of Study Participants

Characteristics	n (%) or mean ± SD	Type of floor covering			Combination of carpet and smooth surface (n = 67)
		Smooth surface (n = 183)	Low pile carpet (n = 1276)	Medium/high pile carpet (n = 172)	
Age	38.2 ± 22.2	32.5 ± 20.0	37.3 ± 22.0	41.1 ± 23.8	45.0 ± 22.7
Gender					
Female	1,180 (50.8%)	81 (44.3%)	647 (50.7%)	77 (44.8%)	38 (56.7%)
Male	1,143 (49.2%)	102 (55.7%)	629 (49.3%)	95 (55.2%)	29 (43.3%)
Race/ethnicity					
Hispanic	623 (26.8%)	78 (42.6%)	332 (26.0%)	49 (28.5%)	11 (16.4%)
Non-Hispanic black	613 (26.4%)	49 (26.8%)	388 (30.4%)	24 (14.0%)	18 (26.9%)
Non-Hispanic white	996 (42.9%)	48 (26.2%)	503 (39.4%)	88 (51.2%)	34 (50.7%)
Others	91 (3.9%)	8 (4.4%)	53 (4.2%)	11 (6.4%)	4 (6.0%)
Education					
< College	1,503 (64.7%)	127 (69.4%)	846 (66.3%)	120 (69.8%)	38 (56.7%)
Some college	513 (22.1%)	41 (22.4%)	279 (21.9%)	34 (19.8%)	16 (23.9%)
> College	305 (13.1%)	15 (8.2%)	150 (11.8%)	18 (10.5%)	13 (19.4%)
Country of birth					
US	1,875 (80.7%)	141 (77.0%)	1028 (80.6%)	136 (79.1%)	60 (89.6%)
Foreign	447 (19.2%)	42 (23.0%)	248 (19.4%)	36 (20.9%)	7 (10.4%)
Veteran/military status					
No	1,625 (70.0%)	130 (71.0%)	876 (68.7%)	115 (66.9%)	46 (68.7%)
Yes	247 (10.6%)	8 (4.4%)	135 (10.6%)	28 (16.3%)	15 (22.4%)
Family PIR	2.5 ± 1.6	2.0 ± 1.5	2.4 ± 1.5	2.4 ± 1.4	2.5 ± 1.8
BMI	27.3 ± 7.0	27.3 ± 7.4	27.1 ± 6.9	27.7 ± 6.9	28.1 ± 8.6
Tap water source					
Don't drink tap water	419 (18.0%)	40 (21.9%)	224 (17.6%)	35 (20.3%)	17 (25.4%)
Community supply	1,335 (57.5%)	103 (56.3%)	739 (57.9%)	91 (52.9%)	38 (56.7%)
Other	338 (0.1%)	22 (12.0%)	190 (14.9%)	30 (17.4%)	9 (13.4%)
Eat out per week					
No	225 (9.7%)	18 (9.8%)	133 (10.4%)	17 (9.9%)	7 (10.4%)

Characteristics	Type of floor covering				
	n (%) or mean \pm SD	Smooth surface (n = 183)	Low pile carpet (n = 1276)	Medium/high pile carpet (n = 172)	Combination of carpet and smooth surface (n = 67)
Yes	2,064 (88.9%)	161 (88.0%)	1126 (88.2%)	154 (89.5%)	60 (89.6%)
Eating shellfish in the past 1 month					
No	1,109 (47.7%)	90 (49.2%)	615 (48.2%)	80 (46.5%)	35 (52.2%)
Yes	1,114 (48.0%)	80 (43.7%)	618 (48.4%)	84 (48.9%)	29 (43.3%)
Eating fish in the past 1 month					
No	793 (34.1%)	68 (37.2%)	445 (34.9%)	48 (27.9%)	17 (25.4%)
Yes	1,431 (61.6%)	103 (56.3%)	788 (61.8%)	116 (67.4%)	47 (70.1%)
Serum Creatinine (mg/dL)	0.9 \pm 0.3	0.8 \pm 0.3	0.8 \pm 0.3	0.9 \pm 0.3	0.9 \pm 0.7
Had at least one period in the past 1 year					
No	1,506 (64.8%)	120 (65.6%)	820 (64.3%)	118 (68.6%)	44 (65.7%)
Yes	706 (30.4%)	51 (27.9%)	396 (31.0%)	49 (28.5%)	23 (34.3%)
Number of pregnancies	1.0 \pm 1.9	0.8 \pm 1.7	1.0 \pm 1.9	1.0 \pm 1.9	0.9 \pm 2.0
Number of children breastfed at least 1 month	0.4 \pm 1.1	0.3 \pm 1.0	0.3 \pm 1.0	0.4 \pm 1.1	0.3 \pm 0.9

Table 2.
Geometric Means of Serum PFAS Concentrations by Type of Floor Covering

Type of Floor Covering	Geometric Mean, 95% CI (ng/ml)						
	PFOA	PFOS	PFHxS	MeFOSAA	PFDA	PFNA	PFNA
Smooth surface	3.45, (2.95, 4.04)	13.67, (10.93, 17.08)	1.29, (0.94, 1.78)	0.31, (0.25, 0.39)	0.30, (0.25, 0.36)	0.94, (0.81, 1.08)	0.94, (0.81, 1.08)
Low pile carpet	3.85, (3.41, 4.35)	17.34, (16.20, 18.55)	1.74, (1.46, 2.08)	0.41, (0.38, 0.45)	0.35, (0.29, 0.41)	1.05, (0.88, 1.25)	1.05, (0.88, 1.25)
Medium to high pile carpet	3.71, (3.00, 4.58)	15.31, (13.22, 17.73)	1.63, (1.20, 2.22)	0.41, (0.31, 0.56)	0.29, (0.25, 0.34)	0.94, (0.83, 1.05)	0.94, (0.83, 1.05)
Combination of carpet and smooth surface	3.75, (3.16, 4.45)	16.66, (13.53, 20.53)	1.56, (1.24, 1.97)	0.44, (0.35, 0.56)	0.33, (0.27, 0.42)	1.08, (0.86, 1.35)	1.08, (0.86, 1.35)

Table 3.

Adjusted Associations* from Multiple Regression Models with Multiple Imputation (k=100) (n=2,323)

% difference in PFAS concentrations ^{***} , 95% CI	PFOA	PFOS	PFHxS	MeFOSAA	PFDA	PFNA
Type of floor covering						
Smooth surface	ref	ref	ref	ref	ref	ref
Low pile carpet	7, (-8, 23)	14, (-2, 33)	32, (3, 70)	25, (7, 45)	12, (-6, 34)	9, (-7, 29)
Medium to high pile carpet	-2, (-19, 17)	-2, (-19, 19)	22, (-15, 75)	23, (-5, 59)	-6, (-22, 12)	-6, (-21, 13)
Combination of carpet and smooth surface	-2, (-21, 22)	2, (-20, 31)	13, (-18, 55)	20, (-8, 57)	3, (-19, 31)	4, (-17, 30)
<i>p-value of Wald Chi-squared test</i>	0.5	0.02	0.1	0.04	0.1	0.3

* We adjusted for age, gender (female/male), race/ethnicity (Hispanic/non-Hispanic black/non-Hispanic white/others), education (less than college/some college/college graduate or above), country of birth (foreign/US), veteran/military status (yes/no), family PIR, tap water source (don't drink tap water/community supply/others), eating out per week (yes/no), eating shellfish during past 30 days (yes/no), eating fish during past 30 days (yes/no), serum creatinine, had at least one period in the past 12 months (yes/no), number of pregnancies, and number of children breastfed at least 1 month in the regression models.

** We exponentiated the regression coefficients, subtracted by one, and multiplied by 100% to estimate the percent difference in PFAS concentrations associated with each predictor.