



Full length article



# Understanding prenatal household exposures to per- and polyfluoroalkyl substances using paired Biological and dust measurements with sociodemographic and housing variables

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## ABSTRACT

Per- and poly-fluoroalkyl substances (PFAS) are chemicals of concern—they are ubiquitous, persistent, with known and suspected health impacts. Well studied, primary sources of exposure to PFAS are drinking water and food. The presence of PFAS in human tissue of general populations suggests other important exposure sources/pathways. House dust measurements suggest widespread presence of PFAS in residences. Limited studies report paired analyses of PFAS occurrence in indoor media and PFAS concentrations in serum. While paired samples of house dust and blood serum are currently rare, the National Children's Study (NCS) contains paired samples, as well as sociodemographic information, from pregnant people that participated in the study. These archived NCS data and specimens for 104 participants collected between 2009 and 2014 were leveraged and analyzed for 16 commonly measured PFAS. We evaluated PFAS levels in the home, and the relationships between PFAS in dust and serum, and sociodemographic or housing variables. In addition, mechanistic exposure models, and then steady-state serum level models with simple parameters were used to estimate dust contributions of PFAS to serum. The geometric means for the most commonly found PFAS (full names in table 1) in serum were: 4.1 ng/mL for PFOS, 1.1 ng/mL for PFOA, 0.87 ng/mL for PFHxS, 0.16 ng/mL for PFDA. The geometric means of PFAS in dust were: 17 µg/kg for PFOS, 16 µg/kg for PFOA, 9.6 µg/kg for PFDS, 4.5 µg/kg for PFHpa, 4.4 µg/kg for PFNA, 3.9 µg/kg for PFHxS, 3.5 µg/kg for PFDA, 2.3 µg/kg for PFDoA, 2.1 µg/kg for PFUdA. PFOA was significantly correlated in serum and dust as was the sum of all PFAS detected in > 50 % of serum and dust. PFAS in serum was significantly associated with: Higher income, recent renovations, years lived in the home, and educational attainment. PFAS in dust was significantly associated with: Higher participant age, type of home, amount of carpet, educational attainment, higher income, recent renovation, and membership in the military. For some PFAS, 25 % of the overall exposure, on average, is from dust, but for others, 3–4 % is attributed to dust.

We were able to identify important associations in PFAS exposure in the homes of pregnant people based on paired serum and dust samples. This built a clearer picture of which PFAS and at what quantities they exist in these homes, how they relate to each other, and how they are tied to sociodemographic and housing factors. Our results demonstrate that exposure to PFAS via house dust may contribute up to 25% of total exposure for adults, highlighting the importance of understanding what drives residential exposures.

## 1. Introduction

Per- and polyfluoroalkyl substances (PFAS) are a class of organic

compounds that are linked to adverse health effects in adults and children and are ubiquitous in consumer products and the built environment (Fabelova et al., 2023; National Academies Press, 2022; DeLuca et al.,

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2022; Gluge et al., 2020; Herzke and Olsson, 2012; Sunderland et al., 2019). Production of PFAS began in the 1930s and these chemicals have since become pervasive in natural and built environments due to their widespread use in hundreds of different applications—including as stain, oil, and water repellent coatings in food containers, and as additives to textiles, furniture, carpet, pharmaceutical packaging, apparel and personal care products (DeLuca et al., 2022; Gluge et al., 2020; Xia et al., 2022).

A recent National Academy of Sciences, Engineering, and Medicine report found considerable evidence that PFAS exposure led to decreased infant and fetal growth, dyslipidemia, increased kidney cancer risk, and decreased antibody response (National Academies Press, 2022). Additionally, many PFAS are highly persistent in the environment and the human body, especially the “legacy” longer chain perfluoroalkyl acids (PFAAs), which are largely out of production in the U.S. and many other countries but can still be found in the environment. A better understanding of the important sources and pathways of PFAS exposure is needed to guide regulation of these chemicals and prioritize exposure mitigation efforts.

Ingestion of contaminated food and water have been identified as key pathways of human exposure to PFAS (Fabelova et al., 2023; Graber et al., 2019; DeLuca et al., 2022; Gluge et al., 2020; Brase and Mullin, 2021; Brennan et al., 2021). However, exposure to house dust may also be a potentially important indoor PFAS exposure pathway (Bjorklund and Thuresson, 2009), especially in communities without significant point source contamination of drinking water. House dust acts as a sink for PFAS found inside the home through transfer from household objects, clothing, or consumer products, acting as an aggregator and sentinel of the level and type of PFAS released into the household environment. Residents can then be exposed to house dust through ingestion, inhalation, or dermal contact (DeLuca et al., 2022; Herzke and Olsson, 2012; Brase and Mullin, 2021; Brennan et al., 2021; Bjorklund and Thuresson, 2009; Fergusson et al., 1986; Rasmussen and Subramanian, 2001; Savvaides et al., 2021; Schultes et al., 2018). To better mitigate indoor pathways of human exposure to PFAS we need a deeper understanding of how dust contamination contributes to PFAS exposure, how it can be used as an indicator of indoor PFAS sources, and how dust PFAS, levels vary based on sociodemographic and household factors.

Indoor PFAS exposure sources and pathways are particularly relevant for pregnant people and their children. Newborns and infants are also especially vulnerable to exposure via dust because of high hand-to-mouth behavior, which can result in greater dust ingestion rates (Goeden and Greene, 2019; Thompson et al., 2010; Chang et al., 2021). Additionally, PFAS have been shown to be transferred to children through the placenta and, later, through breast milk (Fabelova et al., 2023; Goeden and Greene, 2019; Chang et al., 2021; Blomberg et al., 2023; Haug et al., 2011; Jian et al., 2018; Bloom et al., 2022; Rovira et al., 2019; Verner et al., 2016). PFAS health impacts may also be greater, as stages of early development from fertilization to early childhood represent a precarious window where exposures can be especially harmful to development and health later in the child’s life (Chang et al., 2021; Rovira et al., 2019; Dietert et al., 2000; Terry et al., 2019; Tyagi et al., 2021; Wright, 2017).

The National Children’s Study (NCS) was a U.S. nationwide study designed to investigate the effects of environmental exposures on pregnancy outcomes and child health and development (National Institute of Child Health and Human Development, 2016b; Hirschfeld et al., 2011; Mortensen, 2012). As part of the Vanguard Study of the NCS, serum and house dust samples were collected from pregnant NCS participants and health and exposure surveys were administered. After an initial set of targeted chemical analyses, samples were frozen for future research (National Institute of Child Health and Human Development, 2016b). Here, we leveraged the NCS Vanguard Study design to investigate residential PFAS exposure for pregnant people. Frozen, archived serum and house dust samples were analyzed for 16 commonly studied

PFAS. Measured PFAS concentrations in these two media were examined to identify associations between levels of PFAS in the home and PFAS body burden. Serum and dust PFAS levels were then linked with sociodemographic and housing characteristics of these households, to identify possible determinants of exposure. To determine the proportion of overall PFAS exposure that could have occurred through direct exposure to house dust, we used exposure and pharmacokinetic models to estimate the percent of serum levels that could be attributed to dust pathways.

While similar studies exist in the field, there are relatively few studies with access to paired dust and blood serum samples, as noted by a recent systematic review (DeLuca et al., 2022). This unique pairing allows for the comparison of PFAS levels and exposure estimates between these two different media. It also provides insight into the importance of dust exposure, which is emerging as an important secondary avenue of PFAS exposure inside the home that could also act as a sentinel of PFAS containing products inside the home. This research builds our understanding of the importance of indoor PFAS exposure sources and pathways, and the characteristics that may drive these exposures in a highly sensitive subpopulation, pregnant people, and their children.

## 2. Methods

### 2.1. Cohort

We acquired archived questionnaire data and specimens from a pilot phase of the National Children’s Study (NCS), a proposed national study in the United States designed to measure the health effects of environmental exposures on children using parent–child dyads. The NCS intended to investigate exposures for children during and after pregnancy and was not designed to focus specifically on PFAS (National Institute of Child Health and Human Development, 2016b; Hirschfeld et al., 2011; Mortensen, 2012; Branum et al., 2003; Landrigan et al., 2006). People were recruited based on their ability to become pregnant or status as pregnant.

The Vanguard Study pilot of the NCS tested recruitment methods for the main study and recruited 7921 people between 2009 and 2014. Environmental samples and biomonitoring specimens were collected for subsets of participants enrolled in the NCS during pregnancy and were frozen for future use after the initial analyses had been completed (National Institute of Child Health and Human Development, 2016b). Archived samples of settled house dust and serum were available from some participants ( $N = 104$ ) enrolled in NCS and were acquired from the NCS Vanguard Data and Sample Archive and Access System through a Materials Transfer Agreement with NICHD (Moye, 2020; National Institute of Child Health and Human Development, 2016a). Dust was collected by NCS researchers from participant vacuums during study visits. Participant demographic, lifestyle, residence, occupational, and other types of data were available from NCS questionnaire and observational survey instruments, and these data were acquired from National Institute of Child Health and Development’s Data and Specimen Hub (DASH).

### 2.2. PFAS analysis in blood and dust

Table 2 lists the sixteen PFAS measured in samples. House dust and serum samples were analyzed using ultra performance liquid chromatography-mass spectrometry (UPLC-MS). Nine perfluoroalkyl carboxylic acids (PFCA C4-C12) and seven perfluoroalkane sulfonic acids (PFSA C4-C10) were quantified.

#### 2.2.1. Standards and reagents

Liquid chromatography mass spectrometry (LC-MS) grade acetonitrile and formic acid were purchased from Fisher Scientific (Fair Lawn, NJ) and methanol from Honeywell – Burdick & Jackson (Muskegon, MI). Ottawa Sand Standard (20–30 Mesh) was purchased from Fisher

**Table 1**

Summary of questionnaire data used for analysis. Includes a basic summary of the question. The chosen answer and the number of participants who chose that answer. NAs include answers marked as legitimate skip, don't know, refused, missing, blank, not applicable, or other.

Variable	Option	Number of Participants	Variable	Option	Number of Participants	
Age	18–24	21	Income	<\$4,999	8	
	25–30	44		\$5,000–\$9,999	5	
	31–35	20		\$10,000–\$19,999	10	
	36+	9		\$20,000–\$29,999	7	
	NA	10		\$30,000–\$39,999	6	
Building Type	Single Family Home	7		\$40,000–\$49,999	9	
	Apartment /Multifamily Home	3		\$50,000–\$74,999	15	
	Trailer	3		\$75,000–\$99,999	16	
	NA	91		\$100,000–\$199,999	14	
				>\$199,999	1	
Carpet in Home	>1/2 of Home	11		NA	13	
	1/2 of Home	3		Years Lived in Home	<1	22
	<1/2 of Home	5			1–2	26
	NA	85	2.5–5		27	
			5+		14	
		NA	15			
Year Home was Built	2001–now	23	Military Household	Yes	3	
	1981–2000	17		No	60	
	1961–1980	11		NA	41	
	1941–1960	15	Hours/Day with Windows Open	<1	23	
	before 1941	18		1–3	17	
	NA	20		4–12	14	
Education	<High School or GED	5	>12	4		
	High School, GED or Some College	39	none	25		
	Associate Degree	10	NA	21		
	Bachelor's Degree	32	Recent Renovation	Yes	11	
	Post-graduate Degree	18		No	81	
	NA	0		NA	12	

Scientific. ACS reagent ammonium acetate, Supelclean™ ENVI-Carb™ SPE cartridges, and fetal bovine serum were purchased from Sigma-Aldrich (St. Louis, MO). In-house deionized water was used. Native standard solution PFAC-MXC (2000 ng/mL) and mass-labelled PFC extraction standard solution MPFAC-C-ES (2000 ng/mL) were purchased from Wellington Laboratories (Ontario, Canada).

### 2.2.2. Extraction and analysis of house dust

Samples were stored at  $-20^{\circ}\text{C}$  until analyzed in the laboratory during the fall of 2021. Dust was processed using a method modified from a previously described method for PFAS dust analysis (Landrigan et al., 2006). Briefly, dust was sieved through a shaker (Gilson Company, 1-Touch Vibratory Sieve Shaker SS-10) to  $<150\ \mu\text{m}$ , transferred to 20 mL glass scintillation vials, and rotated in the x, y, and z planes for 1 min to assure homogeneous mixing. After rotation, approximately 50 mg of material was relocated to a 15 mL polypropylene centrifuge tube. 2.5 mL of methanol containing 1.2 ng/mL of MPFAC-C-ES IS solution was added to each polypropylene tube. Each tube was vortexed for 3 s followed by sonication for 30 min using a Branson 5510R-MT ultrasonicator. Each tube was then centrifuged at 3500 rpm for 10 min using a Thermo Scientific IEC CL31R Multispeed centrifuge. In the meantime, Supelclean™ ENVI-Carb™ Solid Phase Extraction (SPE) cartridges (bed wt. 250 mg, volume 3 mL) were primed with 5 mL methanol. After centrifugation, samples were poured into SPE cartridges, and the flow-through eluate collected in polypropylene centrifuge tubes. Eluate was evaporated using a Caliper Life Sciences TurboVap LV nitrogen evaporator at  $40^{\circ}\text{C}$  and 12 psi and reconstituted with methanol to a final volume of 0.5 mL. A 100  $\mu\text{L}$  aliquot of the sample was combined with 300  $\mu\text{L}$  of Mobile Phase A (95/5 deionized water/acetonitrile + 2.5 mM ammonium acetate) in a clean autosampler vial and vortexed for 30 s prior to analysis. Method blanks consisted of oOttawa Sand (20–30 mesh) grinded and sieved through a shaker. A

matrix-matched calibration curve was prepared with native standards in Ottawa Sand spiked with mass-labelled internal standards. The calibration curve was extracted exactly as described above. Table S1 provides the method reporting limit (MRL) and detection limit (DL) of each analyte.

### 2.2.3. Extraction and analysis of serum samples

Serum samples were stored at  $-80^{\circ}\text{C}$  until analyzed in the laboratory during the fall of 2021. Serum was processed using a crash-and-dilute method previously described for PFAS analysis (Kotlarz et al., 2020). Briefly, 50  $\mu\text{L}$  of serum was sub-aliquoted to a microcentrifuge tube with 100  $\mu\text{L}$  of 0.1 M formic acid containing 2.5 ng/mL of MPFAC-C-ES internal standard mix. After vortexing, an additional 450  $\mu\text{L}$  of  $-20^{\circ}\text{C}$  acetonitrile was added and further vortexed for 10 s to precipitate protein. The protein was pelleted by centrifugation for 5 min at 10,000 x g and the supernatant decanted. The supernatant was passed through an equilibrated ENVI-Carb SPE cartridge for cleanup (as described for dust), concentrated under nitrogen flow, and then reconstituted with methanol to a final volume of 0.5 mL. Samples were prepared for analysis by combining a 100  $\mu\text{L}$  aliquot of the organic extract with 100  $\mu\text{L}$  of Mobile Phase A in an autosampler vial and mixing thoroughly. Matrix-match calibration curves were prepared by spiking native PFAS into stripped fetal bovine serum and processing as samples. Table S1 provides the MRL and detection limit (DL) of each analyte.

### 2.2.4. Ultra performance (UP) LC-MS analysis

Extracted dust samples were analyzed via UPLC-MS in negative mode with a Waters ACQUITY UPLC BEH C18, 130 Å, 1.7  $\mu\text{m}$ , 2.1 mm  $\times$  50 mm column (Milford, MA) at  $55^{\circ}\text{C}$ . Extracted serum samples were analyzed via UPLC-MS in negative mode with a Restek Raptor C18, 2.7  $\mu\text{m}$ , 3 mm  $\times$  100 mm column (Bellefonte, PA) at  $55^{\circ}\text{C}$ . Both analyses used Thermo Scientific Hypersil GOLD C18, 1.9  $\mu\text{m}$ ,

**Table 2**

Summary table of dust and serum data. Includes detection rate, quartiles of concentrations, Distributed Structure-Searchable Toxicity Database (DSSTox) substance identifier DTXSIDs, and abbreviations for each compound for reference.

Name	DTXSID	Dust				Serum					
		Detection Rate (%)	Quartiles 25 ( $\mu\text{g}/\text{kg}$ )	50 ( $\mu\text{g}/\text{kg}$ )	75 ( $\mu\text{g}/\text{kg}$ )	100 ( $\mu\text{g}/\text{kg}$ )	Detection Rate (%)	Quartiles 25 (ng/mL)	50 (ng/mL)	75 (ng/mL)	100 (ng/mL)
perfluorobutanoic acid (PFBA)	DTXSID4059916	36	0.0	0.0	5.9	310	1.9	0.0	0.0	0.0	0.27
perfluorobutane sulfonic acid (PFBS)	DTXSID5030030	33	0.0	0.29	1.8	50	0.96	0.0	0.0	0.0	0.24
perfluorodecanoic acid (PFDA)	DTXSID3031860	84	1.1	3.5	7.3	310	57	0.1	0.16	0.27	1.3
perfluorododecanoic acid (PFDoDA)	DTXSID8031861	81	0.86	2.2	4.8	130	13	0.28	0.035	0.045	0.14
perfluorodecane sulfonic acid (PFDS)	DTXSID3040148	69	4.1	7.4	16	580	0.0	0.0	0.028	0.046	0.14
perfluoroheptanoic acid (PFHpA)	DTXSID1037303	82	1.3	3.9	11	130	4.8	0.0	0.0	0.0	0.29
perfluoroheptane sulfonate (PFHpS)	DTXSID8059920	42	0.11	0.19	0.59	22	14	0.067	0.12	0.17	0.39
perfluorohexanoic acid (PFHxA)	DTXSID3031862	34	0.0	0.35	8.7	130	0.0	0.0	0.0	0.0	0.0
perfluorohexane sulfonic acid (PFHxS)	DTXSID7040150	96	1.0	2.4	9.6	1200	97	0.52	0.91	1.4	3.6
perfluorononanoic acid (PFNA)	DTXSID8031863	95	1.7	3.5	13	170	3.90	0.46	0.41	0.7	4.1
perfluorononane sulfonic acid (PFNS)	DTXSID60873010	8	0.0	0.024	0.15	6.7	0.00	0.0	0.0	0.0	0.023
perfluorooctanoic acid (PFOA)	DTXSID8031865	97	6.9	13	34	730	100.00	0.74	1.2	1.8	5.2
perfluorooctane sulfonic acid (PFOS)	DTXSID3031864	92	4.9	14	37	1300	99	2.9	4.4	6.4	19
perfluoropentanoic acid (PFPeA)	DTXSID6062599	13	0.0	0.0	0.0	21	2.90	0.0	0.0	0.0	1.8
perfluoro pentanesulfonic acid (PFPeS)	DTXSID8062600	28	0.0	0.12	0.48	13	0.96	0.0	0.0	0.0036	0.13
perfluoroundecanoic acid (PFUnDA)	DTXSID8047553	89	0.77	1.9	4.8	46	0.00	0.0	0.15	0.22	0.62

3 × 50 mm as a delay column (Waltham, MA) as part of standard practice. A reversed-phase binary gradient via a Thermo Vanquish Horizon UPLC was used for all analyses as shown in Tables S2 (dust) and S3 (serum). Mass spectrometry quantitation was conducted on a Thermo Orbitrap Fusion (dust) or Thermo Quantis Triple Quadrupole (serum) using internal standard corrected calibration curves. Table S4 lists the Orbitrap parameters and acquisition settings. Table S5 details the instrument parameters and acquisition settings and Table S6 lists each analyte's mass transitions.

### 2.2.5. QA/QC Criteria

Method blanks were repeatedly analyzed for serum and dust to ensure clean background, every 20 samples for serum, every 60 samples for dust. All reported analytes were consistently > MRL. Replicate samples were prepared for a random 10 % subsample of all analyzed samples and reproducibility calculated with an average relative percentage difference <10 %. Accuracy was maintained with continuous calibration verification standards every ten injections, alternating 15 and 75  $\mu\text{g}/\text{kg}$  for dust and 5 and 15 ng/mL for serum.

### 2.2.6. Data processing

After data collection, chromatograms were processed, and peak areas integrated in Thermo Scientific Xcalibur Quan Browser 4.3. Native standard peak areas were matched against internal standard peak areas according to Table S7 and response ratios were calculated. Statistical analyses were conducted using RStudio (Boston, MA). MRLs and DLs (Table S1) were estimated for each compound/batch/matrix using repeated injections of the calibration curve and the LCMRL package (John et al., 2021).

### 2.3. Questionnaire information

We selected questionnaire questions based on known or suspected connections to house dust or PFAS exposure as informed by previous studies (National Institute of Child Health and Human Development, 2016b; Landrigan et al., 2006). Questions focused on participants' sociodemographic and housing factors to identify possible associations with PFAS exposure.

Multiple questionnaires were administered during the initial study at times ranging from prenatal to thirty-six months postnatal; meanwhile, serum samples in this study were obtained during or prior to pregnancy (Rovira et al., 2019; Verner et al., 2016; Branum et al., 2003; National Institute of Child Health and Human Development, 2016a). Table S8 shows which questions were taken from which questionnaires and the timing of sampling and questionnaire administration in the study. When selecting data for questions that were administered postnatally, we only used questions that could reasonably be assumed to have remained consistent since pregnancy. We assumed that the ratio of carpeted rooms, the type of building that the participant lived in, and the amount of time per day the family had their windows open could all be informed by questionnaires administered 6–36 months postnatally, when no earlier answer was available. Where participants answered the same question multiple times across different questionnaires, we aggregated these answers to a single answer by using only the available answer from the questionnaire administered most contemporaneously to sample collection for each participant. Table S9 shows the percentage of answers used at each time point for each question within the context of the overall sampling timeline. For questions on educational attainment, categories of “high school diploma” and “some college” were combined to reduce the number of variables and increase sample size per response.

Questions about recent renovations focused on several time periods, but we kept and combined only questions focused on “6 months before birth”, “since becoming pregnant”, or “12 months before pregnancy”, because these periods were before house dust sampling. They were combined into a single “recent renovation” variable covering the entire pre-dust sampling period. Some questionnaires were not administered to all participants; for example, relatively few participants provided responses for building type and carpet in the home. A summary of data from the selected questionnaire questions is shown in Table 1.

We modeled relationships between dust and serum PFAS concentrations and explored associations between these concentrations and questionnaire responses. Before statistical analysis, we assessed if the data conformed to the assumptions of linear regression and ANOVA. Normality of residuals was assessed visually using QQ plots; linearity was assessed by plotting the data; heteroscedasticity was checked using a scatterplot of residuals; homogeneity of variance was checked using Levene’s test; sample independence was met through the NCS experimental design. Summary statistics were calculated for all PFAS using base functions in R (John et al., 2021; R Core Team, 2023). When National Health and Nutrition Examination Survey (NHANES) 2011–2012 data was used as a comparator, confidence intervals for mean serum PFAS levels were calculated using the methods described in the NHANES analytical guidelines (National Health and Nutrition Examination Survey, 2018; National Health for Health Statistics, 2011). R code for all analyses can be found in supplementary materials.

### 2.3.1. Data pre-processing

Values below the detection limit (DL) were single value imputed as  $\frac{DL}{\sqrt{2}}$ . Detection limits are listed in Table S1. Because serum and dust PFAS concentration data were log-normally distributed, they were ln-transformed before analysis. Only PFAS with detection frequencies greater than 50 % in the relevant medium (dust or serum) were included in statistical analysis. For tests of associations between dust and serum, only PFAS with detection frequencies greater than 50 % in both media were included.

### 2.3.2. Linear regression

We used linear regression to assess the relationship between ln-transformed PFAS concentrations in house dust and serum. Linear regression was also used to identify relationships between continuous predictors from the questionnaire data (i.e., age of participant and time lived in home) and ln-transformed PFAS concentrations in dust or serum. Linear regression was performed with the *lm* function in R version 4.3.0 (R Core Team, 2023).

### 2.3.3. ANOVA

Analysis of variance (ANOVA) tests were performed using the *aov*

$$\text{Total Serum Concentration} = \frac{\text{Daily Intake}(DP)}{\text{Excretion Rate}(K_p) \times \text{Volume of Distribution}(V_d)} = \frac{\text{total estimated intake}}{K_p \times V_d} \quad (3)$$

function in R version 4.3.0 (R Core Team, 2023) to model univariate relationships between ln-transformed PFAS dust and serum outcomes and categorical variables from the NCS questionnaire. Any significant relationship ( $p < 0.05$ ) identified using ANOVA was further characterized using Tukey’s Honestly Significant Difference (HSD) to identify pairwise relationships between PFAS concentrations and specific questionnaire answers.

### 2.3.4. Multiple regression and model selection

We used model selection to determine the best model to explain

PFAS levels in household dust using NCS questionnaire data. Model selection was performed separately for each compound using stepwise selection in both directions with the *MASS* package in R using the Akaike information criterion (AIC) as the measure of model fit (Venables and Ripley, 2002). Five variables were chosen for the initial model based on significant relationships in the univariate models: participant age, age of home, military household status, income level, and educational attainment. The full suite of variables and their interactions could not be considered due to sample size constraints and available degrees of freedom.

### 2.4. Estimating the percent of serum levels attributable to dust exposure

To estimate the relative importance of PFAS exposure through house dust, we calculated the percent of serum levels attributable to dust pathways using methods previously published by DeLuca et al. (DeLuca et al., 2022). Daily PFAS intake rates due to dust ingestion and dermal absorption were estimated using mechanistic exposure models, and then steady-state serum level models were estimated using a one compartment pharmacokinetic mode (Eqs. (1) and (2)). Parameters for the model and their sources can be found in Table S10 (Fasano et al., 2005; Pang et al., 2002; Jacqueline Moya et al., 2017). PFAS concentrations were assumed to be at steady state at the time of measurement, which may be reasonable due to the long half-lives of PFAS and their ubiquity in the environment which leads to long-term exposures from a range of sources and pathways. For PFAS where volume of distribution ( $V_d$ ) estimates were not available, the  $V_d$  for PFOA was used instead, as studies using animals found that PFAS have very similar  $V_d$ s (Ohmori et al., 2003; Lynch et al., 2023; Poothong et al., 2020). The mean percent of serum levels attributable to dust exposure for each compound was calculated by dividing the estimates of total serum concentration from dust (Eq. (3)) by the geometric means of the PFAS concentrations in the serum of participants.

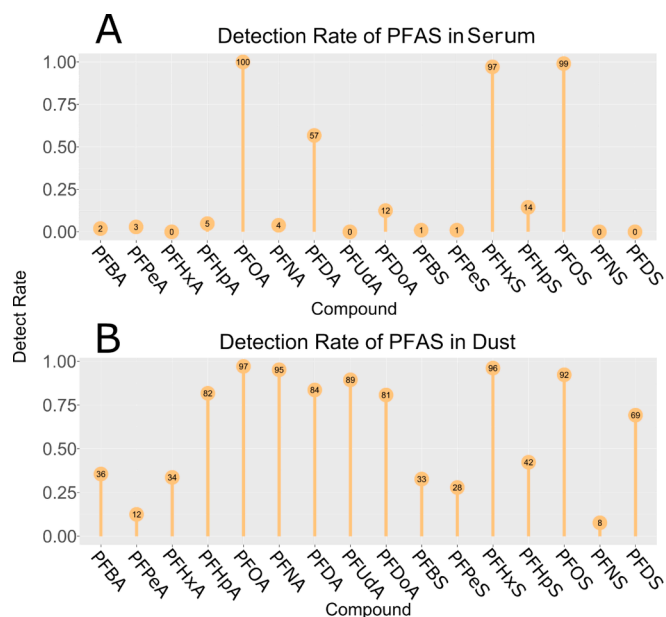
$$\text{Ingestion} \left( \frac{\text{ng}}{\text{day}} \right) = \text{Concentration} \left( \frac{\text{ng}}{\text{ml}} \right) \times \text{Intake Rate} \left( \frac{\text{gdust}}{\text{day}} \right) \times \text{Gastrointestinal Absorbtion Fraction}(\%) \quad (1)$$

$$\text{Dermal Absorbtion} \left( \frac{\text{ng}}{\text{day}} \right) = \text{Concentration} \left( \frac{\text{ng}}{\text{ml}} \right) \times \text{Dust Load} \left( \frac{\text{Dust}}{\text{m}^2} \right) \times \text{Transfer Coefficient} \left( \frac{\text{m}^2}{\text{hr}} \right) \times \text{Time} \left( \frac{\text{hr}}{\text{day}} \right) \times \text{Dermal Absorbtion Fraction}(\%) \quad (2)$$

## 3. Results

### 3.1. Study population

The analyzed subset of the Vanguard Study had participants aged between 18 and 34 years old with a median age of 28. The median time that participants had lived in their homes was 2 years, with 44 % making \$50,000 or more per year, and 48 % having a bachelor’s or post-graduate degree. Data for other sociodemographic and housing characteristics of this population can be found in Table 1.



**Fig. 1.** Lollipop plots of PFAS detection rates in serum (A) and dust (B). The detection rates are represented by the length of the lollipops and the numbers in the top of the lollipop are the actual percent detect.

### 3.2. PFAS in serum

Serum samples were analyzed for 16 PFAS. Detection frequencies and concentration quartiles can be found in Table 2. Serum detection frequencies and concentrations are also presented in Fig. 1a and Fig. 2a. Four PFAS were detected in over 50 % of serum samples: PFOS, PFOA, PFHxS and PFDA. The geometric means (GMs) of those four PFAS were, from highest to lowest: 4.1 ng/mL for PFOS, 1.1 ng/mL for PFOA,

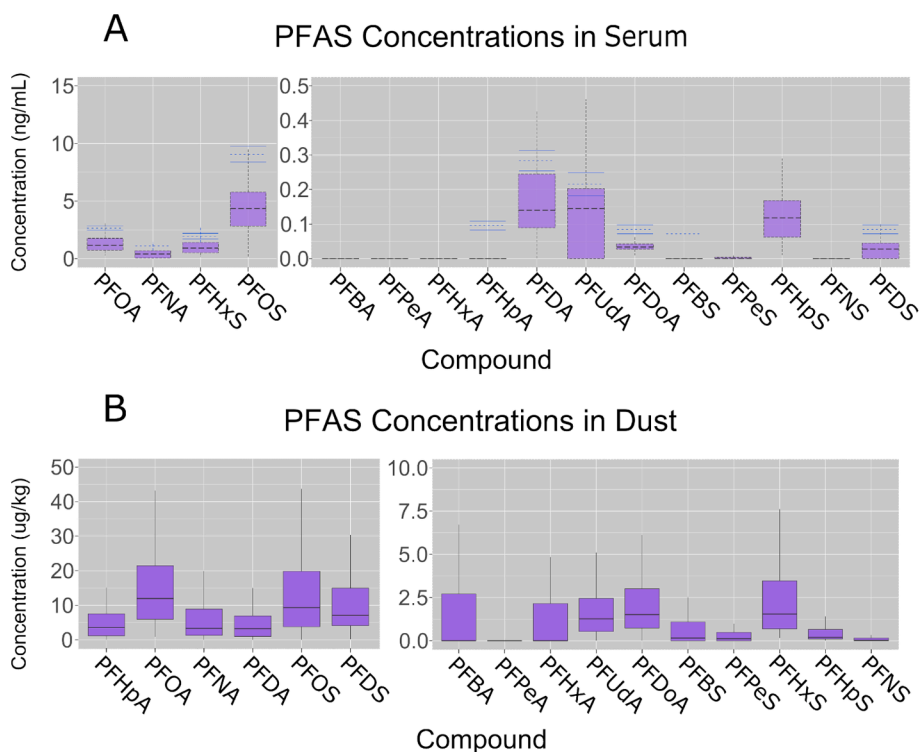
0.87 ng/mL for PFHxS, and 0.16 ng/mL for PFDA.

### 3.3. PFAS in dust

House dust samples were also analyzed for 16 PFAS. Detection frequencies and concentration quartiles can be found in Table 2. Dust detection frequencies and concentrations are also presented in Fig. 1b and Fig. 2b. Of the 16 PFAS analyzed in house dust, nine were detected in over 50 % of homes. The compounds PFOS, PFOA, PFHxS and PFNA were detected in over 90 % of homes, while PFUdA, PFDA, PFHpA, PFDoA and PFDA were detected in between 69–89 % of homes. The geometric means (GMs) of those compounds were, from highest to lowest: 17 µg/kg for PFOS, 16 µg/kg for PFOA, 9.6 µg/kg for PFDS, 4.5 µg/kg for PFHpA, 4.4 µg/kg for PFNA, 3.8 µg/kg for PFHxS, 3.5 µg/kg for PFDA, 2.3 µg/kg for PFDoA, and 2.1 µg/kg for PFUdA. Detection rates and more detailed summary statistics for all analyzed PFAS can be seen in Fig. 1b and Fig. 2b, respectively.

### 3.4. Relationship between serum and dust

Summed levels of the four PFAS that were detected in over 50 % of serum and dust samples were positively correlated ( $p = 0.039$ ) in serum and dust according to linear regression. When considering individual compounds, concentrations of PFOA in serum and dust were positively associated ( $p = 0.017$ ). However, no significant relationships were identified between serum and dust concentrations for PFOS, PFHxS or PFDA ( $p = 0.42$ ,  $p = 0.41$ ,  $p = 0.22$ , respectively). To test if the summed PFAS results were driven entirely by PFOA, PFOA was removed from the sum and the linear regression was repeated. Without PFOA, the p-value ( $p = 0.073$ ) was greater than our significance threshold.



**Fig. 2.** Mean PFAS concentrations in serum (A) and dust (B). Please note the different scales in the plots on the left and right. The PFAS concentration in serum is being compared to the confidence intervals of PFAS measurements available from 2011 to 2012 NHANES which are shown as blue lines bounding the 95% confidence interval in blue with the means as dotted lines.

## Significant Associations Between Blood or Dust and Lifestyle or Demographic Factor

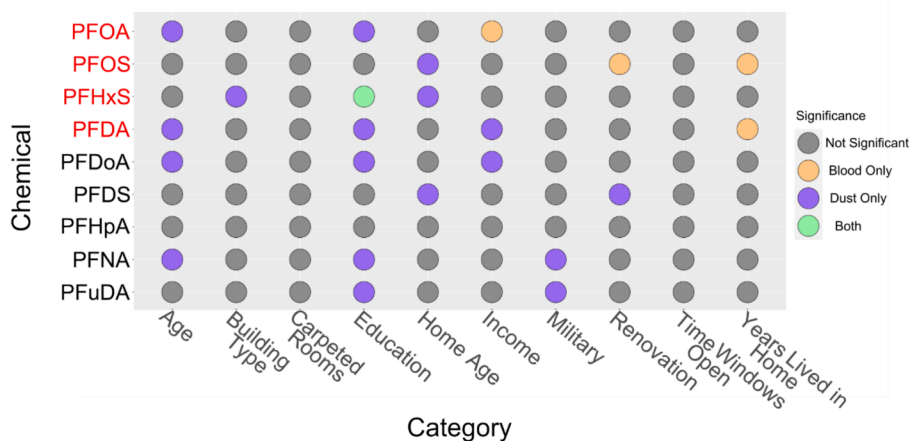


Fig. 3. PFAS with significant ( $p < 0.05$ ) associations for questionnaire data. The PFAS listed in red are the PFAS analyzed in both serum and dust. PFAS detected in  $> 50\%$  of samples in their respective medium were analysed.

### 3.5. Relationships between PFAS in household dust and housing and sociodemographic factors

#### 3.5.1. Univariate and multiple regression models

Significant associations were identified between the concentrations of one or more PFAS in dust and multiple sociodemographic and housing factors (Fig. 3). The best multiple regression models for predicting dust concentrations were chosen based on having the highest AIC. These models varied amongst the individual PFAS.

Higher participant age was significantly associated with increased dust PFOA ( $p = 0.015$ ), PFNA ( $p = 0.037$ ), PFDoA ( $p = 0.023$ ), and PFDA ( $p = 0.016$ ) concentrations. A higher level of educational attainment was tied to higher levels of PFuDA ( $p = 0.015$ ), PFOA ( $p = 0.041$ ), PFNA ( $p = 0.020$ ), PFHxS ( $p = 0.021$ ), PoFDoA ( $p = 0.010$ ), PFDA ( $p = 0.003$ ), and all PFAS generally ( $p = 0.045$ ). The most predictive multivariate model for PFHxS only contained education as a variable. Higher income households ( $\$100,000$ – $\$199,999$ ) were associated with higher levels of PFDA ( $p = 0.0028$ ), and PFDoA ( $p = 0.018$ ) in household dust when compared to the lowest income households. Additionally, if the participant indicated that a current or former military member lived in their home, there were significantly higher levels of PFNA ( $p = 0.0046$ ), and PFuDA ( $p = 0.0044$ ). The best multiple regression models for PFNA and PFuDA simplified down to univariate models with military household. However, this question only had three respondents that indicated they were in a military household out of a total of 63 households with available information.

PFHxS concentration in dust varied significantly ( $p = 0.012$ ) based on the type of building participants lived in. Specifically, trailers had higher concentrations of PFHxS in dust compared to townhome and/or multifamily ( $n = 3$ ). A higher proportion of carpeted rooms in the home was not individually associated with PFAS levels in ANOVA. Older homes were also associated with higher PFAS in the dust for PFDS ( $p = 0.0074$ ), PFHxS ( $p = 0.0037$ ), and PFOS ( $p < 0.001$ ). Multivariate models with the highest AICs for PFOS and PFDS were also univariate models with home age. Furthermore, homes with recent renovations had significantly higher levels of PFDS ( $p = 0.019$ ).

In one case, PFHpA, a model with no variables had the lowest AIC, indicating that none of the models were superior for predicting the concentration of PFHpA over any other. The best model for PFDA included participant age, home age, and annual income category. The only significant ( $p = 0.029$ ) estimate in the model predicted that PFDA in dust was  $1.2 \mu\text{g}/\text{kg}$  lower in homes built in 1981–2000 when compared to homes built after the year 2000. In other variables, higher participant age was estimated to increase dust PFDA concentration by

$0.59 \mu\text{g}/\text{kg}$  per year and higher income was estimated to cause higher PFAS exposure compared to the lowest income bracket. For home age, the categories that weren't significant, all years of which were prior to PFAS going on the market, had coefficients lower than the newest homes in the model.

The best model for PFDoA concentrations in dust according to AIC included participant age, home age, and income. Three categories of income were significantly associated with increased levels of PFAS:  $\$10,000$ – $\$19,000$  ( $p = 0.007$ ),  $\$30,000$ – $\$39,000$  ( $p = 0.032$ ), and  $\$100,000$ – $\$199,000$  ( $p = 0.017$ ), with estimated increases of  $1.95 \mu\text{g}/\text{kg}$ ,  $1.70 \mu\text{g}/\text{kg}$ , and  $1.65 \mu\text{g}/\text{kg}$ , respectively, compared to the lowest income group. For home age, homes built between 1981–2000 were predicted to have significantly lower PFDoA than the newest homes in the study by  $0.99 \mu\text{g}/\text{kg}$ . For each year of increasing participant age, the model predicted a  $0.039 \mu\text{g}/\text{kg}$  increase in PFDoA exposure. For the remaining model coefficients, higher income was primarily associated with higher exposures, while older homes were associated with lower exposures. Table S8 summarizes multiple regression results.

#### 3.6. Percent of serum levels attributable to dust exposure

The estimated mean PFAS concentrations in the participants' serum that were attributable to dust exposure pathways (shown in Table 3) were between  $0.012$  and  $0.14 \text{ ng}/\text{mL}$ . When compared to the actual serum levels, these dust-attributable estimates represented between  $3\%$  (PFNA) and  $25\%$  (PFDA) of the total concentrations. The vast majority of dust PFAS exposure occurred through the ingestion pathway, with dermal exposure accounting for under  $5\%$  of the total serum PFAS.

## 4. Discussion

### 4.1. Associations between dust exposure and serum PFAS levels

Previous research suggests that settled dust can be a significant pathway for PFAS exposure (DeLuca et al., 2022; Sunderland et al., 2019; Poothong et al., 2020; Egeghy, 2011; Lorber, 2011), and here we found that pregnant people with higher total PFAS levels in their house dust had higher total PFAS serum levels, which supports the potential importance of these pathways. Previous studies found that dust related pathways, primarily ingestion but, less so, dermal absorption, may be responsible for up to  $50\%$  of serum PFAS levels for some PFAS (DeLuca et al., 2022; Haug et al., 2011; Egeghy, 2011; Lorber, 2011; Trudel et al., 2008; Kim and Lee, 2019; Gebbink and Berger, 2015; Gomis et al., 2017; Zheng et al., 2020). However, it is important to note that dust can also

**Table 3**

Estimated serum contributions of dust to serum levels for different PFAS. The total percent contribution was summed from the contributions of ingestion and dermal absorption.

compound	Percent from Dust (total)	SE % Dust ( $\pm$ )	Percent from Dust Ingestion	Percent from Dust Dermal	Actual Mean Serum Levels (ng/ml)	Mean Estimated Serum Levels from Dust (ng/ml)
PFHPA	16	2.8	15	0.77	0.076	0.012
PFNA	3.0	0.52	2.8	0.14	1.2	0.038
PFOS	3.8	1.2	3.7	0.19	4.8	0.14
PFDA	25	4.9	24	1.20	0.21	0.044
PFHXS	14	7.3	13	0.68	1.1	0.098
PFOA	7.1	1.4	6.7	0.34	1.40	0.088

act as a PFAS sink and indicator for range of PFAS sources within the home (Fraser et al., 2013; Hall et al., 2020). For example, PFAS in dust can reflect many household and consumer products including building materials, cosmetics, food packaging, clothing, furniture, rugs, and carpets. While the apparent associations between serum and dust PFAS levels may suggest dust as an exposure pathway, they could also reflect that homes with high dust PFAS levels have other relevant indoor exposure sources that are releasing PFAS into the household environment.

While total PFAS levels were correlated for serum and dust, this relationship appeared to be largely driven by PFOA, which was the only compound that had a significant serum-dust relationship when analyzed individually. Based on the myriad possible sources of PFAS exposure within the home, the lack of results for PFHxS, PFOS and PFDA may be caused by differences in exposure and use profiles between the compounds. For example, PFHxS and PFOS, which were measured in high levels in dust, may have many additional residential and non-residential exposure sources and pathways that obscure the signal of dust exposure and reduce our ability to detect this relationship or dust and serum may share a residential source of PFAS deposition that is being detected indirectly here. Alternatively, some PFAS may be absorbed or excreted more readily via dust or other pathways based on their chemical properties. PFAS have been shown to have considerably different half-lives and toxicokinetic properties across different chain lengths and head groups, for example their ability to bind to serum albumin, which could affect the partitioning and excretion of different PFAS (Starnes et al., 2023; Wallis et al., 2023; Andersen et al., 2006; Chang et al., 2012; Chengelis et al., 2009).

While PFOA has been largely phased out of use, it is still found in the environment, probably due to its long environmental half-life and the fact that it is the terminal degradation product of several PFAS precursors (Schellenberger et al., 2022; McDonough et al., 2022). These factors combined with the fact that the samples were taken between 2009 and 2014, when products containing phased out PFAS may not have been replaced yet, may be contributing to the PFOA levels in the dust samples of this study.

#### 4.2. Presence of PFAS in the serum and dust of pregnant people

Levels of PFAS in the NCS participants' serum were slightly lower for most compounds measured than in the 2011–2012 NHANES (Centers for Disease Control and Prevention (CDC), 2020; National Center for Health Statistics, 2011) While the study sampling design did not allow us to consider the study cohort 'nationally representative', participants were broadly recruited from across the U.S. without targeting areas with specific PFAS impacts. While we consider the aggregated results to be roughly reflective of 'typical exposure' for the general population, individual participants could have been located in communities with elevated PFAS levels in media such as drinking water. The median U.S. household income in 2011 was \$50,502, while the median income group in this population was \$50,000–\$74,999 (Noss, 2012). However, the study sample had higher educational attainment than the U.S. population in 2011; 31 % had a bachelor's degree compared to 18 % in the U.S. as a whole, and 17 % had a postgraduate degree compared to 8 % (Digest

of Education Statistics, 2011). The socioeconomic differences in educational attainment could influence the exposure profile of this cohort. A handful of studies have also looked at PFAS levels in house dust in the U.S. (Wu et al., 2015; Karaskova et al., 2016; Minucci et al., 2024; Zheng and Eick, 2023) In general this cohort has lower levels of PFAS in their dust, similar to the levels in their serum. Notable exceptions to this are PFDoA and PFUdA which were similar to levels found in other studies such as Karaskova et al. which found levels of PFDoA and PFUdA at an average of 2 and 3.6 ng/g respectively (Wu et al., 2015; Karaskova et al., 2016; Minucci et al., 2024; Zheng and Eick, 2023). The PFAS levels in the cohort's serum are lower than those in NHANES, despite being a highly educated population, which a recent systematic review suggests may have higher levels of some PFAS (DeLuca et al., 2022). However, other studies have found mixed evidence on the association between education level and PFAS body burden, which suggests a more complicated relationship between PFAS exposure and sociodemographic factors (DeLuca et al., 2022; Sagiv et al., 2015; Park et al., 2019). Pregnant people often avoid certain foods and items that are harmful to the unborn child while they are particularly vulnerable to what the parent interacts with. This could be a cause for differences in PFAS exposure that is specific to this cohort. Recently, researchers published two different studies which analysed dust in households for PFAS. (Hall et al., 2020; DeLuca et al., 2024) Comparing the results here to those, more recent, measurements show that the levels of PFAS in their dust may be comparable to PFAS levels in other households' dust (table S11).

#### 4.3. Associations between housing and socioeconomic factors and PFAS levels

While PFAS are widespread in the environment, we found socio-demographic and housing characteristics that were associated with the levels of PFAS in dust. Older participants in this study had a significantly higher level of PFOA, PFDA, PFDoA, and PFNA in their dust. This may be related to certain age-related behaviors such as reduced frequency of cleaning or owning older products or home furnishings (Zheng and Eick, 2023; Young et al., 2022). Although this cohort is relatively young, with a small age range of 18–34, even a 16-year difference in the average age of home furnishings could impact PFAS levels due to the recent phase out of long chain legacy PFAS and replacement with shorter chain alternatives. Age has been previously linked to higher dust PFAS levels, albeit in an older study population with a much larger range of ages (Minucci et al., 2024).

Older homes were also linked to higher PFAS dust levels. These homes may be more likely to contain older furnishings or building materials from before the legacy PFAS phase out, which could be reflected in dust (Savvaides et al., 2021; Young et al., 2022). Older homes have been tied to higher levels of PFAS in previous studies (DeLuca et al., 2022; DeLuca et al., 2023). Additionally, when homes in this study had been renovated recently, we found higher levels of PFAS in both dust and serum. This suggests that PFAS may have been released from new building materials or furnishings introduced during renovation, or from older materials that were disturbed, leading to increased exposure. Increased levels of lead and other contaminants in the dust of buildings



being renovated have been observed in previous studies (Spanier et al., 2013; Latif et al., 2011).

Carpets have been repeatedly shown to harbor PFAS due to stain-proof treatments applied during or after manufacturing and because carpets can act as a sink for environmental materials like dust more efficiently than other flooring types (Herzke and Olsson, 2012; Savvaides et al., 2021; Young et al., 2022; Zhu and Ro, 2021). Yet we did not find any significant associations between the proportion of the home covered by carpeting and dust PFAS levels for any of the compounds analyzed. However, the NCS questionnaire did not ask specifically about the type of carpet, or the total square footage of the home covered by carpet. This lack of information, along with the low sample size of participants answering this question, may explain why we did not observe an association between carpeting and dust PFAS levels.

For most of the PFAS analyzed, stepwise model selection found that the best model to predict dust concentrations was a univariate model with just one predictor, essentially replicating the findings of the univariate analysis. However, multiple predictors were selected for the best models to predict PFOS and PFOA levels in serum, and PFDA and PFDoA levels in dust. However, model selection largely served to reinforce the univariate model findings and did not produce any new associations with housing or socioeconomic factors.

Many studies investigating PFAS have shown higher levels of PFAS in the serum of military members (Nair et al., 2021; Sunderland et al., 2019; Backe and Day, 2013; Barzen-Hanson et al., 2017; Barton et al., 2020), which has been tied to the use of aqueous film forming foam (AFFF) (Sunderland et al., 2019; Barzen-Hanson et al., 2017). One study showed that PFNA was among the compounds with the highest levels in military member's serum. However, few studies have investigated military households and their families. PFNA, PFuDA and the combined suite of PFAS were higher in house dust from households that identified as having a former or current military member. This finding should be considered tentative, as only three of 63 responding households identified themselves as military households. However, it suggests a possible link between occupational exposure in military personnel and exposure in their home, and even to their family members. Military members could transfer PFAS from their duty stations to their homes, and subsequently to house dust, by carrying it on their clothing, shoes, or bodies. Further studies on military households and PFAS exposure via house dust could elucidate this possible PFAS exposure pathway and inform ways to mitigate this exposure.

#### 4.4. Socioeconomic status and residential PFAS exposure trends

Many complex sociodemographic factors are associated with exposure to environmental contaminants. For example, historical redlining can affect whether someone lives near industrial point sources of pollution, and educational level can determine whether one's job exposes them to noxious chemicals or not (Flanagan and Hallisey, 2020; Sadd et al., 2011; Warner, 2007). Often, lower educational attainment and/or lower socioeconomic status (SES) have been linked to increased chemical exposure risk. (Flanagan and Hallisey, 2020; Sadd et al., 2011; Warner, 2007; Amaro et al., 2021; Perles Roselló and Vías Martínez, 2009; Souza et al., 2021; Wolkin et al., 2015) However, in this study, higher levels of PFDoA, PFDA and PFOA in dust and/or serum were associated with higher income households and higher educational attainment. In the case of these PFAS, higher SES could be linked to greater exposures due to differing consumer product use and purchase patterns. For example, higher SES households may purchase more premium outdoor clothing and equipment, which are often waterproofed with PFAS and have been identified as a possible exposure source (Gluge et al., 2020; Herzke and Olsson, 2012; Xia et al., 2022; van der Veen et al., 2022; DeLuca et al., 2021; Eichler, 2020). However, more research is needed to examine how consumer product use and purchase patterns affect potential PFAS exposures. Additionally, this observed income-to-exposure relationship may not hold for highly impacted communities. It

has been well documented that industrial point source pollution and chemical releases are more likely to occur near vulnerable communities, such as economically disadvantaged groups and people of color (Flanagan and Hallisey, 2020; Sadd et al., 2011; Souza et al., 2021; Wolkin et al., 2015).

#### 4.5. The proportion of PFAS body burden attributable to dust exposure pathways

We estimated PFAS intakes for two residential exposure pathways, dust ingestion and dermal absorption from dust. Of these two pathways, our estimates indicated that dust ingestion was the primary pathway, contributing over 95 % of the total estimated dust related PFAS intakes for each chemical. This is almost certainly due to PFAS' higher rate of absorption in the gastrointestinal system. However, due to a lack of indoor air measurements, we were not able to estimate intakes due to inhalation, which may be a major residential exposure pathway for more volatile PFAS. One shortcoming of our exposure and pharmacokinetic modeling approach is the current lack of detailed understanding of PFAS behavior inside the body. A simple one compartment model may not be reflective of actual conditions, yet data is lacking for most PFAS to parameterize more complex models.

Although food and water ingestion are believed to be the primary PFAS exposure pathways, several recent studies have shown that PFAS exposure via dust is also an important contributor to PFAS burden in the body (DeLuca et al., 2022; Sunderland et al., 2019; Poothong et al., 2020; Egeghy, 2011; Lorber, 2011; Minucci et al., 2024) and houses with drinking water contamination still exhibit a connection between PFAS levels in dust and serum (Minucci et al., 2024). For PFOS, studies have estimated the contribution of dust pathways to range from 1 % to as much as 15 % of total exposure, and here we estimated a contribution of 4 % of total serum PFOS levels in this pregnant cohort (DeLuca et al., 2022; Sunderland et al., 2019; Poothong et al., 2020; Egeghy, 2011; Lorber, 2011). For PFHxS, some estimates put dust contributions as high as 16 % of total exposure, which is comparable to the 14 % estimated in this group as well as the estimate for PFHpA (DeLuca et al., 2022; Sunderland et al., 2019; Poothong et al., 2020; Egeghy, 2011; Lorber, 2011). Individuals' percent contribution from dust likely varied based on the amount of dust in their homes, the PFAS levels in their dust and the magnitude of their other, unmeasured exposure sources such as drinking water accounting for variability in these estimates, while differences across PFAS may be accounted for by differences in how the PFAS are absorbed. Less studied compounds like PFHpA on the other hand, do not have previous estimates and this study provides some important parameters for understanding PFAS exposure in the home.

## 5. Limitations

This study had some limitations that could affect interpretation of the results. The sample size was relatively small at 104 which could have reduced the power of any effects being analyzed. The samples were also collected around ten years before being analyzed for this study, and some PFAS in these samples may have degraded or otherwise been lost over time despite storage at  $-20^{\circ}\text{C}$  (Bach et al., 2015). However, most of the PFAS we analyzed have relatively low volatility and long half-lives and therefore may be less susceptible to losses through time. Additionally, since the dust and serum samples were collected without the knowledge that they would eventually be analyzed for PFAS, sampling measures to prevent PFAS contamination were not taken and we did not have field blanks to analyze, which leads to uncertainty about potential sample contamination. This may have obscured some of the significant relationships being analyzed. While 16 PFAS were analyzed, PFAS precursors were not present in this analysis. Increasingly, these are being recognized as prominent PFAS in indoor environments and sources of PFAS exposure as they can transform into more well known PFASs and PFCAs (Gebbinck and Berger, 2015; McDonough et al., 2022; Butt and

Muir, 2014; Dinglasan et al., 2004; Gebbink and Glynn, 2015).

### 5.1. Residential PFAS exposure and children's health

The NCS was designed to study environmental influences on child health and development, a particularly vulnerable population. House dust is known to be a more important exposure source for children than adults (Hubbard et al., 2022; Ozkaynak et al., 2022) due to greater time spent near the ground and higher hand-to-mouth behavior (Savvaides et al., 2021; Winkens et al., 2018; Haug et al., 2011). Furthermore, this window of exposure to environmental contaminants can be especially detrimental because of early development (Barouki et al., 2012). Environmental measurement via dust and biomonitoring via serum samples are good indicators of PFAS exposure and understanding the link between serum and dust PFAS levels serves as an important step in building a meaningful map of the pathways by which PFAS exposure occurs in children and child-bearing people. This study identified PFAS levels in the serum and dust of a vulnerable population of people, examined the connection between the levels of PFAS in serum and dust, and identified sociodemographic and residential factors that were associated with PFAS levels in serum and dust. Understanding the factors that contribute to PFAS exposure and PFAS levels in dust could help policymakers and medical professionals provide better protections against PFAS exposure. A deeper understanding of the PFAS in serum and dust and a clearer picture of PFAS exposure improves interventions, policy, and helps to establish a sturdier base for future research to build upon.

## 6. Disclaimer

This paper has been reviewed in accordance with Agency policy and approved for publication. The views expressed in this article are those of the authors and do not necessarily represent the views or policies of the U.S. EPA.

## Author contributions

DW drafted the manuscript. DW, JMM and ND performed analysis. CF procured laboratory supplies. KEM designed and developed the analytical methods. KEM and CF conducted sample preparation. KEM performed the experiments and collected the data. JM supervised the experiments and processed the data. ECH and KT designed and conducted sampling. JMM and ECH supervised the analysis and secured funding. DJW, JMM, KT, ECH, JM, and ND contributed scientific expertise during analysis and writing. All authors contributed to the study design and manuscript editing. All authors agreed to accountability for the content.

## CRedit authorship contribution statement

**Dylan James Wallis:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Kelsey E. Miller:** Writing – review & editing, Resources, Methodology, Investigation, Formal analysis, Data curation. **Nicole M. DeLuca:** Writing – review & editing, Formal analysis, Conceptualization. **Kent Thomas:** Writing – review & editing, Resources, Investigation, Data curation, Conceptualization. **Chris Fuller:** Resources. **James McCord:** Writing – review & editing, Methodology, Formal analysis. **Elaine A. Cohen Hubal:** Writing – review & editing, Methodology, Data curation, Conceptualization. **Jeffrey M. Minucci:** Writing – review & editing, Supervision, Resources, Funding acquisition, Data curation, Conceptualization.

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## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.envint.2024.109157>.

## Data availability

The authors do not have permission to share data.

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