HHS Public Access

Author manuscript

Environ Int. Author manuscript; available in PMC 2024 May 13.

Published in final edited form as:

Environ Int. 2024 March ; 185: 108454. doi:10.1016/j.envint.2024.108454.

Associations of dietary intake and longitudinal measures of per- and polyfluoroalkyl substances (PFAS) in predominantly Hispanic young Adults: A multicohort study

Hailey E. Hampson[a], **Elizabeth Costello**[a], **Douglas I. Walker**[b], **Hongxu Wang**[a], **Brittney O. Baumert**[a], **Damaskini Valvi**[b], **Sarah Rock**[a], **Dean P. Jones**[c], **Michael I. Goran**[d], **Frank D. Gilliland**[a], **David V. Conti**[a], **Tanya L. Alderete**[e], **Zhanghua Chen**[a], **Leda Chatzi**[a], **Jesse A. Goodrich**[a,*]

[a]Department of Population and Public Health Sciences, Keck School of Medicine, University of Southern California, Los Angeles, CA, the United States of America

[b]Department of Environmental Medicine and Public Health, Icahn School of Medicine at Mount Sinai, New York, NY, the United States of America

[c]Clinical Biomarkers Laboratory, Division of Pulmonary, Allergy, Critical Care and Sleep Medicine, Emory University, Atlanta, GA, the United States of America

[d]Department of Pediatrics, Keck School of Medicine, USC and The Saban Research Institute, Children's Hospital Los Angeles, Los Angeles, CA 90027, the United States of America

[e]Department of Integrative Physiology, University of Colorado Boulder, Boulder, CO, the United States of America

Abstract

Background: Per- and polyfluoroalkyl substances (PFAS) are pollutants linked to adverse health effects. Diet is an important source of PFAS exposure, yet it is unknown how diet impacts longitudinal PFAS levels.

*Corresponding author at: 1845 N. Soto Street, Los Angeles, 90032, USA. jagoodri@usc.edu (J.A. Goodrich).

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.

CRediT authorship contribution statement

Hailey E. Hampson: Conceptualization, Data curation, Formal analysis, Methodology, Software, Validation, Visualization, Writing – original draft, Writing – review & editing. **Elizabeth Costello:** . **Douglas I. Walker:** Conceptualization, Funding acquisition, Project administration, Writing – review & editing. **Hongxu Wang:** Conceptualization, Data curation, Methodology, Software, Writing – review & editing. **Brittney O. Baumert:** . **Damaskini Valvi:** . **Sarah Rock:** Conceptualization, Investigation, Project administration, Writing – review & editing. **Dean P. Jones:** Conceptualization, Funding acquisition, Project administration, Writing – review & editing. **Michael I. Goran:** Conceptualization, Funding acquisition, Project administration, Writing – review & editing. **Frank D. Gilliland:** Conceptualization, Funding acquisition, Project administration, Writing – review & editing. **David V. Conti:** Conceptualization, Methodology, Writing – review & editing. **Tanya L. Alderete:** Conceptualization, Funding acquisition, Project administration, Writing – review & editing. **Zhanghua Chen:** Conceptualization, Funding acquisition, Investigation, Project administration, Writing – review & editing. **Leda Chatzi:** Conceptualization, Funding acquisition, Investigation, Project administration, Supervision, Writing – review & editing. **Jesse A. Goodrich:** .

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2024.108454.

Objective: To determine if dietary intake and food sources were associated with changes in blood PFAS concentrations among Hispanic young adults at risk of metabolic diseases.

Methods: Predominantly Hispanic young adults from the Children's Health Study who underwent two visits (CHS; n = 123) and young adults from NHANES 2013–2018 who underwent one visit (n = 604) were included. Dietary data at baseline was collected using two 24-hour dietary recalls to measure individual foods and where foods were prepared/consumed (home/restaurant/fast-food). PFAS were measured in blood at both visits in CHS and cross-sectionally in NHANES. In CHS, multiple linear regression assessed associations of baseline diet with longitudinal PFAS; in NHANES, linear regression was used.

Results: In CHS, all PFAS except PFDA decreased across visits (all $p < 0.05$). In CHS, A 1-serving higher tea intake was associated with 24.8 %, 16.17 %, and 12.6 % higher PFHxS, PFHpS, and PFNA at follow-up, respectively (all $p < 0.05$). A 1-serving higher pork intake was associated with 13.4 % higher PFOA at follow-up ($p < 0.05$) Associations were similar in NHANES, including unsweetened tea, hot dogs, and processed meats. For food sources, in CHS each 200-gram increase in home-prepared food was associated with 0.90 % and 1.6 % lower PFOS at baseline and follow-up, respectively, and in NHANES was associated with 0.9 % lower PFDA (all $p < 0.05$).

Conclusion: Results suggest that beverage consumption habits and food preparation are associated with differences in PFAS levels in young adults. This highlights the importance of diet in determining PFAS exposure and the necessity of public monitoring of foods and beverages for PFAS contamination.

Keywords

Per- and polyfluoroalkyl substances; Diet; Food sources; Food packaging

1. Introduction

Per- and polyfluoroalkyl substances (PFAS) are a group of synthetic organofluorine compounds that are widespread in the environment (Linet al., 2020). PFAS are often found in household products such as non-stick cookware, food packaging and water and stain repellents (Linet al., 2020). PFAS are composed of long hydrocarbon chains that are partially or fully fluorinated (Rothet al., 2020). This structure provides stability for PFAS molecules, leading to long half-lives and lengthy degradation processes. Given their abundant use in industrial practices and their high chemical stability, these pollutants persist in the environment for many years leading to an accumulation in humans, animals, fish and shellfish (Linet al., 2020; Rothet al., 2020; Wangetal., 2015). PFAS are detected in more than 98 % of the US population of individuals 12 years of age and older (Calafatet al., 2007). PFAS have similar structures to fatty acids, giving these chemicals the potential to disrupt hormone systems related to energy production, lipid metabolism and inflammation (Decaraet al., 2020; Vanden Heuvelet al., 2006; Wolfet al., 2012). PFAS have been linked to many adverse health effects including increased risk of certain cancers such as liver cancer (Goodrichet al., 2022), high cholesterol (Roth and Petriello, 2022), disrupted glucose homeostasis (Goodrichet al., 2021), Type II Diabetes Mellitus and obesity ((ATSDR) 2022;

Cardenaset al., 2019; Qiet al., 2020), which are some of the leading causes of death in the United States ((CDC) 2023b). Importantly, developing healthy lifestyle behaviors during young adulthood reduces the risk of chronic diseases in later life (Liuet al., 2012). Therefore, identifying modifiable lifestyle factors that impact PFAS exposure in young adults is critical to reducing PFAS levels in the human population and to improving public health.

Diet accounts for a large portion of PFAS exposure in non-occupationally exposed populations(Linet al., 2020; Tittlemieret al., 2007). This may occur through contact with food packaging or non-stick cookware, in foods such as microwave popcorn and snack foods, or through consumption of seafood, meat, and animal products such as eggs and dairy that come from PFAS-exposed animals ((CDC) 2023b; Christensenet al., 2017; Halldorssonet al., 2008; Lasterset al., 2022; Linet al., 2020; Menzelet al., 2021). However, most studies exploring diet as a source of PFAS have examined predominantly self-identified white adults or the general public. Further, many studies examining dietary sources of PFAS do not focus on young adult populations, yet young adults are in a distinct and important behavioral and developmental window (Tohiet al., 2022). While existing research provides dietary guidelines for adult populations of European ancestry or the general public, dietary habits vary across age groups ((USDA) 2023; Larsonet al., 2016), geographic regions (Lackoet al., 2021; Vadivelooet al., 2019), and racial/ethnic groups in the United States (Di Noiaet al., 2016; TaoLiu and Nguyen, 2022; Zimmeret al., 2020). Further, PFAS exposures differ across these factors as well ((EPA) 2023; Parket al., 2019). Importantly, some minority groups experience higher disease burdens, as well as environmental exposures. In particular, Hispanic populations have higher risk of many non-communicable diseases (Fernandez, 2021) and experience higher exposure to environmental toxins including PFAS (LiddieSchaider and Sunderland, 2023). To our knowledge, no studies have examined associations of dietary intake with longitudinal measures of PFAS exposure in predominantly Hispanic young adults.

More targeted research is required to provide guidelines to reduce PFAS exposure for specific groups at high risk of chronic diseases. This study aimed to determine if dietary intake or food source was associated with changes in PFAS concentrations in young adults using a Southern California longitudinal cohort of predominately Hispanic ethnicity. Further, we aimed to provide generalizability of results to other geographic regions using a young adult subset of participants from the National Health and Nutrition Examination Survey (NHANES).

2. Methods

2.1. Study populations

2.1.1. Southern California Children's Health study (CHS)—Data were obtained from a subset of predominantly Hispanic young adults aged 17 to 22 from the Children's Health Study (CHS), described previously (McConnellet al., 2015). Participants were included in the CHS baseline study between 2014 and 2018 if they (a) had overweight or obesity in early adolescence between 2011 and 2012, (b) were not using any medications known to influence body composition or insulin action/secretion, (c) and did not have a

diagnoses of any diseases or illnesses, including type 1 or type 2 diabetes (McConnellet al., 2015). Participants who completed the follow-up visit were included in the longitudinal analyses.

Of the 155 total participants recruited at baseline, 1 had missing dietary data and 31 had missing PFAS data. The total sample used for analysis at baseline had 124 participants with complete diet, PFAS and covariate data (Supplemental Fig. 1A). Eighty-eight participants completed the 4-year follow-up visit and had complete PFAS data at follow-up. Written informed consent was obtained from all participants and the study was approved by the Institutional Review Board at the University of Southern California.

2.1.2. National Health and Nutrition Examination survey (NHANES) 2013–2018—

To test the generalizability of associations between dietary intake and PFAS concentrations in young adults of predominately Hispanic ethnicity from the CHS cohort, data were obtained from young adults from NHANES, which is a nationally representative survey that assesses the health and nutritional status of the US population ((CDC) 2023a). This survey has been conducted continuously since 1999, and data are publicly released in 2-year cycles. For the current study, data were obtained for three two-year cycles from 2013 to 2018 to match the time period of the primary cohort. A key objective of the NHANES cycles during this time period was to increase precision for subgroup estimates (Chen TC;Johnson, 2014). Therefore, Hispanic individuals were oversampled during the 2013–2018 cycles. We selected individuals between 17 and 22 years of age at the time of survey with complete PFAS and covariate data. A total of 604 participants were included from three cycles, 2013–2014, 2015–2016, and 2017–2018 (n = 225, n = 182, n = 197, respectively) (Supplemental Fig. 1B). Data were combined in the analysis across cycles using NHANES survey weights, which take into account sample methods used within each survey cycle ((NCEH) 2023b). The total 604 individuals had complete PFAS data for Perfluorodecanoic acid (PFDA), Perfluorononanoic acid (PFNA) and Perfluorohexanesulphonic acid (PFHxS) (CDC, 2013; CDC, 2015; CDC, 2017). An additional two PFAS, Perfluorooctanesulfonic acid (PFOS) and Perfluorooctanoic acid (PFOA) were later quantified from surplus plasma samples and were available for a total of 577 individuals. Analyses for PFDA, PFNA and PFHxS were performed on the 604 individuals, while analyses for PFOS, PFOA and total summed PFAS were performed on the 577 individuals. Written informed consent was obtained from all participants, and for participants under 18 years of age, consent was given by parents or guardians. The NHANES study protocol was approved by the National Center of Health Statistics Research Ethics Review Board ((NCHS) 2022).

2.2. Dietary assessment

In CHS, dietary data were obtained from two 24-hour dietary recalls on non-consecutive days, one on a weekday and one on a weekend. Recalls were conducted by trained interviewers using the 2014 version of the Nutritional Data System for Research software (NDSR) ((NCC) 2023; Hoffmannet al., 2002). In NHANES, dietary data were also obtained from two 24-hour dietary recalls conducted 3–10 days apart (CDC, 2017; Dietary Interview, 2016; Dietary Interview, 2018; Dietary Interview, 2020).. The U.S. Department of Agriculture's (USDA) Food and Nutrition Database for Dietary Studies (FNDDS) 2013–14

through 2017–18 was used for processing dietary data ((USDA), 2022). For both cohorts, recalls were averaged within each participant to create mean dietary variables for analysis and for participants with only one recall, values from the single recall were used. In CHS, 11 % of participants had only one recall and in NHANES, 18 % had only one recall. The standard, validated 24-hour dietary recall instrument provides an open-ended response structure where participants are asked to recall everything they have consumed in the past 24 h and trained researchers enter this information in the chosen nutrition research software ((NIH) 2014).This provides participants the ability to be specific about their foods consumed including culturally specific foods that may not be included on a close-ended response survey.

In both cohorts, individual foods and beverages were analyzed as amount consumed per day. In CHS, individual foods and beverages were reported by participants and the NDSR software converted participants recall responses into U.S. Food and Drug Administration (FDA) servings of foods and beverages consumed per day. Individual foods and beverages were then examined as continuous exposure variables in FDA servings per day, based on the 2000 Dietary Guidelines for Americans and FDA serving sizes ((USDA) 2000). In NHANES, the FNDDS software produces foods in grams consumed per day. We converted these into 200 g servings per day for appropriate comparability between CHS and NHANES. Individual foods and beverages were thus analyzed as continuous exposure variables in 200 g servings per day. In both cohorts, foods and beverages were further categorized as being prepared at “Home”, at a “Fast-food restaurant” or at a “Non-fast-food restaurant,” and analyzed as 200 g servings consumed per day.

2.3. PFAS assessment

In CHS, plasma PFAS were measured from participant blood samples taken at the time of participants’ first dietary recall visit at baseline and at the follow-up visit (Table 2). PFAS were quantified using Liquid Chromatography-High Resolution Mass Spectrometry (LC-HRMS) and reference standardization (Goet al., 2015; Liuet al., 2020), as described previously (Goodrichet al., 2022). Six PFAS, including Perfluorodecanoic Acid (PFDA), Perfluorohexanesulponic Acid (PFHxS), Perfluorooctanoic Acid (PFOA), Perfluorononanoic Acid (PFNA), Perfluorooctanesulfonic Acid (PFOS) and Perfluoroheptanesulfonic Acid (PFHpS), had greater than 2/3 (67 %) of concentrations above the limits of detection (LOD) and were analyzed as continuous. The LODs were 0.01 ng/dL (PFDA), 0.01 ng/dL (PFHxS), 0.01 ng/dL, 0.01 ng/dL (PFOA), 0.43 ng/dL (PFOS) and 0.05 ng/dL (PFHpS). Values below the LOD were imputed as the LOD/√2. This was only relevant for PFDA, as the other 5 PFAS had 100 % of samples meeting the limit of detection. Three additional PFAS were quantified but were detected in less than 67 % of study samples for at least one study visit, including Perfluoropentane sulfonic acid (PFPeS), Perfluoroheptanoic acid (PFHpA), Perfluorobutane sulfonate (PFBS) (shown in Table 2). For PFPeS, only 19 % of values were below LOD at baseline but more than 95 % were below LOD at follow up. Therefore, PFPeS was analyzed cross sectionally as a continuous variable at baseline only. For PFHpA and PFBS, more than 33 % of values were below LOD at baseline and more than 95 % were below LOD at follow up; therefore, these PFAS at

baseline were dichotomized as "$_{>}$LOD" versus "$_{<}$LOD" and analyzed as binary variables at baseline only.

In NHANES, serum PFAS were measured from participant blood samples at each study visit ((NCEH) 2013; 2016; (NCEH) 2017). Online-solid phase extraction coupled to High Performance Liquid Chromatography-Turbo Ion Spray ionization-tandem Mass Spectrometry (online SPE-HPLC-TIS-MS/MS) was used for quantitative detection of PFAS (KuklenyikNeedham and Calafat, 2005). For NHANES, five of the six PFAS in the primary study were available for the generalizability analysis (PFDA, PFNA, PFHxS, PFOS and PFOA). The LOD for all PFAS was 0.01 ng/dL and PFAS below the LOD were imputed as LOD/√2. All five PFAS in NHANES met quality control standards and were included in analyses. Total summed PFAS were calculated as the sum of all PFAS.

2.4. Covariates

In both cohorts, all participants completed questionnaires on sociodemographic information, including age (in years), sex (male, female), race/ethnicity (Hispanic, Non-Hispanic White, Other), and parental education (missing, less than high school diploma, high school grad, greater than high school diploma). In CHS, less than 2 % of participants, and in NHANES, less than 5 % of participants had missing education values and were included in primary analysis as missing.

2.5. Statistical analysis

In CHS, descriptive statistics were calculated for participant demographics and PFAS concentrations. Differences in PFAS concentrations from baseline to follow-up were tested using paired t-tests. Spearman correlation coefficients were calculated for all dietary variables. To examine associations between baseline diet (individual foods and food sources) and changes in PFAS concentrations for continuous PFAS variables across study visits, linear mixed-effect models with individual-level random intercepts were used (Diggle, 2002). Each PFAS was modeled as a separate outcome and each outcome was modeled as function of each individual food item or food source, baseline age, sex, race/ethnicity, and parental education. For i = $(1, \ldots, N)$ individuals and j = $(1, \ldots, J_i)$ study visits, the following model was used:

$$Y_{ij} = \gamma_0 + \gamma_1 Diet_i + \gamma_2 \text{Visit}_{ij} + \gamma_3 Diet_i \text{Visit}_{ij} + \omega_i + \varepsilon_{ij}$$

where ω_i $N(0, \sigma_{\omega_i})$ is the random effect for each subject and ε_{ij} $N(0, \sigma_\varepsilon)$ are the residuals. Effect estimates for each individual food were determined at both baseline and follow up using the γ_2 parameter from the equation by changing the reference level of time. This model allowed for the inclusion of participants with only baseline or follow-up PFAS measurements, rather than only including participants with outcome measures at both timepoints, which would limit the sample size. Based on a directed acyclic graph, all models were adjusted for baseline age, sex, race/ethnicity, and parental education (as a proxy for socioeconomic status; Supplemental Fig. 2). PFAS concentrations were log-transformed to improve heteroskedasticity and aid in interpretation and comparison across different PFAS. Foods consumed by less than 10 % of participants were removed prior to analysis. PFPeS

was analyzed using the same framework at baseline with multiple linear regression, where PFPeS was modeled as a function of each individual food item or food source, adjusting for age, sex, race/ethnicity and parental education. To examine associations between diet and the additional two dichotomized PFAS, PFHpA and PFBS, the same framework was used at baseline with logistic regression, adjusting for covariates.

To assess generalizability of findings to members of the US population with similar demographics to CHS, we examined cross-sectional associations between diet and PFAS in NHANES. Spearman correlation coefficients were calculated for all dietary variables. To properly account for the complex survey design of NHANES data when pooling data across cycles, cycle-specific sample weights, strata, and primary sampling unit variables were used for NHANES analyses, as described elsewhere ((NCHS) 2023a). After pooling all cycles together, we used weighted linear regression as implemented in the survey package in R to properly analyze these data (Lumley, 2023). For individual foods that are commonly sourced from fast-food and other restaurants, further analyses were performed to determine if source of food may further explain any associations. The following food items were further examined: Pizza, Burritos, Tacos, Fajitas, Burgers, Frankfurters and Hot Dogs, French Fries, Home Fries and Hash Browns. These food items were categorized into the same three source categories as described previously.

For primary analyses of associations between individual foods and PFAS concentrations, associations passed significance if they had a p-value of less than 0.05. Secondarily, we calculated the effective number of tests (M_{eff}) (Wen and Lu, 2011), using principal components analysis (PCA) to adjust for multiple comparisons. We performed PCA on all exposures and all outcomes in both cohorts independently and calculated the eigenvalues for each. We determined the effective number of tests using the Kaiser-Guttman rule (Goretzko and Buhner, 2022), summing all eigenvalues greater than 1 for the exposures, and for the outcomes. Finally, we summed the effective number of tests for the exposures and outcomes, the get the total effective number of tests M_{eff}, in each cohort. The new significance threshold accounting for multiple comparisons is calculated as the p-value divided by M_{eff}. M_{eff} for CHS was 21 and for NHANES was 26. Associations passed the test for multiple comparisons if they had a p-value of less than 0.05/21 =0.0024 for CHS, and 0.05/26 = 0.0019 for NHANES. All analyses were performed using R version 4.3.1 (2023–06–16) ((CRAN) 2021).

2.6. Sensitivity analyses

Sensitivity analyses were performed excluding participants with missing education information in CHS and NHANES. Additional analyses were also performed including energy intake (kilocalories/day) as a covariate.

3. Results

3.1. Study population

In CHS, 123 young adults completed the baseline visit, with a mean age of 19.8 ± 1.2 years. Fifty-eight percent were Hispanic, 54 % were male and a majority had parents with

more than a high school education (66 %) (Table 1). Eighty-eight young adults completed the follow-up visit, with a mean follow-up age of 24.0 ± 0.8 years. In NHANES, 604 young adults were included in analyses, with a mean age of 19.1 ± 1.7 years. Thirty-one percent were Hispanic, 53 % were male and a majority had parents with less than a high school education (40 %) (Table 1). Geometric means of PFAS concentrations for CHS and NHANES are shown in Table 2. All PFAS except for PFDA significantly decreased from baseline to follow-up in CHS, while PFDA significantly increased (all $p < 0.05$; Table 2). All PFAS concentrations were comparable between the CHS baseline assessment and NHANES (Table 2).

3.2. Correlations of individual foods

In both cohorts, dietary intake of several foods and beverages were correlated, providing insights into how individual consumption patterns may influence overall exposure of PFAS. The strongest associations were observed with beverage consumption. In CHS, there were inverse correlations between soft drinks and tea (Spearman correlation coefficient, $r = -0.22$, $p = 0.015$) and soft drinks and tap water (Spearman $r = -0.24$, $p = 0.007$; Supplemental Fig. 3A and B). In NHANES, there were inverse correlations between soft drinks and unsweetened tea (Spearman $r = -0.09$, $p = 0.025$), soft drinks and bottled water (Spearman $r = -0.12$, $p = 0.003$) and soft drinks and tap water (Spearman $r = -0.18$, $p < 0.0001$; Supplemental Fig. 1C and D). We also saw inverse correlations between tea and bottled water (Spearman $r = -0.19$, $p = 0.038$) in CHS and unsweetened tea and bottled water (Spearman $r = -0.10$, $p = 0.017$) in NHANES. In both cohorts, tap water and bottled water were inversely correlated (CHS: Spearman $r = -0.29$, $p = 0.001$; NHANES: Spearman $r = -0.38$, $p < 0.0001$ in NHANES; Supplemental Fig. 3).

3.3. Individual foods & PFAS

In CHS, most associations between individual foods and PFAS at baseline were null; however, there were significant associations at follow up. From the linear mixed effects model, the only significant associations at baseline were with sweet condiments and tap water. A 1-serving higher intake of sweet condiments was associated with a 19.8 % [2.8 %, 40.0 %]) higher PFDA concentration at baseline (Fig. 1A, Supplemental Table 1A). This effect was null at follow-up. Additionally, a 1-serving higher intake of tap water was associated with a 2.79 % [0.97 %, 4.58 %] lower PFOS, 3.18 % [0.048 %, 6.212 %] lower PFHxS and 2.42 % [0.62 %,4.18 %] lower PFHpS concentration at baseline (Fig. 1A, Supplemental Table 1A). From the baseline linear regression model, a 1-serving higher intake of nut and seed butters was associated with a 35.59 % [1.10 %, 81.84 %] higher PFPeS concentration (Supplemental Fig. 4, Supplemental Table 5). From the baseline logistic regression model, a 1-serving higher intake of bread was associated with 1.41 [1.04, 1.89] higher odds of detecting PFHpA. A 1-serving higher intake of soup broths was associated with a 7.30 [1.26, 42.42] higher odds of detecting PFHpA and 5.67 [1.24, 25.84] higher odds of detecting PFBS (Supplemental Fig. 4, Supplemental Table 5). A 1-serving higher intake of pickled foods was associated with a 0.10 [0.01, 0.92] lower odds of detecting PFHpA.

Higher intakes of tea (combined sweetened and unsweetened), pork, sports drinks, nut and seed butters, snack chips and bottled water and lower intakes of whole fruit, fruit juice, pasta, fried potatoes, soft drinks, cooked grains, nuts and seeds, sugar and tap water at baseline were associated with higher concentrations of PFAS at follow up from the linear mixed-effects model (Fig. 1A, Supplemental Table 1B). The strongest positive associations were observed with tea and pork intake. A 1-serving higher intake of tea was associated with 24.8 % [4.0 %, 50.0 %] higher PFHxS, 16.17 % [3.67 %, 30.19 %] higher PFHpS, and 12.6 % [0.5 %, 26.1 %] higher PFNA. A 1-serving higher intake of pork was associated with 13.4 % [3.5 %, 24.2 %] higher PFOA. The strongest negative associations were observed with sugar. A 1-serving higher sugar intake was associated with 18.9 % [12.6 %, 24.7 %] lower PFNA, and 13.9 % [2.4 %, 24.0 %] lower PFHxS.

We observed similar associations in NHANES (Fig. 1B, Supplemental Table 1D). Higher intake of hot dogs (combination of meat types) was associated with higher PFNA, and higher intake of processed meat was associated with higher PFOA. A 1-serving higher intake of hot dogs was associated with a 25.4 % [1.3 %, 55.3 %] higher PFNA concentration and a 1-serving higher intake of processed meat was associated with a 9.8 % [0.5 %, 20.1 %] higher PFOA concentration. Higher intake of unsweetened tea was associated with a higher PFOS concentration, where a 1-serving higher intake of unsweetened tea was associated with a 4.12 % [0.6 %, 7.8 %] higher PFOS concentration. Tap water and bottled water were not significantly associated with PFAS concentrations in NHANES.

3.4. Food sources & PFAS

In CHS, higher intake of food prepared at home was associated with lower PFOS concentrations at baseline and follow-up. A 200 g-serving higher intake of foods prepared at home was associated with a 0.9 % [0.001 %, 1.8 %] lower PFOS at baseline and 1.6 % [0.6 %, 2.5 %] lower PFOS at follow-up (Fig. 2; Supplemental Table 2; Supplemental Table 6).

In NHANES, higher intakes of food prepared at home was also associated with lower PFDA concentrations, where a 200 g-serving higher intake of foods prepared at home was associated with a 0.9 % [0.1 %, 1.7 %] lower PFDA concentration (Supplemental Table 2). In NHANES, burritos, fajitas, tacos, French fries, burgers, and pizza were inversely associated with PFAS (Fig. 1B; Supplemental Table 1D). After further categorizing of these foods based on their sources, burritos, fajitas, tacos, French fries and pizza were inversely associated with PFAS concentrations when prepared at home, while they were positively associated with PFAS when prepared at restaurants and fast-food restaurants (Supplemental Table 3). The strongest inverse association was seen between home-prepared burgers and PFDA, where a 200 g-serving increased consumption of burgers prepared at home was associated with a 22.2 % [2.9 %, 37.7 %] lower PFDA. The strongest positive associations were seen between fast-food prepared hot dogs and PFNA and PFHxS. A 200 g-serving increased consumption of hot dogs prepared at fast-food restaurants was associated with a 36.9 % [8.1 %, 73.5 %] higher PFNA and 63.4 % [3.9 %, 157.1 %] higher PFHxS (Supplemental Table 3).

3.5. Sensitivity analyses

In sensitivity analyses excluding participants with missing parental education information, results were similar. Additionally, in analyses including energy as a covariate, results were unchanged in NHANES and very similar in CHS (Supplemental Table 4).

3.6. Discussion

To our knowledge, this is the first study examining associations of dietary intake with longitudinal PFAS concentrations. This study provides novel findings on dietary PFAS exposures in predominantly Hispanic young adults from longitudinal analyses using a replication cohort to strengthen the generalizability of findings. In independent cohorts, we found that higher intakes of tea and several meats were associated with higher circulating blood PFAS concentrations, while higher intake of food prepared at home was associated with lower circulation blood PFAS concentrations. These findings suggest that both specific foods and dietary sources can impact PFAS exposure in young US populations. Together, these findings may provide a basis for designing dietary interventions to decrease PFAS exposure in Hispanic young adults.

Our study fills an important research gap as one of the first longitudinal studies looking at dietary sources of PFAS in predominantly Hispanic young adults in two independent cohorts across a variety of geographic regions. Previous studies examining how diet impacts PFAS exposure have all been cross-sectional (Christensenet al., 2017; Halldorssonet al., 2008; Lasterset al., 2022; Linet al., 2020; Menzelet al., 2021; Tittlemieret al., 2007). However, the major limitation of cross-sectional studies is an inability to rule out reverse causation. Our longitudinal study reduces this potential bias.

In CHS, we found that PFAS concentrations decreased significantly for five of the six PFAS detected in our sample. These changes are similar to those reported in NHANES over a similar time period with PFOS, PFOA, and PFHxS ((CDC) 2015). Our study took place between 2014 and 2022 in Southern California. This coincides with a period of increased drinking water testing that occurred in the region, increasing public awareness of the issue. For example, in 2012, the U.S. Environmental Protection Agency (EPA) implemented the *Third Unregulated Contaminant Monitoring Rule (*(EPA) 2022), which required monitoring of 30 chemicals including PFOA and PFOS. In 2019, California implemented additional drinking water testing in Los Angeles County, which prompted additional action from local water utilities to reduce high levels of PFAS exposure (Board, 2023). While drinking water PFAS contamination is still of concern in Southern California, participially in areas with higher Hispanic populations (Smallinget al., 2023), the increased testing and awareness may explain the decreases in PFAS levels seen in our study. Additionally, despite the decreases observed over time, our results still found that food and beverage consumption contributed to higher PFAS levels, suggesting that additional public testing is needed.

In our longitudinal study, we found that higher intakes of pork, hot dogs, beef and other processed meats were associated with higher PFAS concentrations in CHS and NHANES. A previous cross-sectional study also using NHANES data for U.S. adults of all race/ethnicities similarly found that higher intakes of meat were associated with higher PFAS

concentrations (Linet al., 2020). It is suggested that PFAS may accumulate in meat products through several exposure sources. Processed meats such as sausages, bacon and hot dogs may accumulate PFAS through contact during processing or cooking ((FDA) 2023; Ramirez Carneroet al., 2021). Other forms of meat, such as unprocessed pork and beef, may come from animals that were raised in PFAS-contaminated areas or were packaged in grease-resistance packaging that contains PFAS (Tittlemieret al., 2007). Further studies are needed to monitor specific foods that may be contributing to PFAS exposure, and the results of this study may be used to inform such future work.

We also found that higher intake of tea was associated with higher PFAS in CHS and NHANES. Previous cross-sectional studies have found similar associations among adults of predominantly European ancestry (Linet al., 2020). PFAS in tea may come from the primary tea components, including the water, tea leaves or tea bags, or from additives, including milk, creamer, or sugar. PFAS exposure from tea could also come from the brewing or preparation processes or through the cups or bottles used to contain pre-made teas. However, both milk, sugar and domestic tap water were inversely associated with PFAS in our study. These results suggest that the PFAS exposures may not be coming from the water used in brewing or the sweeteners or additives. Further, paper products are a major contamination source for PFAS (Glugeet al., 2020) and tea bags are primarily made of paper (JhaDhekne and Patwardhan, 2020). Therefore, it is plausible that PFAS in tea bags may be contributing to the associations seen with tea intake. Previous studies have also seen that tea bags may be an exposure source for a variety of microplastics and nano plastics due to the brewing process with water at high temperatures (Hernandezet al., 2019), suggesting this could also be the case for PFAS contamination in tea. Additionally, we found that bottled water, sports drinks, and coffee were positively associated with PFAS. These results suggest the necessity of public monitoring of beverages as these may be large contributors of PFAS exposures. Results of this study may be used to inform future studies that test and monitor beverages for PFAS contamination.

One unexpected finding in our study was that sugar was inversely associated with PFAS in CHS. This is in contrast with our findings in NHANES which showed a null association between sugar and PFAS levels. It also contrasts with previous literature which has found positive associations with sweets and PFAS (Linet al., 2020). However, we also observed that soft drinks and fruit drinks/fruit juices were associated with greater decreases in PFAS across visits in CHS. Given that sugar sweetened beverages, including soft drinks and fruit juices are the greatest source of sugars in the young adult diet in the United States ((USDA) 2020), one explanation for the strong association seen with sugar is that it is driven by differences in drinking habits. For example, since PFAS-contaminated drinking water is a major source of PFAS exposure in the U.S.(LiddieSchaider and Sunderland, 2023), it is plausible that increased soft drink and fruit juice consumption may reduce PFAS exposure by reducing intake of potentially PFAS-contaminated water. Overall, in our study, we saw many inverse correlations between beverages consumed both in CHS and in NHANES. For example, we saw a significant inverse correlation between soft drinks and tea in CHS and soft drinks and unsweetened tea in NHANES. We also saw significant inverse correlations between tea and tap water in CHS and unsweetened tea and bottled water in NHANES.

These correlations highlight that drinking habits play an important role in the associations seen between PFAS concentrations and certain beverages.

Alternatively, desserts and sweet snacks are the second largest source of sugars among young adults in the United States ((USDA) 2020), and in NHANES, higher intake of cakes and cupcakes were positively associated with PFAS concentrations. Dessert wrappers have been shown to be highly fluorinated however, therefore it is plausible that greater PFAS exposures through desserts is driven by PFAS-contaminated packaging materials (Schaideret al., 2017). Interestingly, we also saw in CHS, that nuts and seeds were associated with lower concentrations of PFOS and PFDA, which is consistent with previous findings that nuts and seeds are inversely associated with several PFAS (Linet al., 2020); However, nut and seed butters were associated with higher PFHpS and PFPeS concentrations. Given that nut and seed butters are packed in grease-resistant containers, it is possible that nut and seed butters may contribute to greater PFAS exposures through the packaging materials rather than the nuts and seeds themselves.

We found that whole fruits, cooked grains such as rice and oatmeal, breads, pastas and some vegetables were inversely associated with PFAS concentrations in CHS and NHANES. Importantly, fruits, vegetables and grains are the major contributors of fiber in the American diet (OASH, 2015), and a previous large scale NHANES study showed that dietary fiber intake has the potential to reduce PFAS concentrations (DzierlengaKeast and Longnecker 2021). Thus, increased consumption of high-fiber foods may contribute to reduced PFAS concentrations. Overall, our findings are generally consistent with previous cross-sectional studies including populations of different ages and racial/ethnic distributions, as well as different geographic areas, providing strong evidence that these foods are associated with PFAS concentrations and require further exploration into their exposure pathways to eliminate PFAS exposures in the population.

In CHS, we saw the strongest associations between diet PFAS concentrations follow up, rather than baseline. This could be due to the fact that several PFAS have long half-lives, and it may take substantial time to change in response to changes in PFAS exposure through diet or water. Additionally, our study sample was made up of individuals 17 to 22 at baseline, a period of transition from adolescent dietary patterns to young adult dietary patterns (Lipskyet al., 2017). With the contribution of new foods to the diet and the removal of other foods from the diet, it may have taken time for PFAS concentrations to change and for that change to be reflected in blood PFAS concentrations. This highlights the need for further studies that monitor changes in PFAS concentrations over longer periods of time, as well as the importance of young adulthood in determining PFAS exposures.

While PFAS exposure can come from the consumption of contaminated foods, food packaging containing PFAS may be an important exposure source as well. Previous studies have shown that PFAS are found in dessert and bread packaging, fast-food sandwich and burger wrappers and paperboards such as pizza boxes (Schaideret al., 2017). Given that packaging materials contaminated with PFAS are frequently used at fast-food restaurants or for restaurant take-out, we examined where foods were prepared (fast-food, restaurant or home) as a proxy for food packaging exposure. We found that higher consumption of food

prepared at home was associated with lower PFAS concentrations in CHS and NHANES. A previous cross-sectional study looking at the general population of U.S. adults using NHANES data similarly found that foods prepared at home were associated with lower PFAS concentrations (Susmannet al., 2019). We expanded on this analysis in NHANES examining association of PFAS with common fast-food and restaurant-sourced food items, including burgers, French fries, hot dogs, burritos, tacos, fajitas, and pizza. In our primary analyses, many of these items were inversely associated with PFAS when consumed from all sources. Further analyses showed that when these foods were prepared from home, they were inversely associated with PFAS, while restaurant and fast-food versions of these foods were positively associated with PFAS. Additionally, trends in our analyses suggest that PFAS exposures were predominantly from fast-food in CHS, with lower PFAS concentrations associated with restaurant and home-prepared food. These results suggest that fast-food may provide higher PFAS exposures, which could be from grease-resistant food packaging containing PFAS (Susmannet al., 2019) or from other food process steps used in preparing fast food. Results for non-fast-food restaurants were less consistent across CHS and NHANES, suggesting that restaurant sourced food may not experience the same PFAS exposures as fast-food. Home-sourced foods were consistently associated with lower PFAS concentrations across CHS and NHANES, suggesting that home cooking may help young adults reduce their exposures to PFAS. However, further studies are needed to monitor specific food packaging materials for PFAS contamination.

Our study has several strengths. Our study found longitudinal associations between dietary intake and PFAS concentrations, with effects increasing in magnitude over time and providing temporal evidence for a relationship between PFAS exposure and diet. We used a second cohort to provide external validity and generalizability of these results to young adults of a variety of race/ethnicities across the United States. Dietary habits differ across populations and geographic regions, and our study provides novel findings that certain beverages and food preparation habits are associated with PFAS exposures among predominantly Hispanic young adults. These findings may inform future work that directly assesses PFAS contamination within common foods consumed by Hispanic young adults. Our results provide a bases for more targeted future studies that assess the PFAS concentrations in popular beverages and foods consumed by this population.

Our study has a few limitations. Our primary cohort had a sample size of 123 participants, which may have resulted in lower power to detect associations. To address this limitation, we included a generalizability cohort with over 600 participants, providing stronger evidence for our main findings. Additionally, in CHS, we found that higher intake of tea was significantly associated with higher PFAS concentrations. However, we were unable to determine which types of tea were driving this relationship (sweetened vs. unsweetened vs. artificially sweetened), due to limitations of the research tool used to assess tea in this cohort. However, in our generalizability cohort, we were able to analyze sweetened and unsweetened tea separately, finding that unsweetened tea was significantly positively associated with PFAS concentrations. Therefore, this provides evidence that the tea itself, rather than any sweeteners that may be added, is driving the positive associations and may be an important exposure pathway. Additionally, in CHS, detailed information on the consumption of specific individual foods was only available at baseline, limiting the ability

to examining associations of PFAS with diet at follow up. At follow up, diet was assessed using the ASA24 ((NIH) 2023), which provides information on broad food categories and individual nutrients, but unlike the NDSR which was used at baseline, does not provide information on individual foods. However, PFAS have long half-lives and decreases in blood PFAS concentrations, which were observed in our study can take years. Therefore, assessing dietary patterns at baseline can provide a good estimate of the effects of diet on changes in blood PFAS. Finally, we were not able to directly measure PFAS contamination in foods or beverages consumed by the study participants, limiting the ability to draw broad conclusions about PFAS contamination in individual foods or beverages. Despite this limitation, our compelling results, which were replicated in two independent cohorts, highlight the importance of additional public monitoring of specific foods and beverages to decrease PFAS exposures in the general population.

4. Conclusion

Our results suggest that both beverage and food intake as well as sources of foods are significantly associated with longitudinal measures of PFAS concentrations in predominantly Hispanic young adults. Our results highlight the need for public monitoring of beverages, processed meats, and food packaging containers, in additional to other well-known sources of PFAS. These findings highlight the importance of specific dietary habits in influencing PFAS exposure in predominantly Hispanic young adults.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The funding for the CHS study came from the NIEHS (R01ES029944), the Southern California Children's Environmental Health Center grants funded by NIEHS (P01ES022845, 5P30ES007048, P01ES011627), the United States Environmental Protection Agency (RD83544101), and the Hastings Foundation. Additional funding from NIH supported JAG (R25GM143298, P30ES007048), LC (R01ES030691, R01ES030364, R21ES029681, R21ES028903, and P30ES007048), ZC (R00ES027870), DV (R01ES033688, P30ES023515), DW (U2CES030859, R01ES032831), DC (P01CA196569, P30ES007048, R01ES030691, R01ES030364, R21ES029681, R21ES028903), TLA (R00ES027853, P50MD017344), MIG (P50MD017344), and DPJ (U2CES030163, P30ES019776, R24ES029490, R01ES032189, R21ES031824), BOB (T32ES013678), EC (T32ES013678), and HEH (T32ES013678).

Data availability

The data that has been used is confidential.

References

(ATSDR), A.f.T.S.a.D.R. What are the health effects of PFAS? ; 2022.

Board SWR.C. Per- and Polyfluoroalkyl Substances. 2023.

Calafat AM, Kuklenyik Z, Reidy JA, Caudill SP, Tully JS, Needham LL, 2007. Serum concentrations of 11 polyfluoroalkyl compounds in the u.s. population: data from the national health and nutrition examination survey (NHANES). Environ. Sci. Tech. 41, 2237–2242.

Cardenas A, Hivert MF, Gold DR, Hauser R, Kleinman KP, Lin PD, Fleisch AF, Calafat AM, Ye X, Webster TF, Horton ES, Oken E, 2019. Associations of Perfluoroalkyl and Polyfluoroalkyl

Substances With Incident Diabetes and Microvascular Disease. Diabetes Care 42, 1824–1832. [PubMed: 31296647]

(CDC), N.H.a.N.E.S. 2013–2014 Data Documentation, Codebook, and Frequencies.

(CDC), N.H.a.N.E.S. 2013–2014 Data Documentation, Codebook, and Frequencies. 2022.

(CDC), N.H.a.N.E.S. 2015–2016 Data Documentation, Codebook, and Frequencies.

(CDC), C.f.D.C.a.P. Fourth National Report on Human Exposure to Environmental Chemicals. 2015.

(CDC), N.H.a.N.E.S. 2017–2018 Data Documentation, Codebook, and Frequencies.

(CDC), C.f.D.C.a.P. Leading Causes of Death. 2023b.

(CDC), C.f.D.C.a.P. About the National Health and Nutrition Examination Survey. 2023a.

Chen TC CJ, Riddles MK, Mohadjer LK, Fakhouri THI. National Health and Nutrition Examination Survey, 2015–2018: Sample design and estimation procedures. Vital Health Stat 2:184.

Christensen KY, Raymond M, Blackowicz M, Liu Y, Thompson BA, Anderson HA, Turyk M, 2017. Perfluoroalkyl substances and fish consumption. Environ. Res. 154, 145–151. [PubMed: 28073048]

cran, r.c.t. r, A language and environment for statistical computing 2021.

Decara J, Rivera P, Lopez-Gambero AJ, Serrano A, Pavon FJ, Baixeras E, Rodriguez de Fonseca F, Suarez J, 2020. Peroxisome proliferator-activated receptors: experimental targeting for the treatment of inflammatory bowel diseases. Front. Pharmacol. 11, 730. [PubMed: 32536865]

Di Noia J, Monica D, Cullen KW, Pérez-Escamilla R, Gray HL, Sikorskii A, 2016. Differences in fruit and vegetable intake by race/ethnicity and by hispanic origin and nativity among women in the special supplemental nutrition program for women, infants, and children, 2015. Prev. Chronic Dis. 13, E115. [PubMed: 27560723]

Dietary Interview - Individual Foods, First Day (DR1IFF_H). 2016.

Dietary Interview - Individual Foods, First Day (DR1IFF_I). 2018.

First Day (DR1IFF_J), 2020.

Diggle P, Heagerty P, Liang K.-Y.Zeger, 2002. Scott Analysis of Longitudinal Data edêds. Oxford University Press.

Dzierlenga MW, Keast DR, Longnecker MP, 2021. The concentration of several perfluoroalkyl acids in serum appears to be reduced by dietary fiber. Environ. Int. 146, 106292. [PubMed: 33395939]

(EPA), U.S.E.P.A. Third Unregulated Contaminant Monitoring Rule. 2022.

(EPA), E.P.A. Our Current Understanding of the Human Health and Environmental Risks of PFAS. 2023.

(FDA), U.S.F.D.A. Analytical Results of Testing Food for PFAS from Environmental Contamination. 2023.

Fernandez ML, 2021. Lifestyle factors and genetic variants associated to health disparities in the hispanic population. Nutrients 13.

Gluge 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 Process Impacts 22, 2345–2373. [PubMed: 33125022]

Go YM, Walker DI, Liang Y, Uppal K, Soltow QA, Tran V, Strobel F, Quyyumi AA, Ziegler TR, Pennell KD, Miller GW, Jones DP, 2015. Reference standardization for mass spectrometry and high-resolution metabolomics applications to exposome research. Toxicol. Sci. 148, 531–543. [PubMed: 26358001]

Goodrich JA, Alderete TL, Baumert BO, Berhane K, Chen Z, Gilliland FD, Goran MI, Hu X, Jones DP, Margetaki K, Rock S, Stratakis N, Valvi D, Walker DI, Conti DV, Chatzi L, 2021. Exposure to perfluoroalkyl substances and glucose homeostasis in youth. Environ. Health Perspect. 129, 97002. [PubMed: 34468161]

Goodrich JA, Walker D, Lin X, Wang H, Lim T, McConnell R, Conti DV, Chatzi L, Setiawan VW, 2022. Exposure to perfluoroalkyl substances and risk of hepatocellular carcinoma in a multiethnic cohort. JHEP Rep 4, 100550. [PubMed: 36111068]

Goretzko D, Buhner M, 2022. Factor retention using machine learning with ordinal data. Appl. Psychol. Meas. 46, 406–421. [PubMed: 35812814]

Halldorsson TI, Fei C, Olsen J, Lipworth L, McLaughlin JK, Olsen SF, 2008. Dietary predictors of perfluorinated chemicals: a study from the Danish National Birth Cohort. Environ. Sci. Tech. 42, 8971–8977.

Hernandez LM, Xu EG, Larsson HCE, Tahara R, Maisuria VB, Tufenkji N, 2019. Plastic teabags release billions of microparticles and nanoparticles into tea. Environ. Sci. Tech. 53, 12300–12310.

Hoffmann K, Boeing H, Dufour A, Volatier JL, Telman J, Virtanen M, Becker W, De Henauw S, 2002. Estimating the distribution of usual dietary intake by short-term measurements. Eur. J. Clin. Nutr. 56 (Suppl 2), S53–S62. [PubMed: 12082518]

Jha DK, Dhekne PP, Patwardhan AW, 2020. Characterization and evaluation of tea bag papers. J. Food Sci. Technol. 57, 3060–3070. [PubMed: 32624608]

Johnson CLD, Sylvia M;Burt Vicki.;Mohadjer Leyla K.; National Center for Health Statistics (U.S.);National Health and Nutrition Examination Survey (U.S.). National Health and Nutrition Examination Survey : sample design, 2011–2014. Vital and health statistics Series 2, Data evaluation and methods research 2014.

Kuklenyik Z, Needham LL, Calafat AM, 2005. Measurement of 18 perfluorinated organic acids and amides in human serum using on-line solid-phase extraction. Anal. Chem. 77, 6085–6091. [PubMed: 16159145]

Lacko A, Delamater P, Gordon-Larsen P, Wen Ng S, 2021. Geographic patterns and socioeconomic differences in the nutritional quality of household packaged food purchases in the United States. Health Place 69, 102567. [PubMed: 33930729]

Larson NI, Miller JM, Watts AW, Story MT, Neumark-Sztainer DR, 2016. Adolescent snacking behaviors are associated with dietary intake and weight status. J. Nutr. 146, 1348–1355. [PubMed: 27281807]

Lasters R, Groffen T, Eens M, Coertjens D, Gebbink WA, Hofman J, Bervoets L, 2022. Home-produced eggs: an important human exposure pathway of perfluoroalkylated substances (PFAS). Chemosphere 308, 136283. [PubMed: 36075366]

Liddie JM, Schaider LA, Sunderland EM, 2023. Sociodemographic factors are associated with the abundance of pfas sources and detection in u.s. community water systems. Environ. Sci. Tech. 57, 7902–7912.

Lin PD, Cardenas A, Hauser R, Gold DR, Kleinman KP, Hivert MF, Fleisch AF, Calafat AM, Sanchez-Guerra M, Osorio-Yanez C, Webster TF, Horton ES, Oken E, 2020. Dietary characteristics associated with plasma concentrations of per- and polyfluoroalkyl substances among adults with pre-diabetes: cross-sectional results from the Diabetes Prevention Program Trial. Environ. Int. 137, 105217. [PubMed: 32086073]

Lipsky LM, Nansel TR, Haynie DL, Liu D, Li K, Pratt CA, Iannotti RJ, Dempster KW, Simons-Morton B, 2017. Diet quality of US adolescents during the transition to adulthood: changes and predictors. Am. J. Clin. Nutr. 105, 1424–1432. [PubMed: 28446498]

Liu K, Daviglus ML, Loria CM, Colangelo LA, Spring B, Moller AC, Lloyd-Jones DM, 2012. Healthy lifestyle through young adulthood and the presence of low cardiovascular disease risk profile in middle age: the Coronary Artery Risk Development in (Young) Adults (CARDIA) study. Circulation 125, 996–1004. [PubMed: 22291127]

Liu S, Yang R, Yin N, Faiola F, 2020. Effects of per- and poly-fluorinated alkyl substances on pancreatic and endocrine differentiation of human pluripotent stem cells. Chemosphere 254, 126709. [PubMed: 32348926]

Lumley T. Package ‘survey’. 2023.

McConnell R, Shen E, Gilliland FD, Jerrett M, Wolch J, Chang CC, Lurmann F, Berhane K, 2015. A longitudinal cohort study of body mass index and childhood exposure to secondhand tobacco smoke and air pollution: the Southern California Children’s Health Study. Environ. Health Perspect. 123, 360–366. [PubMed: 25389275]

Menzel J, Abraham K, Dietrich S, Fromme H, Völkel W, Schwerdtle T, Weikert C, 2021. Internal exposure to perfluoroalkyl substances (PFAS) in vegans and omnivores. Int. J. Hyg. Environ. Health 237, 113808. [PubMed: 34298317]

(NCC), N.C.C. NDSR User Manual. 2023.

(NCEH), N.C.f.E.H. Laboratory Procedure Manual. 2013.

(NCEH), N.C.f.E.H. Laboratory Procedure Manual. 2016.

(NCEH), N.C.f.E.H. Laboratory Procedure Manual. 2017.

(NCHS), N.C.f.H.S. NCHS Ethics Review Board (ERB) Approval. 2022.

(NCHS), N.C.f.H.S. NHANES Survey Methods and Analytic Guidelines. 2023a.

(NCHS), N.C.f.H.S. Weighting. 2023b.

(NIH), N.C.I. 24-hour Dietary Recall (24HR) At a Glance. 2014.

(NIH), N.C.I. Automated Self-Administered 24-Hour (ASA24®) Dietary Assessment Tool. 2023.

(OASH), O.o.D.P.a.H.P. Scientific Report of the 2015 Dietary Guidelines Advisory Committee; Appendix E-3.2: Food Group Contributions to Nutrients in USDA Food Patterns and Current Nutrient Intakes. 2015.

Park SK, Peng Q, Ding N, Mukherjee B, Harlow SD, 2019. Determinants of per- and polyfluoroalkyl substances (PFAS) in midlife women: evidence of racial/ethnic and geographic differences in PFAS exposure. Environ. Res. 175, 186–199. [PubMed: 31129528]

Qi W, Clark JM, Timme-Laragy AR, Park Y, 2020. Per- and polyfluoroalkyl substances and obesity, type 2 diabetes and non-alcoholic fatty liver disease: a review of epidemiologic findings. Toxicol. Environ. Chem. 102, 1–36. [PubMed: 33304027]

Ramirez Carnero A; Lestido-Cardama A; Vazquez Loureiro P; Barbosa-Pereira L; Rodriguez Bernaldo de Quiros A.; Sendon R. Presence of Perfluoroalkyl and Polyfluoroalkyl Substances (PFAS) in Food Contact Materials (FCM) and Its Migration to Food. Foods 2021;10.

Roth K, Imran Z, Liu W, Petriello MC, 2020. Diet as an exposure source and mediator of per- and polyfluoroalkyl substance (PFAS) toxicity. Front Toxicol 2, 601149. [PubMed: 35296120]

Roth K, Petriello MC, 2022. Exposure to per- and polyfluoroalkyl substances (PFAS) and type 2 diabetes risk. Front Endocrinol (lausanne) 13, 965384. [PubMed: 35992116]

Schaider LA, Balan SA, Blum A, Andrews DQ, Strynar MJ, Dickinson ME, Lunderberg DM, Lang JR, Peaslee GF, 2017. Fluorinated compounds in U.S. Fast Food Packaging. Environ Sci Technol Lett 4, 105–111. [PubMed: 30148183]

Smalling KL, Romanok KM, Bradley PM, Morriss MC, Gray JL, Kanagy LK, Gordon SE, Williams BM, Breitmeyer SE, Jones DK, DeCicco LA, Eagles-Smith CA, Wagner T, 2023. Per- and polyfluoroalkyl substances (PFAS) in United States tapwater: comparison of underserved private-well and public-supply exposures and associated health implications. Environ. Int. 178, 108033. [PubMed: 37356308]

Susmann HP, Schaider LA, Rodgers KM, Rudel RA, 2019. Dietary habits related to food packaging and population exposure to PFASs. Environ. Health Perspect. 127, 107003. [PubMed: 31596611]

Tao MH, Liu JL, Nguyen UDT, 2022. Trends in diet quality by race/ethnicity among adults in the united states for 2011–2018. Nutrients 14.

Tittlemier SA, Pepper K, Seymour C, Moisey J, Bronson R, Cao XL, Dabeka RW, 2007. Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. J. Agric. Food Chem. 55, 3203–3210. [PubMed: 17381114]

Tohi M, Bay JL, Tu'akoi S, Vickers MH, 2022. The developmental origins of health and disease: adolescence as a critical lifecourse period to break the transgenerational cycle of ncds-a narrative review. Int. J. Environ. Res. Public Health 19.

(USDA), U.S.D.o.A. 2000 Dietary Guidelines for Americans. 2000.

(USDA), U.S.D.o.A. Dietary Guidelines for Americans, 2020–2025. 2020.

(usda), U.S.D.o.A. Food and Nutrient Database for Dietary Studies (FNDDS) 2022.

(USDA), F.a.N.S. HEI Scores for Americans. 2023.

Vadiveloo M, Perraud E, Parker HW, Juul F, Parekh N, 2019. Geographic differences in the dietary quality of food purchases among participants in the nationally representative food acquisition and purchase survey (FoodAPS). Nutrients 11.

Vanden Heuvel JP, Thompson JT, Frame SR, Gillies PJ, 2006. Differential activation of nuclear receptors by perfluorinated fatty acid analogs and natural fatty acids: a comparison of human, mouse, and rat peroxisome proliferator-activated receptor-alpha, -beta, and -gamma, liver X receptor-beta, and retinoid X receptor-alpha. Toxicol. Sci. 92, 476–489. [PubMed: 16731579]

Wang Z, Cousins IT, Scheringer M, Hungerbuehler K, 2015. Hazard assessment of fluorinated alternatives to long-chain perfluoroalkyl acids (PFAAs) and their precursors: status quo, ongoing challenges and possible solutions. Environ. Int. 75, 172–179. [PubMed: 25461427]

Wen SH, Lu ZS, 2011. Factors affecting the effective number of tests in genetic association studies: a comparative study of three PCA-based methods. J. Hum. Genet. 56, 428–435. [PubMed: 21451529]

Wolf CJ, Schmid JE, Lau C, Abbott BD, 2012. Activation of mouse and human peroxisome proliferator-activated receptor-alpha (PPARα) by perfluoroalkyl acids (PFAAs): further investigation of C4–C12 compounds. Reprod. Toxicol. 33, 546–551. [PubMed: 22107727]

Zimmer MC, Rubio V, Kintziger KW, Barroso C, 2020. Differences in consumption of nasem priority nutrients and food groups by race/ethnicity among women living in wic-participating households. Am. J. Health Promot. 34, 791–795. [PubMed: 32363883]

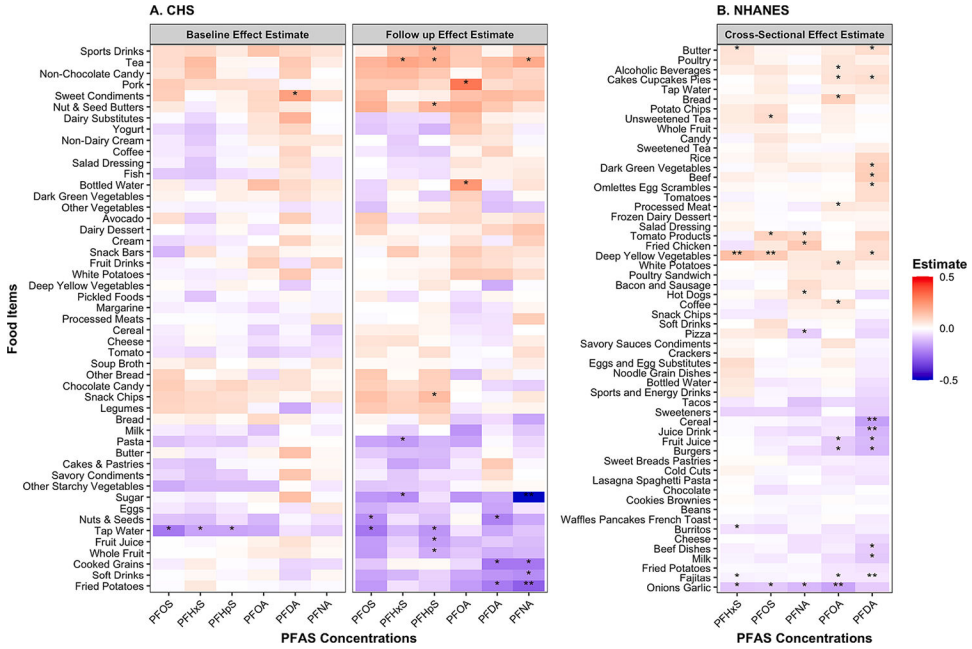

Fig. 1.

Feature expression heatmap of associations between 46 food items in CHS (N = 124 at baseline, N = 88 at follow-up) (A) and 54 food items in NHANES (n = 604) (B) with PFAS concentrations, adjusting for age, sex, race/ethnicity, and parental education. For NHANES, analyses were further adjusted for NHANES cycle. All variables are scaled and PFAS are additionally log transformed. Estimates interpreted as 1 standard deviation increase in intake of food item was associated with a 1 standard deviation increase in log PFAS concentrations for baseline, follow up and cross-sectional effect estimates.. * $p < 0.05$, ** adjusted $p < 0.05$.

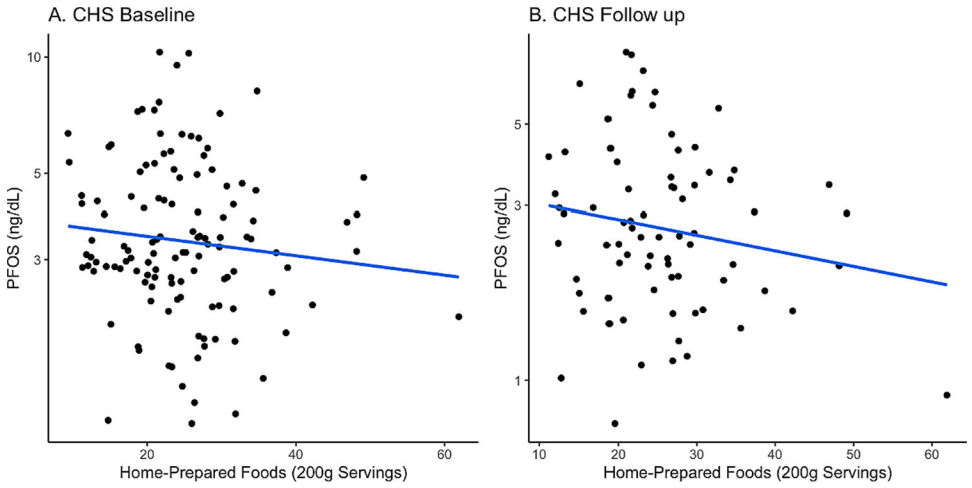

Fig. 2.

Associations of foods prepared at home with PFOS concentration in CHS at A. baseline and B. follow-up. A 200 g serving increase in consumption of home prepared foods was associated with a 0.009 ($p = 0.049$) unit decrease in log PFOS at baseline and a 0.016 ($p = 0.002$) unit decrease in log PFOS at follow up in CHS.

Table 1

Demographic characteristics of study participants in the CHS cohort (N = 124) and NHANES (N = 604).

Variables	CHS (N = 124)	NHANES (N = 604)
	N (%) or Mean (SD)	N (%) or Mean (SD)
Sex		
Male	66 (54 %)	313 (52 %)
Female	57 (46 %)	291 (48 %)
Baseline Age (Years)	19.8 (1.2)	19.1 (1.7)
Follow-Up Age (Years) †	24.0 (0.8)	
Race		
Hispanic	71 (58 %)	185 (31 %)
Non-Hispanic White	45 (37 %)	188 (31 %)
All Other	7 (6 %)	231 (38 %)
Parental Education		
Missing	3 (2 %)	30 (5 %)
< High School	23 (19 %)	243 (40 %)
High School Grad	16 (13 %)	120 (20%)
> High School	81 (66 %)	211 (35 %)
Baseline BMI (kg/m^2)	29.5 (4.8)	27.0 (7.8)
Overweight	103 (84 %)	290 (48 %)

† N = 88 for follow-up time period.

Table 2

Geometric mean PFAS concentrations in CHS (baseline and follow-up plasma PFAS) and NHANES (serum PFAS).

PFAS	CHS Baseline (n = 124)		CHS Follow-up (n = 88)				NHANES (n = 604)[a]	
	Geometric Mean [95 % CI] (ng/dL)	Below LOD [n (%)]	Geometric Mean [95 % CI] (ng/dL)	Below LOD [n (%)]	Mean Difference	P-Value[b]	Geometric Mean [95 % CI] (ng/dL)	Below LOD [n (%)]
PFOS	3.35 [3.08, 3.64]	0 (0 %)	2.68 [2.4, 3.01]	0 (0 %)	−0.62	< 0.0001	3.34 [3.17, 3.52]	2 (0.3 %)
PFOA	1.34 [1.26, 1.43]	0 (0 %)	1.06 [0.98, 1.16]	0 (0 %)	−0.28	< 0.0001	1.35 [1.29, 1.41]	3 (0.3 %)
PFHxS	1.02 [0.9, 1.17]	0 (0 %)	0.52 [0.43, 0.62]	0 (0 %)	−0.73	< 0.0001	1.03 [0.96, 1.10]	4 (0.7 %)
PFNA	0.48 [0.46, 0.50]	0 (0 %)	0.28 [0.25, 0.31]	1 (1 %)	−0.17	< 0.0001	0.44 [0.42, 0.47]	25 (4 %)
PFDA	0.20 [0.18, 0.22]	1 (1 %)	0.25 [0.23, 0.28]	0 (0 %)	0.06	< 0.05	0.14 [0.13, 0.15]	157 (26 %)
PFHpS	0.18 [0.17, 0.19]	0 (0 %)	0.07 [0.06, 0.08]	27 (31 %)	−0.10	< 0.0001		
PFPeS	0.05 [0.04, 0.06]	24 (19 %)	–	88 (100 %)				
PFHpA	–	74 (60 %)	–	88 (100 %)				
PFBS	–	91 (73 %)	–	88 (100 %)				

[a] N = 577 completed PFAS assessment for PFOS and PFOA, N = 604 completed PFAS assessment for PFHxS, PFNA and PFDA

[b] P-Value for paired t-test for difference in mean PFAS concentration between baseline and follow-up in CHS; PFOS, PFOA, PFHxS, PFNA, PFDA and PFHpS analyzed as continuous, PFPeS analyzed as continuous at baseline only, PFHpA and PFBS analyzed as “>LOD” vs. “<LOD” at baseline only; (−) Geometric means for PFAS with > 40 % of values below LOD were not calculated; PFHpS, PFPeS, PFHpA and PFBS not quantified in NHANES.