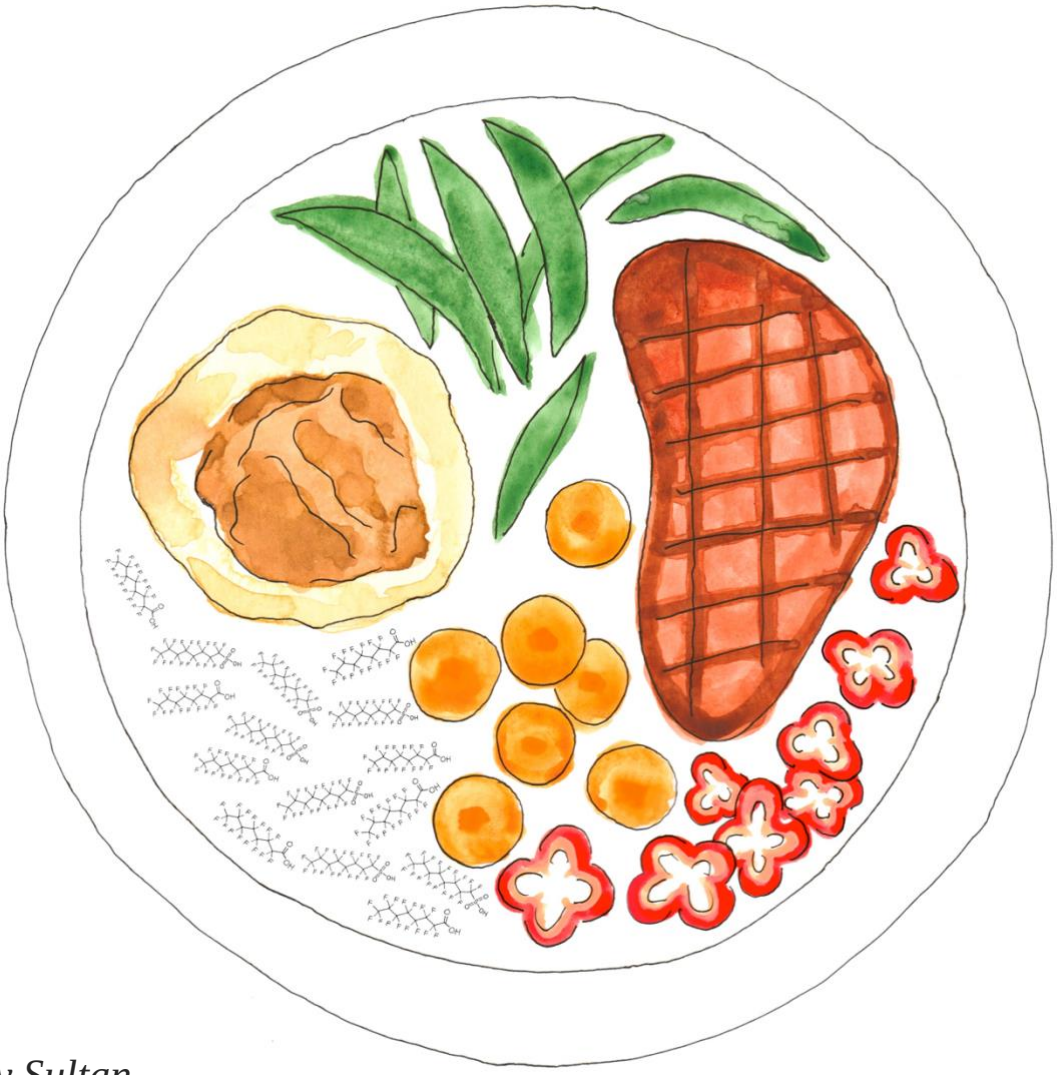
What You Don’t See on the Menu:

Dietary Per- and Polyfluoroalkyl Substance (PFAS) Exposure in Adolescents



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Abstract

Diet is thought to be a primary exposure pathway to per- and polyfluoroalkyl substances (PFAS) in non-occupationally exposed populations. To date, relatively few studies have examined the relationship between dietary intake and PFAS exposure in adolescents in the United States. In this study, we analyzed data obtained from a cohort of 193 Cincinnati metropolitan area children in mid-childhood (median age 12.3 years). Cross-sectional data included food frequency questionnaires, sociodemographic questionnaires, and serum PFAS concentrations. We used bivariate and multivariable regressions to estimate associations of both Healthy Eating Index (HEI) total and component scores and micro- and macro-nutrient variables and serum concentrations of PFOA, PFOS, PFHxS, and PFNA. We found that higher total HEI scores, whole fruit and total fruit HEI component scores, and total dietary fiber intake were associated with lower levels of all four PFAS after adjustment for covariates. We conclude that certain dietary patterns may be associated with lower levels of PFAS exposure, but that wholly eliminating PFAS from the diet is beyond the power of an individual consumer. The endemic nature of PFAS in our environment means that all diets will lead to exposure. Additional research is necessary to better understand the health impacts of low-level PFAS exposure and to identify dietary sources of chemical toxicant exposure.

1. Introduction

Per- and polyfluoroalkyl substances (PFAS) are a large category of anthropogenic chemicals consisting of a hydrophobic carbon chain of variable length which is either partially (poly) or fully (per) fluorinated. At the end of the chain, there is a hydrophilic functional group which can be neutral, positively, or negatively charged. Two of the most common functional groups are carboxylic acid (perfluoroalkyl carboxylic acids) and sulfonic acid (perfluoroalkane sulfonic acids). Due to this unique chemical structure, PFAS are ideal water and oil repellants and have been used in both industrial processes and consumer products. PFAS are found in textiles, furniture, cleaning agents, food packaging, and non-stick cookware as well as aviation hydraulic fuels, fire-fighting foams, and paints (Kotthoff et al., 2015; Jian et al., 2018).

Unfortunately, the same properties that make PFAS useful also make them potentially dangerous. The carbon and fluorine bond is extremely stable, making PFAS highly resistant to thermal desorption, chemical degradation, and biodegradation, thereby allowing them to be incredibly persistent in the environment. PFAS are present in the air, surface water, ground water, and soil (ASTDR, 2018) and bioaccumulate and bio-magnify up the food chain (EFSA, 2020). Once in the human body, PFAS accumulate in areas of the body rich in proteins like the kidneys, liver, and blood (Verreault et al., 2005). PFAS strongly bind to serum proteins, particularly albumin (Salvalaglio et al. 2010). Many PFAS are slowly eliminated from the body: perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA) and perfluorohexane sulfonic acid (PFHxS) have half-lives in human serum of 5.4 years, 3.8 years, and 8.5 years, respectively (Olsen et al., 2007). PFAS have been produced since the late 1940s and they are now nearly ubiquitous in human blood in the United States and the world. PFOS, PFOA, PFHxS,

and perfluorononanoic acid (PFNA) are the most commonly found varieties of PFAS and have been detected in over 98% of the United States population (Calafat et al., 2007).

PFAS have been linked to a litany of adverse health outcomes including cardiovascular disease (Shankar et al., 2012), high cholesterol (Nelson et al. 2010), thyroid hormone disruption (Wen et al. 2013) and hypertension (Min et al., 2012). Of particular concern are adolescents who have been observed to have a higher body burden of PFAS relative to adults (Kato et al., 2011; Kingsley et al., 2018). Exposure to PFAS early in life has the potential to alter health throughout childhood and into adulthood. In children, PFAS have been shown to modulate immune response, with lower antibody levels being detected in vaccinated adolescents with elevated PFAS serum concentrations (Grandjean et al., 2012). PFAS have also been linked with dyslipidemia, a condition that may lead the development of atherosclerosis and cardiovascular disease later in life (Geiger et al., 2014; Frisbee et al., 2010). Other studies have linked PFAS to decreased glomerular filtration rate (Kataria et al., 2015) and delayed age of menarche (Lopez-Espinosa et al., 2011).

Due to evidence of their negative effects and environmental persistence, PFAS have come under increased regulatory scrutiny in recent years. PFOS was banned under the Stockholm Convention in 2009 and PFOA was added in 2019 (Caron-Beaudoin et al. 2020). Since 2000, chemical manufacturers have gradually phased out the production of longer chain PFAS in favor of shorter chain versions that have half-lives ranging from a few days to a month (EFSA 2020). While these shorter chain PFAS may be an improvement on their long-chain counterparts, they have yet to be thoroughly investigated. In any case, due to their persistent nature, these and other PFAS will continue to be endemic in the environment.

Given their persistence and toxicity, it is crucial to understand PFAS exposure pathways. Humans are exposed to PFAS through air, indoor and outdoor dust, drinking water, and food. In cases of local contamination, such as contaminated drinking water, it is water that is the major source of exposure. In cases of low-level exposure in non-occupationally exposed populations, diet is a much more significant factor. A Canadian study found that diet accounted for 61% of total exposure to PFAS (Tittlemier et al., 2007), while a Norwegian study estimated the diet contributed 67-85% of the total intake of PFOA and 88-99% of the total intake of PFOS (Haug et al., 2011).

Prior studies investigating associations between diet and PFAS exposure include market-basket and human exposure assessments. Market-basket studies involve measuring PFAS concentrations directly from food items and then using dietary surveys to estimate the total intake of PFAS from different food groups. Market-basket studies most commonly identified fish as the primary source of PFAS, but there has been a great degree of variation in the results of these studies, particularly across different countries. Studies implicating fish as a source of PFAS exposure were conducted in Norway, the Netherlands, the Faroe Islands, the United Kingdom, France, Spain, and China (Haug et al., 2010; Carlsson et al., 2016; Noorlander et al., 2011; Eriksson et al., 2013; Clarke et al., 2010; Yamada et al., 2014; Domingo et al., 2012; Hlouskova et al., 2013; Papadopoulou et al., 2019; Zhang et al., 2010). High concentrations of PFAS were also found in meats, particularly liver, in the Netherlands, Canada, China, and Europe (Noorlander et al., 2011; Tittlemier et al. 2007; Chen et al., 2018; Hlouskova et al., 2013). Other studies raised concerns about PFAS found in dairy products (Macheka et al., 2021; Wang et al., 2010; Xing et al., 2016), cereals (Haug et al., 2010; Chen et al. 2018), vegetables (Noorlander et

al., 2011; Herzke et al., 2013), eggs (Wang et al., 2008), potatoes (Cornelis et al., 2012), snack food (Ostertag et al., 2009), and popcorn and restaurant food (Sussman et al., 2019).

Human exposure assessment studies typically have participants answer questions about their diet and provide blood samples which are measured for serum or plasma levels of PFAS. Exposure assessment studies examine variations in individuals' dietary consumption in relation to serum or plasma PFAS concentrations. To date, relatively few studies of this kind have examined adolescents in the United States. Many prior studies focused on pregnant women (Tian et al., 2018; Halldorsson et al., 2008; Manzano-Salgado et al., 2016; Caron-Beaudoin et al., 2020) or adult populations (Liu et al. 2017; Thépaut et al., 2021; Sjorgen et al., 2016; Lin et al., 2020). Averina et al. (2018) looked at 940 adolescents aged 15-19 in the Troms region of Northern Norway. This study found that high intake of fish and sugary drinks was positively associated with several kinds of PFAS, high intake of "junk food" such as pizza, hamburgers, and sausages was positively associated with PNFA, and intake of canned foods several times a week was positively associated with PFHxS. Other findings from the Troms study that linked consumption of seagull eggs and reindeer meat to higher levels of PFAS are less applicable to the United States population. Nyström et al. (2022) reported positive associations between PFAS and seafood consumption in 1,098 Swedish adolescents aged 10-21 years. Seshasayee et al. (2021) conducted a study using a cohort of 548 Boston-area children, administering a food questionnaire in early childhood (median age 3.3 years), and then collecting plasma samples in mid-childhood (median age 7.7 years). For individual food items, this study found a positive association of both increased ice cream and soda intake with plasma concentrations of a type of PFAS called methyl-perfluorooctane-sulfonamidoacetic acid (MeFOSAA). The study also found

that children who adhered to a diet high in packaged foods and fish had higher concentrations of all six PFAS examined.

Studies that focus on adults are not necessarily applicable to adolescents because adolescents have different eating habits (Bearer 1995). Furthermore, adolescents may be more vulnerable to the adverse health outcomes driven by PFAS exposure. For these reasons, it is important to further explore the role diet plays, specifically in the exposure of adolescents to PFAS. This present study sought to build upon the limited existing research to better understand associations between children's dietary patterns and their serum PFAS concentrations using data from the Health Outcomes and Measures of the Environment (HOME) Study, a prospective cohort of mother-child pairs from the Cincinnati area who were recruited during pregnancy and returned for follow-up assessments during the first 12 years of life. In contrast to studies published to this point which investigated individual food items, this study considered macro- and micro-nutrient data. Additionally, it examined associations between PFAS serum concentrations and dietary scores (total and component) derived from the Healthy Eating Index (HEI), a measure of dietary quality developed by the United States Department of Agriculture (USDA), with the goal of assessing to what degree prevailing notions of dietary quality serve as predictors of possible chemical exposures.

2. Materials and Methods

2.1 Study Participants

The present study used data from the Health Outcomes and Measures of the Environment (HOME) Study, a prospective cohort study designed to examine the impacts of low-level exposure to environmental chemicals in adolescents. Between March 2003 and January 2006,

468 pregnant women living in the nine-county region of the Cincinnati, Ohio metropolitan area were recruited for the study. HOME Study staff identified women using the medical scheduling systems of nine prenatal practices associated with three hospitals. To be considered eligible for the study, women had to be living in the study region, less than 19 weeks pregnant, older than 18 years, not living in a mobile or trailer home, HIV-negative, not taking medications for seizures or thyroid disorders, planning to continue prenatal care and deliver at the collaborating clinics and hospitals, planning to continue living in the Cincinnati region for the next year, fluent in English, and free of diagnosis of diabetes, bipolar disorder, schizophrenia, or cancer that resulted in radiation treatment or chemotherapy (Braun et al., 2016).

Participation in the study included two clinic visits and one at home visit during pregnancy, a delivery hospital visit, and home or clinic visits at four weeks, and one, two, three, four, and five, eight and 12 years of age. From 2016-2018, the most recent follow-up data were gathered, occurring at roughly the 12-year mark (age range: 11-14 years). This study used cross-sectional data from the 12-year visits (Braun et al. 2020). Of the 468 women who initially enrolled, 412 were invited to participate at the 12-year visit and 256 children returned to the study clinic for the visit (62%). After eliminating twin sets and participants missing covariate or serum PFAS data, 193 adolescents were included in the present study.

2.2 Dietary Assessment

Dietary information was obtained by trained research staff from the Clinical Translation Research Center (CTRC), who performed three 24-hour dietary recall assessments using the Nutrition Data Systems for Research software and foods data base in order to evaluate total daily energy and macro- and micro-nutrient intake (Yolton et al., 2015). The first dietary recall was

conducted in-person during the study visit and the second and third were conducted by telephone. The variables derived from these assessments were largely based on total grams of a given nutrient consumed daily, averaged across the three days. Certain intakes were expressed as the percent of calories that a dietary variable contributed to a participant's overall diet.

In addition to assessing micro- and macro nutrient intake, using dietary recall data, HOME Study staff calculated HEI scores, a measure of dietary quality, based on the degree of dietary conformity with the Dietary Guidelines for Americans (USDA 2020). HEI scores are based on quantities consumed per 1,000 kcal rather than total amounts of food consumed (Krebs-Smith et al., 2018). The HEI is composed of summed scores from thirteen food-group categories: total fruits, whole fruits, total vegetables, greens and beans, whole grains, dairy, total protein foods, seafood and plant proteins, fatty acids, refined grains, sodium, added sugars, and saturated fats (Supplementary Material, Table S1). Added sugars and saturated fats scores were combined into a single category.

2.3 Serum PFAS measurements

Fasting blood samples were collected and processed by the CTRC study staff. Serum was separated from whole blood before being stored at -80°C. Samples were shipped on dry ice to the Centers for Disease Control and Prevention for quantification of PFAS concentration. Serum samples were treated and extracted by solid phase extraction. Then PFAS concentrations were quantified using previously published methods that include dilution of samples with formic acid and analysis by online solid-phase extraction high performance liquid chromatography – tandem mass spectroscopy (Kuklenyik et al., 2005; Kato et al., 2011).

Laboratory staff measured seven different kinds of PFAS: Me-PFOSA-AcOH, PFDeA, PFHxS, PFNA, PFUA, PFOA (branched and unbranched), and PFOS (branched and unbranched). We summed branched and unbranched concentrations to create sums of PFOA and PFOS. Of the 193 children included in this study, PFOA, PFOS, PFHxS, and PFNA were detected in 100% of samples, while PFDeA, Me-PFOSA-AcOH, and PFUA were detected in 46.6%, 34.7%, and 5.2% of samples, respectively. As a result, we only included PFOA, PFOS, PFHxS, and PFNA in further analysis.

2.4 Covariates

Information about age, child sex, child race, mother's education, and child community deprivation index was collected through questionnaires administered by study staff at the 12-year visit. Breastfeeding data were obtained from questionnaires conducted at clinic visits from one to three years of age. Physical activity scores were derived through a questionnaire administered at the 12-year visit as well as through data obtained from an accelerometer—a wearable device that measures the intensity of the activity of a participant.

2.5 Statistical Analysis

We performed statistical analysis using the software package Stata/SE version 16.1. We replaced values of PFAS concentration below the level of detection (LOD) with LOD/$\sqrt{2}$. The distributions of all four PFAS concentrations were right-skewed and were $\log_2$-transformed to approximate normality assumption for statistical analysis.

We constructed tables describing sociodemographic characteristics of study participants as well as the mean HEI scores and geometric mean serum PFOA concentrations based on

demographic category. Additionally, we calculated the median (25^{th} and 75^{th} percentiles) serum concentrations of all four PFAS examined and the median (25^{th} and 75^{th} percentiles) total and component HEI scores and macro- and micro-nutrient variables.

Our bivariate (unadjusted) and multivariable (adjusted) linear regressions estimated the percent difference in serum PFAS concentrations per standard deviation increase of overall HEI scores, HEI component scores, and macro- and micro-nutrient data. Using a directed acyclic graph (DAG), we identified potential confounders *a priori* and included child age (continuous), child sex (male, female), child race (black, white, other), child physical activity score (continuous), child community deprivation index (continuous), duration of any breastfeeding (weeks), mother's education (high school or less, some college/technical school, bachelor's degree/graduate school), and for selected nutritional variables, daily calories consumed (continuous) (Figure 1).

3. Results

3.1 Descriptive Statistics

Participants in this study (N=193) had a median (25^{th}-75^{th}) age of 12.3 (11.9-12.8), included slightly more females than males (53%), and were mostly non-Hispanic White (55%). The median (25^{th}-75^{th}) annual household income was $75,000 ($35,000-$145,000). Median ($25^{th}$-$75^{th}$) community deprivation index was 0.31 (0.22-0.40) on a scale of 0-1, where 1 represented the most deprived community, and the majority of participants' mothers (52%) received a bachelor's or graduate degree (Table 1).

Median (25^{th}-75^{th}) serum concentrations of PFOA, PFOS, PFHxS, and PFNA quantified in participants' serum were 1.3 (1.0-1.6), 2.4 (1.7-3.2), 0.7 (0.5-1.0), and 0.3 (0.2-0.5) ng/mL,

respectively (Table 2). When comparing geometric mean concentrations of PFOA across different demographic groups, non-Hispanic White adolescents and those who had higher household income, lower community deprivation index scores, and mothers with higher levels of education had the highest median PFOA concentrations when compared to other groups. All four $\log_2$-transformed PFAS concentrations were moderately to strongly correlated with each other (r=0.43 to 0.72) (Table 3). Non-Hispanic White adolescents, and those whose mothers had higher levels of education, higher household incomes, and lower community deprivation scores had higher mean HEI total scores than adolescents from other groups (Table 1).

3.2 Unadjusted Associations

In unadjusted models, several HEI dietary components were associated with higher serum PFAS concentrations (Figure 2). Specifically, each standard deviation increase in total HEI score was associated with increases in serum PFOS, PFOS, PFHxS and PFNA concentrations of between 4-8%. Dairy component scores were associated with increases in all four serum PFAS concentrations ranging from 11-15%. Seafood and plant protein scores were associated with increased concentrations of PFOA, PFOS, and PFHxS between 7% and 8%. Lastly, both total fruit and whole fruit scores were associated with increases in PFOA, PFOS, and PFHxS concentrations at a range of 3% to 7% for total fruit and 6% to 7% for whole fruit.

3.3 Adjusted Associations

After adjustment for covariates, many of the associations between PFAS concentrations and dietary variables observed in the unadjusted model were attenuated toward the null and, in some cases, reversed in direction (Figure 3). Notably, total HEI scores were positively associated with

PFAS concentrations in unadjusted models but were associated with 2-9% decreases in PFAS concentrations per standard deviation increase after adjustment for covariates. The magnitude of the positive association between dairy scores and serum PFAS concentrations dropped considerably to a range of -1% to 6%. Similarly, seafood and plant proteins had reduced associations in the adjusted model of -9% to 2%.

Total fruit and whole fruit scores, which were positively associated with PFAS concentrations before adjustment, were negatively associated with PFAS in the adjusted model. One standard deviation increases in total fruit scores was linked to 3% to 11% decreases in serum PFAS concentration. Meanwhile, whole fruit scores were associated with decreases in serum PFAS concentration from 5% to 16%. Total dietary fiber had relatively null associations with the four PFAS in the unadjusted models but was negatively associated with PFAS concentrations in the adjusted models (5-10% increase per standard deviation increase in dietary fiber intake).

Associations between macro-nutrient intake and PFAS serum concentrations were generally null in magnitude. In adjusted models, we observed differences in PFAS concentration of -4% to 1%, -4 to 4%, and -6 to 3% per standard deviation increase in fats, carbohydrates, and proteins consumed, respectively.

We examined which covariates were responsible for the attenuation or reversal of associations between HEI total and component PFOA concentration by adjusting for individual covariates (Table 4). Child race appeared to be the primary factor explaining the difference between unadjusted and adjusted models, followed by mother's education level.

4. Discussion

In this cross-sectional study of adolescents from the Cincinnati, Ohio region, we observed that higher total HEI scores, a measure of overall dietary quality, were associated with lower PFAS concentrations. In addition, several dietary components—total and whole fruit score, and total dietary fiber consumption—were associated with lower serum PFAS concentrations. Seafood and plant protein scores were not meaningfully associated with PFAS concentrations.

The serum PFAS concentrations found in this study are similar to those observed in the NHANES data collected between 2017-2018 for 12-19 year-olds, a similar timeframe as the present study. In NHANES, PFOA, PFOS, PFHxS, and PFNA had median (25^{th}-75^{th}) concentrations of 1.2 (0.7-1.7), 2.6 (1.5-3.7), 0.8 (0.2-1.4), and 0.4 (0.1-0.7) ng/mL, respectively (CDC 2018). However, concentrations among adolescents in this study are lower than those observed in studies of non-contemporary children (Pinney et al., 2014; Gump et al., 2010; Wu et al., 2015). NHANES data from the 2003-2004 cycle reported median (IQR) concentrations in the 12-19-year-old range of 13.9 (2.9-5.4) ng/mL (PFOA), 9.9 (14.4-27.1) ng/mL (PFOS), 2.3 (1.2-4.8) ng/mL (PFHxS), and 0.7 (0.5-1.2) ng/mL (PFNA) (Calafat et al., 2007). As different PFAS are phased out of production, there has been an encouraging trend of decreasing concentrations of these PFAS, and the data from this study, collected between 2016 and 2018, fits into this decline (Vestergren et al., 2009).

The demographic trends observed in this study observing higher serum PFOA concentrations among non-Hispanic White adolescents and those with higher household incomes, lower community deprivation index scores and whose mothers had obtained higher levels of education were similar to previous studies (Pinney et al., 2014; Jackson-Browne et al.,

2010). Duration of breastfeeding was positively associated with PFOA concentrations, a trend that has also been previously observed (Wu et al., 2015; Kingsley et al., 2018).

Compared to a representative sample of Americans aged 2-17 from the 2015-2016 NHANES, HOME Study participants had lower mean HEI scores (44.8 vs. 53.9) and lower scores in every component category except fatty acids and added sugars and saturated fats (USDA 2019) (Table 5).

Other studies have also examined associations between PFAS and dietary scores. In a study of Swedish adolescents, Nyström et al. (2022), found that the Swedish Healthy Eating Index for Adolescents was positively associated with concentrations of PFNA, PFDA, PFUnDA, and lin-PFOS, while a measure scoring dietary diversity was positively associated with PFNA, PFDA, PFUnDA, and lin-PFOS. In a study of pre-diabetic U.S. adults, Lin et al. (2020) found that a Mediterranean-like diet was not associated with serum PFAS concentrations; however, PFHxS was positively associated with a low carbohydrate and high protein diet. In elderly Swedes, Sjorgen et al. (2016) observed positive associations between a Mediterranean-like diet and PFUnDA, PFHxS, PFNA, PFDA, PFOSA and PFOA, as well as positive associations of carbohydrate and high protein diet with PFOS, PFDA, and PFUnDA but not PFHxS. It is possible that the discrepant associations of dietary summary scores and serum PFAS in the present study and these prior studies are due to differences in measures of dietary quality or diet composition in the source populations.

While a high intake of fish has been identified as a key source of PFAS exposure, seafood and plant protein was not associated with serum PFAS concentration in the present study. These findings contrast with numerous other exposure assessment studies which found positive associations between fish consumption and PFAS levels (Lin et a., 2020; Jain et al., 2014;

Averina et al. 2018; Seshasayee et al. 2021; Thépaut et al., 2021; Manzano-Salgado et al., 2016; Liu et al., 2017; Christensen et al., 2017; Brantsæter et al., 2013; Rylander 2010, Nyström et al., 2022, Duffek et al., 2020). It is likely that this study did not observe the same positive association because participants consumed lower amounts of fish—the median HEI seafood and plant protein score was 0.3 on a five-point scale—and our measure of seafood intake combined both seafood and plant protein. Furthermore, Rylander et al. (2010) notes that PFAS concentrations were only elevated for the highest consumers of marine food and that there was no significant difference between the medium and low consumers of marine food. Brantsæter et al. (2013) notes that even between the highest and lowest consuming groups median PFAS levels only varied by between 20-30%.

After adjusting for covariates, dairy was not associated with higher serum PFAS concentrations in this study, likely due to higher consumption of dairy among adolescents who were non-Hispanic white and of higher socioeconomic position; these same factors were associated with higher serum PFAS levels (i.e., confounding). The results of other studies examining associations between dairy and PFAS concentrations are mixed. A market-basket study in the Netherlands reported that 25% of PFOS intake was due to milk consumption, and a similar study in Spain found a similar proportion (21%) (Noorlander et al., 2011, Ericson et al., 2008). Meanwhile, both Jain (2014), examining Americans aged 12 years and above, and Eriksen et al. (2011), examining Danish men, observed negative associations between dairy and PFAS concentrations. Variation in PFAS from dairy could be due to the different environments in which cows are raised which would affect the level to which they are exposed to PFAS through the air, drinking water, or food. It is estimated that 39% of the PFAS in cows eventually leaves the body in their milk (Vestergren et al., 2013).

Higher intake of dietary fiber was linked with lower serum concentrations of PFOS, PFOA, PFHxS and PFNA. Lin et al. (2020) also observed that individuals consuming diets characterized by high fiber consumption had lower PFAS concentrations, while dietary patterns that were low in fiber were associated with increased PFAS concentrations. Dzierlenga et al. (2021) found that dietary fiber was associated with lower concentrations of PFOA, PFOS, and PFNA. This study postulated that fiber enhanced the gastrointestinal excretion of PFAS from the body. Additionally, increased consumption of both total and whole fruit, which are also sources of fiber, was associated with lower serum concentrations of all four PFAS in this study. Similarly, both Lin et al. (2020) and Halldorsson et al. (2008) observed that higher intake of fruit was linked to lower serum PFAS concentration. In contrast, Nyström et al. (2022) found no significant associations between fruit consumption and PFAS levels, and Jain (2018) observed positive associations between fruit and fruit juice intake and levels of PFHxS.

We speculate that increased fruit and dietary fiber consumption as well as higher HEI scores may be indicative of dietary patterns and behaviors associated with consumption of fewer PFAS intensive foods, thus explaining their negative association with these chemicals.

One of the strengths of this study was the wealth of data provided by the HOME Study, which included detailed measures of diet and numerous covariates. A limitation is the relatively modest sample size of the cohort. Additionally, serum PFAS concentrations are long-term biomarkers that result from years of exposure, whereas dietary recall data represent only recent intake. If a participant's diet has changed over time, then their recent dietary intake may not reflect the dietary pattern that led to their current PFAS body burden. The results from the present cohort may also not be generalizable to other populations. However, it should be noted that this population had median PFAS concentrations comparable to other studies at the same

time and relations of socioeconomic factors with PFAS were similar to other studies in the United States (Pinney et al., 2014; Jackson-Browne et al., 2010; Calafat et al., 2007).

An important area for further research is the role that processing and packaging plays in dietary PFAS exposure. In the present study, we did not have information about food processing, so we could not include this factor in our analysis. Seshayasee et al. (2021) observed that children adhering to a dietary pattern including frequently packaged foods had higher serum PFAS concentrations. Susmann et al. (2019) found an association between PFAS and higher intakes of food from fast food and pizza restaurants, which are likely to use to food contact materials containing PFAS. Existing research suggests that migration of PFAS from packaging materials into food is possible (Begley et al., 2008).

Additionally, more research on the effects of background level PFAS is required. As more information about PFAS has been uncovered, the European Food Safety Authority has dramatically lowered its assessment of tolerable daily intakes from 150 ng/kg of bodyweight per day for PFOS and 1500 ng/kg bw per day for PFOA, to 13 ng/kg and 6 ng kg of body weight per day respectively, and most recently to 0.63 ng/kg bw per day for all PFAS combined (EFSA 2008, 2018, 2020). The full extent of the threat presented by low-level exposure to PFAS must be understood to accurately inform future health policy decisions.

Other studies like the present study that sought to identify the key food groups associated with PFAS exposure have had disparate and often contradictory findings. Which food items or groups are identified as being strongly related to PFAS seems to be dependent on the study population itself. For instance, Halldorsson et al. (2008), investigating a cohort of pregnant Danish women, found PFOS and PFOA to be positively associated with higher intakes of red meat, animal fats, and snacks and found no association with fish intake. Meanwhile, in a study of

Norwegian women, high fish diets were linked with higher concentrations of PFOS, PFNA and PFHxS (Rylander et al., 2010). A third study of pregnant women in Shanghai, found that both increased fish and red meat were associated with elevated PFAS levels, but the strongest associations were with animal offal (liver, kidney, blood, and heart) (Tian et al., 2018). The Danish population consumed a median of 69 grams of red meat and 21 grams of fish each day, while the median Norwegian consumed 42 grams of meat (beef and processed meat products) and over 70 grams of fish. In each case, the most popular food item was the strongest predictor of PFAS levels in the population. In addition, for a food item to show a strong association with PFAS levels, it must have reasonably high levels of PFAS but also it must have sufficient variation between individuals in the population. Fish and meat both tend to have higher between-person variation than other food products such as vegetables and grains, which are more evenly consumed across populations (Berg et al, 2014).

The most satisfying conclusion to this study would have been to identify a single food group responsible for PFAS exposure in American adolescents, which could then be summarily avoided. Unfortunately, it appears that no such food group exists. PFAS are endemic in our environment and food systems. The individual consumer has little power to avoid background levels of PFAS exposure simply by shopping in a different aisle at the supermarket. Furthermore, it is unclear to what extent the desire to avoid this low-level chemical exposure should be prioritized in our dietary choices. For example, fish may have high levels of PFAS, but are also associated with a host of health benefits particularly related to cardiovascular and heart disease (Rylander et al., 2010).

This study and studies like it illustrate the pervasiveness of PFAS which have infiltrated all levels of the environment. Ultimately, efforts to avoid exposure should be focused on reducing

the industrial and commercial production and usage of PFAS altogether, rather than the actions and behaviors of individuals.

5. Tables and Figures

Table 1: Characteristics of the 193 eligible participants from the HOME Study

Category	N(%)	GM PFOA (ng/ml)	Mean HEI Total Score
Age			
11 Years	60 (31%)	1.3	45
12 Years	100 (52%)	1.3	45
13 Years Plus	33 (17%)	1.1	44
Sex			
Male	90 (47%)	1.4	45
Female	103 (53%)	1.2	45
Race			
White, non-Hispanic	106 (55%)	1.5	47
Black, non-Hispanic	74 (38%)	1	41
Other	13 (7%)	1.4	48
Household Income			
$0-$50,000	63 (33%)	1	40
$50,000-100,000	49 (25%)	1.4	45
$100,000-150,000	34 (18%)	1.5	49
$150,000+	47 (24%)	1.5	48
Mother's Education			
Highschool or less	26 (14%)	1.1	42
Some College/technical school	66 (34%)	1.1	43
Bachelor's/Graduate Degree	101 (52%)	1.5	47
Deprivation Index			
0-0.245	64 (33%)	1.5	47
0.245-0.357	65 (34%)	1.3	47
0.3569-1	63 (33%)	1.1	40
Physical Activity Score			
1-2.206	65 (34%)	1.1	42
2.206-2.804	64 (33%)	1.3	46
2.804-5	64 (33%)	1.4	46
Duration of Breastfeeding			
0-4 weeks	64 (33%)	1.1	44
4-34 weeks	59 (31%)	1.3	44
24-96 weeks	61 (32%)	1.5	47
Unreported	9 (5%)	0.9	41
Average Daily Calories			
657-1644	62 (32%)	1.2	47
1645-2031	67 (35%)	1.3	43
2031-3252	64 (33%)	1.2	45

Table Note: Deprivation Index, Physical Acitivty Score, Duration of Breastfeeding and Average Daily Calories grouped by tercile. Deprivation Index derived by conducting principal compenent analysis on eight census tract level variables and performing z-score normalization.

Table 2: Univariate statistics of adolescents' serum concentration of selected perfluoroalkyl substances

Category	Median (IQR)	5th-95th Percentile	Minimum-Maximum
PFOA	1.3 (1.0-1.6)	0.7-2.5	0.4-5.2
PFOS	2.4 (1.7-3.2)	1.0-5.7	0.7-11.9
PFHxS	0.7 (0.5-1.0)	0.2-2.3	0.1-20.3
PFNA	0.3 (0.2-0.5)	0.1-0.8	0.1-2.5

Table Note: Measurements given in nanograms per mililiter (ng/ml).

Table 3: Pearson's correlation coefficients of log2-transformed perfluoroalkyl serum concentrations

Category	PFOA	PFOS	PFHxS	PFNA
PFOA	1			
PFOS	0.6712	1		
PFHxS	0.5319	0.7164	1	
PFNA	0.5924	0.602	0.432	1

Table 4: Percent differece in serum PFOA concentrations calculated from linear regressions of selected variables with adjustment from covariates

	Dairy	**Whole Fruit**	**Fatty Acid Ratio**	**Total Score**
Covariates	% Difference (95% CI)	% Difference (95% CI)	% Difference (95% CI)	% Difference (95% CI)
Unadjusted	14 (4, 26)	7 (-3, 18)	-10 (-19, -1)	8 (-2, 19)
Age	15 (4, 26)	7 (-3, 18)	-11 (-20, -2)	7 (-3, 18)
Physical Activity Score	14 (3, 25)	7 (-3, 17)	-11 (-19, -2)	6 (-4, 17)
Sex	12 (2, 24)	8 (-2, 19)	-10 (-18, -1)	8 (-2, 19)
Deprivation Index	11 (1, 22)	1 (-8, 12)	-8 (-17, 1)	4 (-6, 14)
Weeks of Breatfeeding	10 (0, 21)	-1 (-10, 10)	-7 (-16, 2)	3 (-6, 14)
Mother Education Level	9 (-1, 20)	-1 (-10, 9)	-7 (-16, 2)	3 (-6, 13)
Race	2 (-7, 12)	-6 (-14, 3)	-3 (-11, 6)	-3 (-11, 6)
Fully adjusted	1 (-8, 11)	-10 (-18, -1)	-5 (-13, 4)	-7 (-15, 2)

Table Note: Fully adjusted regressions include age (continuous), child sex (male, female), child race (black, white, other), physical activity score (continuous), community deprivation index (continuous), duration of breastfeeding (weeks), and mother's education (high school or less, some college/technical school, bacherlor's degree/graduate school) and total calories (continuous).

Table 5: HEI total and component scores and macro- and micro-nutrient statistics of the cohort

Category (range)	Mean	5th-95th Percentile	Minimum-Maximum
Total Score (0-100)	44.8	28.8-63.0	21.8-75.3
Total Vegetables (0-5)	1.9	0.3-3.9	0.0-5.0
Greens and Beans (0-5)	0.7	0.0-3.3	0.0-4.7
Total Fruit (0-5)	1.8	0.0-4.6	0.0-5.0
Whole Fruit (0-5)	1.8	0.0-5.0	0.0-5.0
Whole Grains (0-10)	3.2	0.0-8.4	0.0-10.0
Dairy (0-10)	6.3	2.2-10.0	0.0-10.0
Total Protein Foods (0-5)	3.7	1.8-5.0	0.0-5.0
Seafood and Plant Proteins (0-5)	1.1	0.0-3.4	0-5
Fatty Acid Ratio (0-10)	4.2	0.4-8.7	0.0-10.0
Sodium (0-10)	4.1	0.7-7.8	0.0-10.0
Refined Grains (0-10)	4.1	0.4-8.3	0.0-10.0
Solid Fats, Alcohol and Added Sugars (0-20)	11.9	4.4-18.8	2.3-20.0

Category	Mean	5th-95th Percentile	Minimum-Maximum
Total Grams (g)	1942.8	1154.4-3001.4	736.2-4117.1
Energy (kcal)	1851.1	1092.8-2635.9	657.2-3251.5
Total Fat (g)	72.5	39.6-108.9	21.3-157.5
Total Carbohydrate (g)	239.0	136.0-349.2	95.6-468.0
Total Protein (g)	66.0	39.4-100.8	22.7-128.4
Animal Protein (g)	44.3	21.7-75.0	10.7-112.6
Vegetable Protein (g)	21.7	12.6-31.4	7.8-42.2
Total Saturated Fatty Acids (g)	24.9	11.8-38.8	6.2-52.1
Total Monounsaturated Fatty Acids (g)	24.4	12.8-38.3	6.6-56.2
Total Polyunsaturated Fatty Acids (g)	17.1	7.0-20.6	4.6-50.9
Total Dietary Fiber (g)	13.0	6.2-20.6	3.6-26.1
Soluble Dietary Fiber (g)	4.2	2.0-6.6	0.9-10.7
Insoluble Dietary Fiber (g)	8.8	4.1-14.9	2.3-27.3
Water (g)	1578.9	846.3-2555.2	557.5-3642.6
% Calories From Fat	34.2	26.1-43.2	19.6-49.3
% Calories from Carbohydrate	51.1	39.7-61.1	29.8-68.9
% Calories from Protein	14.7	9.4-21.8	6.6-28.8
% Calories from SFA	11.8	8.3-16.0	6.2-19.6
% Calories from MUFA	11.5	8.3-15.6	6.7-19.3
% Calories from PUFA	8.0	4.7-12.9	3.5-18.2
Total Grains (ounce equivalents)	7.5	4.1-11.5	2.5-15.0
Whole Grains (ounce equivalents)	1.1	0.0-3.4	0.0-11.9
Refined Grains (ounce equivalents)	6.4	2.9-10	0.9-15.0

Table Note: Fatty Acid Ratio is the ratio of poly- and monounsaturated fatty acids to saturated fatty acids.

Figure 1: Direceted Acyclic Graph of relationship between serum PFAS concentrations, diet, and covariates

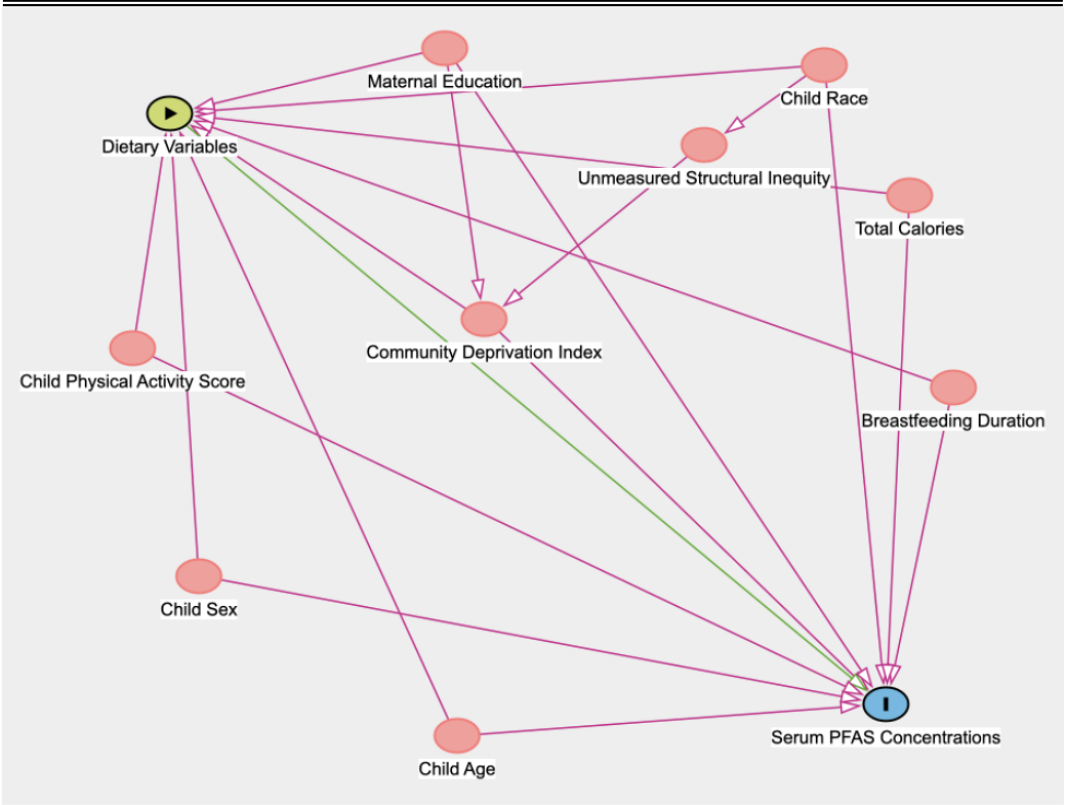

Figure 2: Unadjusted percent difference in serum PFAS concentration per standard deviation increase of nutritional variable

HEI Total and Component Scores	PFOA	PFOS	PFHxS	PFNA
Total Score				
Total Vegetables				
Greens and Beans				
Total Fruit				
Whole Fruit				
Whole Grains				
Dairy				
Total Protein Foods				
Seafood and Plant Proteins				
Fatty Acids				
Sodium				
Refined Grains				
Solid Fats, Alcohol and Added Sugars				

Dietary Composition Variables	PFOA	PFOS	PFHxS	PFNA
Total Grams (g)				
Energy (kcal)				
Total Fat (g)				
Total Carbohydrate (g)				
Total Protein (g)				
Animal Protein (g)				
Vegetable Protein (g)				
Total Saturated Fatty Acids (g)				
Total Monounsaturated Fatty Acids (g)				
Total Polyunsaturated Fatty Acids (g)				
Total Dietary Fiber (g)				
Soluble Dietary Fiber (g)				
Insoluble Dietary Fiber (g)				
Water (g)				
% Calories From Fat				
% Calories from Carbohydrate				
% Calories from Protein				
% Calories from SFA				
% Calories from MUFA				
% Calories from PUFA				
Total Grains (ounce equivalents)				
Whole Grains (ounce equivalents)				
Refined Grains (ounce equivalents)				

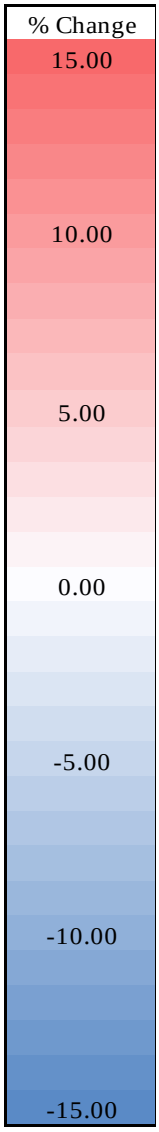

Figure Note: See Table S2 in supplementary material for further detail.

Figure 3: Adjusted percent difference in serum PFAS concentration per standard deviation increase of nutritional variable

HEI Total and Component Scores	PFOA	PFOS	PFHxS	PFNA
Total Score				
Total Vegetables				
Greens and Beans				
Total Fruit				
Whole Fruit				
Whole Grains				
Dairy				
Total Protein Foods				
Seafood and Plant Proteins				
Fatty Acids				
Sodium				
Refined Grains				
Solid Fats, Alcohol and Added Sugars				

Dietary Composition Variables	PFOA	PFOS	PFHxS	PFNA
Total Grams (g)				
Energy (kcal)				
Total Fat (g)				
Total Carbohydrate (g)				
Total Protein (g)				
Animal Protein (g)				
Vegatable Protein (g)				
Total Saturated Fatty Acids (g)				
Total Monounsaturated Fatty Acids (g)				
Total Polyunsaturated Fatty Acids (g)				
Total Dietary Fiber (g)				
Soluble Dietary Fiber (g)				
Insoluble Dietary Fiber (g)				
Water (g)				
% Calories From Fat				
% Calories from Carbohydrate				
% Calories from Protein				
% Calories from SFA				
% Calories from MUFA				
% Calories from PUFA				
Total Grains (ounce equivalents)				
Whole Grains (ounce equivalents)				
Refined Grains (ounce equivalents)				

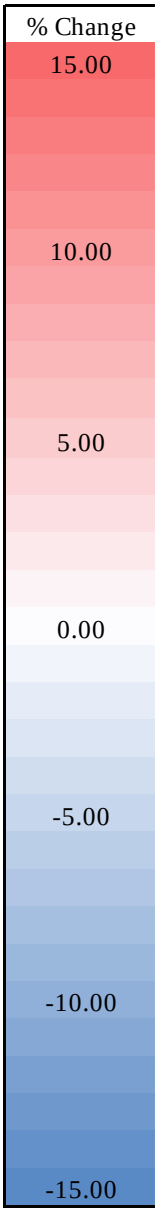

Figure Note: Adjusted for age (continuous), child sex (male, female), child race (black, white, other), physical activity score (continuous), community deprivation index (continuous), duration of breastfeeding (weeks), and mother's education (high school or less, some college/technical school, bacherlor's degree/graduate school). HEI and % Calories variables do not include total calories (continuous) as a covariate. All other variables do. See Table S3 in supplementary material for further detail.

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7. Supplementary Material

Table S1: Healthy Eating Index-2015 components, points values, and standards for scoring

Component	Maximum Points	Standard for maximum score	Standard for minimum score of zero
Total Vegetables	5	≥ 1.1 c equivalents/1,000 kcal	No vegetables
Greens and Beans	5	≥ 0.2 c equivalents/1,000 kcal	No dark green vegetables or beans and peas
Total Fruit	5	≥ 0.8 c equivalents/1,000 kcal	No fruit
Whole Fruit	5	≥ 0.4 c equivalents/1,000 kcal	No whole fruit
Whole Grains	10	≥ 1.5 oz equivalents/1,000 kcal	No whole grains
Dairy	10	≥ 1.3 c equivalents/1,000 kcal	No dairy
Total Protein Foods	5	≥ 2.5 oz equivalents/1,000 kcal	No protein foods
Seafood and Plant Proteins	5	≥ 0.8 c equivalents/1,000 kcal	No seafood or plant proteins
Fatty Acid Ratio	10	(PUFAs + MUFAs)/SFAs ≥ 2.5	(PUFAs + MUFAs)/SFAs ≤ 1.2
Sodium	10	≤ 1.1 g/1,000 kcal	≥ 2.0 g/1,000 kcal
Refined Grains	10	≤ 1.8 oz equivalents/1,000 kcal	≥ 4.3 oz equivalents/1,000 kcal
Added Sugars	10	≤ 6.5% of energy	≥ 26% of energy
Saturated Fats	10	≤ 8% of energy	≥ 16% of energy

Table Note: In this study, added sugars and saturated fats scores were summed together.

Table S2: Unadjusted percent difference in serum PFAS concentrations with each standard deviation increase in nutritional variable

	PFOA	**PFOS**	**PFHxS**	**PFNA**
Variable	% Difference (95% CI)	% Difference (95% CI)	% Difference (95% CI)	% Difference (95% CI)
Total Score	8 (-2, 19)	5 (-5, 16)	6 (-4, 17)	4 (-5, 15)
Total Vegetables	2 (-7, 13)	4 (-6, 15)	3 (-7, 14)	7 (-3, 18)
Greens and Beans	4 (-5, 15)	10 (0, 21)	5 (-5, 16)	6 (-4, 17)
Total Fruit	7 (-3, 18)	3 (-7, 14)	6 (-4, 16)	0 (-10, 10)
Whole Fruit	7 (-3, 18)	6 (-4, 17)	7 (-3, 17)	-1 (-10, 10)
Whole Grains	9 (-1, 20)	3 (-6, 14)	0 (-9, 10)	10 (-1, 21)
Dairy	14 (4, 26)	15 (4, 27)	12 (1, 23)	11 (1, 23)
Total Protein Foods	-4 (-13, 6)	-2 (-11, 8)	-3 (-12, 7)	-3 (-12, 7)
Seafood and Plant Proteins	7 (-3, 18)	8 (-2, 19)	8 (-2, 19)	-1 (-10, 10)
Fatty Acid Ratio	-10 (-19, -1)	-11 (-19, -2)	-6 (-15, 4)	-8 (-16, 2)
Sodium	4 (-5, 15)	1 (-8, 12)	3 (-7, 13)	2 (-8, 12)
Refined Grains	1 (-8, 12)	-4 (-13, 6)	-6 (-15, 4)	-1 (-10, 9)
Solid Fats, Alcohol and Added Sugars	3 (-6, 14)	3 (-7, 13)	6 (-4, 17)	1 (-8, 12)
Variable				
Total Grams (g)	-2 (-11, 8)	-6 (-15, 3)	-1 (-10, 9)	-3 (-12, 7)
Energy (kcal)	-1 (-10, 9)	-7 (-16, 2)	-12 (-20, -3)	-5 (-14, 5)
Total Fat (g)	-2 (-11, 8)	-7 (-15, 3)	-12 (-20, -3)	-5 (-14, 5)
Total Carbohydrate (g)	0 (-10, 10)	-8 (-16, 2)	-11 (-19, -2)	-5 (-14, 5)
Total Protein (g)	1 (-8, 12)	0 (-9, 11)	-5 (-13, 5)	-1 (-10, 9)
Animal Protein (g)	-1 (-10, 9)	0 (-9, 10)	-4 (-13, 6)	-2 (-11, 9)
Vegetable Protein (g)	6 (-4, 17)	1 (-9, 11)	-3(-12, 7)	1 (-8, 12)
Total Saturated Fatty Acids (g)	3 (-7, 13)	0 (-9, 11)	-8 (-16, 2)	1 (-8, 12)
Total Monounsaturated Fatty Acids (g)	-4 (-13, 6)	-9 (-17, 0)	-11 (-19, -2)	-6 (-15, 3)
Total Polyunsaturated Fatty Acids (g)	-5 (-14, 5)	-10 (-18, 0)	-13 (-21, -4)	-8 (-16, 2)
Total Dietary Fiber (g)	5 (-4, 16)	0 (-10, 10)	-3 (-12, 7)	1 (-8, 12)
Soluble Dietary Fiber (g)	7 (-3, 18)	1 (-8, 11)	-3 (-12, 7)	1 (-8, 11)
Insoluble Dietary Fiber (g)	4 (-6, 15)	-1 (-10, 9)	-3 (-13, 6)	1 (-8, 12)
Water (g)	-2 (-11, 8)	-6 (-14, 4)	1 (-8, 11)	-3 (-12, 8)
% Calories From Fat	-2 (-12, 8)	-2 (-12, 8)	-6 (-15, 3)	-1 (-10, 9)
% Calories from Carbohydrate	2 (-8, 12)	-2 (-11, 8)	1 (-8, 11)	-1 (-10, 9)
% Calories from Protein	1 (-9, 11)	8 (-2, 19)	8 (-2, 19)	4 (-6, 14)
% Calories from SFA	6 (-4, 17)	9 (-1, 21)	1 (-8, 11)	8 (-2, 19)
% Calories from MUFA	-5 (-14, 5)	-7 (-16, 2)	-5 (-14, 4)	-4 (-13, 5)
% Calories from PUFA	-6 (-15, 4)	-8 (-16, 2)	-8 (-17, 1)	-5 (-14, 5)
Total Grains (ounce equivalents)	2 (-7, 13)	-2 (-11, 8)	-6 (-15, 3)	-2 (-11, 8)
Whole Grains (ounce equivalents)	6 (-4, 17)	-2 (-11, 8)	-5 (-13, 5)	3 (-7, 13)
Refined Grains (ounce equivalents)	-1 (-10, 9)	-1 (-10, 9)	-4 (-13, 6)	-3 (-12, 7)

Table Note: HEI scores are based on quantities consumed per 1,000 kcal rather than total amounts of food consumed.

Table S3: Adjusted percent difference in serum PFAS concentrations with each standard deviation increase in nutritional variable

	PFOA	**PFOS**	**PFHxS**	**PFNA**
Variable	% Difference (95% CI)	% Difference (95% CI)	% Difference (95% CI)	% Difference (95% CI)
Total Score	-7 (-15, 2)	-6 (-15, 4)	-2 (-11, 9)	-9 (-17, 0)
Total Vegetables	-3 (-11, 6)	0 (-9, 10)	2 (-8, 12)	1 (-7, 11)
Greens and Beans	-1 (-10, 8)	7 (-2, 18)	3 (-7, 13)	2 (-6, 12)
Total Fruit	-3 (-12, 6)	-7 (-15, 3)	-2 (-11, 9)	-11 (-19, -3)
Whole Fruit	-10 (-18, -1)	-7 (-16, 3)	-5 (-15, 5)	-16 (-23, -7)
Whole Grains	1 (-7, 10)	-2 (-11, 8)	-5 (-14, 5)	5 (-4, 15)
Dairy	1 (-8, 11)	6 (-4, 17)	4 (-6, 16)	-1 (-10, 9)
Total Protein Foods	-4 (-12, 5)	-2 (-11, 8)	-3 (-12, 7)	0 (-9, 9)
Seafood and Plant Proteins	-4 (-12, 5)	1 (-8, 11)	2 (-8, 13)	-9 (-17, 0)
Fatty Acid Ratio	-5 (-13, 4)	-8 (-16, 2)	-1 (-10, 10)	-3 (-12, 6)
Sodium	-2 (-10, 7)	-5 (-13, 5)	-1 (-11, 9)	-5 (-13, 5)
Refined Grains	-2 (-10, 7)	-7 (-15, 3)	-7 (-16, 2)	-5 (-14, 4)
Solid Fats, Alcohol and Added Sugars	-5 (-13, 4)	-3 (-12, 7)	3 (-7, 14)	-6 (-14, 3)
Variable				
Total Grams (g)	-11 (-20, -2)	-7 (-17, 4)	5 (-7, 18)	-5 (-15, 6)
Energy (kcal)	-3 (-11, 6)	-6 (-15, 4)	-12 (-20, -3)	-6 (-14, 3)
Total Fat (g)	-1 (-17, 20)	1 (-17, 24)	-4 (-22, 18)	0 (-17, 21)
Total Carbohydrate (g)	4 (-14, 25)	-4 (-22, 18)	0 (-19, 23)	0 (-17, 22)
Total Protein (g)	-6 (-17, 6)	2 (-11, 16)	3 (10, 18)	-2 (-14, 11)
Animal Protein (g)	-4 (-13, 6)	1 (-10, 12)	1 (-10, 13)	-1 (-11, 10)
Vegetable Protein (g)	-2 (-13, 11)	4 (-9, 19)	4 (-9, 20)	-2 (-14, 11)
Total Saturated Fatty Acids (g)	4 (-10, 20)	11 (-5, 30)	-1 (-15, 17)	6 (-9, 23)
Total Monounsaturated Fatty Acids (g)	-6 (-19, 9)	-7 (-21, 9)	-2 (-17, 16)	-5 (-18, 11)
Total Polyunsaturated Fatty Acids (g)	1 (-11, 14)	-3 (-15, 11)	-3 (-15, 12)	0 (-12, 13)
Total Dietary Fiber (g)	-9 (-18, 1)	-6 (-17, 5)	-5 (-15, 7)	-10 (-19, 1)
Soluble Dietary Fiber (g)	-3 (-14, 9)	-3 (-14, 10)	-2 (-13, 12)	-10 (-20, 2)
Insoluble Dietary Fiber (g)	-9 (-18, 0)	-6 (-16, 4)	-5 (-15, 6)	-8 (-17, 2)
Water (g)	-10 (-19, -1)	-6 (-16, 4)	5 (-6, 17)	-4 (-13, 6)
% Calories From Fat	1 (-7, 10)	0 (-9, 10)	-4 (-13, 5)	1 (-8, 11)
% Calories from Carbohydrate	1 (-7, 11)	-2 (-11, 8)	0 (-9, 11)	-1 (-10, 8)
% Calories from Protein	-4 (-12, 5)	3 (-6, 14)	6 (-4, 17)	0 (-8, 10)
% Calories from SFA	3 (-5, 12)	7 (-3, 17)	-2 (-11, 8)	4 (-5, 14)
% Calories from MUFA	-2 (-10, 7)	-5 (-14, 4)	-3 (-13, 7)	-3 (-11, 7)
% Calories from PUFA	0 (-8, 10)	-3 (-12, 7)	-4 (-13, 6)	0 (-9, 10)
Total Grains (ounce equivalents)	-2 (-13, 11)	5 (-9, 20)	1 (-12, 16)	3 (-9, 17)
Whole Grains (ounce equivalents)	-2 (-10, 7)	-4 (-13, 6)	-6 (-15, 4)	2 (-7, 12)
Refined Grains (ounce equivalents)	0 (-11, 12)	8 (-5, 23)	6 (-7, 21)	2 (-10, 15)

Table Note: Adjusted for age (continuous), child sex (male, female), child race (black, white, other), physical activity score (continuous), community deprivation index (continuous), duration of breastfeeding (weeks), and mother's education (high school or less, some college/technical school, bacherlor's degree/graduate school). HEI and % Calories variables do not include total calories (continuous) as a covariate. All other variables do.