

**ASSESSING POTENTIAL EXPOSURE TO PER- AND
POLYFLUOROALKYL SUBSTANCES (PFAS) IN PRODUCE
AND DRINKING WATER IN CHATHAM COUNTY, NC**

by

Yang (Leon) Li

Dr. Heather M. Stapleton, Advisor

April 30, 2021

**Masters project submitted in partial fulfillment of the
requirements for the Master of Environmental Management degree in the
Nicholas School of the Environment of
Duke University**

Executive Summary

Diet constitutes a major human exposure pathway for per- and polyfluoroalkyl substances (PFAS) due to the contamination of drinking water supplies, and their use in food packaging, and accumulation in the food web. Significant PFAS levels have recently been reported in groundwater (Haw River and Cape Fear River) in North Carolina. This has raised concerns for potential exposure for communities consuming drinking water sourced from these rivers and produce grown from lands irrigated with this water. This study sought to evaluate dietary exposure to PFAS from consumption of produce (lettuce, potato and tomato) and drinking water in Chatham County, North Carolina, a previously reported PFAS impacted area. A total of 18 produce samples were collected in local farmer markets and grocery stores. Drinking water PFAS data (N = 40) were abstracted from an ongoing study in Pittsboro, NC collected and analyzed in 2019 and 2020.

PFAS were generally not detected in the produce samples analyzed here, with the exception of perfluorodecanoic acid (PFDA). PFDA was detected in potatoes and tomatoes, ranging from 0.11 to 1.11 ng/g, or parts per billion (ppb). Total PFAS were measured at concentrations ranging from 26.4 ng/L up to 458.1 ng/L in the drinking water samples.

Using the median values of PFDA measured in produce and estimates of produce consumption in the general population (using the 50th and 95th percentiles), exposure to PFDA was estimated. Estimated exposure was highest from potato consumption (median exposure intake varies between 0.42 and 1.40 ng/kg-day). In drinking water, short-chain (<8 carbon) perfluoroalkyl carboxylate acids (PFCA) contributed the most to \sum PFAS exposure. The median exposure intake was 1.40 ng/kg-day for PFHxA and 1.17 ng/kg-day for PFPeA. Higher exposure was generally observed via drinking water compared to produce, and exposures were the highest for young children and decreased with age.

The estimated hazard index suggests that a small portion of the population (~5%) could be at increased risk for adverse effects via produce exposure (in young children) and for all age groups via drinking water exposure.

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Introduction

Per- and polyfluoroalkyl substances (PFASs) are a large class of synthetic chemicals, consisting of a fully or partially hydrophobic alkyl chain and a hydrophilic functional group (Buck et al., 2011). A detailed classification of PFAS classes can be seen in Figure 1. In general, most polymer containing PFASs (fluoropolymers) are less of an environmental and ecological concern due to their reduced solubility and bioavailability (Henry et al., 2018). Currently, the focus of regulatory action and research regarding environmental contamination and adverse health effects are limited to non-polymeric PFASs, especially perfluoroalkyl acids (PFAAs). This is because 1) fluoropolymers and many polyfluoroalkyl substances can be degraded or metabolized to terminal PFAAs (Li et al., 2018; Washington et al., 2018); and 2) PFAAs are chemically stable and highly persistent under normal environmental conditions due to their strong covalent C-F bond. In addition, their water- and grease-repelling properties are popular in retail, and as a consequence, are widely manufactured as surfactants and polymers in fire-fighting foam, paints, food packaging, inks and other consumer products (Banks et al., 1994; Buck et al., 2011; OECD 2018). To date, two major groups of PFAAs have been widely applied in commercial products: perfluoroalkyl carboxylic acids (PFCAs) and perfluoroalkane sulfonic acids (PFASs). In particular, the eight-carbon PFAAs, including perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA), have been of most ecological and human health concern (USEPA 2016).

PFASs have been ubiquitously detected in the environment, biota and human plasma samples since large-scale production began in the 1950s (Giesy and Kannan, 2001; Kato 2015). Certain PFASs have been found in ambient air (Barton et al., 2006), groundwater (USEPA, 2017), surface water (Sun et al., 2016), soil and sediment (Zhu et al., 2019). In the United States, PFASs, especially PFOS and PFOA, are ubiquitously detected in human serum (Kato et al., 2011) and elevated levels are commonly found in drinking water sources (Hu et al., 2016). Such widespread contamination and concern have led to a phase-out of both PFOS and PFOA. However, short-chain PFAAs and other alternatives are increasingly found in the environment, humans and wildlife, which are less studied. In addition, the “background” concentrations might vary greatly depending on proximity to industry and manufacturing plants, patterns of water dispersion and other factors. For example, higher levels of PFASs are often detected in urban

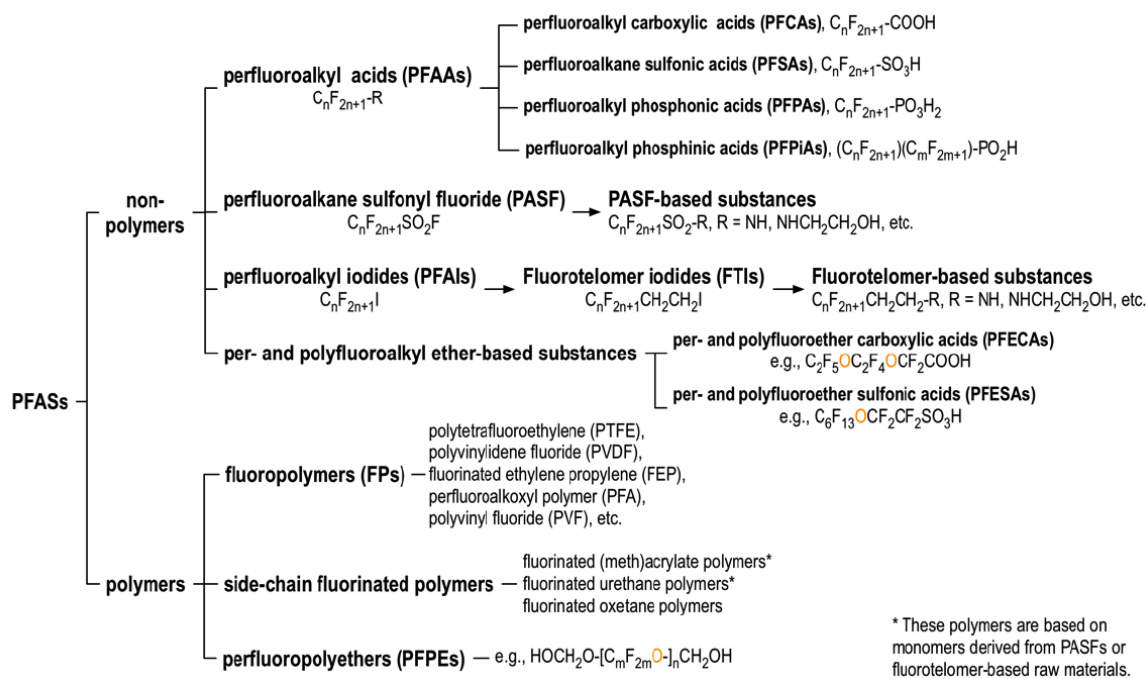


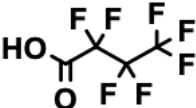
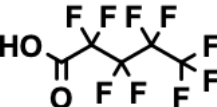

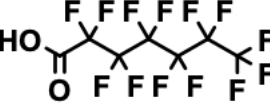



Figure 1. Classification and terminology of PFAS family (From Buck et al. 2011; OECD, 2018).

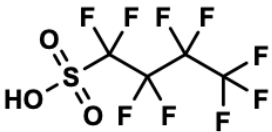


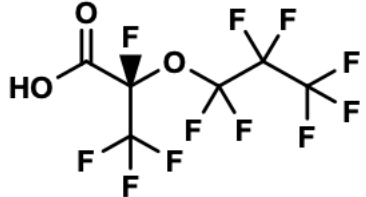
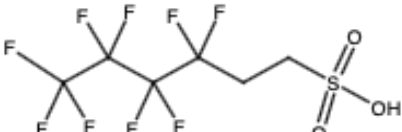

and industrial areas compared to rural and less populated regions (Kannan et al., 2004).

Therefore, identification of contaminated sites and sources is of great importance for exposure and risk assessment of PFASs.

Currently, PFOS and PFOA have been more extensively studied for their toxicity, though certain data and information are also available for other PFAAs. Table 1 lists the properties and toxic effects of the most commonly studied PFASs. Toxicokinetic studies have revealed that PFASs have great potential to bind to proteins, including serum albumin and fatty acid binding proteins (Zhao et al., 2015). As a result, they are not readily metabolized and are less likely to be excreted in urine, leading to long half-lives in the human body (EFSA 2018). In general, PFASs are likely to be transferred and stored in the liver, kidney and blood compartments (Bischel et al., 2011). During pregnancy, these chemicals are likely to cross the placental barrier, although studies have shown that maternal serum levels are often higher than those reaching the fetus (Lau et al. 2003). However, it should be noted that bioaccumulation and half-lives of PFASs vary based on their carbon-chain length and functional groups, and differences will also be observed based on

Table 1. A list of the 13 target PFASs compounds with chemical properties and toxic effects in this study.

Compound	Structure	BCF	Human $t_{1/2}$		Toxic effects
			Male	Female	
PFBA Perfluorobutanoic acid Formula: $C_4HF_7O_2$		-	72 h ^a	87 h ^a	-
PFPeA Perfluoropentanoic acid Formula: $C_5HF_9O_2$		-	-	-	-
PFHxA Perfluorohexanoic acid Formula: $C_6HF_{11}O_2$		-	32 d ^b	-	-
PFHpA Perfluoroheptanoic acid Formula: $C_7HF_{13}O_2$		-	1.5 y ^c	-	-
PFOA Perfluorooctanoic acid Formula: $C_8HF_{15}O_2$		2.28 ^d	4.6 y ^e	3.10 y ^e	Carcinogenicity Potential; Reproductive; Endocrine Disruption Potential ^e
PFNA Perfluorononanoic acid Formula: $C_9HF_{17}O_2$		-	12 y ^f	-	Endocrine Disruption Potential ^e
PFDA Perfluorodecanoic acid Formula: $C_{10}HF_{19}O_2$		4.93 ^d	4.3 y ^e	-	Endocrine Disruption Potential ^e

Compound	Structure	BCF	Human $t_{1/2}$		Toxic effects
			Male	Female	
PFBS Perfluorobutane sulfonic acid Formula: $C_4HF_9O_3S$		-	26 d ^f		-
PFHxS Perfluorohexane sulfonic acid Formula: $C_6HF_{13}O_3S$		-	7.4 y ^e	4.7 y ^e	-
PFOS Perfluorooctane sulfonic acid Formula: $C_8HF_{17}O_3S$		3.44 ^d	4.6 y ^e	3.1 y ^e	Reproductive; Developmental; Endocrine Disruption Potential ^e
GenX (HFPO-DA) Hexafluoropropylene oxide dimer acid Formula: $C_6HF_{11}O_3$		-	-	-	-
4:2 FTS 4:2 fluorotelomer sulfonic acid Formula: $C_6H_5F_9O_3S$		-	-	-	-
6:2 FTS 6:2 fluorotelomer sulfonic acid Formula: $C_8H_5F_{13}O_3S$		-	-	-	-

Note: BCF indicates bioconcentration factor; $t_{1/2}$ indicates half-life; K_{ow} indicates octanol/water partition coefficient; h-hour, d-day, y-year; – indicates that data are not available; a: Chang et al. (2008)); b: Russell, Nilsson, and Buck (2013); c: U.S. EPA ToxCast & Tox21 (2020); d: Li et al. (2018); f: Zhang, Beesoon, et al. (2013); g: Olsen et al. (2009)

species and sex. In general, humans tend to have longer half-lives for PFASs than other mammalian, ranging from a couple days to months for short-chain PFAAs and several years for long-chain PFAAs. In terms of toxicity, studies from laboratory animal and epidemiological studies reported that chronic exposure to PFOS and PFOA, even at low environmental levels, might lead to adverse health effects (EFSA 2018). For example, *in vivo* studies have associated PFASs exposure with hepatotoxic, reproductive, developmental and endocrine disrupting outcomes (Coperchini et al., 2017; Vetvicka et al., 2013; Viberg et al., 2013; Vieira et al., 2013). However, differences in relative potencies among PFASs have been observed. For example, there are differences in the lowest observed effect levels (LOAELs) and the no observed effect levels (NOAELs) among different toxic endpoints (EFSA, 2018). Overall, exposure to PFOS and PFOA have been associated with increased liver enzymes (Gleason, Post and Fagliano, 2015), decreased fetal growth (Johnson et al., 2014), and decreased vaccine response (NTP 2016) in human studies, many of which are consistent with trends observed in animal studies. However, it should be noted that for some health endpoints, there are less consistent results between animal studies and epidemiological studies. For example, exposure to PFOS and PFOA have been negatively associated with blood cholesterol and triglyceride levels in rodents, while positive associations are observed in humans (Wang et al., 2013; Zhang et al., 2013).

Humans are exposed to PFASs via a wide range of pathways. Consumption of contaminated food and drinking water, ingestion of contaminated indoor air, soil and house dust are likely important sources of exposure. Studies have suggested that drinking water and dietary intake are the two major contributors for human exposure to PFAS (Haug et al., 2011; Domingo et al., 2012). According to the European Food Safety Authority, fish and seafood contribute up to 86% of PFOS exposure in European population, followed by meat, eggs and dairy products. Regarding PFOA, drinking water and seafood account for the largest contribution to chronic exposure (EFSA, 2018). In the United States, PFASs contamination in drinking water has been actively investigated and the development of enforceable Maximum Contaminant Levels (MCLs) for drinking water is underway. The first drinking water contamination by PFASs was identified near a PFASs production facility in Washington in 1999. The mean concentration of PFOA was found to be in the range of 1500-7200 ppt.

Exposure to PFASs through dietary intake is not regulated in the United States (Blaine et al., 2014; Sznajder-Katarzyńska et al., 2019); however, diet is expected to contribute significantly to PFASs exposure. Overall, the bioaccumulation potential of PFAS in terrestrial food chains is influenced by 1) chemical functional group and chain length, 2) levels in the irrigation water and soil/biosolids, and 3) organic carbon content. PFASs bioaccumulation in produce largely depends on the soil-water distribution coefficients (K_D) or bioaccumulation factors (BAF). Gebbink et al. (2016) indicated that the BAF for Perfluoroalkyl sulfonic acids (PFSA) from water to Baltic Herring (*Clupea harengus*) increase from 3.3 to 4.1 between 6 to 8 alkyl units. However, the bioaccumulation process is more complicated for terrestrial systems. The study of Blaine et al. (2014) suggests contradictory results; decreased BAFs are associated with increasing chain length in tomato and pear plants. In addition, soil may act as a secondary depot for PFAS and contaminate plants. In the United States, a large amount of sewage sludge containing PFASs from wastewater treatment plants is applied as fertilizer/biosolids in agriculture (Venkatesan et al., 2013). According to U.S EPA National Sewage Sludge Survey, PFASs loading in biosolids ranges from 2749 to 3450 kg/year. Plants grown in contaminated soil are expected to accumulate PFASs through their root systems, and translocate to stems, shoots, leaves and fruiting compartments (Blaine et al., 2014). Studies suggest that low lipophilic, neutral and ionized polar chemicals are more likely to accumulate in the leaves and other parts of the plant. Furthermore, PFASs tend to bind to organic matter in the soil, resulting in reduced bioavailability (Blaine et al., 2014). For example, lettuce grown in 6% organic carbon content was found to have the lowest BAFs of PFAAs compared to 0.4% and 2% organic carbon groups, with a 0.4 to 0.6 log decrease per alkyl bond. Lastly, PFAS can accumulate in livestock and their derived food including milk, meat and eggs via food web (Wen et al., 2016).

PFASs contamination in the Cape Fear River Watershed has been a great concern recently in North Carolina. Elevated levels of legacy PFAS (i.e. PFAAs) have been consistently detected in the Haw River near Pittsboro, NC. Such elevation is assumed to be associated with upstream discharge of PFASs from wastewater treatment plants or other sources. In addition to these legacy PFAAs, other PFASs such as perfluoro-2-propoxypropanoic acid (PFPrOPrA also known as GenX), have been found in the Cape Fear River downstream of the Chemours manufacturing facility (Sun et al., 2016). In general, PFASs levels vary depending on the distance to the point of

release and source concentration. A research study has shown that near contaminated sites, PFAS concentrations in drinking water were 100 times larger than the EPA advisory guideline and account for 75% of total PFAS exposure (Hu et al., 2019). But data regarding PFAS contamination in locally grown produce that may be using contaminated irrigation water and soil is not available. Therefore, the determination of PFAS exposure and associated risks in drinking water and produce within North Carolina are critically needed. The aims of this project are to (1) collect and analyze local produce, including vegetables and eggs, for PFAS; (2) to estimate human exposure to PFASs via dietary consumption of contaminated produce and drinking water in the Pittsboro community; and (3) to assess corresponding risks from PFAS exposure using the EPA hazard quotient and index framework.

Methods

Data on PFASs levels in drinking water from Pittsboro, NC were collected as part of an ongoing exposure study in Dr. Heather Stapleton's laboratory (<https://sites.nicholas.duke.edu/pfas>). Drinking water samples were collected by research participants from their homes in November of 2019 through February of 2020. Detailed information regarding the method used for the laboratory analysis of the water samples can be found in Herkert et al (2020). For the purpose of this analysis, data from 50 tap water samples collected in November of 2019 from residents of Pittsboro, NC were included to estimate drinking water exposure and for the subsequent risk assessment of PFASs. All research participants in this study provided informed consent and all research protocols were approved by the Duke University Institutional Review Board.

Produce collection

Produce samples (n=18) were collected in local farmer's markets or grocery stores in Chatham County in North Carolina between September 26 and December 1, 2020. Three major local farmer markets (Chatham Mills Farmers' Market, Fearington Farmer's Market and Pittsboro Farmer's Market) were sampled. For each farmer's market, three classes of produce (lettuce, potato and tomato), each with conventional and organic types, were purchased. Produce was only considered organic based on the presence of an organic certified label granted by United States Department of Agriculture (USDA). For each subtype of produce, more than 2 samples were required to compose a pool. Samples that were not able to be collected in farmer's markets (n=5)

Table 3. Summary of samples collected for each type of produce.

No	Produce	Class	Type	Source	Origin	Collection Date
L1	Lettuce	Leaf	Conventional	Farm	Chatham Mills Farmer Market	9-26-2020
L2	Lettuce	Leaf	Conventional	Farm	Chatham Mills Farmer Market	9-26-2020
L3	Lettuce	Leaf	Conventional	Farm	Ferrington Farmers' Market	10-6-2020
L4	Lettuce	Leaf	Organic	Farm	Chatham Mills Farmer Market	10-10-2020
L5	Lettuce	Leaf	Organic	Farm	Ferrington Farmers' Market	10-6-2020
L6	Lettuce	Leaf	Organic	Store	Harris Teeter	10-21-2020
P1	Potato	Tuber	Conventional	Farm	Pittsboro Farmer's Market	10-8-2020
P2	Potato	Tuber	Conventional	Farm	Chatham Mills Farmer Market	10-10-2020
P3	Potato	Tuber	Conventional	Farm	Ferrington Farmers' Market	10-13-2020
P4	Potato	Tuber	Conventional	Store	Harris Teeter	10-21-2020
P5	Potato	Tuber	Organic	Store	Harris Teeter	10-21-2020
P6	Potato	Tuber	Organic	Store	Food Lion	12-1-2020
T1	Tomato	Fruit	Conventional	Farm	Pittsboro Farmer's Market	9-24-2020
T2	Tomato	Fruit	Conventional	Farm	Chatham Mills Farmer Market	9-26-2020
T3	Tomato	Fruit	Conventional	Farm	Ferrington Farmers' Market	10-6-2020
T4	Tomato	Fruit	Organic	Farm	Ferrington Farmers' Market	10-6-2020
T5	Tomato	Fruit	Organic	Store	Harris Teeter	10-21-2020
T6	Tomato	Fruit	Organic	Farm	Chatham Mills Farmer Market	10-10-2020

were instead collected from two major local grocery stores (Harris Teeter and Food Lion).

Detailed information for the samples included in this study is listed in Table 3. Samples were all stored at 4°C until homogenization (within 1 week of receipt).

Laboratory methods

Homogenization From each produce item, non-edible parts were removed. Potato samples were left unpeeled for analysis. The edible parts were rinsed with deionized water and homogenized in a pre-cleaned blender. Homogenates were transferred to clean polypropylene jars and stored at -20°C before extraction.

Extraction Homogenized samples were extracted using the protocol from Martin et al. (2016) and Reiner et al. (2009). The mass of each sample (approximately 1.0 g) was first recorded and then the sample was transferred to a pre-labeled polypropylene centrifuge tube. All samples were spiked with an isotopically labelled mixture of PFAS, including GenX [2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-¹³C₃-propanoic acid], 6:2 FTS (sodium 1H, 1H, 2H, 2H-

perfluoro-1-[1,2-¹³C₂]-octane sulfonate) and a mix of isotopically labelled PFAAs from Wellington Laboratories (MPFAC-MXA). This mixture includes PFBA (Perfluoro-n-[1,2,3,4-¹³C₄]butanoic acid), PFPeA (Perfluoro-n-[1,2,3,4,5-¹³C₅]pentanoic acid), PFHxA (Perfluoro-n-[1,2-¹³C₂]hexanoic acid), PFHpA (Perfluoro-n-[1,2,3,4-¹³C₄]heptanoic acid), PFOA (Perfluoro-n-[1,2,3,4-¹³C₄]octanoic acid), PFNA (Perfluoro-n-[1,2,3,4,5-¹³C₅]nonanoic acid), PFDA (Perfluoro-n-[1,2-¹³C₂]decanoic acid), PFBS (Sodium perfluoro-1-[2,3,4-¹³C₃]butane sulfonate), PFHxS (Sodium perfluoro-1-hexane[¹⁸O₂]sulfonate), and PFOS (Sodium perfluoro-1 [1,2,3,4-¹³C₄] octanesulfonate). All standards were purchased from Wellington Laboratories (Guelph, Ontario) and had a purity greater than 95%.

After spiking in the isotopically-labelled standards, 1.0 mL of formic acid (1 M) was added to foster deproteination of the samples. Then 10 mL acetonitrile (chemical grade) was added and samples were vortexed and then centrifuged for 5 min at 3000 rpm using an Eppendorf 5810-R centrifuge. The supernatant was transferred to a clean and labeled polypropylene centrifuge tube. The above step was repeated twice to collect 30 mL of supernatant. After centrifugation, samples were concentrated to dryness using a nitrogen evaporation system. Samples were then reconstituted in 1.0 of methanol.

Extracts were purified using a Solid-Phase Extraction (SPE) cartridge attached to a vacuum manifold. Oasis WAX SPE cartridges (6cc cartridge 500mg 60µm) were used for this analysis. The SPE cartridges were first conditioned with 6 mL 0.1% NH₄OH in MeOH, 6 mL methanol and 6 mL of a sodium acetate (NaOAc) (aq) buffer. Then the 1.0 mL sample was transferred to the SPE column and the cartridges were washed with 6 mL NaOAc (aq) buffer followed by 6 mL methanol. Finally, samples were eluted with 6 mL 0.1% NH₄OH in MeOH.

Following SPE analysis samples were concentrated to 0.3 mL using a nitrogen evaporation system and 0.5 mL of ammonium acetate in water (2mM) was added. Samples were filtered to remove any particulates using a Mini-UniPrep™ Nylon Filter (pore size 0.2 µm). Samples were then spiked with an isotopically labelled PFOA (Perfluoro-n-[1,2-¹³C₂]octanoic acid) and PFOS (Sodium perfluoro-p¹³C₈)octane sulfonate) standard and stored in a -20°C freezer until instrument analysis.

HPLC-MS/MS PFAS compounds were analyzed using an Agilent 1260 Infinity II high-performance liquid chromatograph (HPLC) instrument coupled to an Agilent 6460A triple quadrupole mass spectrometer. The mass spectrometer was operated in negative electrospray ionization mode (HPLC-ESI-MS/MS). Separation of analytes by LC was performed using a 4.6 mm (I.D.) x 50 mm Agilent ZORBAX Eclipse XDB-C18 reversed-phase HPLC column (1.8 μ m particle size) preceded by a 4.6 mm x 5 mm XDB-C18 guard cartridge.

Mobile phases were 2 mM ammonium acetate in water (mobile phase A) and 2 mM ammonium acetate in methanol (mobile phase B) using a flow rate of 0.4 mL/min. Gradient conditions for chromatographic separation were as follows: initial condition (30% B) was increased to 60% B over 1.5 minutes; then increased to 95% B over 2 minutes and held for 5.5 minutes; then increased to 100% B over 3 minutes, returned to initial condition (30% B) over 0.5 minutes, and held for 5.5 minutes. The column temperature was 45°C and the injection volume was 20 μ L. Data were acquired under multiple reaction monitoring (MRM) transitions using optimized parameters.

Quality assurance and control Laboratory blanks (n=9) were prepared and processed with every batch of samples. Method detection limits (MDL) were calculated for each batch of samples using three times the standard deviation of laboratory blanks. MDLs ranged from <0.01 to 3.24 ng/g among the batches (Table S1). Averaged recoveries for labelled PFASs were 53% (22% to 108%) in blanks and 67% (16% to 118%) in samples. Matrix effects can be determined by comparing recoveries between samples and blanks (Table S2). Accuracy of the method was determined by analyzing a Standard Reference Material (SRM 1947, Lake Michigan Fish Tissue, Gaithersburg, MD) in triplicate. PFOS is the only PFAS certified in SRM 1947 and measurements were 81% (75% to 85%) of the certified value.

Statistical analysis All statistical analyses were performed using R Studio (3.6.2 version). Random variables between 0 and one-half the MDL were assigned to samples' values detected below MDL. Wilcoxon rank test (Wilcoxon-Mann Whitney test) was used to compare PFAS levels among different classes of produce and between conventional and organic groups. A p-value <0.05 was considered statistically significant.

Table 4. Summary of age-specific consumption factors for vegetables (lettuce, potato and tomato) and drinking water based on the median and 95th percentile values. Data were extracted from U.S. EPA Exposure Factor Handbook (2011).

Percentile	Lettuce (g/kg-day)		Potato (g/kg-day)		Tomato (g/kg-day)		Tap Water (mL/kg-day)		
	P50	P95	P50	P95	P50	P95	P50	P95	
Age group							Age group		
< 1	0.26	0.71	2.41	12.27	1.33	5.19	<1	43.5	126.5
1 to 2	0.54	2.51	2.89	13.52	1.66	6.29	1 to 3	46.8	101.6
3 to 5	0.62	2.70	2.36	7.93	1.67	9.16	4 to 6	37.9	81.1
6 to 10	0.49	1.45	1.78	5.72	1.16	4.90	7 to 10	26.9	55.2
11 to 15	0.40	2.07	1.06	4.38	0.78	3.14	11 to <14	20.2	41.9
16 to 20	0.39	1.54	0.92	3.23	0.72	3.65	15 to 19	16.4	35
21 to 49	0.41	1.43	0.86	3.57	0.74	3.30	20 to 44	18.6	38.4
50+	0.43	1.41	0.90	3.61	0.67	3.14	45 to 64	22	42.1
							65+	21.8	40

Daily dietary exposure intake

Daily dietary exposure to individual PFAS was estimated using human exposure factors. These exposure factors, including age-specific body weight, age-specific vegetable and drinking water ingestion amount and other parameters, were taken from the EPA Exposure Factors Handbook (USEPA, 2011). Detailed information on these parameters is listed in Table 4. Estimated exposure intake was calculated based on equation (1):

$$EI \left(\frac{\text{ng}}{\text{kg}} \right) = \frac{C * (BW * I)}{BW} \quad (\text{eq-1})$$

where

EI = distribution of daily dietary exposure intake (ng/kg-day);

C = PFAS concentrations for each produce (ng/g for food; ng/L for drinking water);

BW = age-specific body weight (kg);

I = Age-specific averaged ingestion rate (g/kg-day for food; mL/kg-day for drinking water).

Hazard analysis

The hazard quotient (HQ) for exposure to individual PFASs were calculated using equation (2):

$$HQ = \frac{EI}{RfD} \quad (\text{eq-2})$$

Table 5. Summary of human health toxicity reference doses (ng/kg-day) for PFAS reported for various US states and the U.S. EPA.

	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX
EPA ^a					20			2000		20	
CA ^b					0.45					1.8	
MA ^c			5		5	5	5		5	5	
MI ^d					3	3		230	20	2	
MN ^e	2900				18			430	9.7	3.1	
NH ^f					6.1	4.3			4	3	
NJ ^g					2	0.74				1.8	
NC ^h											100
TX ⁱ	2900	3.8	3.8	23	12	12	15	1400	3.8	23	
VT ^j				20	20	20			20	20	
WA ^k					3					3	

Note: Value in boldface were used in the hazard assessment in this study. CA = California; MA = Massachusetts; MI = Michigan; MN = Minnesota; NH = New Hampshire; NJ = New Jersey; NC = North Carolina; TX = Texas; VT = Vermont; WA = Washington

a: https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final-plain.pdf.

https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final-plain.pdf.

b: <https://oehha.ca.gov/media/downloads/water/chemicals/nl/final-pfoa-pfosnl082119.pdf>

c: <https://www.mass.gov/doc/per-and-polyfluoroalkyl-substances-pfas-an-updated-subgroup-approach-to-groundwater-and/download>

d:

https://www.michigan.gov/documents/pfasresponse/MDHHS_Public_Health_Drinking_Water_Screening_Levels_for_PFAS_651683_7.pdf.

e: <https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfoa.pdf>.

<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfbssummary.pdf>.

<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfoa.pdf>.

<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfhxs.pdf>

<https://www.health.state.mn.us/communities/environment/risk/docs/guidance/gw/pfba2summ.pdf>.

f: <https://www4.des.state.nh.us/nh-pfas-investigation/wp-content/uploads/June-PFAS-MCL-Technical-Support-Document-FINAL.pdf>.

g: <https://www.nj.gov/dep/watersupply/pdf/pfoa-recommend.pdf>.

<https://www.state.nj.us/dep/watersupply/pdf/pfos-recommendation-summary.pdf>.

https://www.state.nj.us/dep/wms/bears/docs/pfna_fact_sheet.pdf

h: <https://epi.dph.ncdhhs.gov/oe/pfas/NC%20DHHS%20Health%20Goal%20Q&A.pdf>

i: <https://www.tceq.texas.gov/assets/public/implementation/tox/evaluations/pfcs.pdf>.

j: https://www.healthvermont.gov/sites/default/files/documents/pdf/ENV_DW_PFAS_HealthAdvisory.pdf.

k: <https://www.doh.wa.gov/Portals/1/Documents/4200/PFASToxicologicalAssessment.pdf>

where

HQ = hazard quotient for individual PFASs.

RfD = reference dose (ng/kg-day)

Hazard index (HI) for the mixture of PFASs were calculated using equation (3):

$$HI = \sum HQ \quad (\text{eq-3})$$

For human risk assessment, a HQ (HI) > 1 indicates there is an increased risk for an adverse health outcome. Reference doses (RfD) for each PFASs were based on the available toxicity reference values (Table 5). For consistency, RfDs from Texas for the majority of PFASs and from North Carolina for GenX were used. Alternatively, a tolerable weekly intake of 4.4 ng/kg per week (0.63 ng/kg-day) was also used to evaluate HI (EFSA, 2020). This weekly reference dose takes into account PFAS accumulation and protects against other likely adverse effects reported in human studies.

Results

PFAS concentration

PFAS concentrations measured in produce samples are listed in Table 6. Most PFAS were below MDL in most samples. However, two PFASs were generally found. 4:2 FTS were observed in all lettuce samples (0.15 to 0.40 ng/kg, with an average of 0.28 ng/g) and PFDA in all potato (0.15 to 1.11 ng/kg, with an average of 0.48 ng/g) and tomato samples (0.11 to 0.49 ng/kg, with an average of 0.28 ng/g). PFDA levels were not significantly different between potato and tomato ($p > 0.05$). Outside 4:2 FTS and PFDA, other PFASs were sparsely detected. Two relatively high levels were detected in PFBA in one potato sample (5.29 ng/kg) and in PFBS in one tomato sample (6.5 ng/kg).

Levels of PFAS measured in Pittsboro drinking water are shown in Figure 2 (Summary information was listed in Table S3). Total PFAS ranged from <MDL to 458.1 ng/L, with an average of 189.9 ng/L. The median levels of each PFAS were ranked as follow: PFHxA (38.13 ng/L), PFPeA (32.66 ng/L) > PFBA (16.12 ng/L), PFHpA (15.66 ng/L) > PFOA (5.23 ng/L), PFBS (4.71 ng/L) > PFOS (3.01 ng/L) > PFHxS (1.97 ng/L) > 6:2 FTS (0.78 ng/L) > PFNA (0.57 ng/L) > PFDA (0.41 ng/L) > GenX (0.05 ng/L) > 4:2 FTS (0.01 ng/L) ($p < 0.05$, as shown in Table SI3). For the two highest PFAS, the 95th levels reached 173.3 ng/L for PFHxA and 101.2 ng/L for PFPeA. PFCA levels in general were measured at higher levels than PFSAs. The amounts of PFAS fluorotelomer alternatives (FTS) and ether-based PFAS (GenX) were generally less than 1 ng/L.

Table 6. Summary of PFAS concentrations (ng/g, ppb) in produce.

	Label	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	4:2 FTS	6:2 FTS
Lettuce	L1	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.03	0.40	<MDL
	L2	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.15	<MDL
	L3	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.33	<MDL
	L4	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.26	<MDL
	L5	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.29	<MDL
	L6	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.27	<MDL
Potato	P1	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	1.11	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL
	P2	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.39	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL
	P3	5.29	<MDL	<MDL	<MDL	<MDL	<MDL	0.77	<MDL	<MDL	<MDL	<MDL	<MDL	0.03
	P4	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.30	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL
	P5	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.18	<MDL	<MDL	<MDL	0.06	<MDL	<MDL
	P6	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.15	<MDL	<MDL	0.18	<MDL	<MDL	<MDL
Tomato	T1	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.18	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL
	T2	<MDL	<MDL	<MDL	<MDL	0.48	<MDL	0.42	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL
	T3	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.33	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL
	T4	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.14	6.50	<MDL	<MDL	<MDL	<MDL	<MDL
	T5	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.49	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL
	T6	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL	0.11	<MDL	<MDL	<MDL	<MDL	<MDL	<MDL

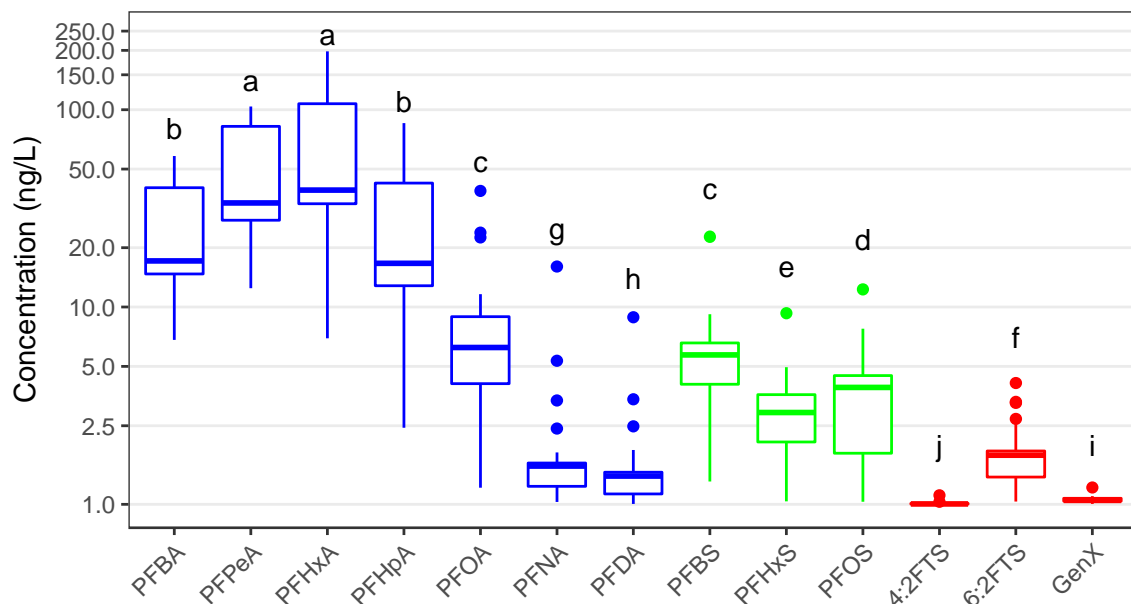


Figure 2. Boxplot of PFAS concentrations (ng/L, ppt) in drinking water. Boxplot was made of 5th, 25th, 50th, 75th and 95th percentiles as well as outliers indicated by dots. Y axis was scaled in log10 (All values were added by 1 for before transformation). Lower case letters indicated significant difference between each PFAS using Wilconxon Rank Test.

It should be noted that concentration units of PFAS are different between drinking water and produce. These values were compared in the unit of ppt. It was found that the averaged PFDA levels in produce (270 ppt) were 3 orders of magnitude higher than in drinking water (0.58 ppt). No comparisons were made for other PFASs. One noted difference was that PFAS with higher concentration in drinking water (such as PFHxA and PFPA) were barely detected in produce, while PFAS detected in produce (4:2 FTS and PFDA) were found in low concentrations in water.

Exposure intake

Daily dietary PFAS intake via produce and drinking water was calculated (Table SI4) and was compared among different age groups (Figure 3). However, PFAS exposure via produce consumption could only be estimated for 4:2 FTS and PFDA. Based on median values, PFDA and 4:2 FTS exposure from produce were higher than those from drinking water among all age groups. It was generally observed that as age increases, exposure intake to PFASs decreases.

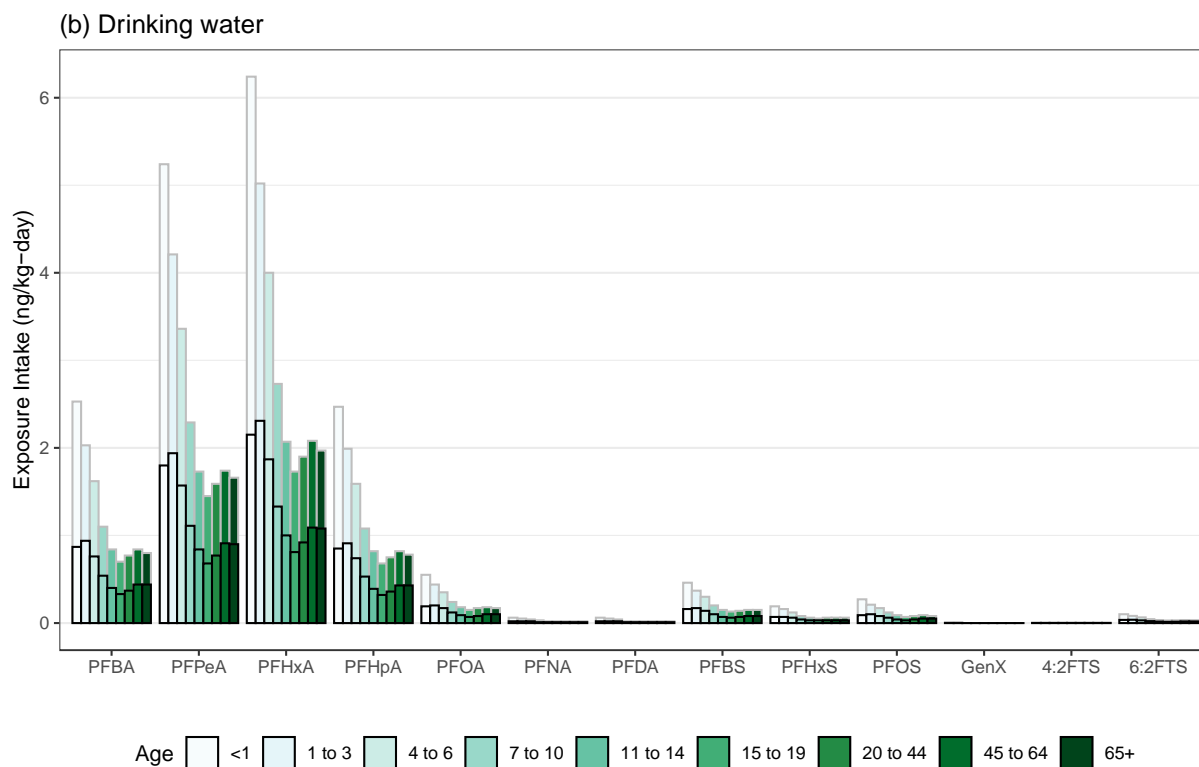
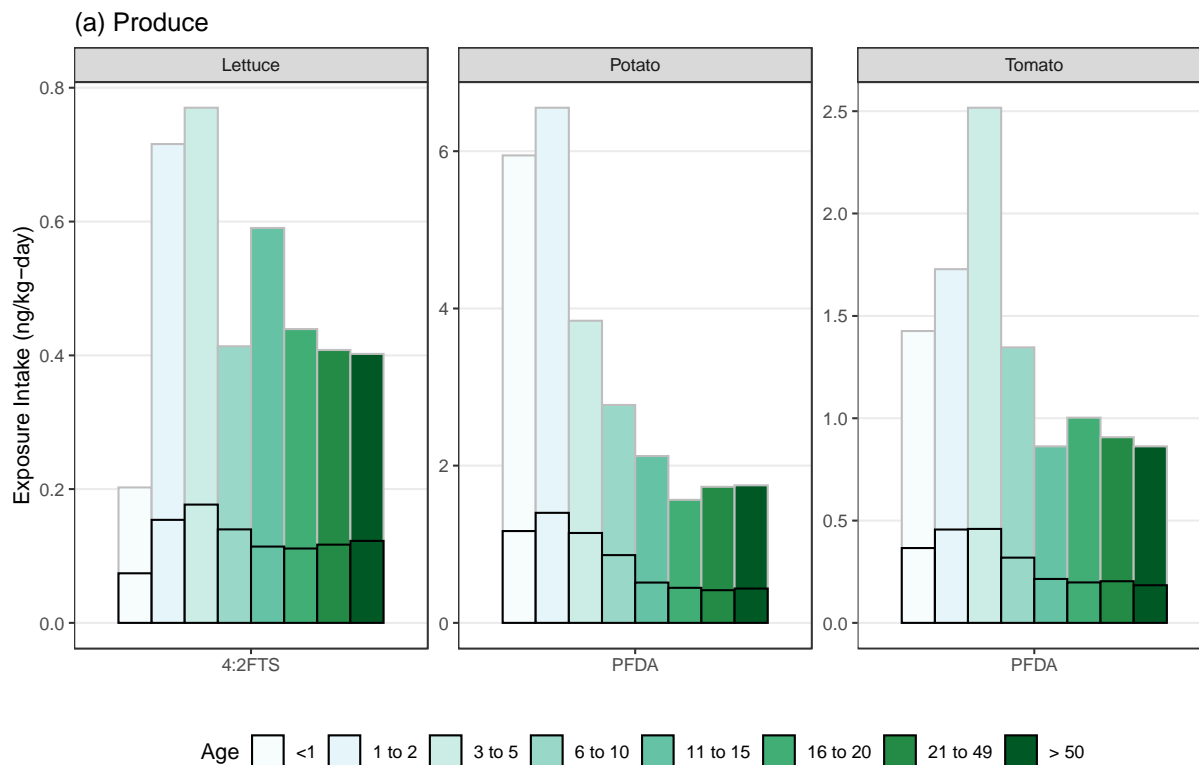


Figure 3. Estimated daily dietary exposure intake (ng/kg-day) of individuals PFAS via (a) produce (lettuce, potato and tomato) and (b) drinking water by age groups (in years). Black bars indicated 50th percentile and grey bars indicated 95th percentile. Exposure estimate are provided in Table S4.

In produce, potato was the major source of exposure to PFDA among all age groups, followed by tomato. The averaged median exposure intake was 0.80 ng/kg-day (0.42 to 1.40 ng/kg-day) for potato, and 0.30 ng/kg-day (0.18 to 0.46 ng/kg-day) for tomato. In terms of age groups, exposure in early life (age <1 to 5) was higher than in later life. However, the peak exposure occurred in age group 1 to 5 instead of during infancy (<1 year). The 95th exposure intake increased median exposure by as much as 4 times and generally follows the pattern of median exposure. However, one notable difference was the 95th percentile exposure estimates from tomato consumption in age group 3 to 5, which was much higher than in age group 1 to 2, while the median exposures were similar. The other difference was that higher 95th exposure of lettuce in age group 11 to 15 was observed. These differences were driven by different produce consumption rates among age groups. For example, the 95th percentile lettuce consumption rate for people aged 11 to 15 was 2.07 g/kg-day, which was higher than that for people aged 6 to 10 (1.45 g/kg-day) and people aged 16 to 20 (1.54 g/kg-day).

In drinking water, the pattern of exposure intake to each PFAS was driven by its concentration. The first exposure tier (>1 ng/kg-day) was found in PFHxA (1.40 ng/kg-day), followed by PFPeA (1.17 ng/kg-day). The second tier (0.1 to 1 ng/kg-day) was found in PFBA (0.57 ng/kg-day), PFHpA (0.55 ng/kg-day), PFOA (0.12 ng/kg-day) and PFBS (0.10 ng/kg-day). The third tier (<0.1 ng/kg-day) was PFOS (0.06 ng/kg-day), PFHxS (0.04 ng/kg-day), 6:2 FTS (0.02 ng/kg-day), PFNA (0.13 ng/kg-day) and PFDA (0.13 ng/kg-day). Lowest exposures were observed for GenX and 4:2 FTS. In terms of age groups, similar age-dependent patterns were observed. Peak median exposure was estimated at age 1 to 3. However, peak 95th exposure occurred at the infant stage (<1). In general, 95th exposure intakes increased median exposure as much as 2 times from drinking water.

Risk analysis

Hazard analysis combines both exposure and toxicity information of chemicals or mixture and was used to identify populations with potential risk. HQ and HI for produce and drinking water among age groups are shown in Figure 4 and Table S5. HQ of 4:2 FTS and 6:2 FTS were not

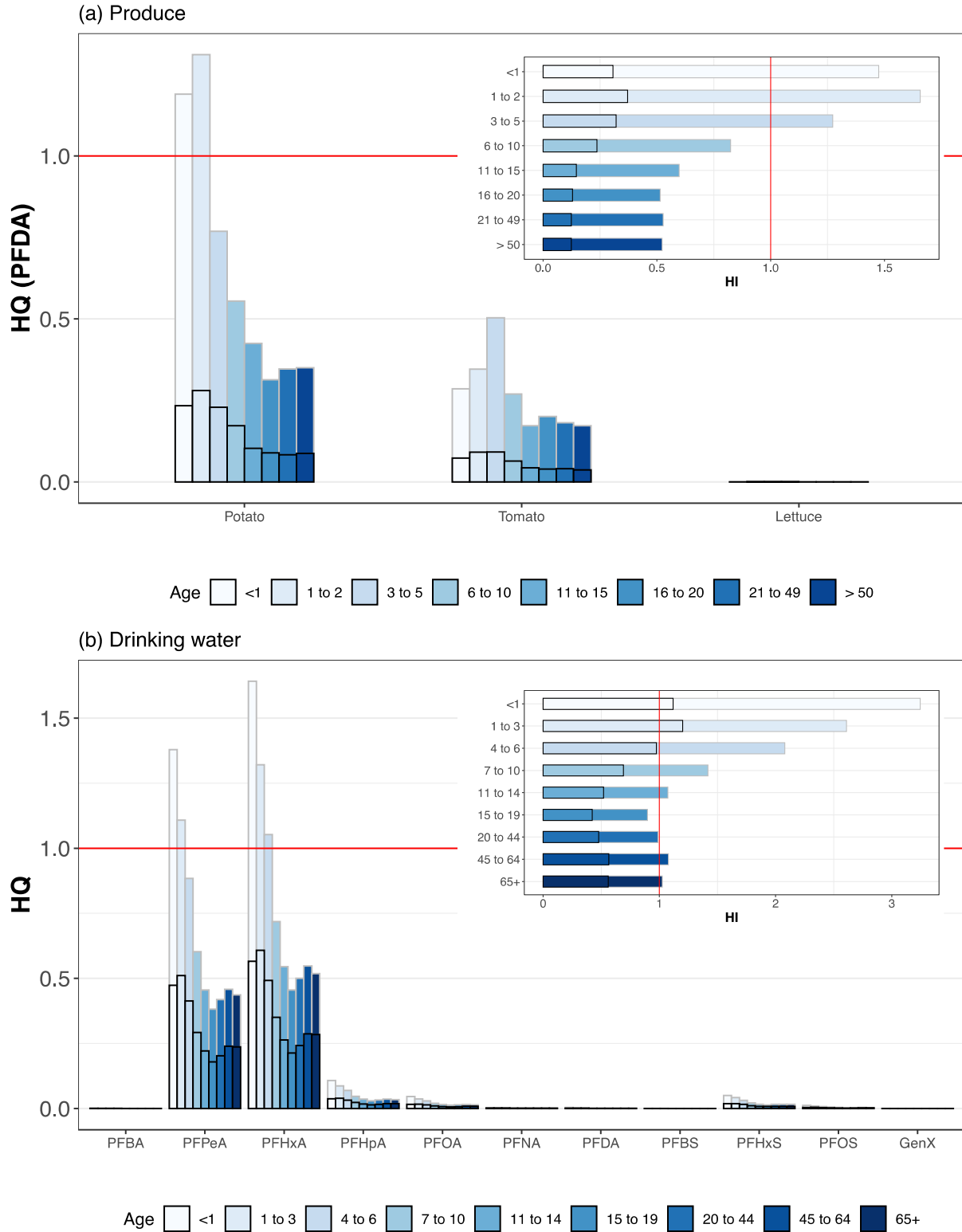


Figure 4. Estimate of the 50th and 95th percentile hazard quotients (HQ) and hazard index (HI) from consumption of (a) produce and (b) drinking water for individual PFAS by age groups. Black bars indicated 50th percentile and grey bars indicated 95th percentile. Estimates are provided Table S5.

calculated due to a lack of RfDs. It was generally observed that infant and children were at elevated risk of PFAS exposure compared to other age groups, indicated by higher HI values. In produce, HQs were calculated for PFDA, which was the only PFAS detected in tomatoes and potatoes. All median HQs fell below a value of 1.0. However, based on the 95th exposure level estimates, HQ values were higher than 1 for infants (age < 1) (1.2) and children aged 1 to 2 (1.3) consuming potatoes. Potato was the major source of hazard among each age group, followed by tomato and lettuce. When taken together, the HI fell between 0.1 to 1.7. A HI >1 was found in infants (1.5), children aged 1 to 2 (1.7) and children aged 3 to 5 (1.3) based on 95th percentile exposures. No HI >1 was found at median levels among each age group.

In drinking water, HQ was calculated for each of the 11 studied PFAS. No potential hazard was identified at median exposure levels. However, at 95th percentile levels, HQ > 1 was found in infants aged < 1 (1.4) and children aged 1 to 3 (1.1) for PFPeA, and in infant aged < 1 (1.6), children aged 1 to 3 (1.3) and children aged 4 to 6 (1.1) for PFHpA. When taken together, it was suggested that drinking water was the major concern for PFAS exposure compared to produce. At 95th exposure levels, HI > 1 was found in nearly all age groups (0.9 to 3.2). At median levels, HI > 1 was only found in infant (1.1), children aged 1 to 3 (1.2) and children aged 4 to 6 (1.0). In conclusion, it was suggested that younger children (<1 to 6) were at higher risk of PFAS exposure compared to other age groups.

Discussion

Studies on PFAS measurements in produce are rather scarce and can be categorized in two types. One type includes field-based studies in which samples are collected in markets and analyzed for PFAS (Heo et al., 2014; Herzke et al., 2013). The other type is greenhouse studies in which produce are cultivated in a laboratory with PFAS contaminated water or amended soils (Blaine et al., 2013, 2014; Felizeter et al., 2012). Inconsistencies were found between the two types of studies. First, larger amounts of PFAS were generally observed in the second type of study where produce was exposed to PFAS under specific growing conditions. For example, concentrations in tomato grown in PFAA amended soil reached 56 ppb (ng/g) for PFBA and 211 ppb (ng/g) for PFPeA in a greenhouse study, while lower levels of 12.1 ppt (ng/kg) PFHxA and 3.2 ppt (ng/kg) PFOA were reported in tomatoes collected from European markets (Herzket et

al., 2013). In Herzket et al. (2013) study, PFOA, PFHxA and PFNA were the most abundant PFAS, with detection rates of 44, 32 and 10%. This study also reported low levels of PFAS in lettuce, tomatoes and potatoes. The most often detected PFAS was PFDA, with a 67% detection rate and a 6 orders magnitude of larger levels. Second, a chain-length dependent pattern of PFAS accumulation was reported in several greenhouse studies (Blaine et al., 2014). For example, short-chain PFCAs are likely to be translocated in the leaf while long-chained PFCAs are retained in the root. However, this pattern was not observed in the market basket studies, nor in the present study. This might be due to low levels and detection frequency in the samples. In general, it is acknowledged that PFAS can be taken up by plants and stored in the edible portion of the organs. However, such accumulation is not fully understood, which is further confounded by soil characteristics, composition of irrigation water and differences in plants. This study reported detectable PFDA levels in tomato and potato samples. Soil, irrigation water and air contaminated by PFAS could all lead to accumulation in produce. Therefore, it is not possible to determine the source of contamination without further research.

This study evaluated dietary exposure to PFAS among age groups. Two patterns were observed. First, exposure to PFAS was higher via drinking water compared to exposure via produce. However, there are several limitations to this study. First, it should be noted that exposure via produce was solely based on PFDA and only three major types of produces were selected. In addition, exposure was calculated with produce samples collected from a regional basis within a limited time frame. Therefore, a larger sample size and comprehensive sampling design would reduce such uncertainty when estimating exposure intake through produce. Second, higher exposure was found in young children compared to adults and the elderly. Such decreasing pattern was also observed in many exposure assessments. This is mainly because exposure was normalized by body weight. Even though adults are exposed to larger total amount of PFAS, higher intake was found in children due to their smaller body weight. In addition, food consumption factors were based on US EPA national exposure handbook rather than local consumption factors in North Carolina. This might affect the precision of exposure estimates.

Currently, there are various studies estimating dietary exposure to PFAS. In 2020, EFSA

Table 7. Summary of HQs for the sum of four PFASs (PFOA, PFNA, PFHxS and PFOS) among age groups.

Drinking Water	Σ HQ ^a		HQ (EFSA) ^b	
	50	95	50	95
Age				
<1	0.04	0.11	0.59	1.70
1 to 3	0.04	0.09	0.62	1.37
4 to 6	0.04	0.07	0.52	1.08
7 to 10	0.02	0.05	0.37	0.75
11 to 14	0.02	0.04	0.27	0.56
15 to 19	0.02	0.03	0.22	0.46
20 to 44	0.02	0.03	0.25	0.52
45 to 64	0.02	0.04	0.30	0.56
65+	0.02	0.03	0.30	0.52

a: Σ HQ = HQ_{PFOA} + HQ_{PFNA} + HQ_{PFHxS} + HQ_{PFOS}

b: HQ (EFSA) = (EI_{PFOA} + EI_{PFNA} + EI_{PFHxS} + EI_{PFOS}) / EFSA tolerable intake (0.63 ng/kg-day)

evaluated total PFAS exposure via food among different age groups. In general, they found that fish and sea food are the major contributors to PFAS exposure. Other important exposures include meat, fruit, eggs, vegetables and drinking water. Based on 95th percentile upper bound median values in the EFSA report, PFDA in studied produce contributed to 24.7% of total dietary exposure in infants. For drinking water, such contributions are: PFBA (11.9%), PFPeA (14.3%), PFHxA (22.5%), PFHpA (9.6%), PFOA (2.0%), PFNA (0.2%), PFDA (0.2%), PFBS (1.4%), PFHxS (0.6%) and PFOS (0.8%). A 4.4 ng/kg-week (0.63 ng/kg-day) tolerable weekly intake was established by EFSA for the sum of four PFAS (PFOA, PFNA, PFHxS and PFOS). In this study, exposures greater than 0.63 ng/kg-day for the 4 combined PFAS via drinking water were found in young children (aged <1, 1 to 3 and 4 to 6) at 95th percentile exposure (Table 7). However, Σ HQ for these 4 PFAS were all below 1 (Table 7). No comparison was made for produce due to unavailable data. Therefore, this study suggests that produce and drinking water are important pathways for human exposure to PFAS.

A hazard analysis was made to characterize health risks related to the presence of PFAS in diet. HQs were calculated with RfDs established by the state of Texas, except for GenX. This is because 1) many studies suggest that the EPA RfDs for PFOS and PFOA are not strict enough to protect human health; and 2) RfDs among different states vary by order of magnitude, which might induce uncertainties from inclusion criteria (Post, 2020). For produce, potato was found to

be of major concern, which is consistent with its high concentration and exposure levels. For drinking water, PFPeA and PFHxA were the only two PFAS with HQ greater than 1 based on 95th percentile levels. Despite its relatively high concentration and exposure levels, PFBA and PFHpA had low HQ values. This study did not find elevated risk induced by PFOS and PFOA, suggesting effective benefits of phase-out of these two PFAS. When taken together, elevated risks were found among children at early age based on 95th percentile exposure ($HI > 1$). It was observed that HI decreases with age. It should be noted that RfDs established by many states (including Texas) are designed to be protective against chronic exposure, either through using data derived from chronic animal exposure studies or by including uncertainty factors (UF) in their estimates. Therefore, HI for each age groups also accounts for risks caused by long-term exposure. In addition, similar results were also reported in several earlier risk assessments. For example, the highest median HI was found in children aged 1 to 2 (15) in a study by Brown et al. (2021). However, one difference was that the lowest HI was found in adolescent aged 12-19 (4.2) and significantly higher HI was reported later in life (9.4). More importantly, based on 95th percentile levels, people in all age groups were at risk of PFAS exposure via drinking water, while such risks were only observed in children at early life. Overall, the study suggested that PFAS exposure via drinking water is more of concern compared to exposure via produce.

Many state, federal and national agencies are starting to develop PFAS regulations for drinking water. In addition to the 70 ng/L Health Advisory for PFOS and PFOA established by US EPA, many states have developed their own more stringent guidelines, mainly for PFAAs. Currently, only NC has established a regulation on GenX. However, there is an order of magnitude difference among the thresholds established by other states due to differences in selection of scientific studies and critical health endpoints. Therefore, more research is needed for PFAS risk assessment and management on a regional basis. Additional toxicity data and guidelines for FTS and other PFAA precursors are expected. This study indicated that only a small portion of the population is at an elevated risk of PFAS exposure from consuming produce. However, it is important to note that exposure estimates were limited to three types of produce. Other food items (including fish and sea food) are possible sources of PFAS exposure and were not included in this study. In addition, it was reported that PFAS in food packaging (e.g hamburger wrappers) are also bioaccumulative and contribute significantly to PFAS exposure, especially for people

who ate out more (Seltenrich et al., 2020). But this was not considered in the exposure assessment. Therefore, PFAS exposure estimate in this study might underestimate the actual risk introduced by produce consumption.

Acknowledgements

This project was supported by the Stapleton Laboratory in the Nichols School of the Environment, Duke University. The author is grateful for all the tremendous support and expertise given by the lab members, including but not limited to, principal investigator Dr. Heather Stapleton, Ph.D candidate Taylor Hoxie, research manager Sharon Zhang, and lab technicians George Tait and Duncan Hay. For people who want to follow up with the latest information and research, the lab link is provide here:

<https://sites.nicholas.duke.edu/stapletonlab/research/pfas-research/>.

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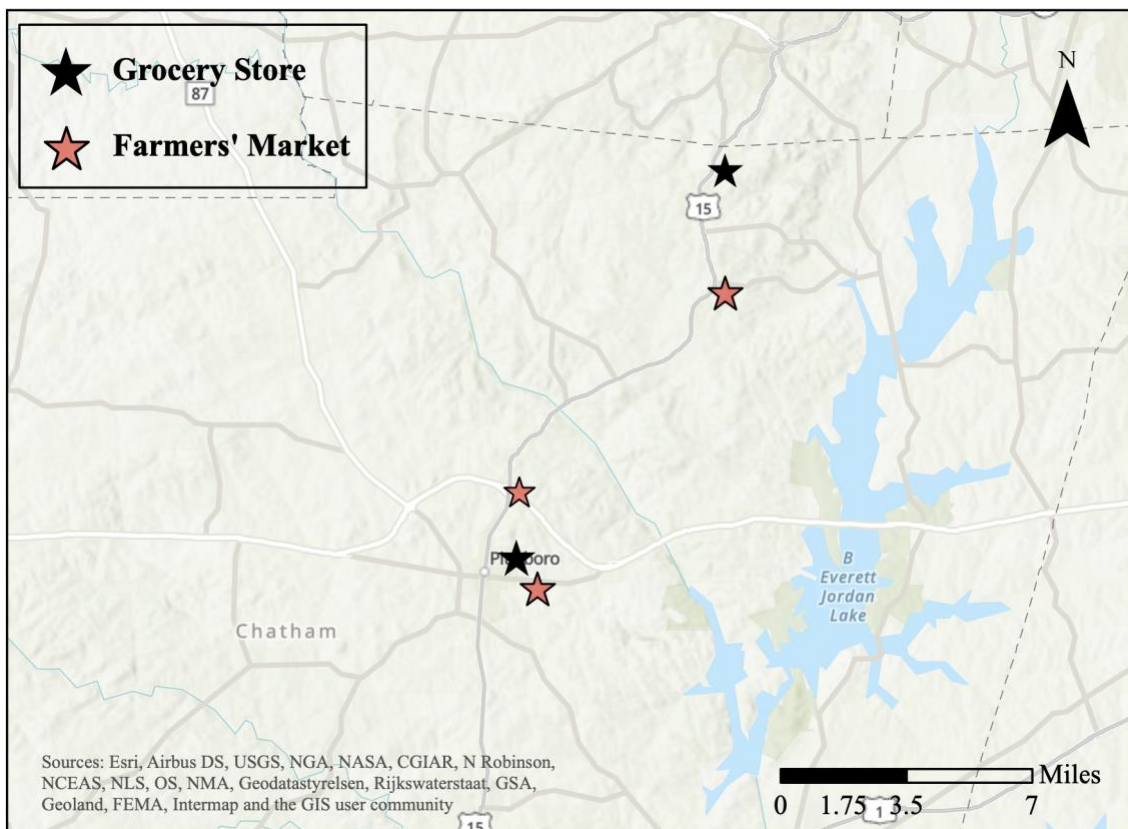


Figure S1. Geographic image of sample collection sites in Chatham County, North Carolina, United States.

Table S1. Summary of MDLs (ng/g) for each batch.

	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	4:2 FTS	6:2 FTS
1	0.63	<0.01	0.07	0.05	0.09	0.06	0.1	3.63	0.03	0.09	<0.01	<0.01	0.04
2	-	<0.01	1.1	1.09	1.2	0.79	0.31	4.24	0.09	0.04	<0.01	<0.01	0.01
3	3.8	<0.01	0	0.25	0.12	0.04	0.05	2.25	0.02	0.01	<0.01	<0.01	0.05
Average	2.22	<0.01	0.39	0.47	0.47	0.3	0.15	3.38	0.05	0.05	<0.01	<0.01	0.03

Table S2. Summary matrix effect index for each type of sample. Matrix effect index was calculated through dividing recoveries of samples by blanks.

	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	6:2 FTS
Tomato	157%	177%	113%	189%	175%	173%	192%	43%	63%	80%	478%	75%
Potato	142%	196%	88%	137%	158%	117%	93%	34%	37%	73%	435%	54%
Lettuce	139%	146%	62%	198%	188%	172%	209%	43%	69%	80%	556%	61%
Average	146%	173%	88%	175%	174%	154%	165%	40%	56%	78%	490%	64%

Table S3. Summary of p-value of Wilcoxon Rank Test between PFASs. Values highlighted by red indicated p-value higher than 0.05.

	PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	4:2 FTS
PFPeA	<0.0001	-	-	-	-	-	-	-	-	-	-	-
PFHxA	<0.0001	0.3831	-	-	-	-	-	-	-	-	-	-
PFHpA	0.8295	0.0013	0.0004	-	-	-	-	-	-	-	-	-
PFOA	<0.0001	<0.0001	<0.0001	<0.0001	-	-	-	-	-	-	-	-
PFNA	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	-	-	-	-	-	-	-
PFDA	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0286	-	-	-	-	-	-
PFBS	<0.0001	<0.0001	<0.0001	<0.0001	0.3831	<0.0001	<0.0001	-	-	-	-	-
PFHxS	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	-	-	-	-
PFOS	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0003	0.0287	-	-	-
GenX	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	-	-
4:2 FTS	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0089	-
6:2 FTS	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0324	0.0038	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

Table S4. Summary of 50th and 95th percentile estimates for exposure intake via produce (lettuce, potato and tomato) and drinking water by age groups.

Age Group	Percentile	PFBA	PFPeA	PFHxA	PHHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	4:2 FTS	6:2 FTS
Lettuce														
<1	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.07	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.20	NA
1 to 2	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.15	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.72	NA
3 to 5	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.18	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.77	NA
6 to 10	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.14	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.41	NA
11 to 15	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.11	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.59	NA
16 to 20	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.11	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.44	NA
21 to 49	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.12	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.41	NA
> 50	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.12	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.40	NA
Potato														
<1	50	NA	NA	NA	NA	NA	NA	1.17	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	5.95	NA	NA	NA	NA	NA	NA
1 to 2	50	NA	NA	NA	NA	NA	NA	1.14	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	6.55	NA	NA	NA	NA	NA	NA
3 to 5	50	NA	NA	NA	NA	NA	NA	1.14	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	3.84	NA	NA	NA	NA	NA	NA

Age Group	Percentile	PFBA	PFPeA	PFHxA	PHHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	4:2 FTS	6:2 FTS
6 to 10	50	NA	NA	NA	NA	NA	NA	0.86	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	2.77	NA	NA	NA	NA	NA	NA
11 to 15	50	NA	NA	NA	NA	NA	NA	0.51	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	2.12	NA	NA	NA	NA	NA	NA
16 to 20	50	NA	NA	NA	NA	NA	NA	0.45	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	1.57	NA	NA	NA	NA	NA	NA
21 to 49	50	NA	NA	NA	NA	NA	NA	0.42	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	1.73	NA	NA	NA	NA	NA	NA
> 50	50	NA	NA	NA	NA	NA	NA	0.44	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	1.75	NA	NA	NA	NA	NA	NA
Tomato														
<1	50	NA	NA	NA	NA	NA	NA	0.37	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	1.43	NA	NA	NA	NA	NA	NA
1 to 2	50	NA	NA	NA	NA	NA	NA	0.46	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	1.73	NA	NA	NA	NA	NA	NA
3 to 5	50	NA	NA	NA	NA	NA	NA	0.46	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	2.52	NA	NA	NA	NA	NA	NA
6 to 10	50	NA	NA	NA	NA	NA	NA	0.32	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	1.35	NA	NA	NA	NA	NA	NA
11 to 15	50	NA	NA	NA	NA	NA	NA	0.21	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	0.86	NA	NA	NA	NA	NA	NA
16 to 20	50	NA	NA	NA	NA	NA	NA	0.20	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	1.00	NA	NA	NA	NA	NA	NA
21 to 49	50	NA	NA	NA	NA	NA	NA	0.20	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	0.91	NA	NA	NA	NA	NA	NA
> 50	50	NA	NA	NA	NA	NA	NA	0.18	NA	NA	NA	NA	NA	NA

Age Group	Percentile	PFBA	PFPeA	PFHxA	PHHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	4:2 FTS	6:2 FTS
	95	NA	NA	NA	NA	NA	NA	0.86	NA	NA	NA	NA	NA	NA
Drinking Water														
<1	50	0.87	1.8	2.15	0.85	0.19	0.02	0.02	0.16	0.07	0.09	<0.01	<0.01	0.03
	95	2.53	5.24	6.24	2.47	0.55	0.06	0.06	0.46	0.19	0.27	0.01	<0.01	0.07
1 to 3	50	0.94	1.94	2.31	0.91	0.2	0.02	0.02	0.17	0.07	0.1	<0.01	<0.01	0.03
	95	2.03	4.21	5.02	1.99	0.44	0.05	0.05	0.37	0.16	0.21	0.01	<0.01	0.06
4 to 6	50	0.76	1.57	1.87	0.74	0.17	0.02	0.02	0.14	0.06	0.08	<0.01	<0.01	0.02
	95	1.62	3.36	4	1.59	0.35	0.04	0.04	0.3	0.12	0.17	<0.01	<0.01	0.05
7 to 10	50	0.54	1.11	1.33	0.53	0.12	0.01	0.01	0.1	0.04	0.06	<0.01	<0.01	0.02
	95	1.1	2.29	2.73	1.08	0.24	0.03	0.02	0.2	0.08	0.12	<0.01	<0.01	0.03
11 to 14	50	0.4	0.84	1	0.39	0.09	0.01	0.01	0.07	0.03	0.04	<0.01	<0.01	0.01
	95	0.84	1.73	2.07	0.82	0.18	0.02	0.02	0.15	0.06	0.09	<0.01	<0.01	0.02
15 to 19	50	0.33	0.68	0.81	0.32	0.07	0.01	0.01	0.06	0.03	0.03	<0.01	<0.01	0.01
	95	0.7	1.45	1.73	0.68	0.15	0.02	0.02	0.13	0.05	0.07	<0.01	<0.01	0.02
20 to 44	50	0.37	0.77	0.92	0.36	0.08	0.01	0.01	0.07	0.03	0.04	<0.01	<0.01	0.01
	95	0.77	1.59	1.9	0.75	0.17	0.02	0.02	0.14	0.06	0.08	<0.01	<0.01	0.02
45 to 64	50	0.44	0.91	1.09	0.43	0.1	0.01	0.01	0.08	0.03	0.05	<0.01	<0.01	0.01
	95	0.84	1.74	2.08	0.82	0.18	0.02	0.02	0.15	0.06	0.09	<0.01	<0.01	0.02
65+	50	0.44	0.9	1.08	0.43	0.1	0.01	0.01	0.08	0.03	0.05	<0.01	<0.01	0.01
	95	0.8	1.66	1.97	0.78	0.17	0.02	0.02	0.15	0.06	0.08	<0.01	<0.01	0.02

Table S5. Summary of 50th and 95th percentile estimates for hazard quotients and hazard index of consuming produce (lettuce, potato and tomato) and drinking water by age groups.

Age Group	Percentile	HQ											HI
		PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	∑ PFAS
Lettuce													
<1	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1 to 2	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
3 to 5	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
6 to 10	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
11 to 15	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
16 to 20	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
21 to 49	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	95	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
> 50	50	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
		NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Potato													
<1	50	NA	NA	NA	NA	NA	NA	0.2	NA	NA	NA	NA	0.2
	95	NA	NA	NA	NA	NA	NA	1.2	NA	NA	NA	NA	1.2
1 to 2	50	NA	NA	NA	NA	NA	NA	0.3	NA	NA	NA	NA	0.3
	95	NA	NA	NA	NA	NA	NA	1.3	NA	NA	NA	NA	1.3
3 to 5	50	NA	NA	NA	NA	NA	NA	0.2	NA	NA	NA	NA	0.2
	95	NA	NA	NA	NA	NA	NA	0.8	NA	NA	NA	NA	0.8
6 to 10	50	NA	NA	NA	NA	NA	NA	0.2	NA	NA	NA	NA	0.2

Age Group	Percentile	HQ											HI
		PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	Σ PFAS
11 to 15	95	NA	NA	NA	NA	NA	NA	0.4	NA	NA	NA	NA	0.4
	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.4	NA	NA	NA	NA	0.4
16 to 20	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.3	NA	NA	NA	NA	0.3
21 to 49	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.4	NA	NA	NA	NA	0.4
> 50	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.4	NA	NA	NA	NA	0.4
Tomato													
<1	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.3	NA	NA	NA	NA	0.3
1 to 2	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.4	NA	NA	NA	NA	0.4
3 to 5	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.5	NA	NA	NA	NA	0.5
6 to 10	50	NA	NA	NA	NA	NA	NA	0.1	NA	NA	NA	NA	0.1
	95	NA	NA	NA	NA	NA	NA	0.3	NA	NA	NA	NA	0.3
11 to 15	50	NA	NA	NA	NA	NA	NA	<0.1	NA	NA	NA	NA	<0.1
	95	NA	NA	NA	NA	NA	NA	0.2	NA	NA	NA	NA	0.2
16 to 20	50	NA	NA	NA	NA	NA	NA	<0.1	NA	NA	NA	NA	<0.1
	95	NA	NA	NA	NA	NA	NA	0.2	NA	NA	NA	NA	0.2
21 to 49	50	NA	NA	NA	NA	NA	NA	<0.1	NA	NA	NA	NA	<0.1
	95	NA	NA	NA	NA	NA	NA	0.2	NA	NA	NA	NA	0.2
> 50	50	NA	NA	NA	NA	NA	NA	<0.1	NA	NA	NA	NA	<0.1
	95	NA	NA	NA	NA	NA	NA	0.2	NA	NA	NA	NA	0.2

Drinking Water

Age Group	Percentile	HQ											HI
		PFBA	PFPeA	PFHxA	PFHpA	PFOA	PFNA	PFDA	PFBS	PFHxS	PFOS	GenX	Σ PFAS
<1	50	<0.1	0.5	0.6	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.1
	95	<0.1	1.4	1.6	0.1	<0.1	<0.1	<0.1	<0.1	0.1	<0.1	<0.1	3.2
1 to 3	50	<0.1	0.5	0.6	0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.2
	95	<0.1	1.1	1.3	0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	2.6
4 to 6	50	<0.1	0.4	0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.0
	95	<0.1	0.9	1.1	0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	2.1
7 to 10	50	<0.1	0.3	0.4	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.7
	95	<0.1	0.6	0.7	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.4
11 to 14	50	<0.1	0.2	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.5
	95	<0.1	0.5	0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.1
15 to 19	50	<0.1	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.4
	95	<0.1	0.4	0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.9
20 to 44	50	<0.1	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.5
	95	<0.1	0.4	0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.0
45 to 64	50	<0.1	0.2	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.6
	95	<0.1	0.5	0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.1
65+	50	<0.1	0.2	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.6
	95	<0.1	0.4	0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1.0