Factsheet on Perfluoroalkyl Substances (PFAS) for Health Professionals

What are PFAS?

Per-and polyfluoroalkyl substances (PFAS) are synthetic chemicals. PFAS have a very strong carbon-fluorine bond that makes them chemically stable, resistant to typical environmental degradation processes, and are persistent in the environment. As of May 2018, researchers had identified ~ 4,730 PFAS-related structures in patent filings and chemical registries, any of which might be in commercial use (OECD, 2018). The compounds classified within this group of substances are resistant to stains, heat, oil, grease, and water, and also act as lubricants. Because they help reduce friction, they are also used in a variety of other industries, including aerospace, automotive, building and construction, and electronics.

PFAS are widely used to make everyday consumer products. For example, PFAS may be used to keep food from sticking to cookware, sofas and carpets more resistant to stains, packaging more resistant to grease, clothes and mattresses more waterproof, and as a component of some firefighting foams (ATSDR, 2018).

Because of their widespread use, most people in the United States have some PFAS in their body. But due to reductions in their incorporation into consumer products, the national trends for PFOA and PFOS in blood serum have been declining for several years (ATSDR 2017).

Data from human studies suggest that some PFAS can take years to be cleared from the body (Bartell et al., 2010; Seals et al., 2010).

Certain PFAS have estimated half-lives that can range from two to nine years:

- Perfluoro-octanoic acid /Perfluorooctanoate (PFOA): 3-4 years
- Perfluorooctane sulfonate (PFOS): 5-6 years
- Perfluorohexane sulfonate (PFHxS): 8-9 years (ATSDR, 2018).

These long half-lives result in body burdens that persist for years, even after identified exposure sources have been reduced (ATSDR 2009; Olsen, 2007).

List of Common PFAS and Their Abbreviations
(https://www.atsdr.cdc.gov/pfas/docs/pfas_fact_sheet.pdf):

Abbreviation	Chemical name
PFOS	Perfluorooctane sulfonic acid
PFOA (or C8)	Perfluorooctanoic acid
PFNA	Perfluorononanoic acid
PFDA	Perfluorodecanoic acid
PFBS	Perfluorobutanoic acid
PFOSA (or FOSA)	Perfluorooctane sulfonaminde
MeFOSAA (aka Me-PFOSA-AcOH)	2-(N-Methyl-perfluorooctane sulfonamido) acetic acid
Et-FOSAA (aka Et-PFOSA-AcOH)	2-(N-Ethyl-perfluorooctane sulfonamido) acetic acid
PFDeA	Perfluorodecanoic acid
PFUnDA	Perfluoroundecanoic acid
PFHxS	Perfluorohexane sulfonic acid

What are PFAS levels in the US population?

Most people in the United States and in other industrialized countries have measurable amounts of PFAS in their blood. PFOA and PFOS are the most prominent.

Since 1999, the National Health and Nutrition Examination Survey (NHANES) has measured blood PFAS in the U.S. population. Based on data collected from previous NHANES 3 cycle years, levels of PFOA and PFOS are generally decreasing in the blood of the general population. This appears to be a result of the 2006 agreement by eight major companies in the PFAS industry to phase out—voluntarily—these two PFAS by 2015. Recent National Health and Nutrition Examination Survey (NHANES) data has shown at least one PFAS in the blood of more than 98% of Americans (Calafat et al., 2019).

Ye et al. (2018) found PFAS in all children in the first nationally representative sample of among U.S. 3-11year old children (most of whom were born after the phase out of PFOS in the United States in 2002). PFAS concentration differences by sex, race/ethnicity, and age suggest lifestyle differences that may impact exposure, and highlight the importance of identifying exposure sources, reducing these exposures, and of studying the environmental fate and transport of PFAS.

Current PFAS levels in the U.S. can be found in the 2018 report at:
https://www.cdc.gov/exposurereport/pdf/FourthReport_UpdatedTables_Volume1_Mar2018.pdf

Tables for PFOA and PFOS with breakdowns by age, ethnicity, and gender are available in Appendix A.

What are the potential health effects of PFAS?

While there are thousands of PFAS compounds, very few have been studied for human health effects. A large number of studies, mainly of PFOA, PFOS, PFNA, and PFHxS, have examined possible relationships between levels of PFAS in blood and harmful health effects in people. https://www.atsdr.cdc.gov/toxprofiles/tp200-c2.pdf.

There is evidence that exposure to PFAS can lead to adverse health outcomes. If humans ingest contaminated food, water, or dust, the PFAS are absorbed, and can accumulate in the body. PFAS remain in the human body for long periods of time.

Studies have indicated that PFAS may affect growth, learning, and behavior of infants and older children; lower a woman's chance of getting pregnant; interfere with the body's natural hormones; increase cholesterol levels; affect the immune system; and increase the risk of some cancers.

From 2005 to 2013, a large epidemiological study (~70,000 participants) was conducted in the Mid-Ohio Valley as an outcome from a class action lawsuit. PFAS (primarily PFOA) was emitted by the active DuPont Washington Works facility near Parkersburg WV. Drinking water supplies in both West Virginia and Ohio were contaminated. The C8 Science Panel analyzed study data and found probable links (as defined by litigation) between elevated PFOA blood levels and high cholesterol (hypercholesteremia), ulcerative colitis, thyroid function, testicular cancer, kidney cancer, preeclampsia, as well as elevated blood pressure during pregnancy (http://www.c8sciencepanel.org/).

The most recent research data suggest that immunotoxicity and developmental toxicity are among the most sensitive endpoints for PFAS (ATSDR, 2018).

Studies indicate that exposure to PFOA and PFOS over certain levels may result in adverse health effects, including:

- **Asthma**
  - PFAS are associated with asthma and related outcomes, including airway hyperresponsiveness, increased serum levels of immunoglobulin E (IgE), and switched type 2 T helper cell (Th2) polarization (Anderson-Mahoney, 2008; Dong et al., 2011; Fairley et al., 2007; Singh et al., 2012).
  - NHANES data showed that serum PFOA concentrations are positively associated with self-reported lifetime asthma prevalence (Humblet et al., 2014).
  - A possible link between reproductive hormones, PFAS levels and asthma among adolescents has also been found. (Zhou et al., 2017).
- **Immune effects**
  - Both animal and epidemiology studies provide strong evidence linking PFOS exposure to immunotoxic effects (ATSDR, 2018; National Toxicology Program (NTP), 2016).

- Evidence is suggestive of a link between serum PFOA, PFOS, PFHxS, and PFDeA levels and decreased antibody responses to vaccines. (Haug et al., 2013).
- PFAS can be transferred to infants via breast milk, increasing risk to the immune system during critical windows of development (Heilman et al., 2017; Mondal et al., 2014).
- NHANES data suggests that PFAS serum concentrations are associated with a 7% greater pathogen burden in adults, and a 28% greater pathogen burden in adolescents (Bulka et al., 2021).
- Higher plasma PFBA concentrations may be associated with a more severe disease outcome (hospitalization, admission to ICU, and death) in individuals who contract COVID19 (Grandjean et al., 2021).

- **Cancer**
  - The International Agency for Research on Cancer (2017) has classified PFOA as "possibly carcinogenic to humans (Group 2B), based on limited evidence in humans that it can cause testicular and kidney cancer, and limited evidence in lab animals."
  - EPA (2016) has determined there is "suggestive evidence of carcinogenic potential" for PFOA. Epidemiology studies show an association between serum PFOA and kidney and testicular tumors in highly exposed individuals.
  - PFOS-exposed workers have demonstrated elevated incidence of bladder cancer mortality following at least one year of exposure (Lindtrom et al., 2011).
  - Epidemiologic cancer studies have also found associations between kidney, testicular, ovarian, prostate, and non-Hodgkins lymphoma with PFOA exposure (Barry et al., 2013; Vieira et al., 2013).
  - Studies in humans have not shown conclusive evidence that PFAS lead to certain cancers such as prostate cancer (Eriksen, 2009; Lundin, 2009; Vieira, 2013; Barry, 2013) and kidney cancer (Leonard, 2008; Lundin, 2009; Raleigh, 2014).
  - Barry et al., (2013) and Vieira et al., (2013) found strong positive associations between PFOA exposure and testicular cancer.
  - Studies suggest associations between PFAS exposure and kidney cancer (Barry et al., 2013; Steenland & Woskie, 2012; Vieira et al., 2013).
    - Findings from Shearer et al. (2020), support a positive association between PFOA exposure and kidney cancer.
    - Mastantonio et al., (2018) identified higher rates of mortality in kidney cancer cases in areas with increased PFAS exposure.
- **Hepatic effects**
  - Increases in serum enzymes and decreases in serum bilirubin, observed in studies of PFOA, PFOS, and PFHxS, are suggestive of liver damage (ATSDR, 2018; Das et al., 2017).
  - In addition, the results of epidemiology studies of PFOA, PFOS, PFNA, and PFDeA suggest a link between PFAS exposure and increases in serum lipid

levels, particularly total cholesterol and LDL cholesterol (ATSDR, 2018; Nelson et al., 2010).
    - Positive associations between PFAS exposure and levels of serum liver enzymes including alanine transaminase (ALT), alkaline phosphatase (ALP), and γ-glutamyltransferase (GGT) were found across multiple studies (Darrow et al., 2016; Gallo et al., 2012; Gleason et al., 2015; Nian et al., 2019; Salihovic et al., 2018; Yamaguchi et al., 2013).
    - Bassler et al., (2019) found positive associations between PFHxS, PFOA, and PFNA, and markers of hepatocyte apoptosis.
    - Studies (Christensen et al., 2019; Fitz-Simon et al., 2013; Fletcher et al., 2013; Maisonet et al., 2012) have consistently shown associations between PFAS exposure and lipid profile in blood including:
        - Increased: triglycerides, cholesterol, low density lipoprotein (LDL).
        - Reductions in: high density lipoprotein (HDL).
- **Cardiovascular effects**
    - There is suggestive epidemiological evidence for an association between serum PFOA and PFOS and pregnancy-induced hypertension and/or pre-eclampsia (ATSDR, 2018).
    - High levels of PFOA exposure in workers has been linked to high levels of uric acid and cholesterol (Nelson et al., 2010), risk factors for cardiovascular disease.
- **Obesity**
    - Of 22 Epidemiological studies, 15 found positive associations between at least one type of PFAS exposure and being overweight or obese (Qi et al., 2020).
        - Various studies have demonstrated the associations of at least one PFAS and increased waist circumference, body mass index (BMI), or weight gain (Cardenas et al., 2018; Christensen et al., 2019; Jaacks et al., 2016; Liu et al., 2018; Tian et al. 2019; Yang et al., 2018).
        - PFOS serum concentration levels may have a positive association with markers of obesity (BMI, and weight circumference) (Domazet et al., 2016; Liu, et al., 2018; Tian et al., 2019).
        - Liu et al., (2018), Tian et al., (2019), and Yang et al., (2018), and found blood levels of PFOA to be positively associated with weight gain/regain.
        - Serum concentration levels of PFNA have been shown to be positively associated with increased obesity markers (Christensen et al., 2019; Liu et al., 2018; Tian et al., 2019; Yang et al., 2018).
        - PFAS exposure in the general population, as well as maternal and childhood exposures, have been shown to be correlated with being overweight and obese (Qi et al., 2020).
- **Endocrine effects**
    - Epidemiology studies provide suggestive evidence of a link between serum PFOA and PFOS and an increased risk of thyroid disease (ATSDR, 2018).

    - In a cross-sectional analysis, Lopez-Espinosa et al. (2012) found that thyroid hormone levels may be affected by serum PFOS and PFNA concentrations, and that hypothyroidism may be associated with serum PFOA concentrations.
    - Tsai et al. (2016), found in that cord blood thyroid hormone levels are affected by PFAS, with a negative association between T4 and PFOS, and a positive association between TSH and PFOS.
- **Mammary gland biology**
    - Altered mammary gland growth from exposure to PFOA may lead to difficulty in breastfeeding and/or an increase in breast cancer later in life (Rudel et al., 2011; Macon & Fention, 2013).
- **Reproductive effects**
    - A suggestive link between serum PFOA and PFOS levels and an increased risk of decreased fertility has been found (ATSDR, 2018).
    - Evaluation of PFAS plasma concentrations in couples undergoing IVF treatment in Ma et al., (2020) showed that:
        - Female partner plasma PFOA concentrations were negatively associated with numbers of: mature oocytes, oocytes retrieved, 2 PN zygotes, and adequate embryos.
- **Kidney health**
    - There is a growing body of evidence to suggest that PFAS are an emerging environmental threat to kidney health (Stanifer et al., 2018):
    - PFAS exposure and kidney health identified 74 studies, including 21 epidemiological, 13 pharmacokinetic, and 40 toxicological studies. PFAS exposure altered several pathways linked to kidney disease.
    - Toxicology studies showing tubular histologic and cellular changes from PFAS exposure, as well as pharmacokinetic studies demonstrated the kidneys were major routes of elimination, with active proximal tubule transport.
- **Bone density**
    - In a representative sample of the U.S. adult population in NHANES 2009-2010, serum PFAS concentrations were associated with lower bone-mineral density, which varied according to the specific PFAS and bone site assessed. Most associations were limited to women. Osteoporosis in women was also associated with PFAS exposure, based on a small number of cases (Khalil et al., 2016).

What are the potential fetal, infant, and maternal health risks?

Developmental effects to fetuses during pregnancy or to breastfed infants include low birth weight, accelerated puberty, skeletal variations have been reported. Notable gaps in the research includes the lack of long-term epidemiological studies in children and adolescents who have greater exposure to PFAS over a lifetime.

Fetal & Maternal Risks

- Increased risk of pre-eclampsia has been associated with PFOA exposure (Stein et al., 2009; C8 Science Panel 2011a,b).
- Matilla-Santander et al. (2017) found that PFAS exposures during pregnancy may influence lipid metabolism and glucose tolerance, impacting the health of the mother and her child:
    - PFOS and PFHxS were positively associated with impaired glucose tolerance.
    - PFOS and PFHxS were positively associated with gestational diabetes mellitus.
    - PFOS and perfluorononanoate (PFNA) were negatively associated with triglyceride levels.
    - PFOA was positively associated with total cholesterol.
    - PFAS were not associated with C-reactive protein.
- Wikström et al., (2021) found that levels of PFOA in early pregnancy were associated with miscarriages in the second half of the first trimester.
- Oh et al., (2021) observed findings that suggest gestational exposure to PFOA and PFNA may increase risk for autism spectrum disorder (ASD) in children.

Birthweight

Evidence from a systematic review of 19 epidemiology studies suggest an inverse association between serum PFOA and PFOS and birth weight https://www.atsdr.cdc.gov/toxprofiles/tp200-c2.pdf.

- Several population-based reproductive outcome studies found statistically significant inverse relationships between exposure to PFOA &/or other PFAS with birthweight and other measures of fetal growth (Apelberg et al., 2007; Olsen et al., 2009; Starling et al., 2017; Washino et al., 2009).
- Fee et al. (2008) found associations between prenatal exposure to PFOS or PFOA and a range of adverse birth outcomes, such as low birth weight, decreased head circumference, reduced birth length, and smaller abdominal circumference.
- Multiple studies have reported an association between elevated maternal blood and cord blood concentrations of PFAS (primarily PFOS and PFOA) and decreased birth weight.
- One meta-analysis suggests that each 1 ng/mL increase in prenatal PFOA levels is associated with up to 18.9 g reductions in birth weight (Johnson, 2014).
- One study revealed a statistically significant association between PFOS and LBW risk (Stein 2009); no studies have found a statistically significant association between PFOA and LBW risk
- https://www.atsdr.cdc.gov/pfas/docs/clinical-guidance-12-20-2019.pdf

Immunosuppression

Elevated exposures to PFAS were associated with induced vaccine-reduced immune protection in children.

- Grandjean et al. (2012) reported that elevated exposures to PFCs were associated with reduced humoral immune response to routine childhood immunizations in children aged 5 and 7 years.
- Haug et al. (2013) reported that PFAS concentrations were associated with reduced antibody levels to the rubella vaccine, suggesting that pre-natal exposure to various PFAS may lead to immunosuppression in early childhood.
- Stein et al. (2015) found that increased exposure to several PFAS was associated with lower levels of mumps and rubella antibody concentrations, possibly indicating a less-robust response to vaccination or greater waning of vaccine-derived immunity over time.
- Heilmann et al. (2017) offered additional evidence on PFAS-associated, deficient antibody responses. They hypothesized that since PFAS are excreted in human milk, serum concentrations in infancy can be estimated from serum analyses at birth and the duration of exclusive breastfeeding. ` Both prenatal exposures and estimated serum levels in early infancy showed clear inverse associations with antibody concentrations against two vaccines at age 5 years. Study results support the high vulnerability of the developing immune system—and of infants—as an extremely vulnerable population.
- In evaluating data from the nationally representative U.S. National Health and Nutrition Examination Survey (NHANES) 1999-2016, (n = 8778), Bulka et al., (2021) explored increased susceptibility to persistent infections. These associations were independent of differences in sociodemographic characteristics and lifestyle factors including body mass index. Among the four PFAS evaluated, PFOS and PFOA were the most strongly tied to higher pathogen burdens:

    - Higher serum concentrations of PFOS in adolescents were positively associated with prevalence of HS1, *Toxoplasma gondii*, and *Toxocara* spp.
    - Positive associations were also seen in PFOA, PFHxS, and PFNA with *Toxocara* spp.

- Dalsager et al., (2021) identified a positive association between maternal serum PFAS and childhood hospitalizations related to infection.
    - The doubling of maternal serum PFOS concentrations increased the rate of childhood hospitalization (age studied: birth-4 years) related to an infection by 23%.
    - PFOS concentrations were associated with a 54% increase, and PFOA concentrations, a 27% increase in risk for hospitalizations due to a lower respiratory tract infection.

Endocrine Disruption

- Higher PFOS and PFNA concentrations are associated with lower levels of IGF-1 and testosterone in children ages 6 to 9 years. PFOS appears to be the most active of the

four compounds studied in affecting sex hormones and IGF-1 in young children (Lopez-Espinosa et al., 2016)
    - Specifically, Lopez-Espinosa et al. (2016) found that PFOS and PFNA concentrations were associated with lower levels of IGF-1 in boys and girls 6–9 years of age.
    - Results also suggest an inverse association between PFAS and sex hormones with a decrease in testosterone in both boys and girls for PFOS and for PFOA in boys.
    - PFOS was also inversely associated with estradiol in boys.
    - Sun et al. (2018) reported that plasma concentrations of PFOS and PFOA were associated with an elevated risk of type 2 diabetes.
- Guo et al., (2021) found that cord serum concentrations of PFAS mixtures were associated with thyroid function.
    - Specifically, cord serum PFAS concentrations were associated with higher TT4 and FT4 levels, and lower TSH levels.
        - PFOS, PFNA, and PFUnDA were the greatest predictors of TT4.
        - PFNA, PFOS, PFDA and PFHpS were the greatest predictors of FT4.
        - PFNA and PFHpS were the greatest predictors of TSH.

Cardiometabolic Disruption

- In a study evaluating PFAS exposures in women working or living near the World Trade Center on 9/11, Spratlen et al., (2019) found significant associations between concentrations of serum PFOS, PFOA, PFHxS, and cord lipids.
    - Specifically, a positive association was shown between PFOA and PFHxS concentrations and triglycerides.
- Results from Kingsley et al., (2019) suggest that PFAS alters lipid metabolism and interferes with biological pathways.
- Li et al., (2021) found that higher PFOA and PFHxS cord serum concentrations during gestation were positively associated with cardiometabolic risk scores in adolescence (12 years of age).
    - Cardiometabolic risk summary scores considered various factors:
        - Traditional: glucose, triglycerides, HDL, systolic and diastolic blood pressure, and waist circumference.
        - Novel: HOMA-IR, triglyceride to HDL ratio, adiponectin to leptin ratio, systolic blood pressure, and cross-sectional area of fat inside the abdominal cavity.
- Findings from Liu et al., (2020) suggest that gestational PFOA and PFHxS concentrations have a positive association with central adiposity and obesity risk in adolescents. Postnatal exposure/serum concentrations of PFOA and PFHxS did not demonstrate this pattern.
- A study focusing on the relationship between gestational PFAS exposure and BMI over the first 12 years of life, Braun et al., (2020) found that:
    - PFOA exposure during gestation was associated with BMI trajectories that indicate potential for adult obesity and cardiometabolic disease.

    - Higher PFOS and PFHxS exposure during gestation were associated with lower BMI.
- Braun et al., (2016), Hartman et al., (2017), Høyer et al., (2015), Lauritzen et al., (2018), and Mora et al., (2016) found a positive association between maternal PFAS exposure during pregnancy and obesity and overweight in school-aged children.
- In adolescents exposed to PFOA as children, Koshy et al., 2017 found a significant, positive association with triglycerides, total cholesterol, and LDL cholesterol. Furthermore:
    - PFHxS levels were associated with decreased insulin resistance;
    - PFOA and PFNA were associated with increased brachial artery distensibility.

What have we learned from animal studies?

In addition to the epidemiological study findings, studies on laboratory animals indicate that PFOA and PFOS can cause reproductive, developmental, hepatic, renal, and immunological effects. Both chemicals have caused tumors in animal studies.

PFOS and PFOA can cause health effects in laboratory animals related to:

- Altered gene expression and testosterone synthesis (Shi et al., 2007)
- Behavior (Ciu et al., 2009; Onischenko et al., 2010)
- Development (Lau et al., 2006),
- Reproductive (Fuentes et al., 2006)
- Neonatal mortality (Luebker et al., 2005)
- Hepatotoxicity, decreased immune function, and/or developmental effects in offspring exposed prenatally to PFOS and PFOA (Lindstrom et al.,, 2011; White et al., 2011)
- Increased liver weight (ATSDR, 2009; Ciu et al., 2009)
- Reduced immunological function (Dewitt et al., 2012)
- Adverse effects on mammary gland development in mice (Post, 2012)
- Neurodevelopment (Koskela, 2016)
- Skeletal development (Onishchenko, 2011)

How do people get exposed?

In the last decade, major manufacturers of PFOA and PFOS products joined EPA in a global stewardship program to phase out production of these agents by 2015. Although PFOA and PFOS are no longer manufactured in the United States, they are still produced internationally and can be imported to the United States in consumer goods such as carpet, leather and apparel, textiles, paper and packaging, coatings, rubber and plastics (USEPA, 2017).

Therefore, multiple PFAS can still be found in drinking water, food, dust, and personal care products, and a person can be concurrently exposed to a variety of PFAS. A single consumer product can contain as many as nine PFAS compounds (Fraser et al., 2013; Liu et al., 2014).

Where PFAS are found

- **Breastmilk**, while breast milk is the optimal food for infants, it provides a route for PFAS excretion for lactating mothers, and therefore, a route of exposure for breastfeeding infants (Koponen et al., 2018; Mondal 2014).
- **Commercial products**, including:
    - stain- and water-repellent fabrics
    - furniture, including mattresses
    - carpets treated for stain resistance
    - nonstick products (e.g., Teflon)
    - polishes, waxes
    - paints
    - cleaning products
    - fire-fighting foams (e.g., aqueous film-forming foam concentrates (AFFF) (a major source of groundwater contamination at airports and military bases where firefighting training occurs)
    - windshield washer fluids
    - some cosmetics and personal care products (shampoo, dental floss)
    - food-packaging materials
- **Workplace**, including production facilities or industries (e.g., chrome plating, electronics manufacturing, oil recovery, aerospace, automotive, electronic, and construction projects)
- **Drinking water**, typically localized and associated with a specific facility (e.g., manufacturer, landfill, wastewater treatment plant, military bases, firefighter training facility)
- **Fish**, where PFAS have the ability to accumulate and persist over time
- **Food**, packaged in PFAS-containing materials, processed with equipment that used PFAS, or grown in PFAS-contaminated soil or water.
- **Environmental residue** (i.e., air, dust, groundwater, soil)
- **Military bases**, where fire-fighting foam is used and can enter drinking water in nearby communities.

Pathways of Exposure

- *Water*
    - Exposure in infants (breast-fed or formula-fed) is higher than in adults using the same drinking water source due to PFOA's presence in breast milk and the greater drinking water intake of infants on a body-weight basis (Post et al., 2012).
    - PFAS contamination of ground water and/or surface water can lead to contamination of drinking water for both public and private drinking water systems, which represents an important source of ingestion exposure in affected populations (US EPA, 2016).

        - In 2013, the EPA began monitoring for PFAS (PFOA, PFOS, PFNA, PFHxS, PFBS, and PFHpA in water systems serving > 10,000 people under the Unregulated Contaminant Monitoring Rule 3 (UCMR3). A single public water system can contain levels above the reporting levels. Approximately 1% of the 5,000 public water systems monitored from 2013 to 2015 had combined PFOA and PFOS concentrations above the EPA Lifetime Health Advisory Level (HAL) of 0.07 µg/L (EPA 2016).
- *Food*
    - PFAS are persistent and known to be contaminants in waterways and can bio-accumulate within fish; fish consumption represents a common exposure route (Christiansen et al., 2017; Egeghy and Lorber, 2011; Trudel et al., 2008).
    - PFAS can migrate from food packaging to food (e.g., microwave popcorn, pizza, and fast food). In January 2016 the Food and Drug Administration amended its regulations to ban PFOA and PFOS in food packaging, which will likely decrease one source of non-drinking water exposure (EPA, 2016).
        - Until recently, consumption of food in PFAS-containing packaging (e.g., popcorn bags, fast-food containers, and pizza boxes) was prevalent. PFAS compounds have been largely phased out of food-packaging materials.
- *Behaviors*
    - Personal behaviors may be important determinants of PFAS exposures (Siebenaler et al., 2017).
        - Serum PFHxS levels were elevated in study participants who reported eating more microwavable foods and in those who vacuumed less often.
        - n addition, the use of water filtration devices was associated with lower levels of PFOA, but higher levels of PFHxA.
        - Boronow et al., 2018 found that in African Americans, frequent consumption of prepared food in coated cardboard containers was associated with higher levels of four PFAS.
        - Flossing with certain brands (Oral-B Glide), having stain-resistant carpet or furniture, were also associated with higher levels of some PFAS (Boronow et al., 2018).
- *Dust*
    - Because of the high number of household and consumer products that contain PFAS (e.g., stain-resistant carpet, textiles), dust ingestion is another route of exposure to consider for young children, who spend a high percentage of time on the floor and have high hand-to-mouth contact (Haug et al., 2011; Shoeib et al., 2011).
    - The use of stain repellant was associated with PFHxS and PFOA concentrations at age 8 years (Kingsley et al., 2018).
    - One investigation found that PFAS may be ubiquitous contaminants in US homes (Knobeloch et al., 2012).

    - Another study found that consumer products contribute most to PFCs in house dust (Minnesota Department of Health, 2014).
- *Air*
    - Wang et al. (2021) concluded that air inhalation may be as important as dust ingestion. An estimated daily intake (EDI) of PFOA via indoor air inhalation was and compared to the strictest tolerable daily intake (TDI) of PFOA (≤0.63 ng/kg bw/day). Potential health risk occurred in the best-case scenario.
- *Occupational exposure*
    - Workers in industries that manufacture or use materials/products containing PFAS may have even greater exposure (ATSDR, 2018; Tanner et al., 2018).
- *Garden soil*
    - PFAS can be released to the environment using PFAS-contaminated water in gardens. Clean water added to the garden may lower the soil concentrations of PFAS (Vermont Agency of Agriculture, Food and Markets, 2016).
    - PFAS can be taken up into plants grown on contaminated soil (e.g., from application of contaminated sewage sludge) or contaminated water. Different types of produce take up different amounts of PFAS from the soil (Yoo et al., 2011).
    - Laboratory studies consistently show that PFAS with short fluorocarbon-chains— such as PFBS—are more readily taken up into plants compared to long-chain PFAS such as PFOS or PFOA. (http://www.health.state.mn.us/divs/eh/hazardous/topics/pfcs/pihgssumm.pdf).
- *Dermal contact*
    - Showering and bathing in water containing PFAS should not increase exposure. Washing dishes in water containing PFAS should not increase exposure (ATSDR, 2018).
    - People can be exposed from dermal contact with carpets or clothing, although this is not considered a primary route of exposure based on PFAS dermal absorption properties. This pathway is of higher concern for young children routinely crawling on treated carpets and who have higher rates of hand-to-mouth activity.
        - Analysis of NHANES 2005-2006 data showed a possible association between low pile carpeting and an increase in PFHxS and MeFOSAA (a precursor to PFOS) serum concentrations (Zhu et al., 2021).

Special exposure risks for fetuses, newborns, and children

- *Breastfeeding*
    - Previous breastfeeding is both a major elimination route for PFAS and a predictor of breastfeeding practices in the current pregnancy (Thomsen et al., 2011; Brantsæter et al., 2013; Mondal et al., 2014; Hackman et al., 2015).

    - Inverse associations between PFOA and PFOS exposure and duration of breastfeeding were found (Fei et al., 2010; Romano, et al., 2016; Timmermann et al., 2017).
    - Breastfeeding duration was positively correlated ($p < 0.001$) with serum concentrations of PFHxS, PFOS, PFOA and PFNA at 1 year of age (Koponen et al., 2018). Kingsley et al. (2018) also found that length of breastfeeding was positively associate with PFAS concentrations in children at ages 3 and 8 years.
    - Rosen et al. (2018) examined the association between prenatal PFAS exposure and duration of breastfeeding. Inverse associations of PFNA, PFDA, and PFUnDA and breastfeeding cessation were observed, as were observed positive associations with PFOS and breastfeeding cessation, but only when accounting for other exposures.
- *Pregnancy*
    - Recent research evaluating possible health effects to fetuses from PFAS exposures have shown that developing fetuses can be exposed when PFAS in maternal blood crosses the placenta and reaches umbilical cord blood. Different PFAS have varying levels of permeability to the placental barrier.
    - Maternal serum PFAS concentrations during pregnancy were strongly correlated with cord serum concentrations (Kingsley et al., 2018).
    - Serum PFAS concentrations in children were associated with mother's age at delivery, parity, race, and child age (Kingsley et al., 2018).

Reducing exposures

Drinking water

A number of options are available to people using public drinking water systems to lower concentrations of PFAS in their drinking water supply. www.nsf.org/newsroom/nsf-international-certifies-first-water-filters-pfoa.

- Public water systems can treat source water with activated carbon or high-pressure membrane systems (e.g., reverse osmosis) to remove PFAS from drinking water. Some home filters remove impurities using granular activated carbon (GAC) and reverse osmosis (RO) (USEPA, 2016).
    - Anumol et al (2015) studied the removal efficiency of various point-of-use devices, and found they can reduce PFOA/PFOS concentrations.
    - A 2017 fact sheet from Michigan includes info about the home filter certification program: https://www.michigan.gov/documents/deq/deq-dwmad-eh-swpu-FilterFactSheet_610096_7.pdf.

Based on studies, there currently are three general types of filtration systems that can reduce PFAS levels in water, if properly maintained:

- Granulated activated carbon (GAC) – either in refrigerator, faucet, or pitcher filters, and some whole-house filtration systems installed on your water line
- Reverse osmosis (RO)
- GAC and RO used together
- Note: Ion exchange (including water softening) and distillation water treatment systems will not reduce PFASs to low levels.

NSF has certified some point-of-use (POU) filters for PFOA and PFOS reduction. Information about these filters can be found at https://www1.villanova.edu/university/nursing/macche.html

Water testing

EPA suggest using a lab that is approved to use the EPA 537 method to detect PFAS. Here is the EPA link to suggested laboratories.

https://www.epa.gov/sites/production/files/2016-10/documents/ucmr3-lab-approval.pdf

What are other ways to reduce exposures to PFAS?

Infant Formula

- To reduce potential exposure to infants, caregivers should use pre-mixed baby formula, or reconstitute using water that is not known to be contaminated with PFAS.
- PFAS can migrate from a mother's blood into her breast milk. However, concentrations of PFAS in the breast milk are lower than in the mother's blood. Currently, the benefits of breastfeeding are expected to outweigh any health effects from potential PFAS exposure via breast milk.

Cooking

- If PFAS have been found in drinking water above the recommended level, use bottled or filtered water for cooking and food preparation. Bottled water treated by reverse osmosis and GAC not contain PFAS. Unless otherwise noted, spring water is not treated to remove chemicals.

Fish

- The consumption of fish is thought to be a potential pathway for PFAS exposures. Be aware of regional fish advisories if eating locally caught fish.
    - While a federal screening level or toxicity value for the consumption of fish has not yet been established, the Dutch National Institute for Public Health and the Environment has calculated a maximum permissible concentration for PFOS of 0.65 nanograms per liter (ng/L) for fresh water (based on consumption of fish by humans as the most critical route) (Moermond, 2010).

- Some states have fish consumption advisories for certain water bodies where PFOS has been detected in fish (MDH 2017; MDHHS 2016).
- Many state health departments also provide site-specific meal advice for tested lakes and rivers (e.g., http://www.health.state.mn.us/divs/eh/fish/eating/sitespecific.html; Minnesota Department of Health and Human Services, 2018).

Consumer Products

- Read consumer product labels and avoid purchasing or using those with PFAS:
  - Stain-resistant carpets, non-stick pans, waterproof garments, some types of furniture and clean products.

What are the current screening levels for PFAS?

EPA Health Advisory Levels

Although US EPA has not issued a maximum contaminant level (MCL) for drinking water, the agency did establish a drinking water lifetime health advisory level (HAL) in May, 2016.

The EPA HAL is set at 70 parts per trillion (0.07 micrograms per liter [µg/L]) for the combined concentrations of PFOS and PFOA. Above these levels, EPA recommends that public drinking water systems take steps to assess contamination, inform consumers, and limit exposure (EPA 2016b, 2016c, 2017).

These levels were based upon the exposure to the chemical for 70 years and assumes that individual exposure to PFAS is derived from drinking water (20%) and home and environmental (80%) sources. The EPA HAL is based on a drinking water intake of more than 3.0 L/day for lactating women, who often drink more water than other people and can pass chemicals to breastfed infants.

HAL is not intended to be a definitive health-effect level, but to provide a margin of protection over a lifetime.

HAL includes uncertainty factors, offering a margin of protection against adverse health effects to the most sensitive populations (fetuses during pregnancy and breastfed infants) https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final_508.pdf.

EPA health advisories are non-regulatory recommendations and are not legally enforceable https://www.gpo.gov/fdsys/pkg/FR-2016-05-25/pdf/2016-12361.pdf.

The EPA HAL is subject to change as new information becomes available (EPA, 2016) https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos.

The EPA UCMR is a program to collect nationally representative data for contaminants suspected to be present in public drinking water, but that do not have regulatory

standards. PFAS are not included on the EPA's fourth UCMR sampling, which began in 2018 and will continue through 2020.

To learn more about the underlying studies for the health advisories, see EPA's Health Effects Support Documents for PFOA and PFOS.

The Agency for Toxic Substances and Disease Registry (ATSDR) Minimum Risk Levels

ATSDR is a federal public health agency, with a role in evaluating whether exposures to contaminants pose a health threat, has draft screening values or minimal risk levels (MRLs) for PFAS. ATSDR MRLs are an estimate of the amount of a chemical a person can eat, drink, or breathe each day without a detectable risk to health. MRLs are intended to serve as a tool to help public health professionals. If an exposure is above an MRL, ATSDR conducts further evaluation to determine if the exposure might harm human health. MRLs do not define regulatory or action levels for ATSDR. ATSDR has developed MRL screening values for PFOA, PFOS, PFHxS, and PFNA that can be converted into drinking water concentrations for adults and children.

- PFOA: 78 ppt (adult) and 21 ppt (child)
- PFOS: 52 ppt (adult) and 14 ppt (child)
- PFHxS: 517 ppt (adult) and 140 ppt (child)
- PFNA: 78 ppt (adult) and 21 ppt (child)

ATSDR bases these calculations on an infant (birth to one year of age) weighing 7.8 kg and an intake rate of 1.113 liters per day. For an adult's drinking water exposure, ATSDR bases this calculation on a body weight of 80 kg and an intake rate of 3.092 liters per day.

Additional information on ATSDR MRLs can be found here:
https://www.atsdr.cdc.gov/pfas/mrl_pfas.html

Is there medical testing for PFAS?

If there are concerns about PFAS exposure, a blood sample can test for the level of PFAS in the body. However, health effects from PFAS at low environmental doses or at bio-monitored levels from low environmental exposures are unknown.

There currently is no established blood level of PFASs, and the tests cannot predict past or future health problems. Blood tests for PFAS are most useful when they are part of a scientific investigation or a health study.

Currently, because PFAS are an emerging contaminant, clinical laboratories do not typically have testing methods for PFAS.

A few laboratories provide blood testing for PFAS and can be found at:
https://www1.villanova.edu/university/nursing/macche.html

Note: In the U.S., the CPT Code for PFOA (C---8, 2014) levels testing is 8254.

Are there any medical treatments for removing PFAS in my patient's blood?

- Currently, there are no medically approved treatments for removing PFAS from the body.
- Reducing exposures are the best remedy. Offer guidance on how to reduce exposures in consumer products and drinking water and perform a thorough history and physical exam.

A fact sheet on Guidance for Clinicians, including responses to common questions, can be found at: https://www.atsdr.cdc.gov/pfas/docs/clinical-guidance-12-20-2019.pdf.

For Citations and Additional Resources Contact:

The Mid-Atlantic Center for Children's Health and the Environment
Phone: 833-362-2243 (toll free)
Email: macche@villanova.edu

Updated: May 2021

Appendix

Serum Perfluorooctane sulfonic acid (PFOS) (2013 – 2014)‡

Geometric mean and selected percentiles of serum concentrations (in µg/L) for the U.S. population from the National Health and Nutrition Examination Survey.

Special Sample of Serum PFAS in Children 3 to 11 Years Old, 2013 – 2014

Categories	Survey years	Geometric mean (95% conf. interval)	50th Percentile (95% conf. interval)	75th Percentile (95% conf. interval)	90th Percentile (95% conf. interval)	95th Percentile (95% conf. interval)	Sample size
Total	13-14	**3.88** (3.53-4.27)	**3.75** (3.44-4.17)	**5.56** (4.83-6.33)	**7.99** (7.02-9.53)	**11.0** (9.03-12.4)	639
Age group							
3-5 years	13-14	**3.38** (3.04-3.77)	**3.41** (2.84-3.78)	**4.78** (3.98-6.32)	**7.18** (5.50-8.71)	**8.82** (7.18-11.0)	181
6-11 years	13-14	**4.15** (3.76-4.58)	**4.02** (3.54-4.45)	**5.77** (5.10-6.43)	**8.78** (6.75-11.8)	**12.4** (9.32-14.1)	458
Gender							
Males	13-14	**4.07** (3.56-4.65)	**4.13** (3.44-4.76)	**6.19** (5.29-7.18)	**8.78** (7.18-11.8)	**11.8** (8.01-15.4)	343
Females	13-14	**3.70** (3.38-4.06)	**3.54** (3.24-3.96)	**4.88** (4.45-5.70)	**7.02** (6.33-8.71)	**9.44** (7.17-12.0)	296
Race/ethnicity							
All Hispanics	13-14	**3.53** (3.13-3.99)	**3.41** (2.95-3.84)	**4.71** (4.13-5.65)	**7.60** (5.43-9.44)	**9.32** (6.50-11.0)	220
Other	13-14	**4.01** (3.62-4.44)	**3.96** (3.44-4.48)	**5.97** (5.02-6.49)	**8.06** (6.98-11.3)	**12.0** (9.10-13.6)	419

‡See Calculation of PFOS and PFOA as the Sum of Isomers for additional information.

Biomonitoring Summary

http://www.cdc.gov/biomonitoring/PFAS_BiomonitoringSummary.html

Factsheet

http://www.cdc.gov/biomonitoring/PFAS_FactSheet.html

Categories	Survey years	Geometric mean (95% conf. interval)	50th Percentile (95% conf. interval)	75th Percentile (95% conf. interval)	90th Percentile (95% conf. interval)	95th Percentile (95% conf. interval)	Sample size
Total	11-12	**2.08** (1.95-2.22)	**2.08** (1.96-2.26)	**3.03** (2.76-3.27)	**4.35** (3.82-4.85)	**5.68** (5.02-6.49)	1904
	13-14‡	**1.94** (1.76-2.14)	**2.07** (1.87-2.20)	**3.07** (2.67-3.37)	**4.27** (3.57-5.17)	**5.57** (4.60-6.27)	2165
Age group							
12-19 years	11-12	**1.80** (1.71-1.91)	**1.74** (1.67-1.89)	**2.41** (2.17-2.62)	**2.93** (2.68-3.19)	**3.59** (2.93-4.25)	344
	13-14‡	**1.66** (1.50-1.84)	**1.67** (1.37-1.97)	**2.20** (1.97-2.57)	**2.87** (2.57-3.40)	**3.47** (2.87-4.37)	401
20 years and older	11-12	**2.12** (1.98-2.28)	**2.16** (2.01-2.33)	**3.15** (2.90-3.36)	**4.64** (3.93-5.25)	**5.94** (5.34-7.45)	1560
	13-14‡	**1.98** (1.79-2.19)	**2.07** (1.90-2.27)	**3.17** (2.77-3.47)	**4.47** (3.70-5.27)	**5.60** (4.67-6.40)	1764
Gender							
Males	11-12	**2.37** (2.22-2.53)	**2.38** (2.26-2.56)	**3.25** (3.00-3.56)	**4.61** (4.11-5.02)	**5.62** (4.85-6.20)	966
	13-14‡	**2.29** (2.09-2.50)	**2.37** (2.17-2.57)	**3.27** (2.87-3.60)	**4.67** (3.77-5.60)	**5.67** (4.67-6.27)	1031
Females	11-12	**1.84** (1.68-2.01)	**1.78** (1.62-1.98)	**2.65** (2.34-3.14)	**3.91** (3.36-4.99)	**5.68** (4.33-8.45)	938
	13-14‡	**1.66** (1.48-1.87)	**1.67** (1.47-1.87)	**2.67** (2.27-3.07)	**3.77** (3.37-4.70)	**5.07** (4.07-6.70)	1134
Race/ethnicity							
Mexican Americans	11-12	**1.66** (1.37-2.02)	**1.71** (1.32-2.23)	**2.43** (1.98-2.98)	**3.38** (2.43-4.48)	**4.08** (2.98-6.15)	211
	13-14‡	**1.36** (1.25-1.47)	**1.37** (1.27-1.47)	**1.97** (1.87-2.10)	**2.70** (2.40-3.10)	**3.17** (2.57-3.77)	332
Non-Hispanic blacks	11-12	**1.80** (1.71-1.90)	**1.94** (1.76-2.09)	**2.82** (2.65-2.95)	**3.94** (3.51-4.40)	**5.11** (4.40-5.79)	485
	13-14‡	**1.52** (1.34-1.73)	**1.67** (1.37-1.97)	**2.57** (2.17-2.97)	**3.60** (3.07-4.50)	**4.60** (3.40-5.77)	455
Non-Hispanic whites	11-12	**2.25** (2.05-2.47)	**2.25** (1.98-2.48)	**3.21** (2.90-3.50)	**4.68** (3.95-5.35)	**6.20** (5.34-7.74)	666
	13-14‡	**2.20** (1.91-2.52)	**2.27** (1.97-2.67)	**3.37** (2.77-3.77)	**4.77** (3.77-5.67)	**5.77** (4.80-6.87)	861
All Hispanics	11-12	**1.70** (1.48-1.95)	**1.79** (1.59-1.95)	**2.46** (2.15-2.91)	**3.60** (2.95-4.48)	**4.70** (3.87-5.94)	406
	13-14‡	**1.45** (1.33-1.59)	**1.47** (1.37-1.67)	**2.10** (1.97-2.40)	**3.07** (2.67-3.27)	**3.47** (3.17-3.97)	537
Asians	11-12	**2.08** (1.83-2.36)	**2.21** (2.04-2.27)	**2.92** (2.55-3.45)	**4.66** (3.42-5.79)	**5.79** (4.93-8.91)	291
	13-14‡	**1.97** (1.75-2.23)	**1.87** (1.67-2.27)	**2.97** (2.47-3.57)	**4.67** (3.97-5.77)	**5.90** (5.00-6.40)	234

Limit of detection (LOD, see Data Analysis section) for Survey year 11-12 is 0.1.
‡See Calculation of PFOS and PFOA as the Sum of Isomers for additional information about Survey years 2013-2014.

Serum Perfluorooctane sulfonic acid (PFOS) (2013 – 2014)‡

Geometric mean and selected percentiles of serum concentrations (in µg/L) for the U.S. population from the National Health and Nutrition Examination Survey.

Special Sample of Serum PFAS in Children 3 to 11 Years Old, 2013 – 2014

Categories	Survey years	Geometric mean (95% conf. interval)	50th Percentile (95% conf. interval)	75th Percentile (95% conf. interval)	90th Percentile (95% conf. interval)	95th Percentile (95% conf. interval)	Sample size
Total	13-14	**3.88** (3.53-4.27)	**3.75** (3.44-4.17)	**5.56** (4.83-6.33)	**7.99** (7.02-9.53)	**11.0** (9.03-12.4)	639
Age group							
3-5 years	13-14	**3.38** (3.04-3.77)	**3.41** (2.84-3.78)	**4.78** (3.98-6.32)	**7.18** (5.50-8.71)	**8.82** (7.18-11.0)	181
6-11 years	13-14	**4.15** (3.76-4.58)	**4.02** (3.54-4.45)	**5.77** (5.10-6.43)	**8.78** (6.75-11.8)	**12.4** (9.32-14.1)	458
Gender							
Males	13-14	**4.07** (3.56-4.65)	**4.13** (3.44-4.76)	**6.19** (5.29-7.18)	**8.78** (7.18-11.8)	**11.8** (8.01-15.4)	343
Females	13-14	**3.70** (3.38-4.06)	**3.54** (3.24-3.96)	**4.88** (4.45-5.70)	**7.02** (6.33-8.71)	**9.44** (7.17-12.0)	296
Race/ethnicity							
All Hispanics	13-14	**3.53** (3.13-3.99)	**3.41** (2.95-3.84)	**4.71** (4.13-5.65)	**7.60** (5.43-9.44)	**9.32** (6.50-11.0)	220
Other	13-14	**4.01** (3.62-4.44)	**3.96** (3.44-4.48)	**5.97** (5.02-6.49)	**8.06** (6.98-11.3)	**12.0** (9.10-13.6)	419

‡See Calculation of PFOS and PFOA as the Sum of Isomers for additional information.

Biomonitoring Summary

http://www.cdc.gov/biomonitoring/PFAS_BiomonitoringSummary.html

Factsheet

http://www.cdc.gov/biomonitoring/PFAS_FactSheet.html

Categories	Survey years	Geometric mean (95% conf. interval)	50th Percentile (95% conf. interval)	75th Percentile (95% conf. interval)	90th Percentile (95% conf. interval)	95th Percentile (95% conf. interval)	Sample size
Total	11-12	**6.31** (5.84-6.82)	**6.53** (5.99-7.13)	**10.5** (9.78-11.1)	**15.7** (14.7-17.5)	**21.7** (19.3-23.9)	1904
	13-14‡	**4.99** (4.50-5.52)	**5.20** (4.80-5.70)	**8.70** (7.90-9.40)	**13.9** (11.9-15.5)	**18.5** (15.4-22.0)	2165
Age group							
12-19 years	11-12	**4.16** (3.70-4.68)	**4.11** (3.48-4.65)	**5.90** (5.14-7.25)	**9.05** (6.49-10.8)	**10.8** (8.52-14.2)	344
	13-14‡	**3.54** (3.17-3.96)	**3.60** (3.10-4.20)	**5.20** (4.60-6.20)	**7.80** (7.00-8.90)	**9.30** (7.90-11.7)	401
20 years and older	11-12	**6.71** (6.24-7.20)	**7.07** (6.65-7.52)	**11.0** (10.4-11.9)	**17.0** (15.3-18.5)	**22.7** (20.4-24.8)	1560
	13-14‡	**5.22** (4.70-5.81)	**5.60** (5.10-6.00)	**9.10** (8.20-10.2)	**14.5** (12.9-16.1)	**19.5** (15.8-23.0)	1764
Gender							
Males	11-12	**7.91** (7.19-8.70)	**8.31** (7.35-9.15)	**12.5** (11.4-13.5)	**19.3** (15.7-21.4)	**24.1** (22.2-28.5)	966
	13-14‡	**6.36** (5.62-7.20)	**6.40** (5.70-7.30)	**10.2** (8.70-11.5)	**15.5** (13.2-19.8)	**22.1** (16.7-26.9)	1031
Females	11-12	**5.10** (4.70-5.53)	**5.27** (4.67-5.64)	**8.57** (7.87-9.30)	**12.5** (11.0-14.9)	**17.5** (14.9-20.5)	938
	13-14‡	**3.96** (3.60-4.35)	**4.00** (3.60-4.60)	**7.20** (6.40-7.70)	**11.8** (9.70-13.6)	**15.1** (13.9-17.3)	1134
Race/ethnicity							
Mexican Americans	11-12	**4.79** (4.07-5.64)	**5.18** (3.92-6.33)	**7.91** (6.18-9.48)	**10.5** (8.50-12.6)	**12.1** (10.0-14.4)	211
	13-14‡	**3.47** (2.90-4.16)	**3.70** (3.00-4.40)	**5.20** (4.60-6.40)	**8.80** (6.40-10.3)	**10.8** (9.20-11.8)	332
Non-Hispanic blacks	11-12	**6.35** (5.41-7.46)	**6.57** (5.71-7.65)	**11.3** (9.74-13.9)	**21.8** (13.9-31.3)	**30.7** (21.6-45.1)	485
	13-14‡	**5.32** (4.12-6.88)	**5.30** (4.30-6.80)	**10.2** (7.60-13.7)	**17.4** (12.4-24.5)	**24.5** (16.3-39.7)	455
Non-Hispanic whites	11-12	**6.71** (6.15-7.32)	**6.83** (6.07-7.73)	**10.7** (9.89-12.2)	**15.7** (14.8-18.1)	**21.3** (18.7-23.5)	666
	13-14‡	**5.31** (4.72-5.98)	**5.70** (5.10-6.40)	**8.90** (8.20-9.90)	**14.1** (12.2-15.6)	**18.0** (15.5-20.4)	861
All Hispanics	11-12	**4.63** (3.86-5.55)	**5.18** (4.41-6.19)	**8.10** (6.64-9.78)	**11.0** (9.96-12.6)	**13.4** (11.5-16.1)	406
	13-14‡	**3.51** (3.09-3.98)	**3.70** (3.20-4.20)	**5.50** (4.90-6.40)	**8.80** (8.00-9.70)	**10.8** (9.70-12.1)	537
Asians	11-12	**7.10** (5.80-8.68)	**7.53** (5.96-9.25)	**12.6** (10.8-17.0)	**24.6** (19.1-33.3)	**35.1** (26.4-42.3)	291
	13-14‡	**6.18** (5.08-7.52)	**6.30** (5.00-7.90)	**13.2** (9.40-15.4)	**23.8** (15.2-33.9)	**33.6** (20.1-69.0)	234

Limit of detection (LOD, see Data Analysis section) for Survey year 11-12 is 0.2.
‡.See Calculation of PFOS and PFOA as the Sum of Isomers for additional information about Survey years 2013-2014.