



Factsheet on Perfluorinated Chemicals (PFCs) for Health Professionals*

What are PFCs?

PFC stands for perfluorinated chemical. The thousands of compounds classified within this branch of substances are resistant to stains, heat, oil, grease, and water, and also act as lubricants. PFCs are widely used to make everyday consumer products. For example, PFCs may be used to keep food from sticking to cookware, to make sofas and carpets resistant to stains, to make clothes and mattresses more waterproof, and may also be used in some food packaging, as well as in some firefighting materials. Because they help reduce friction, they are also used in a variety of other industries, including aerospace, automotive, building and construction, and electronics (NIEHS, 2012).

Scientists refer to PFCs in a number of ways; therefore, look for the following terms when attempting to discern whether or not a substance falls under this category:

- Perfluorinated chemicals
- Polyfluorinated compounds
- Perfluorochemicals
- Polyfluorinated chemicals
- Polyfluoroalkyl substances
- Perfluoroalkyls
- Perfluorinated alkyl acids

PFCs (including PFOS, PFOA, PFHxS, and PFNA) were detected in 98% of a representative sample of U.S. residents in a National Health and Nutrition Examination Survey conducted in 2003-2004 (Calafat *et al.* 2007).

Where are PFCs found?

PFCs are not found naturally, are chemically stable, and persistent in the environment.

Because of their widespread use, most people in the United States have some PFCs in their body. Data from human studies suggest that some PFCs can take years to be cleared from the body (Bartell *et al.*, 2010; Seals *et al.*, 2010). Once the PFCs are in a person's body, it takes from 2 to 9 years before PFC levels go down by half, even if no more is taken in or produced. This half-life results in continued exposure that

could increase body burdens to levels that would result in adverse outcomes (ATSDR 2009; Olsen, 2007).

Potential pathways, which may lead to widespread exposure, include ingestion of food and water, use of commercial products or inhalation from long-range air transport of PFC-containing particulate matter (ATSDR 2009). One primary source of exposure is drinking water (Rumsby et al 2009). Other common sources of exposure include (Fromme et al., 2009):

- Food containers (i.e. pizza boxes, fast food wrappers, popcorn bags)
- Furniture, including mattresses
- Carpets treated for stain resistance
- Water-proof clothing and accessories
- Non-stick cookware (e.g., Teflon)
- Firefighting foams
- Windshield washer fluids
- Aerospace, automotive, electronic, and construction projects
- Environmental residue (i.e. air, dust, groundwater, soil)

Ingestion

- PFOAs are persistent, bio-accumulative, and known to be contaminants in waterways and can bio-accumulate within fish; fish consumption represents a common exposure route (Egeghy and Lorber, 2011; Trudel et al., 2008). 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). Michigan has developed provisional fish consumption screening value ranges for PFOS – See “Michigan Fish Consumption Advisory Program” Guidance Document, available under “Reports and Science” at www.michigan.gov/eatsafefish.
- PFCs can migrate from food packaging to food (with concentrations occurring in high-fat foods, such as microwave popcorn and fast food).
- Traditional drinking water treatment techniques do not remove long chained (LC)-PFCs, so contamination of ground water or surface water, or both, can lead to contamination of drinking water (ATSDR, 2009).
- Because of the high number of household and consumer products that contain PFCs (e.g., 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).

- PFOS and PFOA are unlikely to be taken up by plant roots via contaminated water. However, in one study PFCs were taken up into plants (grasses and barley) grown on contaminated soil (e.g., from application of contaminated sewage sludge). Plant uptake of PFCs decreased with chain length with the shortest PFC in study (C6) having the greatest uptake (Yoo et al., 2011).

Dermal Contact

- People can be exposed from dermal contact with carpets or clothing, although this is not considered a primary route of exposure.

What are the potential health effects of PFCs?

Studies have found PFOS and PFOA in the blood samples of the U.S population, indicating that exposure to the chemicals is widespread (ATSDR 2009; EPA 2006a). In 2003-2004, PFOS and PFOA were detected in 99-100% of blood samples collected from both pregnant and non-pregnant women (Woodruff et al., 2011). Current PFOS and PFOA levels can be found in the 2015 report: http://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Feb2015.pdf. Tables with breakdowns by age, ethnicity, and gender are available in the Appendix at the end of this factsheet.

While current evidence is compelling, causality has not been definitively established for a wide range of health effects. Many uncertainties and data gaps remain and will require further research.

The most consistent findings from epidemiology studies are elevated blood serum total cholesterol levels among exposed populations, with strong evidence for a causal relationship between PFOA exposure and elevations in serum lipids (C8 Science Panel, 2011c).

- General population cross-sectional studies found associations with several health endpoints including increased serum cholesterol (Nelson et al., 2009) and uric acid (Shankar, et al., 2011 a), increased incidence of thyroid disease (Melzer et al., 2010), increased serum liver enzymes (Lin et al., 2010) and decreased renal glomerular filtration (Shankar et al, 2011 b).
- In May 2006, the EPA Science Advisory Board suggested that perfluorooctanoic acid (PFOA) cancer data are consistent with the EPA guidelines for the Carcinogen Risk Assessment descriptor “likely to be carcinogenic to humans.” EPA is still evaluating this information and additional research pertaining to the carcinogenicity of PFOA (USEPA 2006, 2014). In 2011, the Institute of Medicine concluded that currently available data suggests the possibility of a link with breast cancer and PFOA exposure.
- Epidemiologic 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).

- 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.
- PFOS-exposed workers have demonstrated elevated incidence of bladder cancer mortality following at least one year of exposure (Lindstrom et al., 2011).
- Epidemiologic studies have shown an association between PFOS exposure and bladder cancer; however, further research and analysis are needed to understand this association (Alexander, 2003; Lau, 2007).
- Several population-based reproductive outcome studies found statistically significant inverse relationships between birthweight or other measures of fetal growth and PFOA and/or other PFCs (Washino et al., 2009; Olsen et al., 2009; Apelberg et al., 2007). Fee et al. (2008) studying the Danish National Birth Cohort 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.
- 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).
- Elevated exposures to PFCs were associated with reduced vaccine-induced immune protection in children (Grandjean et al., 2012).
- Increased risk of pre-eclampsia was associated with PFOA exposure (Stein et al., 2009; C8 Science Panel 2011a,b).
- Exposure has been linked to endocrine disruption (Casal-Casas et al., 2011).

Animal Studies

In laboratory studies of animals given large doses of PFCs, results indicate that PFOS and PFOA can cause health effects related to:

- Altered gene expression and testosterone synthesis (Shi et al., 2007)
- Behavioral (Ciu et al., 2009; (Onischenko et al., 2010),
- Reproductive, (Fuentes et al., 2006)
- Neonatal mortality (Luebker et al., 2005)
- 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).

Is there medical testing for PFCs?

If there are concerns about PFC exposure, due to proximity to a plant that uses PFCs or due to verified knowledge that their water or environment contains potentially unhealthy levels of PFCs, a blood sample can test for the level of PFCs in the body.

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

Currently, private U.S. clinical laboratories do not perform analyses for other PFCs beyond PFOA. However, a Canadian laboratory provides services for both PFOS and PFOA (http://www.axysanalytical.com/services/emerging_contaminants/).

How can people reduce exposures to PFCs?

- Drinking water: Drinking water treatment for PFCs is complex and can be difficult to implement at a household level. However, a study by the Minnesota Department of Health found that some water filtration devices (point-of-use devices at a single tap, faucet, or outlet) may remove *some* PFCs from water (Olsen and Paulson, 2008). However, household treatment systems need to be carefully maintained to be effective (<http://www.health.state.mn.us/divs/eh/water/factsheet/com/pou.html>), and guidance for private well owners is not currently available regarding appropriate filter change out and maintenance for residential drinking water treatment for PFCs.
- To reduce potential exposure to infants, caregivers should use pre-mixed baby formula, or reconstitute using alternative water sources not containing PFOS and/or PFOA.
- Be aware of fish advisories within area if eating locally caught fish.
- Reduce exposure to consumer products containing PFCs.

What are the current regulatory levels for PFCs?

PFC concentrations are not currently regulated in food, water or air, though the EPA is evaluating some PFCs for potential regulatory action.

In 2009, the US EPA issued Provisional Health Advisory Levels for PFOA and PFOS in drinking water. These values were calculated assuming an exposure scenario in which a 10 kg child consumes 1 L/day of drinking water. The EPA selected an exposure scenario involving children because children consume more water than adults on a body weight basis, and thus will have a higher exposure to contaminants in drinking water on a body weight basis than adults (http://www.epa.gov/opptintr/existingchemicals/pubs/pfcs_action_plan1230_09.pdf)

These EPA advisory levels for drinking water are guidance values only. The EPA provisional drinking water guidelines are as follows: PFOA: 0.4 µg/L and PFOS: 0.2 µg/L.

In 2012, EPA signed a Significant New Use Rule for PFCs to limit their use and continues to evaluate the exposure to PFCs on children and other populations, which are more likely to be more sensitive to PFC exposures.

Where can I find more information?

Agency for Toxic Substances and Disease Registry (ATSDR)

<http://www.atsdr.cdc.gov/PHS/PHS.asp?id=1115&tid=237>

ATSDR TOXFAQs Link for Perfluoroalkyls:

<http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=1116&tid=237#bookmark05>

Centers for Disease Control and Prevention

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

Environmental Protection Agency (USEPA) : Emerging Contaminant: PFOA and PFOS

http://www.epa.gov/fedfac/pdf/emerging_contaminants_pfos_pfoa.pdf

EPA: Provisional Health Advisory Fact Sheet

http://water.epa.gov/action/advisories/drinking/upload/2009_01_15_criteria_drinking_pfa-PFOA_PFOS.pdf

EPA: Unregulated Contaminant Monitoring Sheet:

http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/upload/UCMR3_Fact_Sheet_General.pdf

EPA: Frequently Asked Questions page:

<http://www.epa.gov/oppt/pfoa/pubs/faq.html>

EPA: Warminster, PA

http://www.epa.gov/reg3hwmd/super/sites/PA6170024545/fs/Warminster-Horsham_Joint_Fact_Sheet_FINAL_2-20-15.pdf

EPA: America's Children and the Environment

<http://www.epa.gov/ace/pdfs/Biomonitoring-PFCs.pdf>

NIEHS: National Toxicology Program

<http://ntp.niehs.nih.gov/files/PFOAConcept.pdf>

The Mid-Atlantic Center for Children's Health and the Environment

kidsandenvironment@georgetown.edu or call toll free at: 1-866-622-2431

<https://kidsandenvironment.georgetown.edu>

References

Agency for Toxic Substances and Disease Registry (ATSDR). (2009). *Toxicological Profile for Perfluoroalkyls (Draft for Public Comment)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Alexander, B. H.; Olsen, G. W.; Burris, J. M.; Mandel, J. H.; Mandel, J. S. (2003). Mortality of employees of a perfluorooctanesulphonyl fluoride manufacturing facility *Occup. Environ. Med.* 2004, 60, 722-9.

Apelberg B.J., Goldman L.R., Calafat A.M., Herbstman J.B., Kuklenyik Z., Heidler J., et al. (2007). Determinants of fetal exposure to polyfluoroalkyl compounds in Baltimore, Maryland. *Environ Sci Technol* 41(11), 3891-3897.

Barry, V., Winqvist, A., Steenland, K. (2013). Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. *Environmental Health Perspectives* 121:1313-1318.

Bartell, S.M., A.M. Calafat, C. Lyu, K. Kato, P.B. Ryan, and K. Steenland. (2010). Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. *Environmental Health Perspectives*, 118 (2), 222-8.

C-8 (PFOA) Medical Monitoring Program Coding. (2014). *Health Smart*. Retrieved from <http://healthsmart.com/HealthSmartCustomers/Providers-Service-Locator/C-8-Medical-Monitoring-Program.aspx>.

C8 Science Panel. (2011a). *Status Report: PFOA (C8) Exposure and Pregnancy Outcome Among Participants in the C8 Health Project*. http://www.c8sciencepanel.org/pdfs/Status_Report_C8_and_pregnancy_outcome_15July2011.pdf.

C8 Science Panel. (2011b). *Probable Link Evaluation of Pregnancy Induced Hypertension and Preeclampsia*. http://www.c8sciencepanel.org/pdfs/Probable_Link_C8_PIH_5Dec2011.pdf.

C8 Science Panel. (2011c). *Status Report: Changes in Serum PFOA/PFOS and Serum Lipids Between 2005 and 2010 in the Mid-Ohio Valley*.

http://www.c8sciencepanel.org/pdfs/Status_Report_C8_and_lipid_changes2_5Dec2011.pdf.

- Calafat, A. M., et al. (2007). Polyfluoroalkyl chemicals in the U.S. population: data from the National Health and Nutrition Examination Survey (NHANES) 2003–2004 and comparisons with NHANES 1999–2000. *Environmental Health Perspectives*, 115(11), 1596–602.
- Casals-Casas, C. & Desvergne, B. (2011). Endocrine Disruptors: From Endocrine to Metabolic Disruption *Annu. Rev. Physiol.*, 73,135–62.
- Cui, L, Zhou, Q.F., Liao, C.Y., Fu, J.J., & Jiang, G.B. (2009). Studies on the toxicological effects of PFOA and PFOS on rats using histological observation and chemical analysis. *Archives of Environmental Contamination and Toxicology*, 56(2), 338–49.
- DeWitt, J.C. et al. (2012). Immunotoxicity of perfluorinated compounds: Recent developments. *Toxicol. Pathol.* 40, 300–11.
- Ehgeghy, P. P., and M. Lorber. (2011). An assessment of the exposure of Americans to perfluorooctane sulfonate: A comparison of estimated intake with values inferred from NHANES data. *Journal of Exposure Science and Environmental Epidemiology*, 21 (2), 150–68.
- Fee C, et al. (2008). Fetal growth indicators and perfluorinated chemicals: a study in the Danish National Birth Cohort. *Am. J. Epidemiol.*, 168(1):66–72.
- Fuentes S., Colomina M.T., Rodriguez J., Vicens P., Domingo J.L. (2006). Interactions in developmental toxicology: concurrent exposure to perfluorooctane sulfonate (PFOS) and stress in pregnant mice. *Toxicol. Lett.* 164, 81–89.
- Fromme, Hermann, et al. (2009). Perfluorinated compounds: exposure assessment for the general population in western countries. *International Journal of Hygiene and Environmental Health* 212(3), 239–70.
- Grandjean P, Andersen EW, Budtz-Jørgensen E, Nielsen F, Molbak K, Weihe P, Heilmann C. (2012). Serum vaccine antibody concentrations in children exposed to perfluorinated compounds. *JAMA*, 307(4):391–397.
- Haug, Line S., et al. (2010). Diet and particularly seafood are major sources of perfluorinated compounds in humans." *Environment International*, 36(7), 772–78.
- Institute of Medicine (IOM). (2011). *Breast cancer and the environment: A life course approach*. Washington, DC: The National Academies Press.
- Lau C, Anitole K, Hodes C, Lai D, Pfahles-Hutchens A, Seed J. (2007). Perfluoroalkyl acids: a review of monitoring and toxicological findings. *Toxicol Sci* 99(2), 366–394.

- Lin, C.Y., Lin, L.Y., Chaing, C.K., Wang, W.J., Su, Y.N., et al. (2010). Investigation of the association between low-dose serum perfluorinated chemicals and liver enzymes in U.S. adults. *American Journal of Gastroenterology*, 105(6), 1354-63.
- Lindstrom, A. B., Strynar, M.J. & Libelo, L. (2011). Polyfluorinated Compounds: Past, Present, and Future. *Environmental Science and Technology*, 45 (19), 7954-61.
- Luebker D.J., York R.G., Hansen K.J., Moore J.A., Butenhoff J.L. (2005). Neonatal mortality from in utero exposure to perfluorooctanesulfonate (PFOS) in Sprague-Dawley rats: dose-response, and biochemical and pharmacokinetic parameters. *Toxicology* 215, 149-69
- Melzer O., et al. (2010). Association between serum PFOA and thyroid disease in the NHANES study. *Environmental Health Perspectives*, 118, 682-96.
- Moermond, C., Verbruggem, E., and C. Smit. (2010). *Environmental Risk Limits for PFOS: A Proposal for Water Quality Standards in Accordance with the Water Framework Directive*. National Institute for Public Health and the Environment. www.rivm.nl/bibliotheek/rapporten/601714013.pdf.
- National Institute of Environmental Health Sciences, (September 2012). *Perfluorinated chemicals*. Accessed at: https://www.niehs.nih.gov/health/materials/perfluorinated_chemicals_508.pdf.
- Nelson, J.W., Hatch, E.E., Webster, T.F. (2010). Exposure to polyfluoroalkyl chemicals and cholesterol, body weight, and insulin resistance in the U.S. general population. *Environmental Health Perspectives*, 118, 197-202.
- Olsen, G.W., J.M. Burris, D.J. Ehresman, J.W. Froehlich, A.M. Seacat, J.L. Butenhoff, and L.R. Zobel. (2007). Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. *Environmental Health Perspectives*, 115 (9), 1298-305.
- Onischenko et al. (2010). Prenatal exposure to PFOS or PFOA alters motor function in mice in a sex-related manner. *Neurotox. Research*, 19, 452-61.
- Post G.B., Cohn P.D., Cooper K.R. (2012). Perfluorooctanoic acid (PFOA), an emerging drinking water contaminant: a critical review of recent literature. *Environ Res.* 116, 93-117.
- Rumsby P.C. et al. (2009). Perfluorooctane sulfonate and perfluorooctanoic acid in drinking and environmental waters. *Philos. Transact. A Math. Phys. Eng. Sci.*, 367, 4119-36.
- Seals, R., Bartell, S.M. and Steenland, K. (2011). Accumulation and clearance of perfluorooctanoic acid (PFOA) in current and former residents of an exposed community. *Environmental Health Perspectives* 119(1),119-24.
- Shankar, A., Xiao, J., Ducatman, A. (2011a). Perfluoroalkyl chemicals and elevated serum uric acid in US adults. *Clinical Epidemiology*, 3, 251-258.

- Shankar, A., Xiao, J., Ducatman, A. (2011b). Perfluoroalkyl chemicals and chronic kidney disease in US adults. *American Journal of Epidemiology*. 174, 893-900.
- Shi Z, Zhang H, Liu Y, Xu M, Dai J. 2007. Alterations in gene expression and testosterone synthesis in the testes of male rats exposed to perfluorododecanoic acid. *Toxicol. Sci.* 98, 206-15.
- Shoeib, M., Haarne, T.M., Webster, G., & Lee, S.C. (2011). Indoor sources of poly- and perfluorinated compounds (PFCs) in Vancouver, Canada: Implications for human exposure. *Environment, Science, and Technology* 45, 7999-8005.
- Stein, C.R. et al. (2009). Serum levels of perfluorooctanoic acid and perfluorooctane sulfonate and pregnancy outcome. *American Journal of Epidemiology*, 170, 837-46.
- Steenland K, Fletcher T, Savitz D.A. (2010). Epidemiologic Evidence on the Health Effects of Perfluorooctanoic Acid (PFOA). *Environmental Health Perspectives*, 118(8), 1100-1108.
- Trudel, D., et al. (2008). Estimating consumer exposure to PFOS and PFOA. *Risk Analysis* 28 (2): 251-69.
- USEPA. (2006). *Science Advisory Board of Review of EPA's Draft Risk Assessment of Potential Human Health Effects Associated with PFOA and its Salts*, May 30, 2006. [http://yosemite.epa.gov/sab/sabproduct.nsf/A3C83648E77252828525717F004B9099/\\$File/sab_06_006.pdf](http://yosemite.epa.gov/sab/sabproduct.nsf/A3C83648E77252828525717F004B9099/$File/sab_06_006.pdf)
- USEPA. (2014). *PFOA and Fluorinated Telomers Progress Report*. <http://www.epa.gov/oppt/pfoa/pubs/stewardship/preports8.html>.
- Velez M.P., Arbuckle T.E., & Fraser, W.D. (2015). Maternal exposure to perfluorinated chemicals and reduced fecundity: the MIREC study. *Human Reproduction*, 30(3), 701-709.
- Vieira, V.M., Hoffman, K., Shin, H., Weinberg, J.M., et al. (2013). Perfluorooctanoic Acid Exposure and Cancer Outcomes in a Contaminated Community: A Geographic Analysis. *Environmental Health Perspectives* 121:318-323.
- Washino N, et. al. (2009). Correlations between Prenatal Exposure to Perfluorinated Chemicals and Reduced Fetal Growth. *Environmental Health Perspectives*, 117(4), 660-667.
- Woodruff, T.J., Zota, A.R. & Schwartz, J.M.. (2011). Environmental Chemicals in Pregnant Women in the US: NHANES 2003-2004. *Environmental Health Perspectives* 119 (6), 878-85.
- Yoo, H., et al. (2011). Quantitative determination of perfluorochemicals and fluorotelomer alcohols in plants from biosolid-amended fields using LC/MS/MS and GC/MS." *Environmental Science and Technology* 45 (19), 7985-90.

Appendix A

Serum Perfluorooctane sulfonic acid (PFOS) (2011 - 2012)

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

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
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
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
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
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
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
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
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
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
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
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

Limit of detection (LOD, see Data Analysis section) for Survey year 11-12 is 0.2.

Biomonitoring Summary: http://www.cdc.gov/biomonitoring/PFCs_BiomonitoringSummary.html

Factsheet: http://www.cdc.gov/biomonitoring/PFCs_FactSheet.html

Serum Perfluorooctane sulfonic acid (PFOS) (1999 – 2010)

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

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Total	99-00	30.4 (27.1-33.9)	30.2 (27.8-33.9)	43.7 (37.5-47.3)	57.0 (50.2-71.7)	75.7 (58.1-97.5)	1562
	03-04	20.7 (19.2-22.3)	21.2 (19.8-22.4)	30.0 (27.5-33.0)	41.3 (35.6-50.0)	54.6 (44.0-66.5)	2094
	05-06	17.1 (16.0-18.2)	17.5 (16.8-18.6)	27.2 (24.9-29.6)	39.4 (34.9-43.1)	47.5 (42.7-56.8)	2120
	07-08	13.2 (12.2-14.2)	13.6 (12.8-14.7)	21.0 (18.9-23.3)	32.6 (29.4-36.3)	40.5 (35.4-47.4)	2100
	09-10	9.32 (8.13-10.7)	9.70 (8.50-10.8)	14.8 (12.9-17.3)	23.7 (18.3-30.2)	32.0 (22.6-48.5)	2233
Age group							
12-19 years	99-00	29.1 (26.2-32.4)	29.5 (26.9-34.2)	39.0 (35.9-45.0)	52.7 (46.0-56.2)	57.4 (52.7-66.5)	543
	03-04	19.3 (17.5-21.4)	19.9 (17.8-22.0)	27.1 (23.7-30.2)	36.5 (28.6-45.6)	42.6 (35.1-52.1)	640
	05-06	15.0 (14.3-15.7)	14.9 (13.6-16.6)	22.7 (19.7-24.9)	30.6 (27.8-34.1)	38.5 (33.0-44.6)	640
	07-08	11.3 (10.3-12.3)	11.3 (10.3-13.0)	15.9 (15.1-17.7)	21.7 (17.7-28.2)	28.0 (22.0-32.2)	357
	09-10	6.84 (5.81-8.06)	6.90 (6.00-8.40)	10.7 (8.90-12.5)	14.4 (12.4-18.1)	18.1 (13.5-26.0)	364
20 years and older	99-00	30.6 (27.1-34.4)	30.3 (27.9-33.9)	44.4 (37.9-48.0)	58.0 (50.1-75.7)	78.0 (59.9-107)	1019
	03-04	20.9 (19.3-22.5)	21.4 (19.8-22.8)	30.4 (28.1-33.0)	42.7 (35.7-53.3)	57.8 (45.7-69.4)	1454
	05-06	17.4 (16.2-18.7)	18.0 (17.1-19.4)	27.8 (25.3-30.8)	40.2 (35.6-44.5)	49.6 (42.8-60.7)	1480
	07-08	13.5 (12.4-14.6)	14.0 (13.0-15.5)	21.7 (19.5-24.6)	33.9 (29.9-39.0)	42.8 (37.3-50.3)	1743
	09-10	9.72 (8.45-11.2)	10.1 (8.90-11.2)	15.7 (13.4-18.4)	25.3 (19.3-32.9)	34.1 (23.4-52.7)	1869
Gender							
Males	99-00	33.4 (29.6-37.6)	34.9 (31.1-37.9)	46.3 (41.2-51.6)	58.4 (50.2-78.3)	78.3 (58.0-108)	743
	03-04	23.2 (21.1-25.6)	23.9 (22.4-25.5)	32.2 (28.8-35.9)	45.3 (35.5-62.7)	62.7 (43.8-81.8)	1053
	05-06	20.5 (19.4-21.8)	21.3 (20.0-22.5)	31.4 (27.9-33.7)	43.3 (38.7-49.7)	54.3 (43.5-80.7)	1048
	07-08	16.3 (15.0-17.7)	17.0 (15.7-17.8)	23.9 (21.6-26.9)	36.4 (33.5-41.1)	45.3 (40.4-53.1)	1059
	09-10	11.5 (9.93-13.3)	11.8 (10.6-12.9)	16.8 (14.3-20.4)	25.7 (19.4-39.7)	37.4 (22.5-72.3)	1075
Females	99-00	28.0 (24.6-31.8)	27.8 (24.5-30.2)	39.0 (32.7-46.0)	55.4 (46.3-70.2)	75.7 (56.1-98.4)	819
	03-04	18.4 (17.0-20.0)	18.2 (16.9-19.8)	27.4 (23.8-30.2)	39.8 (34.4-42.6)	46.6 (42.3-61.5)	1041
	05-06	14.4 (13.3-15.4)	14.6 (13.5-15.9)	23.3 (21.1-25.3)	34.2 (30.6-37.7)	42.8 (38.0-46.5)	1072
	07-08	10.7 (9.70-11.7)	10.7 (9.80-11.7)	17.2 (15.4-19.1)	28.7 (21.7-32.5)	33.6 (29.9-41.6)	1041
	09-10	7.65 (6.73-8.71)	7.80 (6.70-9.00)	12.0 (10.8-14.4)	21.1 (16.4-26.9)	28.8 (22.3-34.1)	1158

Limit of detection (LOD, see Data Analysis section) for Survey years 99-00, 03-04, 05-06, 07-08, and 09-10 are 0.2, 0.4, 0.2, 0.2, and 0.2 respectively.

Biomonitoring Summary: http://www.cdc.gov/biomonitoring/PFCs_BiomonitoringSummary.html

Factsheet: http://www.cdc.gov/biomonitoring/PFCs_FactSheet.html

Serum Perfluorooctanoic acid (PFOA) (2011 - 2012)

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

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
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
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
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
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
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
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
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
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
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
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

Limit of detection (LOD, see Data Analysis section) for Survey year 11-12 is 0.1.

Biomonitoring Summary: http://www.cdc.gov/biomonitoring/PFCs_BiomonitoringSummary.html

Factsheet: http://www.cdc.gov/biomonitoring/PFCs_FactSheet.html

Serum Perfluorooctanoic acid (PFOA) (1999 – 2010)

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

	Survey years	Geometric mean	Selected percentiles				Sample size
		(95% conf. interval)	(95% confidence interval)				
			50th	75th	90th	95th	
Race/ethnicity							
Mexican Americans	99-00	3.89 (3.58-4.21)	4.20 (3.80-4.60)	5.80 (5.20-6.30)	7.70 (6.40-8.00)	8.20 (7.70-8.90)	584
	03-04	3.11 (2.84-3.40)	3.30 (3.10-3.70)	4.50 (4.20-5.20)	6.70 (5.70-7.30)	7.60 (6.70-10.5)	485
	05-06	2.62 (2.33-2.95)	2.80 (2.50-3.30)	4.30 (3.80-4.70)	5.80 (5.30-6.70)	7.40 (5.90-8.10)	499
	07-08	3.54 (3.35-3.75)	3.80 (3.50-4.00)	5.20 (4.90-5.60)	6.60 (6.20-7.10)	7.60 (6.80-9.00)	391
	09-10	2.26 (2.00-2.54)	2.40 (2.10-2.60)	3.60 (3.10-3.80)	4.60 (4.00-5.30)	5.40 (4.50-6.60)	461
Non-Hispanic blacks	99-00	4.78 (4.10-5.57)	4.80 (3.80-5.90)	6.40 (5.90-7.50)	9.00 (7.40-11.5)	11.5 (9.20-14.0)	303
	03-04	3.37 (2.99-3.79)	3.70 (3.20-4.20)	5.20 (4.40-6.30)	7.70 (5.30-11.6)	9.60 (6.50-13.9)	538
	05-06	3.27 (2.61-4.08)	3.70 (3.00-4.20)	5.50 (4.40-6.80)	8.10 (6.00-11.3)	10.4 (7.80-12.3)	544
	07-08	3.86 (3.57-4.16)	4.00 (3.50-4.30)	5.90 (5.20-6.50)	7.80 (7.10-8.70)	9.20 (8.50-10.1)	419
	09-10	2.74 (2.47-3.04)	2.80 (2.60-3.00)	4.00 (3.70-4.40)	5.50 (5.00-6.20)	6.70 (5.60-9.40)	391
Non-Hispanic whites	99-00	5.61 (5.06-6.23)	5.60 (4.90-6.20)	7.30 (6.50-8.20)	10.3 (8.40-12.1)	13.0 (11.0-14.9)	519
	03-04	4.18 (3.85-4.53)	4.30 (3.90-4.70)	6.00 (5.50-6.70)	7.90 (7.20-9.20)	9.90 (7.60-13.3)	962
	05-06	4.27 (3.80-4.81)	4.40 (4.00-5.00)	6.60 (5.60-7.80)	9.60 (7.40-12.2)	11.6 (8.80-14.8)	935
	07-08	4.38 (4.20-4.56)	4.60 (4.30-4.70)	6.10 (5.80-6.60)	8.20 (7.80-8.80)	9.90 (9.30-10.6)	931
	09-10	3.36 (3.06-3.69)	3.50 (3.20-3.90)	4.80 (4.40-5.40)	6.60 (5.40-7.80)	7.80 (6.20-10.5)	1031

Limit of detection (LOD, see Data Analysis section) for Survey years 99-00, 03-04, 05-06, 07-08, and 09-10 are 0.1, 0.2, 0.1, 0.1, and 0.1 respectively.

Biomonitoring Summary: http://www.cdc.gov/biomonitoring/PFCs_BiomonitoringSummary.html

Factsheet: http://www.cdc.gov/biomonitoring/PFCs_FactSheet.html

Available at:

http://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Feb2015.pdf

- This material was developed by the Mid-Atlantic Center for Children's Health & the Environment and funded under the cooperative agreement award number 1U61TS000237-01 from the Agency for Toxic Substances and Disease Registry (ATSDR).
- Acknowledgement: The U.S. Environmental Protection Agency (EPA) supports the PEHSU by providing funds to ATSDR under Inter-Agency Agreement number DW-75-92301301. Neither EPA nor ATSDR endorse the purchase of any products or services mentioned in PEHSU materials.