

Perfluorochemicals (PFCs)

Perfluorochemicals (PFCs) are a group of synthetic chemicals that have been used in many consumer products.¹ The structure of these chemicals makes them very stable, hydrophobic (water-repelling), and oleophobic (oil-repelling). These unique properties have led to extensive use of PFCs in surface coating and protectant formulations for paper and cardboard packaging products; carpets; leather products; and textiles that repel water, grease, and soil. PFCs have also been used in fire-fighting foams and in the production of nonstick coatings on cookware and some waterproof clothes.¹ Due in part to their chemical properties, some PFCs can remain in the environment and bioconcentrate in animals.²⁻⁸ Data from human studies suggest that some PFCs can take years to be cleared from the body.⁹⁻¹³

The PFCs with the highest production volumes in the United States have been perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA).¹ PFOS and PFOA are also two of the most frequently detected PFCs in humans.¹⁴ Other PFCs include perfluorohexane sulfonic acid (PFHxS), which is a member of the same chemical category as PFOS; and perfluorononanoic acid (PFNA), which is a member of the same chemical category as PFOA.¹⁵ Chemicals within a given PFC chemical category share similar chemical structures and uses. Although some studies have addressed PFHxS and PFNA specifically, the majority of scientific research has focused on PFOS and PFOA.¹⁵

In 2000, one of the principal perfluorochemical manufacturers, 3M, began phasing out the production of PFOA, PFOS, and PFOS-related compounds. The 3M phaseout of PFOS and PFHxS was completed in 2002, and its phaseout of PFOA was completed in 2008.¹⁶ In 2006, to address PFOA production by other manufacturers, EPA launched the 2010/15 PFOA Stewardship Program, with eight companies voluntarily agreeing to reduce emissions and product content of PFOA, PFNA, and related chemicals by 95% no later than 2010. The industry participants also committed to work toward eliminating emissions and product content of these chemicals by 2015.¹⁷ However, the fact that some of these chemicals may be persistent in the environment and have a long half-life in humans means that they may continue to persist in the environment and in people for many years, despite reductions in emissions.²⁻¹³ EPA is currently evaluating the potential need for regulation of PFCs using the authorities of the Toxic Substances Control Act.¹⁵

The major sources of human exposure to PFCs are poorly understood, but may include food, water, indoor and outdoor air, breast milk, and dust.⁴ Two recent studies pointed to food consumption as the primary pathway of exposure to PFOS and PFOA for Americans and Europeans.^{18,19} PFC-treated food-contact packaging, such as microwave popcorn bags,ⁱ has been a

ⁱ The U.S. Food and Drug Administration recently worked with several manufacturers to remove grease-proofing agents containing C8 perfluorinated compounds from the marketplace. These manufacturers volunteered to stop distributing products containing these compounds in interstate commerce for food-contact purposes as of October 1, 2011. For more information, see <http://www.fda.gov/Food/FoodIngredientsPackaging/FoodContactSubstancesFCS/ucm308462.htm>.

source of PFC exposure.²⁰ Meat and dairy products may also be contaminated with PFCs due to exposure of source animals to air, water, and feed contaminated with PFCs,²¹⁻²³ although a recent study reported that PFCs were undetected in nearly all milk samples tested in the United States.²⁴ In some areas, such as those near industrial facilities that either make or use PFCs, these contaminants have been found at high levels in drinking water, groundwater, and/or surface water.²⁵⁻³¹ PFCs have been detected in human breast milk.³²⁻³⁶ PFCs have been measured in house dust as well, with some perfluorochemicals, such as PFOS, PFOA, PFHxS, perfluorobutane sulfonic acid (PFBS), and perfluorohexanoic acid (PFHxA), found to be present in the majority of dust samples examined.³⁷⁻⁴⁰ Infants and small children may be more highly exposed to the PFCs present in house dust than adults are, due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity.^{18,19,41,42} Children could have increased exposure to PFCs in carpet and carpet protectants, due to the amount of time they spend lying, crawling, and playing on carpet.^{15,41} Limited available data on levels of PFCs in children's blood suggest that the blood serum levels of most PFCs are higher in children ages 3 to 11 years compared with other age groups.^{43,44}

Some PFCs have been widely detected in pregnant women and in umbilical cord blood, suggesting that the developing fetus can be exposed to PFCs while in the womb. However, findings between studies vary. For example, PFOS and PFOA were detected in 99–100% of blood samples collected from both pregnant and non-pregnant women in 2003–2004.⁴⁵ Additionally, PFOS and PFOA were detected in 99% and 100% of umbilical cord blood samples, respectively, collected from newborns in Baltimore.⁴⁶ In another study conducted in Japan, the level of PFOS circulating in a pregnant woman's blood was highly correlated with the level in cord blood. However, PFOA was detected in maternal samples but was not detected in umbilical cord samples in the Japanese study.⁴⁷ Even though studies suggest that the correlation between maternal and fetal exposure may vary, the ubiquitous presence of PFOS, PFOA, and other PFCs in blood of women of child-bearing age and in umbilical cord blood may indicate that fetal exposure to these chemicals is widespread.^{45,46,48}

Some human health studies have 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.⁴⁹⁻⁵² However, there are inconsistencies in the results of these studies, and two other studies did not find an association between prenatal PFC exposure and birth weight.^{53,54} The participants in all of these studies had PFC blood serum levels comparable to levels in the general population. Animal studies echo these findings, though typically at levels much higher than what humans are normally exposed to. Developmental and reproductive effects, including reduced birth weight, decreased gestational length, structural defects, delays in postnatal growth and development, increased neonatal mortality, and pregnancy loss have all been associated with prenatal rodent exposure to PFOS and PFOA.⁵⁵⁻⁶⁵

Findings from a limited number of studies suggest that exposure to PFOS or PFOA may have negative impacts on human thyroid function. However, there are inconsistencies in the findings between these studies. Some studies have found that PFC exposures are associated with

alterations in thyroid hormone levels, as well as an increased risk of thyroid disease in the general public and in workers with occupational exposures.⁶⁶⁻⁶⁸ However, a recent study of pregnant women with exposures comparable to those in the general population found that increasing levels of PFOS, PFOA, and PFHxS were not associated with differences in thyroid hormone levels.⁶⁹ The results from animal studies have been more consistent. Multiple animal studies have found that thyroid hormone levels are altered in animals exposed to PFOS.^{57,62,63,65,70-74} One of these studies also found that PFOA-treated rats have altered thyroid hormone levels.⁷¹ The health risks associated with maternal thyroid hormone disruption during pregnancy may make this a cause for concern. Moderate deficits in maternal thyroid hormone levels during early pregnancy have been linked to reduced childhood IQ scores and other neurodevelopmental effects, as well as unsuccessful or complicated pregnancies.⁷⁵

Both animal and some human studies have found an association between PFCs exposure and cholesterol and/or triglyceride levels, although physiological differences between humans and experimental animals may cause lipid levels to vary in opposite directions.⁷⁶ Structurally, PFCs resemble fatty acids and can bind to receptors that play key roles in lipid metabolism and fat production.⁷⁷ In animal studies involving various species, PFCs are associated with decreased serum levels of these lipids;^{64,65,73} in contrast some human studies show an increase in blood lipid levels with increased presence of PFCs, including PFOS, PFOA, PFHxS, and PFNA, while other human studies show no change in lipid levels with PFC exposure.⁷⁷⁻⁸⁴ This could be a potential concern for children, because the mother's body provides a source of cholesterol and triglycerides to the developing fetus. Cholesterol and fatty acids support cellular growth, differentiation, and adipose accumulation during fetal development.^{49,85} Finally, although human studies have not looked at the associations between PFC exposure and the immune system, animal studies have found an association between PFOS and PFNA exposure (in utero and in adulthood) and immune suppression, including alterations in function and production of immune cells and decreased lymphoid organ weights.⁸⁶⁻⁸⁸

The indicator that follows uses the best nationally representative data currently available on blood serum levels of perfluorochemicals over time for women of child-bearing age. Indicator B6 presents median blood serum levels of PFOS, PFOA, PFHxS, and PFNA for women ages 16 to 49 years.

Indicator B6: Perfluorochemicals in women ages 16 to 49 years: Median concentrations in blood serum, 1999–2008

About the Indicator: Indicator B6 presents concentrations of perfluorochemicals (PFCs) in blood serum of U.S. women ages 16 to 49 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of PFCs in the blood serum. The indicator presents concentrations of PFCs in blood serum over time. The focus on women of child-bearing age is based on concern for potential adverse effects in children born to women who have been exposed to PFCs.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for PFCs. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.²

Perfluorinated Compounds

Indicator B6 presents blood serum levels of four important PFCs: perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), perfluorohexane sulfonic acid (PFHxS), and perfluorononanoic acid (PFNA). These four PFCs were chosen because they are commonly detected in humans, and the bulk of PFCs health effects research in both humans and laboratory animals has focused on these contaminants—especially PFOS and PFOA.

PFCs bind to proteins in the serum of blood. Because PFCs remain in the human body for years, blood serum levels of PFCs are reflective of long-term exposures to these contaminants. Serum accounts for about half the weight of whole blood, so the blood serum concentration of PFCs is about twice the concentration of PFCs in whole blood.⁸⁹ The blood serum PFC levels for this indicator are given in nanograms of PFC per milliliter of blood serum (ng/mL).ⁱⁱ

Concentrations of 12 different PFCs, including PFOS, PFOA, PFHxS, and PFNA, have been measured in blood serum from a representative subset of NHANES participants ages 12 years

ⁱⁱ Most persistent organic pollutants (POPs) are lipophilic, meaning that they accumulate in fatty tissues; however, this is not the case for PFCs, which are both hydrophobic (water-repelling), and oleophobic (oil-repelling). They instead bind to proteins in the serum of blood. While blood levels of lipophilic POPs are commonly lipid-adjusted, the PFC measurements in blood are not.

and older beginning with the 1999–2000 survey cycle, although PFCs were not measured in the 2001–2002 cycle.

In 2007–2008, NHANES collected PFCs biomonitoring data for 2,100 individuals ages 12 years and older, including 495 women ages 16 to 49 years. The four selected PFCs were detected in 99% to 100% of the individuals sampled in NHANES 2007–2008. The median and 95th percentile of blood serum PFC levels for all NHANES participants in 2007–2008 were 14 ng/mL and 41 ng/mL, respectively, for PFOS; 4 ng/mL and 10 ng/mL, respectively, for PFOA; 2 ng/mL and 10 ng/mL, respectively, for PFHxS; 2 ng/mL and 4 ng/mL, respectively, for PFNA.

Birth Rate Adjustment

Indicator B6 uses measurements of PFCs in blood serum of women ages 16 to 49 years to represent the distribution of PFC exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.⁹⁰ A birth rate-adjusted distribution of women’s PFC levels is used in calculating this indicator,ⁱⁱⁱ meaning that the data are weighted using the age-specific probability of a woman giving birth.⁹¹

Data Presented in the Indicator

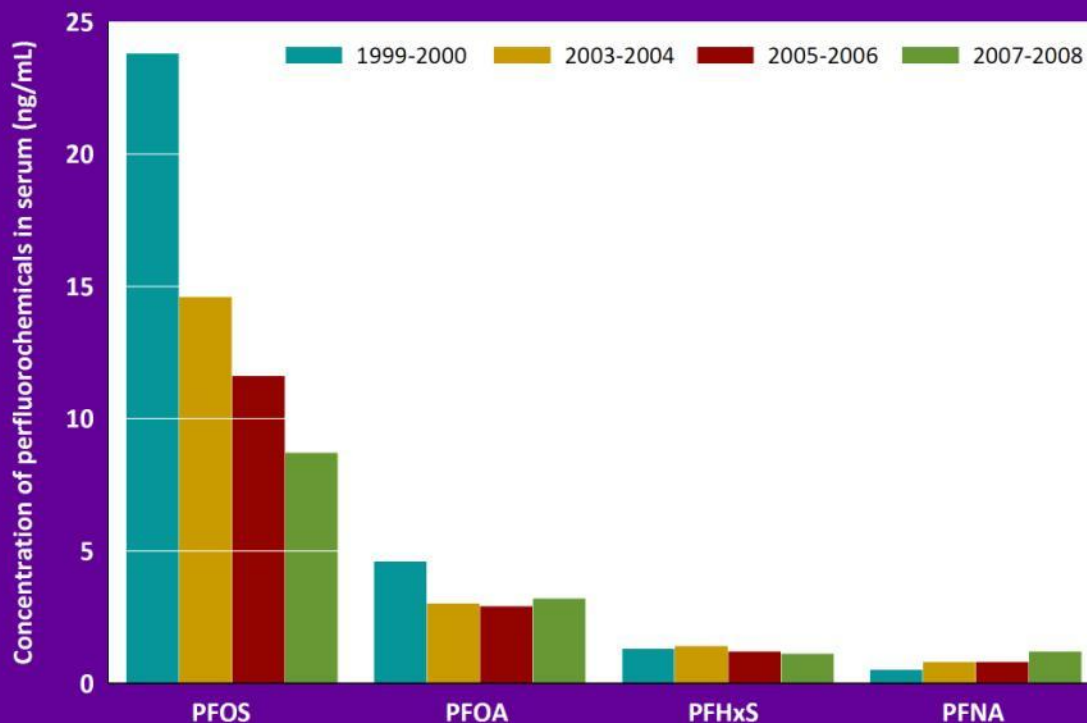
Indicator B6 presents median concentrations of PFOS, PFOA, PFHxS, and PFNA in blood serum over time for women ages 16 to 49 years, using NHANES data from 1999–2008 (excluding the years 2001–2002).

Additional information on the 95th percentile blood serum levels of PFOS, PFOA, PFHxS, and PFNA for women ages 16 to 49 years is presented in the supplemental data tables for this indicator, along with information showing how median and 95th percentile blood serum levels of PFCs in women of child-bearing age vary by race/ethnicity and family income.

Please see the Introduction to the Biomonitoring section for an explanation of the terms “median” and “95th percentile,” a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.

ⁱⁱⁱ There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

Perfluorochemicals in women ages 16 to 49 years: Median concentrations in blood serum, 1999-2008



Data: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

Note: To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth.

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Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- PFCs are measured in blood samples obtained from individual survey participants.

- Between 1999–2000 and 2007–2008, median blood serum levels of PFOS in women of child-bearing age declined from 24 ng/mL in 1999–2000 to 9 ng/mL in 2007–2008. Median blood serum levels of PFOA in women of child-bearing age declined from 5 ng/mL in 1999–2000 to 3 ng/mL in 2007–2008. These decreasing trends were statistically significant.
- The median blood serum levels of PFHxS and PFNA were lower than those of PFOS and PFOA in women of child-bearing age. Median levels of PFHxS have remained relatively constant

over time. Between 1999–2000 and 2007–2008, median blood serum levels of PFNA showed an increasing trend, from 0.5 ng/mL in 1999–2000 to 1.2 ng/mL in 2007–2008.

- The increasing trend in median PFNA levels was statistically significant.
- The concentration of PFOS in blood serum at the 95th percentile in women of child-bearing age showed a decreasing trend from 50 ng/mL in 1999–2000 to 23 ng/mL in 2007–2008. The concentration of PFOA in blood serum at the 95th percentile in women of child-bearing age remained relatively constant between 1999–2000 and 2007–2008. (See Table B6a.)
 - The decreasing trend in 95th percentile PFOS levels was statistically significant.
- For the years 2005–2008, women of child-bearing age living at or above poverty level had higher median and 95th percentile concentrations of PFOS and PFOA in their blood serum compared with women living below poverty level. (See Tables B6b and B6c.)
 - The differences between income groups were statistically significant after adjustment for differences in race/ethnicity and age.
- For the years 2005–2008, median concentrations of PFOA were higher in White non-Hispanic women of child-bearing age (3.5 ng/mL) compared with Black non-Hispanic women (2.7 ng/mL), Mexican-American women (2.3 ng/mL), and women of “All Other Races/Ethnicities” (2.4 ng/mL). (See Table B6b.)
 - These differences in median PFOA concentrations by race/ethnicity were statistically significant. The difference in median concentrations between White non-Hispanic and Black non-Hispanic women was no longer statistically significant after accounting for other demographic characteristics (differences in age and income).
- In 2005–2008, median and 95th percentile concentrations of PFOS were lower in Mexican-American women of child-bearing age at 7.4 ng/mL and 17.3 ng/mL, respectively, compared with White non-Hispanic women at 11.4 ng/mL and 28.4 ng/mL, respectively, Black non-Hispanic women at 11.2 ng/mL and 25.7 ng/mL, respectively, and women of “All Other Races/Ethnicities” at 8.3 ng/mL and 24.9 ng/mL, respectively. (See Tables B6b and B6c.)
 - These differences were statistically significant both with and without adjustment for other demographic characteristics, with the following exceptions: the difference between Mexican-American women and women of “All Other Races/Ethnicities” was statistically significant at the median only after accounting for differences by age and income; and was not statistically significant at the 95th percentile.

Biomonitoring

Perfluorochemicals (PFCs)

1. 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. <http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237>.
2. Centers for Disease Control and Prevention. 2009. *Fourth National Report on Human Exposure to Environmental Chemicals*. Atlanta, GA: CDC. <http://www.cdc.gov/exposurereport/>.
3. Conder, J.M., R.A. Hoke, W. De Wolf, M.H. Russell, and R.C. Buck. 2008. Are PFCAs bioaccumulative? A critical review and comparison with regulatory criteria and persistent lipophilic compounds. *Environmental Science and Technology* 42 (4):995-1003.
4. Fromme, H., S.A. Tittlemier, W. Volkel, M. Wilhelm, and D. Twardella. 2009. Perfluorinated compounds--exposure assessment for the general population in Western countries. *International Journal of Hygiene and Environmental Health* 212 (3):239-70.
5. Kelly, B.C., M.G. Ikonomidou, J.D. Blair, A.E. Morin, and F.A. Gobas. 2007. Food web-specific biomagnification of persistent organic pollutants. *Science* 317 (5835):236-9.
6. Kelly, B.C., M.G. Ikonomidou, J.D. Blair, B. SurrIDGE, D. Hoover, R. Grace, and F.A. Gobas. 2009. Perfluoroalkyl contaminants in an Arctic marine food web: trophic magnification and wildlife exposure. *Environmental Science and Technology* 43 (11):4037-43.
7. Lau, C., K. Anitole, C. Hodes, D. Lai, A. Pfahles-Hutchens, and J. Seed. 2007. Perfluoroalkyl acids: a review of monitoring and toxicological findings. *Toxicological Sciences* 99 (2):366-94.
8. Martin, J.W., S.A. Mabury, K.R. Solomon, and D.C. Muir. 2003. Bioconcentration and tissue distribution of perfluorinated acids in rainbow trout (*Oncorhynchus mykiss*). *Environmental Toxicology and Chemistry* 22 (1):196-204.
9. 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.
10. Brede, E., M. Wilhelm, T. Goen, J. Muller, K. Rauchfuss, M. Kraft, and J. Holzer. 2010. Two-year follow-up biomonitoring pilot study of residents' and controls' PFC plasma levels after PFOA reduction in public water system in Arnsberg, Germany. *International Journal of Hygiene and Environmental Health* 213 (3):217-23.
11. Harada, K., K. Inoue, A. Morikawa, T. Yoshinaga, N. Saito, and A. Koizumi. 2005. Renal clearance of perfluorooctane sulfonate and perfluorooctanoate in humans and their species-specific excretion. *Environmental Research* 99 (2):253-61.
12. Olsen, G.W., J.M. Burriss, 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.
13. Seals, R., S.M. Bartell, and K. Steenland. 2011. Accumulation and clearance of perfluorooctanoic acid (PFOA) in current and former residents of an exposed community. *Environmental Health Perspectives* 119 (1):119-24.
14. Kato, K., L.Y. Wong, L.T. Jia, Z. Kuklennyik, and A.M. Calafat. 2011. Trends in exposure to polyfluoroalkyl chemicals in the U.S. population: 1999-2008. *Environmental Science and Technology* 45 (19):8037-45.
15. U.S. Environmental Protection Agency. 2009. *Long-Chain Perfluorinated Chemicals (PFCs) Action Plan*. Washington, DC: U.S. EPA, Office of Pollution Prevention and Toxics. http://www.epa.gov/oppt/existingchemicals/pubs/pfcs_action_plan1230_09.pdf.
16. 3M. 2010. *What is 3M Doing?* Retrieved January 18, 2010 from http://solutions.3m.com/wps/portal/3M/en_US/PFOS/PFOA/Information/Action/.
17. U.S. Environmental Protection Agency. 2010. *News Release: EPA Announces Substantial Decrease of PFOA* Retrieved January 20, 2010 from <http://yosemite.epa.gov/opa/admpress.nsf/68b5f2d54f3eefd28525701500517fbf/8f9dbdd044050f71852573e50064439f?OpenDocument>.
18. Egeghy, 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.
19. Trudel, D., L. Horowitz, M. Wormuth, M. Scheringer, I.T. Cousins, and K. Hungerbuehler. 2008. Estimating consumer exposure to PFOS and PFOA. *Risk Analysis* 28 (2):251-69.
20. Begley, T.H., K. White, P. Honigfort, M.L. Twaroski, R. Neches, and R.A. Walker. 2005. Perfluorochemicals: potential sources of and migration from food packaging. *Food Additives and Contaminants* 22 (10):1023-31.
21. Tittlemier, S.A., K. Pepper, C. Seymour, J. Moisey, R. Bronson, X.L. Cao, and R.W. Dabeka. 2007. Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. *Journal of Agricultural and Food Chemistry* 55 (8):3203-10.
22. Ericson, I., R. Marti-Cid, M. Nadal, B. Van Bavel, G. Lindstrom, and J.L. Domingo. 2008. Human exposure to perfluorinated chemicals through the diet: intake of perfluorinated compounds in foods from the Catalan (Spain) market. *Journal of Agricultural and Food Chemistry* 56 (5):1787-94.

Perfluorochemicals (PFCs) (continued)

23. Schecter, A., J. Colacino, D. Haffner, K. Patel, M. Opel, O. Papke, and L. Birnbaum. 2010. Perfluorinated Compounds, Polychlorinated Biphenyl, and Organochlorine Pesticide Contamination in Composite Food Samples from Dallas, Texas. *Environmental Health Perspectives* 118:796-802.
24. Young, W.M., P. South, T.H. Begley, G.W. Diachenko, and G.O. Noonan. 2012. Determination of perfluorochemicals in cow's milk using liquid chromatography-tandem mass spectrometry. *Journal of Agricultural and Food Chemistry* 60 (7):1652-8.
25. Konwick, B.J., G.T. Tomy, N. Ismail, J.T. Peterson, R.J. Fauver, D. Higginbotham, and A.T. Fisk. 2008. Concentrations and patterns of perfluoroalkyl acids in Georgia, USA surface waters near and distant to a major use source. *Environmental Toxicology and Chemistry* 27 (10):2011-8.
26. Moody, C.A., G.N. Hebert, S.H. Strauss, and J.A. Field. 2003. Occurrence and persistence of perfluorooctanesulfonate and other perfluorinated surfactants in groundwater at a fire-training area at Wurtsmith Air Force Base, Michigan, USA. *Journal of Environmental Monitoring* 5 (2):341-5.
27. Post, G.B., J.B. Louis, K.R. Cooper, B.J. Boros-Russo, and R.L. Lippincott. 2009. Occurrence and potential significance of perfluorooctanoic acid (PFOA) detected in New Jersey public drinking water systems. *Environmental Science and Technology* 43 (12):4547-54.
28. Shin, H.M., V.M. Vieira, P.B. Ryan, R. Detwiler, B. Sanders, K. Steenland, and S.M. Bartell. 2011. Environmental Fate and Transport Modeling for Perfluorooctanoic Acid Emitted from the Washington Works Facility in West Virginia. *Environmental Science and Technology* 45 (4):1435-42.
29. Sinclair, E., D.T. Mayack, K. Roblee, N. Yamashita, and K. Kannan. 2006. Occurrence of perfluoroalkyl surfactants in water, fish, and birds from New York State. *Archives of Environmental Contamination and Toxicology* 50 (3):398-410.
30. Skutlarek, D., M. Exner, and H. Farber. 2006. Perfluorinated surfactants in surface and drinking waters. *Environmental Science and Pollution Research International* 13 (5):299-307.
31. Steenland, K., C. Jin, J. MacNeil, C. Lally, A. Ducatman, V. Vieira, and T. Fletcher. 2009. Predictors of PFOA Levels in a Community Surrounding a Chemical Plant *Environmental Health Perspectives* 117 (7):1083-1088.
32. Karrman, A., I. Ericson, B. van Bavel, P.O. Darnerud, M. Aune, A. Glynn, S. Lignell, and G. Lindstrom. 2007. Exposure of perfluorinated chemicals through lactation: levels of matched human milk and serum and a temporal trend, 1996-2004, in Sweden. *Environmental Health Perspectives* 115 (2):226-30.
33. Llorca, M., M. Farre, Y. Pico, M.L. Teijon, J.G. Alvarez, and D. Barcelo. 2010. Infant exposure of perfluorinated compounds: levels in breast milk and commercial baby food. *Environment International* 36 (6):584-92.
34. Tao, L., K. Kannan, C.M. Wong, K.F. Arcaro, and J.L. Butenhoff. 2008. Perfluorinated compounds in human milk from Massachusetts, U.S.A. *Environmental Science and Technology* 42 (8):3096-101.
35. Thomsen, C., L.S. Haug, H. Stigum, M. Froshaug, S.L. Broadwell, and G. Becher. 2010. Changes in concentrations of perfluorinated compounds, polybrominated diphenyl ethers, and polychlorinated biphenyls in Norwegian breast-milk during twelve months of lactation. *Environmental Science and Technology* 44 (24):9550-6.
36. Volkel, W., O. Genzel-Boroviczeny, H. Demmelmair, C. Gebauer, B. Koletzko, D. Twardella, U. Raab, and H. Fromme. 2008. Perfluorooctane sulphonate (PFOS) and perfluorooctanoic acid (PFOA) in human breast milk: results of a pilot study. *International Journal of Hygiene and Environmental Health* 211 (3-4):440-6.
37. Bjorklund, J.A., K. Thuresson, and C.A. De Wit. 2009. Perfluoroalkyl compounds (PFCs) in indoor dust: concentrations, human exposure estimates, and sources. *Environmental Science and Technology* 43 (7):2276-81.
38. Strynar, M.J., and A.B. Lindstrom. 2008. Perfluorinated compounds in house dust from Ohio and North Carolina, USA. *Environmental Science and Technology* 42 (10):3751-6.
39. Kato, K., A.M. Calafat, and L.L. Needham. 2009. Polyfluoroalkyl chemicals in house dust. *Environmental Research* 109 (5):518-23.
40. Kubwabo, C., B. Stewart, J. Zhu, and L. Marro. 2005. Occurrence of perfluorosulfonates and other perfluorochemicals in dust from selected homes in the city of Ottawa, Canada. *Journal of Environmental Monitoring* 7 (11):1074-8.
41. Harrad, S., C.A. de Wit, M.A. Abdallah, C. Bergh, J.A. Bjorklund, A. Covaci, P.O. Darnerud, J. de Boer, M. Diamond, S. Huber, et al. 2010. Indoor contamination with hexabromocyclododecanes, polybrominated diphenyl ethers, and perfluoroalkyl compounds: an important exposure pathway for people? *Environmental Science and Technology* 44 (9):3221-31.
42. U.S. Environmental Protection Agency. 2008. *Child-Specific Exposure Factors Handbook (Final Report)*. Washington, DC. EPA/600/R-06/096F. <http://cfpub.epa.gov/ncea/cfm/recorddisplay.cfm?deid=199243#Download>.
43. Kato, K., A.M. Calafat, L.Y. Wong, A.A. Wanigatunga, S.P. Caudill, and L.L. Needham. 2009. Polyfluoroalkyl compounds in pooled sera from children participating in the National Health and Nutrition Examination Survey 2001-2002. *Environmental Science and Technology* 43 (7):2641-7.

Perfluorochemicals (PFCs) (continued)

44. Toms, L.M., A.M. Calafat, K. Kato, J. Thompson, F. Harden, P. Hobson, A. Sjodin, and J.F. Mueller. 2009. Polyfluoroalkyl chemicals in pooled blood serum from infants, children, and adults in Australia. *Environmental Science and Technology* 43 (11):4194-9.
45. Woodruff, T.J., A.R. Zota, and J.M. Schwartz. 2011. Environmental Chemicals in Pregnant Women in the US: NHANES 2003-2004. *Environmental Health Perspectives* 119 (6):878-85.
46. Apelberg, B.J., L.R. Goldman, A.M. Calafat, J.B. Herbstman, Z. Kuklenyik, J. Heidler, L.L. Needham, R.U. Halden, and F.R. Witter. 2007. Determinants of fetal exposure to polyfluoroalkyl compounds in Baltimore, Maryland. *Environmental Science and Technology* 41 (11):3891-7.
47. Inoue, K., F. Okada, R. Ito, S. Kato, S. Sasaki, S. Nakajima, A. Uno, Y. Saijo, F. Sata, Y. Yoshimura, et al. 2004. Perfluorooctane sulfonate (PFOS) and related perfluorinated compounds in human maternal and cord blood samples: assessment of PFOS exposure in a susceptible population during pregnancy. *Environmental Health Perspectives* 112 (11):1204-7.
48. Calafat, A.M., Z. Kuklenyik, J.A. Reidy, S.P. Caudill, J.S. Tully, and L.L. Needham. 2007. Serum concentrations of 11 polyfluoroalkyl compounds in the U.S. population: data from the national health and nutrition examination survey (NHANES). *Environmental Science and Technology* 41 (7):2237-42.
49. Apelberg, B.J., F.R. Witter, J.B. Herbstman, A.M. Calafat, R.U. Halden, L.L. Needham, and L.R. Goldman. 2007. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. *Environmental Health Perspectives* 115 (11):1670-6.
50. Fei, C., J.K. McLaughlin, R.E. Tarone, and J. Olsen. 2007. Perfluorinated chemicals and fetal growth: a study within the Danish National Birth Cohort. *Environmental Health Perspectives* 115 (11):1677-82.
51. Fei, C., J.K. McLaughlin, R.E. Tarone, and J. Olsen. 2008. Fetal growth indicators and perfluorinated chemicals: a study in the Danish National Birth Cohort. *American Journal of Epidemiology* 168 (1):66-72.
52. Washino, N., Y. Saijo, S. Sasaki, S. Kato, S. Ban, K. Konishi, R. Ito, A. Nakata, Y. Iwasaki, K. Saito, et al. 2009. Correlations between prenatal exposure to perfluorinated chemicals and reduced fetal growth. *Environmental Health Perspectives* 117 (4):660-7.
53. Hamm, M.P., N.M. Cherry, E. Chan, J.W. Martin, and I. Burstyn. 2010. Maternal exposure to perfluorinated acids and fetal growth. *Journal of Exposure Science and Environmental Epidemiology* 20 (7):589-97.
54. Monroy, R., K. Morrison, K. Teo, S. Atkinson, C. Kubwabo, B. Stewart, and W.G. Foster. 2008. Serum levels of perfluoroalkyl compounds in human maternal and umbilical cord blood samples. *Environmental Research* 108 (1):56-62.
55. Butenhoff, J.L., G.L. Kennedy, Jr., S.R. Frame, J.C. O'Connor, and R.G. York. 2004. The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat. *Toxicology* 196 (1-2):95-116.
56. Era, S., K.H. Harada, M. Toyoshima, K. Inoue, M. Minata, N. Saito, T. Takigawa, K. Shiota, and A. Koizumi. 2009. Cleft palate caused by perfluorooctane sulfonate is caused mainly by extrinsic factors. *Toxicology* 256 (1-2):42-7.
57. Fuentes, S., M.T. Colomina, J. Rodriguez, P. Vicens, and J.L. Domingo. 2006. Interactions in developmental toxicology: concurrent exposure to perfluorooctane sulfonate (PFOS) and stress in pregnant mice. *Toxicology Letters* 164 (1):81-9.
58. Grasty, R.C., D.C. Wolf, B.E. Grey, C.S. Lau, and J.M. Rogers. 2003. Prenatal window of susceptibility to perfluorooctane sulfonate-induced neonatal mortality in the Sprague-Dawley rat. *Birth Defects Research Part B: Developmental and Reproductive Toxicology* 68 (6):465-71.
59. Hines, E.P., S.S. White, J.P. Stanko, E.A. Gibbs-Flournoy, C. Lau, and S.E. Fenton. 2009. Phenotypic dichotomy following developmental exposure to perfluorooctanoic acid (PFOA) in female CD-1 mice: Low doses induce elevated serum leptin and insulin, and overweight in mid-life. *Molecular and Cellular Endocrinology* 304 (1-2):97-105.
60. Lau, C., J.L. Butenhoff, and J.M. Rogers. 2004. The developmental toxicity of perfluoroalkyl acids and their derivatives. *Toxicology and Applied Pharmacology* 198 (2):231-41.
61. Lau, C., J.R. Thibodeaux, R.G. Hanson, M.G. Narotsky, J.M. Rogers, A.B. Lindstrom, and M.J. Strynar. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. *Toxicological Sciences* 90 (2):510-8.
62. Lau, C., J.R. Thibodeaux, R.G. Hanson, J.M. Rogers, B.E. Grey, M.E. Stanton, J.L. Butenhoff, and L.A. Stevenson. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. II: postnatal evaluation. *Toxicological Sciences* 74 (2):382-92.
63. Luebker, D.J., M.T. Case, R.G. York, J.A. Moore, K.J. Hansen, and J.L. Butenhoff. 2005. Two-generation reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. *Toxicology* 215 (1-2):126-48.
64. Luebker, D.J., R.G. York, K.J. Hansen, J.A. Moore, and J.L. Butenhoff. 2005. Neonatal mortality from in utero exposure to perfluorooctanesulfonate (PFOS) in Sprague-Dawley rats: dose-response, and biochemical and pharmacokinetic parameters. *Toxicology* 215 (1-2):149-69.
65. Thibodeaux, J.R., R.G. Hanson, J.M. Rogers, B.E. Grey, B.D. Barbee, J.H. Richards, J.L. Butenhoff, L.A. Stevenson, and C. Lau. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. I: maternal and prenatal evaluations. *Toxicological Sciences* 74 (2):369-81.

Perfluorochemicals (PFCs) (continued)

66. Olsen, G.W., and L.R. Zobel. 2007. Assessment of lipid, hepatic, and thyroid parameters with serum perfluorooctanoate (PFOA) concentrations in fluorochemical production workers. *International Archives of Occupational and Environmental Health* 81 (2):231-46.
67. Dallaire, R., E. Dewailly, D. Pereg, S. Dery, and P. Ayotte. 2009. Thyroid function and plasma concentrations of polyhalogenated compounds in Inuit adults. *Environmental Health Perspectives* 117 (9):1380-6.
68. Melzer, D., N. Rice, M.H. Depledge, W.E. Henley, and T.S. Galloway. 2010. Association Between Serum Perfluorooctanoic Acid (PFOA) and Thyroid Disease in the NHANES Study. *Environmental Health Perspectives* 118 (686-692).
69. Chan, E., I. Burstyn, N. Cherry, F. Bamforth, and J.W. Martin. 2011. Perfluorinated acids and hypothyroxinemia in pregnant women. *Environmental Research* 111 (4):559-64.
70. Chang, S.C., J.R. Thibodeaux, M.L. Eastvold, D.J. Ehresman, J.A. Bjork, J.W. Froehlich, C. Lau, R.J. Singh, K.B. Wallace, and J.L. Butenhoff. 2008. Thyroid hormone status and pituitary function in adult rats given oral doses of perfluorooctanesulfonate (PFOS). *Toxicology* 243 (3):330-9.
71. Martin, M.T., R.J. Brennan, W. Hu, E. Ayanoglu, C. Lau, H. Ren, C.R. Wood, J.C. Corton, R.J. Kavlock, and D.J. Dix. 2007. Toxicogenomic study of triazole fungicides and perfluoroalkyl acids in rat livers predicts toxicity and categorizes chemicals based on mechanisms of toxicity. *Toxicological Sciences* 97 (2):595-613.
72. Seacat, A.M., P.J. Thomford, K.J. Hansen, L.A. Clemen, S.R. Eldridge, C.R. Elcombe, and J.L. Butenhoff. 2003. Sub-chronic dietary toxicity of potassium perfluorooctanesulfonate in rats. *Toxicology* 183 (1-3):117-31.
73. Seacat, A.M., P.J. Thomford, K.J. Hansen, G.W. Olsen, M.T. Case, and J.L. Butenhoff. 2002. Subchronic toxicity studies on perfluorooctanesulfonate potassium salt in cynomolgus monkeys. *Toxicological Sciences* 68 (1):249-64.
74. Yu, W.G., W. Liu, and Y.H. Jin. 2009. Effects of perfluorooctane sulfonate on rat thyroid hormone biosynthesis and metabolism. *Environmental Toxicology and Chemistry* 28 (5):990-6.
75. Morreale de Escobar, G., M.J. Obregon, and F. Escobar del Rey. 2000. Is neuropsychological development related to maternal hypothyroidism or to maternal hypothyroxinemia? *The Journal of Clinical Endocrinology and Metabolism* 85 (11):3975-87.
76. Vamecq, J., and N. Latruffe. 1999. Medical significance of peroxisome proliferator-activated receptors. *Lancet* 354 (9173):141-8.
77. Nelson, J.W., E.E. Hatch, and T.F. Webster. 2010. Exposure to polyfluoroalkyl chemicals and cholesterol, body weight, and insulin resistance in the general U.S. population. *Environmental Health Perspectives* 118:197-202.
78. Costa, G., S. Sartori, and D. Consonni. 2009. Thirty years of medical surveillance in perfluorooctanoic acid production workers. *Journal of Occupational and Environmental Medicine* 51 (3):364-72.
79. Gilliland, F.D., and J.S. Mandel. 1996. Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins, and cholesterol: a study of occupationally exposed men. *American Journal of Industrial Medicine* 29 (5):560-8.
80. Haugom, B., and O. Spydevold. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic acid (PFOA), perfluorooctane sulphonic acid (PFOSA) and clofibrilic acid. *Biochimica et Biophysica Acta* 1128 (1):65-72.
81. Lin, C.Y., P.C. Chen, Y.C. Lin, and L.Y. Lin. 2009. Association among serum perfluoroalkyl chemicals, glucose homeostasis, and metabolic syndrome in adolescents and adults. *Diabetes Care* 32 (4):702-7.
82. Olsen, G.W., J.M. Burris, M.M. Burlew, and J.H. Mandel. 2003. Epidemiologic assessment of worker serum perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations and medical surveillance examinations. *Journal of Occupational and Environmental Medicine* 45 (3):260-70.
83. Olsen, G.W., J.M. Burris, J.H. Mandel, and L.R. Zobel. 1999. Serum perfluorooctane sulfonate and hepatic and lipid clinical chemistry tests in fluorochemical production employees. *Journal of Occupational and Environmental Medicine* 41 (9):799-806.
84. Sakr, C.J., K.H. Kreckmann, J.W. Green, P.J. Gillies, J.L. Reynolds, and R.C. Leonard. 2007. Cross-sectional study of lipids and liver enzymes related to a serum biomarker of exposure (ammonium perfluorooctanoate or APFO) as part of a general health survey in a cohort of occupationally exposed workers. *Journal of Occupational and Environmental Medicine* 49 (10):1086-96.
85. Woollett, L.A. 2001. The origins and roles of cholesterol and fatty acids in the fetus. *Current Opinion in Lipidology* 12 (3):305-12.
86. Keil, D.E., T. Mehlmann, L. Butterworth, and M.M. Peden-Adams. 2008. Gestational exposure to perfluorooctane sulfonate suppresses immune function in B6C3F1 mice. *Toxicological Sciences* 103 (1):77-85.
87. Fang, X., L. Zhang, Y. Feng, Y. Zhao, and J. Dai. 2008. Immunotoxic effects of perfluorononanoic acid on BALB/c mice. *Toxicological Sciences* 105 (2):312-21.
88. Peden-Adams, M.M., J.M. Keller, J.G. Eudaly, J. Berger, G.S. Gilkeson, and D.E. Keil. 2008. Suppression of humoral immunity in mice following exposure to perfluorooctane sulfonate. *Toxicological Sciences* 104 (1):144-54.
89. Ehresman, D.J., J.W. Froehlich, G.W. Olsen, S.C. Chang, and J.L. Butenhoff. 2007. Comparison of human whole blood, plasma, and serum matrices for the determination of perfluorooctanesulfonate (PFOS), perfluorooctanoate (PFOA), and other fluorochemicals. *Environ Res* 103 (2):176-84.

Perfluorochemicals (PFCs) (continued)

90. National Center for Health Statistics. *Vital Statistics Natality Birth Data, 2003-2004*. Retrieved June 15, 2009 from http://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm.

91. Axelrad, D.A., and J. Cohen. 2011. Calculating summary statistics for population chemical biomonitoring in women of childbearing age with adjustment for age-specific natality. *Environmental Research* 111 (1):149-155.

Biomonitoring

Perfluorochemicals (PFCs)

Table B6. Perfluorochemicals in women ages 16 to 49 years: Median concentrations in blood serum, 1999-2008

Year	Median concentration of PFCs in serum (ng/mL)			
	PFOS	PFOA	PFHxS	PFNA
1999-2000	23.8	4.6	1.3	0.5
2003-2004	14.6	3.0	1.4	0.8
2005-2006	11.6	2.9	1.2	0.8
2007-2008	8.7	3.2	1.1	1.2

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

NOTES:

- PFOS = perfluorooctane sulfonic acid, PFOA = perfluorooctanoic acid, PFHxS = perfluorohexane sulfonic acid, and PFNA = perfluorononanoic acid.
- To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth. The intent of this adjustment is to approximate the distribution of exposure to pregnant women. Results will therefore differ from a characterization of exposure to adult women without consideration of birth rates.

Table B6a. Perfluorochemicals in women ages 16 to 49 years: 95th percentile concentrations in blood serum, 1999-2008

Year	95 th percentile concentration of PFCs in serum (ng/mL)			
	PFOS	PFOA	PFHxS	PFNA
1999-2000	50.1	8.4	4.9	1.3
2003-2004	42.2	8.4	7.1*	NA**
2005-2006	27.8	6.4	5.4	2.2
2007-2008	22.8	7.9	4.9	3.2

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

NOTES:

- PFOS = perfluorooctane sulfonic acid, PFOA = perfluorooctanoic acid, PFHxS = perfluorohexane sulfonic acid, and PFNA = perfluorononanoic acid.
- To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth. The intent of this adjustment is to approximate the distribution of exposure to pregnant women. Results will therefore differ from a characterization of exposure to adult women without consideration of birth rates.

*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

** Not available. The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate), or the RSE cannot be reliably estimated.

Table B6b. Perfluorochemicals in women ages 16 to 49 years: Median concentrations in blood serum, by race/ethnicity and family income, 2005-2008

		Median concentration of PFCs in serum (ng/mL)		
Race / Ethnicity		All Incomes‡ (n=1,121)	< Poverty Level (n=278)	≥ Poverty Level (n=780)
PFOS	All Races/Ethnicities (n=1,121)	10.1	8.1	11.0
	White non-Hispanic (n=453)	11.4	8.1*	11.6
	Black non-Hispanic (n=255)	11.2	NA**	11.2
	Mexican-American (n=272)	7.4	8.1*	7.3
	All Other Races/Ethnicities† (n=141)	8.3	NA**	10.5
PFOA	All Races/Ethnicities (n=1,121)	3.1	2.7	3.2
	White non-Hispanic (n=453)	3.5	3.3*	3.5
	Black non-Hispanic (n=255)	2.7	NA**	2.6
	Mexican-American (n=272)	2.3	2.1*	2.4
	All Other Races/Ethnicities† (n=141)	2.4	NA**	2.6
PFHxS	All Races/Ethnicities (n=1,121)	1.2	1.0	1.2
	White non-Hispanic (n=453)	1.3	1.1*	1.3
	Black non-Hispanic (n=255)	1.1	NA**	1.1
	Mexican-American (n=272)	0.9	0.9*	1.0
	All Other Races/Ethnicities† (n=141)	0.8	NA**	1.1
PFNA	All Races/Ethnicities (n=1,121)	1.0	1.0	1.0
	White non-Hispanic (n=453)	1.1	1.0*	1.1
	Black non-Hispanic (n=255)	1.1	NA**	1.2
	Mexican-American (n=272)	0.8	0.9*	0.8
	All Other Races/Ethnicities† (n=141)	1.1	NA**	1.2

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

NOTES:

- PFOS = perfluorooctane sulfonic acid, PFOA = perfluorooctanoic acid, PFHxS = perfluorohexane sulfonic acid, and PFNA = perfluorononanoic acid.
- To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth. The intent of this adjustment is to approximate the distribution of exposure to pregnant women. Results will therefore differ from a characterization of exposure to adult women without consideration of birth rates.

† The “All Other Races/Ethnicities” category includes all other races or ethnicities not specified, together with those individuals who report more than one race.

‡ Includes sampled individuals for whom income information is missing.

*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

** Not available. The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate), or the RSE cannot be reliably estimated.

Table B6c. Perfluorochemicals in women ages 16 to 49 years: 95th percentile concentrations in blood serum, by race/ethnicity and family income, 2005-2008

		95 th percentile concentration of PFCs in serum (ng/mL)		
Race / Ethnicity		All Incomes‡ (n=1,121)	< Poverty Level (n=278)	≥ Poverty Level (n=780)
PFOS	All Races/Ethnicities (n=1,121)	25.7	22.6	27.8
	White non-Hispanic (n=453)	28.4	23.9*	28.4
	Black non-Hispanic (n=255)	25.7	NA**	25.7
	Mexican-American (n=272)	17.3	17.3*	16.4
	All Other Races/Ethnicities† (n=141)	24.9	NA**	31.0
PFOA	All Races/Ethnicities (n=1,121)	7.5	5.6	7.8
	White non-Hispanic (n=453)	8.1	5.8*	8.1
	Black non-Hispanic (n=255)	6.5	NA**	6.1
	Mexican-American (n=272)	5.5	5.4*	5.6
	All Other Races/Ethnicities† (n=141)	5.8	NA**	5.8
PFHxS	All Races/Ethnicities (n=1,121)	5.1	5.4	5.1
	White non-Hispanic (n=453)	5.6	6.0*	5.6
	Black non-Hispanic (n=255)	4.2	NA**	3.7
	Mexican-American (n=272)	4.6	4.0*	5.2
	All Other Races/Ethnicities† (n=141)	2.1	NA**	2.1
PFNA	All Races/Ethnicities (n=1,121)	2.8	2.7	2.6
	White non-Hispanic (n=453)	2.9	2.0*	2.7
	Black non-Hispanic (n=255)	NA**	NA**	2.3
	Mexican-American (n=272)	2.3	2.3*	2.0
	All Other Races/Ethnicities† (n=141)	2.8	NA**	2.8

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

NOTES:

- PFOS = perfluorooctane sulfonic acid, PFOA = perfluorooctanoic acid, PFHxS = perfluorohexane sulfonic acid, and PFNA = perfluorononanoic acid.
- To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth. The intent of this adjustment is to approximate the distribution of exposure to pregnant women. Results will therefore differ from a characterization of exposure to adult women without consideration of birth rates.

† The “All Other Races/Ethnicities” category includes all other races or ethnicities not specified, together with those individuals who report more than one race.

‡ Includes sampled individuals for whom income information is missing.

*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

** Not available. The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate), or the RSE cannot be reliably estimated.