6. POTENTIAL FOR HUMAN EXPOSURE

6.1 OVERVIEW

Perfluoroalkyls have not been reported at EPA National Priorities List (NPL) sites; however, it is unknown how many of the 1,699 current or former NPL sites have been evaluated for the presence of perfluoroalkyls (HazDat 2008).

Since the early 2000s, companies in the fluorochemical industry have been working with EPA to phase-out the production and use of several perfluoroalkyl compounds (3M 2008a; DuPont 2008; EPA 2007c, 2008f). 3M ceased production of PFOS and related chemicals in 2002 (3M 2008a; EPA 2007c). PFOA, PFOA precursors, and higher homologues are currently being phased out by DuPont and other members of EPA's PFOA Stewardship Program (DuPont 2008; EPA 2008f). Industrial releases of these perfluoroalkyls in the United States are declining based on company reports submitted to EPA (EPA 2008f). In the past, large amounts of perfluoroalkyls were released to the air, water, and soil in and around fluorochemical facilities (3M 2007b, 2008a, 2008b; Barton et al. 2007; Davis et al. 2007; DuPont 2008; EPA 2008f).

Other sources of perfluoroalkyls in the environment have also been considered. Perfluorocarboxylates and sulfonates may be formed from the oxidation of precursors such as fluorotelomer alcohols and perfluoroalkyl sulfonamides in air, water, and soil (D'eon et al. 2006; Ellis et al. 2004; Gauthier and Mabury 2005; Liu et al. 2007; Martin et al. 2006; Wallington et al. 2006; Wang et al. 2005a, 2005b; Wania 2007). The use of perfluoroalkyls in surface protectants such as treatments for carpets and textiles is expected to result in the release of these substances to the air (Barber et al. 2007; Jahnke et al. 2007a; Kubwabo et al. 2005; Moriwaki et al. 2003; Prevedouros et al. 2006; Shoeib et al. 2004). The former use of perfluoroalkyls in aqueous fire-fighting foams has resulted in the release of these substances to soil and groundwater (Moody and Field 1999; Moody et al. 2003).

Perfluoroalkyl carboxylic acids and sulfonic acids are expected to dissociate in the environment based on measured and estimated pKa values of <3 (Kissa 2001; SPARC 2008). Perfluoroalkyl anions will not volatilize from water or soil surfaces (Prevedouros et al. 2006). The unique surfactant properties of these substances may prevent total dissociation of perfluoroalkyls in water (EPA 2005a; Kissa 2001; Prevedouros et al. 2006). Therefore, some volatilization of perfluoroalkyls may occur since the neutral forms of these substances are considered to be highly volatile (Barton et al. 2007; EPA 2005a; Kim and Kannan 2007). Perfluoroalkyls have been detected in air both in the vapor phase and as adsorbed to

particulates (Kim and Kannan 2007). Perfluoroalkyl sulfonamides may partially dissociate in the environment, especially under acidic conditions and are therefore expected to have a higher rate of volatilization compared to the carboxylic acids and sulfonic acids (Martin et al. 2006; SPARC 2008).

Perfluoroalkyls are very stable compounds and are resistant to biodegradation, direct photolysis, atmospheric photooxidation, and hydrolysis (3M 2000; EPA 2008f; OECD 2002, 2007; Schultz et al. 2003). Atmospheric photooxidation half-lives determined for representative perfluoroalkyl sulfonamides ranging from 20 to 50 days indicate that this may be an important degradation mechanism for this group of perfluoroalkyls (D'eon et al. 2006; Martin et al. 2006). Perfluoroalkyls released to the atmosphere are expected to adsorb to particles and settle to the ground through wet or dry deposition (Barton et al. 2007; Hurley et al. 2004; Prevedouros et al. 2006). The chemical stability of perfluoroalkyls and the low volatility of these substances in ionic form indicate that perfluoroalkyls will be persistent in water and soil (3M 2000; Prevedouros et al. 2006). K_{oc} values ranging from 17 to 230 indicate that PFOA will be mobile in soil and can leach into groundwater (Davis et al. 2007; Prevedouros et al. 2006).

Perfluoroalkyls have been detected in environmental media and biota of the Arctic region and in other remote locations such as open ocean waters (Barber et al. 2007; Prevedouros et al. 2006; Yamashita et al. 2005, 2008; Wei et al. 2007a). Proposed source pathways include long-range atmospheric transport of precursor compounds followed by photooxidation to form perfluoroalkyls, direct long-range transport of perfluoroalkyls via oceanic currents, and transport of perfluoroalkyls in the form of marine aerosols (Armitage et al. 2006; Barber et al. 2007; Prevedouros et al. 2006; Wania 2007). Direct transport of perfluoroalkyls in the atmosphere has also been proposed as a source pathway since these substances were recently detected in the vapor phase in outdoor air samples (CEMN 2008; Prevedouros et al. 2006). The actual source of perfluoroalkyls in remote locations is likely to be a combination of these pathways.

PFOA and PFOS have been measured in outdoor urban air samples at concentrations as high as 46 and 919 pg/m^3, respectively (Barber et al. 2007; Harada et al. 2005b, 2006; Kim and Kannan 2007). Concentrations of other perfluoroalkyls measured in outdoor air are generally <1 pg/m^3. Reported concentrations of perfluoroalkyls measured in four indoor air samples were <5 pg/m^3 (Barber et al. 2007). PFOA, PFOS, and PFHxS have been detected in indoor dust samples at concentrations ranging from <2.29–3,700, <4.56–5,065, and <4.56–4,305 ng/g, respectively (Kubwabo et al. 2005; Moriwaki et al. 2003). Reported concentrations of perfluoroalkyls measured in surface water samples are generally below 50 ng/L (Boulanger et al. 2004; Kannan et al. 2005; Kim and Kannan 2007; Nakayama et al. 2007; Simcik and Dorweiler 2005; Sinclair et al. 2004, 2006). Background concentrations of perfluoroalkyls in

groundwater, drinking water, soil, and sediment have not been located. Perfluoroalkyls have been detected in different types of foods at reported concentrations ranging from 0.05 to 10,000 ng/g fresh weight (3M 2001; Food Standards Agency 2006; Fromme et al. 2007b; Tittlemier et al. 2007). Perfluoroalkyls have also been detected in consumer products such as treated carpeting, treated apparel, and paper food packaging (Begley et al. 2005; Washburn et al. 2005). Elevated concentrations of perfluoroalkyls have been measured in air, water, soil, and sediment near fluorochemical industrial facilities (3M 2007b, 2008b, 2008c; Barton et al. 2006; Davis et al. 2007; Hansen et al. 2002).

The highest concentrations of perfluoroalkyls in animals are measured in apex predators, such as polar bears, which indicates that these substances biomagnify in food webs (de Vos et al. 2008; Houde et al. 2006b; Kannan et al. 2005; Kelly et al. 2007; Smithwick et al. 2005a, 2005b, 2006). The bioaccumulation potential of perfluoroalkyls is reported to increase with increasing chain length (de Vos et al. 2008; Furdui et al. 2007; Martin et al. 2004b). In living organisms, perfluoroalkyls bind to protein albumin in blood, liver, and eggs and do not accumulate in fat tissue (de Vos et al. 2008; Kissa 2001).

Mean PFOA, PFOS, and PFHxS serum concentrations reported in various studies from the United States were 2.1–9.6, 14.7–55.8, and 1.5–3.9 ng/mL, respectively (Calafat et al. 2006b, 2007a, 2007b; De Silva and Mabury 2006; Kuklenyik et al. 2004; Olsen et al. 2003a, 2003b, 2004c, 2005, 2007a). Mean concentrations of PFHpA, PFNA, PFDeA, PFUA, PFDoA, PFBuS, PFBA, PFOSA, Me-PFOSA-AcOH, and Et-PFOSA-AcOH are generally <1 ng/mL in these studies. Major PFOS exposure pathways proposed for the general population include food and water ingestion, dust ingestion, and hand-to-mouth transfer from mill-treated carpets (Trudel et al. 2008). For PFOA, the major exposure pathways are proposed to be oral exposure resulting from general food and water ingestion, inhalation from impregnated clothes, and dust ingestion. While migration of residual PFOA in paper packaging and wrapping into food is also a potential route of exposure (Trudel et al. 2008), precursor substances in food packaging can also be metabolized in the body to PFOA (D'eon and Mabury 2007; D'eon et al. 2009). Polyfluoroalkyl phosphoric acids (PAPs) are fluorinated surfactant substances used to greaseproof food-containing paper products. Biotransformation of the 8:2 PAP and the 8:2 fluorotelomer alcohol into PFOA has been demonstrated (D'eon et al. 2009). Based on these proposed exposure pathways, Trudel et al. (2008) estimated that adult uptake doses for high-exposure scenarios were approximately 30 and 47 ng/kg body weight/day for PFOS and PFOA, respectively. The estimated dosage for children under the age of 12 under a high-exposure scenario were estimated to be 101–219 and 65.2–128 ng/kg body weight/day, for PFOS and PFOA, respectively (Trudel et al. 2008). Estimated daily doses for the general population were also estimated by Vestergren et al. (2008) to range from 3.9 to 520 ng/kg body

weight/day for PFOS and from 0.3 to 150 ng/kg body weight/day for PFOA. Infants and toddlers had the highest estimated dosages due to greater hand-to-mouth contact with treated carpeting, mouthing activities of clothes, and greater dust ingestion. Under certain exposure scenarios, it was estimated that up to 80% of the intake could be attributable to exposure to precursor substances followed by subsequent metabolism to PFOS or PFOA. Exposure pathways of perfluorocarboxylate homologues for different exposure settings were modeled by Vestergren and Cousins (2009). Food intake was determined to be the major exposure pathway for the general population, while inhalation of indoor air was reported to be the primary exposure route for occupationally exposed individuals employed in fluorochemical plants. Ingestion of contaminated drinking water was determined to be the primary exposure pathway for individuals residing in communities with high point source contamination of water supplies.

Perfluoroalkyls have been detected in human breast milk and umbilical cord blood. The reported maximum concentrations of PFOS and PFOA measured in human breast milk samples were 0.360–0.685 and 0.210–0.609 ng/mL, respectively (Kärrman et al. 2007; Llorca et al. 2010; So et al. 2006b; Völkel et al. 2008). Maximum concentrations of other perfluoroalkyl compounds were <0.18 ng/mL. PFOS and PFOA have been detected in most umbilical cord blood samples with reported concentrations of 4.9–11.0 and 1.6–3.7 ng/mL, respectively (Apelberg et al. 2007a, 2007b; Fei et al. 2007; Inoue et al. 2004b; Midasch et al. 2007). Other perfluoroalkyls have been detected less frequently, with maximum concentrations of <2.6 ng/mL.

Individuals who perform jobs that require frequent contact with perfluoroalkyl-containing products, such as individuals who install and treat carpets, are expected to have occupational exposure to these substances. Individuals who work at fluorochemical facilities may have higher exposure to perfluoroalkyl compounds than the general population based on elevated concentrations of these substances measured in air, soil, sediment, surface water, groundwater, and vegetation surrounding these facilities (3M 2007b, 2008b, 2008c; Barton et al. 2006; Davis et al. 2007). Studies of individuals living near fluorochemical facilities indicate that drinking water is the major exposure pathway (Emmett et al. 2006a; Holzer et al. 2008; Wilhelm et al. 2009). Estimated off-site exposure of local residents that live near a fluorochemical facility to PFOA from contaminated environmental media ranged from 0.011 to 260 ng/kg/day (3M 2008c).

6.2 RELEASES TO THE ENVIRONMENT

There is no information listed in EPA's Toxic Release Inventory (TRI) on releases of perfluoroalkyls to the environment from manufacturing and processing facilities because these releases are not required to be reported within this program (EPA 2005b).

Perfluoroalkyls are man-made compounds that are not naturally occurring in the environment. Perfluoroalkyls such as PFOS and PFOA have been widely used in the manufacturing of consumer products (Hekster et al. 2003; Schultz et al. 2003). These substances are now detected in both environmental and biological media around the world as well as in serum samples collected from the general population (Calafat et al. 2006b, 2007a, 2007b; De Silva and Mabury 2006; Kuklenyik et al. 2004; Olsen et al. 2003b, 2003c, 2004b, 2004c, 2005, 2007a; Prevedouros et al. 2006). These findings have prompted efforts to reduce and even eliminate emissions of these substances from industrial process streams.

In 2006, the eight major companies of the perfluoropolymer/perfluorotelomer industry agreed to participate in EPA's PFOA Stewardship Program (EPA 2008f). This included voluntary commitments from these companies to reduce facility emissions and product content of PFOA and related chemicals on a global basis by 95% no later than 2010, and to work toward elimination of these substances by 2015 (EPA 2008f). Progress reports were provided in 2007. Data from these reports regarding releases of PFOA, PFOA precursors, and higher PFOA homologues to all media as well as percent reduction in releases are listed in Table 6-1. Total releases of these substances by these companies are uncertain since some of the data are listed as confidential business information.

Prevedouros et al. (2006) estimated the total global historical emissions of perfluoroalkyl carboxylates into the environment from both direct and indirect sources. These data are provided in Table 6-2. Based on these estimations, direct emissions (3,200–6,900 metric tons) have far exceeded indirect emissions (30–350 metric tons). The largest direct emissions identified are from industrial processes such as the manufacture of perfluoroalkyl carboxylates (470–900 metric tons), fluoropolymer manufacture (2,200–5,400 metric tons), and fluoropolymer processing (210–320 metric tons). Direct release of perfluoroalkyl carboxylates from use of aqueous firefighting foams and consumer and industrial products were estimated to be 20–100 and 40–200 metric tons, respectively. The largest indirect emissions identified were from perfluoroalkyl carboxylate residual impurities in perfluorooctylsulfonyl fluoride products (20–130 metric tons) and fluorotelomer-based precursor degradation (6–130 metric tons).

Table 6-1. Reported Emissions of PFOA, PFOA Homologues, or PFOA Precursors in Products from the 2006 U.S. Operations of Fluoropolymer/Fluorotelomer Companies

		Releases to all media from fluorotelomer and telomer manufacturing		
Company	Chemicals	kg	kg of release/100 kg of product produced	Percent reduction in emissions[a]
Arkema, Inc.	PFOA and higher homologues	>1,000–10,000	For fluorotelomer production: >0.1–1	22%
	Precursors	Not applicable	Not applicable	Not applicable
Asahi Glass Company	PFOA, PFOA salts, and higher homologues	4,922	For fluorotelomer production: <1	6%
	Precursors	Not applicable	Not applicable	Not applicable
Ciba Specialty Chemicals Corporation	PFOA	0.05[b]		>99%
	Higher homologues	0.05[b]		>99%
	Precursors	0[b]		>99%
Clariant International Ltd.	Not applicable	Not applicable	Not applicable	Not applicable
Daikin America, Inc.	PFOA	Confidential business information	For fluorotelomer production: 8.0×10^{-3}; for telomer production: 6.4×10^{-7}	94% for FP production; 92% for telomer production
	Precursors and higher homologues	Confidential business information	For telomer production: 6.4×10^{-7}	22% for telomer production
E.I. DuPont de Nemours and Company	PFOA, PFOA salts	1,100	Not reported	98%
	Direct precursors	Confidential business information	Not reported	Confidential business information
3M/Dyneon	PFOA	0	0	100%
Solvay Solexis	PFOA and PFOA salts	Not applicable	Not applicable	Not applicable
	Higher homologues	>1,000–10,000	For fluorotelomer production: 0.161	28%
	Precursors	Not applicable	Not applicable	Not applicable

[a]Percent reduction in product content of these compounds from baseline year levels. The baseline year is the year nearest to the year 2000 for which company data are available.
[b]Total for emissions and product content

PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Source: EPA 2008f

Table 6-2. Global Historical PFCA Production and Emissions Summary[a]

Environmental input source	Historical time period (years)	Estimated total global historical PFCA emissions (t)
Direct PFCA sources		
PFCA manufacture		
PFO/APFO	1951–2004	400–700
PFN/APFN	1975–2004	70–200
Total manufactured		470–900
Industrial and consumer uses		
Fluoropolymer manufacture (APFO)	1951–2004	2,000–4,000
Fluoropolymer dispersion processing (APFO)	1951–2004	200–300
Fluoropolymer manufacture (APFN)	1975–2004	400–1,400
Fluoropolymer processing (APFN)	1975–2004	10–20
Aqueous firefighting foams (AFFF)	1965–1974	50–100
Consumer and industrial products	1960–2000	40–200
Total direct		3,200–6,900
Indirect PFCA sources		
POSF-based products		
PFCA residual impurities	1960–2002	20–130
POSF-based precursor degradation	1960–2002	1–30
POSF-based AFFF	1970–2002	3–30
Fluorotelomer-based products		
PFCA residual impurities	1974–2004	0.3–30
Fluorotelomer-based precursor degradation	1974–2004	6–130
Fluorotelomer-based AFFF	1975–2004	<1
Total indirect		30–350
Total source emissions (direct and indirect)		3,200–7,300

[a]Low and high estimated values as well as the period of use/production for each source are based upon publicly available information cited in the text.

AFFF = aqueous firefighting foams; APFN = ammonium perfluorononanoate; APFO = ammonium perfluoro-octanoate; PFCA = perfluorinated carboxylic acid; PFO = perfluorooctanoate; POSF = perfluorooctanesulfonyl fluoride

Source: Prevedouros et al. 2006

3M ceased production of PFOS and related chemicals in 2002 (3M 2008a; EPA 2008f). EPA has since established the significant new use rule to limit future manufacturing and importation of these substances (EPA 2002, 2007c, 2008f). Included on the current list are PFOS, PFHxS, PFOSA, and Et-PFOSA-AcOH. Therefore, current industrial releases of these perfluoroalkyl sulfonates in the United States are expected to be negligible. In limited applications, products containing PFOS-containing chemicals may be exempted by EPA. Certain chrome plating applications, for example, utilize surfactants containing PFOS to help reduce emissions of hexavalent chromium (Agency for Toxic Substances and Disease Registry 2008b). Information regarding current releases of shorter-chain perfluoroalkyls that are not included under phase-out regulations, such as PFBA and PFBuS, have not been located. Production of PFBA in the United States appears to have ceased, although some is reportedly imported for commercial use (3M 2008a; Agency for Toxic Substances and Disease Registry 2008b).

6.2.1 Air

There is no information listed in the TRI on releases of perfluoroalkyls to the atmosphere from manufacturing and processing facilities because these releases are not required to be reported within this program (EPA 2005b).

According to 3M, PFOA was released to air during manufacturing processes at the Decatur, Alabama facility until use of this substance ceased in 2004 (3M 2008b). This company states that there are currently no air emissions of PFOA at this facility (3M 2008b). PFOA concentrations (75,000–900,000 pg/m^3) measured at the fence line of the DuPont Washington Works facility near Parkersburg, West Virginia in 2004 correlated with values modeled from wind speeds and trajectories surrounding this facility (Barton et al. 2006; Davis et al. 2007; Prevedouros et al. 2006). Based on current EPA regulations and information submitted by companies under EPA's PFOA Stewardship Program, industrial emissions of perfluoroalkyls to air are expected to be decreasing (EPA 2008f). High volume air samples collected at several monitoring stations near the Washington Works facility during nine events between August and October of 2005 contained PFOA at reported concentrations ranging from 10 to 75,900 pg/m^3 (EPA 2007d). The mean and median of these reported concentrations are 5,500 and 240 pg/m^3.

The presence of perfluoroalkyl compounds in indoor air and dust indicates that perfluoroalkyl-containing consumer products such as treated carpets and textiles are sources of release to air (Barber et al. 2007; Jahnke et al. 2007b; Kubwabo et al. 2005; Moriwaki et al. 2003; Prevedouros et al. 2006; Shoeib et al.

2004; Strynar and Lindstrom 2008). Perfluoroalkyl compounds have also been identified on both indoor and outdoor window films (Gewurtz et al. 2009). Disposal of perfluoroalkyl-containing consumer products is also expected to be a source of release to air (Prevedouros et al. 2006). Harada et al. (2005a, 2006) proposed that automobiles may be a source of PFOA in urban air based on elevated levels measured near heavy traffic areas and the widespread use of this substance in automobile materials.

Perfluoroalkyl carboxylic acids and perfluoroalkyl sulfonic acids may be formed by the atmospheric photooxidation of precursor compounds such as fluorotelomer alcohols and perfluoroalkyl sulfonamides (D'eon et al. 2006; Ellis et al. 2004; Martin et al. 2006; Wallington et al. 2006; Wania 2007). Perfluoroalkyl carboxylic acids including PFOA, PFNA, PFHpA, and PFBA were observed as products during a laboratory study involving the photooxidation of 4:2, 6:2, and 8:2 fluorotelomer alcohols (Ellis et al. 2004). D'eon et al. (2006) observed both perfluoroalkyl carboxylic acids and perfluorobutane sulfonate among products of the photooxidation of N-methyl perfluorobutane sulfonamidoethanol.

6.2.2 Water

There is no information listed in the TRI on releases of perfluoroalkyls to water from manufacturing and processing facilities because these releases are not required to be reported within this program (EPA 2005b).

Waste water discharge is also indicated as a release pathway for ammonium perfluorooctanoate (APFO) from the DuPont Washington Works facility (Davis et al. 2007). The average monthly concentrations of APFO measured in surface water from three outlets at the Washington Works facility during 2007 and early 2008 ranged from 3.65 to 377 µg/L (EPA 2008i). Reported concentrations of APFO and PFOA measured in surface water from four separate outlets at this facility during the same period were 3–64 and 2.3–61 µg/L, respectively.

During perfluorochemical operations at the 3M Cottage Grove facility in Minnesota, waste water treatment plant effluent containing perfluoroalkyl compounds was discharged to the Mississippi River. Discharge into Bakers Creek from the waste water treatment plant at the 3M Decatur facility was considered to be a principal source of PFOA release from this facility (3M 2008b). Based on current EPA regulations and information submitted by companies under EPA's PFOA Stewardship Program, industrial emissions of perfluoroalkyls to water are expected to be decreasing (EPA 2008f).

Levels of perfluoroalkyls measured in groundwater near fire-training areas are attributed to the use of these substances in aqueous fire-fighting foams (Moody and Field 1999; Moody et al. 2003). Use and disposal of perfluoroalkyl-containing consumer products is expected to be a source of release to water (Prevedouros et al. 2006).

Both PFOA and PFNA were among the identified products of the aqueous photooxidation of 8:2 fluorotelomer alcohol (Gauthier and Mabury 2005). Wang et al. (2005a, 2005b) measured PFOA as a product of the biodegradation of 8:2 fluorotelomer alcohol in an activated sludge inoculum. These results indicate that both aqueous photooxidation and biodegradation of fluorotelomer alcohols may result in the formation of perfluoroalkyl carboxylic acids in water.

A 2007 study identified perfluoroalkyl compounds in waste water from various waste water treatment plants in Minnesota. Influent, effluent, and sludge samples from 28 public and private facilities were analyzed for 13 perfluoroalkyl compounds. Several facilities, primarily urban treatment plants, were found to have higher concentrations of perfluoroalkyl compounds. Influent, effluent, and sludge from a treatment plant in Brainerd had the highest PFOS levels of all sampled facilities. The PFOS concentration in effluent was found to be 1.51 µg/L. The high levels were attributed to a chrome plating facility using a surfactant containing fluorosulfonate to control hexavalent chromium emissions (Kelly and Solem 2009). Based on these findings, EPA Region 5 began an investigation of whether chromium electroplating facilities were significant sources of PFOS and other perfluoroalkyls in the environment (EPA 2009b). Effluent samples were obtained from seven facilities located in Chicago, Illinois and four facilities in Cleveland, Ohio. It was determined that perfluoroalkyls were being discharged from all 11 facilities at quantifiable levels and that PFOS was detected in waste water from 10 out of 11 facilities at levels of 0.0314–39 µg/L (EPA 2009b). PFOA and PFOS were detected in effluents of six waste water treatment plants located in New York at levels of 0.058–1.05 and 0.003–0.068 µg/L, respectively (Sinclair and Kannan 2006). PFOS and PFOA were detected in effluents of two waste water treatment plants located in Singapore at levels of 0.0053–0.5609 and 0.0112–1.057 µg/L, respectively (Yu et al. 2009c). PFOA, PFOS, and several other perfluoroalkyls were detected in effluent samples of 21 waste water treatment plants and 9 industrial point sources (Clara et al. 2009). PFOA and PFOS were reportedly identified in the effluents of all of the facilities monitored at an average level of 0.060 µg/L for both substances. Polyfluoroalkyl phosphoric acids, a potential precursor to perfluoroalkyls such as PFOA, have also been measured in waste water treatment plant sludge (D'eon et al. 2009).

6.2.3 Soil

There is no information listed in the TRI on releases of perfluoroalkyls to soil from manufacturing and processing facilities because these releases are not required to be reported within this program (EPA 2005b).

Amounts of perfluoroalkyl compounds released to soil from industrial facilities were not located. Between 1978 and 1998, 3M disposed of PFOA-containing sludge from its waste water treatment plant at the Decatur Facility in Alabama through subsurface injection in on-site area fields (3M 2008b). The total amount of sludge applied to the former sludge incorporation area during this time period was 43,149 metric tons, dry weight. Sludge from the Decatur facility has also been disposed of at off-site landfills. During fluorochemical operations at its Cottage Grove facility in Minnesota, 3M disposed of perfluoroalkyl-containing waste at both on- and off-site locations (3M 2007b). Off-site disposal locations included the Washington County Landfill, the Oakdale Dump, and the Woodbury Disposal Site (3M 2008a). Based on current EPA regulations and information submitted by companies under EPA's PFOA Stewardship Program, industrial emissions of perfluoroalkyls to soil are expected to be decreasing (EPA 2008f).

Liu et al. (2007) measured PFOA as a product of the biodegradation of 8:2 fluorotelomer alcohol in soil. This result, along with similar findings in activated sludge tests, indicates that biodegradation of fluorotelomer alcohols may result in the formation of perfluoroalkyl carboxylic acids in soil (Liu et al. 2007; Wang et al. 2005a, 2005b).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

Based on the low pKa values (<3) for the perfluoroalkyl carboxylic acids and sulfonic acids, these compounds are expected to exist primarily as anions in the environment (Kissa 2001; SPARC 2008). Volatilization of perfluoroalkyl anions such as perfluorooctanoate (PFO) from water surfaces is expected to be negligible since ions do not volatilize (Prevedouros et al. 2006). However, due to the surfactant nature of the perfluoroalkyl compounds, some of the amount released to water may form micelles and exist in the associated form despite the low pKa values of these substances (EPA 2005a; Prevedouros et al. 2006). Perfluoroalkyl compounds that associate on water and soil surfaces may volatilize into the

atmosphere (EPA 2005a; Kim and Kannan 2007). The extrapolated vapor pressure of PFOA is 0.017 mm Hg at 20°C, indicating that the neutral form of this substance may be volatile (Barton et al. 2007).

Barton et al. (2007) explored the atmospheric partitioning of PFOA during rain events near an industrial facility and concluded that this substance will be primarily adsorbed to particles in the air since PFOA was not detected in the vapor phase (detection limit of 0.2 ng/m^3). Concentrations of PFOA in raindrops and as particulates were 11.3–1,660 ng/L and 0.09–12.40 ng/m^3. The authors proposed that PFOA or APFO released into air from industrial facilities will be scavenged by atmospheric particles (including aqueous aerosols and raindrops) and dissociate to form the perfluorooctanoate anion. Although Barton et al. (2007) did not detect PFOA in the vapor phase during rain events, low concentrations (<0.12–3.16 pg/m^3) of vapor-phase perfluoroalkyl compounds measured by Kim and Kannan (2007) in urban air provide evidence of a partitioning equilibrium. Wet and dry deposition are expected to be the principal removal mechanisms for perfluoroalkyl carboxylic acids and sulfonic acids in particulate form from the atmosphere. Residence times with respect to these processes are expected to be days to weeks (Barton et al. 2007; Hurley et al. 2004; Kim and Kannan 2007).

Estimated pKa values of 3.92 for Me- and Et-PFOSA-AcOH and 6.24 for PFOSA indicate that these compounds may exist partially in the undissociated form in the environment, especially under acidic conditions (SPARC 2008). Volatilization information are not available for these substances; however, a vapor pressure of 0.05 mm Hg at 25°C for n-ethylperfluorooctane sulfonamide (Et-PFOSA) indicates that undissociated perfluoroalkyl sulfonamides may volatilize into the atmosphere (Martin et al. 2006). Assuming that wet and dry deposition is not important for gas-phase perfluoroalkyl sulfonamides and an atmospheric photooxidation lifetime of 20–50 days, Martin et al. (2006) concluded that perfluoroalkyl sulfonamides could possibly undergo long-range transport in the atmosphere.

K_{oc} values of 17–230 measured for perfluorooctanoate in soils of various organic carbon content indicate that PFOA will be mobile in soil and will not adsorb to suspended solids and sediment in the water column (Davis et al. 2007; Prevedouros et al. 2006). This is supported by the presence of PFOA in groundwater at the Decatur, Cottage Grove, and Washington Works fluorochemical industrial facilities (3M 2007b, 2008b; Davis et al. 2007). In addition to migration to groundwater from plumes near industrial facilities, air emissions followed by atmospheric deposition to soils and subsequent leaching may also contaminate nearby groundwater (Davis et al. 2007). Low volatility, high water solubility (9,500 mg/L at 25°C), and low sorption to solids indicate that the perfluorooctanoate anion will

accumulate in surface waters, especially oceans (Armitage et al. 2006; Kauck and Diesslin 1951; Prevedouros et al. 2006; Wania 2007).

Perfluoroalkyl carboxylic acids and sulfonic acids have been widely detected in both environmental media and biota of the Arctic region (see Table 6-3) and other remote locations. The source of the perfluoroalkyl compounds at these locations is not clear. A number of source pathways for perfluoroalkyl compounds in these remote areas have been proposed and it is likely that the actual source is a combination of these (Barber et al. 2007; Prevedouros et al. 2006).

Long-range atmospheric transport of precursor compounds such as fluorotelomer alcohols and perfluoroalkyl sulfonamides followed by the atmospheric photooxidation of these substances to form perfluoroalkyl carboxylic acids and perfluoroalkyl sulfonic acids results in PFOA and PFOS contamination in remote locations with no direct point sources for these compounds (Barber et al. 2007; D'eon et al. 2006; Dinglasan-Panlilio and Mabury 2006; Ellis et al. 2004; Martin et al. 2006; Simcik 2005; Small et al. 2009; Wallington et al. 2006; Wania 2007). Fluorotelomer alcohols and perfluoroalkyl sulfonamides are relatively volatile and possess long enough atmospheric residence times for long-range transport to be possible (Barber et al. 2007; Yarwood et al. 2007). The presence of fluorotelomer alcohols and perfluoroalkyl sulfonamides in urban and Arctic air offers evidence of long-range atmospheric transport (Loewen et al. 2005; Shoeib et al. 2006; Stock et al. 2004). Photooxidation studies have demonstrated the conversion of these substances to perfluoroalkyl carboxylic acids and sulfonates (see Section 6.2.1). According to Young et al. (2007), the presence of perfluorodecanoic acid and perfluoroundecanoic acid in an Arctic ice cap indicate atmospheric oxidation as a source.

A second source of perfluoroalkyls in remote areas is direct oceanic transport of these substances (Armitage et al. 2006; Barber et al. 2007; Simcik 2005; Wania 2007; Yamashita et al. 2005, 2008). This hypothesis is supported by the presence of perfluoroalkyl compounds measured in ocean water, analysis of ocean currents directed toward the Arctic Ocean, and elevated perfluoroalkyl concentrations measured in coastal waters near industrial regions (Armitage et al. 2006; Barber et al. 2007; Prevedouros et al. 2006; Saito et al. 2003, 2004; Wania 2007; Wei et al. 2007a; Yamashita et al. 2004, 2005, 2008). A third possibility is the transport of perfluoroalkyls in the form of marine aerosols (Barber et al. 2007; CEMN 2008; Prevedouros et al. 2006). This mechanism may be especially relevant for perfluoroalkyl compounds since surfactants have been shown to accumulate in upper sea layers and at water surfaces (Prevedouros et al. 2006). Although direct atmospheric transport of perfluoroalkyl carboxylic acids and sulfonic acids was initially discounted, some researchers are suggesting that this may be a contributing

Table 6-3. Biological Monitoring of PFOA and PFOS in the Arctic

Location and organism	Concentration (ng/g) PFOA	PFOS	Reference
Northeastern Canada, 1996–2002; wet weight[a]			Tomy et al. 2004
Zooplankton (n=5)	2.6	1.8	
Clams (n=5)	ND	0.28	
Shrimp (n=7)	0.17	0.35	
Arctic cod (n=6)	0.16	1.3	
Redfish (n=7)	1.2	1.4	
Walrus (n=5)	0.34	2.4	
Narwhal (n=5)	0.9	10.9	
Beluga (n=5)	1.6	12.6	
Black-legged kittiwake (n=4)	ND	10.0	
Glaucous gulls (n=5)	0.14	20.2	
Northern Canada, 1992–2002[a]			Martin et al. 2004a
Polar bear (n=7)	8.6	3,100	
Arctic fox (n=10)	<2	250	
Ringed seal (n=9)	<2	16	
Mink (n=10)	<2	8.7	
Common loon (n=5)	<2	20	
Northern fulmar (n=5)	<2	1.3	
Black guillemot (n=5)	<2	ND	
White sucker (n=3)	<2	7.6	
Brook trout (n=2)	<2	39	
Lake whitefish (n=2)	<2	12	
Lake trout (n=1)	<2	31	
Northern pike (n=1)	<2	5.7	
Arctic sculpin (n=1)	<2	12	
Northwestern Canada, 2004			Powley et al. 2008
Zooplankton (n=3)	ND	ND–0.2	
Arctic cod (n=5)	ND	0.3–0.7	
Ringed seal (n=5)		2.5–8.6	
Bearded seal (n=1)	ND	1.3	
Northern Norway; ng/g wet weight[a]			Verreault et al. 2005, 2007
Herring gull eggs	<0.091–0.652	21.4–42.2	
Glaucous gulls			
Eggs (n=10)	<0.70	104	
Plasma (n=20)	<0.70–0.74	134	

Table 6-3. Biological Monitoring of PFOA and PFOS in the Arctic

	Concentration (ng/g)		
Location and organism	PFOA	PFOS	Reference
Nanavut, Canada			Butt et al. 2007a, 2007b
Thick-billed murres	<MDL[b]–0.16	<0.40–0.76	
Northern fulmars	<MDL[b]–0.09	<0.40–0.60	
Ringed seals	<0.85–6.2	2–20	
Northern Canada, 2002–2005			Butt et al. 2008
Ringed seal livers (n=110)	<0.7–13.9	0.89–189	
Greenland			Bossi et al. 2005
Ringed seals	<1.2	12.5–95.6	
North American and European Arctic, 1999–2002			Smithwick et al. 2005a
Polar bears (n>72)	<2.3–57.1	263–6,340	
Greenland, 1999–2001			Smithwick et al. 2005b
Polar bears (n=29)[a]	10	2,470	
Greenland, 1972–2002			Smithwick et al. 2006
Polar bears	1.6–4.4	120–1,400	

[a]Reported as mean values
[b]Minimum detection limits for study analytes ranged from 0.03 to 2.3 ng/g. To calculate means, concentrations less than the MDL were replaced with a random value that was less than half the MDL.

MDL = maximum detection limit; ND = not detected; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

source pathway based on recent atmospheric measurements of these compounds in both the vapor phase and as particulates (Barber et al. 2007; Prevedouros et al. 2006).

Perfluoroalkyls compounds have been measured in invertebrates, fish, amphibians, reptiles, birds, bird eggs, and mammals located around the world (Dai et al. 2006; Giesy and Kannan 2001; Houde et al. 2005, 2006a, 2006b; Keller et al. 2005; Kannan et al. 2001a, 2001b, 2002a, 2002b, 2002c, 2002d, 2005, 2006; Sinclair et al. 2006; So et al. 2006a; Wang et al. 2008). The highest concentrations of PFOA and PFOS in animals are measured in apex predators, such as polar bears (Table 6-3), which indicates that these substances biomagnify in food webs (de Vos et al. 2008; Houde et al. 2006b; Kannan et al. 2005; Kelly et al. 2007). The bioaccumulation potential of perfluoroalkyls increases with increasing chain length from 4 to 8 carbon units and then declines with further increases in chain length (Conder et al. 2008; de Vos et al. 2008; Furdui et al. 2007; Martin et al. 2004b). In living organisms, perfluoroalkyls bind to protein albumin in blood, liver, and eggs and do not accumulate in fat tissue, which may explain why bioconcentration factors (BCFs) are lower than expected in aquatic organisms (de Vos et al. 2008; Kissa 2001).

6.3.2 Transformation and Degradation

Perfluoroalkyl compounds are considered to be environmentally persistent chemicals (EPA 2008f; OECD 2002, 2007; Schultz et al. 2003). The carbon atoms of the perfluoroalkyl chain are protected from attack by the shielding effect of the fluorine atoms; furthermore, environmental degradation processes generally do not possess the energy needed to break apart the strong fluorine-carbon bonds (3M 2000; Hekster et al. 2003; Schultz et al. 2003). Perfluoroalkyl compounds are resistant to biodegradation, direct photolysis, atmospheric photooxidation, and hydrolysis (OECD 2002, 2007; Prevedouros et al. 2006).

6.3.2.1 Air

Although transport and partitioning information indicates that air will not be a sink for perfluoroalkyl compounds in the environment, low concentrations of perfluoroalkyl carboxylic acids, sulfonic acids, and sulfonamides have been measured in air both in the vapor phase and as bound to particulates (Barton et al. 2007; Kim and Kannan 2007). Available information indicates that photodegradation will not compete with wet deposition as an atmospheric removal process for perfluoroalkyls (Barton et al. 2007; Hurley et al. 2004; Prevedouros et al. 2006). However, photooxidation may be an important degradation mechanism for perfluoroalkyl sulfonamides (D'eon et al. 2006; Martin et al. 2006).

PFOA does not absorb UV light at environmentally relevant wavelengths (>290 nm); Hori et al. (2004a) reports a weak absorption band for PFOA that ranges from 220 to 270 nm. Based on the measured absorption wavelength of PFOA, perfluoroalkyl carboxylic acids are not expected to undergo direct photolysis. Following irradiation of the potassium salt of PFOS with light of wavelength 290–800 nm for 67–167 hours, it was concluded that there was no evidence of direct photolysis of PFOS under any of the test conditions (OECD 2002). Based on these test results for PFOS, perfluoroalkyl sulfonic acids are not expected to undergo direct photolysis in the atmosphere. Direct photolysis data were not located for perfluoroalkyl sulfonic acids.

A measured photooxidation rate constant is not available for PFOA. Hurley et al. (2004) measured the reaction of short-chain (C1–C4) perfluoroalkyl carboxylic acids with photochemically generated hydroxyl radicals. The proposed mechanism begins with abstraction of the carboxyl hydrogen, which is followed by the removal of the carboxyl group and generation of a perfluoroalkyl radical. Finally, the perfluoroalkyl chain is broken down one carbon atom at a time through an unzipping sequence. The same rate constant, 1.69×10^{-13} cm^3/molecule-second, was measured for the photooxidation of the C2, C3, and C4 molecules, indicating that the chain length may have little effect on the reactivity of perfluoroalkyls with hydroxyl radical. According to the authors, this rate constant corresponds to a half-life of 130 days. Based on the data for the short chain structures, the authors concluded that atmospheric photooxidation of perfluoroalkyl carboxylic acids is not expected to compete with wet and dry deposition, which is predicted to occur on a time scale of the order of 10 days.

Atmospheric photooxidation data are not available for perfluoroalkyl sulfonic acids. Atmospheric photooxidation studies involving n-methyl perfluorobutane sulfonamidoethanol (Me-FBSE) and n-ethyl perfluorobutanesulfonamide (Et-FBSA) indicate possible mechanisms for the reaction of these substances with atmospheric hydroxyl radicals (D'eon et al. 2006; Martin et al. 2006). Products observed from the photooxidation of these compounds indicate the following pathways: removal of an alkyl from the amide (cleavage of the N-C bond); removal of the amido group (cleavage of the S-N bond); and removal of the sulfonamido group (cleavage of the S-C bond) (D'eon et al. 2006; Martin et al. 2006). Each of these pathways would be applicable to the photooxidation of Me- and Et-PFOSA-AcOH. The last two pathways indicate that PFOSA may be photooxidized through removal of the amido or sulfonamido group. The third pathway, cleavage of the S-C bond, also indicates a photooxidation mechanism for perfluoroalkyl sulfonic acids. Martin et al. (2006) proposes an unzipping sequence for the perfluoroalkyl chain following removal of the sulfonyl group.

Measured rate constants for the reaction of Me-FBSE and Et-FBSA with atmospheric hydroxyl radicals are $5.8x10^{-12}$ and $3.74x10^{-13}$ cm^3/molecule-second, respectively (D'eon et al. 2006; Martin et al. 2006). Atmospheric half-lives calculated using these rate constants were 2 days for Me-FBSE and 20–50 days for Et-FBSA.

6.3.2.2 Water

PFOS and PFOA are expected to be stable to hydrolysis in the environment based on half-lives of 41 and 92 years, respectively, calculated from experimental hydrolysis data that were measured over pH 5, 7, and 9 (OECD 2002, 2006b). Based on the data for PFOS and PFOA, hydrolysis is not expected to be an important degradation process for perfluorinated carboxylates and sulfonates in the environment. Hydrolysis data were not located for perfluoroalkyl sulfonamides.

Available information indicates that perfluoroalkyl compounds are resistant to aerobic biodegradation. PFOA and PFNA were not biodegraded during an OECD guideline manometric respirometry screening test for ready biodegradability; 0% of the theoretical oxygen demand was reached after 28 days (Stasinakis et al. 2008). Meesters and Schröder (2004) reported that PFOA and PFOS were not degraded from an initial concentration of 5 mg/L in aerobic sewage sludge in a laboratory scale reactor.

Substances such as perfluorotelomer alcohols and perfluoroalkyl sulfonamides, which are used in a variety of products, are degraded to other substances such as PFOA and PFOS in water and can be considered a source of these substances in the environment (Liu et al. 2007).

6.3.2.3 Sediment and Soil

Data are not available regarding the transformation and degradation of perfluoroalkyl compounds in sediment and soil. Based on the chemical stability of these substances and their resistance to biodegradation in screening tests, environmental degradation processes are not expected to be important removal mechanisms for perfluoroalkyl compounds in sediment and soil (3M 2000; EPA 2008f; Hekster et al. 2003; OECD 2002, 2007; Prevedouros et al. 2006; Schultz et al. 2003).

Substances such as perfluorotelomer alcohols and perfluoroalkyl sulfonamides, which are used in a variety of products, are degraded to other substances such as PFOA and PFOS in soil and sediment and can be considered a source of these substances in the environment (Liu et al. 2007).

6.3.2.4 Other Media

Data are not available regarding the transformation and degradation of perfluoroalkyl compounds in other media.

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to perfluoroalkyls depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of perfluoroalkyls in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on perfluoroalkyls levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. The analytical methods available for monitoring perfluoroalkyls in a variety of environmental media are detailed in Chapter 7.

6.4.1 Air

Perfluoroalkyl levels have been measured in outdoor air at a few locations in the United States, Europe, Japan, and over the Atlantic Ocean (Barber et al. 2007; Barton et al. 2006; Harada et al. 2005a, 2006; Kim and Kannan 2007). Concentrations reported in these studies are provided in Table 6-4.

Mean PFOA levels ranged from 1.54 to 15.2 pg/m^3 in air samples collected in the urban locations in Albany, New York; Fukuchiyama, Japan; and Morioka, Japan and in the rural locations in Kjeller, Norway and Mace Head, Ireland. Higher mean concentrations (101–552 pg/m^3) were measured at the urban locations in Oyamazaki, Japan and Manchester, United Kingdom, and semirural locations in Hazelrigg, United Kingdom. Maximum reported concentrations at Oyamazaki and Hazelrigg were 919 and 828 pg/m^3, respectively. The authors attributed the elevated concentrations at the Hazelrigg location to emissions from a fluoropolymer production plant located 20 km upwind of this semirural community.

PFOA concentrations were above the method quantitation limit (70,000–170,000 pg/m^3) in 6 out of 28 air samples collected along the fence line of the DuPont Washington Works fluoropolymer manufacturing facility, which is located near Parkersburg, West Virginia, in the Ohio River valley (Barton et al. 2006).

Table 6-4. Concentrations of Perfluoroalkyl in Outdoor Air

Location	Mean (range) concentration (pg/m^3)			Reference
	PFOA	PFHpA	PFNA	
Urban				
Albany, New York				
Gas phase (n=8)	3.16 (1.89–6.53)	0.26 (0.13–0.42)	0.21 (0.16–0.31)	Kim and Kannan 2007
Particulate phase (n=8)	2.03 (0.76–4.19)	0.37 (<0.12–0.81)	0.13 (<0.12–0.40)	Kim and Kannan 2007
Oyamazaki, Japan (n=12)	262.7 (72–919); 3,412.8 ng/g in dust	—	—	Harada et al. 2005b
Fukuchiyama, Japan	15.2; 314 ng/g in dust	—	—	Harada et al. 2006
Morioka, Japan (n=8)	2.0 (1.59–2.58)	—	—	Harada et al. 2005b
Manchester, United Kingdom (n=2,1)[a]	341, 15.7	8.2, 0.2	<26.6, 0.8	Barber et al. 2007
Rural				
Kjeller, Norway (n=2)	1.54	0.87	0.12	Barber et al. 2007
Mace Head, Ireland (n=4)	8.9	<0.001	<3.3	Barber et al. 2007
Hazelrigg, United Kingdom (semi-rural) (n=10)	101, 552[b,c]	1.6, 14.4[b]	0.9	Barber et al. 2007
Marine air				
Near Europe (northwest) (n=3)	1.22 (0.5–2.0)	<0.6 (ND–<0.6)	0.3 (ND–0.5)	Jahnke et al. 2007a
Near Africa (east coast) (n=5)	<0.5 (ND–0.7)	ND	<0.2 (ND–0.3)	Jahnke et al. 2007a
Source dominated				
DuPont Washington Works Facility; Parkersburg, West Virginia (n=28)	430,000 (75,000–900,000)[d]	—	—	Barton et al. 2006
DuPont Washington Works Facility; Parkersburg, West Virginia (n=90)	55,260 (10–75,900)	—	—	EPA 2007d

Table 6-4. Concentrations of Perfluoroalkyl in Outdoor Air

Location	Mean (range) concentration (pg/m^3)			Reference
	PFDeA	PFUA	PFDoA	
Urban				
Albany, New York				
Gas phase (n=8)	0.63 (0.24–1.56)	<0.12 (ND–0.16)	0.27 (0.14–0.43)	Kim and Kannan 2007
Particulate phase (n=8)	0.27 (0.13–0.49)	ND	0.12 (<0.12–0.38)	Kim and Kannan 2007
Oyamazaki, Japan (n=12)	—	—	—	Harada et al. 2005b
Fukuchiyama, Japan	—	—	—	Harada et al. 2006
Morioka, Japan (n=8)	—	—	—	Harada et al. 2005b
Manchester, UK (n=2,1)[a]	5.4, <0.8	<0.01, <0.4	<0.01, <0.01	Barber et al. 2007
Rural				
Kjeller, Norway (n=2)	<0.15	<0.12	<0.12	Barber et al. 2007
Mace Head, Ireland (n=4)	<2.8	<0.002	<0.003	Barber et al. 2007
Hazelrigg, United Kingdom (semi-rural) (n=10)	1.0, 8.3[b]	0.7	<0.01	Barber et al. 2007
Marine air				
Near Europe (northwest) (n=3)	<0.6 (ND–0.6)	ND	<0.14 (ND–0.17)	Jahnke et al. 2007a
Near Africa (east coast) (n=5)	ND	0.03 (ND–0.2)	ND	Jahnke et al. 2007a
Source dominated				
DuPont Washington Works Facility – Parkersburg, West Virginia (n=28)	—	—	—	Barton et al. 2006
DuPont Washington Works Facility; Parkersburg, West Virginia (n=90)	—	—	—	EPA 2007d

Table 6-4. Concentrations of Perfluoroalkyl in Outdoor Air

Location	Mean (range) concentration (pg/m^3)				Reference
	PFOS	PFBuS	PFHxS	PFOSA	
Urban					
Albany, New York					
Gas phase (n=8)	1.70 (0.94–3.0)	—	0.31 (0.13–0.44)	0.67 (0.22–2.26)	Kim and Kannan 2007
Particulate phase (n=8)	0.64 (0.35–1.16)	—	<0.12	0.29 (<0.12–0.79)	Kim and Kannan 2007
Oyamazaki, Japan (n=12)	5.2 (2.51–-9.80); 72.2 ng/g in dust	—	—	—	Harada et al. 2005b
Fukuchiyama, Japan	2.2; 46.0 ng/g in dust	—	—	—	Harada et al. 2006
Morioka, Japan (n=8)	0.7 (0.46–1.19)	—	—	—	Harada et al. 2005b
Manchester, United Kingdom (n=2,1)[a]	46, 7.1	2.2, <1.6	1.0, 0.1	<1.6, <0.2	Barber et al. 2007
Rural					
Kjeller, Norway (n=2)	1.0	<0.09	0.05	0.78	Barber et al. 2007
Mace Head, Ireland (n=4)	<1.8	<1.0	0.07	<0.56	Barber et al. 2007
Hazelrigg, United Kingdom (semi-rural) (n=10)	1.6	2.6	0.04	0.2	Barber et al. 2007
Marine air					
Near Europe (north west) (n=3)	1.36 (0.4–2.5)	ND	0.12 (0.02–0.3)	ND	Jahnke et al. 2007a
Near Africa (east coast) (n=5)	0.544 (0.05–1.9)	ND	0.013 (ND–0.05)	ND	Jahnke et al. 2007a

Table 6-4. Concentrations of Perfluoroalkyl in Outdoor Air

Location	Mean (range) concentration (pg/m^3)				Reference
	PFOS	PFBuS	PFHxS	PFOSA	
Source dominated					
DuPont Washington Works Facility, Parkersburg, West Virginia (n=28)	—	—	—	—	Barton et al. 2006
DuPont Washington Works Facility; Parkersburg, West Virginia (n=90)	—	—	—	—	EPA 2007d

[a]Mean values were reported for separate sampling sessions.
[b]The second concentration reported was measured during an earlier sampling session (n=2).
[c]A maximum PFOA concentration of 828 pg/m^3 was measured in air at Hazelrigg, United Kingdom.
[d]Average and range of concentrations in 6 out of 28 samples that contained PFOA above the quantitation limit (70,000–170,000 pg/m^3).

ND = not detected; PFBuS = perfluorobutane sulfonic acid; PFDeA = perfluorodecanoic acid; PFDoA = perfluorododecanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid; PFOSA = perfluorooctane sulfonamide; PFUA = perfluoroundecanoic acid

The reported concentrations in these six samples ranged from 75,000 to 900,000 pg/m^3. The highest concentrations were measured at locations downwind of the facility. High volume air samples collected at several monitoring stations near the Washington Works facility contained PFOA at reported concentrations ranging from 10 to 75,900 pg/m^3 (EPA 2007d). The mean and median of these reported concentrations are 5,500 and 240 pg/m^3.

PFOS was detected above quantitation limits in most of the studies, but concentrations were generally below 5 pg/m^3. A concentration of 46 pg/m^3 was reported in samples from Manchester, United Kingdom. Reported concentrations of other perfluoroalkyls (PFHpA, PFNA, PFDeA, PFUA, PFDoA, PFBuS, PFHxS, and PFOSA) were generally <1 pg/m^3 in these studies. PFHpA was detected at slightly higher concentrations (8.2 and 14.4 pg/m^3) at Manchester and Hazelrigg, United Kingdom, respectively.

Jahnke et al. (2007a) collected eight marine air samples during a cruise between Germany and South Africa (53°N to 33°S). Perfluoroalkyl concentrations steadily declined as the sampling moved further from Europe and toward less industrialized regions. Only PFOS was detected in the two samples collected over the Atlantic Ocean east of southern Africa.

Measurements of perfluoroalkyls in snow samples collected from Canadian Arctic ice caps indicate that these substances may be generated in the atmosphere at these locations (Young et al. 2007). Reported concentrations in these snow samples were 2.6–86 pg/L for PFOS, 12–147 pg/L for PFOA, 5.0–246 ng/L for PFNA, <8–22 pg/L for PFDeA, and <6–27 pg/L for PFUA.

The concentration of PFOS measured in rainwater collected during a rain event in Winnipeg, Manitoba was 0.59 ng/L (Loewen et al. 2005). PFOA, PFNA, PFDeA, PFUA, and PFDoA were not detected in the rainwater. Reported method detection limits for these compounds were 7.2, 3.7, 1.7, 1.2, and 1.1 ng/L, respectively.

Studies of perfluoroalkyl concentrations in indoor environments are available (Table 6-5). The reported mean concentrations of perfluoroalkyls measured in four indoor air samples collected from Tromso, Norway were 0.2 pg/m^3 for PFOSA, <0.5 for PFBuS, <4.1 pg/m^3 for PFHxS, <47.4 pg/m^3 for PFOS, 0.8 pg/m^3 for PFHpA, 4.4 pg/m^3 for PFOA, 2.7 ng/m^3 for PFNA, 3.4 ng/m^3 in PFDeA, <1.3 ng/m^3 for PFUA, and 1.2 ng/m^3 for PFDoA (Barber et al. 2007).

Table 6-5. Concentrations of Perfluoroalkyl in Indoor Air

	Concentration: mean (range); median			
Location	PFOA	PFHpA	PFNA	Reference
Indoor air (pg/m^3)				
Tromso, Norway (n=4)	4.4	0.8	2.7	Barber et al. 2007
Indoor dust (ng/g)				
Ottawa, Canada (n=67)	106.00 (<2.29–1,234); 19.72[a]	—	—	Kubwabo et al. 2005
Japan (n=16)	380 (70–3,700); 165	—	—	Moriwaki et al. 2003
North Carolina and Ohio (n=112)	296 (<10.2–1960); 142 [b]	109 (<12.5–1150); 50.2[b]	22.1 (<11.3–263); 7.99[b]	Strynar and Lindstrom 2008

	Mean (range) concentration (pg/m^3)			
Location	PFDeA	PFUA	PFDoA	Reference
Indoor air (pg/m^3)				
Tromso, Norway (n=4)	3.4	<1.3	1.2	Barber et al. 2007
Indoor dust (ng/g)				
Ottawa, Canada (n=67)	—	—	—	Kubwabo et al. 2005
Japan (n=16)	—	—	—	Moriwaki et al. 2003
North Carolina and Ohio (n=112)	15.5 (<9.40–267); 6.65[b]	30.4 (<10.7–588); 7.57[b]	18.0 (<11.0–520); 7.78[b]	Strynar and Lindstrom 2008

	Mean (range) concentration (pg/m^3)				
Location	PFOS	PFBuS	PFHxS	PFOSA	Reference
Indoor air (pg/m^3)					
Tromso, Norway (n=4)	<47.4	<0.5	<4.1	2.8	Barber et al. 2007
Indoor dust (ng/g)					
Ottawa, Canada (n=67)	443.68 (<4.56–5,065); 37.8[a]	ND[a]	391.96 (<4.56–4,305); 23.1[a]	<1.38[a]	Kubwabo et al. 2005
Japan (n=16)	200 (11–2,500); 24.5	—	—	—	Moriwaki et al. 2003
North Carolina and Ohio (n=112)	761 (<8.93–12,100); 201[b]	41.7 (<12.5–1,150); 9.11[b]	874 (<12.9–35,700); 45.5[b]	—	Strynar and Lindstrom 2008

[a]Method detection limits (MDL) and percent below MDL are as follows: PFOA (2.29 ng/g, 37%), PFOS (4.56 ng/g, 33%), PFBuS (1.38 ng/g, 100%), PFHxS (4.56, 15%), and PFOSA (0.99 ng/g, 90%).
[b]Limit of quantitation (LOQ) and percent above LOQ are as follows: PFHpA (12.5 ng/g, 74.1%), PFOA (10.2 ng/g, 96.4%), PFNA (11.3 ng/g, 42.9%), PFDeA (9.40 ng/g, 30.4%), PFUA (10.7 ng/g, 36.6%), PFDoA (11.0 ng/g, 18.7%), PFOS (8.93 ng/g, 94.6%), PFHxS (12.9 ng/g, 77.7%), PFBuS (12.5 ng/g, 33.0%). Values below the LOQ were assigned a value of LOQ/1.412 when calculating the median.

ND = not detected; PFBuS = perfluorobutane sulfonic acid; PFDeA = perfluorodecanoic acid; PFDoA = perfluorododecanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid; PFOSA = perfluorooctane sulfonamide

Kubwabo et al. (2005) measured the concentrations of selected perfluoroalkyls in dust samples from 67 Canadian homes. PFOA, PFOS, and PFHxS were each detected in 37, 33, and 15% of these samples, respectively (detection limits of 2.29, 4.56, and 4.56 ng/g, respectively). Mean, median, and range of concentrations in these samples were 106, 19.72, and 1.15–1,234 ng/g, respectively, for PFOA; 443.68, 37.8, and 2.28–5,065 ng/g, respectively, for PFOS; and 391.96, 23.1, and 2.28–4,305 ng/g, respectively, for PFHxS. Concentrations were not reported for PFOSA, which was detected above 0.99 ng/g in 10% of the samples. PFBuS was not detected in any of the samples. Moriwaki et al. (2003) measured PFOS and PFOA concentrations in vacuum cleaner dust samples collected from 16 Japanese homes. PFOS and PFOA were detected in every sample with reported concentrations of 11–140 and 69–380 ng/g, respectively, in 15 of the 16 samples. One of the samples contained 2,500 ng/g PFOS and 3,700 ng/g PFOA.

Strynar and Lindstrom (2008) measured perfluoroalkyl levels in 112 indoor dust samples collected from homes and daycare centers in North Carolina and Ohio. These authors detected PFHpA, PFOA, PFNA, PFDeA, PFUA, PFDoA, PFOS, PFHxS, and PFBuS. Mean values ranged from 15.5 to 874 ng/g. PFOS and PFOA were detected in 94.6 and 96.4% of the samples, respectively. Maximum detections in the samples were as high as 12,100 ng/g for PFOS and 35,700 ng/g for PFHxS. Household dust samples collected from the United Kingdom, Australia, Germany, and the United States showed the presence of perfluoroalkyl substances (Kato et al. 2009a). These data are summarized in Table 6-6.

Median levels of PFOS in dust samples collected in homes, apartments, daycare centers, offices, and cars in Sweden were 39, 85, 31, 110, and 12 ng/g, respectively (Bjorklund et al. 2009). Median PFOA levels in dust samples from the same study were 54, 93, 41, 70, and 33 ng/g in homes, apartments, daycare centers, offices, and cars, respectively. The authors concluded that while dietary intake was the major PFOA/PFOS exposure pathway for adults and toddlers in the general population, dust ingestion could become an important pathway under a worst-case scenario (high dust ingestion and maximum dust levels).

6.4.2 Water

PFOS and PFOA have been widely detected in surface water samples collected from various rivers, lakes, and streams in the United States (Boulanger et al. 2004; Kannan et al. 2005; Kim and Kannan 2007; Nakayama et al. 2007; Simcik and Dorweiler 2005; Sinclair et al. 2004, 2006). Less data are available regarding the concentrations of other perfluoroalkyl compounds in surface water. PFHpA and PFHxS

Table 6-6. Concentration (ng/g) of Perfluoroalkyls in 39 Dust Samples[a]

Analyte	25th percentile	50th percentile	75th percentile	Maximum	Frequency of detection (%)
PFBuS	86.3	359.0	782.1	7718	92.3
PFHxS	47.7	185.5	632.2	43,765	79.5
PFOS	31.7	479.6	1,456.6	18,071	74.4
PFHpA	33.9	97.3	532.5	5,195	61.2
PFOA	<LOQ	96.5	667.7	9,818	64.1
PFNA	<LOQ	<LOQ	26.2	832	25.6
PFDeA	<LOQ	<LOQ	61.0	1,965	38.5
PFUA	<LOQ	<LOQ	<LOQ	732	20.5
PFDoA	<LOQ	<LOQ	37.6	1,048	43.6
PFOSA	<LOQ	<LOQ	16.1	184	23.1
Me-PFOSA-AcOH	<LOQ	<LOQ	110.3	4,520	33.3
Et-PFOSA-AcOH	92.4	243.5	417.9	3,795	87.2

[a]The LOQs are 2.6 ng/g except for PFHpA, which is 4.0 ng/g.

Et-PFOSA-AcOH = 2-(N-ethyl-perfluorooctane sulfonamide) acetic acid; LOQ = limit of quanitification; Me-PFOSA-AcOH = 2-(N-methyl-perfluorooctane sulfonamide) acetic acid; PFBuS = perfluorobutane sulfonic acid; PFDeA = perfluorodecanoic acid; PFDoA = perfluorododecanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid; PFOSA = perfluorooctane sulfonamide; PFUA = perfluoroundecanoic acid

were commonly detected in the few studies that analyzed surface water for these compounds (Kim and Kannan 2007; Nakayama et al. 2007; Simcik and Dorweiler 2005). Concentrations of perfluoroalkyls measured in surface water are listed in Tables 6-7 and 6-8. Reported concentrations of perfluoroalkyls in surface water samples are generally below 50 ng/L. Maximum concentrations of PFOS, PFOA, PFHpA, PFNA, PFDeA, PFUA, PFDoA, PFBuS, and PFHxS measured in surface water collected from the Cape Fear Basin, North Carolina were 287, 132, 329, 194, 120, 52.1, 4.46, 9.41, and 35.1 ng/L, respectively (Nakayama et al. 2007). Much higher concentrations of PFOS (198–1,090 ng/L) have been measured in Onondaga Lake in Syracuse, New York (Sinclair et al. 2006). Onondaga Lake is a Superfund site that has become contaminated through industrial activity along its banks. High levels were also reported around Dalton, Georgia, an area with a high density of carpet manufacturing locations (Konwick et al. 2008).

Levels of some perfluoroalkyl compounds measured in surface water and groundwater surrounding perfluorochemical industrial facilities are listed in Table 6-9. Maximum PFOS and PFOA concentrations measured in surface water downstream of the 3M Decatur, Alabama facility were 144 and 598 ng/L, respectively (Hansen et al. 2002).

The average monthly concentrations of APFO measured in surface water from three outlets at the Washington Works facility during 2007 and early 2008 ranged from 3.65 to 377 µg/L (EPA 2008i). Reported concentrations of APFO and PFOA measured in surface water from four separate outlets at this facility during the same period were 3–64 and 2.3–61 µg/L, respectively. Levels of APFO and PFOA measured in groundwater samples collected from three wells at the Washington Works facility during 2007 and early 2008 were 2.9–100 and 2.8–100 µg/L, respectively (EPA 2008i). Information regarding background concentrations of perfluoroalkyls in groundwater in the United States has not been located. PFOS was routinely detected in groundwater samples from the Tokyo, Japan metropolitan area, with its occurrence being traced to the degradation of precursors released to the environment and waste water treatment plants (Murakami et al. 2009).

Yamashita et al. (2005) measured PFOA, PFOS, PFNA, and PFHxS concentrations in ocean water collected from locations in the Atlantic Ocean, Pacific Ocean, and areas near China, Korea, and Japan. These concentrations are listed in Table 6-10. Wei et al. (2007a) measured perfluoroalkyl concentrations in surface seawaters from the western Pacific Ocean, Indian Ocean, and near-Antarctic region. PFOS and PFOA were detected in 60 and 40% of the samples, respectively, with maximum concentrations of 71.7 and 441.6 pg/L, respectively. Concentrations of other perfluoroalkyls (PFHxS, PFBuS, PFDoA, PFDeA, PFNA, PFHpA) were generally below detection in most samples, with the exceptions being in

Table 6-7. Concentrations of PFOA and PFOS in Surface Water (ng/L)

Location	Concentration		Reference
	PFOA	PFOS	
Great Lakes			Boulanger et al. 2004
Lake Ontario (n=8)	15–70	6–121	
Lake Erie (n=8)	21–47	11–39	
New York State waters			Sinclair et al. 2006
Lake Ontario (n=13)	18–34	2.9–30	
Niagara River (n=3)	18–22	3.3–6.7	
Lake Erie (n=3)	13–27	2.8–5.5	
Finger Lakes (n=13)	11–20	1.3–2.6	
Onondaga Lake (n=3)	39–64	198–1,090 (median=756)	
Oneida Lake (n=1)	19	3.5	
Erie Canal (n=3)	25–59	5.7–13	
Hudson River (n=8)	22–173 (median=35)	1.5–3.4	
Lake Champlain (n=4)	10–46	0.8–7.7	
Albany, New York			Kim and Kannan 2007
Lake water (n=11)	3.27–15.8 (median=7.20)	ND–9.30 (median=2.88)	
Surface water runoff (n=14)	0.51–29.3 (median=3.80)	<0.25–14.6 (median=0.81)	
Michigan water regions			Sinclair et al. 2004
Detroit (n=10)	<8–16.14	<0.08–6.13	
Flint (n=4)	<8–23.01	1.50–12.31	
Saginaw Bay (n=5)	<8–24.08	3.10–12.69	
Northeastern Michigan (n=2)	<8	0.87–6.34	
Upper Peninsula (n=7)	<8–13.77	<0.8–3.09	
Northwestern Michigan (n=2)	11.96	<0.8–4.48	
Western Michigan (n=6)	<8–15.17	<0.8–5.32	
Southwestern Michigan (n=5)	8.74–35.86	7.22–29.26	
Lansing (n=3)	<8–13.37	1.04–4.96	

Table 6-7. Concentrations of PFOA and PFOS in Surface Water (ng/L)

Location	Concentration		Reference
	PFOA	PFOS	
Minnesota Waters and Lake Michigan			Simcik and Dorweiler 2005
Remote (n=4)			
Loiten	0.7	ND	
Little Trout	0.3	1.2	
Nipisiquit	0.1	ND	
Tettegouche	0.5	0.2	
Urban (n=4)			
Calhoun	20	47	
Lake Harriet	3.5	21	
Lake of the Isles	0.5	2.4	
Minnesota River	1.2	9	
Lake Michigan (n=4)	<0.6–0.5	1–3.2	
Cape Fear Basin, North Carolina			Nakayama et al. 2007
80 Sites (n=100)			
Mean	43.4	31.2	
Median	12.6	28.9	
Minimum	ND	<1	
Maximum	287	132	
Percent not detected[a]	7.6	0	
Raisin and St. Clair Rivers, Michigan			Kannan et al. 2005
Raisin River	14.7	3.5	
St. Clair River (n=3)	4.0–5.0	1.9–3.9	
Conasauga River, Georgia	253–1,150	192–318	Konwick et al. 2008
Dalton, Georgia	49.9–299	15.8–120	
Several rivers in Japan	0.1–67,000	0.3–59	Harada and Koizumi 2009
Lake Victoria Gulf, Kenya	0.4–96.4 (rivers) 0.4–11.6 (lakes)	<0.4–13.23 (rivers); <0.4–2.53 (lakes)	Orata et al. 2009
River Po, Italy	1–1,270	1–25	Loos et al. 2008

[a]Detection limit is 0.05 ng/L

ND = not detected; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Table 6-8. Concentrations of Other Perfluoroalkyls in Surface Water

	Concentration (ng/L)								
Location (reference)[a]	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Et-PFOSA-AcOH
Great Lakes (Boulanger et al. 2004)									
Lake Ontario (n=8)	—	—	—	—	—	—	—	—	<0.3–10
Lake Erie (n=8)	—	—	—	—	—	—	—	—	3–11
New York State waters (Sinclair et al. 2006)									
Onondaga Lake (n=3)	—	—	—	—	—	—	4.2–8.5	—	—
Erie Canal (n=3)	—	—	—	—	—	—	2.5–5.6	—	—
Other lakes and rivers	—	—	—	—	—	—	0.9–2.8	—	—
Albany, New York (Kim and Kannan 2007)									
Lake water (n=11)	1.15–12.7	ND–3.51	0.25–3.58	ND–1.45	ND–<0.25	—	<0.25–4.05	<0.25	—
Surface water runoff (n=14)	<0.25–6.44	<0.25–5.90	ND–8.39	ND–1.99	ND–1.60	—	ND–13.5	ND–2.14	—
Minnesota waters and Lake Michigan (Simcik and Dorweiler 2005)									
Remote (n=4)									
Loiten	10	ND	ND	—	—	—	—	—	—
Little Trout	4.8	ND	ND	—	—	—	—	—	—
Nipisiquit	0.9	<0.3	ND	—	—	—	—	—	—
Tettegouche	3.1	ND	ND	—	—	—	—	—	—
Urban (n=4)					—	—	—	—	—
Calhoun	11	0.6	0.5	—	—	—	—	—	—
Lake Harriet	2.6	ND	ND	—	—	—	—	—	—
Lake of the Isles	0.4	ND	ND	—	—	—	—	—	—
Minnesota River	0.7	1.9	ND	—	—	—	—	—	—
Lake Michigan (n=4)	<0.6–4.1	<0.6–3.1	ND	–	—	—	—	—	—
Cape Fear Basin, North Carolina (Nakayama et al. 2007)									
80 Sites (n=100)									
Mean	38.7	33.6	22.1	10.4	2.17	2.58	7.29	—	—
Median	14.8	5.70	13.2	5.67	1.95	2.46	5.66	—	—
Maximum	329	194	120	52.1	4.46	9.41	35.1	—	—

Table 6-8. Concentrations of Other Perfluoroalkyls in Surface Water

Location (reference)[a]	Concentration (ng/L)								
	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Et-PFOSA-AcOH
Percent not detected[b]	32.9	10.1	15.2	17.7	53.2	38.0	45.6	—	—
Raisin and St. Clair Rivers, Michigan (Kannan et al. 2005)									
Raisin River	—	—	—	—	—	—	<1	<10	—
St. Clair River (n=3)	—	—	—	—	—	—	<1	<10	—

[a]See Table 6-7 for numbers of samples collected at these locations.
[b]Detection limit = 0.05 ng/L.

Et-PFOSA-AcOH = 2-(N-ethyl-perfluorooctane sulfonamide) acetic acid; ND = not detected; PFBuS = perfluorobutane sulfonic acid; PFDeA = perfluorodecanoic acid; PFDoA = perfluorododecanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOSA = perfluorooctane sulfonamide; PFUA = perfluoroundecanoic acid

Table 6-9. Concentrations of Perfluoroalkyls in Surface Water and Groundwater at Fluorochemical Industrial Facilities

	Percent detection and concentration (µg/L)					
Location	PFOA	PFBA	PFOS	PFHxS	PFBuS	Reference
DuPont Washington Works Facility, West Virginia						
Groundwater						
Borings (n=18)						Davis et al. 2007
Percent detected	89%[a]	—	—	—	—	
Minimum	0.0912[a]	—	—	—	—	
Maximum	78[a]	—	—	—	—	
Wells (n=14)			—	—	—	Davis et al. 2007
Percent detected	100%[a]	—	—	—	—	
Minimum	0.081[a]	—	—	—	—	
Maximum	37.1[a]	—	—	—	—	
Wells (n=3)			—	—	—	EPA 2008i
Percent detected	100%	—	—	—	—	
Minimum	2.8	—	—	—	—	
Maximum	100	—	—	—	—	
Surface water						
Outlets (n=4)						EPA 2008i
Percent detected	100%	—	—	—	—	
Minimum	2.3	—	—	—	—	
Maximum	61	—	—	—	—	
3M Cottage Grove Facility, Minnesota						
Groundwater						
Wells (n=1–7)						3M 2007b
Percent detected	100%	100%	100%	100%	100%	
Minimum	24.6	23.3	26.0	6.47	2.11	
Maximum	619	318	26.0	40.0	26.1	
Surface water						
East and West Cove (n=3–9)						3M 2007b
Percent detected	100%	100%	100%	100%	78%	
Minimum	0.172	0.803	0.227	0.0936	0.304	
Maximum	2.79	1.01	3.12	4.58	9.69	
Mississippi River Shoreline (n=52–80)						3M 2007b
Percent detected	60%	52%	43%	28%	56%	
Maximum	0.760	6.92	0.539	1.04	3.05	
Mississippi River Transect (n=34–44)						3M 2007b
Percent detected	14%	12%	0%	0%	0%	
Maximum	0.0501	0.0530	ND	ND	ND	

Table 6-9. Concentrations of Perfluoroalkyls in Surface Water and Groundwater at Fluorochemical Industrial Facilities

	Percent detection and concentration (µg/L)					
Location	PFOA	PFBA	PFOS	PFHxS	PFBuS	Reference
3M Decatur Facility, Alabama						
Groundwater						
Off-site groundwater (n=18)						3M 2008c
Percent detected	94%	—	—	—	—	
Mean	1.87	—	—	—	—	
Range	0.083–19.8	—	—	—	—	
Surface water						
On-site surface water (n=7)						3M 2008c
Percent detected	100%	—	—	—	—	
Median	2.66	—	—	—	—	
Range	0.32–127	—	—	—	—	
Off-site surface water (n=60)						3M 2008c
Percent detected	98%	—	—	—	—	
Range	0.026–27.7	—	—	—	—	
Tennessee River						Hansen et al. 2002
Upstream of facility (n=19)						
Percent detected	0%	—	100%	—	—	
Range	<25	—	16.8–52.6	—	—	
Downstream of facility (n=21)				—	—	
Percent detected	0%	—	100%	—	—	
Median	355	—	107	—	—	
Range	<25–598	—	30.3–144	—	—	

[a]Analyte was reported as APFO.

APFO = ammonium perfluorooctanoate; ND = not detected; PFBA = perfluorobutyric acid; PFBuS = perfluorobutane sulfonic acid; PFHxS = perfluorohexane sulfonic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Table 6-10. Concentrations of PFOA and PFOS in Ocean Water

	Concentration (pg/L)			
Location	PFOA	PFOS	PFNA	PFHxS
North Atlantic (n=9)	160–338	8.6–36	15–36	4.1–6.1
Mid Atlantic (n=7)	100–439	37–73	—	2.6–12
Central to Eastern Pacific (n=14)	15–62	1.1–20	1.0–16	0.1–1.6
Western Pacific (n=2)	136–142	54–78	—	2.2–2.8
Tokyo Bay (n=8)	1,800–192,000	338–57,700	163–71,000	17–5,600
Offshore Japan (n=4)	137–1,060	40–75	–	3.0–6.1
Coastal Hong Kong (n=12)	673–5450	70–2,600	22–207	<5–311
Coastal China (n=14)	243–15,300	23–9,680	2.0–692	<5–1,360
Coastal Korea (n=10)	239–11,350	39–2,530	15–518	<5–1,390
Sulu Sea (n=5)	88–510	<17–109	—	<0.2
South China Sea (n=2)	160–420	8–113	—	<0.2

PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Source: Yamashita et al. 2005

samples collected near Shanghai, the Philippines, and Indonesia. Maximum concentrations of these perfluoroalkyls ranged from 3.1 to 70.2 pg/L near Shanghai. PFOA, PFOS, and other perfluoroalkyl species were monitored in waste water effluents and 20 rivers located in Japan (Murakami et al. 2008). Perfluoroalkyls were ubiquitous in the river water samples, with concentrations of PFOA as large as 0.054–0.192 µg/L in seven of the river samples with low waste water effluent sources.

The widespread presence of PFOA and PFOS at low concentrations in surface water in the United States indicates that drinking water taken from these sources may contain detectable levels of these substances. PFOA was detected in 12 out of 13 samples collected from four municipal drinking water treatment plants that draw water from the Tennessee River and are located downstream from the 3M Decatur Facility in Alabama. Reported concentrations range from 0.025 to 0.16 µg/L (3M 2008c). PFOA was not detected in any samples collected from a fifth plant located upstream of the 3M Decatur facility (3M 2008c).

Based on a memorandum of understanding with the EPA, DuPont began collecting water monitoring data of both public and private wells near the Washington Works chemical plant. The quarterly reports and monitoring data affiliated with these reports may be obtained from the regulations.gov portal (http://www.regulations.gov/#!home). In samples of water collected at 17 public water facilities from 2002 to 2009 in West Virginia and Ohio, PFOA levels ranged from below the detection limit (0.0023 µg/L) to nearly 100 µg/L in a few test wells in Little Hocking, Ohio (EPA 2010). Emmett et al. (2006a) reported an average PFOA concentration of 3.55 µg/L in residential drinking water from the Little Hocking community, which is located across the Ohio River from the DuPont Washington Works Facility.

According to the Agency for Toxic Substances and Disease Registry (2008), PFOA, PFOS, PFBA, PFHxS, and PFBuS have been detected in the municipal drinking water of communities located near the 3M Cottage Grove fluorochemical facility. According to Chang et al. (2008a), concentrations of PFBA were generally in the low ng/L range in effluent at these locations, but could be in the µg/L range in public and private wells.

PFOS concentrations ranging from 0.1 to 4 ng/L were measured in tap water samples collected from the areas of Morioka City, Iwate, Tokyo, and Kyoto in Japan (Harada et al. 2003).

Concentrations of 43.7 and 50.9 ng/L were measured in samples of tap water originating from the PFOS-contaminated Tama River. PFOA was detected in 65% of the public drinking water systems tested in

New Jersey in 2006 at concentrations ranging from 5 to 39 ng/L (Post et al. 2009). PFOA and PFOS were detected in tap water from 21 cities located in China at concentrations of <0.1–45.9 and <0.1–14.8 ng/L, respectively (Jin et al. 2009). Mak et al. (2009) published a study comparing detections of perfluoroalkyls including PFOA and PFOS in tap water collected in China, Japan, India, Canada and the United States. PFOA and PFOS were the predominant species measured, accounting for 40–50% of the total perfluoroalkyls present in water, with the exception of certain location of India where PFOS or PFOA may not have been present or were present at low levels.

6.4.3 Sediment and Soil

Background environmental levels of perfluoroalkyl compounds in sediment and soil were not located. Levels of some perfluoroalkyl compounds measured in soil and sediment surrounding perfluorochemical industrial facilities are listed in Table 6-11. PFOA was detected in most soil and sediment samples collected on- and off-site at the 3M Decatur facility in Alabama. Maximum soil concentrations were as high as 14,750 ng/g on-site and 7.85 ng/g off-site, and maximum sediment concentrations were as high as 347 on-site and 2,385 ng/g off-site (3M 2008c). The highest levels of PFOA were measured in soil from on-site fields formerly injected with PFOA-containing sludge. Grazing land polluted with perfluoroalkyl-contaminated sludge potentially resulted in contamination of the food supply. Studies of tissue levels of perfluorinated compounds in cattle are ongoing.

PFOA, PFOS, and PFHxS were detected in 90–100% of soil samples collected from a former tar neutralization area, a former sludge disposal area, a former solids burn pit area, a former waste water treatment plant area, and a former fire training area at the 3M Cottage Grove facility in Minnesota (3M 2007b). PFBuS was detected in 60–73% of these samples. Maximum concentrations for these substances were 21,800, 104,000, 3,470, and 139 ng/g, respectively. Levels of PFBuA were only reported for soil in the fire training area; it was detected in 9 out of 11 samples from this location at 0.306–9.07 ng/g. The percent detection of these compounds in sediment from the East and West Cove sites was similar to that in soil. Maximum concentrations of PFOA and PFOS were 1,845 and 65,450 ng/g, respectively. These perfluoroalkyls were also analyzed for in Mississippi River sediment near the Cottage Grove Facility. Levels of these compounds were much greater along the facility shoreline compared to levels in transect samples collected at points crossing the river. Maximum shoreline concentrations for PFOA, PFBuA, PFOS, PFHxS, and PFBuS were 341, 124, 79.0, 11.5, and 29.4 ng/g, respectively. PFHxS, PFBuS, and PFBuA were not detected in any of the transect samples and PFOA was found in only 18%. Although the maximum concentration of PFOS was 3.16 ng/g, it was still detected in 82% of the transect samples.

Table 6-11. Concentrations of Perfluoroalkyls in Soil and Sediment at Fluorochemical Industrial Facilities

	Percent detection and concentration (ng/g)					
Location	PFOA	PFBA	PFOS	PFHxS	PFBuS	Reference
DuPont Washington Works Facility, West Virginia						
Soil						
Boring samples (n=22)						Davis et al. 2007
Percent detected	36%[a]	—	—	—	—	
Minimum	<0.17[a]	—	—	—	—	
Maximum	170[a]	—	—	—	—	
3M Cottage Grove Facility, Minnesota						
Soil						
Boring samples (n=50–108)						3M 2007b
Percent detected	100%	—	95%	90%	60%	
Maximum	21,800	—	104,000	3,470	139	
Fire training area (n=8–11)						3M 2007b
Percent detected	91%	82%	100%	100%	73%	
Maximum	262	11.5	2,948	62.2	24.6	
Sediment						
East and West Cove (n=21–28)						3M 2007b
Percent detected	100%	93%	100%	96%	65%	
Minimum	0.764	ND	40.0	ND	ND	
Maximum	1,845	94.6	65,450	126	9.14	
Mississippi River shoreline (n=84–92)						3M 2007b
Percent detected	70%	44%	80%	28%	29%	
Maximum	341	124	79.0	11.5	29.4	
Mississippi River transect (n=38–40)						3M 2007b
Percent detected	18%	0%	82%	0%	0%	
Maximum	1.09	ND	3.16	ND	ND	

Table 6-11. Concentrations of Perfluoroalkyls in Soil and Sediment at Fluorochemical Industrial Facilities

	Percent detection and concentration (ng/g)					
Location	PFOA	PFBA	PFOS	PFHxS	PFBuS	Reference
3M Decatur Facility, Alabama						
Soil						
On-site former sludge incorporation area (n=357)						3M 2008c
Percent detected	99%	—	—	—	—	
Mean	885–929					
Range	2.91–14,750	—	—	—	—	
On-site background (n=18)						3M 2008c
Percent detected	100%	—	—	—	—	
Mean	3.53–4.1					
Range	1.61–6.03	—	—	—	—	
Off-site soil (n=23)						3M 2008c
Percent detected	100%	—	—	—	—	
Mean	3.68–4.6					
Range	0.72–7.85	—	—	—	—	
Sediment						
On-site sediment (n=8)						3M 2008c
Percent detected	88%	—	—	—	—	
Median	16.8					
Range	1.64–347	—	—	—	—	
Off-site sediment (n=30)						3M 2008c
Percent detected	93%	—	—	—	—	
Range	0.39–2,385	—	—	—	—	

[a]Analyte was reported as APFO.

APFO = ammonium perfluorooctanoate; ND = not detected; PFBA = perfluorobutyric acid; PFBuS = perfluorobutane sulfonic acid; PFHxS = perfluorohexane sulfonic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Paustenbach et al. (2007) estimated PFOA concentrations in environmental media for communities located near the DuPont Washington Works chemical manufacturing facility. From this analysis, the authors concluded that much of the PFOA detected in groundwater near the facility was attributed to deposition to soil surfaces following atmospheric emissions from the plant followed by subsequent leaching into groundwater.

6.4.4 Other Environmental Media

Limited data are available regarding the concentrations of perfluoroalkyl compounds in food. One study has been located that analyzed foods in the United States for PFOS, PFOA, and PFOSA (3M 2001). During this study, over 200 food items were collected from grocery stores in three U.S. test cities having commercial perfluoroalkyl manufacturing or use and from grocery stores in three U.S. control cities that do not have this type of activity. Twelve samples contained perfluoroalkyls above the limit of quantification. Eight of the positive detections were collected in test cities. PFOSA was not detected in any of the food samples. PFOS was detected in four whole milk samples (0.573–0.852 ng/g) and 3 ground beef samples (0.570–0.587 ng/g). PFOA was detected in two ground beef samples (0.504, 1.09 ng/g), two bread samples (0.524, 14.7 ng/g), two apple samples (1.13, 2.35 ng/g), and one green bean sample (0.543 ng/g). The author's state that concentration of 14.7 ng/g measured for PFOA in the one bread sample may have resulted from contamination.

Concentrations of perfluoroalkyls have been reported in foods sampled in Canada, the United Kingdom, and Germany (Food Standards Agency 2006; Fromme et al. 2007b; Tittlemier et al. 2007). Perfluoroalkyls were detected in only 9 out of 54 food composites collected during Canadian Total Diet Studies from 1992 to 2004 (Tittlemier et al. 2007). PFOS was detected in beef steak, ground beef, luncheon meats, marine fish, freshwater fish, and microwave popcorn at concentrations ranging from 0.98–2.7 ng/g, wet weight. PFOA was detected in roast beef, pizza, and microwave popcorn at 0.74–3.6 ng/g, wet weight. PFHpA was detected in pizza and microwave popcorn at 1.5–2.0 ng/g, wet weight. PFNA was detected only in beef steak at 4.5 ng/g, wet weight. PFDeA, PFUA, and PFDoA were analyzed for but not detected in any of the food composites. During the U.K. Food Standards Agency Total Diet Study, PFOS was detected in eggs, sugars and preserves, potatoes, and canned vegetables at 1, 1, 10, and 2 μg/kg, respectively (Food Standards Agency 2006). PFOA was detected only in potatoes at 1 μg/kg. Neither substance was detected in the bread, miscellaneous cereals, carcass meats, offal, meat

products, poultry, fish, oils and fats, green vegetables, other vegetables, fresh fruit, fruit products, beverages, milk, dairy products, or nuts categories. Fromme et al. (2007b) detected PFOS, PFOA, and PFHxS in 33, 45, and 3% of 214 daily duplicate food portions for 31 adults in the city of Munich, Germany. Concentrations were 0.025–1.03 ng/g fresh weight for PFOS, 0.025–118.29 ng/g fresh weight for PFOA, and 0.05–3.03 ng/g fresh weight for PFHxS. Reported 90th percentile values were 0.11 and 0.21 ng/g fresh weight for PFOS and PFOA, respectively (Fromme et al. 2007b).

Limited data are available regarding the levels of perfluoroalkyls in food packaging; however, some measurements have been performed. PFOA was detected in the packaging paper of two microwave popcorn bags at 0.3–4.7 ng/cm^2 uncooked and 0.5–4.3 ng/cm^2 cooked (Sinclair et al. 2007). The mean mass of PFOA in the gas phase of popcorn vapors following popping was 16–17 ng/cm^2. PFHpA, PFNA, PFDeA, PFUA, and PFDoA were detected in one of the bags at 0.4–3.2 ng/cm^2 uncooked and 0.5–4.3 ng/cm^2 cooked; however, these perfluoroalkyls were not detected (<0.2 ng/cm^2) in the second bag. Begley et al. (2005) measured PFOA concentrations of 6–290 μg/kg in microwave popcorn bags. These authors also tested a hamburger wrapper, a sandwich wrapper, a French fry box, and soak-proof paper plates and did not find PFOA above the detection limit in these products. These paper products were not necessarily coated with fluorochemicals. The concentration of PFOA measured in undiluted perfluoro paper coating formulations ranged from 88,000 to 160,000 μg/kg (Begley et al. 2005).

Washburn et al. (2005) measured the concentration of the perfluorooctanoate anion in fluorotelomer treated consumer articles as well as the fluorotelomer formulations used for the treatments. PFOA was detected in mill-treated carpeting (0.2–0.6 mg/kg), carpet-care solution-treated carpeting (0.2–2 mg/kg), treated apparel (<0.02–1.4 mg/kg), treated home textiles (<0.02–1.4 mg/kg), industrial floor waxes and wax removers (0.0005–0.06 mg/kg), latex paint (0.02–0.08 mg/kg), and home and office cleaners (0.005–0.05 mg/kg). The concentrations of PFOA measured in the formulations used for these applications were 30–80, 1–50, <1–40, <1–40, 5–120, 50–150, and 50–150 mg/L, respectively. PFOA was not detected in treated upholstery (<0.034 mg/kg), treated technical textiles (<0.034 mg/kg), treated nonwoven medical garments (<0.034 mg/kg), or stone, tile, and wood sealants (<0.1 mg/kg).

PTFE is a fluoropolymer used in applications such as nonstick cookware coatings and plumbing sealant tape; PFOA has been used as a processing aid in the manufacture of PTFE (DuPont 2008). DuPont states that PFOA is removed from the fluoropolymer material during the baking and curing step of nonstick cookware coatings in a high temperature oven and that there may be trace amounts of residual PFOA in the final coatings (DuPont 2008). Begley et al. (2005) has measured PFOA concentrations of 4–75 μg/kg

in PTFE cookware, 3 μg/kg in PTFE-based dental floss, 4 μg/kg in PTFE-based dental tape, and 1,800 μg/kg in PTFE film/sealant tape. PFOA was not detected in tubing made of a fluoro-ethylene-propene copolymer (Begley et al. 2005).

Studies have been conducted that investigated the release of PFOA from PTFE cookware when heated. Sinclair et al. (2007) reported PFOA release concentrations ranging from 19–287 pg/cm^2 measured using four nonstick frying pans. These concentrations were measured at normal cooking temperatures–within the range of 180–229°C. PFOA was detected in water (7 and 75 ng) boiled for 10 minutes in two out of five non-stick pans (Sinclair et al. 2007). PFOA was not found above the detection limit (0.1 ng/cm^2) during 40 extraction tests on PTFE cookware using an ethanol/water mixture (Washburn et al. 2005). Likewise, Powley et al. (2005) conducted extraction texts on commercial fluoropolymer-treated cookware using water and water/ethanol mixtures at 100 and 125°C. Under simulated cooking conditions, PFOA was not identified above the detection limit of 100 pg/cm^2. Begley et al. (2005) reported that additional PFOA was not generated in the PTFE coating of three empty pans heated to 320°C (DuPont 2008). According to DuPont, the non-stick coating on a pan may begin to deteriorate if the pan is accidentally heated above 348°C, which is well above the maximum recommended cooking temperature of 260°C (DuPont 2008). Although it is possible for an unattended empty pan to reach these high temperatures, overheating non-stick cookware is expected to be prevented in most cases because food oils begin to generate smoke around 190°C (Begley et al. 2005).

Perfluoroalkyl compounds have been identified on both indoor and outdoor window films at urban, suburban, and rural locations near Toronto, Ontario, Canada. The sum of perfluoroalkyls contaminant (ΣPFC) concentrations on outdoor window films ranged from 0.04 to 0.75 pg/cm^2 in winter and from 0.04 to 0.92 pg/cm^2 in summer, with higher values found in urban and suburban locations than in rural locations. Indoors, ΣPFC concentrations on window film ranged from less than the detection limit (which ranged from 25 to 50 pg) to 2.1 pg/cm^2 in winter and from 0.08 to 4.3 pg/cm^2 in summer, although there were no distinct trends between urban and rural for indoor concentrations (Gewurtz et al. 2009).

A comprehensive study that examined 116 articles of commerce (AOC) found perfluorocarboxylic acids, including PFOA, in many commercially available substances, such as carpet care products and waxes (EPA 2009c). Levels of PFOA ranged from nondetectable to 6,750 ng/g, and levels of total perfluorocarboxylic acids (the sum of C5–C12 acids) ranged from nondetectable to 47,100 ng/g. Perfluoroalkyl compounds, including PFOA, have been detected at low levels in personal care products such as cosmetics and sunscreens (Fujii et al. 2013).

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

As a group of compounds, perfluoroalkyls appear to be ubiquitous in human blood based on the widespread detection of these substances in human serum samples (Calafat et al. 2006b, 2007a, 2007b; De Silva and Mabury 2006; Kuklenyik et al. 2004; Olsen et al. 2003b, 2003c, 2004b, 2004c, 2005, 2007a). Tables 6-12 and 6-13 list concentrations of perfluoroalkyl compounds measured in serum samples collected from the general population in the United States. Mean PFOA, PFOS, and PFHxS serum concentrations reported in various studies from the United States were 2.1–9.6, 9.32–55.8, and 1.5–3.9 ng/mL, respectively. Mean concentrations of PFHpA, PFNA, PFDeA, PFUA, PFDoA, PFBuS, PFOSA, Me-PFOSA-AcOH, and Et-PFOSA-AcOH are generally <1 ng/mL in these studies. Biomonitoring data for PFBA in the general population have not been located.

The widespread presence of perfluoroalkyl compounds in blood is well illustrated in studies by Calafat et al. (2007a, 2007b). These authors reported perfluoroalkyl levels in human serum collected during the 1999–2000 and 2003–2004 periods of the National Health and Nutrition Examination Survey (NHANES). The numbers of individuals included in the analyses for each survey period were 1,562 and 2,094, respectively. PFOA, PFOS, PFNA, and PFHxS were detected in 95–100% of serum samples collected during both survey periods. Mean concentrations for PFOA, PFOS, and PFHxS declined by 10–30% in the 2003–2004 survey, while PFNA values doubled from 0.5 to 1.0 ng/mL. NHANES survey data from 2005–2006, 2007–2008, and 2009–2010, have generally continued to show declining levels of PFOA and PFOS in human serum samples (CDC 2014). A dramatic difference in detection frequency was observed for PFOSA, Me-PFOSA-AcOH, and Et-PFOSA-AcOH which were widely detected (91–100%) during the 1999–2000 survey period but were present in only 3.4–27.5% of samples collected during the 2003–2004 survey period. PFDoA and PFBuS were detected in <1% of the NHANES samples. Olsen et al. (2008) reported a nearly 60% decline in PFOS blood levels when comparing data from 2001 to 2006 American Red Cross surveys of participants.

The widespread detection of perfluoroalkyl compounds in the blood of U.S. residents demonstrates that exposure of the general population to these substances is common. Levels of perfluoroalkyl compounds have been measured in indoor air, outdoor air, dust, food, surface water, and various consumer products. Possible exposure pathways have been proposed; however, the relative importance of these pathways including their association with the accumulation of perfluoroalkyls in blood remains unclear (Apelberg

Table 6-12. Concentrations of PFOA and PFOS in Human Serum Collected in the United States

	Detection and concentration (ng/mL [ppb])[a]		
Location	PFOA	PFOS	Reference
U.S. residents—NHANES			
1999–2000 (n=1,562)			Calafat et al. 2007a
Percent >LOD	100%	100%	
Geometric mean	5.2	30.4	
95th percentile	11.9	75.6	
2003–2004 (n=2,094)			Calafat et al. 2007b
Percent >LOD	99.7%	99.9%	
Geometric mean	3.9	20.7	
95th percentile	9.8	54.6	
U.S. residents—NHANES			
Percent >LOD	NR	NR	
Geometric mean	3.07	9.32	
95th percentile	7.50	32.0	
U.S. residents			Calafat et al. 2006b
1990–2002 (n=23)			
Percent >LOD	100%	100%	
Geometric mean	9.6	30.0	
95th percentile	23.0	52.3	
U.S. blood donors			Olsen et al. 2003b
2000–2001 (n=645)			
Percent >LLOQ	100%	92.5%	
Geometric mean	4.6	34.9	
95th percentile[b]	12.1	88.5	
Maximum	52.3	1,656.0	
U.S. residents			Olsen et al. 2003c
(n=24)			
Percent >LLOQ	Not reported	98%	
Geometric mean	2.5	14.7	
Minimum	<3.0	<6.1	
Maximum	7.0	58.3	
Midwestern United States			De Silva and Mabury 2006
2004–2005 (n=16)[c]			
Percent detected	100%	Not detected	
Mean	4.4		
Maximum	8.6		

Table 6-12. Concentrations of PFOA and PFOS in Human Serum Collected in the United States

	Detection and concentration (ng/mL [ppb])[a]		
Location	PFOA	PFOS	Reference
Minneapolis-St. Paul blood donors (plasma)			Olsen et al. 2007b
2005 (n=40)			
Percent >LLOQ	95%	100%	
Geometric mean	2.2	15.1	
75th percentile	3.5	20.2	
Maximum	4.7	36.9	
Atlanta, Georgia			Kuklenyik et al. 2004
2003 (n=20)			
Percent >LOD	100%	100%	
Mean	4.9	55.8	
Minimum	0.2	3.6	
Maximum	10.4	164.0	
Seattle, Washington elderly individuals			Olsen et al. 2004c
(n=238)			
Percent >LLOQ	99.2%	99.5%	
Geometric mean	4.2	31.0	
95th percentile[b]	9.7	84.1	
Maximum	16.7	175.0	
Washington County, Maryland			Olsen et al. 2005
1974 (n=178)			
Percent >LLOQ	71%	100%	
Geometric mean	2.1	30.1	
75th percentile	3.0	40.2	
1989 (n=178)			
Percent >LLOQ	99%	100%	
Geometric mean	5.5	33.3	
75th percentile	6.7	44.0	

Table 6-12. Concentrations of PFOA and PFOS in Human Serum Collected in the United States

	Detection and concentration (ng/mL [ppb])[a]		
Location	PFOA	PFOS	Reference
Boston, Massachusetts; Charlotte, North Carolina; Hagerstown, Maryland; Los Angeles, California; Minneapolis-St. Paul, Minnesota; Portland, Oregon			Olsen et al. 2008
2006 (n=600)	99%	99%	
Percent >LLOQ			
Geometric mean	3.4	14.5	
95th percentile CI geometric mean	3.3–3.6	13.9–15.2	

[a]"Less than" values indicate that the concentration was reported as below the LOD or LLOQ. For cases where samples had concentrations below the limit of detection or lower limit of quantification, a value between zero and the LOD or LLOQ was assigned when calculating the mean concentration.
[b]Reported as bias-corrected estimates.
[c]One sample purchased separately with no origin information supplied.

CI = confidence interval; LLOQ = lower limit of quanitification; LOD = limit of detection; NR = not reported; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Table 6-13. Concentrations of Other Perfluoroalkyls in Human Serum Collected in the United States

	Detection and concentration (ng/mL [ppb])[a]									
Sample population	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Me-PFOSA-AcOH	Et-PFOSA-AcOH
U.S. residents NHANES										
1999–2000 (n=1,562) (Calafat et al. 2007a)										
Percent >LOD	10%	95%	25%	12%	<1%	—	100%	100%	96%	91%
Geometric mean	<0.4	0.5	<0.2	<0.2	<0.2		2.1	0.4	1.0	0.6
95th percentile	NR	1.7	0.5	NR	NR		8.7	1.4	3.2	2.2
2003–2004 (n=2,094) (Calafat et al. 2007b)										
Percent >LOD	6.2%	98.8%	31.3%	9.7%	<0.1%	<0.4%	98.3%	22.2%	27.5%	3.4%
Geometric mean	<0.3	1.0	<0.3	<0.3	<1.0	<0.4	1.9	<0.2	<0.6	<0.4
95th percentile	0.4	3.2	0.8	0.6	<1.0	<0.4	8.3	0.2	1.3	<0.4
2005–2006 (n=2,120) (CDC 2013)										
Percent >LOD	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Geometric mean	—	1.09	0.355	—	—	—	1.67	—	0.41	—
95th percentile	0.7	3.6	1.5	0.7	<LOD	0.1	8.3	0.3	1.57	0.3
2007–2008 (n=2,100) (CDC 2013)										
Percent >LOD	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Geometric mean	—	1.22	0.286	—	—	—	1.95	—	0.301	—
95th percentile	0.5	3.28	0.9	0.6	<LOD	<LOD	9.8	<LOD	1.31	<LOD
2009–2010 (n=2,233) (CDC 2013)										
Percent >LOD	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Geometric mean	—	1.26	0.279	0.172	—	—	1.66	—	0.189	—
95th percentile	0.2	3.77	0.9	0.9	<LOD	<LOD	6.9	<LOD	0.96	0.1
U.S. residents (Calafat et al. 2006b)										
1990–2002 (n=23)										
Percent >LOD	0%	8.7%	0%	13%	0%	—	91.3%	26.1%	13%	56.5%
Geometric mean	NA	<0.3	NA	<0.3	NA		1.6	<0.4	<0.6	<0.4
95th percentile	NA	0.3	NA	1.3	NA		2.7	0.7	1.9	2.5

Table 6-13. Concentrations of Other Perfluoroalkyls in Human Serum Collected in the United States

	Detection and concentration (ng/mL [ppb])[a]									
Sample population	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Me-PFOSA-AcOH	Et-PFOSA-AcOH
U.S. blood donors (Olsen et al. 2003b)										
2000–2001 (n=645)										
Percent >LLOQ	—	—	—	—	—	—	64%	2%	49%	58%
Geometric mean							1.9	NR	<1.8	<2.8
95th percentile[b]							9.5	NR	5.0	7.6
Maximum							66.3	NR	16.4	60.1
U.S. residents (Olsen et al. 2003c)										
(n=24)										
Geometric mean	—	—	—	—	—	—	1.8	3.0	—	—
Minimum							<1.2	<1.3		
Maximum							5.9	22.1		
Midwestern United States (De Silva and Mabury 2006)										
2004–2005 (n=16)										
Percent detected	—	100%	100%	13%	0%	—	—	—	—	—
Mean		0.77	0.17	NR	NA					
Maximum		1.2	0.25	0.067	NA					
Atlanta, Georgia (Kuklenyik et al. 2004)										
2003 (n=20)										
Percent >LOD	10%	100%	75%	85%	10%	—	100%	75%	100%	90%
Mean[b]	<0.3	2.6	0.7	0.8	<1		3.9	0.34	1.7	0.9
Maximum	8.5	3.9	1.2	1.4	1.6		11.2	0.7	5.2	1.4
Seattle, Washington (Olsen et al. 2004c)										
(n=238)										
Percent >LLOQ	—	—	—	—	—	—	76%	"Few"	65%	52%
Geometric mean							2.2	NR	1.2	<1.6
95th percentile[b]							8.3	NR	3.8	7.8
Maximum							40.3	NR	6.6	21.1

Table 6-13. Concentrations of Other Perfluoroalkyls in Human Serum Collected in the United States

	Detection and concentration (ng/mL [ppb])[a]									
Sample population	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Me-PFOSA-AcOH	Et-PFOSA-AcOH
Washington County, Maryland (Olsen et al. 2005)										
1974 (n=178)										
Percent >LLOQ	—	—	—	—	—	—	63%	0%	4%	33%
Geometric mean							1.5	NA	0.5	1.2
75th percentile							2.5	NA	<1.0	1.8
1989 (n=178)										
Percent >LLOQ	—	—	—	—	—	—	82%	0%	38%	93%
Geometric mean							2.5	NA	0.8	3.6
75th percentile							1.6	NA	1.3	4.7

[a]"Less than" values indicate that the concentration was reported as below the LOD or LLOQ. For cases where samples had concentrations below the limit of detection or lower limit of quantification, a value between zero and the LOD or LLOQ was assigned when calculating the mean concentration.
[b]Reported as bias-corrected estimates.
[c]Arithmetic mean of positive concentrations.

Et-PFOSA-AcOH = 2-(N-ethyl-perfluorooctane sulfonamide) acetic acid; LLOQ = lower limit of quanitification; LOD = limit of detection; Me-PFOSA-AcOH = 2-(N-methyl-perfluorooctane sulfonamide) acetic acid; NA = not applicable; NR = not reported; PFBuS = perfluorobutane sulfonic acid; PFDeA = perfluorodecanoic acid; PFDoA = perfluorododecanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOSA = perfluorooctane sulfonamide; PFUA = perfluoroundecanoic acid

et al. 2007b; Begley et al. 2005; Calafat et al. 2006b; Trudel et al. 2008; Washburn et al. 2005). For populations that have elevated levels of perfluoroalkyls in water supplies, the primary route of exposure is expected to be ingestion of contaminated drinking water. Using a stratified random sample of residents in the Little Hocking Water district in Ohio between July 2004 and February 2005, Emmett et al. (2006a) reported median serum PFOA levels of 329 ng/mL in residents' drinking water, with a mean PFOA concentration of 3.55ng/mL. Median serum PFOA levels were 371 ng/mL in residents for whom this was the only residential water source, and 71 ng/mL in those who used bottled, cistern, or spring water. Increased serum PFOA was associated with increasing number of drinks of tap water daily and also with increasing use of water for making soups and stews and in-home canning of fruits and vegetables. Use of a carbon water filter reduced PFOA levels by about 25%. In a follow-up study, 231 study participants in the Little Hocking Water District were evaluated 15 months later with 88% using bottled water exclusively; 8% had made other changes to their ingestion of residential water including use of activated carbon water filters. PFOA levels had decreased an average of 26% from the initial levels (Emmett et al. 2009).

A study conducted by the Minnesota Department of Health reported higher PFOA and PFOS serum levels in residents of two communities with contaminated water supplies as compared to the general population (MN EPHT 2009). Similar findings have been reported by Steenland et al. (2009) in a study of residents in six water districts in the mid-Ohio Valley located near the DuPont Washington Works facility in Washington, West Virginia. The Minnesota Department of Health instituted a program to reduce levels of perfluoroalkyls in drinking water that included using granulated activated carbon (GAC) filters for home use in areas where private wells showed high levels of contamination and large GAC filters for the municipal water supplies (MN EPHT 2009). In two Mid-Ohio Valley locations with PFOA-contaminated drinking water, blood serum levels of PFOA in residents declined significantly following the implementation of GAC filtration of the public water supply (Bartell et al. 2010). The Lubeck, West Virginia and Little Hocking, Ohio public water systems, which were contaminated with PFOA from the DuPont Washington Works facility, began GAC treatment to remove PFOA from the potable water supply in 2007. The average decrease in serum PFOA levels for Lubeck, West Virginia residents primarily consuming public water at home (n=130) was 26% a year after treatment began. Similar trends were reported for residents of Little Hocking, Ohio. The average decrease in PFOA serum levels for residents primarily consuming public water (n=39) was about 11% 6 months after treatment began (Bartell et al. 2010).

Trudel et al. (2008) provide a thorough analysis of general population exposure to PFOS and PFOA based on the available information and have proposed the following possible exposure pathways: food and water consumption, ingestion of house dust, hand-to-mouth transfer from treated carpets, migration into food from PFOA-containing paper or cardboard, inhalation of indoor and ambient air, and inhalation of impregnation spray aerosols. Other pathways proposed to be less significant included oral exposure from hand-to-mouth contact with clothes and upholstery, migration into food prepared with PTFE-coated cookware, dermal exposure from wearing treated clothes, deposition of spray droplets on skin while impregnating, skin contact with treated carpet and upholstery, and deposition of dust onto skin (Trudel et al. 2008). The strong correlation between PFOA and PFOS concentrations in human serum samples indicates that common exposure pathways for these two substances are possible (Calafat et al. 2007a).

In order to estimate human uptake and the major pathways for human exposure to PFOS and PFOA, reported levels of these compounds in various environmental media, including food and consumer products, were analyzed with respect to product use patterns, personal activity patterns, and personal intake rates (Trudel et al. 2008). For PFOS, the major exposure pathways in a high-exposure scenario were proposed to be food and water ingestion, dust ingestion, and hand-to-mouth transfer from mill-treated carpets. Relative contributions of these pathways to the total uptake of PFOS in adults were estimated to be approximately 80, 15, and 5%, respectively (Trudel et al. 2008). For PFOA, the major exposure pathways in a high-exposure were proposed to be oral exposure resulting from migration from paper packaging and wrapping into food, general food and water ingestion, inhalation from impregnated clothes, and dust ingestion. Relative contributions of these pathways to the total uptake of PFOA in adults were estimated to be approximately 60, 15, 15, and 10%, respectively (Trudel et al. 2008). Major exposure pathways for the intermediate and low exposure scenarios were proposed to be through food and drinking water (PFOA and PFOS) and ingestion of house dust (PFOA only).

Based on these proposed exposure pathways, adult uptake doses estimated for low, medium, and high exposure scenarios were approximately 7, 15, and 30 ng/kg body weight/day, respectively, for PFOS and approximately 0.4, 2.5, and 41–47 ng/kg body weight/day, respectively, for PFOA (Trudel et al. 2008). The estimated uptake values were similar for men and women.

Fromme et al. (2009) assessed human exposure to perfluoroalkyls for adults in the general population of western countries. These authors determined average daily exposure levels of 1.6 ng/kg body weight/day for PFOS and 2.9 ng/kg body weight/day for PFOA. Upper daily exposure levels were determined to be 8.8 ng/kg body weight/day for PFOS and 12.6 ng/kg body weight/day for PFOA. These authors

concluded that the oral route, especially diet, was the primary route of exposure to perfluoroalkyls (Fromme et al. 2007a, 2007b, 2009). The geometric mean adult daily intakes for PFOA and PFOS were estimated as 92.6 and 83.3 ng/day, respectively, for residents in Kansai, Japan and 53.7 and 63.8 ng/day, respectively, for residents in Tohoku, Japan (Harada and Koizumi 2009). The most important exposure pathway for both compounds was food ingestion.

Limited information has been located regarding pathways of human exposure to PFBA, PFHpA, PFNA, PFUA, PFDoA, PFBuS, PFHxS, PFOSA, Me-PFOSA-AcOH, and Et-PFOSA-AcOH.

Limited monitoring data are available for PFBA. Monitoring efforts conducted in Washington County, Minnesota near the 3M Cottage Grove Facility revealed widespread contamination of this substance in the groundwater of that area in 2006. This compound has since also been detected along with PFOA, PFOS, PFHxS, and PFBuS in municipal drinking water in Washington County (Agency for Toxic Substances and Disease Registry 2008). Chang et al. (2008a) measured concentrations of PFBA in the serum of 127 former employees and 50 current employees of the 3M Cottage Grove Facility in Minnesota. PFBA serum concentrations were below the detection limit in 73.2% of the former employees and 68.0% of the current employees. Only 4% of the serum samples contained PFBA above 2 ng/mL with maximum concentrations of 6.2 ng/mL for the former employees and 2.2 ng/mL for the current employees.

Another possible source for perfluoroalkyls in human blood is through uptake of precursor compounds and then conversion of these within the human body (Trudel et al. 2008). For example, Me-PFOSA-AcOH and Et-PFOSA-AcOH are the oxidation products of 2-(N-methyl-per-fluorooctane sulfonamido) ethanol and 2-(N-ethyl-per-fluorooctane sulfonamido) ethanol, which have been used in surface treatment applications (Calafat et al. 2006a). Concentrations of Me- and Et-PFOSA-AcOH measured in human serum may have resulted from exposure of individuals to these perfluoroalkyl sulfonamido ethanols and then conversion of the ethanols to the perfluoroalkyl sulfonamido acetates within the body.

Levels of perfluoroalkyl compounds measured in the blood of occupationally exposed individuals are listed in Table 6-14. 3M has estimated doses for various on-site exposure scenarios based on monitoring information collected at the Decatur Facility in Alabama (3M 2008c). Occupational exposure scenarios included groundskeeper/maintenance worker and construction/utility worker exposed to on-site soils, surface water, and sediment. According to 3M, estimated on-site exposure to PFOA ranges from $3.2x10^{-6}$ to 2.4 ng/kg/day, with the highest estimated exposure corresponding to construction/utility

Table 6-14. Concentrations of PFOA, PFOS, and PFHxS in Human Serum for Occupationally Exposed Individuals

	Concentration (µg/mL [ppm])			
Location	PFOA	PFOS	PFHxS	Reference
Decatur, Alabama				
1993 (n=111)	0.00–80.00; 89% <8.92	—	—	Olsen et al. 1998
1995 (n=80)	0.00–114.10; 81% <8.20	—	—	Olsen et al. 1998
1995 (n=90)	—	96% <6.00	—	Olsen et al. 1999
1997 (n=84)	—	94% <6.00	—	Olsen et al. 1999
2000 (n=263)	1.78; 0.04–12.70	1.32; 0.06–10.06	—	Olsen et al. 2003a
1999–2004 (n=26)[a]				Olsen et al. 2007a
Initial	0.691 (0.072–5.1)	0.799 (0.145–3.49)	0.290 (0.016–1.30)	
Final	0.262 (0.017–2.44)	0.403 (0.037–1.74)	1.85 (0.01–0.791)	
Cottage Grove, Minnesota				
1993 (n=111)	0.00–80.00; 88% <8.92	—	—	Olsen et al. 2000
1995 (n=80)	0.00–114.1; 81% <8.20	—	—	Olsen et al. 2000
1997 (n=74)	0.05–81.35; 85% <7.66	—	—	Olsen et al. 2000
2000 (n=122)	4.63 (0.01–92.03)	0.86 (0.03–4.79)	—	Olsen and Zobel 2007
Washington Works, Little Hocking, Ohio				
2004–2005				Emmett et al. 2006a
No occupational exposure (n=312)	0.423 (0.175–0.537)[b]	—	—	
Potential occupational exposure (n=48)	0.406 (0.168–0.623)[b]	—	—	
Substantial occupational exposure (n=18)	0.824 (0.422–0.999)[b]	—	—	
Antwerp, Belgium				
1995 (n=88)	—	75% <6.00	—	Olsen et al. 1999
1997 (n=65)	—	86% <6.00	—	Olsen et al. 1999
2000 (n=255)	0.84 (0.01–7.04)	0.80 (0.04–6.24)	—	Olsen et al. 2003a
Miteni, Trissino, Italy				
2007				
Current occupational exposure (n=39)	5.71[c] (0.20–47.04)	—	—	Costa et al. 2009

Table 6-14. Concentrations of PFOA, PFOS, and PFHxS in Human Serum for Occupationally Exposed Individuals

	Concentration (µg/mL [ppm])			
Location	PFOA	PFOS	PFHxS	Reference
Former occupational exposure (n=11)	4.43[c] (0.53–18.66)	—	—	Costa et al. 2009
2000 (n=25)	11.92[c] (1.54–86.3)	—	—	Costa et al. 2009
2001 (n=42)	11.07[c] (0.73–91.9)	—	—	Costa et al. 2009
2002 (n=46)	10.15[c] (0.34–91.9)	—	—	Costa et al. 2009
2003 (n=41)	6.25[c] (0.38–74.7)	—	—	Costa et al. 2009
2004 (n=34)	6.82[c] (0.54–46.3)	—	—	Costa et al. 2009
2006 (n=49)	5.27[c] (0.54–41.9)	—	—	Costa et al. 2009
2007 (n=50)	3.89[c] (0.20–47.0)	—	—	Costa et al. 2009
Washington Works				
2004				
Current occupational exposure (n=259)	0.494 (0.0174–9.550)	—	—	Sakr et al. 2007b
Intermittent current occupational exposure (n=160)	0.176 (0.0081–2.070)	—	—	Sakr et al. 2007b
Past occupational exposure (n=264)	0.195 (0.0086–2.590)	—	—	Sakr et al. 2007b
No occupational exposure (n=342)	0.114 (0.0046–0.963)	—	—	Sakr et al. 2007b

[a]Data include results from three retirees from the 3M plant in Cottage Grove, Minnesota.
[b]Reported as the interquartile range.
[c]Reported as the median value.

PFHxS = perfluorohexane sulfonic acid; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

workers engaged in projects that involve contact with soil from an onsite field. Individuals who perform jobs that require frequent contact with perfluoroalkyl containing products, such as fire fighters, waste handlers, and individuals who install and treat carpets, are also expected to have occupational exposure to these substances (Emmett et al. 2006a). However, Emmett et al. (2006a) determined that levels of PFOA in the serum of these types of individuals were only slightly higher than the non-occupational exposure group (388 ng/mL compared to 329 ng/mL, respectively) while serum levels in workers at a fluoropolymer manufacturing facility were much higher (775 ng/mL).

6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in Section 3.7, Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993).

Perfluoroalkyl compounds have been detected in childhood serum samples, human breast milk, and umbilical cord blood; reported concentrations are listed in Tables 6-15 and 6-16. Measurements of perfluoroalkyl compounds in amniotic fluid, meconium, neonatal blood, or other tissues have not been located.

A few studies are available that report serum levels of perfluoroalkyls measured in children. Calafat et al. (2007a, 2007b) reported perfluoroalkyl serum concentrations measured in 543–640 adolescents who make up the 12–19-year-old age subpopulation in the 1999–2000 and 2003–2004 NHANES surveys. Olsen et al. (2003a) measured PFOA, PFOS, PFHxS, PFOSA, Me-PFOSA-AcOH, and Et-PFOSA-AcOH in the serum of 598 children of ages 2–12 from various locations in the United States who have been diagnosed with group A streptococcal infections. Mean serum concentrations of perfluoroalkyl compounds

Table 6-15. Percent Detection and Levels of PFOA and PFOS in Children's Serum, Umbilical Cord Blood, and Breast Milk

	Detection and concentration (ng/mL [ppb])		
Location	PFOA	PFOS	Reference
Serum			
U.S. adolescents—NHANES (ages 12–19)			
1999–2000 (n=543)			Calafat et al. 2007a
Percent >LOD	100%	100%	
Geometric mean	5.5	29.1	
95th percentile	11.2	56.8	
2003–2004 (n=640)			Calafat et al. 2007b
Percent >LOD	99.7%[a]	99.9%[a]	
Geometric mean	3.9	19.3	
95th percentile	8.6	42.2	
2005–2006 (n=640)			CDC 2013
Percent >LOD	NR	NR	
Geometric mean	3.59	15.0	
95th percentile	8.40	38.5	
2007–2009 (n=357)			CDC 2013
Percent >LOD	NR	NR	
Geometric mean	3.91	11.3	
95th percentile	7.30	28.0	
2009–2010 (n=364)			CDC 2013
Percent >LOD	NR	NR	
Geometric mean	2.74	6.84	
95th percentile	5.00	18.1	
U.S. children (ages 2–12)			
1994–1995 (n=598)			Olsen et al. 2004b
Percent >LLOQ	97–99%	100%	
Geometric mean	4.9	37.5	
95th percentile[b]	10	89	
U.S. Children (ages 6–11)			Kato et al. 2009b
2001–2002 (n=936)			
Percent >LLOQ	NR	NR	
Arithmetic mean	6.1–7.6	30.45–42.45	
95th percentile	95th percentile	36.51–48.51	

Table 6-15. Percent Detection and Levels of PFOA and PFOS in Children's Serum, Umbilical Cord Blood, and Breast Milk

	Detection and concentration (ng/mL [ppb])		
Location	PFOA	PFOS	Reference
Umbilical cord blood			
Baltimore THREE Study			Apelberg et al. 2007a, 2007b
Cord serum (n=299)			
Percent >LOD	100%	99%	
Geometric mean	1.6	4.9	
Minimum	0.3	<0.2	
Maximum	7.1	34.8	
Maternal serum (n=293)			
Median	1.4–1.6	4.1–5.0	
Germany			Midasch et al. 2007
Cord plasma (n=11)			
Percent detected	100%	100%	
Median	3.4	7.3	
Maternal plasma (n=11)			
Percent detected	100%	100%	
Median	2.6	13.0	
Danish National Birth Cohort			Fei et al. 2007
Cord blood (n=50)			
Mean	3.7	11.0	
Maternal blood (n=200)			
Mean	4.5	29.9	
Japan			Inoue et al. 2004b
Cord serum (n=15)			
Percent detected	0%	100%	
Minimum	<0.5	1.6	
Maximum	No data	5.3	
Maternal serum (n=15)			
Percent detected	20%	100%	
Minimum	<0.5	4.9	
Maximum	2.3	17.6	

Table 6-15. Percent Detection and Levels of PFOA and PFOS in Children's Serum, Umbilical Cord Blood, and Breast Milk

	Detection and concentration (ng/mL [ppb])		
Location	PFOA	PFOS	Reference
Breast milk			
Massachusetts (n=45)			Tao et al. 2008
Milk			
Percent >LOQ	89%	96%	
Median	0.0361	0.106	
Minimum	<0.0301	<0.032	
Maximum	0.161	0.617	
Sweden (n=12)			Kärrman et al. 2007
Milk			
Percent >LOD	8%[c]	100%	
Minimum	<0.209	0.060	
Maximum	0.492	0.470	
Maternal serum			
Percent >LOD	100%	100%	
Minimum	2.4	8.2	
Maximum	5.3	48.0	
China (n=19)			So et al. 2006b
Percent >LOD	100%	100%	
Minimum	0.047	0.045	
Maximum	0.210	0.360	
Germany/Hungary (n=70)			Völkel et al. 2008
Percent >LOQ	16%	100%	
Minimum	<0.200	0.028	
Maximum	0.460	0.639	

[a]Percent detection for the adolescent age group was not specified for the 2003–2004 NHANES samples. Percentages listed here are for the total sample population.
[b]Reported as bias-corrected estimates.
[c]All 12 samples were above the detection limit (0.01 ng/mL); however, levels were only reported for one sample due to a high blank level for this substance (0.209 ng/mL).

LLOQ = lower limit of quantification; LOD = limit of detection; LOQ = limit of quantification; NR = not reported; PFOA = perfluorooctanoic acid; PFOS = perfluorooctane sulfonic acid

Table 6-16. Percent Detection and Levels of Other Perfluoroalkyls in Children's Serum, Umbilical Cord Blood, and Breast Milk

	Detection and concentration (ng/mL [ppb])[a]									
Sample population	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Me-PFOSA-AcOH	Et-PFOSA-AcOH
Serum										
U.S. NHANES (ages 12–19)										
1999–2000 (n=543) (Calafat et al. 2007a)										
Percent >LOD	10%[a]	96%	15%	12%[a]	<1%[a]	—	100%	100%	100%	98%
Geometric mean	—	0.5	<0.2	—	—	—	2.7	0.4	1.3	0.8
95th percentile	—	1.1	0.5	—	—	—	12.9	1.5	3.7	2.4
2003–2004 (n=640) (Calafat et al. 2007b)										
Percent >LOD	6.2%[a]	98.8%[a]	31.3%[a]	9.7%[a]	<0.1%[a]	<0.4%[a]	98.3%[a]	22.2%[a]	27.5%[a]	3.4%[a]
Geometric mean	<0.3	0.9	<0.3	<0.3	<1.0	<0.4	2.4	<0.2	<0.6	<0.4
95th percentile	0.5	2.7	0.7	<0.3	<1.0	<0.4	13.1	0.3	1.4	<0.4
2005–2006 (n=640) (CDC 2013)										
Percent >LOD	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Geometric mean	—	0.929	0.295	—	—	—	2.09	—	0.432	—
95th percentile	1.10	2.7	0.8	0.5	<LOD	0.1	14.1	0.3	1.48	0.2
2007–2008 (n=357) (CDC 2013)										
Percent >LOD	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Geometric mean	—	0.929	0.295	—	—	—	2.09	—	0.432	—
95th percentile	1.10	2.7	0.8	0.5	<LOD	0.1	14.1	0.3	1.48	0.2
2009–2010 (n=357) (CDC 2013)										
Percent >LOD	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Geometric mean	—	1.1	0.22	—	—	—	2.03	—	0.225	—
95th percentile	0.4	2.62	0.6	0.4	<LOD	<LOD	12.3	<LOD	0.87	<LOD

Table 6-16. Percent Detection and Levels of Other Perfluoroalkyls in Children's Serum, Umbilical Cord Blood, and Breast Milk

	Detection and concentration (ng/mL [ppb])[a]									
Sample population	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Me-PFOSA-AcOH	Et-PFOSA-AcOH
U.S. children (ages 2–12)										
1994–1995 (n=598) (Olsen et al. 2004b)										
Percent >LLOQ	—	—	—	—	—	—	85%	14%	77%	92%
Geometric mean	—	—	—	—	—	—	4.5	<2.0	1.9	3.3
95th percentile[b]	—	—	—	—	—	—	65	<2.0	12	10
Umbilical cord blood										
Baltimore THREE Study (Apelberg et al. 2007a, 2007b)										
Cord serum (n=299)										
Percent >LOD	2%	—	24%	34%	5%	3%	—	26%	40%	1%
Minimum	<0.4	—	<0.2	<0.2	<0.2	<0.1	—	<0.05	<0.2	<0.2
Maximum	2.6	—	1.1	1.9	1.7	0.2	—	0.8	1.8	0.5
Japan (Inoue et al. 2004b)										
Cord serum (n=15)										
Percent detected	—	—	—	—	—	—	—	0%	—	—
Maternal serum (n=15)										
Percent detected	—	—	—	—	—	—	—	0%	—	—
Breast milk										
Massachusetts (n=45) (Tao et al. 2008)										
Milk										
Percent >LOQ	<1%	64%	<1%	<1%	<1%	<1%	51%	—	—	—
Minimum	<0.010	<0.0052	<0.00772	<0.00499	<0.00440	<0.0100	<0.0120	—	—	—
Maximum	0.0234	0.0184	0.0111	0.00884	0.00974	0.0198	63.8	—	—	—

Table 6-16. Percent Detection and Levels of Other Perfluoroalkyls in Children's Serum, Umbilical Cord Blood, and Breast Milk

	Detection and concentration (ng/mL [ppb])[a]									
Sample population	PFHpA	PFNA	PFDeA	PFUA	PFDoA	PFBuS	PFHxS	PFOSA	Me-PFOSA-AcOH	Et-PFOSA-AcOH
Sweden (n=12) (Karrman et al. 2007a)										
Milk										
Percent >LOD	—	17%	0%	0%	—	—	100%	67%	—	—
Minimum	—	<0.005	<0.008	<0.005	—	—	0.031	<0.007	—	—
Maximum	—	0.020	<0.008	<0.005	—	—	0.172	0.030	—	—
Maternal serum		—				—	—			—
Percent >LOD	—	100%	100%	100%	—	—	100%	75%	—	—
Minimum	—	0.43	0.27	0.20	—	—	1.8	<0.10	—	—
Maximum	—	2.5	1.8	1.5	—	—	11.8	0.49	—	—
China (n=19) (So et al. 2006b)										
Percent >LOD	37%	100%	100%	100%	—	11%	100%	—	—	—
Minimum	<0.005	0.01	0.0038	0.0091	—	<0.001	0.004	—	—	—
Maximum	0.0067	0.062	0.011	0.056	—	0.0025	0.10	—	—	—

[a]Percent detection for the adolescent age group was not specified for these samples. Percentages listed here are for the total sample population.
[b]Reported as bias-corrected estimates.

Et-PFOSA-AcOH = 2-(N-ethyl-perfluorooctane sulfonamide) acetic acid; LLOQ = lower limit of quantification; LOD = limit of detection; Me-PFOSA-AcOH = 2-(N-methyl-perfluorooctane sulfonamide) acetic acid; ND = no data; NR = not reported; PFBuS = perfluorobutane sulfonic acid; PFDeA = perfluorodecanoic acid; PFDoA = perfluorododecanoic acid; PFHpA = perfluoroheptanoic acid; PFHxS = perfluorohexane sulfonic acid; PFNA = perfluorononanoic acid; PFOSA = perfluorooctane sulfonamide; PFUA = perfluoroundecanoic acid

measured in children from these studies are similar to mean concentrations reported for adults (Calafat et al. 2007a, 2007b; Olsen et al. 2003a). For example, geometric mean concentrations of PFOA and PFOS measured during the NHANES surveys were 3.9–5.5 and 19.3–29.1 ng/mL, respectively, in adolescent serum and 3.9–5.2 and 20.7–30.4 ng/mL, respectively, in serum of the total population. Emmett (2006a) found that 2–5-year-old children had a higher serum PFOA (median 600 ng/mL) in the Little Hocking Water Association district compared with residents in all other age groups (median 321 ng/dL) except for the group aged >60 years, whose levels were similar to those in young children. Several factors may have contributed to the observed high levels in children: infants and young children proportionally drink more water per kg of body weight than adults; children (and also the elderly) tend to spend more time at home with exclusive use of residential water than other age groups; and trans-placental and breast milk exposures could also contribute to levels in children.

Kato et al. (2009b) reported serum levels in children aged 3–5 and 6–11 years from the 2001–2002 NHANES survey for PFNA, PFOA, PFOS, and PFOSA. Highest levels were typically observed for PFOS. The least square mean (LSM is equivalent to arithmetic mean) serum concentrations for PFOS ranged from 30.45 ng/mL for Mexican Americans to 42.45 ng/mL for non-Hispanic whites aged 6–11 years (Kato et al. 2009b). The LSM for PFOA ranged from 6.1 ng/mL for Mexican Americans to 7.6 ng/mL for non-Hispanic whites aged 6–11 years. Among 3–5 year olds, specific data from pooled samples were only presented for PFNA. The LSM serum PFNA serum concentrations for this age group were 0.9, 1.2, and 0.6 ng/mL for non-Hispanic whites, non-Hispanic blacks, and Mexican Americans, respectively (Kato et al. 2009b).

Blood serum levels of PFOA, PFOS, and PFHxS obtained in 2006–2007 from children residing in Australia were reported by Toms et al. (2009). The highest levels tended to occur for PFOS. Mean PFOS serum levels (combined male and female) ranged from 7.0 ng/mL for infants 0–0.5 years of age to 18.3 ng/mL for 6–9 year olds while mean PFOA serum levels ranged from 4.5 ng/mL for infants 0–0.5 years of age to 8.2 ng/mL for 6–9 year olds. Mean serum PFHxS levels ranged from 0.9 ng/mL for infants 0–0.5 years of age to 5.8 ng/mL for 6–9 year olds (Toms et al. 2009).

Although mean serum concentrations of perfluoroalkyl compounds are reported to be similar for older children (12–19 years of age) and adults, estimated 95th percentile values of PFHxS measured in childhood serum were noted to be higher than values estimated for adults. Olsen et al. (2003a) reported bias-corrected 95th percentile estimates of 65 ng/mL for PFHxS in the serum of children ages 2–12. This value is higher than bias-corrected 95th percentile estimates of 9.5 and 8.3 ng/mL based on PFHxS

measurements in the serum of 645 U.S. adult blood donors and 238 elderly individuals from the Seattle, Washington area, respectively (Olsen et al. 2003b, 2004b, 2004c). The difference is less extreme in the NHANES data, with PFHxS 95th percentile values of 12.9–13.1 ng/mL reported for children compared to values of 8.3–8.7 ng/mL reported for the total population. Olsen et al. (2004b) also noted statistically higher levels of Me-PFOSA-AcOH measured in children citing estimated 95th percentile values of 12.0, 5.0, and 3.8 ng/mL for serum concentrations of this substance measured in children, adult donors, and elderly individuals, respectively (Olsen et al. 2003b, 2004b, 2004c).

Reasons for the observed differences of PFHxS and Me-PFOSA-AcOH levels in childhood serum samples compared to adult samples have not been determined. Olsen et al. (2004b) states that different exposure and activity patters between children and adults should be considered. For example, children may have a higher exposure than adults to PFHxS, a substance that has been used in postmarket carpet cleaning applications, since they are lower to the ground and have increased contact with carpeted floors (Calafat et al. 2007a; Olsen et al. 2004b).

When estimating PFOS and PFOA uptake doses for children, Trudel et al. (2008) assumes the same exposure pathways for children as were proposed for adults, but considers exposure from hand-to-mouth transfer from treated carpets to be much larger in children. This pathway was estimated to contribute 40–60% of the total uptake of both PFOS and PFOA in infants (0–1 years), toddlers (1–4 years), and children (5–11 years) in the high exposure scenario. Exposure via human breast milk was included in the food consumption pathway for infants. Exposure via mouthing of clothes, carpet, and upholstery was also considered for children <12; however, this was considered to be a minor pathway of exposure. PFOS uptake doses estimated for the low, medium, and high exposure scenarios were 18.1–219 ng/kg body weight/day for infants, 14.8–201 ng/kg body weight/day for toddlers, and 9.7–101 ng/kg body weight/day for children. PFOA uptake doses estimated for the low, medium, and high exposure scenarios were 2.2–121 ng/kg body weight/day for infants, 1.2–128 ng/kg body weight/day for toddlers, and 0.8–65.2 ng/kg body weight/day for children. In contrast with the estimates for children under age 12, relative exposure pathways and uptake doses estimated for teenagers (12–20 years) were approximately the same as for adults.

Tao et al. (2008) measured perfluoroalkyl concentrations in 45 human breast milk samples collected from Massachusetts. PFOS, PFOA, PFHxS, and PFNA were each detected in 96, 89, 51, and 64% of the samples, respectively, with median concentrations of 106, 36.1, 12.1, and 6.97 pg/mL, respectively. PFHpA, PFDeA, PFUA, PFDoA, and PFBuS were each detected in <1% of the samples. Perfluoroalkyls

have also been measured in the human breast milk of individuals from Sweden, China, and Germany/ Hungary (Kärrman et al. 2007; So et al. 2006b; Völkel et al. 2008). PFOS was detected in all samples while detection of PFOA ranged from 8–100% in these studies. The reported maximum concentrations of PFOS and PFOA measured in human breast milk samples collected during these studies were 0.360–0.639 and 0.210–0.490 ng/mL, respectively (Kärrman et al. 2007; So et al. 2006b; Völkel et al. 2008). Other perfluoroalkyls detected in human breast milk included PFHpA, PFNA, PFDeA, PFUA, PFBuS, PFHxS, and PFOSA. Maximum concentrations of these compounds were reported to be <0.18 ng/mL.

The presence of perfluoroalkyl compounds in umbilical cord blood indicates that these substances can cross the placental barrier resulting in the exposure of babies *in utero* (Apelberg et al. 2007a, 2007b; Fei et al. 2007; Inoue et al. 2004b; Midasch et al. 2007). In most studies, PFOS and PFOA have been detected in 99–100% of umbilical cord blood samples with reported concentrations were 4.9–11.0 and 1.6–3.7 ng/mL, respectively (Apelberg et al. 2007a, 2007b; Fei et al. 2007; Inoue et al. 2004b; Midasch et al. 2007). Inoue et al. (2004b) did not detect PFOA in 15 cord blood samples from Japan; however, this compound was only detected in the maternal serum of three mothers. Apelberg et al. (2007a) also reported concentrations of other perfluoroalkyl compounds measured in 299 cord serum samples collected during the Baltimore THREE Study. Of these compounds, PFDeA, PFUA, PFOSA, and Me-PFOSA-AcOH were detected most frequently (24, 34, 26, and 40%, respectively). Maximum concentrations in these samples ranged from 1.1 to 1.8 ng/mL. PFHpA, PFDoA, PFBuS, and Et-PFOSA-AcOH were each detected in <6% of the samples with maximum concentrations ranging from 0.2 to 2.6 ng/mL.

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Individuals who work at or are located near fluorochemical facilities may have higher exposure to perfluoroalkyl compounds than the general population based on elevated concentrations of these substances measured in air, soil, sediment, surface water, groundwater, and vegetation surrounding these facilities (3M 2007b, 2008b, 2008c; Barton et al. 2006; Davis et al. 2007). PFOA, PFOS, PFBA, PFBuS, and PFHxS have been detected in the municipal drinking water of some communities located near fluorochemical facilities (3M 2008c; Agency for Toxic Substances and Disease Registry 2008; Emmett et al. 2006a; Holzer et al. 2008; Steenland et al. 2009; Wilhelm et al. 2009). Emmett et al. (2006a) compared PFOA serum levels to various types of exposure for individuals living in the Little Hocking community (near DuPont's Washington Works facility) and concluded that residential water source was the primary determinant of serum PFOA at this location. These authors reported that the mean human serum PFOA level was 105 times the level in residential drinking water. In residents with residential

drinking water but without occupational exposure, the model of best-fit for serum PFOA also varied significantly by age (highest in children ≤5 years old, and those over 60 years old), use of carbon home water filters (negative effect), number of servings of home-grown fruits and vegetables (positive effect), and number of tap water-based drinks per day (positive effect). Median PFOA serum levels for residents currently residing in six water districts located in the mid-Ohio Valley near the Washington Works facility ranged from 12.1 to 224.1 ng/mL, while the median concentration ranged from 10.5 to 33.7 ng/mL for residents who previously worked or resided in these districts (Steenland et al. 2009). PFOA serum levels tended to be highest for children aged 0–9 years and persons over 50 years old. These authors also reported that former employees at the chemical plant had much higher levels (median=75 ng/mL) than people who had not worked at the plant (median=24 ng/mL), but lower levels than those who continued to be employed at the plant during the monitoring period (median=148 ng/mL). The serum levels of the 69,030 residents participating in this study categorized by age are provided in Table 6-17. Additional blood serum levels of PFOA and PFOS for residents in selected areas of Ohio, West Virginia, New Jersey, and Minnesota whose residential source of drinking water may have been contaminated are available from the EPA docket on PFOA and related perfluoroalkyl substances (EPA-HQ-OPPT-2003-0012) (Bilott 2004, 2005a, 2005b, 2007).

Individuals involved in activities with prolonged use of perfluoroalkyl-containing products, such as the application of protective coatings for fabrics and carpet and the use of paper coatings, may have higher levels of exposure to perfluoroalkyl compounds than the general population (Calafat et al. 2006a).

3M estimated doses for various off-site exposure scenarios based on monitoring information collected at the Decatur Facility in Alabama (3M 2008c). Exposure scenarios include local children and adult residents exposed to PFOA in off-site soils, groundwater, municipal water, fish from the Tennessee River, and surface water and sediments in the Tennessee River. According to 3M, estimated off-site exposure of local residents to PFOA ranges from 0.011 to 260 ng/kg/day with the highest estimated exposure corresponding to children whose source of drinking water is groundwater adjacent to the southern side of the facility.

6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether

Table 6-17. Blood Serum Levels for 69,030 Current and Former Residents of Six Water Districts in the Mid-Ohio Valley (2005–2006)

Age (years)	Number (percentage of total)	Median PFOA level (ng/mL)
0–9	4,915 (7.1)	32.8
10–19	9,658 (14.0)	26.6
20–29	10,073 (14.6)	21.0
30–39	10,547 (15.3)	22.7
40–49	12,113 (17.6)	28.0
50–59	10,515 (15.2)	33.6
60–69	6,881 (10)	42.9
≥70	4,328 (6.3)	40.1

PFOA = perfluorooctanoic acid

Source: Steenland et al. 2009

adequate information on the health effects of perfluoroalkyls is available. Where adequate information is not available, ATSDR, in conjunction with NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of perfluoroalkyls.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

6.8.1 Identification of Data Needs

Physical and Chemical Properties. Perfluoroalkyl compounds have unique and complex physical and chemical properties (Kissa 2001; Schultz et al. 2003). Sources are available that provide helpful insights into the structural aspects and surfactant nature of these substances; however, many of the properties are still not well understood (CEMN 2008; Kissa 2001; Schultz et al. 2003). In general, specific properties such as physical state, melting point, boiling point, density, solubility, vapor pressure, micelle formation, and acid dissociation in water have not been determined or are not well described for these compounds. Measurements of these end points are needed. Information regarding the potential association of these species in water would be useful. Where determination of a particular end point is not possible, a thorough description of the physical and chemical properties as they relate to that end point would be helpful. Perfluoroalkyl substances discussed in this profile exist as a mixture of linear and branched isomers. Isomer-specific data would also be useful for the various physical-chemical properties.

Production, Import/Export, Use, Release, and Disposal. According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit substance release and off-site transfer information to the EPA. The TRI, which contains this information for 2006, became available in May of 2008. This database is updated yearly and should provide a list of industrial production facilities and emissions.

United States production volume ranges as of 2002 are available for PFOA, APFO (PFOA salt), PFBA, and PFOS (EPA 2008g). Production volume information for other perfluoroalkyls has not been located.

Perfluoroalkyl production is expected to be declining since companies have begun phasing out these substances (EPA 2008f). Continued reporting on perfluoroalkyl production is expected to provide evidence of this decline.

No information has been located regarding the import and export of perfluoroalkyl compounds. Uses of perfluoroalkyls are well described in the literature; no further information is needed (3M 1999; DuPont 2008; EPA 2008f; Hekster et al. 2003; Schultz et al. 2003). Recommended methods for the disposal of perfluoroalkyl compounds have not been located. In the past, perfluoroalkyl-containing waste has been disposed of in on- and off-site landfills, through sludge incorporation, and through incineration (3M 2007b, 2008b; Agency for Toxic Substances and Disease Registry 2005). New disposal methods that avoid release of these substances into the open environment and prevent contamination of nearby soil, sediment, and groundwater should be developed.

Environmental Fate. Perfluoroalkyls are very stable compounds and are resistant to biodegradation, direct photolysis, atmospheric photooxidation, and hydrolysis (3M 2000; EPA 2008f; OECD 2002, 2007; Schultz et al. 2003). The chemical stability of perfluoroalkyls and the low volatility of these substances in ionic form indicate that perfluoroalkyls will be persistent in water and soil (3M 2000; Prevedouros et al. 2006). K_{oc} values ranging from 17 to 230 indicate that PFOA will be mobile in soil and can leach into groundwater (Davis et al. 2007; Prevedouros et al. 2006). Environmental fate and potential pathways of PFOA exposure at and near the DuPont Washington Works site have been discussed (Small et al. 2009).

Bioavailability from Environmental Media. Perfluoroalkyls are widely detected in humans and animals indicating that these substances are bioavailable. The bioaccumulation potential of perfluoroalkyls is reported to increase with increasing chain length (de Vos et al. 2008; Furdui et al. 2007; Martin et al. 2004b). In living organisms, perfluoroalkyls bind to protein albumin in blood, liver, and eggs and do not accumulate in fat tissue (de Vos et al. 2008; Kissa 2001). The mechanism of perfluoroalkyl uptake in animals is not fully understood; additional study would be helpful (de Vos et al. 2008). Perfluoroalkyl substances discussed in this profile exist as a mixture of linear and branched isomers. Data regarding the bioavailability of branched versus linear substances would be useful.

Food Chain Bioaccumulation. High levels of certain perfluoroalkyls in animals have been measured in apex predators, such as polar bears, which indicates that some perfluoroalkyls possess the ability to bioaccumulate (de Vos et al. 2008; Houde et al. 2006a; Kannan et al. 2005; Smithwick et al. 2005a, 2005b, 2006). Perfluoroalkyl sulfonates with carbon chain length lower than 8 tend to

bioaccumulate less than PFOS. Ongoing monitoring of perfluoroalkyl levels in animals may help to determine whether efforts to phase out these substances will have had an effect on their biomagnification.

Exposure Levels in Environmental Media. Reliable monitoring data for the levels of perfluoroalkyls in contaminated media at hazardous waste sites are needed so that the information obtained on levels of perfluoroalkyls in the environment can be used in combination with the known body burden of perfluoroalkyls to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Concentrations of perfluoroalkyls have been measured in surface water from several locations across the United States (Boulanger et al. 2004; Kannan et al. 2005; Kim and Kannan 2007; Nakayama et al. 2007; Simcik and Dorweiler 2005; Sinclair et al. 2004, 2006). Continued monitoring for perfluoroalkyls in surface water would be useful. Data are available regarding levels of perfluoroalkyls in outdoor air, indoor air, indoor dust, food, food packaging, and consumer products (3M 2001; Barber et al. 2007; Begley et al. 2005; Food Standards Agency 2006; Fromme et al. 2007b; Harada et al. 2005b, 2006; Jogsten et al. 2009; Kim and Kannan 2007; Kubwabo et al. 2005; Moriwaki et al. 2003; Tittlemier et al. 2007; Washburn et al. 2005). Comprehensive studies monitoring for perfluoroalkyls in these matrices within the United States are needed. Background concentrations of perfluoroalkyls in groundwater, drinking water, soil, and sediment have not been located and therefore are a data need. Elevated concentrations of perfluoroalkyls have been measured in air, water, soil, and sediment near fluorochemical industrial facilities (3M 2007b, 2008b, 2008c; Barton et al. 2006; Davis et al. 2007; Hansen et al. 2002). Continued monitoring for perfluoroalkyls in these matrices are needed to assess exposure of individuals working at these locations and individuals who live near these facilities.

Exposure Levels in Humans. Trudel et al. (2008) provided a thorough assessment of the exposure of the general population to PFOS and PFOA. 3M (2008b) provided an assessment of exposure of individuals to PFOA on-site at a fluoropolymer facility. Uptake values and exposure pathways determined in these studies should be examined further. Conclusions made in these assessments are expected to be adjusted as future monitoring data are made available. Large-scale monitoring of perfluoroalkyls in human serum in the United States is ongoing (Calafat et al. 2006a). Future results of human monitoring studies would be useful for assessing human exposure to these substances over time. The results of these studies can be examined for correlations between human perfluoroalkyl levels and the phasing out of perfluoroalkyl compounds by companies of the fluorochemical industry. Concentrations of perfluoroalkyls measured in urine have not been located. Higher exposure levels for individuals who

reside in areas where substances such as PFOA contaminated both public and private water supplies have been documented (Emmett et al. 2006a, 2009).

This information is necessary for assessing the need to conduct health studies on these populations.

Exposures of Children. Trudel et al. (2008) provided a thorough assessment of the exposure of children to PFOS and PFOA. These conclusions should be reexamined with respect to future biomonitoring data when they become available. Data are available regarding the levels of perfluoroalkyls in young children (Kato et al. 2009b; Olsen et al. 2004b; Toms et al. 2009). The recent NHANES surveys did not include perfluoroalkyl serum levels for children below 12 years of age (Calafat et al. 2007a, 2007b). Future NHANES efforts are scheduled to include children of ages 3–11 years in the sample population (Calafat et al. 2007a). Data provided from these efforts will be useful in assessing the exposure of young children to perfluoroalkyls.

Concentrations of perfluoroalkyls have been measured in human breast milk and cord blood (Apelberg et al. 2007a, 2007b; Fei et al. 2007; Inoue et al. 2004b; Kärrman et al. 2007; Midasch et al. 2007; So et al. 2006b; Völkel et al. 2008). Additional monitoring for perfluoroalkyls in these media would be useful.

Child health data needs relating to susceptibility are discussed in Section 3.12.2, Identification of Data Needs: Children's Susceptibility.

Exposure Registries. No exposure registries for perfluoroalkyls were located. This substance is not currently one of the compounds for which a sub-registry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for sub-registries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance.

6.8.2 Ongoing Studies

The NIH RePORTER (2014) database provides additional information obtainable from a few ongoing studies that may fill in some of the data needs identified in Section 6.8.1. These studies are summarized in Table 6-18.

As part of the EPA PFOA Stewardship Program, member companies have agreed to reduce facility emissions and product content of PFOA and related chemicals on a global basis by 95% no later than 2010, and to work toward elimination of these substances by 2015. These companies have also agreed to provide progress reports to EPA on a regular basis. DuPont and 3M are currently working with EPA to develop thorough assessments of perfluoroalkyl environmental contamination and human exposure to these substances surrounding major fluorochemical facilities such as the Decatur, Alabama facility and the Washington Works facility (3M 2008a, 2008b, 2008c, 2008d; DuPont 2008; EPA 2008f).

As part of the Third National Health and Nutrition Evaluation Survey (NHANES III), the Environmental Health Laboratory Sciences Division of the National Center for Environmental Health, Centers for Disease Control and Prevention, will be analyzing human blood samples for perfluoroalkyls. These data will give an indication of the frequency of occurrence and background levels of these compounds in the general population.

Table 6-18. Ongoing Studies on Perfluoroalkyls

Investigator	Affiliation	Research description	Sponsor
Shankar, A	West Virginia University	Perfluoroalkyl chemicals (PFC) are detectable in the blood of >98% of U.S. adults. This project will study the association between blood PFCs and kidney disease and cardiovascular disease.	National Institute of Environmental Health Sciences (NIEHS)
Frisbee, SJ	West Virginia University	The primary objective of this proposal is to determine the associations between non-8-carbon perfluoroalkyl acids (PFAAs) and serum parameters of lipid, liver, and kidney function in children. The proposed study will perform secondary analysis on data collected as part of the C8 Health Project, a community cohort study with 69,030 participants, including more than 12,000 children.	National Institute of Environmental Health Sciences (NIEHS)
Sagiv, SK	Boston University Medical Campus	This study ill measure levels of four perfluorinated compounds—perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorohexane sulfonate (PFHxS), and perfluorononanoate (PFNA) —in prenatal maternal plasma collected in early gestation and estimate associations with fetal and infant somatic growth, childhood adiposity, and metabolic outcomes such as serum cholesterol and insulin resistance, and neurodevelopment, including cognition and behavior.	National Institute of Environmental Health Sciences (NIEHS)

Source: NIH RePORTER 2014