Long-Chain Perfluorinated Chemicals (PFCs) Action Plan

I. Overview

Long-chain perfluorinated chemicals (PFCs)[1] are found world-wide in the environment, wildlife, and humans. They are bioaccumulative in wildlife and humans, and are persistent in the environment. To date, significant adverse effects have not been found in the general human population; however, significant adverse effects have been identified in laboratory animals and wildlife. Given the long half-life of these chemicals in humans (years), it can reasonably be anticipated that continued exposure could increase body burdens to levels that would result in adverse outcomes.

Since 2000, the Agency has taken various actions to help minimize the potential impact of PFCs on human health and the environment, including the publication of three Significant New Use Rules on perfluoroalkyl sulfonate (PFAS) chemicals and the review of substitutes for long-chain PFCs as part of its review process for new chemicals under EPA's New Chemicals Program. Although such actions are important steps to reducing exposure to these chemicals, EPA continues to be concerned with long-chain PFCs. Consequently, EPA intends to propose actions in 2012 under the Toxic Substances Control Act (TSCA) to address the potential risks from long-chain PFCs.

EPA intends to consider initiating TSCA section 6 rulemaking for managing long-chain PFCs. If EPA can make certain findings with respect to these chemicals (further analysis of the information will be performed as part of TSCA section 6 rulemaking), TSCA section 6 provides authority for EPA to ban or restrict the manufacture (including import), processing, and use of these chemicals. A rule addressing the PFAS sub-category could expand beyond the reach of the SNURs that the Agency has promulgated over the past decade. For example, the rule could address PFAS-containing articles. A rule addressing the perfluoroalkyl carboxylate (PFAC) sub-category could expand the reach of the 2010/15 PFOA Stewardship Program beyond the eight participating companies and further address the concerns for potential PFAC exposure through the use of PFAC-containing articles. EPA will develop more detailed assessments to support the TSCA section 6(a) "presents or will present an unreasonable risk" findings. If these more detailed assessments indicate that a different approach to risk management is appropriate, EPA will consider additional approaches.

Long-chain PFCs are a concern for children's health. Studies in laboratory animals have demonstrated developmental toxicity, including neonatal mortality. Children's exposures are greater than adults due to increased intakes of food, water, and air per pound of body weight, as well as child-specific exposure pathways such as breast milk consumption, mouthing and ingestion of non-food items, and increased contact with the floor. Biomonitoring studies have found PFCs in cord blood and breast milk, and have reported that children have higher levels of

[1] The terms long-chain PFCs, long-chain perfluoroalkyl sulfonate (PFAS), and long-chain perfluoroalkyl carboxylate (PFAC) chemicals in this document refer only to chemicals described in the chemical identity section, including certain polymers that contain perfluorinated moieties. They do not include other PFCs, particularly those having shorter chain lengths.

some PFCs compared to adults. Thus, given the pervasive exposure to PFCs, the persistence of PFCs in the environment, and studies finding deleterious health effects, EPA will examine the potential risks to fetuses and children.

II. Introduction

As part of EPA's efforts to enhance the existing chemicals program under the Toxic Substances Control Act (TSCA)[2], the Agency identified an initial list of widely recognized chemicals, including PFCs, for action plan development based on their presence in human blood; persistent, bioaccumulative, and toxic (PBT)[3] characteristics; use in consumer products; production volume; and other similar factors. This Action Plan is based on EPA's initial review of readily available use, exposure, and hazard information[4] on PFCs. EPA considered which of the various authorities provided under TSCA and other statutes might be appropriate to address potential concerns with PFCs in developing the Action Plan. The Action Plan is intended to describe the courses of action the Agency plans to pursue in the near term to address its concerns. The Action Plan does not constitute a final Agency determination or other final Agency action. Regulatory proceedings indicated by the Action Plan will include appropriate opportunities for public and stakeholder input, including through notice and comment rulemaking processes.

III. Scope of Review

Continuing contributions of PFAS/PFAC to the environmental/human reservoir are best addressed using a category approach.

The PFAS/PFAC precursors may be polymers that are coated on a specific substrate. This action is considering only the contribution of precursors as a source of PFAS/PFAC, and not the inherent toxic effects of the polymer or exposure to dust that contains fluorinated polymers.

Long-Chain Perfluoroalkyl Sulfonate (PFAS) Sub-Category

The PFAS sub-category includes perfluorohexane sulfonic acid (PFHxS)[5], perfluorooctane sulfonic acid (PFOS)[6], and other higher homologues. The category also includes the acid salts and precursors.

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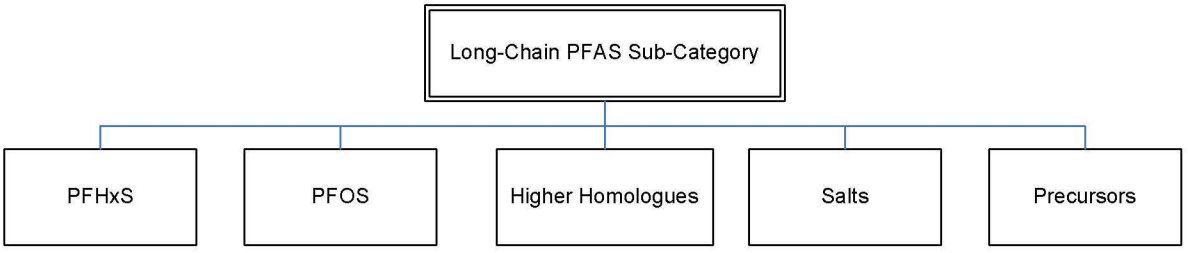
[2] 15 U.S.C. §2601 *et seq.*

[3] Information on PBT chemicals can be found on the EPA website at http://www.epa.gov/pbt/.

[4] Information sources customarily employed include Inventory Update Reporting (IUR) submissions; Toxic Release Inventory (TRI) reporting; data submitted to the HPV Challenge Program; existing hazard and risk assessments performed by domestic and international authorities including but not limited to U.S. Federal government agencies, the Organization for Economic Cooperation and Development, the Stockholm Convention on Persistent Organic Pollutants, Health and Environment Canada, the European Union; and others. Action plans will reference specific sources used.

[5] CF_3-$(CF_2)_5$-SO_3H; CAS RN: [355-46-4].

[6] CF_3-$(CF_2)_7$-SO_3H; CAS RN: [1763-23-1].



The similarities of the chemicals within the PFAS sub-category can be established when reviewing representative structures of the different category member compounds:

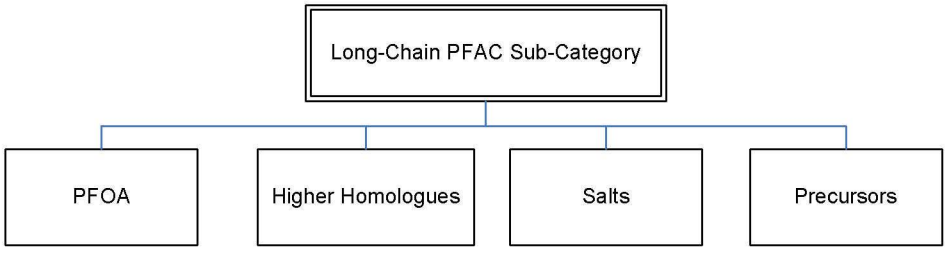
a. $CF_3(CF_2)_n$-SO_3^-M where M = H^+ or any other group where a formal dissociation can be made; and

b. $CF_3(CF_2)_n$-$S(=O)_y$-X where y = 0 – 2 and X is any chemical moiety.

where n > 4.

Long-Chain Perfluoroalkyl Carboxylate (PFAC) Sub-Category

The PFAC sub-category includes perfluorooctanoic acid (PFOA) [7] and other higher homologues. The category also includes the acid salts and precursors.



These similarities within the PFAC sub-category can be established by reviewing representative structures of the different category member compounds:

a. $CF_3(CF_2)_n$-COO^-M where M = H^+ or any other group where a formal dissociation can be made;

b. $CF_3(CF_2)_n$-$CH=CH_2$;

c. $CF_3(CF_2)_n$-C(=O)-X where X is any chemical moiety;

d. $CF_3(CF_2)_m$-CH_2-X where X is any chemical moiety; and

e. $CF_3(CF_2)_m$-Y-X where Y = non-S, non-N hetero atom and where X is any chemical moiety.

[7] CF_3-$(CF_2)_6$-COOH; CAS RN: [335-67-1].

where n > 5 or m > 6.

IV. Uses and Substitutes Summary

Production Volume

PFAS Chemicals

Commercial production of PFAS chemicals began over half a century ago. Total production from 1970 to 2002 was estimated to be about 100,000 tons (Paul A.G., 2009). By 2003, PFOS chemicals were no longer manufactured by 3M, the principal U.S. producer. However, production of PFOS-related chemicals is still ongoing in other countries, though to a much smaller extent than before 2003 (POPRC, 2007). As PFOS-based products became more strictly regulated in developed countries, production shifted to other countries. For example, manufacturers in China began large scale production in 2003 at the advent of 3M's 2002 global PFOS phase-out. China had an annual production in 2004 of less than 50 tons, but has increased production dramatically in recent years, with an estimated production of more than 200 tons in 2006. Approximately 100 tons of that amount is designated for export (POPs, 2008).

PFAC Chemicals

World-wide production of fluorotelomers was estimated at 20 million pounds in 2006. The United States accounts for more than 50 percent of world-wide fluorotelomer production. Textiles and apparel account for approximately 50 percent of the volume, with carpet and carpet care products accounting for the next largest share in consumer product uses. Coatings, including those for paper products, are the third largest category of consumer product uses.

Fluorotelomer release sources, and consequent exposure to fluorotelomers, can be explained through the examination of the life cycle of this category of chemicals:

Manufacture of Monomers ➔ Manufacture of Polymers ➔ Processing and Use ➔ Product Life

The manufacture of non-polymeric chemicals (surfactants, wetting agents, cleansers, etc.) is included in the manufacture of monomers. Some residual monomers are present in the various raw materials and final products of the different steps of manufacturing. Because each intermediate contains the same R_f moiety, the polymers also contain this moiety. The 2010/15 PFOA Stewardship Program encourages the elimination of PFAC precursors in product content. Companies reporting under PFOA Stewardship Program differentiate between the amounts of PFAC precursors present in the final polymer product as residuals and the amount present in the polymer as R_f moities. The availability of PFAC precursor from the content of residuals in fluorotelomer based polymer products (FTBP) would be small in comparison to the amount released should polymeric materials biodegrade in the environment. Potentially all monomeric, not just the small amounts of residual monomers and other monomer raw material and intermediates released at each of the four steps in the sequence above, could be PFAC precursors.

Uses

PFCs are substances with special properties that have thousands of important manufacturing and industrial applications. They impart valuable properties, including fire resistance and oil, stain, grease, and water repellency. For example, they are used to provide non-stick surfaces on cookware and waterproof, breathable membranes for clothing, and are used in many industry segments, including the aerospace, automotive, building/construction, chemical processing, electronics, semiconductors, and textile industries.

PFAS Chemicals

PFAS are synthetic chemicals that do not occur naturally in the environment. Long-chain PFAS chemicals, as defined in this action plan, are no longer manufactured in United States. However, there is a limited set of existing uses for which alternatives are not yet available, and which are characterized by low volume, low exposure potential, and low releases.

The existing SNUR regulations on PFAS chemicals do not affect the continued use of existing stocks of the listed chemicals that had been manufactured or imported into the United States prior to the effective date of the SNURs. Existing products and formulations already in the United States containing these chemicals – for example, PFOS-based fire fighting foams produced before the rules took effect in 2002 – can also still be used without providing notice to the Agency. Because the PFAS SNURs exempt articles, PFOS may be imported or processed as part of an article without the Agency receiving prior notice.

PFAC Chemicals

PFAC are synthetic chemicals that do not occur naturally in the environment. PFOA is manufactured for use primarily as an aqueous dispersion agent [as the ammonium salt] in the manufacture of fluoropolymers, which are substances with special properties that have thousands of important manufacturing and industrial applications.

PFOA also be produced unintentionally by the degradation of some fluorotelomers, which are not manufactured using PFOA but could degrade to PFOA. Fluorotelomers are used to make polymers that impart soil, stain, grease, and water resistance to coated articles. Some fluorotelomer based products are also used as high performance surfactants in products where an even flow is essential, such as paints, coatings, cleaning products, and fire-fighting foams for use on liquid fuel fires. Fluorotelomer-based products can be applied to articles both at the factory and by consumers and commercial applicators in after-market uses such as carpet treatments and water repellent sprays for apparel and footwear.

Fluoropolymers, such as polytetrafluoroethylene (PTFE), which may contain some PFAC contamination, or that use PFOA as an emulsion stabilizer in aqueous dispersions, have a large U.S. market. The wire and cable industry is one of the largest segments of the fluoropolymer market, accounting for more than 35 percent of total U.S. fluoropolymer use. Apparel makes up about 10 percent of total fluoropolymer use, based on total reported production volume. Fluoropolymers are used in a wide variety of mechanical and industrial components, such as

plastic gears, gaskets and sealants, pipes and tubing, O-rings, and many other products. Total U.S. demand for fluoropolymers in 2004 was between 50,000 and 100,000 metric tons. The United States accounted for less than 25 percent of the world consumption of PTFE in 2007, and between 25 and 50 percent of the world consumption of other fluoropolymers. PTFE is the most commonly used fluoropolymer, and the United States consumed less than 50,000 metric tons of PTFE in 2008.

Substitutes

EPA is reviewing substitutes for PFOS, PFOA, and other long-chain PFCs under the New Chemicals Program. EPA established the program under section 5 of TSCA to help manage the potential risk from chemicals new to the marketplace.

EPA's review of alternatives to long-chain PFCs has been ongoing since 2000 and is consistent with the approaches to alternatives encouraged under the PFOA Stewardship Program. Through 2009, EPA has received and reviewed over 100 perfluorinated alternatives of various types. EPA reviews the new substances against the range of toxicity, fate, and bioaccumulation issues that have caused past concerns with perfluorinated substances, as well as any issues that may be raised by new chemistries (EPA, 2009b).

V. Hazard Identification Summary

The information used by EPA for this Action Plan includes the Organisation for Economic Co-operation and Development's (OECD) assessments of PFOS (OECD, 2002) and PFOA (OECD, 2006), EPA's Office of Pollution Prevention and Toxics' (OPPT) draft risk assessment of PFOA (EPA, 2009d), Environment Canada's assessment (Canada, 2006), the assessment of PFOS by the Stockholm Convention on Persistent Organic Pollutants (POPs, 2009), and other sources. The summary of the toxicity information is based on these previous assessments, and where appropriate, additional information on short- and long-chain lengths is provided.

World-Wide Distribution of PFAS and PFAC

Presence in Humans

PFAS and PFAC have been detected in human blood samples throughout the world. Blood samples have been collected in countries world-wide including the United States, Japan, Canada, Peru, Colombia, Brazil, Italy, Poland, Germany, Belgium, Sweden, India, Malaysia, Korea, China, and Australia. In addition, PFAS and PFAC have been detected in breast milk, liver, umbilical cord blood, and seminal plasma. In most cases, the analytes most often detected in human matrices, and usually in the highest concentrations, were PFOS, PFOA, and PFHxS. Other PFAS and PFAC detected in human tissue include perfluorooctane sulfonamide (PFOSA), 2-(N-methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH), 2-(N-ethylperfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH or PFOSAA), perfluoroheptanoic acid (PFHpA), perfluorononanoate (PFNA), perfluorodecanoic acid (PFDeA or PFDA), perfluoroundecanoic acid (PFUA), perfluorododecanoic acid (PFDoA),

perfluoropentanoic acid (PFPeA), perfluorohexanoic acid (PFHxA), and perfluorobutane sulfonate (PFBS).

National Health and Nutrition Examination Survey (NHANES) data show that mean levels of PFOS, PFOA and PFHxS in the general U.S. population older than 12 years declined between the sampling period of 1999-2000 and 2003-2004 (Calafat, 2007). In addition, 3M reported a decline of the same chemicals from 2000 to 2006 in a group of 600 adult American Red Cross (ARC) blood donors (G. W. Olsen, Mari DC, Church TR, Ellefson ME, Reagen WK, Boyd TM, Herron RM, Medhdizadehkashi Z, Nobiletti JB, Rios JA, Butenhoff JL, Zobel LR 2008). The biggest drop reported in both surveys was in PFOS (~30% in NHANES and ~60% in the ARC study). Both reported ~25% decline in PFOA. NHANES reported a 10% decrease in PFHxS while the ARC study reported a 30% drop. Conversely, PFNA increased by approximately 50% over 4 years in NHANES and by 100% over 6 years in the ARC study. 3M also reported a 100% increase in PFDeA, while the increase in NHANES was 60%. 3M reported an 80% increase in PFUA.

It appears that most of PFAS and PFAC do not vary much across adolescents participating in NHANES; however, pooled data from 2001-2002 indicate that most of the levels of perfluorinated compounds are higher in children ages 3-11 years compared to adults (individual samples 2001-2002), especially for PFHxS (Kato, 2009). More recent data on children are not available.

It is clear that there are individuals who have been exposed to perfluorinated compounds at levels much higher than the majority of the population. Recent data indicate that individuals living near a U.S. facility that uses PFOA may have much higher PFOA serum concentrations than those currently reported for the general population (Calafat, 2007; Emmett, 2006).

Presence in the Environment and Wildlife

Water

Log K_{ow} values for PFOA, PFOS and other commercially available ammonium salts range from -0.52 to > 6.8 (De Silva, 2008; Tomlin, 2005) and have water solubilities that range from 0.10 to > 500,000 (Hekster, 2003; Kissa, 2001). Long-chain PFAC have been measured in surface waters of remote areas such as the north shore of Lake Superior, the Hudson Bay region of Northeastern Canada, tributaries of the Pearl River in Guangzhou, China and the Yangtze River. Ice surface samples in the Canadian Arctic (Northwest Territories and Nunavut) had levels of that ranged from 5-246 pg/L for C9-C11 compounds.

Multiple studies have reported a global distribution of PFAC and PFAS that have been reported in wildlife tissue and blood samples. PFAS have also been found in a variety of aquatic organisms. Most recently, four perfluorinated analytes (PFOS and PFAS: C10, C11, and C12) were found in fillets from bluegill in selected rivers in Minnesota and North Carolina (Delinsky, 2009). In general, the highest concentrations in wildlife have been found in the livers of fish-eating animals close to industrialized areas.

Soil and Sediment

PFOA and PFOS are considered to be resistant to degradation in soil. Levels of C9-C11 PFAC have been found in remote Arctic region sediment ranging from 0.68 µg/kg – 2.58 µg/kg. PFAC are known to increase over time in sediment as observed in a 22-year study (1980-2002) of the Niagara River discharge. Sediment dwelling invertebrates such as amphipods, zebra mussels, and crayfish have also been found to have PFOA concentrations ranging from 2.5 – 90 ng/g ww in the Raisin, St. Clair, and Calumet Rivers (MI)(Kannan, 2005). At the 3M Decatur, AL site, PFOA concentrations in Asiatic clams ranged from 0.51 ng/g to 1.01 ng/g. Mussels and oysters in Tokyo Bay were found to contain PFOA concentrations 0.660 ng/g ww and worms from the Ariake Sea in western Japan had concentrations of PFOA of 82 ng/g ww.

PFAS and PFAC are Persistent, Bioaccumulative, and Toxic

Persistence and Bioaccumulation in Humans and Laboratory Animals

Animal studies of the straight-chain PFAS and PFAC have shown that these compounds are well absorbed orally, but poorly eliminated; they are not metabolized, and they undergo extensive uptake from enterohepatic circulation. Studies of PFOS and PFOA have shown that these compounds are distributed mainly to the serum, kidney, and liver, with liver concentrations being several times higher than serum concentrations; the distribution is mainly extracellular. Both compounds have a high affinity for binding to B-lipoproteins, albumin, and liver fatty acid-binding protein. Studies have reported PFOS, PFOA, and several other PFAS and PFAC in umbilical cord blood indicating these chemicals cross the placenta.

The elimination half-lives of several PFAS and PFAC are summarized in Table 1. In general, the rate of elimination decreases with increasing chain length, although the half-life of PFHxS (C6) is longer than the half-life of PFOS (C8) in humans. There is a tremendous species difference in elimination, and elimination is greatly reduced in humans. Thus, the half-life of PFOS is 7 days in rats, 150 days in monkeys, and 5.4 years in humans. There is a gender difference in the elimination of PFOA and other PFAC in laboratory animals. Studies of PFOA in rats have shown that the gender difference is developmentally regulated, and the adult pattern is achieved by sexual maturation. The reason for the species and gender differences in elimination are not well understood. These differences are hormonally controlled, and may also be due to the actions of organic anion transporters. A gender difference has not been found in humans, although uncertainty exists due to the small sample size.

Table 1. Comparative Rates of Elimination*

Serum Half-life	PFHxS (C6)	PFOS (C8)	PFOA (C8)	PFNA (C9)	PFDA (C10)
Rat		7 days	2-4 hours 6-7 days	2 days 31 days	59 days 40 days
Mouse			16 days 22 days	41 days 64 days	
Monkey	87 days 141 days	150 days	30 days 21 days		

Human	8.5 years	5.4 years	2.3-3.8 years		

***Red – females; blue - males**

Regardless of chain length, it is critical to note that the half-lives of these compounds are measured in hours to days to months in rats, mice and monkeys, but years in humans. This means that these compounds will persist and bioaccumulate in humans, and comparatively low exposures can result in large body burdens. The gender and species differences in elimination also indicate that comparisons of toxicological effects must utilize some measure of body burden rather than administered dose.

Persistence and Bioaccumulation in the Environment

PFOS and longer chain PFAC (> C8) bioaccumulate and persist in protein-rich compartments of fish, birds, and marine mammals such as carcass, blood, and liver (Conder, 2008). Studies have found fish bioconcentration factor (BCF) values for C8 to C14 PFAC ranging from 4 – 40,000 in rainbow trout (Martin, 2003). Fish BCF values for C8-C11 PFAS are relatively lower (4-4900). There are two BCF study results for long chain PFAC with BCF values from 4,7000 to 4,800 for perfluorohexadecanic acid (C16) in carp and BCF values from 320 to 430 for perfluorooctadecanoic acid (C18) in carp (Martin, 2003). Available evidence shows the likely potential for bioaccumulation or biomagnifications in marine or terrestrial species. This is due to conformational changes into a helical structure in the molecule resulting in a smaller cross-sectional diameter as chain length increases which can lead to the ability to accumulate in organisms (NITE, 2002a, 2002b). Additional evidence that C14 and C15 PFAC bioaccumulate and are bioavailable is their presence in fish, invertebrates, and polar bears. The bioaccumulation of PFOS and PFAC (C8 through C14) in air-breathing animals (e.g., birds and mammals) is thought to represent biomagnification due to high gastrointestinal uptake and slow respiratory elimination (B. Kelly, MG Ikonomou, JD Blair, B Surridge, F Hoover, R Grace, APC Gobas 2009; B. C. Kelly, Ikonomou MG, Blair JD, Morin AE, Gobas APC, 2007). In addition, Conder et al. state that the bioaccumulation and bioconcentration potential of PFAC are directly related to the length of the perfluorinated chain, and PFAS are more bioaccumulative than PFAC of the same chain length (Conder, 2008).

Within the PFAC and PFAS categories, the perfluorinated carboxylic and sulfonic acids (R_f from C5 to C20) are persistent chemicals that are resistant to degradation under environmental conditions. Even the reaction of PFAS/PFAC precursors with hydroxyl radicals in the atmosphere are considered to be so slow that long range transport is considered a viable exposure pathway (Hurley, 2004; G. W. Olsen, DC Mari, WK Reagen, ME Ellefson, DJ Ehresman, JL Butenhoff, LR Zobel, 2007).

Toxicity in Humans

Until recently, epidemiological and medical surveillance studies have been conducted primarily in the United States on workers occupationally exposed to POSF-based fluorochemicals. These studies specifically examined PFOS or PFOA exposures and possible adverse outcomes. One occupational study of exposures to a PFNA surfactant blend was

undertaken. The studies on PFOS and PFOA include mortality and cancer incidence studies, a study examining potential endocrine effects, an ''episodes-of-care'' study evaluating worker insurance claims data, and worker surveillance studies examining associations between primarily PFOS and/or PFOA serum concentrations and hematology, hormonal and clinical chemistry parameters. The PFNA study examined liver enzymes and blood lipid levels. In general, no consistent association between serum fluorochemical levels and adverse health effects has been observed.

Toxicity in Laboratory Animals

PFOA

The toxicity of PFOA has been extensively studied. Repeated-dose studies in rats have shown reduced body weight, hepatotoxicity, reduced cholesterol, and a steep dose-response curve for mortality. Due to gender differences in elimination, adult male rats exhibit effects at lower administered doses than adult female rats. Thus, dietary exposure for 90 days resulted in significant increases in liver weight and hepatocellular hypertrophy in female rats at 1000 ppm (76.5 mg/kg-day) and in male rats at doses as low as 100 ppm (5 mg/kg-day). Studies in nonhuman primates have shown similar effects at doses as low as 3 mg/kg-day, although the reduction in cholesterol has not been observed.

The carcinogenic potential of PFOA has been investigated in two dietary carcinogenicity studies in Sprague-Dawley rats, and has been shown to induce hepatocellular adenomas, Leydig cell tumors, and pancreatic acinar tumors. It has not been shown to be mutagenic in a variety of assays. There is sufficient evidence to indicate that PFOA is a PPARα-agonist and that the liver carcinogenicity (and toxicity) of PFOA is mediated by PPARα in the liver in rats. There is no evidence that the liver toxicity in nonhuman primates is due to PPARα-agonism. There is controversy over the relevance of this particular mode of action for humans. The mode of action for the Leydig cell tumors and pancreatic acinar tumors has not been established, and therefore these are assumed to be relevant for humans.

Several studies have shown that PFOA is immunotoxic in mice. PFOA causes thymic and splenic atrophy, and has been shown to be immunosuppressive in both *in vivo* and *ex vivo* systems. Studies using transgenic mice showed that the PPARα was involved in causing the adverse effects to the immune system.

Standard prenatal developmental toxicity studies in rats and rabbits in which pregnant animals are exposed only during gestation and sacrificed prior to the birth of the pups have not shown many effects. Thus, there was no evidence of developmental toxicity after exposure to doses as high as 150 mg/kg-day in an oral prenatal developmental toxicity study in rats. In a rat inhalation prenatal developmental toxicity study, the NOAEL and LOAEL for developmental toxicity were 10 and 25 mg/m3, respectively. In a rabbit oral prenatal developmental toxicity study there was a significant increase in skeletal variations after exposure to 5 mg/kg-day, and the NOAEL was 1.5 mg/kg-day.

However, the potential developmental toxicity of PFOA is evident when the pups are evaluated during the postnatal period. Thus, a two-generation reproductive toxicity study in rats

showed a reduction in F1 pup mean body weight during lactation at 30 mg/kg-day group and during the post-weaning period at 10 mg/kg-day. In addition, there was a significant increase in mortality mainly during the first few days after weaning, and a significant delay in the timing of sexual maturation for F1 male and female pups at 30 mg/kg-day.

Due to the rapid elimination of PFOA in female rats, many researchers have examined the developmental toxicity of PFOA in mice. These studies have shown a pattern of developmental effects similar to those observed with PFOS. Full liter resorptions were noted at 40 mg/kg-day and the percent of live fetuses and fetal body weight were reduced at 20 mg/kg-day. The most notable effect of prenatal exposure to PFOA was the severe compromise of postnatal survival at doses as low as 5 mg/kg-day, and the postnatal growth impairment and developmental delays noted among the survivors; the BMD_5 and $BMDL_5$ for neonatal survival were estimated at 2.84 and 1.09 mg/kg-day, respectively. Additional studies in mice have shown that PFOA exposure causes a significant reduction in mammary gland differentiation in the dams and stunted mammary gland development in the female pups.

Several studies have examined the mode of action for the developmental effects. These have shown that exposure to a dose of 20 mg/kg-day for 2 days late in gestation is sufficient to cause the neonatal mortality in mice. Studies with PPARα knockout mice have shown that the PPARα is required for the neonatal mortality and expression of one copy of this gene is sufficient. This is in contrast to the studies showing that PPARα is not involved in the neonatal mortality associated with PFOS exposure. Although there is controversy over the human relevance of the PPARα-agonist hepatotoxicity observed in rodents, the role of PPARα in development and particularly in the PFOA-induced neonatal mortality observed in mice is unknown; therefore this mode of action is assumed to be relevant for humans.

Other PFAC Chemicals

Although there is an extensive database for PFOA, few studies have examined the toxicity of the shorter or longer chained PFAC. However, the data suggest that the toxicity profile is quite similar to that of PFOA, albeit at different dose levels presumably due to the differences in elimination half-life.

Although standard repeated-dose toxicity studies have not been conducted on the PFAC with chain lengths greater than PFOA, many studies have been conducted examining the potential for hepatomegaly and peroxisome proliferation (a marker for the activation of PPARα). Kudo et. al. found that PFOA, PFNA, and PFDA induced the activity of peroxisomal B-oxidation in male rats (2000). Kudo et al. showed that all PFAC with six- to nine-carbon length chains induced hepatomegaly and peroxisomal B-oxidase activity in mice, and the potency was in the order of PFNA > PFOA > perfluoroheptanoic acid (2006). Permadi et al. also showed that PFDA induces hepatomegaly and hepatic peroxisomal palmitoyl-CoA oxidase (1993). Thus, these studies indicate that the PFAC with a carbon chain length of eight and greater activate PPARα. The differences in potency probably reflect the differences in the half-life of the varying chain lengths. Despite the lack of traditional toxicity studies, it is reasonable to conclude that these compounds would likely produce similar effects as those observed with PFOA.

With respect to the potential developmental effects of PFAC with carbon chain lengths greater than C8, EPA is completing a developmental toxicity study of PFNA in mice (C. Lau, personal communication, 2009). Maternal body weight gain was reduced at 3 mg/kg-day, and severe toxicity was observed at 10 mg/kg-day. Neonatal survival was compromised at 5 mg/kg-day, and significant lags in neonatal growth were observed at 3 mg/kg-day. Thus, this study shows a pattern of effects very similar to those observed with PFOA. It is likely that PFAC with carbon chain lengths greater than nine would also result in similar effects, and that the potency would be dependent on the half-life of the compound.

PFOS

The toxicity of PFOS has also been extensively studied and was summarized in OECD report (2002) and by Lau et al. (2006). Repeated-dose studies in rats and nonhuman primates have shown reduced body weight, hepatotoxicity, reduced cholesterol, and a steep dose-response curve for mortality. These effects occur in nonhuman primates at doses as low as 0.75 mg/kg-day, and in rats at 2 mg/kg-day.

The carcinogenic potential of PFOS has been investigated in a dietary carcinogenicity study in Sprague-Dawley rats, and has been shown to induce hepatocellular adenomas at 20 ppm. In addition, thyroid follicular cell adenomas were observed in male rats that had been allowed to "recover" for a year following treatment for one year; the reason for this is unclear. However, thyroid follicular tumors have also been observed in rats exposed to N-EtFOSE, a major precursor of PFOS. PFOS has not been shown to be mutagenic in a variety of assays. Although PFOS can activate PPARα, the data are not sufficient to establish a PPARα-agonist mode of action for the liver tumors.

A standard prenatal developmental toxicity study in rats has shown a significant decrease in fetal body weight and significant increase in external and visceral anomalies, delayed ossification, and skeletal variations; a NOAEL of 1 mg/kg-day and a LOAEL of 5 mg/kg-day for developmental toxicity were indicated. In rabbits, significant reductions in fetal body weight and significant increases in delayed ossification were observed; a NOAEL of 1.0 mg/kg-day and a LOAEL of 2.5 mg/kg-day for developmental toxicity were indicated.

A two-generation reproductive toxicity study in rats showed neonatal mortality. All F1 pups at the highest dose of 3.2 mg/kg-day died within a day after birth, while close to 30% of the F1 pups at 1.6 mg/kg-day died within 4 days after birth. As a result of the pup mortality in the two top dose groups, only the two lowest dose groups, 0.1 and 0.4 mg/kg-day, were continued into the second generation. The NOAEL and LOAEL for the F2 pups were 0.1 mg/kg-day and 0.4 mg/kg-day, respectively, based on reductions in pup body weight.

The results of this study prompted additional research. Studies in which pregnant rats and mice were dosed during gestation and the pups were followed postnatally provided a BMD_5 and $BMDL_5$ for neonatal survival of 1.07 and 0.58 mg/kg-day in rats, respectively, and 7.02 and 3.88 mg/kg-day in mice, respectively. Studies have shown that the critical period of exposure is during late gestation. Mode of action studies initially focused on the lung and found significant histological and morphometric differences in the lungs of pups treated with PFOS. However,

subsequent studies did not find any effect on lung phospholipids and rescuing agents failed to mitigate the neonatal mortality. Thus, the mortality does not appear to be related to lung immaturity. In contrast to PFOA, studies with PPARα knockout mice have shown that the PPARα is not involved in the neonatal mortality. Current research is focusing on the possibility that the physical properties of PFOS may interfere with the normal function of pulmonary surfactant, leading to neonatal mortality.

Other PFAS Chemicals

A combined reproductive/developmental toxicity study of PFHxS has been conducted in rats. In the parental males there was a significant reduction in cholesterol at doses as low as 0.3 mg/kg-day, and hepatotoxicity at doses as low as 3 mg/kg-day. There was no evidence of developmental or reproductive toxicity at doses as high as 10 mg/kg-day.

Toxicity to Wildlife

Adverse effects on exposed populations of organisms have been observed with exposure to perfluorinated compounds in the parts per million range. Studies have shown a reduction in hatchability of chickens when they were exposed *in ovo* to PFOS, and a reduction in survival in 14-day old Northern bobwhite quail from hens exposed to 10 ppm of PFOS in the diet. In addition, a delay in growth and metamorphosis in the Northern leopard frog exposed to 3 mg/L of PFOS has been reported, as well as reduced cumulative fecundity and fertility effects in fathead minnows exposed to 0.1 mg/L PFOS. Further evidence of potential reproductive effects has been observed with exposure to C9-C11 PFAC. A significant induction of vitellogenin in rainbow trout was observed in a dose-dependent manner at concentrations of C10 PFAC 0.0256-2000 μg/g in the diet as well as a weak affinity demonstrated for the hepatic estrogen receptor from C9-C12 PFAC.

Mortality in sediment dwelling organisms such as the nematode, *Caenorhabditis elegans* has been observed with concentrations of C9 up to 0.66 mM and subsequent effects in offspring generations were found at concentrations up to 1nM as evidence by a 70 % decline in fecundity.

VI. Fate Characterization Summary

The PFAS and PFAC acids are strong acids that exist in equilibrium between the neutral form and the anionic form. Both the anionic and neutral forms of PFOA are soluble in water. While the Henry's law constant values suggests partitioning to air for the neutral, protonated form, predicting the amount that partitions into air is complicated because there is uncertainty over the degree to which carboxylic and sulfonic acids partition from the water to atmosphere. The uncertainty arises with regard to the value of the acid dissociation constant (i.e., pK_a), or the fraction of the acid form present at environmentally relevant pH. PFAC and PFAS have been detected in air, water, and soil samples collected throughout the world. The oceans have been suggested as the final sink and route of transport for perfluorinated carboxylic and sulfonic acids, where they have been detected on the surface and at depths > 1,000 meters (Yamashita, 2005).

Some PFAS/PFAC have the potential for long-range transport. They are transported over

long distances (i.e., long-range transport) by a combination of dissolved-phase ocean and gas-phase atmospheric transport; however, determining which is the predominant transport pathway is complicated by the uncertainty over water to atmosphere partitioning. Furthermore, there is evidence that transport and subsequent oxidation of volatile alcohol PFAS/PFAC precursors may contribute to the levels of PFAS / PFAC in the environment.

Studies by industry and academic researchers have shown that fluorotelomer alcohols (FTOH) can be degraded by microorganisms and by abiotic processes. 8-2 FTOH and FTOH of other chain lengths, and related chemicals in mixed microbial cultures, activated sludge and soil systems have been shown to be easily degraded to form PFOA and related perfluorinated acids. Some studies have also shown that $-CF_2-$ groups can be mineralized, forming shorter chain perfluoro acids. If FTOH are absorbed from ingestion, inhalation, dermal or ocular exposure or formed in vivo by from other compounds they can be metabolized by mammals and other organisms to form perfluorinated acids and other fluorinated compounds. FTOH can be degraded by abiotic processes in water and air to produce PFAC and various intermediates. FTOH are fairly volatile. Based on atmospheric half-lives determined in chamber studies, FTOH can be transported globally. Deposition or degradation in areas far from the source can result in PFAC contamination in high latitudes and other remote locations and contribute to global background levels of PFAC and PFAS.

Data submitted by industry and in the open literature show that perfluorooctane sulfonyl fluoride (POSF) and its derivatives can be degraded under environmental conditions to form perfluoroalkyl sulfonates and carboxylic acids. Reaction of POSF ($CF_3(CF_2)_n-SO_2F$) with methyl or ethyl amines is used to produce N-ethyl or N-methyl perfluorooctane sulfonamidoethanols (FOSE). Similar reactions are used to make shorter and longer chain analogs to POSF and POSF derivatives. FOSE compounds, (or $CF_3(CF_2)_n-SO_2N(R1)(R2)$, where R1 and R2 can be hydrogen, methyl or longer alcohols or other organic chains), such as N-methyl and N-ethyl FOSEs can be degraded though a series of intermediates to form both perfluoro carboxylic acids and perfluoroalkyl sulfonates. Data on the degradation of individual intermediates has been used to identify these pathways and has confirmed that these compounds can be degraded by a number of microbial and abiotic mechanisms. Reaction with other chemical intermediates produces other FOSA derivatives, including phosphate esters, fatty acids esters, silanes, carboxylates, and polymers with acrylate, urethane and other linkages. Longer and shorter chain perfluoro sulfonyl derivatives have also been produced intentionally and as unintended reaction products. Based on existing data from the open literature and CBI data, it is expected that that most, if not all, of these POSF and other chain length sulfonyl fluorides and their derivatives will be degraded to carboxylic acids and/or sulfonate over time. Most of these compounds will have environmental and metabolism half-lives of weeks to months. Some will be degraded faster and some will degrade more slowly, but all will eventually be degraded.

Very little data is available on the behavior of other perfluorochemicals in the environment and in vivo but the existing data suggest that they will also be degraded to form PFAC. For example, recent studies have shown that ingested mono and di polyfluoroalkyl phosphates (PAPs) can be degraded in rats to form PFOA and other PFAC in the body. They can also be degraded by microbial processes in soil and wastewater to form perfluorinated acids (D'eon, 2007).

A limited number of studies on the degradation of fluorotelomer-based polymers have been submitted in support of PMN submissions and existing chemicals, and published in the open literature. Based on studies, some fluorotelomer-based polymers are subject to hydrolysis, photolysis and biodegradation to some extent. Studies have shown half-lives of a few days to hundreds of years.

In addition, preliminary research on degradation of fluorotelomers has shown that some urethanes and acrylates biodegrade; however, half-lives and kinetics of the fluorotelomers are not yet well-defined. Ongoing research by EPA's Office of Research and Development (ORD) research is designed to generate high quality data that will help the Agency address some key uncertainties in pathways of exposure and potential risks from PFOA (Washington, 2009).

These studies have shown that the perfluorinated portion of some polymers is released as the polymer is degraded by microbial or abiotic processes to form telomer alcohols or other intermediates and that they eventually form PFAC. Polymers based on POSF and other chain length chemistries show similar degradation rates and release intermediates that further degrade to form perfluorinated acids and sulfonates. Studies have shown that some polymers can undergo indirect photolysis in soil and in aquatic systems and be degraded with half-lives of days to several years.

VII. Exposure Characterization Summary

The pattern of PFAS and PFAC contamination varies with location and among species, which suggests multiple sources of emission and patterns of migration into environmental media from the sources of emission. Major pathways that enable PFOA and PFOS to get into human blood in small quantities are not yet fully understood. Manufacturing releases are known to have contaminated local drinking water supplies in the immediate vicinity of some industrial plants, leading to localized elevated blood levels. The widespread presence of PFOA and PFOS precursors in human blood samples nationwide suggests other pathways of exposure, possibly including long range air transport, and the release of PFOA and PFOS from treated articles.

Summary of Exposure to Consumers and Children from PFCs in Indoor Environments

PFCs in Articles of Commerce

EPA's ORD has conducted research on 116 articles of commerce documenting that PFCs contained in articles of commerce have the potential to be released from those articles. Articles tested and found to contain the highest levels of PFAC were carpet and carpet treatment products, various types of apparel, home textiles, thread sealant tape, floor wax and other sealants, and food contact paper and paper coatings. Carpet and carpet treatment products contained individual PFAC in levels from 0.04-14100 ng/g; food contact paper and paper coatings: 0.05-160,000 ng/g; thread sealant tape and apparel: ND (non-detect)-3488 ng/g and ND-4640ng/g respectively; floor wax and sealer: 0.03-3720 ng/g; and home textiles: ND-519 ng/g. Some of the more commonly found PFAC measured in these articles were PFHxA, PFHpA, PFNA, PFDA, PFUnDA, PFOA and PFOS. Inhalation levels of PFOA and total PFCs

measured in carpet were 5385 pg/cm^3 and 32500 pg/cm^3 respectively (Guo, 2009).

Children are particularly susceptible to exposure from inhalation of PFC off-gassing from carpet and carpet protectants during their earliest years when they are lying, crawling and spending large amounts of time playing on the carpet. The significantly high levels of PFC found by ORD in carpet and carpet protectants pose an exposure concern for children through this pathway. Adults can also be exposed to PFCs in carpets through inhalation and dermal contact. Consumers and children may also be exposed to PFCs in apparel, home textiles, thread sealant tape, floor wax, contact paper and paper coatings. Some of these articles such as paper coatings for foods cannot be ruled out for the ingestion exposure pathways for children and adults depending upon how the PFCs in the paper contacts the food and subsequently humans.

PFCs in Indoor Air

Another source of PFCs to the indoor environment is dust containing not only PFAC and PFAS but also fluorotelomer alcohols. Maximum indoor dust air measurements of 6:2 FTOH were found at 804 ng/g in the house dust of eastern United States (Strynar, 2008). The PFAS (ET-FOSA, Et-FOSE, MeFOSE) chemicals were measured at 646 ng/g, 75440 ng/g, and 8860 ng/g respectively in indoor air in Canada (Shoeib, 2005). PFOA was found at 3700 ng/g in Japanese household vacuum cleaner dust (Moriwaki, 2003).

Summary of Exposure to the General Population

PFCs in Groundwater, Freshwater, Saltwater, and Rainwater

PFAC and PFAS have been found in many countries as well as in Unites States in untreated groundwater, rivers, streams, bays, estuaries, oceans and rain water. Levels of PFAC in groundwater near the 3M Cottage Grove, MN industrial site have been measured as high as 846,000 ng/l (PFOA) and in freshwater as high as 178,000 ng/l (PFBA) (Department of Health and Human Services, 2005). PFOS has been found near Cottage Grove, MN in groundwater at levels of 371,000 ng/l and in freshwater at 18,200 ng/l. PFAC in rainwater has been measured in the United States between 0.1 and 1006 ng/l (PFHpA) (Scott BF, 2006).

Saltwater levels of PFOS have been measured in the Pacific Ocean at 57,700 ng/l and in precipitation from snow and rain in China at 545 ng/l (Liu W, 2009; Yamashita, 2005). While the general population may not directly ingest these groundwater, freshwater and saltwater levels as drinking water, the ground water and freshwater containing PFCs may discharge to surface waters from which municipalities withdraw drinking water. The general population may also experience dermal, ingestion and inhalation exposures when coming into contact with freshwater containing PFCs. Rainwater containing PFCs may contribute PFCs to vegetables and fruits in home gardens, crops grown on commercial crop lands, drinking water reservoirs, and surface waters from which drinking water is withdrawn.

PFCs in Freshwater and Saltwater Fish

Freshwater fish have been found to contain levels of PFAS and PFAC. The highest levels

of PFAS measured in the United States to date were near the 3M Cottage Grove, MN site (Oliaei F, 2006). Liver samples of bass, walleye and carp ranged from 130-6350 ng/g PFOS wet weight. Blood samples of these same fish ranged from PFOS levels of 136-29600 ng/ml in serum. Total PFCs for the blood of freshwater fish in the same area was measured at 32248 ng/ml serum. The highest levels of PFAC for freshwater fish were found near the 3M Cottage Grove, MN site and were measured for blood samples of bass, walleye, and carp in the range of 2.53-210 ng/ml serum. For comparison, saltwater fish in Danish seas had measured levels of PFOS up to 156 ng/g and saltwater fish in Charleston Harbor South Carolina were found with PFOS levels up to 101 ng/g (Bossi R, 2005; Houde M, 2006).

VIII. Risk Management Considerations

Current Risk Management Summary

PFAS Chemicals

Following the voluntary 3M phase-out of PFAS chemicals in the United States in 2002, EPA issued SNURs to control the reintroduction of these chemicals into the U.S. market. Final rules were published on March 11, 2002 (EPA, 2002b) and December 9, 2002 (EPA, 2002a), to limit any future manufacture or importation of 88 PFAS chemicals specifically included in that phase-out. On October 9, 2007, EPA published another SNUR on 183 additional PFAS chemicals (EPA, 2007). Those actions were necessary because data showed that certain alkyl chain lengths of the PFAS chemicals are toxic to human health, bioaccumulate, and are persistent in the environment. PFAS chemicals are no longer manufactured in United States. However a limited set of existing uses was excluded from the SNURs because alternatives were not yet available.

Similar to the PFAS SNURs in United States, PFOS has also been restricted in the European Union, Canada, Australia and other countries, and has been nominated for inclusion in the Stockholm Convention and the Convention on Long-Range Transboundary Air Pollution (LRTAP) Persistent Organic Pollutants (POPs) protocol. At the fourth Conference of the Parties (COP) to the Stockholm Convention on POPs, held in May 2009, delegates agreed to add PFOS, its salts, and perfluorooctane sulfonyl fluoride (PFOSF) to Annex B, subjecting it to restrictions on production and use. Parties agreed that while the ultimate goal is the elimination of PFOS, production of the chemical may continue for limited purposes, including coatings for semiconductors, firefighting foam, photo imaging, aviation hydraulic fluids, metal plating, and certain medical devices. Countries must notify the Convention Secretariat whether they intend to continue production for acceptable purposes. Countries can also ask for specific exemptions allowing the production of PFOS for use in the production of chemical substances used in goods such as carpets, leather and apparel, textiles, paper and packaging, coatings, and rubber and plastics (POPs, 2009).

PFAC Chemicals

OPPT's core strategy for working towards the elimination of PFAC chemicals has been through the PFOA Stewardship Program. Under the program, eight major companies operating

in the United States committed to reduce global facility emissions and product content of PFAC chemicals by 95 percent by 2010, and to work toward eliminating emissions and product content by 2015 (EPA, 2009a). Companies provide annual progress reports, and most companies have reported significant progress in meeting program goals.

On March 7, 2006, EPA published a proposal to amend the polymer exemption rule to exclude polymers containing certain perfluoroalkyl moieties from eligibility for the exemption (EPA, 2006). Under this proposal, polymers containing these perfluoroalkyl moieties would need to go through the pre-manufacture notification (PMN) review process so that EPA can better evaluate these polymers for potential effects on human health and the environment. This change to the current regulation is necessary because, based on current information, EPA can no longer conclude that these polymers "will not present an unreasonable risk of injury to health or the environment" under the terms of the polymer exemption rule, which is the determination necessary to support an exemption under section 5(h)(4) of TSCA. This amendment to the polymer exemption rule is a necessary complement to the PFOA Stewardship Program and will give EPA the necessary tools to review and control risk of PFC-based and related polymers, including those PFAS and PFAC containing polymers.

In January 2009, EPA's Office of Water (OW) developed Provisional Health Advisory (PHA) values for PFOA and PFOS to mitigate potential risk from exposure to these chemicals through drinking water (EPA, 2009c). Due to limited information on the toxicity of PFCs other than PFOA and PFOS, no attempt was made by OW at that time to develop PHA values for the other PFCs. OPPT and OW are working together to determine whether revised health advisory values are needed for PFOA and PFOS.

In October 2009, EPA's Office of Solid Waste and Emergency Response (OSWER) used OW's PHA's to derive sub-chronic R_fD values for PFOA and PFOS. These values may be used in the Superfund program's risk-based equations to derive Removal Action Levels and/or Screening Levels for water and other media, as appropriate.

EPA has taken the leadership role in raising the profile of PFCs at an international level stemming from Agency concerns about the role of long range transport in the environmental distribution of PFCs, and U.S. importation of products containing these chemicals (UNEP, 2009b). As a result of these activities, in May 2009, during the International Conference on Chemicals Management (ICCM2), delegates to the Strategic Approach to International Chemicals Management (SAICM) agreed to consider the development of stewardship programs and regulatory approaches to reduce emissions and content of PFAC and PFAS chemicals in products and to work towards their elimination, where feasible (UNEP, 2009a).

Remaining Issues and Concerns

PFAS Chemicals

PFAS chemicals are no longer manufactured in the United States but continue to be manufactured outside of the United States. Although the PFAS SNURs are an important step toward controlling any future manufacture or import of PFAS chemicals, these chemicals may

continue to be imported into United States in articles, such as carpets, leather and apparel, textiles, paper and packaging, coatings, and rubber and plastics.

Possible scenarios of concern:
- Direct releases to the environment from U.S. facilities as a result of few existing uses.
- Direct releases to the environment from non-U.S. facilities, resulting in transboundary environmental transport to United States.
- Articles containing PFAS chemicals. Recent research by EPA's ORD has shown that consumer articles could release PFCs, significantly increasing the magnitude and duration of exposure to humans and the environment to these chemicals.

PFAC Chemicals

Although the 2010/15 PFOA Stewardship Program is expected to eliminate the production of C8-based fluorotelomers by the eight participating companies by 2015, the potential remains for continued environmental and human loading of PFAC in the United States. This is in part because companies not participating in the PFOA Stewardship Program may follow the market opportunity presented when the eight PFOA Stewardship Program companies leave the PFAC market by 2015. This occurred with PFAS production in some Asian countries after the 3M 2002 phase-out of PFAS chemicals in United States (Wenya, 2008).

Possible scenarios of concern:
- Direct releases to the environment from U.S. facilities not participating in PFOA Stewardship Program.
- Direct releases to the environment from non-U.S. facilities not participating in PFOA Stewardship Program, resulting in transboundary environmental transport to United States.
- Articles, including imports, containing PFAC chemicals. These articles could release PFAC as a result of their residual content in fluorotelomer-based products and/or as the fluorotelomers-based polymers in articles biodegrade.

IX. Next Steps

To date, significant adverse effects have not been found in general human population; however, significant adverse effects have been identified in laboratory animals and wildlife. Given the long half-life of these chemicals in humans (years), it can reasonably be anticipated that continued exposure could increase body burdens to levels that would result in adverse outcomes. Consequently, EPA intends to propose actions in 2012 under TSCA to address the potential risks from long-chain PFCs.

EPA intends to consider initiating TSCA section 6 rulemaking for managing long-chain PFCs. If EPA can make certain findings with respect to these chemicals (further analysis of the information will be performed as part of TSCA section 6 rulemaking), TSCA section 6 provides authority for EPA to ban or restrict the manufacture (including import), processing, and use of these chemicals. A rule addressing the PFAS sub-category could expand beyond the reach of the SNURs that the Agency has promulgated over the past decade. For example, the rule could address PFAS-containing articles. A rule addressing the PFAC sub-category could expand the

reach of the 2010/15 PFOA Stewardship Program beyond the eight participating companies and further address the concerns for potential PFAC exposure through the use of PFAC-containing articles. EPA will develop more detailed assessments to support the TSCA section 6(a) "presents or will present an unreasonable risk" findings. If these more detailed assessments indicate that a different approach to risk management is appropriate, EPA will consider additional approaches.

EPA will continue with the 2010/15 PFOA Stewardship Program to work with companies toward the elimination of long-chain PFCs from emissions and products. EPA will also continue to evaluate alternatives under EPA's New Chemicals Program and collaborate with other countries on managing PFCs.

As part of the Agency's efforts to address these chemicals, EPA also intends to evaluate the potential for disproportionate impact on children and other sub-populations.

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