Product – Chemical Profile for Perfluoroalkyl and Polyfluoroalkyl Substances (PFASs) in Carpets and Rugs

SAFER CONSUMER PRODUCTS

Department of Toxic Substances Control

CalEPA California Environmental Protection Agency

February 2018 • DISCUSSION DRAFT

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ABOUT THIS REPORT

The Department of Toxic Substances Control (DTSC) identifies product-chemical combinations for consideration as Priority Products in accordance with the process identified in Article 3 of the Safer Consumer Products (SCP) regulations[1] (Process for Identifying and Prioritizing Product-Chemical Combinations). Based on the findings presented in this Product-Chemical Profile (Profile), DTSC finds PFASs in carpets and rugs to meet the key prioritization criteria[2] for listing a Priority Product:

(1) There must be potential public and/or aquatic, avian, or terrestrial animal or plant organism exposure to the Candidate Chemical(s) in the product; and

(2) There must be the potential for one or more exposures to contribute to or cause significant or widespread adverse impacts.

This Profile explains DTSC's rationale for considering a product-chemical combination prior to initiating a Priority Product rulemaking. DTSC will finalize this Profile after considering public comments, and may then start the rulemaking process. Once the regulation is adopted, the responsible entities must follow the reporting requirements pursuant to section 69503.7 and Article 5 (Alternatives Analysis) in the SCP regulations.

Readers should consider the following:

1. This Profile is not a regulatory document and does not impose any regulatory requirements.
2. The Profile summarizes information compiled by DTSC as of February 2018.
3. DTSC requests that stakeholders provide data on the chemical and product described in this document to assist us in the discernment process that will lead to our regulatory proposal. Written comments can be submitted using our information management system, CalSAFER,[3] prior to March 23, 2018.
4. By proposing to list this product-chemical combination as a Priority Product containing a Chemical of Concern, DTSC is not asserting that the product cannot be used safely, only that there is a potential for exposure of people or the environment to the Chemical of Concern in the Priority Product and that such exposure has the potential to cause or contribute to significant or widespread adverse impacts.

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[1] CAL. CODE REGS. tit. 22, Division 4.5, Chapter 55.

[2] CAL. CODE REGS. tit. 22, § 69503.2(a).

[3] https://calsafer.dtsc.ca.gov/cms/searchpackages/?documentType=4

SUMMARY OF THE RATIONALE FOR PRODUCT-CHEMICAL SELECTION

The Department of Toxic Substances Control (DTSC) proposes to list carpets and rugs containing perfluoroalkyl and polyfluoroalkyl substances (PFASs) as a Priority Product. This product-chemical combination meets the identification and prioritization factors outlined in the Safer Consumer Products Regulations: (1) there is potential for human and other organism exposure to PFASs in carpets and rugs; and (2) the exposure has the potential to contribute to or cause significant and widespread adverse impacts. The class of PFASs contains more than 3,000 man-made chemicals with at least one fully fluorinated carbon atom. All PFASs are Candidate Chemicals because the California Environmental Contaminant Biomonitoring Program lists the entire class as Priority Chemicals for measuring in the blood or urine of Californians.

Potential for exposure to PFASs in carpets and rugs

DTSC has identified carpets and rugs as sources of significant and widespread human and ecological PFAS exposures. Carpets and rugs constitute nearly half of all floor coverings in U.S. homes and workplaces. A large percentage of the PFASs produced worldwide are used to treat carpets, rugs, and other home textiles to confer stain-, soil-, oil- or water-resistance.

PFASs have a wide variety of applications, including in numerous consumer products. Regardless of their sources, due to their unique physicochemical properties, PFASs released to the environment end up virtually everywhere in aquatic, atmospheric, and terrestrial environments, including remote locations far from any point source. They are present, for instance, in indoor dust, drinking water sources, food, wildlife, and humans—including the milk and serum of breastfeeding women.

Once released to the environment during product manufacture, use, or disposal, PFASs become part of a virtually closed cycle leading to chronic, lifelong human and ecological exposures. Carpets and rugs contribute to the widespread environmental contamination and exposures, as do other consumer products such as food packaging, cosmetics, and waterproof clothing. Because persistent PFASs lack a natural degradation route, their levels in the environment, humans, and biota may continue to rise for as long as PFASs are produced and used in consumer products.

Once released to the environment during product manufacture, use, or disposal, PFASs become part of a virtually closed cycle leading to chronic, lifelong human and ecological exposures. Carpets and rugs contribute to the widespread environmental contamination and exposures.

Human exposure to PFASs begins early in a person’s life, since mothers transfer these chemicals to their babies via the placenta and breastfeeding. In the general population, PFAS exposure occurs mainly via ingestion of contaminated food and drinking water. This contamination is partly due to releases of PFASs from treated consumer products at various points in their lifecycle, including manufacturing, use, and disposal. Through normal use, treated carpets, rugs, and other consumer products release PFASs into indoor air and dust, which people inhale or ingest. Most (75 per cent in 2016) waste or end-of-life carpets and rugs in California are disposed of in landfills, where they become sources of PFASs to the environment via leachates and gaseous emissions. Wastewater treatment plants that collect landfill leachates, surface runoff, and residential and commercial wastewater cannot effectively remove PFASs. As a result, when wastewater effluent is discharged into surface waters, PFASs are released into the environment, contaminating aquatic ecosystems and drinking water sources. Sewage sludge also contains PFASs, thus the application of biosolids on soil can contaminate terrestrial ecosystems, drinking water, and human food supplies.

Carpets and rugs are a major source of exposure for infants and children via direct contact and indoor dust ingestion. Young children have been shown to ingest more soil and dust than adults, due to greater hand-to-mouth transfer; this can result in higher exposure to PFASs found in these contaminated environmental media. Industrial workers, carpet installers, carpet cleaners and workers in upholstered furniture, furnishings, outdoor clothing, and carpet stores may also experience above average PFAS exposure levels.

Potential for significant or widespread adverse impacts

Due to the strength of the carbon-fluorine bond, PFASs are characterized by high environmental persistence, which leads to continuous and poorly-reversible accumulation in the environment, and hence to likely increasing exposures. Most PFASs display significant mobility in environmental media, which makes them widespread in the environment and in living organisms. Many members of the PFAS class bioaccumulate significantly in animals or plants, including in foods consumed by humans, and undergo lactational or transplacental transfer from mothers to offspring. Certain PFASs also have high global warming potential, or may contribute to global warming by increasing cloud albedo.

PFASs show evidence for several toxicological and environmental hazard traits. In humans, reported adverse health effects from chronic exposure to the most extensively studied PFASs include increased serum cholesterol, thyroid disease, immune system disruption, pregnancy-induced hypertension, and kidney and testicular cancers. Animal studies have associated PFAS exposure with carcinogenicity, developmental toxicity, reproductive toxicity, cardiovascular toxicity, endocrine toxicity, hematotoxicity, hepatotoxicity and digestive system toxicity, immunotoxicity, nephrotoxicity and other urinary system toxicity, neurodevelopmental toxicity, and respiratory toxicity. Some PFASs have also been linked to phytotoxicity, aquatic toxicity, and terrestrial ecotoxicity.

Under a voluntary agreement with the U.S. Environmental Protection Agency (U.S. EPA), in 2015, major PFAS manufacturers phased out the production and emission of longer-chain PFASs with known adverse health impacts and their precursors. As a result, biomonitoring studies have shown decreasing levels of certain longer-chain PFASs in human tissues. However, most replacements are also PFASs, including fluorinated ethers and shorter-chain PFASs. Fluorinated ethers were thought to degrade easily, but recent studies found they also persist indefinitely in the environment. Shorter-chain PFASs are marketed as less toxic compared to the longer-chains, mainly because they appear to bioaccumulate less and to be more readily eliminated from some organisms. Nevertheless, they are equally persistent and more mobile in the environment than the chemicals they are replacing, and also show potential for toxicity. Toxicological and epidemiological data clearly indicating the safety of aggregate, chronic and low-dose exposures to PFASs found in stain- and soil-repellents are lacking.

Based on the criteria in the Safer Consumer Products Regulations, DTSC has determined that PFASs have the potential to cause significant and widespread adverse impacts to sensitive subpopulations, including fetuses, infants, young children, pregnant women, carpet installers, and carpet retail sector workers; to environmentally-sensitive habitats; and to threatened and endangered species. Given the known hazard traits and the absence of data demonstrating the safety of any member of the class, replacing currently-used PFASs in carpets and rugs with other members of the PFAS class could constitute a regrettable substitution. Hence, this proposal covers the entire class of PFASs in carpets and rugs.

1 PRODUCT-CHEMICAL DEFINITIONS AND SCOPE

1.1 Scope of Candidate Chemical

This proposal covers all perfluoroalkyl and polyfluoroalkyl substances (PFASs) in current or future production. All PFASs are Candidate Chemicals (DTSC 2016) under the Safer Consumer Products (SCP) program due to their designation on December 22, 2015 as Priority Chemicals under the California Environmental Contaminant Biomonitoring Program (California Biomonitoring Program or CECBP). The CECBP listing is based on the chemicals' potential for widespread exposures, persistence, bioaccumulation, and emerging evidence for toxicity. In addition, DTSC's Candidate Chemicals database includes 73 other listings for individual PFASs (see Appendix 1).

All PFASs are Candidate Chemicals under the Safer Consumer Products (SCP) program due to their designation on December 22, 2015 as Priority Chemicals under the California Environmental Contaminant Biomonitoring Program.

PFASs are a class of fluorinated organic chemicals containing at least one fully fluorinated carbon atom (CECBP 2015a). This class includes more than 3,000 chemicals (KEMI 2015), although fewer are currently in production. PFASs are manufactured and not found naturally in the environment. All PFASs contain carbon and fluorine atoms, in addition to other elements such as oxygen, hydrogen, nitrogen, sulfur, or chlorine.

PFASs vary in chain length, i.e., the number of carbon atoms forming the backbone of their molecule, from a chain of two carbons to large molecular weight polymers. Based on common characteristics, and for the purposes of this designation, PFASs can be roughly subdivided into four main categories (Buck et al. 2011; Wang et al. 2017) listed below and depicted in Figure 1. However, any other PFAS that exists or will be developed in the future also falls under the scope of this proposal:

1 **Perfluoroalkyl acids (PFAAs)**. These are perfluorinated substances in which fluorine atoms have replaced all hydrogen atoms attached to carbon atoms (except for those associated with functional groups). As a result, these compounds are recalcitrant to degradation and extremely persistent in the environment. This subgroup includes (see Appendix 2 for examples with CAS numbers):

    - Perfluoroalkyl carboxylic acids (PFCAs) such as perfluorooctanoate (PFOA);[4]
    - Perfluoroalkyl sulfonic acids (PFSAs) such as perfluorooctane sulfonate (PFOS);[4]
    - Perfluoroalkyl sulfinic acids (PFSiAs);
    - Perfluoroalkyl phosphonic acids (PFPAs);
    - Perfluoroalkyl phosphinic acids (PFPiAs);
    - Perfluoroether carboxylic acids (PFECAs); and
    - Perfluoroether sulfonic acids (PFESAs).

2. **PFAA precursors.** These are mostly polyfluoroalkyl substances, meaning fluorine atoms have replaced all hydrogen atoms attached to at least one (but not all) carbon atoms. Polyfluorinated substances have the

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[4] PFOA and PFOS are referred to as "C8" because they contain eight carbon atoms in their molecules.

potential to degrade into perfluoroalkyl substances, i.e., they are precursors to perfluoroalkyl substances. Examples include:

- PFASs used for surface treatments:
    - Side-chain fluorinated polymers (e.g., fluorinated acrylate, methacrylate, urethane, oxetane polymers);
    - N:2[5] polyfluoroalkyl phosphoric acid esters, also known as polyfluoroalkyl phosphates, or fluorotelomer phosphates (PAPs);
    - Polyfluorinated organosiloxanes; and
    - Fluorotelomer ethoxylates (FTEOs).

- PFASs commonly used as raw materials for surface treatment products:
    - *N*-alkyl perfluoroalkane sulfonamides (FASAs);
    - Perfluoroalkane sulfonamide ethanols (FASEs) and *N*-alkyl perfluoroalkane sulfonamidoethanols;
    - *N*-alkyl perfluoroalkane sulfonamidoethyl acrylates and methacrylates (FAS(M)ACs);
    - Fluorotelomer iodides (n:2 FTIs);
    - Fluorotelomer olefins (n:2 FTOs);
    - Fluorotelomer alcohols (n:2 FTOHs); and
    - Fluorotelomer acrylates (n:2 FTACs) and methacrylates (n:2 FTMACs).

- PFASs formed as intermediate environmental transformation products:
    - Fluorotelomer aldehydes (n:2 FTALs) and unsaturated aldehydes (n:2 FTUALs);
    - Fluorotelomer carboxylic acids (n:2 FTCAs) and unsaturated carboxylic acids (n:2 FTUCAs);
    - Fluorotelomer sulfonic acids (n:2 FTSs);
    - Fluorotelomer ethoxycarboxylates (FTEOCs); and
    - n:3 saturated and unsaturated acids.

3. **Perfluoropolyethers (PFPEs).** These are perfluoroalkyl substances that are highly persistent in the environment, with large molecular size (oligomers, polymers, and copolymers), and ether linkages. They cannot degrade to PFAAs, but PFAAs may be feedstock in manufacturing or impurities.

4. **Fluoropolymers.** These polymers are materials (as opposed to surface treatments), and are highly persistent in the environment. They cannot degrade to PFAAs, but certain PFAAs have been used in their manufacturing and can occur as impurities. Examples include polytetrafluoroethylene (PTFE), polyvinylidene fluoride (PVDF), and polyvinyl fluoride (PVF).

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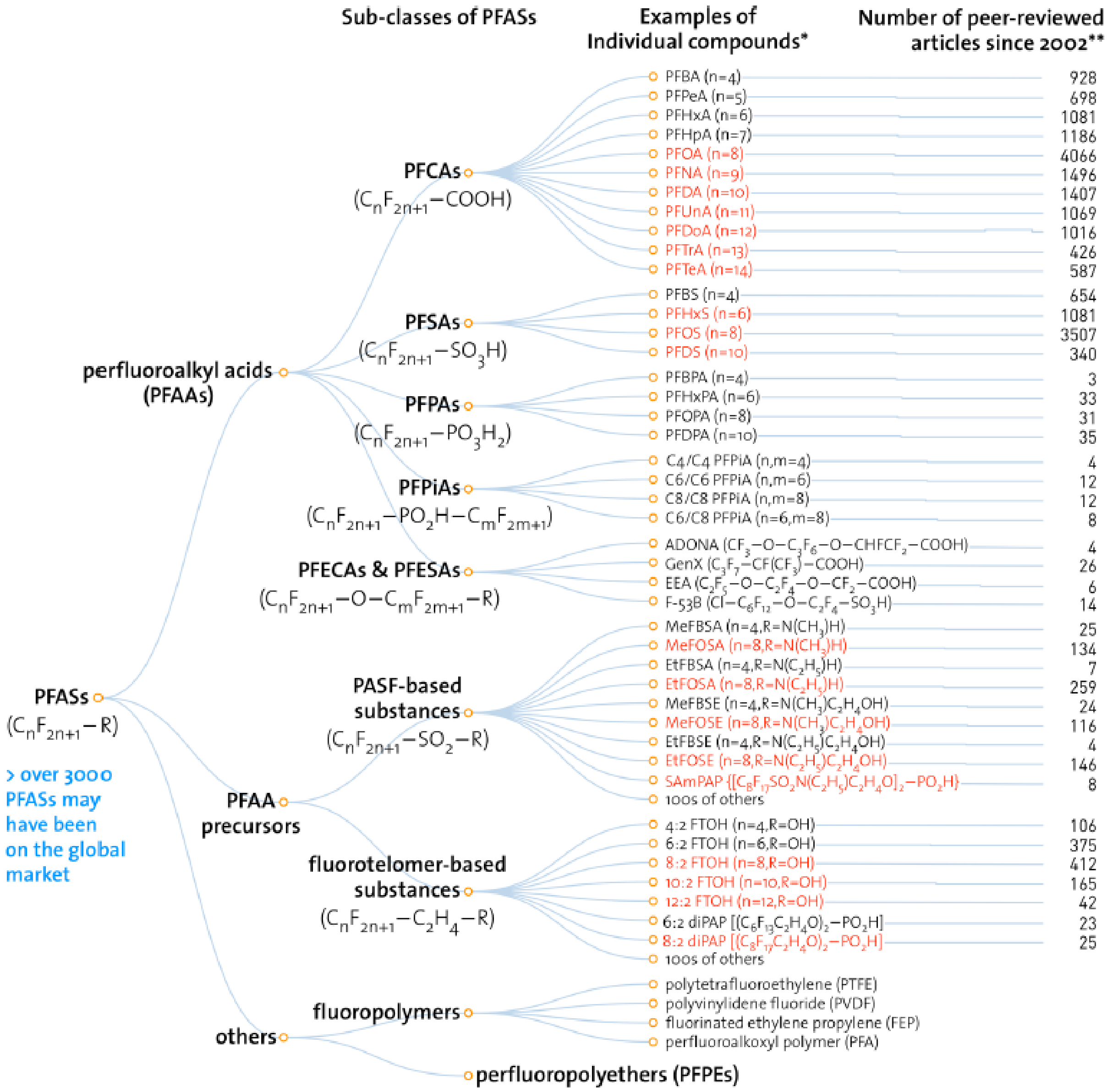
[5] “N” refers to the number of perfluorinated carbon atoms; the number after the colon refers to the number of non-fluorinated carbon atoms in the molecule.

PFAAs and some of their precursors are frequently subdivided into longer- and shorter-chain PFASs. The longer-chain PFSAs have six or more perfluorinated carbons; longer-chain PFCAs, PFPAs, and PFPiAs have seven or more perfluorinated carbons (Bowman 2017a).



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* PFASs in RED are those that have been restricted under national/regional/global regulatory or voluntary frameworks, with or without specific exemptions (for details, see OECD (2015), Risk reduction approaches for PFASs. http://oe.cd/1AN).

** The numbers of articles (related to all aspects of research) were retrieved from SciFinder® on Nov. 1, 2016.

Figure 1: General classification of PFASs (Wang et al. 2017).

1.2 Scope of Product

Carpets and rugs are any consumer product made from natural or synthetic fabric intended to be used as a floor covering inside commercial or residential buildings [adapted from the U.S. Consumer Product Safety Commission's Carpets and Rugs Business Guidance (U.S. CPSC 2017)]. This includes carpeted door mats used indoors and outdoors.

The Global Product Classification (GPC) categorizes carpets as Building Materials (Flooring - Carpet), and rugs as Household / Office Furniture / Furnishings (Floor Rugs/Mats – Detachable). The relevant Brick Codes are (GS1 2016):

- Carpets: 10002446
- Rugs: 10002218.

The relevant North American Industrial Classification System (NAICS) codes for the carpet and rug industry, as designated by the U.S. Census Bureau (U.S. Census Bureau 2017), include:

- 31-33 – Manufacturing
    - 314 – Textile Product Mills
        - 3141 – Textile Furnishings Mills
            - 31411 – Carpet and Rug Mills.

This proposal does not include: carpets and rugs for outdoors use; carpets and rugs for use inside planes, trains, buses, automobiles, or any other indoor environments besides buildings; resilient floor coverings (e.g., linoleum and vinyl tile); artificial turf; wall hangings and coverings; table mats; or camping sleeping mats.

1.3 Chemical and Product Use and Trends

PFASs possess unique physicochemical characteristics that confer increased stability in extreme temperatures, anti-static properties, and resistance to wettability, staining, and corrosion (Buck 2012; Buck et al. 2011). PFASs are used in a wide variety of applications, including (FluoroCouncil 2017a; KEMI 2015):

- surface treatments for leather and textiles
- surface treatments for food packaging, processing, and preparation
- metal (chromium) plating fume suppressants
- detergents, pharmaceuticals, pesticides
- aqueous firefighting foams (AFFFs)
- aerospace, automotive, construction, and electronics manufacturing industries.

In carpets and rugs, PFASs have been used since the early 1980s to impart stain-, soil-, and grease-resistance (Dusaj 1988; U.S. EPA 2012). According to the Carpet and Rug Institute, the industry trade group representing 90 percent of U.S. carpet manufacturers, "most residential and commercial carpets are treated" with PFAS-based stain- and soil-repellents (Yarbrough 2017). PFAS-containing treatments can be applied to carpets at four different stages: (1) during the manufacturing of the carpet fibers; (2) during the carpet and rug manufacturing process, at the carpet and rug mill; (3) after the carpet and rug manufacturing process, at a separate finishing facility or in stores at the time of sale; or (4) post-sale of the carpet or rug by consumers or professional cleaners (U.S. EPA 2012). This proposal considers only the first three applications.

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In the mid-1950s, 3M developed Scotchgard Fabric Protector, the first PFAS-based stain- and soil-repellent for use on textiles. This was followed, in 1963, by the development of Zepel by Du Pont, for use on clothing. Later, in 1972, 3M produced Scotchgard Carpet Protector, the first product specifically marketed for use on carpets (Maitland 1982). The floor covering sector has been receptive to using surface treatments because carpet fibers are commonly made of synthetic materials such as nylon, polypropylene, acrylic, and polyester, which can readily absorb water- or oil-based compounds (Hilton 2017). Thus, most commercial and residential carpets and rugs sold in the U.S. are treated with PFASs to provide resistance to soil and to oil- or water-based stains, by preventing adhesion of solid and liquid contaminants to fibers. However, according to a major carpet manufacturer (Davis 2016), PFAS-based treatments increase product performance only for lower quality carpet fibers, and only for a limited amount of time because, when the coating begins to wear off, it traps soil underneath, creating permanent stains.

The United States produces more than half the world's fluorotelomers and fluorotelomer-based polymers (U.S. EPA 2015a). Globally, 50 percent of these fluorotelomers and associated side-chain fluorinated polymers are used in the treatment of textiles and apparel, including carpets and rugs (U.S. EPA 2009). The PFAS polymers used in carpets, rugs, and other textiles can contain various amounts of mobile residual raw materials, impurities, or degradation products, including PFAAs and other PFAA precursors such as fluorotelomer alcohols (FTOHs) and perfluoroalkyl sulfonamide alcohols (Buck and Schubert 2009; Posner 2017).

Prior to the year 2000, a 3M-commissioned study estimated a 50 percent PFAS treatment loss from carpets due to walking and vacuuming over a typical nine-year product lifespan (Battelle Memorial Institute 2000). Since then, several industry-led improvements have significantly reduced the mobility of the fluorinated treatment applied to carpets and rugs.

During the early to mid-2000s, fluorotelomer-based side-chain fluorinated polymers replaced non-polymeric PFASs, such as sulfonamides, in treatments for carpets and rugs (FluoroCouncil 2017b). According to the

FluoroCouncil,[6] a trade association representing the world's leading PFAS manufacturers, all uses of non-polymeric PFASs in carpets and rugs have ceased in both domestic and imported products, because they are more expensive, less effective, and less stable than the polymers. Side-chain fluorinated polymers are now the most common carpet and rug treatments on the U.S. market. However, other polymeric PFASs such as PFPEs and fluoropolymers may also be used. For instance, a patent filed on January 31, 2013 by INVISTA North America S.a.r.l. for clay nanoparticle and wax-based "liquid and soil repellent compositions for fibers," including carpets and rugs, contains the following statement (Iverson et al. 2017):

> *"Even though a fluorochemical is not needed to achieve the desired soil and liquid repellent properties, one or more may be included. For example, the soil repellent composition can include a fluorochemical. A suitable fluorochemical may be derived from any of the classes specific to fluorinated matter including fluoropolymers, perfluoropolyethers (PFPEs), and side-chain-fluorinated polymers."*

Since the mid-2000s, industry has shifted from sprayed topical application to an "exhaust" application using heat to more firmly bind the PFAS treatment to the carpet and rug, thus increasing the effective life of the carpet treatment (FluoroCouncil 2017b). As a result, less of the PFAS treatment is lost during product life span, and many carpet manufacturers now offer life-time warranties for stain resistance. Further improvements to the PFAS treatment application process have significantly decreased the amount of fluorine needed to maintain performance compared to methods used in the 1990s (FluoroCouncil 2017b).

As adverse health effects associated with exposure to PFOA and PFOS became well characterized, the U.S. EPA's 2010/2015 Stewardship Program (U.S. EPA 2010a) led most U.S.-based manufacturers to switch to shorter-chain PFASs. However, the manufacturing capacity of longer-chain PFASs has expanded in China, Russia, and India, resulting in increased environmental contamination in those countries (Liu et al. 2016; OECD 2015; Wang et al. 2013). Based on available data, U.S. EPA asserted that the import of carpets and rugs containing longer-chain PFASs is unlikely (U.S. EPA 2015a), though not impossible (Liu et al. 2016; OECD 2015; Wang et al. 2013). Carpets and rugs made from recycled material may still contain longer-chain PFASs.

Since the U.S. phaseout of PFOA, PFOS, and their precursors, most manufacturers have shifted to PFASs with six carbons in their molecule, such as perfluorohexanoic acid (PFHxA) and other shorter-chain PFAS chemistries. Fluorinated ethers and branched alternatives to longer-chain PFASs are also being synthesized, but little is known about their physicochemical or toxicological properties, or their effects on humans, biota, and the environment. Some scientists advocate for identifying "essential" and "non-essential" uses of PFASs, while developing safe alternatives and processes for those essential uses (Blum et al. 2015; Wang et al. 2017).

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[6] https://fluorocouncil.com/About-the-Fluoro-Council

2 PROPERTIES AND POTENTIAL ADVERSE IMPACTS OF THE CANDIDATE CHEMICAL AND RELATED CHEMICALS

2.1 Physicochemical Properties

Reference: CAL. CODE REGS. tit. 22, § 69503.3(a)(1)(D).

PFASs possess unique properties that contribute to their environmental persistence, global distribution, and accumulation in water, soils, plants, and animals. Compared to other organic chemicals, PFASs are very persistent in the environment and biota, partly because fluorine (F) forms the strongest single bond with carbon (Kiplinger et al. 1994). Due to its high electronegativity, the F atom pulls the shared electrons of the covalent C-F bond towards itself, which renders it partially negative and the C atom partially positive, creating a polar covalent bond. This bond requires high energy, up to 544 kJ/mol, to dissociate (Lemal 2004), compared to the 337 kJ/mol needed to separate carbon and hydrogen (Dean 1999). Consequently, PFASs are thermally, chemically, and biologically stable, and resistant to abiotic degradation such as atmospheric photo-oxidation, direct photolysis, and hydrolysis (Wang et al. 2016). Perfluoroethers are more thermally stable than other PFASs due to their strong C-O bonds (Ceretta 2013). Polymeric PFASs also have high thermal, chemical, aging, and weather resistance; and are inert to solvents, hydrocarbons, acids and bases (Banks et al. 1994). Even though bacteria and fungi can degrade organic chemicals by utilizing the carbon present in the compounds as an energy source, the F atoms in PFASs surround and shield the carbon backbone, so organisms are largely unable to fully biodegrade these chemicals (Frömel and Knepper 2010). PFASs that can degrade in the environment typically transform into smaller PFASs that do not degrade further under environmental conditions, such as PFAAs (Buck et al. 2011; D'Eon and Mabury 2011; Krafft and Riess 2015a; Wang et al. 2014a; Washington et al. 2015).

For most PFASs, physicochemical properties data are absent from the public domain. Table 1 lists those available for PFAAs. Less is known about PFECAs and PFESAs, but despite structural differences, their physicochemical properties are believed to be similar to those of their PFCA or PFSA counterparts (Gomis et al. 2015). In particular, they appear to be similarly persistent and mobile in the environment as the longer-chain PFASs (Gomis et al. 2015).

Most PFASs are solid at room temperature. Notable exceptions include 6:2 FTOH, which exists as a liquid at room temperature, and 8:2 FTOH, which exists as a solid but can sublimate and volatilize at room temperature (Concawe 2016). The boiling points of PFASs are similar to those of the homologous hydrocarbons (i.e., molecules with the same structure but only carbon-hydrogen bonds instead of carbon-fluorine bonds), except for perfluoroethers and perfluoroketones, which boil at significantly lower temperatures compared to their hydrocarbon homologues due to lower intermolecular forces (Ceretta 2013).

Most PFASs exhibit low vapor pressures, but compared to homologous hydrocarbons they tend to be similarly or somewhat more volatile, despite higher molecular weights (Krafft and Riess 2015a). Most PFAAs are semi-volatile and can adsorb onto indoor dust (Bohlin-Nizzetto et al. 2015; Dreyer et al. 2015; Haug et al. 2011; Jogsten et al. 2012; Knobeloch et al. 2012; Liu et al. 2015a; Liu et al. 2014; Strynar and Lindstrom 2008). Some of their precursors however, including FTUCAs and FTOHs, tend to be more volatile and can undergo atmospheric long-range transport (Ding and Peijnenburg 2013).

PFASs display a range of solubility in water, from sparingly soluble to miscible, though models may significantly underestimate PFAS solubility (Xiao 2017). Solubility tends to increase with decreasing carbon chain length and molecular weight (Table 1). PFCAs and PFSAs are relatively more soluble than other PFASs of similar chain length due to the hydrophilicity of the carboxylate and sulfonate groups on these molecules. Solubility of PFCAs and PFSAs tends to decrease with increasing molecular weight due to a concomitant increase in the length of the hydrophobic perfluorinated alkyl chains (Concawe 2016). Their water solubility makes these PFASs mobile in the environment and capable of undergoing long-range transport via ocean currents (Ahrens et al. 2009a; Armitage et al. 2009; Krafft and Riess 2015b; Prevedouros et al. 2006). Model results suggest, however, that PFPiAs are highly hydrophobic; therefore sediments may be an important sink for PFPiAs in the aquatic environment (Xiao 2017). PFAAs ionize in water at environmentally relevant pH, according to their negative pK_a (Gomis et al. 2015; Wang et al. 2017). For PFPAs, full deprotonation (i.e., two negative charges) occurs at a pH of 5.13-7.11 (Xiao 2017).

The bonds between the carbon-fluorine (CF_3 and CF_2) groups present in PFASs create very low critical surface energy, resulting in lipophobic and hydrophobic characteristics. This means PFASs repel both oil and water (Audenaert et al. 1999), making them surfactants useful for resisting stains from soils, liquids and grease on the surface of carpets, rugs, and other textiles. Due to their low critical surface energy, PFASs tend to accumulate and form micelles at liquid (e.g., water)/air phase boundaries (Concawe 2016; Eftaiha et al. 2012; Mason Chemical Company 2017).

Their hydrophilic and hydrophobic properties pose challenges to determining PFAS partitioning coefficients experimentally. leading to reliance on modeled data and estimates (Shoeib et al. 2004). For example, K_{ow}, which represents a chemical's partitioning behavior between octanol and water, cannot be determined experimentally for PFASs because they form a third phase in addition to octanol and water. All PFAAs listed in Table 1 except for PFBA, PFPeA, PFBS, PFBPA, and PFHxPA have modeled log K_{ow} >4, which means they are considered bioaccumulative according to the California Code of Regulations, Title 22, Division 4.5, Chapter 54, Article 5. However, K_{ow} – a common screening criterion of a neutral compound's ability to partition from water into lipid-rich tissues within an organism (Mackay and Fraser 2000) – has limited applicability to PFAAs, due to their unique properties. This is because, in general, PFASs have a low potential to accumulate in adipose (lipid) tissues, unlike most other persistent organic pollutants. Instead, PFASs are proteinophilic (protein-binding), accumulating particularly in blood, liver, stomach, kidneys, lungs, gall bladder, brain, muscle, and yolk sac tissues (Chen and Guo 2009; Greaves et al. 2012; Hebert and MacManus-Spencer 2010; Jones et al. 2003).

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Table 1. Physicochemical properties of Some PFAAs [vii]

Chemical Name	Water Solubility (20 - 25 °C) [g/L]	Melting Point [°C]	Boiling Point [°C]	Vapor Pressure [Pa]	log K_{ow} [-]	log K_{oc} [L/kg]	K_d (pH 7)	Dissociation Constant (pK_a)	Soil Adsorption Coefficient (L/kg)
PFCAs									
Perfluorobutanoic Acid (PFBA)	Miscible	-18	121	1307	2.8	1.9	--	-0.2 to 0.7	48*
Perfluoropentanoic Acid (PFPeA)	113	14*	124	1057	3.4	1.4	--	-0.06	96*
Perfluorohexanoic Acid (PFHxA)	22	14	143	457	4.1	1.9	--	-0.13	1,070*
Perfluoroheptanoic Acid (PFHpA)	4.2	30	175	158	4.7	2.2	0.4 - 1.1	-0.15	2,110*
Perfluorooctanoic Acid (PFOA)	3.4 - 9.5	37 - 60	188 - 192	4 - 1300	5.3	1.3 - 2.4	0 - 3.4	-0.16 to 3.8	1,160*
Perfluorononanoic Acid (PFNA)	9.5	59 - 66	218	1.3	5.9	2.4	2.6 - 5.9	-0.17	2,830*
Perfluorodecanoic Acid (PFDA)	9.5	77 - 88	218	0.2	6.5	2.8	2.0 - 31	-0.17	397*
Perfluoroundecanoic Acid (PFUnA)	0.004	83 - 101	160 - 230	0.1	7.2	3.3	12 - 103	-0.17	16,700*

[vii] Data excluding those marked with an asterisk are from peer-reviewed literature as summarized by Concawe (2016) Environmental fate and effects of poly and perfluoroalkyl substances (PFAS). Concawe Soil and Groundwater Taskforce, Brussels, Belgium. Prepared by ARCADIS: T. Pancras, G. Schrauwen, T. Held, K. Baker, I. Ross, H. Slenders, Report # 8/16. Accessed March 15, 2017. Concawe (www.concawe.eu) is a division of the European Petroleum Refiners Association based in Brussels, Belgium. Entries marked with an asterisk (*) were predicted using QSAR models by U.S. EPA (2017a) U.S. Environmental Protection Agency (U.S. EPA): Chemistry Dashboard. Available at: https://comptox.epa.gov/dashboard. Data are calculated and updated regularly by OPERA [OPEn (quantitative) Structure-activity Relationship Application], a standalone free and open source command line application in Matlab (Version 8.2) providing QSAR models predictions. Model validation data set may be found here: http://esc.syrres.com/interkow/EpiSuiteData.htm. Accessed November 6, 2017.

Chemical Name	Water Solubility (20 - 25 °C) [g/L]	Melting Point [°C]	Boiling Point [°C]	Vapor Pressure [Pa]	log K_{ow} [-]	log K_{oc} [L/kg]	K_d (pH 7)	Dissociation Constant (pK_a)	Soil Adsorption Coefficient (L/kg)
Perfluorododecanoic Acid (PFDoA)	0.0007	107 - 109	245	0.01	7.8	--	24 - 269	-0.17 to 0.8	85,500*
Perfluorotridecanoic Acid (PFTrDA)	0.0002	99*	249*	0.3	8.3	--	--	--	184,000*
Perfluorotetradecanoic Acid (PFTeDA)	0.00003	98*	276	0.1	8.9	--	--	--	233,000*
PFSAs									
Perfluorobutane Sulfonic Acid (PFBS)	46 - 57	76 - 84	211	631	3.9	1	--	-6.0 to -5.0	288*
Perfluorohexane Sulfonic Acid (PFHxS)	2.3	58*	231*	59	5.2	1.8	0.6 - 3.2	-6.0 to -5.0	2,300*
Perfluorooctane Sulfonic Acid (PFOS)	0.52 - 0.57	54	237* to > 400	6.7	6.4	2.5 - 3.1	0.1 - 97	-6.0 to -2.6	1,460*
Perfluorodecane Sulfonic Acid (PFDS)	0.002	--	--	0.7	7.7	3.5	--	--	19,200*
PFPAs									
Perfluorobutyl Phosphonic Acid (PFBPA)	14260	--	--	0.18	2.2	--	--	--	--
Perfluorohexyl Phosphonic Acid (PFHxPA)	515	--	--	0.04	3.5	--	--	--	--

Chemical Name	Water Solubility (20 - 25 °C) [g/L]	Melting Point [°C]	Boiling Point [°C]	Vapor Pressure [Pa]	log K_{ow} [-]	log K_{oc} [L/kg]	K_d (pH 7)	Dissociation Constant (pK_a)	Soil Adsorption Coefficient (L/kg)
Perfluorooctyl Phosphonic Acid (PFOPA)	25	96*	210*	0.01 5.7e-03*	4.7 4.8*	--	--	--	658*
Perfluorodecyl Phosphonic Acid (PFDPA)	0.5	87*	210*	0.0002 1.5e-03*	6.0 3.9*	--	--	--	18,100*
PFPiAs									
Perfluorobutyl phosphinic acid (PFBPiA)	2.3e-06 to 8.2e-03*	-9.9 to 70.1*	208 to 245*	6.8e-07 to 1.2e-02*	2.4 to 11.9*	--	--	--	1,390*
Perfluorohexyl phosphinic acid (PFHxPiA)	3.6e-07 to 3.1e-04*	-25.1 to 106*	213 to 301*	8.5e-11 to 9.6e-03*	4.4 to 15.1*	--	--	--	231,000*
Perfluorooctyl phosphinic acid (PFOPiA)	1.7e-09 to 2.8e-04*	-9.9 to 138*	206 to 353*	9.0e-13 to 1.7e-02*	7.2 to 18.6*	--	--	--	134,000*
Perfluorodecyl phosphinic acid (PFDPiA)	2.9e-08*	10.4*	265*	1.9e-09*	7.6*	--	--	--	132,000*
Perfluorododecyl phosphinic acid (PFDODPiA)	5.4e-07*	50.7*	290*	2.2e-09*	7.5*	--	--	--	133,000*

2.2 Environmental Fate and Transport

2.2.1 Environmental fate

Reference: Cal. Code Regs. tit. 22, § 69503.3(a)(1)(E).

Numerous factors, including intrinsic chemical properties combined with physical and biogeochemical processes, collectively determine the occurrence and fate of PFASs. Studies distinguish between PFAAs entering the environment from direct and indirect sources (Buck et al. 2011; Prevedouros et al. 2006). “Direct” sources refer to PFAA emissions from the life cycle of products containing PFAAs or their derivatives as ingredients, unreacted raw materials, or unintended by-products; “indirect” sources refer to PFAA formation from the degradation of precursors (OECD 2013). Monitoring data suggest that a large number of PFASs are PFAA precursors, meaning they can transform into PFAAs in the environment (Ahrens 2011).

Regardless of their sources, due to their unique physicochemical properties (see Section 2.1), PFASs released to the environment end up virtually everywhere, including air, dust, wastewater treatment plant (WWTP) effluent, biosolids, soil, inland and ocean waters, drinking water, and food (Lindstrom et al. 2011b; Wang et al. 2016). PFAAs and their precursors are found in the deep ocean and in underground aquifers, in rainwater and snow, and in pristine Arctic lakes, far from any point source (Kim and Kannan 2007; Prevedouros et al. 2006; Zhao et al. 2012). Wildlife and humans are often sinks of environmental PFASs (Cariou et al. 2015; Chang et al. 2014; Fraser et al. 2013; Giesy and Kannan 2001; Kang et al. 2016; Liu et al. 2011a; Monroy et al. 2008; OECD 2013; Olsen et al. 2007; Tao et al. 2008a; Tao et al. 2008b), as discussed in Chapter 3 of this document.

Volatile PFAA precursors, including FTOHs, are distributed globally as gases or adsorbed to airborne particulates (Dreyer et al. 2015; Liu et al. 2015a; Wang et al. 2014), and can re-volatilize back into the atmosphere from water or snow (Xie et al. 2015). PFAAs can become airborne and deposit onto water surfaces (Kim and Kannan 2007). Global distribution also occurs due to movement of oceanic currents, with PFHxA and PFBS being among the most frequently detected compounds in seawater (Zhao et al. 2012).

The following sections summarize publicly available data on the behavior of PFASs in different environmental compartments and sinks, without following a particular pathway since the transport and cycling mechanisms of all PFASs in the environment are not fully understood. The available environmental fate data are limited to PFAAs and their precursors. However, since PFAAs are used in the manufacturing of fluoropolymers and PFPEs, the following discussion can be relevant to the life cycle of all PFASs.

PFASs in air

As discussed in Section 2.1, PFASs display a wide range of airborne behaviors. Some PFASs, such as FTOHs, are relatively volatile and detected in indoor air and dust (Ericson et al. 2012; Fraser et al. 2013; Karásková et al. 2016). Release of these PFASs into air can occur during chemical manufacturing, consumer product manufacturing, product use, around WWTPs, and under landfill conditions after disposal (Ahrens 2011; Ahrens et al. 2011b), as described in more detail in Section 3.3.2. The most volatile PFASs are PFAA precursors, which can oxidize to PFAAs in the atmosphere (Ellis et al. 2004; Wallington et al. 2006). PFAAs are semi-volatile and dissociate in water, as the perfluoroalkyl chain forms micelles on the water surface and partitions into the air (Eftaiha et al. 2012; Mason Chemical Company 2017).

Once PFASs enter the air, they can be carried long distances by air currents or adsorb to particulate matter (Barber et al. 2007; Dreyer et al. 2015; Liu et al. 2015a; Shoeib et al. 2010; Wang et al. 2014), which may be directly inhaled or deposited in rain and snow (Casal et al. 2017; Sammut et al. 2017; Taniyasu et al. 2013). According to the limited publicly available data, PFASs have long atmospheric half-lives, for instance approximately 20 days for 8:2 FTOH (Ellis et al. 2003).

This potential for long-range atmospheric transport followed by deposition onto land and water has resulted in PFAS-contaminated food chains and accumulation in plants and animals, even in remote Arctic and Antarctic regions.

This potential for long-range atmospheric transport followed by deposition onto land and water has resulted in PFAS-contaminated food chains and accumulation in plants and animals, even in remote Arctic and Antarctic regions (Butt et al. 2010; Casal et al. 2017; Houde et al. 2006). Despite the U.S.-wide phaseout of longer-chain PFASs, atmospheric emissions of these compounds from offshore manufacturing could potentially still impact the United States. Trans-Pacific atmospheric transport from China leading to adverse impacts on air quality in California has previously been documented for other pollutants (Lin et al. 2014b). Considering the large number of PFASs in commerce today, their continuous air emissions, and their relatively long atmospheric half-lives, atmospheric transport is of significant concern due to the potential for widespread PFAS environmental distribution and contamination impacting humans and other organisms.

PFASs in water

PFASs enter surface water and groundwater through a variety of routes, including industrial discharges from manufacturing sites, WWTP effluent, runoff and leaching from contaminated soil or landfills, and deposition of contaminated particulates onto water bodies (see Chapters 3 and 4). Their individual solubility and partitioning behavior, combined with the physical and chemical characteristics of receiving waters, determine whether specific PFASs are found dissolved or suspended in the water column, in micelles at the air/water interface (Eftaiha et al. 2012; Mason Chemical Company 2017), or adsorbed to solid particles (Campo et al. 2016).

During the wastewater treatment process, volatile PFASs can be released into the air, while others selectively partition into biosolids and are partially removed from wastewater. WWTP effluent can have higher concentrations of PFAAs than the influent because of transformation during the treatment process (Appleman et al. 2014; Arvaniti and Stasinakis 2015). PFASs also enter surface and groundwater via landfill leachate (Ahrens et al. 2016; Banzhaf et al. 2017).

Few data on the aqueous photolysis of PFASs in water are publicly available; for instance the half-life for 8:2 FTOH is 93.2 ± 10.0 hours (Gauthier and Mabury 2005). PFAAs appear to not degrade at all under environmental conditions, even in activated sewage sludge, and can be more persistent than other legacy persistent organic pollutants such as dichlorodiphenyltricloroethane (DDT), polychlorinated biphenyls (PCBs), and dieldrin (European Commission 2017a; EWG 2003).

Like other persistent organic pollutants, oceans are considered the ultimate long-term reservoir for PFASs (Prevedouros et al. 2006), including PFAAs and their precursors (Gonzalez-Gaya et al. 2014). Marine organisms can be chronically exposed via a contaminated food chain (Ahrens et al. 2009a; Casal et al. 2017; Houde et al. 2011; Xiao 2017). Many physical and biogeochemical processes and factors collectively determine the oceanic occurrence and fate of PFASs, including distance to coastal urban industrial regions, oceanic subtropical gyres, currents, and biogeochemical processes (Gonzalez-Gaya et al. 2014). Oceanic currents can transport PFASs over long distances, resulting in PFAS contamination of water in distant pristine regions of the world, far from industrial activities (Ahrens et al. 2011a; Prevedouros et al. 2006). Snow and ice in the northern latitudes also accumulate PFASs, including the shorter-chains (Del Vento et al. 2012). Climate change resulting in snow, ice, and permafrost melt can remobilize PFASs and increase their concentrations in coastal areas (Zhao et al. 2012).

Sediments are an important sink and reservoir of PFASs in the aquatic environment (Chen et al. 2016; Pan et al. 2015). Freshwater and marine sediments can become contaminated when PFASs dissolved in water adsorb onto particulates and are deposited. PFASs exhibit a range of sediment-water partitioning coefficients that influence their environmental fate. Long-term production and use patterns for PFASs are documented in sediment cores (Codling et al. 2014).

Spatial distribution of PFASs in sediments and the occurrence of “hot spots” in sampling areas can provide evidence of specific manufacturing activities and point source discharges (Munoz et al. 2015; White et al. 2015). Monitoring studies reported in Section 3.3.1 suggest that urbanization and industrial activities contribute to PFAS accumulation in sediments, particularly in coastal regions and estuaries near urban centers. PFAS-contaminated coastal waters can be carried to deep sea regions through deep shelf water cascading events (Sanchez-Vidal et al. 2015). Persistent organic pollutants similar to PFASs have been found in ocean sediments deeper than 10,000 m, where they can bioaccumulate in marine organisms and biomagnify through food chains (Jamieson et al. 2017).

The ubiquitous presence of PFASs in surface water, groundwater, oceans, sediments, and air has also resulted in drinking water contamination, especially with the more soluble and mobile shorter-chain PFAAs (Gellrich et al. 2013). Contamination of drinking water with PFASs and irrigation of food crops with contaminated water is generally considered a major source of human exposure, as discussed in subsequent sections of this document.

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PFASs in soil

PFASs can contaminate soil via atmospheric transport followed by deposition, or directly through application of biosolids, soil conditioners, or other treatments. From soil, they can migrate into surface water or groundwater, including drinking water sources (Lilienthal et al. 2017). The fate and transport of a specific PFAS in soil depends on its physicochemical properties. Longer-chain PFASs tend to partition preferentially into soil organic matter, and shorter-chain PFASs into soil water (Ahrens et al. 2009b; Guo et al. 2015). Shorter-chain PFCAs have been observed to migrate out of soil following biosolid application, with disappearance half-lives ranging from 88 to 866 days (Venkatesan and Halden 2014). According to the study authors, the fate of these PFCAs after loss from soils could include leaching, plant uptake, and volatilization.

Publicly available data on the half-lives of PFASs in soils are scarce and vary widely across models and experiments. For example, one industry study reports a half-life of 1,200 to 1,700 years for the biodegradation of a side-chain fluoroacrylate polymer to PFOA in aerobic soil (Russell et al. 2008). Other researchers who evaluated the degradability of two side-chain fluoroacrylate polymers in saturated soil estimated half-lives of 33 to 112 years (Washington et al. 2015). Russell et al. (2010) estimated the half-life in soil of a side-chain fluorourethane polymer to be 28 to 241 years.

PFASs in biota

Plants and animals can bioaccumulate PFASs, with large inter-species differences, particularly between aquatic and terrestrial organisms (Concawe 2016). The tendency of PFAAs to bioaccumulate can partially be explained by (i) their higher affinity for phospholipids, which are major components of biological membranes, and (ii) their similarity to fatty acids, which makes them bind to proteins within the organism (Ng and Hungerbühler 2014).

The extent of bioaccumulation is traditionally measured using several different indices. The bioconcentration factor (BCF) is a measure of the amount of a contaminant found in an aquatic organism compared to the amount found in water. The biomagnification factor (BMF) compares the amount of the contaminant in the organism to that in its diet. The bioaccumulation factor (BAF) combines uptake from all sources, including water, diet, and environmental exposures (Arnot and Gobas 2006). Chemicals with BAF or BCF values >1000 or BMF >1 meet the bioaccumulation hazard trait criteria outlined in the CAL. CODE REGS. tit. 22, Division 4.5, Chapter 54, Article 5. However, these traditional measures of bioaccumulation have limited applicability when gauging the potential for PFAS exposures to cause adverse health effects (Cousins et al. 2016). Although the elimination kinetics and toxicity of some single PFASs have been experimentally determined, the ubiquity and extreme persistence of PFASs in the environment lead to continuous internal exposures to mixtures of PFASs, which have not been adequately assessed (Wang et al. 2017) (see Section 2.1).

Table 2 summarizes BCFs, BMFs, and BAFs for some PFASs with publicly available data. The BCFs of some longer-chain PFAAs approach 1,000, and a few – PFOS and perfluorohexadecanoic acid (PFHxDA) – exceed it. Animals tend to have greater concentrations of PFASs in their bodies compared to their diets. BAFs in aquatic organisms depend on the type and concentration of specific PFASs in water, and are generally proportional to the length of the carbon chain or molecular weight, with PFSAs showing higher bioaccumulation than PFCAs of the same chain length – although there is inter-species variation (Hong et al. 2015).

Once ingested via food or drinking water, some PFASs remain in the human body for years. Limited studies suggest that serum elimination half-life can range from a few days for shorter-chain PFASs (Chang et al. 2008; Nilsson et al. 2010; Olsen et al. 2007a) to several years for longer-chain PFASs (Bartell et al. 2010; Olsen et al. 2007a; Seals et al. 2011), and even 56 years for a chlorinated PFESA (Shi et al. 2016). Reported half-lives for

PFAAs and FTOHs in other organisms such as rats, mice, and monkeys are shorter than in humans, typically hours, days, or months (Butenhoff et al. 2004; Chang et al. 2008; Chang et al. 2012; Gannon et al. 2011; Ohmori et al. 2003; Sundström et al. 2012; Tatum-Gibbs et al. 2011).

Table 2. A selection of BCFs, BAFs, and BMFs for some PFASs, as cited in Concawe (2016)

Bioconcentration Factor (BCF)	
PFOS	
Bluegill	1,866 – 4,312 (Drottar et al. 2001)
Rainbow Trout	1,100 – 5,400 (Drottar et al. 2001)
Catfish and largemouth bass (Decatur, Alabama)	830 – 26,000 (Giesy and Newsted 2001)
Rainbow Trout	2,900 (liver) 3,100 (blood) (Martin et al. 2003)
PFOA	
Water breathing animals	1.8 – 8.0 (ECHA 2014)
Rainbow Trout	12 (liver), 25 (blood) (Martin et al. 2003)
PFHxDA	
Carp	4,700 – 4,800 (U.S. EPA 2009)
Perfluorooctadecanoic acid (PFODA)	
Carp	320 – 430 (U.S. EPA 2009)
Bioaccumulation Factor (BAF) (within a trophic level)	
PFOS	
Zooplankton/water	240 (Houde et al. 2008)
Mysis/water	1,200 (Houde et al. 2008)
Sculpin/water	95,000 (Houde et al. 2008)
Lake trout/water	16,000 (Houde et al. 2008)
PFOA	
Water breathing animals	0.9 – 266 (ECHA 2014)

Biomagnification Factor (BMF) (across trophic levels)	
PFOS	
Arctic cod/zooplankton (Western Canadian Arctic)	8.7 (Powley et al. 2008)
Caribou/lichen (Canada)	2.0 – 9.1 (Müller et al. 2011)
Wolf/caribou(Canada)	0.8 – 4.5 (Müller et al. 2011)
Dolphin/seatrout (2 U.S. locations)	0.9 (Houde et al. 2006)
Seatrout/pinfish (2 U.S. locations)	4.6 (Houde et al. 2006)
Walrus/clam (Eastern Arctic Food Web)	4.6 (Tomy et al. 2004)
Narwhal/Arctic cod (Eastern Arctic Food Web)	7.2 (Tomy et al. 2004)
Beluga/Arctic cod (Eastern Arctic Food Web)	8.4 (Tomy et al. 2004)
Beluga/redfish (Eastern Arctic Food Web)	4 (Tomy et al. 2004)
Polar bear/seal (Canadian Arctic)	177 (Martin et al. 2004)
PFOA	
Water breathing animals	0.02 – 7.2 (ECHA 2014)
Caribou/lichen (Canada)	0.9 – 11 (Müller et al. 2011)
Wolf/caribou (Canada)	0.9 – 3.8 (Müller et al. 2011)
Walrus/clam (Eastern Arctic Food Web)	1.8 (Tomy et al. 2004)
Narwhal/Arctic cod (Eastern Arctic Food Web)	1.6 (Tomy et al. 2004)
Beluga/Arctic cod (Eastern Arctic Food Web)	2.7 (Tomy et al. 2004)
Beluga/redfish (Eastern Arctic Food Web)	0.8 (Tomy et al. 2004)
Beluga whale/Pacific herring (Western Canadian Arctic Food Web)	1.3 (Tomy et al. 2004)
Arctic cod/marine arctic copepod (Western Canadian Arctic Food Web)	2.2 (Tomy et al. 2004)
Dolphin/seatrout (2 US locations)	1.8 (Houde et al. 2006)

Seatrout/pinfish (2 US locations)	7.2 (Houde et al. 2006)
Polar bears/ringed seal (2 US locations)	45 – 125 (Butt et al. 2008)
Polar bear/seal (Canadian Arctic)	8.6 (Martin et al. 2004)

2.2.2 Potential for the Candidate Chemical to degrade, form reaction products, or metabolize into another Candidate Chemical or a chemical that exhibits one or more hazard traits

Reference: CAL. CODE REGS. tit. 22, § 69503.3(a)(1)(G).

PFASs that degrade into PFAAs are termed PFAA precursors, and provide a significant indirect source of PFAAs to the environment (see Section 1.1 for examples of PFAA precursors). Degradation to PFAAs can occur for instance in the atmosphere, consumer products, landfills, or WWTPs; mainly via hydroxylation or photo-oxidation (Nielsen 2014; Prevedouros et al. 2006). The degradation half-life of PFAA precursors can range from days to centuries (Li et al. 2017; Nielsen 2014; Washington et al. 2015). PFAA precursors can persist in the environment and in biological compartments for various lengths of time, but eventually degrade through biotic and abiotic transformation to PFAAs, via intermediate metabolites suspected of high acute toxicity (Butt et al. 2014; D'Eon and Mabury 2007; Li and Guo 2016; Rand and Mabury 2012a; Rand and Mabury 2012b; Rand and Mabury 2013; Rand and Mabury 2014; Rand and Mabury 2016). Fluorotelomer-based compounds degrade (Phillips et al. 2007; Young et al. 2007) or undergo biotransformation into PFCAs (Chen et al. 2017; Kim et al. 2012a; Liu et al. 2010a; Liu et al. 2010b; Rand and Mabury 2014; Ruan et al. 2014; Russell et al. 2015; Zhao and Zhu 2017). The intermediates in this process include saturated and unsaturated fluorotelomer aldehydes (FTALs and FTUALs, respectively), saturated and unsaturated fluorotelomer carboxylic acids (FTCAs and FTUCAs, respectively), and n:3 acids[8] (Frömel et al. 2016; Nilsson et al. 2013; Rand et al. 2014).

Laboratory studies of fluorotelomer carboxylic acid and aldehyde intermediates indicate they are more acutely toxic than PFCAs, including PFOA. One study found that FTCAs and FTUCAs are one to five orders of magnitude more toxic to freshwater organisms than PFCAs (Phillips et al. 2007). In a human liver cell toxicity assay, FTUALs were found to be up to 200 times more toxic than FTUCAs, FTCAs, and PFCAs (Rand et al. 2014).

PFASs that degrade into PFAAs are termed PFAA precursors, and provide a significant indirect source of PFAAs to the environment. Degradation to PFAAs can occur for instance in the atmosphere, consumer products, landfills, or WWTPs.

Fluorotelomer-based side-chain fluorinated polymers can also degrade to FTOHs or other fluorotelomer compounds, with PFCAs as terminal degradation products (Washington et al. 2015). Similarly, perfluoroalkane sulfonamide-based side-chain fluorinated polymers may degrade into FASAs in the environment (Russell et al. 2008). Because of their potential to be mobilized, interact with their

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[8] These intermediates are also referred to as x:3 acids. See Buck et al. (2011) for definition.

surrounding environment, and degrade (KEMI 2015; Russell et al. 2010), side-chain fluorinated polymers are sometimes referred to as "functionalized oligomers" to distinguish them from true polymers.

2.2.3 Potential for the Candidate Chemical or its degradation products to be released into, migrate from, or distribute across environmental media, and accumulate and persist in biological or environmental compartments

Reference: CAL. CODE REGS. tit. 22, § 69503.3(b)(4)(H).

PFASs are widespread in indoor and outdoor environments, humans, and biota. They are found even in raindrops and snowflakes, in high altitude atmospheric wind currents (Taniyasu et al. 2013), and the deep sea (Sanchez-Vidal et al. 2015; Zhao et al. 2012). The accumulation of PFASs in different environmental compartments and living organisms appears to depend on their partitioning behavior, which depends in part on the perfluoroalkyl chain length and the functional group of the parent compound (EFSA 2011; Wang et al. 2017). See Section 3.3.1 for monitoring data evidencing the potential for PFASs to distribute across environmental media and accumulate in biological and environmental compartments.

2.3 Hazard Traits and Environmental or Toxicological Endpoints

Reference: CAL. CODE REGS. tit. 22, § 69503.3(a)(1)(A).

PFASs display multiple hazard traits according to the Office of Environmental Health Hazard Assessment (OEHHA)'s Green Chemistry Hazard Traits regulations (CAL. CODE REGS. tit. 22, §§ 69401 et seq.). These include toxicological hazard traits, (Articles 2 and 3), environmental hazard traits (Article 4), and exposure potential hazard traits (Article 5).

2.3.1 Exposure potential hazard traits

Environmental persistence

PFAAs are extremely persistent in the environment, with the exception of PFPiAs, which degrade into PFPAs and potentially PFCAs (Scheringer et al. 2014; Wang et al. 2016). All other PFAAs degrade significantly only under environmentally irrelevant conditions. PFHxS and C11 through C14 PFCAs are listed as very persistent (vPvB) on the European Chemicals Agency (ECHA)'s Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Candidate List of Substances of Very High Concern (SVHC); PFOA, ammonium perfluorooctanoate (APFO), PFNA, and PFDA are listed as persistent (PBT) (ECHA 2017). Limited data on some PFECAs and PFESAs suggest resistance to biodegradation (Beekman et al. 2016). PFPEs and fluoropolymers are similarly persistent, whereas PFAA precursors degrade into extremely persistent PFAAs.

Mobility in environmental media

As discussed in Section 2.2.1, PFAAs are capable of long-range transport. Depending on their physicochemical properties, such as water solubility, K_{oc}, and volatility, PFASs can be transported in the dissolved phase (e.g., by rivers and oceanic currents), in the particulate phase (e.g., via suspended sediments), or in the atmosphere.

Bioaccumulation

PFAAs tend to bioaccumulate, with elimination half-lives increasing with perfluoroalkyl chain length. In humans, known serum elimination half-lives range from three days to 56 years (see Section 2.2.1). PFPiAs may be an exception, since they can biotransform to PFPAs (Joudan et al. 2017; Yeung and Mabury 2016). ECHA's REACH Candidate List of SVHCs designates PFHxS and C11 through C14 PFCAs as very bioaccumulative (vPvB), and PFOA, APFO, PFNA, and PFDA as bioaccumulative (PBT) (ECHA 2017). PFAAs, including PFBA and PFHxA, differ in their uptake and accumulation in human tissues (Burkemper et al. 2017; Perez et al. 2013). PFASs can be transported across the brain blood barrier and accumulate in animal brain tissue (Greaves et al. 2013). Biomagnification, which is the increase in contaminant concentration higher in the food chain, has been reported for PFAAs in freshwater and marine organisms (Houde et al. 2006). Less is known about the potential for bioaccumulation or biomagnification of PFECAs and PFESAs (Beekman et al. 2016). To our knowledge, the bioaccumulation potential of other PFASs besides PFAAs has not been studied.

Lactational or transplacental transfer

PFASs, including PFAAs, non-polymeric PFAA precursors, and chlorinated polyfluorinated ether sulfonates, can undergo transplacental transfer in humans (Chen et al. 2017; Midasch et al. 2007; Yang et al. 2016; Zhang et al. 2013; Zhao et al. 2017). Lactational transfer has also been documented for PFAAs in humans (Karrman et al. 2007; Llorca et al. 2010; Mogensen et al. 2015; Mondal et al. 2014; Tao et al. 2008b), but to our knowledge has not been studied for PFAA precursors or other PFASs. Transplacental and lactational transfer can be significant routes of exposure for newborns (Cariou et al. 2015; Papadopoulou et al. 2016; Winkens et al. 2017).

Global warming potential

Some PFPEs, which are increasingly used as replacements for PFAAs or their precursors, may have high global warming potential. The global warming potential of perfluoropolymethylisopropyl ether, a type of PFPEs, ranges from 7,620 over 20 years to 12,400 over 500 years, relative to CO_2 (IPCC 2007). Some non-polymeric fluorinated ethers, which can be used in the production of PFPEs and released to the environment, also have high global warming potential (ranging, relative to CO_2, from 207 to 13,800 over 20 years, from 59 to 14,900 over 100 years, and from 18 to 8,490 over 500 years) (IPCC 2007). Additionally, CHF_3, with a global warming potential of 11,700 to 14,800 relative to CO_2 over 100 years (IPCC 2007), is a degradation product of fluorinated polymers from incomplete combustion (Huber et al. 2009). Other PFASs ubiquitous in the ocean and transferred onto sea spray aerosols can significantly increase the concentration of cloud condensation nuclei, thus increasing cloud albedo or reflectance. This increases the efficiency of solar radiation absorption in the atmosphere, contributing to global warming (MacLeod et al. 2014).

2.3.2 Toxicological hazard traits

Appendix 3 lists toxicological hazard traits for the PFASs with publicly available data (mostly PFAAs and some precursors).

A systematic review of 64 epidemiological studies that assessed children's exposure to PFASs and associations with specific health outcomes found positive correlations between PFAS exposure and suppressed immune system response, dyslipidemia, impaired kidney function, and delayed menarche.

Potential adverse human health effects from PFAS exposure include:

- increased serum cholesterol (Skuladottir et al. 2015; Winquist and Steenland 2014);
- thyroid disease (Winquist and Steenland 2014);
- immune dysregulation (Grandjean and Clapp 2014; Grandjean et al. 2016; Grandjean et al. 2017);
- pregnancy-induced hypertension (C8 Science Panel 2011); and
- kidney and testicular cancers (C8 Science Panel 2012).

Epidemiological studies suggest a link to adverse health effects, but often entail uncertainties in exposure or confounding factors. A systematic review of 64 epidemiological studies that assessed children's exposure to PFASs and associations with specific health outcomes found positive correlations between PFAS exposure and suppressed immune system response, dyslipidemia, impaired kidney function, and delayed menarche (Rappazzo et al. 2017). PFOA also appears to be associated with low birth weight in humans (Malits et al. 2017).

Possibly due to their shorter observed biological half-lives, shorter-chain PFASs were expected to have decreased toxicity compared to the longer-chains (DeWitt 2015; Stahl et al. 2011; Wolf et al. 2008). Notable exceptions are PFHxS (C6) and PFBS (C4) in some toxicokinetic studies and receptor binding assays (Danish Environmental Protection Agency 2015a; Wolf et al. 2008). PFBS was reported to be developmentally toxic in zebrafish (Hagenaars et al. 2011). In rodents, delayed pupil response (Butenhoff et al. 2012) [and retinal degeneration][9] (3M 2006) may indicate a potential for ocular toxicity in humans. Rodent studies have also identified reproductive and developmental (fetal resorption, delayed eye opening) (Das et al. 2008), hematotoxic (reduced RBC, hemoglobin, and hematocrit) (Lieder et al. 2009), and respiratory (nasal degeneration) (Loveless et al. 2009) hazards. Neurodevelopmental toxicity has been demonstrated *in vitro* by dose-dependent suppression of neuronal differentiation (Slotkin et al. 2008).

Scientific review of the limited data on the PFECA GenX (a PFOA replacement in some applications) and related perfluorinated ether substances from Chemours (Beekman et al. 2016) indicates potential associations with

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[9] Unpublished data mentioned in the cover letter accompanying a 3M submission to the TSCA 8(e) docket.

adverse health effects in laboratory animals, including cancer, body weight gain, changes to the immune system and cholesterol levels, increased weights of kidneys and livers, and liver cell changes (Rae et al. 2015).

When differences in rodent toxicokinetics are taken into consideration however, PFECAs and shorter-chain PFAAs may have similar or higher toxic potency than the longer-chain PFAAs they are replacing. Using a toxicokinetic model and existing toxicity data sets, a recent study found that PFBA, PFHxA, and PFOA have the same potency to induce increased liver weight, whereas GenX is more potent (Gomis et al. 2018). The authors concluded that previous findings of lower toxicity of fluorinated alternatives in rats were primarily due to the faster elimination rates and lower distribution to the liver compared to PFOA and other longer-chain PFAAs.

Less is known about the toxicity of most PFAA precursors. Laboratory studies of fluorotelomer carboxylic intermediates indicate that they are more acutely toxic than PFCAs in non-mammalian systems (Phillips et al. 2007; Rand et al. 2014). Industry studies report increased mortality for 6:2 FTOH (O'Connor et al. 2014), and liver and kidney toxicity for 8:2 FTOH (Ladics et al. 2008), at the highest doses only. Other FTOH studies have found a potential for endocrine disruption, interfering with thyroid and sex steroid hormone systems (Ishibashi et al. 2008; Rosenmai et al. 2016; Weiss et al. 2009).

Activation of the nuclear peroxisome proliferation-activated (PPAR alpha) receptor is hypothesized as a mode of action that causes adverse health effects from PFAS exposure, but other biological interactions may be at play that have not yet been identified (Guyton et al. 2009; Rappazzo et al. 2017). PFASs have been shown to bind competitively to the human thyroid hormone transport protein (Weiss et al. 2009), and thus may affect thyroid hormone levels and early life brain development (Winkens et al. 2017).

2.3.3 Environmental hazard traits

PFAS toxicity in aquatic organisms – as a hazard trait – has been reported across a broad range of species (microorganisms, algae, plants, invertebrates, amphibians, fish, marine mammals) and adverse impacts (e.g., impaired growth, mortality, developmental effects, and reproductive effects). However, the environmental effects on terrestrial wildlife are not as well studied. See Appendix 3 for details.

A 2011 study testing the structure-activity relationship of PFOA, PFOS, PFBS, and PFBA in fish development found that all four PFASs were teratogens, leading to malformations of the tail and an uninflated swim bladder, causing fish to swim abnormally; exposure to PFBS and PFOS also resulted in fish head malformations (Hagenaars et al. 2011). Swimming and buoyancy are necessary for fish to compete for food and escape predators.

Studies in zebrafish embryos found higher potential for adverse developmental effects for PFASs with sulfonic groups, such as PFBS and PFOS (Ulhaq et al. 2013), and potential endocrine disrupting effects for 6:2 FTOH (Liu et al. 2009). Studies show increased acute toxicity in microalgae and zebra fish embryos with increasing chain length (Latala et al. 2009; Ulhaq et al. 2013). A review of fish toxicity data concluded that shorter-chain PFASs have moderate to low acute toxicity at exposures of less than seven days; data on longer exposures are unavailable (Danish Environmental Protection Agency 2015a).

2.4 Related Chemicals and Their Adverse Impacts

Reference: Cal. Code Regs. tit. 22, § 69503.3(a)(1)(C).

Individual PFASs rarely occur in isolation. People and biota are exposed to mixtures of PFASs, potentially leading to cumulative adverse impacts. Although simultaneous exposure to different PFASs may result in additive effects, only a few studies have investigated the toxicity of PFAS mixtures, with varying results. For instance, a cumulative health risk assessment of seventeen PFASs in the Swedish population concluded there was a threat of hepatotoxicity or reproductive toxicity in a subpopulation that routinely consumed PFOS-contaminated fish, but not in the general public (Borg et al. 2013).

Exposure to PFAS mixtures may exert greater toxicity than exposure to a single PFAS (Wang et al. 2011). Evidence of increased mixture toxicity includes results from an *in vitro* study in which binary combinations of PFAAs at low concentrations behaved additively in activating the PPAR alpha receptor (Wolf et al. 2014). A cytotoxicity assay found that a mixture of PFASs altered the cellular lipid pattern of human placental cells at levels below those that induce toxic effects (Gorrochategui et al. 2014). An *in vitro* study of PFOA, PFOS, and PFNA found that toxicity in a human macrophage cell line and acute toxicity in zebrafish were greater for mixtures than individual compounds (Rainieri et al. 2017).

Mixture toxicity studies of PFASs and other toxicants are limited. The results include:

- cumulative adverse effects *in vitro* of PFOS, PFOA, and several other contaminants (Hg^{2+}, Cd^{2+}, 2,4-D, propylparaben, mitomycin C, and furazolidone) in a bioluminescent cyanobacterial toxicity test (Rodea-Palomares et al. 2012);
- cumulative neurotoxic and adverse behavioral effects of PFHxS, endosulfan, cypermethrin, chlorpyrifos or carbaryl in mice after a single exposure, even at dosages where the individual chemicals did not elicit a significant adverse impact (Lee 2015);
- increased severity of oxidative stress and apoptosis to zebrafish embryos following exposure to PFOS and zinc oxide nanoparticles (Du et al. 2017); and
- adverse impacts on mitochondrial function in juvenile Chinook salmon after exposure to a mixture of contaminants of emerging concern, including several PFASs, pharmaceuticals, and personal care products, at environmentally-relevant concentrations (Yeh et al. 2017).

2.5 Specific Populations That May Be Harmed by the Candidate Chemical

2.5.1 Human populations or aquatic, avian, or terrestrial animal or plant organisms for which the Candidate Chemical has the potential to contribute to or cause adverse impacts

Reference: Cal. Code Regs. tit. 22, § 69503.3(a)(1)(F).

Due to their widespread presence in the environment and biota, PFASs may contribute to or cause adverse impacts in all humans and other organisms. A recent review paper (Krafft and Riess 2015b) states that:

"Because of ubiquitous PFAS presence, there is no unexposed control population and many studies compare outcome data for high-dose sub-groups with a low-dose sub-group within the same population, a practice that can weaken dose-response correlations."

Wildlife, particularly apex predators, could also be vulnerable to adverse effects from chronic exposure to PFASs in food and water (Tartu et al. 2017; Tipton et al. 2017). The potential for adverse impacts of PFASs on aquatic species has been well-documented, with toxic effects noted in aquatic plants (Ding and Peijnenburg 2013; Ding et al. 2012a; Hoke et al. 2012) and widespread bioaccumulation in aquatic animals. Fish-eating birds, including eagles and ospreys, are especially vulnerable to PFAS exposure via food chain contamination (Giesy et al. 2010). Adverse impacts to other birds have also been found, including reduced chicken embryo survival (Norden et al. 2016). Shorter-chain PFASs generally show increased uptake by leafy plants compared to the longer-chains (Blaine et al. 2014), though longer-chains may preferentially accumulate in roots (Zhao et al. 2017). Less is known about potential impacts to terrestrial animals and plants, especially at the population level.

2.5.2 Potential impacts to sensitive subpopulations, environmentally sensitive habitats, endangered and threatened species, or impaired California environments

Reference: CAL. CODE REGS. tit. 22, §§ 69503.3(a)(1)(F) and 69503.3(a)(2).

Infants, toddlers, and small children comprise a sensitive subpopulation because of their increased ingestion and inhalation rates per unit of body weight, rapid development, immature physiological ability to detoxify environmental contaminants, and behavioral characteristics that predispose them to increased exposures to environmental contaminants (U.S. EPA 2011). This results in a higher body burden of PFASs as compared to adults (Rappazzo et al. 2017). Breasted infants are susceptible to increased exposures to PFASs in breast milk, because breastfeeding is a route of PFAS excretion for lactating women (Kang et al. 2016; Karrman et al. 2007; Mogensen et al. 2015; Mondal et al. 2014). Infants, toddlers, and small children often have increased exposures due to hand-mouth behaviors that can lead to increased ingestion of dust and soil with environmental contaminants, and higher cumulative exposure doses relative to bodyweight compared to adults (U.S. EPA 2011). Pregnant women and fetuses are also sensitive subpopulations because of transplacental migration and the vulnerability of the rapidly developing fetus (Slotkin et al. 2008). Table 3 lists the main routes of PFAS exposure for children.

Individuals with certain preexisting conditions such as elevated serum cholesterol, high blood pressure, and compromised kidney function may be unusually sensitive to PFAS exposure (ATSDR 2015). Workers in the carpet industry, including retail workers, also comprise a sensitive subpopulation because of their potentially higher exposure, particularly to volatile PFASs emitted from carpets and rugs. Studies demonstrate substantial human exposure in occupational settings and communities relying on contaminated drinking water sources (Heydebreck et al. 2016). Over 44 million residents utilize private wells for drinking water and may be disproportionately impacted by PFAS-contamination of groundwater. However, U.S. EPA did not monitor private wells for contamination (Hu et al. 2016). People living near PFAS chemical manufacturers, or in other areas contaminated by PFASs, can have higher-than-average levels of PFASs (Fromme et al. 2009). Residents of low-income communities may also be disproportionately exposed to hazardous chemicals and experience greater cumulative adverse impacts from these exposures. Manufacturing facilities, including those that release PFASs, are often located in these communities. As a result, residents may be chronically exposed to multiple industrial chemicals concurrently, increasing the likelihood of adverse health effects (U.S. EPA 2016d). Environmental justice concerns also arise regarding contamination in areas far from manufacturing sites, such as the remote regions of the Arctic. For instance, the traditional diet of the Nuuk Inuit of Greenland and the Faroese includes foods heavily contaminated with PFASs that originated from thousands of miles away (Grandjean et al. 2012; Grandjean et al. 2016; Long et al. 2012).

Table 3: Prenatal and early-childhood PFAS exposure routes[10]

Exposure Pathway	Basis
Transplacental migration	Exposure to PFASs begins before birth. Transplacental passage can be a significant route of human exposure to PFAAs (Kim et al. 2011) and their precursors (Yang et al. 2016). Transfer to the fetus is one of the major PFAS elimination routes for women, particularly for PFOA (Lee et al. 2013).
Ingestion – Breast Milk	Breast milk ingestion is a significant route of PFAS exposure for infants (Mondal et al. 2014). Infants fed breast milk may have higher PFAS dietary exposure than those fed infant formula (Fromme et al. 2010).
Ingestion – Food	Food ingestion is considered a primary route of exposure for the general population, with higher relative exposures in infants, toddlers, and small children because of their low body weight (Egeghy and Lorber 2011). Due to widespread contamination, PFASs are found in a variety of animal and plant food (Blaine et al. 2014; Perez et al. 2014). PFASs are readily absorbed after ingestion (ATSDR 2015; Danish Environmental Protection Agency 2015a).
Ingestion – Drinking water	Numerous studies conclude that drinking water is a major source of PFAS intake (DeWitt 2015; Trudel et al. 2008). In 2016, U.S. EPA issued drinking water health advisories for PFOA and PFOS of 70 ng/L (combined concentrations), to protect the most sensitive populations – fetuses during pregnancy and breastfed infants (U.S. EPA 2016a). Some states have adopted, or are considering adopting, lower limits.
Inhalation and ingestion of dust	PFASs are widely found in house dust, with higher concentrations in homes with treated carpets (Haug et al. 2011). Exposure via dust is higher in toddlers and small children than adults, and can be significant due to children's lower body weights, increased inhalation rate, higher dust ingestion rates, increased floor contact and hand-to-mouth behavior (Mercier et al. 2011; Tian et al. 2016). PFASs are nearly completely absorbed following inhalation and ingestion (ATSDR 2015).

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[10] Protecting children, a sensitive subpopulation, is one of the Policy Priorities identified in SCP's 2015-2017 Work Plan. Childhood is a life stage that all members of a population experience, although children constitute a population subgroup when evaluating exposures at a specific time.

California’s endangered and threatened species could be adversely affected by exposure to PFASs associated with carpet and rug treatments, especially considering the adverse effects on reproduction and development demonstrated for some PFAAs. This could contribute to the current biodiversity crisis in aquatic ecosystems (Abell 2002; Mora and Sale 2011; Valentini et al. 2016). Threatened and endangered species of fish and marine mammals are particularly vulnerable to population-level adverse impacts if they cannot obtain food or avoid predators. Mammals and birds occupying the highest trophic level, including orcas, wolves, grizzly bears, eagles and condors, are vulnerable to adverse health effects from ingestion of food and water contaminated with PFASs that bioaccumulate and biomagnify (Kannan et al. 2006; Kelly et al. 2009).

Environmentally sensitive habitats in California, including estuaries and other wetlands, can receive surface water contaminated with PFASs via wastewater plant effluent or surface runoff, as noted in Section 3.3.2, leading to contaminated water and sediments, as noted in Section 3.3.1. These ecosystems are important breeding, spawning, and nesting sites, and feeding grounds for millions of migratory birds – including threatened and endangered species – that transit California during their annual migration.

3 FACTORS RELATED TO POTENTIAL EXPOSURE TO THE CANDIDATE CHEMICAL IN THE PRIORITY PRODUCT

3.1 Presence and Use Patterns of the Product

3.1.1 Market presence of the product

Reference: CAL. CODE REGS. tit. 22, §§ 69503.3(b)(1)(A) and (B).

Carpets and rugs are the most commonly used floor covering product in the United States. In 2016, they accounted for approximately 42 percent ($8.78 billion) and 12 percent ($2.5 billion) of the $21.2 billion U.S. flooring products market, respectively (Floor Covering News 2017; Ryan 2017). Of 2016 carpet sales, 44.9 percent ($3.94 billion) were for commercial spaces, 46.7 percent ($4.10 billion) for residential replacement, 7.4 percent ($0.65 billion) for new residential uses, and one percent ($0.09 billion) for other uses (Ryan 2017).

In 2016, carpets and rugs accounted for 58.8 percent of flooring industry in volume, with 11.22 billion square feet sold in the United States. An estimated 94.1 million square yards of carpet were sold in California in 2016 (CCSP 2016). According to the Carpet and Rug Institute, the industry trade group representing 90 percent of U.S. carpet manufacturers, “most residential and commercial carpets are treated” with PFAS-based stain- and soil-repellents (Yarbrough 2017).

3.1.2 Intended use of the product

Reference: CAL. CODE REGS. tit. 22, §§ 69503.3(b)(1)(C) and 69503.3(b)(4)(D)1.

Some carpets are designed for residential use, others for office spaces and other commercial applications. Depending on the type of carpet, performance lifetime ranges from less than ten to more than 25 years (Arcuri 2015). The sheer volume of carpets and rugs sold annually represents a huge potential source of daily exposure to PFASs in homes, workplaces, and other commercial spaces.

3.1.3 Household and workplace presence of the product and other products containing the Candidate Chemical, and aggregate effects

Reference: CAL. CODE REGS. tit. 22, §§ 69503.3(a)(1)(B) and 69503.3(b)(3).

Californians are exposed to PFASs from a variety of sources. Carpets and rugs are found in many indoor spaces, including residential, office, academic, and commercial buildings. Most have been treated with PFASs to resist water, stains, and dirt (see Section 3.1.1). PFASs are found in many frequently-used consumer products in households and workplaces, including flooring, furniture, clothing, cookware, food packaging, cleaning products, personal care products, and electronics (KEMI 2015), as well as in food (Christensen et al. 2017) and drinking water (Schwanz et al. 2016) (see Section 3.3.1). This can result in multiple source exposure to complex PFAS mixtures, including through other consumer products besides carpets and rugs (Becanova et al. 2016) . These other potential exposure sources are also of interest to DTSC.

3.2 Populations That May Be Exposed to the Candidate Chemical through Release from the Product

3.2.1 Targeted customer base

Reference: CAL. CODE REGS. tit. 22, § 69503.3(b)(1).

Because of the prevalence of stain- and soil-repellent carpets and rugs in residential and commercial spaces, all Californians are potentially exposed, directly or indirectly, to PFASs from these products. U.S. Census data from 2012 identifies more than 850 stores in California selling carpets and rugs, representing a potential source of increased exposure to PFASs for thousands of retail workers (U.S. Census Bureau 2017).[11] The U.S. Bureau of Labor Statistics reported in 2016 that California led the nation in the number of carpet installers employed in that sector, with 5,270 workers (U.S. BLS 2016).

Because of the prevalence of stain- and soil-repellent carpets and rugs in residential and commercial spaces, all Californians are potentially exposed, directly or indirectly, to PFASs from these products.

3.2.2 Potential exposure to the Candidate Chemical during the product's life cycle, considering uses by sensitive subpopulations, workers, customers, clients, and the general public who use the product at home, schools, workplaces, or other locations

Reference: CAL. CODE REGS. tit. 22, § 69503.3(b)(4)(D).

PFASs from carpets and rugs can be found in home and office air samples, and in the blood of residents and office workers (Fraser et al. 2012; Fraser et al. 2013). Compared to outdoor air, indoor air can have >1,000 times higher levels of FTOHs (Fraser et al. 2012; Müller et al. 2012). Carpets and rugs often contain FTOHs as manufacturing impurities or intermediate degradation products (Herzke et al. 2012; Kotthoff et al. 2015; Liu et al. 2015b; Vestergren et al. 2015). Carpet installation can result in significantly higher concentrations of PFAAs in indoor compared to outdoor air (Gewurtz et al. 2009). During normal product use, surface abrasion can release PFAAs from treated carpet and rug fibers via tiny particulates, which can become resuspended (Rosati et al. 2008). Human exposure to PFASs can occur via inhalation of PFAS-containing fine particulates and ingestion of

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[11] We performed an advanced search by Industry Codes using the 2012 NAICS code "442210: Floor covering stores," then refined the search results by limiting them to California, and selecting the document ID EC1244SLLS11 ("Retail Trade: Subject Series – Product Lines: Product Lines Statistics by Industry for the U.S. and States: 2012"). The table lists 852 establishments under Products and Services Codes 20365 (soft-surface (textile) floor coverings to be installed)," and 641 establishments under Products and Services Code 20366 (carpets & rugs not requiring installation).

household and office dust, which concentrates PFASs (Fraser et al. 2012; Harrad et al. 2010; Haug et al. 2011; Tian et al. 2016).

Knobeloch et al. (2012) measured PFASs in vacuum dust samples from 39 homes, finding correlations between the amount of PFNA and PFUnA in dust and the presence of carpeting. Later, Karásková et al. (2016) measured 20 PFASs in dust from homes and found that exposure doses via dust ingestion were significantly higher in toddlers than adults. This indicates an increased potential for adverse impacts in this vulnerable subgroup.

Regarding inhalation exposures, Fraser et al. (2012) found that FTOH concentrations in office air can predict PFOA concentrations in the blood of workers. Carpet used in institutional settings may contain higher amounts of PFASs (Gewurtz et al. 2009), presumably due to its use in high traffic areas.

Carpet stores have higher PFAA concentrations in air than other locations (Gewurtz et al. 2009). As a result, people working in the carpet manufacturing and retail sector, including store workers, carpet installers, and carpet cleaners, may be disproportionately impacted.

3.3 Actual or Potential Exposures to the Candidate Chemical in the Product

3.3.1 Occurrence or potential occurrence of exposure to the Candidate Chemical in the product

Reference: C*AL.* C*ODE* R*EGS. tit. 22, § 69503.3(b)(2).*

PFASs are found ubiquitously in the environment, plants, animals, and humans (Campo et al. 2016; Lindstrom et al. 2011a; Lindstrom et al. 2011b). Carpets and rugs contribute to widespread environmental contamination and exposures, as do other consumer products such as food packaging, cosmetics, and waterproof clothing. Once released to the environment during product manufacture, use, or disposal, PFASs become part of a virtually closed cycle leading to chronic, lifelong human and ecological exposures (Figure 2). Because PFAAs and other persistent PFASs lack a natural degradation route, their levels in the environment, humans, and biota may continue to rise for as long as PFASs are used in consumer products.

Between 2009 and 2017, 455 new PFASs have been detected in environmental media and commercial products. However, most of the PFASs that contribute to total organic fluorine in the environment, wildlife, and human blood samples remain unidentified (Xiao 2017). Thus, only a small fraction (sometimes less than five percent) of the PFASs in these media are likely reflected in the data summarized in this section. The full extent of the contamination, despite extensive research, remains poorly understood.

Once released to the environment during product manufacture, use, or disposal, PFASs become part of a virtually closed cycle leading to chronic, lifelong human and ecological exposures. Because PFAAs and other persistent PFASs lack a natural degradation route, their levels in the environment, humans, and biota may continue to rise for as long as PFASs are used in consumer products.

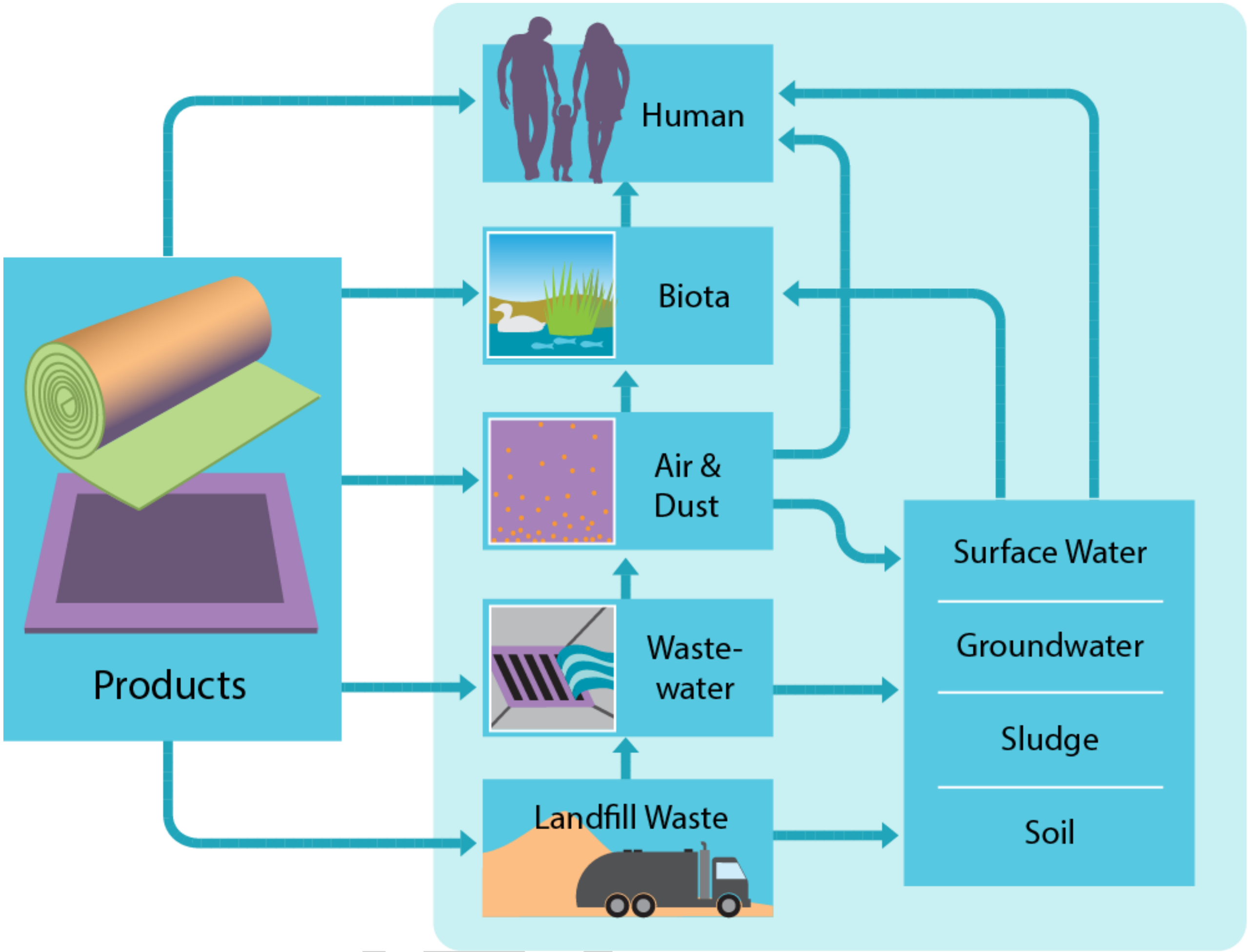

Figure 2: Key routes of PFAS exposure from treated carpets and rugs.

Evidence that the Candidate Chemical is present in or released from the product

Accurate information on the current production volume of PFASs used in domestically-produced and imported carpets and rugs is lacking, although Paul et al. (2009) estimated that a large percentage produced globally is used by the carpet industry. The amount of PFASs in treated carpets and rugs varies widely, depending on the carpet and its age. Stain- and soil-repellents can contribute approximately fifteen percent of fiber weight in synthetic carpets (KEMI 2015).

According to the FluoroCouncil (Bowman 2017a; FluoroCouncil 2017b), only polymeric PFASs are used as carpet treatments. While the majority are side-chain fluorinated polymers, according to a patent filed on January 31, 2013 by INVISTA North America S.a.r.l. (Iverson et al. 2017), a “suitable fluorochemical may be derived from any of the classes specific to fluorinated matter including fluoropolymers, perfluoropolyethers (PFPEs), and side-chain-fluorinated polymers.”

Although only polymeric PFASs are currently used in the treatment of U.S. carpet and rugs, non-polymeric PFASs can be present as manufacturing impurities or degradation products (Kotthoff et al. 2015; Vestergren et al. 2015). For example, carpets purchased in the United States in 2011 and 2013 were found to contain up to 1,300 ng/g 6:2 FTOH, 1,500 ng/g 8:2 FTOH, and 1,210 ng/g 10:2 FTOH (Liu et al. 2015b). FTOHs, used as manufacturing intermediates, are also impurities or degradation intermediates of side-chain fluorinated polymers and can off-gas from fluorotelomer-based products (Dinglasan-Panlilio and Mabury 2006; Sinclair et al. 2007). An analysis of the sum total of PFAAs found in carpet samples detected a wide range of concentrations, with one carpet used in institutional settings having the highest sum of fourteen PFASs (Gewurtz et al. 2009), presumably due to its use in high traffic areas.

Monitoring data showing that the Candidate Chemical is widespread in the indoor and outdoor environment and accumulates in terrestrial and aquatic organisms

Numerous published studies have found that PFASs are ubiquitous in various types of environmental media, in biota, and in the indoor environment. A whole suite of PFAAs and precursors, including FTOHs, FTOs, FTCAs, FTUCAs, FASAs, FASEs, FTSs, fluorotelomer phosphate diesters (diPAPs), perfluoroalkyl iodides (PFAIs), FTIs, and perfluoroalkyl sulfinates (PFSIs) were found in various matrices such as air, WWTP effluent and sludge, landfill leachate, seawater, rivers, lakes, rain, snow, ice, and soil [see (Frömel et al. 2016) and Table 4 for examples].

Air and dust: PFASs are ubiquitous in air and dust. A review and analysis of literature data published after 2010 concluded that neutral PFASs, mainly FTOHs, FASAs, and FASEs are most dominant in indoor air due to their low water solubility and high vapor pressure (Jian et al. 2017). 6:2, 8:2, and 10:2 FTOHs have been detected in indoor and outdoor ambient air (Jahnke et al. 2007; Karásková et al. 2016; Kim and Kannan 2007; Liu et al. 2013; Shoeib et al. 2011; Tian et al. 2016). The estimated atmospheric lifetime of FTOHs indicates that atmospheric transport can contribute to far-field human exposures and environmental burdens (Ellis et al. 2003). Human exposure to PFAAs, FASAs, FOSEs, and other PFASs can also occur through house dust (Jian et al. 2017), which can have significantly higher PFAS levels than background concentrations in urban soils (Tian et al. 2016; Xu et al. 2013). PFAA precursors in air and dust were observed to correlate with PFAA levels in human serum (Makey et al. 2017). Dust contains various classes of chemicals with hazard traits similar to PFASs, such as flame retardants, phthalates, and environmental phenols (Mitro et al. 2016). Because Americans spend, on average, more than 90 percent of their time indoors (Klepeis et al. 2001), ingestion and inhalation of indoor dust represent potentially significant exposure pathways to multiple contaminants along with PFASs.

Surface water and groundwater: PFASs have been detected in creeks and rivers (D'Eon et al. 2009), lakes (De Silva et al. 2011), oceans (Benskin et al. 2012b), and groundwater (Eschauzier et al. 2013; Xiao et al. 2015). Surface waters can contain high levels of PFASs, especially downstream of industrial facilities such as carpet manufacturing sites (Konwick et al. 2008). In the raw water of a drinking water treatment plant in the Cape Fear River watershed, North Carolina, Sun et al. (2016) found the PFECA GenX, a PFOA replacement in fluoropolymer manufacturing, at very high levels (631 ng/L on average, max ~4,500 ng/L). In fresh water and coastal water, PFHxA and PFHpA have been reported at levels comparable to or higher than PFOA (Cousins et al. 2011). PFPAs have also been detected in surface water at levels ranging from 0.088 to 3.4 ng/L (D'Eon et al. 2009). In the San Francisco Bay Area of California, PFASs were detected in bay water (Sedlak et al. 2017) and groundwater, with concentrations ranging from 19 to 192 ng/L (PFOS), <LOD to 22 ng/L (PFOA), and <20 ng/L (PFHxS, PFDS, PFDA, and two PFOS precursors) (Plumlee et al. 2008). Contaminated surface waters can be directly ingested by livestock and wild animals, leading to exposure up the food chain. PFAS contamination extends to drinking water sources, as discussed below.

WWTP effluent: PFASs are commonly found in municipal and industrial wastewaters (Appleman et al. 2014; Arvaniti et al. 2015; Chen et al. 2017; Clara et al. 2008; Sun et al. 2012). Industrial wastewater can have PFOA and PFOS levels above 1,000 ng/L (Kim et al. 2012b; Lin et al. 2014a). PFPAs have also been detected in WWTP effluent at levels up to 6.5 ± 1.1 ng/L (D'Eon et al. 2009). WWTPs are major point sources for PFAS contamination of aquatic environments (Ahrens 2011; Hamid and Li 2016). Degradation of PFAA precursors, such as FTOHs, within WWTPs can lead to increases in effluent PFAA concentrations (Ahrens 2011; Frömel et al. 2016; Guerra et al. 2014). Despite the phaseout of PFOS- and PFOA-containing stain- and soil-repellent carpet and rug treatments, these PFASs are frequently detected in wastewater, indicating their ongoing release into the environment (Guerra et al. 2014). A study of effluent from WWTPs in the San Francisco Bay Area, detected several PFAAs including PFHxA, PFOA, PFBA, and PFOS with highest median effluent concentrations of 24, 23, 19 and 15 ng/L, respectively (Houtz et al. 2016). A subsequent study (Sedlak et al. 2017) of three San Francisco Bay WWTPs found mean PFOA, PFOS, PFHxA, and PFPeA concentrations as high as 39.7, 42.3, 30.1, and 21.2 ng/L, respectively.

Landfill leachate: PFASs are commonly detected in landfill leachate around the world (Benskin et al. 2012a; Fuertes et al. 2017; Gallen et al. 2017; Hamid et al. 2018). A study of 95 leachate samples from eighteen U.S. landfills estimated the annual release of PFCAs, FTCAs, and PFSAs and precursors to be 291 kg/yr, 285 kg/yr, and 84 kg/yr, respectively (Lang et al. 2017). A Swedish study of 26 PFASs in samples from groundwater, surface water, WWTP effluent, and landfill leachate found landfill leachates had the highest average total PFASs concentrations (487 ng/L) (Ahrens et al. 2016). Shorter-chain PFAAs tend to be the most abundant PFASs in landfill leachate (Hamid et al. 2018).

Soils: PFASs are ubiquitous in soils. A survey of soils around the world, including remote locations such as Antarctica, found PFASs in all samples (Rankin et al. 2016). Side-chain fluorinated polymers were detected in all biosolid-treated soil samples measured in a Canadian study (Chu and Letcher 2017). Soil and water contaminated with PFASs can lead to plant and soil organism uptake and transfer to grazing animals potentially consumed by humans (Navarro et al. 2017; Yoo et al. 2011). Some plant species show a very high PFAS accumulation potential, with higher BCFs for shorter-chain PFASs. For example, the BCF (plant/soil ratio) of 6:2 FTSA in birch leaves can be up to 143,700 (Gobelius 2016). Based on their ability to uptake and sequester PFASs in above-ground biomass, some plants have been proposed for use in the phytoremediation of PFAS-contaminated soil (Gobelius 2016).

Sediments: Sediments can also become contaminated. White et al. (2015) analyzed PFASs in estuarine sediments from the Charleston Harbor and the Ashley and Cooper Rivers (n=36) in South Carolina, finding higher concentrations of eleven PFASs than previously reported for U.S. urban areas. The PFASs were dominated by PFOS, followed by PFDA and PFOA (White et al. 2015). Eighteen PFCAs and PFSAs were detected in the archived surface sediments of five major rivers in China (Pan et al. 2014a). Similar to other studies of sediments from lakes, reservoirs, and estuaries, the most frequently detected PFASs were PFOA and PFOS (detection frequency of 100 and 83 percent, respectively), with greater concentrations in urban areas, consistent with other studies (Pan et al. 2014b). PFPAs and PFPiAs have been measured in lake sediments, but in smaller concentrations compared to other PFAAs (Guo et al. 2016). In San Francisco Bay sediments, PFPiAs were detected at concentrations ranging between 8.75 and 14.2 ng/g (Sedlak et al. 2017). Concentrations of PFAAs, including PFHxA and PFBA, have been found at higher concentrations at ocean depths than at the surface, indicating the potential for deep ocean sediments to serve as the ultimate sink for PFAAs that adsorb to particulates (Prevedouros et al. 2006; Sanchez-Vidal et al. 2015).

Biota: PFAAs are found in numerous marine species, including plankton (Casal et al. 2017), sea turtles (Keller et al. 2005; Keller et al. 2012; O'Connell et al. 2010), seals (Routti et al. 2016), California sea otters (Kannan et al. 2006), whales (Hart et al. 2008), fish (Wong et al. 2017), sharks (Kumar et al. 2009), polar bears (Dietz et al. 2008), dolphins (Adams et al. 2008; Houde et al. 2005), and marine bird eggs (Verreault et al. 2007); this indicates potential for widespread adverse impacts throughout the marine food web. PFPiAs are also found in the blood of fish, birds, and dolphins (De Silva et al. 2016). As shorter-chain PFAA precursors have gained market share, shorter-chain PFAA concentrations in the marine environment have increased, as evidenced by increasing concentrations of PFBS in cetaceans from 2002-2014 (Lam et al. 2016). The PFBS precursor perfluorobutane sulfonamide (FBSA) also bioaccumulates in fish (Chu et al. 2016). 6:2 Cl-PFESA, currently used as a PFOS substitute in the chrome plating industry, has been detected in a wide range of marine organisms, including several species of gastropods, bivalves, crabs, shrimps, cephalopods, and fish, and tends to magnify along the food chain (Liu et al. 2017).

PFAAs are found in numerous marine species, including plankton, sea turtles, seals, California sea otters, whales, fish, sharks, polar bears, dolphins, and marine bird eggs; this indicates potential for widespread adverse impacts throughout the marine food web.

PFCAs are found in Arctic seals, presumably due to long-range transport of precursors such as FTOHs and FASAs via oceanic and atmospheric currents (Routti et al. 2016). Increased coastal PFAS concentrations can follow ice and snow melt in the Arctic, resulting in an increased exposure potential for marine biota in Arctic coastal regions (Zhao et al. 2012). Numerous studies have documented high concentrations of PFASs in Arctic mammals. For instance, up to seventeen PFASs were found in the livers of marine mammals off the coast of Greenland, including killer whales, polar bears, and ringed seals (Gebbink et al. 2016). PFASs can be transported across the brain blood barrier and accumulate in animal brain tissue, leading to concerns about potential neurotoxicity (Greaves et al. 2013). A study of 128 young polar bears (between three and five years old) sampled between 1984 and 2006 estimated that longer-chain PFASs in polar bears will reach levels associated with adverse impacts in lab animals by 2014-2024 (Dietz et al. 2008). PFASs were found to biomagnify throughout the Arctic food chain, with increasing concentrations in wolves that consume caribou contaminated with PFASs via their lichen-based diet (Müller et al. 2011). Researchers also evaluated the effect of cyclic seasonal emaciation on the distribution and composition of PFASs in Arctic foxes, finding significant differences that could indicate increased potential for health effects from PFASs when foxes have reduced access to food (Aas et al. 2014).

Table 4: Monitoring studies found varying levels of PFASs in the environment and biota.

Sample type	PFAS type	Concentration	Reference
Indoor air (homes)	6:2 FTOH 8:2 FTOH 10:2 FTOH 8:2 FTAC 10:2 FTAC	1.8 ng/m³ (median) 8.7 ng/m³ (median) 2.5 ng/m³ (median) 0.27 ng/m³ (median) 0.12 ng/m³ (median)	Fromme et al. (2015)
Indoor air (schools)	6:2 FTOH 8:2 FTOH 10:2 FTOH 8:2 FTAC 10:2 FTAC	3.3 ng/m³ (median) 4.4 ng/m³ (median) 1.8 ng/m³ (median) 0.45 ng/m³ (median) 0.31 ng/m³ (median)	Fromme et al. (2015)
Indoor air (offices)	6:2 FTOH 8:2 FTOH 10:2 FTOH	1.3 ng/m³ (geometric mean) 9.9 ng/m³ (geometric mean) 2.9 ng/m³ (geometric mean)	Fraser et al. (2012)
House dust	Sum of nine PFCAs (C6 to C14)	<1.0 – 37,400 µg/kg	Liu et al. (2011b)
Daycare center dust	PFOA PFOS	31 – 110 µg/kg 23 – 65 µg/kg	Bjorklund et al. (2009)
Outdoor air	FTOHs	0.06 – 0.19 ng/m³	Müller et al. (2012)
North American soils	Sum of PFCAs Sum of PFSAs	145 – 6,080 ng/kg 35 – 1,990 ng/kg	Rankin et al. (2016)
U.S. river (Cape Fear, North Carolina), used for drinking water	GenX (PFPrOPrA)	631 ng/L (mean)	Sun et al. (2016)
WWTP effluent (San Francisco Bay Area, California)	PFBA PFPeA PFHxA PFBS PFHxS PFOS	16 ng/L (mean) 12 ng/L (mean) 26 ng/L (mean) 2.7 ng/L (mean) 4.8 ng/L (mean) 13 ng/L (mean)	Houtz et al. (2016)
WWTP effluent (Germany)	PFBA PFPeA	<4.2 ng/L <254 ng/L	Frömel et al. (2016)
Sediments (South Bay area, California)	PFOS	0.60 – 2.61 µg/kg dry weight	Sedlak et al. (2017)
Sewage sludge	Sum of fourteen PFAAs	126 – 809 µg/kg	Yan et al. (2012)

Sample type	PFAS type	Concentration	Reference
Fish (whole fish from the Ohio, Missouri, and Upper Mississippi Rivers)	Sum of ten PFAAs	5.9 – 1,270 µg/kg 53.4 µg/kg (median)	Ye et al. (2008)
Prey fish (San Francisco Bay, California)	PFOS	11.8 µg/kg (geometric mean)	Sedlak et al. (2017)
Cormorant eggs (San Francisco Bay, California)	PFOS	36.1 – 466 µg/kg	Sedlak et al. (2017)
Harbor seals serum (San Francisco Bay, California)	PFOS	12.6 – 796 µg/kg	Sedlak et al. (2017)
Arctic lichen and plants	Sum of six PFCAs (C8 to C13)	0.02 – 0.26 µg/kg	Müller et al. (2011)
Arctic caribou (liver)	Sum of six PFCAs (C8 to C13)	6 – 10 µg/kg	Müller et al. (2011)
Arctic wolf (liver)	Sum of six PFCAs (C8 to C13)	10 – 18 µg/kg	Müller et al. (2011)

Monitoring data showing that the Candidate Chemical is present in human food

Exposure modeling studies have concluded that the ingestion of contaminated food and drinking water is the largest contributor of PFAAs to the overall human body burden (Trudel et al. 2008). A study of 2013-2014 NHANES data concluded that diet is the dominant and most important source of PFAA exposure for children aged three to eleven, even more so than for adolescents and adults (Jain 2018).

Contamination of food with PFASs occurs via contact with PFAS-treated food packaging materials (Trier et al. 2011), as well as from plant and animal uptake from contaminated soil and water. The Minnesota Department of Health found that the use of PFAS-contaminated irrigation water results in elevated PFAS concentrations in home-grown produce (Scher et al. 2018) . Shorter-chain PFASs such as PFBA showed the highest potential to translocate to and bioaccumulate in edible plants, thus entering the terrestrial food chain.

The PFASs currently used in U.S. carpet and rug treatments degrade into shorter-chain PFAAs that show greater bioaccumulation in vegetable crops than the longer-chain PFAAs from older treatments.

Biosolids from WWTPs can be heavily contaminated with PFASs that adsorb to the organic matter (Arvaniti et al. 2012; Sun et al. 2011), which can subsequently contaminate agricultural land when applied as a soil amendment (Blaine et al. 2014). The application of PFAA- and PFAA precursor-contaminated biosolids to land can lead to bioaccumulation of PFAAs in grasses (Yoo et al. 2011) and food crops (Navarro et al. 2017). Animal feed can become contaminated when grown in soil treated with PFAS-containing irrigation water or biosolids (Gobelius 2016; Lee et al. 2014; Yoo et al. 2011). PFAA levels in irrigation water correlate significantly with levels in vegetables (Zhang et al. 2016). The PFASs currently used in U.S. carpet and rug treatments degrade into shorter-chain PFAAs that show greater bioaccumulation in vegetable crops than the longer-chain PFAAs from older treatments (Blaine et al. 2013).

Measurements of PFASs in foods (see examples in Table 5) found them in wide-ranging concentrations (Domingo and Nadal 2017; EFSA 2012). Most available studies are from Europe and Asia, but levels are expected to be similar in the United States, including California. From 2006-2012, the European Food Safety Agency (EFSA 2012) tested 7,560 food samples and found PFASs in most of them. The most prevalent PFAS contaminants were PFOS (29 percent) and PFOA (nine percent). PFBS and PFHxA were found to accumulate in food crops, including sugar beets, broccoli, spinach, lettuce, celery, potato, pea, fruit, and green beans (EFSA 2012). A review and analysis of literature data published after 2010 found that shorter-chain PFAAs, including PFBA, PFPeA, and PFHxA, were frequently detected at high concentrations in vegetables, fruits, and beverages (Jian et al. 2017). Fish appears to be the most frequently PFAS-contaminated food (Yamada et al. 2014a; Yamada et al. 2014b), estimated to contribute the most to dietary exposure (Domingo and Nadal 2017; EFSA 2012), though terrestrial animals also have high PFOS concentrations (EFSA 2012; Haug et al. 2010). One study measured PFASs in 21 foods and beverages in Norway, reporting concentrations of PFOS in cod and cod liver that were much higher than in other foods, including other types of fish (Haug et al. 2010).

A 2012 European Union study estimated that PFOA and PFOS dietary exposure doses were much lower than the tolerable daily intake (TDI) established by EFSA in 2008. Dietary exposure to PFOS in toddlers was estimated to contribute almost 20 percent of the TDI in the most exposed group (EFSA 2012). The TDI was based on a sub-chronic study in Cynomolgus monkeys, which did not consider exposure sources other than dietary, nor exposure to PFOA and PFOS precursors (EFSA 2008). TDIs for other PFASs have not been established.

Dietary exposure studies in the United States are generally lacking (Domingo and Nadal 2017), although one study in California found significant positive associations between PFAS serum concentrations for children and adults and consumption of certain foods including butter/margarine, fish, meat products, and microwave popcorn (Wu et al. 2015). The globalization of the human food chain, combined with widespread contamination of foods, indicates the potential for lifetime exposure to PFASs for the people of California.

Table 5: Monitoring studies found varying levels of PFASs in human food.

Sample Type	PFAS Type	Concentration	Reference
Potatoes	PFOA	0.07 µg/kg	Schecter et al. (2010)
Vegetables	PFHxA	<LOD – 0.42 µg/kg	EFSA (2012)
Fruit	PFHxA	<LOD – 0.17 µg/kg	EFSA (2012)
Fruit	PFBS	<LOD – 0.067 µg/kg	EFSA (2012)
Fruit	PFHxS	<LOD – 0.20 µg/kg	EFSA (2012)
Meat products	PFOA	<LOD – 0.24 µg/kg	Schecter et al. (2010)
Crustaceans	PFOA	<LOD – 8.0 µg/kg	EFSA (2012)
Fish	PFOA	<LOD – 0.30 µg/kg	Schecter et al. (2010)

Sample Type	PFAS Type	Concentration	Reference
Fish	PFHxA	<LOD – 23 µg/kg	EFSA (2012)
Fish	PFOA	<LOD – 18.2 µg/kg	EFSA (2012)
Fish	PFOS	<1 ng/g – >100 µg/kg	Berger et al. (2009)
Fish and other seafood[12]	PFOS	<LOD – 310 µg/kg	EFSA (2012)
Eggs	Sum of eleven PFASs	27 – 160 µg/kg	Wang et al. (2008)
Eggs and products	PFOA	<LOD – 25.5 µg/kg	EFSA (2012)
Eggs and products	PFOS	<LOD – 6.4 µg/kg	EFSA (2012)
Home produced eggs (yolks) two km from industrial site	Sum of twelve PFAAs	8.99 - 482 µg/kg	Su et al. (2017)
Butter	PFOA	1.07 µg/kg	Schecter et al. (2010)
Margarine	PFOA	0.19 µg/kg	Schecter et al. (2010)
Olive oil	PFOA	1.80 µg/kg	Schecter et al. (2010)
Honey	PFOA	0.00 – 0.47 µg/kg	EFSA (2012)

Monitoring data showing that the Candidate Chemical is present in drinking water

PFASs are detected in drinking water around the world, typically in the ng/L range (Xiao 2017). Drinking water can become contaminated with PFAAs and their precursors from carpet and rug stain- and soil-repellents via several routes, including direct discharge of industrial effluent into waterways, leaching of land-applied wastewater or biosolids into groundwater, leachate draining from landfills that contain discarded carpets, and discharge of WWTP effluent into streams and rivers (Lindstrom et al. 2011a). A review and analysis of literature data published after 2010 found that, out of all PFASs measured to date, PFOS, PFHxA, PFHpA, PFNA, PFDA, and PFOA had the highest concentrations and detection frequencies in drinking water (Jian et al. 2017). A study of 133 PFASs in drinking water (bottled and tap) from nine countries, including the United States, found shorter-chain PFAAs in 64 to 92 percent of the samples (Kaboré et al. 2018).

Drinking water can become contaminated with PFAAs and their precursors from carpet and rug stain- and soil-repellents via several routes, including direct discharge of industrial effluent into waterways, leaching of land-applied wastewater or biosolids into groundwater, leachate draining from landfills that contain discarded carpets, and discharge of WWTP effluent into streams and rivers.

As part of the third Unregulated Contaminant Monitoring Rule (UCMR3) program, U.S. EPA (2017c) monitored public water supplies for six PFAAs from 2013 until 2015, reporting their presence in approximately three percent of samples tested (Table 6). PFOA and PFOS concentrations equaled or exceeded the lifetime total health advisory of 70 ng/L, combined, in water serving approximately 16.5 million people, with most detects (28 out of 452 samples, or 6.2 percent detection rate) in California (Hu et al. 2016; Hurley et al.

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[12] PFOS mean concentrations were “constantly higher” in freshwater fish than in marine fish.

2016). The most significant predictors of observed PFAS detection frequencies and concentrations were: (1) proximity to industrial sites that manufacture or use PFASs; (2) proximity to military fire training areas; and (3) the number of nearby WWTPs (Hu et al. 2016).

The Minimum Reporting Levels (MRLs) for the six PFASs measured by the UCMR3 program ranged from ten to 90 ng/L, whereas the method used has a limit of quantification in the two to five ng/L range. Eurofins Eaton Analytical (2017), which was responsible for analyzing approximately 40 percent of the UCMR3 data, found PFASs in more than a tenth of the samples they analyzed. However, fewer than two percent of those samples showed values above the UCMR3 MRL and were reported to U.S. EPA. This suggests that the UCMR3 program may have significantly underestimated the prevalence of PFASs in U.S. drinking water.

Drinking water contamination can linger for decades. In August 2017, the town of Belmont, MI, measured PFASs in some of their drinking water at levels 540 times above the EPA health advisory level (32,000 ng/L PFOS and 5,800 ng/L PFOA). The contamination is linked to a hazardous waste site used in the 1960s by a former tannery. No PFASs have been added to the site since the 1970s (Chawaga 2017; Ellison 2017).

Table 6: Monitoring studies found varying levels of PFASs in drinking water.

Sample Type	PFAS Type	Concentration	Reference
U.S. drinking water[13]	PFOA PFOS PFNA	<MRL – 349 ng/L <MRL – 1,800 ng/L >MRL – 56 ng/L	Hu et al. (2016)
European drinking water	PFBS PFHxS PFOS	<LOD – 0.24 µg/kg <LOD – 0.011 µg/kg <LOD – 0.016 µg/kg	EFSA (2012)

Monitoring data showing that the Candidate Chemical is present in California media at levels of concern

Numerous California drinking water sources are contaminated with PFAAs according to the UCMR3 study. 40 percent of the positive UCMR3 study detects in California are above the U.S. EPA advisory level of 70 ng/L for PFOA and PFOS combined (Hurley et al. 2016).

California sea otters were found to have PFOA and PFOS levels ranging from <5 to 147 ng/g and <1 to 884 ng/g, respectively, significantly correlated with incidence of disease (Kannan et al. 2006). Historically, San Francisco Bay wildlife has shown some of the highest PFOS levels ever measured (Sedlak and Greig 2012). In 2010, contaminant monitoring of the San Francisco Bay surface water detected several PFAAs, including: PFBS at concentrations up to 7.89 ng/L (average 1.58 ng/L), PFBA at up to 62.20 ng/L (average 12.96 ng/L), and PFHxA ranging from 1.37 ng/L to 221.0 ng/L (SFEI 2010). Harbor seal serum sampled in 2014 still contained PFOS at

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[13] The UCM3 MRLs (Minimum Reporting Levels) for PFASs in U.S. drinking water are 10 – 90 ng/L depending on the PFAS being tested.

concentrations ranging from 12.6 to 796 ng/g, more than a decade after the PFOS phaseout (Sedlak et al. 2017). A 2016 study of PFAAs and precursors in wastewater effluent discharged to San Francisco Bay highlighted the local impact of AFFF,[14] and noted that levels of PFBA, PFPeA, and PFHxA in WWTP effluent had increased by 150 to 220 percent since 2009 (Houtz et al. 2016). Due to this widespread contamination, the San Francisco Bay Regional Monitoring Program designated PFOS as “moderate concern” for the San Francisco Bay, and all other PFASs as “possible concerns” (Sutton et al. 2017).

Human biomonitoring data

Nearly all humans ever studied show evidence of exposure to some PFASs (Calafat et al. 2007), which can accumulate in human lungs, kidneys, liver, brain, and bone tissue (Perez et al. 2013). Biomonitoring studies have been limited to certain PFASs with available analytical methods (see Table 7 for some examples), however humans are exposed to many more PFASs that haven’t yet been measured (Yeung and Mabury 2016).

The Centers for Disease Control and Prevention (CDC), which monitors Americans’ exposure to PFASs as part of the National Health and Nutrition Examination Survey (NHANES), has detected PFASs in the blood of all people tested (Calafat et al. 2007). NHANES blood samples collected in 2011-2012 averaged PFHxS, PFOA, and PFOS from 1.3 to 6.3 µg/L (ATSDR 2017).

The Centers for Disease Control and Prevention (CDC), which monitors Americans’ exposure to PFASs as part of the National Health and Nutrition Examination Survey (NHANES), has detected PFASs in the blood of all people tested.

Shorter-chain PFAAs such as PFHxA have rarely been measured or detected in human serum. However, while PFHxA may be below detection in human serum and plasma, it can be found at concentrations higher than most longer-chain PFASs in human whole blood samples (Poothong et al. 2017). Other studies demonstrate that shorter-chain PFAAs (PFBA, PFPeA and PFHxA) are detectable in human urine at levels comparable to or higher than PFOA (Hartmann et al. 2017; Kim et al. 2014; Perez et al. 2012). As the unique behaviors of PFAAs in biological systems and their human toxicokinetics become better understood, CDC may revise its biomonitoring protocols to sample tissues most relevant to assessing exposures to shorter-chain PFAAs (Calafat 2017).

The California Biomonitoring Program, administered jointly by the California Department of Public Health, DTSC, and OEHHA, has measured PFAA levels in several subpopulations (CECBP 2017). The California Teachers Study is a statewide collaborative research project evaluating health-related factors in the development of breast cancer. For more than 20 years, over 133,000 female school teachers and employees have participated in the study. As of 2015, the California Biomonitoring Program has found PFHxS, PFOA, and PFOS in >99 percent of 1,759 blood samples provided by study participants (CECBP 2015b). Hurley et al. (2016) analyzed blood samples from a subset of 1,333 participants who lived in areas where PFASs in drinking water concentrations were tested. Of these participants, 109 women lived in an area where at least one PFAS was detected in drinking

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[14] AFFF may be the largest direct point source of PFASs, according to a Swedish Environmental Protection Agency study (Goldenman et al. 2017). Generally, AFFF contains high levels of PFSAs, including PFOS and PFHxS, but only low levels of PFCAs (Hu et al. 2016).

water from public water systems. The analysis showed an association between detectable levels of PFOA and PFOS in drinking water and the women's blood levels (Hurley et al. 2016). In a subset of 1,257 women aged 40 to 94 from the California Teachers Study, serum levels of longer-chain PFASs decreased from 2011 to 2015 on average by ten to 20 percent per year – except for PFHxS, which did not decline significantly (Hurley et al. 2018). Another study of 139 California households detected PFASs in all study participants, including young children and adults (Wu et al. 2015). Exposure levels correlated with diet and use of consumer products such as fire extinguishers and stain-repellent or waterproof clothing, and residential dust exposure in children.

In North Carolina, a study of 37 young adults detected PFHxS (1.07 – 12.55 ng/mL), PFOS (0.39 – 31.35 ng/mL), PFOA (0.30 – 4.07 ng/mL), and PFNA (0.23 – 4.02 ng/mL) in all serum samples, and PFHxA (ND – 1.00 ng/mL) and PFDA (ND – 1.60 ng/mL) in 83.8 percent and 97.3 percent of serum samples, respectively (Siebenaler et al. 2017). In Europe, biomonitoring of pregnant and nursing women from 1996 to 2010 found that PFBS, used in the manufacture of Scotchgard brand stain- and soil-repellent, doubled its blood concentrations every 6.3 years, concurrent with the phaseout of PFOS (Glynn et al. 2012).

PFASs in carpets and rugs may impact children more than adults due to their frequent hand-to-mouth behavior, more direct contact with carpets and rugs over a larger body surface area, and other physiological differences.

A study of more than 600 children aged six to ten from Eastern Massachusetts found higher blood concentrations of certain PFAAs and PFAA precursors in those children having a carpet or a rug in their bedroom (Harris et al. 2017). PFASs in carpets and rugs may impact children more than adults due to their frequent hand-to-mouth behavior, more direct contact with carpets and rugs over a larger body surface area, and other physiological differences (Moya et al. 2004).

Table 7: Human biomonitoring studies found widespread exposure to PFASs.

Sample Type	PFAS Type	Concentration	Reference
Serum of women aged 40 to 94	PFHpA	0.084 ng/mL (mean); 1.16 ng/mL (max)	Hurley et al. (2018)
	PFOA	3.0 ng/mL (mean); 27.6 ng/mL (max)	
	PFNA	1.07 ng/mL (mean); 10.4 ng/mL (max)	
	PFDA	0.28 ng/mL (mean); 3.9 ng/mL (max)	
	PFUnDA	0.17 ng/mL (mean); 1.3 ng/mL (max)	
	PFHxS	2.2 ng/mL (mean); 21.8 ng/mL (max)	
	PFOS	8.5 ng/mL (mean); 99.8 ng/mL (max)	
Adult human blood	PFOA	2.1 – 9.6 ng/mL (means reported in various studies)	ATSDR (2015)
	PFOS	14.7 – 55.8 ng/mL (means reported in various studies)	
Adult human blood (near contaminated site)	PFOA	3.09 ng/mL (mean); 32 ng/mL (max)	NHDHHS (2016)
	PFOS	8.59 ng/mL (mean); 95.6 ng/mL (max)	
	PFHxS	4.12 ng/mL (mean); 116 ng/mL (max)	

Sample Type	PFAS Type	Concentration	Reference
Cord blood	PFOA	1.6 – 3.7 ng/mL	ATSDR (2015)
Breast milk	PFPeA	0.053 ng/mL (geometric mean)	Kang et al. (2016)
	PFHxA	0.047 ng/mL (geometric mean)	
	PFHpA	0.030 ng/mL (geometric mean)	
	PFOA	0.071 ng/mL (geometric mean)	
	PFOS	0.049 ng/mL (geometric mean)	

Evidence that the Candidate Chemical exhibits certain hazard traits (persistence, bioaccumulation, lactational/transplacental transfer)

As discussed in Section 2.2, PFAAs are highly persistent and tend to accumulate in the environment and biota, due to the extremely strong bond between carbon and fluorine atoms that does not degrade under typical environmental conditions (Giesy et al. 2010; Liu and Mejia Avendano 2013).

Due to transplacental transfer, the developing fetus is exposed to mixtures of PFAAs *in utero* (Bach et al. 2014; Mamsen et al. 2017). Fluorinated ethers, such as Cl-PFESA, can also transfer from mother to fetus via the placenta (Pan et al. 2017). PFAAs are found in breast milk, potentially adding to significant cumulative early-life exposures. A 2016 study reported detectable concentrations of PFHxA in >70 percent of breast milk samples (n=264), and detectable PFOA levels in the breast milk of ~99 percent of breast-feeding mothers, associated with exposure to PFASs in consumer products (Kang et al. 2016). Breastfed newborns can have higher body burdens of PFAAs compared to those fed only infant formula (Fromme et al. 2010). Kim et al. (2014) analyzed concentrations of PFASs in the serum and urine of children, and found a correlation between longer duration of breastfeeding and serum concentrations.

Transfer to the fetus and breastfeeding appear to be significant PFAA elimination routes for women; serum concentrations of PFOA and PFOS are estimated to decrease by two to three percent per month while breastfeeding (Mondal et al. 2014). Biomonitoring studies of infants and small children suggest that peak concentrations of PFOS and PFOA occur before children turn two years old (Winkens et al. 2017).

PFASs are released into air, water, and soil during the manufacture of stain- and soil-repellents, and during the manufacture, consumer use, and landfilling of carpets and rugs.

3.3.2 Potential exposure to the Candidate Chemical during the product's life cycle, considering manufacturing, use, storage, transportation, waste, and end-of-life management practices

Reference: CAL. CODE REGS. tit. 22, § 69503.3(b)(4)(A).

The lifecycle of carpets and rugs can expose humans and other living organisms to PFASs via several routes (Figure 3). PFASs are released into air, water, and soil during the manufacture of stain- and soil-repellents, and during the manufacture, consumer use, and landfilling of carpets and rugs (Ahrens et al. 2011b; Oliaei et al. 2013). As explained in Section 2.2, PFAAs can be emitted to the environment either directly during the life cycle of the product (manufacture, use, disposal), or indirectly via the degradation of precursors (OECD 2013). 3M Company, a major PFAS manufacturer, estimated that 85 percent of indirect emissions of PFOS-related compounds occurred during the use and disposal of consumer products such as carpets, while only fifteen percent resulted from manufacturing releases into the environment (Paul et al. 2009).

PFASs from the manufacture and use of stain- and soil-repellents in carpets and rugs can enter waterways via direct discharge from manufacturers, release of WWTP effluent, or following land application of contaminated biosolids, resulting in PFAS-contaminated surface water and groundwater (Hoffman et al. 2011; Lindstrom et al. 2011a). Most WWTPs cannot adequately remove PFASs, making them a common source of PFASs to surface waters (Dauchy et al. 2012; Guo et al. 2010; Rayne and Forest 2009).

Manufacturing-related emissions

Decades of manufacturing and use of PFASs, including as stain- and soil-repellents for carpets and rugs, have contributed to global environmental contamination (Cousins et al. 2016; Hoffman et al. 2011; Oliaei et al. 2013). Volatile PFASs can be released into the air during the manufacture of stain- and soil-resistant chemical treatments, impregnation of carpet fibers, and application of repellent on carpets and rugs (Stock et al. 2004).

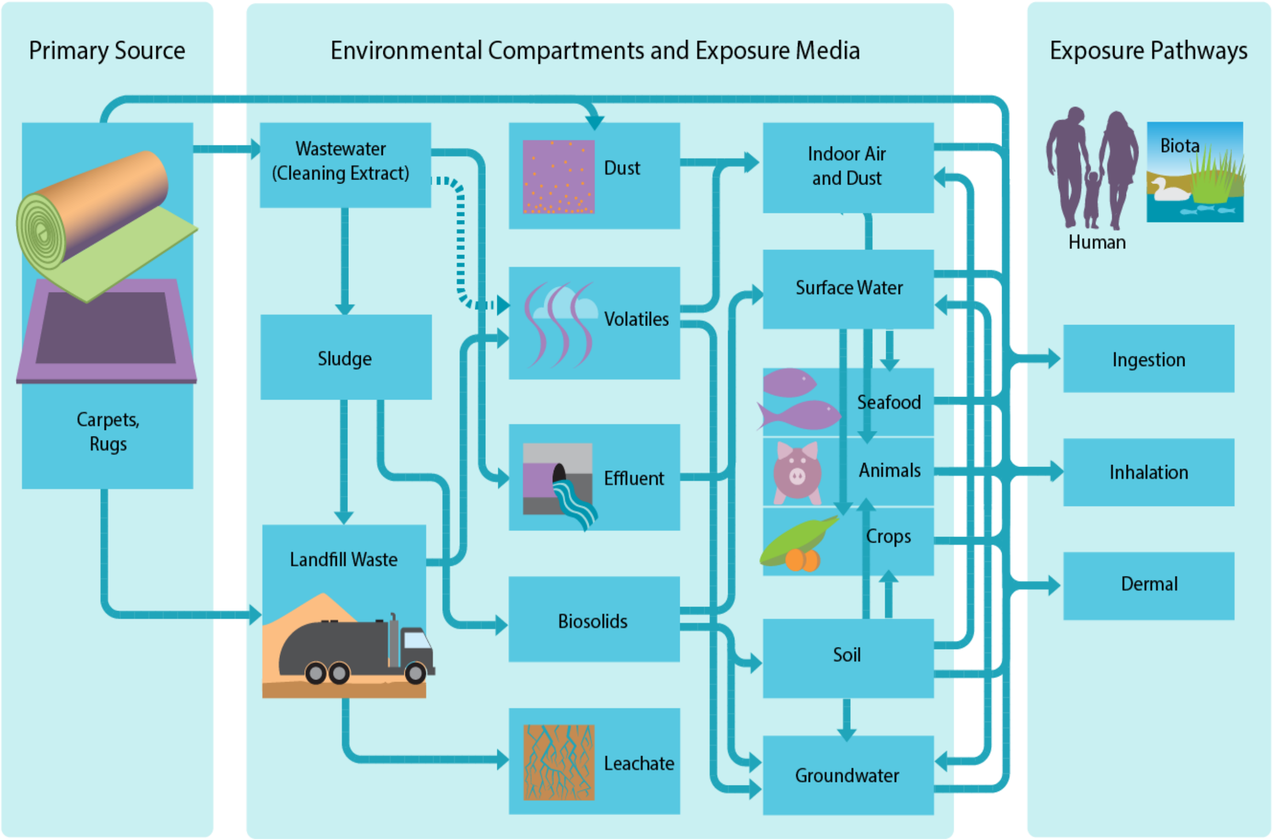

Figure 3: Possible pathways of exposure to PFASs from the life cycle of carpets and rugs.

Manufacturing wastewater discharges into WWTPs can contaminate rivers and other waterways (Konwick et al. 2008). Once released into air and water, PFAAs can be highly mobile and undergo long-range transport to distant regions via atmospheric circulation and oceanic currents (Ahrens et al. 2011a; Prevedouros et al. 2006), as explained in more detail in Section 2.2.1.

PFAAs and their precursors emitted during carpet and rug manufacturing can contaminate soil and sediments via deposition of contaminated particulates, use of contaminated irrigation water, and land-application of contaminated biosolids (Armitage et al. 2009; Blaine et al. 2014; Lindstrom et al. 2011a; Zhang et al. 2016). One study of 262 surface sediment samples from 48 lakes and two reservoirs in China (Qi et al. 2016) found high levels of sediment PFASs (C4 through C14) correlated with proximity of the sampling locations to industrial activities in food packaging, textile, electroplating, firefighting, semiconductor, precious metals processing, and coating industries.

Exposure during use of carpets and rugs

The air in homes, offices, and retail businesses can become contaminated with volatilized FTOHs, which are impurities or intermediate degradation products of the stain- and soil-repellents used on carpets and rugs (Bohlin-Nizzetto et al. 2015; Schlummer et al. 2015). PFAAs from carpet and rug treatments can also adsorb to house dust or be released as tiny particulates from surface abrasion during normal use (Rosati et al. 2008; Tian et al. 2016). Household and office dust can concentrate PFASs, leading to exposure via inhalation of PFAA-containing fine particulates and ingestion of dust (Haug et al. 2011; Rosati et al. 2008; Shoeib et al. 2005).

A study of young adults in North Carolina found a statistically significant 57 percent increase in PFHxS levels in participants who reported vacuuming less frequently (Siebenaler et al. 2017). Another study of Canadian pregnant women found that levels of PFAA precursors in indoor air and dust correlated with participants' PFOA, PFNA, and PFOS serum levels (Makey et al. 2017). Although dust ingestion is generally considered a minor pathway of exposure to PFASs in adults, ingestion in toddlers and young children can be significantly higher (Tian et al. 2016; Wu et al. 2015).

Carpet cleaning operations may dispose of PFAS-contaminated wastewater in drains, entering WWTPs in areas served by public water systems. When contaminated wastewater is discharged into residential septic tanks, water soluble organic chemicals like PFAAs may migrate into nearby soil and waterways adjacent to the leach field (Schaider et al. 2016; Schaider et al. 2014). WWTP discharge can have concentrations of PFOA up to hundreds of ng/L (Hamid and Li 2016), compared to the U.S. EPA lifetime health advisory of 70 ng/L for PFOA and PFOS combined, and the New Jersey PFOA Maximum Contaminant Level for drinking water of 14 ng/L. As explained in Section 3.3.1, discharge of PFAA-contaminated effluent into surface waters can lead to contamination of streams, rivers, and sediments (Ahrens et al. 2011b; Sun et al. 2012).

Contamination resulting from carpet and rug disposal

In 2016, 343 million pounds of post-consumer carpet were discarded in California, out of which 257 million pounds (75 percent) were landfilled (CCSP 2016). Of the carpet that was not landfilled, eleven percent (38 million pounds) was recycled, 0.27 percent (926,000 pounds) was reused, and 6.1 percent (21 million pounds) was combusted for energy recovery (CCSP 2016). The California Carpet Stewardship Program, established in 2011 pursuant to the California Carpet Stewardship Law (California Public Resources Code §§ 42970-42983), aims to increase the proportion of discarded carpet diverted from landfills. California State Assembly Bill 1158, approved on October 14, 2017, creates a goal of a 24 percent recycling rate by 2020.

In landfills, the side-chain fluorinated polymers currently used in most carpet and rug stain- and soil-resistant treatments can degrade to form first non-polymeric fluorotelomer-based PFASs such as FTOHs, which then further degrade to intermediates such as FTCAs and FTUCAs, and finally to PFAAs (Hamid et al. 2018; Washington et al. 2015). The degradation of these side-chain fluorinated polymers in landfills and other waste stocks can be a significant long-term (years to centuries) source of PFAAs to the environment (Lang et al. 2017; Li et al. 2017). Estimated release rates from waste stocks are almost two orders of magnitude higher for shorter-chain PFCAs, due to their greater mobility in water, compared to longer-chain PFCAs (Li et al. 2017).

Landfills have been identified as a source of PFAS contamination to nearby air, dry deposition, and plant leaves (Tian et al. 2018). PFAAs and their precursors from discarded carpets and rugs can be released into the atmosphere during carpet decomposition in landfills, and become widely dispersed (Ahrens et al. 2011b). PFAAs can also become mobilized in landfill leachate (Fuertes et al. 2017; Lang et al. 2016; Shoaeioskouei 2012). Contaminated leachate collected from lined landfills for treatment at WWTPs can result in contaminated waterways, as wastewater treatment does not completely remove effluent PFAAs (Hamid and Li 2016; Huset et al. 2011).

PFAAs and their precursors have been detected in landfill leachate all over the world, including Germany (Busch et al. 2010), Canada (Benskin et al. 2012a), and Australia (Gallen et al. 2017). In the United States, an estimated 61.1 million m^3 of leachate was generated in 2013, containing approximately 600 kg total of 70 PFASs measured (Lang et al. 2017). 5:3 FTCA, an intermediate in the degradation of fluorotelomer-based substances to PFAAs, showed the highest mean concentration in leachate from surveyed landfills, followed by PFHxA; the following PFASs were detected in similar amounts: PFBA, PFPeA, PFHpA, PFOA, and 6:2 FTCA (Lang et al. 2017). An evaluation of PFASs leaching from carpet in simulated landfill conditions found increased leaching concentrations over time (Kim et al. 2015). The relatively slow release of PFASs from landfills compared to input rates means that landfilled waste continues to release PFASs more than a decade or longer (Lang et al. 2017). With 50 percent of fluorotelomers and related side-chain fluorinated polymers used in treatment of textiles, including carpets and rugs, the release of contaminated leachate into the environment will continue to be a pervasive and long-lasting threat to drinking water sources.

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Furthermore, biosolids from WWTPs can be heavily contaminated with PFAAs and precursors that adsorb to organic matter (Sun et al. 2011). When these materials are land-applied or landfilled, PFASs can migrate from biosolids into soil and leach into adjacent waterways, potentially leading to widespread contamination and ecological impacts. Land application of biosolids contaminated with PFAAs and their precursors can lead to bioaccumulation of PFAAs in grasses (Yoo et al. 2011) and food crops (Blaine et al. 2014; Lee et al. 2014). PFAA precursors such as those used in treated carpets and rugs can also undergo transformation to PFAAs in soils (Washington and Jenkins 2015).

Recycling of post-consumer carpet is a preferred alternative to landfilling, but can perpetuate the presence of PFASs in products made from the recycled material (Herzke et al. 2012). Carpet incineration for energy recovery may also release PFASs and other hazardous chemicals in the environment. A literature survey regarding the combustion of various fluorinated polymers reported emissions of C3-C14 PFCAs, ozone depleting substances such as chlorofluorocarbons (CFCs), and greenhouse gases such as fluorocarbons (Huber et al. 2009).

3.3.3 Frequency, extent, level, and duration of potential exposure for each use and end-of-life scenario

Reference: CAL. CODE REGS. tit. 22, § 69503.3(b)(4)(E).

Everyone can experience potential exposures to PFASs frequently over a lifetime, including from placental transfer, breastfeeding, drinking water, food, and contact with consumer products and environmental media. Biota can also experience potential exposures via environmental media and diet. This section highlights exposure scenarios for the following human subpopulations at risk of higher-than-average exposures, or more vulnerable to these exposures: fetuses, infants, toddlers, office workers, workers in carpet stores, carpet installers, and carpet cleaners.

With more than 3.9 million births in the U.S. in 2016, fetuses and infants constitute a large subpopulation highly vulnerable to exposures to PFASs released during the manufacturing and use of stain- and soil-repellents in carpets and rugs. Because PFASs undergo transplacental migration (see Section 3.3.1), fetuses can be continuously exposed from conception until birth. PFASs are also frequently detected in breast milk, with nursing infants exposed daily over many months. The CDC reported that 79 percent of infants born in 2011 were breastfed, with 27 percent still breastfeeding at twelve months of age. The amount of breast milk ingested by newborns averages 76 g, six to eighteen times per day (CDC 2014).

Toddlers can experience increased exposures to PFASs due to behavioral characteristics. Children between the ages of one and three spend a lot of time in contact with the floor, where dust settles, and engage in indoor hand-mouth behaviors an average of sixteen times per day (U.S. EPA 2011). Thus, dust ingestion can be a significant route of exposure for young children (Mercier et al. 2011; Tian et al. 2016). While adults are estimated to ingest an average of 50 mg/day of soil and dust, children under six years of age may ingest 100 mg/day (U.S. EPA 2011). PFAS doses via dust ingestion could be approximately an order of magnitude higher for toddlers than for adults (Karásková et al. 2016). A study of toddlers' cumulative exposure to PFOA and PFOS estimated a daily intake of 53.6 ng/day of PFOA and 14.8 ng/day of PFOS (Tian et al. 2016).

Office workers are another large group of Californians possibly experiencing chronic exposures to PFASs while on the job. Office air can be contaminated with FTOHs from carpet treatments, leading to involuntary, chronic inhalation exposures in workers, which has not been well-characterized (Bohlin-Nizzetto et al. 2015; Schlummer et al. 2015). PFASs can also adsorb to office dust or be released from surface abrasion as tiny particulates, which office workers may ingest (Fraser et al. 2013). Due to their long usage life, commercial carpets could be a near-daily source of PFOA, PFOS, and other phased-out longer-chain PFAS exposure for office workers.

Workers in carpet stores and carpet installers are also vulnerable to chronic inhalation exposures to PFASs found in carpets and rugs. These workers could potentially experience PFAS exposures for the entire duration of their work shift. According to the latest U.S. Census data, there are more than 850 stores in California that sell carpets and rugs (U.S. Census Bureau 2017).[15] The U.S. Bureau of Labor Statistics estimates that there are 5,270 carpet installers in California, more than in any other U.S. state (U.S. BLS 2016). These statistics indicate that thousands of Californians employed in these two business sectors could be highly exposed to PFASs at work. Exposure factors that influence actual internal PFAS dosages in an individual can only be accurately determined through in-depth epidemiological and workplace studies that monitor individual exposures from numerous sources, which have not been conducted to date.

3.4 Factors That May Mitigate or Exacerbate Exposure to the Candidate Chemical

3.4.1 Containment of the Candidate Chemical within the product

Reference: CAL. CODE REGS. tit. 22, § 69503.3(b)(4)(F).

PFASs are not fully contained within the product, and have the potential to be released during product use, and end-of-life, as discussed above.

3.4.2 Engineering and administrative controls that reduce exposure concerns

Reference: CAL. CODE REGS. tit. 22, § 69503.3(b)(4)(G).

Administrative and engineering controls are unlikely to address exposure concerns during product use and disposal.

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[15] We performed an advanced search by Industry Codes using the 2012 NAICS code "442210: Floor covering stores," then refined the search results by limiting them to California, and selected the document ID EC1244SLLS11 ("Retail Trade: Subject Series – Product Lines: Product Lines Statistics by Industry for the U.S. and States: 2012"). The table lists 852 establishments under Products and Services Codes 20365 (soft-surface (textile) floor coverings to be installed)," and 641 establishments under Products and Services Code 20366 (carpets & rugs not requiring installation).

4 ADVERSE WASTE AND END-OF-LIFE EFFECTS

Reference: CAL. CODE REGS. tit. 22, §§ 69503.2(b)(1)(B) and 69501.1 (a)(8).

4.1 Discharges or Disposals to Storm Drains or Sewers That Adversely Affect Operation of Wastewater or Stormwater Treatment Facilities

PFASs are not routinely removed by WWTPs, and removal is expensive (Arvaniti and Stasinakis 2015; Mudumbi et al. 2017). Following contamination from carpet manufacturing in Dalton, Georgia, Alabama water utility Gadsden Water stated that it “has suffered substantial economic and consequential damage, including expenses associated with the future installation and operation of a filtration system capable of removing the chemicals from the water and lost profits and sales” (Chapman 2016).

4.2 Releases of the Chemical into the Environment from Solid Waste Handling, Treatment, or Disposal of the Product, or from Product-Related Discharge or Disposal to Storm Drains or Sewers

In California, carpets are one of the top ten components of the landfill waste stream – approximately 3.2 percent of waste by volume (CalRecycle 2017; GAIA and Changing Markets 2017). From 2015 to 2016, due to the 2010 California Carpet Stewardship Law, post-consumer carpet collection and carpet tile recycling increased by four and 31 percent, respectively. In 2016, 343 million pounds of carpet were discarded in California, of which 75 percent (257 million pounds) were disposed to landfills (CCSP 2016). Of the 86 million pounds diverted from California landfills in 2016, 21 million pounds were combusted for energy recovery (CCSP 2016). This combustion occurs in incinerators often located in low income communities, where emissions burden people already impacted by cumulative chemical exposures from other sources, creating environmental justice concerns (GAIA and Changing Markets 2017).

In California, carpets are one of the top ten components of the landfill waste stream – approximately 3.2 percent of waste by volume. In 2016, 343 million pounds of carpet were discarded in California, of which 75 percent (257 million pounds) were disposed to landfills.

As detailed in Section 3.3.2, municipal landfill leachate is a known point source of PFAS emissions into the environment (Benskin et al. 2012a; Busch et al. 2010; Eggen et al. 2010; Fuertes et al. 2017; Hamid et al. 2018; Huset et al. 2011; Lang et al. 2017), with waste carpets and rugs as significant contributors (Gallen et al. 2017; Lang et al. 2016; Shoaeioskouei 2012). This impacts unlined landfills and those with a leachate management system, but also those that collect and treat the leachate in WWTPs, which often fail to adequately remove PFASs (Arvaniti and Stasinakis 2015; Huset 2007) and discharge PFAS mixtures into receiving waters, as explained in Section 3.3.1 (Dauchy et al. 2017; Hamid and Li 2016; Pan et al. 2016). WWTPs are also significant sources of volatile PFASs, as are landfilled carpets and rugs (Ahrens et al. 2011b; Weinberg et al. 2011). A study of PFASs in digested sewage sludge from

45 WWTPs in Switzerland found that carpet protection and textile finishing industries were the most relevant point sources of PFCAs (Alder and van der Voet 2015).

PFASs in landfill leachates, land-applied wastewater, and biosolids can contaminate streams, rivers, and other drinking water sources (Hoffman et al. 2011; Lindstrom et al. 2011a). Stormwater runoff containing PFASs released from consumer products may be a significant pathway for PFAS transport and release to surface waters (Houtz and Sedlak 2012).

5 ADDITIONAL CONSIDERATIONS

5.1 Other Relevant Factors Not Identified by the Regulation

Although PFAS manufacturers have been aware of these chemicals' adverse impact potential for decades, national and international action to limit human and ecological exposures has only recently started. In 2015, more than 200 scientists from 40 countries signed The Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs) (Blum et al. 2015), a scientific consensus on the persistence and potential for harm of this class of chemicals, and a roadmap for international cooperation to prevent further harm.[16]

To address concerns about potential health effects from cumulative exposures to PFOA and PFOS, U.S. EPA established individual chronic reference doses (RfDs) for PFOA and PFOS of 0.00002 mg/kg/day (U.S. EPA 2016b; U.S. EPA 2016c). These are estimates of daily human exposure expected to be without adverse health effects over a lifetime. In Fall 2016, after concerns that more than 16 million Americans are exposed to PFOA and PFOS via contaminated drinking water, U.S.EPA reviewed new toxicological studies on PFOA-induced developmental effects and reduced its 2009 400 ng/L provisional Health Advisories to a lifetime drinking water Health Advisory of 70 ng/L for combined concentrations of both chemicals (U.S. EPA 2016a). These Health Advisories are recommendations only, and are not legally binding. Also in fall 2016, Congressman Brendan Boyle of Philadelphia, PA introduced legislation (H.R. 6125) to require an enforceable nationwide standard for PFASs in drinking water to replace the U.S. EPA's voluntary health advisories (Boyle 2016). The bill is still pending in the House Committee on Energy and Commerce. On December 4, 2017, U.S. EPA announced a cross-agency effort to address PFASs, including filling data gaps, supporting communities impacted by drinking water contamination, and proactively communicating with states, tribes, partners, and the public about the health effects of PFAS exposure (U.S. EPA 2017d).

Several U.S. states have set lower health advisories than U.S. EPA, but most of these are also not legally binding. In May 2016, the State of Vermont issued a drinking water health advisory of 20 ng/L of PFOA and PFOS combined, which was also based on developmental endpoints, including reduced ossification and accelerated puberty in males and low birth weight (Vermont Department of Health 2016). In May 2017, the Minnesota Department of Health released revised drinking water guidance values of 35 ng/L for PFOA and 27 ng/L for PFOS (MDH 2017), to "reflect new state-level analysis of the potential for mothers to pass along the chemicals to fetuses and nursing infants." In November 2017, New Jersey became the first state to set formal Maximum Contaminant Levels of 14 ng/L for PFOA, and 13 ng/L for PFNA (NJDEP 2017). On December 13, 2017, Michigan Democrats introduced House Bill 5373, which would establish a legal drinking water limit of 5 ng/L for PFOA and perfluorooctane (MHR 2017). Nevertheless, some scientists have argued that the limit should be even lower to protect sensitive subpopulations. For example, Grandjean and Clapp (2015) derived a limit of 1 ng/L based on evidence of immunotoxicity in children, combined with uncertainties from incomplete toxicity testing and lack of epidemiological studies.

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[16] One of the authors of this document, Simona Bălan, co-wrote the Madrid Statement prior to joining DTSC.

PFASs have also become a concern for environmental cleanup across the United States due to contamination from firefighting, manufacturing, and other activities. PFAAs and other PFASs used in AFFFs were found to contaminate surface- and groundwater and soils, and to have numerous adverse impacts. Among others, they can significantly inhibit bacteria performing trichloroethylene dechlorination, thus hindering the bioremediation of soils contaminated with chlorinated solvents (Harding-Marjanovic et al. 2016).

On August 1, 2017, a bipartisan group of eight Democrats and six Republicans in the U.S. House of Representatives signed a letter asking the White House to address PFAS contamination caused by Department of Defense firefighting activities (Kildee et al. 2017). Also in August 2017, the New York State Department of Health and state officials from Alaska, Michigan, New Hampshire, Pennsylvania, and Vermont asked CDC's Agency for Toxic Substances and Disease Registry to launch a longitudinal study of PFAS-impacted communities (Zucker et al. 2017). On December 12, 2017, the Fiscal Year 2018 National Defense Authorization Act was signed into law including a $7 million budget for the CDC and the Agency for Toxic Substances and Disease Registry (ATSDR) to run, in consultation with the Department of Defense, a national study on the health impacts of human exposure to PFASs, especially from contaminated drinking water (U.S. Senate 2017). Washington State currently has two bills under consideration by the House Environment Committee: House Bill 2793, which would prohibit as of 2020 the sale of firefighting foams with intentionally-added PFASs (State of Washington 2018a), and House Bill 2658, which would prohibit as of 2021 the sale of food packaging materials with intentionally-added PFASs (State of Washington 2018b).

On January 30, 2018, South Australia became the first state to ban PFAs-containing firefighting foams (Government of South Australia 2018). European countries are also taking a closer look at PFASs under REACH and national regulatory frameworks. ECHA is currently considering a proposed restriction on long-chain PFAAs, their salts, and precursors (the public consultation period ends June 20, 2018) (ECHA 2018). The European Commission is also currently considering a proposal to add PFASs as a class to the EU Drinking Water Directive (European Commission 2017b). In April 2017, the trade association Swedish Water, representing Sweden's municipal water supply companies, called for a ban on PFASs in all consumer products because of the threat to drinking water supplies (Chemical Watch 2017a). In June 2017, 37 Swedish government agencies and research institutions signed a memorandum of understanding to collaboratively study PFASs and reduce their associated risks (Chemical Watch 2017b). Other EU member countries, including Norway, Denmark, and Germany have ongoing programs to study and regulate the use of PFASs.

Major international companies are beginning to remove PFASs from the products they manufacture or sell. Carpet manufacturers Interface and Tandus Centiva have successfully phased out PFASs in their products without sacrificing performance (Davis 2016; Wilkinson 2016). Several apparel manufacturers, such as Levi Strauss & Co., and manufacturers of furniture and furnishings, such as IKEA and Crate and Barrel, have also phased out PFASs from their products (Blum 2016). In 2016, outdoor gear manufacturer Columbia introduced its first PFAS-free rain jacket (Jurries 2016). In January 2017, Target announced that, as part of its new chemical management policy, it will stop selling textile products with added PFASs by 2022 (Target Inc. 2017). In May 2017, 18 major purchasers including Kaiser Permanente, the City and County of San Francisco, LinkedIn Corporation, and the University of California Santa Cruz signed the Center for Environmental Health (CEH)'s pledge to "preferentially purchase furniture made without toxic chemicals," including "fluorinated stain treatments" (i.e., PFASs) (CEH 2017).

5.2 Key Data Gaps

Physicochemical properties and environmental fate

Data on the physicochemical properties of PFASs are limited to mostly model results, which vary widely because the underlying dataset is based on hydrocarbons. Models and experimental data that can adequately characterize PFASs, especially beyond longer-chain PFCAs and PFSAs, are still lacking (Xiao 2017).

Although a considerable number of studies document levels of some PFASs in the environment and various biological media, the sorption/desorption patterns and mechanisms of transport, transformation, and fate of most PFASs have not been investigated. Consequently, the relationships between PFAS environmental presence, levels in biota and humans, and adverse health impacts related to elevated levels remain unclear. Major data and knowledge gaps remain regarding the sources, transformation, distribution, accumulation, and temporal and spatial trends of various PFASs in wildlife and humans.

Relatively few studies are available on PFASs in sediments, as environmental studies worldwide have focused more attention on the quantities of PFASs in biota and water, and less on sediments and their partitioning behavior and fate (Nakata et al. 2006; Zareitalabad et al. 2013). Data on persistence and half-life estimates in biological and environmental media are also limited, and are often conflicting or inaccurate. Half-lives are estimated based on acute exposures rather than on the low-dose chronic exposures experienced by most people and biota. Data on bioaccumulation and potential for lactational or transplacental transfer are limited to some PFAAs and a few precursors.

Toxicology

Toxicological data are limited to a few PFASs – mostly longer-chain PFAAs such as PFOA and PFOS. The toxicities of polymers, their intermediate degradation products and processing aids are poorly understood. In general, there are very little mammalian toxicological data on PFASs, especially regarding polymers.

PFASs occur in complex mixtures of multiple compounds, yet toxicological endpoints are typically assessed using single PFASs. Most toxicological studies evaluate exposure to single PFASs for 90 days or less, even though humans and animals are chronically exposed to PFAS mixtures over a lifetime. The adverse human and environmental health effects resulting from cumulative exposures to PFAS mixtures have not been well characterized, and toxicological reference values for PFAS mixtures are lacking. Toxicological and epidemiological data clearly indicating safety of cumulative, chronic, and low-dose exposures to the PFASs found in stain- and soil-repellents, particularly for infants and workers, are lacking. Data on potential adverse effects from in-utero exposures in newborns are also scarce.

The mechanism of toxic action of various PFASs and the impact of chain length, functional groups, and other chemical properties on toxicity have yet to be fully understood. Because toxicological studies do not uniformly use experimental animals most sensitive to specific PFASs, and their mechanisms of toxic action are still poorly understood, it is difficult to extrapolate adverse effects from laboratory studies to humans or biota under real-life exposure scenarios. It remains unclear if the levels commonly found in humans and biota are associated with potential health effects or adverse impacts at the population level.

Exposure and adverse impacts

Despite extensive documentation of the widespread environmental contamination with PFASs associated with use in consumer products such as carpets and rugs, longitudinal studies that evaluate aggregate exposures to these chemicals from all sources through different life-stages are still lacking. Current exposure assessments focus on single PFAS chemicals, and studies on simultaneous exposure to PFAS mixtures are rare. PFAS exposure sources in humans and variations in exposure among ethnic groups are also rarely identified (Houde et al. 2006). Although existing monitoring programs may help track the sources and environmental behavior of PFASs, they only add to the large database of environmental concentrations, which alone cannot predict adverse impacts to humans, biota, and the environment.

The relationship between exposure and toxicological outcomes, particularly for PFASs other than PFOA and PFOS, is a major data gap (DeWitt et al. 2015). It remains unclear whether serum concentration or other exposure metrics are useful for estimating exposures and potential adverse impacts. Also missing are early life longitudinal exposure studies measuring internal and external exposure for multiple pathways and PFASs (Winkens et al. 2017).

A lack of comprehensive data on source emissions and the distribution of PFASs through all environmental compartments makes it challenging to accurately estimate cumulative exposures over a lifetime. The frequency, level, and duration of individual or population-level PFAS exposures associated with the use of stain- and soil-repellents in carpets and rugs have not been estimated using either deterministic or probabilistic exposure modeling. The PFASs released from stain- and soil-repellents in carpets and rugs are also found in many other consumer products, are widespread in the environment, and are found in drinking water and food sources. Thus, chronic, daily exposures to PFAS mixtures are highly relevant when evaluating the potential for adverse health effects, but such exposures have yet to be adequately assessed by PFAS chemical manufacturers or public health researchers.

5.3 Conflicting Studies

Physicochemical properties and environmental fate

Data on physicochemical properties and environmental fate range widely in literature depending on the measurement methods or model used for the estimates. Half-lives and environmental persistence values reported in literature sometimes vary by orders of magnitude due to lack of analytical standards and inconsistent experimental approaches and analytical methods (Washington et al. 2015). For example, one study reported a half-life of 1,200 – 1,700 years for the degradation of a side-chain fluoroacrylate in soil (Russell et al.

2008), whereas another estimated half-lives of 33 to 112 years for two side-chain fluoroacrylate polymers in soil (Washington et al. 2015).

Toxicology

As shown in Appendix 3, the evidence of adverse health effects from PFAS exposure in humans is sometimes conflicting (DeWitt 2015). Studies that examine associations between maternal serum, blood, or breast milk PFAS concentrations and child health outcomes sometimes show mixed results (Rappazzo et al. 2017). For example, one study found an association between increased maternal serum PFOS concentrations and increased hyperactivity in children (Hoyer et al. 2015), while another reported a null association between PFOS in cord blood and the neurobehavioral symptoms of attention deficit/hyperactivity disorder (ADHD) (Lien et al. 2013). Studies have also suggested detrimental, beneficial, or no effects on metabolism. For instance, PFAS mechanisms of action can both exacerbate insulin resistance and improve insulin sensitivity (Fleisch et al. 2017).

Exposure and adverse impacts

Academic scientists, environmental consulting firms employed by chemical manufacturers, and government agencies around the world have conducted risk assessments using a variety of methods with the goal of determining whether the levels of PFASs found in different environmental media pose a health risk to humans or animals. Depending on the type of model used to calculate risk, the assumptions underlying the assignment of exposure factors in exposure dose calculations, and the uncertainties in the data used in the dose-response assessment, contradictory conclusions have been drawn, leading to different regulatory levels for allowable concentrations in drinking water.

6 DISCUSSION OF POTENTIAL FOR SIGNIFICANT OR WIDESPREAD ADVERSE IMPACTS

PFASs associated with stain-and soil-repellents in carpets and rugs are released into air, water, and soil during chemical and carpet manufacturing, product use, and disposal (see Section 3.3.2). These extremely persistent chemicals are now global pollutants that can be found in all environmental compartments. Atmospheric and oceanic currents, as well as international trade, have distributed PFASs to areas of the world far from manufacturing sites, including pristine Arctic lands and lakes (Stock et al. 2007). Once PFASs enter the environment, there is no practical way to remove them. Their widespread presence has led to contaminated drinking water and human food chains (Section 3.3.1), resulting in a growing public health concern (APHA 2016).

Exposing humans and aquatic, avian, or terrestrial animal or plant organisms to PFASs associated with carpets and rugs has the potential to contribute to or cause significant and widespread adverse impacts according to multiple factors considered by the Safer Consumer Products regulations.

Given (1) the sheer number of PFASs being produced or explored for use worldwide, (2) the potential for irreversible human and ecological exposures to PFASs throughout the life cycle of carpets and rugs, and (3) the potential for these exposures to contribute to or cause significant or widespread adverse impacts, DTSC proposes to list carpets and rugs containing PFASs as a Priority Product.

6.1 Adverse Impacts Linked to Exposure Potential Hazard Traits, Physicochemical Properties, and Environmental Fate

As detailed in Sections 2.2, 2.3, and 3.3, ***PFASs associated with carpets and rugs show several exposure potential hazard traits:***

- PFASs are either extremely persistent in the environment, or degrade into other extremely persistent PFASs;
- most PFASs display significant mobility in environmental media, which makes them widespread in the environment and in living organisms;
- many members of the PFAS class bioaccumulate significantly in animals or plants, including in foods consumed by humans;
- the PFASs evaluated for this trait display significant lactational or transplacental transfer, which appears to be a widespread issue given the ubiquitous presence of PFASs in breastmilk and cord blood; and
- some PFASs have high global warming potential, or may contribute to global warming by increasing cloud albedo.

Exposing humans and aquatic, avian, or terrestrial animal or plant organisms to PFASs associated with carpets and rugs has the potential to contribute to or cause significant and widespread adverse impacts according to multiple factors considered by the Safer Consumer Products regulations.

Persistence has been called “the most important single criterion affecting chemical exposure and risk via the environment,” because persistent chemicals may travel longer distances and affect a larger population, have increased potential for bioaccumulation across food webs, and are very slow to remove from the environment through natural processes or engineered remediation (Mackay et al. 2014). PFAAs are extremely persistent, with no known natural degradation pathway under environmental conditions; most other PFASs degrade to PFAAs in the environment and within living organisms, i.e., they are PFAA precursors (Buck et al. 2011; D'Eon and Mabury 2011; Krafft and Riess 2015a; Wang et al. 2014a; Washington et al. 2015). The extreme environmental persistence of PFAAs leads to chronic background exposures and increases the potential for significant adverse impacts.

These exposure potential hazard traits stem from the PFASs’ unique physicochemical properties and environmental fate, detailed in Sections 2.1 and 2.2. Once PFASs enter the environment, there is no practical way to remove them. Their widespread presence has led to contaminated food chains and drinking water (Lindstrom et al. 2011b). The accumulation of these chemicals in the sediments of lakes, rivers, harbors, oceans (Xiao 2017), and drinking water sources (Cousins et al. 2016) may pose significant environmental and human health hazards (Xiao 2017).

6.2 Adverse Impacts Linked to Toxicological Hazard Traits

As detailed in Section 2.3 and Appendix 3, ***PFASs associated with carpets and rugs show evidence for the following toxicological hazard traits:***

- carcinogenicity;
- developmental toxicity;
- reproductive toxicity;
- cardiovascular toxicity;
- endocrine toxicity;
- hematotoxicity;
- hepatotoxicity and digestive system toxicity;
- immunotoxicity;
- nephrotoxicity and other urinary system toxicity;
- neurodevelopmental toxicity; and
- respiratory toxicity.

Increasing reports of toxicity in humans and experimental animals, sometimes at or below current exposure levels, is leading to growing public health concern (APHA 2016) and expanded regulatory interest (Corsini et al. 2014; Zushi et al. 2012). The adverse impacts can be widespread and significant. For instance, the total cost of hospitalization for medical concerns and loss of IQ points due to PFOA-attributable low weight births between 2003 and 2014 in the United States was estimated at $13.7 billion (Malits et al. 2017).

6.3 Adverse Impacts Linked to Environmental Hazard Traits

As detailed in Section 2.3 and 2.5***, many PFASs associated with carpets and rugs show the following significant environmental hazard traits:***

- phytotoxicity, especially in green algae, which are the foundation of the aquatic food chain (Casal et al. 2017; Ding et al. 2012a); and
- terrestrial ecotoxicity and aquatic toxicity, including impairment of wildlife developmental, growth, reproduction, or survival.

Discharges of PFAS-contaminated WWTP effluents into streams and rivers has led to contamination of water and sediments at concentrations considered potentially hazardous to wildlife and a potential threat to biodiversity (Rodriguez-Jorquera et al. 2016). PFAAs bioaccumulate in phyto- and zooplankton and are toxic to green algae, which are the foundation of the aquatic food chain (Casal et al. 2017; Ding et al. 2012b). In general, aquatic invertebrates, which comprise the largest percentage of animal biomass in aquatic ecosystems, are particularly susceptible to adverse impacts because they are often concomitantly exposed to complex mixtures of waterborne contaminants (Bradley et al. 2017). Water-filtering species of aquatic animals are the most highly exposed to PFAAs (Renzi et al. 2013). The ubiquitous presence of PFASs in aquatic environments and the limited toxicological knowledge about cumulative impacts to aquatic species have led to increasing concern about the potential for irreversible adverse aquatic impacts (Scheringer et al. 2014).

The increasing concentrations of PFASs associated with stain- and soil-repellents found in environmental media, animal tissues, and biota can contribute to or cause significant or widespread adverse impacts in people and the environment.

6.4 Adverse Impacts Linked to Aggregate Effects

Stain- and soil-repellents contain PFAS mixtures that contaminate the environment, leading to chronic lifelong exposures in humans and biota. Toxicological and epidemiological data clearly indicating the safety of aggregate, chronic, and low-dose exposures to PFASs found in stain- and soil-repellents, particularly in infants and workers, are lacking. Traditionally, the science of toxicology has been predicated on the principle that “the dose makes the poison.” Increasing aggregate exposures to mixtures of extremely persistent PFASs in contaminated media could result in more frequent and higher exposure doses, and increase the potential for significant adverse toxicological effects. Therefore, the increasing concentrations of PFASs associated with stain- and soil-repellents found in environmental media, animal tissues, and biota can contribute to or cause significant or widespread adverse impacts in people and the environment.

6.5 Adverse Impacts Linked to the Human Populations, or to the Aquatic, Avian, or Terrestrial Animal or Plant Organisms Exposed to PFASs

As detailed in Section 2.5, ***PFASs have the potential to cause significant and widespread adverse impacts to:***

- sensitive subpopulations including fetuses, infants, children, pregnant women, carpet installers, carpet cleaners, carpet retail sector workers, and people with certain preexisting conditions;
- environmentally sensitive habitats; and
- threatened and endangered species.

Fetuses, infants, toddlers, and young children experience higher relative exposure levels and are more vulnerable to the effects of toxicants; carpet installers, carpet cleaners, and carpet retail workers can also experience high exposures on the job, while individuals with certain preexisting conditions (e.g., elevated cholesterol, high blood pressure, poor kidney function) may be especially sensitive to PFASs (see Sections 2.5.2 and 3.3.3). Pregnant women's exposure to PFASs is of concern due to transplacental and lactational transfer to the offspring (see Sections 2.3.1 and 3.3.1). Endangered and threatened species in California are vulnerable to contamination of waterways and food webs with PFASs from stain- and soil-repellent carpets and rugs.

7 ALTERNATIVES

Reference: CAL. CODE REGS. *tit. 22, § 69503.2(b)(3).*

Given the potential for exposures and significant or widespread adverse impacts, replacing currently used PFASs in carpets and rugs with other members of the PFAS class could constitute a regrettable substitution. Hence, this proposal covers the entire class of PFASs in carpets and rugs. Several chemical and non-chemical options are available to meet the stain- and soil-resistance requirements for carpets and rugs. According to a patent filed by INVISTA North America S.a.r.l. for clay nanoparticle and wax-based "liquid and soil repellent compositions for fibers," including for carpets and rugs (Iverson et al. 2017), "a fluorochemical is not needed to achieve the desired soil and liquid repellent properties."

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7.1 Chemical Alternatives

Sulfonation

Acid-based stains, the most problematic for three-dimensional structures like carpets and rugs (Cooke and Weigmann 1990), can be effectively prevented via sulfonation. Aromatic and aliphatic sulfonates have been used since 1986 as stain blockers, in addition to PFASs, to increase the nylon carpet fibers' resistance to acid stains from foodstuff (Cooke and Weigmann 1990; Dusaj 1988; Schindler and Hauser 2004).

Sulfonation can be achieved through various methods (Hangey et al. 1989; Summers 1999). The Healthy Building Network researched several patents and reported that the sulfonation process typically uses a metal salt of sulfoisophthalic acid, for example salts of 5-sulfoisophthalic acid, diaminium bis-3,5-dicarboxybenzensulfonate, and tri-diaminium bis-3,4-dicarboxybenzensulfonate, which "are not associated with significant health or environmental hazards, but they have not been fully assessed" (Vallette et al. 2017).

Sulfonation blocks all fiber dye sites with colorless sulfonates, making the carpet or rug impossible to stain by acidic colorants (McSheehy 2017). However, this also limits the color options since the fibers cannot be dyed, decreases softness, and may increase product cost (Davis 2016). The carpet manufacturer Interface, who uses this technology, says that there's "no need for an additional topical treatment" (Interface 2017). Universal Fibers, one of Interface's suppliers, produces PFAS-free carpet yarn using a sulfonated nylon copolymer (Universal Fibers unknown date).

Other non-fluorinated chemical alternatives

PFAS-free chemical alternatives that prevent soils from adhering and staining agents from penetrating the fiber surface are becoming available. DTSC does not currently have complete information on the chemical content of these alternatives, and has not evaluated them for safety.

Tandus-Centiva, a carpet manufacturer serving 80 percent of available educational space in California, has found that the performance of these fluorine-free alternatives is "equivalent or superior to the fluorinated treatments."

Examples include Invista's non-fluorinated Duratech® and Tandus Centiva's Eco-Ensure. Tandus-Centiva, a carpet manufacturer serving 80 percent of available educational space in California, has found that the performance of these fluorine-free alternatives is "equivalent or superior to the fluorinated treatments" (Winkens et al. 2017). Eco-Ensure qualified for Gold Level Cradle to Cradle Certification,[17] and Duratech® is under evaluation (Winkens et al. 2017).

A patent from February 2017 describes "fluorine-free compositions rendering textile articles, such as carpets and other textile floor coverings made from synthetic fibres or natural fibres which are water repellent, soils resistant and stain resistant," based on (a) a nanoparticle silicate clay, (b) an anionic acrylic-based copolymer binding agent, and (c) water (Invista North America 2017).

Universal Fibers uses a sulfonated nylon copolymer (i.e., sulfonation) to prevent acid dyes and stains, and a low-soil finish technology based on amide functional waxes to prevent soiling (McSheehy 2017; Universal Fibers unknown date).

The Danish Environmental Protection Agency reviewed a non-fluorinated repellent called NEOSEED from Nicca Chemical Co., with potential applications including carpet, and ingredients such as a non-ionic polymer, ester compound, hydrocarbon compound, organic solvent, and water. Insufficient ingredient information was available for a health hazard assessment (Danish Environmental Protection Agency 2015b).

Other potential alternatives to PFASs in carpet treatments may include siloxane and silicone polymers, hydrocarbons (e.g., paraffin waxes), polyurethanes, and dendrimers (hydrocarbon- or polyurethane-based) (Danish Environmental Protection Agency 2015b; KEMI 2015; U.S. EPA 2012). These are all durable water repellents without oil and alcohol repellency. Some non-fluorinated (meth)acrylates/urethanes have stain release properties for both oil- and water-based stains (Danish Environmental Protection Agency 2015b). For example, a copolymer of ethylenesulfonic acid with methyl methacrylate has been marketed for use as stain proofing agent for nylon fibers (WeylChem Höchst GmbH 2017).

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[17] The Cradle to Cradle Certified™ Product Standard guides designers and manufacturers through continuous product improvement with respect to material health, material reutilization, renewable energy and carbon management, water stewardship, and social fairness. For each of these five categories, the product receives an achievement level of Basic, Bronze, Silver, Gold, or Platinum. The lowest achievement level represents the product's overall mark. More information available at http://www.c2ccertified.org/get-certified/product-certification. Accessed January 5, 2018.

A Danish Environmental Protection Agency review concluded that the main ingredients in paraffin-based repellents are mostly harmless, readily biodegradable, non-bioaccumulative, and non-toxic to aquatic organisms; however, some products may contain potentially hazardous ingredients or impurities such as isocyanates, dipropylene glycol, and metal salts (Danish Environmental Protection Agency 2015b). Similarly, they found that most silicones used in textile impregnation are inert, generally without adverse effects, but toxic manufacturing intermediaries such as D4 (suspected of damaging fertility) and D5 (potential carcinogen) may be present at trace levels. For dendrimer-based repellents, they lacked sufficient information for an assessment, but found that some products may contain potentially toxic siloxanes, cationic polymers, isocyanates, or powerful irritating organic acids.

7.2 Non-Chemical Alternatives

PFAS treatments are used in carpets mainly for stain- and soil-resistance. Soil-resistance can also be achieved without chemical treatment by modifying the shape of the yarn. Thicker lobes make the carpet or rug more durable and inherently soil-resistant. The carpet or rug looks good longer, but is less soft (Davis 2016).

Some carpet fibers, such as polypropylene fibers, are inherently stain-resistant and may not need chemical treatment (Davis 2016). Wool fibers are naturally hydrophobic and can resist water-based stains, but not oil-based stains or soil (Johnson and Russell 2009).

Fibers made of polyethylene terephthalate (PET), also known as polyester, are naturally stain resistant. A comparative study found that recycled PET carpet fibers performed better in appearance retention tests compared to recycled and new nylon carpet fibers (Watson and Warnock 2003).

Polytrimethylene terephthalate (PTT) fibers have superior stain resistance compared to nylon, and are similarly durable, resilient, and soft (Bhattad et al. 2013). In the early 2000's, DuPont's Bio-Based Materials business unit developed Sorona®, a family of PTT polymers. According to the manufacturer, Sorona® "fiber is highly resistant to most stains without the need for surface treatment with additives or coatings" (Kurian 2005).

8 OTHER REGULATORY PROGRAMS

Reference: CAL. CODE REGS. tit. 22, § 69503.2(b)(2).

DTSC has identified the following state and federal programs that regulate the product or the Candidate Chemical in the product to protect public health and the environment. The proposed regulation does not duplicate or conflict with these regulations.

8.1 Applicable California State Laws and Regulations

No California state laws or regulations currently address the use of PFASs in carpets and rugs. However, a few current laws or regulations apply to carpet recycling.

AB 2398, California's Law for Carpet Stewardship

California AB 2398 was signed into law September 30, 2010 to increase the diversion and recycling of carpet in the state of California. The law generates funding to meet its goals through an assessment on each square yard of carpet sold in California. It does not address the use of PFASs in carpets and rugs.

AB 1158

California AB 1158 was signed into law October 14, 2017, setting a goal for the state to achieve a 24 percent recycling rate for postconsumer carpet by January 1, 2020, and to meet and exceed that rate continually thereafter. It requires a carpet stewardship plan to achieve 24 percent recycling rate for postconsumer carpet by January 2020, and quantifiable 5-year and annual goals. It does not address the use of PFASs in carpets and rugs.

8.2 Applicable Federal Laws and Regulations

Toxic Substances Control Act (TSCA)

Section 5 of TSCA authorizes U.S. EPA to issue Significant New Use Rules (SNURs) for new or existing chemicals used in a significant new way. A SNUR requires companies to notify U.S. EPA at least 90 days prior to manufacturing, importing, or processing substances for a significant new use, and submit a notification including information about the chemical's identity, physical characteristics, processing and use, and available toxicity data. U.S. EPA has 90 days to evaluate the new use and can request more data, prohibit or limit the manufacture, or allow the use. The following SNURs relate to PFASs:

- December 9, 2002 – SNUR regarding any future manufacture (including imports) of 75 PFASs specifically included in the 2000-2002 voluntary phaseout of PFOS by 3M (U.S. EPA 2002a);

- March 11, 2002 – SNUR regarding any future manufacture (including imports) of 13 PFASs specifically included in the 2000-2002 voluntary phaseout of PFOS by 3M (U.S. EPA 2002b);
- October 9, 2007 – SNUR for 183 PFASs believed to no longer be manufactured, imported, or used in the United States (U.S. EPA 2007);
- September 30, 2013 – SNUR requiring companies to report their intent to manufacture certain PFOA-related chemicals to treat carpets, and to import carpets containing these PFASs (U.S. EPA 2013); and
- (Proposed) January 21, 2015 – proposed SNUR affecting manufacturers (including importers) of PFOA and PFOA-related chemicals, including as part of articles, and processors of these chemicals (U.S. EPA 2015a).

In 2006, U.S. EPA developed a 2010/2015 Stewardship Program for reducing emissions of PFOA, its precursors, and related higher homologues (U.S. EPA 2010a). Through a Memorandum of Understanding (MOU) with U.S. EPA, eight major U.S. manufacturers agreed to voluntarily eliminate PFOA in their emissions and products by 2015. Participating companies include: Arkema, Asahi, BASF Corporation, Clariant, Daikin, 3M/Dyneon, DuPont, and Solvay Solexis.

Since 2000, U.S. EPA has been reviewing hundreds of substitutes for PFOA, PFOS, and other longer-chain PFASs, particularly regarding their toxicity, fate, and bioaccumulation under the New Chemicals Program (U.S. EPA 2017b). For many PFASs, U.S. EPA has been using TSCA §5(e) Consent Orders to require testing while allowing production and use. U.S. EPA is also investigating substitutes for certain direct uses of PFOA.

On January 27, 2010, U.S. EPA amended the Polymer Exemption Rule for new chemicals under TSCA to exclude certain side-chain fluorinated polymers, due to potential risk to human health or the environment (U.S. EPA 2002b).

8.3 Applicable International Treaties or Agreements with the Force of Domestic Law

Stockholm Convention on Persistent Organic Pollutants

There are no applicable international treaties or agreements with the force of domestic law. However, the Stockholm Convention on Persistent Organic Pollutants (POPs), which the United States has not ratified, is a global treaty to protect human health and the environment from chemicals that remain intact in the environment for long periods, become widely distributed geographically, accumulate in the fatty tissue of humans and wildlife, and have harmful impacts on human health or the environment. In 2009, PFOS, its salts, and perfluorooctane sulfonyl fluoride were added to Annex B of the Stockholm Convention, which restricts the production and use, as well as import and export, of listed POPs (SSCCH 2017). Production and use of these chemicals is still allowed for acceptable purposes after Annex B listing, which included use in carpets until 2015.

The EU Council has also submitted a proposal to list PFOA, its salts, and related compounds in Annex A of the Stockholm Convention (European Commission 2015). During its thirteenth meeting on October 17-20, 2017 (UNEP/POPS/POPRC 2017), the POPs Review Committee recommended that the Conference of the Parties list

PFOA, its salts, and PFOA-related compounds in Annex A or B to the Stockholm Convention, with specific exemptions. The recommendations will be considered at the ninth meeting of the Conference of the Parties to the Stockholm Convention, in Spring 2019. Also at the October 2017 meeting, the POPs Review Committee agreed with a proposal by Norway to list PFHxS, its salts, and PFHxS-related compounds in Annexes A, B, and/or C to the Convention, and has established a working group to review the proposal further and prepare a draft risk profile.

ABBREVIATIONS

PFAS (sub)classes	
diPAP(s)	Fluorotelomer phosphate diester(s)
FASA(s)	Perfluoroalkane sulfonamide(s)
FASE(s)	Perfluoroalkane sulfonamide ethanol(s)
FAS(M)AC(s)	Perfluoroalkane sulfonamidoethyl (meth)acrylate(s)
FOSE(s)	Perfluorooctane sulfonamide ethanol(s)
FTAC(s)	Fluorotelomer acrylate(s)
FTAL(s)	Saturated fluorotelomer aldehydes
FTCA(s)	Saturated fluorotelomer carboxylic acids
FTEO(s)	Fluorotelomer ethoxylates
FTEOC(s)	Fluorotelomer ethoxycarboxylate(s)
FTI(s)	Fluorotelomer iodide(s)
FTMAC(s)	Fluorotelomer methacrylate(s)
FTO(s)	Fluorotelomer olefin(s)
FTOHs	Fluorotelomer alcohol(s)
FTS(s)	Fluorotelomer sulfonic acid(s)
FTUAL(s)	Unsaturated fluorotelomer aldehydes
FTUCA(s)	Unsaturated fluorotelomer carboxylic acids
PAP(s)	Polyfluoroalkyl phosphate(s)
PASF	Perfluoroalkane sulfonyl fluoride
PFAA(s)	Perfluoroalkyl acid(s)
PFAI(s)	Perfluoroalkyl iodide(s)
PFAS(s)	Perfluoroalkyl and polyfluoroalkyl substance(s)
PFECA(s)	Perfluoroether carboxylic acid(s)
PFESA(s)	Perfluoroether sulfonic acid(s)
PFCA(s)	Perfluoroalkyl carboxylic acid(s)
PFPA(s)	Perfluoroalkyl phosphonic acid(s)
PFPE(s)	Perfluoropolyether(s)
PFPiA(s)	Perfluoroalkyl phosphinic acid(s)
PFSA(s)	Perfluoroalkyl sulfonic acid(s)
PFSI(s)	Perfluoroalkyl sulfinate(s)
PFSiA(s)	Perfluoroalkyl sulfinic acid(s)

Individual PFASs	
APFO	Ammonium perfluorooctanoate
FBSA	Perfluorobutane sulfonamide
PASF	Perfluoroalkane sulfonyl fluoride

PFBA	Perfluorobutanoic acid
PFBPA	Perfluorobutyl phosphonic acid
PFBPiA	Perfluorobutyl phosphinic acid
PFBS	Perfluorobutane sulfonic acid
PFDA	Perfluorodecanoic acid
PFDS	Perfluorodecane sulfonic acid
PFDoA	Perfluorododecanoic acid
PFDODPiA	Perfluorododecyl phosphinic acid
PFDPA	Perfluorodecyl phosphonic acid
PFDPiA	Perfluorodecyl phosphinic acid
PFHpA	Perfluoroheptanoic acid
PFHxA	Perfluorohexanoic acid
PFHxPA	Perfluorohexyl phosphonic acid
PFHxPiA	Perfluorohexyl phosphinic acid
PFHxS	Perfluorohexane sulfonic acid
PFNA	Perfluorononanoic acid
PFOA	Perfluorooctanoate or perfluorooctanoic acid
PFOPA	Perfluorooctyl phosphonic acid
PFOPiA	Perfluorooctyl phosphinic acid
PFOS	Perfluorooctane sulfonate or perfluorooctane sulfonic acid
PFPeA	Perfluoropentanoic acid
PFTeDA	Perfluorotetradecanoic acid
PFTrDA	Perfluorotridecanoic acid
PFUnA	Perfluoroundecanoic acid
PTFE	Polytetrafluoroethylene
PVDF	Polyvinylidene fluoride
PVF	Polyvinyl fluoride

Other abbreviations used in this document	
ADHD	Attention deficit/hyperactivity disorder
AFFF(s)	Aqueous firefighting foam(s)
ATSDR	Agency for Toxic Substances and Disease Registry
BAF(s)	Bioaccumulation factor(s)
BCF(s)	Bioconcentration factor(s)
BMF(s)	Biomagnification factor(s)
CDC	Centers for Disease Control and Prevention
CECBP	California Environmental Contaminant Biomonitoring Program
CEH	Center for Environmental Health
CFCs	Chlorofluorocarbons

CPSC	Consumer Product Safety Commission
DDT	Dichlorodiplenyltricloroethane
DTSC	Department of Toxic Substances Control
ECHA	European Chemicals Agency
EPA	Environmental Protection Agency
F	Fluorine
GPC	Global Product Classification
MOU	Memorandum of Understanding
MRL(s)	Minimum Reporting Level(s)
NAICS	North American Industrial Classification System
NHANES	National Health and Nutrition Examination Survey
OECD	Organisation for Economic Cooperation and Development
OEHHA	Office of Environmental Health Hazard Assessment
PBT	Persistent, bioaccumulative and toxic
PCBs	Polychlorinated biphenyls
PET	Polyethylene terephthalate PET
PIGE	Particle-induced γ-ray emission
POPs	Persistent organic pollutants
PPAR	Peroxisome proliferator-activated receptor
PTT	Polytrimethylene terephthalate
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
RfD	Reference dose
SCP	Safer Consumer Products
SNUR	Significant New Use Rule
SVHC	Substances of Very High Concern
TDI	Tolerable daily intake
TSCA	Toxic Substances Control Act
UCMR3	Unregulated Contaminant Monitoring Rule 3
U.S.	United States
vPvB	Very persistent, very bioaccumulative
WWTP	Wastewater treatment plant

Units	
°C	Degrees Celsius
Pa	Pascal
kg	Kilograms
m	Meters
m^3	Meters cubed
$Pa \cdot m^3/mol$	Pascals times meters cubed per mol

kJ/mol	Kilojoules per mol
µg/m^2	Micrograms per meter squared
ng/m^3	Nanograms per meter cubed
nmol/cm^2	Nanomols per centimeter squared
L/kg	Liters per kilogram
g/L	Grams per liter
ng/L	Nanograms per liter
ng/mL	Nanograms per milliliter
ng/g	Nanograms per gram
µg/kg	Micrograms per kilogram
ng/kg	Nanograms per kilogram
ng/day	Nanograms per day
mg/day	Milligrams per day
mg/kg/day	Milligrams per kilogram per day
kg/yr	Kilograms per year

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APPENDIX 1 – ADDITIONAL PFAS CANDIDATE CHEMICALS DATABASE LISTINGS

Chemical Name	CAS RN	Authoritative List	Hazard Traits
10:2 Fluorotelomer acetate	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
10:2 Fluorotelomer acrylate	17741-60-5	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
10:2 Fluorotelomer carboxylic acid	53826-13-4	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
10:2 Fluorotelomer unsaturated carboxylic acid	70887-94-4	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
2-(N-Ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH)	2991-50-6	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
2-(N-Methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH)	2355-31-9	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy) propanoic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
4:2 Fluorotelomer sulfonic acid	757124-72-4	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
5:3 Fluorotelomer carboxylic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
6:2 Fluorotelomer acetate	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
6:2 Fluorotelomer acrylate	17527-29-6	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
6:2 Fluorotelomer carboxylic acid	53826-12-3	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
6:2 Fluorotelomer phosphate diester	57677-95-9	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
6:2 Fluorotelomer phosphate monoester	57678-01-0	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
6:2 Fluorotelomer sulfonic acid	27619-97-2	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
6:2 Fluorotelomer unsaturated carboxylic acid	70887-88-6	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
6:2/8:2 Fluorotelomer phosphate diester	943913-15-3	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
7:3 Fluorotelomer carboxylic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
7:3 Fluorotelomer carboxylic acid	No CAS RN	ATSDR Neurotoxicants (3/3/2011)	Immunotoxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
8:2 Fluorotelomer acetate	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
8:2 Fluorotelomer acrylate	27905-45-9	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
8:2 Fluorotelomer carboxylic acid	27854-31-5	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
8:2 Fluorotelomer phosphate diester	678-41-1	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
8:2 Fluorotelomer sulfonic acid	39108-34-4	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
8:2 Fluorotelomer unsaturated carboxylic acid	70887-84-2	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
Ammonium 4,8-dioxa-3H-perfluorononanoate (ADONA)	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Ammonium nonadecafluorodecanoate	3108-42-7	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence
Ammonium pentadecafluorooctanoate (APFO)	3825-26-1	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence
Ammonium pentadecafluorooctanoate (APFO)	3825-26-1	EC Annex VI CMRs – Cat. 1B (7/19/2016)	Reproductive Toxicity
Bis(perfluorohexyl) phosphinic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Bis(perfluorooctyl) phosphinic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Sum of branched perfluorooctanoic isomers	No CAS RN	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Heptacosafluorotetradecanoic acid	376-06-7	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence
Nonadecafluorodecanoic acid, sodium salt	3830-45-3	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence
n-Perfluorooctane sulfonic acid (n-PFOS) and n-Perfluorooctanoic acid (n-PFOA)	No CAS RN	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Pentacosafluorotridecanoic acid	72629-94-8	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence

Chemical Name	CAS RN	Authoritative List	Hazard Traits
Perfluorobutane sulfonic acid (PFBS)	375-73-5	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorodecanoic acid (PFDeA)	335-76-2	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence
Perfluorodecanoic acid (PFDeA)	335-76-2	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorodecylphosphonic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluorododecanoic acid (PFDoA)	307-55-1	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence
Perfluorododecanoic acid (PFDoA)	307-55-1	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluoroethylcyclohexane sulfonic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluoroheptane sulfonic acid	375-92-8	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluoroheptanoic acid (PFHpA)	375-85-9	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
Perfluorohexadecanoic acid	67905-19-5	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluorohexane sulfonic acid (PFHxS)	355-46-4	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorohexanoic acid (PFHxA)	307-24-4	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluorohexyl-perfluorooctylphosphinic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluorohexylphosphonic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Sum of perfluoromethylheptane sulfonic acid isomers	No CAS RN	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorononane sulfonic acid	474511-07-4	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
Perfluorononanoic acid (PFNA)	375-95-1	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorononanoic acid (PFNA)	375-95-1	EC Annex VI CMRs – Cat. 1B (7/19/2016)	Reproductive Toxicity
Perfluorooctadecanoic acid	16517-11-6	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluorooctane sulfonamide (PFOSA)	754-91-6	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorooctane sulfonic acid (PFOS)	1763-23-1	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorooctane sulfonic acid (PFOS)	1763-23-1	EC Annex VI CMRs – Cat. 1B (7/19/2016)	Reproductive Toxicity
Perfluorooctane sulfonic acid (PFOS)	1763-23-1	OSPAR Priority Action Part A (1/1/2002)	Bioaccumulation, Environmental Persistence
Perfluorooctane sulfonic acid (PFOS)	1763-23-1	WA PBT (1/3/2006)	Bioaccumulation, Environmental Persistence
Perfluorooctanoic acid (PFOA)	335-67-1	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Perfluorooctanoic acid (PFOA)	335-67-1	EC Annex VI CMRs – Cat. 1B (7/19/2016)	Reproductive Toxicity
Perfluorooctanoic acid (PFOA)	335-67-1	EC PBTs (1/12/2017)	Bioaccumulation, Environmental Persistence
Perfluorooctanoic acid (PFOA)	335-67-1	IARC Carcinogens – 2B (5/19/2017)	Carcinogenicity
Perfluorooctylphosphonic acid	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
Perfluoropentane sulfonic acid	2706-91-4	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluoropentanoic acid	2706-90-3	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluorotetradecanoic acid	376-06-7	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluorotridecanoic acid	72629-94-8	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Perfluoroundecanoic acid (PFUA)	2058-94-8	CDC 4th National Exposure Report (1/1/2017)	Developmental Toxicity, Hepatotoxicity, Digestive System Toxicity, Reproductive Toxicity
Potassium 1,1,2,2,-tetrafluoro-2-({1,1,1,2,3,3-hexafluoro-3-[(trifluorovinyl)oxy]-2-propanyl}oxy)ethanesulfonate tetrafluoroethene	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity
Sodium 2-(N-ethylperfluorooctane-1-sulfonamido)ethyl phosphate	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity

Chemical Name	CAS RN	Authoritative List	Hazard Traits
Sodium bis-[2-(N-ethylperfluorooctane-1-sulfonamido)ethyl] phosphate	No CAS RN	CECBP - Priority Chemicals (12/22/2015) Priority Chemicals that are identified under the California Environmental Contaminant Biomonitoring Program	Bioaccumulation, Carcinogenicity, Developmental Toxicity, Endocrine Toxicity, Hepatotoxicity, Digestive System Toxicity, Immunotoxicity, Reproductive Toxicity

APPENDIX 2 – EXAMPLES OF PERFLUOROALKYL ACIDS (PFAAS)

Sources: Supplemental information in Wang et al. (2011) and Sun et al. (2016)

Perfluorinated carbons #	Chemical Name	Abbreviation	CAS Number
Perfluoroalkyl carboxylic acids (PFCAs)			
3	Perfluorobutanoic acid	PFBA	375-22-4
4	Perfluoropentanoic acid	PFPeA	2706-90-3
5	Perfluorohexanoic acid	PFHxA	307-24-4
6	Perfluoroheptanoic acid	PFHpA	375-85-9
7	Perfluorooctanoic acid	PFOA	335-67-1
8	Perfluorononanoic acid	PFNA	375-95-1
9	Perfluorodecanoic acid	PFDA	335-76-2
10	Perfluoroundecanoic acid	PFunA	2058-94-8
11	Perfluorododecanoic acid	PFDoA	307-55-1
12	Perfluorotridecanoic acid	PFTrDA	72629-94-8
13	Perfluorotetradecanoic acid	PFTeDA	376-06-7
14	Perfluoropentadecanoic acid	PFPeDA	141074-63-7
Perfluoroalkyl sulfonic acids (PFSAs)			
4	Perfluorobutane sulfonic acid	PFBS	375-73-5
6	Perfluorohexane sulfonic acid	PFHxS	355-46-4
7	Perfluoroheptane sulfonic acid	PFHpS	357-92-8
8	Perfluorooctane sulfonic acid	PFOS	1763-23-1
10	Perfluorodecane sulfonic acid	PFDS	333-77-3
Perfluoroalkyl sulfinic acids (PFSiAs)			
4	Nonafluoro-1-butanesulfinic acid	PFBSi	34642-43-8
6	Tridecafluoro-1-hexanesulfinic acid	PFHxSi	115416-67-6
8	Heptadecafluoro-1-octanesulfinic acid	PFOSi	647-29-0
10	Heneicosafluoro-1-decanesulfinic acid	PFDSi	558-98-5
Perfluoroalkyl phosphonic acids (PFPAs)			
4	Perfluorobutyl phosphonic acid	PFBPA	52299-24-8
6	Perfluorohexyl phosphonic acid	PFHxPA	40143-76-8
8	Perfluorooctyl phosphonic acid	PFOPA	40143-78-0
10	Perfluorodecyl phosphonic acid	PFDPA	52299-26-0
12	Perfluorododecyl phosphonic acid	PFDoDPA	63225-55-8
Perfluoroalkyl phosphinic acids (PFPiAs)			
8	Perfluorobutyl phosphinic acid	PFBPiA	52299-25-9
12	Perfluorohexyl phosphinic acid	PFHxPiA	40143-77-9
16	Perfluorooctyl phosphinic acid	PFOPiA	40143-79-1
20	Perfluorodecyl phosphinic acid	PFDPiA	52299-27-1
24	Perfluorododecyl phosphinic acid	PFDODPiA	63225-54-7
Perfluoroether carboxylic acids (PFECAs)			
2	Perfluoro-2-methoxyacetic acid	PFMOAA	674-13-5

Perfluorinated carbons #	Chemical Name	Abbreviation	CAS Number
3	Perfluoro-3-methoxypropanoic acid	PFMOPrA	377-73-1
3	Perfluoro(3,5-dioxahexanoic) acid	PFO_2HxA	39492-88-1
4	Perfluoro-4-methoxybutanoic acid	PFMOBA	863090-89-5
4	Perfluoro(3,5,7-trioxaoctanoic) acid	PFO_3OA	39492-89-2
5	Perfluoro-2-propoxypropanoic acid	GenX (PFPrOPrA)	62037-80-3
5	Perfluoro(3,5,7,9-tetraoxadecanoic) acid	PFO_4DA	39492-90-5
6	3H-Perfluoro-3-[(3-methoxy-propoxy) propanoic acid]	ADONA	958445-44-8
9	Perfluoro triether carboxylic acid	PFTECA	329238-24-6
10	Perfluoro[(2-ethyloxy-ethoxy)acetic acid] (in ammonium salt form)	PFEEA	908020-52-0
11	Not available	F-53	754925-54-7
12	Not available	F-53B	73606-19-6
13	1-Butanesulfonamide, N,N-phosphinicobis(oxy-2,1-ethanediyl)bis1,1,2,2,3,3,4,4,4-nonafluoro-N-methyl-	PFBSaPA	120945-47-3
14	N-[3-(Dimethylamino)propyl]-3,3,4,4,5,5,6,6,7,7,8,8,8-tridecafluorooctanesulphonamide N-oxide	Forafac	80475-32-7
15	N-(3-Trimethoxysilylpropyl)Perfluorohexanamide	RM720	154380-34-4

APPENDIX 3 – PFAS HAZARD TRAITS

Health Hazard Category	Summary of Findings	Positive or Negative Adverse Impact Finding	Reference
Carcinogenicity	Limited evidence in humans for the carcinogenicity of perfluorooctanoic acid (PFOA). A positive association was observed for cancers of the testis and kidney. Overall evaluation: Perfluorooctanoic acid (PFOA) is possibly carcinogenic to humans (Group 2B). Review by authoritative body.	Positive	IARC (2016)
	Suggestive evidence of carcinogenic potential for PFOA. Epidemiology studies demonstrate an association of serum PFOA with kidney and testicular tumors among highly exposed members of the general population. Two chronic bioassays of PFOA support a positive finding for its ability to be tumorigenic in one or more organs of rats, including the liver, testes, and pancreas. Review by authoritative body.	Positive	U.S. EPA (2016b)
	Suggestive evidence of carcinogenic potential for PFOS. In a chronic oral toxicity and carcinogenicity study of PFOS in rats, liver and thyroid tumors, and mammary fibroadenomas were identified. Review by authoritative body.	Positive	U.S. EPA (2016c)
	Positive association of kidney cancer with PFOA exposure as observed in high PFOA exposure cohorts in occupational and community settings.	Positive	Barry et al. (2013); C8 Science Panel (2012); Steenland and Woskie (2012); Vieira et al. (2013)
	Positive association of testicular cancer with PFOA exposure in high PFOA exposure cohort.	Positive	Barry et al. (2013); C8 Science Panel (2012)
	Increased risk of thyroid cancer in occupational cohort exposure to PFOA.	Positive	Barry et al. (2013)
	Possible increase in prostate cancer mortality in workers with 10 years occupational exposure to PFOA.	Positive	Gilliland and Mandel (1993)

	Higher PFOA serum levels associated with testicular, kidney, prostate and ovarian cancer, and non-Hodgkin lymphoma. (High PFOA exposure cohort)	Positive	Lundin et al. (2009); Vieira et al. (2013)
	Higher risk of prostate cancer in individuals with PFAS levels above median and a first degree relative with prostate cancer. Positive of genetic susceptibility. PFHxS showed a statistically significant interaction.	Positive	Hardell et al. (2014)
	Weak positive association of breast cancer risk with PFOS exposure in Danish National Birth Cohort.	Positive	Bonefeld-Jorgensen et al. (2014)
	Tumor triad in male rats (liver, Leydig cells, pancreatic) dosed with APFO.	Positive	Biegel et al. (2001); Sibinski (1987)
	Increase in mammary tumors in rats with 2-year PFOA oral administration.	Positive	Sibinski (1987)**
	Ammonium 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)-propanoate was tested for chronic toxicity and carcinogenicity in Sprague–Dawley rats. Level of evidence sufficient to show that the findings of liver, pancreas, testicular tumors are relevant for humans.	Positive	Beekman et al. (2016); Rae et al. (2015)
	General Danish population study did not indicate increased incidence of prostate, bladder, pancreatic, liver cancers.	Negative	Eriksen et al. (2009)
	No association between PFOA levels and breast cancer risk in mothers from Danish National Birth Cohort.	Negative	Bonefeld-Jorgensen et al. (2014)
	No increased prostate cancer risk compared to controls (Gleason score and prostate-specific antigen (PSA) levels).	Negative	Hardell et al. (2014)
	No association of occupational APFO exposure with liver, pancreatic or testicular cancers.	Negative	Lundin et al. (2009)
	Study reports lack of concordance between community exposures and occupational exposures one or two magnitudes higher than those for the general population. The discrepant findings across the study populations were described as likely due to chance, confounding, and/or bias.	Negative	Chang et al. (2014); (3M-funded independent tox/epi review)
	No evidence of genotoxicity from NaPFHx in bacterial mutagenicity assays (333-5000 µg/mL), nor chromosome aberrations in human peripheral lymphocytes (with or without metabolic activation) (5-3860 µg/mL).	Negative	Loveless et al. (2009)

	PFASs modestly produce reactive oxygen species and DNA damage in the HepG2 cell line. Increase in ROS production was not concentration-dependent and the compounds did not generate DNA damage that could be detected by the alkaline comet assay.	Negative	Eriksen et al. (2010)
Developmental Toxicity	PFOA and PFOS are "known to the State of California to cause reproductive toxicity", based on formal identification by an authoritative body (U.S. EPA). Review by authoritative body.	Positive	OEHHA (2017)
	The EPA Office of Water selected a RfD of 0.00002 mg/kg-day for PFOA based on reduced ossification and accelerated puberty (in male mice) (Lau et al. 2006).	Positive	U.S. EPA (2016b)
	EPA derived a reference dose for PFOS of 0.00002 mg/kg-day based on decreased neonatal rat body weight from the 2-generation study (Luebker et al. 2005).	Positive	U.S. EPA (2016c)
	Treatment of pregnant CD-1 mice with NH4+ PFBA by oral gavage was associated with delayed eye opening in pups and delayed vaginal opening in 175 or 350 mg/kg-day dose groups.	Positive	Das et al. (2008)
	Decreased birth weight with increased maternal/cord blood PFOA levels in general population.	Positive	Apelberg et al. (2007); Fei et al. (2007); Johnson et al. (2014); Maisonet et al. (2012)
	No association between PFOA levels and birth weight in high-exposure C8 community population.	Negative	Darrow et al. (2013); Nolan et al. (2009); Savitz et al. (2012a); Savitz et al. (2012b); Stein et al. (2009)
	K+ PFHxS given to Sprague Dawley rats by oral gavage. No treatment-related effects (reproductive, developmental, neurological) in dams or offspring compared with controls.	Negative	Butenhoff et al. (2009)
	2,3,3,3-Tetrafluoro-2-(heptafluoropropoxy)propanoate (a PFOA replacement technology) in rats at 100 and 1000 mg/kg-day decreased fetal weights and produced early delivery of offspring. Offspring were alive, with no increase in resorptions. Reproductive effects were observed at dose levels also inducing maternal toxicity, which "do not normally warrant classification".	Negative	Beekman et al. (2016)

Reproductive Toxicity	Decreased fertility in humans with increased concentrations of PFOS, PFOA and PFHxS in maternal plasma.	Positive	Fei et al. (2009); Velez et al. (2015)
	Probable link between PFOA exposure and pregnancy-induced hypertension in humans.	Positive	C8 Science Panel (2012)
	Treatment of pregnant CD-1 mice with PFBA by oral gavage was associated with increased offspring liver weight at postnatal day (PND) 1 (but not by PND 10). Delayed eye opening in pups was observed in all dose groups (35, 175 and 350 mg/kg-day) and delayed puberty in highest two dose groups.	Positive	Das et al. (2008)
	Increased ovarian follicular fluid levels of PFHxS were associated with reduced baseline follicle count and post-fertilization success in women receiving assistive reproductive technology.	Positive	McCoy et al. (2017)
Cardiovascular Toxicity	Probable link between PFOA exposure and elevated cholesterol.	Positive	C8 Science Panel (2012)
	No increase in relative risk of heart disease. Significant 'trend' for increased risk in heart disease after 10-year lag in PFOA exposure.	Positive	Sakr et al. (2009)
	Positive correlation of serum cholesterol with PFOA, PFOS and PFHxS exposure.	Positive	Fisher et al. (2013); Costa et al. (2009); Nelson et al. (2010); Sakr et al. (2007b); Skuladottir et al. (2015); Steenland et al. (2010); Wang et al. (2012); Winquist and Steenland (2014a)
	No indication of increased serum lipids/cholesterol in humans with elevated PFOS/PFOA serum levels.	Negative	Emmett et al. (2006) (exposed residents); Olsen et al. (2000); Olsen and Zobel (2007)
	No electrocardiogram (EKG) alterations in PFOA-exposed workers (5-9550 ng/mL serum PFOA).	Negative	Sakr et al. (2007a); (Sakr et al. 2007b)
	No increased mortality ratio in exposed workers for heart disease.	Negative	Lau et al. (2007); Lundin et al. (2009); Steenland and Woskie (2012)

Endocrine Toxicity	Probable link between PFOA exposure and thyroid disease in human based on occupational and community cohort studies.	Positive	C8 Science Panel (2012); Lopez-Espinosa et al. (2012); Melzer et al. (2010); Winquist and Steenland (2014b)
	Increased risk of gestational diabetes development with serum PFOA	Positive	Zhang et al. (2015)
	Increased mortality from diabetes in occupational exposure cohorts. Authors specify that mortality may not be a good surrogate for incidence.	Positive	Lau et al. (2007); Leonard et al. (2008); Lundin et al. (2009) (SMR = 197); Steenland and Woskie (2012) (SMR = 1.90)
	Positive association of serum estradiol and testosterone with serum PFOA in men. Authors indicated circadian variations (collection time) may confound data.	Positive	Sakr et al. (2007a); Sakr et al. (2007b)
	Decreased free and total T4 in PFOA-treated Cynomolgus monkeys.	Positive	Butenhoff et al. (2002)
	PFBS (IC50 = 68 µM) and PFHxS are aromatase inhibitors in placental cells. Inhibitory effect of PFBS important despite low uptake in cells.	Positive	Gorrochategui et al. (2014)
	Activation of estrogen receptor in reporter gene assay for 4:2, 6:2 and 8:2 FTOH. C4+ PFCAs all activate PPAR-alpha; no PFBA activation of PPAR-gamma.	Positive	Rosenmai et al. (2016)
	PFHxSK inhibited 11 -HSD2 in human and rat kidney microsomes. PFHxSK was more potent in human than rat microsomes.	Positive	Zhao et al. (2011)
	Competitive binding to transthyretin (for TH transport) in binding assay, reducing free thyroxine in blood. Competitive binding of PFHxS > PFOS/PFOA > PFHpA. Binding of PFASs 12-300 times lower than thyroxine. No effect from PFBA and FTOHs.	Positive Negative	Weiss et al. (2009)
	PFBA/PFBS had lowest potency of activation of PPAR-alpha in COS-1 cells in multiple chain lengths tested.	Negative	Wolf et al. (2008)
	The potassium salts of PFBS and PFHxS had no effect on 3β or 17β-HSD activity in human or rat testes microsomes, even at high concentrations.	Negative	Zhao et al. (2010)

	No evidence of altered thyroid function with occupational exposure, serum thyroid stimulating hormone (TSH), T3, T4.	Negative	Mundt et al. (2007); Olsen et al. (2003); Olsen et al. (1998); Olsen and Zobel (2007); Sakr et al. (2007a); Sakr et al. (2007b)
	No association between serum PFOA and Type 2 diabetes incidence in general or worker populations.	Negative	MacNeil et al. (2009); Steenland et al. (2015)
	No association between serum PFOA and measures of metabolic syndrome in general or worker populations.	Negative	Lin et al. (2009)
Hematotoxicity	Reduced red blood cell (RBC) count, hemoglobin and hematocrit in male rats (90 day PFBS dosage).	Positive	Lieder et al. (2009)
	No correlation in hematological parameters (including hematocrit, hemoglobin, red blood cells, white blood cells, platelets) and serum PFOS in plant workers.	Negative	Olsen et al. (2003); Olsen et al. (1998); Olsen and Zobel (2007)
	No alterations in blood counts in workers exposure to PFOA.	Negative	Sakr et al. (2007a); Sakr et al. (2007b)
Hepatotoxicity and Digestive System Toxicity	Probable link between PFOA exposure and ulcerative colitis.	Positive	C8 Science Panel (2012); Steenland et al. (2015)
	Positive minor association of serum PFOA with serum liver enzyme levels (AST, ALT, GGT).	Positive	Costa et al. (2009); Gallo et al. (2012); Olsen et al. (2000); Olsen et al. (2003); Olsen and Zobel (2007); Sakr et al. (2007a); Sakr et al. (2007b)
	Small, increased liver weight in Cynomolgous monkeys (PFOA capsules).	Positive (low N, 1-2% increase)	Butenhoff et al. (2002)
	Hepatonecrosis, hepatic tumors in Cynomolgous monkeys. At termination of dosing, the mean PFOS serum level corresponding to the LOAEL was 171,000 ng/mL in females and 173,000 ng/mL in males (K-PFOS capsules).	Positive (low N)	Seacat et al. (2002)
	In rats, PFHxS was more toxic to liver than PFOS and PFBS.	Positive	Danish Environmental Protection Agency (2015)

	In a 90-day study, PFHxA was administered to rats daily by oral gavage. The NOAEL was 50 mg/kg/day for males and >200 mg/kg/day for females based on increased liver weight, peroxisome beta oxidation activity and centrilobular hepatocyte hypertrophy.	Positive	Chengelis et al. (2009)
	Reversible increased liver weight at 30 and 150 mg/kg-day APFB, slight hepatocellular hypertrophy and decreased serum total cholesterol in male S-D rats at 150 mg/kg-day for 28 days APFB.	Positive	Butenhoff et al. (2012)
	Various repeated dose studies of varying duration (but 90 days or less) and dose in rodents of FRD-902, ammonium 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)propanoate reveals increased liver and kidney weights, hepatocellular hypertrophy and alterations in blood chemistries. Chronic feeding study revealed various liver pathologies.	Positive	Beekman et al. (2016)
	K+ PFHxS to Sprague Dawley rats by oral gavage. Effects in parental males included reduced serum cholesterol (all doses), increased liver-to-body and liver-to brain weight ratio, centrilobular hypertrophy, hypertrophy AND hyperplasia of thyroid follicular cells, decreased hematocrit (3 and 10 mg/kg b.w. per day), decreased triglycerides and increased albumin, urea nitrogen, alkaline phosphatase, Ca2+ and albumin/globulin ratio (10 mg/kg b.w. per day)	Positive	Butenhoff et al. (2009)
	4:2, 6:2 and 8:2 FTOHs were incubated in freshly isolated rat hepatocytes, causing cell and mitochondrial toxicity. Cytotoxicity was found to be related to cytochrome P450-mediated bioactivation. LC50s 0.66, 3.7 and 1.4 mM, respectively.	Positive	Martin et al. (2009)
	Compared with PFOA and PFOS, long-chain PFAS alternatives 6:2 chlorinated polyfluorinated ether sulfonate, hexafluoropropylene trimer acid, HFPO tetramer acid, and 6:2 fluorotelomer sulfonic acid showed greater cytotoxicity on human liver HL-7702 cells.	Positive	Sheng et al. (2017)
	6:2 chlorinated polyfluorinated ether sulfonate (6:2 Cl-PFESA) and hexafluoropropylene oxide (HFPO) showed unique binding modes and higher binding affinity to human liver fatty acid binding protein (hl-FABP), and higher cytotoxicity in human liver cells, than PFOA and PFOS.	Positive	Sheng et al. (2018)

	PPAR-alpha induction in human hepatocellular carcinoma cells was found to be similar for C4 and C5 (“short-chain”) PFCAs as for C12 and C14 (“long-chain”), but was induced at lower cellular concentrations by the C4 and C5 compounds tested. Other PFCAs tested (C6-C10) induced PPAR-alpha greater than 2.5-fold.	Positive	Rosenmai et al. (2018)
	No significant relationship between serum PFOA and liver function in residents of community water district.	Negative	Emmett et al. (2006)
	No association of PFOA exposure with liver disease. U-shaped dose-response for serum bilirubin observed in cohort may explain inverse responses in occupational cohorts.	Negative	C8 Science Panel (2012)
	No reported gastric ulcers or colon polyps in workers exposed to PFOS.	Negative	Grice et al. (2007)
Immunotoxicity	Depressed antibody production post-vaccination, or increased odds of, with increasing PFOA, PFOS and PFHxS serum concentrations.	Positive	Grandjean et al. (2012); Grandjean et al. (2016); Granum et al. (2013); Looker et al. (2014)
	Several PFASs were associated with an increased number of respiratory tract infections in the first 10 years of life, suggesting immunosuppressive effects.	Positive	Impinen et al. (2018)
	In human peripheral blood leukocytes, PFBS inhibited the release of TNF-α and IL-10, but IL-6 and IFN-γ were unaffected. In THP-1 cells, PFBS also inhibited NF-κB activation (by inhibiting LPS-induced phosphorylation of P65, necessary for NF-κB transcription), and prevented I-κB degradation.	Positive	Corsini et al. (2012)
	Probable link between PFOA exposure and ulcerative colitis.	Positive	C8 Science Panel (2012); Steenland et al. (2015)
	Prenatal exposure to PFOA, PFDA, PFDoA and PFHxS increased the risk of childhood atopic dermatitis in female children during the first 24 months of life in a prospective birth cohort study.	Positive	Chen et al. (2018)

	Oral administration of 2,3,3,3-Tetrafluoro-2-(Heptafluoropropoxy)-Propanoate in mice for 28 days. Evaluated for T-cell dependent antibody response (TDAR) and splenic lymphocyte subpopulations 5 days post-antigen treatment. TDAR was suppressed in females at 100 mg/kg. T lymphocyte numbers were increased in males at 100 mg/kg.	Positive	Rushing et al. (2017)
Nephrotoxicity and Other Urinary System Toxicity	Increased mortality from renal disease (SMR 3.11) in workers at PFOA facility. PFOA exposure estimated based on job history and data from biomonitoring survey. Significant positive trend in nonmalignant kidney disease.	Positive	Steenland and Woskie (2012)
	No association of kidney function, (measured blood urea nitrogen and serum creatinine) with occupational PFOS/PFOA exposure.	Negative	Costa et al. (2009); Mundt et al. (2007); Olsen et al. (2003); Olsen et al. (1998)
Neurodevel-opmental Toxicity	Positive association with Attention Deficit Hyperactivity Disorder (ADHD) in children in a highly PFOA-exposed community.	Positive	Hoffman et al. (2010)
	PFBS uniquely suppressed differentiation of Ach and Dopa phenotypes in vitro PC12 (rat neuronal) cells. Concentration-dependent decrease in expression of tyrosine hydroxylase and choline acetyltransferase. Unlikely for one shared mechanism of toxicity across perfluorinated chemicals.	Positive	Slotkin et al. (2008)
	No finding of adverse association between PFOA exposure and neuropsychological test performance in children.	Negative	Stein et al. (2013)
Neurotoxicity	PFASs may induce neurobehavioral effects, particularly in developmentally exposed animals.	Positive	Mariussen (2012)
	Neurobehavioral endpoints were not affected in Sprague Dawley rats following exposure to PFHxA (0, 20, 100 or 500 mg/kg b.w. per day NaPFHx) for 90 days.	Negative	Loveless et al. (2009)
Respiratory Toxicity	90-day evaluation of NaPFHxA in Sprague Dawley rats following oral gavage, indicated a NOAEL of 20 and 100 mg/kg-day in males and females, respectively, based on mild to minimal degeneration of the nasal epithelium.	Positive	Loveless et al. (2009)

	Normal pulmonary function in fluoropolymer plant workers with serum PFOA.	Negative	Sakr et al. (2007a); Sakr et al. (2007b)
Other Toxicological Hazard Traits	Increased PPAR-alpha activation in human-transfected COS-1 monkey kidney cells by PFHxA, PFBA, PFHxS, and PFBS. PFDA inactive.	Positive	Wolf et al. (2008)
	28-day 150 mg/kg-d APFB repeat-dose oral study in S-D rats. Delay in pupillary reflex response. Outer retinal degeneration and loss of photoreceptors. Decrease in inner retinal, nuclear and synaptic terminal layer thicknesses. No mortality.	Positive	3M (2006); (3M 2006); Butenhoff et al. (2012)
	Ammonium 4,8-dioxa-3H-perfluorononanoate (ADONA) toxicity was evaluated in acute and repeat-dose studies of up to 90-days duration. It was a mild skin irritant and a moderate to severe eye irritant in rabbits.	Positive	Gordon (2011)
	No generation of reactive oxygen species (ROS) nor DNA damage by PFBS and PFHxA in HepG2 cells.	Negative	Eriksen et al. (2010)
Terrestrial Animal (Ecological) Toxicity	Exposure to PFOS produced toxic effects in diverse endpoints after acute and chronic dietary exposures in mallard (Anas platyrhynchos) and northern bobwhite quail (*Colinus virginianus*). Authors were able to establish no-effect and effect-based exposure concentrations ("toxicity reference values") protective of bird populations (useful in ecological risk assessment).	Positive	Newsted et al. (2007); Newsted et al. (2005)
	Egg concentrations of PFAS are negatively correlated with hatching success in wild and laboratory birds.	Positive	Custer et al. (2012); Custer et al. (2014); Norden et al. (2016)
	Pollinators, including honeybees (*Apis millifera*) and bumblebees (*Bombus terrestris*), are adversely affected by exposure to PFOS via oral and contact routes.	Positive	Mommaerts et al. (2011); Wilkins (2001a); Wilkins (2001b)
	A PFOS no-effect concentration in soil of 77 mg/kg was derived for earthworm.	Positive	Sindermann et al. (2002)
	One-day-old chickens (*Gallus gallus*) were exposed for three weeks to PFOS, PFOA, or PFDA but exhibited no adverse effects on body weight, organ characteristics, blood chemistry, or histopathology.	Negative	Yeung et al. (2009)

Aquatic Toxicity	Exposures to PFOS and PFOA, and, to a lesser extent, other longer-chain PFASs produce a broad range of acute and chronic toxicity responses in diverse aquatic organisms: microorganisms, algae, plants, invertebrates, amphibians, and fish.	Positive	Beach et al. (2006); Ding and Peijnenburg (2013); Environment Canada (2006); Environment Canada (2012); Giesy et al. (2010); UNEP/POPS/POPRC (2016a); UNEP/POPS/POPRC (2016b); UNEP/POPS/POPRC (2016c); UNEP/POPS/POPRC (2016d)
	8:2 FTCA, 8:2 FTUCA, 6:2 FTCA, 6:2 FTUCA, 7:3 acid, 5:3 acid, PFPeA, and PFDA were found to be of low to medium concern to aquatic organisms (EC/LC50 typically between 1 and >100 mg/L)	Positive	Hoke et al. (2012)
	Aquatic toxicity of PFHxA, PFHpA, PFOA and PFNA to several species of marine microalgae increased with increasing chain length: for every extra perfluoromethylene group in the alkyl chain, toxicity increased twofold. EC50 ranged from 0.28 mM to 12.84 mM.	Positive	Latala et al. (2009)
	Toxicity of PFBS, PFOS, PFHxA, PFOA, PFDoA and PFTeA on the membrane system of the freshwater algae *Scenedesmus obliquus* increased with chain length. PFOS, PFDoA and PFTeA inhibited algal growth in a concentration-dependent manner.	Positive	Liu et al. (2008)
	Tail deformation and uninflated swim bladders in zebrafish exposed to PFBA. Lowered heart rates and cardiac output. Malformations of the head in zebrafish exposed to PFBS. No structure-activity relationship for some endpoints. Exposure concentrations 50-3000 mg/L.	Positive	Hagenaars et al. (2011)

	F–53B [6:2 chlorinated polyfluorinated ether sulfonate] exposure (1.5 - 12 mg/L) induced developmental toxicity, including delayed hatching, increased occurrence of malformations, and reduced survival. Malformations, including pericardial and yolk sac edemas, abnormal spines, bent tails, and uninflated swim bladders increased with time course and dose. Continuous exposure resulted in high accumulation levels in zebrafish embryos, suggesting an inability for embryos to eliminate this compound and a high cumulative risk to fish. F–53B induced cardiac toxicity and reduced heart rate.	Positive	Shi et al. (2017)
	EC50 values obtained are far above concentrations typically found in surface waters, the PFASs tested are not expected to have acute harmful effects to *C. sphaericus* or *D. magna* in the real environment.	Negative	Ding et al. (2012a); Ding et al. (2012b)
	Short chain PFCAs (PFBA, PFPeA and PFHxA) never reach environmental concentrations comparable to proposed Environmental Quality Standards which are in the order of thousands of ng/L for freshwaters. In the case of PFBS thousands of ng/L were determined only in an Italian river basin directly impacted by a fluorochemical plant, but concentrations up to 200–400 ng/L were sometimes measured in rivers in Northern Europe and China. PFAS with carbon chain ≤C6, due to their low bioaccumulative potential and low acute and chronic aquatic toxicity, do not seem to be a direct concern for the aquatic environment.	Negative	Valsecchi et al. (2017)

Phytotoxicity	A broad range of acute and chronic toxicity responses in algae and aquatic plants have been observed, with stronger effects associated with increased perfluoroalkyl chain length.	Positive	Beach et al. (2006); Ding and Peijnenburg (2013); Environment Canada (2006); Environment Canada (2012); Giesy et al. (2010); UNEP/POPS/POPRC (2016a); UNEP/POPS/POPRC (2016b); UNEP/POPS/POPRC (2016c); UNEP/POPS/POPRC (2016d); Ding et al. (2012a); Ding et al. (2012b); Hoke et al. (2012); Latala et al. (2009); Liu et al. (2008)
	No-effect PFOS concentrations in soil ranged from 4 mg/kg to 60 mg/kg for seven species of crop plants.	Positive	Brignole et al. (2003)
	The aquatic toxicity of seven PFASs was investigated on the root elongation of lettuce (*L. sativa*) seeds and photosynthesis of green algae (*P. subcapitata*). The toxic effects on lettuce seeds and green algae were found to be similar in a relative sense and were shown to have a good relationship with the fluorinated carbon-chain length. The toxicity of these chemicals increased with increasing fluorinated carbon chain length. It is interesting to observe that PFBA is more toxic than expected, which may be related with acidification of the test solution. Because the unicellular *P. subcapitata* is sensitive to the pH value of the test solution, the actual EC50 value of PFBA is lower and deviates from the relationship between log EC50 value and chain length.	Positive	Ding et al. (2012a)
Other Environmental Hazard Traits	Exposure to PFOS decreased hatching success of chickens, but dose-related activation of PPAR-alpha did not occur.	Negative	O'Brien et al. (2009)

APPENDIX 4 – REPORT PREPARATION

Preparers:

Simona A. Balan (SCP), Ph.D., Senior Environmental Scientist (Specialist)

Julia Gress (SCP), Ph.D., Environmental Scientist

Dennis Guo (SCP), Ph.D., Research Scientist III

Vivek Mathrani (HERO), Ph.D., Staff Toxicologist (Specialist)

Jeff Wong (SCP), Ph.D., Staff Toxicologist (Specialist) – Retired Annuitant

Reviewers:

Andre Algazi, M.S., Project Leader

Karl Palmer, M.S., Branch Chief for Safer Consumer Products Program

Meredith Williams, Ph.D., Deputy Director for Safer Products and Workplaces Program

APPENDIX 5 – APPENDIX REFERENCES

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