Product – Chemical Profile for Treatments Containing Perfluoroalkyl or Polyfluoroalkyl Substances for Use on Converted Textiles or Leathers

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SAFER CONSUMER PRODUCTS

Department of Toxic Substances Control

CalEPA California Environmental Protection Agency

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ABOUT THIS PROFILE

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 (California Code of Regulations, title 22, Division 4.5, Chapter 55, Article 3). DTSC has determined that treatments containing any member of the class of perfluoroalkyl and polyfluoroalkyl substances (PFASs) for use on converted[1] textiles or leathers meet the key prioritization criteria (California Code of Regulations. title 22, section 69503.2(a)) 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 Product-Chemical Profile (Profile) demonstrates that the regulatory criteria have been met and serves as the basis for Priority Product rulemaking. The Profile does not provide a comprehensive assessment of all available adverse impact and exposure literature on PFASs or on treatments for converted textiles or leathers. If this Priority Product regulation is adopted, the responsible entities must follow the reporting requirements pursuant to the SCP regulations (California Code of Regulations. title 22, section 69503.2(a)).

Candidate Chemical: A chemical that exhibits a hazard trait and is listed on one or more authoritative lists in the SCP regulations.

Product-Chemical Profile: A report generated by DTSC to explain its determination that a proposed Priority Product meets the SCP regulatory criteria for potential significant or widespread adverse impacts to humans or the environment.

Priority Product: A product-chemical combination, as identified in regulations by DTSC, that has the potential to contribute to significant or widespread adverse impacts to humans or the environment.

[1] The term "converted" indicates textiles and leather that manufacturers and craftspeople have turned into consumer products such as clothing, shoes, upholstery, etc.

Readers should consider the following:

- This Profile is not a regulatory document and does not impose any regulatory requirements.
- The Profile summarizes information compiled by DTSC as of January 2021 and includes consideration of stakeholder feedback provided during the comment period that closed on January 14, 2020. [2] In preparation for rulemaking, DTSC also requested feedback on the scientific basis of this document from four external scientific peer reviewers. Their feedback was provided to DTSC on June 29, 2020.
- Since the public draft from November 8, 2019, DTSC:
    - Made several editorial changes to improve the clarity of the writing;
    - Corrected a few minor errors identified by DTSC staff and public commenters;
    - Made some clarifications and changes to address points raised by public commenters and the external peer reviewers; and
    - Added several new references subsequently identified.
- 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. The proposal indicates only that there is a potential for people or the environment to be exposed to the Chemical of Concern in the Priority Product; that such exposure has the potential to cause or contribute to significant or widespread adverse impacts; and that safer alternatives should be explored.

[2] https://calsafer.dtsc.ca.gov/cms/commentpackage/?rid=12746

1. RATIONALE FOR PRODUCT-CHEMICAL SELECTION

The Department of Toxic Substances Control (DTSC) proposes to list treatments containing any member of the class of perfluoroalkyl and polyfluoroalkyl substances (PFASs) for use on converted textiles or leathers as a Priority Product. This product-chemical combination meets the identification and prioritization factors outlined in the Safer Consumer Products (SCP) regulations: (1) there is potential for human and other organism exposure to PFASs from treatments for converted textiles or leathers; and (2) the exposure has the potential to contribute to or cause significant or widespread adverse impacts.

PFASs are a class of manufactured chemicals with at least one fully fluorinated carbon atoms (Buck et al. 2011; Biomonitoring California 2015a). All PFASs are Candidate Chemicals under the SCP program because the Biomonitoring California program lists the entire class as Priority Chemicals for measuring in the blood or urine of Californians. While not all PFASs are used in treatments for converted textiles and leathers, any PFASs used in these products are of concern for the reasons detailed in this document. Because PFAS ingredients are not typically listed on Safety Data Sheets, DTSC does not know which specific PFASs are used in these treatments or might be used in the future. Also, these intentionally added ingredients often contain other PFASs as impurities or degradation products. Therefore, DTSC is taking a chemical class approach to PFASs.

DTSC is also taking a class approach to PFASs because:

- PFASs are a wide and varied group of chemicals used in many applications. All PFASs share one common trait – highly stable carbon-fluorine (C-F) bonds that make them or their final degradation products highly persistent in the environment.
- Persistence is a hazard trait identified in the Office of Environmental Health Hazard Assessment's Green Chemistry Hazard Traits regulation 69405.3: "Persistence of a chemical in the environment promotes sustained exposure and contributes to accumulation in the environment."
- Within the overall PFAS class, perfluoroalkyl acids (PFAAs) are the most problematic subclass. They are the most widely and thoroughly characterized subclass and are associated with a number of different health hazards, including endocrine disruption, developmental and reproductive toxicity, and immune dysregulation.
- In the case of PFAAs, their ubiquity in combination with their persistence results in continuous exposure from multiple sources, including contaminated drinking water sources and the food supply. They can accumulate in meat, plants, and drinking water, and ultimately in humans and wildlife.
- The vast majority of PFASs degrade into PFAAs, which typically increases their mobility in the environment, making containment or removal a challenge.

- While persistence alone warrants enough concern to include any member of the PFAS class in product prioritization, the health hazards associated with exposure to PFAAs are additionally concerning and underlie our listing.

Potential for exposure to PFASs in treatments for converted textiles or leathers

> Treatments for converted textiles or leathers … contribute to the ubiquitous environmental PFAS contamination and exposures…. Once released to the environment during product manufacture, use, or disposal, PFASs become part of a virtually closed cycle leading to chronic human and ecological exposures.

DTSC has identified treatments for converted textiles or leathers as significant sources of human and ecological PFAS exposures, especially via inhalation during product use. Treatments for converted textiles or leathers are frequently used in homes and in several occupations. They contribute to the ubiquitous environmental PFAS 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 human and ecological exposures. They are found in the aquatic, atmospheric, and terrestrial environments, including remote locations far from any point source. They are present in indoor dust, drinking water sources, food, wildlife, and humans – including the milk and serum of breastfeeding women. Because persistent PFASs lack a natural degradation route, their levels in the environment, humans, or biota will continue to rise for as long as PFASs are produced and used in consumer products, and even after production of these compounds has ceased.

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, but other sources of exposure may contribute. This contamination is partly due to releases of PFASs from the manufacturing, use, and disposal of treatments for converted textiles or leathers, as well as from the use and disposal of the treated consumer products. When used, treatments for converted textiles or leathers release PFASs into indoor air and dust, which people inhale or ingest. Most waste or end-of-life converted textiles or leathers 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 do not 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, rugs, upholstery, clothing, shoes, and other consumer products to which treatments containing PFASs have been applied become major sources of exposure for infants and children via direct contact and incidental 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. Carpet and upholstery cleaners, workers in stores selling upholstered furniture, furnishings, carpets, clothing, or shoes, and auto dealership workers and auto detailing technicians may also experience above-average PFAS exposure levels.

Potential for significant or widespread adverse impacts

All PFASs display at least one of the hazard traits identified in California's Green Chemistry Hazard Traits Regulations (California Code of Regulations, title 22, section 69401 et seq.): they are either extremely persistent in the environment or they degrade into extremely persistent PFASs. Most PFASs are mobile in environmental media such as air and water, and thus are widespread in living organisms and the environment. Several PFASs bioaccumulate significantly in animals or plants, including those consumed by humans as food. Certain PFASs also contribute to global warming.

Studies indicate that some PFASs can cause reproductive and developmental, liver and kidney, and immunological effects, as well as tumors in laboratory animals. The most consistent finding from human epidemiology studies is a small increase in serum cholesterol levels among exposed populations, with more limited findings related to infant birth weights, effects on the immune system, cancer, and thyroid hormone disruption. Some PFASs have also been linked to phytotoxicity, aquatic toxicity, and terrestrial ecotoxicity.

Under a voluntary agreement with the United States (U.S.) Environmental Protection Agency (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.

All PFASs are Candidate Chemicals under the SCP program because the Biomonitoring California program lists the entire class as Priority Chemicals for measuring in the blood or urine of Californians.

Based on the criteria in the Safer Consumer Products regulations, DTSC has determined that treatments containing PFASs for use on converted textiles or leathers have the potential to cause significant and widespread adverse impacts to sensitive subpopulations, including fetuses, infants, young children, pregnant women, carpet and upholstery cleaners, workers in stores selling upholstered furniture, furnishings, carpets, clothing, or shoes, and auto dealership workers and auto detailing technicians; to environmentally sensitive habitats; and to threatened and endangered species. Given the known hazard traits, replacing currently used PFASs in treatments for converted textiles or leathers with other members of the PFAS class could constitute a regrettable substitution. Hence, this proposal covers treatments containing any member of the class of PFASs intended for use on converted textiles or leathers.

2. PRODUCT DEFINITION AND SCOPE

This section describes the product that forms the basis for the proposed product-chemical combination.

"Treatments containing PFASs for use on converted[3] textiles or leathers" means any product containing PFASs placed into commerce in California that may be marketed or sold for the purpose of:

- Eliminating dirt or stains from carpets, rugs, clothing, shoes, upholstery, or other converted textiles and leathers; or
- Repelling stains, dirt, oil, or water from carpets, rugs, clothing, shoes, upholstery, or other converted textiles and leathers.

Note that these treatments are sometimes also referred to as aftermarket treatments (ECY 2020) or impregnating agents (Jensen et al. 2008).

Subproducts include:

- Cleaner: a product marketed or sold for the purpose of eliminating dirt or stains;
- Protectant: a product marketed or sold to protect the surface from soiling when in contact with dirt or other impurities, or to reduce liquid absorption;
- Spot remover: a product marketed or sold to clean localized areas, or to remove localized spots or stains; and
- Water proofer or water repellant: a product marketed or sold to repel water.

"Treatments containing PFASs for use on converted textiles or leathers" does not include products marketed or sold exclusively for use during the process of carpet, rug, clothing, shoe, or furniture manufacturing.

[3] The term "converted" indicates textiles and leather that manufacturers and craftspeople have turned into consumer products such as carpets, upholstery, furnishings, clothing, shoes, etc.

3. CANDIDATE CHEMICAL DEFINITION AND PROPERTIES

This section introduces the Candidate Chemical(s) in the proposed product-chemical combination.

This proposal covers all perfluoroalkyl and polyfluoroalkyl substances (PFASs)[4] in current or future production. While not all PFASs are used in treatments for converted textiles and leathers, any PFASs used in these products are of concern, for the reasons detailed below. Because PFAS ingredients are not typically listed on Safety Data Sheets, DTSC does not know which specific PFASs are used in these treatments or might be used in the future. Also, these intentionally added ingredients often contain other PFASs as impurities or degradation products. Therefore, DTSC is taking a chemical class approach to PFASs.

As of 2015, a few individual PFASs were included on the Safer Consumer Products (SCP)'s Candidate Chemical list. In December of that year, all PFASs were added as Candidate Chemicals under the SCP Program due to their designation as Priority Chemicals under the Biomonitoring California program (Biomonitoring California 2015a). The Biomonitoring California listing is based on the chemicals' potential for widespread exposures, persistence, bioaccumulation, and emerging evidence for toxicity.

PFASs are a class of fluorinated organic chemicals containing at least one fully fluorinated carbon atom (Biomonitoring California 2015a). This class includes more than 3,000 chemicals (KEMI 2015), although fewer may be in current production. The Organisation for Economic Cooperation and Development (OECD) has identified 4,730 Chemical Abstracts Service (CAS) numbers related to individual PFASs or commercial PFAS mixtures available on the global market (OECD 2018). 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. 2017b) listed below and depicted in Figure 1. However, any other PFAS that exists or will be developed in the future and is used in treatments for converted textiles or leathers also falls under the scope of this proposal.

[4] Note that U.S. EPA and others use the acronym "PFAS" for this class of chemicals. For the purpose of this document, we use the acronym "PFASs" to emphasize that this is a class of chemicals, and to make it clear when we refer to multiple members of this class rather than an individual chemical.

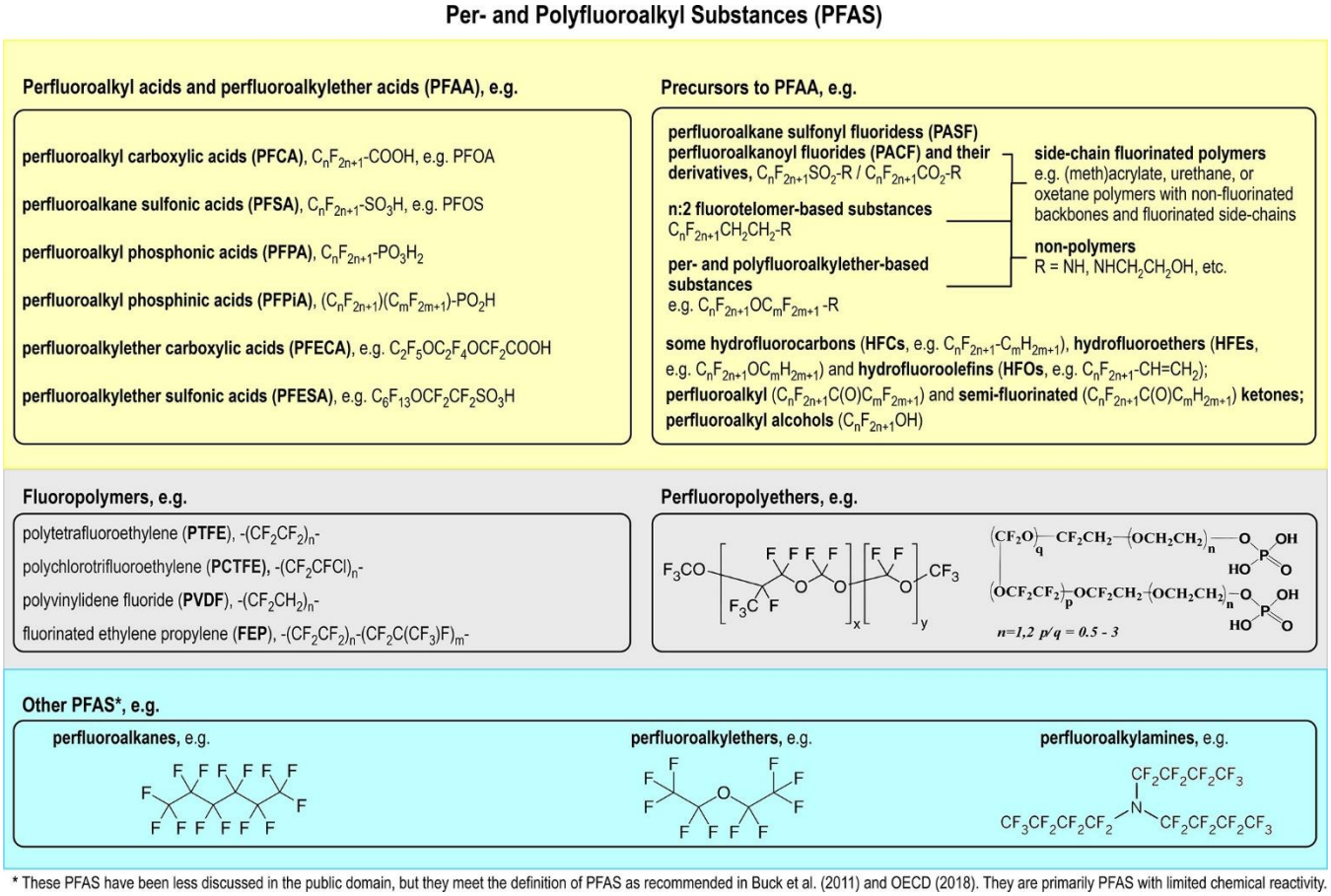

Figure 1: General classification of PFASs. Reprinted with permission Kwiatkowski et al. (2020), available at https://pubs.acs.org/doi/10.1021/acs.estlett.0c00255. Further permissions related to the material excerpted should be directed to the ACS.

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:

    i. Perfluoroalkyl carboxylic acids (PFCAs) such as perfluorooctanoate (PFOA);[5]
    ii. Perfluoroalkyl sulfonic acids (PFSAs) such as perfluorooctane sulfonate (PFOS);[5]
    iii. Perfluoroalkyl sulfinic acids (PFSiAs);
    iv. Perfluoroalkyl phosphonic acids (PFPAs);
    v. Perfluoroalkyl phosphinic acids (PFPiAs);
    vi. Perfluoroether carboxylic acids (PFECAs); and
    vii. Perfluoroether sulfonic acids (PFESAs).

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

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 potential to degrade into perfluoroalkyl substances, i.e., they are precursors to perfluoroalkyl substances. See examples in Figure 2.
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 are unlikely to degrade to PFAAs under typical environmental conditions, but may contain PFAA impurities and may release hazardous products during combustion (Nordic Council of Ministers 2020).
4. **Fluoropolymers.** These polymers are highly persistent in the environment. They cannot degrade to PFAAs under typical environmental conditions, but certain PFAAs have been used in their manufacturing and can occur as impurities. Moreover, fluoropolymers may release PFCAs, including PFOA, during combustion at temperatures between 180 and 800°C (Feng et al. 2015; Schlummer et al. 2015). Examples of fluoropolymers include polytetrafluoroethylene (PTFE), polyvinylidene fluoride (PVDF), and polyvinyl fluoride (PVF).

There are also a few highly persistent PFASs that might not fall into these categories (Figure 1).

Thus, while PFAAs constitute a small subset (approximately 1 percent) of PFASs, they are terminal degradation products, manufacturing aids/feedstocks, or impurities of other PFAS class members, which makes their hazard traits relevant to the entire class. 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 2017).

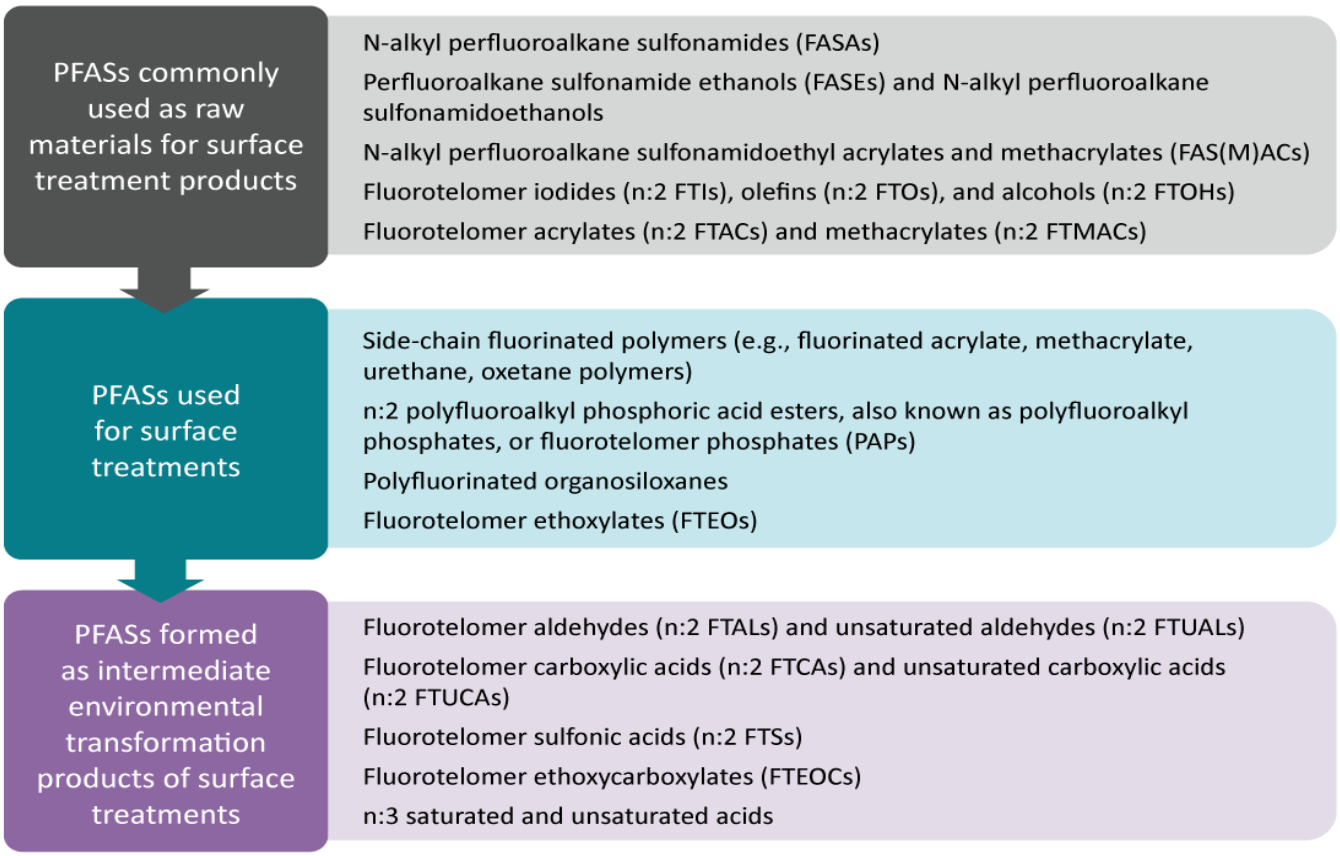

Figure 2: Examples of PFAA precursors, as described in Buck et al. (2011).

Relevant physicochemical properties

Reference: California Code of Regulations, title 22, section 69503.3(a)(1)(D).

Physicochemical properties can be helpful in predicting a chemical's behavior. A chemical's behavior in humans, wildlife, ecosystems, and the environment may indicate potential adverse public health and environmental impacts.

PFASs possess unique physicochemical characteristics that confer increased stability in extreme temperatures, antistatic properties, and resistance to wettability, staining, and corrosion (Buck et al. 2011; Buck et al. 2012). PFASs are used in a wide variety of applications, including (KEMI 2015; FluoroCouncil 2020):

- 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); and
- Aerospace, automotive, construction, and electronics manufacturing industries.

These unique properties of PFASs also 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; Frömel and Knepper 2010; Krafft and Riess 2015a; Wang et al. 2014a; Washington et al. 2015).

For most PFASs, physicochemical properties data are absent from the public domain. The data available in the literature can range widely depending on the measurement methods or model used for the estimates. Table 1 lists data 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, according to modeling studies (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 and 4:2 FTOH, which exist 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 (NILU 2015; Dreyer et al. 2015; Ericson et al. 2012; Fraser et al. 2013; Haug et al. 2011; Knobeloch et al. 2012; Liu et al. 2014; Liu et al. 2015a; 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). In general, shorter-chain PFASs are more volatile than longer chains, and thus more likely to be released from products (U.S. EPA 2020a).

PFAAs are proteinophilic (protein-binding), accumulating particularly in blood, liver, kidney, lung, brain, and muscle tissues.

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. 2017b). 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 surface active agents (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. PFAAs are proteinophilic (protein-binding), accumulating particularly in blood, liver, kidney, lung, brain, and muscle tissues (Chen and Guo 2009; Hebert and MacManus-Spencer 2010; Greaves et al. 2012; Jones et al. 2003; Pérez et al. 2013). Additionally, the concept of K_{ow} for PFAAs has little to no practical meaning or usefulness because PFAAs exist in anion rather than neutral form at environmentally relevant conditions (Valsecchi et al. 2017).

Data on the physicochemical properties of PFASs are limited to mostly model results, which vary widely because the underlying data set 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).

Table 1. Physicochemical properties of some PFAAs[6]

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)
Perfluorobutanoic acid (PFBA)	Miscible	-18	121	1307	2.8	1.9	--	-0.2 – 0.7
Perfluoropentanoic acid (PFPeA)	113	9.6 – 25*	124	1057	3.4	1.4	--	-0.06
Perfluorohexanoic acid (PFHxA)	22	14	143	457	4.1	1.9	--	-0.13
Perfluoroheptanoic acid (PFHpA)	4.2	30	175	158	4.7	2.2	0.4 – 1.1	-0.15
Perfluorooctanoic acid (PFOA)	3.4 – 9.5	37 – 60	188 – 192	4 – 1300	5.3	1.3 – 2.4	0 – 3.4	-0.16 – 3.8
Perfluorononanoic acid (PFNA)	9.5	59 – 66	218	1.3	5.9	2.4	2.6 – 5.9	-0.17
Perfluorodecanoic acid (PFDA)	9.5	77 – 88	218	0.2	6.5	2.8	2.0 – 31	-0.17
Perfluoroundecanoic acid (PFUnA)	0.004	83 – 101	160 – 230	0.1	7.2	3.3	12 – 103	-0.17

[6] Data excluding those marked with an asterisk are from peer-reviewed literature as summarized by Concawe (2016). Entries marked with an asterisk (*) were predicted using QSAR models by U.S. EPA (2020a).

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)
Perfluorododecanoic acid (PFDoA)	0.0007	107 – 109	245	0.01	7.8	--	24 – 269	-0.17 to 0.8
Perfluorotridecanoic acid (PFTrDA)	0.0002	0.26 – 119*	224 – 261*	0.3	8.3	--	--	--
Perfluorotetradecanoic acid (PFTeDA)	0.00003	52 – 130*	276	0.1	8.9	--	--	--
Perfluorobutane sulfonic acid (PFBS)	46 – 57	76 – 84	211	631	3.9	1	--	-6.0 – -5.0
Perfluorohexane sulfonic acid (PFHxS)	2.3	26.7 – 190*	218 –238*	59	5.2	1.8	0.6 – 3.2	-6.0 – -5.0
Perfluorooctane sulfonic acid (PFOS)	0.52 – 0.57	54	219 –244*	6.7	6.4	2.5 – 3.1	0.1 – 97	-6.0 – -2.6
Perfluorodecane sulfonic acid (PFDS)	0.002	--	--	0.7	7.7	3.5	--	--
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)
Perfluorooctyl phosphonic acid (PFOPA)	25	--	210 –244*	0.01	4.7	--	--	--
Perfluorodecyl phosphonic acid (PFDPA)	0.5	187*	210 –272*	0.0002	6.0	--	--	--

Environmental fate

Reference: California Code of Regulations, title 22, section 69503.3(a)(1)(E).

Environmental fate describes a chemical's mobility in environmental media, transformation (physical, chemical, or biological), or accumulation in the environment or biota. A chemical's environmental fate in air, water, soil, and living organisms relates to its exposure potential hazard traits, as defined in the California Code of Regulations, Title 22, Chapter 54.

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. 2017b).

Numerous factors, including intrinsic chemical properties combined with physical and biogeochemical processes, collectively determine the occurrence and fate of PFASs (ITRC 2020a). Longer-chain PFASs bioaccumulate up the food chain (Concawe 2016). Shorter-chain PFASs are particularly mobile in water and air, can undergo long-range transport, and are difficult to remove from contaminated water (Brendel et al. 2018; Li et al. 2020a). PFAAs are the terminal environmental degradation products of most PFASs, and all are extremely persistent in the environment (Wang et al. 2017b). Shorter-chain PFAAs are distributed globally by oceanic currents and sea spray, or by the degradation of volatile precursors that undergo long-range atmospheric transport (Ateia et al. 2019a). Long-range atmospheric transport followed by wet or dry deposition has led to the ubiquity of PFBA and other PFAAs in snow, water, soil, and sediment samples from a remote alpine nature reserve in China (Wang et al. 2019).

Some 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; ITRC 2020a).

Regardless of their sources, due to their unique physicochemical properties, 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, snow, ice, glaciers, and pristine Arctic lakes, far from any point source (Kim and

Kannan 2007; Kirchgeorg et al. 2016; Muir et al. 2019; 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. 2007a; Tao et al. 2008a; Tao et al. 2008b).

Volatile PFAA precursors, including FTOHs, are distributed globally in the gas phase or adsorbed to airborne particles (Dreyer et al. 2015; Liu et al. 2015a; Wang et al. 2014b), 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). Strong inverse correlations between salinity and the concentrations of most PFASs measured indicate that ongoing continental discharges are the major sources of PFASs to the marine environment (Zhang et al. 2019c).

Although a considerable number of studies document levels of some PFASs in the environment and various biological media, knowledge regarding the sorption/desorption patterns and mechanisms of transport, transformation, and fate of most PFASs is limited. 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 sometimes 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. Major data and knowledge gaps remain regarding the sources, transformation, distribution, accumulation, and temporal and spatial trends of various PFASs in wildlife and humans.

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 a few of their precursors. However, since PFAAs are manufacturing aids, impurities, or degradation products of most other PFASs, the following discussion can be relevant to the life cycle of the entire class.

Due to their potential for long-range atmospheric transport followed by deposition onto land and water, PFASs have contaminated food chains and accumulated in plants and animals, even in remote Arctic and Antarctic regions.

PFASs in air

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. 2011a). 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; ITRC 2020a; 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. 2014b), which may be directly inhaled or deposited in rain and snow (Casal et al. 2017; Sammut et al. 2017; Taniyasu et al. 2013). Neutral volatile PFAS precursors, such as FTOHs, are the dominant PFASs found in the gas phase (Ahrens et al. 2012), whereas ionic PFASs, such as PFOA and PFOS, characterized by low vapor pressure and high water solubility, tend to dominate in airborne particulate matter (ITRC 2020a). 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).

Due to their potential for long-range atmospheric transport followed by deposition onto land and water, PFASs have contaminated food chains and accumulated in plants and animals, even in remote Arctic and Antarctic regions (Houde et al. 2006; Butt et al. 2010; Casal et al. 2017; Muir et al. 2019; Roscales et al. 2019). Despite the U.S.-wide phaseout of longer-chain PFASs, atmospheric emissions of these compounds from offshore manufacturing may still impact the U.S. 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, deposition of contaminated particulate matter onto water bodies, and glacial meltwater. 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). Longer-chain PFAAs tend to partition into suspended

particles and sediments, while shorter-chain PFAAs tend to remain in the dissolved phase (Chen et al. 2019).

PFAAs can contaminate groundwater through groundwater recharge (Liu et al. 2016) or be transported to the oceans where they become globally distributed by ocean currents (Benskin et al. 2012a). Stormwater runoff may contribute significantly to the PFAS load in surface waters (Wilkinson et al. 2017; Zushi and Masunaga 2009). During the treatment of municipal and industrial wastewater, volatile PFASs can be released into the air, while others selectively partition into biosolids and are partially removed from wastewater. WWTP effluent tends to have higher concentrations of PFAAs than the influent because of the transformation of precursors during the treatment process (Appleman et al. 2014; Arvaniti and Stasinakis 2015; Sinclair and Kannan 2006). PFASs also enter surface water and groundwater via landfill leachate (Ahrens et al. 2016a; Banzhaf et al. 2017).

PFAAs appear to not degrade 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 (EWG 2003; European Commission 2017). Like for other persistent organic pollutants, oceans are considered the ultimate long-term reservoir for PFASs (Prevedouros et al. 2006), including PFAAs and their precursors (González-Gaya et al. 2014). Marine organisms are chronically exposed via contaminated food chains (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 (González-Gaya et al. 2014). PFASs are also transported downwards from the ocean surface mainly by sinking zooplankton fecal pellets and decaying phytoplankton, but these removal mechanisms are slow (González-Gaya et al. 2019). 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 (Prevedouros et al. 2006; Ahrens et al. 2011b).

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).

The ubiquity 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 (Boone et al. 2019; Gellrich et al. 2013). Shorter-chain PFASs are generally more mobile in groundwater and will leach faster from soil to groundwater (NGWA 2017). 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.

PFASs in sediments

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 particulate matter 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). Longer-chain PFASs such as PFOS and PFOA are typically the predominant PFASs found in surface sediments (Rankin et al. 2016). 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 (see Chapter 4 below) 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).

The ubiquity 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.

PFASs in soil

PFASs can contaminate the soil via atmospheric transport followed by deposition, or directly through application of biosolids (Sepulvado et al. 2011), soil conditioners, or other treatments. From soil, they can migrate into surface water or groundwater, including drinking water sources (Lilienthal et al. 2017). The sorption of PFASs to soil is influenced by several factors, including soil organic matter content, clay content, pH, and the presence of cations (Higgins and Luthy 2006; Li et al. 2018). The fate and transport of a specific PFAS in soil depends on its physicochemical properties and is site-specific (Mejia-Avendaño et al. 2020). Longer-chain PFASs tend to partition preferentially into soil (Dalahmeh et al. 2018), while shorter-chain PFASs partition into soil water (Ahrens et al. 2009b; Guo et al. 2015). Longer-chain PFAAs added to an agricultural soil were found to sorb strongly to the soil, whereas the shorter-chain PFAAs were readily transported in water from the soil surface to the root zone and into the groundwater (McLachlan et al. 2019; Muir et al. 2019). Shorter-chain PFCAs have also 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 (Zhang et al. 2019a).

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). A team of U.S. EPA scientists (Washington et al. 2009) pointed out several issues with that study and arrived at an estimated half-life for fluoroacrylate polymers of 10-17 years based on experimental results and modeling. Subsequently, they evaluated the degradability of two commercial side-chain fluoroacrylate polymers in saturated soil and found 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. More recently, U.S. EPA scientists calculated an environmental half-life for these polymers between nine and 60 years and concluded that the side-chain fluorinated polymers "now in production might constitute considerable sources to the environment of the new generation of PFASs" (Washington et al. 2019). Dasu and Lee (2016) studied the inherent biotransformation potential of two urethane monomers and found negligible degradation in agricultural soil, but significant degradation in forest soil, with an estimated half-life ranging between three and 22.2 months. Royer et al. (2015) studied the biotransformation of 8:2 FTAC and 8:2 FTMAC to 8:2 FTOH in soils, finding half-lives of five days or less and 15 days, respectively.

PFASs in biota

Plants and animals can bioaccumulate PFASs, with large interspecies differences, particularly between aquatic and terrestrial organisms (Concawe 2016). PFAS uptake has been documented in fruits and vegetables grown in backyard and garden soils irrigated with contaminated water (Scher et al. 2018), agricultural soils spiked with PFASs (McLachlan et al. 2019), potted soils spiked with PFASs (Stahl et al. 2009; Zhang et al. 2019a), and biosolids-amended soil (Blaine et al. 2013; Blaine et al. 2014a; Yoo et al. 2011). Crops grown in agricultural soils without significant PFAS contamination were found to contain small levels of mainly shorter-chain PFASs from rainwater and atmospheric deposition (Eun et al. 2020). Longer-chain PFAAs, especially PFSAs, tend to partition in the roots, whereas the more soluble, shorter-chain PFAAs, especially PFCAs, partition in other parts of the plant (Blaine et al. 2013; Blaine et al. 2014a; Gobelius et al. 2017; Lechner and Knapp 2011; Scher et al. 2018; Stahl et al. 2009; Yoo et al. 2011). Chen et al. (2020) found evidence that the PFECA GenX can bioaccumulate in plants and translocate into different plant tissues, depending on the plant species. Studies have also reported PFAA uptake in tree leaves, possibly from a combination of soil uptake and atmospheric deposition, in an urban environment (Zhang et al. 2015) and near industrial emission sources (Shan et al. 2014). PFAS absorption by plants depends, among other factors, on the PFASs' concentration, chain length, and functional group; the plant's physiological characteristics; and the characteristics and amendments of the soil or growth media (Ghisi et al. 2019; Wang et al. 2020).

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 greater than 1,000 or BMF greater than 1 meet the bioaccumulation hazard trait criteria outlined in the California Code of Regulations, title 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. 2017b).

Table 2(a-c) 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. Earthworms were found to accumulate PFASs from soil, including shorter-chain compounds, with BAFs ranging from 1.1 to 96, depending on the compound (Munoz et al. 2020). 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 interspecies variation (Hong et al. 2015). However, this relationship between PFAS chain length and bioaccumulation is reversed in aquatic and terrestrial plants. For instance, Atlantic Ocean plankton displays higher BAFs for PFPeA and PFHxA than for PFOA, possibly due to uptake and metabolism of precursor compounds (Zhang et al. 2019c). BAFs for PFCAs in vegetables grown near a fluorochemical industrial park in China also decreased with increasing chain length (Zhang et al. 2020).

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. 2007b) to several years for longer-chain PFASs (Bartell et al. 2010; Olsen et al. 2007b; Seals et al. 2011). It can take up to 56 years for total elimination of 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 2a. Examples of bioconcentration factors (BCFs)

Chemical	Organism	BCF	Reference
PFOS	Bluegill	1,866 – 4,312	Drottar (2001)
PFOS	Rainbow trout	1,100 – 5,400	Drottar (2001)
PFOS	Rainbow trout	2,900 (liver), 3,100 (blood)	Martin et al. (2003)
PFOA	Water breathing animals	1.8 – 8.0	ECHA (2014)
PFOA	Rainbow trout	12 (liver), 25 (blood)	Martin et al. (2003)
PFHxDA	Carp	4,700 – 4,800	U.S. EPA (2009a)
Perfluorooctadecanoic acid (PFODA)	Carp	320 – 430	U.S. EPA (2009a)

Table 2b. Examples of bioaccumulation factors (BAFs)

Chemical	Organism	BAF	Reference
PFOS	Zooplankton/water	240	Houde et al. (2008)
PFOS	Mysis/water	1,200	Houde et al. (2008)
PFOS	Sculpin/water	95,000	Houde et al. (2008)
PFOS	Lake trout/water	16,000	Houde et al. (2008)
PFOA	Water breathing animals	0.9 – 266	ECHA (2014)
PFOA	Copepods/water	200 – 500	Munoz et al. (2019)
PFOA	Copepods/suspended solids	251	Munoz et al. (2019)
PFOA	Mysids/water	316	Munoz et al. (2019)
PFOA	Mysids/suspended solids	3.16 – 79.4	Munoz et al. (2019)

Chemical	Organism	BAF	Reference
PFOA	Shrimp/water	125 – 631	Munoz et al. (2019)
PFOA	Shrimp/suspended solids	2.5 – 31.6	Munoz et al. (2019)
PFHpA	Copepods/water	200 – 500	Munoz et al. (2019)
PFHpA	Copepods/suspended solids	251	Munoz et al. (2019)

Table 2c. Examples of biomagnification factors (BMFs)

Chemical	Organism	BMF	Reference
PFOS	Arctic cod/zooplankton (Western Canadian Arctic)	8.7	Powley et al. (2008)
PFOS	Caribou/lichen (Canada)	2.0 – 9.1	Müller et al. (2011)
PFOS	Wolf/caribou (Canada)	0.8 – 4.5	Müller et al. (2011)
PFOS	Dolphin/seatrout (2 U.S. locations)	0.9	Houde et al. (2006)
PFOS	Seatrout/pinfish (2 U.S. locations)	4.6	Houde et al. (2006)
PFOS	Walrus/clam (Eastern Arctic Food Web)	4.6	Tomy et al. (2004)
PFOS	Narwhal/Arctic cod (Eastern Arctic Food Web)	7.2	Tomy et al. (2004)
PFOS	Beluga/Arctic cod (Eastern Arctic Food Web)	8.4	Tomy et al. (2004)
PFOS	Beluga/redfish (Eastern Arctic Food Web)	4	Tomy et al. (2004)
PFOS	Polar bear/seal (Canadian Arctic)	177	Martin et al. (2004)
PFOA	Water-breathing animals	0.02 – 7.2	ECHA (2014)
PFOA	Caribou/lichen (Canada)	0.9 – 11	Müller et al. (2011)
PFOA	Wolf/caribou (Canada)	0.9 – 3.8	Müller et al. (2011)
PFOA	Walrus/clam (Eastern Arctic Food Web)	1.8	Tomy et al. (2004)
PFOA	Narwhal/Arctic cod (Eastern Arctic Food Web)	1.6	Tomy et al. (2004)
PFOA	Beluga/Arctic cod (Eastern Arctic Food Web)	2.7	Tomy et al. (2004)

Chemical	Organism	BMF	Reference
PFOA	Beluga/redfish (Eastern Arctic Food Web)	0.8	Tomy et al. (2004)
PFOA	Beluga whale/Pacific herring (Western Canadian Arctic Food Web)	1.3	Tomy et al. (2004)
PFOA	Arctic cod/marine Arctic copepod (Western Canadian Arctic Food Web)	2.2	Tomy et al. (2004)
PFOA	Dolphin/seatrout (2 U.S. locations)	1.8	Houde et al. (2006)
PFOA	Seatrout/pinfish (2 U.S. locations)	7.2	Houde et al. (2006)
PFOA	Polar bears/ringed seal (2 U.S. locations)	45 – 125	Butt et al. (2008)
PFOA	Polar bear/seal (Canadian Arctic)	8.6	Martin et al. (2004)

Degradation, reaction, or metabolic products of concern

Reference: California Code of Regulations, title 22, section 69503.3(a)(1)(G).

A Candidate Chemical may degrade, form reaction products, or metabolize into other chemicals that have one or more hazard traits. These metabolites, degradation products, and reaction products (which may or may not be Candidate Chemicals) may cause different adverse impacts from those of the parent chemical. In some cases, a Candidate Chemical's degradation or reaction products or metabolites may have the same hazard trait, and may be more potent or more environmentally persistent, or both, than the parent chemical. In such cases, adverse impacts may be more severe, or may continue long after, the Candidate Chemical's release to the environment.

Of the 4,730 individual PFASs or commercial PFAS mixtures identified by OECD on the global market, 4,186 likely degrade to PFAAs in the environment or biota (OECD 2018). PFASs that degrade into PFAAs are termed PFAA precursors and provide a significant indirect source of PFAAs to the environment (see examples of PFAA precursors in Figure 2). 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; Rand and Mabury 2012a; Rand and Mabury 2012b; Rand and Mabury 2013; Rand and Mabury 2014; Rand and

Mabury 2017). Fluorotelomer-based compounds degrade (Phillips et al. 2007; Young et al. 2007) or undergo biotransformation into PFCAs (Chen et al. 2017b; Dasu and Lee 2016; Kim et al. 2012a; Kim et al. 2014b; Lee et al. 2010; Liu et al. 2010a; Liu et al. 2010b; Rand and Mabury 2014; Royer et al. 2015; Ruan et al. 2014; Russell et al. 2015; Zhao and Zhu 2017; Hamid et al. 2020). The intermediates in this process include saturated and unsaturated fluorotelomer aldehydes (FTALs and FTUALs, respectively), and saturated and unsaturated fluorotelomer carboxylic acids (FTCAs and FTUCAs, respectively) (Frömel et al. 2016; Nilsson et al. 2013; Rand et al. 2014; Hamid et al. 2020). FTUCAs were detected in all 81 indoor air samples collected in 2015 during a study in China, suggesting the transformation of PFAA precursors such as FTOHs in the indoor environment (Yao et al. 2018).

Pharmacokinetic data from rat and human studies on 6:2 FTOH revealed that, of its main metabolites, the 5:3 fluorotelomer carboxylic acid has the highest internal exposure and slowest clearance, which decreases with increasing exposure (Kabadi et al. 2018). The study also found that 5:3 FTCA may reach steady state following repeated exposure to 6:2 FTOH, therefore resulting in biopersistence, with potential for systemic toxicity (Kabadi et al. 2018). In rat plasma, liver, and fat, 5:3 FTCA reaches steady state after approximately one year following repeated oral doses of 6:2 FTOH (Kabadi et al. 2020).

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).

Side-chain fluorinated polymers, which are fluorotelomer-based, can also degrade to FTOHs or other fluorotelomer compounds, with PFCAs as terminal degradation products (Washington et al. 2015; Washington et al. 2019). Because of their potential to be mobilized, interact with their 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.

Intentional or accidental combustion of PFASs also forms hazardous chemicals. 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 when fluoropolymers are combusted at temperatures representative of municipal incinerators (Huber et al. 2009). In a review of how fluoropolymers differ from nonpolymeric PFASs, the authors acknowledged that end-of-life considerations should be further investigated because hazardous substances may arise from the combustion of fluoropolymers (Henry et al. 2018). For instance, during incineration at temperatures above 450°C, PTFE forms additional hazardous substances including the ultra-short-chain PFAA tetrafluoroacetic acid (TFA) (Huber et al. 2009) and hydrofluoric acid (HF) (Henry et al. 2018) which appears on DTSC’s Candidate Chemicals list. An industry-sponsored

experimental study in a rotary kiln test facility simulating municipal incinerators found that PTFE polymer pellets begin to decompose at around 500˚C, and by around 650˚C they completely convert to HF gas and F-containing ash, with no significant PFAA emissions (Aleksandrov et al. 2019). Other authors report that at lower temperatures, as could occur during accidental landfill fires, fluoropolymers such as PTFE can break down into PFCAs, including PFOA (Feng et al. 2015; Schlummer et al. 2015).

Thus, while PFAAs constitute a small subset (approximately 1 percent) of PFASs, they are terminal degradation products, manufacturing aids/feedstocks, or impurities of other PFAS class members. This makes their hazard traits relevant to the entire class (see Figure 3).

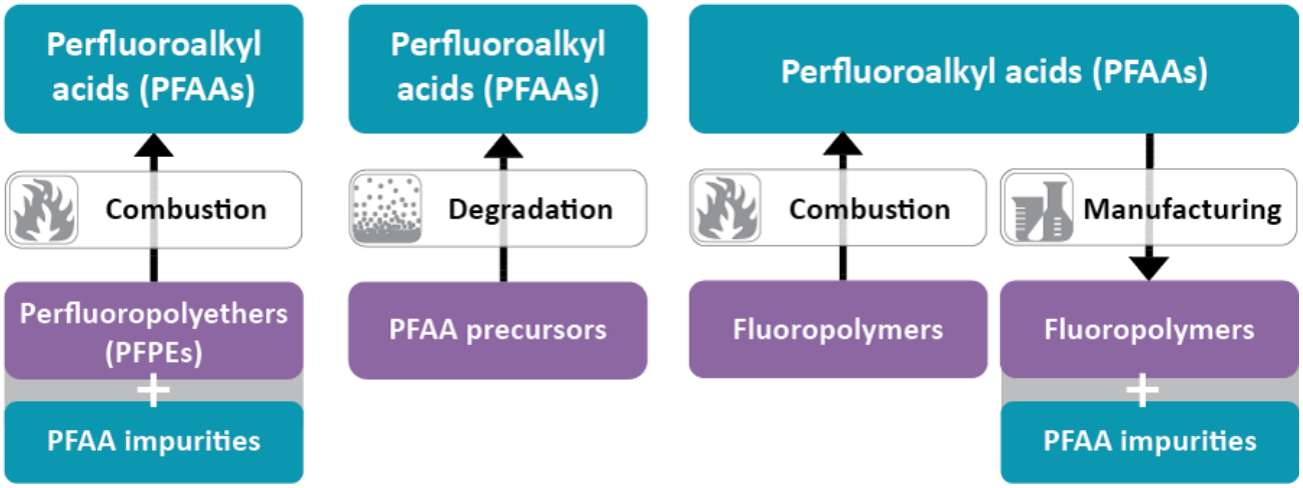

Figure 3: Use of all PFAS subclasses can lead to exposure to PFAAs at some point in the chemicals' life cycle; they either degrade to form PFAAs, release PFAAs if incinerated, or require PFAAs for their manufacture, often resulting in PFAA impurities in the final product. Specifically:

- *Fluoropolymers are made using PFAAs;*
- PFPEs and fluoropolymers can contain PFAA impurities;
- *PFAAs can be released from PFPEs and fluoropolymers during combustion; and*
- *PFAA precursors such as fluorotelomer-based substances, including side-chain fluorinated polymers, degrade to PFAAs.*

Hazard traits and environmental or toxicological endpoints

Reference: California Code of Regulations, title 22, section 69503.3(a)(1)(A).

The hazard traits and environmental or toxicological endpoints summarized in this section are defined in the SCP regulations in sections 69501.1(a)(36) and (33), respectively, both of which refer to the Office of Environmental Health Hazard Assessment's (OEHHA) Green Chemistry Hazard Trait regulations

(California Code of Regulations, Title 22, Chapter 54). These include exposure potential, toxicological, and environmental hazard traits.

PFASs display multiple hazard traits according to OEHHA's Green Chemistry Hazard Traits regulations (California Code of Regulations, title 22, section 69401 et seq.). These include toxicological hazard traits, (Articles 2 and 3), environmental hazard traits (Article 4), and exposure potential hazard traits (Article 5).

Exposure potential hazard traits

Environmental persistence

Environmental persistence is one of the hazard traits identified in the Green Chemistry Hazard Traits Regulations. According to OEHHA's Statement of Reasons for these regulations (OEHHA 2012):

"*Persistence of a chemical in the environment promotes sustained exposure and contributes to accumulation in the environment. Because persistence is an inherent property of a chemical in the environment that results in increased exposure to the chemical and consequently potential for health risks, it can appropriately be identified as a hazard trait. Legacy chemicals such as DDT and PCBs remain public health concerns decades after their production was banned because of their ability to persist in the environment*."

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. PFOS and its salts, perfluorooctane sulfonyl fluoride, PFOA and its salts, and PFOA-related compounds are designated as persistent organic pollutants (POPs) under the Stockholm Convention (UNEP 2020). PFHxS and C11 through C14 PFCAs are listed as very persistent and very bioaccumulative (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, bioaccumulative, and toxic (PBT) (ECHA 2020a). Limited data on some PFECAs and PFESAs suggest resistance to biodegradation (RIVM 2016). PFPEs and fluoropolymers are similarly persistent, whereas PFAA precursors degrade into extremely persistent PFAAs.

Mobility in environmental media

As discussed in Chapter 3, PFAAs are mobile, capable of long-range transport, and have been found in many environmental compartments far away from their sources. Once released into air and water, PFAAs and some of their precursors are highly mobile and undergo long-range transport to distant regions via atmospheric circulation and oceanic currents (Prevedouros et al. 2006; Ahrens et al. 2011b; Muir et al. 2019). Depending on their physicochemical properties, such as water solubility, K_{oc}, and

volatility, PFASs can be transported in the dissolved phase (e.g., through soil water, by rivers and oceanic currents), in the particulate phase (e.g., via suspended sediments or airborne particles), or in the atmosphere. Shorter-chain PFAAs have higher aquatic persistence and mobility than the longer chains, and thus are more frequently detected in the aquatic environment (Li et al. 2020a) and preferentially taken up by plants (Blaine et al. 2013), including food crops (Wang et al. 2015b). Shorter-chain PFAAs are especially mobile in soil and water, hence they accumulate in edible plant parts and are difficult and expensive to remove during water treatment (Brendel et al. 2018; Boone et al. 2019; Ateia et al. 2019a; Crone et al. 2019).

Bioaccumulation

PFAAs tend to bioaccumulate, with elimination half-lives increasing with perfluoroalkyl chain length. In humans, known serum elimination half-lives range from a few days to several years (Bartell et al. 2010; Chang et al. 2008; Olsen et al. 2007b; Nilsson et al. 2010; Seals et al. 2011). 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 vPvB, and PFOA, APFO, PFNA, and PFDA as bioaccumulative (PBT) (ECHA 2020a). PFAAs differ in their uptake and accumulation in human tissues (Burkemper et al. 2017; Pérez et al. 2013). PFAAs can cross the brain blood barrier and are present in animal brain tissue (Greaves et al. 2013). PFAAs, especially the shorter-chain ones, tend to bioaccumulate in plants, as evidenced by findings of higher short-chain PFAA concentrations in plants compared to environmental levels (Blaine et al. 2013; Blaine et al. 2014a; Gobelius et al. 2017; Lechner and Knapp 2011; Scher et al. 2018; Yoo et al. 2011; Chen et al. 2020). The bioaccumulation hazard trait is enhanced by the extreme persistence combined with the high environmental mobility of these chemicals. Biomagnification, which is the increase in contaminant concentration higher in the food chain, has been reported for longer-chain PFAAs in freshwater and marine organisms (Houde et al. 2006). Shorter-chain PFAAs appear not to undergo biomagnification in an Antarctic ecosystem (Gao et al. 2020). Less is known about the potential for bioaccumulation or biomagnification of PFECAs and PFESAs (RIVM 2016). To our knowledge, the bioaccumulation potential of other PFASs besides PFAAs has not been evaluated.

Lactational or transplacental transfer

Transplacental and lactational transfer can be significant routes of exposure for newborns (Cariou et al. 2015; Papadopoulou et al. 2016; Winkens et al. 2017a). PFASs, including PFAAs, nonpolymeric PFAA precursors, and chlorinated polyfluorinated ether sulfonates, undergo transplacental transfer in humans (Midasch et al. 2007; Zhang et al. 2013; Yang et al. 2016; Chen et al. 2017a; Zhao et al. 2017; Eryasa et al. 2019; Cai et al. 2020). A recent study found that up to 30.3 percent of the PFASs transferred from mother to fetus via the placenta were novel compounds that lack information regarding their toxicity and environmental fate (Li et al. 2020c). This suggests that more PFASs could be

undergoing transplacental transfer, beyond those that have been studied to date. The placenta-to-maternal serum ratios of PFOS, PFOA, and PFNA increased during gestation, more so in pregnancies with male fetuses compared to female ones, suggesting bioaccumulation in the placenta and increasing exposure with fetal age (Mamsen et al. 2019). Lactational transfer has also been documented for PFAAs in humans (Kärrman 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.

Global warming potential

Some PFPEs, which are increasingly used as replacements for PFAAs or their precursors, may have high global warming potential (GWP).[7] The GWP of perfluoropolymethylisopropyl ether, a type of PFPE, ranges from 7,620 over 20 years to 12,400 over 500 years, relative to carbon dioxide (CO_2) (IPCC 2007). Some nonpolymeric fluorinated ethers, which can be used in the production of PFPEs and released to the environment, also have high GWP (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, fluoroform (CHF_3), with a GWP 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 reflectance. This increases the efficiency of solar radiation absorption in the atmosphere, contributing to global warming (MacLeod et al. 2014).

Toxicological hazard traits

Toxicological data are limited to a few PFASs, mostly PFAAs. The toxicities of fluorinated polymers, their intermediate degradation products, and processing aids are poorly understood. Appendix C lists toxicological hazard traits for the PFASs with publicly available data (mostly PFAAs and some precursors). 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.

Potential adverse human health effects and risk factors from longer-chain PFAS exposure include:

- Increased serum cholesterol (Skuladottir et al. 2015; Winquist and Steenland 2014);
- Thyroid disease (Winquist and Steenland 2014);

[7] The term GWP is defined by the California Air Resources Board (CARB) at https://ww3.arb.ca.gov/cc/inventory/background/gwp.htm. The GWP value represents the climate forcing of a kilogram of emissions of a substance relative to the same mass of carbon dioxide (CO_2), which has a GWP value of 1. In California, GWPs are calculated over a 100-year time frame.

- Immune dysregulation (Grandjean and Clapp 2014; Grandjean et al. 2017a; Grandjean et al. 2017b);
- 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 first occurrence of menstruation (Rappazzo et al. 2017). PFOA also appears to be associated with low birth weight in humans (Malits et al. 2018).

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 first occurrence of menstruation.

The health effects listed above were observed in studies focused on longer-chain PFASs. Possibly due to their shorter observed biological half-lives, shorter-chain PFASs were expected to have lower toxicity compared to the longer chains (DeWitt 2015; Stahl et al. 2011; Wolf et al. 2008). Notable exceptions are PFHxS (C6) (considered a longer-chain due to its high bioaccumulation potential) and PFBS (C4) in some toxicokinetic studies and receptor binding assays (Wolf et al. 2008; Danish Environmental Protection Agency 2015a). PFBS was reported to be developmentally toxic in zebrafish (Hagenaars et al. 2011) and hematotoxic (reduced red blood cell count, hemoglobin, and hematocrit) (Lieder et al. 2009). The neurodevelopmental toxicity potential of PFBS has been demonstrated *in vitro* by dose-dependent suppression of neuronal differentiation (Slotkin et al. 2008). Delayed pupil response (Butenhoff et al. 2012) and retinal degeneration[8] in rodents (3M 2006) indicate a potential for ocular toxicity in humans from exposure to the ammonium salt of PFBA. Rodent studies have also identified reproductive and developmental (fetal resorption, delayed eye opening) (Das et al. 2008), and respiratory (nasal degeneration) (Loveless et al. 2009) hazards for the ammonium salt of PFBA.

Scientific review of the limited data on the PFECA GenX (a PFOA replacement in some applications) and related perfluorinated ether substances from Chemours (RIVM 2016) indicates potential associations with adverse health effects in laboratory animals, including cancer, body weight gain, changes to the

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

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, 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.

As shown in Appendix C, 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 (Høyer et al. 2015), while another reported a null association between PFOS in cord blood and the neurobehavioral symptoms of attention deficit/hyperactivity disorder (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).

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 nonmammalian 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. Rice et al. (2020) present evidence that PFHxA toxicity and toxicokinetics alone would not adequately inform toxicity characterization of fluorotelomer precursors for which PFHxA is a terminal product (e.g. 6:2 FTOH). 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).

The mode of action of PFAAs has not been fully characterized. Activation of the nuclear peroxisome proliferation-activated receptor alpha (PPAR-alpha) has been associated with some of the hepatic effects of PFOA and PFOS, although other biological interactions associated with PFAAs' neurodevelopmental and reproductive toxicity 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. 2017a).

Environmental hazard traits

Appendix C lists several studies documenting the following environmental hazard traits:

- Phytotoxicity;
- Wildlife developmental impairment;
- Wildlife reproductive impairment; and
- Wildlife survival impairment.

Laboratory studies of environmentally relevant species of algae, aquatic plants, terrestrial plants, fish, amphibians, mammals, birds, and important pollinating insects, as well as studies of birds exposed in the wild, show an association between PFAS exposure and several adverse environmental impacts, including developmental, reproductive, or survival impairment (Annunziato et al. 2019; Eggers Pedersen et al. 2016; Liu et al. 2015b; Sanchez et al. 2015; Soloff et al. 2017). Additionally, PFAS toxicity has been reported across a broad range of aquatic organisms (microorganisms, algae, plants, invertebrates, amphibians, fish, and marine mammals) and adverse impacts (e.g., impaired growth, mortality, developmental effects, and reproductive effects), while the environmental effects on terrestrial wildlife are not as well-studied. See Appendix C 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, ultimately impacting their survival rate. Developmental toxicity was also observed in zebrafish embryos following exposure to 6:2 FTCA (Shi et al. 2017) and to several perfluoropolyether carboxylic acids (Wang et al. 2020). Studies of 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 zebrafish 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 were unavailable (Danish Environmental Protection Agency 2015a). A 2015 study found that levels of PFCAs and PFSAs, including PFHxA and PFBS, found in various brain regions affected neurochemical markers in Greenland polar bears (Eggers Pedersen et al. 2015).

Structural or mechanistic similarity to chemicals with known adverse impacts

Reference: California Code of Regulations, title 22, section 69503.3(a)(3).

Some chemicals may lack sufficient data to definitively establish presence or absence of harm. In such cases, DTSC may also consider data from other chemicals closely related structurally to the Candidate Chemical to identify potential public health and environmental impacts.

While physicochemical properties and hazard traits for many PFASs are either uncharacterized or unavailable in the public domain, the data available for structurally similar PFASs raise sufficient concerns for DTSC to consider PFASs as a class. In particular, longer- and shorter-chain PFAAs share three key structural and mechanistic properties: their structural similarity to fatty acids (DeWitt et al. 2015), their potential to activate PPAR-alpha (Rosenmai et al. 2018; Wolf et al. 2008; Wolf et al. 2014), and their persistence in the environment (Krafft and Riess 2015a). The carbon-fluorine bonds characteristic of the structure of all PFASs endow their extreme environmental persistence.

4. POTENTIAL FOR EXPOSURES TO THE CANDIDATE CHEMICAL IN THE PRIORITY PRODUCT

Reference: California Code of Regulations, title 22, section 69503.3(b).

The SCP regulations direct the Department to evaluate the potential for public or aquatic, avian, or terrestrial animal or plant organism exposure to the Candidate Chemical(s) in the product by considering one or more factors for which information is reasonably available.

Market presence and trends

Reference: California Code of Regulations, title 22, section 69503.3(b)(2).

Product market presence information may be used as a surrogate to assess potential exposures to the Candidate Chemical in the product. This information may include statewide sales by volume, the number of units sold or amount of sales generated, or information on the targeted customer base.

Tests in the 1940s found that fluorochemicals performed best in imparting resistance to the spreading of liquids on solid surfaces. This led 3M to develop the Scotchgard brand of oil and water repellents (Audenaert et al. 1999). The first commercial Scotchgard rain and stain repellent, called FC-208, was released in 1960. Subsequent formulations with increased ability to withstand laundering fall under three major categories: esters, polyurethanes, and acrylate copolymers (Audenaert et al. 1999). But scientific research highlighting the dangers posed by treatments containing PFASs challenged the industry. As a result, 3M announced in 2000 that it was discontinuing the manufacture and sale of Scotchgard, which at that time was based on C8 chemistry; however, the company reintroduced another PFAS-based formulation 16 months later (Singh et al. 2005).

According to data on two broad North American Industry Classification System (NAICS) codes,[9] the number of U.S. manufacturers and sales revenues for these industry sectors have steadily declined from 2007 to 2017, by 17.2 percent and 32.5 percent, respectively (U.S. Census Bureau 2007; U.S. Census Bureau 2012; U.S. Census Bureau 2017). Foreign imports of treatment products, however, may meet any unsatisfied demand for these products. Moreover, market data gaps remain substantial. Accessible government, nongovernmental, academic, and corporate data sources reveal only scarce sales information for specific treatment products.

A September 2019 search of the Mintel Global New Products Database identified approximately 50 new treatment product releases over the previous five-year span (Mintel 2019). These products

[9] These two NAICS codes – 325612 and 325613 – comprise 'polish and other sanitation good' and 'surface active agent' manufacturing. Some nontreatment manufacturers fall within these codes.

included stain removers and protectors and leather protectors. Detecting the presence of PFASs in these types of products, however, remains difficult. Many product labels and Safety Data Sheets fail to provide complete lists of ingredients. In some cases, companies provide no ingredient information whatsoever. Screening methods such as mass spectrometry can be used to only quantify PFASs in a known library and require complicated workflows to accurately identify undiscovered PFASs (Lee et al. 2019). Governmental and academic research offers some level of insight into the presence of PFASs in treatments. For example, a 2008 Danish report estimated that at most 10 percent of the treatments for textiles and leather contain PFASs (Jensen et al. 2008). However, a more recent study reported finding one or more PFASs in 63 percent of the 60 products tested, even though only 10 percent of those products' labels mentioned PFASs (Favreau et al. 2017).

Treatment products containing PFASs often exist in homes and places of work throughout California. Even a person who chooses not to purchase a PFAS-containing upholstery protector may own several spray bottles of PFAS-containing stain remover. A wide range of occupations rely on treatment products that contain PFASs; these include shoe and leather workers and repairers, vehicle cleaners, and building cleaning workers.[10] The vehicle cleaning sector (Standard Occupational Code 53-7061) in California employed nearly 53,000 workers in 2016. While nationally, trends in employment for most of these occupations are projected to move downward through 2028, employment levels in California for nearly all of these occupations are rising substantially (U.S. Bureau of Labor Statistics 2019). For instance, government analysts estimate that building cleaning worker jobs in California will increase by over 15 percent between 2016 and 2026 (California Employment Development Department 2019).

Potential exposures to the Candidate Chemical during the product's life cycle

Reference: California Code of Regulations, title 22, sections 69503.3(b)(3); 69503.3(b)(4)(A-H).

Potential exposures to the Candidate Chemical or its degradation products may occur during various product life cycle stages, including manufacturing, use, storage, transportation, waste, and end-of-life management practices. Information on existing regulatory restrictions, product warnings, or other product use precautions designed to reduce potential exposures during the product's life cycle may also be discussed here.

During their life cycle, products used to treat converted textiles or leathers can expose humans and other living organisms to PFASs via several pathways and routes (Figure 4). PFASs are released into air, water, and soil during the manufacture, consumer use, and landfilling, incineration, or recycling of

[10] Additional occupations that risk exposure to treatments containing PFASs include carpet installers and automotive service technicians and mechanics. 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. Bureau of Labor Statistics 2016).

converted textile or leather products (Ahrens et al. 2011a; Oliaei et al. 2013), including via evaporation during the product use phase and when washing treated textiles or leathers (Favreau et al. 2017). Administrative and engineering controls are unlikely to address exposure concerns during product use and disposal. PFASs released during the manufacture and use of treatments for converted textiles or leathers can enter waterways via direct discharge from manufacturers, release of WWTP effluent, or following land application of contaminated biosolids (Sepulvado et al. 2011), 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).

PFASs are released into air, water, and soil during stain- and soil-repellent manufacturing, consumer use, and landfilling of treatments used in converted textile or leather products.

Manufacturing

Volatile PFASs can be released into the environment during the manufacture of treatments for textiles or leathers (Stock et al. 2004). Manufacturing wastewater discharges into WWTPs can contaminate rivers and other waterways (Konwick et al. 2008). Once released into air and water, PFAAs and some of their precursors are highly mobile and undergo long-range transport to distant regions via atmospheric circulation and oceanic currents (Prevedouros et al. 2006; Ahrens et al. 2011b; Muir et al. 2019).

PFAAs and their precursors can contaminate soil and sediments via deposition of contaminated particulate matter, use of contaminated irrigation water, and land application of contaminated biosolids as fertilizers (Armitage et al. 2009; Blaine et al. 2014a; Lindstrom et al. 2011a; Sepulvado et al. 2011; 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 PFASs (C4 through C14) in sediment correlated with proximity of the sampling locations to industrial activities in food packaging, textile, electroplating, firefighting, semiconductor, precious metals processing, and coating industries.

Use

Manufacturers of converted textile and leather products generally recommend reapplication of treatment products every few years to ensure optimal stain and soil resistance. This suggests that PFASs migrate from converted textile or leather products. Migration of PFASs from products is exacerbated in certain instances such as competitive skiing, where frequent reapplication of treatments on ski clothing and associated products contributes to very high levels of PFASs in snow and contamination of the food chain, water, and soil (Hanssen et al. 2019).

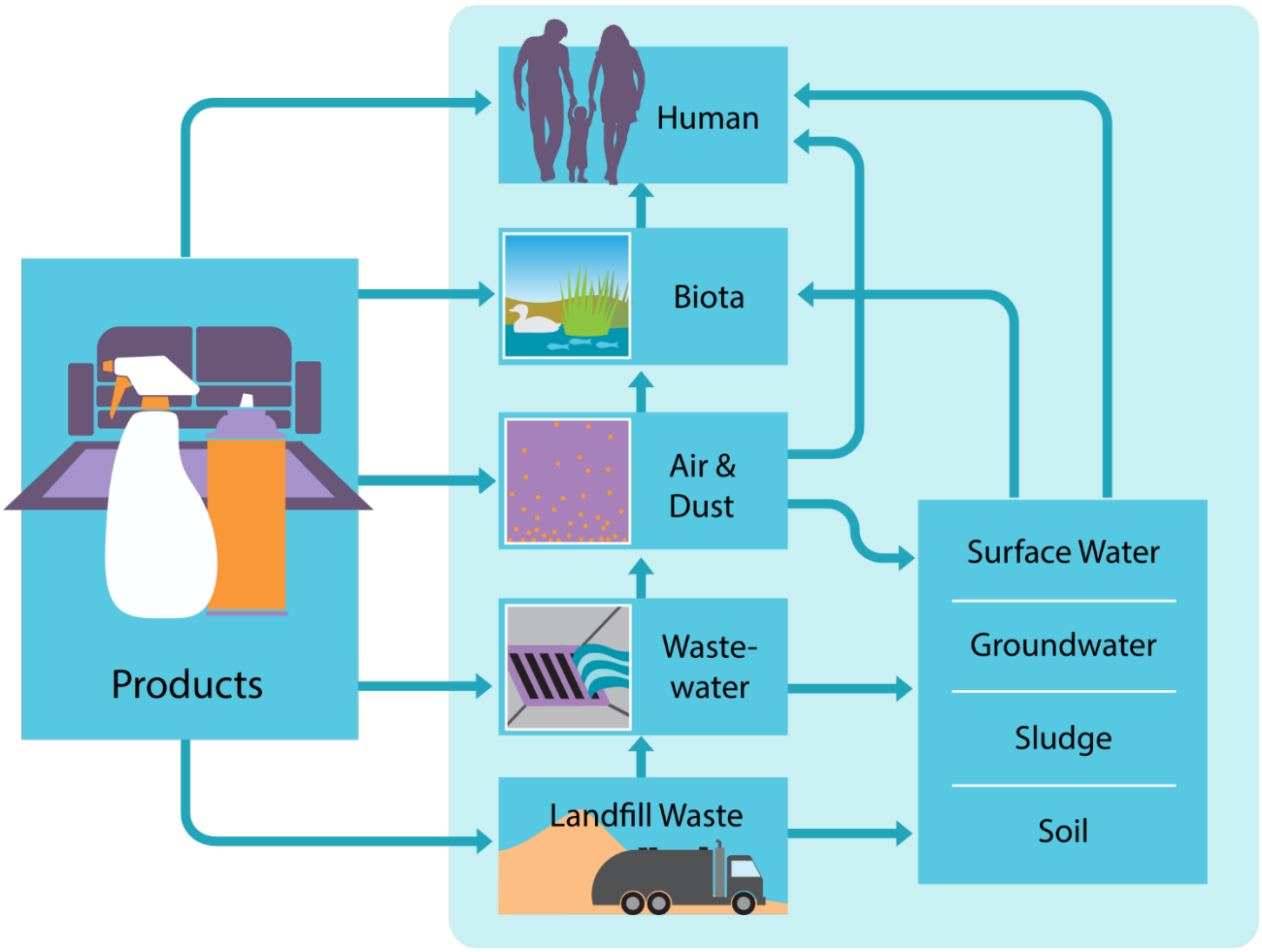

Figure 4: Key PFAS exposure pathways from treatments for converted textiles or leathers.

Weather conditions like sunlight, high temperature, and humidity may accelerate the degradation of precursors, including the side-chain fluorinated polymers applied to textiles or leathers, into PFAAs (van der Veen et al. 2020). One study found that the side-chain fluorinated polymer surfactant contained in the Scotchgard fabric protector metabolizes *in vitro* into the PFBS precursor perfluorobutane sulfonamide (FBSA) (Chu and Letcher 2014). This suggests that, if inhaled during application, the fluorinated polymeric surfactants contained in these treatments can metabolically degrade into PFAAs and accumulate inside the human body.

U.S. EPA researchers studied the PFASs found in 13 consumer product categories and 116 household products. They concluded that many of the PFASs detected in household dust can be attributed to the use of treatment products applied to converted textiles and leathers, such as carpeting, apparel, and upholstery. Carpet and fabric care liquids were among the greatest sources of exposure. It is estimated that the average household has 1 kg of household carpet or fabric care liquids for spot treatment, which can introduce up to 950 µg of PFCAs during application. Furthermore, a single professional

treatment uses about 6 kg of commercial-grade carpet/fabric care liquids, resulting in the addition of up to 71,800 µg of PFCAs to the home (U.S. EPA 2009b). A Canadian family that routinely treated its carpets with a Scotchguard formulation was found to have very high PFHxS levels in household dust and serum. The family's young children had exceptionally high serum PFAS levels, which is likely attributed to the increased contact with treated articles and opportunity for exposure via dust ingestion or inhalation (Beesoon et al. 2012). The inhalation exposure pathway to treatment products containing PFASs is particularly significant due to the aerosolization of the product formulations during application (Trudel et al. 2008). One study estimated that exposure to FTOHs via inhalation of spray droplets from textile treatments ranges from 52 to 2,522 ng/kg body weight (Favreau et al. 2017).

Due to consumer use of treated textiles and leathers, PFASs are found in home and office air samples, and in the blood of residents and office workers (Beesoon et al. 2012; Fraser et al. 2012; Fraser et al. 2013). Human exposure to PFASs occurs via inhalation of volatile PFASs and PFAS-containing fine particles, as well as via incidental ingestion of indoor dust, which concentrates PFASs (Fraser et al. 2012; Harrad et al. 2010; Haug et al. 2011; Tian et al. 2016; Rosati et al. 2008; Shoeib et al. 2005; Zheng et al. 2020).

PFAAs from converted textile or leather treatments can also adsorb to house dust or be released as tiny particles from surface abrasion during normal use (Rosati et al. 2008; 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. 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 incidental dust ingestion is generally considered a minor pathway of exposure to PFASs in adults, it can be significant in toddlers and young children (Tian et al. 2016; Wu et al. 2015; Wu et al. 2020). Karásková et al. (2016) measured 20 PFASs in dust from homes and found that exposure doses via incidental dust ingestion were significantly higher in toddlers than adults. Also, children's mouthing of treated textile and leather articles could lead to PFAS exposure, particularly to the shorter-chain PFAAs (CEC 2017). This indicates an increased potential for adverse impacts in this vulnerable subgroup.

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 converted textiles or leathers (Herzke et al. 2012; NILU 2015; Vestergren et al. 2015; Schlummer et al. 2015; Kotthoff et al. 2015). Compared to outdoor air, indoor air can have more than 1,000 times higher levels of FTOHs (Fraser et al. 2012; Müller et al. 2012). Fraser et al. (2012) found that FTOH concentrations in office air predicted PFOA concentrations in the blood of workers.

An air and dust sampling study in several indoor locations in China estimated the daily intake of PFASs for adults (Yao et al. 2018). The study found that exposure via inhalation ranges between 1.04 and 14.1 ng/kg body weight per day, while exposure via incidental dust ingestion ranges between 0.10 and 8.17 ng/kg body weight per day. Biota can also experience potential exposures via environmental media and diet.

Cleaning of treated textile and leather may dispose of PFAS-contaminated wastewater in drains, entering WWTPs in areas served by public water systems (CCCSD 2020; CASA 2020). Migration studies have been conducted for textile articles treated with side-chain fluorinated polymers. Shorter-chain PFAAs, due to their greater solubility, migrate more readily from textiles during simulated laundering and when exposed to artificial saliva (CEC 2017). A European study on accelerated washing found that side-chain fluorinated polymers used to treat rain jackets cleave into shorter-chain PFAAs and emit roughly 0.7 tons of fluorotelomer alcohol (6:2 FTOH) per year into the environment in Europe alone (Schellenberger et al. 2019b). While polymeric PFASs form a chemical bond with the surfaces they are applied to, they have shown a decrease in water repellency as treated products are put through repeated wash cycles; concurrently, higher concentrations of PFASs have been measured in wash water (Knepper et al. 2014). 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. 2014; Schaider et al. 2016). WWTP discharge can have PFOA concentrations up to hundreds of ng/L (Hamid and Li 2016). Discharge of PFAA-contaminated effluent into surface waters can lead to contamination of streams, rivers, and sediments (Ahrens et al. 2011a; Sun et al. 2012).

End-of-life

In landfills, the side-chain fluorinated polymers currently used in most stain- and soil-resistant treatments for converted textiles and leathers can degrade, first, to form nonpolymeric fluorotelomer-based PFASs such as FTOHs, 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 source (years to even centuries) of PFAAs to the environment (Lang et al. 2017; Li et al. 2017; Washington et al. 2019). 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; Li et al. 2020a).

Landfills have been identified as a source of PFAS contamination to nearby air and plant leaves (Tian et al. 2018). PFAAs and their precursors from discarded treatment containers and treated textile and leather products can be released into the atmosphere during decomposition in landfills, and become widely dispersed (Ahrens et al. 2011a). In a study conducted in Vermont, PFASs were detected in waste samples at levels ranging from 0.043 to 2,030 parts per billion (ppb), with the highest concentrations

measured from bulky items such as furniture, textiles, carpeting, and leather (Sanborn, Head & Associates, Inc. 2019). PFAAs are also 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 PFAAs from the effluent (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), Norway (Knutsen et al. 2019), Canada (Benskin et al. 2012b), Uganda (Dalahmeh et al. 2018), and Australia (Gallen et al. 2017; Hepburn et al. 2019). In the U.S., an estimated 61.1 million cubic meters 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 under simulated landfill conditions found increased concentrations leaching 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 for more than a decade or longer (Lang et al. 2017; Washington et al. 2019).

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Furthermore, biosolids from WWTPs can be heavily contaminated with PFAAs and precursors that adsorb to organic matter (Arvaniti et al. 2012; Gallen et al. 2018; Kim Lazcano et al. 2019; Sun et al. 2011; Kim Lazcano et al. 2020). The side-chain fluorinated polymer surfactants that are the main components of Scotchgard fabric protector products were found in high concentrations in all biosolid samples tested from WWTPs across Canada (Letcher et al. 2020). When these materials are land-applied or landfilled, shorter-chain PFASs can migrate from biosolids into soil and leach into adjacent waterways (Sepulvado et al. 2011), 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. 2014a; Lee et al. 2014). PFAA precursors such as those used in treatments for converted textiles or leathers can also undergo transformation to PFAAs in soils (Washington et al. 2015).

Waste incineration may also release PFASs and other hazardous chemicals to the environment. A literature survey regarding the combustion of various fluorinated polymers reported emissions of C3-C14 PFCAs, ozone depleting substances such as CFCs, and greenhouse gases such as fluorocarbons (Huber et al. 2009). During incineration at temperatures above 450°C, PTFE forms TFA (Huber et al.

2009) and other hazardous thermal degradation products such as hydrofluoric acid (HF) (Henry et al. 2018), which is on DTSC's Candidate Chemicals list. Other studies show that at lower temperatures, as could occur during accidental landfill fires, fluoropolymers can break down into PFCAs, including PFOA (Feng et al. 2015; Schlummer et al. 2015). A 2015 study found that, even at temperatures above 400˚C and in the presence of calcium hydroxide, PTFE does not undergo complete mineralization (Wang et al. 2015a).Recycling of postconsumer converted textiles or leathers such as carpet, upholstery, and clothing is a preferred alternative to landfilling or incineration, but can perpetuate the presence of PFASs in products made from the recycled material (Herzke et al. 2012). Carpet recycling includes several processing steps that generate high amounts of dust, such as hammer milling, shearing, and shredding (CARE 2012). Workers engaged in recycling activities, including waste handlers, could be exposed to PFASs in dust, while nearby residents could be exposed via off-site dust migration (HBN 2017).

Aggregate effects

Reference: California Code of Regulations, title 22, section 69503.3(a)(1)(B) and sections 69503.3(b)(3).

Multiple sources of exposure to the Candidate Chemical may increase the potential for significant or widespread adverse impacts.

PFASs are found in many frequently used consumer products in Californian households and workplaces, including carpets, rugs, flooring, furniture, clothing, cookware, cleaning products, personal care products, electronics, and food packaging (KEMI 2015), as well as in food (Christensen et al. 2017) and drinking water (Schwanz et al. 2016; Boone et al. 2019; Domingo and Nadal 2019). The indoor environment is a significant source of PFAS exposure due to the presence of PFAS-containing consumer products. The PFASs can evaporate from these products and partition to the indoor air, dust, or surfaces (Zheng et al. 2020). Overall, humans can be exposed to PFASs through a combination of dietary and nondietary pathways, including ingestion and inhalation. To add to this complexity, exposure to PFAAs can be categorized as direct or indirect. Direct exposure occurs when PFAAs enter the body, for example, through inhalation or ingestion. Indirect exposure means exposure to one or more PFAA precursors that are subsequently biotransformed to PFAAs in our bodies (Poothong et al. 2020). Since PFASs are ubiquitous in both indoor and outdoor environments, the general population is exposed to complex PFAS mixtures via multiple exposure pathways simultaneously. This makes it difficult to model and predict aggregate exposure scenarios for individuals. Better understanding of population-wide aggregate exposures requires both generating more human biomonitoring data and expanding analytical methods to include more PFAS analytes. Nevertheless, DTSC considers all *potential* sources of PFAS exposure under its mission to protect the people and biota of California.

Because of the prevalence of PFAS-treated products in residential and commercial spaces, as well as in food and drinking water sources, all Californians are potentially exposed to PFASs, directly or indirectly. The Chinese textile industry is estimated to use about 10,000 metric tons of finishing treatments containing fluorinated chemicals (Lim et al. 2011). These materials and products are then exported globally. High PFAS concentrations have been detected in textile products originating from China, including cotton and leather clothing, bedding, curtains, furniture coverings, and most notably carpeting (Vestergren et al. 2015).

Despite extensive documentation of the widespread environmental PFAS contamination from consumer products such as treatments for converted textiles or leathers, longitudinal studies that evaluate aggregate exposures to these chemicals from all sources through different life stages are still lacking. A lack of comprehensive data on source emissions and the distribution of PFASs through all environmental compartments makes it challenging to accurately estimate aggregate exposures over a lifetime. The frequency, level, and duration of individual or population-level PFAS exposures associated with the use of treatments for converted textiles or leathers have not been estimated using either deterministic or probabilistic exposure modeling.

Indicators of potential exposures to the Candidate Chemical in the product

Reference: California Code of Regulations, title 22, section 69503.3(b)(2).

The SCP regulations consider various data that indicate potential for exposure to the Candidate Chemical or its degradation products, including: the Candidate Chemical's presence in and release from the product; monitoring data indicating the Candidate Chemical's presence in the indoor and outdoor environment, biota, humans (e.g., biomonitoring studies), human food, drinking water, and other media; and evidence of persistence, bioaccumulation, lactational and transplacental transfer.

PFASs are found ubiquitously in the environment, plants, animals, and humans (Campo et al. 2016; Lindstrom et al. 2011a; Lindstrom et al. 2011b). Treatments for converted textiles or leathers contribute to widespread environmental contamination and exposures, as do other consumer products such as carpets, rugs, furniture, 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 human and ecological exposures (Figure 4). 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 consumer products (Xiao 2017). However, most of the PFASs that contribute to total organic fluorine in the environment, wildlife, and human blood samples remain unidentified due to analytical limitations (Xiao 2017; Spaan et al. 2019). Thus, only a small fraction (sometimes less than 5 percent) of the PFASs in

these media are likely reflected in the data summarized in this section. Nakayama et al. (2019) discussed the available analytical methods for measuring PFASs in air, water, sediments, soil, sludge, dust, and biological samples and identified a great need for new methods that expand the suite of PFASs studied, especially in humans and wildlife. As of now, 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 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.

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 treatments is lacking. Using data from the Danish Product Register, the Danish EPA calculated the total content of fluorinated compounds in treatments for shoes to be between 0.03 and 60 g/kg (Jensen et al. 2008). Another study conducted on several product categories detected PFAS in sprays for fabrics and textiles (Ye et al. 2015).

Although only polymeric PFASs are currently used in treatments for converted textiles or leathers (Bowman 2017; FluoroCouncil 2017), nonpolymeric PFASs can be present as manufacturing impurities or degradation products (Kotthoff et al. 2015; Vestergren et al. 2015). FTOHs, which are used as manufacturing intermediates, are also present as 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). A weather-protector spray was found to contain high levels of fluorine and PFASs: over 14,000 parts per million (ppm) total fluorine, over 46,000 ppb 6:2 FTOH, and over 65,000 ppb of the related chemical 6:2 fluorotelomer methacrylate (Peaslee 2019). The same research group investigated six pairs of shoes and determined that four of them contained total fluorine levels as high as 1,219 ppm, with 8:2 FTOH and 10:2 FTOH levels of 3,516 ppb and 738 ppb respectively (Peaslee 2019). A study in Switzerland found FTOHs at levels up to 1,840 ppm in treatment products (Favreau et al. 2017). Studies in Europe and Canada found FTOH levels as high as 9,420 ng/L, and the sum of PFCAs as high as 8.4 ng/L in treatments for converted textiles or leathers (Jensen et al. 2008). A sampling of textiles used as materials for outdoor products showed a PFHxA concentration of up to 17,100 ng/m^2, while leather samples exhibited up to 241,800 ng/m^2 PFBA and 143,000 ng/m^2 PFBS (Kotthoff et. al. 2015). Carpet/fabric care liquids and foams in the U.S. in 2011 and 2013 were found to contain up to 105,000 ng/g 6:2 FTOH, 2,950 ng/g 8:2 FTOH, and 1,460 ng/g 10:2 FTOH (Liu et

al. 2015c). Treated home textiles and upholstery were found to contain up to 40,900 ng/g 6:2 FTOH, 21,200 ng/g 8:2 FTOH, and 12,100 ng/g 10:2 FTOH (Liu et al. 2015c).

Until relatively recently, the side-chain fluorinated polymers used in treatments for converted textiles or leathers were made with longer-chain PFASs (more than seven carbon atoms) and included impurities such as PFOS, PFOA, and 8:2 FTOH (Jensen et al. 2008). Because of these side chains' potential to cause adverse health effects, PFOS-based substances were phased out by major U.S. producers over a decade ago (Prevedouros et al. 2006). Currently, domestically produced treatments are made with shorter-chain side-chain fluorinated polymers, mainly urethane-based polymers and some (meth)acrylates. These degrade to short-chain FTOHs, such as 6:2 FTOH, which are more volatile than the longer-chain ones and thus more likely to be released from products (U.S. EPA 2020a).

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 3 for examples]. Despite the phaseout of longer-chain PFASs that started more than a decade ago, concentrations in environmental samples and biota have not declined (Land et al. 2018). This indicates that ecosystems continue to be exposed to previously released PFASs for a long time, even after their use is discontinued.

Table 3: Monitoring studies found varying levels of PFASs in the environment and biota.

Sample Type	PFAS Type: Concentration	Reference
Indoor air (homes)	6:2 FTOH: 1.8 ng/m³ (median) 8:2 FTOH: 8.7 ng/m³ (median) 10:2 FTOH: 2.5 ng/m³ (median) 8:2 FTAC: 0.27 ng/m³ (median) 10:2 FTAC: 0.12 ng/m³ (median)	Fromme et al. (2015)
Indoor air (homes)	Sum of FTOHs: 3.21 ng/m³ (median) Sum of longer-chain PFCAs: 0.34 ng/m³ (median) Sum of shorter-chain PFCAs: 0.35 ng/m³ (median) Sum of PFSAs: 0.23 ng/m³ (median) Sum of diPAPs: 0.0012 ng/m³ (median)	Yao et al. (2018)

Sample Type	PFAS Type: Concentration	Reference
Indoor air (hotels)	Sum of FTOHs: 3.33 ng/m³ (median) Sum of longer-chain PFCAs: 0.21 ng/m³ (median) Sum of shorter-chain PFCAs: 0.26 ng/m³ (median) Sum of PFSAs: 0.12 ng/m³ (median) Sum of FOSE/FOSAs: 0.0053 ng/m³ (median) Sum of diPAPs: 0.0011 ng/m³ (median)	Yao et al. (2018)
Indoor air (schools)	6:2 FTOH: 3.3 ng/m³ (median) 8:2 FTOH: 4.4 ng/m³ (median) 10:2 FTOH: 1.8 ng/m³ (median) 8:2 FTAC: 0.45 ng/m³ (median) 10:2 FTAC: 0.31 ng/m³ (median)	Fromme et al. (2015)
Indoor air (offices)	6:2 FTOH: 1.3 ng/m³ (geometric mean) 8:2 FTOH: 9.9 ng/m³ (geometric mean) 10:2 FTOH: 2.9 ng/m³ (geometric mean)	Fraser et al. (2012)
Indoor dust (homes)	Sum of nine PFCAs (C6 to C14): <1.0 – 37,400 ng/g	Liu et al. (2011b)
Indoor dust (homes)	Sum of longer-chain PFCAs: 59.5 ng/g (median) Sum of shorter-chain PFCAs: 87.4 ng/g (median) Sum of PFSAs: 17.1 ng/g (median) Sum of diPAPs: 1.13 ng/g (median)	Yao et al. (2018)
Indoor dust (hotels)	Sum of FTOHs: 149 ng/g (median) Sum of longer-chain PFCAs: 25.8 ng/g (median) Sum of shorter-chain PFCAs: 3.25 ng/g (median) Sum of PFSAs: 10.3 ng/g (median) Sum of FOSE/FOSAs: 2.1 ng/g (median) Sum of diPAPs: 10.2 ng/g (median)	Yao et al. (2018)
Indoor dust (child care centers)	Sum of PFCAs: 8.37 – 386 ng/g Sum of PFSAs: 1.29 – 190 ng/g Sum of 42 PFASs: 46.9 – 6470 ng/g	Wu et al. (2020)
Indoor dust (child care centers)	Sum of PFCAs: 3.4 – 32 ng/g Sum of PFSAs: 0.36 – 37 ng/g Sum of FTS: not detected – 82 ng/g Sum of FTOH: not detected – 3100 ng/g Sum of FOSA/FOSE: not detected – 380 ng/g	Zheng et al. (2020)

Sample Type	PFAS Type: Concentration	Reference
Indoor dust (day care center)	PFOA: 31 – 110 ng/g PFOS: 23 – 65 ng/g	Björklund et al. (2009)
Indoor dust (classrooms)	PFOA: <0.05 – 31 ng/g PFNA: <0.05 – 0.71 ng/g PFBS: <0.25 – 49 ng/g PFHxS: <0.1 – 120 ng/g PFOS: <0.1 – 21 ng/g	Harrad et al. (2019)
Indoor dust (cars)	PFOA: <0.05 – 14 ng/g PFNA: <0.05 – 3.1 ng/g PFBS: <0.25 – 170 ng/g PFHxS: <0.1 – 49 ng/g PFOS: <0.1 – 82 ng/g	Harrad et al. (2019)
Outdoor air	FTOHs: 0.06 – 0.19 ng/m^3	Müller et al. (2012)
North American soils	Sum of PFCAs: 145 – 6,080 ng/kg Sum of PFSAs: 35 – 1,990 ng/kg	Rankin et al. (2016)
Sediments (South Bay Area, California)	PFOS: 0.60 – 2.61 ng/g dry weight	Sedlak et al. (2017)
Sediments (Cape Fear River, North Carolina)	GenX (PFPrOPrA): 3.1 – 21.6 ng/g dry weight	UNCW (2018)
Sediments (from aMatikulu and uMvoti estuaries, South Africa)	PFBA: 0.15 – 1 ng/g PFOA: 0.26 – 2.5 ng/g PFOS: 0.05 – 0.99 ng/g PFDoA: 0.03 – 0.63 ng/g PFTrDA: 0.03 – 0.37 ng/g	Fauconier et al. (2019)
South African estuaries (aMatikulu and uMvoti)	aMatikulu – PFOA: 171 – 258 ng/L uMvoti – PFOA: 711 – 788 ng/L	Fauconier et al. (2019)
U.S. river (Cape Fear, North Carolina), used for drinking water	GenX (PFPrOPrA): 631 ng/L (mean)	Sun et al. (2016)

Sample Type	PFAS Type: Concentration	Reference
WWTP effluent (San Francisco Bay Area, California)	PFBA: 16 ng/L (mean) PFPeA: 12 ng/L (mean) PFHxA: 26 ng/L (mean) PFBS: 2.7 ng/L (mean) PFHxS: 4.8 ng/L (mean) PFOS: 13 ng/L (mean)	Houtz et al. (2016)
WWTP effluent (Germany)	PFBA: <4.2 ng/L PFPeA: <254 ng/L	Frömel et al. (2016)
Sewage sludge	Sum of 14 PFAAs: 126 – 809 µg/kg	Yan et al. (2012)
Fish (whole fish from the Ohio, Missouri, and Upper Mississippi Rivers)	Sum of 10 PFAAs: 5.9 – 1,270 µg/kg; 53.4 µg/kg (median wet weight)	Ye et al. (2008)
Prey fish (San Francisco Bay, California)	PFOS: 11.8 µg/kg (geometric mean wet weight)	Sedlak et al. (2017)
Cormorant eggs (San Francisco Bay, California)	PFOS: 36.1 – 466 µg/kg (wet weight)	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)[11]: 0.02 – 0.26 µg/kg	Müller et al. (2011)
Baltic Sea cod (liver)	PFOS: 6.4 – 62 µg/kg	Kowalczyk et al. (2019)
Eastern Arctic beaked redfish (liver)	PFOS: 0.5 – 2.5 µg/kg	Kowalczyk et al. (2019)
Polar cod (liver)	PFOS: 1.89 – 2.15 µg/kg	Kowalczyk et al. (2019)
Arctic caribou (liver)	Sum of six PFCAs (C8 to C13): 6 – 10 µg/kg	Müller et al. (2011)

[11] C8 to C13 refers to the number of carbon atoms in the PFCA molecule. This range thus covers: PFOA, PFNA, PFDA, PFUnA, PFDoA, PFTrDA.

Sample Type	PFAS Type: Concentration	Reference
Arctic wolf (liver)	Sum of six PFCAs (C8 to C13): 10 – 18 µg/kg	Müller et al. (2011)
Arctic reindeer	PFBA: 0.079-14 µg/kg	Ahrens et al. (2016b)

Air and dust

PFASs are ubiquitous in air and dust, as evidenced by numerous recent studies. All dust samples collected from 18 California child care centers in 2018 contained at least seven PFCAs (PFPeA, PFHxA, PFHpA, PFOA, PFNA, PFTrDA, and PFTeDA), two PFSAs (PFBS and PFOS), and two PFAA precursors (6:2 FTOH and 8:2 FTOH), with 6:2 FTOH and 6:2 FTSA found at highest levels (Wu et al. 2020). Similarly, all dust samples collected from eight child care facilities in other U.S. states (Washington and Indiana) contained PFASs, and 6:2 FTOH was the dominant compound (Zheng et al. 2020). All indoor air and dust samples collected in 2015 from residences, hotels, outdoor equipment stores, curtain stores, and carpet stores in China contained at least one PFAA or precursor (FTOHs, FOSE/FOSAs, or diPAPs) (Yao et al. 2018). A recent study identified 34 emerging PFASs (including PFECAs and short-chain PFAA precursors) in indoor and outdoor airborne particulate matter from five Chinese cities (Yu et al. 2018). PFASs were found in indoor dust in homes sampled on four continents, as well as in British cars, classrooms, and offices (Goosey and Harrad 2011). 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 FTAC and 6:2 FTMAC were also frequently detected, sometimes at high concentrations, in children's bedroom air sampled in Finland during 2014/2015 (Winkens et al. 2017b). 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 widespread 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). According to a meta-analysis, U.S. household dust can contain hundreds of chemicals (Moschet et al. 2018), including some with hazard traits similar to those of 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), incidental 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), glacial meltwater and snow (Skaar et al. 2018), oceans (Benskin et al. 2012a; González-Gaya et al. 2019; Zhang et al. 2019c), estuaries (Fauconier et al. 2019), 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 manufacturing sites (Konwick et al. 2008). 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 (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). 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 high levels (631 ng/L on average, with a maximum of approximately 4,500 ng/L). In a recent U.S. EPA – U.S. Geological Survey study, PFBS and PFOA were detected in the source waters of all 25 drinking water treatment plants tested from across the U.S., while PFHxS, PFOS, PFBA, PFPeA, PFHxA, PFHpA, PFNA, and PFDA were detected at over 90 percent of the sites (Boone et al. 2019). Certain PFECAs and PFESAs were detected in surface waters in all countries on three continents surveyed recently, indicating global distribution and contamination (Pan et al. 2018). The ultra-short-chain PFAS TFA was also detected in seawater, rain, and melted snow on several continents (Ateia et al. 2019a). Contaminated surface waters can be ingested by livestock and wild animals, leading to exposure up the food chain.

WWTP effluent

PFASs are commonly found in municipal and industrial wastewaters (Appleman et al. 2014; Arvaniti et al. 2015; Chen et al. 2017b; Clara et al. 2008; Sun et al. 2012; Coggan et al. 2019). 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 (D'eon et al. 2009). WWTPs are major point sources for PFAS contamination of aquatic environments (Ahrens 2011; Dalahmeh et al. 2018; Frömel et al. 2016; Gallen et al. 2018; Guerra et al. 2014). Degradation of PFAA precursors, such as FTOHs, within WWTPs can lead to increases in effluent PFAA concentrations (Ahrens 2011; Dalahmeh et al. 2018; Frömel et al. 2016; Gallen et al. 2018; Guerra et al. 2014). Despite the phaseout of PFOS- and PFOA-containing stain- and soil-repellent treatments for converted textiles or leathers, these PFASs are frequently detected in wastewater, indicating their ongoing release into the environment (Gallen et al. 2018; 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 samples collected in 2012

from 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. 2012b; Fuertes et al. 2017; Gallen et al. 2017; Hamid et al. 2018; Knutsen et al. 2019). A study of 95 leachate samples from 18 U.S. landfills estimated the annual release of PFCAs, FTCAs, and PFSAs plus 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 that landfill leachates had the highest average total PFAS concentration (487 ng/L) (Ahrens et al. 2016a). Shorter-chain PFAAs have become the most abundant PFASs in landfill leachate, reflecting the relatively recent market shift from longer- to shorter-chain compounds (Hamid et al. 2018; Knutsen et al. 2019). A study of leachate from municipal and industrial landfills in Sweden also detected ultra-short-chain PFASs (with three or fewer carbon atoms) such as TFA, perfluoropropanoic acid (PFPrA), and trifluoromethane sulfonic acid (TFMS) (Björnsdotter et al. 2019).

Soils

PFASs are ubiquitous in soils. Surveys of soils around the world, including remote locations such as Antarctica and the Arctic Circle, found PFASs in all samples (Llorca et al. 2012; Rankin et al. 2016; Hanssen et al. 2019). Short-chain PFAAs were found in higher concentrations than PFOS in soil samples collected from residential areas throughout China, reflecting their widespread use (Li et al. 2020b). Side-chain fluorinated polymers were detected in all biosolid-treated soil samples measured in a Canadian study (Chu and Letcher 2017a). 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). An evaluation of agricultural lands in Kampala, Uganda, found PFASs in the terrestrial food chain, including in plants such as maze, yam, and sugar cane (Dalahmeh et al. 2018). 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 aboveground 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 11 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). Consistent with 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 (Pan et al. 2014b). PFPAs and PFPiAs have also been detected in lake sediments, but at lower concentrations compared to other PFAAs (Guo et al. 2016). A recent study found that Cape Fear River sediments appear to act as a repository for GenX, potentially releasing it into the water column (UNCW 2018). 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 those PFAAs that adsorb to particulate matter (Prevedouros et al. 2006; Sanchez-Vidal et al. 2015).

PFAAs are found in numerous species, including plankton, sea turtles, seals, California sea otters, whales, fish, penguins, sharks, polar bears, dolphins, seabirds, white-tailed eagles, and bird eggs. This indicates potential for widespread adverse impacts throughout food webs.

Biota

PFAAs are found in numerous species, including plankton (Casal et al. 2017; Zhang et al. 2019c), sea turtles (Keller et al. 2005; O'Connell et al. 2010; Keller et al. 2012), seals (Routti et al. 2016), California sea otters (Kannan et al. 2006), whales (Hart et al. 2008), fish (De Silva et al. 2016; Wong et al. 2017), penguins (Llorca et al. 2012), sharks (Kumar et al. 2009), polar bears (Dietz et al. 2008; Liu et al. 2018b; Tartu et al. 2018), dolphins (Adams et al. 2008; Houde et al. 2005; Lynch et al. 2019; De Silva et al. 2016), seabirds (Escoruela et al. 2018; Roscales et al. 2019), white-tailed eagles (Sun et al. 2019), and bird eggs (Verreault et al. 2007; Wu et al. 2019). This indicates potential for widespread adverse impacts throughout food webs.

The Washington State Department of Ecology surveyed regional lakes and analyzed water, fish tissue, and osprey eggs for PFAS contamination. They found that PFOS biomagnified through trophic levels by several orders of magnitude, but they did not observe any bioaccumulation of shorter-chain PFASs (State of Washington 2017).

As shorter-chain PFAA precursors have gained market share, the concentrations of shorter-chain PFAAs in the marine environment have increased, as evidenced by increasing PFBS levels in cetaceans from 2002-2014 (Lam et al. 2016). The PFBS precursor 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). 6:2 Cl-PFESA and hexafluoropropylene oxide trimer acid (a PFOA replacement) accumulate in frogs, particularly in male

frog skin, liver, and muscle, and in female frog ovaries, raising concerns regarding potential developmental toxicity (Cui et al. 2018).

PFCAs are found in Arctic seals and Antarctic seabirds, presumably due to long-range transport of precursors such as FTOHs and FASAs via oceanic and atmospheric currents (Llorca et al. 2012; Routti et al. 2016; Roscales et al. 2019). In an Antarctic ecosystem, shorter chains dominated the PFAA content of sediments, algae, gastropods, and fish (Gao et al. 2020). In the Arctic, increased coastal PFAS concentrations can follow ice and snow melt, 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 17 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). A long-term study of archived white-tailed eagle feathers collected in Greenland, Norway, and Sweden showed increasing levels of PFCAs over time, but a decrease in PFOS levels in feathers from Greenland and Norway, consistent with the 3M phaseout that took place in 2000 (Sun et al. 2019). In biological samples taken from Norwegian animals, the highest PFAS levels were measured in arctic foxes, otters, and polar bears; the study also detected PFASs in wolves for the first time in Norway (Hanssen et al. 2019). A Swedish study showed that contamination is exacerbated by point sources such as manufacturing plants and firefighting activities; PFASs were detected in the impacted surface water samples collected near such sources at levels up to 1,920 ng/L and in invertebrates at levels up to 767 ng/g (Koch et al. 2019).

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 3 and 5 years old) sampled between 1984 and 2006 estimated that longer-chain PFASs in polar bears would 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).

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 National Health and Nutrition Examination Survey (NHANES) data concluded that diet is the dominant and most important source of PFAA exposure for children aged 3 to 11, 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 (Dalahmeh et al. 2018). Animal feed can also become contaminated when grown in soil treated with PFAS-containing irrigation water or biosolids (Gobelius 2016; Lee et al. 2014; Yoo et al. 2011).

The use of PFAS-contaminated irrigation water has been linked to elevated PFAA concentrations in fruits and vegetables (Blaine et al. 2014b; Zhang et al. 2016; Scher et al. 2018; Zhang et al. 2020). The application of PFAS-contaminated biosolids to land leads to the bioaccumulation of PFAAs in grasses (Yoo et al. 2011) and food crops (Blaine et al. 2014a; Navarro et al. 2017).

The PFASs currently used in U.S. treatments for converted textiles or leathers degrade into shorter-chain PFAAs that show greater bioaccumulation in vegetable crops than the longer-chain PFAAs from older treatment products (Blaine et al. 2013). Composted biosolid samples from the U.S. and Canada had three times higher levels of PFHxA in 2018 compared to 2016, while PFOA and PFOS remained nearly the same (Kim Lazcano et al. 2019). The study authors interpreted this finding as a reflection of the recent market shift to shorter-chain compounds.

Studies of PFASs in foods (see examples in Table 4) found them in wide-ranging concentrations (Domingo and Nadal 2017; EFSA 2012). Most of the available studies are from Europe and Asia, but levels are expected to be similar in the U.S., including in 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 (9 percent). Fifty-three samples collected in various developing countries were analyzed with a novel test method, with limits of detection as low as 3.1 pg/g for PFAAs. PFOS and PFOA were detected in all these samples, and PFHxS in 80 percent of samples (Sadia et al. 2019). 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). PFAAs, including longer and shorter chains, were found to accumulate in the roots and shoots of wheat (Zhao et al. 2018; Zhang et al. 2019b).

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). 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 study in Germany estimated that people who consume 150-300 g of beaked redfish or cod fillet per week are exposed to up to 322 and 198 ng of PFAAs each week, respectively (Kowalczyk et al. 2019). A study of pregnant women and children from six European countries concluded that fish consumption during certain stages of pregnancy is associated with higher concentrations of PFASs in

serum (Papadopoulou et al. 2019). Nevertheless, terrestrial animals can also have high PFAS concentrations (EFSA 2012; Haug et al. 2010). For instance, several PFAAs were found in the liver and muscle of beef cattle in China (Wang et al. 2017a).

The PFASs currently used in U.S. treatments for converted textiles or leathers degrade into shorter-chain PFAAs that show greater bioaccumulation in vegetable crops than the longer-chain PFAAs from older treatment products.

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). In 2018, however, EFSA proposed to lower its TDI from 150 ng/kg body weight per day to 13 ng/kg body weight per week for PFOS, and from 1,500 ng/kg body weight per day to 6 ng/kg body weight per week for PFOA (Knutsen et al. 2018). According to EFSA, "exposure of a considerable proportion of the population" exceeds the proposed limits for both compounds (Knutsen et al. 2018). In 2020, EFSA set a new limit of 4.4 ng/kg body weight per week for PFOA, PFOS, PFNA, and PFHxS, combined (EFSA 2020).

Dietary exposure studies in the U.S. 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 4: 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)
Beef	PFOS	0.0832 µg/kg	Sadia et al. (2019)
Crustaceans	PFOA	<LOD – 8.0 µg/kg	EFSA (2012)

Sample Type	PFAS Type	Concentration	Reference
Fish	PFOA	<LOD – 0.30 µg/kg	Schecter et al. (2010)
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)
Fish	PFOS	0.3 – 62 µg/kg	Kowalczyk et al. (2019)
Fish	PFNA	<LOD – 18 µg/kg	Kowalczyk et al. (2019)
Fish	PFOS	0.087 µg/kg	Sadia et al. (2019)
Chicken	PFOS	0.0257 µg/kg	Sadia et al. (2019)
Milk	PFOS	0.0899 µg/kg	Sadia et al. (2019)
Eggs	Sum of 11 PFASs	27 – 160 µg/kg	Wang et al. (2008)
Eggs	PFOS	0.212 µg/kg	Sadia et al. (2019)
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) 2 km from industrial site	Sum of 12 PFAAs	8.99 – 482 µg/kg	Su et al. (2017)
Butter	PFOA	1.07 µg/kg	Schecter et al. (2010)
Butter	PFOS	0.114 µg/kg	Sadia et al. (2019)
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 nanogram per liter (ng/L) range (Domingo and Nadal 2019). Drinking water can become contaminated with PFAAs and their precursors from treatments for converted textiles or leathers via several routes, including direct discharge of industrial effluent into waterways; leaching of land-applied wastewater or biosolids; leachate draining

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

from landfills that contain discarded products; 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 U.S., 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 treatments for converted textiles or leathers via several routes, including direct discharge of industrial effluent into waterways; leaching of land-applied wastewater or biosolids; leachate draining from landfills that contain discarded products; and discharge of WWTP effluent into streams and rivers.

As part of the third Unregulated Contaminant Monitoring Rule (UCMR 3) program, U.S. EPA (2017a) monitored public water supplies for six PFAAs from 2013 until 2015, reporting their presence in approximately 4 percent of samples tested (Hu et al. 2016). PFOA and PFOS concentrations equaled or exceeded the lifetime total health advisory of 70 ng/L, combined, in water serving approximately 6 million people, while 16.5 million people were served water above the UCMR 3 Minimum Reporting Levels (MRLs), with most detects (28 out of 452 samples, or 6.2 percent detection rate) in California (Hu et al. 2016; Hurley et al. 2016). The most significant predictors of observed PFAS detection frequencies and concentrations were: proximity to industrial sites that manufacture or use PFASs; proximity to military fire training areas; and the number of nearby WWTPs (Hu et al. 2016).

The MRLs for the six PFASs measured by the UCMR 3 program ranged from 10 to 90 ng/L, whereas the method used has a limit of quantification in the 2 to 5 ng/L range. Eurofins Eaton Analytical (2017), which was responsible for analyzing approximately 40 percent of the UCMR 3 data, found PFASs in more than a tenth of the samples it analyzed. However, fewer than 2 percent of those samples showed values above the UCMR 3 MRL and were reported to U.S. EPA. This suggests that the UCMR 3 program may have significantly underestimated the prevalence of PFASs in U.S. drinking water. A U.S. EPA-U.S. Geological Survey joint study (Boone et al. 2019) retested 25 of the drinking water treatment plants sampled under UCMR 3. The authors found PFBS, PFHxA, and PFOA in 100 percent of the treated water samples, and PFPeA, PFHpA, PFNA, and PFOS in over 90 percent of those samples. The shorter-chain PFAAs showed no significant decline between the source and the treated water (Boone et al. 2019). A recent U.S. EPA literature review found that, in addition to the six PFASs measured during UCMR 3, 23 other PFASs in drinking water were reported to date (Crone et al. 2019).

Drinking water contamination can linger for decades. In August 2017, the town of Belmont, Michigan, measured PFASs in some of its drinking water at levels 540 times above the U.S. 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 5: Monitoring studies found varying levels of PFASs in drinking water.

Sample Type	PFAS Type: Concentration	Reference
U.S. drinking water[13]	PFOA: <MRL – 349 ng/L PFOS: <MRL – 1,800 ng/L PFNA: <MRL – 56 ng/L	Hu et al. (2016)
U.S. drinking water	PFBS: <0.032 – 11.9 ng/L PFHxS: ND – 21.1 ng/L PFOS: ND – 36.9 ng/L PFBA: ND – 104 ng/L PFPeA: ND – 514 ng/L PFHxA: 0.09 – 60.8 ng/L PFHpA: ND – 177 ng/L PFOA: <0.56 – 104 ng/L PFNA: ND – 38.6 ng/L PFDA: ND – 24.7 ng/L	Boone et al. (2019)
European drinking water	PFBS: <LOD – 0.24 µg/kg PFHxS: <LOD – 0.011 µg/kg PFOS: <LOD – 0.016 µg/kg	EFSA (2012)

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

Numerous drinking water sources in California are contaminated with PFAAs, according to the UCMR 3 study. Forty percent of the positive UCMR 3 study detections in California are above the U.S. EPA advisory level of 70 ng/L for PFOA and PFOS combined (Hurley et al. 2016). In February 2020, the California State Water Resources Control Board reported data from more than 600 water system sites adjacent to airports with fire training areas or near municipal solid waste landfills (California State Water Resources Control Board 2020a). Sampling was conducted in two rounds, the first between April and June 2019 (Q1) and the second between July and September 2019 (Q2). In the Q1 data, 180 of the water system sites sampled exceeded the state's notification level of 6.5 ppt for PFOS, while 149 samples exceeded the notification level of 5.1 ppt for PFOA. In the Q2 samples, the number of sites

[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.

exceeding notification levels increased to 213 for PFOS and 185 for PFOA. Two shorter-chain PFAAs – PFHxA and PFBS – were detected in 186 and 199 of the Q1 samples, respectively. In the Q2 samples, detections increased: PFHxA was measured in 279 samples and PFBS in 281 samples. In both sampling periods, most samples with positive detections contained more than one PFAS.

California sea otters were found to have PFOA and PFOS levels ranging from less than 5 to 147 ng/g and from less than 1 to 884 ng/g, respectively, and these concentrations were 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). Harbor seal serum sampled in 2014 still contained PFOS at concentrations ranging from 12.6 to 796 ng/g, more than a decade after the PFOS phaseout (Sedlak et al. 2017).

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 at up to 221.0 ng/L (SFEI 2010). A 2016 study of PFAAs and precursors in wastewater effluent discharged to San Francisco Bay highlighted the local impact of aqueous film-forming foam (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). Monitoring by the San Francisco Estuary Institute in the Bay Area has continued to detect PFASs in bay water, stormwater, wastewater, bird eggs, and sport fish (SFEI 2020b). Due to this widespread contamination and high persistence of all PFAS class members, the San Francisco Bay Regional Monitoring Program has designated all PFASs as "moderate concern" for the San Francisco Bay, meaning they "have a high probability of at least a low level impact on Bay aquatic life" (SFEI 2020a; SFEI 2020b).

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.

Human biomonitoring data

Nearly all humans show evidence of exposure to some PFASs (Calafat et al. 2007), which can accumulate in human lungs, kidneys, liver, brain, and bone tissue (Pérez et al. 2013). A recent study found PFOS, PFOA, PFNA, PFDA, PFUnA, and PFHxS in human embryonic and fetal organs, with the highest burden in lung tissue in the first trimester samples, and in the liver in the second and third trimester samples (Mamsen et al. 2019). Biomonitoring studies have been limited to certain PFASs with

[14] AFFF may be the largest direct point source of PFASs (European Commission 2017). Generally, AFFF contains high levels of PFSAs and their precursors, but only low levels of PFCAs (Hu et al. 2016).

available analytical methods (see Table 6 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 NHANES, has detected PFASs in the blood of all people tested (Calafat et al. 2007). In NHANES blood samples collected in 2011-2012, average PFHxS, PFOA, and PFOS levels ranged between 1.3 and 6.3 µg/L each (ATSDR 2018c). In a Norwegian blood donor study, PFOS and PFOA were the most commonly detected PFASs, with average concentrations of 5.71 mg/mL and 1.41 mg/mL, respectively, and higher concentrations in older donors (Averina et al. 2020). PFBA and PFHxA were found in 13.3 percent and 22.6 percent, respectively, of 2682 urine samples collected from 2013-2014 NHANES participants (Calafat et al. 2019).

A biomonitoring study of over 65,000 people enrolled in the C8 Health Project, which evaluated exposures to PFASs in drinking water contaminated by DuPont, found PFHxA in the serum of more than 50 percent of the people tested (Frisbee et al. 2009). Shorter-chain PFAAs such as PFHxA, PFPeA, and PFBA often fall below the level of detection in human serum and plasma, but can be found at concentrations higher than most longer-chain PFASs in human whole blood samples (Poothong et al. 2017). They have also been detected in human hair samples at similar or higher levels compared to longer-chain PFAAs (Alves et al. 2015; Ruan et al. 2019; Martín et al. 2019). Shorter-chain PFAAs are also detected in human urine at levels comparable to or higher than PFOA (Hartmann et al. 2017; Kim et al. 2014a; Pérez et al. 2012), and may accumulate in certain organs at levels exceeding those of longer-chain PFAAs. In human autopsy tissues, out of 21 PFAAs studied, PFBA was the dominant compound in kidney and lung (median of 263 and 807 ng/g, respectively) and PFHxA in liver and brain (median of 68.3 and 141 ng/g, respectively), whereas PFOA dominated in bone (median of 20.9 ng/g) (Pérez et al. 2013).

Children may be more impacted than adults by exposure to PFASs from treatments for use on converted textiles or leathers, due to their frequent hand-to-mouth behavior, more direct contact with the treated products over a larger body surface area, and other physiological differences.

The California Biomonitoring Program, administered jointly by the California Department of Public Health, DTSC, and OEHHA, has measured PFAA levels in several subpopulations (Biomonitoring California 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 schoolteachers and employees have participated in the study. As of 2015, the California Biomonitoring Program has found PFHxS, PFOA, and PFOS in more than 99 percent of 1,759 blood samples provided by study participants (Biomonitoring California 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 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 10 to 20 percent per year – except for PFHxS, which did not change 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 the Scotchgard brand stain and soil repellent, doubled its blood concentrations every 6.3 years, concurrent with the phaseout of PFOS (Glynn et al. 2012). A U.S. national biomonitoring study of midlife women showed that serum concentrations of legacy PFASs, such as PFOS and PFOA, decreased significantly from 1999 to 2011, while concentrations of emerging compounds such as PFNA, PFDA, and PFUnA significantly increased over the same period (Ding et al. 2020).

A study of more than 600 children aged 6 to 10 from Eastern Massachusetts found higher blood concentrations of certain PFAAs and PFAA precursors in those children who had a carpet or rug in their bedroom (Harris et al. 2017). Children may be more impacted than adults by exposure to PFASs from treatments for use on converted textiles or leathers, due to their frequent hand-to-mouth behavior, more direct contact with the treated products over a larger body surface area, and other physiological differences (Moya et al. 2004).

Table 6: Human biomonitoring studies found widespread exposure to PFASs.

Population	Sample Type	PFAS Type: Concentration	Reference
Women aged 40 to 94	Serum	PFHpA: 0.084 ng/mL (mean); 1.16 ng/mL (max) 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)	Hurley et al. (2018)
337 California adults	Serum	Me-PFOSA-AcOH: 0.12 ng/mL (geometric mean) PFDA: 0.19 ng/mL (geometric mean) PFHxS: 1.03 ng/mL (geometric mean) PFNA: 0.79 ng/mL (geometric mean) PFOA: 1.49 ng/mL (geometric mean) PFOS: 5.21 ng/mL (geometric mean)	Biomonitoring California (2018)
Children aged 3 to 11	Serum	PFHxS: 0.84 ng/mL (geometric mean) PFNA: 0.79 ng/mL (geometric mean) PFOA: 1.92 ng/mL (geometric mean) PFOS: 3.88 ng/mL (geometric mean) MeFOSAA: 0.110 ng/mL (50^{th} percentile) PFDA: 0.100 ng/mL (50^{th} percentile, girls ages 3-5 years; boys <LOD)	CDC (2018)
Adults	Blood (near contam-inated site)	PFOA: 3.09 ng/mL (mean); 32 ng/mL (max) PFOS: 8.59 ng/mL (mean); 95.6 ng/mL (max) PFHxS: 4.12 ng/mL (mean); 116 ng/mL (max)	NHDHHS (2016)
Adult men	Serum	PFOA: 4.50 ng/mL (mean) PFOS: 20.80 ng/mL (mean) PFHxS: 2.88 ng/mL (mean) PFNA: 1.52 ng/mL (mean)	He et al. (2018)
Adult women	Serum	PFOA: 3.46 ng/mL (mean) PFOS: 14.51 ng/mL (mean) PFHxS: 1.94 ng/mL (mean) PFNA: 1.30 ng/mL (mean)	He et al. (2018)

Population	Sample Type	PFAS Type: Concentration	Reference
Pregnant women	Plasma	PFHxS: 0.37 ng/mL (mean) PFHxA: 0.16 ng/mL (mean) PFHpA: 0.18 ng/mL (mean) PFOS: 5.74 ng/mL (mean) PFOA: 2.64 ng/mL (mean) PFNA: 1.35 ng/mL (mean) PFDA: 0.57 ng/mL (mean) PFUnDA: 1.50 ng/mL (mean) PFDoA: 0.21 ng/mL (mean) PFTrDA: 0.35 ng/mL (mean) PFTeDA: 0.12 ng/mL (mean)	Tsai et al. (2018)
Pregnant women	Cord serum	PFOA: 1.59 ng/mL (mean) PFNA: 0.13 ng/L (mean) PFOS: 1.43 ng/L (mean) Sum of 11 PFASs: 3.67 ng/L mean	Cao et al. (2018)
Newborn	Umbilical cord plasma	PFOA: 7.65 ng/L (mean); 29.97 ng/L (max) PFOS: 2.93 ng/L (mean); 65.61 ng/L (max) PFNA: 0.69 ng/L (mean); 3.29 ng/L (max) PFDA: 0.44 ng/L (mean); 5.73 ng/L (max) PFUnA: 0.45 ng/L (mean); 5.27 ng/L (max) PFDoA: 0.10 ng/L (mean); 1.14 ng/L (max) PFHxS: 0.18 ng/L (mean); 0.85 ng/L (max) PFBS: 0.05 ng/L (mean); 0.46 ng/L (max)	Liu et al. (2018a)
Breastfeeding women	Breast milk	PFPeA: 0.053 ng/mL (geometric mean) 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)	Kang et al. (2016)

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

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

PFAAs and nonpolymeric PFAA precursors undergo transplacental transfer in humans (Midasch et al. 2007; Zhang et al. 2013; Yang et al. 2016; Zhao et al. 2017; Eryasa et al. 2019; Cai et al. 2020). Fluorinated ethers, such as Cl-PFESA, can also transfer from mother to fetus via the placenta (Chen et

al. 2017a; Pan et al. 2017; Cai et al. 2020). The placenta-to-maternal serum ratios of PFOS, PFOA, and PFNA were observed to increase during gestation – more so in pregnancies with male fetuses compared to female ones – suggesting bioaccumulation in the placenta and increasing exposure with fetal age (Mamsen et al. 2019). Through transplacental transfer, the developing fetus is exposed to mixtures of PFAAs *in utero* (Bach et al. 2015; Mamsen et al. 2017). Up to 70.1 percent of these transplacental mixtures can come from legacy PFASs (Li et al. 2020c). Gestational diabetes appears to significantly increase the rate of PFAS transplacental transfer (Eryasa et al. 2019).

PFAAs, including the shorter chains, are found in breast milk (Nyberg et al. 2018; Jin et al. 2020), potentially adding to significant cumulative early-life exposures. A 2016 study reported detectable concentrations of PFHxA in over 70 percent of breast milk samples (n=264), and detectable PFOA levels in the breast milk of nearly 99 percent of lactating mothers, associated with exposure to PFASs in consumer products (Kang et al. 2016). A study of lactating mothers in Korea found that PFAS concentrations in breast milk increased significantly after the first month of breastfeeding, likely due to a change in diet (Lee et al. 2018). A similar study in Ireland detected four PFASs in breast milk (Abdallah et al. 2020). Breastfed newborns can have higher body burdens of PFAAs compared to those fed only infant formula (Fromme et al. 2010). Kim et al. (2014b) analyzed concentrations of PFASs in the serum and urine of children and found a correlation between longer duration of breastfeeding and serum concentrations.

Transplacental 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 2 to 3 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 2 years old (Winkens et al. 2017a).

5. POTENTIAL FOR SIGNIFICANT OR WIDESPREAD ADVERSE IMPACTS

Reference: California Code of Regulations, title 22, section 69503.2(a).

This section integrates the information provided in the profile to demonstrate how the key prioritization principles, as identified in the SCP regulations, are met. The SCP regulations are not based on a weight of evidence approach. A single piece of reliable evidence can be sufficient to demonstrate potential for significant or widespread adverse impacts.

PFASs associated with treatments for converted textiles or leathers are released into air, water, and soil during chemical and product manufacturing, product use, and disposal. 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. 2004; Muir et al. 2019).

Exposing humans and other organisms to PFASs associated with the treatments for converted textiles or leathers has the potential to contribute to or cause significant and widespread adverse impacts, based on multiple factors considered by the Safer Consumer Products regulations.

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, resulting in a growing public health concern (APHA 2016). Conventional, aeration, oxidation, and biological treatment are ineffective for removing PFASs from drinking water (Crone et al. 2019). Nanofiltration was the only treatment that efficiently removed PFASs from contaminated drinking water in a recent study (Boiteux et al. 2017). Anion exchange resins and high-pressure membranes such as nanofiltration and reverse osmosis can remove over 90 percent of PFAAs from drinking water, including shorter-chain ones. However, these methods are expensive, perform poorly at removing neutral PFAA precursors, and generate large waste streams with concentrated PFAAs that need to be disposed of (Crone et al. 2019).

Exposing humans and other organisms to PFASs associated with the treatments for converted textiles or leathers has the potential to contribute to or cause significant and widespread adverse impacts, based on 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 treatments for converted textiles or leathers, and (3) the potential for these exposures to contribute to or cause

significant or widespread adverse impacts, DTSC proposes to list treatments containing PFASs for use on converted textiles or leathers as a Priority Product.

Adverse impacts linked to the Candidate Chemical's hazard traits

Reference: California Code of Regulations, title 22, section 69503.3(a).

The SCP regulations direct the Department to evaluate the potential for the Candidate Chemical to contribute to or cause adverse impacts by considering several adverse impact factors for which information is reasonably available.

As discussed in Chapter 3, the PFASs associated with treatments for converted textiles or leathers show several exposure potential hazard traits, including persistence, mobility, bioaccumulation, and lactational and transplacental transfer. These exposure potential hazard traits stem from the PFASs' unique physicochemical properties and environmental fate (see Chapter 3). PFAAs are extremely persistent, with no known natural degradation pathways 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 2010; Krafft and Riess 2015a; Wang et al. 2014a; Washington et al. 2015). The extreme environmental persistence of PFAAs, regardless of chain length, leads to their continuous presence in the environment, hence to continuous chronic background exposures for humans and biota (Brendel et al. 2018). 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). Also, removal of chemicals of concern is only feasible for hotspots and cannot address background environmental contamination (Cousins et al. 2019).

These extremely persistent chemicals are now global pollutants that can be found in all environmental compartments. 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, resulting in a growing public health concern (APHA 2016). In particular, shorter-chain PFASs are highly mobile in soil and water, accumulate in edible plant parts, and are difficult and expensive to remove during water treatment (Brendel et al. 2018; Boone et al. 2019; Ateia et al. 2019a; Crone et al. 2019). Conventional, aeration, oxidation, and biological treatment are ineffective for removing PFASs from drinking water (Crone et al. 2019). Nanofiltration was the only treatment that efficiently removed PFASs from contaminated drinking water in a recent study (Boiteux et al. 2017). Anion exchange resins and high-pressure membranes such as nanofiltration and reverse osmosis can remove over 90 percent of PFAAs from drinking water, including shorter-chain ones. However, these methods are expensive, perform

poorly at removing neutral PFAA precursors, and generate large waste streams with concentrated PFAAs that need to be disposed of (Crone et al. 2019).

Furthermore, as discussed in Chapter 3 and detailed in Appendix C, shorter-chain PFAAs, which are the final degradation products of the PFAS formulations used in domestic treatments for converted textiles or leathers, display several toxicological hazard traits, including developmental toxicity, endocrine toxicity, hematotoxicity, immunotoxicity, neurodevelopmental toxicity, reproductive toxicity, and ocular toxicity. Longer-chain PFAAs, which may be found in imported products, display the following additional toxicological hazard traits: carcinogenicity, cardiovascular toxicity, hepatotoxicity and digestive system toxicity, nephrotoxicity and other urinary system toxicity, and respiratory toxicity. Inhalation exposure to PFASs from the use of treatments for converted textiles and leathers is of particular concern for several reasons, including the propensity of the side-chain fluorinated polymers to metabolically degrade into PFAAs inside the body (Chu and Letcher 2014); the tendency of PFASs, particularly shorter-chain PFAAs, to accumulate in lung tissue (Pérez et al. 2013); and the potential for PFASs to modify lung surfactant function (Sørli et al. 2020). Several cases of injury and illness in workers and consumers acutely exposed to waterproof sprays, aerosols, and other treatment products are documented in the literature. Symptoms include cough, fever, tachypnea, alveolar hemorrhage, desquamative interstitial pneumonia, acute respiratory distress syndrome, acute pneumonitis, lung edema, and general acute pulmonary injuries (Vernez et al. 2006; Epping et al. 2011; Nakazawa et al. 2014; Kondo et al. 2014; Kikuchi et al. 2015; Walters et al. 2017; Harada et al. 2017; Scheepers et al. 2017; Sawamoto et al. 2018).

The environmental hazard traits of PFASs include phytotoxicity and impairment of wildlife development, reproduction, or survival, however data are limited to a very small number of PFASs and organisms (ITRC 2020b). Discharges of PFAS-contaminated WWTP effluents into streams and rivers have led to contamination of water and sediments at concentrations considered potentially hazardous to wildlife and threatening to biodiversity (Rodriguez-Jorquera et al. 2016). Studies have also suggested that PFAAs, including PFHxA, may contribute to mixture toxicity and enhance the adverse impacts associated with other hazardous compounds (see the section on Cumulative Effects below). The ubiquity 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). Discharges of PFAS-contaminated WWTP effluents into streams and rivers have led to contamination of water and sediments at concentrations considered potentially hazardous to wildlife and threatening to biodiversity (Rodriguez-Jorquera et al. 2016).

Biosolids, especially from WWTPs that treat leachate from landfills and composting facilities, can be heavily contaminated with PFAAs and precursors that adsorb to organic matter (Arvaniti et al. 2012; Gallen et al. 2018; Kim Lazcano et al. 2019; Sun et al. 2011). Side-chain fluorinated polymers were detected in all biosolid-treated soil samples in a recent Canadian study (Chu and Letcher 2017b),

suggesting that when biosolids are applied for land use, the contained PFASs can partition into the soil. Shorter-chain PFAAs 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). They can also leach into adjacent waterways (Sepulvado et al. 2011), 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. 2014a; Lee et al. 2014).

Some studies indicate the potential for shorter-chain PFASs to modulate biological responses, which we recognize is not a robust indicator of toxicological hazard. The relationship between exposure and toxicological outcomes, particularly for PFASs other than PFOA and PFOS, is a major data gap (DeWitt 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. 2017a). Nevertheless, increasing reports of toxicity in humans and experimental animals, sometimes at or below common exposure levels, is leading to growing public health concern (APHA 2016) and expanded regulatory interest (Corsini et al. 2014; Zushi et al. 2012).

Cumulative effects

Reference: California Code of Regulations, title 22, section 69503.3(a)(1)(C).

Cumulative effects occur from exposures to the Candidate Chemical and other chemicals with similar hazard traits or endpoints.

Individual PFASs rarely occur in isolation. Instead, people and biota are exposed to mixtures of PFASs and other chemicals, potentially leading to cumulative adverse impacts, yet toxicological endpoints are typically assessed using single PFASs. The adverse human and environmental health effects resulting from exposures to PFAS mixtures have not been well-characterized, and toxicological reference values for PFAS mixtures are lacking. Some have argued that exposure to PFAS mixtures may exert greater toxicity than exposure to a single PFAS (Wang et al. 2011). Although exposure to multiple 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 17 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). One effort to estimate cumulative toxicity is presented in the work of RIVM (2018), who used a Relative Potency Factor (RPF) method to compare exposure to a PFAS mixture to exposure to a comparable amount of PFOA. This method is currently limited by the availability of toxicological data for the many different PFASs, as well as by several assumptions about the toxicity mechanism and mode of action.

Its calculations rely heavily on subacute and subchronic oral liver toxicity studies conducted in rodents. So far, RPFs have been calculated for only 20 individual PFASs.

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 PPAR-alpha (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). The cytotoxicity of individual PFASs and of binary mixtures using an amphibian fibroblast cell line showed mixtures were approximately additive, except with PFOS and PFOA, which were weakly synergistic (Hoover et al. 2019).

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

- Effects on gene expression patterns in zebrafish embryos co-exposed to a mixture of PFHxA, PFOS, and PCB126; lowered oxidative stress response from exposure to PCB126 and PFHxA, and to PCB126, PFOS, and PFHxA (but not from the individual compounds, nor from PCB126 and PFOS), suggesting PFHxA plays a synergistic role, although the exact mechanism remains unknown (Blanc et al. 2017);
- Endocrine disruption in developing rats co-exposed to a mixture of PFHxS and 12 endocrine-disrupting chemicals (including bisphenol A, dibutyl phthalate, di-2-ethylhexyl phthalate, and butyl paraben), at doses relevant for human exposure, including doses at which PFHxS alone and the mixture of endocrine disruptors alone showed no or only weak effects (Ramhøj et al. 2018);
- Clinically relevant perturbations of normal cell function of benign breast epithelial cells co-exposed to a mixture of PFOA, bisphenol A, and methylparaben at concentrations relevant to human exposure; these effects were not observed when cells from the same lines were exposed to the chemicals individually (Dairkee et al. 2018);
- 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);
- 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).

While the above data suggest a potential for cumulative harm from exposure to mixtures of PFASs or of PFASs and other chemicals, it is unclear whether the outcomes from the cell lines, zebrafish, or juvenile salmon are associated with adverse phenotypes. In spite of the limitations of these approaches, as described by the National Academy of Sciences (NAS 2017), these studies suggest that exposure to certain combinations of PFASs, or co-exposure to certain PFASs and other chemicals, has the potential to contribute to disease by perturbing various pathways or activating some mechanisms. In particular, PFAAs may have cumulative impacts with one another and with other hazardous chemicals, but co-exposures that dramatically affect adverse impacts are not known at this time and require more scientific research.

Adverse waste and end-of-life effects

Reference: California Code of Regulations, title 22, sections 69503.2(b)(1)(B) and 69501.1 (a)(8).

This section summarizes findings related to the waste materials and byproducts generated during the life cycle of the product and their associated adverse effects, as described in the SCP regulations. These considerations can form part of the basis for proposing the product-chemical combination.

Effects on solid waste and wastewater disposal, treatment, and recycling

Conventional wastewater treatment systems such as sedimentation, flocculation, coagulation, and biological degradation are ineffective for PFAS removal (Rahman et al. 2014; Ng et al. 2019). Adsorption and ion-exchange have also been recently explored as methods that could increase the speed and efficiency of PFAS removal (Ji et al. 2018; Ateia et al. 2019b). While adsorption and separation technologies combined with destructive technologies show promise for removing a wider range of PFASs, their large-scale application poses several challenges (Horst et al. 2018; Li et al. 2020a), including reliance on chemicals and energy, high cost, and generation of harmful byproducts (Lu et al. 2020). Shorter-chain PFAAs are especially challenging to remove from aquatic environments and to destroy (Li et al. 2020a).

Biosolid treatments are also ineffective at reducing the levels of PFAAs. A study of commercial biosolids from the U.S. and Canada found that thermal hydrolysis had no apparent effect on the PFAA concentration, whereas heat treatment and composting increased PFAA concentrations (especially PFHxA) via the degradation of precursors; only blending with PFAS-free material decreased the concentration of PFAAs in the commercial biosolids, by diluting it (Kim Lazcano et al. 2019). Regardless of the treatment used, the side-chain fluorinated polymer surfactants that are the main components of Scotchgard fabric protector products were found in high concentrations in all biosolid samples tested from WWTPs across Canada (Letcher et al. 2020). The researchers concluded that the main source of these side-chain fluorinated polymer surfactants was domestic wastewater.

Removal of PFASs from the waste stream is expensive. For instance, following contamination from carpet manufacturing in Dalton, Georgia 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). Wastewater effluent can contribute significantly to the PFAA contamination of U.S. drinking water sources (Guelfo and Adamson 2018).

Discharges or disposal to storm drains or sewers that adversely impact the 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). In fact, wastewater treatment typically transforms PFAA precursors into PFAAs, resulting in higher PFAA concentrations in WWTP effluent compared to the influent (Ahrens 2011; Frömel et al. 2016; Guerra et al. 2014).

Releases of the Candidate Chemical during product end-of-life

In California, textiles are one of the top five largest components of single-family municipal waste, by weight (CalRecycle 2020). In 2018, synthetic and organic textiles (clothing, towels, sheets, rope, etc.) contributed an estimated 1.08 million tons (representing 2.7 percent of the solid waste disposed in the state's landfills), with an additional 627,926 tons (1.6 percent) from carpets (CalRecycle 2020). From 2015 to 2016, due to the 2010 California Carpet Stewardship Law, postconsumer carpet collection and carpet tile recycling increased by 4 and 31 percent, respectively. Nevertheless, in 2016, an estimated 75 percent of the carpet discarded in California was disposed of in landfills (CCSP 2016). Leather products are grouped within a broad category called "composite organic" waste, and their proportion in the landfill waste stream cannot be easily quantified. In 2018, shoes, purses, and belts alone accounted for an estimated 0.3 percent (120,032 tons) of the state's overall solid waste disposal (CalRecycle 2020).

The use of incinerators has become popular as a method to reduce landfill volumes while increasing energy recovery (CCSP 2016). Incinerating textile or leather products (i.e., carpets, shoes, furniture, upholstery) treated with fluorinated polymers, including shorter-chain side-chain fluorinated polymers, can result in air emissions. Potential air toxicants include PFCAs, ozone depleting substances such as CFCs, and potent greenhouse gases such as fluorocarbons, since the mineralization (defluorination) of PFASs is incomplete even at high temperatures and in the presence of calcium hydroxide (Huber et al. 2009). This combustion occurs in incinerators often located in low-income communities, where emissions burden people already impacted by aggregate chemical exposures from other sources, creating environmental justice concerns (GAIA and Changing Markets 2017).

Empty containers of treatments for converted textiles or leathers may contain PFAS residue, which may enter the environment when the containers are discarded. Municipal landfill leachate is a known point source of PFAS emissions to the environment (Benskin et al. 2012; Busch et al. 2010; Eggen et al. 2010; Fuertes et al. 2017; Hamid et al. 2018; Huset et al. 2011; Lang et al. 2017; Boucher et al. 2019), with treated carpets, rugs, furnishings, and clothing as significant contributors (Gallen et al. 2017; Lang et al. 2016; Shoaeioskouei 2012; Mumtaz et al. 2019). 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 (Dauchy et al. 2017; Hamid and Li 2016; Pan et al. 2016). WWTPs are also meaningful sources of volatile PFASs, as are landfilled carpets and rugs (Ahrens et al. 2011a; Weinberg et al. 2011). A study of PFASs in digested sewage sludge from 45 WWTPs in Switzerland found that the 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).

Populations that may be adversely impacted

Reference: California Code of Regulations, title 22, sections 69503.3(a)(1)(F) and 69503.3(a)(2).

This section identifies specific populations of humans and environmental organisms that may be harmed if exposed to the Candidate Chemical in the product. Sensitive subpopulations, environmentally sensitive habitats, endangered and threatened species, and impaired environments in California have special consideration as they may be more vulnerable.

Exposures to PFASs occur frequently and from multiple sources over a lifetime, including from placental transfer, breastfeeding, drinking water, food, and contact with consumer products and other environmental media. PFASs have the potential to cause significant and widespread adverse impacts especially to:

- Sensitive subpopulations (which include fetuses, infants, children, pregnant women, some 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. Workers, including carpet and upholstery cleaners; workers in

upholstered furniture, furnishings, clothing, shoe, and carpet stores; auto dealership workers; and auto detailing technicians can experience high PFAS exposures on the job. Individuals with certain preexisting conditions (e.g., elevated cholesterol, high blood pressure, poor kidney function) may be especially sensitive to PFASs. Pregnant women's exposure to PFASs is of concern due to transplacental and lactational transfer to the offspring. Endangered and threatened species in California are vulnerable to contamination of waterways and food webs with PFASs that originate from stain- and soil-repellent treatments for converted textiles or leathers.

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.

Due to their widespread presence in the environment and biota, PFASs may contribute to or cause adverse impacts in all humans and other organisms. In their review paper, Krafft and Riess (2015b) state 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."

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. 2012; Hoke et al. 2012) and widespread bioaccumulation in aquatic animals. Less is known about potential impacts to terrestrial animals and plants, especially at the population level. Shorter-chain PFASs generally show increased uptake by leafy plants compared to the longer chains (Blaine et al. 2014a), though longer chains may preferentially accumulate in roots (Zhao et al. 2017). PFASs have been shown to bioaccumulate in earthworms, indicating the potential for biomagnification in terrestrial species (Karnjanapiboonwong et al. 2018; Munoz et al. 2020). Wildlife, particularly apex predators, may suffer adverse effects from chronic exposure to PFASs in food and water (Tartu et al. 2017; Tipton et al. 2017). Fish-eating birds, including eagles and ospreys, are especially vulnerable to PFAS exposure via food chain contamination (Giesy et al. 2010). Known impacts to birds from PFAS exposure include reduced embryo survival in chicken (Norden et al. 2016).

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). Pregnant women and fetuses are also sensitive subpopulations because of transplacental migration and the vulnerability of the rapidly developing fetus (Slotkin et al. 2008). Breastfed 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; Kärrman et al. 2007; Mogensen et al. 2015; Mondal et al. 2014; Abdallah et al. 2020). Infants, toddlers, and small children often have increased exposures due to hand-mouth behaviors that can lead to increased incidental ingestion of dust and soil with environmental contaminants, and higher doses relative to body weight compared to adults (U.S. EPA 2011). Infants who crawl over carpets and upholstery are at increased risk of ingesting or inhaling dust (Wu et al. 2018), which may contain PFASs. A study involving over 1,000 children up to 7 years old found a negative correlation between plasma concentrations of most PFAAs and age, indicating higher exposures in younger children (Zhang et al. 2018). Table 7 lists the main routes of PFAS exposure for children.

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 the PFASs released during the treatment of converted textiles or leathers. Because PFASs undergo transplacental migration (Midasch et al. 2007; Zhang et al. 2013; Yang et al. 2016; Chen et al. 2017a; Zhao et al. 2017; Eryasa et al. 2019; Cai et al. 2020), fetuses are continuously exposed from conception until birth. PFASs are also frequently detected in breast milk (Nyberg et al. 2018; Jin et al. 2020), 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 12 months of age. The amount of breast milk ingested by newborns averages 76 g, six to 18 times per day (CDC 2014).

Toddlers can experience increased exposures to PFASs due to behavioral characteristics. Children between the ages of 1 and 3 spend a lot of time in contact with the floor, where dust settles, and engage in indoor hand-mouth behaviors an average of 16 times per day (U.S. EPA 2011). Thus, incidental dust ingestion can be a significant route of exposure for young children (Mercier et al. 2011; Tian et al. 2016; Wu et al. 2020). While adults are estimated to ingest an average of 50 mg/day of soil and dust, children under 6 may ingest 100 mg/day (U.S. EPA 2011). PFAS doses via incidental 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). A study of U.S. child care environments estimated median daily intakes for toddlers via dust ingestion of up to 0.85 ng/kg body weight per day for the sum of 28 PFASs (Zheng et al. 2020). Similarly, a study at California child care centers estimated that the children's daily intake of PFAAs via dust ingestion ranges from 0.023 – 1.9 ng/kg body weight (Wu et al. 2020).

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 textile or leather treatments, leading to involuntary, chronic inhalation exposures in workers, which has not been well-characterized (NILU 2015; Schlummer et al. 2015). PFASs can also adsorb to office dust or be released from surface abrasion as tiny particles, which office workers may ingest (Fraser et al. 2013). Due to their long usage

life, commercial carpets could be a near-daily source of exposure to PFOA, PFOS, and other phased-out longer-chain PFASs for office workers.

Table 7: Prenatal and early-childhood PFAS exposure routes[15]

Exposure Pathway	Basis
Transplacental migration	Exposure to PFASs begins before birth. Transplacental passage is 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 foods (Blaine et al. 2014a; Pérez et al. 2014). PFASs are readily absorbed after ingestion (ATSDR 2018a; 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 2020b). Some states have adopted, or are considering adopting, lower limits.

[15] Protecting children, a sensitive subpopulation, is one of the goals identified in SCP's 2018-2020 Priority Product 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.

Exposure Pathway	Basis
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 incidental dust ingestion rates, increased floor contact and hand-to-mouth behavior (Mercier et al. 2011; Tian et al. 2016). PFASs are readily absorbed following inhalation and ingestion (ATSDR 2018a).

Workers in the textile or leather industry, including retail workers and recyclers, also comprise a sensitive subpopulation because of their potentially higher exposure, particularly to volatile and semi-volatile PFASs emitted from these products. The highest levels of volatile PFASs, such as FTOHs, have been found in stores selling outdoor clothing, carpets, or furniture (Langer et al. 2010; Schlummer et al. 2013). One study estimated that for people living or working in environments with high levels of FTOHs in indoor air, such carpet stores, exposure to PFOA resulting from FTOH degradation is in the same order of magnitude as dietary PFOA exposure (Schlummer et al. 2013).

Studies demonstrate substantial human exposure in workers at fluorochemical manufacturing facilities (Gao et al. 2018) and communities relying on contaminated drinking water sources (Heydebreck et al. 2016). People living near PFAS chemical manufacturers, or in other areas contaminated by PFASs, have higher-than-average levels of PFASs (Fromme et al. 2009). Individuals with biomarkers of susceptibility for certain health conditions may be unusually sensitive to PFAS exposure (ATSDR 2018a).

Residents of low-income communities may be disproportionately impacted by chronic exposures to multiple hazardous industrial chemicals, increasing the likelihood of cumulative adverse health effects (U.S. EPA 2016a). Manufacturing facilities, including those that release PFASs, are often located in these communities. 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 Inuit in Nunavut, Canada, the Nuuk Inuit in Greenland, and the Faroese includes foods heavily contaminated with PFASs that originated from thousands of miles away (Grandjean et al. 2012; Grandjean et al. 2017a; Long et al. 2012; Ostertag et al. 2009).

California's endangered and threatened species could be adversely affected by exposure to PFASs associated with treatments for converted textiles or leathers, 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, leading to contaminated water and sediments. 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.

6. OTHER REGULATORY PROGRAMS

Reference: California Code of Regulations, title 22, section 69503.2(b)(2).

DTSC has assessed all applicable state and federal laws and regulations and international treaties or agreements with the force of domestic law related to the product or the Candidate Chemical in the product. DTSC has determined that these programs do not overlap or conflict with this proposal to list treatments containing members of the class of PFASs for use on converted textiles or leathers, nor with any subsequent regulation that may result from such listing.

No California state laws or regulations currently address the use of PFASs in treatments for converted textiles or leathers. Also, there are no applicable international treaties or agreements with the force of domestic law.

At the federal level, Section 5 of the Toxic Substances Control Act (TSCA) authorizes U.S. EPA to issue Significant New Use Rules (SNURs) for new chemicals 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 to 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 U.S. (U.S. EPA 2007);
- October 22, 2013 – SNUR requiring companies to report their intent to manufacture certain PFOA-related chemicals to treat carpets, as well as their intent to import carpets containing these PFASs (U.S. EPA 2013);
- (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 2015);
- (Proposed) February 20, 2020 – supplemental to a SNUR issued on certain PFASs in 2015; amendment includes regulation on imported products that contain certain PFASs used as part of surface coatings (U.S. EPA 2020f); and
- June 22, 2020 – final SNUR requiring notice before anyone can (1) resume the manufacturing or processing of specific long-chain PFASs that have been phased out in the U.S. or (2) import

products containing certain long-chain PFASs as surface coatings or import carpets containing perfluoroalkyl sulfonates (U.S. EPA 2020c).

Since 2000, U.S. EPA has reviewed 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 worked with individual submitters pursuant to TSCA section 5(e) to develop Consent Orders, which typically contain certain requirements such as testing, while allowing production and use.

Section 5 of TSCA and pursuant consent orders do not provide the same level of public health and environmental protection that would be provided by designating a Priority Product. TSCA is based on a risk assessment approach. SCP, however, considers the potential human and ecological exposures and the potential for significant or widespread adverse impacts, which provides a greater level of protection. TSCA's focus is solely on prohibiting or limiting the manufacture, processing, distribution in commerce, use, or disposal of a substance. It does not address the creation of safer products.

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).

The National Defense Authorization Act for Fiscal Year 2020 (NDAA), signed into law on December 20, 2019, contains multiple PFAS-related amendments, including a ban on the use of PFASs in food packaging for military ready-to-eat meals and the phaseout of PFAS-containing firefighting foam (Inhofe 2019). Additionally, section 7321 of the NDAA added several PFASs to the Toxics Release Inventory (TRI) under section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA), effective January 1, 2020. The TRI allows the public to become more informed about the environmental and safety concerns that may occur from handling and storage of toxic chemicals. The NDAA specifically added 14 PFASs to the TRI, plus any additional PFASs that are subject to a SNUR in the Code of Federal Regulations (CFR) on or before December 20, 2019, under either 40 CFR 721.9582 or 721.10536, and are identified as active in commerce on the TSCA inventory list published in February 2019. The TRI list currently contains 172 PFASs. Reporting forms are due by July 1, 2021 (U.S. EPA 2020e). Other PFASs will be automatically added to the TRI if any of the following occur (Inhofe 2019):

- EPA finalizes a toxicity value for a perfluoroalkyl or polyfluoroalkyl substance or class of PFASs;
- EPA finalizes a SNUR for a perfluoroalkyl or polyfluoroalkyl substance or class of PFASs;
- EPA adds a perfluoroalkyl or polyfluoroalkyl substance or class of PFASs to an existing SNUR; or
- EPA designates a perfluoroalkyl or polyfluoroalkyl substance or class of PFASs as an active chemical substance on the TSCA Inventory.

7. POTENTIAL ALTERNATIVES

Reference: California Code of Regulations, title 22, section 69503.2(b)(3).

This section summarizes information available to DTSC regarding alternatives that may or may not be safer than the Candidate Chemical. DTSC does not need to ensure that these alternatives are safer and may summarize their associated hazards to illustrate readily available information. The sections below may include information such as how readily available an alternative is, product functions addressed by the alternative, and implications for manufacturers using the alternative (e.g., use limitations, product reformulation, different equipment needs).

Given the potential for exposures and significant or widespread adverse impacts, replacing currently used PFASs in treatments for converted textiles or leathers with other members of the PFAS class could constitute a regrettable substitution. Therefore, to encourage identification and evaluation of non-PFAS alternatives, this proposal applies to treatments for converted textiles or leathers containing any member of the class of PFASs.

Given the potential for exposures and significant or widespread adverse impacts, replacing currently used PFASs in treatments for converted textiles or leathers with other members of the PFAS class could constitute a regrettable substitution. Therefore, to encourage identification and evaluation of non-PFAS alternatives, this proposal applies to treatments for converted textiles or leathers containing any member of the class of PFASs.

PFAS-free chemical alternatives that prevent soils from adhering to and staining agents from penetrating the fiber surface are already available. Examples include TriPlex's Go Green Guard and Go Clean 2X Rinse; Vectra Enterprises, Inc.'s Vectra Spray; and Innovative Chemical Technologies, Inc.'s Flexipel HR-100. DTSC does not currently have complete information on the chemical content of these alternatives and has not evaluated their safety. The exact formulation of these products is largely unknown because manufacturers withhold the information as proprietary trade secrets.

Depending on the application, natural alternatives to PFASs in treatments for converted textiles and leathers include mink oil and kakishibu (fermented persimmon tannin) (Kawamoto 2020). Other potential alternatives to PFASs in treatments for converted textile or leather may include siloxane and silicone polymers, hydrocarbons (e.g., paraffin waxes), derivatives of fatty acids (e.g., long-chain fatty acid esters), polyurethanes, and dendrimers (hydrocarbon- or polyurethane-based) (Danish Environmental Protection Agency 2015b; KEMI 2015; Teli 2018; U.S. EPA 2012; Atav 2018). These materials function as nonfluorinated, biodegradable durable water repellents with performance

comparable to short-chain fluorinated polymers. They repel stains from high and intermediate surface tension liquids such as orange juice and red wine, but they provide no repellency against lower surface tension stains such as olive oil (Schellenberger et al. 2019a). Some nonfluorinated (meth)acrylates and urethanes have stain release properties for both oil- and water-based stains (Danish Environmental Protection Agency 2015b). These alternatives appear promising if they can be scaled for high throughput manufacturing.

A Danish Environmental Protection Agency review concluded that the main ingredients in paraffin-based repellents are mostly harmless, readily biodegradable, nonbioaccumulative, and nontoxic 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 (a 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.

Other potential PFAS alternatives have been patented but may not yet be commercially available. 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, soil 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). Another patent mentions a “superhydrophobic” agent containing nonfluorinated, water-based hydrophobic components that have useful fluid management properties, including stain repellency. It goes on to call this application a viable treatment product that incorporates titanium oxide nanoparticles for maintaining “a translucent-white superhydrophobic surface treatment that does not require subsequent pH-modification” (Qin et al. 2018).

8. ADDITIONAL CONSIDERATIONS

This section summarizes other relevant information not captured under the adverse impact and exposure factors named in section 69503.3 of the Safer Consumer Products regulations.

The Safer Consumer Products Program's focus on treatments for use on converted textiles or leathers is part of a larger effort by California and other U.S. states, the U.S. federal government, other nations, and international organizations to evaluate a wide range of products and determine whether their use of PFASs is necessary and, when it is not, to adopt safer alternatives. Some of these other efforts are briefly summarized below. While these other actions may not relate directly to treatment products, PFASs released from the products and activities discussed below contribute to aggregate exposures, as explained earlier in this document.

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]

U.S. EPA research and action plan

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 2020d). Through a Memorandum of Understanding 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, Clariant, Daikin, 3M/Dyneon, DuPont, and Solvay Solexis.

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 2017c). On March 13, 2018, U.S. EPA also announced a partnership with the National Toxicology Program to develop a tiered testing approach to characterize the toxicity and toxicokinetics of 75 PFASs that represent the entire class (U.S. EPA 2018). In February 2019, U.S. EPA revealed its PFAS Action Plan. In it, EPA commits, among other things, to: (1) a regulatory determination by the end of 2019 whether to set a maximum contaminant level (MCL) for PFOA and PFOS; (2) beginning regulatory work to list PFOA and PFOS as hazardous substances under the Comprehensive

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

Environmental Response, Compensation, and Liability Act (CERCLA); (3) publishing draft toxicity assessments for PFBA, PFHxA, PFHxS, PFNA, and PFDA in 2020; and (4) continuing to improve analytical methods, monitoring, and risk communication (U.S. EPA 2019). In September 2019, U.S. EPA announced it is awarding approximately $6 million to eight grantees to research PFASs in waste streams and identify practical approaches to manage their potential impacts (U.S. EPA 2019).

Restrictions on PFASs in products

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). In September 2019, Home Depot announced that it will stop selling carpets and rugs containing PFASs in the U.S. and Canada beginning in December 2019 (Home Depot 2019). Similarly, in October 2019, Lowe's announced that it will stop purchasing PFAS-containing carpets and rugs in the U.S. and Canada by January 2020 (Floor Daily 2019).

Related laws and regulations

On September 29, 2020, California Governor Newson signed SB 1044 (Stats. 2020, ch. 308). As of January 1, 2022, the new law requires labeling of all firefighter personal protective equipment that contains intentionally-added PFASs and bans the manufacture, sale, distribution, and use of class B firefighting foams containing intentionally-added PFASs, except where required by federal law. Certain facilities are exempt from the law until either 2024 or 2028 (Allen 2020).

In March 2018, the San Francisco Department of the Environment adopted regulations regarding environmentally preferable purchasing, which include a requirement that all carpet purchased for city projects contain no intentionally added PFASs (SFE 2018). Also in March 2018, two PFAS-related bills were signed into law in Washington state. Engrossed Substitute House Bill 2658 prohibits the sale of food packaging containing PFASs as of January 1, 2022, provided the state Department of Ecology identifies safer alternatives by January 1, 2020 (Washington State Legislature 2018a). Engrossed Substitute Senate Bill 6413 prohibits the use of PFAS-containing firefighting foam for training purposes

as of July 1, 2018, and, as of July 1, 2020, prohibits the sale of firefighting foams with intentionally added PFASs, with exemptions for the military, airports, oil refineries, and chemical plants (Washington State Legislature 2018b). In 2020, this law was amended by Engrossed Substitute House Bill 2265 to, among others, limit the exemption for federal facilities (including Department of Defense facilities and airports) to two years after federal regulations begin allowing the use of non-PFAS foams, and the exemptions for oil terminals, oil refineries, and chemical plants to until 2024 (Washington State Legislature 2020). In 2019, the state of Washington enacted Substitute Senate Bill 5135, which authorizes the state's departments of Ecology and Health to regulate classes of chemicals in consumer products (Washington State Legislature 2019). The law identifies PFASs as one of the five initial priority chemical classes. In September 2019, the state Department of Ecology announced that it will be focusing its initial research on PFASs in carpets and aftermarket carpet treatments, among other products (ECY 2019).

PFBS, PFHxS, PFNA, plus a GenX precursor and their salts have been added to the REACH Candidate List of substances of very high concern (SVHCs) for authorization (ECHA 2020a). In September 2019, Denmark announced a ban on PFASs in cardboard and paper used as food contact materials, which became effective in July 2020 (Danish Ministry of Environment and Food 2019; Keller and Heckman LLP 2020). On January 30, 2018, South Australia became the first state to ban PFAS-containing firefighting foams (Government of South Australia 2018).

The Stockholm Convention on POPs, which the U.S. 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 (UNEP 2020). Production and use of these chemicals are still allowed for acceptable purposes after Annex B listing, which included use in carpets until 2015. Following the recommendation of the POPs Review Committee (UNEP/POPS/POPRC 2017), the Conference of the Parties decided in the spring of 2019 to list PFOA, its salts, and PFOA-related compounds in Annex A to the Stockholm Convention, with some time-limited exemptions (COP 2019). The POPs Review Committee also agreed in October 2017 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 (POPRC 2020).

Relevant proposed laws and regulations

In November 2019, The House Committee on Energy and Commerce proposed a legislative package, the PFAS Action Act of 2019, which would, among other things, require EPA to conduct comprehensive

toxicity testing on all PFASs; designate PFOA, PFOS, and their salts as hazardous substances under CERCLA and as hazardous air pollutants under the Clean Air Act; determine whether any or all other PFASs should be designated as hazardous substances under CERCLA, or as hazardous air pollutants under the Clean Air Act, no later than five years after the date of enactment of the Act; and set a nationwide drinking water standard for at least PFOA and PFOS (Dingell 2020; NYU Law 2020). In January 2020, the PFAS Action Act passed in the House. Also in January 2020, Senator Bernie Sanders introduced the Preventing Future American Sickness Act of 2020, which addresses several strategies for limiting the presence of PFASs including grants for treating drinking water contamination, a prohibition on food-contact substances, listing PFASs as hazardous air pollutants under the Clean Air Act, and a prohibition of waste incineration (Sanders 2020).

European countries are also taking a closer look at PFASs under REACH and national regulatory frameworks. The German Federal Environment Agency has made the case for designating shorter-chain PFASs as SVHCs and regulating them under REACH as chemicals of equivalent concern to PBT or vPvB substances (Brendel et al. 2018). 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 European Union member countries, including Norway, Denmark, and Germany, have ongoing programs to study and regulate the use of PFASs. On February 1, 2018, the European Commission adopted a proposal for a revised drinking water directive that adds the entire class of PFASs to its list of regulated compounds (European Commission 2018). On May 11, 2020, the national authorities of Germany, the Netherlands, Norway, Sweden, and Denmark initiated a call for evidence to inform a joint REACH restriction proposal to limit the human health and environmental risks associated with the manufacture and use of PFASs. The proposal is expected to be submitted to ECHA in 2022, with restrictions possibly becoming effective as of 2025 (ECHA 2020b).

Exposure limits

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. Contradictory conclusions have been drawn depending on several factors: 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. This has led to different regulatory levels for allowable concentrations in drinking water.

For instance, to address concerns about potential health effects from cumulative exposures to PFOA and PFOS, U.S. EPA established individual chronic reference doses 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 the fall of 2016, after concerns that more than 16 million Americans are exposed to PFOA and PFOS via contaminated drinking water, U.S. EPA reviewed new studies of developmental effects and reduced its 2009 provisional health advisory of 400 parts per trillion (ppt)[17] to a lifetime drinking water health advisory of 70 ppt for PFOA and PFOS combined (U.S. EPA 2020b). In 2018, the Agency for Toxic Substances and Disease Registry (ATSDR) developed MRLs (ATSDR 2018b) that correspond to drinking water advisory levels of 11 ppt for PFOA and 7 ppt for PFOS (Sunderland et al. 2019).

These health advisories are recommendations only and are not legally binding or enforceable. On April 13, 2018, 25 U.S. senators wrote a letter to U.S. EPA Administrator Pruitt asking him to "expeditiously declare an MCL for all PFAS, based on rigorous scientific evidence" (U.S. Senate 2018). Several U.S. states have set their own regulatory standards or advisory guidelines for one or more PFASs in one or more environmental media such as drinking water, groundwater, surface water, soil, air, fish, or wildlife (Longsworth 2020). In 2018, New Jersey became the first U.S. state to adopt a drinking water MCL for any PFAS – 13 ppt for PFNA; in June 2020, New Jersey also adopted an MCL of 14 ppt for PFOA and 13 ppt for PFOS (New Jersey Department of Environmental Protection 2020). In March 2020, Vermont adopted an MCL of 20 ppt for the sum of five PFASs: PFOA, PFOS, PFHxS, PFHpA, and PFNA (Vermont Department of Environmental Conservation 2020). In July 2020, New York adopted MCLs for PFOA and PFOS of 10 ppt each (New York State 2020). In August 2020, Michigan adopted MCLs for the following seven PFASs: 6 ppt for PFNA, 8 ppt for PFOA, 16 ppt for PFOS, 51 ppt for PFHxS, 370 ppt for a GenX compound, 420 ppt for PFBS, and 400,000 ppt for PFHxA (Michigan PFAS Action Response Team 2020). In September 2020, Massachusetts established an enforceable drinking water standard of 20 ppt for the sum of six PFASs (PFOS, PFOA, PFHxS, PFNA, PFHpA, and PFDA) (Massachusetts Department of Environmental Protection 2020).

In California, OEHHA released in 2018 a recommendation for interim notification levels of 14 ppt for PFOA and 13 ppt for PFOS in drinking water based on evidence for developmental toxicity, immunotoxicity, cancer, and liver toxicity (OEHHA 2018). In August 2019, OEHHA updated its research and concluded that the concentration in drinking water that would pose a lifetime cancer risk of no more than one in one million is 0.1 ng/L (or ppt) for PFOA and 0.4 ng/L (or ppt) for PFOS (OEHHA 2019b). However, since those concentrations are lower than current analytical limits of quantification, OEHHA recommended that notification levels for drinking water be set at the lowest levels at which PFOA and PFOS can be reliably detected in drinking water using currently available and appropriate technologies (OEHHA 2019a). As a result, the California State Water Resources Control Board set its

[17] 1 ppt is equivalent to 1 ng/L water.

drinking water notification levels to 5.1 ppt for PFOA and 6.5 ppt for PFOS (California State Water Resources Control Board 2020a). In February 2020, the Board also lowered the response levels for water systems from 70 ppt for PFOA and PFOS combined to 10 ppt for PFOA and 40 ppt for PFOS (California State Water Resources Control Board 2020b).

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. In 2020, EFSA set a tolerable weekly intake limit of 4.4 ng/kg body weight per week for PFOA, PFOS, PFNA, and PFHxS combined (EFSA 2020). However, in the U.S. there are currently no dietary guidelines or tolerable intake limits.

9. CONCLUSIONS

DTSC has determined that treatments for use on converted textiles or leathers that contain any member of the class of PFASs meet the key prioritization criteria (California Code of Regulations, title 22, section 69503.2(a)) 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.

Treatments for converted textiles or leathers are widespread in Californian homes and workplaces and can be significant sources of human and ecological PFAS exposures, including human exposures via inhalation during product use and ecological exposures via discharges to wastewater treatment plants.

The degradation products of the PFASs currently used in treatments for converted textiles or leathers have the potential to cause significant and widespread adverse impacts to sensitive subpopulations, including fetuses, infants, young children, and pregnant women; carpet and upholstery cleaners; workers in upholstered furniture, furnishings, clothing, shoes, and carpet stores; auto dealership workers; and auto detailing technicians. Exposure to PFAS degradation products may also adversely impact environmentally sensitive habitats and threatened and endangered species. Given the known hazard traits of all members of the class, replacing currently used PFASs in the treatments for converted textiles or leathers with other PFASs could constitute a regrettable substitution. Therefore, to encourage identification and evaluation of non-PFAS alternatives, this Priority Product proposal applies to treatments for converted textiles or leathers containing any member of the class of PFASs.

While additional data on product formulations and available alternatives could be helpful in informing a future regulatory proposal, DTSC has sufficient information on potential PFAS exposures from treatments for converted textiles or leathers, and on adverse impacts that could result from these exposures, to support listing this product-chemical combination as a Priority Product.

ACRONYMS AND ABBREVIATIONS

PFAS (sub)classes

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

Individual PFASs

APFO	Ammonium perfluorooctanoate
FBSA	Perfluorobutane sulfonamide
PFBA	Perfluorobutanoic acid
PFBPA	Perfluorobutyl phosphonic acid
PFBS	Perfluorobutane sulfonic acid
PFDA	Perfluorodecanoic acid
PFDS	Perfluorodecane sulfonic acid
PFDoA	Perfluorododecanoic acid
PFDPA	Perfluorodecyl phosphonic acid
PFHpA	Perfluoroheptanoic acid
PFHxA	Perfluorohexanoic acid
PFHxDA	Perfluorohexadecanoic acid
PFHxPA	Perfluorohexyl phosphonic acid
PFHxS	Perfluorohexane sulfonic acid
PFNA	Perfluorononanoic acid
PFOA	Perfluorooctanoate or perfluorooctanoic acid
PFODA	Perfluorooctadecanoic acid
PFOPA	Perfluorooctyl phosphonic acid
PFOS	Perfluorooctane sulfonate or perfluorooctane sulfonic acid
PFPeA	Perfluoropentanoic acid
PFPrA	Perfluoropropanoic acid
PFPrOPrA	Perfluoro-2-propoxypropanoic acid (GenX)
PFTeDA	Perfluorotetradecanoic acid
PFTrDA	Perfluorotridecanoic acid
PFUnA	Perfluoroundecanoic acid
PTFE	Polytetrafluoroethylene
PVDF	Polyvinylidene fluoride
PVF	Polyvinyl fluoride
TFA	Tetrafluoroacetic acid
TFMS	Trifluoromethane sulfonic acid

Other abbreviations used in this document

AA	Alternatives Analysis
AFFF	Aqueous film-forming foam
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	Bioaccumulation factor
BCF	Bioconcentration factor
BMF	Biomagnification factor
C	Carbon
CARB	California Air Resources Board
CAS	Chemical Abstracts Service
CDC	Centers for Disease Control and Prevention
CEH	Center for Environmental Health
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFCs	Chlorofluorocarbons
CFR	Code of Federal Regulations
DDT	Dichlorodiplenyltricloroethane
DTSC	Department of Toxic Substances Control
ECHA	European Chemicals Agency
EPA	Environmental Protection Agency
EPCRA	Emergency Planning and Community Right-to-Know Act
F	Fluorine
GWP	Global warming potential
HF	Hydrofluoric acid
MCL	Maximum contaminant level
MRL	Minimum Reporting Level
NDAA	National Defense Authorization Act
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
PCB	Polychlorinated biphenyl
POPs	Persistent organic pollutants
PPAR	Peroxisome proliferator-activated receptor
ppm	Parts per million
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
RPF	Relative Potency Factor
SB	Senate Bill

SCP	Safer Consumer Products
SNUR	Significant New Use Rule
SVHC	Substances of Very High Concern
TDI	Tolerable daily intake
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
UCMR 3	Unregulated Contaminant Monitoring Rule 3
vPvB	Very persistent, very bioaccumulative
WWTP	Wastewater treatment plan

Units

°C	Degrees Celsius
µg	Micrograms
µg/kg	Micrograms per kilogram
g	Grams
g/L	Grams per liter
kg	Kilograms
kg/yr	Kilograms per year
kJ/mol	Kilojoules per mol
L/kg	Liters per kilogram
m	Meters
m^3	Meters cubed
mg/day	Milligrams per day
mg/kg/day	Milligrams per kilogram per day
mg/L	Milligrams per liter
ng/day	Nanograms per day
ng/g	Nanograms per gram
ng/kg	Nanograms per kilogram
ng/L	Nanograms per liter
ng/mL	Nanograms per milliliter
ng/m^2	Nanograms per meter squared
ng/m^3	Nanograms per meter cubed
Pa	Pascal
ppb	Parts per billion
ppm	Parts per million
ppt	Parts per trillion

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APPENDIX A – POTENTIAL RELEVANT FACTORS

Non-exhaustive list of adverse impact factors that may be relevant to this proposed Priority Product

Relevant Factors are used in SCP's Alternatives Analysis (AA) to make a focused and meaningful comparison of adverse impacts during the product's lifecycle between the Priority Product and alternative. This Profile has identified adverse impacts in the following categories:

- Adverse environmental impacts
- Adverse public health impacts
- Adverse waste and end-of-life effects
- Environmental fate
- Physicochemical properties
- Associated exposure pathways and life cycle segments, including:
    - Manufacture
    - Use
    - Waste generation and management
    - Reuse and recycling
    - End-of-life disposal

At a minimum, all AAs submitted for this product-chemical combination must include a discussion of these impacts and how they compare between the Priority Product and whatever alternative(s) have been identified at the appropriate point in the lifecycle. This list is not intended to be comprehensive. Also, alternatives evaluated in the AA report will likely have additional adverse impacts that don't apply to the Priority Product; these will also need to be assessed in the AA report. Product performance and economics are generally not evaluated in the Profile.

APPENDIX B – REPORT PREPARATION*

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* This document is based on information presented in the Product-Chemical Profile for Carpets and Rugs Containing Perfluoroalkyl or Polyfluoroalkyl Substances.

APPENDIX C – PFAS TOXICOLOGICAL HAZARD TRAITS

This appendix lists the key studies identified regarding the toxicological hazard traits defined in the California Code of Regulations, title 22, section 69503.3(a)(1)(A). Both studies showing positive and negative adverse impact findings are included here for completeness, however the SCP regulations are not based on a weight of evidence approach, nor on a systematic review methodology.

Table A1. Findings regarding carcinogenicity

Summary of Findings	Adverse Impact Finding	Reference
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 (2020)
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 (2016)
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)

Summary of Findings	Adverse Impact Finding	Reference
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-Jørgensen et al. (2014)
Tumor triad in male rats (liver, Leydig cells, pancreatic) dosed with APFO.	Positive	Biegel et al. (2001)
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	RIVM 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-Jørgensen et al. (2014)

Summary of Findings	Adverse Impact Finding	Reference
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)
No evidence of carcinogenicity in either male or female Sprague-Dawley rats treated with PFHxA when administered orally daily for 2 years.	Negative	Klaunig et al. (2015)
No observed association between occupational APFO exposure and the risk of dying or developing liver, pancreatic, testicular, kidney, prostate, breast, bladder, and thyroid, cancers.	Negative	Raleigh et al. (2014)
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)

Table A2. Findings regarding developmental toxicity

Summary of Findings	Adverse Impact Finding	Reference
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 (2016a)
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 (2016)
Treatment of pregnant CD-1 mice with NH4+ PFBA by oral gavage is 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)

Summary of Findings	Adverse Impact Finding	Reference
2,3,3,3-Tetrafluoro-2-(heptafluoropropoxy)propanoate (a PFOA replacement technology) in rats at 100 and 1000 mg/kg-day decreases fetal weights and produces 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	RIVM et al. (2016)

Table A3. Findings regarding reproductive toxicity

Summary of Findings	Adverse Impact Finding	Reference
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)
PFOS, PFDoA, PFNA, and PFOA are cytotoxic in JEG-3 human placental cells (EC50s range from 107 - 647 µM). PFOS, PFOA, and PFBS are potential CYP19 aromatase inhibitors (IC50s 57 - 80 µM). Notable inhibitory effect of PFBS with relatively low uptake in cells.	Positive	Gorrochategui et al. (2014)
Treatment of pregnant CD-1 mice with PFBA by oral gavage was associated with increased incidence of fetal resorption and 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)

Summary of Findings	Adverse Impact Finding	Reference
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)

Table A4. Findings regarding cardiovascular toxicity

Summary of Findings	Adverse Impact Finding	Reference
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. (2007a); Skuladottir et al. (2015); Steenland et al. (2010); Wang et al. (2012); Winquist and Steenland (2014)
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. (2007b); Sakr et al. (2007a)
No increased mortality ratio in exposed workers for heart disease.	Negative	Lau et al. (2007); Lundin et al. (2009); Steenland and Woskie (2012)

Table A5. Findings regarding endocrine toxicity

Summary of Findings	Adverse Impact Finding	Reference
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. (2007b); Sakr et al. (2007a)
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)
PPAR-alpha activities are induced at lower cellular concentrations for short-chain homologs relative to long-chains.	Positive	Rosenmai et al. (2016)

Summary of Findings	Adverse Impact Finding	Reference
PFHxSK inhibits 11 -HSD2 in human and rat kidney microsomes. PFHxSK is more potent in human than rat microsomes.	Positive	Zhao et al. (2010)
Non-cytotoxic levels of PFHxDI (dodecafluoro-1,6-diiodohexane) significantly promote the proliferation of MCF-7 breast cancer cells. PFHxDI's preferential binding affinity for estrogen receptor α and β isoforms mediated this cellular response.	Positive	Song et al. (2018)
PFHxS can disrupt the thyroid system of Wistar rats, as measured by T4 levels in dams and offspring. PFHxS potentiated the antiandrogenic effect of an endocrine disruptor mixture in male offspring.	Positive	Ramhøj et al. (2018)
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.	Positive	Weiss et al. (2009)
No effect from PFBA and FTOHs on binding to transthyretin (for TH transport) in binding assay.	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 has 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. (1998); Olsen et al. (2003); Olsen and Zobel (2007); Sakr et al. (2007a); Sakr et al. (2007a)

Summary of Findings	Adverse Impact Finding	Reference
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)

Table A6. Findings regarding hematotoxicity

Summary of Findings	Adverse Impact Finding	Reference
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. (2007b); Sakr et al. (2007a)

Table A7. Findings regarding hepatotoxicity and digestive system toxicity

Summary of Findings	Adverse Impact Finding	Reference
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. (2007b); Sakr et al. (2007a)

Summary of Findings	Adverse Impact Finding	Reference
Probable link between PFOA exposure and ulcerative colitis.	Positive	C8 Science Panel (2012); Steenland et al. (2015)
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 is 171,000 ng/mL in females and 173,000 ng/mL in males (K-PFOS capsules).	Positive (low N)	Seatcat et al. (2002)
In rats, PFHxS is 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 is 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 revealed increased liver and kidney weights, hepatocellular hypertrophy and alterations in blood chemistries. Chronic feeding study revealed various liver pathologies.	Positive	RIVM et al. (2016)

Summary of Findings	Adverse Impact Finding	Reference
Increased incidence of focal cystic degeneration, and centrilobular hepatocellular hypertrophy and necrosis in male Sprague-Dawley rats given 50 mg/kg-day ammonium 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)-propanoate orally for 2 years. Similar results in female rats given 500 mg/kg-day. Clinical chemistry evaluations among males receiving 50 mg/kg-d showed increases in enzymes indicative of liver injury (alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase, and sorbitol dehydrogenase).	Positive	Rae et al. (2015)
K+ PFHxS to Sprague Dawley rats by oral gavage. Effects in parental males include 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 is 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 show greater cytotoxicity on human liver HL-7702 cells.	Positive	Sheng et al. (2017)

Summary of Findings	Adverse Impact Finding	Reference
6:2 chlorinated polyfluorinated ether sulfonate (6:2 Cl-PFESA) and hexafluoropropylene oxide (HFPO) show 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 is similar for C4 and C5 ("short-chain") PFCAs as for C12 and C14 ("long-chain"), but is induced at lower cellular concentrations by the C4 and C5 compounds tested. Other PFCAs tested (C6-C10) induce 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. The observed U-shaped dose-response for serum bilirubin 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)

Table A8. Findings regarding immunotoxicity

Summary of Findings	Adverse Impact Finding	Reference
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)

Summary of Findings	Adverse Impact Finding	Reference
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. (2017); Granum et al. (2013); Looker et al. (2014)
In human peripheral blood leukocytes, PFBS inhibits the release of TNF-α and IL-10, but IL-6 and IFN-γ are unaffected. In THP-1 cells, PFBS also inhibits NF-κB activation (by inhibiting LPS-induced phosphorylation of P65, necessary for NF-κB transcription), and prevents 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 increases 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 is suppressed in females at 100 mg/kg. T lymphocyte numbers are increased in males at 100 mg/kg.	Positive	Rushing et al. (2017)

Table A9. Findings regarding nephrotoxicity and other urinary system toxicity

Summary of Findings	Adverse Impact Finding	Reference
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)

Summary of Findings	Adverse Impact Finding	Reference
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)

Table A10. Findings regarding neurodevelopmental toxicity

Summary of Findings	Adverse Impact Finding	Reference
Positive association with Attention Deficit Hyperactivity Disorder (ADHD) in children in a highly PFOA-exposed community.	Positive	Hoffman et al. (2010)
A single exposure to PFHxS (9.2 mg/kg) on postnatal day 10 affected the cholinergic system and altered adult spontaneous behavior and cognitive function in male and female mice.	Positive	Viberg et al. (2013)
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)

Table A11. Findings regarding neurotoxicity

Summary of Findings	Adverse Impact Finding	Reference
PFASs such as PFOA and PFOS 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)

Table A12. Findings regarding respiratory toxicity

Summary of Findings	Adverse Impact Finding	Reference
90-day evaluation of NaPFHxA in Sprague Dawley rats following oral gavage, indicates 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)
Three cases of young healthy adults, following occupational inhalation of a new waterproofing formulation containing an acrylate fluoropolymer, developed a rapidly progressive dyspnea; two had hypoxemia and flu-like reactions within 1-2 hours of exposure.	Positive	Lazor-Blanchet et al. (2004)
A commercial nanofilm spray product containing a perfluorosilane induced a concentration-dependent decrease of the tidal volume, significant increases of protein level in bronchoalveolar lavage fluid, reduced body weight, atelectasis, emphysema, and lung hemorrhages in BALB/cJ mice compared to a formulation containing alkylsilane.	Positive	Nørgaard et al. (2010)
Normal pulmonary function in fluoropolymer plant workers with serum PFOA.	Negative	Sakr et al. (2007b); Sakr et al. (2007a)

Table A13. Findings regarding other toxicological hazard traits

Summary of Findings	Adverse Impact Finding	Reference
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)
Chlorinated polyfluorinated ether sulfonates (6:2 Cl-PFAES and 8:2 Cl-PFAES) bound to PPAR-α, -β, and -γ with affinity higher than PFOS and has agonistic activity toward PPAR signaling pathways with similar or greater potency.	Positive	Li et al. (2018)
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); 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 is a mild skin irritant and a moderate to severe eye irritant in rabbits.	Positive	Gordon (2011)
Survival rate of female Sprague-Dawley rats significantly decreased at presumed maximally tolerated dose of PFHxA (200 mg/kg-d) in 2-year study.	Positive	Klaunig et al. (2015)
Experimental and QSAR modeling results showed PFAS mixture cytotoxicity in amphibian fibroblasts was approximately additive, except with PFOS and PFOA, which were weakly synergistic.	Positive	Hoover et al. (2019)
Dopaminergic neurodegeneration in nematodes (*C. elegans*) exposed to PFOS.	Positive	Sammi et al. (2019)
No generation of reactive oxygen species (ROS) nor DNA damage by PFBS and PFHxA in HepG2 cells.	Negative	Eriksen et al. (2010)

Table A14. Findings regarding phytotoxicity

Summary of Findings	Adverse Impact Finding	Reference
Aquatic toxicity of PFHxA, PFHpA, PFOA and PFNA to several species of marine microalgae increases with increasing chain length: for every extra perfluoromethylene group in the alkyl chain, toxicity increases twofold. EC50 ranges 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 inhibits algal growth in a concentration-dependent manner.	Positive	Liu et al. (2008)
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); Ding et al. (2012a); Ding et al. (2012b); Environment Canada (2006); Environment Canada (2012); Giesy et al. (2010); Hoke et al. (2012); Latala et al. (2009); Liu et al. (2008); UNEP/POPS/POPRC (2016);

Summary of Findings	Adverse Impact Finding	Reference
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 increases with increasing fluorinated carbon chain length. PFBA was more toxic than expected, likely due to 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)

Table A15. Findings regarding wildlife developmental impairment

Summary of Findings	Adverse Impact Finding	Reference
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)
Adverse impacts on embryonic development, hatchability, and time to hatch of Japanese medaka (*O. latipes*) eggs from exposure to PFOA and PFOS	Positive	Ji et al. (2008)

Summary of Findings	Adverse Impact Finding	Reference
Developmental toxicity observed in zebrafish embryos exposed to 6:2 FTCA. Observed effects include: decreased hatching and survival percentages, reduced heart rate, increased malformations (especially pericardial edema), decreased erythrocyte number, and disrupted erythroid differentiation during zebrafish embryonic development.	Positive	Shi et al. (2017a)
Delayed development (time to metamorphosis) in northern leopard frog (Rana pipiens) tadpoles exposed to 10 ug/L PFHxS for 40 days.	Positive	Hoover et al. (2017)
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. (2017b)
Dopamine decreased significantly in the brains of Northern Leopard frogs treated with PFOA and PFOS. Significant increases in dopamine turnover also resulted from PFOA and PFOS treatment.	Positive	Foguth et al. (2019)

Summary of Findings	Adverse Impact Finding	Reference
PFECAs (e.g., perfluoro (3,5,7-trioxaoctanoic) acid (PFO3OA), perfluoro (3,5,7,9-tetraoxadecanoic) acid (PFO4DA), and perfluoro (3,5,7,9,11-pentaoxadodecanoic) acid (PFO5DoDA)) tested on zebrafish embryos damaged swim bladder mesothelium and significantly lowered thyroid hormone levels in larvae. Exogeneous T3 or T4 supplementation partly rescued PFECA-induced posterior swim bladder malformation.	Positive	Wang et al. (2020)

Table A16. Findings regarding wildlife reproductive impairment

Summary of Findings	Adverse Impact Finding	Reference
Decreased fecundity of female Japanese medaka (*O. latipes*) after exposure to PFOS and PFOA.	Positive	Ji et al. (2008)
Egg concentrations of PFASs are negatively correlated with hatching success in wild and laboratory birds.	Positive	Custer et al. (2012); Custer et al. (2014); Nordén et al. (2016)
PFOS has adverse impacts on the reproduction of bumblebees (*Bombus terrestris*), including reduced ovarian size.	Positive	Mommaerts et al. (2011)

Table A17. Findings regarding wildlife survival impairment

Summary of Findings	Adverse Impact Finding	Reference
Decreased hatching and survival percentages observed in zebrafish embryos exposed to 6:2 FTCA and to F–53B [6:2 chlorinated polyfluorinated ether sulfonate].	Positive	Shi et al. (2017a); Shi et al. (2017b)

Summary of Findings	Adverse Impact Finding	Reference
Adverse impacts on fecundity, embryonic development, hatchability, and time to hatch of Japanese medaka (*O. latipes*) eggs from exposure to PFOA and PFOS.	Positive	Ji et al. (2008)
Survival rate of female Sprague-Dawley rats significantly decreased at presumed maximally tolerated dose of PFHxA (200 mg/kg-d) in two-year study.	Positive	Klaunig et al. (2015)

APPENDIX D – APPENDIX REFERENCES

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