A primer on perfluoroalkyl substances (PFAS) – emerging contaminants in drinking water

EXECUTIVE SUMMARY

Poly- and perfluorinated substances (PFAS) are man-made chemicals used globally since the 1960s in a variety of industrial and commercial products. These compounds were designed to resist heat, oil, stains, grease, and water and have vastly improved the quality of modern life. The unique ability of PFAS to repel both oil and water has led to their principal application in stain repellant products (carpet, furniture/upholstery), nonstick cookware, water-proof clothing (e.g., Gortex, Tyvek) and coated paper. In addition, PFAS will produce a stable foam that can flow across liquid solvents, which has led to their widespread use as a high performance “aqueous film-forming foam” (AFFF). AFFF was used for firefighting burning fuels at military bases, airports, oil refineries, and firefighting training facilities.

The carbon-fluorine bond is the strongest in nature, making these compounds highly persistent in the environment. The length of the carbon chain helps determine both its toxicity and fate in the environment. The “C8” compounds, which include perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), contain eight carbons and are the most persistent, bioaccumulative and biologically active compounds within this class of chemicals. As the PFAS molecule is analogous to dietary fatty acids, high doses in laboratory animals appear to affect the components of cells and tissues that typically metabolize or accumulate lipids.

Currently, PFAS issues are appearing in the news on a weekly basis. To date, there are hundreds of research studies on PFAS that have been performed on both animals and humans (U.S. EPA health effects documents cite over 300 references for both PFOS and PFOA). In 2016, the EPA published a very low drinking water advisory (0.07 μg/L), which has generated significant public concern near manufacturing or waste sources.

This whitepaper presents a general “primer” on both PFOA and PFOS, including a) chemical characteristics, use and production b) environmental disposition and fate c) environmental exposure d) toxicological effects and e) ecological effects and aquatic toxicity. This paper also highlights the needs, based on developing regulatory frameworks and public concern, for additional research on toxicity, the growing need for environmental consulting related to PFAS for both industry and the public, and the potential for a burgeoning remediation market.
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1. BACKGROUND

Poly- and perfluorinated substances (PFAS) are man-made chemicals that have been used globally since the 1960s in a wide variety of industrial and commercial products. They were purposefully developed to have properties which make them resistant to heat, oil, stains, grease, and water. PFAS compounds and associated fluoropolymers are critical to modern life as they have greatly enhanced the function and durability of items society takes for granted, such as airplanes, automobiles, machinery, cell phones and water-repellant materials. Many chemicals in this group, the most common being perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), are resistant to degradation and therefore have become ubiquitous in the environment. Although the molecular backbone of PFOA and PFOS resembles fatty acids (normal components of lipids), they do not bioaccumulate in fat. Studies have shown that nearly all (~95%) people tested have detectable levels (low parts per billion) of PFAS in their blood and, although epidemiological studies have “linked” several health conditions to high exposures of PFOS/PFOA, no adverse effects on health have yet to become evident in children or adults. Toxicity has also been shown to be very low in fish and wildlife.

Currently, PFAS are appearing in the news on a weekly basis. This phenomenon has been exacerbated by EPA’s recent unprecedented development of drinking water advisories in the low part per trillion (ppt) levels, and the subsequent recognition that it is not uncommon to find PFOA and PFOS in groundwater at levels close to the health advisories. In the Northeast, people are afraid to drink water with parts per trillion concentrations of PFOA even though higher levels of more toxic conventional constituents (e.g., arsenic, lead and cadmium) may be found in groundwater.

The purpose of this paper is to present a general overview of the scope of the PFAS “problem.” The amount of both peer-reviewed and grey literature publications on PFAS is daunting. Over 400 peer-reviewed manuscripts addressing sources, environmental disposition, behavior (principally in groundwater), fate, exposure and risk are published each year. EPA health effects documents for PFOA and PFOS cite over 300 human and animal health studies for each individual compound. Consequently, introductory remarks may help put this current “crisis” in perspective.

In the 20th century, headlines on “unregulated contaminants” were shown to be cyclical. Fears about health effects following low level exposure to potentially toxic compounds (e.g., asbestos, arsenic, PCBs, dioxins, mercury, etc.) were often “linked” to adverse health effects but causality was difficult to prove. For example, perchlorate was used as a successful drug to treat hyperthyroidism in the 1940’s but, after discovery of low levels in food and drinking water, it was rebranded as a “rocket fuel” or a “potent thyroid toxin.” After a decade of high profile stories in the media and hundreds of millions of dollars in remediation costs, the EPA has recently concluded that low level chronic exposure is not a hazard to humans. Perchlorate has since been removed from the EPA’s Unregulated Contaminant List.

Another important aspect of PFAS which has been over-emphasized is that they bioaccumulate into higher organisms, including humans. The “background” levels of PFAS in human blood, however, are within the same range as other trace contaminants, including arsenic, cadmium, mercury, lead and formaldehyde. PFAS in the tissues of carnivorous birds (gulls, eagles) and mammals (seals, polar bears) are about one thousand times lower (parts per billion) compared to legacy compounds such as PCBs and mercury (parts per million). These low levels of PFAS have not been shown to adversely affect survival, growth or reproduction in wildlife.

Finally, though PFAS has been studied for decades, scientists are still at a loss to identify any “mode-of-action,” i.e., by what internal mechanism does PFAS cause toxicity. Laboratory studies have shown that PFOA and PFOS activate small organelles called peroxisomes. These subcellular structures are responsible for the metabolism of fatty acids and the breakdown of some types of environmental contaminants. PFOA and PFOS stimulate the alpha form of the “peroxisome proliferator-activated receptor” (PPARα), which is very active in rodents but weakly active in humans. It is thus questionable whether the use of mice and rats, which appear to be more sensitive to very high doses of PFAS, are the appropriate biological model for deriving a drinking water criterion.

2. PFAS CHARACTERISTICS, USE AND PRODUCTION

PFOS and PFOA are compounds in the broader class of PFAS chemicals, which in turn are a subset of perfluorinated compounds (PFCs). Fluorine is the most electronegative element in the periodic table. Consequently, perfluoroalkyl compounds are extremely stable owing to the strength of the carbon-fluorine bonds (the strongest in nature), the presence of the three electron pairs surrounding each fluorine, and the shielding of the carbon atoms by the fluorine...
atoms (3M 1999; Kissa 2001). These chemical properties make PFAS resistant to degradation by microorganisms, sunlight (photolysis) and destruction by reaction with acids, bases, oxidants, and reductants (3M 2000; EPA 2008f; OECD 2002, 2007; Schultz et al. 2003). The strong electronegativity also allows analytical laboratories to detect PFAS in the low parts per trillion range.

The unique properties of PFOA and PFOS are due to a water-soluble “head” and a “tail” that is insoluble in both water and lipid:

Unlike PCBs or dioxins, the number of carbons in the molecular chain can vary between two and 14. Thus the number of permutations of chain length, number of fluorine molecules on the chain, and type of water soluble “head” (e.g., carbonic, sulfonic, alcohol, phosphoric) accounts for over 6,000 different types of PFAS. PFOA and PFOS fit in the “middle range” of carbon length and, because of their unique properties, are the two most widely produced and studied PFAS. The overall consensus in the current literature is that PFAS chains with six carbons or less do not appear to pose a hazard to human health or the environment.

Past processes used for the production of PFAS include electrochemical fluorination (ECF) and telomerization. The ECF process generally results in even- and odd-numbered, branched and linear chains of perfluoroalkyl compounds, whereas telomerization produces even-numbered, linear chains. Most production today is via the telomerisation process (Buck et al., 2011).

The terminology can be very confusing. The Latin prefix “per” means “beside” while “poly” means many. Thus, a perfluorinated compound is one where all of the hydrogens on a carbon chain are replaced by fluorine molecules, which are all beside one another (e.g., PFOA and PFOS below). A polyfluorinated compound, however, is not fluorine saturated so will have either an unfluorinated carbon group in the chain, or has a “break” in the molecule, such as an ester or ketone group:

The presence of an oxygen molecule in the chain or unsaturated carbons (containing hydrogens rather than a fluorine) make the compound vulnerable to microbial degradation. Thus the newer replacements for PFAS (e.g., “GenX”) are designed to be more environmentally friendly as the breakdown products typically have fluorine chains of less than six carbons.

The unique ability of PFAS to repel both oil and water has led to their principal application in stain repellant products for carpet, furniture/upholstery, nonstick cookware, clothing (e.g., Gortex), Tyvek and coated paper. They are also used as wetting agents (surfactants) in industrial processes and fluoropolymer production (principally Teflon products).

2.1 PFOS

Low water concentrations of PFAS will produce a stable foam that can flow across liquid solvents, which has led to their widespread use as a high performance “aqueous fire-fighting foam” (AFFF). In the 1960s, the Department of Defense issued specifications for AFFF for use in extinguishing fires at military bases, airports, oil refineries, and firefighting training facilities throughout the U.S. In 2002, the 3M Company, the largest manufacturer of AFFF in the world, voluntarily phased out manufacture of PFOS. AFFF
has not been manufactured in the U.S. since 2002 (Place & Field, 2013; Houtz et al., 2014) and eight other major global manufacturers phased out production between 2006 and 2015.

AFFF products containing PFOS (and PFOA) may still be in use. Although AFFF was reformulated in the early 2000s and no longer contains PFOS, civilian and military airports continue to maintain an inventory of PFOS-based AFFF. In recent years, EPA issued Significant New Use Rules (SNURs) under its Toxic Substances Control Act (TSCA) authority to restrict the production and use of products that contain PFOS and its precursors; however, the EPA, Federal Aviation Authority, and other regulatory agencies continue to allow its use (FAA, 2011).

Table 1 presents 2011 estimates on the quantities of AFFF at U.S. airports and other facilities that store flammable hydrocarbons (e.g. refineries, bulk storage terminals) (Darwin, 2011).

<table>
<thead>
<tr>
<th>Use Sector</th>
<th>PFOS-based AFFF 2004</th>
<th>PFOS-based AFFF 2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Military &amp; Other Federal</td>
<td>2,100,000</td>
<td>1,094,700</td>
</tr>
<tr>
<td>Civil Aviation (ARFF)</td>
<td>130,000</td>
<td>20,000</td>
</tr>
<tr>
<td>Oil Refineries</td>
<td>950,000</td>
<td>152,000</td>
</tr>
<tr>
<td>Other Petro-Chem</td>
<td>1,000,000</td>
<td>500,000</td>
</tr>
<tr>
<td>Civil Aviation (Hangers)</td>
<td>190,000</td>
<td>70,300</td>
</tr>
<tr>
<td>Fire Departments</td>
<td>120,000</td>
<td>60,000</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>150,000</td>
<td>75,000</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>4,600,000</strong></td>
<td><strong>1,972,000</strong></td>
</tr>
</tbody>
</table>

Their unique fire extinguishing properties make it difficult to find equally effective replacement compounds for fire-fighting foam applications. Compounds with six carbon fluorinated chains, which have short environmental half-lives and are less toxic, have been approved for commercial use by EPA’s PFC Stewardship Program.

### 2.2 PFOA

PFOA has been manufactured since the 1940s, and unlike PFOS, PFOA manufacture continues although most companies are phasing it out. In the past, PFOA was used primarily as an aqueous dispersion agent (additive) in the manufacturing of fluoropolymers, which are substances with special properties that have a wide variety of commercial and industrial applications (e.g., fluoropolymers like Teflon®, Gore-Tex® fabric, Stainmaster® carpets, and Scotchgard®). PFOA can also be created by the degradation of some fluorinated telomers that are not manufactured using PFOA. Fluorinated telomers are used in fire-fighting foams and as surface protection to provide soil, stone, greases, and water resistance in products such as tile, stone, textiles, and paper packaging (EPA, 2014).

In 2006, EPA partnered with eight chemical companies to launch the 2010/2015 PFOA Stewardship Program to reduce emissions and product content of PFOA and long-chain PFCs that break down to PFOA by 95% in 2010, and to eliminate long-chain PFCs by 2015. As of January 2015, the program was on track to meet its goal of phasing out the use of PFOA (EPA, 2015).

### 3. ENVIRONMENTAL DISPOSITION AND FATE

As discussed, PFAS are very stable compounds that are resistant to biodegradation, direct photolysis, atmospheric photooxidation, and hydrolysis. The chemical stability and physical-chemical properties of PFAS (negligible vapor pressure, high solubility in water, and moderate sorption to solids) suggest that the final sink for these compounds is surface water and, to a lesser extent, soils/sediment (3M 2000; Prevedouros et al. 2006). Koc values ranging from 17 to 230 indicate that PFOA will be mobile in soil and can leach into groundwater (Davis et al. 2007; Prevedouros et al. 2006).

Because of their water solubility, long production history and propensity to bioaccumulate, low concentrations of PFCs have been found worldwide in soil, groundwater, surface water, rain, ice caps, air, plants, animal tissue, and blood serum (including remote environments) and detections have been observed at relatively high concentrations in animals at the top of the food chain (e.g., polar bears) (Furl & Meredith, 2010).

Although there have been many papers addressing both the type and strength of the various sources of PFAS, there is very little information on the mass flux of PFAS from one media to another; inventories of global releases are currently not important as levels are rapidly declining since manufacturing of the most persistent perfluoroalkyl congeners are being phased out. On a historical scale, global production of PFOA has been about 10 times greater than global emissions (Prevedouros et al., 2006). To date, it is known that the majority of these emissions (>95%) are directly released into surface waters, whereas deposition from atmospheric emissions is considered to be marginal (<5%) (Ahrens and Bundschuh, 2014). Studies in both
the U.S. and Europe show major sources of PFAS include industrial and municipal wastewater treatment plants (WWTP), fire-fighting incidents/training areas and landfills (Eschauzier et al., 2012; Ahrens and Bundschuh, 2014; Hu et al., 2016).

Municipal WWTP effluents and infiltration of urban runoff and leachate are thus the major sources of diffuse pollution to rivers and aquifers. WWTPs are an important source as PFAS are not removed by standard treatment methods and labile precursors biodegrade, increasing concentrations in effluent relative to influent. PFOA is not retained by activated sludge and is essentially discharged unchanged into receiving waters, while about half of the PFOS load is retained in sewage sludge. Median concentrations of PFOA and PFOS in European wastewaters are similar (~12 nanograms per liter (ng/L; one nanogram per liter is equal to one part per trillion [PPT]) while levels in receiving waters (~3 ng/L) were lower (Eschauzier et al., 2012). Levels in surface waters in the U.S. are similar, generally near the detection limit (~1 ng/L) in rural areas, less than 20 ng/L in U.S. rivers and streams, and higher levels near wastewater treatment plants or near contaminated properties such as Superfund sites (USEPA, 2016a). Concentrations found in the open ocean for PFOA are generally less than 0.4 ng/L, while PFOS levels are typically less than 0.07 ng/L.

The widespread presence of PFOA and PFOS at low concentrations in surface water in the U.S. indicates that drinking water taken from these sources will often contain detectable levels of these substances (ATSDR, 2014). Detection of PFAS in drinking water within the U.S. has been closely linked to industrial sites, military fire training areas, and wastewater treatment plants (Hu et al., 2016). Consequently, drinking waters in rural areas will typically be below the limit of detection whereas suburban/urban areas may see a higher frequency of detections. Generally speaking, if detected, surface water and groundwater will have very low levels (e.g., typically <10 ng/L).

The table below presents an overall summary of the environmental disposition of PFAS and compares them to persistent legacy compounds typically found in the environment.

<table>
<thead>
<tr>
<th>Highly water soluble</th>
<th>PFCs</th>
<th>Dioxins &amp; PCBs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bind well to soil &amp; sediments</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>Degradation in environment to some extent</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>Bioaccumulates in fish</td>
<td>NO<em>YES</em></td>
<td>YES</td>
</tr>
<tr>
<td>Bioaccumulates in lipids</td>
<td>NO</td>
<td>YES</td>
</tr>
<tr>
<td>Drinking water is major exposure route</td>
<td>YES</td>
<td>NO</td>
</tr>
</tbody>
</table>

Unlike legacy compounds like PCBs and dioxins, the unique characteristics of PFAS means they are highly water soluble and lipid insoluble, will not bind well to soil and sediment, will not degrade over time, and that longer chain perfluoralkyls will bioaccumulate in fish (at relatively low levels).

4. ENVIRONMENTAL EXPOSURE

It is clear from the literature that every living organism has been exposed to many types of PFAS in one way or another. The available literature on PFAS exposure and effects is dominated by PFOS and PFOA due to their production and concern that they could biomagnify to a level whereby humans consuming fish may be affected. Less information is available on the exposure of other short- or long-chain PFAS derivatives. For the less investigated polyfluorinated chemicals, toxicology is often estimated based on structure-activity relationships or homologues.

4.1 EXPOSURE OF HUMANS

There is still a lot of misinformation and debate on how humans are exposed to PFAS, and even well-designed studies can be confounded by the analytical limitations of measuring PFAS (e.g., lack of available standards, cross-contamination, interferences). In 2009, EPA conducted a market basket study entitled “Perfluorocarboxylic Acid Content in 116 Articles of Commerce” (EPA, 2009) with the goal to “identify the major PFCA sources in non-occupational, indoor environments by determining the content of these chemicals in a variety of articles of commerce and rank them in terms of source strengths.” The following table presents the results of the concentrations, as well as the total mass, of PFAS in household items thought to contain the compounds.

### COMPARISON OF SOURCE STRENGTHS FOR TOTAL AMOUNT OF PFCA (TPFCA) IN A HYPOTHETICAL “TYPICAL” AMERICAN HOME (USEPA, 2009)

<table>
<thead>
<tr>
<th>Article Category</th>
<th>TPFCA in Article</th>
<th>Article Quantity</th>
<th>TPFCA in Home (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-treated Carpeting</td>
<td>48.4 ng/cm²</td>
<td>130 cm²</td>
<td>72.6</td>
</tr>
<tr>
<td>Commercial carpet-care liquids</td>
<td>12000 ng/g</td>
<td>6 kg</td>
<td>71.8</td>
</tr>
<tr>
<td>Treated floor waxes and stone/tiler/wood sealants</td>
<td>2430 ng/g</td>
<td>1 kg</td>
<td>2.42</td>
</tr>
<tr>
<td>Treated home textile and upholstery</td>
<td>336 ng/g</td>
<td>5 kg</td>
<td>1.68</td>
</tr>
<tr>
<td>Household carpet/fabric-care liquids and foams</td>
<td>953 ng/g</td>
<td>1 kg</td>
<td>0.95</td>
</tr>
<tr>
<td>Treated apparel</td>
<td>198 ng/g</td>
<td>2 kg</td>
<td>0.4</td>
</tr>
<tr>
<td>Membranes for apparel</td>
<td>124 ng/g</td>
<td>1 kg</td>
<td>0.12</td>
</tr>
<tr>
<td>Treated food contact paper</td>
<td>3100 ng/g</td>
<td>0.01 kg</td>
<td>0.03</td>
</tr>
<tr>
<td>Thread seal tape and pastes</td>
<td>603 ng/g</td>
<td>0.02 kg</td>
<td>0.01</td>
</tr>
<tr>
<td>Non-stick cookware</td>
<td>0.028 ng/cm²</td>
<td>1 m²</td>
<td>0.0003</td>
</tr>
<tr>
<td>Dental floss and plaque removers</td>
<td>31.3 ng/g</td>
<td>0.005 kg</td>
<td>0.0002</td>
</tr>
</tbody>
</table>

Source: G. Post, NJDEP
A wide disparity in concentration can be seen between materials like carpeting, upholstery treatment and floor waxes vs. nonstick cookware, food paper and dental floss. Since 2009, other studies have shown that human exposure to PFAS is mainly by ingestion of food and inhalation of household dust; consumption of drinking water is only significant if the water source is contaminated (Lorber and Egeghy, 2011; Frommea et al., 2009; Post et al., 2012). For adults, the median PFOA intake is estimated to be 70 ng/day. The contribution from diet and drinking water are reported to be about 46 and 24 ng/day, respectively (below, from Lorber and Egeghy, 2011).

For children, the intakes appear to be moderately higher for diet and drinking water, most likely due to the fact that they weigh much less than adults so, pound-for-pound, they proportionally consume more food and water than adults.

Once in the body, PFAS are not metabolized; they bind strongly to blood albumen (not fat) and are mainly concentrated in the liver and kidneys. In European studies, observed serum and plasma concentrations range from 1 to 116 μg/L for PFOS and from 0.5 to 40 μg/L for PFOA. In the U.S., mean blood levels for all age groups are 9.3 μg/L (PFOS) and 3.1 μg/L (PFOA). A study of retired fluorochemical workers estimated the half-life of PFOS and PFOA at 5.4 years and 3.8, respectively (Olsen, 2007). The half-life of PFOS and PFOA in laboratory animals (mice, rats, monkeys) is much shorter, in the range of months, which can cause extrapolation issues in tests.

4.2 EXPOSURE OF AQUATIC ORGANISMS

In the aquatic environment, PFAS are subject to partitioning processes, whereby short-chain compounds are mainly distributed in the water phase, whereas long-chain derivatives tend to bind to particles. Although few bioconcentration studies with PFAS exist for aquatic organisms, laboratory studies with fish have shown that the bioaccumulation potential of perfluorinated carboxylates and perfluorinated sulfonates is related to chain length, with the greatest accumulation being observed for those compounds with the longest fluorinated carbon chains (Martin et al. 2003a, 2003b; Condor et al. 2008).

In rainbow trout, fluorinated carbon chains shorter than seven do not bioaccumulate and typically have BCFs less than 1.0.

In the wild, PFOS (eight carbon chain) is typically the dominant PFAS accumulating in fish with the concentration increasing along the food chain, showing its high bioaccumulation potential. In contrast, PFOA has a low bioaccumulation potential and is relatively similar among species from different trophic levels (Ahrens and Bundschuh, 2014). On a relative basis, concentrations of PFOS are greater in higher trophic level predators (e.g., seals, bears, mink, cormorants, gulls, ospreys, eagles). Compared to legacy contaminants (PCBs, DDT, mercury), however, PFAS are about an order of magnitude lower (low to mid parts per billion).

5. TOXICOLOGICAL EFFECTS

There are data available showing a positive correlation between (sub)chronic PFOS and PFOA exposure and reproductive and/or developmental (growth) effects in animals. In humans, the results must be gleaned from ecologic and epidemiological studies, which are not consistent. Animal studies show dose-related effects from PFOS and PFOA on the liver, the gastrointestinal tract and on thyroid hormone levels as well as developmental changes (e.g., low pup birth weight) following exposure of the pregnant dams. In general, the development of a “reference dose,” or a daily human intake considered safe over a lifetime, results in similar values (both extremely low) for both PFOA (30 ng/
kg body weight/day) and PFOS (20 ng/kg body weight/day). The very low RfD derived for PFOA and PFOS, discussed below, is mainly driven by the fact that a) PFOA/PFOS have a much longer half-life in humans than in rodents or primates b) human equivalent doses are ‘back-calculated’ from PFOA concentrations measured in rodent serum at the no- or low-effect dose and c) uncertainty factors (accounting for interhuman variability and species-specific differences) are judiciously applied. The reference doses are unique in that they result in the lowest drinking water health advisory ever issued by the EPA, much lower than legacy compounds that include PCBs, toxic metals and pesticides.

It is important to point out that, unlike many other chemicals the EPA has developed protective benchmarks for, there is no known "mode-of-action" for PFOA/PFOS. It is not metabolized at all by any organ system, being excreted in the same form it was administered. It is currently thought that peroxisome proliferator-activated receptor (PPARα), which is active in rodents but only weakly active in humans, is responsible for causing adverse effects, including some types of tumors, following PFOA/PFOS exposure to laboratory animals. Additionally, the dosing levels result in blood concentrations in the test animals that are at least 100 times higher (mg/L) than concentrations ever observed in humans (μg/L).

The developmental effects seen in rats and mice on which the EPA based their Health Advisories include: lower birth weight, minor skeletal (bone formation) changes, and faster onset of puberty in male mice. These health effects, however, have not all been found or confirmed in humans.

5.1 EFFECTS IN HUMANS – PFOA

One of the earliest human epidemiological studies on PFOA was the “C8 Health Project.” Drinking water for communities in the mid-Ohio Valley region of West Virginia was contaminated due to PFOA (C8) emissions from DuPont’s Washington Works facility (releases had been ongoing since the 1950s). C8 reached drinking water supplies by entering the groundwater and was detected in six water supplies in 2002. From 2005 to 2013 a series of epidemiologic (exposure and health) studies were conducted on almost 70,000 individuals using questionnaires and blood samples from people who had been drinking water contaminated with PFOA. The goal of the project was to assess the “links” between PFOA (commonly referred to as C8) and a number of diseases (EPA, 2016a,b). It is important to note that these types of epidemiological studies cannot identify whether PFAS compounds actually cause an adverse health effect or disease.

The results of the epidemiological studies showed that there was no association between PFOA exposure and over 20 different health endpoints (e.g., neurological, reproductive, immune, infectious) and 18 types of cancer (e.g., blood, respiratory, GI tract, breast, etc). The studies did find a “probable link” between PFOA exposure and high blood cholesterol, ulcerative colitis, thyroid disease, testicular cancer, kidney cancer and preeclampsia (pregnancy-induced hypertension). However, upon inspection of the details of each “probable link” within the report, the associations were inconsistent and/or weak. For example, the relative risks for elevated blood cholesterol and kidney cancer never rose above an odds ratio of 2.0 (1.0 or lower would be no effect). There was an “elevated risk for testicular cancer in higher exposure area” but the findings “were based on small numbers since testicular cancer is rare.” Additionally, there were no corrections for exposure to other co-contaminants, including PFOS (generally, if diet is the route of exposure, co-contaminants will often correlate).

Since low birth weight is a consistent finding in laboratory rodents, Bach et al. (2015) reviewed 14 studies to look at the association between PFOA and/or PFOS and human fetal growth. Higher PFOS and PFOA concentrations were weakly associated with decreased average birth weight in most studies, and only a few results were statistically significant. A second study of the “Arhus Birth Cohort” (Bach et al., 2016a) did not find any strong or consistent dose-response patterns between PFAS and any routine measurements of fetal growth. Recent studies make a strong case that the “mixture” of other pollutants in the blood (e.g., arsenic, thallium, phthalates) contribute more to observations on birth weight than each single chemical alone (Lenters et al., 2015; Govarts et al., 2016). Other reviews show negative or inconsistent results on exposure outcomes with regard to female fertility, reproductive hormones or male semen quality (Bach et al., 2016b).

5.2 EFFECTS IN HUMANS – PFOS

For PFOS, there are many epidemiology studies that evaluated large cohorts of highly exposed populations, observing the association of PFOS exposure versus a variety of health endpoints. Like PFOA, the epidemiologic evidence supports an association between PFOS and increased total cholesterol. The EPA (2016b) suggests that study limitations, which included high correlation between PFOA and PFOS...
and not controlling for other persistent environmental chemicals (e.g., polybrominated diphenyl ethers), may confound the results.

Numerous epidemiologic studies evaluated thyroid hormone levels and/or thyroid disease in association with serum PFOS concentrations. The results are inconsistent and it is difficult to judge whether a subclinical change in circulating thyroid hormone levels constitute an "adverse effect." EPA (2016b) concludes that “the association between PFOS and altered thyroid hormone levels is stronger in people at risk for thyroid insufficiency or disease” and that changes in “thyroid hormones (i.e., TSH, T3 or T4) show mixed effects across cohorts.” Studies of thyroid disease and thyroid hormone concentrations in children and pregnant women also found inconsistent effects; TSH was the bioindicator most frequently associated with PFOS in studies of pregnant women.

The EPA (2016b) reports mixed results of studies that examined effects on the fetus using neonatal measurements (e.g., mean birth weight, small for gestational, head circumference, etc.). Olsen et al. (2009) recently reviewed all of the published epidemiologic literature as it pertains to the association of exposure to PFOS and PFOA with human fetal development. Eight studies were examined which focused on six general (non-occupational) and two occupational populations. The occupational studies focused on a perfluorochemical manufacturing site (Decatur, AL) with exposure categorized from work history and biomonitoring data. There were inconsistent associations reported for several different birth outcomes, including birth weight and length, head circumference, and ponderal index, among the five general population studies that measured PFOS and PFOA in the study subjects. No association with birth weight or gestational age was reported in the community drinking water study. Only one general population study examined infant Apgar scores and developmental milestones at six and 18 months of age with no associations reported.

Studies evaluating associations of PFOS with measures indicating immunosuppression are generally unremarkable. A recent article reviewing PFOA and PFOS exposure versus immunological health conditions in humans concluded that “with few, often methodologically limited studies of any particular health condition, generally inconsistent results, and an inability to exclude confounding, bias, or chance as an explanation for observed associations, the available epidemiologic evidence is insufficient to reach a conclusion about a causal relationship between exposure to PFOA and PFOS and any immune-related health condition in humans” (Chang et al., 2016).

As discussed above, associations between PFOA and/or PFOS, human fetal growth, fertility and male semen quality were weak or inconsistent. Rarely do investigators consider the effects of the “mixture” of other pollutants in the blood (e.g., arsenic, thallium, phthalates, PCBs) which may contribute more to observations on birth weight than the effect of a single chemical alone (Lenters et al., 2015; Govarts et al., 2016).

Although some human studies suggest an association with bladder, colon, and prostate cancer, the literature is inconsistent and some studies are confounded by failure to control for risk factors such as smoking (EPA, 2016b). Altogether, there is no convincing evidence in the scientific literature that exposure to PFOS or PFOA causes cancer in humans.

5.3 SUMMARY OF HUMAN STUDIES

The associations between PFAS and most epidemiology endpoints are, at best, inconsistent. Although mean serum values are presented in the human studies, actual estimates of PFAS exposure (i.e., doses/duration) are not available. For PFOA, the EPA notes that it is likely that some of the human exposures that contribute to serum PFOA values may come from “derivatives or precursors” that break down metabolically to PFOA (possibly from PFOA in diet and materials used in the home, which creates potential for confounding). In addition, most of the subjects of the epidemiology studies have other contaminants in their blood that were not factored into the ecological analyses. Although the study designs state that they adjust for other confounding factors (e.g., drinking, smoking), the presence of other contaminants constitutes a level of uncertainty that is absent in the animal studies.

For the Health Advisories, the EPA only used the human epidemiological studies as “qualitative support” as they concluded that the human epidemiology data for PFOS/PFOA do not provide adequate quantifiable dose-response information for use as the basis of deriving a drinking water advisory “because of uncertainty regarding the routes, levels and timing of exposures plus the confounding influences of other PFCs present in serum.” Despite the plethora of studies available, most authors state that “additional research is warranted” to determine if definitive epidemiological effects exist and how they are related to PFAS exposure.
6. USEPA HEALTH ADVISORIES FOR PFOA AND PFOS

In 2016, following very extensive peer-review and public comment periods, the EPA published two comprehensive drinking water health advisories for both PFOA and PFOS (USEPA, 2016a,b). These documents are supported by companion “health assessment support” documents that provide the technical basis for the derivation of both drinking water health advisories (EPA, 2016c,d). Because the chemical structure and toxicological effects on laboratory animals for both compounds was virtually identical, the final drinking water advisory for both PFOA and PFOS is estimated to be 0.070 μg/L. Thus, assuming the risk is “additive,” EPA also advised the sum of both compounds not exceed 0.070 μg/L if both are found to be present in drinking water.

6.1 EFFECTS IN LABORATORY ANIMALS – PFOA

For PFOA, there are many animal studies of acute, subchronic, and chronic duration available for multiple species including monkeys, rats and mice following oral dosing regimens. These studies report developmental effects (survival, body weight changes, reduced ossification, delays in eye opening, altered puberty, and delayed mammary gland development), liver toxicity (hypertrophy, necrosis, and effects lipid metabolism), kidney toxicity (weight), immune effects, and cancer (liver, testicular, and pancreatic). Overall, the toxicity studies available for PFOA demonstrate that the developing fetus is particularly sensitive to PFOA-induced toxicity, albeit using doses that are selected to be very high to insure an “adverse effect.”

6.2 DERIVATION OF REFERENCE DOSE - PFOA

The available endpoints in the development of the RfD including liver effects (weight changes with histopathology), body weight changes in adults and offspring, reproductive outcomes such as fertility, developmental effects (altered puberty, survival, and developmental delays) and immune effects. The candidate studies were selected based on their NOAEL and/or LOAEL values, a duration of 11 to 91 days, use of a control, and two or more doses. The average serum concentrations were then converted into an oral equivalent dose for humans by recognizing that clearance from the body equals dose to the body. In other words, the acceptable dose in humans will be lower than a weight-adjusted dose in laboratory animals because the half-life in humans is in years while the half-life in animals is in months.

The “Point of Departure” (POD) selected was a study on mice (Lau et al., 2006) where a dose of 1 mg/kg/day resulted in a “lowest observed adverse effect” (LOAEL) in the pups (a decrease in bone density and accelerated male puberty). This daily dose in mice was back-calculated using a pharmacokinetic model (using mouse blood concentrations) to obtain a “human equivalent dose” (HED) of 0.0053 mg/kg/day.

Interestingly, the HED’s calculated from other studies were all within a factor of three. Even more remarkable is that all of the RfDs were virtually the same. They were so close that one of the expert peer-reviewers stated that “Despite all of these scientifically-credible exercises and deliberations, the end result (RfD) seems to this reviewer to have been predetermined – to be extremely low.”

The RfDs from all of the candidate studies (including the POD) ranged from 0.00002 to 0.00015 mg/kg/day across multiple endpoints. The RfD of 0.00002 mg/kg/day calculated from the HED serum values from Lau et al. (2006) was selected. This RfD is derived from reduced ossification of the proximal phalanges (“fingertips”) and accelerated puberty in male pups (four days earlier than controls) as the critical effects.

6.3 PFOA DRINKING WATER HEALTH ADVISORY

A health advisory (HA) is a concentration in drinking water that is not expected to cause adverse effects based on a specified exposure duration. HAs are developed to be protective of even the most sensitive populations. HAs are informal guidance and not regulatory or legally enforceable. In addition, HAs can evolve based on new science (e.g., exposure or toxicity). Drinking Water Health Advisories are intended to inform public health officials who are wrestling with chemicals in drinking water that do not have Maximum Contaminant Levels (MCLs) or other regulatory criteria. So they can take the appropriate actions to protect their residents, those public health officials include drinking water system operators, and regulators who have the primary responsibility for overseeing these systems.

Due to perceived susceptibility of pregnant women, EPA used a drinking water intake and body weight for lactating women in the estimation of the lifetime HA for this potential critical time period for PFOA. EPA used the rate of 54 mL/kg-day, which is the 90th percentile for drinking water ingestion for lactating women. They believe the lactating woman is the more protective scenario given the increased water intake rate needed for her body weight to support
milk production. This was deemed relevant as human studies demonstrate that PFOA is transferred from mother to infant via cord blood and breast milk (a recent study by Haug et al. (2011) showed that breast milk contributed >83% of the PFOA exposure in six-month-old infants). A “relative source contribution” of 20% was also assumed for drinking water (to be protective of additional intake of PFAS from other sources).

The lifetime HA for PFOA was calculated by EPA (2016a) as follows:

A DWEL was derived from the RfD and assumes that 100% of the exposure comes from drinking water.

\[
\text{DWEL} = \frac{\text{RfD} \times \text{BW}}{\text{DWI}}
\]

Where:

- RfD = 0.00002 mg/kg/day (based on the RfD derived from Lau et al. (2006)
- DWI/BW = 0.054 L/kg-day (90th percentile for consumers only estimate of combined direct and indirect community water ingestion for lactating women)

The lifetime HA was then calculated after application of a 20% RSC as follows:

\[
\text{Lifetime HA} = \text{DWEL} \times \text{RSC}
\]

\[
= 0.000037 \text{mg/L} \times 0.2
\]

\[
= 0.000074 \text{mg/L} \text{ (rounded to 0.00007 mg/L)}
\]

\[
= 0.07 \mu g/L
\]

Because the critical effect identified for PFOA is a developmental endpoint and can potentially result from a short term exposure during a critical period of development, EPA concludes that the lifetime HA for PFOA is applicable to both short-term and chronic risk assessment scenarios. Thus, the lifetime HA of 0.07 μg/L also applies to short-term exposure scenarios (weeks to months) to PFOA in drinking water, including during pregnancy and lactation.

6.4 EFFECTS IN LABORATORY ANIMALS – PFOS

For PFOS, oral animal studies of short-term and subchronic duration are available in multiple species including monkeys, rats and mice. These studies report developmental effects (decreased body weight, survival, and increased serum glucose levels and insulin resistance in adult offspring), reproductive (mating behavior), liver toxicity (liver weight co-occurring with decreased cholesterol, hepatic steatosis), developmental neurotoxicity (altered spatial learning and memory), immune effects, and cancer (thyroid and liver). Of all of these studies, it was determined that the developing fetus is particularly sensitive to PFOS-induced toxicity. As discussed, human epidemiology data report inconsistent or weak associations between PFOS exposure and high cholesterol, thyroid disease, immune suppression, and some reproductive and developmental parameters (fertility, fecundity). Although some human studies suggest an association with bladder, colon, and prostate cancer, the literature is inconsistent and some studies are confounded by failure to control for risk factors such as smoking.

The process and equations used for the derivation of the Rfd for PFOS was the same as presented above for PFOA. The study selected as the POD was a two-generation rat study (Luebker et al., 2005), which showed a decrease in pup birth weight following a daily dose of 0.1 mg/kg/day. The average serum value in rat pups (6.26 mg/L) was converted using a pharmacokinetic model to a HED of 0.00051 mg/kg/day. This daily dose in rats was then back-calculated using a pharmacokinetic model (using the average rat serum concentrations) to obtain a HED of 0.00051 mg/kg/day. An RfD of 0.00002 mg/kg/day was calculated by dividing the HED by an uncertainty factor of 30 (a factor of three for interspecies uncertainty and a factor of 10 for human sensitivity; no factor was required to convert a subchronic dose as the NOAEL was used).

As with PFOA, because the critical effect identified for PFOS is a developmental endpoint that can result in exposure during a critical period of development, EPA concludes that the lifetime HA for PFOS is applicable to both short-term and chronic risk assessment scenarios.

6.5 PFOS DRINKING WATER HEALTH ADVISORY

Adverse effects observed following exposures to PFOA and PFOS are the same or similar, and epidemiological studies in humans have attempted to “link” exposure with effects on serum lipids, birth weight, and antibodies. At high doses, common effects on animals include the liver, neonate development, and responses to immunological challenges. Both compounds were also associated with tumors in long-term animal studies. The effects serving as the basis for the RfDs for both PFOA and PFOS are developmental endpoints (e.g., reduced ossification and accelerated puberty in males for PFOA and decreased pup birth weight for PFOS; see
USEPA 2016c, 2016d). Because the RfDs for both PFOA and PFOS are based on similar developmental effects and are essentially numerically identical, when these two chemicals co-occur at the same time and location in a drinking water source, a conservative and health-protective approach that EPA recommends would be to compare the sum of the concentrations ([PFOA] + [PFOS]) to the HA (0.07 μg/L). This drinking water advisory is protective of any carcinogenic effects as this level is below concentrations that would be known to cause cancer in humans based on derivations of cancer slope factors from animal bioassays.

EPA developed the HAs for PFOS to protect the developing fetus. As with PFOA, the RfD for PFOS was the same, so the derivation of the HA was the same, i.e., for both PFOS and PFOA, the HA is 0.07 ppb in drinking water. In addition, because the chemicals are related and are associated with the same adverse effects, EPA intended that the HA for the sum of PFOA concentration plus PFOA concentration is 0.07 ppb. The HAs are considered to be protective for all adverse effects and all populations. The HA documents for PFOS and PFOA also include steps to be taken should PFOS or PFOA be detected in a drinking water supply or system.

The HAs for PFOS and PFOA are intended to apply to all exposure durations, short or long. This is different from most HAs, which are set for a specific time duration, such as short-term versus lifetime. The reason that PFOS and PFOA are unusual in this way is that the most sensitive endpoint is developmental (low birth weight). In theory, exposure to a sufficient amount of a developmental toxicant at just the wrong moment during gestation is sufficient to cause the adverse effect.

7. PERSPECTIVE ON UNCERTAINTIES

To put these health advisory values in perspective, from a modeling standpoint, the uncertainty factor used in going from the dose of PFOA administered to the mouse (1 mg/kg/day) to the HED (0.0053 mg/kg/day) is a factor of 188. This is mainly due to the model differences in the half-life between the rodent (weeks) vs. the human (years). For PFOA, another factor of 300 is then applied to convert the HED to a “safe” concentration for lactating women (i.e., 10 for human sensitivity, three to extrapolate from animals to humans, and 10 to convert from a LOAEL to a NOAEL). In other words, to go from a LOAEL in a mouse to a NOAEL for a pregnant or post-partum woman, a factor of at least 56,400 (300 x 188) is used to derive a “safe” lifetime drinking water health advisory.

Another way to view the level of conservatism in this RfD is to compare the difference between the mouse blood concentration for the LOAEL (38 mg/L) and the average PFOA level in human blood in the U.S. (3.07 μg/L, taken from NHANES for “all ages”). The difference between the two equals a margin-of-safety of 12,400!

Finally, some additional perspective can be gained by examining the “background” levels of PFOA, PFOS and conventional “toxics” in human blood that are not anticipated to incur any adverse effects over the long term. The following graph presents, on a log scale, contaminants reported in blood serum of humans in the U.S. (references provided in Attachment A):

The graph above shows that the concentrations of potentially “toxic” contaminants in human blood ranges from the low part per trillion range (0.01 μg/L for MTBE) to the low part per million range (2.61 mg/L for formaldehyde). Formaldehyde and ammonia are natural by-products of the metabolism of dietary nutrients containing carbon (e.g. carbohydrates) and nitrogen (e.g., amino acids). BTEX compounds (benzene, toluene, ethylbenzene and xylene) enter our bodies through inhalation while driving in traffic or perhaps refueling automobiles.

What is interesting in this graphic is that the concentration of PFOA (~2 μg/L) is lower than a normal blood level for cyanide (~4 μg/L) and the concentration of PFOS (6.3 μg/L) is slightly lower than the normal blood level of lead (~11.2 μg/L). Also presented is the level of lead and cyanide in blood that would be considered “clinically elevated” (200 μg/L for cyanide and 100 μg/L for lead). The margin-of-
safety between the “level of concern” and the normal blood level for cyanide and lead are a factor of 50 and nine, respectively. Interestingly, the levels of PFOA measured in human blood from “high-exposure community” (e.g., as seen in the “C8 Study”) following exposure to contaminated groundwater would be about 10 times the level presented above (or less than 100 μg/L as cited in Olsen, 2015). Based on this analysis, and knowing that both lead and cyanide are clearly more toxic than PFOA/PFOS in terms of acute toxicity (cyanide) and chronic effects (lead), one can reason that the current levels of PFOA and PFOS in the bloodstream of average U.S. individuals presents neither a short-term nor a long-term risk. Additionally, compared to formaldehyde, which is a reactive molecule and a “known carcinogen,” the concentrations of PFOA/PFOS are two orders of magnitude lower than levels known to occur naturally in human blood.

8. ECOLOGICAL EFFECTS AND AQUATIC TOXICITY OF PFAS

As discussed above, PFOA and PFOS are fully fluorinated fatty acid analogues that are resistant to hydrolysis, photolysis, microbial degradation, and metabolism by animals, which makes them highly persistent in the environment (Giesy and Kannan, 2002). Since PFAS are chemically stabilized by strong covalent C-F bonds, they were historically considered to be metabolically inert and non-toxic (Sargent and Seffl, 1970). Recent evidence has demonstrated that, at high doses in laboratory animals, they can be biologically active and may cause peroxisomal proliferation, increased activity of fat metabolizing enzymes, and alterations in other important biochemical processes (Giesy et al. 2010). In both fish and wildlife, however, PFOS is more widely distributed and at higher concentrations in their tissues (liver, blood, eggs) than PFOA. Consequently, in the environment, PFOS is much more relevant and therefore this discussion on aquatic and ecological effects will focus on this chemical.

Laboratory bioconcentration studies with fish have demonstrated that PFOS accumulates in a time- and concentration-dependent manner and that the primary route of accumulation of PFOS is from the water; dietary sources of PFOS appear to be secondary and may not significantly enhance the overall accumulation of PFOS. As with laboratory mammals, PFOS primarily concentrates in blood, liver and kidney with much lower concentrations in gonads, fat and muscle. BCFs in the laboratory (carp, rainbow trout, blue-gill) are relatively low compared with legacy contaminants, generally ranging between 500 and 4,000 L/kg. Half-lives in fish vary widely with the species tested (typically less than 150 days) and PFOS levels decrease rapidly when moved to cleaner water (Giesy et al. 2010). In the wild, biomagnification up the food chain plays a much bigger role than direct transfer from the surface water to an aquatic organism.

In the laboratory, the toxicity of PFOS to aquatic organisms exposed via surface water is very low. Giesy et al. (2010) conducted a comprehensive literature review of both the acute and chronic effects following exposure of aquatic plants, macroinvertebrates, amphibians, fish and shellfish to PFAS, including PFOS. The authors conclude that:

- a concentration of 8.2 mg PFOS/L would be protective of aquatic plants
- a concentration of 0.78 mg PFOS/L should be protective of aquatic organisms under acute exposure scenarios
- chronic water concentrations less than or equal to 0.46 mg PFOS/L should not pose a significant adverse risk to fish or invertebrates and
- a critical body residue level for PFOS in fish tissues, calculated from a bluegill accumulation study, estimated a tissue PFOS concentration of 87 mg/kg wet weight as a “toxic threshold” value.

These levels are thousands of times higher than concentrations reported in natural waters, including water bodies close to contaminated waste sites or areas where past fire-fighting exercises have significantly increased raised environmental levels. Using very conservative methods to estimate acute and chronic criteria developed under the Great Lakes Initiative, they calculated acute and chronic levels of 21 μg/L and 5.1 μg/L, respectively. Even these levels, which they state are biased low, are still hundreds of times greater than the parts per trillion levels reported in freshwater systems.

Although invertebrates and fish appear to be safe from exposure to PFOS, biomagnification does clearly occur, although the mechanism is not via partitioning into lipid but apparently the affinity the compound has for proteins. The parts per billion levels reported in the tissues of in upper trophic level receptors still do not compare to the parts per million levels of legacy compounds like mercury, PCBs, and DDT derivatives. Swallows have always been a good receptor to study from the perspective of food chain transfer as they will nest in manmade birdhouses, will feed
locally, and their eggs are easy to collect. A recent study by Custer et al. (2014) showed a negative association between concentrations of PFOS in eggs and hatching success in tree swallows. They showed the concentration at which effects became evident (150–200 ug/kg wet weight) were apparently lower than effect levels found in laboratory feeding trials or egg-injection studies of bird types. The State of Michigan sampling of birds adjacent to the Former Wurtsmith Air Force Base in Michigan revealed some of the highest concentrations of PFAS ever recorded in tree swallows (maximum PFOS = 1220 ug/kg in eggs; 1840 ug/kg in plasma). One strong caveat of studies of birds in the wild is that other contaminants present in the birds (dioxins, PCBs, mercury, etc.) may also correlate with PFOS and so, without additional analyses and statistical interpretation, the results can be overstated if the co-contaminants, or the mixture thereof, is responsible for any negative effects on hatching success.

9. IMPLICATIONS FOR THE REGULATED COMMUNITY

What can be gleaned from the above information in terms of how issues surrounding PFOA/PFOS may affect industry and the regulated community? First, based on chemical properties and environmental fate, this “problem” will always be defined as a groundwater/drinking water issue (EPA recently released a memorandum clarifying the “appropriate application” of the HA is only for drinking water). Second, it is not anticipated that an MCL for drinking water will be promulgated for PFOA/PFOS because, nationwide, PFOA and PFOS are detected way too infrequently and, when they are detected, levels are generally observed below 70 ppt. Thirdly, economically viable treatment technologies exist for the efficient removal of low levels of PFAS from large-scale drinking water systems (e.g., Emerging Compounds Treatment Technologies). Fourth, like dioxins, it should not come as a complete surprise if researchers identify natural degradation pathways for C8 compounds (degradation of TCDD was not thought possible until the discovery of white rot fungus). Finally, using a comparative toxicity approach, it appears, based on known measurements of levels typically seen in human blood, that PFOA and PFOS would never rise to a level of clinical concern from the standpoint of both an acute or a chronic effect.

PFAS compounds, however, present a real threat from the public’s perception of risk, which have may pressure some states to promulgate enforceable drinking water standards even though a Federal MCL has not been issued (MN and VT are the only two with levels less than EPA’s HAs). Accordingly, PFAS expertise is being sought by the regulated community and the public to understand the nature and extent of PFAS contamination once identified and then to apply effective remedial technology(s). There will likely be significant academic research on all aspects of PFAS, making teaming with universities an option to move to the forefront of PFAS consulting and treatment. Accordingly, recent graduates may be valuable employees for environmental consulting firms who engage in the PFAS market.

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