PETITION TO REQUIRE HEALTH AND ENVIRONMENTAL TESTING UNDER THE TOXIC SUBSTANCES CONTROL ACT ON CERTAIN PFAS MANUFACTURED BY CHEMOURS IN FAYETTEVILLE, NORTH CAROLINA

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October 13, 2020
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EXECUTIVE SUMMARY

This petition is submitted by Center for Environmental Health, Cape Fear River Watch, Clean Cape Fear, Democracy Green, the NC Black Alliance, and Toxic Free NC. Petitioners are non-profit public health, environmental and environmental justice groups based in North Carolina. Their petition is filed under Section 21 of the Toxic Substances Control Act (TSCA). It requests that the Environmental Protection Agency (EPA) require health and environmental effects testing on 54 Per- and Polyfluoroalkyl Substances (PFAS) manufactured by The Chemours Company (Chemours) at its chemical production facility in Fayetteville, North Carolina. The petition seeks issuance of a rule or order under section 4 of TSCA compelling Chemours to fund and carry out this testing under the direction of a panel of independent scientists. As demonstrated in the petition, the 54 PFAS meet the criteria for testing in section 4(a) of TSCA.

PFAS have raised significant concern in the US and globally because of their persistence and potential to bio-accumulate, widespread presence in living organisms, products, and the environment, and demonstrated adverse health effects at low doses. EPA and many other authoritative bodies have noted the common characteristics of PFAS as a class. The Fayetteville chemical manufacturing facility, which is located on the Cape Fear River upstream of Wilmington, North Carolina, has long been a major producer and user of PFAS under the ownership of E. I. DuPont de Nemours & Company, Inc. (DuPont) and, since 2015, Chemours, a DuPont spinoff.

In the last few years, several of these PFAS have been identified in drinking water sources serving over a quarter of a million people in the Cape Fear watershed, in human blood and in environmental media, including air emissions, surface water, sediment, stormwater, groundwater and locally grown produce. Significant attention has been focused on “GenX” compounds. These chemicals have been produced as byproducts at the Fayetteville since the early 1980s. They were recently commercialized as a replacement for perfluorooctanoic acid (PFOA), a surfactant in the polymerization of fluoropolymers that was phased out in 2015 in response to serious health and environmental concerns. However, GenX compounds are only a portion of the PFAS produced at the facility which have been shown to have actual or likely human exposure and presence in the environment. Petitioners have identified a total of 54 PFAS (not including legacy substances) that are attributable to the Chemours facility and have been detected in environmental media and/or people in the Cape Fear River watershed adjacent to and downstream of the plant site. These 54 PFAS are identified in the attached Master Chemical List, with citations to the evidence demonstrating actual or likely exposure.

Under a consent order between EPA and Chemours, GenX compounds have undergone some toxicological testing but, as EPA has recognized, available studies are incomplete. There is also some testing underway on a small number of other PFAS under a North Carolina consent order, but these studies are limited in scope. No health or environmental effects testing has been conducted on the remainder of the 54 PFAS. Thus, for all 54 substances, there is an absence of sufficient data to determine risks to the large exposed population within range of the Fayetteville facility and the
surrounding ecosystem and to set risk reduction targets and other protective measures. For residents and their families, the inability to determine the health impacts of their historical, ongoing and future PFAS exposure is a deep source of anxiety and concern.

To date, EPA has failed to use its testing authorities under TSCA section 4 to fill the extensive data-gaps on PFAS. Congress included these tools in TSCA to assure that responsibility for developing information on the health and environmental impacts of chemicals is assigned to those businesses engaged in their production and commercial use. While the federal government and academic institutions have an important role to play in PFAS research, they should not and cannot shoulder the entire testing burden. A full understanding of this large and problematic chemical class will be impossible unless industry contributes its sizable resources to determining their risks to human health and the environment. The goal of this petition is to compel EPA to use its TSCA testing authorities to assure that industry assumes this responsibility.

Leading authorities have recognized that, because of the similarities in persistence, mobility, and toxicity among PFAS, all members of the class have the potential to cause the same adverse effects as well-characterized compounds such as PFOA and perfluorooctane sulfonate (PFOS). Based on the known hazards of these analogues, untested PFAS with potential for exposure would meet the criteria for testing in section 4(a)(1)(A) of TSCA because they (1) “may present an unreasonable risk of injury” and (2) have “insufficient information and experience” to determine their effects on health or the environment. Indeed, EPA took this very approach in reviewing GenX compounds under the related “new chemicals” provisions in section 5 of TSCA: it issued a consent order requiring testing based on findings that these compounds “may present an unreasonable risk” because of their similarities to PFOS and PFOA and “the information available to the Agency is insufficient to permit a reasoned evaluation of the[ir] human health and environmental effects.”

The same conclusions are required under TSCA section 4(a)(1)(A) for the 54 PFAS in the Master Chemical List based on their similarities to other well-studied PFAS, evidence of actual or likely human exposure and lack of sufficient data for informed determinations of risk. For all these substances, testing is “necessary” under section 4(a)(1)(A) of TSCA because there is no other scientifically sound and reliable method for determining their health and environmental effects. Thus, this petition asks EPA to issue a test rule or order requiring Chemours to fund studies necessary to understand the likely health and environmental risks from past and ongoing exposure to the 54 PFAS.

Some of the 54 PFAS proposed for testing are intended commercial products while others are byproducts created during the manufacture of commercial products. The testing authority in section 4 of TSCA applies to both commercial products and byproducts from a commercial chemical manufacturing process. Thus, both types of PFAS are subject to section 4 testing requirements.

The scope of testing proposed in this petition would differ depending on whether compounds fall into Tier 1 (detection in human sera, food or drinking water) or Tier 2 (significant potential for human exposure based on detection in environmental media and other evidence).

The petition proposes the following testing program:

*Experimental Animal Studies*
• Compounds in both Tiers would undergo 28-day repeated dose rodent toxicology studies coupled with reproductive and developmental toxicity screening assays, examining critical PFAS endpoints including hormone disruption, liver and kidney damage, developmental and reproductive harm, changes in serum lipid levels, and immune system toxicity.

• These studies would also be conducted on three mixtures of PFAS representative of the groups of substances to which residents have been exposed through drinking water, human sera and other pathways.

• Multigeneration or extended one-generation and 2-year rodent carcinogenicity studies would be conducted on the 14 Tier 1 substances in recognition of the evidence of direct and substantial human exposure and the concerns for these endpoints demonstrated by other PFAS.

• Most studies would be carried out in two species (mice and rats) and by oral routes of administration, except inhalation would be used for volatile chemicals.

• Toxicokinetic studies would be conducted to characterize relationships between serum concentrations and dermal, oral and inhalation exposures in the test species, and to evaluate biological half-life and potential for bioaccumulation.

• Testing requirements would be based on EPA and OECD guidelines, with appropriate adjustments to reflect sensitive endpoints that have been reported for PFOA, PFOS, and GenX.

_Human Studies_

• A human health study for the Cape Fear watershed would be conducted using a similar study design to that used for the Parkersburg, WV PFOA (C8) study. The goal of the study would be to determine the relationship between exposure to the mixtures of PFAS that characterize current and historical exposure in the Cape Fear watershed and health outcomes among exposed populations.

• Testing would also be performed to determine human half-lives of the listed chemicals through longitudinal biomonitoring and exposure estimation in workers.

_Ecological Effects/Fate and Transport and Physical-Chemical Properties Studies_

• Testing would include ecological effects studies, similar to studies conducted on GenX.

• EPA would require development of analytical standards where not currently available, physical-chemical properties tests, and fate and transport studies in order to identify and predict exposures.

_Avoidance of Duplication_

• Chemours would not be required to repeat studies already conducted or in progress on GenX and the five substances subject to the North Carolina consent order but, because these studies are insufficient, additional studies on these PFAS would be conducted.
Independent Science Panel

- To maximize the credibility and objectivity of the data and key findings, EPA would contract with the National Academy of Sciences (NAS) to form an independent expert science panel with responsibility for overseeing all aspects of the testing program. The public and Chemours would have the opportunity to submit nominations for membership on the panel.

Chemours is taking steps to control environmental releases of PFAS under the consent order issued by the State of North Carolina in February 2019. These measures are critical to reduce human exposure to the 54 PFAS and should not be delayed while the testing proposed by this petition is underway. At the same time, it is important to recognize that Chemours’ actions to reduce exposure do not eliminate the need for testing because PFAS exposure is continuing despite these actions and understanding the health impacts of both ongoing and historical exposure remains essential to protect exposed communities in the Cape Fear Watershed.

The body of this petition –

- describes the petitioning organizations and their concerns about PFAS exposure;
- reviews relevant TSCA provisions supporting the petition;
- provides an overview of health and environmental impacts of PFAS as a chemical class;
- identifies the 54 PFAS covered by the petition and the basis for their selection;
- describes the rationale for requiring testing of these substances under section 4 of TSCA;
- outlines the proposed framework for testing and specific studies to be required; and
- addresses how the test rule or order would be structured to maximize the relevance, quality and independence of the testing conducted.

I. CONCERNS OF THE PETITIONERS ABOUT PFAS EXPOSURE IN THE CAPE FEAR WATERSHED

The six petitioners are grassroots non-profit organizations committed to protecting North Carolina communities and ecosystems from the threat of toxic pollution. They are deeply concerned about the contamination of the Cape Fear River and resulting harm to human health from PFAS released into the environment by the Chemours Fayetteville chemical manufacturing facility.

More details on the goals and concerns of the petitioners are presented below.

- The Center for Environmental Health is a non-profit organization working to protect children and families from harmful chemicals in air, food, water and in everyday products. Its vision and mission are a world where everyone lives, works, learns and plays in a healthy environment; we protect people from toxic chemicals by working with communities, businesses, and the government to demand and support business practices that are safe for human health and the environment. CEH is headquartered in
Oakland, California and has offices in North Carolina, where it works closely with local groups on toxic pollution threats to North Carolina citizens.

➢ **Cape Fear River Watch** is a grassroots environmental nonprofit based in Wilmington, North Carolina whose mission is to protect and improve the water quality of the Cape Fear River Basin for all people through education, advocacy and action. Since our founding, over 25 years ago, we have worked on a wide variety of water quality issues – educating and organizing our community to take action, partnering with researchers, influencing decision makers, and holding polluters accountable. Since learning of the nearly four decades of PFAS contamination of our river, the drinking water supply for about 300,000 people, and a vital ecological and economical resource to our region, Cape Fear River Watch, in partnership with academia and the Southern Environmental Law Center, has worked to stop the source of pollution, understand and explain the impacts to human health and the ecosystem, and ensure that those responsible are held accountable.

➢ **Clean Cape Fear** is an all volunteer, grassroots community group based in the Wilmington, NC area. We're educators, environmentalists, doctors, faith leaders, scientists, veterans, and concerned residents all working together to hold Chemours/DuPont accountable for decades of pollution. We formed shortly after learning toxic chemicals linked to cancer and other serious health problems were detected in our finished tap water. These discharges and air emissions impact five counties with over 300,000 residents still drinking contaminated tap water downstream from Chemours and over 3,500+ well owners with contaminated groundwater near the Fayetteville, NC area. Chemours and DuPont did this for nearly 40 years until a local journalist alerted the public in 2017.

➢ **Democracy Green** is an organization created and run by native North Carolinians-of-color to address the systemic impacts burdening our most climate impacted and disenfranchised communities across North Carolina. We work in partnership with communities, groups and organizations across the historic U.S. South, in addition to areas hailing the descendants of U.S. chattel slavery and Indigenous sovereign nations. We have seen the horrific damage to communities wrought by *Per- and Polyfluoroalkyl Substances* on North Carolinians and we cannot stand idly by while the corporations responsible are not held accountable. Democracy Green stands against corporate polluters and the harmful impact of their pollutants and chemicals on frontline communities and low-wealth populations.

➢ **The NC Black Alliance** is working toward state-level systemic change by strengthening the network of elected officials representing communities of color throughout the state and collaborating with a progressive, grassroots networks on intersecting issues. We know, oftentimes the same communities impacted by climate disasters are the same
neighborhoods facing the direct impact of health disparities created by exposure to dangerous chemicals, such as *Per- and Polyfluoroalkyl Substances*. We believe all people have the right to clean air, clean water, access to health care, and a thriving economy.

➢ **Toxic Free NC** advances environmental health and justice in North Carolina by advocating for safe alternatives to harmful pesticides and chemicals. Founded in 1986, the organization has played a leading role in state-wide pesticide reform and has contributed to national efforts strengthening regulatory protections to protect vulnerable communities and the environment from petrochemical pollution. PFAS contamination is at the nexus of clean water concerns in North Carolina. Three years ago, we learned that GenX, an industrial PFAS chemical, was dumped for years into the Cape Fear waterway by the polluting Chemours company. While high levels of PFAS have been detected in drinking water across the state, the full health impact on the exposed residents of North Carolina is still unknown. Together with other organizations in North Carolina, Toxic Free NC advocates for regulatory solutions to prevent further PFAS discharge into our environment and cleanup the PFAS already present. We represent thousands of North Carolina residents whose drinking water has been contaminated and are deeply concerned about the consequences for their health.

II. **KEY TSCA AUTHORITIES SUPPORTING THE PETITION**

A. **Testing Rules and Orders Under TSCA Section 4**

As stated in section 2(b)(1), a core policy of TSCA is that “adequate information should be developed with respect to the effect of chemical substances and mixtures on health and the environment and that the development of this information should be the responsibility of those who manufacture and those who process such chemical substances and mixtures.” This policy is embodied in section 4 of TSCA, which provides EPA with broad authority to require industry to test its chemicals to determine their risks to human health and the environment. Recognizing the need for more testing to support chemical risk determinations, the 2016 TSCA amendments streamline section 4 by authorizing EPA to issue orders in addition to rules requiring development of data.

Of most relevance to this petition, section 4(a)(1)(A)(i) authorizes EPA to require testing where it determines that—

the manufacture, distribution in commerce, processing, use, or disposal of a chemical substance or mixture, or that any combination of such activities, may present an unreasonable risk of injury to health or the environment (emphasis added).

Since the purpose of testing is to inform the assessment of a chemical’s risks, the standard for a “may present” finding is a low one. In *Chemical Manufacturers Association v. U.S. Environmental Protection Agency*, 859 F.2d 977 (1988), the DC Circuit concluded that “[b]oth the wording and structure of TSCA reveal that Congress did not expect that EPA would have to document to a certainty the existence of an ‘unreasonable risk’ before it could require testing.” It added that TSCA’s legislative
history demonstrates that “the word ‘may’ in section 4 was intended to focus the Agency’s attention on chemical substances ‘about which there is a basis for concern, but about which there is inadequate information to reasonably predict or determine the effects of the substance or mixture on health or the environment.”

The DC Circuit acknowledged that “Congress did not intend to authorize EPA to issue test rules on the basis of mere hunches” but stressed that EPA need not demonstrate that exposure or toxicity is “probable.” Instead, EPA may “rely on inferences in issuing a section 4 test rule, so long as all the evidence . . . indicates a more-than-theoretical probability of exposure.” Inferences can also support findings of potential toxicity; this can include toxicity data on chemical analogs since “Congress explicitly contemplated that EPA would base test rules on comparisons among structurally similar chemicals.” Indeed, EPA has repeatedly used Structure Activity Relationships (SAR) to support “may present” findings under both section 4 and the parallel new chemical review provisions of section 5.

In addition to a “may present” finding, section 4(a)(1)(A)(i) directs EPA to make two further determinations before requiring testing: (1) there is “insufficient information and experience” with which the chemical’s effects on health and the environment “can reasonably be determined or predicted”; and (2) testing is “necessary to develop such information.” The first determination will be justified whenever data either do not exist or are inadequate to support scientifically supportable conclusions about the chemical’s adverse effects. The second determination will be warranted where EPA concludes that testing is the only way to obtain sufficient information about these effects and that such information cannot be derived from other sources.

Once EPA makes these findings, it must require that testing be conducted “to develop information with respect to the health and environmental effects for which there is an insufficiency of information and experience” and which are “relevant to a determination” whether the substance “does or does not present an unreasonable risk to health and the environment.”

Under section 4(b)(2)(A), a broad range of studies may be required under test rules or orders. These may include studies to determine “carcinogenesis, mutagenesis, teratogenesis, behavioral disorders, cumulative or synergistic effects, and any other effect which may present an unreasonable risk of injury to health or the environment.” Studies to be conducted may include “epidemiologic studies, serial or tiered testing, in vitro tests, and whole animal tests.” The rule or order can also require development of information “for the assessment of exposure or exposure potential to humans or the environment.”

Under section 4(b)(3), testing rules or orders must place responsibility for developing the required data on the entities who manufacture and/or process the chemical to be tested. Section 4(b)(1) provides that the rule or order must prescribe the “protocols and methodologies” for conducting testing and procedures and deadlines for submitting interim and final test results. These requirements are enforceable under TSCA and non-compliance may give rise to civil and criminal penalties under section 16 and specific enforcement under section 17.

Testing under TSCA section 4 can be required on chemicals produced for intentional use or as byproducts during a commercial chemical manufacturing operation. EPA defines “byproduct” under TSCA as “any chemical substance or mixture produced without a separate commercial intent during
the manufacture, processing, use, or disposal of another chemical substance or mixture.” 40 CFR § 712.3(a).

B. Recognition of Chemical Categories under TSCA 26(c)

Section 26(c)(1) of TSCA authorizes EPA to treat a group of chemical substances as a “category” under section 4 and other TSCA provisions. If the Agency designates chemicals as a “category,” testing or other requirements prescribed by EPA would apply to each substance in the category. Under section 26(b)(2), “category” treatment is warranted if chemicals are “similar in molecular structure, in physical, chemical or biological properties, or in mode of entrance into the human body or into the environment” or “in some other way are suitable for classification as such for purposes of this Act.”

The TSCA authority to address “categories” could be applied to all PFAS or to subgroups, such as the PFAS manufactured at the Chemours Fayetteville facility. Uniform testing or other requirements could then be applied to all PFAS in the category.

C. Citizens’ Petitions under TSCA Section 21

This petition is filed under the authority of section 21 of TSCA, which enables “any person” to petition EPA to issue a rule or order requiring testing under section 4. The petition must demonstrate why the rule or order is “necessary.” EPA must grant or deny the petition within 90 days. If EPA grants the petition, it must promptly begin the process to require testing by rule or order. If it denies the petition, EPA must publish a Federal Register notice explaining the reasons for the denial.

Where EPA denies the petition for a testing rule or order in whole or in part or fails to act in 90 days, the petitioner may file suit in a US District Court to challenge the petition denial or lack of action. The petitioner is entitled to a de novo proceeding on the merits of its petition, and the Court must direct EPA to initiate a proceeding to develop a test rule or order if it concludes, based on a preponderance of the evidence, that the chemical meets the criteria for requiring testing under section 4(a)(1)(A) of TSCA.

III. SERIOUS HEALTH AND ENVIRONMENTAL CONCERNS PRESENTED BY PFAS AS A CLASS

A. Production and Use

PFAS have a unique set of properties with an unusual ability to cause serious and widespread harm to public health and the environment. A defining feature of PFAS is their carbon-fluorine bonds, which impart high thermal stability and resistance to degradation. Because of their pronounced ability to repel oil and water, PFAS have been used in a variety of industries in the US and around the globe. They function as surfactants, friction reducers, and repellents of water, dirt, and oil. As a result, they are added to consumer products to impart nonstick (waterproof, greaseproof, and stainproof) and low-friction characteristics. Examples of products that contain or are coated with PFAS include carpets, glass, paper, clothing and other textiles, plastic articles, cookware, food packaging, electronics, and personal care products. PFAS also have many industrial applications, including as dispersants and emulsifiers, membranes, and firefighting foams.
EPA’s 2019 PFAS Action Plan estimates that over 4,000 PFAS have been manufactured and used in a variety of industries and products worldwide since the 1940s and that over 1,000 PFAS are listed on the TSCA inventory (600 of which were reported for the Active Substance Inventory). These estimates do not reflect the large number of PFAS produced as byproducts and transformation products, which are not reportable for the TSCA Inventory but contribute to human exposure and environmental release.

The EPA Action Plan identifies numerous human exposure pathways for PFAS, including:

- Drinking water from public water and private water systems, typically localized and associated with a release from a specific facility (e.g., manufacturer, processor, landfill, wastewater treatment, or facilities using PFAS-containing firefighting foams);
- Consumption of plants and meat from animals, including fish that have accumulated PFAS;
- Consumption of food that came into contact with PFAS-containing products (e.g., some microwaveable popcorn bags and grease-resistant papers);
- Use of, living with, or otherwise being exposed to commercial household products and indoor dust containing PFAS, including stain- and water-repellent textiles (including carpet, clothing and footwear), nonstick products (e.g., cookware), polishes, waxes, paints, and cleaning products;
- Employment in a workplace that produces or uses PFAS, including chemical production facilities or utilizing industries (e.g., chromium electroplating, electronics manufacturing, or oil recovery); and
- In utero fetal exposure and early childhood exposure via breastmilk from mothers exposed to PFAS.

B. Adverse Effects

PFAS are often called “forever” chemicals because they do not break down or degrade over time and therefore are highly persistent. Thus, they build up in the natural environment and in biological systems if they are bioaccumulative. These characteristics, combined with the high mobility of many PFAS, have resulted in their widespread distribution and pervasive presence both in environmental media and in people and wildlife around the globe, including many remote locations. Thus, PFAS have been detected in the blood of workers and the general population, with 99 percent of those sampled showing detectable levels of these compounds. This PFAS body burden is a function of multiple exposure pathways, including air emissions, food and water consumption, consumer products like carpet or clothing and house dust. Because of their resistance to degradation, there is no known safe method of disposal of PFAS that would prevent build-up in the environment at the end of their useful lives.

In addition to their persistence, the threat posed by production and use of PFAS is amplified by their high mobility, especially in water. Their high water solubility and environmental persistence together make PFAS a ubiquitous pollutant of surface and groundwater. As a result, PFAS-contaminated drinking water is a widespread threat across the US; a growing number of drinking water suppliers have detected PFAS in source water or tap water, raising concerns about drinking water safety and resulting in use of costly treatment systems in numerous communities across the country.
Animal studies demonstrate that PFAS are linked to many serious health effects, including cancer, hormone disruption, liver and kidney damage, developmental and reproductive harm, changes in serum lipid levels, and immunotoxicity, often at low doses. Human studies of populations with elevated blood levels of PFAS have shown associations with a variety of health conditions, including kidney and testicular cancer, elevated cholesterol, liver disease, decreased fertility, thyroid problems and changes in hormone levels and immune systems. Moreover, concurrent exposure to multiple PFAS may have additive or synergistic effects.

C. Transition to Short-chain and Other Replacement Chemistries

Initially, health and environmental concerns about PFAS were focused on “long-chain” substances, consisting of perfluoroalkylcarboxylic acids (PFCA) and perfluoroalkanesulfonic acids (PFSA) with six or more fluorinated carbons. Within these chemical classes, PFOA and PFOS (along with their precursors and degradation products) received the greatest attention because of their commercial prominence and widespread detection in people and the environment. As scientific and regulatory concerns surfaced, 3M Corporation stopped producing PFOA and PFOS in the early 2000s, and DuPont and other producers agreed in 2006 to phase out PFOA and other PFCA compounds by 2015.6

The shift away from long-chain PFCA and PFSA was accompanied by the increased use of shorter-chain substances as substitutes in many historical PFAS applications. Prominent short-chain substitutes include perfluorobutanoic acid (PFBA), perfluorobutanesulfonic acid (PFBS), perfluorohexanoic acid (PFHxA), and GenX. There has also been substitution of long-chain per- and polyfluoroalkyl ether carboxylic acids (PFECAs) as well as per- and polyfluoroalkyl sulfonic acids (PFESAs). Although initially assumed to be less harmful, growing evidence indicates that short-chain PFAS, PFECAs, and PFESAs have characteristics similar to those of PFOS, PFOA and other long-chain PFAS. In its PFAS Action Plan EPA recognized that, although the “toxicities of short-chain PFAS have generally been less thoroughly studied,” they are “as persistent in the environment as their longer-chain analogues and are highly mobile in soil and water.”7 Moreover, as production has increased, short-chain PFAS, PFECAs, and PFESAs have been found in human sera and the environment and the limited testing conducted has demonstrated health effects common to long-chain PFAS, including hepatic and renal effects, suppressed immune function and changes to liver weight, serum cholesterol, and thyroid hormones.3,4

IV. PFAS PRODUCTION AT THE CHEMOURS FAYETTEVILLE FACILITY AND CONTAMINATION OF THE CAPE FEAR WATERSHED

The Chemours plant is located on a 2,150-acre site in a rural area south of Fayetteville, North Carolina, adjacent to the west bank of the Cape Fear River. The river continues for over 110 km to the City of Wilmington and then broadens into an estuary that ultimately flows into the Atlantic Ocean. Residents of Wilmington and other population centers downstream from the facility use the river as a source of drinking water. The facility was built and operated by DuPont and started producing fluoropolymers in 1971. In 2015, DuPont spun off its performance chemicals business to Chemours, a newly created company which then acquired the Fayetteville plant and other former DuPont facilities.

The plant is a major producer and user of PFAS. Its PFAS-based product lines are Fluoromonomers, Fluorinated Vinyl Ethers and Nafion® Polymers, which are used as membranes in fuel cells and chlorine production.8 The mix of precursors, byproducts, degradation products and commercial substances associated with these product lines is complex and not well-understood but likely involves hundreds if
not thousands of individual PFAS, many of which have chemical structures that are as yet unidentified. A chart describing the major chemical manufacturing streams at the facility is provided in Attachment 1.

The Fayetteville facility began producing PFOA in 2001 following the 3M phaseout, but as concerns about PFOA mounted, efforts were undertaken to find a shorter-chain PFAS that could replace PFOA as a processing aid (surfactant) in manufacture of Teflon fluoropolymers. The result was commercialization of a group of compounds with the GenX tradename. According to the North Carolina consent order, these chemicals consist of the C3 Dimer Acid (also known as Hexafluoropropylene Oxide (HFPO) Dimer Acid), CAS No. 13252-13-6; the C3 Dimer Acid Fluoride (also known as HFPO Dimer Acid Fluoride), CAS No. 2062-98-8; and the C3 Dimer Acid Ammonium Salt (also known as HFPO Dimer Acid Ammonium Salt), CAS No. 62037-80-3. (HFPO is also a building block chemical for several other PFAS-based products manufactured at the facility and is sold commercially).

GenX compounds have been produced as byproducts of the Nafion product line since 1980. However, anticipating their introduction as a commercial product, in 2008, DuPont filed two premanufacture notices (PMNs) for these chemicals under section 5 of TSCA (P-08-0508 and P-08-0509). Based on similarities between GenX and PFOS and PFOA, EPA and DuPont entered into a consent order in January 2009 requiring toxicological testing of GenX and limitations on production, worker exposure and environmental release. With the consent order in place, DuPont began commercial production of GenX at the Fayetteville plant, touting it as a “sustainable replacement” for PFOA. GenX produced in North Carolina was supplied to the Dupont fluoropolymer manufacturing facility in Parkersburg, West Virginia, a site where prior use of PFOA had previously resulted in widespread contamination of drinking water.

The 2009 EPA consent order restricted discharges of wastewater containing GenX compounds but this restriction did not apply when these chemicals were produced as byproducts in other plant operations. GenX chemicals were detected in the Cape Fear River and its tributaries in the summer of 2012 and follow-up sampling in 2013 identified significant levels of GenX in source water at drinking water treatment plants using surface water from the river. Subsequent sampling showed that conventional and advanced water treatment processes did not measurably reduce GenX concentrations in finished water. GenX has also been detected in tap water in Louisville, Kentucky, in drinking water near Chemours plant in Parkersburg, West Virginia, and near a Chemours production facility in the Netherlands. GenX contamination has spread globally, with these compounds now being detected in the Arctic Ocean. GenX was detected in fish in the Cape Fear Drainage Basin as early as 2007. Despite the attention it has received, GenX is only one of the PFAS that have been detected in environmental media in the Cape Fear watershed and may not be the contaminant of greatest concern. During the initial work of Strynar et al. and Sun et al., nine other PFAS were identified in the Cape Fear River and drinking water downstream of the Fayetteville plant. In some cases, concentrations of these PFAS were above those of GenX. In further sampling of the river downstream of the plant, McCord et al. (2019) found 37 unique PFAS molecules. Based on their structures, the detected PFAS were grouped into three categories corresponding to distinct segments of the plant’s operations (fluoromonomer, Nafion membrane and fluorinated vinyl ether production). Several of these compounds were also detected in the blood of residents of the Cape Fear region, confirming human exposure. Sampling in the Cape fear River indicated that total PFAS concentrations (all substances combined) were 130,000 parts per trillion (ppt).
As concern increased about surface water and drinking water contamination, monitoring of other environmental media for the presence of PFAS produced at the Fayetteville plant was initiated. As determined in Chemours’ own compliance testing under the North Carolina consent order, several additional PFAS associated with the Fayetteville Works facility have been detected in private wells, wastewater, stormwater, sediment, groundwater, soil, air emissions, and local produce, including a large number of compounds of uncertain chemical composition.

The 2019 consent order between Chemours and the North Carolina Department of Environmental Quality (DEQ) requires controls on wastewater discharges and air emissions of PFAS, directs Chemours to identify constituents of wastewater and process streams and to conduct environmental monitoring, provides for groundwater remediation, and requires health and environmental effects testing of five PFAS. Sampling of drinking water systems and private wells since the order was issued indicates the continuing presence of GenX and several other PFAS.

V. SELECTION OF PFAS FOR HEALTH AND ENVIRONMENTAL EFFECTS TESTING

As described above, multiple PFAS linked to Chemours operations have been found in human blood, drinking water and/or other environmental media downstream of the Fayetteville plant. These chemicals warrant health and environmental effects studies because data on their effects are insufficient or unavailable and they may present unreasonable risks because of the combination of potential toxicity and exposure. As a foundation for testing to fill these data gaps, petitioners have developed a list of 54 PFAS produced at the plant for which there is evidence of known or anticipated human exposure as demonstrated by available data on their presence in human sera, drinking water, surface water, air emissions, rainwater, private wells, groundwater and produce. The list was divided into Tier 1 substances (for which there is known human exposure based on detection in blood, food or drinking water) and Tier 2 substances (for which human exposure is probable based on detection in environmental media). The detailed justification for assigning substances to these Tiers is provided in two related spreadsheets in Attachment 2, the Chemours PFAS Master Testing List. The 54 testing candidates are summarized below in Table 1:

<table>
<thead>
<tr>
<th>#</th>
<th>Abbreviation</th>
<th>Name</th>
<th>DTXSID</th>
<th>Tier</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>HFPO-DA (GenX)</td>
<td>perfluoro-2-propoxypropanoic acid (related to GenX: ammonium perfluoro-2-methyl-3-oxahexanoate 62037-80-3</td>
<td>DTXSID70880215</td>
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<td>2</td>
<td>PFO4DA</td>
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<td>3</td>
<td>PFO5DoDA (aka TAF)</td>
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</tr>
<tr>
<td></td>
<td>Name</td>
<td>Description</td>
<td>Code</td>
<td></td>
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<tr>
<td>4</td>
<td>Nafion byproduct 2</td>
<td>2-[1-[difluoro(1,2,2,2-tetrafluoroethoxy)methyl]-1,2,2,2-tetrafluoroethoxy]-1,1,2,2-tetrafluoroethanesulfonic acid</td>
<td>DTXSID10892352</td>
<td>1</td>
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<tr>
<td>5</td>
<td>Hydro-EVE acid</td>
<td>3-[1-[difluoro(1,2,2-tetrafluoroethoxy)methyl]-1,2,2-tetrafluoroethoxy]-2,2,3,3-tetrafluoro-propanoic acid</td>
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<tr>
<td>6</td>
<td>Nafion byproduct 1</td>
<td>1,1,2,2-tetrafluoro-2-[(1,1,1,2,3,3-hexafluoro-3-[(1,2,2-trifluoroethenyl)oxy]propan-2-yl]oxy]ethane-1-sulfonic acid</td>
<td>DTXSID30892354</td>
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<td>PFO2HxA</td>
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<td>PFMOPrA</td>
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<td>11</td>
<td>NaDONA</td>
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<td>12</td>
<td>PFMOBA</td>
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<td>PEPA</td>
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<td>N1AF</td>
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<td>PEVE</td>
<td>pentafluoroethyl trifluorovinyl ether</td>
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<td>PES</td>
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<td>TFE</td>
<td>tetrafluoroethylene</td>
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<td>(was MMF)</td>
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<td>PSEPVE</td>
<td>perfluoro (4-methyl-3, 6-dioxaoct-7-ene)sulfonyl fluoride</td>
<td><a href="#">DTXSID3044596</a></td>
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<td>24</td>
<td>PPVE</td>
<td>heptafluoropropyl trifluorovinyl ether</td>
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<td>25</td>
<td>PEPF</td>
<td>2,3,3,3-tetrafluoro-2-(1,1,2,2,2-pentafluoroethoxy)propanoyl fluoride (aka perfluoroethoxypropionyl fluoride)</td>
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<td>26</td>
<td>HFPO-DAF</td>
<td>2,3,3,3-tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)propanoyl fluoride</td>
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<td>fluoroether E2</td>
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<td>fluoroether E3</td>
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<td>n-perfluorobutane</td>
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<td>34</td>
<td>MA (?)</td>
<td>tetrafluoro-2-[tetrafluoro-2-(fluorosulfonyl)ethoxy]-propanoylfluoride</td>
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<td>8-fluorosulfonylperfluoro(2,5-dimethyl-3,6-dioxoanoyl) fluoride</td>
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<td>DFSA</td>
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<td>39</td>
<td>HFPO</td>
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<tr>
<td>40</td>
<td>EVE</td>
<td>methyl perfluoro(3-(1-ethenloxypropan-2-yloxy)propanoate)</td>
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<td>41</td>
<td>RSU</td>
<td>2,2-difluoro-2-(fluorosulfonyl) acetyl fluoride</td>
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<td>42</td>
<td>MMF</td>
<td>2-fluoro-2-methylpropanediol difluoride (aka methyl-2, 2-difluoromalonyl fluoride or 2-fluoro-2-methylpropanediol difluoride)</td>
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<td>43</td>
<td>MAE</td>
<td>methylperfluoro(5- (fluoroformyl)-4-oxahexanoate)</td>
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<tr>
<td>44</td>
<td>DAE</td>
<td>methyl perfluoro(8-(fluoroformyl)-5-methyl- 4 7-dioxanonanoate</td>
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<td>45</td>
<td>SU</td>
<td>2- hydroxytetrafluoroethane sulfonic acid sultone</td>
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<td>46</td>
<td>NVHOS</td>
<td>1,1,2,2-tetrafluoro-2-(1,2,2,2-tetrafluoro-ethoxy)ethane sulfonate</td>
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<td>47</td>
<td>MTP</td>
<td>2,2,3,3-tetrafluoro-3-methoxypropanoic acid</td>
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<td>48</td>
<td>Byproduct 4</td>
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<td>49</td>
<td>Byproduct 5</td>
<td>2-fluoro-2-{1,1,2,3,3,3-hexafluoro-2-(1,1,2,2-tetrafluoro-2-sulfoethoxy)propoxy}acetic acid</td>
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<td>2</td>
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<tr>
<td>50</td>
<td>Byproduct 6</td>
<td>1,1,2,2-tetrafluoro-2-[(1,1,1,2,3,3,4,4-octafluorobutan-2-yl)oxy]ethyl-1-sulfonic acid</td>
<td>-</td>
<td>2</td>
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</tbody>
</table>
Acid 1,1,2,2-tetrafluoro-2-[(1,1,1,2,3,3,4,4-octafluorobutan-2-yl)oxy]ethane-1-sulfonic acid

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>51</td>
<td>EVE Acid</td>
<td>2,2,3,3-tetrafluoro-3-[1,1,1,2,3,3-hexafluoro-3-(1,2,2-trifluoroethoxy)propan-2-yl]oxypropanoic acid</td>
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<td>52</td>
<td>R-EVE</td>
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</tr>
<tr>
<td>53</td>
<td>PFECA B</td>
<td>Perfluoro-3,6-dioxoheptanoic acid</td>
</tr>
<tr>
<td>54</td>
<td>PFECA G</td>
<td>4-(Heptafluoroisopropoxy)hexafluorobutanoic acid</td>
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</tbody>
</table>

In addition to these 54 compounds, there are numerous PFAS with likely human exposure associated with the Fayetteville facility that cannot be tested at the present time because they lack adequate chemical characterization. For example, a recent report of sampling conducted by Chemours under the North Carolina consent order identified 21 “unknown” PFAS present in General Facility Discharge samples and 250 “unknown PFAS” present in Chemours Process Wastewater samples, for a total of 257 potential unique “unknown” PFAS (14 unknown PFAS were present in both types of samples).\(^{39}\) As the report explains, “the compounds are considered to be unknown because the analytical method has not been calibrated for them (for example, because authentic standards do not exist)” but there is sufficient information to determine that the unknown compounds are PFAS. This non-targeted analysis was conducted to “develop standards and methods to facilitate the quantitative analysis of” the unknown PFAS. As standards are developed and these PFAS can be fully identified, they may also warrant testing based on their presence in discharges from the Chemours facility.

VI. RATIONALE UNDER TSCA FOR REQUIRING TESTING ON THE 54 PFAS

A. Present an Unreasonable Risk of Injury to Health or the Environment

As shown below, all of the 54 PFAS individually meet the criteria in TSCA section 4(a)(1)(A) for requiring testing because (1) they may present an unreasonable risk of injury to health or the environment, (2) they lack sufficient data to determine their adverse health and environmental effects and risks of injury, and (3) testing is necessary to provide sufficient information for these determinations. In addition, it is likely that some PFAS activate similar biological processes and have additive or other interactive effects when there is concurrent exposure through drinking water or other pathways. These PFAS mixtures would likewise meet the TSCA testing criteria based on their potential for unreasonable risk and the insufficiency of available data on their effects on human health.

As described above, the “may present” standard in section 4 is not difficult to satisfy. The focus is on whether there is a reasonable basis for concern about a chemical, not whether there is certainty about
its risks to health or the environment. Neither hazard nor exposure need be conclusively demonstrated. It is only necessary to show that the possibility of both is more than theoretical; this can be established by inference rather than direct evidence. Thus, testing will be warranted where (1) data or structure-activity analysis indicate potential adverse effects and (2) there is reason to anticipate exposure to the chemical by people or the environment. For groups of chemicals that qualify as a “category” under section 26(c) because of similarities in chemical structure and/or toxicity, these determinations need not be made for every individual substance but can be based on the common characteristics of the class.

1. **Health Effects of PFAS as a Class**

Available data on the PFAS class demonstrate a range of serious health effects in people and animals, including cancer, hormone disruption, liver and kidney damage, developmental and reproductive harm, changes in serum lipid levels, and immune system toxicity, some of which occur at extremely low levels of exposure. Consistent evidence of these health effects has been observed in experimental animals as well as in people exposed at work or from drinking PFAS-contaminated water or in the general population, and this consistency increases confidence in a causal relationship between PFAS and health effects in humans.

PFOA and PFOS are two specific PFAS that have been studied most intensively. However a similar spectrum of health effects are associated with several other chemicals within the PFAS class, such as GenX, PFHxS, PFHxA, PFBuS, PFBuA, and PFNA. These effects have been observed consistently in both short-chain and long-chain substances and should be presumed to be of concern for all PFAS. Thus, recent EPA and other governmental toxicity assessments and health advisories for PFOS, PFOA, GenX and PFBS identify similar health effect concerns over a range of PFAS compositions.

Liver toxicity is the most common effect observed across the PFAS family, in both humans and experimental animals, likely because it is commonly measured. For example, a study in China found altered liver function markers in people with exposure to PFOA, PFOS, and PFNA and abnormal prealbumin associated with exposures to PFPeA, PFHxA, PFNA, PFDoDA, PFTrDA and PFTeDA. Similarly, EPA’s toxicity evaluation for GenX selected liver effects as the basis for the assessment, and also noted that other effects observed included kidney toxicity, immunological effects, developmental effects, and cancer, similar to PFOA, PFOS, and other PFAS that have been studied.

As another example of consistent effects across the PFAS class, in a recent review article about PFAS chemicals, the US National Toxicology Program (NTP) concluded that findings of suppressed vaccine response in humans and T cell-dependent antibody response in experimental animals warranted classifying PFOA and PFOS as presumed immune hazards to humans. In a recent draft toxicological profile, the ATSDR extended this finding to PFHxS and perfluorodecanoic acid (PFDeA), identifying all four compounds as suppressants of antibody response in humans.

PFOA and GenX have been shown to cause cancers in experimental animals, particularly in the pancreas, and a large study of people exposed to contaminated drinking water near the DuPont facility in Parkersburg WV also found elevated rates of two cancers. A recent review of the carcinogenic potential of PFAS as a class concluded that: “The most well-studied member of the PFAS class, perfluorooctanoic acid (PFOA), induces tumors in animal bioassays and has been associated with elevated risk of cancer in human populations. GenX, one of the PFOA replacement chemicals, induces tumors in animal bioassays as well. Using the Key Characteristics of Carcinogens framework for cancer hazard identification, we considered the existing epidemiological, toxicological and mechanistic data for 26 different PFAS. We
found strong evidence that multiple PFAS induce oxidative stress, are immunosuppressive, and modulate receptor-mediated effects."

EPA has analogized well-characterized PFAS like PFOS and PFOA to other chemicals in the class and concluded that, because these chemicals have the potential for the same adverse effects, they should be tested for the same endpoints and/or controlled to limit risks. For example, the 2009 TSCA section 5(e) consent order for GenX chemicals\textsuperscript{9} -- which are short-chain PFAS -- noted that: “EPA has concerns that these PMN substances will persist in the environment, could bioaccumulate, and be toxic ("PBT") to people, wild mammals, and birds. EPA’s concerns are based on data on the PMN substances, analogy to other [PFAS] chemicals, and to perfluorooctanoic acid ("PFOA") and perfluorooctane sulfonate ("PFOS") which are both currently under review by EPA for PBT concerns.” According to EPA:

“Toxicity studies on the analogs PFOA and PFOS indicate developmental, reproductive and systemic toxicity in various species. Cancer may also be of concern. These factors, taken together, raise concerns for potential adverse chronic effects in humans and wildlife” from exposure to GenX. \textsuperscript{9}

On this basis, the order determined that, “In light of the potential risk of human health and environmental effects posed by” GenX chemicals, their manufacture and processing “may present an unreasonable risk of injury to human health and the environment” and “the information available to the Agency is insufficient to permit a reasoned evaluation of the[ir] human health and environmental effects.” To address these findings, the order placed controls on manufacture, use and disposal and required extensive health and environmental effects testing, including chronic/carcinogenicity studies.\textsuperscript{9}

EPA applied the same approach in its 2015 proposed TSCA Significant New Use Rule (SNUR) for long-chain perfluoroalkyl carboxylate and perfluoroalkyl sulfonate substances.\textsuperscript{48} Explaining why new uses of these substances should be prohibited without EPA review, EPA relied on their similarities to PFOS and concern that other PFAS would have the same health effects:

“While most studies to date have focused primarily on PFOS, structure-activity relationship analysis indicates that the results of those studies are applicable to the entire category of PFAS chemical substances, which includes PFOS. Available test data have raised concerns about their potential developmental, reproductive, and systemic toxicity.” \textsuperscript{48}

Given the recognition of EPA and other authorities that all PFAS have the potential for causing the adverse health and environmental effects linked to well-characterized substances like PFOS and PFOA because of their common structural characteristics, there is a strong basis to conclude that the 54 PFAS covered by this petition “may present an unreasonable risk of injury” under TSCA section 4(a)(1)(A).

Magnifying this potential risk is the co-occurrence of multiple PFAS in drinking and surface water, other environmental media and the blood of humans and wildlife. This co-occurrence is amply demonstrated in the Cape Fear watershed, where numerous PFAS manufactured at the Fayetteville facility have been detected in the same samples of drinking water and surface water and human sera. Where exposure is to multiple PFAS simultaneously, their toxic effects may be additive or synergistic, resulting in greater overall risk than exposure to any individual PFAS alone.
2. **Demonstrated and Anticipated Exposure to the 54 PFAS**

As noted above, potential exposure is a component of a “may present” finding under section 4(a)(1)(A). Such exposure can be inferred from a substance’s properties and circumstances of manufacture and use as well as from direct evidence of exposure. In the case of the 14 Tier 1 compounds, their presence in human blood, produce and/or drinking water, including for large community water supplies, demonstrates human exposure by a large population in the Cape Fear watershed, amply supporting a “may present” finding. For the remaining 40 PFAS in Tier 2, a strong inference of exposure can be drawn from their presence in surface water, stormwater, wastewater, sediment, groundwater, soil, private wells, and/or air emissions. These pathways are highly likely to result in direct exposure by residents of the surrounding region as well as by fish, birds, and wildlife. Moreover, over time, the probability of exposure will be heightened by the persistence, high mobility and bio-accumulative properties of PFAS, which result in their long-term residence and wide distribution in environmental media.

Although information is limited about the commercial chemical products that leave the Chemours facility, there are also multiple pathways that could result in exposure to the 54 PFAS outside of the Cape Fear watershed:

- GenX chemicals are transported to Chemours’ Parkersburg, West Virginia facility for use in fluoropolymer production and have been detected in drinking water systems and private wells in the vicinity of the plant. GenX has also been detected downstream in Louisville, Kentucky and in other locations.

- Twelve of the 54 PFAS are used to manufacture products, like plastics (PSEPVE, PPVE), chemicals (PEPF, HFPO-DAF), and/or other raw materials (PSEPVE, PPVE, PEPF, HFPO-DAF) that are distributed in commerce. These PFAS could be released into the environment or result in worker or population exposure at sites where the products are processed or used or at the end of their life-cycles, for example by incineration, landfilling or wastewater treatment. They could also result in food contact if they migrate from food packaging, an application in which PPVE is used.

- Four of the 54 PFAS are listed directly as ingredients in consumer products and can be expected to result in widespread exposure, while others are used in manufacturing of consumer goods.

Specific uses of these 54 PFAS as reported in EPA’s Chemistry Dashboard based on Dionisio (2018) are listed below in Table 2.

<table>
<thead>
<tr>
<th>CHEMICAL NAME</th>
<th>SUMMARY OF USES</th>
</tr>
</thead>
<tbody>
<tr>
<td>trifluoro(pentafluoroethoxy)ethylene</td>
<td>plastics manufacturing</td>
</tr>
<tr>
<td>Chemical</td>
<td>Use</td>
</tr>
<tr>
<td>----------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>tetrafluoroethylene</td>
<td>food and drinking water contact materials, plastics, sewage treatment</td>
</tr>
<tr>
<td>hexafluoropropene</td>
<td>food and drinking water contact materials, plastics, automotive, interior</td>
</tr>
<tr>
<td>trifluoro(trifluoromethoxy)ethylene</td>
<td>food and drinking water contact materials, plastics</td>
</tr>
<tr>
<td>1,1,2,2-tetrafluoro-2-[1,2,2-trifluoro-1-(trifluoromethyl)-2-[(trifluorovinyl)oxy]ethoxy]ethanesulphonyl fluoride</td>
<td>plastics manufacturing</td>
</tr>
<tr>
<td>1,1,1,2,2,3,3-heptafluoro-3-[(trifluorovinyl)oxy]propane</td>
<td>food and drinking water contact materials, plastics</td>
</tr>
<tr>
<td>propanoyl fluoride, 2,3,3,3-tetrafluoro-2-(pentafluoroethoxy)-</td>
<td>chemical manufacturing</td>
</tr>
<tr>
<td>2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)propionyl fluoride</td>
<td>chemical manufacturing</td>
</tr>
<tr>
<td>2,3,3,3-tetrafluoro-2-(trifluoromethoxy)propionyl fluoride</td>
<td>chemical manufacturing</td>
</tr>
<tr>
<td>decafluorobutane</td>
<td>drug manufacturing</td>
</tr>
<tr>
<td>2,3,3,3-tetrafluoro-2-[1,1,2,3,3,3-hexafluoro-2-[1,1,2,2-tetrafluoro-2-(fluorosulphonyl)ethoxy]propoxy]propionyl fluoride</td>
<td>chemical manufacturing</td>
</tr>
<tr>
<td>trifluoro(trifluoromethyl)oxirane</td>
<td>chemical and plastics manufacturing</td>
</tr>
</tbody>
</table>

- Chemours is now transporting its process wastewater off-site for deep well injection in Deer Park, Texas. Spills or accidents at the facility during the manufacturing process and during transport could release PFAS into the environment, and releases are also possible during or after injection. Furthermore, Chemours has stated its intention to resume wastewater discharges to the Cape Fear River, which will result in PFAS discharges even if the waste is treated first to reduce levels of PFAS.51

Thus, in addition to their presence in environmental media and biota in the Cape Fear region adjacent to and downstream of the Chemours facility, several of the 54 PFAS are likely a common source of exposure for a broad segment of the US population.

B. Insufficiency of Information and Experience

To justify testing, TSCA section 4(a)(1)(A) also requires EPA to determine that there are “insufficient information and experience” to reasonably determine or predict a chemical’s effects on health or the
environment. For the 54 PFAS, the sufficiency of available information should be determined by comparing available data with the known adverse effects of other PFAS. The goal should be to conduct a scientifically sound assessment of each of the 54 chemicals for the critical toxic endpoints that have been identified in studies on PFOS, PFOA and other well-characterized substances. If such an assessment cannot be conducted for the 54 substances because of the lack of data, available information on these substances should be deemed “insufficient” under TSCA section 4(a).

As EPA stressed in its PFAS Action Plan,7 “[t]here are many PFAS of potential concern to the public that may be found in the environment. Most of these PFAS lack sufficient toxicity data to inform our understanding of the potential for adverse human or ecological effects.” The absence of toxicological data was underscored by ATSDR in its draft 2018 Toxicological Profile for PFAS,4 which identifies numerous critical gap gaps across the PFAS class. The 54 substances covered by this petition fit this pattern. Either they lack any health and ecological effects data or the available studies are limited and incomplete and do not provide an adequate basis for hazard and risk assessment. Key data gaps include measurement of physical-chemical properties, methods of analysis, assessment of partitioning, bioaccumulation, and degradation, pharmacokinetics, and toxicity, especially for the endpoints commonly observed for the better studied PFAS, such as liver toxicity, and effects on the immune system, lipid metabolism, kidney, thyroid, development, reproduction, and cancer. In addition, despite their widespread detection in environmental media, ecotoxicity data are generally lacking.

While testing has been conducted on GenX and Chemours is required to test five other PFAS under the North Carolina consent order, this testing is inadequate to fully evaluate the risks of these substances. For example, EPA’s draft 2018 Toxicity Assessment for GenX chemicals44,52 highlights the need for additional carcinogenicity studies:

“One study is available on evaluating carcinogenicity of HFPO dimer acid and its ammonium salt in rats (DuPont-18405-1238, 2013). In this study, liver and pancreatic tumors were noted at the highest doses tested. The available data for HFPO dimer acid ammonium salt suggest that mice might be more sensitive to exposure to these GenX chemicals than rats. Given the evidence that the liver is the target organ for toxicity and the primary organ for tumor development, there is a need for additional research using chronic duration exposures in mice.”

Echoing concerns in EPA’s PFAS Action Plan, the EPA draft GenX toxicity assessment44,52 also notes that “[n]o data are available to evaluate cancer risk via dermal or inhalation exposure” and that “[d]ata for the elucidation of differential susceptibility dependent on life stage (e.g., developing fetus, women of reproductive age, or pregnant women) are not available.”

Comments on the EPA draft GenX toxicity assessment52 by CEH and other groups also highlighted the “[l]ack of toxicity data from inhalation and dermal exposure routes” and underscored the need for additional developmental toxicity and immunotoxicity studies:

“Developmental toxicity and immunotoxicity are common health effects associated with PFAS exposure, both of which can occur at extremely low levels of exposure.4 Two developmental toxicity studies, only one of which was in mice, and a single study that specifically assesses immune effects is a serious database limitation.
One critical data gap is the lack of a full 2-generation toxicity study evaluating exposures during early organogenesis. Additionally, there are many developmental and immune effects that have yet to be assessed, including reproductive system development (i.e. mammary gland development and function), neurodevelopment, autoimmunity, infectious disease resistance, and immune hypersensitivity (i.e. asthma and allergies).

A recent study comparing the toxicity of PFOA and GenX in pregnant mice and their developing embryo–placenta units demonstrated a similar increase in placental abnormalities relative to controls for both compounds, underscoring concern about GenX’s reproductive effects and the need for additional data. The testing program proposed by petitioners would address these GenX data-gaps.

Similarly, while the testing program for 5 PFAS called for by the North Carolina consent order is a step in the right direction, it falls short of providing sufficient information for an informed assessment of these chemicals’ health effects. The five substances are all listed in Tier 1 because of demonstrated human exposure, as evidenced by their presence in human sera, produce and/or drinking water. Nonetheless, the studies to be conducted consist only of 28-day oral immunotoxicity studies and 90-day oral repeated dose toxicity studies in rats and mice. Not included are studies on reproduction/developmental toxicity, carcinogenesis, toxicokinetics and fate and transport, which are essential for risk evaluation. In addition, testing is only by the oral route and so inhalation and dermal toxicokinetic studies are important for those substances for which exposure is predominantly by these routes. These gaps would limit understanding of the health effects of the five substances and impede assessment of the risks of historical, ongoing and future exposure which is essential for public awareness, public health protection and informed risk management. The testing program proposed in this petition for Tier 1 chemicals would develop this missing information.

C. The Need for Testing to Develop Sufficient Information

A final prerequisite for issuing a test rule under section 4(a)(1)(A) of TSCA is a finding that testing “is necessary” to determine the health and environmental effects for which sufficient information is lacking. The studies proposed by petitioners are the minimum necessary for a full understanding of the health risks from past present and future exposure to the 54 PFAS by petitioners, their families and the communities they represent and for health protective reductions in risk and exposure going forward.

A strong focus of this petition is toxicity testing in experimental animals. These studies are “necessary” under TSCA because no alternative to animal testing is capable of developing reliable health effects information on individual PFAS at this time.

As described above, PFOA, PFOS and other PFAS are known to cause common modes of toxicity in vivo, such as effects on liver biochemistry and cholesterol, pup survival, and immunotoxicity. However, the mechanisms and molecular initiating events associated with these effects have not been defined. As a result, in vitro assays or computational approaches that reliably predict in vivo effects are not currently available or validated for PFAS. A recent peer-reviewed paper from EPA scientists explains that the Agency is conducting in vitro testing on 75 PFAS in order to develop and validate approaches to predict PFAS toxicity based on molecular structure. However, this work is still at an early stage and only two of the PFAS covered by this petition are included in the EPA testing.
In addition, the toxicokinetics of PFAS are unpredictable, making it difficult to anticipate relationships between intake and serum or tissue concentrations.\(^4,28,54,58\) Testing in experimental animals, including toxicokinetics and endpoint-specific studies, is necessary to address these uncertainties. A recent scientific meeting report focused on PFAS concluded that: “Differences in bioactivity, and/or kinetics that are not consistently explained by structure alone necessitate prioritization of collection of additional toxicity data associated with representative molecules.” The report adds “that traditional tools used for organic molecules to predict fate and transport are generally not helpful in predicting outcome for this group of substances highlighting the need for new approaches and models that could be useful for fluorinated organic molecules.”\(^58\)

Animal studies alone, however, are not sufficient for understanding the human health effects of PFAS exposure. The half-lives of PFAS may vary between chemicals and between species, sexes, and developmental stages, and this variability appears to drive some of the apparent differences in toxicity (e.g. see ATSDR 2018).\(^4,54\) Moreover, human half-lives are not confidently predicted from animal studies, although these studies can provide important information about biological persistence.\(^4\) Thus, the best approach is a combination of toxicokinetic studies in rats and mice and longitudinal biomonitoring studies in workers when they are starting or ending their exposure. These studies, accordingly, are likewise “necessary” under TSCA for informed risk assessment and management. This petition would require Chemours to fund and facilitate them.

Finally, exposures around the Fayetteville facility and by the downstream communities that petitioners represent have been to multiple PFAS simultaneously. Although animal toxicity studies on individual PFAS are essential, they do not account for the synergistic and cumulative effects of multi-compound exposures. In order to understand the effects of exposure to the PFAS mixtures originating from the Chemours facility, two additional studies are necessary. First, the petition proposes animal toxicity testing of three PFAS mixtures that represent characteristic exposures of residents in the Cape Fear watershed. Second, the petition proposes a study of PFAS-associated health outcomes in exposed residents in the watershed. Both of these studies are “necessary” under TSCA because they are the only way to examine the health impacts from exposure to the mix of PFAS chemistries uniquely associated with the North Carolina operations of DuPont and Chemours. The petition proposes to study three PFAS mixtures that replicate actual exposure pathways using the same toxicology tests specified for the 54 individual PFAS, as specified below. The proposed epidemiologic study would be based on the approach successfully used in Parkersburg WV to examine the human health impacts of PFOA exposure (the “C8 Study”).\(^59, 60\)

As discussed below, this petition also proposes ecotoxicity studies, which EPA has previously determined are necessary because of the widespread presence and mobility of PFAS in environmental media, and studies of physical-chemical properties and fate and transport, which are necessary for both human health and environmental risk assessment for the 54 substances.

**VII. FRAMEWORK FOR PROPOSED TESTING PROGRAM**

**A. General Approach to Testing**

Once EPA makes the findings justifying testing under section 4(a)(1)(A), it must require studies that will “develop information with respect to the health and environmental effects for which there is an insufficiency of information and experience” and which are “relevant to a determination” whether the substance “does or does not present an unreasonable risk to health and the environment.” Consistent
with this requirement, the petition calls for Chemours to perform toxicity studies on the 54 chemicals which address the endpoints associated with critical effects that have previously been the basis for evaluating the risks of PFOA, PFOS, and other studied PFAS. As discussed above, studies on these compounds show a generally similar or overlapping set of adverse effects, including cancer, hormone disruption, liver and kidney damage, developmental and reproductive harm, changes in serum lipid levels, and immune system toxicity.\cite{3,4,42,44} These effects have in turn been selected as the basis for health-based guidelines for drinking water and other pathways of exposure.\cite{3,40,42} The proposed testing approach for the 54 PFAS takes advantage of this existing knowledge by specifying studies that target critical effects that likely overlap with those that have been demonstrated by more extensive testing of PFOA, PFOS and other studied PFAS, recognizing that potency and toxicokinetics may vary by compound and endpoint. The endpoints to be addressed in these studies are described below:

B. Human Health Effects Studies on the 54 PFAS in Experimental Animals

For all 54 compounds, the following critical human health endpoints should be investigated:

1. **Liver toxicity**: Effects on liver are commonly reported for PFAS studies in humans and animals\cite{3,4} and this effect was selected as the basis for risk assessments for drinking water guidelines for several PFAS in several states, including California, Maine, New Jersey, and North Carolina (PFOA); Canada (PFOS); and EPA (GenX).\cite{42,44} Blood biochemistry markers that reflect liver toxicity, such as ALT, are altered in animal and human studies of PFAS exposures, e.g. Nian (2019).\cite{46} Studies in exposed humans have also shown that PFAS affect serum levels of cytokeratin 18 M30, tumor necrosis factor α, and Interleukin 8.\cite{61}

2. **Serum biochemistry including cholesterol, lipids, glucose**: PFAS appear to affect metabolism and effects on cholesterol and lipid levels have been reported in humans and animals.\cite{3,4,17,62} Other serum biochemical markers are related to PFAS effects on liver and kidney.

3. **Immunotoxicity**: Immunotoxicity, especially reduced antibody response, has been reported for PFOS, GenX, and other PFAS in animals and humans, and US NTP has categorized PFOA and PFOS as presumed human immunotoxicants.\cite{4,38,44,47,63-65} The effects reported for PFOS in the Dong (2009) animal study\cite{38} are the basis for the New Jersey drinking water guideline for PFOS.

4. **Developmental and reproductive toxicity, especially pup body weight and mortality**: Risk assessments to set drinking water guideline levels for PFOA and PFOS have often been based on pup weight and pup mortality in the 4 days after birth, as well as on other developmental effects.\cite{4,40,42} Specifically, the Lau (2006) study\cite{55} of PFOA shows important effects on pup viability at low exposures and this study has driven several governmental risk assessments.

5. **Kidney toxicity**: While kidney toxicity does not appear to be the critical effect for PFOA, PFOS, or GenX, kidney effects are commonly reported in rodents and humans.\cite{3,4}
6. **Thyroid hormones**: PFAS exposures affect thyroid hormone levels in multiple studies. Thyroid was a critical effect for EPA’s PFBS risk assessment.\(^{44}\)

For Tier 1 compounds with demonstrated human exposure in human sera and/or drinking water, the following additional critical endpoints would be addressed:

1. **Developmental neurotoxicity**: Michigan set its drinking water guideline for PFOA based on developmental neurotoxicity and Texas set its PFOS guideline based on altered hippocampus synapse structure.\(^{42}\) Maine set a drinking water remediation guideline for PFOA based on thyroid hormone changes,\(^{40}\) and EPA added a 3X uncertainty factor in the GenX risk assessment because of the lack of data for developmental neurotoxicity and immunotoxicity assessments.\(^{44}\) Thyroid was a critical effect for EPA’s PFBS risk assessment.\(^{44}\)

2. **Developmental immunotoxicity**: As noted above immunotoxicity has been observed in rodent and human studies for several PFAS, and EPA added a 3X uncertainty factor in the GenX risk assessment because of the lack of data for developmental neurotoxicity and immunotoxicity assessments.\(^{44}\)

3. **Developmental reproduction including mammary gland development**: US EPA and Minnesota set their drinking water guidelines for PFOA based on accelerated puberty. Texas set theirs based on altered mammary gland development and New Jersey highlighted altered mammary gland development as a critical effect of concern. The altered mammary gland development from early life exposure is linked to impaired lactation in rodent studies.\(^{66,67}\) Effects on the developing male and female reproductive system have been reported for several PFAS.\(^{4}\)

4. **Carcinogenesis**: PFOA and GenX have been tested in cancer bioassays and found to cause pancreatic, liver, and other tumors, and studies in humans showed elevated kidney and testicular cancers.\(^{4,44,45}\) California has based drinking water guidelines on these cancer effects.\(^{42}\)

For all 54 compounds, an informed risk evaluation will also require studies to characterize toxicokinetics in the test animal species, including parameters necessary to anticipate internal doses associated with oral, dermal, and inhalation exposures in humans. Also, all toxicity studies would verify internal doses with serum or other tissue concentrations.

Previous PFAS animal testing has been conducted mainly by the oral route of exposure. This route is appropriate for evaluating risks of ingesting PFAS-contaminated drinking water. However, inhalation and dermal contact are also known pathways of exposure for some PFAS. Previous testing has rarely examined the effects of PFAS exposure by these routes. As EPA acknowledges in its PFAS Action Plan, “[l]imited data exist on health effects associated with inhalation or dermal exposure to PFAS.”\(^{38}\) As noted above, EPA’s draft toxicity assessment for GenX and public commenters have highlighted the absence of information to assess risks from inhalation and dermal exposure.\(^{44}\) For the testing proposed in this petition, studies would be carried out by oral routes of administration, except inhalation would be used for volatile chemicals. (Within the set of 54 chemicals, ten have an estimated boiling point above 30° C. and an additional nine have an estimated vapor pressure above 10 mm Hg, indicating these may be candidates for inhalation testing.)
For several end-points and routes of exposure, testing should be conducted in two species, i.e. rats and mice, as EPA indicated in its GenX toxicity assessment. The proposed testing plan specifies which tests should be conducted in both rats and mice vs. in just one species.

All PFAS studies should be designed to measure levels of the test compound and any potential metabolites (though these are unlikely to occur) in sera or urine in order to verify internal dose and correlate administered and internal doses with adverse effects, including for different routes of administration.

C. Animal Studies on PFAS Mixtures

As noted above, while studies on individual PFAS provide critical information, they do not account for the synergistic and additive effects of simultaneous exposure to multiple PFAS and likely will underestimate the severity of health effects as a result. In the Cape Fear watershed, actual exposure has been to mixtures of PFAS in drinking water, augmented by concurrent exposure to PFAS in ambient air and local produce. To capture the interactions between the multiple PFAS to which local populations have been exposed, this petition proposes that representative PFAS mixtures undergo the same set of animal studies as the 54 individual compounds. To conduct this testing, three PFAS mixtures would be formulated and administered, reflecting distinct subgroups in the exposed population: 1) the mixture of PFAS detected in drinking water consumed by Cape Fear communities downstream of the Chemours plant; 2) the mixture of PFAS found in the blood of area residents during bio-monitoring; and 3) the mixture of compounds to which residents living near the Chemours facility have been exposed as a result of plant emissions and discharges (i.e. PFAS measured in ambient air, private wells, and local produce).

D. Human Studies of Communities Exposed to PFAS-contaminated Drinking Water

TSCA section 4(b)(2)(A) authorizes EPA to require human epidemiology studies. Because of the extensive exposure to PFAS by communities in the larger Cape Fear watershed, it is important to better understand the levels and extent of PFAS exposure, the specific PFAS present in blood and urine and the medical histories of individuals in this population and to examine the association between these indicators of PFAS exposure and health outcomes. Studies in humans are an important way to identify health effects associated with the combined exposure to many PFAS, and to take into account toxicokinetics or susceptibilities that are unique to humans but not measured in rodent toxicity studies.

A research study to determine the health impacts of PFOA exposure was conducted under the settlement in the class action suit against DuPont for PFOA drinking water contamination near the DuPont/Chemours Washington Works in Parkersburg, West Virginia. To conduct this study, medical histories and blood samples were obtained from residents of the affected water districts (roughly 80,000 people) and then the data were reviewed by a Science Panel comprised of three respected epidemiologists. The Panel conducted a number of additional studies based on the data and ultimately reached a conclusion that it was “probable” that exposure to PFOA in drinking water was linked to testicular and kidney cancer in the impacted communities, along with additional serious non-cancer human diseases.

The West Virginia study – taken together with recent studies in other contaminated regions that show links between PFAS exposure and immune response, serum lipids, and birth outcomes (all
outcomes that are also observed in toxicology studies although generally humans appear more sensitive) – confirms the value of epidemiological data for other populations exposed to PFAS, including in Eastern North Carolina. The Cape Fear River is a source of drinking water for over 250,000 local residents downstream of Fayetteville and has been contaminated by multiple PFAS linked to the Chemours facility. Thus, there is an opportunity to examine whether health effects have occurred in this population due to drinking water exposure to a unique set of PFAS chemicals. A study modeled after the C8 Study in Parkersburg, West Virginia would provide important data about associations between measured and historically reconstructed PFAS exposure levels and selected health outcomes.

This study should recruit at least 100,000 children and adults (equally of both sexes for both children and adults) from communities exposed to PFAS-contaminated drinking water. The study should obtain blood samples from participants to measure PFAS serum levels and several effect biomarkers such as lipids, and thyroid, kidney, immune and liver function. The study would also obtain urine samples from participants to measure PFAS levels and kidney function biomarkers. Based on this information, the study would examine associations between exposure to PFAS compounds and lipids, renal function and kidney disease, thyroid hormones and disease, liver function and disease, glycemic parameters and diabetes, as well as immune response and function and cancers in both children and adults. In addition, the study would investigate PFAS differences in sex hormones and sexual maturation, vaccine response, and neurobehavioral outcomes in children. In adults, additional outcomes of interest would include cardiovascular disease, osteoarthritis and osteoporosis, endometriosis, and autoimmune disease.

ATSDR has selected research teams to implement an epidemiologic study in seven communities where people have experienced drinking water contamination from PFAS. Unfortunately, no research proposal was submitted for the Cape Fear/Wilmington communities despite their history of drinking water contamination with PFAS from the Chemours Fayetteville facility. We believe that TSCA section 4 provides authority to direct Chemours to fund an epidemiological study of these communities. Such a requirement would be based on the finding (discussed above) that PFAS releases from the facility “may present an unreasonable risk of injury” to the health of impacted communities. In addition, since animal toxicology studies do not reflect the effects of combined exposure to multiple PFAS human and animal response may differ because of toxicokinetics and other factors, there is “insufficient information and experience” to reasonably determine or predict the health effects of the 54 PFAS in the absence of human data. We request that EPA therefore include a human study in the test rule or order it issues in response to this petition.

E. Human Half Life Studies

As noted above, the half-lives of PFAS may vary between chemicals and between species, sexes, and developmental stages, and this variability appears to drive some of the apparent differences in toxicity. Moreover, half-lives in humans may not be predicted from animal studies. Thus, to determine half-lives in humans, we propose that Chemours conduct longitudinal studies in its workers to detect the rate of increase and rate of decay of serum or tissue levels as exposure begins or ceases.

F. Physical-Chemical Properties and Fate and Transport Studies and Test Standards

i. Fate and Transport Studies
EPA’s PFAS Action Plan recognizes that “Information for many PFAS sources, fate and transport, and human and ecological exposure is sparse, both spatially and temporally.” In addition to toxicity testing in animals and humans, conducting risk evaluations for these 54 chemicals will require the ability to effectively identify and quantify concentrations of the chemicals in various media. Thus, additional testing is necessary to evaluate fate and transport for the 54 PFAS, including their propensity to bioaccumulate, bind to organic material, partition to air or water, and degrade under various conditions.

The EPA OPPTS 835 series of tests addresses fate and transport characteristics and notes that “Information on the degradability of organic chemicals may be used for hazard assessment or for risk assessment under TSCA.” Here too, testing conducted on GenX at EPA direction provides a model for fate and transport studies on other PFAS. In the GenX PMNs, Chemours (then DuPont) submitted studies for thermal transformation byproduct ready biodegradability and activated sludge respiration inhibition. The EPA consent order following review of these PMNs required several additional studies as listed in Table 3 below:

<table>
<thead>
<tr>
<th>ENVIRONMENTAL FATE TESTING</th>
<th>OPPTS OR OECD GUIDELINE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Modified Semi-Continuous Activated Sludge (SCAS) with Analysis for degradation products</td>
<td>OPPTS 835.5045, OPPTS 835.3210 or OECD 302A</td>
</tr>
<tr>
<td>Aerobic and Anaerobic Transformation in Soil</td>
<td>OECD 307</td>
</tr>
<tr>
<td>Aerobic and Anaerobic transformations in Aquatic Sediment Systems</td>
<td>OECD 308</td>
</tr>
<tr>
<td>Direct Photolysis in Water (if wavelengths &gt;290 nm are absorbed)</td>
<td>OPPTS 835.2210</td>
</tr>
<tr>
<td>Indirect Photolysis in Water</td>
<td>OPPTS 835.5270</td>
</tr>
<tr>
<td>Phototransformation of Chemicals on Soil Surfaces</td>
<td>OECD Jan. 2002 Draft</td>
</tr>
<tr>
<td>Simulation test-Acrobic Sewage Treatment (Activated Sludge Units)</td>
<td>OECD 303A</td>
</tr>
<tr>
<td>Anaerobic biodegradability of organic compounds in digested sludge</td>
<td>OECD 311</td>
</tr>
<tr>
<td>Fish Bioconcentration test</td>
<td>OPPTS 850.1730</td>
</tr>
</tbody>
</table>

The studies included in the GenX PMNs and consent order should be performed on the 54 PFAS. (See Table 4a below.)

2. Physical-Chemical Properties Studies

Tests to characterize these chemicals’ physical-chemical properties should also be conducted if they have not already been performed by Chemours. The EPA recently issued a test order for physical-
chemical data for Pigment Violet 29, one of the first chemicals subject to a TSCA risk evaluation, indicating the importance of this type of data for assessing risk under TSCA.72

According to the 2009 consent order issued by EPA,9 the PMNs for GenX compounds included the following tests of physical-chemical properties: water solubility, vapor pressure, and octanol water partition coefficient. The consent order required two additional physical/chemical property tests: for UV visible absorption (OPPTS 830.7050 or OECD 101) and hydrolysis as a function of pH (OPPTS 830.7050 or OECD 111). We are adding one additional test to determine the octanol:air partition coefficient. Taken together, these comprise a minimum set of physical-chemical properties tests that should be performed on the 54 PFAS, if not previously conducted.

3. Test Standards

While Chemours has apparently developed test standards for several PFAS measured in environment media, these standards are not readily available to the public and their adequacy cannot be assessed. To the extent they do not now exist, Chemours should develop valid analytical tools for detecting and measuring the presence of the 54 PFAS in the environment. For example, for pesticide registrations, EPA requires registrants to provide an analytical standard to ensure that the chemical can be identified with confidence in environmental or tissue samples. The EPA method notes that: “Proper analytical reference grade materials [should be] available for the Agency to validate residue and environmental chemistry analytical methods and that Federal and State enforcement laboratories have a known consistent source of analytical reference standards to validate methods employed in enforcement and monitoring activities”(EPA method OPPTS 860.1650).

G. Eco-toxicity Testing

EPA’s PFAS Action Plan recognizes that “[e]cological toxicity information is also needed by stakeholders to inform risk assessment and management to protect ecosystems, animals, and plant resources they support, and ultimately the human benefits that stem from these resources, including, for example, the prevention of potential PFAS risks associated with consuming game animals and fish.”41 An understanding of the eco-toxicity of the 54 PFAS is critical because many of them have been detected in surface water and in aquatic species and, if persistent, bio-accumulative and mobile, will be widely found in fish, wildlife and other biota and may migrate up the food chain.

The 2009 consent order for GenX compounds finds that “there is high concern for possible environmental effects over the long-term,” citing the GenX analog PFOA, “which has been detected in a number of species of wildlife, including marine mammals [and] is toxic to mammalian and other species.”9 Noting that the 2008 Dupont PMNs contained the results of acute toxicity testing of fish (Rainbow trout, daphnia, and algae), the consent order requires three additional studies – a Fish Early Life Stage Toxicity test (OPPTS 850.1400), a Daphnid Chronic Toxicity test (OPPTS 850.1300), and an Avian Reproduction test-Bobwhite Quail (OPPTS 850.2300). A similar but narrower set of studies is required for the 5 PFAS to be tested under the North Carolina consent order.27

The ecotoxicity end-points addressed in the 2008 PMNs and the 2009 consent order define a minimum set of environmental effects studies that should be performed on the 54 PFAS covered by this petition.
F. Avoiding Duplicative Testing

As discussed above, 5 of the 54 listed chemicals in this petition are also designated for testing in the Chemours North Carolina consent decree. These tests would not need to be replicated in response to this petition. Similarly, GenX has undergone several studies under EPA’s 2009 PMN consent order and those tests would also not be repeated. However, GenX and the five PFAS subject to testing under the Chemours consent decree would require additional studies under the framework presented in this petition and these studies would need to be conducted under the test rule or order issued if the petition is granted.

In addition, the North Carolina consent order requires Chemours to submit all known analytical test methods and lab standards for PFAS in air emissions and process wastewater. Under a TSCA consent order or rule, Chemours would not need to develop additional test methods and standards where they have already been developed and submitted under the consent order. This would also be the case for fate and transport, physical-chemical properties and ecotoxicity studies already conducted.

VIII. SPECIFIC STUDIES TO BE CONDUCTED AND RELEVANT TEST GUIDELINES

Based on the testing framework described above, Table 4 lists the specific studies to be required and relevant test guidelines if the petition is granted.

TABLE 4A- TESTING NEEDED FOR ALL PFAS IN THE PETITION (TIERS 1 AND 2) AS WELL AS THREE PFAS MIXTURES

<table>
<thead>
<tr>
<th>Type of test</th>
<th>EPA or other method number</th>
<th>Special requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic chemistry, physical-chemical properties, fate and transport, analytical standards</td>
<td>OPPTS 860.1650</td>
<td></td>
</tr>
<tr>
<td>Provide analytical standard to EPA</td>
<td>OPPTS 830.1550, 1600, 1620, 1650, 1670, 1700, 1750, 1800, 1900</td>
<td></td>
</tr>
<tr>
<td>Product identity, composition, and analysis</td>
<td>OPPTS 830.7200, 7220, 7300, 7840, 7860, 7950, 7370, 7550, 7560, 7000, 7050</td>
<td>Melting point, boiling point, density, water solubility, vapor pressure, dissociation constant, octanol-water partition coefficient, octanol-air partition coefficient, pH, UV/Vis absorption, hydrolysis as a function of pH</td>
</tr>
<tr>
<td>Toxicity</td>
<td>EPA method 870.3650, with modification</td>
<td>2 species</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>----------------------------------------</td>
<td>---------------------------------------------------------------------------</td>
</tr>
<tr>
<td><em>Combined repeated dose toxicity study with repro/dev tox screening test, oral</em></td>
<td>Oral (or by inhalation for higher vapor pressure chemicals)</td>
<td>For this study, must use a modified protocol similar to that required for GenX in the TSCA test order to DuPont(^9) and similar to the Lau et al. 2006 PFOA study. (^5) Requirements for the study include (1) include 45 dams in control group, 25+ dams in each treatment group; (2) the duration of the study should be extended to until the pups have reached sexual maturation; (3) dosing of the parental animals should be continued through lactation and then the pups should be directly dosed until they reach sexual maturation; (5) pup body weight should be recorded on lactation days 0, 4, 7, 14, and 21 and then at weekly intervals, (6) litter size can be standardized to 4 male and 4 female pups/litter on lactation day 4 (optional); (7) at weaning one pup/sex/litter shall be randomly selected to follow until sexual maturation; and (8) the time of sexual maturation should be recorded (i.e. vaginal opening and preputial separation).</td>
</tr>
<tr>
<td>This study would be conducted on all 54 chemicals as well as on three PFAS mixtures that represent what local residents have been exposed to.</td>
<td>Critical endpoints:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>liver weight and histology</td>
<td></td>
</tr>
<tr>
<td></td>
<td>liver lipid content</td>
<td></td>
</tr>
<tr>
<td></td>
<td>kidney weight</td>
<td></td>
</tr>
<tr>
<td></td>
<td>serum biochemistry, including ALT, lipids (including subfractions and lipoproteins),(^6) glucose, cytokeratin 18 M30, tumor necrosis factor (\alpha), and Interleukin (^8) pup weight and survival, pup liver/body weight ratio, and all other maternal and pup endpoints affected by PFOA in Lau et al 2006 pup and maternal liver gene expression spleen and thymus weights bone marrow cellularity maternal and pup hormone levels including thyroid hormones T4 and TSH, estradiol, and androgens mammary gland histopathology at puberty Plus all other standard endpoints</td>
<td></td>
</tr>
</tbody>
</table>
Serum or urine concentrations of the dosed chemicals must be measured to ascertain internal dose; and tissue concentrations on some of the repeated dose animals is needed to understand absorption, distribution, and excretion.

### Immunotoxicity

This 28-day test in mouse includes T-cell dependent antibody responses and natural killer cell activity.

This study would be conducted on all 54 chemicals as well as on three PFAS mixtures that represent the groups of PFAS that local residents have been exposed to.

**EPA method 870.7800**

Oral (or by inhalation for higher vapor pressure chemicals)

Use similar number of animals as in Dong et al. (2009) PFOS study

Standard endpoints in addition adding any additional endpoints reported in Dong et al. (2009) PFOS study

Serum or urine concentrations of the dosed chemicals must be measured to ascertain internal dose

### Toxicokinetics

Toxicokinetics investigations should determine half-life in test species used for the toxicity testing and measure tissue concentrations including in fetal liver.

This study would be conducted on all 54 chemicals as well as on three PFAS mixtures that represent the groups of PFAS that local residents have been exposed to.

**EPA 870.7485 tier 1, with modification to include pregnant animals**

This testing should be conducted in all the species used for toxicity testing, in males and females, by oral, inhalation, and dermal routes of exposure, and in pregnant animals.

Determine serum half-life, any tissues where bioaccumulation occurs, and any metabolites or transformation products.

Determine the relationship between administered dose and serum and tissue concentrations.

Based on this information, propose best method and matrix for biomonitoring in humans

### Ecotoxicity

Same testing as was included in the GenX PMNs and required

1) acute toxicity testing of fish (Rainbow trout), daphnia, and algae
These studies would be conducted on all 54 chemicals as well as on three PFAS mixtures that represent the group of PFAS that local residents have been exposed to.

2) a fish early life stage toxicity test (OPPTS 850.1400),
3) a daphnid chronic (reproduction) toxicity test (OPPTS 850.1300),
4) sediment 10-day freshwater invertebrates toxicity test
5) an avian reproduction test—bobwhite quail (OPPTS 850.2300)

### TABLE 4B: ADDITIONAL TESTING NEEDED FOR PFAS WITH DOCUMENTED HUMAN EXPOSURE (TIER 1)

<table>
<thead>
<tr>
<th>Type of test</th>
<th>EPA or other method number</th>
<th>Special requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extended one-generation with developmental immunotoxicity, reproductive toxicity, and neurotoxicity batteries and assessment of mammary gland development and hormone concentrations</td>
<td>NTP multigeneration and OECD 443</td>
<td>Rats should be used for this assessment, except mice should be used for for developmental neurotoxicity assessment. Parental males should be dosed for 10 weeks prior to mating; standard endpoints plus hormone measurements (estradiol, androgens, T4, T3, TSH, glucocorticoids) mammary gland developmental and lactational effects (specifically, must assess whether the F1 can successfully feed the F2) pup and maternal liver gene expression and PPAR receptor levels developmental immunotoxicity developmental reproduction developmental neurotoxicity in mice (learning and memory, motor, behavioral) Serum or urine concentrations of the dosed chemicals must be measured to ascertain internal dose</td>
</tr>
</tbody>
</table>

### TABLE 4C: HUMAN STUDIES

**Human half-life determination**

Design longitudinal studies in Chemours workers to detect rate of increase and rate of decay of serum or tissue levels as exposure begins or ceases in order to determine half-lives in humans.
**Epidemiological study of residents exposed to PFAS from contaminated drinking water from Cape Fear River and other pathways**

Study health outcomes associated with past and current exposures in residents exposed to contaminated drinking water from Cape Fear River. A design similar to the C8 Study in Parkersburg WV is appropriate, given that an estimated 250,000 people downstream of the Chemours plant are served by drinking water with PFAS contamination. The study will include biomonitoring and historical exposure reconstruction taken together with reporting of relevant health outcomes including birth weights, ability to breast feed, liver and kidney function, cholesterol and blood chemistry, immune function, vaccine response, autoimmune diseases, and cancer. To be adequately powered, the study should enroll 100,000 or more residents. The study should examine a separate subgroup of residents close to the Chemours plants whose exposures were different from the rest, dominated by emissions of PFAS to air and subsequent inhalation and drinking water contamination from private wells and contamination of locally grown foods.

### IX. MECHANISMS FOR CONDUCTING TESTING

As required under TSCA, Chemours would be legally responsible for carrying out testing in compliance with the EPA rule or order, which would prescribe in detail the protocols and methodologies for conducting testing, deadlines for completing testing and submitting results and requirements for filing progress reports and describing data and findings.

However, as a landmark testing program on a visible and important class of chemicals, it will be critical to assure that the required studies are performed independently and according to the highest scientific standards and are subject to rigorous oversight by EPA and outside experts. To maximize the credibility and objectivity of the data and key findings, EPA should ask the National Academy of Sciences (NAS) to create an independent expert science panel with responsibility for overseeing all aspects of the testing program. The panel’s members would be appointed by NAS, with input from Chemours, the co-petitioners and other members of the public. This panel would provide direction on all aspects of study design, including selection of laboratories, protocols and methodologies, selection of dose levels, histopathology and statistical analysis of data and data interpretation and findings.

### CONCLUSION

Petitioners look forward to meeting with EPA to discuss this petition and the Agency’s response and appreciate the opportunity to present our concerns and recommendations for testing under TSCA section 4 on PFAS of great concern to the citizens of Eastern North Carolina.

Respectfully submitted,
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Science Advisors to Petitioners
REFERENCES


US National Toxicology Program. Toxicology and Carcinogenesis Studies of Perfluorooctanoic Acid Administered in Feed to Sprague Dawley (Hsd:Sprague Dawley SD) Rats. (2020).


US National Toxicology Program. Immunotoxicity Associated with Exposure to Perfluorooctanoic Acid (PFOA) or Perfluorooctane Sulfonate (PFOS). (2016).

US EPA. Long-Chain Perfluoroalkyl Carboxylate and Perfluoroalkyl Sulfonate Chemical Substances; Significant New Use Rule. (Federal Register 2885, 2890, 2015).


North Carolina DEQ. Permit NC0089915 to Chemours Fayetteville Works to discharge wastewater under the NPDES. (2020).


72 US EPA. C.I. Pigment Violet 29 (Anthr[a2, 1,9-def:6,5, 1 Od'e' f ]diisoquinoline-1 ,3,8, 10(2H,9H)-tetrone) TSCA Section 4 Test Order (Docket ID Number: EPA-HQ-OPPT-2020-0070). (2020).

**Monoether PFECAs**
Strynar et al., 2015

<table>
<thead>
<tr>
<th>Substance</th>
<th>CAS</th>
<th>Related Substance</th>
<th>Structural Isomer</th>
<th>Precursor-Aryl Fluoride</th>
<th>Vinyl Ether</th>
<th>Polyether PFECAs</th>
<th>PFESAs</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFMOOA</td>
<td>13140</td>
<td>PFMOOA</td>
<td>Sun et al.</td>
<td>Sun et al.</td>
<td>PEVE</td>
<td>PFO4DA</td>
<td>Nafion Monomer</td>
</tr>
<tr>
<td>PMPA</td>
<td>874</td>
<td>PEPA</td>
<td>Sun et al.</td>
<td>Sun et al.</td>
<td>PEVE</td>
<td>PFO4DA</td>
<td>Nafion Monomer</td>
</tr>
<tr>
<td>PFEC-2</td>
<td>715</td>
<td>PFEC-2</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>PFEC-3</td>
<td>578</td>
<td>PFEC-3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Polyether PFECAs**
Strynar et al., 2015

**PFESAs**
Strynar et al., 2015

**Other**
McCord and Strynar, 2018

**LEGEND:**
- **Structural Isomer or Related Substance:** Found in water
- **Measured by LC/MS:** Found in water
- **Precursor; acyl or sulfonyl fluoride:** Found in air
- **Volatile; GC/MS:** Found in air
ATTACHMENT 2
<table>
<thead>
<tr>
<th>Target Compound Abbreviation</th>
<th>Target Compound Name</th>
<th>CAS</th>
<th>Tier</th>
<th>Justification for inclusion (details next sheet)</th>
<th>Some testing required in Chemours consent decree or EPA GenX consent order (*If somer and only one must be tested)</th>
<th>EPA has analytical standard</th>
<th>Known to be in consumer products or used for manufacturing</th>
</tr>
</thead>
<tbody>
<tr>
<td>HFPO-DA (GenX)</td>
<td>perfluoro-2-propoxypropanoic acid (related to GenX: ammonium hexafluoroprop-2-yl-3- oxidehexanezolate 62037-80-3 )</td>
<td>13252-13-6</td>
<td>1</td>
<td>2,4,5,6,7,9,10,12,13,14,15,16,17,18,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>PFOAEDI</td>
<td>perfluoro(3,5,7,9-tetraaflourodecane) acid</td>
<td>39492-90-5</td>
<td>1</td>
<td>2,4,5,6,7,9,17,18,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>PFOA(Da) (aka TAF)</td>
<td>perfluoro(3,5,7,9,11-pentaoxaflourodecanooic) acid</td>
<td>39492-91-6</td>
<td>1</td>
<td>2,4,5,6,17,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>Nafion byproduct 2</td>
<td>3,1,1,2,3,3,3,4,4,4,4-didecafluorooctyltrifluoroethyl-1,1,1,2,2,2-hexafluorooctane-1-sulfonic acid</td>
<td>749836-20-2</td>
<td>1</td>
<td>2,4,5,6,14,15,17,18,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>Hydro-EVE acid</td>
<td>3,1,1,2,3,3,3,4,4,4,4-didecafluorooctyltrifluoroethyl-1,1,1,2,2,2-hexafluorooctane-1-sulfonic acid</td>
<td>77304-62-9</td>
<td>1</td>
<td>2,4,5,17,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>Nafion byproduct 1</td>
<td>1,1,2,3,3,3,4,4,4,4-didecafluorooctyltrifluoroethyl-1,1,1,2,2,2-hexafluorooctane-1-sulfonic acid</td>
<td>29311-67-9</td>
<td>1</td>
<td>2,4,5,6,9,14,15,17,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>POFOHEMA</td>
<td>perfluoro(3,5,9,11-tetraoxafluorodecanooic) acid</td>
<td>39492-88-1</td>
<td>1</td>
<td>2,4,5,6,7,9,13,14,15,16,17,18,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>PFOSOA</td>
<td>perfluoro(3,5,7-trioxoventacacetic) acid</td>
<td>39492-89-2</td>
<td>1</td>
<td>2,5,6,7,9,13,14,17,18,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>PFMOADA</td>
<td>perfluoro-2-methoxyacetic acid</td>
<td>1074-13-5</td>
<td>1</td>
<td>2,4,5,6,7,9,13,14,15,16,17,18,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>PFPSOFA</td>
<td>perfluoro-2-methylpropanoic acid</td>
<td>3773-73-1</td>
<td>1</td>
<td>2,4,5,6,7,9,13,14,15,16,17,18,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>NaDONA</td>
<td>sodium dodecafluorotetrahydro-4,8-dioxanenonanoate</td>
<td>95845-44-8</td>
<td>1</td>
<td>2,10</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
</tr>
<tr>
<td>PFDBA</td>
<td>perfluoro-4-methoxybutanonoic acid</td>
<td>863090-89-5</td>
<td>1</td>
<td>2,7,9,13,18</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
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<tr>
<td>PEPA</td>
<td>perfluoro-2-propoxypolyacrylic acid</td>
<td>267239-61-2</td>
<td>1</td>
<td>2,5,14,15,16,17,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
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<td>PMPA</td>
<td>perfluoro-2-propoxypolyacrylic acid</td>
<td>13140-29-9</td>
<td>1</td>
<td>2,5,14,15,16,17,19,20</td>
<td>yes</td>
<td>yes</td>
<td>GenX</td>
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<tr>
<td>NIAF</td>
<td>N/A</td>
<td>2</td>
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<tr>
<td>PMCP</td>
<td>perfluoromethylxyloptopane</td>
<td>1380-22-7</td>
<td>2</td>
<td>12</td>
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<tr>
<td>PEVE</td>
<td>pentfluorooethyl trifluoroxy vinyl ether</td>
<td>10493-43-3</td>
<td>2</td>
<td>6,12</td>
<td></td>
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<tr>
<td>PES</td>
<td>perfluoro(2-ethoxethyl) sulphonic acid</td>
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<td>1,5,17,19,20</td>
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<td>yes</td>
<td>GenX</td>
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<tr>
<td>TFE</td>
<td>tetrafluoroethylene</td>
<td>116-14-3</td>
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<td>HFP</td>
<td>hexafluoroxyphene</td>
<td>116-15-4</td>
<td>2</td>
<td>12</td>
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<tr>
<td>PMVE</td>
<td>perfluoroxyphene/perfluorovinyl ether</td>
<td>1187-93-5</td>
<td>2</td>
<td>6,12</td>
<td></td>
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<tr>
<td>(was MMF)</td>
<td>difluoropropandioido acid</td>
<td>1514-85-8</td>
<td>2</td>
<td>5,8</td>
<td></td>
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<tr>
<td>PSEPVE</td>
<td>perfluoro(4-methyl-3,6-dioxaoc-7-en)-sulfonyl fluoride</td>
<td>16090-14-5</td>
<td>2</td>
<td>6,12</td>
<td></td>
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<td>PPVE</td>
<td>heptafluoropropyl trifluoroxyl ether</td>
<td>1625-05-8</td>
<td>2</td>
<td>6,12</td>
<td></td>
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<tr>
<td>PPFP</td>
<td>2,3,3,3-tetrafluoro-2-(1,1,2,2,2-pentafluoroxyloxy)propanoyl fluoride (aka perfluoroxyphenoxypropyl fluoride)</td>
<td>1682-78-6</td>
<td>2</td>
<td>6,12</td>
<td></td>
<td></td>
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<td>HFPO-DA</td>
<td>2,3,3,3-tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)propanoyl fluoride</td>
<td>2062-98-8</td>
<td>2</td>
<td>6,12</td>
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<td>PMFMP</td>
<td>2,3,3,3-tetrafluoro-2-(trifluoroxyloxy)propanoyl fluoride (aka perfluoroxyphenoxypropyl fluoride)</td>
<td>2927-83-5</td>
<td>2</td>
<td>6,12</td>
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<tr>
<td>E2</td>
<td>fluoroether E2</td>
<td>3330-14-1</td>
<td>2</td>
<td>1,12</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>E1</td>
<td>heptafluoroxypropyl 1,2,2,2-tetrafluoroxyethyl ether</td>
<td>3330-15-2</td>
<td>2</td>
<td>3,11,12</td>
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<td>E3</td>
<td>fluoroether E3</td>
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<tr>
<td>carbon fluoride</td>
<td>333-50-4</td>
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<td>12</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>PAF</td>
<td>perfluorocycyl fluoride</td>
<td>354-34-7</td>
<td>2</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n-perfluorobutane</td>
<td>355-25-9</td>
<td>2</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MA (7)</td>
<td>tetrafluoro-2,3,3,3-tetrafluoro-2,3-fluorosulfonyloxyethyl) propanoyl fluoride</td>
<td>4089-57-2</td>
<td>2</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3,4-dimethyl-6-dioxoaactyl fluoride (aka nafion monomer precursor)</td>
<td>4089-58-1</td>
<td>2</td>
<td>6,12</td>
<td></td>
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<tr>
<td>PFP</td>
<td>perfluoropropyl fluoride</td>
<td>422-61-7</td>
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<td>12</td>
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<td>PFOA</td>
<td>perfluoropropyl fluoride</td>
<td>422-64-0</td>
<td>2</td>
<td>6,12</td>
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<td>FOSA</td>
<td>difluorosulfacetic acid</td>
<td>422-67-3</td>
<td>2</td>
<td>1,5,8</td>
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<td>HFPO</td>
<td>hexafluoropropyne oxide</td>
<td>428-59-1</td>
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<tr>
<td>Target Compound Abbreviation</td>
<td>Target Compound Name</td>
<td>DTXSID</td>
<td>CAS</td>
<td>Tier</td>
<td>Justification for inclusion (details next sheet)</td>
<td>Some testing required in Chemours consent decree or EPA GenX consent order (*If isomer and only one must be tested)</td>
<td>EPA has analytical standard</td>
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<td>EVE</td>
<td>methyl perfluoro(3-(1-ethenyloxypropan-2-yloxy)propanoate)</td>
<td>DTXSID8044969</td>
<td>63863-43-4</td>
<td>2</td>
<td>12</td>
<td>yes</td>
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<td>RSU</td>
<td>2,2-difluoro-2-(fluorosulfonyl) acetyl fluoride</td>
<td>DTXSID360881</td>
<td>677-67-8</td>
<td>2</td>
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<td>MMF</td>
<td>2-fluoro-2-methylpropanedioyl difluoride (aka methyl 2, 2-difluoromalonyl fluoride or 2-fluoro-2-methylpropanedioyl difluoride)</td>
<td>DTXSID0060981</td>
<td>69116-71-7 (?</td>
<td>2</td>
<td>12</td>
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<td>MAE</td>
<td>methyl hexafluoroprop-1-fluoropropanoate</td>
<td>DTXSID0060981</td>
<td>69116-72-9</td>
<td>2</td>
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<td>DAE</td>
<td>methyl perfluoro(8-(fluoroformyl)-5-methyl-4 7-dioxanonoate</td>
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<td>69116-73-0</td>
<td>2</td>
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<td>SU</td>
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<td>697-38-7</td>
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<td>NVHS</td>
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<td>801209-99-4</td>
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<td>5,6,17,19,20</td>
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<td>MTP</td>
<td>2,2,3,3-tetrafluoro-3-methoxy-propanoic acid</td>
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<td>91449-21-9 (?</td>
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<td>3,5,14</td>
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<td>Byproduct 4</td>
<td>2,2,3,4,5,5,5-4(1,1,2,2-tetrafluoro-2-sulfoethyl)pentanoate</td>
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<td>-</td>
<td>NA</td>
<td>2</td>
<td>5,17,19,20</td>
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<td>Byproduct 5</td>
<td>2-fluoro-2-(1,1,2,3,3,3-hexafluoro-2(1,1,2,2-tetrafluoro-2-sulfoethyl)propanoic acid</td>
<td></td>
<td>-</td>
<td>NA</td>
<td>2</td>
<td>5,17,19,20</td>
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<td>Byproduct 6</td>
<td>1,1,2,2-tetrafluoro-2-[[1,1,1,2,3,3,4,4-octafluorobutan-2-yl]oxy]ethane-1-sulfonic acid,1,2,2-tetrafluoro-2-[[1,1,1,2,3,3,4,4-octafluorobutan-2-yl]oxy]ethane-1-sulfonic acid</td>
<td></td>
<td>-</td>
<td>NA</td>
<td>2</td>
<td>5,17,19,20</td>
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<td>EVE Acid</td>
<td>2,3,3,3-tetrafluoro-3(1,1,1,2,3,3,3-hexafluoro-3(1,2,2-trifluoroethenoyl)propan-2-yl)sulfonic acid</td>
<td>DTXSID0060981</td>
<td>69087-46-3</td>
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<td>R-EVE</td>
<td>5-(2-carboxy-1,1,2,2,2-tetrafluoroethoxy)-2,2,3,3,5,7,7-octafluorohexanoic acid</td>
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<td>-</td>
<td>NA</td>
<td>2</td>
<td>5,17,19,20</td>
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<td>PFCA B</td>
<td>Perfluoro-3,6-dioxaheptanoic acid</td>
<td>DTXSID0060981</td>
<td>151727-58-6</td>
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<td>5,17,19,20</td>
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<td>PFCA C</td>
<td>4-(Heptafluorosiloxy)hexafluorobutanoic acid</td>
<td>DTXSID0060981</td>
<td>601212-59-9</td>
<td>2</td>
<td>5,17,19,20</td>
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<td>ID</td>
<td>Full citation</td>
<td>Source/Justification</td>
<td>Links or notes (if applicable)</td>
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<tr>
<td>1</td>
<td>McCord, J. and M. Strynar (2019). &quot;Identification of Per- and Polyfluoroalkyl Substances in the Cape Fear River by High Resolution Mass Spectrometry and Nontargeted Screening&quot;. Environmental Science &amp; Technology 53(9): 4717-4727.</td>
<td>Samples were collected from surface water in May of 2017 from the Cape Fear River. Samples were collected upstream and downstream of the Chemours facility so that they could filter out &quot;background levels&quot; of PFAS in the river. They identified 37 to 58 PFAS in the river. Many were novel compounds that had not yet been described. Some do not have CAS numbers because they are likely degradants or byproducts. Compounds that are named &quot;Compound 84&quot; could not have their identity determined because the potential isomers could not be distinguished from each other.</td>
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<tr>
<td>3</td>
<td>Kotlarz, N., et al. (2020). &quot;Measurement of Novel, Drinking Water-Associated PFAS in Blood from Adults and Children in Wilmington, North Carolina.&quot; Environmental Health Perspectives 128(7): 077005.</td>
<td>Identified by the GenX Exposure Study at NCSU as being in drinking water or human blood. They tested for 17 specific PFAS in quantitative and semi-quantitative measurements. They reported the findings for drinking water and human serum via letters to participants. The first round of results from this study have been published as Kotlarz et al. 2020.</td>
<td></td>
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<tr>
<td>5</td>
<td>Strynar, M., et al. (2015). &quot;Identification of Novel Perfluoroalkyl Ether Carboxylic Acids (PFECAs) and Sulfonic Acids (PFESAs) in Natural Waters Using Accurate Mass Time-of-Flight Mass Spectrometry (TOFMS).&quot; Environmental Science &amp; Technology 49(19): 11622-11630.</td>
<td>Strynar et al. (2015) found in Cape Fear River in surface water samples from Summer 2012. Samples were taken upstream of Chemours, downstream of Chemours, and further downstream of a tributary that feeds into the Cape Fear River to determine if it could be contributing to the contamination. This report found several legacy PFAS and replacement PFAS in the Cape Fear River.</td>
<td></td>
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</tr>
<tr>
<td>6</td>
<td>Sun, M., et al. (2016). &quot;Legacy and Emerging Perfluoroalkyl Substances Are Important Drinking Water Contaminants in the Cape Fear River Watershed of North Carolina.&quot; Environmental Science &amp; Technology Letters 3(12): 415-419.</td>
<td>Sun et al. (2016) found these compounds in the Cape Fear River using extended sampling periods between June and December of 2013. Water came from drinking water treatment plants of which the Cape Fear River was the source. This is the seminal paper that found GenX in the drinking water for the citizens of Wilmington. Figure 1 is shown regularly at conferences to this day.</td>
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<tr>
<td>8</td>
<td>Cape Fear Public Utility Authority. (2019). &quot;Raw and Finished PFAS Data: 15 May 2019.&quot; Retrieved July 2019.</td>
<td>Compounds detected in raw and finished water in Cape Fear Public Utility Authority or H2GO in Brunswick County. The Lower Cape Fear Water &amp; Authority sells to CFPUA, Brunswick PU, and Pender PU. Brunswick PU sells to H2GO. Documents cited here are from July 3, 2018 (H2GO) and May 5, 2019 (CFPUA): they were provided by a Wilmington community member.</td>
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</tr>
</tbody>
</table>

Compounds detected by FDA in produce that was grown near Chemours facility in Fayetteville, NC. In total, 20 samples were tested for 16 different PFAS, one of which was a control sample of lettuce grown outside of the area. The other 19 all had some level of PFAS detected.


This report details the levels of E1 that were released via the air from the Chemours Fayetteville Site. E1 is the thermal breakdown product of GenX. It is volatile and released into the air. It has only been detected by Chemours, not government or academic researchers.


NC DEQ Air Emissions permit review from March 14, 2019. This is the air emissions report Chemours provided to NC DEQ to describe their air scrubbing system for PFAS removal. It details various PFAS that are volatile and could be released into the air. It also details the estimated amount of PFAS (in pounds per year) that will be released after air scrubbing. This means that these compounds are produced by Chemours and released into the air by Chemours.


Results of Cape Fear Public Utility Authority testing which is ongoing. Chemicals with this justification were found in drinking water according to the May 5, 2020 testing report.


Results of Brunswick County testing of raw and finished water which is ongoing. Chemicals with this justification were found in raw water in the May 21, 2020 report.


Results of Brunswick County testing of raw and finished water which is ongoing. Chemicals with this justification were found in finished [drinking] water in the May 21, 2020 report.


Results of the Chemours wastewater testing from January, February, March 2020.


Appendix A of the Chemours Corrective Action Plan under the NC Consent Order has testing of soil and groundwater for PFAS.

Private well data. Provided by Fayetteville, NC, community member.


Results of the Chemours sediment testing from January 2020.


NPDES Permit No. NC0003573 provided by Southern Environmental Law Center.