COMMENTS ON CHEMOURS' CORRECTIVE ACTION PLAN (DEC. 31, 2019) SUBMITTED PURSUANT TO THE CONSENT ORDER IN State of North Carolina v. The Chemours Company FC, LLC, Case No. 17-CVS 580 (Bladen County Superior Court)

I. <u>Executive Summary</u>

These comments—which have been prepared by counsel for plaintiffs and the proposed class in *Carey, et al. v. E.I. du Pont de Nemours and Co. and The Chemours Co. FC, LLC*, No. 7:17-cv-00189 (E.D.N.C. filed Oct. 23, 2017), in consultation with experts Dr. Stephen B. Ellingson of Vatten Associates and Dr. Richard DeGrandchamp of Scientia Veritas—address Chemours' failure to "comply with the requirements of the 2L Rules and guidance provided by [the North Carolina Department of Environmental Quality ("DEQ")]." Consent Order ¶ 16; *see* 15A NCAC 02L .0103 ("2L Rules") ("The rules established in this Subchapter are intended to maintain and preserve the quality of the groundwaters, prevent and abate pollution and contamination of the water of the state, protect public health, and permit management of the groundwaters for their best usage by the citizens of North Carolina.").

As explained in detail below, Chemours' proposed Corrective Action Plan ("CAP"):

- fails to adequately protect the human health of residential users of municipal water supplies drawn from the Cape Fear River in Brunswick, Columbus, New Hanover, and Pender Counties ("Downstream Residential Consumers" or "DRCs");
- fails to adequately address the full extent of PFAS contamination originating from Fayetteville Works (both from the decades that DuPont operated the facility, and since Chemours took over its operations after DuPont spun Chemours off as a separate company); and
- 3) fails to provide an adequate, unbiased, and scientifically sound risk assessment.

For these reasons, the only way to adequately protect the human health and property of DRCs is to require Chemours to provide the same level of protection it has agreed to provide well owners with PFAS contamination: installation of RO filtration to the same extent required by Paragraph 20 of the Consent Order, with bottled water provided until filters are installed. Additionally, to protect human health in the Cape Fear River area, Chemours (and its predecessor DuPont) should be directed to: (1) fund and participate in independent toxicity assessments for each of the chemicals of concern, as well as any synergistic effects between those chemicals; and (2) fund and participate in epidemiological studies regarding the effects of contamination of residents in the Cape Fear River area, including contamination resulting from well-established toxic PFOA and PFOS that have been found in residents' blood as a result DuPont's and Chemours' conduct.

A. The CAP Fails to Protect Human Health.

An important starting point for understanding the failings of Chemours' CAP is Paragraph 20 of the Consent Order in *State of North Carolina v. The Chemours Company FC, LLC*, Case No. 17-CVS 580 (the "Consent Order" or "CO"). Paragraph 20 requires Chemours to install reverse osmosis ("RO") filtration systems at homes that obtain water from private drinking water wells if the wells are contaminated with:

a. combined quantifiable concentrations of PFAS listed in Attachment C [of the CO] in exceedance of 70 ng/L [or parts per trillion, "ppt"]; or
b. quantifiable concentrations of any individual PFAS listed in Attachment C [of the CO] in exceedance of 10 ng/L.

CO \P 20. These requirements are referred to in these comments as the "10/70 Action Levels" or "Action Levels." The twelve specific PFAS underlying those Action Levels are set forth in

Attachment C to the CO, which is reproduced below. These twelve PFAS are referred to in these

comments as "Attachment C PFAS."

| | | ienny. | | | | | |
|--------------------------------|---------|--|---|-----------------------------|---------------------|---------------------|--|
| Common Name PFMOAA | | Chemical Name Perfluoro- 2-methoxyacetic acid | | CASN 674-13-5 | | Chemical Formula | |
| | | | | | | C3HF5O3 | |
| РМРА | PFMOPrA | Perfluoro-2- methoxypropanoic acid | Perfluoro-3- methoxypropanoi | 13140-29- 9 | 377- 73-1 | C4HF7O3 | |
| PFO2HX | ζΑ. | Perfluoro(3.5-dioxahexanoic) acid | | 39492-88-1 | | C4HF7O4 | |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro- 2- (pentafluoroethoxy) propanoic acid | Perfluoro-4- methoxybutanoic acid | 267239- 61-2 | 8630 90- 89-5 | C5HF9O3 | |
| PFO3OA | | Perfluoro(3,5,7-trioxaoctanoic) acid | | 39492-89-2 | | C5HF9O5 | |
| PFO4DA | | Perfluoro(3,5,7,9-tetraoxadecanoic) acid | | 39492-90-5 | | C6HF11O6 | |
| PFESA-BP1 / Nafion BP #1 | | Nafion Byproduct 1 | | 66796-30-3; 29311-67-9 | | C7HF13O5S | |
| PFESA-BP2 / Nafion BP #2 | | Nafion Byproduct 2 | | 749836-20-2 | | C7H2F14O5S | |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4,5,5,6,6- decafluoro-6- (trifluoromethoxy)-; Butanoic acid, 2,2,3,3,4,4- hexafluoro-4-[1,2,2,2- tetrafluoro-1- (trifluoromethyl)ethoxy]- | | 174767-10-3; 801212-59-9 | | C7HF13O3 | |
| TAFN4 / PF05DA | | Perfluoro(3,5,7,9,11-pentadodecanoic) acid | | 39492-91-6 | | C7HF13O7 | |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | C7HF13O2 | |
| HFPO-DA / PFPrOPrA / "GenX" | | 2,3,3,3-Tetrafluoro-2 (1,1,2,2,3,3,3- heptafluoropropoxy)-propanoic acid) | | 13252-13-6 | | C6HF11O3 | |

The 10/70 Action Levels are consistent with standards needed to protect human health. In prior litigation stemming from its predecessor DuPont's contamination of the Ohio River Valley with PFAS originating from the Parkersburg, West Virginia manufacturing facility ("Washington Works"), a Science Panel jointly appointed by DuPont found a probable causal link between PFOA, or C8, and testicular cancer, kidney cancer, ulcerative colitis, thyroid disease, high cholesterol, and pregnancy-induced hypertension at exposure levels in excess of 50 parts per trillion. DuPont moved its production of toxic C8 to the Fayetteville Works facility in 2002 and continued manufacturing C8 at that facility until 2013. The Attachment C PFAS at issue in this case are closely related to C8 and have been linked to similar adverse health effects: the chemicals

that have resulted in contamination throughout the Cape Fear River area result from manufacturing C8 and its successor, GenX, at Fayetteville Works. As detailed below, the Attachment C PFAS share toxicity characteristics with C8, and the 10/70 Action Levels are appropriate and necessary measures to protect human health throughout the area, particularly in view of the fact they are being distributed to and consumed by a population that already has been exposed to PFOS and PFOA contamination emanating from Fayetteville Works.

Chemours has made a *binding contractual commitment* to remediate the effects of its PFAS contamination for residents who obtain water from private wells with test results in excess of the Action Levels. Yet, Chemours has failed to take any measures to protect residential properties served by utilities who draw their water from the Cape Fear River downstream of Chemours' Fayetteville Works plant—*even though those residences are contaminated with Attachment C PFAS above the 10/70 Action Level*.

In the summer and fall of 2019, Plaintiffs' counsel and their consulting technical experts collected and analyzed water samples from 36 residences in Bladen, Brunswick, Columbus, Cumberland, New Hanover, and Pender Counties to determine whether and at what concentrations these homes were contaminated with Attachment C PFAS. All 27 residences that were sampled in Brunswick, Columbus, New Hanover, and Pender Counties are serviced by municipal water providers including the Cape Fear Public Utility Authority ("CFPUA") and Brunswick County Public Utilities ("BCPU"). The samples were collected from either the tap or water heaters of the residences.

All samples collected and analyzed from Brunswick, Columbus, New Hanover, and Pender Counties show contaminant concentrations exceeding the threshold for installation of RO systems pursuant to Paragraph 20 of the CO; *every single sample had PFAS concentrations exceeding* *the 10/70 Action Levels*. Alarmingly, these residential water supplies are contaminated two years after Chemours claims to have ceased discharging PFAS into the Cape Fear River from Outfall 002.

The residential samples collected by plaintiffs' counsel and their experts are consistent with results published by municipal water providers in the area. Tests of finished water by the Brunswick County and the Cape Fear Public Utility Authority water systems consistently have identified Attachment C PFAS in finished water at levels well in excess of the 10/70 Action Levels.¹

Importantly, the residents serviced by municipal water with PFAS contamination exceeding the 10/70 Action Levels receive drinking water that has already been treated by the municipal water providers. PFAS contamination nonetheless persists. Moreover, there is no date certain as to when these municipal water service providers will provide replacement treatment systems designed to remediate PFAS. Nor is there any certainty that such replacement systems— when installed—will be able to remove PFAS concentrations to below the health-based criteria required by the Consent Order.

The public health concerns associated with continuing contamination of the water supply with PFAS originating from Fayetteville Works are particularly acute in light of the GenX Exposure Study PFAS Blood Sample Results published on November 18, 2018.² That study involved an analysis of blood samples from 310 Wilmington residents (44 of whom were sampled

¹ See https://www.cfpua.org/761/Emerging-Compounds (showing combined Attachment C PFAS levels in CFPUA finished water exceeding 100 ppt and as high as 300 ppt since June 2019); https://www.brunswickcountync.gov/genx/ (showing finished water at Brunswick treatment plant exceeding 400 ppt for combined Attachment C PFAS in August 2019 and exceeding 100 ppt through December 2019).

² North Carolina State University Center for Human Health and the Environment, GenX Exposure Study PFAS Blood Sample Results, *available at* https://chhe.research.ncsu.edu/wordpress/wp-content/uploads/ 2018/11/Community-event-BLOOD-slides.pdf (published November 18, 2018).

twice) for 23 PFAS. The results consistently showed newly identified PFAS (including Nafion Byproduct 2, PFO4DA, and PFO5DoDA) in residents' blood.³ Additionally, the sample found that legacy PFAS (including well-established toxins PFOA and PFOS, as well as PFHxS, PFNA, and PFDA) that were previously used at Fayetteville Works remain in residents' blood at levels substantially in excess of background levels for the United States years after C8 manufacturing was discontinued, suggesting that area residents historically were exposed to high levels of those chemicals in their drinking water.⁴ As part of the class action, the *Carey* plaintiffs allege that residents in the area should be entitled to blood tests to ascertain the amount of PFAS in their blood as a result of DuPont's and Chemours' conduct and determine whether additional medical treatment is needed. It is uncertain when or whether that relief (which Chemours opposes) may be available in the class action, and the CAP currently does not include any measures to address the health of area residents. As part of the CAP, Chemours should be required to (a) fund blood tests to ascertain the amount of PFAS in area residents' blood; (b) fund a public health study to assess the health effects of PFAS in residents' blood, including the prevalence of health conditions linked with PFAS in the community and any synergistic effects between newer PFAS and historical PFAS such as PFOA and PFOS that remain in residents' blood; and (c) toll the statute of limitations for any personal injury claims that may exist as a result of PFAS contamination until after the completion of those public health studies.

B. The CAP Fails to Provide Adequate Plans to Remediate Ongoing PFAS Contamination from the Site.

There are three primary flaws in the CAP's remedial proposal with respect to remediation of ongoing PFAS contamination originating from the Fayetteville Works site.

³ *Id.*, slide 26.

⁴ *Id.*, slide 39.

First, the CAP proposes no reduction in PFAS loadings to the Cape Fear River from aerial deposition on more than 70 square miles of the Cape Fear River watershed. Although Chemours maintains that it has reduced loadings to the environment over the last two years, it has taken at best nominal measures to abate the thousands, if not hundreds of thousands of tons of PFAS already emitted into the air. The PFAS deposited on the ground surface has vertically migrated into groundwater that is flowing into the Cape Fear River. As a result, PFAS will continue to impact the DRCs for an indefinite time unless and until RO systems are installed.

Second, the effectiveness of Chemours' measures to remediate PFAS at its own Fayetteville Works facility are highly speculative, unlikely to work, and are projected to extend over an indefinite period. Chemours has not implemented—or even completed investigating—any of the temporary or permanent measures necessary to prevent PFAS contamination from migrating into the Cape Fear River. On the contrary, Chemours' own documentation indicates that PFAS contamination will continue to migrate to the underlying groundwater and into the Cape Fear River even if Chemours manages to prevent contamination from migrating directly into the Cape Fear River from its Fayetteville Works facility. As discussed in further detail below, none of the remedies proposed in the CAP will prevent PFAS from contaminating residences at concentrations exceeding the 10/70 Action Levels for many years. Chemours maintains throughout the CAP that it is impracticable to remediate the large tracts of the Cape Fear River watershed contaminated with PFAS. This PFAS has entered the groundwater and is discharging directly into the Cape Fear River and will do so for decades to come. Because this source of PFAS contamination will continue to affect DRCs for the foreseeable future, there is all the more need to protect DRCs by installing RO systems now.

Third, the CAP fails to account for the differing rates of PFAS migration through air, soil, groundwater, sediment, and river water. PFAS will reach the DRCs not as a single "slug" but rather gradually over many years. This means that even if GenX concentrations in the DRCs' tap water declines below the 140 ppt provisional level or the 10/70 Action Levels, other PFAS will continue to impact the DRCs' tap water for years to come.

Providing the DRCs with the same level of protection afforded to residents drinking well water near the facility is the only means of protecting human health. Chemours admits that RO systems remove more than 92% of HFPO-DA, ensuring human receptor exposures remain below regulatory limits. There is no reason why the DRCs should not be provided with the same level of protection afforded to residents drinking well water near Fayetteville Works. In order to protect the DRCs, the only viable option is to provide them with RO systems including replacement of filters until such time that *at a minimum* PFAS concentrations decline below the 10/70 Action Levels.

C. The CAP Fails to Provide an Adequate, Scientifically Sound, and Unbiased Risk Assessment.

Chemours has yet to complete satisfactory risk assessments as required by paragraph 14 of the CO. Chemours has yet to properly quantify the risks of PFAS exposure to DRCs and all other individuals affected by Chemours' contamination. Chemours has failed to comply with CO Paragraph 14's requirement to establish that Attachments B and C PFAS do not pose an unacceptable risk to human health. Chemours fails to calculate toxicity values (and risks) for 19 out of 20 PFAS, focusing all of its efforts on GenX, the one PFAS for which DuPont and Chemours have produced at least *some* toxicity evaluations. Chemours' analysis fails to (a) follow standard EPA guidance for deriving toxicity values; (b) adequately address the past decade of scientific literature on GenX's toxicity; (c) properly weigh and account for the toxicity and human health risks of GenX, including immunotoxicity; and (d) account for all necessary risks and toxicity information associated with drinking contaminated water. Chemours also manipulates its conclusions by making improper and scientifically unsound assumptions that mask the true risks associated with drinking PFAS-contaminated water and fail to account for exposure risks to sensitive subpopulations.

D. DEQ Must Require Chemours to Implement the Economically and Technologically Feasible Solution of Installing RO Filters for DRCs.

Chemours' claims of technical and economic infeasibility are deeply flawed. Underlying Chemours' claims is an assumption that, if *source-based* remediation is infeasible for any reason, then *no* remedial actions may be required. This is simply false. Even assuming remediation is not feasible, Chemours has failed to analyze or propose any alternative means of protecting human health, property, and the environment, including solutions that *Chemours has already admitted are both technologically and economically feasible: installing household-level RO filters to protect all citizens and homes from ongoing PFAS contamination*. Chemours' failure to consider this alternative is a glaring defect in its CAP, and one that must be addressed particularly given Chemours' repeated suggestions that other remedial measures are or will be either technically or economically infeasible.

Chemours admits that installing RO systems is the only practical method for protecting homes near its facility on well water with PFAS concentrations exceeding the 10/70 Action Levels. This is the only practical method for protecting the DRCs as well. Therefore, the CAP should also require Chemours to pay for the acquisition, installation, operation and maintenance of RO systems for all residences in the counties above that are serviced by municipal water authorities drawing water from the Cape Fear River. During the interim period until Chemours compensates the DRCs for installing RO systems, Chemours should also compensate the DRCs for the cost of purchasing bottled water. Additionally—and particularly in light of Chemours' inadequately supported excuses for failing to propose adequate remedial measures—DEQ must seek to hold Chemours' predecessor, DuPont, liable for its role in contaminating the Cape Fear River watershed with PFAS. As yet, DuPont has not been required to account for or contribute to the remediation of the contamination caused by its activities, for which it is jointly liable along with its successor, Chemours' claims of economic infeasibility cannot be properly evaluated without considering DuPont's joint and several liability for the historical discharges of PFAS, which have spanned decades, and for which DuPont must also be held responsible.

* *

The comments below provide further detail on the points raised above, including an introductory section briefly summarizing the history of Chemours' and DuPont's production of toxic PFAS and subsequent contamination of surrounding water supplies with these harmful chemicals for decades. This history is critical, as it shows DuPont's and Chemours' historical disregard for the health and safety of residents affected by their unlawful discharges and emissions of PFAS and the significant health risks posed by these chemicals (including the Attachment C PFAS or "second generation PFAS," such as GenX). More important is that it also reveals that Chemours' recent and ongoing communications with North Carolina residents affected by Chemours' contamination are misleading in their claims that the PFAS are non-toxic and not harmful to human health.

II. <u>DuPont's and Chemours' History of PFAS Contamination and Corporate</u> <u>Irresponsibility</u>

In evaluating the CAP and, more generally, the accountability of DuPont and Chemours for PFAS contamination throughout the Cape Fear River area, understanding DuPont's long history of PFAS contamination and legacy of corporate irresponsibility in addressing serious threats to human health and the environment is critical. In particular, the CAP must be evaluated alongside: (a) the state of scientific knowledge regarding the toxicity of PFAS, including DuPont's stipulated acknowledgement that exposure to C8 (the predecessor chemical to GenX and other Attachment C PFAS that was manufactured at Fayetteville Works until 2010) in drinking water at concentrations of 50 ppt and above presents risks to human health; (b) the growing body of scientific literature confirming that second generation PFAS, such as those originating from Fayetteville Works, pose health risks substantially identical to those posed by C8; and (c) that the mishandling of these toxic chemicals by DuPont and Chemours has resulted in the presence of PFAS in drinking water throughout the area at levels exceeding 70 ppt, significantly above the level at which C8 was determined to be dangerous to human health by a jointly appointed C8 Science Panel ("C8 Panel") in prior litigation.

DuPont's and Chemours' history with PFAS began in 1951, when DuPont began using C8 at its Washington Works plant in Parkersburg, West Virginia.⁵ C8 was used as a manufacturing aid in the production of TeflonTM.⁶ Concerns about the toxicity of C8 surfaced internally within DuPont as early as 1954, and DuPont confirmed C8's toxicity to animals at least as of 1961.⁷ By 1978, the manufacturer of DuPont's C8, the 3M Company, had confirmed that C8 was detectable in workers' blood, and by 1980, DuPont confirmed it was toxic to humans, accumulated in human bodies, and that exposure to C8 was intolerable.⁸ Despite the toxicity of C8, DuPont continued using it as a processing aid.

⁵ See Leach v. E.I. du Pont de Nemours & Co., No. 01-C-608, 2002 WL 1270121, at *3 (W. Va. Cir. Ct. Apr. 10, 2002) (findings of fact from Order on Class Certification).

⁶ See In re E. I. du Pont de Nemours & Co. C-8 Pers. Injury Litig., No. 2:13-CV-170, 2016 WL 659112, at *1 (S.D. Ohio Feb. 17, 2016).

⁷ Leach, 2002 WL 1270121, at *4.

 $^{^{8}}$ *Id* at 4.

DuPont began discharging PFAS, including both C8 and newer PFAS such as hexafluoropropylene oxide dimer acid (HFPO-DA) (also known as GenX), from its vinyl ether manufacturing process at Fayetteville Works into the Cape Fear River as early as 1980. Yet, DuPont did not make any comprehensive report of its historical discharges or conduct any healthbased study on any of these PFAS discharged into the Cape Fear River, there is not yet a comprehensive study of the PFAS to which Cape Fear River area residents have been exposed as a result of the historical discharges from Fayetteville Works, and there has not yet been any study of any epidemiological impacts caused by DuPont and Chemours.

In May of 2000, when the 3M Company announced it would stop manufacturing C8 (after internal studies increasingly raised concerns about its biopersistence and toxicity), DuPont made the decision to manufacture C8 at Fayetteville Works in North Carolina.⁹ According to DEQ's internal timeline of DuPont's Clean Water Act National Pollution Discharge Elimination System ("NPDES") permitting changes, DuPont's May 2001 NPDES permit application sought to permit discharges of process wastewater containing C8 from a "new Teflon® facility" at Fayetteville Works.¹⁰

Around the time when DuPont began manufacturing PFAS in North Carolina in 2000/2001, a series of lawsuits were filed against DuPont to hold it accountable for contaminating a drinking water supply in West Virginia with C8 and for causing personal and property injury as a result of that contamination.¹¹ The *Leach* case in particular involved a class action brought by a group of individuals who alleged common law tort claims (under West Virginia law) for equitable, injunctive, and declaratory relief, along with compensatory and punitive damages, as a result of

⁹ Id.

¹⁰ See Chemours NPDES Permit File Timeline, https://assets.adobe.com/public/08e2e4d7-eeca-4164-70fb-8b9cee2d3629.

¹¹ See Leach, 2002 WL 1270121, at *1.

alleged drinking water contamination. The Wood County Circuit Court certified the class in April 2002.¹² After several years of litigation, the parties reached a settlement (the "*Leach* Settlement Agreement") that established a procedure to decide whether the approximately 80,000 class members would be permitted to proceed with individual actions against DuPont based on any of the human diseases alleged to have been caused by exposure to C8.¹³

The procedure required the parties to establish a Science Panel composed of three independent epidemiologists to study whether there was a link between exposure to C8 in drinking water (of .05 parts per billion, or **50 ppt** over the course of one year) and human disease among the *Leach* class.¹⁴ The Settlement Agreement contractually bound both parties to the results of the Science Panel's findings. Specifically, if the Science Panel issued a "Probable Link Finding"— that is, a finding that it was more likely than not that there is a link between exposure to C8 and a particular human disease (for class members exposed to C8 at 50 ppt over the course of one year)— then DuPont waived its right to challenge in individual cases whether a particular Class Member's dose of C8 (at 50 ppt) was sufficient to be capable of causing a disease with a "Probable Link" finding.¹⁵ "Probable Link" findings ultimately were issued for kidney cancer, testicular cancer, thyroid disease, ulcerative colitis, diagnosed high cholesterol (hypercholesterolemia), and pregnancy-induced hypertension and preeclampsia.¹⁶

 $^{^{12}}$ *Id*.

¹³ See Class Action Settlement Agreement, *Leach v E.I. du Pont de Nemours & Co.* (Nov. 17, 2004) (No. 01-C-608), https://www.hpcbd.com/dupont/Settlement-Agreement.pdf.

¹⁴ See Leach Settlement Agreement §§ 2.1.1; 12.

¹⁵ In re E. I. du Pont de Nemours & Co. C-8 Pers. Injury Litig., 314 F. Supp. 3d 868, 873 (S.D. Ohio 2014) ("If the Science Panel found that it was 'more likely than not that there is a link between exposure to C–8 and a particular Human Disease among Class Members,' the Panel then issued a Probable Link Finding for that specific disease and DuPont waived its right to challenge whether 'it is probable that exposure to C–8 is capable of causing' the Linked Disease, *i.e.*, general causation. ([Settlement Agreement] § 3.3).").

¹⁶ See C8 Probable Link Reports, C8 Science Panel, http://www.c8sciencepanel.org/prob_link.html.

Because the Science Panel made numerous Probable Link findings, the Settlement Agreement provided that individual class members could pursue personal injury claims individually.¹⁷ Following several bellwether trials—first for Carla Bartlett (resulting in a \$1.6 million jury verdict), and second for David Freeman (resulting in a \$5.1 million jury verdict)— DuPont and its new spin-off, Chemours, agreed to a joint global settlement of the individual personal injury suits flowing out of the *Leach* settlement, to the tune of **\$670 million**, split evenly between DuPont and Chemours.¹⁸

DuPont was also being pursued by the U.S. Environmental Protection Agency ("EPA") to remediate the harmful effects of its contamination of the water supply in West Virginia. In March 2009, DuPont and the EPA reached a Consent Order ("West Virginia 2009 Consent Order") in which DuPont agreed to offer water treatment or bottled water to people on *private or public* water systems if the level of C8 reached **40 ppt**.¹⁹

In an attempt to shed future liabilities associated with C8, DuPont began searching for replacements to C8. In 2009—shortly before it agreed to remediate water contaminated with C8 at 40 ppt—DuPont and the EPA reached a separate Consent Order under Section 2619 of the Toxic Substances Control Act (the "2009 TSCA Consent Order") that permitted DuPont to begin manufacturing GenX as a replacement PFAS for C8.²⁰ EPA noted that the scientific studies submitted by DuPont were "insufficient to permit a reasoned evaluation" of the human health

¹⁷ See Leach Settlement Agreement § 3.3.

¹⁸ See The Chemours Company, Investor Presentation at 2, 10 (March 2017), https://s21.q4cdn.com/411213655/files/doc_presentations/March-2017-Chemours-Investor-Presentation.pdf.

¹⁹ See EPA Order on Consent, In the Matter of E.I. du Pont de Nemours and Company, No. SDWA-05-2009-0001; SDWA-03-2009-0127 DS (Mar. 10, 2009), https://www.epa.gov/sites/production/files/2016-05/documents/dupont-finalorder09.pdf.

²⁰ See Consent Order and Determinations Supporting Consent Order, In the Matter of DuPont Company, Premanufacture Notice Nos. P-08-508 and P-08-509 (Jan. 28, 2009), https://chemview.epa.gov/chemview/proxy?filename=sanitized_consent_order_p_08_0508c.pdf.

effects of GenX, and "in light of the potential risk of human health and environmental effects," limited the manufacture, distribution, and disposal of the chemical.²¹ In particular, it was obliged to "recover and capture (destroy) or recycle" GenX "at an overall efficiency of 99% from all effluent process streams and . . . air emissions."²²

But as DEQ well knows, the Consent Order in this case is attributable to the fact that DuPont and its successor, Chemours, failed to follow the EPA Order and NPDES permit it agreed to abide by, discharging untold sums of PFAS into the Cape Fear River watershed and placing the health and safety of affected residents at risk.

Equally problematic is the fact that Chemours is **representing to local residents and the general public** that "there is no indication of any harmful health effects of PFAS at these low levels," with "low levels" referring to "any household with per- and polyfluoroalkyl substances ('PFAS') that are (1) at or above 10 ppt for any one PFAS, or (2) at or above 70 ppt for total PFAS."²³ It has made this representation despite agreeing specifically in the Consent Order to conduct third party toxicity studies "informative to human health," *see* Consent Order ¶ 14, despite the fact PFAS have been found in the water supply at levels well in excess of the 50 ppt exposure standard identified in the "probable link" findings by the Science Panel in the C8 litigation, and despite the availability of numerous studies demonstrating the harmful health effects of GenX and newer, second-generation PFAS.²⁴ And the California Department of Toxic Substances Control has reviewed emerging scientific studies on GenX and found that

²¹ *Id.* at xv, 36.

²² *Id.* at 36.

²³ See, e.g., C3 Dimer Acid and PFAS, The Chemours Company, https://www.chemours.com/en/aboutchemours/global-reach/fayetteville-works/fayetteville-works-toxicology (last visited Feb. 26, 2020); Letter 2B to local residents, https://files.nc.gov/ncdeq/GenX/consentorder/paragraph19/Letter-2B-offering-ROand-Table-3-results-Over-10-ppt---represented.docx.

²⁴ See, e.g., Melisa Gomis et al., Comparing the toxic potency in vivo of long-chain perfluoroalkyl acids and fluorinated alternatives, 113 Environ. Int'l 1 (2018); Gloria Post et al., Key scientific issues in

PFECAs and shorter-chain PFAAs may have similar or higher toxic potency than the longer-chain PFAAs they are replacing. Using a toxicokinetic model and existing toxicity data sets, a recent study found that PFBA, PFHxA, and PFOA have the same potency to induce increased liver weight, whereas *GenX is more potent*. The authors concluded that previous findings of lower toxicity of fluorinated alternatives in rats were primarily due to the faster elimination rates and lower distribution to the liver compared to PFOA and other longer-chain PFAAs.²⁵

Chemours' representations that there is "*no indication*" of any harmful health effects of PFAS at the Action Levels set by the Consent Order is highly misleading at best, particularly without a disclosure or explanation that there is no indication that PFAS at the Action Levels are *not* harmful to human health. Chemours should be required to make corrective disclosures to area residents regarding the potential adverse health effects resulting from exposure to PFAS at levels in excess of the Action Levels, and the lack of any basis to represent that there is not a risk of adverse health effects from exposure at those levels.

Geography

As the CAP states, "The Cape Fear River is a water source for a number of communities downstream of the Site. Raw water intakes are located at Bladen Bluffs and Kings Bluff Intake Canals, located approximately 5 miles and 55 miles downstream from the Site, respectively."²⁶

developing drinking water guidelines for perfluoroalkyl acids: Contaminants of emerging concern, 15 PLoS Biol e2002855 (2017); Melissa Gomis, From emission sources to human tissues: modelling the exposure to per- and polyfluoroalkyl substances, (2017); Nan Sheng et al., Cytotoxicity of novel fluorinated alternatives to long chain, 92 Archives of Toxicol. 359 (2017); Melisa Gomis et al., A modeling assessment of the physicochemical properties and environmental fate of emerging and novel per- and polyfluoroalkyl substances, 505 Sci. of the Total Environ. 981 (2014); J.M. Rae et al., Evaluation of chronic toxicity and carcinogenicity of ammonium 2,3,3,3- tetrafluoro-2-(heptafluoropropoxy)-propanoate in SpragueDawley rats, 2 Toxicol. Rep. 939 (2015).

 ²⁵ Product – Chemical Profile for Perfluoroalkyl and Polyfluoroalkyl Substances (PFASs) in Carpets and Rugs at 29, California Department of Toxic Substances Control (2018).
 ²⁶ CAP at xii

These raw water intakes supply water to the CFPUA, the BCPU, and the Pender County Utility, among others.

The PFAS in the Fayetteville Works facility's wells and surface water drainage features, and found in the drinking water supplies of DRCs, originated from both Chemours and DuPont. DuPont began discharging GenX and other PFAS into the Cape Fear River as early as 1980. Such releases continued to occur after Chemours was spun off from DuPont in 2015.

Historically there have been three release routes of PFAS from Fayetteville Works to the environment, and these release routes continue to impact DRCs:

- emissions to air that have settled on more than 70-plus square miles in the Cape Fear River watershed, and migrate into the Cape Fear River;
- releases of process water to subsurface soil and groundwater that migrates into the Cape Fear River; and
- 3) releases of process wastewater directly into the Cape Fear River.

These release pathways are now being controlled by Chemours, and as Chemours states,

they "have resulted in secondary sources of PFAS in the environment to groundwater and surface

water receptors."²⁷ As stated in the CAP:

Historical releases resulted in the following secondary sources of PFAS being present in the environment:

• PFAS in unsaturated soils from aerial deposition infiltrating to groundwater. Aerial deposition has resulted in a distributed, nonpoint source of PFAS in onsite and offsite soils that represent a secondary source to groundwater. Infiltrating rainfall has transported these PFAS downward to groundwater.

• PFAS in soils and groundwater from Site process water releases. Process water leaks in the manufacturing areas resulted in PFAS in Site soil and groundwater. Based on the hydrogeology of the Site, these PFAS are detected in the Perched Zone, Surficial Aquifer, or

²⁷ CAP at xii.

Black Creek Aquifer and then migrate towards primarily the Cape Fear River and Old Outfall 002 with some component reaching Willis Creek.²⁸

III. The CAP Fails to Protect Human Health

The CAP fails to protect human health, because it does nothing to address the ongoing contamination of DRCs' water supplies with PFAS.

To address PFAS in the environment from past (*i.e.*, legacy) releases, the CAP developed objectives and cleanup goals to guide the evaluation and selection of corrective actions. The CO's remedial and management goals for Fayetteville Works are:

- reduce the total loading of PFAS originating from the facility to the Cape Fear River by at least 75 % from 2017 levels (CO paragraph 16);
- provide whole-building filtration units and RO units to qualifying surrounding residents with water exceeding the 10/70 Action Levels (CO paragraphs 19 and 20);
- comply with 15A NCAC 02L .0103 ("2L Rules") (CO paragraph 16), including following the policy for the intention of the 2L Rules "to maintain and preserve the quality of the groundwaters, prevent and abate pollution and contamination of the water of the state, protect public health, and permit management of the groundwaters for their best usage by the citizens of North Carolina"; and
- comply with other requirements of the CO.²⁹

However, nowhere in the CAP does Chemours comply with the 2L Rules, including following the policy for the intention of the 2L Rules to "prevent and abate pollution and contamination" of the Cape Fear River so that is safe for consumption by the DRCs.

²⁸ CAP at 19-20.

²⁹ CAP at 48.

A. DRC Sampling Results.

DRCs' water remains contaminated with PFAS approximately two years after Chemours ceased discharging PFAS directly into the Cape Fear River watershed via Outfall 002.

In August and October 2019, Plaintiffs' counsel collected and analyzed samples from 36 residences in Bladen, Brunswick, Columbus, Cumberland, New Hanover, and Pender Counties. Samples were collected from residences serviced by private well water and municipal water. Most of the residences that were sampled are serviced by municipal water providers including the CFPUA and the BCPU. All residences were sampled for PFAS listed in Attachment C to the Consent Order filed in *State of North Carolina, ex rel., Michael S. Regan, Secretary, North Carolina Dept. of Envtl. Quality v. The Chemours Company FC, LLC, No.* 17 CVS 580.³⁰ The results are summarized in the spreadsheet attached as Appendix A.

Twenty-seven (27) residences serviced by municipal water providers including the CFPUA and BCPU were sampled in Brunswick, Columbus, New Hanover, and Pender Counties. The samples were collected from either the tap or water heaters of the residences.

All samples collected from the taps of these residences had contaminant concentrations exceeding the threshold for installation of RO systems pursuant to Paragraph 20 of the CO; *every single sample had PFAS concentrations exceeding the 10/70 Action Levels*. It is important to recognize that the residents serviced by municipal water exceeding the Paragraph 20 criteria are drinking water that has *already been treated* by the municipal water providers. Further, these residences are contaminated *two years* after Chemours purportedly ceased its discharge from Outfall 002.

³⁰ Samples collected in June 2018 and March 2019 were analyzed in accordance with EPA Method 537 and samples collected in August 2019 and October 2019 were analyzed in accordance with EPA Method 537.1. All analysis was conducted by GEL Laboratories, LLC (Charleston, SC).

IV. The CAP Fails to Provide Adequate Plans to Remediate PFAS Contamination

The CAP fails to provide adequate plans and proposals to remediate ongoing PFAS contamination in order to protect DRCs. In some instances, Chemours offers *no solution* to a major source of continuing PFAS contamination (from aerial depositions outside Fayetteville Works), and in others offers proposals that are so tentative and under-investigated as to provide no assurance of any remedial efficacy at all. Nowhere in the CAP has Chemours calculated how long the Cape Fear River will continue to be impacted by PFAS migrating from the Cape Fear River watershed or the seeps at and near Fayetteville Works. Nor has Chemours determined how long the DRCs' water will continue to exceed the 10/70 Action Levels. Given the absence of such critical information, it is safe to assume that Chemours knows that the river and the DRC water supplies will be exceeded for decades, if not indefinitely.

Three major flaws are highlighted in the subsections that follow.

A. The CAP Proposes No Reduction in PFAS Loadings to the Cape Fear River from Aerial Deposition on the Cape Fear River Watershed and Consequently PFAS Will Continue to Impact the DRCs for an Indefinite Time Until RO Systems Are Installed.

The CAP proposes no reduction in PFAS loadings to the Cape Fear River from aerial deposition on the Cape Fear River watershed. As a result, PFAS will continue to impact the DRCs for an indefinite time unless and until RO systems are installed.

Aerial depositions of PFAS are a substantial source of ongoing PFAS contamination. In addition to PFAS being discharged directly into the Cape Fear River from outfalls and groundwater seeps at and near Fayetteville Works, Chemours also discharged PFAS into the air from its process operations. These PFAS then settled on the surrounding land within the Cape Fear River watershed. As stated in the CAP, "the aerial PFAS signature [*sic*] are diffuse, at lower concentrations *over a* 70+ *square mile area*..."³¹

PFAS deposition on land makes its way into groundwater. As the CAP itself states, "Historical releases resulted in . . . PFAS in unsaturated soils from aerial deposition infiltrating to groundwater. Aerial deposition has resulted in a distributed, non-point source of PFAS in onsite and offsite soils that represent a secondary source to groundwater. *Infiltrating rainfall has transported these PFAS downward to groundwater*."³² Below is a diagram from the CAP showing, with blue dots, the location of groundwater contamination—detected to date—caused by the vertical migration of PFAS from aerial deposition on the surface down to the groundwater below:

³¹ CAP at 23 (emphasis added).

³² CAP at 19 (emphasis added).



Figure 2: PFAS Signatures by Primary Source

Source: CAP at 24.

The contribution of these PFAS from aerial deposition on the Cape Fear River watershed over such a large area means that the PFAS which has now migrated vertically into the groundwater will continue to be discharged from the groundwater and over-land flow into the Cape Fear River for years to come.

The CAP acknowledges that, at a minimum, the deposition of PFAS-contaminated air emissions from Fayetteville Works have reached and contaminated soil and groundwater over 70 square miles.³³ This is a low estimate. Groundwater samples from an offsite drinking water well approximately 9.3 miles away from the Fayetteville Works facility (Well *Cumberland-1D*) tested positive for GenX.^{34 35} Therefore, the areal extent of PFAS impacts to soil and groundwater is at least 272 square miles (using the 9.3 mile distance from the facility to the contaminated well as the radius).³⁶

Although Chemours maintains that it has reduced loadings to the environment over the last two years, it has taken at best nominal measures to abate the thousands of pounds, if not tons of PFAS already emitted into the air. For example, Chemours admits that its air emissions likely contained 5 tons per year of HFPO-DA (GenX): "Air emission reductions to date, on an annualized basis for 2019, have resulted in an estimated yearly reduction of 2,150 pounds of HFPO-DA, a greater than 93% reduction."³⁷ Chemours has provided no information on the amount of other PFAS that was emitted into the air over the years and settled on the Cape Fear River watershed.

The PFAS deposited on the soil have already migrated a significant downward distance to the underlying groundwater. Although discontinuous is some areas, there is a subsurface confining layer of lower permeability silty or sandy clay that separates the surficial, shallow aquifer from a more extensive, deeper aquifer.³⁸ For example, offsite Well *Bladen-2D* was screened at 70 to 75 feet below the ground surface in the more extensive, deeper Black Creek Aquifer.³⁹ Groundwater samples from this well are contaminated with GenX and other PFAS.⁴⁰ This and many other of the

³³ CAP at xii, xvi, 23, 56, 57 and 76; and Table 4.

³⁴ CAP at App. A – On and Offsite Assessment Tables, Table A 9-4.

 $^{^{35}}$ CAP at App. A – On and Offsite Assessment Tables, Figure A4-2B. Chemours incorrectly shows on this figure that samples from Well Cumberland-1D contains < 3.8 ng/L of GenX. Samples from this well actually contain up to 5 ng/L of GenX; *see* Table A 9-4.

 $^{^{36}}$ 9.3² x π = 271.

³⁷ CAP at 29.

³⁸ CAP at 11.

³⁹ CAP at App. A – On and Offsite Assessment Tables, Table A 6-3.

⁴⁰ CAP at App. A – On and Offsite Assessment Tables, Table A 9-4.

deep on- and offsite wells were screened in the Black Creek Aquifer.⁴¹ This aquifer is composed of high permeability fine to medium sand.⁴² This aquifer is hydraulically connected to the Cape Fear River,⁴³ and groundwater in this aquifer can flow toward the Cape Fear River at 28.0 feet per day.⁴⁴

Rather than address this extensive source of contamination, Chemours has thrown up its

hands: Chemours plainly believes that it simply may never be possible to remediate the PFAS that

has been deposited on the surface-and migrated through the groundwater-into the Cape Fear

River. In the CAP, it maintains that:

The technical and economic infeasibility of Table 3+ PFAS remediation is driven by two factors, (a) the large areal extent PFAS are detected and (b) the lack of remedial technologies that are effective over large areas and effectively destroy PFAS mass in-situ at a technically achievable and affordable scale. To date Table 3+ PFAS have been detected over an area of 70+ square miles (over 45,000 acres). The size of the area encompasses hundreds of private land parcels and any remedial construction activities using currently available remedial technologies (excavation and groundwater extraction) would be very disruptive to the local community and this disruption would continue for a lengthy period of time. Any remedy which in principle could help make progress towards PQLs over this large area would cost in the billions to tens of billions of dollars.... Additionally, there are no currently available remedies that are expected to be able to meet **POLs over an area this large.**⁴⁵

Simply put, Chemours is proposing no measures to remediate the 70+ square miles of

historic PFAS contamination that has percolated down into the groundwater and remains a source

of contaminant loading to the river.

⁴¹ CAP at App. A – On and Offsite Assessment Tables, Table A 6-3.

⁴² CAP at 11.

⁴³ CAP at 12 and 70.

⁴⁴ CAP at App. A – On and Offsite Assessment Tables, Table A 6-4.

⁴⁵ CAP at 56.

To avoid taking any measures to remediate this contamination, Chemours has simply said, *first*, that it is not technologically and economically feasible, and *second*, that its Human Health Screening Level Exposure Assessment (HH-SLEA) and Ecological Assessment show that there is *no need to do anything* to remedy these harms.

Chemours is wrong. Not only are its assessments flawed, *see* Section V below, but it is clear that these sources of contamination *continue to contaminate the Cape Fear River water supply and, as a result, the homes of DRCs*. What Chemours has *not* addressed is the technological and economic feasibility of installing RO systems for DRCs. And it is clear that, in fact, the only means of protecting these DRCs is to provide them with RO systems.

B. Remediation of the Groundwater Seeps at Fayetteville Works Will Also Span an Indefinite Timeframe Further Necessitating the Installation of RO Systems at the DRCs.

Unlike the offsite aerial depositions discussed above, Chemours has agreed to engage in some remedial measures to clean up PFAS at its own Fayetteville Works facility. But as explained below, these measures are highly speculative, unlikely to work, and are projected to extend over a long period of time. Because this source of PFAS contamination will continue to affect DRCs for the foreseeable future, there is all the more need to protect DRCs by installing RO systems now.

The CAP presents nine purported remedial actions and two interim actions for discharges at and near Fayetteville Works including groundwater seeps, Willis Creek, Georgia Branch Creek and Old Outfall 002. The overall schedule for implementation and expected reductions are shown below in Table ES2.

| | Loading | Duration | Year | | | | | |
|---|-----------------|----------|----------|------|------|------|------|------|
| Proposed and Provisional Remedial Alternatives | Reduction | (Years) | 2019 | 2020 | 2021 | 2022 | 2023 | 2024 |
| Air Abatement Controls and Thermal Oxidizer ¹ | | 1 | ~ | | | | | |
| Conveyance Network Sediment Removal - Outfall 002 ² | NQ ³ | 1 | - | | | | | |
| Capture and Treat Old Outfall 002 | | 1 | | | | | | |
| Terracotta Pipe Replacement - Outfall 002 | | 2 | | | | | | |
| Stomswater Pollution Prevention Plan - Outfall 002 | | 1 | | | | | | |
| Groundwater Intrusion Mitigation - Outfall 002 | 0.7% | 2 | | | | | | |
| Interim Action - CFR Seeps | NQ ³ | 2 | | | | | | |
| Interim Action - Onsite Groundwater | NQ ³ | 1 | | | | | | |
| Targeted Stomnwater Control - Outfall 002 | 1.3% | 4 | | | | | | |
| Ex Situ Capture and Treatment - CFR Seeps ⁴ | 33% | 4 | | | | | | |
| Onsite Groundwater Treatment | 18% | 5 | | | | | | |
| Cumulative Estimated Total Table 3+ PFAS River Reductions to River ⁵ | | | <2% | 26% | 27% | 43% | 60% | 79% |

 Table ES2: Overall Estimated Reductions Plan Schedule and Reductions to Cape Fear River Total Table 3+ PFAS

 Loadings

Notes

- Schedule for multiple alternatives are dependent upon permitting requirements.

- Loading reductions to CFR based on average of May, June, Sep. 2019 data

- Duration listed for implementation

1 - Scheduled implementation is December 31, 2019.

2 - Completed October 2019.

3 - Anticipated reduction from action cannot be quantified at present.

4 - Assumed to be Ex Situ Capture as the permanent remedial alternative for seeps.

5 - Cumulative estimated reductions assumes:

a) that reductions are achieved at the end of the implementation period; and

b) that the time period for contingent actions is not needed.

Source: CAP at xix.

As can be seen from Table ES2, many of these 11 actions have merely "*planned* action implementation period[s]," or "time periods for *contingent* actions" with no definitive end dates and will take an indeterminate amount of time.⁴⁶ In fact, the only remedial measures that have been implemented—namely, Air Abatement Controls and Thermal Oxidizer, and Conveyance and Capture Sediment Removal—collectively mitigates *less than a 2% reduction* in loadings to the Cape Fear River.⁴⁷

Even longer time frames are indicated in Consent Order Table 10 (site cleanup goals), in which many items have planning periods and contingency periods that extend beyond *5 years*:



Action Complete Planned Action Implementation Period Time Period for Contingent Actions

⁴⁶ CAP at xvii.

⁴⁷ CAP at 33, Table 7.

| Media / Pathway | Cleanup Goal Basis | Near Term (2 years) | Intermediate Term (up to 5 years) | Long-Term (>5 years) |
|--------------------------|---|--|--|--|
| Cape Fear River | CO paragraph 16: minimum 75% reduction of Table 3+ PFAS Loading Reduce HFPO-DA and Table 3+ PFAS loading concentrations such that exposures continue to decrease as provided in SLEAs. | Begin implementation of interim actions proposed in this CAP to decrease Table 3+ PFAS loading to the Cape Fear River. | Complete implementation of interim actions and proposed corrective actions outlined here to reduce Table 3+ PFAS loading to the Cape Fear River by at least 75% from baseline. | Achieve 75% Table 3+ PFAS Loading Reduction; Maintain HFPO-DA and other Table 3+ PFAS in accordance with surface water standards in the Cape Fear River. |
| Old Outfall 002 | CO paragraph 12: capture dry weather flows of Outfall 002 and treat to 99% removal of HFPO-DA and PFMOAA before subsequent discharge. Supports CO paragraph 16 requirement of minimum 75% Table 3+ PFAS loading reduction in Cape Fear River. Comply with NPDES permit. | - Implement dry weather flows capture and treat system. | Maintain dry weather flows capture and treat system as long as needed. | Maintain dry weather flows capture and treat system as long as needed |
| Onsite Groundwater Seeps | As per Paragraph 12 Cape Fear River PFAS Loading Reduction Plan reduce Total Table 3+ PFAS mass loading to Cape Fear River. Supports CO paragraph 16 requirement of minimum 75% loading reduction in Cape Fear River. | Begin implementing and optimizing interim actions and long-term remedies. | Seep treatment remedy operating to reduce Table 3+ PFAS loading as long as needed | - Maintain seep treatment remedy as needed |
| Willis Creek | Creek - Achieve economically and technically feasible reductions to support CO paragraph 16 requirement of minimum 75% Table 3+ PFAS mass loading reduction in Cape Fear River. - Implement thermal or abatement controls to groundwater concentur offsite groundwater concentur offsite groundwater of creek. - Reduce discharge to Willis Creek of onsite Table 3+ PFAS with a process water signature - Design and begin con for onsite groundwater will reduce PFAS mas Black Creek Aquifer | | Maintain air abatement controls. On Site Groundwater Remedy will address PFAS loading to Willis Creek. | Maintain air abatement controls. Maintain groundwater remedy as needed |

| Media / Pathway | Cleanup Goal Basis | Near Term (2 years) | Intermediate Term (up to 5 years) | Long-Term (>5 years) |
|--------------------------|--|--|---|---|
| Onsite and Offsite Soils | Maintain human exposures to HFPO- DA below the NCDHHS reference dose (achieved per HH-SLEA results and replacement drinking water actions) | Implement thermal oxidizer and other air abatement controls to reduce PFAS deposition rates to on and offsite soils. | Maintain thermal oxidizer and other air abatement controls to reduce PFAS deposition rates to on and offsite soils. | Maintain thermal oxidizer and other air abatement controls to reduce PFAS deposition rates to on and offsite soils. |
| | Maintain ecological exposures below adverse effects levels (achieved per Ecological SLEA results) | | | |
| | - 2L requires removal or control of secondary sources to groundwater such as contaminated soils. Per information presented in Section 3.6 much more mass is in groundwater than in soils suggesting soil remediation would have a reduced benefit. | | | |
| Outfall 002 | The NPDES permit will develop effluent limits for Outfall 002 Outfall 002 actions proposed in Chemours CO paragraph 12 Cape Fear River PFAS Loading Reduction Plan | Comply with NPDES permit Begin implementing actions proposed in the Reduction Plan | Comply with NPDES permit (permit is for 5 years) Implement actions proposed in the Reduction Plan | Re-apply for NPDES permit Maintain actions proposed in the Reduction Plan |

| Media / Pathway | Cleanup Goal Basis | Near Term (2 years) | Intermediate Term (up to 5 years) | Long-Term (>5 years) |
|----------------------|--|--|--|--|
| Georgia Branch Creek | Achieve economically and technically feasible reductions to support CO paragraph 16 requirement of minimum 75% Table 3+ PFAS mass loading reduction in Cape Fear River | Implement thermal oxidizer and other air abatement controls to reduce offsite groundwater concentrations over time; | - Maintain air abatement controls. | - Maintain air abatement controls. |
| Onsite Groundwater | Reduce discharge of PFAS with a PFAS process water signature to Cape Fear River and to Willis Creek to support CO paragraph 16 requirement of minimum 75% Table 3+ PFAS mass loading reduction in Cape Fear River (Process water signature discharge to Old Outfall 002 is addressed by Old Outfall 002 capture and treatment system; PFAS historically released in process water does not discharge to Georgia Branch Creek) - Comply with 2L Rules | Implement interim actions. Conduct pre-design investigations for on- site groundwater remedy and treatment. | - Implement groundwater remedy. | Evaluate 2L cleanup standards based on potentially existing cleanup standards developed from newly available scientific studies and potentially more effective remedial approaches recently developed. Presently both technically and economically infeasible to cleanup onsite groundwater to PQLs. |
| Offsite Groundwater | Provide replacement drinking water to surrounding residents where groundwater based on requirements of CO paragraphs 19 and 20 Maintain human exposures to HFPO- DA below the North Carolina Department of Health and Human Services (NCDHHS) reference dose (achieved per HH-SLEA results and replacement drinking water actions) | Provide replacement drinking water. Implement thermal oxidizer and other air abatement controls to reduce offsite groundwater concentrations over time. | Maintain provision of replacement drinking water as long as needed Maintain air abatement controls. | Maintain provision of replacement drinking water as long as needed Maintain air abatement controls. |

Source: CAP at 53, et seq.

Even worse, the CAP couches much of its language about the efficacy of its remedial plans in highly tentative language filled with caveats and escape hatches.

For instance, according to the CAP, the full extent of offsite PFAS contamination originating from Fayetteville Works is still being investigated.⁴⁸ Chemours acknowledges that extensive investigations and design adaptations will be necessary before contamination can be remediated.

As another example, Chemours states that before groundwater discharges to the Cape Fear

River can be addressed, it must "proceed in developing the detailed design, including collection

of <u>extensive</u> pre-design data, for a long-term groundwater containment approach."⁴⁹ Chemours

continues:

Extensive investigation, analysis, and numerical model refinement would be required to properly design a remedy of this scale. A geotechnical investigation would be required along the alignment (anticipated boring frequency every 100 linear feet) to

⁴⁸ CAP at 34, Sec. 4.1.1.

⁴⁹ CAP at xvii.

determine the depth and penetration resistance of the confining unit. Additional delineation consisting of borings, wells, and in-river flux analyses may also be utilized to properly target the optimal areas for containment needed to achieve the corrective action objectives. Finally, pilot testing, consisting of extraction well drilling and aquifer testing at multiple locations along the alignment, would be performed to determine the optimal well spacing and extraction rates. It is anticipated that in the course of two years, these activities would allow for model refinement and completion of design and permitting effort. In the absence of this pre-design data, the following discussion of a long-term groundwater remedy is *still highly conceptual*.⁵⁰

The CAP is replete with other examples of Chemours' signaling that its remediation plans are indefinite and will take a long time. The CAP proposes both short term interim remedial measures and long-term remedial alternatives, but both types of measures have long time horizons. As an example, the *long-term* remedial alternative for Black Creek Aquifer consists of the construction, operation and maintenance of a barrier wall and groundwater capture. But Chemours proposes no deadline for this proposal. As another example, the "interim remedial alternative advanced for groundwater consists of installing submersible electric pumps in seven existing Black Creek monitoring wells and pumping the water to the OOF2 treatment plant for treatment and discharge."⁵¹ Chemours estimates that this *interim* remedial measure will take *two years to complete*.

Critically, Chemours states openly that it has no estimated time for completion of this remedy: "The schedule for implementation of a groundwater remedy is included in Section 6.5 of this document; the pre-design investigation through detailed design and permitting is expected to take two years. *At the conclusion of the effort*, Chemours would present a detailed onsite remedial design to DEQ for approval."⁵²

⁵⁰ CAP at 71.

⁵¹ CAP at 70.

⁵² CAP at 75

Even more problematic than the extended timeline is that these proposed remedial actions are unlikely to be effective. As an example, the CAP proposes an interim action of extracting groundwater from existing monitoring wells screened in the Black Creek Aquifer-which has one of the largest PFAS loading contributions to the Cape Fear River-and treatment prior to discharge. As an interim remedial approach, Chemours proposes to place small submersible pumps in seven existing onsite groundwater monitoring wells. In an effort to capture a small portion of the PFAS-contaminated groundwater before it reaches the Cape Fear River, these wells would be pumped at a total of 14 gallons per minute (gpm).⁵³ Following DEQ's approval of the CAP, Chemours expects it will take 12 months to install and operate these small submersible pumps. The operation of these pumps would be monitored for another 12 months. These two years are considered by Chemours to be a "contingent action" and apparently could be modified or discontinued if the pumps do not operate appropriately.⁵⁴ Regardless, this interim remedial approach or contingent action is unlikely to measurably mitigate the discharge of PFAScontaminated groundwater to the Cape Fear River. Chemours' own analysis states that a series of purpose-built extraction wells spaced at 50-foot intervals near the Cape Fear River would have to pump at least 4,430 gpm to effectively remediate PFAS contamination.⁵⁵ This groundwater pumping rate is 317-times higher than Chemours' proposed interim measure of 14 gpm. The contrast between Chemours' plan and the reality of its implementation is highlighted in the table below:

| Pumping Rate Needed to | Pumping Rate Proposed | | |
|--------------------------|-----------------------|--|--|
| Remediate PFAS | by Chemours | | |
| 4,430 gallons per minute | 14 gallons per minute | | |

⁵³ CAP at 70.

⁵⁴ CAP at Table 13.

⁵⁵ CAP at Table 8.

With respect to long-term permanent remedial measures, in an effort to downplay

Chemours' commitment to effectively remediate onsite contamination, the CAP states openly that

the efficacy of a long-term remedy is simply uncertain:

The corrective actions proposed in this CAP will be refined over time as both remedial technologies and understanding advance. PFAS are an *emerging class of contaminant*, with the Table 3+ PFAS present at the Site from this facility one of the newer sets of PFAS being examined by the remediation industry. *The state of knowledge regarding the fate and transport properties, toxicological characteristics, and potential remedial approaches for PFAS and Table 3+ PFAS are continuing to evolve and advance.*⁵⁶

In addition, the CAP also states openly that the time horizon for remediation is highly

uncertain:

Extensive investigation, analysis, and numerical model refinement would be required to properly design a remedy of this scale, including but not limited to geotechnical borings, contamination distribution investigations, in-river flux analyses, and pilot testing. It is anticipated that in the course of two years, these activities would allow for model refinement and completion of the design and permitting effort. *In the absence of this data, the proposed long-term groundwater remedy is still highly conceptual, and it is not presently possible to conclude with confidence whether this alternative is economically feasible.* At the conclusion of the PDI, Chemours will either present a detailed onsite remedial design or a remedial alternative to DEQ for approval...⁵⁷

In other words, even though the Consent Order required concrete plans for remedial action,

Chemours has effectively said: more studies are needed, and, even if we conduct them, there's no

guarantee they will be technically or economically feasible.

Most alarming, however, is Chemours' suggestion that it should not be held to the 2L Rules

at all. Specifically, the CAP states that "NCDEQ and Chemours may need to consider alternate

cleanup standards conceived under 15A NCAC 02L .0106 (a) and (i) together and 15A NCAC

⁵⁶ CAP at 1.

⁵⁷ CAP at xvii.

02L .0106 (k) individually or risk-based remediation as described by N.C.G.S. § 130A-310.66 *et seq*.^{*58} Chemours suggests rewriting the Consent Order rather than complying with its obligations to protect public health, as required by the 2L Rules and the Consent Order. In short, *Chemours is reserving its rights to never restore the Cape Fear River to levels that will protect the DRCs*. In light of Chemours' sidestepping of its responsibilities under the Consent Order—and the tenuousness of the proposals made in the CAP—the only method to protect the DRCs in the short and foreseeable future is for them to be provided with RO units.

There is also no certainty that total loadings from groundwater into the Cape Fear River will decrease within a certain timeframe. That is, there is no definitive decreasing trend in PFAS-contaminated water reaching the Cape Fear River. For example, PFAS-contaminated surface water is present in Georgia Branch Creek, which discharges to the Cape Fear River. For example, while "Total Table 3+ PFAS concentrations from wells PW-02 and PW-14 were approximately 100 times lower in the resampled results compared to the original samples (15,000,000 to 140,000 ng/L and 18,000,000 to 160,000 ng/L respectively),"⁵⁹ there is no indication that the purported decrease is due to factors outside of chemical loadings, such as dilution from increased groundwater flowage rates and volumes.

Chemours also has not evaluated whether the decrease is asymptotic and will reach a plateau which still contributes extensive loadings to the Cape Fear River—in other words, Chemours has provided no analysis of whether PFAS reductions have stabilized and are likely to decrease only nominally over time, or whether PFAS reductions will follow a downward trend. Indeed, Chemours has not even attempted to conduct this analysis at all. Accordingly, all that Chemours can offer is that "The concentrations in these wells will continue to be monitored as part

⁵⁸ CAP at xvi.

⁵⁹ CAP at 20.

of monitoring plan activities described in Section 7.^{w60} As another example, according to the CAP, in some instances, the concentration of PFAS in monitoring wells is actually *increasing* with time. As stated in the CAP, "Total Table 3+ PFAS concentrations for wells PIW-7S and PW-06 following redevelopment and resampling were greater than previous results. For example, total Table 3+ PFAS concentrations for well PW-06 increased from 3,000 ng/L to 4,400 ng/L while well PIW-7S increased from 17,000 ng/L to 54,000 ng/L.^{w61}

There is also no certainty that total loadings from groundwater into the Cape Fear River

will decrease with distance from Fayetteville Works. As stated in the CAP:

Onsite there are four seep features with channelized flow that enter the Cape Fear River. In October 2019, ten offsite groundwater seeps - the Lock and Dam Seep and Seeps E to M - were identified on the west bank of the Cape Fear River to the south of the Site. The seeps were identified by performing a visual survey from a boat on the western side of the Cape Fear River between Old Outfall 002 and Georgia Branch Creek. Flow from these seeps ranged from seeping water from an embankment (*i.e.* trickles) to a visible small stream in one of the seeps. Results from samples collected from the seeps indicate Total Table 3+ PFAS concentrations ranged between 2,600 to 6,800 ng/L. **The seven southernmost seeps (G to M) had similar concentrations to the mouth of Georgia Branch Creek sampled in September (2,100 ng/L).⁶²**

Chemours incorrectly states that there is a decreasing trend in PFAS concentrations while moving

southward toward Georgia Branch Creek.⁶³ Although the first few seeps near the Old Outfall 002

(*i.e.*, Seeps E to G) do exhibit higher PFAS concentrations (average 1,000 ng/L of GenX), all of

⁶⁰ CAP at 20.

⁶¹ CAP at 21.

⁶² CAP at 21.

⁶³ CAP at App. D – Southwestern Offsite Seeps Assessment, Offsite Seeps Assessment Memo, December 31, 2019, at 3.

the next six downstream seeps over the next 0.6 miles exhibit similar PFAS concentrations (average 572 ng/l GenX).⁶⁴

Further, although the ongoing *discharge* of PFAS-containing water from Outfall 002 has been reduced, the outfall is still providing about 5 percent of the mass-loading to surface water in the adjacent Cape Fear River. And the concentration of GenX and other PFAS in samples collected from Outfall 002 remain elevated.





Source: *GenX Surface Water Sampling Sites*, North Carolina Department of Environmental Quality, https://deq.nc.gov/news/key-issues/genx-investigation/genx-surface-water-sampling-sites, last visited Jan. 25, 2020.

⁶⁴ CAP at App. D – Southwestern Offsite Seeps Assessment, Offsite Seeps Assessment Memo, December 31, 2019, at Table 1 and Figure 2.

The CAP makes the completely unsupported statement that PFAS contamination will naturally reduce over time, stating, "While other media were not identified as significantly contributing to overall intake, human exposure to PFAS in all environmental media will continue to decrease over time as a result of Facility air emissions reductions."⁶⁵ However, Chemours has provided *no analytic data, statistics, calculations or regression analyses to support this conclusion*. In fact, as discussed elsewhere throughout these comments, it is likely that the reservoirs of PFAS existing in soil, groundwater and discharges throughout the Cape Fear River watershed will discharge into the Cape Fear River and be consumed by the DRCs for years if not decades to come. Conversely, according to the CAP, "Table 3+ PFAS are not expected to degrade in a reasonable time period in the environment, and therefore this is not a mechanism that will support concentration reductions."⁶⁶ In short, the CAP states: "Based on professional opinion the costs for on and offsite remediation to PQLs would exceed billions to potentially tens of billions of dollars *and the timeframe would be on the order of multiple decades*."⁶⁷ (emphasis added)</sup>

Once again, Chemours' inability to remediate the Cape Fear River within any given timeframe means that DRCs will be exposed to PFAS unless they are provided with RO systems. The DRCs simply cannot wait until Chemours eventually—if ever—effectively implements these permanent remedial measures. In the meantime, the DRCs should be provided with RO systems and bottled water. This solution is both economically and technologically feasible, and DEQ should order it pursuant to Chemours' obligations under the CO.

⁶⁵ CAP at 38.

⁶⁶ CAP at 58.

⁶⁷ CAP at 58.

C. The Differing Rates of PFAS Migration Through Air, Soil, Groundwater, Sediment and River Water Means That PFAS Will Reach the DRCs Not as a Single "Slug" but Rather over Many Years.

The differing rates of PFAS migration through air, soil, groundwater, sediment, and river water means that PFAS will reach the DRCs not as a single "slug" but rather gradually over many years. In lay terms, each PFAS has a different "stickiness" coefficient, meaning that although some PFAS adhere strongly to surfaces, others are less adherent. The technical term for this is "retardation." Chemours neglects to consider these disparate migration rates.

To explain their variations, Section 3.2 of the CAP provides a description of the physical and chemical properties of Table 3+ PFAS found in the air, soil, groundwater, sediment, and river water and their fate and transport. This table makes clear that PFAS will continue to reach DRCs for an indefinite amount of time due to the differing retardation rates for different PFAS. Pursuant to CO Paragraph 27, Chemours funded a study analyzing the fate and transport characteristics of identified PFAS compounds originating from Fayetteville Works in air, surface water, and groundwater.⁶⁸ The findings of this study establish that although many of the Attachment C PFAS are highly mobile (which explains why they will continue to migrate from and near Fayetteville Works to the municipal water intakes), some of the other Attachment C PFAS are less mobile and thus will continue to be released and reach the intakes for years to come.

Section 3.2 of the CAP summarizes the PFAS values for the octanol-water partition coefficient (K_{ow}), organic carbon-water partition coefficient (K_{oc}), and surface tension of water, which determine the propensity and degree to which PFAS bind to organic carbon in the soil, groundwater, sediment, and river water:

Generally, Table 3+ PFAS are expected to be mobile in the environment given the presence of charged head groups and ether

⁶⁸ Geosyntec, 2019c. Site Associated PFAS Fate and Transport Study Pursuant to Consent Order Paragraph 27 (June 24, 2019).
bonds, but they will experience some retardation due to sorption to soils. For some Table 3+ PFAS, mobility may be enhanced relative to straight-chain, non-ether PFAS by their branched structure and the presence of two charged head groups. The mobility of the Table 3+ PFAS will be retarded by various chemical processes but will likely have lower retardation than long-chain PFAS without ether bonds. Chemical processes expected to have the most impact on mobility are sorption to naturally occurring organic carbon in soil and, in the unsaturated soil zone, preferential partitioning to the air-water interface.⁶⁹

The CAP continues, "Kow is a standard parameter used for estimating bioconcentration

factors. . . Other mechanisms of sorption can also include the potential for PFAS, including Table

3+ compounds to bioaccumulate in organisms."⁷⁰

Finally, Chemours' Table 2 demonstrates that PFAS has differing "Measured Log Kow and

Calculated Log Koc Values" which indicates that Chemours' PFAS will reach the DRCs over an

extended period of time.

⁶⁹ CAP at 13.

⁷⁰ CAP at 14.

| Table 3+ PFAS | ${ m Log}~{ m K_{ow}}^1$ at pH 5 | $Log {K_{ow}}^2$ at pH 8 | $Log \ K_{\text{oc}} \ (L/Kg)^2 \ at \ pH \ 5$ |
|----------------|----------------------------------|--------------------------|--|
| MMF | <2.92 (1.08)* | <3.11 (1.09)* | |
| DFSA | <2.90 (1.19)* | <3.11 (1.05)* | |
| MTP | <2.90 (2.19)* | <3.11 (2.42)* | 0.52 |
| PPF | <2.93 (2.43)* | <2.98 (2.48)* | 0.67 |
| PFMOAA | <2.82 (2.45)* | <2.83 (2.43)* | 0.89 |
| NVHOS | 2.92 | 2.93 | 0.95 |
| R-EVE | 3.04 | 3.14 | 1.01 |
| PMPA | 3.05 | 3.05 | 1.02 |
| Byproduct 4 | 3.09 | 3.19 | 1.04 |
| Byproduct 5 | 3.14 | 3.23 | 1.07 |
| PFO2HxA | 3.32 | 3.30 | 1.17 |
| PEPA | 3.63 | 3.60 | 1.35 |
| PES | 3.80 | 3.78 | 1.44 |
| PFECA B | 3.98 | 3.95 | 1.54 |
| PFO3OA | 4.17 | 4.13 | 1.65 |
| HFPO-DA | 4.24 | 4.23 | 1.69 |
| Byproduct 6 | 4.61 | 4.57 | 1.90 |
| Hydro-EVE Acid | 4.68 | 4.66 | 1.94 |
| Byproduct 2 | 4.72 | 4.68 | 1.96 |
| PFECA-G | 4.79 | 4.77 | 2.00 |
| PFO4DA | 4.98 | 4.95 | 2.11 |
| PFESA-BP1 | 5.09 | 5.06 | 2.17 |
| EVE Acid | 5.10 | 5.06 | 2.17 |
| PFO5DA | 5.78 | 5.72 | 2.56 |

Table 2: Table 3+ Measured Log Kow and Calculated Log Koc Values

Source: CAP at 15.

Thus, Chemours itself admits that different PFAS will be transported at different rates due to different retardation factors. As provided in the CAP, "The retardation factor estimates suggest in the saturated zone *approximately half* of the Table 3+ PFAS will experience minimal retardation where travel times will be similar to groundwater travel times; *i.e.*, factors were close to 1."⁷¹ The remaining half will experience a wide array of travel times with many likely taking years to reach the DRCs' water supply.

⁷¹ CAP at 28.

This means that different PFAS, traveling at different speeds, will continue to impact the water consumed by the DRCs at differing times for years if not decades to come. The only means to protect the DRCs during this extended time period is to provide them with RO systems.

V. <u>The CAP Fails to Provide an Adequate, Scientifically Sound, and Unbiased</u> <u>Risk Assessment as Required by CO Paragraph 14.</u>

Chemours has yet to properly quantify the risks of PFAS exposure to DRCs and all other individuals affected by Chemours' contamination. In particular, Chemours has failed to comply with CO Paragraph 14's requirement to establish that Attachments B and C PFAS do not pose an unacceptable risk to human health. And more importantly, because there is no basis to conclude that consumption of water contaminated with Attachment C PFAS at levels in excess of the Action Levels does not pose an unacceptable risk to human health, Chemours must provide the DRCs with RO systems and bottled water while an evaluation of the health risks and toxicity values is being executed.

Chemours' toxicity assessment found in Appendix F to the CAP, also referred to as the HH-SLEA, contains numerous errors and underestimates risks to human health. In particular, Chemours has yet to properly quantify the risks of PFAS exposure to DRCs and all other individuals affected by Chemours' contamination. Chemours fails to calculate toxicity values (and risks) for 19 out of 20 PFAS, focusing all of its efforts on GenX, the one PFAS for which DuPont and Chemours have produced at least *some* toxicity evaluations. In addition to Chemours' complete failure to assess toxicity for most of the chemicals at issue (or the interplay between those chemicals amongst one another and with legacy PFAS contamination that remains in area residents' bodies), Chemours' analysis fails to (a) follow standard EPA guidance for deriving toxicity values; (b) adequately address the past decade of scientific literature on GenX's toxicity;

(c) properly weigh and account for the toxicity and human health risks of GenX, including immunotoxicity; and (d) account for all necessary risks and toxicity information associated with drinking contaminated water. Chemours also manipulates its conclusions by making improper and scientifically unsound assumptions that mask the true risks associated with drinking PFAS-contaminated water and fail to account for exposure risks to sensitive subpopulations.

Incredibly, Chemours also fails to address its own data, identified in its TSCA Section 8(e) submissions, demonstrating that GenX-induced toxic effects include liver toxicity (*e.g.*, hypertrophy, single-cell necrosis, peroxisome proliferation, and increased liver weight relative to body weight), hematological effects (*e.g.*, decreased red blood cell count, hemoglobin, and hematocrit), kidney toxicity (*e.g.*, increased kidney weight, necrosis, and hyperplasia), developmental effects (*e.g.*, body weight changes), immune effects (*e.g.*, T cell-dependent antibody response [TDAR] suppression and lymphocyte increases), and suggestive evidence of tumor formation (*e.g.*, liver and pancreatic acinar cell tumors).⁷² Yet the HH-SLEA fails to address any of these impacts.

Chemours' deeply flawed HH-SLEA purports to quantify the risks of exposure of offsite human receptors to 20 PFAS listed in Table 3+ of the HH-SLEA (only one of which is GenX) but not the synergistic effect of these chemicals upon one another (or indeed other PFAS and chemicals found in the DRCs' tap water) or together with PFAS (including PFOS and PFOA) that have bioaccumulated in residents' bodies as a result of DuPont's and Chemours' historical contamination of the water supply. The HH-SLEA purports to quantify exposures of offsite human receptors to released PFAS for several receptor-exposure scenarios, and to provide a provisional

⁷² Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3) Also Known as 'GenX Chemicals', EPA-823-P-18-001 Public Comment Draft at 47, U.S. EPA (Nov. 2018), https://www.epa.gov/sites/production/files/2018-11/documents/genx_public_comment_draft_toxicity_assessment_nov2018-508.pdf.

human health hazard characterization for GenX (HFPO-DA) based on quantified intakes and the North Carolina Department of Health and Human Services ("DHHS") 2017 draft oral reference dose (RfDo).

The HH-SLEA violates fundamental, generally accepted principles of toxicology and riskassessment practice and ignores standard regulatory guidance, as the below subsections make clear.

First, Chemours fails to calculate toxicity values (and risks) for 19 out of 20 PFAS listed in Table 3+, focusing all of its efforts on GenX, the one PFAS for which DuPont and Chemours have produced at least *some* toxicity evaluations.⁷³

Second, even setting aside Chemours' failure to adequately analyze the toxicity of 95% of the PFAS listed in Table 3+, Chemours' analysis itself is deeply flawed, including by its failure to (a) follow standard EPA guidance for deriving toxicity values; (b) adequately address the past decade of scientific literature on GenX's toxicity; (c) properly weigh and account for the toxicity and human health risks of GenX, including immunotoxicity and potential carcinogenicity; and (d) account for all necessary risks and toxicity information associated with drinking contaminated water.

Third, Chemours also manipulates its conclusions by making improper and scientifically unsound assumptions that mask the true risks associated with drinking PFAS-contaminated water.

Fourth, Chemours fails to consider its own studies prepared pursuant to its consent decree with the EPA pursuant to Section 8(e) of TSCA.

Fifth, Chemours' methodological flaws are underscored by its failure to account for exposure risks to sensitive subpopulations.

 $^{^{73}}$ CAP at xv; Section 4.2.

A. The HH-SLEA Violates Generally Accepted Principles of Toxicology and Risk-Assessment and Ignores Standard Regulatory Guidance.

The CAP and HH-SLEA provide an insufficient analysis of the toxicity associated with both GenX exposures and exposure to the other 19 PFAS. Chemours has also failed to account for the synergistic effects of the 19 PFAS—as well as other contaminants in the DRCs' water supply and, critically, with PFOS, PFOA and other legacy PFAS that remain in residents' blood as a result of historical contamination of the water supply by DuPont and Chemours, as was found in the blood samples taken as a part of the GenX Exposure Study. The HH-SLEA does not meet generally accepted standards of toxicology and risk assessment. Considerable and fundamental efforts must be made to conduct adequate toxicity studies to reduce the uncertainty in the current GenX toxicological database, and Chemours must also conduct the necessary toxicity studies to derive toxicity values for the 19 PFAS compounds listed in Table 3+ for which no toxicity information is presented in the CAP or HH-SLEA. In the meantime, Chemours must provide the DRCs with bottled water and RO systems because as admitted by Chemours in the CAP, "supplying whole building filtration systems and reverse osmosis units for qualifying residents offsite reduces HFPO-DA (and Table 3+ PFAS) intake by over 92%, ensuring human receptor exposures remain below hazard limits for HFPO-DA, based on the NC DHHS draft RfDo."74

> 1. Chemours fails to calculate toxicity values (and risks) for 19 out of 20 PFAS listed in Table 3+, focusing all of its efforts on GenX, the one PFAS for which DuPont and Chemours have produced at least *some* toxicity evaluations.⁷⁵

The HH-SLEA provides little information regarding actual human health risks. This is because, although the 20 PFAS contaminants listed in Table 3+ have been *identified*, Chemours

⁷⁴ CAP at xv.

⁷⁵ CAP at xv; Section 4.2.

has only presented toxicity information and data for a single PFAS (GenX), while omitting any such information for the other 19, despite the fact that thousands of North Carolina residents have been drinking water contaminated with these PFAS for years if not decades. Chemours appears to dismiss the absence of toxicity data for these 19 PFAS compounds listed in Table 3+ as unimportant; however, it is likely one or more of them, alone or in the combinations present in area drinking water, may be shown to be even more toxic than GenX alone. For this reason, the HH-SLEA does not even meet the standard of a screening risk assessment. Chemours' conclusions about the risks of drinking water contaminated with GenX and other PFAS are incorrect and grossly underestimate the risks of such exposure, including the fact that Chemours fails to differentiate between the hazard of being exposed to a single PFAS versus the hazards associated with exposure to multiple PFAS at the same time (*i.e.*, the combined effect of PFAS exposure) and with introducing additional PFAS into the blood of residents who already have accumulated PFOS, PFOA, and other PFAS in their blood.

In its HH-SLEA, Chemours states that the calculated hazard quotients, or "HQs," were "**less than 1** for residents, farmers, and gardeners exposed to soil, produce, and well water in exposure unit ("EU") 1 through EU 12, indicating potential HFPO-DA exposure is unlikely to pose a hazard, even in the absence of drinking water treatment."⁷⁶

This conclusion—of a HQ for GenX of less than 1—is wholly unsupported for the simple reason that Chemours has yet to derive *any* toxicity values for 19 of the 20 PFAS listed in Table 3+. And even accepting the existence of *some* toxicity values for GenX, significant uncertainty exists even as to that data. By its own admission, Chemours states that the toxicity data—which was taken from DuPont's 2010 EPA TSCA Section 8(e) filing—may well underestimate GenX's

 $^{^{76}}$ CAP at Section 4.2.4.

risks because (1) the studies are outdated; (2) they are based only on liver pathology (when the immune system is likely a more sensitive target organ); (3) there is no human data; and (4) toxicity was based on subchronic rather than chronic animal studies. Chemours states:

Toxicity Data. The SLEA provisional hazard characterization is based on the HFPO-DA RfDo of 1E-04 mg/kg-day adopted by the NC DHHS, which is predicated on liver toxicity endpoints from two subchronic studies in mice. There is inherent uncertainty in the use of animal toxicity data to characterize potential human health hazards and the RfDo *could potentially change as new information becomes available*. (emphasis added)⁷⁷

Chemours' statement both (a) acknowledges that the current value could potentially change as "new information becomes available," and (b) that Chemours intends to wait for other entities to generate new toxicity information (RfD) for GenX.⁷⁸ Chemours is responsible for GenX pollution, and should be responsible for funding the independent studies needed to generate "new" toxicity information.

Chemours also downplays the impact of the lack of toxicity information for the 19 other

PFAS compounds listed in Table 3+ by referring to it as mere "uncertainty":

In addition to the uncertainty associated with the HFPO-DA RfDo, the lack of toxicity information for other Table 3+ PFAS also introduces uncertainty to the HH-SLEA but data are not available to evaluate the potential effect, if any, on the conclusions [of the] hazard characterization [*sic*].⁷⁹

With regard to the "screening" levels of GenX, Chemours also insinuates that a 10 ppt

GenX level in drinking water is "safe" because it is based on an agreement:

[Hazard Quotient] estimates based on an assumption of 10 ng/L of HFPO-DA in drinking water, which is the maximum concentration in well water that would not require a treatment system, range from

⁷⁷ CAP at 39.

⁷⁸ *Id.* at 39.

⁷⁹ *Id.* at 39-40.

0.003 to 0.07 and, hence are more than an order of magnitude below a level of concern (unity or 1). 80

Although an *agreement of treatment* may have been reached with the State, a drinking water level of 10 ppt for GenX may be insufficiently protective of human health. For example, the Natural Resources Defense Council ("NRDC") conducted an independent analysis to calculate the "safe" concentration of GenX in drinking water and concluded the level should be *less* than 1 ppt. This is 10 times less than the current 10/70 Action Level for GenX. Furthermore, the NRDC's analysis was based on DuPont's 2010 TSCA Section 8(e) and toxicity studies:

If uncertainty factors that properly reflected the deficiencies in toxicity data (database, sub-chronic to chronic, children's vulnerability, human variability, animal to human differences) were used, *the combined uncertainty factor could be as high as 100,000, which would result in a MCLG of less than 1 ppt for GenX chemicals* (see Appendix F for calculations). This highlights the current considerable level of uncertainty in determining a safe level of exposure for GenX chemicals.⁸¹

The last point underscores the importance of summing health risks when multiple contaminants are present in drinking water, as set forth in the EPA's risk assessment guidance and generally accepted toxicology practice.⁸² Chemours' assumption that a GenX concentration in drinking water is safe is based on an underlying (incorrect) assumption that GenX is the *only* PFAS contaminant to which a population is exposed. When there are multiple contaminants, the EPA requires further reductions in screening risk assessments to account for similar contaminants that may also pose risks. The EPA guidance states:

5.15 Screening Sites with Multiple Contaminants

⁸⁰ *Id.* at 39.

⁸¹ A. Reade, T. Quinn, and J. S. Schreiber, *PFAS in Drinking Water 2019: Scientific and Policy Assessment for Addressing Per-and Polyfluoroalkyl Substances (PFAS) in Drinking Water* at 43, Natural Resources Defense Council (Apr. 12, 2019), https://www.nrdc.org/sites/default/files/media-uploads/ nrdc_pfas_report.pdf.

⁸² Regional Screening Levels (RSLs) User's Guide at 5.15, U.S. EPA (Nov. 2019), https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide.

The screening levels in the tables are calculated under the assumption that only one contaminant is present. Users needing to screen sites with multiple contaminants should consult with their regional risk assessors. The following sections describe how target risks can be changed to screen against multiple contaminants and how the ratio of concentration to RSL can be used to estimate total risk.⁸³

The EPA provides guidance on how to adjust the HQs for single chemicals when multiple

contaminants are present (which depends on how many of the other 19 PFAS listed in Table 3+

are detected):

The calculator on this website can be used to generate SLs based on any THQ [target hazard quotient] or target cancer risk (TR) deemed appropriate by the user. The THQ input to the calculator can be modified from the default of 1. How much it should be modified is a user decision, but it could be based upon the number of contaminants being screened together. For example, if one is screening two contaminants together, then the THQ could be modified to 0.5. If ten contaminants are being screened together, then the THQ could be modified to 0.1. The above example weights each chemical equally; it is also possible to weight the chemicals unequally, as long as the total risk meets the desired goal. The decision of how to weight the chemicals is likely to be site-specific, and it is recommended that this decision be made in consultation with the regional risk assessor.⁸⁴

The other option the EPA provides for assessing the risk of exposure to multiple, related

chemical compounds (and which Chemours also failed to follow) is to first develop "safe" drinking water concentrations for each of the 20 PFAS listed in Table 3+ (which are sometimes referred to as maximum contaminant level goals [MCLGs] or screening levels [SLs], as shown below). The detected concentration in drinking water for each of the individual PFAS compounds is then divided by those concentrations (shown as Cx, Cy,...Cz); finally, the quotients are added together, or summed. If the summed THQ exceeds 1.0, the contaminants may pose an unacceptable risk.

⁸³ Id.

⁸⁴ *Id.* at 5.15.1.

What is important to recognize about this approach is that the summed THQ can far exceed acceptable risk levels even when each of the individual PFAS compounds does not exceed its own MCLG (or SL).

4. For non-cancer hazard estimates, divide the concentration term by its respective non-cancer SL designated as 'n' and sum the ratios for multiple contaminants. The cumulative ratio represents a non-carcinogenic hazard index (HI). A hazard index of 1 or less is generally considered 'safe'. A ratio greater than 1 suggests further evaluation. Note that carcinogens may also have an associated non-cancer SL that is not listed in the SL Table. To obtain these values, the user should view the Supporting Tables. See equation below $Total Hazard Index = \left[\left(\frac{C_x}{SL_x} \right) + \left(\frac{C_y}{SL_y} \right) + \left(\frac{C_z}{SL_y} \right) \right] \times THQ$

Source: Regional Screening Levels (RSLs) User's Guide at 5.15.1, U.S. EPA (Nov. 2019), https://www.epa.gov/risk/regional-screening-levels-rsls-users-guide.

This error is critical. This issue is compounded because other chemicals have been found in the DRCs' water supply that originated from Fayetteville Works. Chemours followed none of these approaches, and its assertion that the Action Level for GenX is safe is unsupported and contrary to the established methodological approaches when populations are exposed to multiple, similar compounds.

Chemours should be required to generate adequate toxicity information for the 19 untested PFAS listed in Table 3+ that DuPont (and later Chemours) emitted and discharged into the environment. Chemours, as the manufacturer, is not only most familiar with the chemical/physical properties of its own chemical products, but has profited from their use or sale for more than a decade, which is more than sufficient time to have completed toxicological testing on the remaining 19 PFAS compounds listed in Table 3+. The HH-SLEA and CAP do not explain why DuPont and Chemours failed to perform any such testing in the past, and there is no explanation for why Chemours continues to delay testing today. It is incumbent on Chemours to explain how

it intends to finalize the HH-SLEA and CAP risk assessments and the necessary underlying data. Simply put, no risk assessment can be conducted without PFAS-specific toxicity studies and the toxicity values that are extracted from those studies. Those studies do not yet exist, and there is no concrete plan for their completion.⁸⁵

Moreover, given the similarity of the molecular structure of the Table 3+ PFAS to PFOA (also known as C8) and GenX, Chemours should also be required to comply with the requirement in Section 8(e) of TSCA to immediately notify the EPA when substances or mixtures present a substantial risk of injury to health or the environment. TSCA Section 8(e) states, "Any person who manufactures, [imports,] processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment shall immediately inform the [EPA] Administrator of such information unless such person has actual knowledge that the Administrator has been adequately informed of such information." 15 U.S.C. § 2607(e). EPA's guidance states that such "Substantial Risk Notifications" under TSCA Section 8(e) should be submitted within 30 calendar days.

Until Chemours conducts foundational toxicity tests on the 19 PFAS compounds listed in Table 3+, there is no path forward to derive toxicity values. And without this information, human health risks cannot be determined, the current calculated risks are not even a good faith guess, and

⁸⁵ Chemours and its predecessor, DuPont, are well equipped to generate such data. DuPont contends that its *Haskell Laboratory for Toxicology and Industrial Medicine* is one of the most advanced industrial toxicology testing facilities in the world. Indeed, in its very first mission statement in 1935, Haskell Laboratory stated that the purpose of its facilities was to test DuPont's chemical products *before* they were placed on the market. *Haskell Laboratory of Industrial Toxicology*, 13 Chem. Eng. News 3, 44-46 (1935) (The purpose of Haskell Labs is "*to test thoroughly from a health standpoint all products produced by the company before they are placed on the market.*") (emphasis added).

to ensure they are adequately protected from the future health risks of these toxic chemicals, DRCs are entitled to the installation of RO systems and bottled water.

2. The Carcinogenetic Impacts of GenX and PFAS Must Be Addressed

In addition to noncancer systemic toxicity, however, the HH-SLEA does not address or discuss whether GenX or any of the other 19 PFAS listed in Table 3+ are, like their closely related predecessor C8, likely carcinogens. Indeed, the EPA's draft toxicity assessment of GenX (which Chemours relies on) states:

[T]here is Suggestive Evidence of Carcinogenic Potential of oral exposure to GenX chemicals in humans, based on the female hepatocellular adenomas and hepatocellular carcinomas and male combined pancreatic acinar adenomas and carcinomas.⁸⁶

Moreover, because the cancer studies the EPA refers to were rat studies, even the EPA's

assessment may underestimate GenX's cancer risk, as it is well-known that mice are more sensitive

to the effects of GenX than rats. *GenX's potential carcinogenicity was not even mentioned in the*

HH-SLEA.

A review of DuPont's and Chemours' knowledge of the carcinogenicity of GenX is

instructive.

a. Pursuant to the 2009 consent decree between DuPont and the EPA, DuPont was required to conduct a series of tests on GenX. The tests demonstrated significant health and environmental dangers associated with GenX, and yet DuPont concealed, misrepresented, and downplayed these dangers, all while continuing to discharge toxic chemicals into the Cape Fear River.

⁸⁶ Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3) Also Known as 'GenX Chemicals', EPA-823-P-18-001 Public Comment Draft at 47, U.S. EPA (Nov. 2018), https://www.epa.gov/sites/production/files/2018-11/documents/genx_public_comment_draft_toxicity_assessment_nov2018-508.pdf.

b. On July 15, 2010, pursuant to the consent decree, DuPont submitted a letter report to the EPA summarizing the results of studies of the impacts of GenX on both fetal and adult laboratory rats. The study found a direct correlation between the dosage of GenX and early deliveries, fetal weight, and skeletal deformations:

There was a dose-related increase in the number of dams [female mice] found with **early deliveries** on GD 21.

In addition, mean fetal weight was **8 and 28% lower** (statistically significant) than controls at 100 and 1000 mg/kg/day, respectively.

A higher mean litter proportion of 14th rudimentary ribs was observed in the 1000 mg/kg/day group, resulting in a higher mean litter proportion of **total skeletal variations and total developmental variations**....

c. As for the maternal laboratory rats, the study found that:

Focal necrosis [small areas of dead tissue such as cysts] of the liver was noted in some females in the 100 and 1000 mg/kg/day groups in a dose-related manner.

d. On July 20, 2010, pursuant to the consent decree, DuPont submitted a report to the

EPA on a further rodent study which found numerous instances of cellular deformation indicative of liver disease and early-stage cancer. Pathological findings included focal necrosis, which are small areas of dead liver cells undergoing disintegration, and an increase of peroxisome proliferators which have been shown to cause liver disease and induce tumors in livers.

e. To address these adverse findings, DuPont performed a follow-up study which it reported to the EPA on January 28, 2011. The results differed little from the July 20, 2010 letter report and portended the results of a far more detailed analysis in 2014:

Hepatocellular hypertrophy was characterized by cytoplasmic eosinophilic stippling that is consistent with **peroxisome proliferation**. In the 5 mg/kg/day F0 males and females, other **liver lesions** included increases in single cell **necrosis**, **mitotic figures**, lipofuscin pigment, and **focal necrosis** (females only). Increases in mitotic figures indicate that a cell population is proliferating and is used as an index of tumor aggression.

f. On January 8, 2013, DuPont completed another study. The results further confirmed the dangerous health effects of exposure to GenX:

Under the conditions of this study, the no-observed-adverse-effect level (NOAEL) was considered to be 1 mg/kg/day in male and female rats. Test substance-related **neoplastic changes** were observed at the high dose (500 mg/kg/day in females; 50 mg/kg/day in males) and included **hepatocellular tumors** in females and, in males, equivocal **increases in pancreatic acinar cell tumors and testicular interstitial cell tumors**.

But DuPont dismissed the results as not being relevant to human health:

Based on the high dose threshold for these tumor responses in this study, the lack of genotoxicity of the test material across a battery of *in vitro* and *in vivo* tests, and the known responses of the rat versus other species, including humans, to these PPAR(a) associated tumor responses, these tumor findings are not considered relevant for human risk assessment.

g. The January 2013 study also found uterine polyps, which is a potential indicator of uterine cancer, but dismissed the results on statistical grounds. DuPont did not, however, provide a basis for selecting the statistical tests or any evidence that it had run the tests, or the results of the tests. DuPont swept its own dire findings under the rug, while citing no authority and conducting no tests supporting these broad dismissals. Moreover, DuPont failed to acknowledge the large body of science that is contrary to DuPont's purported conclusion that its rodent studies are irrelevant to human health.

h. In 2014, DuPont scientists conducted yet another evaluation of the toxic effects of GenX, "Evaluation of chronic toxicity and carcinogenicity of ammonium 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)-propanoate in Sprague–Dawley rats" ("GenX Report"). This study was

51

designed to be far more detailed than the last half-dozen studies, and was presumably designed to put to bed any lingering doubts about the carcinogenicity of GenX. But the opposite occurred. The GenX Report found "[i]ncreases in enzymes indicative of **liver injury.** . ." It also found a gradual deterioration of specific tissues, cells, and organs with a corresponding impairment of function, and small areas of dead liver tissue. Blood sampling analysis and results also found that the rats were in a diseased state.

i. Tumors were also discovered in the rats:

At the interim necropsy, **non-neoplastic** test substance-associated effects were present in the liver of males at 50 mg/kg and in the liver and kidneys of females at 500 mg/kg.

j. "Non-neoplastic" refers to new growth in tissue that does not serve a useful purpose

- *i.e.*, tumors. Neoplasms may be malignant or benign; some benign tumors may progress to malignancy. The report later indicated that these tumors were indeed carcinogenic. DuPont also found the livers to be enlarged, lesions and dying cells—all indicators of liver disease.

k. DuPont also found cells in the early stages of kidney cancer:

Kidney changes in females at 500 mg/kg included tubular dilation, edema of the renal papilla, **transitional cell hyperplasia in the renal pelvis**, tubular mineralization, **renal papillary necrosis** and CPN. Tubular dilation frequently occurred in an ascending pattern extending from the papilla to the outer cortex, while at other times it was present only in the papilla. **Edema of the papilla** was characterized by increased rarefaction or myxomatous change in the papillary interstitium, sometimes with polypoid protrusions from the lateral surface of the papilla. The **edema** and tubular dilation were often associated with hyperplasia of the transitional cell epithelium lining the papilla and pelvis. Small foci of tubular mineralization were often present and, in some animals, necrosis of the tip of the papilla was present.

Transitional cell hyperplasia in the kidney is often an initial stage in the development of cancer.

1. The report also found that, in addition to tumors in the liver, tumors were also found

in the kidney, stomach, and tongues of females:

In addition, in female rats given 500 mg/kg, statistically significant increases in **hyperplasia** of squamous epithelium were observed in the nonglandular stomach (limiting ridge only) and tongue (in association with subacute/chronic inflammation in the tongue).

Hyperplasia is the enlargement of an organ or tissue caused by an increase in the reproduction rate

of its cells, often as an initial stage in the development of cancer.

m. DuPont ultimately concluded that the lesions in the liver were carcinomas—that

GenX caused liver disease and cancer in the livers of females and males:

Compound-related neoplastic changes occurred in the livers of females administered 500 mg/kg and included **increased incidences of hepatocellular adenoma and carcinoma.** These tumors occurred in association with the degenerative and necrotic liver lesions observed at this dose as described above. Hepatocellular tumors and test substance-associated degenerative and necrotic lesions were not observed in females at lower doses and **the incidences of hepatocellular tumors were similar in all male groups.**

n. The report also found that in males, GenX causes pancreatic cancer, but then

attempted to minimize the impact of its findings:

In males administered 50 mg/kg, **a statistically significant increase in the combined incidence of pancreatic acinar cell adenomas and carcinomas was seen**, but neither the incidence of adenoma or carcinoma alone was statistically increased, although the incidence of carcinomas (2.9%) was slightly outside the historical range of 0-1.7%.

o. DuPont's study also found evidence of testicular cancer, but again tried to minimize

its significance:

The incidence of **Leydig cell adenomas** (11.4%) was increased above historical control ranges for this tumor (0-8.3%) in males administered 50 mg/kg, although this increase was not statistically significant compared to controls. In addition, a Leydig cell adenoma

was present in 1 male at the interim necropsy in the 50 mg/kg group. The incidence of Leydig cell hyperplasia was also increased above historical control range in this group at terminal sacrifice (also 0-8.3%, although again, this incidence was not statistically significant versus controls. However, comparison to within-study controls was complicated by the fact that controls had a relatively high incidence of Leydig cell hyperplasia (10%). Based on the above considerations and the known activity of PPAR α agonists to produce Leydig cell hyperplasia and adenomas in rats, the relationship to the test compound for **these lesions was considered equivocal in this study**.

Leydig cell tumors are usually benign, but approximately 10% are malignant. As with germ cell tumors, they spread throughout the lymphatic system. However, unlike germ cell tumors, Leydig cell tumors show relative lack of sensitivity to radiotherapy and chemotherapy agents.

p. DuPont likewise tried to minimize its finding on pancreatic cancer and Leydig cell tumors by claiming that "less robust" evidence "suggests" that the results were "likely" not relevant to humans:

While there is less definitive mechanistic data on the role PPAR α plays in the induction of pancreatic acinar cell tumors in rats, the available data involving altered bile flow and increased cholecystokinin *suggests* that this mode of action is also *likely* to be non-relevant for humans. While *less robust*, research considering comparative biology and mechanism of action of Leydig cell tumor induction in rodents by a wide variety of chemical classes *suggests* these tumors most *likely* have low relevance to humans.

q. DuPont's GenX report ultimately concluded: "The test chemical belongs to a class of compounds known as peroxisome proliferators (PPAR α agonists) which are known to produce liver, pancreatic, and testicular tumors in rats and liver tumors in mice." However, faced with its findings that GenX is carcinogenic, DuPont concluded, without any epidemiological study on rodents impregnated with human proteins, that "these compounds have not been shown to be carcinogenic in other species including humans. Based on the extensive research into the comparative biology of peroxisome proliferator-induced hepatic carcinogenesis, the induction of

liver tumors in rodents by non-genotoxic peroxisome proliferators (this compound was shown to be inactive in a battery of genotoxicity assays) is not considered relevant to humans."

r. DuPont never tested for the synergetic impact of GenX and other PFASs.

DuPont wrongly dismissed all these results as not being relevant to human health. s. DuPont claimed that the observed increase in cancer in rodents exposed to GenX was irrelevant based on the single argument that the PPAR mode of action in rodents is irrelevant to human cancers. But DuPont ignored the fact that the PPAR mode of action only applies to liver cancer and not to pancreatic and testicular cancer. Moreover, it was DuPont who selected the rodents for the cancer study, and DuPont ignored the fact that there are rodents with modified signaling that are more conducive to determining the test's applicability to humans. Scientific studies by independent researchers have found carcinogenic impacts from PFOA exposure to these modified rodents. DuPont also concluded that the high doses used in the rodent studies were not representative of human exposures. This argument is not only scientifically untrue but defies common sense for several reasons. First, all two-year cancer rodent studies follow the protocol developed by the U.S. National Toxicology Program, which requires dosing rodents at elevated dose. This requirement is necessary to increase the probability of detecting cancers in humans. Further, humans in many instances are even more susceptible to cancer and other pathologies than laboratory rodents. Moreover, it has been well-established that when exposures to carcinogens occur during the early-life stage, critical exposure carries a much greater risk of developing cancer. The EPA requires a factor of 10 to be applied to calculating risk for these early life exposures. Finally, DuPont's claim that rodent cancers only occur at high doses and are therefore irrelevant to human exposures is absurd from a common-sense standpoint-the EPA required DuPont to conduct the studies on rodents because they were relevant to determining health impacts of GenX exposure to humans. Nevertheless, DuPont dismissed its toxicology results as not being relevant to human health, and DuPont neglected to notify area residents, drinking water providers, or state and local officials of the significant dangers posed by the polluted water supply.

t. In 2012, a series of studies further demonstrated the negative health impacts of exposure to PFOA and perfluorooctanesulfonic acid (PFOS). Tests showed immunotoxic effects in a variety of species and models. Additionally, the C8 Health Project, which was created as part of the settlement of another lawsuit against DuPont, found a significant positive exposure-response relationship between PFOA and kidney cancer.

u. A 2013 population-based case-control analysis supported the association between PFOA exposure and both kidney and testicular cancer and suggested an association with prostate and ovarian cancer and non-Hodgkin lymphoma.

v. Despite all of this scientific evidence that DuPont's secret dumping of GenX into the Cape Fear River posed serious health consequences for the hundreds of thousands of people who depended on the river for their water supply, DuPont continued to conceal its actions and failed to warn regulators or the public.

w. As noted above, DuPont developed GenX primarily because it was thought to be more biodegradable than PFOAs, which had spawned extensive litigation. DuPont's logic was that GenX would pass through the body more quickly, and thus cause less damage than PFOAs.

x. According to DuPont's own March 15, 2010 report, however, written pursuant to its consent decree with the EPA, GenX is not inherently biodegradable. The purpose of this test was to evaluate the inherent biodegradability of the test substance via a 28-day test. The test was designed to meet the requirements of SEPA HJIT 153-2004, "the guidelines for the testing of chemicals," OECD Procedure 302C, "Inherent Biodegradability: Modified MITI Test (II),"

56

adopted May 1981. The report concluded that: ". . . Based on the residue analysis, **the biodegradation of the test substance was 0%** and there was hardly any change for the test substance in the 'abiotic' vessel during the testing period. The BOD results showed that **biodegradation of the test substance was both** <**1% after 14 and 28 days**. The test was valid because the level of biodegradation of the reference substance aniline exceeded 40% after 7 days, and 65% after 14 days. Therefore, the test substance was not inherently biodegradable under this test condition." In other words, DuPont's own test found that GenX was not biodegradable, that is, it was not capable of being broken down (decomposed) rapidly by the action of microorganisms. The implications for North Carolina residents—who depend on the Cape Fear River for their water supply—was that their exposures would be long-lasting.

y. DuPont's results were consistent with those of other researchers, which have found that GenX is not only not biodegradable, but that it bonds with protein in the cells of living organisms and adheres to sediment, scale and pipes, and then reenters the water supply. These living cells include biofilms that cling to pipes and water heaters. Moreover, there is no method that is known with any degree of certainty that will remove the biofilms from the water heaters and plumbing in homes.

Furthermore, even though the EPA has determined that GenX causes liver and pancreatic cancers in animals, no carcinogenic toxicity value (*i.e.*, a cancer slope factor) has yet been developed for GenX, let alone for the other 19 PFAS listed in Table 3+. Thus, most efforts to derive toxicity values for the purpose of establishing safe exposure levels for soils, surface and groundwater, tap water, air, and biota are limited to noncancer health effects, which by itself is a source of great scientific uncertainty. The HH-SLEA should identify this data or information gap and should explain why the issue of GenX-induced cancer was not included in its comments.

The issue of GenX-induced cancer will be of critical importance in the future because epidemiology studies must focus on both noncancer systemic toxicity (*i.e.*, organ damage) and cancer when such studies are finally undertaken.

3. GenX Is Likely More Toxic Than PFOS and PFOA

Recent studies—which Chemours entirely omitted from the HH-SLEA and CAP—indicate the toxicity of GenX has already been underestimated. For example, Gomis et al.⁸⁷ compared the toxic potency of long-chain perfluoroalkyl acids to the shorter-chain, second-generation fluorinated compounds in 2018; based on the severity of liver pathology, she concluded that GenX was even more toxic than the first-generation PFAS compounds it was designed to replace because it was "thought" to be less toxic. She stated:

Dose-response curves of liver enlargement from sub-chronic oral toxicity studies in male rats were converted to internal dose in serum and in liver to examine the toxicity ranking of [PFAS] and fluorinated alternatives. Converting administered doses into equivalent serum and liver concentrations reduced the variability in the dose-response curves for PFBA, PFHxA, PFOA and GenX. The toxicity ranking using modeled serum (GenX > PFOA > PFHxA > PFBA) and liver (GenX > PFOA ≈ PFHxA ≈ PFBA) concentrations indicated that some fluorinated alternatives have *similar or higher toxic potency than their predecessors* when correcting for differences in toxicokinetics.⁸⁸

The researchers concluded that "some fluorinated alternatives have similar or higher toxic

potency than their predecessors when correcting for differences in toxicokinetics." Neither the

HH-SLEA nor the CAP, however, discuss toxicokinetic differences for any of the 20 PFAS compounds, and Gomis' analysis showing GenX is <u>more</u> toxic than PFOA means that the current 140 ppt Health Advisory Level for GenX is far too high. The GenX level should be set

 ⁸⁷ M. I. Gomis, R. Vestergren, D. Borg, and I. T. Cousins, *Comparing the Toxic Potency in Vivo of Long-Chain Perfluoroalkyl Acids and Fluorinated Alternatives*, 113 Environ. Int. 1-9 (Jan. 2018).
 ⁸⁸ Id.

even lower than the current safe drinking water levels that some states have developed for PFOA of 10 to 15 ppt.

Because Chemours did not conduct a detailed toxicokinetic study, it inferred that elimination of GenX from the body is rapid. However, Chemours' analysis does not and cannot be interpreted to mean that the recent blood sampling tests performed by the N.C. State University in which GenX was not detected in participants' *blood*—should be interpreted to mean that the exposed population does not have elevated levels of GenX in their *bodies*. It has now been well established that PFAS compounds *bioaccumulate* in different organs and tissues in the body and that this bioaccumulation essentially prevents PFAS from circulating in the blood. It is thus possible to have an undetectable level of GenX based on blood tests *because* GenX has bioaccumulated in different *organs*. Thus, while the blood levels for GenX may be low or undetectable, there may be noncirculating GenX stored in organs bound to tissue.

Examples of this toxicokinetic phenomenon abound. A similar phenomenon was described by Perez et al.,⁸⁹ and their following illustration shows that different organs bioaccumulate different types of PFAS; although blood levels for PFAS compounds like GenX may be low or non-detectable, that in itself does not mean that the body burden is insignificant.

⁸⁹ F. Pérez, et al., *Accumulation of Perfluoroalkyl Substances in Human Tissues*, 59 Environ. Int. 354-62 (2013).



Source: F. Pérez, et al., *Accumulation of Perfluoroalkyl Substances in Human Tissues*, 59 Environ. Int. 354-62 (2013).

Finally, it is a fundamental toxicological principle that the absence of a detectable amount of a toxin in blood samples does not mean there is no ongoing risk of health hazards. This is because toxic compounds may simply trigger toxic effects: that is, once a toxin triggers disease, continuous exposures to that toxin are not necessary for illness and disease to manifest. For example, a heavy smoker may smoke for decades and quit; even though the person no longer smokes (exposure stops), he or she can develop lung cancer. That is, lung cancer is triggered by cigarette smoke, but cancer may develop even in the absence of ongoing exposure, as the latency period between smoking and the onset of lung cancer is about 45 years. Thus, setting aside the toxicokinetic principles discussed above, and even if blood levels of GenX do not show *current* exposure, the toxicological damage may have already been triggered. Illness and disease can progress even when exposure to GenX stops. It should also be emphasized that the HH-SLEA and CAP do not calculate *any* risks associated with past historical exposures—which were very high for GenX and related PFAS compounds—because Chemours uses *current PFAS levels to predict the future risks*. This vastly understates the risk associated with past exposures to PFAS, which occurred at a time when Chemours was dumping directly into the Cape Fear River, emitting to the air and spilling to ground.

4. Chemours Failed to Follow EPA Standards for Deriving Toxicity Values for GenX and Other PFAS.

Chemours did not follow, let alone cite or reference, EPA or North Carolina guidance that toxicity values must be derived from primary peer-reviewed toxicity studies. For GenX, Chemours made no attempt to verify toxicity values and instead used *draft* preliminary noncancer RfD levels developed by the EPA and DHHS, which were based on DuPont's 2009-2010 toxicity study submissions provided to the EPA pursuant to the TSCA premanufacture notice procedure, often referred to as "8(e)." *Despite the fact that many toxicology studies have been published in the past decade, Chemours did not conduct a literature review to identify any of these subsequent studies*; these studies might have resulted in a different RfD for one or more of the 20 PFAS compounds identified by Chemours in its Appendix F, Table 1 shown below.

| Chemical Abbreviation | Chemical Name | Chemical Formula | Consent Order Constituent | Table 3+ Constituent | |
|---|--|------------------|------------------------------|-------------------------|--|
| HFPO-DA | Hexafluoropropylene oxide dimer acid | C6HF11O3 | Х | Х | |
| PEPA | Perfluoroethoxypropyl carboxylic acid | C5HF9O3 | Х | Х | |
| PFECA-G | Perfluoro-4-isopropoxybutanoic acid | C12H9F9O3S | Х | Х | |
| PFMOAA | Perfluoro-2-methoxyaceticacid | C3HF5O3 | Х | Х | |
| PFO2HxA | Perfluoro(3,5-dioxahexanoic) acid | C4HF7O4 | Х | Х | |
| PFO3OA | Perfluoro(3,5,7-trioxaoctanoic) acid | C5HF9O5 | Х | Х | |
| PFO4DA | Perfluoro(3,5,7,9-tetraoxadecanoic) acid | C6HF11O6 | Х | Х | |
| PMPA | Perfluoromethoxypropyl carboxylic acid | C4HF7O3 | Х | Х | |
| Hydro-EVE Acid | Perfluoroethoxsypropanoic acid | C8H2F14O4 | | Х | |
| EVE Acid | Perflouroethoxypropionic acid | C8HF13O4 | | Х | |
| PFECA B | Perfluoro-3,6-dioxaheptanoic acid | C5HF9O4 | | Х | |
| R-EVE | R-EVE | C8H2F12O5 | | Х | |
| PFO5DA | Perfluoro-3,5,7,9,11-pentaoxadodecanoic acid | C7HF13O7 | Х | Х | |
| Byproduct 4 | Byproduct 4 | C7H2F12O6S | | Х | |
| Byproduct 6 | Byproduct 6 | C6H2F12O4S | | Х | |
| Byproduct 5 | Byproduct 5 | C7H3F11O7S | | Х | |
| NVHOS | Perflouroethoxysulfonic acid | C4H2F8O4S | | Х | |
| PES | Perfluoroethoxyethanesulfonic acid | C4HF9O4S | | Х | |
| PFESA-BP1 | Byproduct 1 | C7HF13O5S | Х | Х | |
| PFESA-BP2 | Byproduct 2 | C7H2F14O5S | Х | Х | |
| Notes: CASN - Chemical Abst SLEA - Screening Leve | ract Service Number I Exposure Assessment | | | | |

Source: CAP, App. F, Table 1. Note that there is an error in this table: PFHpA is listed in the CO but was omitted from the table.

Further, by not conducting toxicological analyses of the 19 PFAS compounds listed in Table 3+, Chemours is simply assuming—with no basis for doing so, and contrary to the body of available evidence—they are not toxic *at any concentration*. The sole purpose of a quantitative risk assessment is to establish the concentration or dose at which contaminants are toxic and then determine whether the site-related dose exceeds the toxicity value.

Chemours' false and completely baseless assumption—that the 19 PFAS compounds listed in Table 3+ do not produce toxic effects at any dose—is deeply troubling from a practical standpoint. Although many risk assessments are conducted based on a *hypothetical* assumption that people *could be* exposed to contaminants, it is a fact that thousands of North Carolina residents and DRCs *have been exposed* to the 19 PFAS compounds listed in Table 3+, likely for many years and at doses much higher than the present levels that Chemours relies on for its exposure assessment. The associated health risks from this known exposure are therefore *not hypothetical* but rather are an established fact.

Chemours' failure to follow basic EPA or North Carolina risk assessment guidance is both inexplicable and indicative of the HH-SLEA's inadequacy. In both the HH-SLEA and the CAP, Chemours makes no mention of EPA or North Carolina risk assessment guidance that must be followed to properly derive toxicity values for the 19 PFAS compounds listed in Table 3+. This omission is untenable. The HH-SLEA and CAP do not cite this EPA directive, nor does Chemours cite the detailed and more recent guidance developed by the EPA to guide the derivation of a toxicity value.⁹⁰

Chemours also fails to discuss, follow, or cite the more than 10 EPA guidance documents that have since been issued which present in great detail how primary peer-reviewed studies should be evaluated, together with the protocols and methods for extrapolating toxicity data from animal studies to characterize human toxicity. Toxicity values form the basis of not only human health risk assessments but drinking water standards and advisories. Without toxicity values, neither assessment can be completed.

The HH-SLEA and CAP do not discuss why Chemours deviated from the hierarchal procedures required by the EPA in the *OSWER Directive 9285.7-Memorandum* to derive toxicity values. Chemours does not explain why it did not contact the Office of Research and Development/National Center for Environmental Assessment/Superfund Health Risk Technical Support Center (STSC)—which is responsible for assisting the scientific community in developing

⁹⁰ *Tier 3 Toxicity Value White Paper*, Regional Tier 3 Toxicity Value Workgroup, OSWER Human Health Regional Risk Assessors Forum, U.S. EPA (May 16, 2013), https://semspub.epa.gov/work/HQ/163525.pdf.

provisional toxicity values—to request technical assistance in developing provisional toxicity values. Nor did Chemours discuss whether it conducted a review of other available Tier 3 sources of toxicity information as required by EPA guidance. Rather, *Chemours is waiting for others in the scientific community to develop toxicity values for chemical products that Chemours itself produced and profited from*. Chemours' "wait and see" approach is a prime example of its corporate irresponsibility and, more importantly, has resulted in an incomplete risk assessment.

5. The HH-SLEA Underestimates the Toxicity and Human Health Risks for GenX.

Despite Chemours' failure to conduct toxicity testing for the 19 PFAS compounds, it did conduct toxicity studies on GenX. But these toxicity and human health risk assessments are flawed and starkly underestimate the actual risk to human health of exposure to GenX.

As shown below in CAP Appendix F, Table 4 (highlighted cells), the values were based on three sources of GenX animal toxicity data compiled by the EPA,⁹¹ DEQ/DHHS,⁹² and Thompson et al.⁹³ These include the following RfDs: EPA draft RfDo = 0.00008 mg/kg-day; DHHS RfDo = 0.00010 mg/kg-day; and Thompson, et al. RfDo = 0.01000 mg/kg-day:

⁹¹ Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3) Also Known as 'GenX Chemicals', EPA-823-P-18-001 Public Comment Draft at 47, U.S. EPA (Nov. 2018), https://www.epa.gov/sites/production/files/2018-11/documents/genx_public_comment_draft_toxicity_assessment_nov2018-508.pdf.

⁹² Secretaries' Science Advisory Board Review of the North Carolina Drinking Water Provisional Health Goal for GenX, N.C. DEQ/DHHS (2018).

⁹³ C. M. Thompson, S. E. Fitch, C. Ring, W. Rish, J. M. Cullen, and L. C. Haws, *Development of an Oral Reference Dose for the Perfluorinated Compound GenX*, 39 J. Appl. Toxicol. 9, at 1267-82 (2019).

| | | HFPO-DA Intake (mg/kg-day) [2] | | HFPO-DA Hazard | | | | | | |
|---------------------|-----------------------|--------------------------------|--------------------------|-------------------------|--------------------------------------|-------------------------------|---------------------------------------|------------------------------|-------------------------------|------------------------------|
| Exposure | EUDescription | Receptor [1] | Untreated Well | Current | Untreated Well Water (RME EPC) [3,5] | | | Current Conditions (10 ng/L) | | |
| Unit (EU) | | | Water Co (RME EPC) (1 | Conditions (10 ng/L) | USEPA Draft RfDo = 8.00E-05 | NC DHHS RfDo = 1.00E-04 | Thompson et al. RfDo = 1.00E-02 | USEPA Draft RfDo = | NC DHHS RfDo = 1.00E-04 | Thompson et al. RfDo = |
| EU1 | 2.5 km, Northeast | | 8.00E-05 | 7.00E-06 | 1 | 0.8 | 0.008 | 0.08 | 0.07 | 0.0007 |
| EU2 | 2.5 km, Southeast | | 6.00E-05 | 5.00E-07 | 0.7 | 0.6 | 0.006 | 0.006 | 0.005 | 0.00005 |
| EU3 | 2.5 km, Southwest | | 3.00E-05 | 1.00E-06 | 0.4 | 0.3 | 0.003 | 0.02 | 0.01 | 0.0001 |
| EU4 | 2.5 km, Northwest | | 1.00E-05 | 5.00E-07 | 0.1 | 0.1 | 0.001 | 0.006 | 0.005 | 0.00005 |
| EU5 | 5 km, Northeast | | 2.00E-05 | 5.00E-07 | 0.3 | 0.2 | 0.002 | 0.006 | 0.005 | 0.00005 |
| EU6 | 5 km, Southeast | Offsite Child | 9.00E-06 | 5.00E-07 | 0.1 | 0.09 | 0.0009 | 0.006 | 0.005 | 0.00005 |
| EU7 | 5 km, Southwest | Gardener (Age 0-6) | 2.00E-05 | 5.00E-07 | 0.2 | 0.2 | 0.002 | 0.006 | 0.005 | 0.00005 |
| EU8 | 5 km, Northwest | | 5.00E-06 | 5.00E-07 | 0.06 | 0.05 | 0.0005 | 0.006 | 0.005 | 0.00005 |
| EU9 | 10 km, Northeast | | 5.00E-06 | 5.00E-07 | 0.07 | 0.05 | 0.0005 | 0.006 | 0.005 | 0.00005 |
| EU10 | 10 km, Southeast | | 1.00E-06 | 5.00E-07 | 0.01 | 0.01 | 0.0001 | 0.006 | 0.005 | 0.00005 |
| EU11 | 10 km, Southwest | | 2.00E-06 | 5.00E-07 | 0.03 | 0.02 | 0.0002 | 0.006 | 0.005 | 0.00005 |
| EU12 | 10 km, Northwest | | 1.00E-06 | 5.00E-07 | 0.01 | 0.01 | 0.0001 | 0.006 | 0.005 | 0.00005 |
| EU13 | CFR, 10 mi. Upstream | | 5.00E-09 | n/a | 0.00006 | 0.00005 | 0.0000005 | n/a | n/a | n/a |
| EU14 | CFR, Site-Adjacent | | 3.00E-08 | n/a | 0.0004 | 0.0003 | 0.000003 | n/a | n/a | n/a |
| EU15 | CFR, 4 mi. Downstream | Offsite Child | ND | n/a | ND | ND | ND | n/a | n/a | n/a |
| EU16 | CFR, Bladen Bluffs | Recreationalist | 1.00E-05 | n/a | 0.2 | 0.1 | 0.001 | n/a | n/a | n/a |
| EU17 | CFR, Kings Bluffs | (Age 0-6) | 2.00E-08 | n/a | 0.0002 | 0.0002 | 0.000002 | n/a | n/a | n/a |
| EU18 | Onsite Pond 1 | | 8.00E-07 | n/a | 0.01 | 0.008 | 0.00008 | n/a | n/a | n/a |
| EU19 | Offsite Pond B | | 3.00E-07 | n/a | 0.004 | 0.003 | 0.00003 | n/a | n/a | n/a |
| EU16 (Intake Point) | CFR, Bladen Bluffs | Offsite Child | 2.00E-05 | n/a | 0.2 | 0.2 | 0.002 | n/a | n/a | n/a |
| EU17 (Intake Point) | CFR, Kings Bluffs | Resident (Age 0-6) | 9.00E-07 | n/a | 0.01 | 0.009 | 0.00009 | n/a | n/a | n/a |

Source: CAP at App. F, Table 4.

The toxicity values derived by the EPA and DEQ/DHHS are largely based on the toxicity

data presented in the documents Chemours submitted in the TSCA Section 8(e) consent order.

These studies are both old and limited in scope, and the EPA concluded that:

Data from these available studies indicate that the liver is the most sensitive target of GenX chemicals toxicity. Liver effects were observed in both male and female mice and rats at varying durations of exposures and doses. *These effects occurred at the lowest doses* of exposure to GenX chemicals.⁹⁴

Likewise, DHHS also primarily relied on the decade-old Chemours studies and concluded that the

liver was the most sensitive organ and determined where liver pathology (hepatotoxicity) occurred

at the lowest dose.

⁹⁴ Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3) Also Known as 'GenX Chemicals', EPA-823-P-18-001 Public Comment Draft at 47, U.S. EPA (Nov. 2018), https://www.epa.gov/sites/production/files/2018-11/documents/genx_public_comment_draft_toxicity_assessment_nov2018-508.pdf.

Table 1. No observed adverse effects levels (NOAELs) and proposed benchmark dose lower bound (BMDL) for selected endpoints from seven repeat oral dose studies in rodents dosed with GenX. BMDS Wizard output reports, which include BMD, BMD-to-BMDL ratio, and model visualizations for each modeled endpoint, can be found in Appendix B.

| | 28-day Mice 0, 0.1, 3, 30 | | 28-day Rats M - 0, 0.3, 3, 30 F - 0, 3, 30, 300 | | 90-day Mice 0, 0.1, 0.5, 5 | | 90-day Rats | | |
|---------------------------------------|------------------------------|---------------------------------|---|---------------------------------|-------------------------------|---------------------------------|---|---------------------------------|--|
| Study dose groups (mg/kg/day) | | | | | | | M - 0, 0.1, 10, 100 F - 0, 10, 100, 1000 | | |
| | NOAEL (mg/kg/day) | Proposed BMDL (mg/kg/day) | NOAEL (mg/kg/day) | Proposed BMDL (mg/kg/day) | NOAEL (mg/kg/day) | Proposed BMDL (mg/kg/day) | NOAEL (mg/kg/day) | Proposed BMDL (mg/kg/day) | |
| Hematology | | | | | - | | | | |
| Hemoglobin | 0.1 (M) | 0.0492* | 0.3 (M) | 1.19 | NS | | 0.1 (M) | 2.12 | |
| Hematocrit | 0.1 (M) | 0.00589* | 0.3 (M) | 0.357 | NS | | 0.1 (M) | | |
| Hepatotoxicity | | | | | | | | | |
| Albumin/Globulin ratio | 0.1 (M&F) | 0.464 (M); 0.595 (F) | 0.3 (M) | 0.945 | NS | | 0.1 (M) | 0.0741* | |
| Alkaline Phosphatase | 3 (M&F) | (M); 5.55 (F) | NS | | 0.5 (M&F) | 0.480 (M); 0.859* (F) | 10 (M) | 1.43 | |
| Alanine Aminotransferase | 3 (M) | | NS | | 0.5 (M&F) | (M&F) | NS | | |
| Aspartate Aminotransferase | 3 (M) | 1.18 | NS | | 0.5 (M) | 0.169 | NS | | |
| Sorbitol dehydrogenase | 3 (M&F) | (M); 6.29 (F) | NS | | 0.5 (M&F) | (M&F) | NS | | |
| Hepatocellular necrosis (single cell) | 0.1 (M)^ | 0.299* | 3 (M)^ | 2.70* | 0.5 (M)^ | 0.126# | | | |
| Liver weight to brain weight | 0.1 (M&F) | 0.194 (M); 0.751 (F) | 0.3 (M) | 0.611 | 0.5 (M&F) | 0.300 (M); 0.929 (F) | 0.1 (M) | | |
| Developmental | | | | | - | | | | |
| Combined fetal weights | | | | | | | | | |
| Skeletal variations | | | | | | | | | |
| Offspring weight PND 21 | | | | | | | | | |
| F1 body weight PND 40 | | | | | | | | | |

Source: Secretaries' Science Advisory Board Review of the North Carolina Drinking Water Provisional Health Goal for GenX – Final, DEQ/DHHS (Oct. 30, 2018).

EPA has noted that there were significant deficiencies in the toxic endpoints that Chemours studied. Most importantly, Chemours *did not evaluate immunotoxicity*, particularly with regard to antibody response. In identifying this deficiency, the EPA noted that although gross (nonspecific) hematological damage was investigated, specific immunotoxicity involving antibody production in response to an antibody challenge was not evaluated:

[I]mmune and hematological effects were also observed at low doses; however, these endpoints are not as consistently observed compared to liver effects... *Evaluation of additional immune function assays, histopathology, and immune endpoints such as antibody levels are not available.* The combined dataset was found to be weak as it did not include sufficient measures of immunopathology, humoral immunity, cell-mediated immunity, nonspecific immunity, or host resistance. Data on the potential for these GenX chemicals to impact aspects of immune function beyond immunosuppression are lacking. *Additional studies, therefore,*

would be useful to support a more conclusive determination of immunotoxic potential. $(emphasis added)^{95}$

Rectifying this gap in knowledge is critical and underscores the need for further analysis of GenX's immunotoxicity to ensure adequate protection of human health from the effects of GenX exposure.

The absence of this information—and its failure to be incorporated into the risk assessment for GenX—follows the historical pattern that DuPont followed in the case of C8. Like GenX, early studies of C8 appeared to indicate that the most sensitive toxic effect identified in PFOA studies was liver damage. Because there were no immunotoxicity studies available for C8 to assess this important toxicological endpoint, however, the C8 toxicity values were based on limited studies in which only liver pathology was identified. Since those early incomplete studies, immunotoxicity has been shown to have a significant impact, especially in children – particularly their ability to effectively immunize against disease.

North Carolina experts like Dr. DeWitt (East Carolina University) concur that immunotoxic effects are the most sensitive toxic endpoints for deriving toxicity values for PFOA or PFOS, which suggests that the immunotoxic effects of GenX must be considered when deriving its toxicity value. Dr. DeWitt recently delivered a December 2, 2019 presentation to the North Carolina Secretaries' Science Advisory Board entitled, *Immunotoxicological Findings of PFAS: A Focus on PFOA and PFOS*. The graph below from one of her studies shows that there is a striking decrease in the circulating antibody blood levels with increasing PFOA dose, which means that there is a reduction in the ability of humans to effectively immunize against disease.

⁹⁵ Human Health Toxicity Values for Hexafluoropropylene Oxide (HFPO) Dimer Acid and Its Ammonium Salt (CASRN 13252-13-6 and CASRN 62037-80-3) Also Known as 'GenX Chemicals', EPA-823-P-18-001 Public Comment Draft at 47, U.S. EPA (Nov. 2018), https://www.epa.gov/sites/production/files/2018-11/documents/genx_public_comment_draft_toxicity_assessment_nov2018-508.pdf.



Source: J. C. DeWitt, C. B. Copeland, M. J. Strynar, and R. W. Luebke, *Perfluorooctanoic Acid-Induced Immunomodulation in Adult C57BL/6J or C57BL/6N Female Mice*, 116 Environ. Health Perspect. 5, at 644-50 (May 2008).

Just as DuPont did with C8, Chemours in its HH-SLEA relies on deficient and incomplete toxicological databases in conducting its risk assessments—a practice that is unacceptable in light of the established health risks from exposure to similar PFAS. In a recent publication, Grandjean characterizes the problems with relying on deficient and incomplete toxicological databases for risk assessments. Even though Chemours has produced a wide range of PFAS compounds for 60 years—including the 20 PFAS listed in Table 3+ at issue here—Chemours has done little to conduct the type of toxicity studies needed to assess their risks and protect human health:

Identification and characterization of environmental hazards that impact human health *must rely on the best possible science* to inform and inspire appropriate public health intervention. The perfluorinated alkylate substances (PFASs) are persistent emerging pollutants that are now being recognized as important human health hazards. Although the PFASs have been produced for over 60 years, academic research on environmental health aspects has appeared only in the most recent 10 years or so. . . .

Some early studies, *e.g.*, on population exposures and toxicity, were not released to the public until after year 2000. Still, the first PFAS risk assessments ignored these reports and relied on scant journal publications. The first guidelines and legal limits for PFAS exposure, *e.g.*, from drinking water, were proposed 10 years ago. They have decreased substantially since then, but remain higher than suggested by data on human adverse effects, especially on the immune system, that occur at background exposure levels....

By now, the best-known PFASs are being phased out, and related PFASs are being introduced *as substitutes*. *Given the substantial delays in discovery of PFAS toxicity, in dissemination of findings, and in regulatory decisions, PFAS substitutes and other persistent industrial chemicals should be subjected to prior scrutiny before widespread usage.*⁹⁶

6. The HH-SLEA's Conceptual Exposure Model Ignores Key Exposure Pathways and Manipulates Inputs to Artificially Minimize Risk.

In addition to the analytical flaws in Chemours' toxicology assessments, Chemours also underestimates exposure risks by ignoring—or purposefully omitting—key exposure pathways. Specifically, in preparing the HH-SLEA, Chemours prepared a "Conceptual Exposure Model ["CEM"] for Human Exposure to PFAS Historically Deposited Offsite."⁹⁷ The CEM purportedly identifies a complete set of exposure pathways by which human receptors could come into contact with PFAS in environmental media offsite. But the CEM limits the scope of these pathways to *only those receptors that are within 10 kilometers (6.2 miles) of Fayetteville Works (i.e.*, EU 1 to 12), or at specific locations along the Cape Fear River (EU 13 to EU 19). The CEM thus excludes DRCs that consume tap water supplied to them by local water utilities whose treated water exceeds the CO's 10/70 Action Levels. Risks to these other receptors must be considered.

In the HH-SLEA, Chemours also mathematically attenuated the actual concentration of GenX present in the drinking water from individual wells near Fayetteville Works by using aggregation. Chemours accomplishes this by first grouping individual wells into arbitrarily

⁹⁶ P. Grandjean, *Delayed Discovery, Dissemination, and Decisions on Intervention in Environmental Health: a Case Study on Immunotoxicity of Perfluorinated Alkylate Substances,* 17 Environ. Health 62 (2018) (emphasis added).

⁹⁷ CAP at App. F, Section 3 - Conceptual Exposure Model, at 10 *et seq.*, and App. F, Figure 2.

assigned EU, which ranged in size from 0.48 to 5.2 square miles.⁹⁸ Once the numerous wells were grouped into these large EUs, either the average PFAS concentration or an upper-bound estimate of the PFAS concentration was calculated. These two calculations, whose results were incorporated into subsequent risk estimates by Chemours, artificially diminished the elevated PFAS concentrations detected in many of the well samples. These aggregated and mathematically attenuated GenX results were used by Chemours to estimate non-cancer risks to older children and adults drinking this contaminated well water. For example, Chemours' EU 1, located northeast of Fayetteville Works, included 24 sample results from 22 different wells.⁹⁹ Samples of drinking water from 17 of the 22 (77 percent) different wells in EU 1 contained GenX at a concentration above the DEQ/DHHS Health Advisory Level ("HAL") of 140 ng/L. In addition, the HH-SLEA only estimates risks from exposure to GenX. In EU 1, for example, eight other PFAS listed in the CO were detected in well samples, but they are not included in Chemours' risk estimates. The highest concentration of these other Attachment C PFAS was 4,400 ppt. All of these eight residences contained other (non GenX) individual PFAS above the 10/70 Action Levels.

By not considering these other (non GenX) PFAS, the HH-SLEA underestimates the risk to those drinking contaminated water. Chemours should not group individual wells into arbitrary EUs and then aggregate the GenX concentrations before estimating risks to the residences. Rather, they should estimate risks (and the applicability of the 10/70 Action Levels in the CO) from drinking water from each individual well.

Chemours also failed to consider important exposure routes accounted for in the State's 140 ppt HAL. Specifically, the HAL incorporates a relative source contribution factor for PFAS

⁹⁸ CAP at App. F, Section 4 and Figure 3.

⁹⁹ CAP at App. F, Table F-3-2.

exposure via drinking water.¹⁰⁰ This means that 20 percent of a receptor's PFAS exposure is from drinking contaminated water and the other 80 percent is from air and food. However, Chemours did not consider these other exposure routes.¹⁰¹ Instead, Chemours assumes that the only source of PFAS is from drinking water, thus excluding the risk of exposure from air and food. By not considering the ongoing and background exposure to PFAS in air and food, Chemours substantially underestimates the risk contribution to receptors from Fayetteville Works-related PFAS.

B. Chemours Must Provide RO Systems Until an Adequate Risk Assessment Is Prepared, and Must Conduct Additional Epidemiological and Toxicity Testing.

Chemours is currently far short of fulfilling its obligations under Paragraph 14 of the CO. As a result, the population of exposed residents continues to be exposed to PFAS contamination without the underlying data necessary to ensure human health and safety. Because adequate toxicity and risk assessments are unlikely to be completed for many years, DEQ should require Chemours to install RO systems and purchase bottled water for DRCs until Chemours' Paragraph 14 obligations are satisfied.

Paragraph 14 grants DEQ "the right to seek additional toxicity studies or additional health, chemical persistence and environmental fate information beyond the scope of the initial set of studies required by this paragraph. DEQ shall consider public comments in determining what additional toxicity studies or additional health, chemical persistence and environmental fate information are needed." Chemours' own studies have found significant instances of a variety of carcinogenic and non-carcinogenic impacts from GenX, and numerous epidemiological studies

¹⁰⁰ Methodology for Deriving Ambient Water Quality Criteria for Protection of Human Health, U.S. EPA Office of Water (Oct. 2000).

¹⁰¹ There is no Relative Source Contribution factor incorporated into Chemours' equations to calculate the intake of drinking water. *See* CAP at App. F, Table F-2-1.

have been conducted in large communities where DuPont has manufactured PFAS compounds, contaminating regional air and public and private drinking water.

Paragraph 14 of the CO requires Chemours to conduct toxicology studies of the chemicals listed in Attachment B of the CO. Such studies should also be expanded to include epidemiological studies. To ensure transparency and reliability, the studies should be conducted under the auspices of a truly independent science panel of neutral experts, similar to the process used in connection with DuPont's contamination of the Ohio River Valley area surrounding its Washington Works plant with C8. In addition, Chemours should also be required to conduct toxicological and epidemiological studies on all chemicals listed in Attachment C of the CO.

C. Parallels Between the Health Studies for C8 (*i.e.*, PFOA) Conducted for DuPont's Washington Works plant in West Virginia and the Present Investigation Now Being Conducted for the Chemours Fayetteville Works plant.

There are important parallels between the early health studies for C8 conducted for DuPont's Washington Works plant in West Virginia and the present investigation now being conducted for the Chemours Fayetteville Works plant. The lessons that were learned in the Washington Works investigation should be addressed by the current HH-SLEA and the CAP or at least identified as areas of uncertainty. By ignoring those earlier extensive and robust animal and epidemiology studies, it appears that the mistakes DuPont made in the Washington Works investigations are being repeated in the current HH-SLEA. Lessons learned from the C8 investigation should be applied in assessing the risk of second generation PFAS like GenX and the Attachment C PFAS. Specifically, in the case of C8, early past studies that relied solely on animal studies significantly underestimated the risks to human health, particularly for those exposed to PFAS-contaminated drinking water. Indeed, past PFAS health studies that relied solely on animal studies were disastrous because numerous illnesses and diseases that went completely undetected
in animals produced a high incidence of disease (including cancer) in the human cohort that was actually exposed (for decades) to PFAS. Chemours in its HH-SLEA fails to learn from these past lessons, which have been heeded by most toxicologists and health professionals, and continues to rely solely on animal studies that are incomplete or nonexistent (*i.e.*, they have not yet been performed).

Like the present CAP, which presents the early screening stages of investigations into the health risks posed by PFAS based on incomplete animal toxicity studies, the early DuPont Washington Works studies (which focused on PFOA toxicity and threats to human health) made similar conclusions.

This conclusion was ultimately rejected by one of the largest epidemiological studies of all time. As noted above in Section II, the C8 Science Panel emerged as a result of DuPont's settlement with a class of plaintiffs in the *Leach* action. *Leach v. E. I. du Pont de Nemours & Co.*, No. 01-C-698 (Wood County W. Va. Cir. Ct.). The three epidemiologists appointed to the C8 Science Panel studied the toxicity of C8 to characterize human exposure risks for actual residents, and found a "probable link" between human illness and exposure to C8 (at a dose of 50 parts per trillion (ppt) or nanograms per liter (ng/L) over the course of one year) for the following diseases among exposed residents:

- High cholesterol;
- Ulcerative colitis;
- Thyroid disease;
- Testicular cancer;
- Kidney cancer; and

• Pregnancy-induced hypertension.¹⁰²

The C8 Science Panel's findings made crystal clear that the animal studies DuPont relied on in the Washington Works studies vastly underestimated the health threat to humans of exposure to even small quantities of PFAS. Indeed, many of the toxic effects, illness, and disease that were ultimately characterized in the human cohort exposed to PFOA at Washington Works were never even identified in the animal studies DuPont relied on in its earlier health assessments.

With each passing year, the field of toxicology concludes that more and more PFAS are far more toxic than previously thought. For example, the following graph shows how the "assumed" safe level of PFAS in drinking water has dropped precipitously over the past decade, which parallels the advancement of toxicological research. It shows that the "health-protective" exposure levels deemed safe by the EPA for PFOA and PFOS in drinking water in 2009 decreased from 400 and 200 ppt, respectively, to 70 ppt in 2016.



¹⁰² See C8 Probable Link Reports, C8 Science Panel, http://www.c8sciencepanel.org/prob_link.html (last visited Feb. 21, 2020).

Source: P. Grandjean and E. Budtz-Jorgensen, *Immunotoxicity of Perfluorinated Alkylates: Calculation of Benchmark Doses Based on Serum Concentration in Children*, 12 Environ. Health 35 (2013).

Moreover, even the EPA's four-year-old level of 70 ppt (for the combined levels of PFOA and PFOS) has now been shown to significantly underestimate the health threat of these PFAS. Many states are urgently taking swift action to protect their citizens by setting acceptable levels far below 70 ppt. Indeed, many are setting permissible levels of PFOA and PFOS at parts-per-trillion levels in the low teens.

Because the current state-of-the-science toxicological database for GenX is in its infancy, the toxicity values used in the HH-SLEA to calculate risks are highly uncertain and likely underprotective; most certainly, future studies will show our current knowledge is very limited. Therefore, the CAP and the HH-SLEA must include a commitment by Chemours to continue to update its risk assessment as new toxicity and epidemiological information becomes available.

It is likely that these PFAS will be shown to produce *similar toxic effects* as listed above when such studies become available. This assumption is based on the physical/chemical structural similarities of all PFAS compounds, which toxicologists rely on to determine or predict whether similar toxic effects will occur. For example, the NRDC states:

However, issues related to the entire PFAS class, which has now grown to an estimated 4,700 chemicals, have been of increasing concern for researchers and health authorities. Although there is not a robust toxicity database for the suite of PFAS, *it is generally recognized that these chemicals are structurally similar, and it is reported that the health risks associated with one PFAS are expected for other PFAS as well.*¹⁰³

¹⁰³ A. Reade, T. Quinn, and J. S. Schreiber, *PFAS in Drinking Water 2019: Scientific and Policy Assessment for Addressing Per-and Polyfluoroalkyl Substances (PFAS) in Drinking Water* at 9, Natural Resources Defense Council (Apr. 12, 2019), https://www.nrdc.org/sites/default/files/media-uploads/ nrdc_pfas_report.pdf.

VI. <u>Providing the DRCs with the Same Level of Protection Afforded to Residents</u> <u>Drinking Well Water Near Fayetteville Works Is the Only Means for</u> <u>Protecting Human Health</u>

Providing the DRCs with the same level of protection afforded to residents drinking well water near Fayetteville Works is the only means of protecting human health. As stated in the CAP, "Untreated well water was identified as the primary source of potential PFAS intake and hazard."¹⁰⁴ When the HH-SLEA accounts for the effectiveness of the Chemours-provided drinking water treatment systems that are currently in place, PFAS intake via drinking water and associated hazards are substantially reduced and may be as low as zero."¹⁰⁵ But the HH-SLEA fails to recognize the same risks posed to the DRCs. The same approach should be taken with respect to DRCs, who should also be provided with point-of-use treatment. Chemours' studies indicate "that supplying whole building filtration systems and reverse osmosis units for qualifying residents offsite reduces HFPO-DA (and Table 3+ PFAS) intake by over 92%, ensuring human receptor exposures remain below hazard limits for HFPO-DA, based on the NC DHHS draft RfDo."¹⁰⁶ There is no reason why the DRCs should not be provided with the same level of protection afforded to residents drinking well water near Fayetteville Works.

A. RO Is the Only Reliably Effective Point-of-Use PFAS Exposure Reduction Method.

A recent study¹⁰⁷ evaluated the effectiveness of point-of-use (POU) (*i.e.*, at the drinking water tap) in removing a suite of three perfluoroalkyl sulfonic acids, seven perfluoroalkyl carboxylic acids, and six per- and polyfluoroalkyl ether acids in homes in central and southeastern

¹⁰⁴ CAP at 35.

¹⁰⁵ CAP at 35.

¹⁰⁶ CAP at xv.

¹⁰⁷ Herkert, N.J., et al., Assessing the Effectiveness of Point-of-Use Residential Drinking Water Filters for Perfluoroalkyl Substances (PFASs), 2020 Environ. Sci. and Technol. Lett., https://dx.doi.org/10.1021/acs.estlett.0c00004.

North Carolina. POU filtration systems included countertop and pitcher filters, faucet-mounted filters, activated carbon block refrigerator filters, activated carbon block under-sink filters, undersink dual-stage filters, and under-sink RO filters. The study found that "PFASs are difficult to remove in full-scale water treatment systems because of their physicochemical properties." But it also found that the under-sink dual-stage and RO filters tested showed near complete removal of all PFASs evaluated. In contrast, it found that all other filters containing activated carbon exhibited variable PFAS removal. In these filters, PFAS removal efficiency was dependent on chain length, with long-chain PFASs (~60-70% removal) being more efficiently removed than short-chain PFASs (~40% removal). A few whole-house activated carbon POE systems (n = 8) were also evaluated; however, results were variable, and in some cases (four of eight systems), increased PFAS levels were observed in the filtered water.

RO is superior to these POU and POE methods, and is the only reliably effective method to protect DRCs.

CONCLUSION

For the forgoing reasons, Plaintiffs respectfully request that DEQ compel Chemours to pay for the acquisition, installation, operation and maintenance of three under-sink RO systems for each residence in the municipal water supply districts and the past and future costs of bottled water pending the installation of such systems.

Appendix A - Summary of PFAS Detected in Water Heater and Tap Water Sampling near Wilmington, NC.



Table A-1. Summary of PFAS Detected in Water Heater and Tap Water Sampling near Wilmington, NC. units = ng/L

| | | | | | | | Property: | Α | А | А | А | В | В | В |
|--------------|-------------------|---------------------------------------|--------------------------|------------------|------------|-------------------------------|---------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| | | | | | | | Smpl ID: | WAMP | WAMP | WAMP | WAMP | SAND | SAND | SAND |
| | | | | | | | Location: | Tap-F | Tap-S | Heat-B | Heat-T | Heat-B.a | Heat-B.b | Heat-T.c |
| | | | | | | | Wtr. Source: | Municipal |
| _ | | | | | | | City: | Calabash | Calabash | Calabash | Calabash | Hampstead | Hampstead | Hampstead |
| | | | | | | Chemical | County: | Bruns. | Bruns. | Bruns. | Bruns. | Pend. | Pend. | Pend. |
| Comm | ion Name | Chemio | cal Name | C | ASN | Formula | Smpl Date: | 8/29/19 | 8/29/19 | 8/29/19 | 8/29/19 | 3/13/19 | 3/13/19 | 3/13/19 |
| Detected PF/ | AS listed in Atta | chment C of 2/25/19 Con | sent Order | | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | C3HF5O3 | | 29.4 | 30.3 | 30.0 | 37.1 | 33.1 | 35.8 | 36.3 |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | C4HF7O3 | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | | | 8.15 | 8.99 | 11.4 | 13.5 | 6.71 | 6.31 | 8.82 |
| PFO2HxA | | Perfluoro(3,5-dioxahexar | noic) acid | 39492-88 | -1 | C4HF7O4 | | 22.4 | 25.4 | 21.2 | 30.4 | 4.18 | 3.75 | 4.90 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | C5HF9O3 | | | | | | | | |
| | | (pentafluoroethoxy) propanoic acid | methoxybutanoic acid | 61-2 | 89-5 | | | 9.36 | 11.9 | 12.0 | 18.1 | ND | ND | 2.13 |
| PFO3OA | | Perfluoro(3,5,7-trioxaoc | tanoic) acid | 39492-89 | -2 | C5HF9O5 | | 6.97 | 10.6 | 7.33 | 7.97 | 5.09 | 5.21 | 5.61 |
| PFO4DA | | Perfluoro(3,5,7,9-tetraox | xadecanoic) acid | 39492-90 | -5 | C6HF11O6 | | 3.47 | 2.92 | 2.77 | 2.53 | ND | ND | ND |
| PFESA-BP1/ | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 67-9 | -3; 29311- | C7HF13O5S | | ND |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 0-2 | C7H2F14O5S | | 2.11 | 2.17 | 1.53 | 1.98 | ND | ND | ND |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | 4,5,5,6,6-decafluoro-6- | 174767-1 | 0-3; | C7HF13O3 | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | anoic acid, 2,2,3,3,4,4- | 801212-5 | 9-9 | | | ND | ND | | | ND | ND | ND |
| | | hexafluoro-4-[1,2,2,2-tet | rafluoro-1- | | | | | ND | ND | ND | ND | ND | | ND |
| | | (trifluoromethyl)ethoxy]- | - | | | | | | | | | | | |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pen | tadodecanoic) acid | 39492-91 | -6 | C7HF13O7 | | 1.55 | 1.92 | 1.45 | 1.50 | ND | ND | ND |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | C7HF13O2 | | 19.4 | 27.1 | 20.9 | 24.7 | 2.22 | 2.04 | 2.46 |
| HFPO-DA / P | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | 1,2,2,3,3,3- | 13252-13 | -6 | C6HF11O3 | | 40.0 | 26.2 | 22.4 | 25.0 | E OF | F 24 | C 75 |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | | | 19.8 | 26.3 | 22.1 | 25.8 | 5.95 | 5.34 | 6.75 |
| | | | | | Attacher | ment C Total <mark>(Ex</mark> | ceeds 70 ppt) | 123 | 148 | 131 | 164 | 57 | 58 | 67 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USEPA) Method 537 and samples collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laboratories, LLC (Charleston, SC). Locations - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outside spigot/tap, Out-F = First-flush from outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water sample. Wtr. Source - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | В | С | С | С | С | С | С | D | D |
|------------------------|-------------------|---|---|----------------------|-----------------|------------------------------------|-------------------------------|-------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| | | | | | | SAND | ERGE | ERGE | ERGE | ERGE | ERGE | ERGE | НАМР | HAMP |
| | | | | | | Heat-T.d Municipal Hampstead | Heat-B Municipal Leland | Heat-T Municipal Leland | Heat-B.a Municipal Leland | Heat-B.b Municipal Leland | Heat-T.c Municipal Leland | Heat-T.d Municipal Leland | Heat-B.a Municipal Leland | Heat-B.b Municipal Leland |
| | | | | | | Pend. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. |
| Comm | on Name | Chemi | cal Name | C | ASN | 3/13/19 | 6/21/18 | 6/21/18 | 3/12/19 | 3/12/19 | 3/12/19 | 3/12/19 | 3/12/19 | 3/12/19 |
| Detected PFA | AS listed in Atta | chment C of 2/25/19 Con | sent Order | | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 39.0 | 21.5 | 13.7 | 5.87 | 5.75 | 8.22 | 5.21 | 9.52 | 9.70 |
| ΡΜΡΑ | PFMOPrA | Perfluoro-2- methoxypropanoic acid | Perfluoro-3- methoxypropanoic acid | 13140-29 9 | - 377-73-1 | 9.58 | ND | ND | 16.7 | 7.01 | 6.03 | 6.27 | 8.76 | 8.42 |
| PFO2HxA | | Perfluoro(3,5-dioxahexar | noic) acid | 39492-88 | -1 | 4.57 | 17.1 | 8.89 | 6.44 | 6.47 | 6.52 | 5.89 | 7.71 | 7.87 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- (pentafluoroethoxy) propanoic acid | Perfluoro-4- methoxybutanoic acid | 267239- 61-2 | 863090- 89-5 | 3.80 | ND | ND | ND | ND | ND | ND | 2.85 | ND |
| PFO3OA | | Perfluoro(3,5,7-trioxaoc | tanoic) acid | 39492-89 | -2 | 6.05 | 14.1 | 6.61 | 2.35 | 2.1 | 1.36 | 1.81 | 2.25 | 2.81 |
| PFO4DA | | Perfluoro(3,5,7,9-tetraox | kadecanoic) acid | 39492-90 | -5 | ND | 4.98 | 2.57 | ND | ND | ND | ND | ND | ND |
| PFESA-BP1 / | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 67-9 | -3; 29311- | ND | 202 | 3.37 | ND | ND | ND | ND | ND | ND |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 0-2 | ND | 8.40 | ND | ND | ND | ND | ND | ND | ND |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 (trifluoromethoxy)-; Buta hexafluoro-4-[1,2,2,2-tet (trifluoromethyl)ethoxy]- | 4,5,5,6,6-decafluoro-6- noic acid, 2,2,3,3,4,4- rafluoro-1- | 174767-1 801212-5 | 0-3; 9-9 | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pen | tadodecanoic) acid | 39492-91 | -6 | ND | 7.89 | ND | ND | ND | ND | ND | ND | ND |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 2.64 | 16.8 | 18.1 | 1.79 | 1.62 | 1.92 | 1.96 | 2.54 | 1.99 |
| HFPO-DA / PI "GenX" | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 heptafluoropropoxy)-pro | .,2,2,3,3,3- panoic acid | 13252-13 | -6 | 6.84 | 15.3 | 16.7 | 8.70 | 8.79 | 7.71 | 7.71 | 10.1 | 10.0 |
| | | | | | Attache | 72 | 308 | 70 | 42 | 32 | 32 | 29 | 44 | 41 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | D | D | D | D | D | D | E | E |
|--------------|-------------------|----------------------------|--------------------------|------------------|-------------|---------------------------------------|---------------------------------------|---------------------------------|---------------------------------|---------------------------------------|---------------------------------------|------------------------------|------------------------------|
| | | | | | | НАМР | НАМР | HAMP | HAMP | HAMP | НАМР | LEE | LEE |
| | | | | | | Heat-B.a/Dup_c Municipal Leland | Heat-B.b/Dup_d Municipal Leland | Heat-T.e Municipal Leland | Heat-T.f Municipal Leland | Heat-T.e/Dup_g Municipal Leland | Heat-T.f/Dup_h Municipal Leland | Tap-F Municipal Leland | Tap-S Municipal Leland |
| | | | | | | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. |
| Comm | on Name | Chemi | cal Name | C | ASN | 3/12/19 | 3/12/19 | 3/12/19 | 3/12/19 | 3/12/19 | 3/12/19 | 10/24/19 | 10/24/19 |
| Detected PFA | AS listed in Atta | chment C of 2/25/19 Con | sent Order | 1 | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 9.90 | 9.38 | 8.16 | 9.54 | 6.68 | 8.66 | 53.1 | 56.6 |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | 8.15 | 8.42 | 7.38 | 7.67 | 7.04 | 7.71 | 14.1 | 16.6 |
| PFO2HxA | | Perfluoro(3,5-dioxahexar | noic) acid | 39492-88 | 8-1 | 8.64 | 7.96 | 6.83 | 7.14 | 3.91 | 6.56 | 45.3 | 44.8 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | |
| | | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | ND | ND | ND | ND | ND | ND | | |
| | | propanoic acid | | | | | | | | | | 12.0 | 12.4 |
| PFO3OA | | Perfluoro(3,5,7-trioxaoc | tanoic) acid | 39492-89 |)-2 | 3.24 | 2.59 | 2.35 | 1.99 | 2.33 | 1.72 | 15.4 | 18.0 |
| PFO4DA | | Perfluoro(3,5,7,9-tetrao | xadecanoic) acid | 39492-90 |)-5 | ND | ND | ND | ND | ND | ND | 6.06 | 5.70 |
| PFESA-BP1/ | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 67-9 |)-3; 29311- | ND | ND | ND | ND | ND | ND | 2.41 | 2.23 |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 20-2 | ND | ND | ND | ND | ND | ND | 3.07 | 2.95 |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | 4,5,5,6,6-decafluoro-6- | 174767-1 | .0-3; | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | anoic acid, 2,2,3,3,4,4- | 801212-5 | 9-9 | | | | | | | | |
| | | hexafluoro-4-[1,2,2,2-tet | rafluoro-1- | | | ND | ND | ND | ND | ND | ND | | |
| | | (trifluoromethyl)ethoxy]- | - | | | | | | | | | ND | ND |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pen | tadodecanoic) acid | 39492-91 | -6 | ND | ND | ND | ND | ND | ND | 2.7 | 2.74 |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 1.70 | 1.63 | 1.78 | 2.25 | 1.87 | 1.52 | 30.2 | 30.1 |
| HFPO-DA / PI | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | 1,2,2,3,3,3- | 13252-13 | 8-6 | | | 0.5- | | | 0 | | |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | 9.56 | 10.1 | 8.67 | 8.90 | 8.50 | 8.25 | 47.8 | 46.4 |
| | | | | 1 | Attache | 41 | 40 | 35 | 37 | 30 | 34 | 232 | 239 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | E | E | E | F | | | | G | G | |
|-------------|------------------|----------------------------|--------------------------|----------|-----------------|-----------|--------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|--|
| | | | | | | LEE | LEE | LEE | FOUR | FOUR | FOUR | FOUR | SELL | SELL | |
| | | | | | | Heat-B | Heat-B/Dup_2 | Heat-T | Tap-F | Tap-S | Heat-B | Heat-T | Tap-F | Tap-S | |
| | | | | | | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | |
| | | | | | | Leland | Leland | Leland | Oak Is. | |
| | | | | | | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | |
| Comm | on Name | Chemi | cal Name | <u> </u> | ASN | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | |
| Detected PF | AS listed in Att | achment C of 2/25/19 Con | sent Order | | | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 36.7 | 42.9 | 53.8 | 32.3 | 43.6 | 27.8 | 9.75 | 76.5 | 73.4 | |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | 9-377-73-1 | | | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | | | | | | | | | | |
| | | | | | | 17.2 | 20.5 | 23.7 | ND | 8.31 | 11.2 | 4.11 | 18.6 | 15.0 | |
| PFO2HxA | | Perfluoro(3,5-dioxahexar | noic) acid | 39492-88 | 3-1 | 35.4 | 43.3 | 51.1 | 24.0 | 33.3 | 26.8 | 10.3 | 57.1 | 61.5 | |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | | | |
| | | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | | | | | | | | | | |
| | | propanoic acid | | | | 4.96 | 5.56 | 6.64 | 7.1 | 8.0 | ND | ND | 13.4 | 13.3 | |
| PFO3OA | | Perfluoro(3,5,7-trioxaoc | tanoic) acid | 39492-89 |)-2 | 11.3 | 12.3 | 15.1 | 9.14 | 13.8 | 9.39 | 3.85 | 21.1 | 20.6 | |
| PFO4DA | | Perfluoro(3,5,7,9-tetrao | xadecanoic) acid | 39492-90 |)-5 | 3.93 | 3.46 | 4.05 | 3.39 | 4.23 | 2.37 | 1.4 | 7.03 | 8.06 | |
| PFESA-BP1/ | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 |)-3; 29311- | | | | | | | | | | |
| | | | | 67-9 | | ND | ND | 1.40 | ND | 1.47 | ND | ND | 1.94 | 2.00 | |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 20-2 | 1.57 | 1.68 | 2.03 | 2.49 | 2.74 | ND | ND | 5.02 | 4.67 | |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | 4,5,5,6,6-decafluoro-6- | 174767-1 | LO-3; | | | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | anoic acid, 2,2,3,3,4,4- | 801212-5 | 59-9 | | | | | | | | | | |
| | | hexafluoro-4-[1,2,2,2-tet | rafluoro-1- | | | | | | | | | | | | |
| | | (trifluoromethyl)ethoxy]- | | | | ND | ND | ND | ND | ND | ND | ND | ND | ND | |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pen | tadodecanoic) acid | 39492-91 | L-6 | 1.19 | 1.54 | 2.75 | ND | 2.3 | ND | ND | 3.88 | 3.23 | |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 19.6 | 23.2 | 26.0 | 14.6 | 21.3 | 13.4 | 5.49 | 36.1 | 37.4 | |
| HFPO-DA / P | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | L,2,2,3,3,3- | 13252-13 | 3-6 | | | | | | | | | | |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | 26.5 | 30.9 | 37.1 | 21.5 | 32.2 | 18 | 7.69 | 52.4 | 54.2 | |
| | | · | | · | Atta <u>che</u> | 158 | 185 | 224 | 115 | 171 | 109 | 43 | 293 | 293 | |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | G | G | Н | н | н | Н | I | 1 | 1 | |
|---------------|------------------|----------------------------|--------------------------|----------|-------------|--------------------------------|--------------------------------|----------------------------------|----------------------------------|-----------------------------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|---|
| | | | | | | SELL | SELL | WATE | WATE | WATE | WATE | GOOS | GOOS | GOOS | |
| | | | | | | Heat-B Municipal Oak Is. | Heat-T Municipal Oak Is. | Tap-F Municipal Ocean Isle | Tap-S Municipal Ocean Isle | Heat-B Municipal Ocean Isle | Heat-T Municipal Ocean Isle | Tap-F Municipal Riegelwood | Tap-S Municipal Riegelwood | Heat-B Municipal Riegelwood | |
| Comm | on Namo | Chomi | | | ACN | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Bruns. | Colum. | Colum. | Colum. | |
| Detected PFA | S listed in Atta | chment C of 2/25/19 Con | sent Order | | ASN | 10/24/19 | 10/24/19 | 8/23/13 | 0/23/15 | 6/25/15 | 0/25/15 | 10/24/19 | 10/24/15 | 10/24/19 | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 17.6 | 14.4 | 29.7 | 29.1 | 35.2 | 30.3 | 59.3 | 63.5 | 52.4 | |
| PMPA | PFMOPrA | , Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | | | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | | | 7.00 | 6.19 | 7.56 | 6.84 | | | | |
| | | | | | | 5.01 | 5.52 | | | | | 22.7 | 19.1 | 22.6 | |
| PFO2HxA | | Perfluoro(3,5-dioxahexar | noic) acid | 39492-88 | 8-1 | 16.9 | 13.6 | 24.1 | 23.2 | 27.9 | 26.3 | 47.0 | 51.8 | 50.0 | _ |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | | | |
| | | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | | | 11.6 | 13.4 | 4.69 | 3.35 | | | | |
| | | propanoic acid | | | | ND | ND | | | | | 11.4 | 11.0 | 3.94 | |
| PFO3OA | | Perfluoro(3,5,7-trioxaoct | tanoic) acid | 39492-89 | 9-2 | 5.13 | 4.37 | 8.99 | 9.21 | 9.01 | 8.19 | 16.9 | 18.2 | 15.2 | |
| PFO4DA | | Perfluoro(3,5,7,9-tetraox | xadecanoic) acid | 39492-90 |)-5 | 2.00 | 1.28 | 4.09 | 4.07 | 4.06 | 3.01 | 5.89 | 5.89 | 5.41 | |
| PFESA-BP1 / N | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 |)-3; 29311- | | | ND | ND | ND | ND | | | | |
| | | | | 67-9 | | ND | ND | | | | | 1.82 | 1.51 | ND | |
| PFESA-BP2 / N | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 20-2 | ND | ND | 1.99 | 1.79 | 1.69 | 1.91 | 3.39 | 3.85 | 3.02 | |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | 4,5,5,6,6-decafluoro-6- | 174767-1 | .0-3; | | | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | anoic acid, 2,2,3,3,4,4- | 801212-5 | 59-9 | | | ND | ND | ND | ND | | | | |
| | | hexafluoro-4-[1,2,2,2-tet | rafluoro-1- | | | | | ND | ND | ND | | | | | |
| | | (trifluoromethyl)ethoxy]- | - | | | ND | ND | | | | | ND | ND | ND | |
| TAFN4 / PFO5 | 5DA | Perfluoro(3,5,7,9,11-pent | tadodecanoic) acid | 39492-91 | -6 | ND | ND | ND | 2.03 | 1.42 | 1.77 | 2.60 | 2.34 | 2.83 | |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 8.51 | 8.83 | 18.5 | 17.0 | 16.8 | 17.7 | 31.2 | 31.4 | 24.3 | |
| HFPO-DA / PF | PrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | L,2,2,3,3,3- | 13252-13 | 8-6 | | | 20.0 | 21.0 | 20.7 | 20.2 | | | | |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | 9.02 | 11.3 | 20.9 | 21.6 | 20.7 | 20.3 | 41.2 | 44.5 | 33.6 | |
| | | | | | Attache | 64 | 59 | 127 | 128 | 129 | 120 | 243 | 253 | 213 | |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | l l | J | J | J | J | К | | | | |
|-----------------------------------|----------------------------|-------------------------|----------|-----------------|------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|--|
| | | | | | GOOS | ODEL | ODEL | ODEL | ODEL | RIVE | RIVE | RIVE | RIVE | |
| | | | | | Heat-T | Tap-F | Tap-S | Heat-B | Heat-T | Tap-F | Tap-S | Heat-B | Heat-T | |
| | | | | | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | |
| | | | | | Riegelwood | Shalotte | |
| | | | | | Colum. | Bruns. | |
| Common Name | Chemic | cal Name | <u> </u> | ASN | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 8/29/19 | 8/29/19 | 8/29/19 | 8/29/19 | |
| Detected PFAS listed in At | achment C of 2/25/19 Cons | sent Order | | | | <u> </u> | | | | | | | | |
| PFMOAA | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 48.1 | 20.6 | 15.3 | 64.0 | 98.3 | 22.4 | 31.6 | 36.3 | 34.0 | |
| PMPA PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | | | | | | | | | | |
| | methoxypropanoic acid | methoxypropanoic acid | 9 | | | | | | | 6.06 | 7.97 | 8.17 | 8.67 | |
| | | | | | 17.9 | 2.71 | ND | 19.3 | 24.1 | | | | | |
| PFO2HxA | Perfluoro(3,5-dioxahexar | noic) acid | 39492-88 | 8-1 | 43.8 | 15.9 | 9.64 | 57.8 | 74.2 | 18.9 | 25.4 | 29.6 | 26.2 | |
| PEPA PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | | | |
| | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | | | | | | 14.3 | 8.49 | 4.55 | 5.68 | |
| | propanoic acid | | | | 5.34 | 5.38 | 2.53 | 12.5 | 17.2 | | | | | |
| PFO3OA | Perfluoro(3,5,7-trioxaoct | tanoic) acid | 39492-89 |)-2 | 13.9 | 6.97 | 4.37 | 19.5 | 26.0 | 5.35 | 10.7 | 8.89 | 9.11 | |
| PFO4DA | Perfluoro(3,5,7,9-tetrao> | xadecanoic) acid | 39492-90 |)-5 | 4.73 | 2.56 | 1.23 | 5.98 | 7.54 | 2.23 | 4.39 | 3.62 | 3.54 | |
| PFESA-BP1 / Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 |)-3; 29311- | | | | | | | | | | |
| | | | 67-9 | | 1.26 | ND | ND | 1.66 | 1.71 | ND | ND | ND | ND | |
| PFESA-BP2 / Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 20-2 | 3.55 | 1.76 | ND | 4.32 | 5.31 | 1.79 | 2.22 | 2.21 | 2.05 | |
| PFECA-G | Hexanoic acid, 2,2,3,3,4,4 | 4,5,5,6,6-decafluoro-6- | 174767-1 | .0-3; | | | | | | | | | | |
| | (trifluoromethoxy)-; Buta | noic acid, 2,2,3,3,4,4- | 801212-5 | 59-9 | | | | | | | | | | |
| | hexafluoro-4-[1,2,2,2-tet | rafluoro-1- | | | | | | | | ND | ND | ND | ND | |
| | (trifluoromethyl)ethoxy]- | | | | ND | ND | ND | ND | ND | | | | | |
| TAFN4 / PFO5DA | Perfluoro(3,5,7,9,11-pent | tadodecanoic) acid | 39492-91 | -6 | 3.29 | ND | ND | 3.00 | 3.2 | ND | 2.69 | 1.85 | 1.75 | |
| PFHpA | Perfluoroheptanoic acid | | 375-85-9 | | 24.7 | 12.8 | 6.93 | 35.4 | 39.5 | 14.0 | 18.9 | 20.2 | 21.9 | |
| HFPO-DA / PFPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | L,2,2,3,3,3- | 13252-13 | 8-6 | | | | | | | | | | |
| "GenX" | heptafluoropropoxy)-pro | panoic acid | | | 33.1 | 17.6 | 9.43 | 48.5 | 63.3 | 16.2 | 22.7 | 20.9 | 20.7 | |
| | | | | At <u>tache</u> | 200 | 86 | 49 | 272 | 360 | 101 | 135 | 136 | 134 | |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | K | L | L | L | L | М | М | М |
|------------------------|-------------------|---|---|----------------------|-----------------|---------------------------------------|----------------------------------|----------------------------------|-----------------------------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|
| | | | | | | RIVE | ADAM | ADAM | ADAM | ADAM | ARBO | ARBO | ARBO |
| | | | | | | Heat-T/Dup_4 Municipal Shalotte | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington | Heat-T Municipal Wilmington | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington |
| Comm | on Name | Chemio | cal Name | c | ASN | Bruns. 8/29/19 | New Han. 10/24/19 | New Han. 10/24/19 | New Han. 10/24/19 | New Han. 10/24/19 | New Han. 10/25/19 | New Han. 10/25/19 | New Han. 10/25/19 |
| Detected PFA | AS listed in Atta | achment C of 2/25/19 Cons | sent Order | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 31.1 | 63.9 | 68.9 | 55.9 | 72.3 | 71.6 | 68.7 | 62.8 |
| ΡΜΡΑ | PFMOPrA | Perfluoro-2- methoxypropanoic acid | Perfluoro-3- methoxypropanoic acid | 13140-29 9 | 9- 377-73-1 | 8.90 | 25.9 | 17.8 | 30.4 | 36.4 | 28.4 | 36.5 | 27.8 |
| PFO2HxA | | Perfluoro(3,5-dioxahexar | noic) acid | 39492-88 | 3-1 | 26.7 | 57.9 | 56.5 | 54.5 | 70.9 | 68.5 | 72.6 | 46.8 |
| ΡΕΡΑ | PFMOBA | 2,3,3,3-Tetrafluoro-2- (pentafluoroethoxy) propanoic acid | Perfluoro-4- methoxybutanoic acid | 267239- 61-2 | 863090- 89-5 | 18.4 | 11.3 | 11.2 | 9.66 | 11.8 | 8.47 | 12.6 | 7.44 |
| PFO3OA | | Perfluoro(3.5.7-trioxaoct | anoic) acid | 39492-89 | 9-2 | 9.78 | 18.7 | 17.1 | 15.4 | 23.9 | 18.1 | 21.5 | 16.5 |
| PFO4DA | | Perfluoro(3,5,7,9-tetrao | (adecanoic) acid | 39492-90 |)-5 | 3.87 | 4.56 | 4.21 | 3.40 | 5.28 | 4.18 | 4.78 | 3.58 |
| PFESA-BP1/ | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 67-9 |)-3; 29311- | ND | ND | ND | ND | ND | ND | ND | ND |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 20-2 | 2.01 | 2.14 | 2.04 | 1.54 | 1.77 | 1.71 | 2.21 | 1.84 |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 (trifluoromethoxy)-; Buta hexafluoro-4-[1,2,2,2-tet (trifluoromethyl)ethoxy]- | I,5,5,6,6-decafluoro-6- noic acid, 2,2,3,3,4,4- rafluoro-1- | 174767-1 801212-5 | 10-3; 59-9 | ND | ND | ND | ND | ND | ND | ND | ND |
| TAFN4 / PFO | 5DA | Perfluoro(3.5.7.9.11-pent | tadodecanoic) acid | 39492-91 | L-6 | 1.92 | 1.56 | ND | ND | ND | ND | 1.31 | 1.40 |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 18.8 | 21.6 | 20.5 | 15.3 | 19.9 | 16.7 | 20.8 | 15.1 |
| HFPO-DA / PI "GenX" | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 heptafluoropropoxy)-pro | .,2,2,3,3,3- panoic acid | 13252-13 | 3-6 | 20.0 | 40.3 | 40.2 | 27.1 | 32.7 | 29.8 | 33 | 28.3 |
| | | ÷ | | | Attache | 141 | 248 | 238 | 213 | 275 | 247 | 274 | 212 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | М | Ν | Ν | N | N | 0 | 0 | Ο |
|-------------|-------------------|---------------------------------------|---------------------------------------|------------------|-----------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|
| | | | | | | ARBO | BOHN | BOHN | BOHN | BOHN | BOTS | BOTS | BOTS |
| | | | | | | Heat-T Municipal Wilmington | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington | Heat-T Municipal Wilmington | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington |
| | | | | | | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. |
| Comm | ion Name | Chemic | cal Name | C. | ASN | 10/25/19 | 8/28/19 | 8/28/19 | 8/28/19 | 8/28/19 | 8/27/19 | 8/27/19 | 8/27/19 |
| Detected PF | AS listed in Atta | achment C of 2/25/19 Cons | sent Order | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 57.4 | 41.2 | 38.5 | ND | ND | 43.9 | 47.0 | 45.6 |
| PMPA | PFMOPrA | Perfluoro-2- methoxypropanoic acid | Perfluoro-3- methoxypropanoic acid | 13140-29 9 | - 377-73-1 | 27.6 | 11.5 | 11.8 | ND | ND | 16.2 | 15.5 | 13.1 |
| PFO2HxA | | Perfluoro(3,5-dioxahexan | oic) acid | 39492-88 | -1 | 52.3 | 36.1 | 35.7 | ND | ND | 42.6 | 41.2 | 37.3 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- 61-2 | 863090- 89-5 | | 8 37 | 6.03 | ND | ND | 8.01 | 6.03 | 5 28 |
| | | propanoic acid | | 012 | 05 5 | 8.57 | 0.57 | 0.03 | | | 0.01 | 0.05 | 5.20 |
| PFO3OA | | Perfluoro(3,5,7-trioxaoct | anoic) acid | 39492-89 | -2 | 17.7 | 12.4 | 11.3 | ND | ND | 11.0 | 10.9 | 11.4 |
| PFO4DA | | Perfluoro(3,5,7,9-tetrao> | adecanoic) acid | 39492-90 | -5 | 2.93 | 2.35 | 2.50 | ND | ND | 2.17 | 1.86 | 2.04 |
| PFESA-BP1/ | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 67-9 | -3; 29311- | ND | ND | ND | ND | ND | ND | ND | ND |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 0-2 | 1.77 | ND | ND | ND | ND | ND | ND | ND |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | ,5,5,6,6-decafluoro-6- | 174767-1 | .0-3; | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | noic acid, 2,2,3,3,4,4- | 801212-5 | 9-9 | | ND | ND | ND | ND | ND | ND | ND |
| | | hexafluoro-4-[1,2,2,2-tet | rafluoro-1- | | | | | ND | ND | ND | | ND | |
| | | (trifluoromethyl)ethoxy]- | | | | ND | | | | | | | |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pent | adodecanoic) acid | 39492-91 | -6 | ND | ND | ND | ND | ND | ND | ND | ND |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 17.1 | 10.1 | 9.89 | ND | ND | 9.91 | 9.78 | 8.02 |
| HFPO-DA / P | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | ,2,2,3,3,3- | 13252-13 | -6 | | 19.0 | 16.9 | ND | ND | 20.2 | 10.0 | 16.9 |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | 26.3 | 10.0 | 10.8 | ND | ND | 20.2 | 19.0 | 10.0 |
| | | | | | Attache | 212 | 140 | 133 | - | - | 154 | 151 | 140 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | 0 | Р | | Q | Q | Q | Q | R |
|---------------|------------------|----------------------------|-------------------------|----------|------------|------------|------------|------------|------------|------------|------------|------------|------------|
| | | | | | | BOTS | CARR | CARR | HYAN | HYAN | HYAN | HYAN | JAYB |
| | | | | | | Heat-T | Tap-F | Tap-S | Tap-F | Tap-S | Heat-B | Heat-T | Tap-F |
| | | | | | | Municipal |
| | | | | | | Wilmington |
| | | | | | | New Han. |
| Comm | on Name | Chemic | al Name | С | ASN | 8/27/19 | 10/24/19 | 10/24/19 | 10/25/19 | 10/25/19 | 10/25/19 | 10/25/19 | 8/28/19 |
| Detected PFA | S listed in Atta | achment C of 2/25/19 Cons | sent Order | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 46.0 | 79.6 | 78.8 | 68.0 | 71.1 | 63.6 | 65.8 | 42.5 |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | 14.2 | | | | | | | 11.6 |
| | | | | | | | 28.9 | 25.4 | 22.3 | 23.3 | 21.5 | 20.6 | |
| PFO2HxA | | Perfluoro(3,5-dioxahexan | noic) acid | 39492-88 | 8-1 | 43.8 | 68.4 | 68.2 | 64.8 | 60.5 | 55.5 | 61.3 | 39.4 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | |
| | | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | 6.64 | | | | | | | 6.17 |
| | | propanoic acid | | | | | 11.6 | 12.3 | 10.7 | 11.0 | 10.7 | 11.5 | |
| PFO3OA | | Perfluoro(3,5,7-trioxaoct | anoic) acid | 39492-89 | -2 | 12.0 | 19.3 | 20.2 | 18.3 | 18.7 | 16.7 | 21.0 | 10.7 |
| PFO4DA | | Perfluoro(3,5,7,9-tetrao> | (adecanoic) acid | 39492-90 |)-5 | 3.10 | 3.64 | 4.62 | 4.86 | 4.02 | 4.37 | 3.97 | 2.68 |
| PFESA-BP1/I | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 | -3; 29311- | | | | | | | | ND |
| | | | | 67-9 | | ND |
| PFESA-BP2 / I | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 0-2 | ND | 1.91 | 2.48 | 2.24 | 2.32 | 1.90 | 2.25 | ND |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | I,5,5,6,6-decafluoro-6- | 174767-1 | .0-3; | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | noic acid, 2,2,3,3,4,4- | 801212-5 | 9-9 | ND | | | | | | | ND |
| | | hexafluoro-4-[1,2,2,2-tet | rafluoro-1- | | | ND | | | | | | | |
| | | (trifluoromethyl)ethoxy]- | | | | | ND | ND | ND | ND | ND | ND | |
| TAFN4 / PFO5 | 5DA | Perfluoro(3,5,7,9,11-pent | tadodecanoic) acid | 39492-91 | 6 | ND | ND | ND | 1.55 | ND | ND | ND | ND |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 9.55 | 20.2 | 22.6 | 19.5 | 18.1 | 18.9 | 19.6 | 9.63 |
| HFPO-DA / PF | PrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | .,2,2,3,3,3- | 13252-13 | -6 | 10.0 | | | | | | | 107 |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | 18.8 | 35.6 | 40.6 | 36.8 | 34.5 | 34.9 | 39.8 | 16./ |
| | | | | ÷. | Attache | 154 | 269 | 275 | 249 | 244 | 228 | 246 | 139 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | R | R | R | R | S | S | S | S |
|--------------|-------------------|---------------------------------------|-------------------------|------------------|------------|--|----------------------------------|-----------------------------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|-----------------------------------|
| | | | | | | JAYB | JAYB | JAYB | JAYB | LAUR | LAUR | LAUR | LAUR |
| | | | | | | Tap-F/Dup_1 Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington | Heat-T Municipal Wilmington | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington | Heat-T Municipal Wilmington |
| Comm | on Name | Chemic | al Name | с. | ۵SN | New Han. 8/28/19 | New Han. 8/28/19 | 8/28/19 | 8/28/19 | New Han. 8/27/19 | 8/27/19 | 8/27/19 | 8/27/19 |
| Detected PFA | AS listed in Atta | achment C of 2/25/19 Cons | sent Order | | | 0, 20, 20 | 0, 20, 20 | 0,20,20 | 0,20,20 | 0, 1, 10 | 0,21,20 | 0, 1, 1, 20 | 0,27,20 |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 45.7 | 47.1 | 28.8 | 44.2 | 35.8 | 43.7 | ND | 46.9 |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | 13.1 | 12.5 | 7.61 | 12.9 | 15.7 | 13.7 | ND | 13.5 |
| PFO2HxA | | Perfluoro(3,5-dioxahexan | oic) acid | 39492-88 | -1 | 42.2 | 43.4 | 23.1 | 37.8 | 34.4 | 39.1 | ND | 41.6 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | |
| | | (pentafluoroethoxy) propanoic acid | methoxybutanoic acid | 61-2 | 89-5 | 5.93 | 5.69 | 2.99 | 5.70 | 4.72 | 6.24 | ND | 4.77 |
| PFO3OA | | Perfluoro(3,5,7-trioxaoct | anoic) acid | 39492-89 | -2 | 12.8 | 10.7 | 6.64 | 10.2 | 6.68 | 12.1 | ND | 9.22 |
| PFO4DA | | Perfluoro(3,5,7,9-tetraox | (adecanoic) acid | 39492-90 | -5 | 2.08 | 3.13 | 1.59 | 2.54 | 2.63 | 3.19 | ND | 2.31 |
| PFESA-BP1/ | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 67-9 | -3; 29311- | ND | ND | ND | ND | ND | ND | ND | ND |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 0-2 | ND | ND | ND | ND | ND | ND | ND | ND |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | ,5,5,6,6-decafluoro-6- | 174767-1 | 0-3; | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | noic acid, 2,2,3,3,4,4- | 801212-5 | 9-9 | ND | ND | | ND | | | ND | ND |
| | | hexafluoro-4-[1,2,2,2-tetr | rafluoro-1- | | | ND | ND. | ND | | ND | ND | ND | ND |
| | | (trifluoromethyl)ethoxy]- | | | | | | | | | | | |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pent | adodecanoic) acid | 39492-91 | -6 | ND | ND | ND | ND | ND | ND | ND | ND |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 9.82 | 8.63 | 6.33 | 7.98 | 8.70 | 9.76 | ND | 9.98 |
| HFPO-DA / PF | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | ,2,2,3,3,3- | 13252-13 | -6 | 16 0 | 10 0 | 12.2 | 16.2 | 20.0 | 10.2 | | 17 7 |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | 10.8 | 10.0 | 12.3 | 10.2 | 20.0 | 15.2 | ND | 1/./ |
| | | | | | Attache | 148 | 150 | 89 | 138 | 129 | 147 | - | 146 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | Т | Т | Т | Т | U | U | U | U |
|-------------|-------------------|----------------------------|-------------------------|----------|-------------|------------|------------|------------|------------|------------|------------|------------|------------|
| | | | | | | LULL | LULL | LULL | LULL | MASO | MASO | MASO | MASO |
| | | | | | | Tap-F | Tap-S | Heat-B | Heat-T | Tap-F | Tap-S | Heat-B | Heat-T |
| | | | | | | Municipal |
| | | | | | | Wilmington |
| | | | | | | New Han. |
| Comm | non Name | Chemic | al Name | C | ASN | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/25/19 | 10/25/19 | 10/25/19 | 10/25/19 |
| Detected PF | AS listed in Atta | achment C of 2/25/19 Cons | sent Order | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 70.9 | 63.9 | 54.4 | 59.6 | 73.5 | 63.3 | 67.8 | 72.5 |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | | | | | | | | |
| | | | | | | 23.0 | 20.5 | 25.7 | 29.7 | 37.7 | 34.3 | 34.9 | 38.3 |
| PFO2HxA | | Perfluoro(3,5-dioxahexan | oic) acid | 39492-88 | 8-1 | 66.6 | 55.4 | 49.8 | 62.7 | 68.1 | 66.1 | 69.1 | 74.3 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | |
| | | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | | | | | | | | |
| | | propanoic acid | | | | 12.7 | 10.1 | 9.30 | 9.67 | 12.7 | 11.1 | 11.8 | 12.8 |
| PFO3OA | | Perfluoro(3,5,7-trioxaoct | anoic) acid | 39492-89 |)-2 | 20.8 | 17.3 | 17.4 | 17.4 | 20.7 | 20.0 | 19.7 | 24.2 |
| PFO4DA | | Perfluoro(3,5,7,9-tetraox | adecanoic) acid | 39492-90 |)-5 | 5.62 | 3.85 | 3.4 | 3.21 | 3.87 | 4.53 | 4.35 | 4.45 |
| PFESA-BP1/ | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 |)-3; 29311- | | | | | | | | |
| | | | | 67-9 | | ND |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 20-2 | 2.3 | 2.11 | 1.48 | 1.71 | 2.15 | 1.86 | 2.10 | 2.10 |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 | ,5,5,6,6-decafluoro-6- | 174767-1 | .0-3; | | | | | | | | |
| | | (trifluoromethoxy)-; Buta | noic acid, 2,2,3,3,4,4- | 801212-5 | 9-9 | | | | | | | | |
| | | hexafluoro-4-[1,2,2,2-teti | rafluoro-1- | | | | | | | | | | |
| | | (trifluoromethyl)ethoxy]- | | | | ND |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pent | adodecanoic) acid | 39492-91 | -6 | ND | 1.24 | ND | 1.63 | ND | 1.41 | 1.67 | ND |
| PFHpA | | Perfluoroheptanoic acid | - | 375-85-9 | | 17.7 | 19.4 | 15.5 | 17.7 | 18.4 | 17.9 | 20.1 | 21.2 |
| HFPO-DA / P | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 | ,2,2,3,3,3- | 13252-13 | 8-6 | | | | | | | | |
| "GenX" | | heptafluoropropoxy)-pro | panoic acid | | | 44.2 | 35.7 | 27.8 | 29.7 | 32.4 | 30.1 | 31.7 | 34.5 |
| | | | | | Attache | 264 | 230 | 205 | 233 | 270 | 251 | 263 | 284 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | V | V | V | V | W | | | |
|-----------------------|-------------------|--|---|----------------------|-----------------|----------------------------------|----------------------------------|-----------------------------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|-----------------------------------|
| | | | | | | ONEI | ONEI | ONEI | ONEI | ΟΥST | ΟΥST | ΟΥST | ΟΥST |
| | | | | | | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington | Heat-T Municipal Wilmington | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B Municipal Wilmington | Heat-T Municipal Wilmington |
| | | | | | | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. |
| Comm | non Name | Chemic | cal Name | C | ASN | 8/27/19 | 8/27/19 | 8/27/19 | 8/27/19 | 10/25/19 | 10/25/19 | 10/25/19 | 10/25/19 |
| Detected PF | AS listed in Atta | achment C of 2/25/19 Cons | sent Order | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyace | tic acid | 674-13-5 | | 38.9 | 43.4 | 51.3 | 46.4 | 56.3 | 59.3 | 64.5 | 72.5 |
| РМРА | PFMOPrA | Perfluoro-2- methoxypropanoic acid | Perfluoro-3- methoxypropanoic acid | 13140-29 9 | - 377-73-1 | 22.0 | 12.8 | 14.4 | 13.7 | 32.0 | 33.0 | 31.3 | 34.3 |
| PFO2HxA | | Perfluoro(3,5-dioxahexan | oic) acid | 39492-88 | 8-1 | 33.4 | 43.1 | 40.1 | 41.0 | 58.9 | 59.3 | 69.1 | 67.6 |
| ΡΕΡΑ | PFMOBA | 2,3,3,3-Tetrafluoro-2- (pentafluoroethoxy) | Perfluoro-4- methoxybutanoic acid | 267239- 61-2 | 863090- 89-5 | 8.18 | 4.08 | 4.67 | 4.11 | 10.4 | 10.4 | 10 5 | 10.4 |
| ΡΕΟЗΟΔ | | Perfluoro(3 5 7-trioxaoct | anoic) acid | 39497-80 | 1-7 | 6.5 | 12.2 | 10.4 | 11.2 | 17.3 | 16.5 | 18.2 | 17.6 |
| PFO4DA | | Perfluoro(3 5 7 9-tetraox | (adecanoic) acid | 39492-90 |)-5 | 2.09 | 3 21 | 2.9 | 2 32 | 4.29 | 4.47 | 4.46 | 4.29 |
| PFESA-BP1 / | Nafion BP #1 | Nafion Byproduct 1 | | 66796-30 67-9 | -3; 29311- | ND | ND | ND | ND | ND | ND | ND | ND |
| PFESA-BP2 / | Nafion BP #2 | Nafion Byproduct 2 | | 749836-2 | 0-2 | ND | 1.22 | ND | ND | 1.87 | 1.88 | 1.75 | 2.07 |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4 (trifluoromethoxy)-; Buta hexafluoro-4-[1,2,2,2-tetr (trifluoromethyl)ethoxy]- | 1,5,5,6,6-decafluoro-6- noic acid, 2,2,3,3,4,4- rafluoro-1- | 174767-1 801212-5 | .0-3; 9-9 | ND | ND | ND | ND | ND | ND | ND | ND |
| TAFN4 / PFO | 5DA | Perfluoro(3,5,7,9,11-pent | adodecanoic) acid | 39492-91 | 6 | ND | ND | ND | ND | ND | ND | 1.44 | ND |
| PFHpA | | Perfluoroheptanoic acid | , | 375-85-9 | | 5.71 | 10.2 | 10.6 | 9.93 | 16.7 | 18.4 | 18.6 | 20.5 |
| HFPO-DA / P "GenX" | FPrOPrA / | 2,3,3,3-Tetrafluoro-2 (1,1 heptafluoropropoxy)-pro | ,2,2,3,3,3- panoic acid | 13252-13 | 8-6 | 18.7 | 18.3 | 21.1 | 16.4 | 28.4 | 29.1 | 29.9 | 35.0 |
| | | | | | Attache | 135 | 149 | 155 | 145 | 226 | 232 | 250 | 264 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | Х | X | Х | Х | X | Y | Y | Z |
|---|---------|--|-----------------------|--------------|----------------------------------|----------------------------------|--|-----------------------------------|-----------------------------------|----------------------------------|----------------------------------|-------------------------------------|----------|
| | | | | | | RAND | RAND | RAND | RAND | RAND | TREE | TREE | WIND |
| | | | | | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Tap-S/DUP_1 Municipal Wilmington | Heat-B Municipal Wilmington | Heat-T Municipal Wilmington | Tap-F Municipal Wilmington | Tap-S Municipal Wilmington | Heat-B.a Municipal Wilmington | |
| | | | | | New Han. | | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. | New Han. |
| Common Name | | Chemical Name | | C | ASN | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/24/19 | 10/25/19 | 10/25/19 | 3/13/19 |
| Detected PFAS listed in Attachment C of 2/25/19 Consent Order | | | | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyacetic acid | | 6/4-13-5 | | 67.9 | 60.3 | 62.7 | 74.0 | 72.0 | 48.0 | 60.9 | 21.7 |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | - 377-73-1 | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | | | | | | | | 5.01 |
| | | | | | | 22.4 | 18.6 | 18.9 | 21.9 | 24.3 | 29.6 | 32.7 | |
| PFO2HxA | | Perfluoro(3,5-dioxahexanoic) acid | | 39492-88 | 5-1 | 62.5 | 55.2 | 60.5 | 64.4 | 62.4 | 47.4 | 60.9 | 6.62 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | |
| | | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | | | | | | | | 2.74 |
| | | propanoic acid | | | | 11.3 | 11.0 | 11.1 | 12.5 | 11.0 | 6.74 | 11.7 | |
| PFO3OA | | Perfluoro(3,5,7-trioxaoctanoic) acid | | 39492-89-2 | | 18.4 | 16.5 | 17.4 | 19.2 | 16.7 | 13.5 | 22.4 | 12.5 |
| PFO4DA | | Perfluoro(3,5,7,9-tetraoxadecanoic) acid | | 39492-90-5 | | 4.6 | 3.74 | 4.63 | 3.69 | 4.3 | 3.16 | 4.84 | 4.74 |
| PFESA-BP1 / Nafion BP #1 | | Nafion Byproduct 1 | | 66796-30 | -3; 29311- | | | | | | | | ND |
| | | | | 67-9 | | ND | ND | ND | ND | ND | ND | ND | |
| PFESA-BP2 / Nafion BP #2 | | Nafion Byproduct 2 | | 749836-20-2 | | 2.25 | 1.89 | 2.12 | 2.16 | 2.49 | ND | 1.84 | ND |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4,5,5,6,6-decafluoro-6- | | 174767-10-3; | | | | | | | | | |
| | | (trifluoromethoxy)-; Butanoic acid, 2,2,3,3,4,4- | | 801212-5 | 9-9 | | | | | | | | |
| | | hexafluoro-4-[1,2,2,2-tetrafluoro-1- | | | | | | | | | | | ND |
| | | (trifluoromethyl)ethoxy]- | | | | ND | ND | ND | ND | ND | ND | ND | |
| TAFN4 / PFO5DA | | Perfluoro(3,5,7,9,11-pentadodecanoic) acid | | 39492-91-6 | | 1.25 | ND | ND | 1.47 | ND | ND | ND | ND |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 21.2 | 21.0 | 20.8 | 19.5 | 19.0 | 12.7 | 18.9 | 2.43 |
| HFPO-DA / PFPrOPrA / | | 2,3,3,3-Tetrafluoro-2 (1,1,2,2,3,3,3- | | 13252-13 | -6 | | | | | | | | |
| "GenX" | | heptafluoropropoxy)-propanoic acid | | | | 38.4 | 36.5 | 39.8 | 40.8 | 40.5 | 26.7 | 31.2 | 5.05 |
| | | | | | At <u>tache</u> | 250 | 225 | 238 | 260 | 253 | 188 | 245 | 61 |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.



units = ng/L

| | | | | | | Z | Z | Z | Z | AA | AA | AA | AA |
|--------------------------|-------------------|---|-----------------------|-------------|-------------|------------|------------|------------|-----------|-----------|-----------|-----------|-----------|
| | | | | | WIND | WIND | WIND | WIND | LIBE | LIBE | LIBE | LIBE | |
| | | | | | Heat-B.b | Heat-B.c | Heat-T.e | Heat-T.f | Tap-F | Tap-S | Heat-B | Heat-T | |
| | | | | | | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal | Municipal |
| | | | | | Wilmington | Wilmington | Wilmington | Wilmington | Winnabow | Winnabow | Winnabow | Winnabow | |
| | | | | | | New. Han. | New. Han. | New. Han. | New. Han. | Bruns. | Bruns. | Bruns. | Bruns. |
| Common Name | | Chemical Name | | C | ASN | 3/13/19 | 3/13/19 | 3/13/19 | 3/13/19 | 8/27/19 | 8/27/19 | 8/27/19 | 8/27/19 |
| Detected PF/ | AS listed in Atta | achment C of 2/25/19 Con | sent Order | | | | | | | | | | |
| PFMOAA | | Perfluoro- 2-methoxyacetic acid | | 674-13-5 | | 21.9 | 20.7 | 18.2 | 19.3 | 42.4 | 37.5 | 42.5 | 36.6 |
| PMPA | PFMOPrA | Perfluoro-2- | Perfluoro-3- | 13140-29 | 9-377-73-1 | | | | | | | | |
| | | methoxypropanoic acid | methoxypropanoic acid | 9 | | 7.15 | 7.36 | 7.27 | 8.46 | 11.5 | 9.84 | 9.38 | 10.1 |
| PFO2HxA | | Perfluoro(3,5-dioxahexanoic) acid | | 39492-88-1 | | 4.87 | 3.69 | 3.03 | 2.52 | 34.0 | 30.6 | 34.6 | 30.8 |
| PEPA | PFMOBA | 2,3,3,3-Tetrafluoro-2- | Perfluoro-4- | 267239- | 863090- | | | | | | | | |
| | | (pentafluoroethoxy) | methoxybutanoic acid | 61-2 | 89-5 | 2.77 | 2.71 | 2.05 | 2.43 | 7.29 | 5.84 | 7.12 | 5.91 |
| | | propanoic acid | | | | | | | | | | | |
| PFO3OA | | Perfluoro(3,5,7-trioxaoctanoic) acid | | 39492-89-2 | | 12.0 | 11.3 | 10.2 | 9.83 | 11.2 | 10.6 | 10.3 | 10.8 |
| PFO4DA | | Perfluoro(3,5,7,9-tetraoxadecanoic) acid | | 39492-90-5 | | 4.94 | 5.03 | 3.32 | 3.41 | 4.91 | 4.19 | 4.26 | 4.68 |
| PFESA-BP1 / Nafion BP #1 | | Nafion Byproduct 1 | | 66796-30 |)-3; 29311- | ND | ND | ND | ND | ND | ND | ND | ND |
| | | | | 67-9 | | ND | | | | | | | |
| PFESA-BP2 / Nafion BP #2 | | Nafion Byproduct 2 | | 749836-20-2 | | ND | ND | ND | ND | 2.66 | 2.58 | 2.34 | 2.48 |
| PFECA-G | | Hexanoic acid, 2,2,3,3,4,4,5,5,6,6-decafluoro-6- 17 | | 174767-1 | LO-3; | | | | | | | | |
| | | (trifluoromethoxy)-; Butanoic acid, 2,2,3,3,4,4- | | 801212-5 | 59-9 | ND | ND | ND | ND | ND | ND | ND | ND |
| | | hexafluoro-4-[1,2,2,2-tetrafluoro-1- (trifluoromethyl)ethoxy]- | | | | | | | | | | | |
| | | | | | | | | | | | | | |
| TAFN4 / PFO5DA | | Perfluoro(3,5,7,9,11-pentadodecanoic) acid | | 39492-91-6 | | ND | ND | ND | ND | 1.81 | 2.00 | ND | 1.86 |
| PFHpA | | Perfluoroheptanoic acid | | 375-85-9 | | 1.95 | 1.68 | 1.51 | 1.31 | 24.8 | 24.9 | 27.2 | 26.3 |
| HFPO-DA / PFPrOPrA / | | 2,3,3,3-Tetrafluoro-2 (1,1,2,2,3,3,3- | | 13252-13 | 8-6 | 4 74 | 5.01 | 5.50 | 5.21 | 27.7 | 25.1 | 25.0 | 25.2 |
| "GenX" | | heptafluoropropoxy)-propanoic acid | | | | 4.74 | | | | | | | |
| Attache | | | | | 60 | 57 | 51 | 52 | 168 | 153 | 163 | 155 | |

Notes/Comments:

999 = Sample result for single PFAS above 10 ng/L limit in Consent Order (Para. 20.a)

999 = Sample result for combination of PFAS above 70 ng/L limit in Consent Order (Para. 20.b)

CASN = Chemical abstracts service registry number

ng/L = Nanograms per liter (a.k.a. parts per trillion or pptr)

ND = Not detected

PFAS = Per- and polyfluoroalkyl substances

Samples collected in June 2018 and March 2019 tested consistent with U.S. Environmental Environmental Protect Agency (USE collected in August 2019 and October 2019 tested consistent with USEPA Method 537.1. All tests completed by GEL Laborator **Locations** - Dup = Blind duplicate, Heat-B = Bottom of water heater sample, Heat-T = Top of water heater sample, O.S. = Outsic outside tap, Out-S = Follow-up outside tap water sample, Tap-F = First-flush sample from tap, and Tap-S = Follow-up tap water **Wtr. Source** - Municipal = Minicipal water provider.

