Public Comments Received Regarding Addendum to Consent Order

Public Comment period:
August 17, 2020- September 17, 2020

From: Hope Taylor
To: comments.chemours
Subject: [External] Clean Water for NC Comments on Addendum to Chemours Consent Order
Date: Thursday, September 17, 2020 11:57:59 PM
Attachments: image001.png

Comments of Clean Water for North Carolina on Addendum to Consent Order Paragraph 12

Prepared by Hope Taylor, MSPH, Executive Director **Submitted September 17, 2010**

The continued production of PFAS with a wide range of side products of poorly characterized human health and ecological effects has caused contamination of both surface water and groundwater drinking water sources downstream and for miles around the Chemours facility. As an organization that worked closely with the surrounding community and other non-profits from 2005 through 2008 to hold then-DuPont Fayetteville Works accountable for the almost immediate onsite groundwater contamination after DuPont began C-8 (PFOA) production, and sought to have production of PFOA and related compounds halted, we were hopeful that the PFOA Stewardship agreement negotiated with EPA would significantly decrease environmental and human exposures to PFOA and related compounds. Instead, we learn that the Agreement essentially dropped the leash on PFAS production at the Fayetteville Works. DEQ, formerly DENR, had been singularly unresponsive to calls for additional oversight and accountability. Even staff within the agency who knew part of the story and sought to protect NC waters and air appear to have been marginalized and their concerns buried. Further, extensive contamination in other parts of the state due to use of PFAS in manufacture of a wide range of products has continued to show up more widely.

The greatest impact on drinking water supplies may even be outside North Carolina, in Michigan, a state that doesn't manufacture these products. Given the persistence in the environment, the health effects that ARE known at low concentrations and the completely unregulated consumption and use of these products by other manufacturers, the regulatory and health burden borne by the public is completely unacceptable. The "quality of life" improvements claimed by Chemours and its customers for these products do not justify the costs in human and ecological health. The only just solution to this ongoing contamination, continuing to accumulate over time, is to cease manufacturing of these substances or any products that could result in PFAS by-products. Internationally known scientists, including NIEHS retired Director have called for a complete phase out of all of this entire class of chemicals and we agree there is no convenience that they offer that can possibly justify their continued production. For DEQ to continue to allow appallingly weakly regulated production that has consistently contaminated the public's resources from the start of production for nearly two decades is unconscionable.

Comment on pages 1-3

The purpose of Paragraph 12 of the Consent Order was to require Chemours to submit a plan for maximally feasible reductions of PFAS releases that can be achieved within two years. On repeated occasions, Chemours submitted plans to DEQ that did NOT come close to achieving maximum feasible reductions. DEQ and CFRW have been far too tolerant of Chemours' continued efforts to escape accountability, while proposing inadequate controls of all releases of PFAS. Given that the public has already been deprived of considerable value through the

contamination of its air, surface water and groundwater, with future health effects that will be nearly impossible to quantify, DEQ has all of the information it needs to require production to be greatly curtailed or stopped UNTIL Chemours can demonstrate complete removal of all PFAS species from its air, groundwater and surface water releases. **The company has demonstrated that either there is NO safe way to produce these persistent, toxic compounds, or that they don't accept the responsibility for doing so.**

Further, the public has been bearing the regulatory costs of "managing" completely avoidable impacts to the environment, including drinking water sources—this is a massive economic injustice, in addition to the criminal legacy of health effects that residents of several states face. Rather than taking Chemours' word for what is feasible, DEQ must demand financial information about all production costs, profits and a list of every customer for its products that include any compounds in the PFAS class , and that list must be made public in order to enable customers of a wide range of products to choose to protect their health and environmental receptors from exposure to these substances. Rather than allow for up to five years of continuing reductions, the only serious leverage that DEQ must exercise, given the continuing accumulation of PFAS in ecosystems and in the blood and other tissues of humans and other animals, is to **stop production of PFAS entirely**. To continue to grant an irresponsible corporation additional time to achieve inadequate reductions which, based on long experience, Chemours will likely to continue to evade, is wishful thinking and completely fails to protect the public health and natural resources.

PFAS reduction measures

As of this date, it appears DEQ is still totally dependent on Chemours for determining mass loading of PSAF from the seeps, other discharge points, stormwater and air emissions, an appalling weak position for a regulatory agency that should have been corrected long ago in permitting actions, and certainly within a month of the reports of PSAF in drinking water supplies downstream.

All sampling must be witnessed by DEQ or a delegated and trained representative of CFRW, whose salary and travel expenses must be reimbursed by Chemours.

All samples must be split with DEQ and CFRW which shall be independently submitted to certified laboratory and DEQ/CFRW will be reimbursed for the cost of analyses.

DEQ should have notified the public at least through their website if the required actions by August 31, 2020 have been satisfactorily completed .

The schedule for achieving interim control of seeps is too generous, and can be achieved more quickly and in parallel for the four seeps if needed. As soon as plans that should have been submitted by Aug. 31 are approved, construction must begin and be completed by Dec. 1, 2020. If plans submitted are late or inadequate to result in seep control, Chemours must be assessed stipulated penalty immediately.

All inspections and sampling events must be noticed to DEQ and CFRW at least 24 hours in advance and observed by a representative of DEQ and/or CFRW with Chemours responsible for reimbursing DEQ and CFRW for all documented expenses and salary.

All reporting to DEQ and CFRW must happen within 24 hours of any "upset" or uncontrolled even that may have resulted in a release, with immediate investigation and reporting to NC

DEQ and CFRW within 48 business hours of receipt of any analytical results.

Reporting to downstream utilities must occur within 4 hours of any upset or uncontrolled release, rather than within 24 hours, to allow for prompt closure of intakes and connection of water supply to alternative sources.

Evaluation of Interim Effectiveness

Removal efficiency of only 80% is completely unacceptable, even on an interim basis. That low expectations removal requirement would still allow for significant discharge of PFAS to the environment, further adding to levels of persistent PFAS in soils, sediments and fish, with potential remobilization into groundwater and human exposure. If a removal efficiency of 95% is not sufficient to give a final discharge concentration of less than 70 ppt., the discharge must be collected and further treated to this level before release to public waters. If the interim demonstration has not been achieved by June 1, 2021, the full installation of a complete capture and treatment must be required by December 31at, 2021.

On site stormwater must be treated to achieve a standard of less than 70, ppt for all PFAS species, rather than simply a 99% reduction, given the high volume of stormwater that may be discharged.

Regulation and loading based on all PFAS species, testing costs to be born by Chemours, sampling by water utilities and other independent entities

Additionally, Chemours must demonstrate their ability to measure all PFAS compounds in their effluent (not limited to GenX, PMPA, and PFMOAA).and provide detailed scientific evidence for any compounds used as indicators and the appropriate factor to calculate total PFAS loading. Measuring only certain groups of PFAS chemicals does not prevent the downstream communities from contamination of other PFAS chemicals.

Chemours must be responsible for the costs of all testing for the full suite of PFAS compounds in drinking water sources, with all sampling to be done by water utility staff.

Chemours must achieve PFAS removal efficiency of at least 99.9%, during both dry and wet weather conditions. Defining dry and wet weather can be challenging. Failure to reach a clear definition for enforcement will expose downstream communities to risk of PFAS exposure and accumulation. Therefore, the standard should be clear and enforceable all the time. Again, Chemours must be required to quantify all PFAS chemicals, not just GenX, PMPA, and PFMOAA.

Reporting monitoring results quarterly is far from sufficient. Chemours should be responsible for biweekly monitoring and be responsible for all costs associated with water quality analyses, with split samples to DEQ.

Barrier Wall and Groundwater Extraction and Treatment

The Barrier Wall and Groundwater Extraction system design must be submitted with review and DEQ approval by August 15, 2021, and construction completed by June of 2022. Time

allowed to design and construct this important system and start operations is far too generous. Treatment system must have a removal efficiency of 99.99 % efficiency.

Stipulated penalties.

The stipulated penalties for non-compliance with the required activities and performance standards are too low to prevent Chemours from gaining a net benefit from its failure to comply. In additional to paying penalties, Chemours must be required to install water filtration systems for all downgradient households within ½ mile until the facility demonstrates for a period of at least 3 years that it is discharging total PFAS less than 70 ppt, with 20% lower standard each year following, due to PFAS persistence in the environment. Any failure to meet subsequent lowered discharge standards should require installation of whole house filtration systems and maintenance for all downgradient households for another ½ mile radius within 3 months.

Thank you for the opportunity to comment on the Addendum to Paragraph 12 of the Consent Order. Clean Water for North Carolina has always believed in the stated goal of agency enforcement policy, that the scale of penalties assessed must be sufficient to compensate for the value of damage caused by a polluter's activities, and that the permittee must see no net benefit from its non-compliance. This Addendum as currently written, and the Consent Order fall far short of this goal, as well as failing to protect human and ecological health. Chemours has, for many years, been allowed to profit massively while degrading the natural resources and health of the people of North Carolina. The only appropriate action commensurate with the scale of the damage that has been done is to require production of all PFAS and any substances that have PFAS by-products to cease. Only then will NC DEQ have the leverage to require this corporation that has profited greatly from production at the public's expense to perform fully accountable clean up, and to only return to production of materials that do not force the people of NC and other states to bear burdenss that so greatly outweigh any convenience the PFAS have provided.

Yours truly,

Hope Taylor, MSPH hope#cwfnc.org

Executive Director, Clean Water for North Carolina

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From: bredl@skybest.com
To: comments.chemours
Cc: Holman, Sheila
Subject: [External] Chemours Public Comments
Date: Thursday, September 17, 2020 8:28:31 PM
Attachments: 200917_BREDL_comments PFAS NCDEQ Add SOC .pdf
Importance: High

September 17, 2020, 8:20 PM

Sheila Holman, DEQ Assistant Secretary
Assistant Secretary's Office
1601 Mail Service Center
Raleigh, NC 27699-1601

RE: Chemours Public Comments

Dear Ms. Holman:

Please find attached to this email comments on the proposed Addendum to the Consent Order Paragraph 12, which requires additional actions by Chemours to prevent PFAS pollution from entering the Cape Fear River via contaminated groundwater from the Fayetteville Works Site.

Thank you for the opportunity to provide these remarks.

Be well,

Louis A. Zeller
Louis A. Zeller, Executive Director
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Founded in 1984, we have projects and chapters in Alabama, Georgia, Tennessee, South Carolina, North Carolina and Virginia

We should impart our courage, and not our despair, our health and ease, and not our disease, and take care that this does not spread by contagion.—Henry David Thoreau, Walden

Blue Ridge Environmental Defense League

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September 17, 2020

Sheila Holman, DEQ Assistant Secretary
Assistant Secretary's Office
1601 Mail Service Center
Raleigh, NC 27699-1601

RE: Chemours Public Comments

Dear Ms. Holman:

On behalf of the Blue Ridge Environmental Defense League, I write to comment on the proposed Addendum to the Consent Order Paragraph 12, which requires additional actions by Chemours to prevent PFAS pollution from entering the Cape Fear River via contaminated groundwater from the Fayetteville Works Site.

As you know, PFAS, perfluoroalkyl substances, are manmade, fluorinated compounds, also known as PFC, polyfluorinated compounds. The term PFAS includes a class of more than 5,000 compounds, such as Perfluorooctane Sulfonic Acid (PFOS) and Perfluorooctonoic Acid (PFOA), GenX (in which the PFAS hexafluoropropylene oxide is used to manufacture) and many more. The problems are that PFAS are persistent chemicals which do not break down in soil or water, which can bioaccumulate in the food chain and which have negative impacts on human health.

According to the US EPA: "Studies indicate that PFOA and PFOS can cause reproductive and developmental, liver and kidney, and immunological effects in laboratory animals. Both chemicals have caused tumors in animals." [1]

Further, the national Center for Health and Environmental Justice finds,

> Human studies found similar results to animal studies, namely associations between PFCs and liver, hormone, and immune system function. Some epidemiological studies have linked PFC exposure to kidney and testicular cancers in people. In addition, PFC exposure has been linked to hypertension in pregnant women, slightly lower birth weight in infants and elevated blood cholesterol levels. Other studies have found that higher PFC levels are associated with a potential decrease in vaccine efficacy.[2] (citations omitted)

In 2016 an EPA health advisory set a limit of 0.07 ug/L for both PFOS and PFOA combined in water.[3]

[1] USEPA, "Basic Information on PFAS," https://www.epa.gov/pfas/basic-information-pfas
[2] Center for Health and Environmental Justice "PFC Fact Sheet," http://chej.org/wp-content/uploads/PFC-Fact-Sheet-Toxicity.pdf
[3] USEPA Fact Sheet PFOA & PFOS Drinking Water Health Advisories, May 2016, EPA 800-F-16-003. https://www.epa.gov/sites/production/files/2016-06/documents/drinkingwaterhealthadvisories_pfoa_pfos_updated_5.31.16.pdf

Going the EPA one better, on July 24, 2019, the state of New York adopted a rule which set enforceable limits for PFOA and PFOS at 10 parts per trillion, and for 1,4-dioxane at 1 part per billion. And these maximum contaminant limits apply to all 278 water systems in the state, with testing to have begun within six months.

Effective ways of removing PFAS from contaminated drinking water are available: activated carbon treatment, ion exchange resins, nanofiltration or reverse osmosis. The question of who is to pay must be answered. We believe that the source should be the deep pockets of the manufacturers who invented and mass-produced the problem.

Contaminants of Emerging Concern is a term used by water quality professionals to describe pollutants that have been detected in water bodies that may cause ecological or human health impacts and typically are not regulated under current environmental laws.[4]

The discovery of PFAS in the Cape Fear watershed, along with 1,4 dioxane and other emerging contaminants, should spur a rapid science-based response by state and local public officials in North Carolina. And the response must include a statewide public system which affords people the ability to find their potential exposure and health risk in order to take steps to protect themselves, their families and their communities.

Respectfully,

Louis A. Zeller
Executive Director

[4] Contaminants of Emerging Concern including Pharmaceuticals and Personal Care Products". Water Quality Criteria. Washington, D.C.: U.S. Environmental Protection Agency (EPA). 2019-08-19, https://www.epa.gov/wqc/contaminants-emerging-concern-including-pharmaceuticals-and-personal-care-products

From: Bell, Cori
To: comments.chemours
Subject: [External] Chemours Public Comments: Consent Order Addendum
Date: Thursday, September 17, 2020 5:12:35 PM
Attachments: image001.png
NRDC Chemours Addendum comment .pdf

Assistant Secretary Holman,

Attached, please find NRDC's comments regarding the proposed Addendum to the Chemours Consent Order. Please feel free to reach out to me if you have any questions or concerns.

Best,
Cori

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Sheila Holman
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Via email: comments.chemours@ncdenr.gov

September 17, 2020

RE: Chemours Public Comments

Ms. Holman:

On behalf of the Natural Resources Defense Council (NRDC) and its 69,000+ members and activists in North Carolina, I am writing to submit comments on the Addendum to Chemours Consent Order Paragraph 12. We appreciate the opportunity to submit comments on this important aspect of the Consent Order, which seeks to address PFAS loading from the Fayetteville Works Facility (Facility) via groundwater, stormwater, and on-site streams.

We are generally supportive of the proposed Addendum, with the exception of some aspects listed below. The actions outlined in the Addendum would help curb significant sources of PFAS pollution from the Facility, benefiting North Carolinians and the Cape Fear River ecosystem. Specifically, we are supportive of the use of interim benchmarks, enforceable requirements, and stipulated penalties. The Addendum could be strengthened by addressing the concerns noted below.

The Addendum should require more prompt submission and public disclosure of sampling data. Reporting on a bimonthly or quarterly basis is not frequent enough to identify and correct potential problems. Monitoring results should be submitted to DEQ monthly, and DEQ should post submitted data on its website within a week of receipt. The public availability of this information is vital. Additionally, sampling during rainfall events should occur within the first hour of the rainfall event to ensure an accurate measure of pollutants leaving the site. Further, it should be made clear that the stipulated penalties on page 22 of the Addendum apply for the failure to collect and/or report sampling data.

Finally, the Addendum does not directly address contaminated drinking water sources, particularly sources for drinking water wells and downstream drinking water systems. Wells in the area around the Fayetteville Works Facility have had their drinking water source contaminated with PFAS, and the same is true for hundreds of thousands of people downstream from the Facility. While the Consent Decree requires Chemours to address certain drinking water issues for well owners, it does not require the cleanup of drinking water sources for wells or downstream water users. Exposure to PFAS-laden drinking water must be adequately addressed to ensure that the hundreds of thousands of residents who rely on these drinking water sources are no longer exposed to PFAS contamination; if the Addendum is not the appropriate document to address this issue, then the Corrective Action Plan and other enforcement tools must.

If edited accordingly, the Addendum together with the Corrective Action Plan (and potentially other enforcement actions) will help address the major sources of PFAS pollution from the Fayetteville Works Facility. However, DEQ could take a more protective course of action by requiring the installation of the Alternate Interim Seep Remediation System, as this system is technologically and economically feasible. This would be consistent with the Consent Decree and would ensure efficient and effective PFAS removal. Relatedly, a removal efficiency above 99% is technologically and economically feasible and therefore should be required.

Addressing the abovementioned concerns would result in a more effective Addendum, protecting North Carolinians from additional, preventable PFAS exposure. Thank you for this opportunity to comment and please feel free to reach out to me if you have any questions or concerns.

Regards,

Cori Bell
Program Attorney, Water
Natural Resources Defense Council

From:	Martin, Sharon L.
To:	comments.chemours
Subject:	FW: [External] Comments on the Addendum to the Consent Order Paragraph 12 from Counsel in Carey v. E.I. du Pont de Nemours & Co., No. 7:17-cv-189 (E.D.N.C.)
Date:	Thursday, September 17, 2020 6:53:27 PM
Attachments:	Letter to Sheila Holman re Comments on Consent Order Addendum.pdf Comments on Addendum to Consent Order Paragraph 12 from Counsel in Carey v E.I. du Pont.pdf

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From: Holman, Sheila
Sent: Thursday, September 17, 2020 6:12 PM
To: Martin, Sharon L. <sharon.martin@ncdenr.gov>
Subject: Fw: [External] Comments on the Addendum to the Consent Order Paragraph 12 from Counsel in Carey v. E.I. du Pont de Nemours & Co., No. 7:17-cv-189 (E.D.N.C.)

Sheila Holman
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From: Jamie Bowers <jbowers@cohenmilstein.com>
Sent: Thursday, September 17, 2020 5:10 PM
To: Holman, Sheila <sheila.holman@ncdenr.gov>
Cc: Theodore Leopold <tleopold@cohenmilstein.com>; S. Douglas Bunch <dbunch@cohenmilstein.com>; Steve Seigel <sseigel@susmangodfrey.com>; Alison Deich <ADeich@cohenmilstein.com>; Steve Morrissey <smorrissey@susmangodfrey.com>; Neal Weinfeld <NHW@dedendumgroup.com>; Abraczinskas, Michael <michael.abraczinskas@ncdenr.gov>; Scott, Michael <michael.scott@ncdenr.gov>; Culpepper, Linda <Linda.Culpepper@ncdenr.gov>; Lane, Bill F <Bill.Lane@ncdenr.gov>; Benzoni, Francisco <fbenzoni@ncdoj.gov>; kemp@cfrw.us <kemp@cfrw.us>; ggisler@selcnc.org <ggisler@selcnc.org>
Subject: [External] Comments on the Addendum to the Consent Order Paragraph 12 from Counsel in Carey v. E.I. du Pont de Nemours & Co., No. 7:17-cv-189 (E.D.N.C.)

Good afternoon,

Please find attached comments on the Addendum to the Consent Order Paragraph 12 from counsel in *Carey v. E.I. du Pont de Nemours and Co.*, No. 7:17-cv-189-D (E.D.N.C.), and the attached letter.

Best regards,

Jamie Bowers

Jamie Bowers
Associate
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From:	Jamie Bowers
To:	comments.chemours
Cc:	Theodore Leopold; S. Douglas Bunch; Steve Seigel; Alison Deich; Steve Morrissey; Neal Weinfeld
Subject:	[External] Comments on the Addendum to the Consent Order Paragraph 12 from Counsel in Carey v. E.I. du Pont de Nemours & Co., No. 7:17-cv-189 (E.D.N.C.)
Date:	Thursday, September 17, 2020 5:07:44 PM
Attachments:	Letter to Sheila Holman re Comments on Consent Order Addendum.pdf Comments on Addendum to Consent Order Paragraph 12 from Counsel in Carey v E.I. du Pont.pdf

Good afternoon,

Please find attached comments on the Addendum to the Consent Order Paragraph 12 from counsel in *Carey v. E.I. du Pont de Nemours and Co.*, No. 7:17-cv-189-D (E.D.N.C.), and the attached letter.

Best regards,

Jamie Bowers

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September 17, 2020

VIA ELECTRONIC MAIL

The Honorable Sheila Holman
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Re: Chemours Public Comments – Comments from Class Counsel in *Carey v. E.I. du Pont de Nemours & Co.*, No. 7:17-CV-00189-D (E.D.N.C.)

Dear Assistant Secretary Holman:

We are the Court-appointed interim co-lead counsel for the putative Class in *Carey v. E.I. du Pont de Nemours & Co.*, No. 7:17-CV-00189-D, currently pending in the U.S. District Court for the Eastern District of North Carolina ("*Carey* Counsel"). The *Carey* action plaintiffs ("Plaintiffs") seek to hold Chemours and its predecessor, DuPont, liable for polluting North Carolina residents' bodies and property with GenX and other Per- and Polyfluoroalkyl Substance ("PFAS") compounds originating from Chemours' Fayetteville Works plant, thereby endangering these residents' health. Plaintiffs seek to represent several putative classes of individuals—including property owners who receive their water from wells as well as property owners who receive their water from public utilities—whose health and property have been injured by Chemours' and DuPont's wrongful contamination of the Cape Fear River area with PFAS.

On behalf of those putative Classes, Plaintiffs respectfully submit the attached comments in response to the ADDENDUM TO CONSENT ORDER PARAGRAPH 12 (the "Addendum") in *State of North Carolina v. The Chemours Company FC, LLC*, Case No. 17-CVS 580 (Bladen County Superior Court).

Briefly summarized, *Carey* Counsel provide the following comments:

First, Chemours should be required to provide downstream water consumers with under-sink reverse osmosis systems pending Chemours' satisfactorily demonstrating achievement of the action levels set forth in Paragraph 20 of the Consent Order.

Second, the Addendum's Seep Remediation Systems will not prevent migration of PFAS into the Cape Fear River from the seeps. The Interim Seep Remediation System will not prevent contaminated groundwater from discharging to the Cape Fear River. The Long-Term Seep Remediation System will not prevent contaminated groundwater from being discharged to the Cape Fear River. And the Barrier Wall and Groundwater Extraction System will not mitigate the discharge of contaminated groundwater into the Cape Fear River.

Third, the Addendum should require Chemours to remediate sediment contamination.

Fourth, the PFAS mass loading measurements set forth in Section 1 of the Addendum will vastly underestimate PFAS loadings to the Cape Fear River.

Fifth, the Addendum should provide an opportunity for notice and comment on Chemours' submittals pursuant to the Addendum's requirements.

In sum, because the Addendum's proposals will not fully remediate the PFAS contamination Chemours has caused in the immediate or long term, Chemours must take responsibility to ensure clean, uncontaminated water reaches property owners and individuals who rely on the Cape Fear River for their water supply.

For the reasons set forth below, 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 where tap water was found to exceed the 10/70 Action Levels (described below), and bottled water pending the installation of such systems, and incorporate *Carey* Counsel's suggestions to ensure the Addendum more adequately addresses the remedial measures that will be necessary to address Chemours' and DuPont's decades-long history of contaminating the environment and water with PFAS.

Respectfully submitted,

/s/ Theodore J. Leopold
Theodore J. Leopold

/s/ Stephen E. Morrissey
Stephen E. Morrissey

Cc:

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COMMENTS ON ADDENDUM TO CONSENT ORDER PARAGRAPH 12
State of North Carolina v. The Chemours Company FC, LLC
Case No. 17-CVS 580 (Bladen County Superior Court)

The following comments submitted by counsel ("*Carey* Counsel") for the plaintiffs in the putative class action *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), address the proposed Addendum to Paragraph 12 of the Consent Order ("Addendum").

First, *Carey* Counsel appreciates the adoption by the North Carolina Department of Environmental Quality ("DEQ") of certain aspects of *Carey* Counsel's comments on Section IV.B of the Corrective Action Plan ("CAP") in the Addendum. This includes, in particular, DEQ's efforts to compel Chemours to implement its obligations under the CAP in an expeditious manner.

Carey Counsel nevertheless provides these comments as a result of concerns about certain provisions of the Addendum which permit Chemours to avoid its obligations to remediate Fayetteville Works in the manner prescribed in the Addendum.

In addition, as noted in our initial comments on the CAP, *Carey* Counsel believes it is critically important for the health and welfare of residents in the Cape Fear River area for toxicological and epidemiological studies to be conducted as soon as possible. Contrary to any reasonable standard for handling dangerous chemicals, DuPont and Chemours discharged massive amounts of PFAS into residents' drinking water sources for decades without first establishing that it was safe to do so by conducting toxicological assessments of each chemical and the synergistic effects among the various chemicals. The findings of the C8 Panel that DuPont agreed to appoint as part of the resolution of the Washington Works matter highlight the serious public health risks posed by ongoing exposure to PFAS, and the need for and benefits of long-term health studies of chemicals like these. As requested in *Carey* Counsel's initial comments, the State should require

Chemours and DuPont to fund studies of the toxicological effects of these chemicals and epidemiological studies of their impacts in affected communities.

I. Comment 1: Pending the achievement of the action levels set forth in Paragraph 20 of the Consent Order, Chemours should be required to provide downstream water consumers with under-sink reverse osmosis systems.

It will take years to fully implement the various mitigation measures set forth in the Addendum—if, as explained below, they will be effective at all—because of contamination ongoing while those mitigation measures are being implemented, and likely continuing long afterwards due to residual PFAS contamination in the river, soil, and groundwater resulting from decades of pollution emanating from Fayetteville Works. Chemours' obligations set forth in the draft Addendum will not adequately protect individuals consuming water drawn from the Cape Fear River downstream of Fayetteville Works ("Downstream River Consumers" or "DRCs"). The Addendum's remedial measures do not mandate that Chemours remediate or prevent migration of per- and polyfluoroalkyl substances (PFAS) into the Cape Fear River from: (1) the "daylighting" of groundwater from the Perched Zone and Surficial Aquifer; (2) the Black Creek Aquifer north and south of the Barrier Wall depicted in Exhibit 5 to the Addendum; (3) the ten offsite groundwater seeps, namely the Lock and Dam N. 2 Seep and Seeps E to M; (4) the eastern bank of the river; (5) overland or groundwater flowage from the hundreds of square miles impacted by Chemours' air emissions; or (6) river sediment.

Because the Addendum's requirements do not adequately protect DRCs from drinking contaminated water, and as explained in additional detail in *Carey* Counsel's comments in the CAP, DEQ should order Chemours to provide DRCs with under-sink Reverse Osmosis Systems ("RO Systems") and bottled water pursuant to Paragraph 20 of the Consent Order. The *Carey* plaintiffs are seeking the installation of reverse-osmosis filters as part of the relief sought in the

class action litigation, but requiring that remedy as part of the Consent Order would provide that relief to area residents much more quickly and could facilitate an earlier resolution of the litigation.

II. Comment 2: The Addendum's Seep Remediation Systems will not prevent migration of PFAS into the Cape Fear River from the seeps.

Pursuant to Section 2.a. of the Addendum, Chemours was required to install an Interim Seep Remediation System by August 31, 2020. This Interim Seep Remediation System is essentially a "flow-through cell system" whereby contamination passes through a porous barrier composed of carbon that is designed to reduce the concentration of only three PFAS by 80%.[1] The flow-through cell system is located only next to the four seeps along the river bank adjacent to Fayetteville Works (labeled as Seeps A, B, C, and D).[2] Each seep measures approximately 100 feet long.

The "extraction wells" are in actuality just narrow-diameter monitoring wells with short-length screens that will likely extract very small quantities of water.[3] Moreover, the seven extraction wells are spaced approximately 1000 feet apart and spread in a line over a distance of approximately 6000 linear feet, and there is no evidence to suggest that they will be able to capture groundwater located more than a few dozen feet—at most—from the wells.

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[1] If the Interim Seep Remediation System fails to a achieve an 80% removal efficiency, then Chemours is required to install an Alternative Interim Seep Remediation System designed to capture and treat 99% of the PFAS in the groundwater.

[2] The seeps were identified by a visual observation, and Chemours did not sample other locations along the bank to determine whether contamination, while not visible, was still migrating from the plant into the river.

[3] Pursuant to Section 3.a., groundwater is to be actively pumped from seven small-diameter wells, with small screens, currently used for passive groundwater monitoring. According to the Legend on Attachment 4: "Extraction Wells and Conceptual Piping Route", the wells are to be screened in the Black Creek Aquifer only.

Pursuant to the Addendum's terms, a determination of the effectiveness of the seep remediation system (in terms of its ability to remove 15 PFAS) is to be measured by the concentration of only three PFAS (GenX, PFMOAA, and PMPA) rather than the full list of the Consent Order's 12 Attachment C chemicals, or the Addendum's 20 Attachment 3: Table 3+ SOP Compounds.

A. Comment 2(a): The Interim Seep Remediation Systems will not prevent contaminated groundwater from discharging to the Cape Fear River.

The Addendum's Interim Seep Remediation Systems are inadequate to prevent contaminated groundwater from discharging to the Cape Fear River. These flaws are particularly significant because if DEQ, Cape Fear River Watch, or this Court concur with Chemours' claim of impracticability as to the Long-Term Seep Remediation Plan, Chemours may resort to the Interim Seep Remediation Plan as its only purported remedial solution. However, the seep remedy is riddled with flaws, such that it will effectively provide no guarantee of remediating the extensive PFAS contamination emanating from Fayetteville Works. These flaws are detailed below.

First, there is no description of the chemical and physical composition of the: (a) "flow-through cell system", (b) porous barrier, or (c) filtration or reactive medium. As discussed in other comments by *Carey* Counsel, PFAS is extremely difficult to filter out of water, with reverse osmosis providing the most effective mechanism of doing so. As indicated in numerous studies, and *Carey* Counsel's comment on the Proposed Consent Order, dated January 10, 2019, granular activated carbon and other environmental media are extremely inefficient in capturing PFAS. In July 2018, scientists from North Carolina State University, the University of North Carolina at Charlotte, and East Carolina University published a peer-reviewed study on the effectiveness of

various treatment technologies for removing GenX and other short-chained PFAS ("NC Treatment Study").[4] The NC Treatment Study found that:

- "Both the full-scale and pilot-scale results illustrate that **GAC is only somewhat effective for controlling GenX** in the context of treating coagulated Cape Fear River water. Recognizing that the adsorbability of PFASs decreases with decreasing perfluorinated carbon chain length. . . **GAC will be only marginally effective for shorter-chain PFEAs such as PFMOAA, PFMOPrA, and PFEOPrA as well as the diether PFO2HxA**. For Nafion byproduct 2, effective GAC performance can be expected for at least 5,000 BV, but data collected at the top sampling port of the full-scale adsorber as well as data from the pilot study show **substantial breakthrough of the Nafion byproduct 2** in the range 10,000-15,000 BV (>82% breakthrough at full scale, >74% breakthrough at pilot scale)."

- "**An additional concern with GAC adsorption processes is the potential for desorption** when PFEA concentrations change in the source water and/or more strongly adsorbing compounds displace weakly adsorbed PFEAs as the mass transfer zones of the more strongly adsorbing compounds migrate through the GAC bed. In this study, the first scenario applied; PFEA concentrations in the source water decreased dramatically as a result of source reduction efforts. At the beginning of the evaluation period (June 19, 2017; ~3,500 BV of water treated), PFMOAA removal was approximately 70%. As PFMOAA concentrations in the GAC influent decreased from approximately 26,000 to approximately 680 ng/L, **PFMOAA removal quickly ceased, and during the last three weeks of the monitoring period, PFMOAA levels in the GAC effluent were approximately 10 times those measured in the GAC influent.**"

- "On average, GenX concentrations in the plant effluent were 28% higher than those in the plant influent. Given that the adsorber containing the youngest GAC was able to remove GenX during the study period. . . **the results suggest that the two adsorbers containing older GAC were desorbing GenX**."

Second, the Addendum provides no description of the dimensions or placement of the flow-through cell system. Although the Addendum states that the flow-through cell system is to be placed at the seeps, there is no description as to how far it must extend above or below the visually identified seep. Without specifying the dimensions, it is likely that the actual installation of the

[4] Hopkins, Z. R., Sun, M., Dewitt, J.C., & Knappe, D.R.U, *Recently detected drinking water contaminants: GenX and other per- and polyfluoroalkyl ether acids*, Journal of the American Water Works Association (June 14, 2018), 110(7), 13-28, https://dx.doi.org/10.1002/awwa.1073.

system will permit PFAS to slip below the bottom or around the sides of the cell into the Cape Fear River. There is also no description of the width of the cell; the thinner the cell, the lower the likelihood that it will capture and filter out PFAS.

Third, the identified seeps likely underrepresent the actual PFAS contamination on site. The lateral extent of PFAS contamination flowing into the Cape fear River is unknown. The location of the seeps was identified by visual observations only, and it is very likely that PFAS contamination is migrating into the Cape Fear River along a much longer stretch of the river. The selected seeps, A, B, C, and D, are also not the only visually identified seeps of PFAS migrating from Chemours.[5] There are nine additional seeps that must be remediated. In particular, the extraction system should include a system designed to extract groundwater from all seeps. Chemours incorrectly states that there is a decreasing trend in PFAS concentrations while moving southward toward Georgia Branch Creek. 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 the next six downstream seeps over the next 0.6 miles exhibit similar PFAS concentrations

[5] 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).

CAP at 22.

(average 572 ng/L GenX). As a result, the extraction system should include a system designed to extract groundwater from all seeps:

Fourth, there is no description of the radius (or cone) of influence of the seven monitoring wells to be converted to groundwater extraction wells covering the 6,000 linear feet of the Cape Fear River. The wells have a narrow diameter, and small screens in the aquifer.

Fifth, there is no information about the efficiency of pumping, and Chemours should be directed to conduct a pumping test immediately to test the system's efficiency.[6] If Chemours fails

[6] A *pumping test* is a field experiment in which a well is pumped at a controlled rate and water-level response (drawdown) is measured in one or more surrounding observation wells and optionally in the pumped well (control well) itself; response data from pumping tests are used to estimate the hydraulic properties of aquifers, evaluate well performance, and identify aquifer boundaries. The goal of a pumping test is to estimate hydraulic properties of an aquifer system

to conduct such a test, the wells may have minimal, if any, impact on removing contaminated groundwater from the aquifer.

Sixth, as proposed, the wells are likely to be ineffective in capturing PFAS contamination. Given that the water is being extracted from a monitoring well with small screens, it is possible if not likely that the groundwater being extracted from the well is only approximately ten feet in each direction around the well—if not less. There is no indication that Chemours will be able to capture groundwater located 500 feet from each side of the well—6,000 feet in total. There is also no indication that the wells will capture groundwater from each of the aquifers at the site. In addition to the Black Creek Aquifer, Fayetteville Works is also overlain by the Surficial Aquifer and the Perched Zone. There is no indication that the seven monitoring wells in each of these aquifers are screened. These wells will very likely not be effective. The "extraction" wells are only two inches in diameter with short, 10-foot screens. Four of the wells are "up on the hill" and have an average depth of 120 feet. There is also no description of the depth at which the wells in the Black Creek Aquifer must be screened. It is possible if not likely that the PFAS will migrate not only around the extraction wells, but also beneath the wells into the Cape Fear River.

Seventh, the effectiveness of the extraction wells should be measured against *all* PFAS identified on site, not merely three "representative" PFAS. In the Addendum, the "extraction" wells' effectiveness is to be determined by measuring its efficacy for three parameters only—GenX, PFMOAA, and PMPA—rather than the full list of PFAS identified in Consent Order Attachment C (12 chemicals), or Addendum Attachment 3: Table 3+ (20 compounds). Each of

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based upon information developed from on-site measurements such as permeability, grain size, groundwater flow velocity and gradient geometry, incongruities in the soil, and many other factors. For the pumped aquifer, one seeks to determine transmissivity, hydraulic conductivity (horizontal and vertical), and storativity (storage coefficient).

the listed PFAS have different toxicity and chemical fate and transport characteristics. Chemours has yet to determine the toxicity for many of the Consent Order Attachment C chemicals, or even the five chemicals listed on Consent Order Attachment B. The fate and transport characteristics of these chemicals have also yet to be determined, even though they have been detected in the DRCs' tap water nearly 100 miles downstream from Fayetteville Works.

Eighth, in order to determine whether the seep remediation system is meeting its stated 80% (or 99%) removal efficiency requirements, a pre-treatment baseline must be established. The baseline must be determined on a temporal basis, *i.e.*, before the system has become operational, and on a geographic basis, *i.e.*, immediately upgradient and downgradient of the cells.

Significantly, the Addendum does not specify when the "baseline" will be established for the Cape Fear River. Section 1 of the Addendum does not establish whether the Cape Fear River "baseline" will be established before seep and groundwater mitigation activities begin. The monthly Cape Fear River sampling to establish the "baseline" for PFAS mass-loading does not begin until seven days after DEQ approves the August 31, 2020 updated mass-loading model for the Cape Fear River and Outfall 002. The Addendum does not specify how many months (for the Cape Fear River) and for how many weeks (for Outfall 002) samples will need to be collected and analyzed to establish the PFAS mass-loading baseline for the Cape Fear River. The Addendum also does not specify whether the baseline will be established before PFAS discharges from the seeps are to be mitigated, which begins on November 16, 2020 (Sec. 2.a), or before the onsite wells begin extracting groundwater on November 30, 2020 (Sec. 3.a).

Additionally, the Addendum does not specify that samples must be collected directly up-gradient and down-gradient from the flow-through cells, and collected at the same time. The Addendum relies upon sampling from the Cape Fear River, but there are numerous factors that can

impact the concentration of PFAS in the Cape Fear River including river-flow volume, whether that river flow is increasing or decreasing, location of the sample, organic content in the river water, and many other factors. In general, the lower the groundwater flowage volume, the higher the concentration of the chemical (in this case PFAS). Conversely, if the flowage of the groundwater is high, the concentration of the chemical will appear low (although mass flow downstream could still be high). In order to ensure that parties are extracting a chemical to meet a particular goal, regulators require that the influent and effluent streams be sampled as closely as possible to the flow-through system. Regulators also require that the concentration of chemicals in the treated sample be compared to the concentration of chemicals in the untreated sample at the same point in time. Further, because the Cape Fear River is used for recreational purposes, the effectiveness of the remedial action should be determined before the PFAS reaches the river, not once it is in the river. Such requirements are absent from the Addendum, and should be incorporated.

B. Comment 2(b): The Long-Term Seep Remediation System will not prevent contaminated groundwater from being discharged to the Cape Fear River.

Pursuant to Sections 2(c) and 3(a) of the Addendum, regardless of whether Chemours satisfies its Interim Seep Remediation System (80% removal efficiency) or Alternate Interim Seep Remediation System (99% removal efficiency) objectives, by March 15, 2025, Chemours must establish that GenX, PFMOAA, and PMPA are reduced between 95% and 99% (depending upon when and where the sample is collected) to satisfy the Long-Term Seep Remediation Plan. However, the sampling and efficiency requirements for the Long-Term Seep Remediation Plan suffer from flaws similar to those in the Interim Seep Remediation System:

1. There is no requirement to determine the lateral extent of PFAS contamination outside of the four seeps.
2. There is no requirement that the horizontal and vertical extent of PFAS contamination around the seven monitoring/extraction wells capture all of the contaminated groundwater at Fayetteville Works.
3. It is unclear where the wells will be placed, and therefore there is no requirement that the wells capture PFAS in the three aquifers underlying the site: the Perched Zone, the Surficial Aquifer, and the Black Creek Aquifer.
4. There is no requirement to determine the effectiveness of the seep remediation system for any PFAS other than GenX, PFMOAA, and PMPA.
5. There is no requirement that Chemours demonstrate compliance with the 95% to 99% removal efficiency by collecting the remediated and unremediated samples from the groundwater upgradient of the seeps rather than in the river, and at the same time.

   There is no requirement that the monitoring/extraction wells capture contamination being discharged from the Lock and Dam Seep and Seeps E through M.

There are also at least three additional flaws with the Long-Term Seep Remediation Plan beyond those flaws it shares with the Interim Seep Remediation Plan.

First, Chemours is not required to follow any specific method for calculating compliance with removal efficiency. Instead Chemours is allowed to simply "propose a methodology" at some later date. There is no reason that this methodology should not be set forth in the Addendum now given that such methodologies have been used thousands of times at thousands of sites for more than 20 years.

Second, after submitting the "initial determination," Chemours need only demonstrate the 95% to 99% removal effectiveness for five years[7]even if at the end of that five-year period, the seeps continue to discharge groundwater contaminated with PFAS into the river. Rather than setting an arbitrary termination date, treatment of the seeps should continue until the concentrations of PFAS in the untreated seeps meet the 95% to 99% removal efficiencies. And again, as noted above, sampling should occur and be measured in groundwater, not river water.

Third, the Addendum does not explain why, after six months of operation, the influent and effluent sampling of the flow-through cells cannot be changed to measure the PFAS listed in Table 3+ of Attachment 3. Granted, Table 3+ includes the three indicator PFAS; however, that table does not include all of the PFAS listed in Consent Order Attachment C (*e.g.,* perfluoroheptanoic acid (PFHpA), CASRN 375-85-9, is not included in Table 3+).

C. Comment 2(c): The Barrier Wall and Groundwater Extraction System will not mitigate the discharge of contaminated groundwater into the Cape Fear River.

Section 3(b) of the Addendum requires Chemours to install a Barrier Wall at Fayetteville Works to capture not only groundwater migrating from Seeps A-D, but nearly all groundwater from the plant. However, the Addendum provides none of the detail necessary to establish a benchmark to measure and guarantee compliance with the stated goal of preventing groundwater from migrating into the Cape Fear River. Furthermore, should Chemours establish that the prevention of groundwater migration into the Cape Fear River is "impracticable"—a term that is undefined in the Addendum—Chemours is allowed to fall back to merely the seep remediation systems. As explained above, however, such remediation systems are likely to be ineffective at remediating even the seeps. The end result is that under the terms of the Addendum, Chemours

[7] *See* ¶ 2(c)(i).

may be able to avoid the requirement that it prevent any PFAS contamination from flowing from Fayetteville Works into the Cape Fear River and contaminating DRCs' downstream tap water. The flaws in the Barrier Wall plan are detailed below.

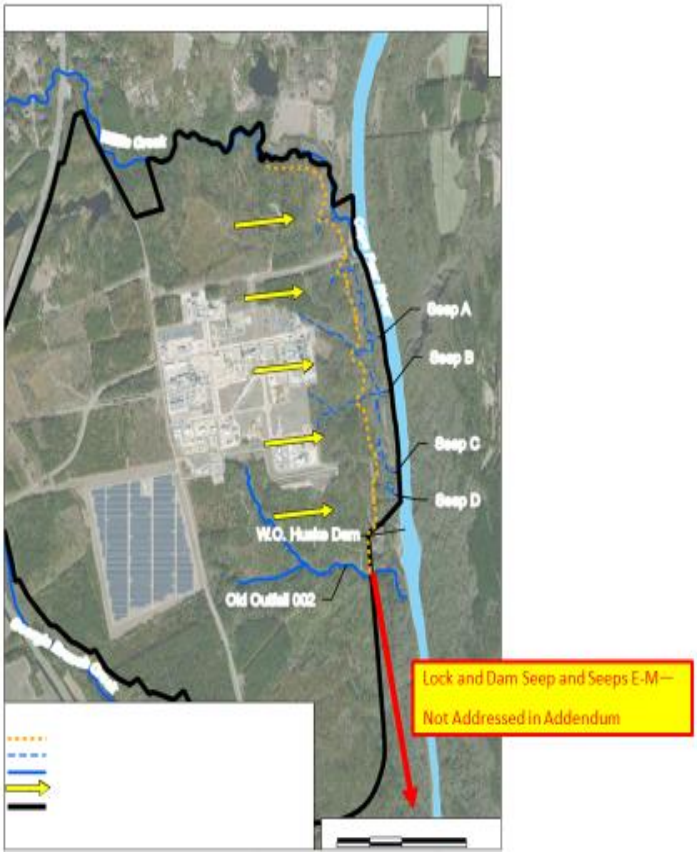
First, the Addendum provides no detail on the construction of the Barrier Wall, in particular no detail concerning its depth. This is problematic, because in order to be effective, the Barrier Wall must extend nearly 100 feet below grade where it will be anchored into the Upper Cape Fear Confining Unit.[8] Further, according to Addendum Attachment F, it appears that the Barrier Wall will be approximately 750 feet west (upstream) of the river. However, some of the highest groundwater contamination levels remain to the east (downgradient) of the Barrier Wall.[9] Because PFAS contamination has been detected in the Black Creek Aquifer, which discharges directly into the Cape Fear River, the Addendum should require that the Barrier Wall be anchored into the Upper Cape Fear Confining Unit.

Second, pursuant to Addendum Section 3(b)(iv), Chemours can simply propose an alternative design for the Barrier Wall if it believes that the design set forth in the Addendum is "impracticable"—an undefined and ambiguous standard. Because there is no benchmark for establishing practicability, Chemours has broad latitude in designing an alternate barrier or other containment system, as long as the alternative design removes PFAS loading to the "maximum extent ***possible***." Like the word "impracticable," the phrase "maximum extent possible" is both vague and also undefined in the Addendum. Although the parties to the Addendum can contest the practicability of the alternate remedy in court, such a proceeding could take months if not years,

[8] Geosyntec Consultants, *On and Offsite Assessment for Fayetteville Works*, Version 1 (Sept. 30, 2019), at Figures 10-2, 10-3, 10-4, 10-5, 10-6.
[9] Geosyntec Consultants, *On and Offsite Assessment for Fayetteville Works*, Version 2 (Oct. 31, 2019), at Figure 6-3 and Appendix at Table I-1, *available at* https://files.nc.gov/ncdeq/GenX/consentorder/paragraph18/2019.11.01---18-NCDEQ---CFRW-20191031.pdf.

and because the seep remediation systems will likely be ineffective, the DRCs will continue to be exposed to harmful levels of PFAS contamination for years to come.

Finally, like the wells for the Interim Seep Remediation Plan, neither the Barrier Wall nor Extraction Wells extend far enough south to the heavily contaminated Lock and Dam Seep and Seeps E-M, and therefore extensive contamination will continue migrating into the Cape Fear River directly from the groundwater originating beneath Fayetteville Works.



Without these measures, the DRCs will continue to be exposed to harmful levels of PFAS for years to come.

III. Comment 3: The Addendum should require Chemours to remediate sediment.

The Addendum also fails to address sediments in the Cape Fear River that are laden with PFAS from Chemours' operations. Until the sediments are remediated, the sediments will serve as a source of PFAS in the DRCs' drinking water.

GenX is a ubiquitous component of the Cape Fear River sediments.[10] These contaminated sediments act as a repository of GenX that may be released into the overlying water column. "The deepest zone at the Site established to have detectable concentration of PFAS is the Black Creek Aquifer" which is "in direct connection with the Cape Fear River."[11] Because PFAS is miscible in water, and because the Black Creek Aquifer discharges to the Cape Fear River from a variety of seeps and groundwater, it is highly likely that PFAS has contaminated the sediment in the river.

There are numerous studies indicating that PFAS contaminate sediment.[12] "When PFAS attaches to sediments, sediments can act as a continuing source of PFAS to water, fish and biota. That is because the river is a dynamic system where sediment is frequently re-suspended, both naturally and by human-caused activities such as dredging. Aquatic creatures and fish in the water

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[10] *Report to the Environmental Review Commission from the University of North Carolina at Wilmington Regarding the Implementation of Section 20(a)(2) of House Bill 56 (S.L. 2017-209)*, *available at* https://www.ncleg.gov/documentsites/committees/ERC/ERC%20Reports%20Received/2018/CFPUA%20and%20UNC-W/2018-April%20HB%2056%20UNCW%20Rpt.pdf.

[11] Geosyntec Consultants, *On and Offsite Assessment for Fayetteville Works*, Version 1 (Sept. 30, 2019), at 53.

[12] *Investigation of Levels of Perfluorinated Compounds in New Jersey Fish, Surface Water, and Sediment*, New Jersey Department of Environmental Protection, Division of Science, Research, and Environmental Health (June 18, 2018, updated April 9, 2019), *available at* https://www.nj.gov/dep/dsr/publications/Investigation%20of%20Levels%20of%20Perfluorinated%20Compounds%20in%20New%20Jersey%20Fish,%20Surface%20Water,%20and%20Sediment.pdf; *PFAS Found in Sediment and Surface Water at Milwaukee Estuary Area of Concern*, Wisconsin Department of Natural Resources (May 7, 2020), *available at* https://dnr.wi.gov/news/releases/article/?id=5122.

interact with sediments, which both exposes the biota and fish to PFAS and "stirs up" the sediment, potentially releasing sediment-bound PFAS in the water."[13]

Specifically, with respect to the Cape Fear River, the University of North Carolina at Wilmington found "that GenX is a ubiquitous component of the sediments sampled to date," noting that, based on the samples it had collected:[14]

- "GenX is present" in sediment;
- "Point and non-point sources are likely contributors" to GenX contamination;
- "Sediments appear to be acting as a repository of GenX that may be released into the overlying water column"; and
- ***"GenX is a ubiquitous component of the sediments sampled to date."***[15]

Because Chemours has contaminated river sediment with PFAS, in addition to addressing the seeps and groundwater entering the Cape Fear River, Chemours should also be required to remediate contaminated river sediment.

IV. Comment 4: The PFAS mass loading measurements set forth in Section 1 of the Addendum will vastly underestimate loadings to the Cape Fear River.

The Addendum should also require that measurements of PFAS mass loading to the Cape Fear River occur more often and for a longer period of time.

First, the mass loadings analysis should evaluate not only Chemours PFAS loadings detected in the Cape Fear River now, but also PFAS loadings that have occurred in the past and

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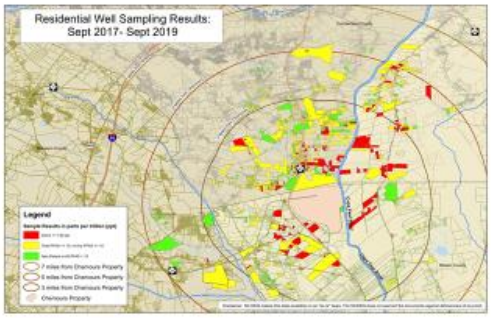
[13] Minnesota 3M PFC Settlement, "Frequently Asked Questions," *available at* https://3msettlement.state.mn.us/frequently-asked-questions; *see also* Harry Behzadi, Ph.D., SGS, *The next frontier on PFAS contamination in sediment, surface water and fish tissue*.

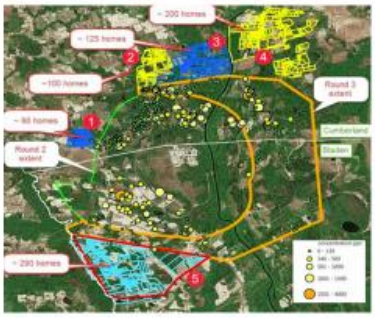
[14] *Report to the Environmental Review Commission from the University of North Carolina at Wilmington Regarding the Implementation of Section 20(a)(2) of House Bill 56 (S.L. 2017-209), available at* https://www.ncleg.gov/documentsites/committees/ERC/ERC%20Reports%20Received/2018/CFPUA%20and%.

[15] *Id*. at 3.

will occur in the future. Chemours' PFAS have been emitted into the air and discharged into the water for more than 40 years across locations covering hundreds of square miles. The full extent of the contamination has not been even remotely fully determined. PFAS has been detected over an area covering hundreds of square miles, including in groundwater near a school located ten miles east of Fayetteville Works, in rainwater puddles at UNC Wilmington, in surface water and sediment more than 100 miles downstream of the plant, and even in the Atlantic Ocean. These locations all fall within the Cape Fear River watershed, and there is no study to suggest that these contaminants will not make their way to the Cape Fear River through groundwater migration and overland flow for decades to come.

Groundwater contamination has been discovered more than 9 miles northeast of the Chemours plant, and the aquifer flows toward the River.





Second, measurements of PFAS mass loading to the Cape Fear River should occur far more often than mandated in the Addendum. Pursuant to Section 1 of the Addendum, Chemours sampling at the Tar Heel Ferry Road Bridge is only required when there is a 1½-inch rain event. On the one hand, a large volume of clean water will dilute the concentration of PFAS in the sample. On the other hand, PFAS concentrations attached to particulates will typically increase with rising flow as they are scoured from the riverbanks and bottom. Often the largest mass loading occurs during relatively infrequent, short-duration flow events. Accordingly, the sampling frequency and river flow measurements (before and during the sampling) must be frequent enough to capture the entire range of mass loading events.

The Addendum provides that with respect to water collected from Kings Bluff (Cape Fear River Lock & Dam #1) and Bladen Bluffs, only grab samples need be collected. In order to avoid dilution of the samples, multiple discrete samples should be collected. This should include at least one sample collected from the top one foot of the sediment.

The Addendum also provides that Chemours can terminate sampling after five years, and that even during the five-year period, "Chemours may apply to DEQ for modifications of protocol, including with respect to sampling frequency." Samples should be collected for 30 years, if not more, because different PFAS will reach the river, and thus the DRCs, at different times. PFAS are "forever chemicals," are very stable, and will not degrade for decades. As mentioned in *Carey* Counsel's comments on the CAP:

> 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.
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> 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.[16] 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. . .
>
> 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.

[16] Geosyntec Consultants, *Site Associated PFAS Fate and Transport Study for Chemours Fayetteville Works* (June 24, 2019), *available at* https://files.nc.gov/ncdeq/GenX/consentorder/paragraph27/P27-PFAS-FT-Report.pdf (prepared pursuant to Consent Order Paragraph 27).

PFAS sampling should thus occur monthly, at all locations specified in the Addendum, and continue until the concentration of all PFAS listed in Consent Order Attachment C and the Addendum's 20 Attachment 3: Table 3+ SOP Compounds declines by 99%.

V. Comment 5: There should be opportunity for notice and comment on Chemours' submittals under the Addendum.

The Addendum requires the submittal of more than 10 design, maintenance, monitoring, and sampling plans; hundreds of samplings data sets; and numerous reports and other submittals. As shown by the *Carey* Counsel's comments on the Proposed Consent Order, the 93 pages of comments on Chemours' CAP, and these comments on the draft Addendum, Chemours has made numerous errors in its interpretation of the data and its selection of remedial alternatives. All of this information is relevant to determining the impact of Chemours' contamination on the DRCs' health and safety. The *Carey* Counsel therefore respectfully request that the Addendum provide that *Carey* Counsel: (1) be notified of, and promptly provided with, all of Chemours' submissions of plans, data, and reports related to releases of PFAS from Fayetteville Works; and (2) be provided with 30 days to comment on these submittals.

From: boergirl@aol.com
To: comments.chemours
Subject: [External] Comments on Chemours Addenum to Consent order
Date: Thursday, September 17, 2020 3:43:03 PM

Your website was unresponsive for comment on the Chemours agreement.
I lived in Cumberland/Bladen County at the time questions originally arose about the plant discharges from the Dupont Plant. These concerns were raised more than 20 years ago.

I now live in New Hanover County that is still dealing with the GenX-Chemours pollution problems in the Cape Fear River.
The proposed testing and monitoring in the addendum is still insufficient to protect the public and needs further discussion.

There are no water utilities listed in the contract including PWC in Fayetteville or the CFPU in Wilmington. This is a glaring omission.

As with other chemical pollutants discharged into the Cape Fear River public water supplies, this has gone on far to long.

Sharon Valentine
3755 Old Sand Mine Dr.
Wilmington, NC 228412

From: homebull@aol.com
To: comments.chemours; homebull@aol.com
Subject: [External] My comments
Date: Thursday, September 17, 2020 3:11:42 PM

NCDENR (CHEMOURS CONTINUAL ENVIRONMENTAL DEGRADATION):

I am submitting these comments on September 17 at 2:40 PM.

My name is Leonard Bull and I am a retired faculty member and administrator from North Carolina State University. I retired in 2009 and live in Wilmington. I am a member of the Cape Fear River Assembly Board and Executive Committee, but I am writing representing myself. At NCState, the last 10 years of my tenure were as Associate Director of the Animal and Poultry Waste Management Center which you are very familiar with, I know. In that capacity I had MANY contacts with NCDENR. And I have to say that the behavior of NCDENR in that activity was similar to the way that it is continuing to abdicate its responsibility to protect the and REPRESENT the taxpayers who fund the agency! Many NCDENR employees with whom I worked and interacted would share the comments that I make here!

My reason for writing is to express my continued dismay at the snail's pace that you (NCDENR) have taken and are again proposing to take to REQUIRE Chemours to CEASE AND DESIST its emissions (air, water and soil) of GenX (and possibly other PFAS) as well as its former form into the environment. There is absolutely NO reason why there should have ever been ANY delay in prompt action, YEARS AGO, based on the similar tragic events of West Virginia that you are all well aware of. And yet, deadlines and excuses have continued to dribble from your agency and be ignored, all the while allowing Chemours to emit and a dangerous and potentially lethal chemicals into the environment.

The most recent "abuse" of your mandate was the secret way that the discussions took place, apparently without ANY involvement of either PWC or CFPUA, as well as other water processing users in the basin, while expecting those agencies (and their taxpaying customers) to clean up the water that they did not pollute (the same for municipalities regarding air and soil contamination).

It is an **insult** that you are proposing a snails pace to move forward in the proposed procedures. There is NO justifiable reason why:a. that timeline cannot be shortened SIGNIFICANTLY; b. the paltry penalties for missing deadlines cannot be INCREASED TO A SUM THAT WILL ENCOURAGE ACTION.

I urge you to do the job that you are charged with doing and take drastic action to SUBSTANTIALLY reduce the time allowed for Chemours to clean up their operating practices and impose penalties of such a financial amount that they will take them seriously and act. There are people who are contracting serious illnesses as a result of your lax and insufficient action!

Thank you for your consideration and I believe required action!!

Sincerely,

LEONARD S BULL, PhD, PAS
Emeritus Professor/Head, NCState Department of Animal Science
Emeritus Associate Director, NCState University Animal and Poultry Waste Management Center
3755 Old Sand Mine Drive
Wilmington, NC 28412
homebull@aol.com

From:	Vaughn Hagerty
To:	comments.chemours
Cc:	Jim Flechtner
Subject:	[External] Comments on Proposed Addendum to the Chemours Consent Order
Date:	Thursday, September 17, 2020 2:34:31 PM
Attachments:	CFPUA-Comments-Consent-Order-Addendum-9-17-2020.pdf

The attached comments on the Proposed Addendum to the Chemours Consent Order are submitted on behalf of Jim Flechtner, CFPUA Executive Director.

Vaughn Hagerty
Public Information Officer
Cape Fear Public Utility Authority
o: 910-332-6704 | c: 910-264-8338
235 Government Center Dr., Wilmington, NC 28403
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Cape Fear Public Utility Authority
Stewardship. Sustainability. Service.

James R. Flechtner, PE
Executive Director
235 Government Center Drive
Wilmington, NC 28403
910-332-6669
jim.flechtner@cfpua.org

September 17, 2020

Assistant Secretary's Office
RE: Chemours Public Comments
1601 Mail Service Center
Raleigh, NC 27699-1601

Re: Public Comments on Proposed Addendum to Paragraph 12 of Chemours Consent Order

To NCDEQ:

On August 13, 2020, Cape Fear Public Utility Authority (CFPUA) was told that the State had negotiated changes to the Consent Order governing actions Chemours must take to address the damage done by decades of PFAS releases from Chemours' Fayetteville Works Plant.

Changes being proposed in the Addendum to Paragraph 12 of the Consent Order (the Addendum) relate mainly to measures meant to address the flow of highly contaminated groundwater and stormwater from Chemours' industrial site into the Cape Fear River, the source of drinking water for hundreds of thousands of North Carolinians, including CFPUA's customers. CFPUA's comments on the Addendum are outlined and detailed below.

1. The Addendum proposes relief for downstream water users that is neither as timely nor as certain as that provided to private well owners near Chemours' plant. Most would say the measures offered to well owners are appropriate to protect human health. Can the State now say the measures being proposed in the Consent Order will reduce PFAS in the Cape Fear River to levels that are protective of human health?

We and our community arguably have far more at stake in the outcome of these discussions than any of the three parties negotiating the terms of the Consent Order: More than 200,000 New Hanover County residents depend on CFPUA for drinking water, the majority of which is sourced from the Cape Fear River. We and our community bear the burdens for Chemours' pollution of the river. Yet we have been excluded from the discussions that have shaped the Consent Order and the Addendum. This may explain why – as in the case of other measures that have been proposed to mitigate or remediate Chemours' contamination – remedies in the Addendum that the State and Cape Fear River Watch say will provide CFPUA's customers relief continue to be less immediate and less definitive than those the State is rightly forcing Chemours to take for a few thousand private well owners near the Fayetteville works.

In a nutshell: Chemours must provide clean water – bottled water within three days and a permanent solution within six months – to owners of private wells where PFAS is detected at 10 parts per trillion (ppt) for one PFAS compound or 70 ppt for a combination of PFAS compounds (the 10/70 level). For

many of these well owners, this relief already has occurred. Meanwhile, CFPUA consistently detects PFAS in concentrations exceeding the 10/70 level in raw, untreated water it withdraws from the Cape Fear River. Our relief is coming, we are told, though each time this relief is revealed, it is nowhere near as immediate or as certain as what the State has secured for the private well owners. We have repeatedly expressed these concerns to the State. As expressed in CFPUA's Motion to Intervene and the proposed Complaint, which are attached to and incorporated into these comments, the State has the authority and right to require action by Chemours to immediately abate harm to its residents. The Complaint filed by the State in Bladen County Superior clearly set forth that the State is aware of such harm and has the authority to act. To date, including at a September 1, 2020, meeting with the State, we have received no satisfactory explanation for why hundreds of thousands of North Carolina residents downstream who rely on the Cape Fear River for drinking water are being treated unequally. We have asked the State if the measures being proposed in the Consent order and the Addendum will reduce PFAS concentrations in the Cape Fear River to levels the State believes are protective of human health, absent the measures CFPUA is undertaking at our ratepayers' expense to treat Chemours' PFAS in our community's drinking water. To date, we and our community have received no satisfactory answer. This question is not rhetorical, and if the State cannot provide confident affirmation, we must conclude the actions taken thus far and those being proposed are inadequate and incomplete.

2. Chemours has until late 2021 to prove the interim seep remediation system can remove 80 percent of the PFAS flowing into the Cape Fear River at concentrations in the hundreds of thousands of parts per trillion (ppt). What is known about the system's design raises doubts about the likelihood of reaching this goal. Any alternative measure would come months after the completion of the additional filters at Sweeney Water Treatment Plant under construction specifically to treat Chemours' PFAS – and funded by CFPUA's ratepayers.

To be sure, the most significant projects being proposed in the Addendum sound positive, at least in terms of the reductions in PFAS loading from the Chemours facility into the Cape Fear River.

The first of these projects is a "seep remediation system" Chemours says will remove 80 percent of PFAS from groundwater reaching the river from four seeps. According to sample data[1] provided to the State by Chemours, each day, each of these seeps pollutes the river with anywhere from more than 91,000 gallons to almost 250,000 gallons of water with PFAS concentrations ranging as high as 340,000 ppt. (It should be noted that these PFAS totals take into account only 20 specific compounds. Chemours recently told[2] the state it had found 21 as-yet-unknown PFAS compounds in water that likely migrates to the river; none of these is accounted for in the sampling analysis on the seeps. The remediation system is to be completed by April 5, 2021. Chemours then has four months to show it can achieve the promised 80 percent removal efficiency. The State, however, will grade Chemours' work on a curve, since removal efficiency will be measured for only three compounds: GenX, PMPA, and PFMOAA.

But what happens if the interim seep remediation system is not successful? This is not idle speculation. Based on the limited technical information available on the interim seep remediation system and the proposed volume and depth of granular activated carbon (GAC) in its filter system, we have serious

[1] "2020 Q1 MLM Assessment" https://files.nc.gov/ncdeq/GenX/consentorder/coaddendumsubmittals/2020-Q1-MLM-Assessment.xlsx

[2] "PFAS Non-Targeted Analysis and Methods Interim Report" https://www.chemours.com/en/-/media/files/corporate/fayetteville-works/pfas-nontargeted-analysis-and-methods--interim-report-20200630.pdf

concerns about their ability to achieve the 80 percent removal efficiency given the extremely high concentrations of PFAS in water flowing through the seeps. In a tacit admission of this uncertainty, the Addendum gives the State until March 31, 2022, to determine that any individual seep system has failed to live up to its promise. By that time, the $43 million GAC filters under construction at the Sweeney Plant should be online and effectively treating Chemours PFAS in our customers' drinking water. Chemours has paid none of the $43 million or the millions of CFPUA ratepayer dollars already spent to address Chemours' pollution. Chemours has no plans to pay any of millions of dollars to operate the filters that will be removing its PFAS.

We noted that the Addendum states that the Alternate System, including "ex situ capture and treatment," must be completed within eight months. The PFAS removal goal for the Alternate System is 99 percent compared with 80 percent for the interim remediation seep remediation system. It is unclear what advantages the interim seep remediation system offers downstream water users over the Alternate System, which is supposed to be more effective at PFAS removal and can be installed within a comparable timeframe. Why not require the Alternate System in the first place and stipulate the less-effective interim seep remediation system as Plan B?

3. The project promising the most significant reduction in PFAS loading of the Cape Fear River is a Barrier Wall and Groundwater Extraction System. This project is sketched only in broad outlines, with details to "be determined." Even assuming it is not "technically impracticable in light of geological and other site conditions that are unknown," as the Addendum states, the barrier wall and groundwater extraction system does not have to be completed until March 2023 – more than a year after the GAC filters at Sweeney will be effectively protecting CFPUA's customers.

The second significant project in the Addendum is a "Barrier Wall and Groundwater Extraction System." This "permanent" measure combines a 1½-mile underground wall, meant to keep the Chemours facility's PFAS-laden groundwater from migrating to the river, with a series of wells, from which groundwater is to be extracted and treated before discharge. This system is supposed to reduce the PFAS load in the Cape Fear River coming from Chemours' contaminated groundwater by at least 99 percent – once it is completed in March 2023, more than a year after Sweeney's GAC filters have begun operation. If Chemours determines that building this barrier wall system is "technically impracticable,", it must propose an alternate system by June 30, 2021, that will reduce PFAS loading by the "maximum extent possible" by March 15, 2023.

We have seen very little information about the barrier wall. Perhaps the State has not seen much information about it either, since the Addendum states: "It is understood that the precise contours, locations, and structure of the barrier wall will be determined as part of the design and will be subject to DEQ approval".

Essentially, once again, we and our customers are being asked to wait years for an uncertain outcome. The message from Chemours to our community seems to be: "Trust us." If so, our response is: "When it comes to Chemours, we trust only what we can verify for ourselves."

Moreover, to date the State has not been able, or even tried, to assure CFPUA and its customers that if Chemours achieves the efficiency goals measured in these percentages, PFAS levels in the Cape Fear River will remain low enough for our community to receive water that meets the 10/70 level without the

interim treatment measures ongoing and the permanent GAC filters under construction at the Sweeney Plant, which we must pay for.

4. Mention of potential PFAS contamination related to DuPont raises concerns about the other companies operating at the Fayetteville Works.

The Addendum includes a number of other measures, from completing the decommissioning of a terracotta pipe (a project under discussion for more than a year), implementing an industrial Stormwater Pollution Prevention Plan (somewhat surprising that this would not be standard operating procedure), and completing an investigation of "significant remaining sources of PFAS loading" of the river.

In particular, we have some concerns about an investigation into whether non-contact cooling water from DuPont's operations at the Fayetteville Works is "causing groundwater containing PFAS to infiltrate the outfall channel." Previous documents submitted in connection with the Consent Order have mentioned PFAS contamination found near the operations of the third tenant at the site, Kuraray[3]. Given that both DuPont and Kuraray continue to discharge process wastewater under Chemours' NPDES permit, we would ask the State and Chemours to provide more details about these items, as well as results of sampling of DuPont's and Kuraray's discharges.

5. The State should require Chemours to provide relief for CFPUA's customers that is equivalent to and just as timely and certain as the relief it is requiring for private well owners – *not in lieu of but in addition to* what is being done to address the PFAS contamination emanating from Chemours' highly contaminated industrial site.

On September 2, 2020, the N.C. Department of Environmental Quality issued a news release[4] about a meeting with CFPUA Board members and staff. Among those at the meeting were NCDEQ Secretary Michael S. Regan and representatives of the N.C. Attorney General's office.

At that meeting, Secretary Regan asked CFPUA what the State could do for downstream water users such as CFPUA's customers. The answer is plain: Use the 10/70 level to require Chemours to provide both immediate, interim relief and permanent relief measures equivalent to those the Consent Order requires Chemours to provide to private well owners near Chemours' plant.

Immediate, interim relief could take the form of funds from Chemours to provide individual stipends to affected downstream water users to purchase bottled water or install under-the-sink filtration systems until permanent relief is provided. This is equivalent to what is provided to the private well owners.

Permanent relief is apparent: Require Chemours to fully fund the upgrades currently underway at the Sweeney Plant to add GAC filters to effectively treat raw water contaminated by Chemours' PFAS and to pay for the costs to operate them. This also is equivalent to what is provided to private well owners. As noted above, the additional GAC filters at Sweeney will be operational long before Chemours must demonstrate it has successfully achieved the PFAS loading reduction goals for the barrier wall.

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[3] "Outfall 002 Assessment" https://www.chemours.com/en/-/media/files/corporate/ncdeq-cfrw-submission-consent-order-para-12-11-1.pdf

[4] "DEQ Secretary's Statement on PFAS discussion with CFPUA" https://deq.nc.gov/news/press-releases/2020/09/02/deq-secretarys-statement-pfas-discussion-cfpua

Granting these immediate and the permanent measures should in no way relieve Chemours of its responsibility to take any of the actions stipulated in the Consent Order or the Addendum. PFAS deposited by air emissions from Chemours' operations are largely responsible for the contamination of the private wells. The requirement that Chemours provide relief to the owners of those wells did not absolve the company from its responsibility to address the air emissions that caused the problem in the first place. Likewise, providing relief to downstream water users in a timely and direct manner should not absolve Chemours from addressing the groundwater, runoff, discharge, contaminated sediment in the more than 50 miles of riverbed between Chemours and CFPUA's intake at Kings Bluff, and other pathways sending Chemours' PFAS into the Cape Fear River.

Regards,

James R. Flechtner, PE
Executive Director

Attachments

NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

COMES NOW Cape Fear Public Utility Authority and renews and amends its Motion to Intervene in this action as a party pursuant to N. C. Gen. Stat. § 1A-1, Rule 24. In support of this Motion, Cape Fear Public Utility Authority shows the following to the Court:

1. Cape Fear Public Utility Authority ("CFPUA") is a public utility authority created by New Hanover County and the City of Wilmington pursuant to North Carolina General Statutes Chapter 162A, and is vested with authority to sue in its own name. N.C. Gen. Stat. § 162A-6. CFPUA exercises public and essential governmental functions to provide for the public health and welfare of its customers by providing potable water for residents of and businesses in New Hanover County and the City of Wilmington.

2. CFPUA owns and operates a raw water intake located on the Cape Fear River, downstream of the Defendant's Fayetteville Works Facility ("Facility" or "Chemours Facility"), and a water treatment plant to provide potable water to CFPUA's customers. CFPUA currently provides potable water to approximately 200,000 people and the businesses within its service area in New Hanover County.

3. Attached to this motion is CFPUA's proposed Amended Intervenor Complaint alleging the claims that CFPUA seeks to assert in this action.

Impacts to CFPUA Caused by Chemours and Acknowledged by the State

4. The State of North Carolina commenced this action against defendant The Chemours Company FC, LLC ("Chemours") on September 7, 2017. The original Complaint ("State's Original Complaint") was brought by the State pursuant to its delegated authority under the federal Clean Water Act ("CWA"), 33 U.S.C. §§ 1251 *et seq.*, to administer and enforce the National Pollution Discharge Elimination System ("NPDES") program, 33 U.S.C. § 1342, as specified in Article 21 of Chapter 143 of the North Carolina General Statutes. *See* State's Original Complaint ¶¶ 6-10.

5. In the State's Original Complaint, the State alleged (among other things): (a) the surface water into which the Chemours Facility discharges wastewater is used as a public water supply source that serves residents and businesses in several counties [Paragraph 48]; (b) Chemours and its predecessor knew for years that GenX and related compounds were being discharged into surface waters of the State [Paragraphs 56, 88]; (c) water samples collected at various times from the Cape Fear River showed concentrations of GenX were present in the Cape Fear River at levels in excess of the Department of Health and Human Services ("DHHS") health goal [Paragraphs 63, 87]; (d) GenX and related compounds discharged from the Chemours Facility have been and are present in public drinking water supplied to residents and businesses in several counties [Paragraph 55]; (e) from at least the beginning of 2009, Chemours' predecessor was aware of EPA's concern regarding the toxic effects of GenX on human health and the environment [Paragraphs 78-80]; (f) Chemours' continuing violations of North Carolina water quality laws adversely affect the public interest [Paragraph 128]; and (g) the State is entitled to injunctive relief against Chemours to prevent and abate Chemours' unpermitted discharges [Paragraph 129].

6. Pursuant to N.C. Gen. Stat. § 143-215.6C, in an enforcement action asserted by the State, if the Court determines that a violation of the North Carolina water laws or rules has occurred or is threatened, the Court "shall grant the relief necessary to prevent or abate the violation or threatened violation" (emphasis added).

7. GenX and related compounds are within a family of chemicals known as per- and polyfluoroalkyl substances or "PFAS." These chemicals are commonly used in the manufacture of nonstick coatings, stain- and water-resistant products, in fire-fighting foams, and for other consumer and commercial purposes.

8. Beginning the last week of June 2017, the CFPUA has undertaken periodic sampling and analysis of Cape Fear River water, both the intake "raw" river water and treated "finished" water for distribution to its customers. A spreadsheet of the analytical results for PFAS concentrations in samples of raw and finished water is attached to CFPUA's proposed Amended Intervenor Complaint as Exhibit A. The spreadsheet reflects that samples of the raw and finished Cape Fear River water have contained at least 23 different specific PFAS compounds in the water samples. The spreadsheet also shows the continuing variability over time of concentrations of PFAS compounds in the raw water and the finished water.

9. Additionally, graphs charting historic PFAS levels in the Cape Fear River against river flows at the raw water intake are attached to CFPUA's proposed Amended Intervenor Complaint as Exhibits B and C. As these data demonstrate, PFAS concentrations are largely a function of river flows. Higher river flows dilute PFAS in the river water, leading to lower concentrations. Conversely, lower flows result in higher PFAS concentrations. Accordingly, the levels of PFAS that CFPUA and its customers are exposed to are largely dependent on weather.

10. CFPUA's water treatment plant does not have the capability to treat and remove the PFAS pollutants that currently exist in the Cape Fear River. Although CFPUA can take certain

interim measures to reduce PFAS levels in finished water by periodically replacing biofilters designed for other water treatment purposes, those measures are not only unsustainably expensive but also reduce the biofilters' capacity to remove contaminants for which they were designed.

11. After conducting pilot testing on treatment options to remove the PFAS pollutants from Cape Fear River water, CFPUA determined that the addition of a granular activated carbon ("GAC") filter system would be its best, lowest cost option for treatment. The cost of designing, constructing, testing, implementing, and operating a GAC system to remove PFAS pollutants from the raw Cape Fear River water will be at least $70 million over a ten year period.

State's Actions Following its Original Complaint Have Left CFPUA Unprotected

12. On September 8, 2017 – less than 24 hours after the State filed its Original Complaint – a hearing was held at which a Consent Order was entered ("Original Consent Order"), which recites that it "partially resolves this matter." Original Consent Order at 1.

13. Prior to the State's commencement of this enforcement action, CFPUA and its counsel were in frequent contact with various representatives of the North Carolina Department of Environmental Quality (DEQ) to provide information, especially emphasizing the vulnerable population served by CFPUA, and urging the State to take prompt and comprehensive enforcement action. Neither CFPUA nor its counsel were informed by the State of the filing of the State's Original Complaint, the hearing scheduled for September 8, 2017, or the proposed Original Consent Order. CFPUA learned of the action and the Original Consent Order only after the Original Consent Order had been entered and filed.

14. On October 16, 2017, CFPUA filed its own action against Chemours and E.I. du Pont de Nemours and Company (DuPont), Chemours' predecessor in interest, in federal court in the Eastern District of North Carolina. *Cape Fear Public Utility Authority v. The Chemours Company FC, LLC and E.I. du Pont de Nemours and Company,* 7:17-cv-195, Federal District

Court, Eastern District of North Carolina (the "CFPUA Federal Suit"). CFPUA alleged (among other things): (a) Chemours has discharged, directly and via the groundwater, pollutants into the State's groundwater and the Cape Fear River, in violation of federal and state law and applicable permits; (b) CFPUA is a downstream riparian owner that uses water from the Cape Fear River; (c) the Cape Fear River water is adversely affected by the past and current discharges of pollutants by Chemours; (d) as a riparian owner, CFPUA has a right to use water from the Cape Fear River whose quality is not unreasonably diminished; (e) the sediments in the river have accumulated and hold substantial quantities of the pollutants discharged by Chemours and its predecessor, and this will continue to adversely affect the groundwater and the waters of the Cape Fear River; (f) the current and prior pollutant discharges have caused and continue to cause damage to CFPUA; (g) CFPUA is entitled to damages for the harm caused by the prior pollution; (h) CFPUA is entitled to an order requiring Chemours to restore the river and its sediments to an unpolluted state; and (i) CFPUA is entitled to prospective relief such that CFPUA does not continue to suffer injury and damage as a result of the actions and inactions of Chemours and its predecessor.

15. Brunswick County filed a lawsuit similar to the CFPUA Federal Suit against Chemours and DuPont (7:17-cv-209) in federal district court in the Eastern District of North Carolina. Thereafter, the Brunswick County lawsuit and the CFPUA Federal Suit were consolidated in the Eastern District of North Carolina and a Master Complaint of Public Water Suppliers was filed.

16. On October 17, 2017, CFPUA filed a Motion to Intervene in this action (the "Original Motion to Intervene"). In its Original Motion to Intervene: (a) CFPUA asserted it has an interest in the relief granted in this action to assure that such relief adequately protects its interests and those of its customers; (b) CFPUA asserted its ability to obtain relief (including injunctive relief that would compel removal of the sources of on-going PFAS contamination of the Cape Fear

River) might be impaired if the State fails to prevail in whole or in part in this action or if the State compromises this action in a manner detrimental to CFPUA; and (c) CFPUA sought intervention to protect its right to notice and opportunity to comment on any future settlement of this action.

17. On November 13, 2017, the State, Chemours, and CFPUA executed and filed a *Stipulation of All Parties Regarding Settlement Procedures and Withdrawal of Motion to Intervene* in this action. Pursuant to this Stipulation: (a) DEQ agreed (among other things) to provide written notice and at least 30 days for public comment with respect to any proposed settlement between the State and Chemours of this action; and (b) CFPUA withdrew its Original Motion to Intervene.

18. On or around April 9, 2018, the State filed an *Amended Complaint and Motion for Interim Preliminary Relief* ("State's Amended Complaint") in this action. In the State's Amended Complaint, the State alleged (among other things) many of the same or similar allegations it had alleged in the State's Original Complaint (as described in Paragraph 5 of this motion) regarding Chemours' knowing discharges of GenX and other PFAS into the Cape Fear River, the toxic effects of PFAS on human health and the environment, the use of the river water as a public water supply source that serves residents and businesses in several counties, and the presence of PFAS discharged from the Chemours Facility in public drinking water. The State's Amended Complaint also alleged that: (a) the State has obtained additional evidence of the extent of contamination caused by Chemours' release of PFAS into the environment [Paragraph 5]; (b) Chemours has identified the migration of groundwater from the Chemours Facility to the Cape Fear River as the most significant current source of contaminant loading in the river [Paragraph 126]; and (c) a major source of groundwater contamination, both onsite and offsite of the Chemours Facility, is Chemours' air emissions from the Facility [Paragraph 132].

19. On June 11, 2018, the State published a proposed *Order for Preliminary Injunctive Relief* for public comment. On July 10, 2018, CFPUA (through its counsel) provided written

comments in response to the State's proposed *Order*. CFPUA's comments generally supported the preliminary relief sought by the State, but also requested revisions to the proposed *Order* that would seek additional information and provide additional preliminary relief for the downstream water utilities.

20. On November 21, 2018, the day before Thanksgiving, DEQ announced on its website its proposal to enter into a proposed Consent Order ("PCO") with Chemours and Cape Fear River Watch (an environmental organization that also signed the PCO, which provided for the organization's intervention in the action by consent). *See* https://deq.nc.gov/news/press-releases/2018/11/21/release-state-officials-require-chemours-provide-permanent-drinking.

DEQ's Thanksgiving announcement states, "The proposed consent order is a comprehensive resolution regarding per- and polyfluoroalkyl substances (PFAS) contamination originating from Chemours' Fayetteville Works facility." The announcement also stated that DEQ would accept public comment on the PCO until December 21, 2018.

21. CFPUA was unaware that the parties to this action had reached a proposed settlement or had agreed to propose a Consent Order until the PCO was published by DEQ on the day before Thanksgiving 2018. CFPUA was not consulted about or notified of the status of the parties' settlement negotiations, the potential terms of a proposed settlement, or the impending publication of the PCO. DEQ did not seek input from CFPUA regarding how the terms of the proposed settlement might (or might not) address the harm or provide relief to CFPUA and its customers. CFPUA provided timely comments on the deficiencies of the PCO.

22. Paragraph 12 of the PCO was targeted at reducing PFAS loading to the Cape Fear River. If PFAS loading to the river is reduced, this would provide at least some relief to CFPUA. However, the PCO allowed for a five year implementation period with proposed interim actions that would theoretically provide downstream PFAS reductions in river water. The PCO also

obligated Chemours to take certain actions intended to: (a) reduce future discharges of PFAS pollutants from the Chemours Facility and (b) prevent current and future consumption of contaminated groundwater by citizens who live around the Facility and obtain potable water from water supply wells in the vicinity of the Facility. But the PCO did not include any provision that requires Chemours to prevent the current and ongoing use or consumption of PFAS-contaminated Cape Fear River water by downstream citizens and other users (including CFPUA) – even though the State acknowledges this harm, acknowledges CFPUA's current inability to remove these pollutants from Cape Fear River water, and requests relief for this harm in the State's Original and Amended Complaints in this action.

23. On December 20, 2018, CFPUA filed a Motion to Intervene and calendared it for hearing on January 14, 2019, in light of the failure of the PCO to address the ongoing harm to CFPUA and its customers. Following discussions with DEQ regarding the terms of the PCO, CFPUA agreed to remove its Motion to Intervene from the calendar, but not withdraw the motion itself, to allow the parties time to consider amendments to the PCO that would protect CFPUA and its customers from exposure to PFAS-contaminated river water. *See* Tr. of Hrg. on Mot. for Entry of Consent Order ("Hrg. Tr.") at 31.

24. Then, on February 20, 2019, counsel for DEQ notified counsel for CFPUA that DEQ had revised the terms of the PCO and had filed a motion for entry of a Revised Proposed Consent Order ("Revised PCO"), to be heard five days later. CFPUA had not previously seen or been notified of the revised terms, nor was there time for CFPUA to call a Board meeting to allow the Board to consider the proposed Revised PCO or to determine whether CFPUA should pursue its Motion to Intervene. Counsel for CFPUA advised the Court of this predicament at the hearing on February 25, 2019. *See* Hrg. Tr. at 30-31. The Court did not to rule on CFPUA's motion at the

February 25 hearing, but approved the Revised PCO ("Revised Consent Order") without CFPUA's participation.

25. For CFPUA, the Revised Consent Order is an improvement over the Original Consent Order, in that the Revised Consent Order mandates interim benchmarks that Chemours would have to meet to reduce certain pathways for PFAS loading to the river. However, the Revised Consent Order still has the same fundamental deficiencies described in this Motion – it leaves CFPUA customers exposed to PFAS in their drinking water for years, and any relief it does provide to CFPUA and its customers is both uncertain and insufficient (as discussed below).

26. The Revised Consent Order does, however, provide immediate relief for a different set of North Carolina citizens. Indeed, one of the most significant aspects of the Revised Consent Order is the requirement for replacement water supplies for certain citizens exposed to PFAS-contaminated water, as set forth in Section F of the Revised Consent Order. For fourteen PFAS identified on Attachment C of the Revised Consent Order, the Revised Consent Order established drinking water standards of 10 parts per trillion (ppt) for an individual PFAS, and 70 ppt for combined PFAS levels (the "Bladen County Limit").[1] Consent Order ¶ 20. For one set of persons whose water is contaminated in excess of the Bladen County Limit, Chemours is obligated to provide interim replacement water within three days of being notified, and permanent reverse osmosis systems within six months. Consent Order ¶¶ 20 and 23.

27. Inexplicably, the Bladen County Limit only applies to groundwater users. The result is that Bladen County residents whose groundwater is contaminated in excess of the Bladen County Limit standard receive near-immediate relief. Conversely, CFPUA and its customers, whose raw and finished water regularly exceeds the Bladen County Limit, must wait years for the

[1] For purposes of calculating the Bladen County Limit, four of the PFAS are grouped into two sets based on their molecular similarity.

theoretical possibility of clean water. The Revised Consent Order creates, in effect, two classes of North Carolina citizens that have suffered the same harm, and it treats the two classes of citizens differently for no good reason. DEQ's unequal treatment of two classes of citizens who have suffered the same harm is unexplained, unjustified, and arbitrary and capricious.

28. The Revised Consent Order has three primary deficiencies with respect to CFPUA and its customers: (1) a flawed premise, (2) deferred relief to CFPUA customers, and (3) no certainty of adequate relief.

29. *First*, the Revised Consent Order is based on a flawed premise. As justification for entry of the Revised Consent Order, DEQ and Chemours both assured this Court that its implementation would result in the continued reduction of PFAS levels in the Cape Fear River. For instance, counsel for DEQ asserted that "[a]s a result of DEQ requiring cessation of the discharge of process wastewater, there were dramatic reductions in the concentrations of GenX in Chemours' discharge," and "similar reductions" in CFPUA's finished water. Hrg. Tr. at 8. Similarly, counsel for Chemours stated that the cessation of its PFAS-laden wastewater discharges "resulted in truly dramatic reductions in the levels of GenX in the river." Hrg. Tr. at 23. DEQ further emphasized to the Court that Paragraph 12 of the Revised Consent Order requires Chemours to prepare a plan to achieve the maximum feasible reduction in PFAS loading from the Chemours Facility to the Cape Fear River and was "of central importance for downstream communities." Hrg. Tr. at 14.

30. In other words, DEQ and Chemours informed the Court that two remedial measures would lead to continued reduction of PFAS levels in the river and thereby provide the relief sought by CFPUA and its customers: (a) Chemours' cessation of discharges of PFAS contaminated process wastewater into the Cape Fear River (discharges that had occurred for over 30 years); and (b) Chemours would study and then address PFAS loading from its Facility to the Cape Fear River

thereafter. Based on those two remedial measures, DEQ and Chemours argued to the Court that PFAS levels in the river had dropped in prior months, and they theorized that PFAS levels would continue to drop in the immediate near term.

31. While both measures are helpful and, indeed, necessary, the theory (as explained to the Court) has not matched reality. As demonstrated by the continued monitoring of PFAS over the past 18 months since the hearing, PFAS levels in the Cape Fear River have been variable – not decreasing – and are largely dependent on river flows. PFAS in groundwater, surface water runoff, and sediment continues to migrate into the river from within and around the Facility and from accumulated sediment in the Cape Fear River bed caused by decades of Chemours' discharges and emissions of PFAS pollutants into the environment.

32. The data demonstrate that, in the months preceding the February 2019 hearing, high river flows were largely responsible for the "dramatic reductions" in PFAS concentrations presented to the Court. Following the hearing, PFAS levels in the Cape Fear River later increased significantly due to drier weather and resulting decreased river flow. PFAS levels in the river have not continued their decline, as was represented to the Court.

33. Of the 58 raw water sampling events since the hearing on the Revised PCO, 47 exceeded the Bladen County Limit standard for the Attachment C PFAS. Of the 44 finished water samples, 32 exceeded this standard.

34. *Second*, the relief to CFPUA offered by the Revised Consent Order will not be realized for years, unlike the relief provided to Bladen County residents. The Revised Consent Order allows Chemours five years to implement a plan to reduce PFAS loading to the Cape Fear River from groundwater at the Facility. Revised Consent Order ¶ 12.a.

35. As required by the Revised Consent Order, Chemours submitted to DEQ a *Cape Fear River PFAS Loading Reduction Plan* on August 26, 2019, and the related *Corrective Action*

Plan ("CAP") on December 31, 2019, detailing its proposals to remediate the groundwater at the Facility and reduce PFAS loading to the river. Under Chemours' own estimates (which CFPUA's consultant Tetra Tech has opined are not scientifically supported, *see* Exhibit D and Exhibit E, attached to the proposed Amended Intervenor Complaint), it will take through 2022 for Chemours to control just 43% of the PFAS loading from its Facility to the Cape Fear River. By the end of 2024, Chemours estimated it will have controlled just 79% of PFAS releases from its Facility to the river. The full extent of Chemours' proposed remedial actions are expected to take between 5 and 10 years, if not longer. All the while, the water of the Cape Fear River will regularly exceed the Bladen County Limit.

36. Conversely, the Revised Consent Order requires that Chemours provide temporary replacement water supplies to the citizens of Bladen County within 3 days of becoming aware that an affected user's groundwater exceeds the Bladen County Limit, and a permanent replacement within 6 months. Consent Order ¶¶ 20 and 23.

37. Under the Revised Consent Order, the citizens of Bladen County are assured relief within days. Meanwhile, CFPUA customers have been, and will continue to be, subject to water in excess of the Bladen County Limit for many years.

38. *Third*, the Revised Consent Order and Chemours' *Loading Reduction Plan* and CAP do not assure adequate relief to CFPUA. As an initial matter, even assuming Chemours can meet its projections, its remedial actions were projected to reduce PFAS loading from its Facility by just 79%. But Chemours itself acknowledges that its proposed long-term groundwater remedy is "still highly conceptual," and that "it is not presently possible to conclude with confidence whether this alternative is economically feasible." *See* CAP at 71, 74. Moreover, the PFAS *Loading Reduction Plan* and CAP do nothing to address the extensive soil, groundwater, and sediment contamination surrounding the Facility and in the riverbed, which will continue releasing

PFAS to the Cape Fear River for decades. Therefore, the Chemours *Loading Reduction Plan* and CAP represent an uncertain, unreliable remedy for the downstream water utilities and constitute a violation of the intent of Paragraph 12 of the Revised Consent Order.

39. As such, CFPUA and its customers will continue being subjected to river water contaminated with PFAS for the foreseeable future. And given the limits of the remediation proposed by Chemours, there is no assurance that even after its completion the water of the Cape Fear River will meet the Bladen County Limit. The only way to assure that CFPUA's finished water will meet that standard is to provide every customer with clean bottled water (as required for the Bladen County users) for all domestic uses or build a treatment system designed to remove PFAS as quickly as possible from the raw river water, as CFPUA is doing.

40. Once it became clear that the rosy projections of DEQ and Chemours did not prove valid, the CFPUA Board authorized continuing the design of the GAC system, and on September 11, 2019, awarded a construction contract for the GAC and approved revenue bonds to finance its cost. The bond sale was held on October 17, 2019, and CFPUA customers began incurring a charge for the amortization of the bonds on July 1, 2020. Construction on the GAC began on November 4, 2019, and the plant is expected to be completed and operational in 2022.

The Proposed Addendum Does Not Ensure Relief to CFPUA

41. Since the Revised Consent Order was entered, CFPUA has continued to share its monitoring results and concerns with DEQ, including its data showing that the high variability of PFAS concentrations in the Cape Fear River over time are largely dependent on the river's flow rate. Chemours has conducted site assessments which indicate that the majority of PFAS loading from the Chemours Facility into the Cape Fear River is due to groundwater contamination. Chemours also estimated that the Facility has been responsible for between 68% and 84% of PFAS concentrations at CFPUA's water intake in the river.

42. In its discussions with CFPUA, DEQ recognized that the Revised Consent Order in its current form is deficient. DEQ has now proposed additional modifications, releasing a draft Addendum to the Revised Consent Order for public comment on August 13, 2020 (the "proposed Addendum"). However, consistent with its past practice, DEQ again did not consult with CFPUA on the terms of the proposed Addendum, instead notifying CFPUA of its existence just hours before its public release.

43. The proposed Addendum requires concrete remediation directed toward limiting groundwater migration from the Chemours Facility into the Cape Fear River. Yet, as it relates to CFPUA and other downstream water users, the proposed Addendum still suffers from exactly the same major deficiency as the Revised Consent Order: There is still no assurance, or even reasonable expectation, that PFAS levels in the Cape Fear River will ever consistently reach the Bladen County Limit, and there is still no immediate relief for CFPUA or its customers. CFPUA is left in the same position as it was before the proposed Addendum: wait an indeterminate number of years for an indeterminate level of relief from an indeterminate number of PFAS compounds.

44. Based on the evidence in this action and the studies undertaken since implementation of the Revised Consent Order, the State is aware that: (a) hundreds of PFAS pollutants exist in the surface water in the Cape Fear River; (b) even if the Chemours Facility immediately ceases all emissions and discharges of PFAS pollutants into the Cape Fear River and completes the remediation improvements now promised, those pollutants will continue to contaminate the surface water in the Cape Fear River for decades to come (because PFAS pollutants in the vegetation, soils, and groundwater in a large and unknown radius around the Chemours Facility will continue to migrate into the river water through groundwater flow and surface run-off and PFAS pollutants entrained in the Cape Fear River sediment will continue to be released into the river water); (c) Cape Fear River water is being used downstream from the

Chemours Facility by CFPUA, customers of CFPUA, and other citizens; and (d) downstream utilities like CFPUA do not have the ability to consistently remove PFAS pollutants from the drinking water supplied to their customers without the construction of additional treatment systems, such as the GAC system currently being constructed by CFPUA. Yet again, the State has left CFPUA to its own devices in dealing with current and reasonably foreseeable PFAS contamination in the river.

45. As part of the Addendum requirements and as described in the proposed NPDES Permit # NC 0089915 that was sent to public notice on July 10, 2020 ("proposed Permit"), DEQ is requiring Chemours to analyze for 59 distinct PFAS compounds found in Chemours' proposed wastewater discharge (the "Full Suite = Table 3+ Lab SOP +Method 537 Compounds"). This proposed Permit only covers stormwater and groundwater seepage in old outfall 2, and does not govern the main Chemours wastewater treatment plant discharge that is current being captured and shipped offsite for disposal (which is covered by a separate permit). CFPUA has filed a written objection to DEQ's failure to place any controls over the remaining 56 PFAS compounds and the fact that the proposed Permit would allow a maximum daily discharge of the three regulated compounds (GenX, PFMOAA and PMPA) of 964 ppt. In addition, in its June 30, 2020, *PFAS Non-Targeted Analysis and Methods Interim Report on Process and Non-Process Wastewater and Stormwater,* Chemours identified 21 new and previously unknown PFAS compounds in its "General Facility Discharge" and 250 new and unknown PFAS compounds in its "Process Wastewater."

46. The Secretaries' Science Advisory Board ("SAB") continues to review studies related to the appropriate health and regulatory standards for GenX and other PFAS compounds. At its August 31, 2020 meeting, the SAB reviewed studies by two groups of scientists (Exhibits F and G) on the probable toxicological impacts of GenX (and PFOA) on mice and rats and studies

by another group of scientists (Exhibit H) on the probable toxicological impacts of BFESA-BP2 (Nafion Byproduct 2) on mice. Both of these PFAS compounds are found in significant amounts in Chemours' discharges and releases. And these are but two of the hundreds of PFAS compounds now identified as emanating from the Chemours Facility and likely entrained in the sediments of the riverbed. What health and environmental impacts these compounds will have individually and synergistically is going to take decades to determine.

47. The State's own Original and Amended Complaints in this action acknowledge that North Carolina citizens and water utilities downstream from the Chemours Facility are using PFAS contaminated Cape Fear River water. But DEQ has chosen not to seek further near-term relief for the harms to CFPUA and its customers and, through the proposed Permit, intends to allow Chemours to discharge a combined concentration of 964 ppt of three PFAS compounds (GenX, PFMOAA and PMPA) – without express limits on other PFAS – from proposed Outfall 003 at the Chemours Facility. Since the proposed Addendum and proposed Permit do not provide reasonably certain or adequate relief for the harms suffered by CFPUA and its customers, CFPUA is entitled to participate in this action as a full party and to present its claims and evidence to the Court requiring such abatement.

48. This motion is timely as it is now apparent that: (a) PFAS levels in the Cape Fear River are not declining as previously represented to the Court, and (b) DEQ and Chemours will not provide any immediate relief to CFPUA and its customers, and (c) any relief that is provided pursuant to the Revised Consent Order is uncertain and inadequate. Following the hearing on February 25, 2019, CFPUA has continued monitoring PFAS levels in the Cape Fear River to determine whether the relief in the Revised Consent Order to the downstream water providers would suffice. It has not sufficed, nor will it in any reasonable amount of time, if ever. The recent proposed Addendum was negotiated and agreed to without the input or consent of CFPUA, the

person it most significantly impacts. CFPUA cannot protect its interests unless it is allowed to participate in this action as a party.

49. At its August 12, 2020 meeting, the CFPUA Board approved the preparation and filing of this Motion to Intervene and attached proposed Amended Intervenor Complaint.

Intervention of Right Pursuant to Rule 24(a)(2).

50. North Carolina law provides that a person may intervene in a lawsuit as a matter of right under certain circumstances:

> When the applicant claims an interest relating to the property or transaction which is the subject of the action and he is so situated that the disposition of the action may as a practical matter impair or impede his ability to protect that interest, unless the applicant's interest is adequately represented by existing parties.

N.C. Gen. Stat. § 1A-1, Rule 24(a)(2).

51. North Carolina law requires that a motion to intervene be timely and that the applicant establish that: "(1) it has a direct and immediate interest relating to the property or transaction, (2) denying intervention would result in a practical impairment of the protection of that interest, and (3) there is inadequate representation of that interest by existing parties." *Virmani v. Presbyterian Health Services Corp.,* 350 N.C. 449, 459, 515 S.E.2d 675, 683 (1999).

52. *Timeliness of motion.* Whether an application to intervene is timely is left to the discretion of the trial court, which "will consider the following factors: (1) the status of the case, (2) the possibility of unfairness or prejudice to the existing parties, (3) the reason for the delay in moving for intervention, (4) the resulting prejudice to the applicant if the motion is denied, and (5) any unusual circumstances." *Procter v. City of Raleigh Board of Adjustment,* 133 N.C. App. 181, 183, 514 S.E.2d 745, 746 (1999), *citing State Employees' Credit Union, Inc. v. Gentry,* 75 N.C. App. 260, 330 S.E.2d 645 (1985).

53. CFPUA's motion to intervene is timely in this case. *First,* a motion to intervene is "rarely denied as untimely prior to the entry of judgment...." *Hamilton v. Freeman,* 147 N.C. App. 195, 201, 554 S.E.2d 856, 859-60 (2001), *petitions and appeal dismissed,* 355 N.C. 285, 560 S.E.2d 803 (2002). In this action, judgment has not yet been entered. Rather, the State has negotiated and is administering a Revised Consent Order, and has now proposed an Addendum, under the continuing oversight of this Court that are inadequate to protect the interests of CFPUA.

54. *Second,* there has been no delay by CFPUA in filing this motion. CFPUA has continuously monitored both implementation of the Revised Consent Order and its impacts on PFAS levels in the Cape Fear River. There can be no question now that the relief to CFPUA is inadequate, as DEQ itself has acknowledged in publishing the proposed Addendum. CFPUA and its customers will continue to be exposed to PFAS concentrations that DEQ has determined, through the Revised Consent Order, require action. That DEQ understood CFPUA's predicament but excluded it from participating in the preparation of the terms of the proposed Addendum confirms that CFPUA must be a party to this action for its interests to be protected. CFPUA is acting accordingly.

55. *Third,* there is no risk of unfairness or prejudice to the existing parties. To the contrary, DEQ and Chemours have been aware of CFPUA's interests and concerns since before the State's Original Complaint was filed, and DEQ and Chemours continue to discuss and negotiate the terms and requirements of the Revised Consent Order, the scope of remediation for Chemours' releases of PFAS contaminants into the environment, and a proposed Addendum that directly impacts CFPUA and its customers, but without CFPUA at the table. The only prejudice is to CFPUA, by not being a party to this action.

56. *Fourth,* there are "unusual circumstances" here. In spite of assurances of its concerns for CFPUA and its customers, DEQ has proven that it has its own enforcement agenda

in this case, and that agenda does not include any immediate and certain relief for CFPUA and its customers. It is "unusual" that a state regulatory agency would admit in court filings that Chemours' pollutant releases are causing ongoing significant harm to CFPUA and its customers, but deliberately and repeatedly exclude CFPUA from the negotiations to remedy the harm. DEQ now has shown a consistent, carefully considered unwillingness to confer with CFPUA about the remediation measures DEQ is contemplating and that directly impact CFPUA and its customers.

57. *First requirement of intervention as of right:* CFPUA has "a direct and immediate interest relating to the property or transaction" involved in this enforcement action. The "property" which is the subject of this action is groundwater and the waters of the Cape Fear River. CFPUA withdraws raw water from the Cape Fear River for treatment and distribution of treated water to approximately 200,000 people and to businesses in New Hanover County. For the past 30 years, Chemours and its predecessor have discharged PFAS pollutants directly into the river and via the groundwater in violation of their NPDES permit, the Clean Water Act, and state law. The quality of the waters of the Cape Fear River is unreasonably diminished by these current and past discharges of pollutants. As a riparian owner, CFPUA has a right to use water from the Cape Fear River whose quality is not unreasonably diminished. In addition, sediments in the river have accumulated PFAS pollutants discharged by Chemours and its predecessor over time, and the contaminated sediments will continue to adversely affect the groundwater and unreasonably diminish the quality of the waters of the Cape Fear River into the indefinite future. Chemours also has released PFAS contaminants by air emissions over an extensive area surrounding the Facility, and those PFAS contaminants have been deposited on the land surface and are reaching the Cape Fear River by overland surface run-off and groundwater migration. The current and prior emissions and discharges of pollutants have caused and continue to cause harm to CFPUA, as alleged in CFPUA's Federal Suit and in the attached proposed Amended Intervenor Complaint.

58. A transaction which is the subject of the pending action is the historic and current discharges and emissions of PFAS pollutants by Chemours and its predecessor from the Chemours Facility in violation of law; the historic, current, and future contamination of the Cape Fear River arising from those emissions and discharges; and the State's effort to obtain relief to abate Chemours' emissions and discharges of pollutants to the Cape Fear River. CFPUA has a direct and undeniable interest in the State's action (including the relief granted pursuant to this action) to ensure that the harms to CFPUA and its customers resulting from Chemours' emissions and discharges of pollutants are considered, comprehensive evidence of the harms and potential remedial options are presented and evaluated in their appropriate context, and any relief obtained in this action adequately protects CFPUA's interests and abates the harms caused to CFPUA and its customers.

59. *Second requirement of intervention as of right:* For several reasons, denying intervention to CFPUA would result in the "practical impairment" of CFPUA's ability to protect its interests.

(a) *First,* as explained in this motion, DEQ and Chemours continued to negotiate over the terms and implementation of the Revised Consent Order and have reached agreement with Chemours (without input from CFPUA) on a proposed Addendum whose implementation directly impacts CFPUA. CFPUA deserves a seat at the table and cannot adequately protect its interests without a seat.

(b) *Second,* by setting the Bladen County Limit for certain PFAS contaminants in groundwater in the vicinity of the Chemours Facility, DEQ arguably has established maximum concentrations of those compounds it has determined to be safe for human consumption and use for water supply. If one result of this action may be the explicit or implicit creation of drinking water standards for surface or groundwater contaminated by

Chemours' pollutant discharges, CFPUA has a direct interest in participating in the evaluation and setting of those standards – particularly if CFPUA may be required to treat raw Cape Fear River water to meet those standards.

(c) *Third,* one water quality standard applicable to fresh surface water that DEQ must enforce pursuant to the Clean Water Act and state law is: deleterious substances may be discharged "*only*" in such amounts that will "not render the waters injurious to public health ... or impair the waters for any designated uses." 15A NCAC 2B .0211(12). One designated use of the Cape Fear River surface water segment from which CFPUA withdraws water is "a source of water supply for drinking...." 15A NCAC 2B .0216(1). CFPUA has a direct and immediate interest in the State's enforcement of its water quality standards, including this particular standard, since (i) CFPUA treats and distributes drinking water, (ii) the PFAS pollutants discharged into the Cape Fear River are injurious to public health and impair the Cape Fear River water for its use as drinking water, and (iii) CFPUA's ability to provide safe drinking water will be undermined if DEQ is unable or unwilling to seek or accomplish adequate enforcement of the State's water quality standards.

(e) *Fourth,* in its CFPUA Federal Suit, CFPUA seeks damages from Chemours and its predecessor for their PFAS pollutant discharges, injunctive relief to restore the Cape Fear River and its sediments to an unpolluted state, and prospective relief such that CFPUA does not continue to suffer harms and damage as a result of the actions and inactions of Chemours and its predecessor. CFPUA's ability to obtain relief in the CFPUA Federal Suit may be impaired if the State compromises this underlying action in a manner detrimental to CFPUA's interests.

60. *Third requirement for intervention as of right:* It is clear now that CFPUA's interests are not adequately represented by the State or current intervenor in this action.

(a) *First,* the State's Original and Amended Complaints in this enforcement action acknowledge the contamination of the Cape Fear River and the harm to downstream river water users, including public water utilities. Based on the State's complaints and the State's statutory duty to protect the environment of North Carolina and to seek enforcement for environmental violations on behalf of the State's citizens, CFPUA had the reasonable expectation (at least at the outset of this action) that the State would seek to remedy all the significant harms caused by the PFAS pollutant discharges, not just some of them. Yet the Revised Consent Order and proposed Addendum do not require prompt cleanup of the contamination in the raw Cape Fear River water, nor do they provide any relief to CFPUA to assist with its treatment of raw Cape Fear River water. It has become evident in the course of this action that the relief provided in the Revised Consent Order and proposed Addendum is undependable and inadequate to protect CFPUA's interests or remedy the harms to CFPUA and its customers. To the contrary, the Revised Consent Order appears to establish drinking water standards that CFPUA currently cannot meet and that DEQ knows CFPUA currently cannot meet.

(b) *Second,* the relief provided in the Revised Consent Order and proposed Addendum is not adequate to enforce the State's water quality standards. Those standards require that discharges of deleterious substances to surface water (such as the Cape Fear River) be limited to amounts that do not injure public health or impair the water's use as a source of drinking water. But because the relief described in the Revised Consent Order and proposed Addendum does not require prompt abatement of the contamination in the raw or finished Cape Fear River water, the water quality standard currently cannot be met

and the ongoing, unremediated contamination of the Cape Fear River will continue to impair the river's use as a source of drinking water for CFPUA's customers for the foreseeable future.

(d) *Third,* while the Revised Consent Order provides specific, immediate relief to some citizens (those exposed to PFAS-contaminated groundwater in Bladen County), CFPUA is left to fend for itself. A private action is subject to different defenses and legal constraints than a State enforcement action; and, in any event, DEQ's decision to secure a higher degree of relief for groundwater users than surface water users necessarily means its interests have diverged from CFPUA's interests in this enforcement action.

61. In sum, CFPUA's interests in the remediation of the Cape Fear River and the use of river water for potable water purposes have been impaired by a Revised Consent Order that has failed to live up to its promise of relief to CFPUA, and a proposed Addendum that suffers from the same defects. CFPUA's interests are no longer adequately represented by the State, and CFPUA therefore is entitled to intervene in this enforcement action as a matter of right pursuant to N.C. Gen. Stat. § 1A-1, Rule 24(a)(2).

Permissive Intervention pursuant to Rule 24(b)(2)

62. In the alternative, CFPUA should be allowed to intervene pursuant to Rule 24(b) of the North Carolina Rules of Civil Procedure, which allows intervention:

> When an applicant's claim or defense and the main action have a question of law or fact in common. … In exercising its discretion the court shall consider whether the intervention will unduly delay or prejudice the adjudication of the rights of the original parties.

N.C. Gen. Stat. § 1A-1, Rule 24(b)(2).

63. CFPUA's claims asserted in the attached proposed Amended Intervenor Complaint involve the proper administration and enforcement of the North Carolina water protection laws

and the CWA on the facts alleged in the State's Amended Complaint, and they involve the same pollutant impacts to the Cape Fear River and the appropriate remedies for those impacts as are the subject of the State's Amended Complaint. As such, the same questions of law and fact are involved. Furthermore, CFPUA's intervention will not unduly delay or prejudice the adjudication of the rights of the existing parties, for the reasons previously stated in this motion.

Prayer for Relief

For the foregoing reasons, the Cape Fear Public Utility Authority requests that the Court grant the following relief:

> (a) an order granting this motion to intervene authorizing CFPUA to participate in this action as a party as a matter of right; or, in the alternative an order granting this motion allowing CFPUA to intervene permissively as a party in this action;
>
> (b) an order authorizing CFPUA to file the attached proposed Amended Intervenor Complaint;
>
> (c) the Court's consideration of the claims in the attached proposed Amended Intervenor Complaint, and an opportunity for CFPUA to present evidence and argument to the Court regarding its claims and the relief provided (and not provided) pursuant to the Revised Consent Order and the impacts of the Revised Consent Order on the interests of CFPUA and its customers; and
>
> (d) deferral of the Court's review of and decision on the proposed Addendum until CFPUA has had the opportunity to present evidence and argument for the Court's consideration on the impacts of the proposed Addendum on the interests of CFPUA and its customers and on necessary changes and additions to the proposed Addendum that would adequately remedy the harms to CFPUA and its customers.

CFPUA also requests that the Court: (i) grant such other and further relief as the Court deems just and proper, and (ii) tax the cost of this action, including attorneys' fees, if allowable, against the Plaintiff and/or Defendant.

Respectfully submitted this the _____ day of September, 2020.

Respectfully submitted this the 8 day of September, 2020.

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VERIFICATION

James R. Flechtner, PE, having been duly sworn, deposes and says that he is Executive Director of the Cape Fear Public Utility Authority (the proposed intervenor in this action), that he has read the foregoing RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED), that in his position as Executive Director he is familiar with and has knowledge of the facts stated in the foregoing RENEWED AND AMENDED MOTION, and those facts are true of his own personal knowledge.

James R. Flechtner

Subscribed and sworn to before me

this the 8th day of September, 2020

Notary Public

NOTARY PUBLIC
KRISTEN L. MAJORS
GUILFORD COUNTY, NC

My Commission Expires: June 19, 2024

CERTIFICATE OF SERVICE

The undersigned hereby certifies that a copy of the foregoing RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED) was served upon the parties in this action by depositing a copy of the same in the United States Mail, First Class Postage Prepaid, addressed as follows:

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This the 8 day of September, 2020.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant. CAPE FEAR PUBLIC UTILITY AUTHORITY, Intervenor-Plaintiff, v. STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Defendant.	**INTERVENOR COMPLAINT BY CAPE FEAR PUBLIC UTILITY AUTHORITY FOR DECLARATORY AND INJUNCTIVE RELIEF**

COMES NOW Cape Fear Public Utility Authority ("CFPUA"), through counsel, and alleges and says:

BACKGROUND FACTS

1. CFPUA is a public utility authority created by New Hanover County and the City of Wilmington pursuant to North Carolina General Statutes Chapter 162A, and is vested with authority to sue in its own name. N.C. Gen. Stat. § 162A-6. CFPUA exercises public and essential

governmental functions to provide for the public health and welfare of its customers by providing potable water for residents of New Hanover County and the City of Wilmington. CFPUA owns and operates a water intake located on the Cape Fear River, downstream of the Defendant's Fayetteville Works Facility, and a water treatment plant to provide potable water to 200,000 North Carolinians and the schools, hospitals, industry, and other businesses and institutions that serve them.

2. Defendant The Chemours Company FC, LLC ("Chemours") is a corporation organized and existing under the laws of Delaware, and registered to do business as a foreign corporation in the State of North Carolina. Chemours currently owns and operates the Fayetteville Works Facility, located at 22828 NC Highway 87 W., Fayetteville, North Carolina.

3. The State's original Complaint ("State's Original Complaint") in this action was brought on behalf of the Department of Environmental Quality ("DEQ"), an agency of the State of North Carolina, pursuant to its delegated authority under the federal Clean Water Act ("CWA"), 33 U.S.C. §§ 1251 *et seq.*, to administer and enforce the National Pollution Discharge Elimination System ("NPDES") program, 33 U.S.C. § 1342, as specified in Article 21 of Chapter 143 of the North Carolina General Statutes. *See* Original Complaint ¶¶ 6-10.

4. As alleged in the State's Original Complaint in this action, this matter arises out of Defendant's operation of the Fayetteville Works Facility (the "Facility"), a chemical manufacturing facility located adjacent to the Cape Fear River just south of Fayetteville, North Carolina.

5. In the State's Original Complaint, the State alleged (among other things):

a. The surface water into which Defendant's Fayetteville Works Facility discharges wastewater is used as a public water supply source that serves residents and businesses in several counties [Paragraph 48];

b. Chemours and its predecessor knew for years that GenX and related compounds were being discharged from the Facility into surface waters of the State, in violation of North Carolina water quality laws [Paragraphs 56, 88];

c. Water samples collected at various times from the Cape Fear River showed concentrations of GenX were present in the Cape Fear River at levels in excess of the health goal established by the North Carolina Department of Health and Human Services ("DHHS") [Paragraphs 63, 87];

d. GenX and related compounds discharged from the Facility have been and are present in public drinking water supplied to residents and businesses in several counties [Paragraph 55];

e. On information and belief, public water supply treatment plants are ineffective at removing GenX and related compounds from Cape Fear River water [Paragraph 54];

f. From at least the beginning of 2009, Chemours' predecessor was aware of EPA's concern regarding the toxic effects of GenX on human health and the environment [Paragraphs 78-80];

g. Chemours' continuing violations of North Carolina water quality laws adversely affect the public interest [Paragraph 128]; and

h. The State is entitled to injunctive relief against Chemours to prevent and abate Chemours' unpermitted discharges [Paragraph 129].

6. GenX and related compounds are within a family of chemicals known as per- and polyfluoroalkyl substances or "PFAS." These chemicals are commonly used in the manufacture of nonstick coatings, stain- and water-resistant products, in fire-fighting foams, and for other consumer and commercial purposes.

7. Beginning the last week of June 2017, the Cape Fear Public Utility Authority has undertaken periodic sampling and analysis of Cape Fear River water, both the intake "raw" river water and treated "finished" water for distribution. A spreadsheet of the analytical results for samples of raw and finished water is attached as Exhibit A. The spreadsheet reflects that samples of the raw and finished Cape Fear River water have contained at least 23 different specific PFAS compounds in the water samples, The spreadsheet also shows the continuing variability of concentrations of PFAS compounds in the raw water and the finished water.

8. Additionally, graphs charting historic PFAS levels in the Cape Fear River against river flows at the raw water intake is attached as Exhibits B and C. As these exhibits demonstrate, PFAS concentrations are largely a function of river flows. Higher river flows dilute PFAS in the river, leading to lower concentrations. Conversely, lower flows result in higher PFAS concentrations. Accordingly, the levels of PFAS that CFPUA and its customers are exposed to are largely dependent on weather.

9. CFPUA's water treatment plant does not have the capability to treat and remove the PFAS pollutants that currently exist in the Cape Fear River. Although CFPUA can take certain interim measures to reduce PFAS levels in finished water by periodically replacing biofilters designed for other purposes, those measures are not only unsustainably expensive but also reduce the biofilters' capacity to remove contaminants for which they were designed.

10. After conducting pilot testing on treatment options to remove the PFAS pollutants from Cape Fear River water, CFPUA determined that the addition of a granular activated carbon ("GAC") filter system would be its best option for treatment. The cost of designing, constructing, testing, implementing, and operating a GAC system will be at least $70 million over a ten year period.

11. The Cape Fear Public Utility Authority board approved a resolution authorizing CFPUA to proceed with the design, permitting, and construction of a GAC. CFPUA has since completed the GAC designs, sold revenue bonds to finance the cost of the GAC, executed a construction contract, begun charging customers for the amortization of the bonds, and begun construction on the plant. The GAC is expected to be completed and operational in 2022.

State's Actions Following its Original Complaint Have Left CFPUA Unprotected

12. On September 8, 2017 – less than 24 hours after the State filed its Original Complaint – a hearing was held at which a Consent Order was entered ("Original Consent Order"), which recites that it "partially resolves this matter." Original Consent Order at 1.

13. Prior to the State's commencement of this enforcement action, the Cape Fear Public Utility Authority and its counsel were in frequent contact with various representatives of the North Carolina Department of Environmental Quality (DEQ) to provide information, especially emphasizing the vulnerable population served by CFPUA, and urging the State to take prompt and comprehensive enforcement action. Neither CFPUA nor its counsel were informed by the State of the filing of this action, the hearing scheduled for September 8, 2017, or the proposed Original Consent Order. CFPUA learned of the action and the Original Consent Order only after the Original Consent Order had been entered and filed.

14. On October 16, 2017, the Cape Fear Public Utility Authority filed a separate action against Chemours and its predecessor in interest, E. I. du Pont de Nemours ("DuPont") in federal court in the Eastern District of North Carolina. *Cape Fear Public Utility Authority v. The Chemours Company FC, LLC and E.I. du Pont de Nemours and Company*, 7:17-cv-195 ("CFPUA's Federal Suit"). Following a similar action initiated by Brunswick County against Chemours and DuPont, 7:17-cv-209, the two actions were consolidated and a Master Complaint of Public Water Suppliers (the "Master Complaint") was filed, in which Town of Wrightsville Beach and Lower Cape Fear Water & Sewer Authority joined.

15. The claims alleged in the Master Complaint are common law claims arising under State law. As alleged in the Master Complaint and in CFPUA's Notice to Conform to Master Complaint:

a. Chemours and DuPont have discharged PFAS, directly and via the groundwater and air emissions, into the State's groundwater and the Cape Fear River, in violation of federal and state law and applicable permits;

b. CFPUA is a downstream riparian owner that uses water from the Cape Fear River;

c. The quality of the waters of the Cape Fear River water is unreasonably diminished by the past and current discharges and other releases of PFAS by Chemours and DuPont;

d. As a riparian owner, CFPUA has a right to use water from the Cape Fear River whose quality is not unreasonably diminished;

e. PFAS discharged by Chemours and DuPont have accumulated in the sediment of the Cape Fear River, the groundwater that feeds the River, and in deposits in the

watershed from the air emissions from the Facility, and this will continue to unreasonably diminish the quality of the waters of the Cape Fear River;

f. CFPUA's water treatment plant does not have the technical capability to treat and remove the PFAS pollutants that currently exist in the Cape Fear River;

g. The current and prior PFAS discharges have caused and continue to cause harm and damages to CFPUA;

h. CFPUA is entitled to damages for the prior pollution caused by Chemours and its predecessor and to injunctive relief to prevent and abate continuing harm and damages to CFPUA.

16. On or around April 9, 2018, the State of North Carolina filed an Amended Complaint and Motion for Interim Preliminary Relief ("Amended Complaint") in this action. In its Amended Complaint, the State alleged (among other things) many of the same or similar allegations it had alleged in its Original Complaint (as described in Paragraph 5 of this Complaint) regarding Chemours' knowing discharges of GenX and other PFAS into the Cape Fear River, the toxic effects of PFAS on human health and the environment, the use of the river water as a public water supply source that serves residents and businesses in several counties, and the presence of PFAS discharged from the Chemours Facility to the public drinking water. The State also alleged in its Amended Complaint that: (a) it has obtained additional evidence of the extent of contamination caused by Chemours' release of PFAS into the environment [Paragraph 5]; (b) Chemours has identified the migration of groundwater from the Chemours Facility to the Cape Fear River as the most significant current source of contaminant loading in the river [Paragraph 126]; and (c) a major source of groundwater contamination, both onsite and offsite, is Chemours' air emissions [Paragraph 132].

17. On June 11, 2018, the State published a proposed Order for Preliminary Injunctive Relief for public comment. On July 10, 2018, CFPUA (through its counsel) provided written comments in response to the State's proposed order. The comments generally supported the preliminary relief sought by the State, but also requested revisions to the proposed order that would seek additional information and provide additional preliminary relief for the downstream water utilities.

18. On November 21, 2018, the day before Thanksgiving, DEQ announced on its website its proposal to enter into a proposed Consent Order ("PCO") with Chemours and Cape Fear River Watch (an environmental organization that also signed the PCO and that seeks to intervene in this action). *See* https://deq.nc.gov/news/press-releases/2018/11/21/release-state-officials-require-chemours-provide-permanent-drinking. DEQ's announcement states, "The proposed consent order is a comprehensive resolution regarding per- and polyfluoroalkyl substances (PFAS) contamination originating from Chemours' Fayetteville Works facility." The announcement also states that DEQ will accept public comment on the PCO until December 21, 2018.

19. The Cape Fear Public Utility Authority was unaware that the parties to this action had reached a proposed settlement or had agreed to propose a Consent Order until the PCO was published by DEQ on the day before Thanksgiving. CFPUA was not consulted about or notified of the status of the parties' settlement negotiations, the potential terms of a proposed settlement, or the impending publication of the PCO. DEQ did not seek input from CFPUA regarding how the terms of the proposed settlement might (or might not) provide relief to CFPUA and its customers.

20. Paragraph 12 of the PCO was targeted toward reducing PFAS loading to the Cape Fear River, which would theoretically reduce the PFAS entering CFPUA's raw water intake.

However, the PCO allowed for a five year implementation period with limited interim reductions. The PCO also included requirements that seek to reduce future discharges of PFAS pollutants from the Chemours Facility and to prevent current and future consumption of contaminated groundwater by citizens who live around the Facility and obtain potable water from water supply wells in the vicinity of the Facility. But the PCO did not include requirements to prevent the current and ongoing use or consumption of contaminated Cape Fear River water by downstream citizens and other users (including CFPUA) – even though the State acknowledges this harm, acknowledges CFPUA's current inability to remove these pollutants from Cape Fear River water, and requests relief for this harm in the State's complaints in this action.

21. On December 20, 2018, CFPUA filed a Motion to Intervene and calendared it for hearing on January 14, 2019, in light of the deficiencies in the proposed Consent Order. Following discussions with DEQ regarding the terms of the PCO, CFPUA agreed to remove its Motion to Intervene from the calendar but not withdraw the motion itself to allow the parties time to consider further improvements to the PCO. *See* Tr. of Hrg. on Mot. for Entry of Consent Order ("Hrg. Tr.") at 31.

22. Then, on February 20, 2019, counsel for DEQ notified counsel for CFPUA that DEQ, Chemours and the River Watch had agreed upon revised the terms of the PCO and had filed a motion for entry of a proposed Revised Consent Order ("Revised PCO" or "Consent Order"), to be heard five days later. CFPUA had not previously seen or been notified of the revised terms, nor was there time for CFPUA to advise the Board on the revised terms of the PCO being proposed or get board approval or disapproval to pursue its Motion to Intervene and so advised the Court. *See* Hrg. Tr. At 30–31. The Court did not rule on CFPUA's motion at the February 25 Hearing.

23. An improvement over the prior version, the Revised PCO provided for more protections to downstream users, such as interim benchmarks in the reduction of PFAS loading to the river. However, the Revised PCO still had the same fundamental deficiencies described above—it left CFPUA customers exposed to PFAS in their drinking water for years, while ensuring clean water for the citizens of Bladen County.

Deficiencies in the Revised Consent Order

24. One of the most significant aspects of the Revised Consent Order is the requirement for replacement water supplies, set forth in Section F. For fourteen PFAS identified on Attachment C, the Revised Consent Order established drinking water standards of 10 parts per trillion (ppt) for any individual PFAS, and 70 ppt for combined PFAS levels (the "Bladen County Limit"). Revised Consent Order ¶ 20. For persons whose water is contaminated in excess of the Bladen County Limit, Chemours is obligated to provide interim replacement water within three days of being notified, and permanent reverse osmosis systems within six months. Revised Consent Order ¶¶ 20 and 23.

25. Inexplicably, the Bladen County Limit only applies to groundwater users. The result is that Bladen County residents whose groundwater exceeds the Bladen County Limit standard receive near-immediate relief. Conversely, CFPUA and its customers, whose raw and finished water regularly exceed the Bladen County Limit standard, must wait years for clean water. This unequal treatment of North Carolina citizens that have suffered similar harm because of the actions and inactions of Chemours and DuPont is still unexplained and arbitrary and capricious.

26. The Revised Consent Order is based on a flawed premise. As justification for entry of the Revised PCO, DEQ and Chemours both assured this Court that the implementation of the provisions in the Revised PCO had reduced and would continue to reduce downstream PFAS

levels in the Cape Fear River. For instance, counsel for DEQ asserted that "[a]s a result of DEQ requiring cessation of the discharge of process wastewater, there were dramatic reductions in the concentrations of GenX in Chemours' discharge," and "similar reductions" in CFPUA's finished water. Hrg. Tr. at 8. Similarly, counsel for Chemours opined that the cessation of its PFAS-laden wastewater discharges "resulted in truly dramatic reductions in the levels of GenX in the river." Hrg. Tr. at 23. DEQ further emphasized to the Court that Paragraph 12 of the Revised PCO requires Chemours to demonstrate a plan to achieve the maximum feasible reduction in PFAS loading from the facility to the Cape Fear River, and was "of central importance for downstream communities." Hrg. Tr. at 14.

27. In other words, by turning off the PFAS spigot into the Cape Fear River that was Chemours' process wastewater in the first instance, and by requiring Chemours to study and then address PFAS loading from its facility to the Cape Fear River thereafter, DEQ theorized that PFAS levels in the river had dropped and would continue to drop in the immediate near term as it had in the prior 6 months..

28. The reality has not matched the representations made to the Court. As demonstrated by the continued monitoring of PFAS over the past 18 months, PFAS levels in the Cape Fear River have been variable and are largely dependent on river flows. PFAS in groundwater, surface water runoff, and sediment continues to migrate into the river from and around the Facility and from accumulated sediment in the Cape Fear River bed due to decades of contamination.

29. Accordingly, in the months preceding the February 2019 hearing, high river flows were largely responsible for the "dramatic reductions" in PFAS concentrations presented to the Court, rather than merely a matter of Chemours having halted its process wastewater discharges.

Following the hearing, PFAS levels in the Cape Fear River later increased significantly due to drier weather, rather than continuing their decline as was represented to the Court.

30. Chemours and DEQ both theorize that migration of groundwater from the Chemours Facility to the Cape Fear River is the most significant source of PFAS contamination in the river, which Chemours has yet to resolve. Am. Compl. ¶ 126. It is therefore no surprise that, of the 58 raw water sampling events since the hearing on the Revised PCO, 47 exceeded the Bladen County Limit. Of the 44 finished water samples, 32 exceeded this standard.

31. Further, the relief to CFPUA offered by the Revised Consent Order will not be realized for years, unlike the relief provided to Bladen County residents. The Order allows Chemours five years to implement a plan to reduce PFAS loading to the Cape Fear River from groundwater at the Facility. Consent Order ¶ 12.a.

32. As required by the Consent Order, Chemours submitted to DEQ a Cape Fear River PFAS Loading Reduction Plan on August 26, 2019, and the related Corrective Action Plan ("CAP") on December 31, 2019, detailing its proposals to remediate the groundwater at the Facility and reduce PFAS loading to the river. Under Chemours' own estimates (which CFPUA's consultant Tetra Tech has opined is not scientifically supported (*see* Exhibit D and Exhibit E, attached)), it will take through 2022 for them to control just 43% of the PFAS loading from their facility to the Cape Fear River. By the end of 2024, Chemours estimates it will have controlled just 79% of the current PFAS releases from its Facility to the river. The full extent of Chemours' proposed remedial actions are expected to take between 5 and 10 years, if not longer. All the while, the water of the Cape Fear River at CFPUA's intake regularly exceeds the Bladen County Limit.

33. Conversely, the Revised Consent Order requires that Chemours provide temporary replacement water supplies to the citizens of Bladen County within 3 days of becoming aware that an affected user's groundwater exceeds the Bladen County Limit, and a permanent replacement within 6 months. Consent Order ¶¶ 20 and 23.

34. Finally, the Revised Consent Order and Chemours' Loading Reduction Plan and CAP fall short of assuring adequate relief to CFPUA. As an initial matter, even assuming Chemours can meet its projections, its remedial actions would reduce PFAS loading from its Facility by just 79%. But Chemours itself acknowledges that its proposed long-term groundwater remedy is "still highly conceptual," and that "it is not presently possible to conclude with confidence whether this alternative is economically feasible." *See* CAP at 71, 74. Moreover, those plans do nothing to address the extensive soil, groundwater, and sediment contamination in the larger area surrounding the Facility and in the riverbed, which will continue releasing PFAS to the Cape Fear River for decades. Therefore, the PFAS Loading Reduction Plan and CAP represent a future and possible solution for the downstream water utilities.

35. As such, CFPUA and its customers will continue being subjected to river water contaminated with PFAS. And given the limits of the remediation proposed by Chemours, there is no assurance that even after its completion the water of the Cape Fear River will meet the Bladen County Limit. The only way to assure that CFPUA's finished water will meet that standard is to build a treatment system designed to remove PFAS, as CFPUA is doing.

36. Based on the evidence in this action and the studies arising from implementation of the Consent Order and Revised Consent Order, the State is aware that: (a) PFAS pollutants exist in the surface water in the Cape Fear River; (b) even if the Chemours Facility immediately ceases all emissions and discharges of PFAS pollutants into the Cape Fear River, those pollutants will

continue to contaminate the surface water in the Cape Fear River for decades to come (since pollutants in the vegetation, soils, and groundwater in a large and unknown radius around the Chemours Facility and in riverbed sediments will continue to migrate into the river water through groundwater flow and surface run-off); (c) Cape Fear River water is being used downstream from the Chemours Facility by CFPUA, customers of CFPUA, and other citizens; and (d) downstream utilities like CFPUA do not have the ability to consistently remove these pollutants from the drinking water supplied to their customers. Yet the State has left CFPUA to its own devices in dealing with the PFAS contamination in the river.

The Proposed Addendum Does Not Ensure Relief to CFPUA

37. Since the Revised Consent Order was entered, CFPUA has continued to share its monitoring results and concerns with DEQ, including its data showing that the high variability of PFAS concentrations in the Cape Fear River over time are largely dependent on the volume of flow. Chemours has conducted site assessments which indicate that the majority of PFAS loading from the Chemours Facility into the Cape Fear River is due to groundwater contamination. Chemours itself also calculated that it has been and is the primary contributor to PFAS in the Cape Fear River, estimating that Facility has been responsible for between 68% and 84% of PFAS concentrations at CFPUA's water intake in the river.

38. In its discussions with CFPUA, DEQ recognized that the Revised Consent Order in its current form is deficient. DEQ and Chemours now proposed additional modifications, releasing a draft Addendum to the Revised Consent Order for public comment on August 13, 2020 (the "proposed Addendum"). However, consistent with its past practice, DEQ again did not consult with CFPUA on the terms of the proposed Addendum, instead notifying CFPUA of its existence just hours before its public release.

39. The proposed Addendum requires concrete remediation directed toward limiting groundwater migration from the Chemours Facility into the Cape Fear River. Yet, as it relates to CFPUA and other downstream water users, the proposed Addendum still suffers from exactly the same major deficiency as the Revised Consent Order: There is still no assurance, or even reasonable expectation, that PFAS levels in the Cape Fear River will ever consistently reach the Bladen County Limit, and there is still no immediate relief for CFPUA or its customers. CFPUA is left in the same position as it was before the proposed Addendum: wait an indeterminate number of years for an indeterminate level of relief from an indeterminate number of PFAS compounds.

40. As part of the Addendum requirements and as described in the proposed NPDES Permit # NC 0089915 that was sent to public notice on July 10, 2020 ("proposed Permit"), DEQ is requiring Chemours to analyze for 59 distinct PFAS compounds found in Chemours' proposed wastewater discharge (the "Full Suite = Table 3+ Lab SOP +Method 537 Compounds"). This proposed Permit only covers stormwater and groundwater seepage in old outfall 2, and does not govern the main Chemours wastewater treatment plant discharge that is currently being captured and shipped offsite for disposal (which will be covered by a separate, subsequent permit). CFPUA has filed a written objection to DEQ's failure to place any controls over the remaining 56 PFAS compounds and the fact that the proposed Permit would allow a maximum daily discharge of the three regulated compounds (GenX, PFMOAA and PMPA) of 964 ppt.

41. In addition, in its June 30, 2020, PFAS Non-Targeted Analysis and Methods Interim Report on Process and Non-Process Wastewater and Stormwater, Chemours identified 21 new and previously unknown PFAS compounds in its "General Facility Discharge" and 250 new and unknown PFAS compounds in its "Process Wastewater."

42. The Secretaries' Science Advisory Board ("SAB") continues to review studies related to the appropriate health and regulatory standards for GenX and other PFAS compounds. At its August 31, 2020 meeting, the SAB reviewed studies by two groups of scientists (Exhibits F and G) on the probable toxicological impacts of GenX (and PFOA) on mice and rats and studies by another group of scientists (Exhibit H) on the probable toxicological impacts of BFESA-BP2 (Nafion Byproduct 2) on mice. Both of these PFAS compounds are found in significant amounts in Chemours' discharges and releases. And these are but two of the hundreds of PFAS compounds now identified as emanating from the Chemours Facility and likely entrained in the sediments of the riverbed. What health and environmental impacts these compounds will have individually and synergistically is going to take decades to determine.

43. Based on the evidence in this action and the studies undertaken since implementation of the Revised Consent Order, the State is aware that: (a) hundreds of PFAS pollutants exist in the surface water in the Cape Fear River; (b) even if the Chemours Facility immediately ceases all emissions and discharges of PFAS pollutants into the Cape Fear River and completes the remediation improvements now promised, those pollutants will continue to contaminate the surface water in the Cape Fear River for decades to come (because PFAS pollutants in the vegetation, soils, and groundwater in a large and unknown radius around the Chemours Facility will continue to migrate into the river water through groundwater flow and surface run-off and PFAS pollutants entrained in the Cape Fear River sediment will continue to be released into the river water); (c) Cape Fear River water is being used downstream from the Chemours Facility by CFPUA, customers of CFPUA, and other citizens; and (d) downstream utilities like CFPUA do not have the ability to consistently remove PFAS pollutants from the drinking water supplied to their customers without the construction of additional treatment

systems, such as the GAC system currently being constructed by CFPUA. Yet again, the State has left CFPUA to its own devices in dealing with current and reasonably foreseeable PFAS contamination in the river.

Mandatory abatement of violations under N.C. Gen. Stat. § 143-215.6C

44. As alleged in the State's Amended Complaint, the past and ongoing unpermitted discharges and releases of PFAS by Chemours violate the State laws implementing the Clean Water Act. Am. Compl. ¶¶ 145–164.

45. The State further alleged that North Carolina has the authority to take enforcement action against violations of the Clean Water Act and the implementing State laws, which prohibit the discharge of unpermitted pollutants. Am. Compl. ¶ 14.

46. Water from the Cape Fear River is withdrawn by CFPUA and treated in its treatment plant, and the treated water is then distributed to its customers for drinking and other public uses. The relevant stream segment of the Cape Fear River from which the water is withdrawn by CPFUA is classified WS-IV CA.

47. One State water quality standard applicable to all fresh surface waters is: "Oils, deleterious substances, colored, or other wastes: *only* such amounts as *shall not* render the waters injurious to public health, secondary recreation, or to aquatic life and wildlife, or adversely affect the palatability of fish, aesthetic quality, or impair the waters for any designated uses." 15A NCAC 2B .0211(12) (italics added). One designated use of class WS-IV surface water segments is "a source of water supply for drinking." 15A NCAC 2B .0216(1). The PFAS pollutants discharged and released into the Cape Fear River by Chemours and its predecessor: (a) are deleterious substances within the meaning of this water quality standard; (b) are present in the Cape Fear River

in amounts that render the Cape Fear River waters injurious to public health; and (c) are present in the Cape Fear River in amounts that impair the Cape Fear River waters for its designated use.

48. Under North Carolina's water quality laws implementing the Clean Water Act, DEQ is authorized to institute a civil action for injunctive relief to restrain and abate violations of the applicable water quality laws. N.C. Gen. Stat. § 143-215.6C. Upon a determination by the Court that an alleged violation "has occurred or is threatened, the court shall grant the relief necessary to prevent or abate the violation." *Id.* (emphasis added); Am. Compl. ¶ 46.

49. DEQ expressly brought the Amended Complaint under, *inter alia*, N.C. Gen. Stat. § 143-215.6C.

FIRST CLAIM FOR RELIEF
(Declaratory Judgment-Consent Order is Arbitrary and Capricious)

50. The allegations set forth in the preceding paragraphs are realleged and incorporated by reference.

51. Pursuant to the Declaratory Judgments Act, N.C. Gen. Stat. § 1-253 *et seq.*, and for the reasons stated above, CFPUA seeks an order declaring that the State's decision to settle this enforcement action on the terms stated in the Addendum to the Revised Consent Order is arbitrary and capricious and an abuse of discretion under the North Carolina Administrative Procedure Act.

52. *First*, the proposed Addendum to the Revised Consent Order fails to provide effective remedial requirements for off-site PFAS contamination in the Cape Fear River, river sediment, and air depositions in the soil and groundwater, which will continue to impact the waters of the Cape Fear River and the downstream users of the Cape Fear River for decades into the future. Instead, the State has left CFPUA and other downstream users to the uncertainties and expense of private litigation, to vindicate their rights on their own, and has thereby abandoned its

obligations to enforce the State's environmental laws (including the State's water quality standards) on behalf of all citizens of the State.

53. *Second*, the Addendum to the Revised Consent Order implicitly continues the established drinking water remedial requirements (to the Bladen County Limit) for residents in the vicinity of the Fayetteville Works Facility whose groundwater is impacted by PFAS, but does not establish the same requirements for everyone downstream whose drinking water is also impacted by the same PFAS contaminants. The State's decision to resolve this enforcement action in a manner that mandates unequal treatment of North Carolina citizens is arbitrary and capricious, irrational, and an abuse of discretion.

54. An actual controversy exists based on the State's decision not to fully address the immediate and continuing harms to CFPUA and its customers.

55. CFPUA has no adequate or effective administrative remedy against the State or its agency DEQ. The subject of this Complaint is the underlying historic and ongoing releases of PFAS by Chemours, the public health and environmental harms caused by those releases, and the State's efforts to seek relief for the violations of North Carolina water quality laws in this enforcement action. Jurisdiction to consider and determine the outcome of this action lies in Bladen County Superior Court, over which the Office of Administrative Hearings ("OAH") has no authority. Accordingly, there is no adequate administrative remedy available to CFPUA, an administrative claim in OAH would be futile, and this Court has jurisdiction to determine this action.

56. CFPUA seeks an order declaring that the State's decision to resolve this enforcement action pursuant to the terms of the Addendum to the Revised Consent Order is arbitrary and capricious, irrational, and an abuse of discretion under the North Carolina

Administrative Procedure Act since it (a) does not assure that the existing harm to downstream Cape Fear River water users is abated and (b) implicitly establishes differing and irrational levels of PFAS contamination that are safe for human consumption and use depending on whether a user's exposure to PFAS contaminants arises from use of surface water or groundwater.

SECOND CLAIM FOR RELIEF
(Declaratory Judgment–Equal Protection Violation)

57. The allegations set forth in the preceding paragraphs are realleged and incorporated by reference.

58. The Revised Consent Order and the Addendum thereto implicitly establishes two different sets of drinking water safety levels – one set (the Bladen County Limit) for residents in the vicinity of the Fayetteville Works Facility whose groundwater is impacted by PFAS, and a different set with higher or no levels for everyone downstream whose water is also impacted by PFAS, including CFPUA and its customers.

59. With regard to the safety of their drinking water supply, CFPUA and its customers are similarly situated to residents in the vicinity of the Fayetteville Works Facility who rely on potable water from water supply wells that are contaminated with PFAS, in that: (a) both groups of residents reside in the area of PFAS impact from the Fayetteville Works Facility; (b) both groups of residents rely on drinking water supplies contaminated with PFAS; (c) the drinking water used by both groups of residents has been contaminated by PFAS discharges and releases from the same Facility; and (d) without relief, the drinking water of both groups of residents will continue to be contaminated with PFAS for decades into the future.

60. While the Revised Consent Order requires Chemours to remediate or replace the water supply of nearby residents whose groundwater is contaminated with certain PFAS compounds above the Bladen County Limit, the Addendum and the Revised Consent Order

include no similar requirement for downstream users whose water supply is also contaminated with the same PFAS compounds from the same Facility.

61. The Revised Consent Order's (continued with the Addendum) disparate treatment of North Carolinians exposed to PFAS-contaminated drinking water supplies constitutes discrimination in that the Consent Order's protections do not apply equally to all similarly situated persons, do not reflect a rational distinction between such persons, and therefore, violate equal protection as guaranteed by the Equal Protection Clause of Article I, Section 19 of the North Carolina Constitution and the Equal Protection Clause of Section 1 of the Fourteenth Amendment to the United States Constitution.

62. Upon information and belief, the Revised Consent Order's (continued with the Addendum) distinctions between nearby and downstream groups of residents are not related to a legitimate purpose.

63. CFPUA seeks a judgment declaring that the Addendum and the Revised Consent Order constitute a violation of the United States and North Carolina Constitutions.

THIRD CLAIM FOR RELIEF
(Declaratory Judgment-Abatement of Violation)

64. The allegations set forth in the preceding paragraphs are realleged and incorporated by reference.

65. Under North Carolina's statutes and rules implementing the Clean Water Act, DEQ is authorized by N.C. Gen. Stat. § 143-215.6C to request the Attorney General to institute a civil action for injunctive relief to restrain and abate a violation of the State's water quality laws. Pursuant to this statute, the Attorney General instituted this enforcement action on behalf of the State. Upon a determination by the Court that the alleged violation "has occurred or is threatened,

the court shall grant the relief necessary to prevent or abate the violation." N.C. Gen. Stat. § 143-215.6C (emphasis added); Am. Compl. ¶ 46.

66. The Amended Complaint expressly seeks to enforce, and requests relief pursuant to, N.C. Gen. Stat. § 143-215.6C.

67. Although the Amended Complaint and the terms of the Consent Order are premised on violations of North Carolina's water quality laws by Chemours, which resulted in widespread PFAS contamination in the Cape Fear River, the Consent Order does not prevent or abate the violation. In particular, the Consent Order fails to provide effective relief for off-site PFAS contamination in the Cape Fear River, river sediment, air depositions, and possible future surface water discharges which will continue to impact the waters of the Cape Fear River and the downstream users of the Cape Fear River for decades into the future.

68. An actual controversy exists based on the State's failure to seek effective abatement of the violations of Chemours. As a result, the waters of the Cape Fear River will continue to be impacted by PFAS historically released by Chemours, in violation of North Carolina water quality laws, which will reach CFPUA's intake within the river and affect the quality of CFPUA's finished water, and thereby cause current and future harm to CFPUA and its customers.

69. The State's Amended Complaint alleges the basis for the Court's jurisdiction under N.C. Gen. Stat. § 143-215.6C, and the record shows that the facts alleged by the State will be proved by the evidence that will be presented in this case. However, the State's decision to seek to settle this enforcement action on the basis of the Revised Consent Order (continued with the Addendum) irrationally and arbitrarily fails or refuses to seek the "relief necessary to prevent or abate the violation[s]" alleged in the Amended Complaint. The Revised Consent Order (continued with the Addendum) irrationally and arbitrarily and without justification precludes the Court from

entering the "relief necessary" as required by the enforcement statute under which this action was instituted.

70. Pursuant to the Declaratory Judgments Act, N.C. Gen. Stat. § 1-253 *et seq.*, and for the reasons stated above, CFPUA seeks an order of the Court declaring that: (a) the statutory and regulatory violations alleged by the State in this action have occurred or are threatened; and (b) the Revised Consent Order (continued with the Addendum) fails to meet the mandate of N.C. Gen. Stat. § 143-215.6C, to prevent or abate the violations of North Carolina's water quality laws and rules by Chemours; and (c) the State's decision to agree to the Revised Consent Order (continued with the Addendum) is irrational, arbitrary, and unsupported by the record in this case because the Revised Consent Order (continued with the Addendum) does not seek or accomplish the "relief necessary to prevent or abate the violation" and, if allowed by the Court as agreed-to by the State, would prevent the grant of the "relief necessary" as required by N.C. Gen. Stat. § 143-215.6C.

PRAYER FOR RELIEF

WHEREFORE, Intervernor CFPUA respectfully prays the Court for the following relief:

1. A judicial declaration, pursuant to N.C. Gen. Stat. § 1-253 *et seq*., that the State's decision to agree to the Revised Consent Order (continued with Addendum) was arbitrary and capricious;

2. A judicial declaration, pursuant to N.C. Gen. Stat. § 1-253 *et seq.*, that the Revised Consent Order (continued with Addendum) violates the Equal Protection Clause of Article I, Section 19 of the North Carolina Constitution and the Equal Protection Clause of Section 1 of the Fourteenth Amendment to the United States Constitution to the extent it arbitrarily and irrationally

treats similarly situated citizens differently for purposes of addressing and abating PFAS discharges or releases to drinking water;

3. A judicial declaration and determination, pursuant to N.C. Gen. Stat. § 1-253 *et seq*., that: (a) the statutory and regulatory violations alleged by the State in this action have occurred or are threatened; and (b) the Revised Consent Order (continued with the Addendum) fails to meet the mandate of N.C. Gen. Stat. § 143-215.6C, to prevent and abate the violations of North Carolina's water quality laws and rules by Chemours; and (c) the State's decision to agree to the Revised Consent Order (continued with the Addendum) is irrational, arbitrary, and unsupported by the record in this case because the Revised Consent Order (continued with the Addendum) does not seek or accomplish the "relief necessary to prevent or abate the violation" and, if allowed by the Court as agreed-to by the State, would prevent the grant of the "relief necessary" as required by N.C. Gen. Stat. § 143-215.6C.

4. An order, following the trial of this case and pursuant to N.C. Gen. Stat. § 143-215.6C, granting the relief necessary to prevent and abate Chemours' violations of the water quality laws of this State;

6. Such other and further relief as to the Court may seem just and proper.

Respectfully submitted this the _____ day of _____, 2019.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT A
TO AMENDED INTERVENOR COMPLAINT

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	9/11/2018	ND	ND	ND	17.5	ND	ND	ND	ND	ND	ND	ND	12.8	ND	ND	ND	ND	67.7	167
Sweeney Raw	9/12/2018	ND	ND	ND	16.9	ND	ND	ND	ND	ND	ND	ND	1.7	ND	ND	ND	ND	3.41	10.9
Sweeney Raw	9/14/2018	ND	ND	ND	15.5	ND	ND	ND	ND	ND	ND	ND	3.29	ND	ND	ND	ND	14.6	43.4
Sweeney Raw	9/15/2018	ND	ND	ND	18.8	ND	ND	ND	ND	ND	ND	ND	1.85	ND	ND	ND	ND	5.84	22.4
Sweeney Raw	9/16/2018	ND	ND	ND	15.2	ND	ND	ND	ND	ND	ND	ND	1.97	ND	ND	ND	ND	8.67	28
Sweeney Raw	9/17/2018	ND	ND	ND	33.8	ND	ND	ND	ND	ND	ND	1.33	2.35	ND	ND	ND	ND	3.19	7.35
Sweeney Raw	9/18/2018	ND	ND	ND	18.6	ND	ND	ND	ND	ND	ND	ND	1.34	ND	ND	ND	ND	2.32	5.66
Sweeney Raw	9/19/2018	ND	ND	ND	17.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.77	4.43
Sweeney Finished	9/19/2018	ND	ND	ND	19.8	ND	ND	ND	ND	ND	ND	ND	9.51	ND	ND	ND	ND	51.9	120
Sweeney Raw	9/20/2018	ND	ND	ND	18.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.16	5.14
Sweeney Raw	9/21/2018	ND	ND	ND	12.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.67	5.28
Sweeney Raw	9/22/2018	ND	ND	ND	8.44	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.09	5.9
Sweeney Raw	9/23/2018	ND	ND	ND	5.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.96	3.22
Sweeney Raw	9/24/2018	ND	ND	ND	6.32	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.88
Sweeney Finished	9/24/2018	ND	ND	ND	7.34	ND	ND	ND	ND	ND	ND	ND	6.8	ND	ND	ND	ND	37.9	96.4
Sweeney Raw	9/25/2018	ND	ND	ND	8.54	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.25	3.63
Sweeney Raw	9/26/2018	ND	ND	ND	15.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.66	7.87
Sweeney Raw	9/27/2018	ND	ND	ND	17.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.05	5.14
Sweeney Raw	9/28/2018	ND	ND	ND	27.7	ND	ND	ND	ND	ND	ND	ND	1.68	ND	ND	ND	ND	3.95	9.97
Sweeney Raw	9/29/2018	ND	ND	ND	25.1	ND	ND	ND	ND	ND	ND	ND	1.4	ND	ND	ND	ND	3.46	8.66
Sweeney Raw	9/30/2018	ND	ND	ND	12.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.69	4.6
Sweeney Raw	10/1/2018	ND	ND	ND	10.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.2	4.37
Sweeney Finished	10/1/2018	ND	ND	ND	16.9	ND	ND	ND	ND	ND	ND	ND	10.7	ND	ND	ND	ND	56.1	125
Sweeney Raw	10/2/2018	ND	ND	ND	9.79	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.74	ND
Sweeney Raw	10/3/2018	ND	ND	ND	11.7	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.3	3.66
Sweeney Raw	10/4/2018	ND	ND	ND	10.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.5
Sweeney Raw	10/5/2018	ND	ND	ND	11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.2	3.77
Sweeney Raw	10/6/2018	ND	ND	ND	10.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.56	4.74
Sweeney Raw	10/7/2018	ND	ND	ND	10.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.22	4.63

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	9/11/2018	126	139	9.5	3.76	5.25	11.9	ND	2.53	ND	ND	16.3	ND	6.95	23.3	ND	2.85		ND	20.1	12.2
Sweeney Raw	9/12/2018	13.4	14.3	4.47	7.29	5.44	10.7	ND	1.43	ND	ND	12.5	ND	7.55	22.3	ND	2.28		ND	17.8	9.08
Sweeney Raw	9/14/2018	43.9	51.5	ND	2.83	4.98	11.7	ND	1.57	ND	ND	13.6	ND	7.05	22.9	ND	2.18		ND	17.7	10.5
Sweeney Raw	9/15/2018	25.5	26.3	5.14	5.37	4.77	12.2	ND	1.7	ND	ND	17.9	ND	6.43	28.9	ND	2.53		ND	16.2	12.1
Sweeney Raw	9/16/2018	28.1	33.4	ND	9.7	4.37	9.04	ND	1.44	ND	ND	15.1	ND	5.63	23.5	ND	1.64		ND	13.5	8.81
Sweeney Raw	9/17/2018	11.2	13.9	7.61	13.8	2.54	6.59	ND	1.07	ND	ND	9.27	ND	3.71	14.2	ND	0.831		ND	9.72	5.54
Sweeney Raw	9/18/2018	9.06	10.6	ND	ND	1.96	5.8	ND	0.683	ND	ND	6.48	ND	2.87	9.8	ND	ND		ND	7.83	4.41
Sweeney Raw	9/19/2018	6.94	9.9	ND	ND	1.52	4.73	ND	ND	ND	ND	2.97	ND	2.59	5.2	ND	0.777		ND	6.45	3.35
Sweeney Finished	9/19/2018	93.9	104	11.8	7.62	2.66	6.51	ND	2.23	ND	ND	8.68	ND	4	12.4	ND	1.99		ND	13.4	6.43
Sweeney Raw	9/20/2018	8.44	10.2	ND	ND	1.69	5.1	ND	0.693	ND	ND	2.99	ND	2.62	5.17	ND	0.824		ND	7.03	3.34
Sweeney Raw	9/21/2018	7.05	10.7	10.2	ND	1.99	5.08	ND	0.736	ND	ND	3	ND	3.27	5.14	ND	0.999		ND	7.36	3.2
Sweeney Raw	9/22/2018	6.89	6.82	ND	ND	1.5	4.86	ND	ND	ND	ND	2.48	ND	1.66	3.83	ND	0.973		ND	7.97	3.23
Sweeney Raw	9/23/2018	4.59	5.73	ND	ND	1.43	4.12	ND	ND	ND	ND	1.73	ND	2.16	2.49	ND	0.996		ND	6.42	3.23
Sweeney Raw	9/24/2018	4.96	4.05	ND	8.71	1.73	5.39	ND	ND	ND	ND	2.06	ND	2.07	4.05	ND	0.914		ND	7.31	3.39
Sweeney Finished	9/24/2018	59.5	69.7	8.63	ND	1.75	4.27	ND	1.68	ND	ND	3.52	ND	3.52	3.53	ND	1.84		ND	10.2	5.43
Sweeney Raw	9/25/2018	6.31	6.7	3.35	ND	1.53	6.79	ND	ND	ND	ND	2.1	ND	1.97	5.32	ND	1.07		ND	7.15	3.38
Sweeney Raw	9/26/2018	13.4	13.7	8.6	24.8	1.79	10.3	ND	ND	ND	ND	3.37	ND	1.97	7.1	ND	1.03		ND	7.47	4.28
Sweeney Raw	9/27/2018	12.6	16	2.12	21	1.75	6.93	ND	ND	ND	ND	2.56	ND	2.36	3.84	ND	0.926		ND	7.59	3.95
Sweeney Raw	9/28/2018	18.9	21.2	12.2	ND	2.43	10.2	ND	ND	ND	ND	3	ND	3.96	5.68	ND	0.744		ND	10.4	6.12
Sweeney Raw	9/29/2018	18.6	17.5	9.19	9.31	2.74	9.95	ND	ND	ND	ND	3.34	ND	4.29	6.33	ND	1.02		ND	10.6	6.13
Sweeney Raw	9/30/2018	9.26	7.53	5.32	14.3	2.64	7.49	ND	0.966	ND	ND	5.36	ND	4.81	8.74	ND	1.4		ND	14.6	6.14
Sweeney Raw	10/1/2018	7.05	8.03	4.82	10.3	2.82	7.35	ND	1.15	ND	ND	7.63	ND	4.34	13.6	ND	1.68		ND	16	7.05
Sweeney Finished	10/1/2018	81.6	92.8	17	5.28	2.93	9.12	ND	1.37	ND	ND	6.31	ND	5.26	9.19	ND	2		ND	15.8	8.51
Sweeney Raw	10/2/2018	4.93	7.02	5.32	17.1	2.85	9.1	ND	1.08	ND	ND	7.95	ND	4.25	13.7	ND	1.39		ND	14.2	7.17
Sweeney Raw	10/3/2018	4.93	7.19	4.81	17.3	2.89	10.1	ND	1.37	ND	ND	9.66	ND	3.89	13	ND	1.55		ND	14.9	6.96
Sweeney Raw	10/4/2018	5.45	6.83	4.15	21.7	3.31	9.97	ND	1.46	ND	ND	9.95	ND	4.25	15.4	ND	1.76		ND	14.7	8.17
Sweeney Raw	10/5/2018	4.23	8.27	4.59	25.3	2.61	10.2	ND	1.33	ND	ND	10	ND	3.75	15.2	ND	1.81		ND	14.8	7.43
Sweeney Raw	10/6/2018	5.29	7.77	4.58	23.6	3.34	10.9	ND	1.59	ND	ND	12.2	ND	3.9	18.6	ND	1.83		ND	16	9.59
Sweeney Raw	10/7/2018	5.63	6.17	3.9	15.9	3.54	12	ND	1.63	ND	ND	14.3	ND	4.52	23.2	ND	2.06		ND	16.8	9.89

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	9/11/2018	1.03	25.9	ND	ND	0.665		ND	**672.24**	**559.56**	**83**
Sweeney Raw	9/12/2018	1.33	27.8	ND	ND	0.58		ND	**191.16**	**84.87**	**44**
Sweeney Raw	9/14/2018	1.1	29	ND	ND	ND		ND	**297.30**	**188.62**	**63**
Sweeney Raw	9/15/2018	1.21	32	ND	ND	ND		ND	**247.14**	**129.10**	**52**
Sweeney Raw	9/16/2018	0.833	24.2	ND	ND	ND		ND	**233.10**	**140.14**	**60**
Sweeney Raw	9/17/2018	0.602	15.3	ND	ND	ND		ND	**163.90**	**103.80**	**63**
Sweeney Raw	9/18/2018	ND	9.85	ND	ND	ND		ND	**97.26**	**54.06**	**56**
Sweeney Raw	9/19/2018	ND	5.48	ND	ND	ND		ND	**74.71**	**44.61**	**60**
Sweeney Finished	9/19/2018	0.632	14	ND	ND	ND		ND	**491.46**	**427.21**	**87**
Sweeney Raw	9/20/2018	ND	5.55	ND	ND	ND		ND	**79.85**	**47.83**	**60**
Sweeney Raw	9/21/2018	ND	5.46	ND	ND	ND		ND	**83.74**	**50.50**	**60**
Sweeney Raw	9/22/2018	ND	4.03	ND	ND	ND		ND	**60.67**	**32.62**	**54**
Sweeney Raw	9/23/2018	ND	3.69	ND	ND	ND		ND	**46.88**	**22.34**	**48**
Sweeney Raw	9/24/2018	ND	4.2	ND	ND	ND		ND	**58.03**	**28.98**	**50**
Sweeney Finished	9/24/2018	ND	5.3	ND	ND	ND		ND	**327.31**	**289.79**	**89**
Sweeney Raw	9/25/2018	ND	8.08	ND	ND	ND		ND	**67.17**	**31.88**	**47**
Sweeney Raw	9/26/2018	ND	16.1	ND	ND	ND		ND	**140.34**	**90.30**	**64**
Sweeney Raw	9/27/2018	ND	7.3	ND	ND	ND		ND	**113.62**	**78.97**	**70**
Sweeney Raw	9/28/2018	0.787	9.15	ND	ND	ND		ND	**148.07**	**98.60**	**67**
Sweeney Raw	9/29/2018	ND	6.82	ND	ND	ND		ND	**144.44**	**96.56**	**67**
Sweeney Raw	9/30/2018	0.936	8.08	ND	ND	ND		ND	**116.26**	**60.46**	**52**
Sweeney Raw	10/1/2018	0.661	9.11	ND	ND	ND		ND	**117.46**	**53.70**	**46**
Sweeney Finished	10/1/2018	0.792	9.2	ND	ND	ND		ND	**475.86**	**411.69**	**87**
Sweeney Raw	10/2/2018	0.657	13.6	ND	ND	ND		ND	**121.85**	**53.85**	**44**
Sweeney Raw	10/3/2018	0.596	13.2	ND	ND	ND		ND	**129.01**	**60.55**	**47**
Sweeney Raw	10/4/2018	0.639	15.4	ND	ND	ND		ND	**137.04**	**61.98**	**45**
Sweeney Raw	10/5/2018	0.637	14.7	ND	ND	ND		ND	**141.83**	**69.36**	**49**
Sweeney Raw	10/6/2018	0.686	18.4	ND	ND	ND		ND	**155.18**	**70.34**	**45**
Sweeney Raw	10/7/2018	0.699	20.5	ND	ND	ND		ND	**156.79**	**61.95**	**40**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Raw	10/8/2018	ND	ND	ND	11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.55	ND
Sweeney Finished	10/8/2018	ND	ND	ND	11.2	ND	ND	ND	ND	ND	ND	ND	9.91	ND	ND	ND	ND	52.3	117
Sweeney Raw	10/9/2018	ND	ND	ND	10.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.83	4.49
Sweeney Raw	10/10/2018	ND	ND	ND	9.94	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.33	ND
Sweeney Raw	10/11/2018	ND	ND	ND	9.58	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.02	4.53
Sweeney Raw	10/12/2018	ND	ND	ND	10.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.29	4.47
Sweeney Finished	10/15/2018	ND	ND	ND	19.4	ND	ND	ND	ND	ND	ND	ND	10	ND	ND	ND	ND	61.3	131
Sweeney Finished	10/23/2018	ND	ND	ND	8.39	ND	ND	ND	ND	2.22	ND	ND	6.14	ND	ND	ND	ND	28.7	77.9
Sweeney Finished	10/31/2018	ND	ND	ND	8.7	ND	ND	ND	ND	ND	ND	ND	4.48	ND	ND	ND	ND	23	48.4
Sweeney Finished	11/5/2018	ND	ND	ND	9.68	ND	ND	ND	ND	ND	ND	ND	4.54	ND	ND	ND	ND	20.6	50.4
Sweeney Finished	11/13/2018	ND	ND	ND	9.30	ND	ND	ND	ND	ND	ND	ND	4.65	ND	ND	ND	ND	28.7	55.8
Sweeney Finished	11/19/2018	ND	ND	ND	5.46	ND	ND	ND	ND	ND	ND	ND	3.06	ND	ND	ND	ND	13.8	28.1
Sweeney Raw	11/20/2018	ND	ND	ND	3.96	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.02
Sweeney Finished	11/21/2018	ND	ND	ND	3.57	ND	ND	ND	ND	ND	ND	ND	3.0	ND	ND	ND	ND	10.5	26.8
Sweeney Raw	11/27/2018	ND	ND	ND	12.0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.59
Sweeney Finished	11/28/2018	ND	ND	ND	8.24	ND	ND	ND	ND	ND	ND	ND	2.9	ND	ND	ND	ND	8.69	21.7
Sweeney Raw	12/3/2018	ND	ND	ND	6.93	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	12/4/2018	ND	ND	ND	4.44	ND	ND	ND	ND	ND	ND	ND	2.0	ND	ND	ND	ND	7.24	19.8
Sweeney Raw	12/10/2018	ND	ND	ND	16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.38
Sweeney Finished	12/11/2018	ND	ND	ND	8.71	ND	ND	ND	ND	ND	ND	ND	2.7	ND	ND	ND	ND	10.2	23.5
Sweeney Raw	12/17/2018	ND	ND	ND	25.6	ND	ND	ND	ND	ND	ND	ND	1.39	ND	ND	ND	ND	1.2	ND
Sweeney Finished	12/18/2018	ND	ND	ND	9.53	ND	ND	ND	ND	ND	ND	ND	1.9	ND	ND	ND	ND	6.19	15.1
Sweeney Raw	12/24/2018	ND	ND	ND	5.46	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	12/26/2018	ND	ND	ND	2.82	ND	ND	ND	ND	ND	ND	ND	1.99	ND	ND	ND	ND	7.35	17.5
Sweeney Raw	12/31/2018	ND	ND	ND	12.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	1/2/2019	ND	ND	ND	4.08	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	10.1	22.4
Sweeney Raw	1/7/2019	ND	ND	ND	6.92	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.87
Sweeney Finished	1/8/2019	ND	ND	ND	3.98	ND	ND	ND	ND	ND	ND	ND	2.2	ND	ND	ND	ND	8.07	18.7
Sweeney Raw	1/14/2019	ND	ND	ND	6.83	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.34

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Raw	10/8/2018	5.23	7.69	4.15	ND	3.77	12.9	ND	1.57	ND	ND	16.4	ND	3.88	25.1	ND	2.13		ND	17.2	8.97
Sweeney Finished	10/8/2018	49.4	81.5	5.28	ND	3.63	13.7	ND	2.22	ND	ND	15.2	ND	5.54	23.5	ND	2.51		ND	17	10.8
Sweeney Raw	10/9/2018	5.57	6.15	4.21	ND	3.67	10.6	ND	1.67	ND	ND	16.8	ND	4.35	26.2	ND	1.86		ND	19.1	10.3
Sweeney Raw	10/10/2018	4.32	5.42	4.01	ND	4.02	9.46	ND	1.68	ND	ND	17.3	ND	4.64	27.3	ND	2.15		ND	18.2	11.6
Sweeney Raw	10/11/2018	4.54	6.27	2.95	ND	3.72	10.4	ND	1.8	ND	ND	16.8	ND	5.0	26	ND	2.14		ND	16.8	10.9
Sweeney Raw	10/12/2018	8.25	9.81	4.87	ND	3.73	10.1	ND	1.92	ND	ND	16.8	ND	4.67	25.4	ND	2.27		ND	18	10.4
Sweeney Finished	10/15/2018	61.3	82.4	10.2	ND	4.85	12.7	ND	2.71	ND	ND	19.3	ND	6.44	27.4	ND	3.34		ND	20.9	14.3
Sweeney Finished	10/23/2018	38.6	47.6	5.35	ND	3.93	10.7	ND	1.94	ND	ND	15.1	ND	5.51	22.8	ND	2.8		ND	19.3	11.6
Sweeney Finished	10/31/2018	25.5	30.7	5.2	ND	4.66	14	ND	2.24	ND	ND	24	ND	6.13	34.6	ND	2.73		ND	18.8	13.7
Sweeney Finished	11/5/2018	27	33.8	6.07	ND	3.8	8.83	ND	2.06	ND	ND	15.4	ND	5.12	21.5	ND	2.39		ND	15.4	11.4
Sweeney Finished	11/13/2018	57.7	70	4.58	ND	3.01	6.24	ND	1.49	ND	ND	8.43	ND	3.92	12.7	ND	1.75		ND	12.8	7.81
Sweeney Finished	11/19/2018	31.9	49.2	ND	ND	1.75	9.33	ND	1.03	ND	ND	4.75	ND	2.23	7.7	ND	1.25		ND	7.7	4.56
Sweeney Raw	11/20/2018	4.65	5.21	ND	ND	1.64	ND	ND	ND	ND	ND	2.55	ND	2.05	3.41	ND	0.89		ND	5.45	3.14
Sweeney Finished	11/21/2018	29.8	36.3	3.0	ND	1.35	ND	ND	0.738	ND	ND	2.69	ND	1.85	3.71	ND	1.12		ND	6.64	3.56
Sweeney Raw	11/27/2018	6.14	12.3	5.48	ND	3.0	3.86	ND	0.711	ND	ND	7.53	ND	3.75	9.46	ND	1.13		ND	12.6	7.5
Sweeney Finished	11/28/2018	32.8	39.8	5.2	ND	2.0	3.52	ND	0.799	ND	ND	5.36	ND	3.0	7.88	ND	1.13		ND	8.22	5.18
Sweeney Raw	12/3/2018	4.21	6.34	ND	ND	2.8	5.0	ND	0.854	ND	ND	10.7	ND	3.01	12.9	ND	1.16		ND	12.8	7.36
Sweeney Finished	12/4/2018	18.2	24.5	2.1	ND	1.67	4.11	ND	ND	ND	ND	6.01	ND	2.23	9.16	ND	0.858		ND	7.24	4.94
Sweeney Raw	12/10/2018	11.7	15.3	ND	13.6	2.54	5.36	ND	0.807	ND	ND	8.88	ND	3.19	11.6	ND	1.41		ND	9.44	6.41
Sweeney Finished	12/11/2018	23.3	35.5	7.17	ND	1.7	3.79	ND	0.657	ND	ND	6.23	ND	1.99	9.02	ND	1.22		ND	6.4	4.54
Sweeney Raw	12/17/2018	9.33	15.3	9.99	ND	1.73	4.09	ND	ND	ND	ND	3.17	ND	2.81	4.79	ND	0.749		ND	6.67	4.01
Sweeney Finished	12/18/2018	17.9	24	5.2	ND	1.07	ND	ND	ND	ND	ND	2.09	ND	1.75	3.33	ND	0.617		ND	4.57	2.69
Sweeney Raw	12/24/2018	3.09	5.98	ND	ND	2.29	ND	ND	ND	ND	ND	3.87	ND	3.85	5.38	ND	0.91		ND	11.7	5.73
Sweeney Finished	12/26/2018	22	22.7	2.62	ND	0.717	1.31	ND	ND	ND	ND	1.44	ND	1.35	2.42	ND	ND		ND	3.96	2.08
Sweeney Raw	12/31/2018	6.3	8.87	ND	ND	2.65	3.97	ND	0.736	ND	ND	6.21	ND	3.45	8.56	ND	1.09		ND	13	7.09
Sweeney Finished	1/2/2019	28.9	31.7	8.34	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	5.01	ND	ND		ND	6.47	3.37
Sweeney Raw	1/7/2019	5.58	6.33	3.38	ND	2.55	4.62	ND	ND	ND	ND	7.08	ND	3.73	9.83	ND	0.826		ND	12.6	7.05
Sweeney Finished	1/8/2019	28.8	28.3	3.7	1.23	1.31	3.38	ND	ND	ND	ND	3.93	ND	1.77	5.25	ND	0.962		ND	7.98	4.61
Sweeney Raw	1/14/2019	5.59	8.19	ND	ND	2.64	6.53	ND	ND	ND	ND	7.58	ND	3.71	10.8	ND	1.23		ND	11.8	6.78

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Raw	10/8/2018	0.703	22.1	ND	ND	ND		ND	**144.34**	**46.02**	**32**
Sweeney Finished	10/8/2018	0.902	20.6	ND	ND	ND		ND	**442.19**	**341.79**	**77**
Sweeney Raw	10/9/2018	0.929	24.8	ND	ND	ND		ND	**152.73**	**49.25**	**32**
Sweeney Raw	10/10/2018	0.875	25	ND	ND	ND		ND	**147.25**	**42.32**	**29**
Sweeney Raw	10/11/2018	0.814	22.7	ND	ND	ND		ND	**146.96**	**46.69**	**32**
Sweeney Raw	10/12/2018	0.818	20.5	ND	ND	ND		ND	**155.10**	**57.29**	**37**
Sweeney Finished	10/15/2018	1.09	26.9	ND	ND	ND		ND	**515.53**	**394.90**	**77**
Sweeney Finished	10/23/2018	0.867	20.8	ND	ND	0.679		ND	**330.93**	**227.78**	**69**
Sweeney Finished	10/31/2018	0.919	31.4	ND	ND	ND		ND	**299.16**	**169.98**	**57**
Sweeney Finished	11/5/2018	0.893	21.1	ND	ND	ND		ND	**259.98**	**167.49**	**64**
Sweeney Finished	11/13/2018	0.669	13.5	ND	ND	ND		ND	**303.05**	**239.16**	**79**
Sweeney Finished	11/19/2018	ND	7.97	ND	ND	ND		ND	**179.79**	**136.27**	**76**
Sweeney Raw	11/20/2018	ND	3.98	ND	ND	ND		ND	**39.95**	**19.39**	**49**
Sweeney Finished	11/21/2018	ND	4.7	ND	ND	ND		ND	**139.32**	**115.65**	**83**
Sweeney Raw	11/27/2018	0.696	8.55	ND	ND	ND		ND	**97.30**	**46.04**	**47**
Sweeney Finished	11/28/2018	ND	6.48	ND	ND	ND		ND	**162.84**	**124.67**	**77**
Sweeney Raw	12/3/2018	0.613	11.2	ND	ND	ND		ND	**85.85**	**28.18**	**33**
Sweeney Finished	12/4/2018	ND	7.55	ND	ND	ND		ND	**121.99**	**84.23**	**69**
Sweeney Raw	12/10/2018	ND	12.3	ND	ND	ND		ND	**122.92**	**69.86**	**57**
Sweeney Finished	12/11/2018	ND	8.53	ND	ND	ND		ND	**155.16**	**117.31**	**76**
Sweeney Raw	12/17/2018	ND	4.94	ND	ND	ND		ND	**95.77**	**65.98**	**69**
Sweeney Finished	12/18/2018	ND	3.46	ND	ND	ND		ND	**99.40**	**81.91**	**82**
Sweeney Raw	12/24/2018	0.646	6.13	ND	ND	ND		ND	**55.04**	**18.40**	**33**
Sweeney Finished	12/26/2018	ND	2.06	ND	ND	ND		ND	**92.32**	**78.42**	**85**
Sweeney Raw	12/31/2018	0.632	7.83	ND	ND	ND		ND	**83.19**	**34.18**	**41**
Sweeney Finished	1/2/2019	ND	4.06	ND	ND	ND		ND	**124.43**	**105.52**	**85**
Sweeney Raw	1/7/2019	ND	8.38	ND	ND	ND		ND	**80.75**	**31.16**	**39**
Sweeney Finished	1/8/2019	ND	4.99	ND	ND	ND		ND	**129.16**	**98.91**	**77**
Sweeney Raw	1/14/2019	ND	9.51	ND	ND	ND		ND	**82.53**	**29.53**	**36**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	1/15/2019	ND	ND	ND	4.58	ND	ND	ND	ND	ND	ND	ND	1.83	ND	ND	ND	ND	9.18	17
Sweeney Raw	1/21/2019	ND	ND	ND	9.69	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.03
Sweeney Finished	1/22/2019	ND	ND	ND	6.02	ND	ND	ND	ND	ND	ND	ND	1.43	ND	ND	ND	ND	5.91	15.1
Sweeney Raw	1/28/2019	ND	ND	ND	4.31	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.35
Sweeney Finished	1/29/2019	ND	ND	ND	3.54	ND	ND	ND	ND	ND	ND	ND	1.48	ND	ND	ND	ND	6.45	15.7
Sweeney Raw	2/4/2019	ND	ND	ND	11.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.72	5.46
Sweeney Finished	2/5/2019	ND	ND	ND	7.2	ND	ND	ND	ND	ND	ND	ND	1.86	ND	ND	ND	ND	7.63	18.1
Sweeney Raw	2/11/2019	ND	ND	ND	19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.27	4.75
Sweeney Finished	2/12/2019	ND	ND	ND	11.5	ND	ND	ND	ND	ND	ND	ND	2.34	ND	ND	ND	ND	7.17	21
Sweeney Raw	2/18/2019	ND	ND	ND	11.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.5	3.73
Sweeney Finished	2/19/2019	ND	ND	ND	10.6	ND	ND	ND	ND	ND	ND	ND	2.05	ND	ND	ND	ND	8.24	22.8
Sweeney Raw	2/25/2019	ND	ND	ND	4.08	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	2/26/2019	ND	ND	ND	3.49	ND	ND	ND	ND	ND	ND	ND	1.87	ND	ND	ND	ND	8.36	19.9
Sweeney Raw	3/4/2019	ND	ND	ND	8.59	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.61
Sweeney Finished	3/5/2019	ND	ND	ND	5.28	ND	ND	ND	ND	ND	ND	ND	1.82	ND	ND	ND	ND	7.25	17.2
Sweeney Raw	3/11/2019	ND	ND	ND	6.75	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.63
Sweeney Finished	3/12/2019	ND	ND	ND	5.50	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.32
Sweeney Raw	3/18/2019	ND	ND	ND	7.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.67
Sweeney Finished	3/19/2019	ND	ND	ND	5.25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.17
Sweeney Raw	3/25/2019	ND	ND	ND	3.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	3/26/2019	ND	ND	ND	3.35	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.70
Sweeney Raw	4/1/2019	ND	ND	ND	7.90	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.87
Sweeney Finished	4/2/2019	ND	ND	ND	3.81	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.27
Sweeney Raw	4/8/2019	ND	ND	ND	18.70	ND	ND	ND	ND	ND	ND	ND	4.17	1.46	ND	ND	ND	1.52	4.48
Sweeney Finished	4/9/2019	ND	ND	ND	9.18	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.91
Sweeney Raw	4/15/2019	ND	ND	ND	8.39	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.78
Sweeney Finished	4/16/2019	ND	ND	ND	2.87	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.86
Sweeney Raw	4/22/2019	ND	ND	ND	18.0	ND	ND	ND	ND	ND	ND	ND	ND	1.48	ND	ND	ND	1.78	5.84
Sweeney Finished	4/23/2019	ND	ND	ND	7.22	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.43

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	1/15/2019	24.4	22	5.66	1.64	1.52	4.26	ND	0.638	ND	ND	5.32	ND	1.99	8.64	ND	0.837		ND	6.23	4.72
Sweeney Raw	1/21/2019	8.39	11.1	13.1	ND	2.16	4.61	ND	0.593	ND	ND	5.26	ND	2.73	8.11	ND	0.952		ND	8.92	6.68
Sweeney Finished	1/22/2019	21	24.6	4.6	ND	1.28	3.18	ND	ND	ND	ND	3.21	ND	1.88	5.27	ND	0.813		ND	5.67	3.38
Sweeney Raw	1/28/2019	3.69	5.08	9.74	ND	2.51	3.67	ND	ND	ND	ND	7.91	ND	3.7	10.2	ND	0.953		ND	10.4	6.74
Sweeney Finished	1/29/2019	20.1	24.3	6.88	ND	1.12	2.66	ND	ND	ND	ND	3.46	ND	1.51	5.41	ND	0.697		ND	4.31	3.18
Sweeney Raw	2/4/2019	10.7	16.9	4.25	6.17	2.28	ND	ND	ND	ND	ND	5.6	ND	3.18	7.7	ND	0.972		ND	11.1	6.06
Sweeney Finished	2/5/2019	21.7	26.3	4.81	ND	1.41	3.04	ND	ND	ND	ND	3.54	ND	1.93	5.5	ND	0.759		ND	6.95	4.03
Sweeney Raw	2/11/2019	13.6	17.8	5.96	ND	2.63	ND	ND	ND	ND	ND	4.64	ND	4.28	7.49	ND	0.735		ND	12.6	6.32
Sweeney Finished	2/12/2019	27.6	37.8	7.01	ND	1.56	ND	ND	ND	ND	ND	2.85	ND	2.72	5.16	ND	ND		ND	7.5	4.3
Sweeney Raw	2/18/2019	7.79	12.4	4.79	7.23	2.26	3.68	ND	ND	ND	ND	3.96	ND	4.25	6.24	ND	ND		ND	9.24	4.58
Sweeney Finished	2/19/2019	29.5	33.1	7.5	ND	1.72	4.15	ND	ND	ND	ND	3.39	ND	2.52	5.97	ND	ND		ND	5.85	3.91
Sweeney Raw	2/25/2019	2.78	3.82	1.61	ND	1.6	ND	ND	ND	ND	ND	2.13	ND	2.1	4.02	ND	0.809		ND	9.54	4.41
Sweeney Finished	2/26/2019	26	27.7	3.17	ND	1.25	ND	ND	0.698	ND	ND	2.1	ND	1.61	3.73	ND	0.782		ND	5.34	3.99
Sweeney Raw	3/4/2019	6.54	7.68	5.21	3.57	1.58	ND	ND	ND	ND	ND	2.07	ND	2.26	3.28	ND	0.723		ND	7.93	4.68
Sweeney Finished	3/5/2019	23.6	28.7	4.51	1.94	1.08	1.8	ND	ND	ND	ND	1.37	ND	1.79	2.77	ND	ND		ND	5.14	2.93
Sweeney Raw	3/11/2019	5.36	4.69	10.6	2.31	1.61	4.39	ND	ND	ND	ND	2.22	ND	2.5	3.74	ND	0.629		ND	8.69	4.64
Sweeney Finished	3/12/2019	5.57	8.09	5.93	2.57	0.853	2.12	ND	ND	ND	ND	1.09	ND	0.775	2.66	ND	ND		ND	2.61	1.92
Sweeney Raw	3/18/2019	5.29	7.07	4.26	4.04	2.30	3.76	ND	0.747	ND	ND	3.59	ND	3.51	5.86	ND	0.991		ND	13.6	6.86
Sweeney Finished	3/19/2019	7.25	7.35	5.75	2.10	1.11	2.74	ND	ND	ND	ND	1.80	ND	1.13	3.76	ND	ND		ND	3.43	3.04
Sweeney Raw	3/25/2019	3.08	4.36	1.32	ND	2.03	3.87	ND	ND	ND	ND	3.26	ND	3.03	5.57	ND	0.893		ND	10.5	5.4
Sweeney Finished	3/26/2019	5.31	4.79	4.39	1.57	0.718	2.46	ND	ND	ND	ND	1.32	ND	0.730	2.68	ND	ND		ND	1.67	1.78
Sweeney Raw	4/1/2019	9.98	12.2	4.09	1.72	1.95	ND	ND	0.673	ND	ND	3.14	ND	2.98	5.32	ND	0.857		ND	11.1	6.32
Sweeney Finished	4/2/2019	6.40	5.39	4.31	1.35	0.617	2.27	ND	ND	ND	ND	1.21	ND	ND	2.42	ND	ND		ND	1.18	1.67
Sweeney Raw	4/8/2019	13.0	14.7	10.6	6.02	2.55	3.08	ND	ND	ND	ND	5.86	ND	4.26	6.54	ND	0.929		ND	12.2	5.82
Sweeney Finished	4/9/2019	8.26	8.63	11.8	2.42	1.04	2.70	ND	ND	ND	ND	2.22	ND	0.817	3.75	ND	ND		ND	1.87	1.95
Sweeney Raw	4/15/2019	7.65	7.20	5.05	2.17	2.05	ND	ND	ND	ND	ND	2.81	ND	2.50	3.67	ND	0.830		ND	8.55	3.69
Sweeney Finished	4/16/2019	6.38	6.50	5.15	1.28	0.766	2.06	ND	ND	ND	ND	0.959	ND	ND	2.48	ND	ND		ND	0.708	1.09
Sweeney Raw	4/22/2019	11.3	15.5	12.1	ND	1.96	ND	ND	ND	ND	ND	2.6	ND	2.69	3.59	ND	0.727		ND	9.38	4.63
Sweeney Finished	4/23/2019	8.00	8.81	12.20	ND	0.811	2.46	ND	ND	ND	ND	1.24	ND	ND	2.22	ND	ND		ND	1.06	1.24

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	1/15/2019	ND	6.19	ND	ND	ND		ND	**126.64**	**91.61**	**72**
Sweeney Raw	1/21/2019	ND	6.72	ND	ND	ND		ND	**92.05**	**50.57**	**55**
Sweeney Finished	1/22/2019	ND	5	ND	ND	ND		ND	**108.34**	**81.87**	**76**
Sweeney Raw	1/28/2019	0.658	7.52	ND	ND	ND		ND	**78.43**	**32.08**	**41**
Sweeney Finished	1/29/2019	ND	4.52	ND	ND	ND		ND	**105.32**	**81.91**	**78**
Sweeney Raw	2/4/2019	ND	6.84	ND	ND	ND		ND	**100.73**	**62.60**	**62**
Sweeney Finished	2/5/2019	ND	5.24	ND	ND	ND		ND	**120.00**	**91.14**	**76**
Sweeney Raw	2/11/2019	0.725	7.2	ND	ND	ND		ND	**109.00**	**67.02**	**61**
Sweeney Finished	2/12/2019	ND	4.71	ND	ND	ND		ND	**143.22**	**117.27**	**82**
Sweeney Raw	2/18/2019	0.738	6.2	ND	ND	ND		ND	**90.49**	**53.30**	**59**
Sweeney Finished	2/19/2019	ND	6.03	ND	ND	ND		ND	**147.33**	**117.18**	**80**
Sweeney Raw	2/25/2019	ND	4.09	ND	ND	ND		ND	**40.99**	**14.42**	**35**
Sweeney Finished	2/26/2019	ND	3.91	ND	ND	ND		ND	**113.90**	**92.59**	**81**
Sweeney Raw	3/4/2019	ND	2.73	ND	ND	ND		ND	**58.45**	**35.27**	**60**
Sweeney Finished	3/5/2019	ND	2.55	ND	ND	ND		ND	**109.73**	**91.67**	**84**
Sweeney Raw	3/11/2019	ND	3.53	ND	ND	ND		ND	**63.29**	**33.56**	**53**
Sweeney Finished	3/12/2019	ND	2.75	ND	ND	ND		ND	**44.76**	**31.07**	**69**
Sweeney Raw	3/18/2019	ND	5.19	ND	ND	ND		ND	**75.86**	**33.04**	**44**
Sweeney Finished	3/19/2019	ND	3.60	ND	ND	ND		ND	**50.48**	**31.67**	**63**
Sweeney Raw	3/25/2019	0.663	5.4	ND	ND	ND		ND	**52.52**	**15.16**	**29**
Sweeney Finished	3/26/2019	ND	3.03	ND	ND	ND		ND	**35.50**	**22.43**	**63**
Sweeney Raw	4/1/2019	ND	5.60	ND	ND	ND		ND	**77.70**	**42.90**	**55**
Sweeney Finished	4/2/2019	ND	2.92	ND	ND	ND		ND	**34.82**	**23.74**	**68**
Sweeney Raw	4/8/2019	0.715	6.11	ND	ND	ND		ND	**122.71**	**80.51**	**66**
Sweeney Finished	4/9/2019	ND	3.93	ND	ND	ND		ND	**60.48**	**44.42**	**73**
Sweeney Raw	4/15/2019	ND	3.55	ND	ND	ND		ND	**60.89**	**36.05**	**59**
Sweeney Finished	4/16/2019	ND	2.30	ND	ND	ND		ND	**34.40**	**25.00**	**73**
Sweeney Raw	4/22/2019	ND	4.11	ND	ND	ND		ND	**95.69**	**68.60**	**72**
Sweeney Finished	4/23/2019	ND	2.91	ND	ND	ND		ND	**50.60**	**39.90**	**79**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Raw	4/29/2019	ND	ND	ND	8.64	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.83
Sweeney Finished	4/30/2019	ND	ND	ND	3.27	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.48
Sweeney Raw	5/6/2019	ND	ND	ND	6.79	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.73
Sweeney Finished	5/7/2019	ND	ND	ND	3.67	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.99
Sweeney Raw	5/13/2019	ND	ND	ND	12.0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.90	6.09
Sweeney Finished	5/14/2019	ND	ND	ND	4.88	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.53
Sweeney Raw	5/20/2019	ND	ND	ND	19.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.57	7.27
Sweeney Finished	5/21/2019	ND	ND	ND	7.24	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.65
Sweeney Raw	5/27/2019	ND	ND	ND	23.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.59	8.01
Sweeney Finished	5/28/2019	ND	ND	ND	9.32	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.25	4.62
Sweeney Raw	6/3/2019	ND	ND	ND	31.9	ND	ND	ND	ND	ND	ND	ND	ND	3.51	ND	ND	ND	6.14	16.7
Sweeney Finished	6/4/2019	ND	ND	ND	13.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.58	6.55
Sweeney Raw	6/10/2019	ND	ND	ND	36.9	ND	ND	ND	ND	ND	ND	ND	ND	5.43	ND	ND	ND	7.09	18.4
Sweeney Finished	6/11/2019	ND	ND	ND	15.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.31	6.69
Sweeney Raw	6/17/2019	ND	ND	ND	9.66	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.82
Sweeney Finished	6/18/2019	ND	ND	ND	7.73	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.90
Sweeney Raw	6/24/2019	ND	ND	ND	9.67	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.65	4.13
Sweeney Finished	6/25/2019	ND	ND	ND	8.03	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.76	7.30
Sweeney Raw	7/1/2019	ND	ND	ND	54.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	11.3
Sweeney Finished	7/2/2019	ND	ND	ND	8.61	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.41	6.53
Sweeney Raw	7/8/2019	ND	ND	ND	28.6	ND	ND	ND	ND	ND	ND	ND	ND	3.08	ND	ND	ND	3.88	9.71
Sweeney Finished	7/9/2019	ND	ND	ND	14.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.60	8.25
Sweeney Raw	7/15/2019	ND	ND	ND	24.8	ND	ND	ND	ND	ND	ND	ND	ND	2.70	ND	ND	ND	3.31	7.43
Sweeney Finished	7/16/2019	ND	ND	ND	16.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.80	8.75
Sweeney Raw	7/29/2019	ND	ND	ND	24.8	ND	ND	ND	ND	ND	ND	ND	ND	2.10	ND	ND	ND	2.42	8.85
Sweeney Finished	7/30/2019	ND	ND	ND	19.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.25	10.0
Sweeney Raw	8/12/2019	ND	ND	ND	15.7	ND	ND	ND	ND	ND	ND	ND	ND	1.57	ND	ND	ND	2.04	5.10
Sweeney Finished	8/13/2019	ND	ND	ND	13.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.43	13.4
Sweeney Raw	8/26/2019	ND	ND	ND	28.3	ND	ND	ND	ND	ND	ND	ND	ND	2.70	ND	ND	ND	3.15	9.93

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Raw	4/29/2019	8.79	11.8	6.06	4.04	2.77	4.65	ND	0.718	ND	ND	8.29	ND	4.15	9.01	ND	1.42		ND	16.6	8.79
Sweeney Finished	4/30/2019	4.83	6.11	3.81	ND	0.621	2.24	ND	ND	ND	ND	1.47	ND	ND	3.13	ND	ND		ND	1.26	1.41
Sweeney Raw	5/6/2019	5.76	8.2	2.47	3.03	2.77	5.37	ND	0.730	ND	ND	6.82	ND	3.30	9.42	ND	1.17		ND	15.1	7.87
Sweeney Finished	5/7/2019	5.81	5.64	4.14	2.21	0.910	3.39	ND	ND	ND	ND	2.08	ND	ND	3.92	ND	ND		ND	1.18	1.66
Sweeney Raw	5/13/2019	14.5	18.7	4.61	ND	3.05	ND	ND	0.932	ND	ND	7.93	ND	4.13	10.9	ND	1.17		ND	18.2	8.24
Sweeney Finished	5/14/2019	9.26	9.56	5.21	1.92	0.916	3.60	ND	ND	ND	ND	1.85	ND	ND	4.10	ND	ND		ND	1.09	1.60
Sweeney Raw	5/20/2019	19.4	25.2	8.76	3.96	2.64	5.64	ND	ND	ND	ND	4.98	ND	4.70	8.69	ND	1.24		ND	14.0	7.03
Sweeney Finished	5/21/2019	11.3	11.8	10.1	3.73	0.806	3.57	ND	ND	ND	ND	1.57	ND	0.64	3.07	ND	ND		ND	0.852	1.15
Sweeney Raw	5/27/2019	20.3	29.3	6.78	1.82	3.15	6.52	ND	0.750	ND	ND	7.35	ND	4.46	11	ND	0.95		ND	13.8	6.97
Sweeney Finished	5/28/2019	17.3	17.8	12.7	4.47	1.04	4.04	ND	ND	ND	ND	1.83	ND	0.626	3.93	ND	ND		ND	0.874	1.29
Sweeney Raw	6/3/2019	43.5	52.2	14.6	6.34	3.75	6.89	ND	0.775	ND	ND	9.99	ND	5.83	16.5	ND	1.29		ND	17.4	7.97
Sweeney Finished	6/4/2019	24.3	21.7	18.0	7.36	1.15	6.03	ND	ND	ND	ND	2.27	ND	0.628	5.36	ND	ND		ND	0.940	1.26
Sweeney Raw	6/10/2019	46.8	57.0	14.7	8.23	3.72	5.29	ND	0.890	ND	ND	9.47	ND	6.25	14.4	ND	1.02		ND	18.4	8.27
Sweeney Finished	6/11/2019	28.8	25.8	18.2	7.82	1.22	5.99	ND	ND	ND	ND	2.23	ND	0.592	5.73	ND	ND		ND	1.08	1.26
Sweeney Raw	6/17/2019	7.70	8.02	2.07	2.40	3.44	6.07	ND	0.783	ND	ND	9.57	ND	4.52	15.3	ND	1.07		ND	14.0	6.51
Sweeney Finished	6/18/2019	16.2	15.1	7.0	3.39	1.41	5.35	ND	ND	ND	ND	2.70	ND	0.815	6.58	ND	ND		ND	1.28	1.48
Sweeney Raw	6/24/2019	13.7	14.7	5.46	2.41	2.41	6.43	ND	1.25	ND	ND	13.5	ND	3.58	20.3	ND	1.42		ND	14.4	8.61
Sweeney Finished	6/25/2019	20.0	20.6	10.3	3.45	1.37	6.61	ND	ND	ND	ND	4.76	ND	0.903	10.6	ND	ND		ND	0.908	2.25
Sweeney Raw	7/1/2019	57.7	63.0	64.9	22.6	10.3	6.86	ND	ND	ND	ND	2.22	ND	2.08	5.16	ND	ND		ND	1.56	3.36
Sweeney Finished	7/2/2019	22.6	21.1	8.1	2.43	1.84	8.13	ND	ND	ND	ND	4.51	ND	1.070	11.9	ND	ND		ND	1.280	2.39
Sweeney Raw	7/8/2019	32.5	36.2	8.07	9.52	4.45	7.22	ND	1.29	ND	ND	17.7	ND	5.73	25.7	ND	1.48		ND	19.6	9.63
Sweeney Finished	7/9/2019	30.7	34.5	15.1	6.44	2.19	8.64	ND	ND	ND	ND	5.41	ND	1.130	12.70	ND	ND		ND	1.28	2.59
Sweeney Raw	7/15/2019	24.7	34.9	7.58	7.15	5.53	5.20	ND	1.01	ND	ND	10.9	ND	9.39	16.0	ND	1.37		ND	21.4	8.59
Sweeney Finished	7/16/2019	36.5	35.2	16.5	5.92	2.56	8.18	ND	ND	ND	ND	4.47	ND	1.30	11.5	ND	ND		ND	1.21	2.22
Sweeney Raw	7/29/2019	25.3	30.0	6.67	7.90	5.43	7.70	ND	0.97	ND	ND	12.7	ND	8.54	19.3	ND	1.28		ND	19.7	7.20
Sweeney Finished	7/30/2019	35.2	42.5	13.6	5.14	3.07	8.47	ND	ND	ND	ND	5.41	ND	2.18	12.2	ND	ND		ND	2.36	2.60
Sweeney Raw	8/12/2019	15.5	22.2	5.51	5.79	4.84	8.28	ND	0.967	ND	ND	10.5	ND	7.05	14.2	ND	1.13		ND	15.7	6.62
Sweeney Finished	8/13/2019	32.7	29.5	11.6	5.68	3.10	7.80	ND	ND	ND	ND	5.23	ND	1.90	10.8	ND	ND		ND	1.64	2.51
Sweeney Raw	8/26/2019	28.7	38.9	8.30	ND	5.54	11.2	ND	1.42	ND	ND	24.8	ND	6.24	34.7	ND	1.74		ND	18.4	9.95

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Raw	4/29/2019	1.01	8.21	ND	ND	ND		ND	**108.78**	**51.45**	**47**
Sweeney Finished	4/30/2019	ND	2.93	ND	ND	ND		ND	**33.56**	**21.97**	**65**
Sweeney Raw	5/6/2019	0.726	8.21	ND	ND	ND		ND	**90.46**	**35.79**	**40**
Sweeney Finished	5/7/2019	ND	4.55	ND	ND	ND		ND	**41.15**	**25.54**	**62**
Sweeney Raw	5/13/2019	0.644	10.2	ND	ND	ND		ND	**123.20**	**65.73**	**53**
Sweeney Finished	5/14/2019	ND	4.67	ND	ND	ND		ND	**51.19**	**35.21**	**69**
Sweeney Raw	5/20/2019	0.704	10.2	ND	ND	ND		ND	**146.58**	**91.74**	**63**
Sweeney Finished	5/21/2019	ND	4.58	ND	ND	ND		ND	**64.06**	**49.39**	**77**
Sweeney Raw	5/27/2019	0.749	12.2	ND	ND	ND		ND	**160.30**	**99.75**	**62**
Sweeney Finished	5/28/2019	ND	5.85	ND	ND	ND		ND	**86.94**	**69.29**	**80**
Sweeney Raw	6/3/2019	0.888	16.3	ND	ND	ND		ND	**262.47**	**184.88**	**70**
Sweeney Finished	6/4/2019	ND	8.11	ND	ND	ND		ND	**118.84**	**95.36**	**80**
Sweeney Raw	6/10/2019	0.93	18.0	ND	ND	ND		ND	**281.19**	**204.02**	**73**
Sweeney Finished	6/11/2019	ND	8.39	ND	ND	ND		ND	**130.71**	**106.45**	**81**
Sweeney Raw	6/17/2019	0.747	13.2	ND	ND	ND		ND	**107.88**	**42.24**	**39**
Sweeney Finished	6/18/2019	ND	8.45	ND	ND	ND		ND	**82.41**	**57.04**	**69**
Sweeney Raw	6/24/2019	0.656	16.4	ND	ND	ND		ND	**140.68**	**65.22**	**46**
Sweeney Finished	6/25/2019	ND	12.3	ND	ND	ND		ND	**111.14**	**76.20**	**69**
Sweeney Raw	7/1/2019	0.788	5.25	ND	ND	ND		ND	**311.88**	**276.52**	**89**
Sweeney Finished	7/2/2019	ND	14.8	ND	ND	ND		ND	**116.65**	**75.24**	**65**
Sweeney Raw	7/8/2019	1.04	22.2	ND	ND	ND		ND	**247.60**	**149.26**	**60**
Sweeney Finished	7/9/2019	ND	15.5	ND	ND	ND		ND	**160.93**	**116.9**	**73**
Sweeney Raw	7/15/2019	1.29	18.9	ND	ND	ND		ND	**212.15**	**123.47**	**58**
Sweeney Finished	7/16/2019	ND	14.6	ND	ND	ND		ND	**167.11**	**125.54**	**75**
Sweeney Raw	7/29/2019	0.980	21.3	ND	ND	ND		ND	**213.14**	**120.74**	**57**
Sweeney Finished	7/30/2019	ND	16.3	ND	ND	ND		ND	**180.78**	**133.6**	**74**
Sweeney Raw	8/12/2019	1.31	16.6	ND	ND	ND		ND	**160.61**	**83.91**	**52**
Sweeney Finished	8/13/2019	ND	13.9	ND	ND	ND		ND	**156.09**	**114.44**	**73**
Sweeney Raw	8/26/2019	1.19	33.7	ND	ND	ND		ND	**268.86**	**144.78**	**54**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	8/27/2019	ND	ND	ND	18.2	ND	ND	ND	ND	ND	ND	ND	1.29	ND	ND	ND	ND	3.26	12.7
Sweeney Raw	9/4/2019	ND	ND	ND	11.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.75	4.08
Sweeney Raw	9/5/2019	ND	ND	ND	11.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.69	3.91
Sweeney Raw	9/6/2019	ND	ND	ND	11.1	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.69
Sweeney Raw	9/9/2019	ND	ND	ND	76.0	ND	ND	ND	ND	ND	ND	3.78	6.14	ND	ND	ND	ND	7.98	14.3
Sweeney Finished	9/10/2019	ND	ND	ND	25.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.70	12.4
Sweeney Raw	9/23/2019	ND	ND	ND	28.4	ND	ND	ND	ND	ND	ND	ND	2.98	ND	ND	ND	ND	3.20	8.80
Sweeney Finished	9/24/2019	ND	ND	ND	20.6	ND	ND	ND	ND	ND	ND	ND	1.62	ND	ND	ND	ND	2.76	13.2
Sweeney Raw	10/1/2019	ND	ND	ND	39.4	ND	ND	ND	ND	ND	ND	ND	3.41	ND	ND	ND	ND	3.74	14.30
Sweeney Finished	10/2/2019	ND	ND	ND	28.4	ND	ND	ND	ND	ND	ND	ND	2.05	ND	ND	ND	ND	3.99	16.7
Sweeney Raw	10/7/2019	ND	ND	ND	38.8	ND	ND	ND	ND	ND	ND	ND	2.91	ND	ND	ND	ND	5.29	17.5
Sweeney Finished	10/8/2019	ND	ND	ND	34.2	ND	ND	ND	ND	ND	ND	ND	1.96	ND	ND	ND	ND	3.99	18.5
Sweeney Raw	10/21/2019	ND	ND	ND	41.7	ND	ND	ND	ND	ND	ND	ND	3.33	ND	ND	ND	ND	6.13	17.6
Sweeney Finished	10/22/2019	ND	ND	ND	36.3	ND	ND	ND	ND	ND	ND	ND	2.07	ND	ND	ND	ND	4.13	17.4
Sweeney Raw	11/4/2019	ND	ND	ND	24.8	ND	ND	ND	ND	ND	ND	ND	1.68	ND	ND	ND	ND	2.65	9.52
Sweeney Finished	11/5/2019	ND	ND	ND	12.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.54	9.16
Sweeney Raw	11/18/2019	ND	ND	ND	35.0	ND	ND	ND	ND	ND	ND	ND	1.62	ND	ND	ND	ND	4.38	9.97
Sweeney Finished	11/19/2019	ND	ND	ND	12.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.38	8.25
Sweeney Raw	12/2/2019	ND	ND	ND	22.50	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.26	6.01
Sweeney Finished	12/3/2019	ND	ND	ND	11.30	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.40	6.54
Sweeney Raw	12/16/2019	ND	ND	ND	19.90	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.46	7.89
Sweeney Finished	12/17/2019	ND	ND	ND	11.70	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.83	5.43
Sweeney Raw	12/30/2019	ND	ND	ND	8.04	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.69
Sweeney Finished	12/31/2019	ND	ND	ND	6.40	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.26	3.92
Sweeney Raw	1/13/2020	ND	ND	ND	6.36	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.30
Sweeney Finished	1/14/2020	ND	ND	ND	6.01	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.79
Sweeney Raw	1/27/2020	ND	ND	ND	12.70	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.33	5.12
Sweeney Finished	1/28/2020	ND	ND	ND	9.77	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.71	5.84
Sweeney Raw	2/10/2020	ND	ND	ND	4.47	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.36

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	8/27/2019	40.3	46.2	14.0	6.62	3.82	13.2	ND	ND	ND	ND	9.84	ND	1.81	21.9	ND	ND		ND	2.05	3.29
Sweeney Raw	9/4/2019	11.5	14.8	5.97	3.24	4.31	10.8	ND	0.97	ND	ND	21.9	ND	4.80	26.4	ND	1.54		ND	14.5	8.44
Sweeney Raw	9/5/2019	12.9	16.5	7.41	6.60	4.7	11.5	ND	1.04	ND	ND	23.1	ND	5.72	28.2	ND	1.60		ND	15.5	8.42
Sweeney Raw	9/6/2019	12.7	15.8	6.77	ND	4.22	11.2	ND	1.07	ND	ND	24.2	ND	4.22	30.9	ND	1.53		ND	15.4	7.70
Sweeney Raw	9/9/2019	36.1	52.3	21.7	16.0	5.00	12.5	ND	0.94	ND	ND	26.0	ND	4.75	35.2	ND	1.45		ND	15.5	8.30
Sweeney Finished	9/10/2019	39.0	44.9	16.7	8.82	3.57	16.4	1.20	ND	ND	ND	12.3	ND	1.84	25.4	ND	ND		ND	1.93	3.44
Sweeney Raw	9/23/2019	24.1	34.2	8.74	15.6	3.39	16.1	ND	ND	ND	ND	7.98	ND	5.03	11.3	ND	0.714		ND	11.4	5.19
Sweeney Finished	9/24/2019	39.7	44.8	15.8	7.30	2.87	9.75	ND	ND	ND	ND	6.51	ND	2.24	10.8	ND	ND		ND	2.52	2.88
Sweeney Raw	10/1/2019	40.3	46.8	10.4	25.7	5.60	9.16	ND	1.01	ND	ND	14.9	ND	6.73	21.1	ND	1.29		ND	16.0	7.58
Sweeney Finished	10/2/2019	52.6	63.4	17.6	8.37	4.05	10.8	ND	ND	ND	ND	10.2	ND	2.57	17.0	ND	ND		ND	3.69	4.18
Sweeney Raw	10/7/2019	50.2	57.4	15.7	11.0	6.73	13.8	ND	0.820	ND	ND	22.1	ND	7.71	33.4	ND	1.40		ND	15.2	9.63
Sweeney Finished	10/8/2019	65.1	60.1	28.6	11.4	6.52	12.6	ND	ND	ND	ND	13.7	ND	2.90	29.6	ND	ND		ND	3.78	5.11
Sweeney Raw	10/21/2019	47.7	55.6	11.4	11.1	6.61	18.3	ND	1.05	ND	ND	30.2	ND	7.41	45.5	ND	1.67		ND	14.9	10.20
Sweeney Finished	10/22/2019	57.4	65.7	18.0	10.5	5.66	18.2	ND	ND	ND	ND	19.5	ND	3.45	33.4	ND	0.646		ND	3.68	5.92
Sweeney Raw	11/4/2019	23.0	26.7	9.21	5.47	6.52	17.1	ND	0.961	ND	ND	22.3	ND	9.18	32.6	ND	1.41		ND	17.1	9.84
Sweeney Finished	11/5/2019	24.5	29.1	6.14	3.19	3.27	8.31	ND	ND	ND	ND	9.52	ND	2.47	16.8	ND	ND		ND	2.50	3.68
Sweeney Raw	11/18/2019	34.2	41.8	17.1	ND	5.99	2.05	ND	0.737	ND	ND	14.3	ND	5.97	24.4	ND	1.61		ND	12.9	7.96
Sweeney Finished	11/19/2019	25.3	24.8	11.3	3.50	2.46	6.18	ND	ND	ND	ND	4.98	ND	1.59	10.9	ND	ND		ND	2.08	2.55
Sweeney Raw	12/2/2019	20.60	24.00	13.00	ND	4.24	5.95	ND	ND	ND	ND	6.43	ND	5.51	12.40	ND	0.78		ND	11.00	5.08
Sweeney Finished	12/3/2019	18.40	18.90	15.10	3.38	1.93	4.41	ND	ND	ND	ND	3.87	ND	1.88	7.67	ND	ND		ND	2.24	2.09
Sweeney Raw	12/16/2019	19.80	21.30	6.72	6.02	6.06	10.60	ND	0.87	ND	ND	15.70	ND	4.26	30.80	ND	1.07	ND	ND	13.00	7.92
Sweeney Finished	12/17/2019	14.50	13.70	7.09	4.23	2.55	7.57	ND	ND	ND	ND	6.24	ND	1.68	13.30	ND	ND	ND	ND	2.41	3.04
Sweeney Raw	12/30/2019	7.98	8.52	ND	2.86	4.02	7.07	ND	ND	ND	ND	11.90	ND	3.78	18.30	ND	1.03	ND	ND	10.10	6.60
Sweeney Finished	12/31/2019	10.30	9.65	4.91	2.84	2.21	6.11	ND	ND	ND	ND	6.50	ND	1.56	12.60	ND	ND	ND	ND	2.50	2.71
Sweeney Raw	1/13/2020	6.86	6.98	3.50	1.49	5.01	5.92	ND	ND	ND	ND	7.95	ND	4.14	15.10	ND	0.97	ND	ND	11.30	8.92
Sweeney Finished	1/14/2020	10.40	10.20	4.81	2.34	2.24	5.61	ND	ND	ND	ND	4.02	ND	1.53	9.44	ND	ND	ND	ND	2.46	3.15
Sweeney Raw	1/27/2020	12.40	16.00	8.50	ND	3.76	5.31	ND	ND	ND	ND	9.10	ND	4.02	12.90	ND	0.97	ND	ND	13.10	7.22
Sweeney Finished	1/28/2020	13.30	15.00	11.10	3.22	2.69	5.99	ND	ND	ND	ND	5.90	ND	2.05	11.90	ND	ND	ND	ND	4.42	3.94
Sweeney Raw	2/10/2020	4.17	5.82	7.71	ND	2.40	3.29	ND	ND	ND	ND	5.34	ND	2.93	5.79	ND	0.71	ND	ND	8.18	4.37

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	8/27/2019	ND	25.9	ND	ND	ND		ND	**224.38**	**152.41**	**68**
Sweeney Raw	9/4/2019	0.79	24.4	ND	ND	ND		ND	**171.49**	**74.54**	**43**
Sweeney Raw	9/5/2019	1.07	25.3	ND	ND	ND		ND	**186.76**	**83.71**	**45**
Sweeney Raw	9/6/2019	0.96	26.7	ND	ND	ND		ND	**178.16**	**74.26**	**42**
Sweeney Raw	9/9/2019	0.71	32.2	ND	ND	ND		ND	**376.85**	**260.3**	**69**
Sweeney Finished	9/10/2019	ND	28.6	ND	ND	ND		ND	**244.40**	**162.02**	**66**
Sweeney Raw	9/23/2019	0.665	15.6	ND	ND	ND		ND	**203.39**	**134**	**66**
Sweeney Finished	9/24/2019	0.640	11.9	ND	ND	ND		ND	**195.89**	**152.29**	**78**
Sweeney Raw	10/1/2019	1.23	21.0	ND	ND	ND		ND	**289.65**	**198.95**	**69**
Sweeney Finished	10/2/2019	0.651	22.8	ND	ND	ND		ND	**269.05**	**203.31**	**76**
Sweeney Raw	10/7/2019	1.24	31.8	ND	ND	ND		ND	**342.63**	**220.9**	**64**
Sweeney Finished	10/8/2019	0.812	31.2	ND	ND	ND		ND	**330.07**	**237.55**	**72**
Sweeney Raw	10/21/2019	1.51	45.1	ND	ND	ND		ND	**377.01**	**224.76**	**60**
Sweeney Finished	10/22/2019	1.04	41.6	ND	ND	ND		ND	**344.60**	**231**	**67**
Sweeney Raw	11/4/2019	1.60	36.5	ND	ND	ND		ND	**258.14**	**125.33**	**49**
Sweeney Finished	11/5/2019	ND	19.7	ND	ND	ND		ND	**153.48**	**96.75**	**63**
Sweeney Raw	11/18/2019	1.13	26.9	ND	ND	ND		ND	**248.02**	**158.37**	**64**
Sweeney Finished	11/19/2019	ND	13.4	ND	ND	ND		ND	**130.97**	**91.81**	**70**
Sweeney Raw	12/2/2019	0.88	13.80	ND	ND	ND		ND	**154.43**	**94.8**	**61**
Sweeney Finished	12/3/2019	ND	8.60	ND	ND	ND		ND	**107.71**	**78.89**	**73**
Sweeney Raw	12/16/2019	1.00	25.40	ND	ND	ND	ND	ND	**200.76**	**99.79**	**50**
Sweeney Finished	12/17/2019	ND	14.30	ND	ND	ND	ND	ND	**109.57**	**64.72**	**59**
Sweeney Raw	12/30/2019	0.91	14.30	ND	ND	ND	ND	ND	**108.10**	**41.99**	**39**
Sweeney Finished	12/31/2019	ND	11.60	ND	ND	ND	ND	ND	**85.07**	**45.78**	**54**
Sweeney Raw	1/13/2020	1.01	11.30	ND	ND	ND	ND	ND	**99.11**	**35.44**	**36**
Sweeney Finished	1/14/2020	ND	8.95	ND	ND	ND	ND	ND	**74.95**	**41.57**	**55**
Sweeney Raw	1/27/2020	0.68	10.80	ND	ND	ND	ND	ND	**123.91**	**65.15**	**53**
Sweeney Finished	1/28/2020	ND	11.20	ND	ND	ND	ND	ND	**108.03**	**65.84**	**61**
Sweeney Raw	2/10/2020	ND	4.70	ND	ND	ND	ND	ND	**61.24**	**28.87**	**47**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	2/11/2020	ND	ND	ND	4.73	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.11	4.36
Sweeney Raw	2/24/2020	ND	ND	ND	4.61	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	2/25/2020	ND	ND	ND	4.35	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.06
Sweeney Raw	3/9/2020	ND	ND	ND	13.00	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.64	3.61
Sweeney Finished	3/10/2020	ND	ND	ND	8.35	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.27	3.80
Sweeney Raw	4/6/2020	ND	ND	ND	11.30	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.37	4.93
Sweeney Finished	4/7/2020	ND	ND	ND	5.04	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.89
Sweeney Raw	5/4/2020	ND	ND	ND	9.42	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.38	3.58
Sweeney Finished	5/5/2020	ND	ND	ND	4.84	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.22
Sweeney Raw	6/1/2020	ND	ND	ND	4.19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	6/2/2020	ND	ND	ND	3.67	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Raw	7/6/2020	ND	ND	ND	17.80	ND	ND	ND	ND	ND	ND	ND	1.37	ND	ND	ND	ND	2.10	7.26
Sweeney Finished	7/7/2020	ND	ND	ND	11.30	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.07
Sweeney Raw	7/22/2020	ND	ND	ND	23.50	ND	ND	ND	ND	ND	ND	ND	1.41	ND	ND	ND	ND	2.25	6.65
Sweeney Raw	7/23/2020	ND	ND	ND	23.90	ND	ND	ND	ND	ND	ND	ND	1.47	ND	ND	ND	ND	2.71	6.45
Sweeney Raw	7/24/2020	ND	ND	ND	24.90	ND	ND	ND	ND	ND	ND	ND	1.69	ND	ND	ND	ND	2.70	8.61
Sweeney Raw	7/25/2020	ND	ND	ND	22.50	ND	ND	ND	ND	ND	ND	ND	1.75	ND	ND	ND	ND	2.63	8.51
Sweeney Raw	8/2/2020	ND	ND	ND	16.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.25	5.36
Sweeney Raw	8/3/2020	ND	ND	ND	15.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.50	3.94
Sweeney Raw	8/4/2020	ND	ND	ND	13.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.45	3.70
Sweeney Raw	8/5/2020	ND	ND	ND	13.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.23	3.43
Sweeney Finished	8/5/2020	ND	ND	ND	12.1	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.11

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	2/11/2020	9.69	9.73	6.02	ND	1.78	4.53	ND	ND	ND	ND	4.29	ND	1.60	7.01	ND	ND	ND	ND	3.37	3.17
Sweeney Raw	2/24/2020	4.04	9.89	2.01	1.50	2.61	3.43	ND	ND	ND	ND	5.45	ND	3.11	6.54	ND	0.75	ND	ND	8.93	5.40
Sweeney Finished	2/25/2020	7.79	18.30	2.78	2.04	2.03	4.47	ND	ND	ND	ND	3.43	ND	1.33	7.05	ND	ND	ND	ND	3.12	3.56
Sweeney Raw	3/9/2020	11.60	18.70	5.05	6.75	2.93	ND	ND	ND	ND	ND	4.38	ND	3.61	6.03	ND	0.90	ND	ND	9.48	5.64
Sweeney Finished	3/10/2020	11.40	12.10	6.46	4.55	1.88	4.29	ND	ND	ND	ND	3.37	ND	1.80	6.91	ND	ND	ND	ND	3.04	3.26
Sweeney Raw	4/6/2020	12.70	9.11	6.98	4.49	4.33	5.53	ND	ND	ND	ND	11.30	ND	4.27	13.70	ND	1.42	ND	ND	15.20	8.68
Sweeney Finished	4/7/2020	6.79	4.81	9.41	1.75	1.15	3.71	ND	ND	ND	ND	2.88	ND	ND	4.78	ND	ND	ND	ND	1.23	1.70
Sweeney Raw	5/4/2020	9.68	10.90	11.10	3.54	4.42	5.75	ND	ND	ND	ND	6.69	ND	5.38	12.00	ND	0.82	ND	ND	12.40	7.92
Sweeney Finished	5/5/2020	5.64	8.49	6.42	2.14	1.13	3.60	ND	ND	ND	ND	1.81	ND	0.64	4.85	ND	ND	ND	ND	1.06	1.53
Sweeney Raw	6/1/2020	3.77	3.40	2.67	ND	2.38	3.05	ND	ND	ND	ND	2.69	ND	3.51	4.90	ND	1.02	ND	ND	11.30	4.63
Sweeney Finished	6/2/2020	5.44	7.34	7.03	1.43	1.08	2.92	ND	ND	ND	ND	1.70	ND	0.68	3.93	ND	ND	ND	ND	1.22	1.32
Sweeney Raw	7/6/2020	20.00	23.80	9.82	7.50	3.95	5.88	ND	0.65	ND	ND	3.90	ND	4.83	7.40	ND	0.93	ND	ND	14.80	7.90
Sweeney Finished	7/7/2020	15.00	16.50	12.70	5.44	2.23	6.43	ND	ND	ND	ND	2.23	ND	0.97	6.73	ND	ND	ND	ND	1.41	2.34
Sweeney Raw	7/22/2020	24.10	24.80	12.40	ND	4.18	5.76	ND	0.73	ND	ND	3.96	ND	5.02	7.46	ND	1.12	ND	ND	16.40	6.83
Sweeney Raw	7/23/2020	24.90	22.10	12.30	ND	4.41	6.06	ND	0.75	ND	ND	3.75	ND	5.41	7.82	ND	1.34	ND	ND	17.90	6.51
Sweeney Raw	7/24/2020	26.50	34.60	7.12	9.18	4.14	6.58	ND	0.61	ND	ND	4.65	ND	5.05	7.67	ND	1.31	ND	ND	13.20	6.41
Sweeney Raw	7/25/2020	25.90	30.00	7.41	7.17	4.03	6.42	ND	0.69	ND	ND	3.83	ND	4.43	7.47	ND	0.98	ND	ND	13.80	6.32
Sweeney Raw	8/2/2020	17.3	22.0	10.80	2.45	4.09	4.86	ND	ND	ND	ND	3.31	ND	4.46	6.11	ND	0.834	ND	ND	13.0	6.09
Sweeney Raw	8/3/2020	11.7	19.0	10.30	2.58	4.52	5.66	ND	ND	ND	ND	3.74	ND	4.59	7.65	ND	0.947	ND	ND	12.1	6.59
Sweeney Raw	8/4/2020	13.3	15.8	9.73	ND	5.12	5.63	ND	0.790	ND	ND	4.46	ND	4.67	8.59	ND	1.22	ND	ND	14.7	7.70
Sweeney Raw	8/5/2020	12.1	15.4	9.28	ND	4.42	5.71	ND	0.817	ND	ND	4.49	ND	4.65	8.22	ND	1.28	ND	ND	12.6	7.01
Sweeney Finished	8/5/2020	19.6	24.3	16.6	3.79	3.03	7.04	ND	ND	ND	ND	2.63	ND	1.14	7.91	ND	ND	ND	ND	1.37	2.12

Raw and Finished PFAS Data

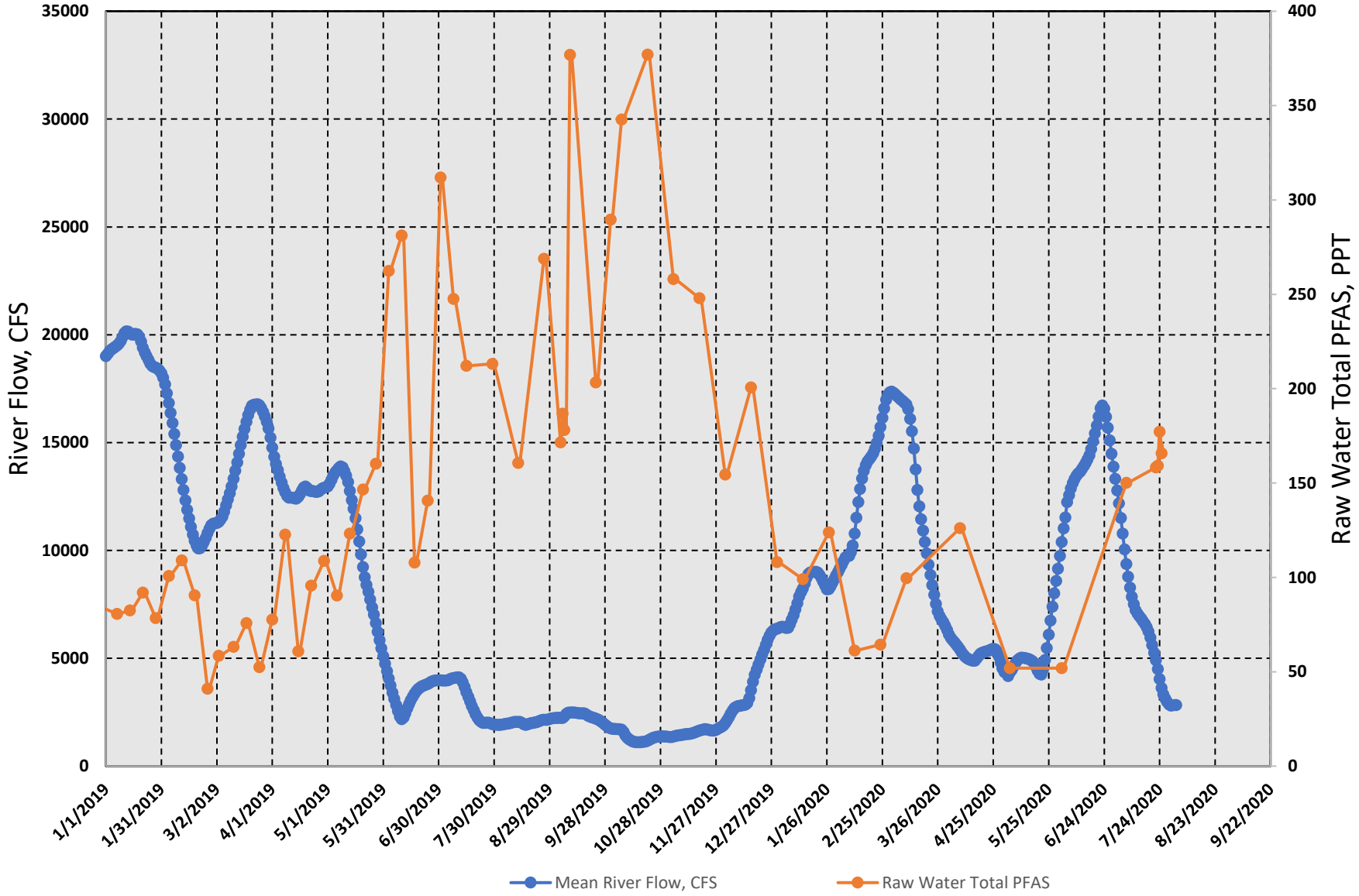
Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	2/11/2020	ND	6.90	ND	ND	ND	ND	ND	**69.29**	**40.93**	**59**
Sweeney Raw	2/24/2020	ND	6.13	ND	ND	ND	ND	ND	**64.40**	**27.5**	**43**
Sweeney Finished	2/25/2020	ND	6.62	ND	ND	ND	ND	ND	**69.93**	**41.75**	**60**
Sweeney Raw	3/9/2020	ND	6.32	ND	ND	ND	ND	ND	**99.64**	**64.73**	**65**
Sweeney Finished	3/10/2020	ND	6.84	ND	ND	ND	ND	ND	**79.32**	**51.3**	**65**
Sweeney Raw	4/6/2020	0.66	10.10	ND	ND	ND	ND	ND	**126.07**	**62.18**	**49**
Sweeney Finished	4/7/2020	ND	5.23	ND	ND	ND	ND	ND	**50.37**	**32.57**	**65**
Sweeney Raw	5/4/2020	1.20	11.40	ND	ND	ND	ND	ND	**117.58**	**56.29**	**48**
Sweeney Finished	5/5/2020	ND	5.19	ND	ND	ND	ND	ND	**48.56**	**30.56**	**63**
Sweeney Raw	6/1/2020	ND	4.36	ND	ND	ND	ND	ND	**51.87**	**16.72**	**32**
Sweeney Finished	6/2/2020	ND	3.74	ND	ND	ND	ND	ND	**41.50**	**26.61**	**64**
Sweeney Raw	7/6/2020	0.71	8.35	ND	ND	ND	1.19	ND	**150.15**	**93.55**	**62**
Sweeney Finished	7/7/2020	ND	7.50	ND	ND	ND	ND	ND	**93.85**	**66.24**	**71**
Sweeney Raw	7/22/2020	0.99	9.51	ND	ND	ND	1.25	ND	**158.32**	**99.07**	**63**
Sweeney Raw	7/23/2020	0.77	9.31	ND	ND	ND	1.32	ND	**159.18**	**97.58**	**61**
Sweeney Raw	7/24/2020	0.83	9.87	ND	ND	ND	1.58	ND	**177.20**	**119.95**	**68**
Sweeney Raw	7/25/2020	0.68	9.39	ND	ND	ND	1.91	ND	**165.82**	**109.7**	**66**
Sweeney Raw	8/2/2020	0.675	7.49	ND	ND	ND	ND	ND	**127.48**	**79.87**	**63**
Sweeney Raw	8/3/2020	0.804	8.94	ND	ND	ND	ND	ND	**119.76**	**67.96**	**57**
Sweeney Raw	8/4/2020	0.771	9.51	ND	ND	ND	ND	ND	**120.64**	**61.94**	**51**
Sweeney Raw	8/5/2020	0.593	8.94	ND	ND	ND	ND	ND	**113.47**	**59.23**	**52**
Sweeney Finished	8/5/2020	ND	9.69	ND	ND	ND	ND	ND	**115.43**	**83.13**	**72**

NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT B
TO AMENDED INTERVENOR COMPLAINT

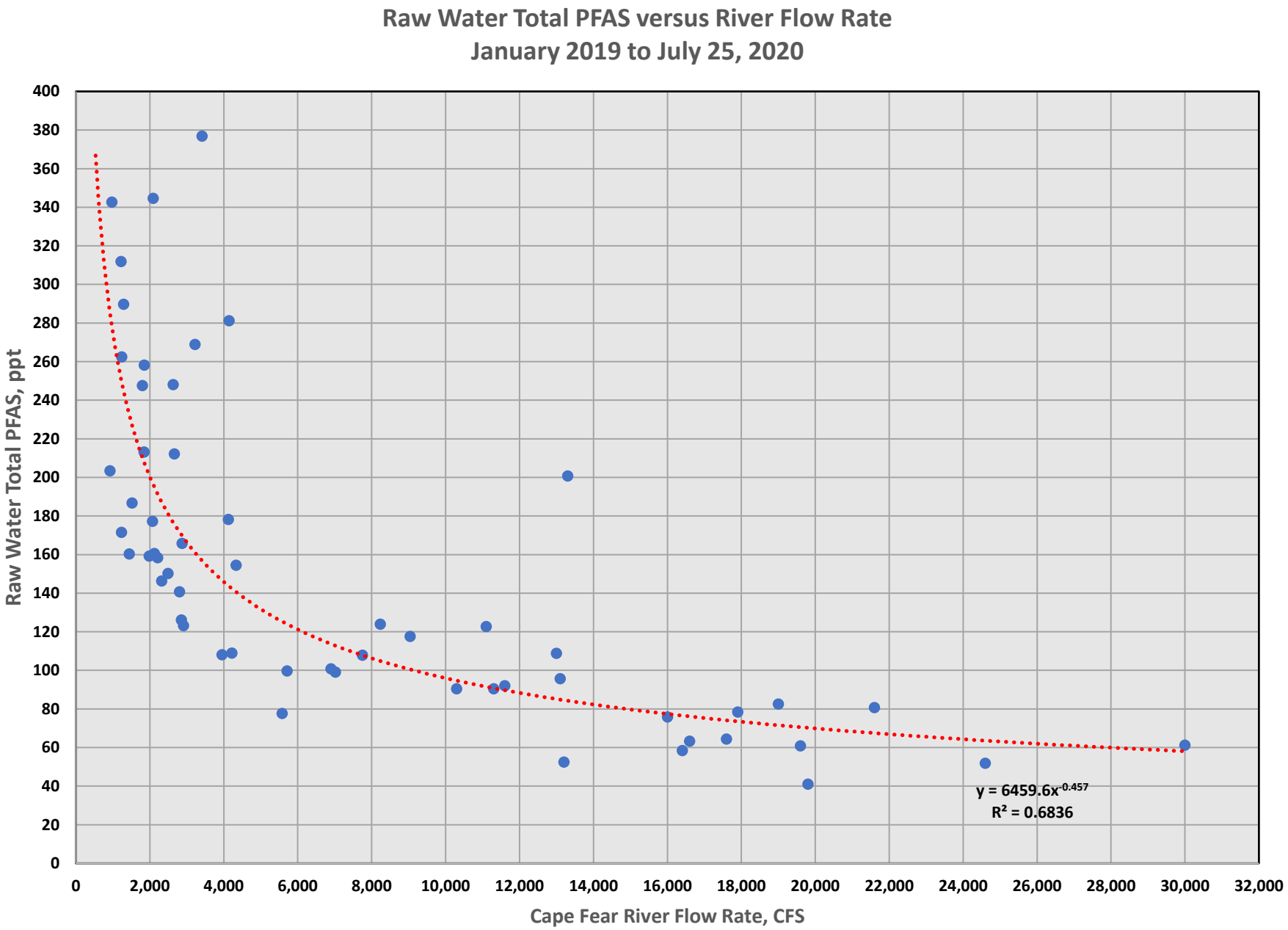
Cape Fear River Flow vs Raw Water Total PFAS

USGS 02105769 Cape Fear River at L&D #1, Kelly, NC



NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT C
TO AMENDED INTERVENOR COMPLAINT


Raw Water Total PFAS versus River Flow Rate
January 2019 to July 25, 2020
Raw Water Total PFAS, ppt
Cape Fear River Flow Rate, CFS
y = 6459.6x^-0.457
R² = 0.6836
0
20
40
60
80
100
120
140
160
180
200
220
240
260
280
300
320
340
360
380
400
0
2,000
4,000
6,000
8,000
10,000
12,000
14,000
16,000
18,000
20,000
22,000
24,000
26,000
28,000
30,000
32,000

NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT D
TO AMENDED INTERVENOR COMPLAINT

Technical Review of Cape Fear River PFAS Loading Reduction Plan for Cape Fear Public Utility Authority (CFPUA)

September 27, 2019

PREPARED FOR

Cape Fear Public Utility Authority
235 Government Center Drive
Wilmington, NC 28403

PREPARED BY

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TETRA TECH

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1.0 BACKGROUND

Chemours Company issued the Cape Fear River PFAS Loading Reduction Plan (Geosyntec, 2019) to the North Carolina Department of Environmental Quality (NCDEQ) and Cape Fear River Watch (CFRW) on August 26, 2016 in response to the Consent Order (CO) entered by the Bladen County Superior Court (paragraphs 12 and 11.1) on February 25, 2019. The CO was issued regarding emissions and discharges of per- and polyfluoroalkyl substances (PFAS), including hexafluoropropylene oxide dimer acid (HFPO-DA; 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)propanoic acid) and the ammonium salt of HFPO-DA, which has the trade name of GenX®, from the Fayetteville Works facility. GenX is used to manufacture high performance fluoropolymers. GenX replaces the ammonium salt of perfluorooctanoic acid (PFOA), which was phased out of production in 2009 because PFOA is persistent in the environment, bioaccumulates, and is toxic. At that time the Fayetteville Works facility was owned and operated by E.I. du Pont de Nemours and Company (DuPont). The Chemours Company was founded in July 2015 as a spin-off from DuPont.

In 2009 EPA authorized the manufacture of GenX; however, EPA also issued an order that required DuPont to capture, at an overall efficiency of 99%, new chemical substances from wastewater effluent and air emissions (premanufacture notice numbers P-08-508 and P-08-509). News broke regarding high levels of GenX and PFAS in the Cape Fear River and downstream potable waters in 2017 – spurring further environmental investigations and facility inspections. Shortly thereafter NCDEQ filed a Complaint alleging violations of the premanufacture order due to evidence in downstream waters of PFAS discharges by Chemours and DuPont, ultimately leading to the August 26, 2016 CO.

The Fayetteville Works facility is located in Bladen County, NC on the west side of the Cape Fear River just upstream of the William O, Huske Lock and Dam (Lock and Dam #3). The facility includes two Chemours manufacturing areas, the Monomers IXM area and the Polymer Processing Aid Area (PPA area), as well as an onsite process Wastewater Treatment Plant (WWTP) and Power Area (Geosyntec, 2019). In addition, manufacturing areas on the facility grounds are leased to Kuraray America Inc. for Butacite® and SentryGlas® production and to DuPont for polyvinyl fluoride (PVF) resin manufacturing.

The Chemours Fayetteville Works facility is located about 55 miles upstream of the Kings Bluff water intake on the Cape Fear River where the Cape Fear Public Utility Authority (CFPUA) withdraws water for treatment and potable use distribution. Elevated levels of PFAS have been observed in both the raw source water from the Cape Fear River and finished water at the CFPUA's Water Treatment Plants (WTPs). Traditional water treatment processes do not successfully remove GenX and other PFAS (Hopkins et al., 2018). The effectiveness of currently implemented and proposed PFAS pollution control strategies adopted by Chemours directly affect the quality of CFPUA's intake water and community exposure to these substances.

In light of these concerns, CFPUA engaged Tetra Tech to conduct a technical review of the PFAS Loading Reduction Plan and associated environmental assessments. Specifically, CFPUA requested input on the technical soundness of the surface and groundwater modeling, reasonableness of the assumptions applied in the analyses, reasonableness of the seven proposed strategies for reducing PFAS loads, identification of critical gaps in the analyses, and recommendations for additional studies related to reducing PFAS loads.

The Cape Fear River PFAS Loading Reduction Plan itself consists of 33 pages plus a cover letter, but is supported by five technical appendices: 1) PFAS Mass Loading Model, 2) Seeps and Creeks

Investigation Report, 3) Outfall 002 Assessment, 4) Terracotta Pipe Grouting Report, and 5) HFPO-DA Loading Reduction Estimates, all of which were completed by Chemours' consultant, Geosyntec Consultants of NC, P.C. The PFAS Loading Reduction Plan includes seven proposed actions aimed to reduce PFAS loading to the Cape Fear River. Findings from the review of the plan and supporting technical reports are discussed in this memorandum.

To better understand the relationship between river flow rate at the Kings Bluff intake and PFAS concentrations, CFPUA has developed a correlation analysis between the variables. CFPUA requested a technical review of the correlation analysis, which is also discussed in this memorandum as are implications related to the loading reduction plan.

2.0 TECHNICAL REVIEW

The PFAS loading reduction plan is informed by the PFAS Mass Loading Model (MLM), which evaluates contributions of PFAS to the Cape Fear River from nine pathways (Figure 1):

- Upstream river water and groundwater
- Willis Creek (north of the facility)
- Direct atmospheric deposition on the river in the vicinity of the facility
- Outfall 002
- Onsite upwelling groundwater
- Four identified onsite channelized seeps
- Old Outfall 002
- Offsite groundwater
- Georgia Branch Creek (south of the facility)

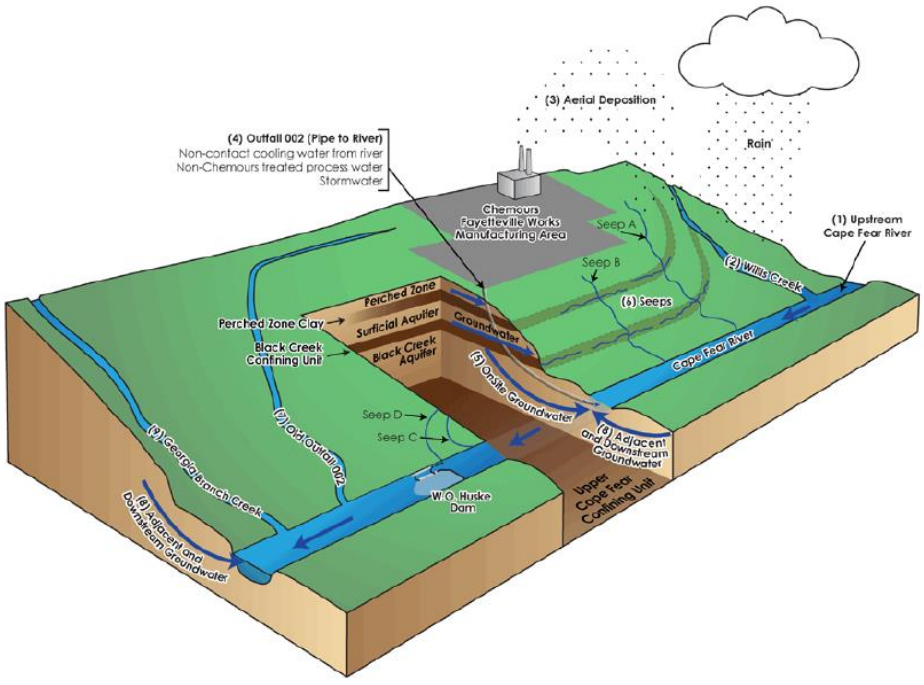

Figure 1. PFAS Transport Pathways (Geosyntec, 2019; Figure 5)

The MLM incorporates analyses and findings from the other appendices, such as the Seeps and Creeks Investigation Report that is used for characterizing groundwater conditions and contributions. Comments on the technical soundness, reasonableness of the assumptions applied, and critical gaps are discussed in the sections below. Key comments are summarized in Table 1.

Table 1. Key Comments from the Technical Review

Brief Description of Comment	Section (Comment Number)
Lack of adequate groundwater monitoring data and application of post-Hurricane Florence data.	2.1 (#1) and 2.2 (#1 and #5)
The modeling applied insufficient extents for resurfacing groundwater, resulting in potentially underestimated loads to the river.	2.2 (#2 and #3)
Limited scope of atmospheric deposition modeling (e.g., only HFPO-DA; seemingly conservative application of October 2018 conditions; limited spatial extent)	2.1 (#4)
Lack of information about the extent, magnitude, and impacts of offsite PFAS groundwater and soil contamination that may continue to contribute PFAS to the river.	2.2 (#4) and 2.3 (#7)
Lack of information to characterize PFAS contamination of sediment in the Cape Fear River bed and riparian wetlands.	2.2 (#6) and 2.3 (#7)
Implementation timing and ongoing risks for untreated sources.	2.3 (#1 and #2)
Lack of information regarding the effectiveness of treatment technologies.	2.3 (#3)
Need for notification requirements regarding spills or other releases since no production related changes have been required to date.	2.3 (#5)
Concerns regarding discharges of Kuraray process wastewater shown to contain elevated PFAS concentrations.	2.3 (#6)

2.1 TECHNICAL SOUNDNESS

This section summarizes our concerns regarding the technical soundness of data that has been assembled and cited to support conclusions in Cape Fear River PFAS Loading Reduction Plan and supporting appendices.

1. Onsite groundwater sampling data used to estimate mass loading to the river is based on a single round of samples collected primarily post Hurricane Florence – four of the five well samples in Appendix A are from late October – early November 2018, while the hurricane occurred in September 2018 with over 12 inches of rain recorded in nearby Fayetteville during the hurricane. This rainfall (and associated infiltration) may have significantly impacted short-term groundwater

sampling data, thus the representativeness of the data used is in question, especially since no other sampling data for the wells were provided for comparison purposes.

2. Onsite and offsite groundwater (transport pathways 5 and 8) PFAS concentrations used for the mass loading model are not provided in Table 3 of the MLM report. Is there a reason why these were specifically excluded while all other transport pathways had concentrations provided? What are the concentrations that were used?
3. It is unclear how groundwater south of the plant between Old Outfall 2 and Georgia Bank Creek was handled. Was groundwater in this area included in the onsite or offsite groundwater mass loading calculations? What parameters were used in the evaluation of contributions to the river from this area?
4. Previously reported deposition contours for air emissions from the Fayetteville Works facility were used to quantify the atmospheric deposition load in the MLM (ERM, 2018). Estimated deposition rates were combined with the average Cape Fear River surface area and estimated residence time to estimate a mass loading from aerial deposition to the river. The deposition load to the river surface was only evaluated for a ~3.5 km segment of the river near the facility. Key concerns regarding the modeling analysis follow, and critical gaps in the overall study related to atmospheric deposition are discussed in the next subsection. Note that some information discussed here is presented in the atmospheric deposition modeling report (ERM, 2018).
    a. The atmospheric deposition modeling focuses solely on HFPO-DA (ERM, 2018). To estimate the atmospheric deposition load of other PFAS compounds (non-HFPO-DA) for the MLM, concentration ratios derived from well monitoring samples are applied. The report, however, lacks proof that ratios from well measurements are directly applicable to air concentrations. Indeed, the ratios are likely to be different as PFAS compounds volatility, airborne transport, and subsurface soil sorption characteristics are not linearly related (ITRC, 2018). Therefore, this is not a reasonable assumption given the lack of evidence. The report also does not describe how the air transport and deposition of other PFAS compounds (non-HFPO-DA) differs from that of HFPO-DA.
    b. The MLM applies expected not actual emissions from the facility for October 2018. The MLM does not thoroughly discuss how factors that influence variability in air transport and deposition (e.g., fluctuations due to weather) are addressed. It is unclear if the results applied represent a single month (i.e., October 2018) extrapolated to represent annual deposition or if annual deposition is characterized by modeling emissions, transport, and deposition over a multi-year period. If it is the former, the application of October 2018 seems to be conservative; simulations of PFAS deposition for May 2018 are more widespread compared to October 2018. According to Table C-1 the same emission rates are applied for both (May and October 2018) scenarios, which means the differences in the extent of deposition are due to atmospheric conditions. Application of conditions for a single month is not reasonable for evaluating the annual load and the MLM should account for variability in conditions that impact the load. If in fact the atmospheric deposition modeling used to inform the MLM simulated a multi-year period, the report should clarify the methods. In addition, it is important that the impacts of intra- and inter-annual variability are discussed, including fluctuating emissions from the facility (i.e., due to operations cycling) and weather (e.g., wind direction and speed).
    c. Dilution factors are applied to estimate resulting concentrations in groundwater wells surrounding the property for various atmospheric deposition scenarios, however, the approach assumes zero concentration in existing aquifer water. Thus, the resulting

groundwater concentrations presented are biased low. [Note this information does not seem to be applied in the MLM.]

5. It is noted in Section 2.1.5. of the "Seeps and Creeks" appendix that samples were collected to avoid inclusion of suspended solids. In the final bullet of Section 3.4 of the Outfall 002 Assessment report it is stated that no relationship between TSS and total or dissolved PFAS was found (although details of the analysis are not provided). However, this conflicts with the fact that elevated PFAS concentrations at Location 22 are attributed to sediment clogging the autosampler (Outfall 002 Assessment report). Sorption of PFAS compounds is complex because the compounds have a lipophilic head and a hydrophobic tail. Thus, a clear relationship to TSS is not expected. A relationship to organic carbon on a PFAS species-by-species basis is likely yet was not examined.
6. The MLM approximates loading rates for each pathway based on PFAS concentration and flow data. The validity of the results for certain pathways is impacted by sparse monitoring records. For example, only a single sample was applied to characterize the upstream load (Section 4.1), even though elevated PFAS levels have been observed in upstream waters such as the Haw River (Barnes, 2019). Using a single sample to estimate the long-term load is not sufficient and additional monitoring should be conducted to characterize the upstream load across various seasons and flow regimes. It is stated in Section 4.5 that all EPA 537 PFAS compounds did not originate from the site as these were present in intake water. Therefore, EPA 537 PFAS compounds were assigned a zero concentration for the MLM. It can be deferred (although it is not explicitly stated) that this finding is based on the single upstream sample. Additional sampling is needed to evaluate the potential contribution of EPA 537 PFAS from the site.
7. No explanation is provided as to why some EPA 537 PFAS sampling method substances are reported as "NS" – defined as compound was not analyzed for in collected sample(s) or sample was not collected. Due to the lack of monitoring for these compounds, the total PFAS concentrations and loads reported in the study may be an underestimate of actual total PFAS concentrations and loads.
8. The DVM Narrative Reports show that many of the collected samples applied in the MLM did not meet sampling protocols (e.g., due to exceeded hold time). In addition, there are several cases where the dissolved concentration exceeds that of the total concentration for a PFAS substance (Table 10 Analytical Results – Stormwater Sampling). These data quality concerns contribute uncertainty to the monitoring and modeling results.
9. Results from TestAmerica were pending from the Outfall 002 monitoring at the time the report was issued. Results presented are from the onsite Chemours lab. The report does not specify if the Chemours lab is approved through the Resource Conservation and Recovery Act (RCRA). The report and modeling should be updated to incorporate the TestAmerica records.
10. HFPO-DA reductions from 2017 and 2019 in the load to the Cape Fear River are presented in the HFPO-DA Loading Reduction Estimates report. For both 2017 and 2019 monitoring from a single day was applied to estimate a typical daily load, which was directly extrapolated to generate an annual load (by multiplying by the number of days per year). The river flow applied to compute the annual load estimate for 2019 was less than one-third of the river flow applied to compute the annual load estimate for 2017, which falsely skews (overestimates) the reported percent reductions in loading to the Cape Fear River. It is not reasonable to assume that monitoring from a single day can be used to compute an accurate annual load. Recent load estimates computed by CFPUA based on more frequent monitoring at Lock and Dam #1 are higher. The analysis

should be redone and samples from multiple monitoring events spanning various seasons and flows should be applied for characterizing baseline and current loads and associated reductions.

2.2 CRITICAL GAPS

1. Overall, there is a significant lack of site-specific data regarding groundwater conditions at the facility. The report indicates that a total of five monitoring wells were available and used in the mass loading evaluation, which is not nearly adequate for delineating site geologic/hydrogeologic conditions and groundwater impacts considering the three groundwater flow systems involved. The report also indicates that additional groundwater characterization work is planned/underway for the site, which should provide data to more accurately portray onsite groundwater impacts to the river and improve the representativeness of the loading model. Hydrogeologic characteristics were in many cases estimated based on literature values and/or empirical evidence – generic ranges for hydraulic conductivity were used from general hydrogeology references, and groundwater flow gradients were estimated from water levels in riverside wells and a river gauging level remote from the site. It is important to collect adequate site-specific data to use in developing a technically sound detailed hydrogeologic conceptual site model that encompasses all three groundwater flow zones identified at the site (perched zone, surficial aquifer, and Black Creek aquifer) for quantifying groundwater flow rates and volumetric discharges/mass loading to the river.
2. Using observed mass loading at Bladen Bluffs, the MLM was calibrated through the adjustment of the following parameters: hydrologic conductivity for the Upper and Lower portions of the Black Creek Aquifer, groundwater discharge length (i.e., area contributing resurfacing groundwater to the river), and an offsite gradient adjustment factor. The rationale for modifying the discharge area for groundwater during model calibration iterations (only 40% to 75% of the total area was used) is unclear – all groundwater in the three flow zones identified (perched zone, surficial aquifer, Black Creek aquifer) should eventually discharge to the Cape Fear River either via direct discharge (Black Creek aquifer) or via seeps and surface water. Clearly the onsite groundwater discharge area length is significantly under-represented as described in Table D-2 of the onsite groundwater flow estimate (2,900 feet), which results in an under-estimation of onsite groundwater discharge from the Chemours site to the river. The calibration process was used as the rationale for this reduced length, however, the calibration process should be constrained to accurately reflect site conditions. Assuming 100% discharge of the Black Creek aquifer to the river would increase discharge/mass loading to the river significantly.
3. Similar to the previous comment, groundwater upwelling to the river is assumed to be less than 100%. Based on a USGS report regarding groundwater flow in the Coastal Plain Aquifer System of North Carolina, some shallow groundwater in the area may resurface as baseflow to the Cape Fear River while some may resurface further downstream (Giese et. al., 1991); however, additional field information is needed to support this parameterization. The assumed aquifer thickness for offsite groundwater discharge to the river is not provided – what was assumed and what is the basis for the assumption? Finally, a hydraulic conductivity value of 2.55 x 10-4 m/s was used for calculating offsite groundwater discharge to the river; however much lower K values were assumed for onsite groundwater (Black Creek aquifer). It is reasonable to assume that offsite shallow groundwater across the river is from the same formation; why the difference in K values? This would underestimate the relative mass loading via onsite groundwater versus

offsite groundwater. In addition, the Black Creek aquifer is likely to be slightly thicker on the other side of the River as it is generally down-dip; was this taken into account?

4. The loading analysis excludes deposition to surrounding land (wet or dry) that is stored in offsite soils, transported to streams via erosion, and leached into groundwater. These mechanisms and associated loadings have yet to be properly quantified. An investigation for the DuPont Washington Works plant near the Ohio-West Virginia border found contamination from atmospheric deposition up to 20 miles from the plant (Zevitas and Zemba, 2018). It is plausible that air emissions at the Fayetteville Works facility were/are transported further than assumed in the loading analysis, deposited, stored in soils, and leached into groundwater that resurfaces as baseflow to the river. Wells exhibiting high levels of PFAS contamination opposite of observed groundwater pathways (e.g., wells on the east side of the river) support this concept (ERM, 2018). This also could explain why concentrations and loads of some PFAS compounds are higher at the Kings Bluff intake compared to Bladen Bluffs, specifically during June 2019 (Table 7-A and Table 7-B), but the MLM was only calibrated at the Bladen Bluffs intake located about five miles downstream of the facility. CFPUA analyzed the relationship between raw water total PFAS and river flow rate using 2019 monitoring records (Figure 2). Elevated PFAS concentrations occur during periods of low flow. Given the halting of the release of process wastewater by Chemours, the elevated concentrations are likely attributable to onsite and offsite groundwater, releases from sediment bed stores, and/or currently unidentified other point sources. Therefore, a critical gap in the current analysis framework is that the extent, magnitude, and impacts of offsite PFAS groundwater and soil contamination has not been evaluated. Releases of contaminated groundwater, diffusion from contaminated sediment, and erosion of contaminated soils may contribute PFAS to the CFPUA's intake water following the implementation of the proposed control strategies (Section 2.3). Additional offsite monitoring and modeling is needed to understand the long-term implications on downstream water quality.

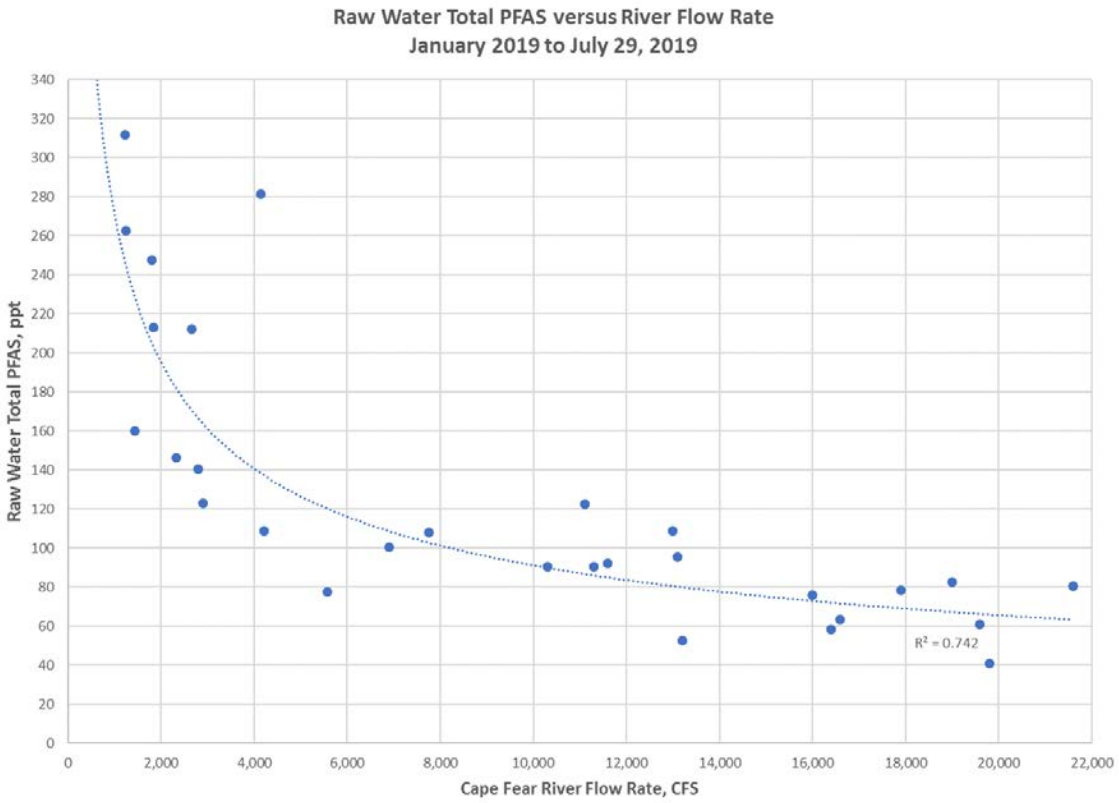

Figure 2. PFAS Concentrations and Cape Fear River Flow (provided by CFPUA)

5. For offsite groundwater where airborne deposition is considered to be the mechanism for PFAS transport to groundwater, prevailing wind directions should be utilized to estimate groundwater concentrations and mass loading to the river through offsite groundwater discharge to the river (see supplemental wind rose). For example, the predominant wind directions measured at nearby Fayetteville are from the southwest and from the northeast, which generally correlates with Figure E-2. For the area east and southeast of the site, however, there is very little data (few residential wells) and a review of Figure E-2 suggests that PFAS loading to groundwater in this area may be underestimated. The sampling data for wells west and northwest of the site (a much larger data set) could, however, be used to project/estimate groundwater concentrations/mass loading due to airborne deposition in the east-southeast area as the proportion of west and northwest winds (from west to east) is similar to/slightly higher than east/southeast winds (1998 – 2019 data). As currently configured, it appears that offsite groundwater mass loading to the river from east/southeast of the site may be underestimated.
6. A critical gap in the technical framework is that no sampling has been reported to characterize PFAS contamination of sediment in the Cape Fear River bed or riparian wetlands. It is anticipated that historic emissions and discharges from the facility have accumulated and caused long-term residual contamination of the river and riparian wetlands. Diffusion from such contaminant stores could provide a long-term source of PFAS contamination to the river. Scouring of contaminated sediment from the river bed or banks during high flow events could also elevate PFAS concentrations in downstream intake water. Sediment sampling along the mainstem should be conducted to characterize the extent and magnitude of sediment bed and riparian wetland contamination and the potential associated risks. Areas prone to excess build-

up of organic matter, such as sluggish riverine swamps and pools behind the locks and dams, face a higher risk of exhibiting elevated sediment PFAS concentrations. A comprehensive study is needed to characterize sediment PFAS contamination in the Cape Fear River bed that includes assessment of potential contamination hot-spots, such as the Kings Bluff intake canal situated near the Cape Fear River Lock and Dam #1. In addition, onsite sediment sampling has been sparse and should be extended to all concentrated surface flow pathways (e.g., open channel to Outfall 002).

7. A flow-based PFAS loading curve prepared by CFPUA for 2019 is shown in Figure 3. Higher PFAS loads are associated with higher flows, which indicates that stormwater and/or sediment bed erosion (as described in the previous comment) contributes PFAS to the river. Yet, these sources are poorly quantified, including both onsite and offsite stormwater contributions.

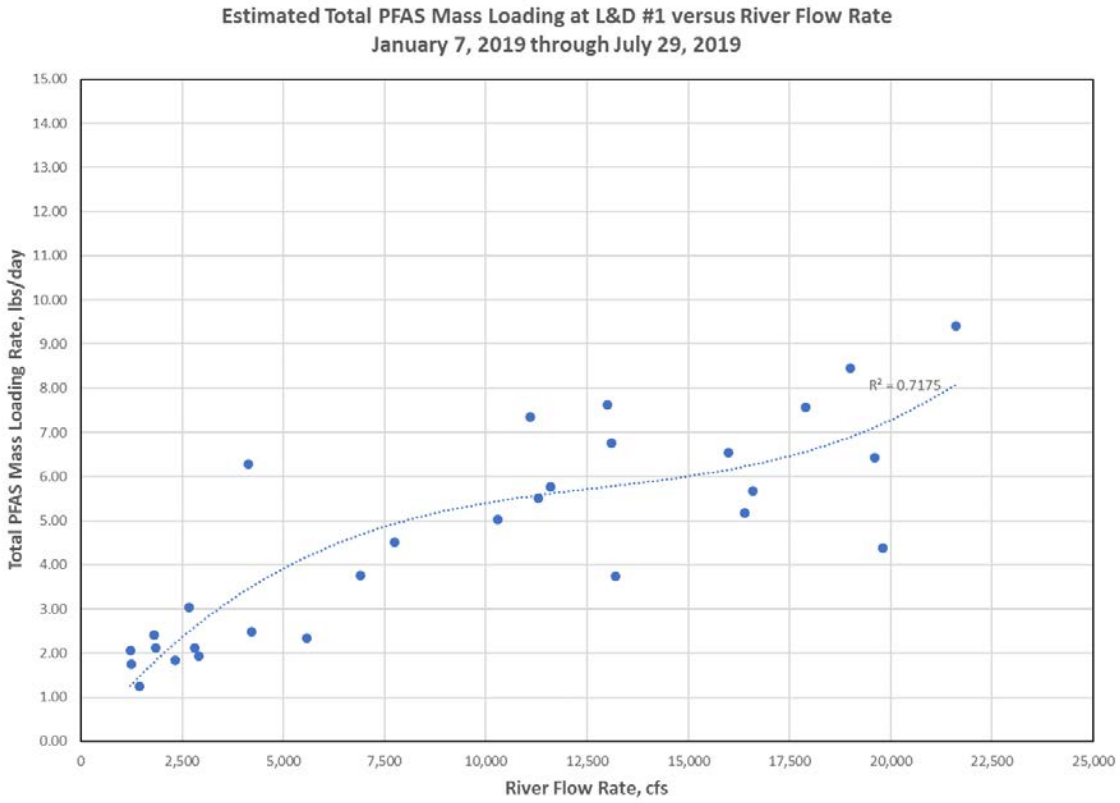

Figure 3. Flow-based PFAS Loading Rate (provided by CFPUA)

8. A mass balance evaluation of flow from the facility to the river is not provided in the Geosyntec (2019) report and is needed to verify the overall annual flow balance applied in the MLM. Such an evaluation should incorporate flow sources, storages, and discharges surface and subsurface discharges from the facility study area.
9. The possibility of additional diffuse discharges from the perched zone/shallow aquifer in other areas along the river should be investigated.

2.3 LOADING REDUCTION PLAN AND STRATEGIES

Chemours has previously implemented PFAS loading control measures: 1) eliminating process wastewater discharges (excluding those from site tenants Kuraray and DuPont), 2) air emission controls, 3) lining the facility's cooling water channel and sediment ponds, and 4) extraction of groundwater discarded offsite.

Seven new control strategies are proposed for the Chemours Fayetteville Works facility in the current plan (Geosyntec, 2019): 1) capture and treat Old Outfall 002 water (within two years), 2) capture and treat groundwater from seeps (within five years), 3) targeted sediment removal from conveyance network (within one year), 4) develop a stormwater pollution prevention plan (within one year), 5) targeted stormwater source control and/or treatment (within four years), 6) decommission and replacement of remaining terracotta piping (that carried industrial process wastewater; within two years), and 7) assessment of potential groundwater intrusion into the conveyance network (within five years). All proposed actions are to be implemented within five years and are onsite controls (on the Fayetteville Works property). Key comments regarding the plan and strategies follow.

1. It is stated on page v. regarding the control strategies that "Four of these actions would be implemented within two years of Consent Order Amendment and three of the actions would be implemented within five years of Consent Order Amendment (assuming all necessary permits and authorizations are provided in a timely manner)." Control actions may not be implemented on schedule due to the ambiguity of this statement, which poses a risk to downstream users.
2. The actions related to groundwater (#2 and #7) are set to take the longest time to implement yet are the top loading sources according to the MLM. Plans to evaluate and address groundwater and stormwater are still being developed, thus, loadings from these sources remain a vulnerability to downstream water supplies.
3. No specific treatment option is listed for captured onsite surface and groundwater, nor is the effectiveness of the proposed treatment methods demonstrated. Without these specifications it is uncertain if the loading reduction plan will effectively mitigate PFAS pollution. An onsite study evaluating the proposed treatment technologies and observed effectiveness (i.e., percent removal, treated concentrations and loads) should be required.
4. The onsite perched zone pumping described in the report (Section 3; Completed Reduction Actions) amounts to <0.1 gpm. Has there been any evaluation to determine whether the pumping rate can be increased via more aggressive pumping or additional groundwater extraction points to enhance capture of this highly impacted groundwater?
5. No manufacturing process changes have been required to date. Spills or unknown leaks or emissions at the facility remain a risk to CFPUA's source water. In paragraph 15 of the CO, Chemours is to provide notification to downstream water utilities in the event of elevated PFAS releases through Outfall 002. However, CFPUA should consider requesting spill (or other contaminant release) notification requirements that are more comprehensive.
6. Discharge of Chemours' process wastewater has been halted and the waste is injected into subsurface storage out-of-state. However, elevated HFPO-DA and PFMOAA concentrations were also observed in Kuraray process wastewater, which continues to be discharged from the onsite WWTP (page 18 of the Outfall 002 Assessment) via Outfall 002. Sources causing contamination of Kuraray process wastewater have not been identified and quantified. Furthermore, control strategies have not been required or proposed for the Kuraray process wastewater.

7. No PFAS loading control strategies are recommended for contaminated offsite soils, offsite groundwater, or river sediment due to the lack of evaluation of these sources (see Section 2.2). Additional strategies may be needed following the evaluation of these sources to ensure protection of downstream water quality.

All monitoring applied in the assessment appears to have been conducted by Geosyntec and contracted labs for Chemours. DEQ can require split sampling (samples provided to DEQ for parallel testing) per the Consent Order. Split sampling would be beneficial from the perspective of CFPUA for quality assurance and control checking, therefore, CFPUA should inquire about completed split sampling and the findings, or the rationale for why split sampling has not occurred to date.

3.0 REFERENCES

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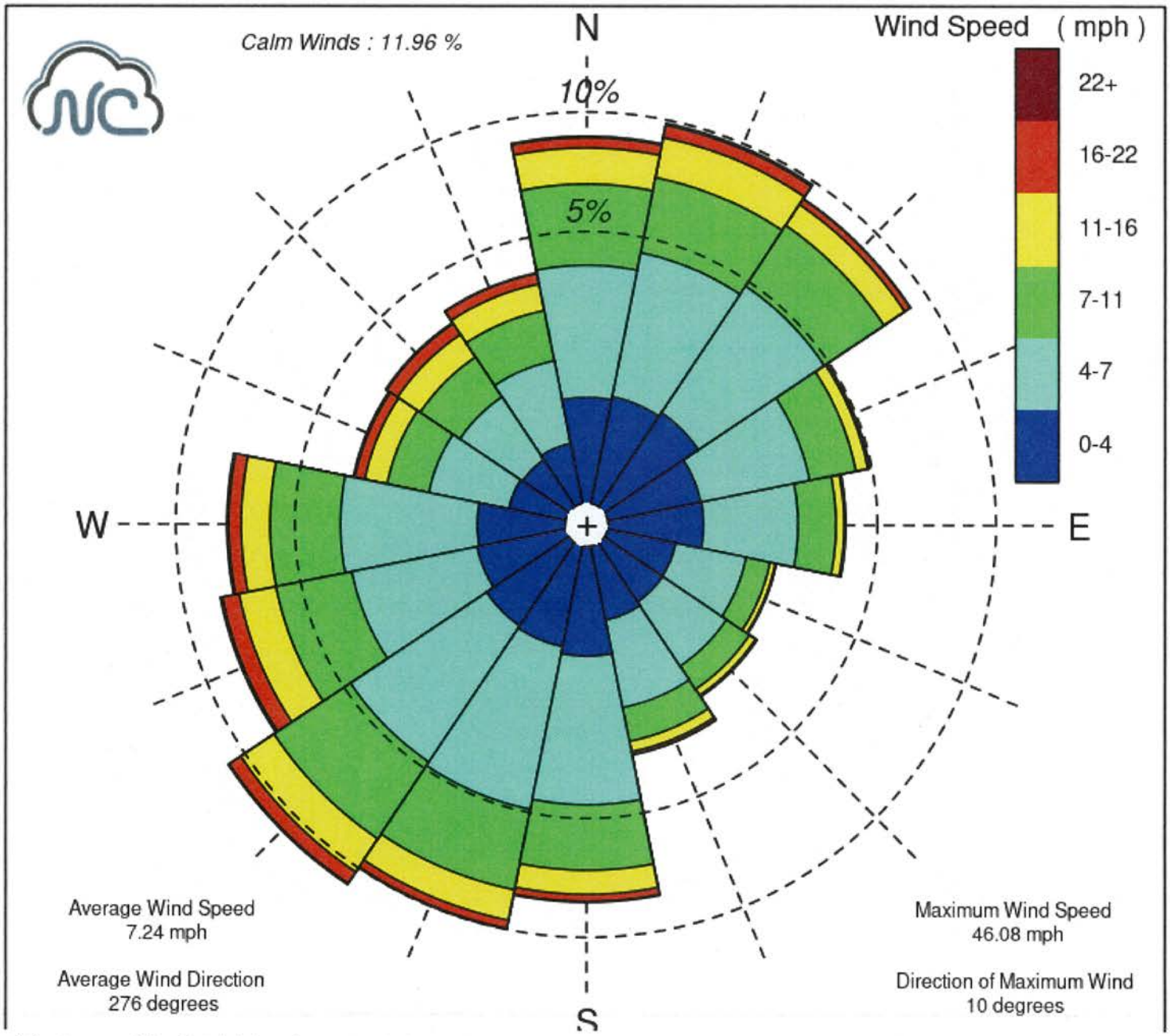
Wind Rose for Fayetteville Airport (KFAY)
Jan. 10, 1998 to Sep. 19, 2019



Shows wind blowing from a particular direction

U.S. Department of Commerce
National Oceanic & Atmospheric Administration
National Environmental Satellite, Data, and Information Service
Current Location: Elev: 192 ft. Lat: 35.1092° N Lon: -78.8944° W
Station: **FAYETTEVILLE 2.6 N, NC US US1NCCM0038**

Record of Climatological Observations

These data are quality controlled and may not be identical to the original observations.
Generated on 09/19/2019

National Centers for Environmental Information
151 Patton Avenue
Asheville, North Carolina 28801

Observation Time Temperature: Unknown Observation Time Precipitation: Unknown

Year	Month	Day	Temperature (F)			Precipitation					Evaporation		Soil Temperature (F)					
			24 Hrs. Ending at Observation Time		At Observation	24 Hour Amounts Ending at Observation Time				At Obs. Time			4 in. Depth			8 in. Depth		
			Max.	Min.		Rain, Melted Snow, Etc. (in)	Flag	Snow, Ice Pellets, Hail (in)	Flag	Snow, Ice Pellets, Hail, Ice on Ground (in)	24 Hour Wind Movement (mi)	Amount of Evap. (in)	Ground Cover (see *)	Max.	Min.	Ground Cover (see *)	Max.	Min.
2018	09	01																
2018	09	02				0.00												
2018	09	03																
2018	09	04				0.00												
2018	09	05				0.00												
2018	09	06				0.00												
2018	09	07				0.00												
2018	09	08				0.00												
2018	09	09																
2018	09	10				0.00												
2018	09	11				0.00												
2018	09	12				0.50												
2018	09	13																
2018	09	14				0.78												
2018	09	15				3.67												
2018	09	16				3.02												
2018	09	17				5.07												
2018	09	18				1.59												
2018	09	19				0.00												
2018	09	20				0.00												
2018	09	21				0.00												
2018	09	22				0.00												
2018	09	23				0.00												
2018	09	24				0.00												
2018	09	25				0.00												
2018	09	26				0.00												
2018	09	27				0.00												
2018	09	28				0.33												
2018	09	29				0.00												
2018	09	30				0.00												
		Summary				14.96		0.0										

Empty, or blank, cells indicate that a data observation was not reported.

*Ground Cover: 1=Grass; 2=Fallow; 3=Bare Ground; 4=Brome grass; 5=Sod; 6=Straw mulch; 7=Grass muck; 8=Bare muck; 0=Unknown

"s" This data value failed one of NCDC's quality control tests.

"T" values in the Precipitation or Snow category above indicate a "trace" value was recorded.

"A" values in the Precipitation Flag or the Snow Flag column indicate a multiday total, accumulated since last measurement, is being used.

Data value inconsistency may be present due to rounding calculations during the conversion process from SI metric units to standard imperial units.

NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT E
TO AMENDED INTERVENOR COMPLAINT

Technical Review of Cape Fear River PFAS Corrective Action Plan for Cape Fear Public Utility Authority (CFPUA)

February 28, 2020

PREPARED FOR

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1.0 EXECUTIVE SUMMARY

This report is a technical review of the Corrective Action Plan (CAP; Geosyntec, 2019a) for remediation of per- and polyfluoroalkyl substances (PFAS) discharged by the Chemours Company Fayetteville Works facility. Comments regarding the technical soundness of the assessments presented in the CAP and critical gaps are discussed in Section 3.0. The main concerns relevant to the Cape Fear Public Utility Authority's (CFPUA) downstream raw water intake are summarized below. Based on the information provided and information lacking, the adequacy of the modeling and CAP cannot be judged.

- The CAP and past reports use an **inconsistent application of PFAS analyte groups** for monitoring, loading analyses, and remediation planning (Section 3.1 #1). It is stated that, except for HFPO-DA, Modified EPA 537 method PFAS do not originate from onsite manufacturing; however, this is inconsistent with some process water samples presented in Characterization of PFAS in Process and Non-process Wastewater and Stormwater Quarterly Report #1 (Table 4, Location ID 16). Loads from the Modified EPA 537 method PFAS are excluded from the mass balance model. As a result, the model may underestimate PFAS loading from the site that impacts downstream water quality.
- The CAP **does not clearly define a baseline period**. The PFAS Loading Reduction Plan and CAP are also missing important information; relative contributions are presented by transport pathway, however, flows, concentrations, and loads to the river (mass of total PFAS per time) are not specified. Without a clear definition of the baseline period and loads, results could be interpreted in a manner that misrepresents progress and the effectiveness of remediation strategies (Section 3.1 #2).
- Multiple **technical issues related to the numerical groundwater model** are discussed in Section 3.1 #7 and Section 3.2 #2 that raise questions about the validity of the model and simulated remediation strategies. The model lacks a validation period to establish the robustness of the calibration. The report does not provide a rationale for the selection of proposed remedies and, based on the limited information provided, it is uncertain if the strategies will effectively capture and treat the PFAS-contaminated groundwater plumes.
- The onsite treatment strategies described in the CAP **neglect components of onsite pathways that may continue to contribute PFAS to the river** (Section 3.2 #1). The strategy specified for Old Outfall 002, for example, targets dry weather flows for treatment and excludes the treatment of wet weather flows that have the potential to transport contaminated sediment to the river. No creek-specific controls are planned for Willis Creek and Georgia Creek and no treatment plans are described for the newly identified seeps (E to M) south of the site. The effectiveness of the proposed treatment measures is uncertain and cannot be evaluated from the material provided in the CAP.
- There is a gap regarding the extent, magnitude, and **loading of PFAS from offsite contaminated soils and groundwater** that could act as long-term sources of PFAS to the river, continuing to impact the quality of raw intake water for CFPUA (Section 3.2 #1 and #4). PFAS contamination from Chemours has been detected in an area of 70 square miles (or more) surrounding the facility. However, because of the extent of the contamination, lack of scalable remediation technologies, and because no groundwater standards have been issued, it is claimed in the CAP that restoring groundwater conditions to PQLs is not feasible, which does not seem to comply with 2L Rules as required by the CO (paragraph 16). PFAS contamination of sediment in the bed and riparian wetlands of the river also remains uncertain. A comparative PFAS loading assessment just downstream of the site and at the CFPUA raw water intake is needed to evaluate offsite loading contributions to the river.

2.0 BACKGROUND

Chemours Company submitted the Cape Fear River PFAS Corrective Action Plan (Geosyntec, 2019a) to the North Carolina Department of Environmental Quality (NCDEQ) and Cape Fear River Watch (CFRW) on December 31, 2019, in response to the Consent Order (CO) entered by the Bladen County Superior Court (paragraphs 11.1 and 12) on February 25, 2019. The CO was issued regarding emissions and discharges of PFAS, including HFPO-DA and the ammonium salt of HFPO-DA, which has the trade name of GenX®, from the Fayetteville Works facility. GenX is used to manufacture high-performance fluoropolymers. GenX replaces the ammonium salt of perfluorooctanoic acid (PFOA), which was phased out of production in 2009 because PFOA is persistent in the environment, bioaccumulates, and is toxic. At that time the Fayetteville Works facility was owned and operated by E.I. du Pont de Nemours and Company (DuPont). The Chemours Company was founded in July 2015 as a spin-off from DuPont.

In 2009 EPA authorized the manufacture of GenX; however, EPA also issued an order that required DuPont to capture new chemical substances from wastewater effluent and air emissions at an overall efficiency of 99 percent (premanufacture notice numbers P-08-508 and P-08-509). News broke regarding elevated levels of GenX and PFAS in the Cape Fear River in 2017 – spurring further environmental investigations and facility inspections. Shortly thereafter, NCDEQ filed a Complaint alleging violations of Title 15A of the North Carolina Administrative Code Subchapter 02L .0202 Groundwater Quality Standards due to evidence of PFAS discharges by Chemours and DuPont, ultimately leading to the CO.

The Fayetteville Works facility is in Bladen County, North Carolina, on the west side of the Cape Fear River just upstream of the William O. Huske Lock and Dam (Lock and Dam #3). The facility includes two Chemours manufacturing areas, the Monomers IXM area and the Polymer Processing Aid Area (PPA area), as well as an onsite process wastewater treatment plant (WWTP) and power area (Geosyntec, 2019b). Manufacturing areas on the facility grounds are leased to Kuraray America Inc. for Butacite® and SentryGlas® production and to DuPont for polyvinyl fluoride (PVF) resin manufacturing.

The Chemours Fayetteville Works facility is located about 55 miles upstream of the Kings Bluff water intake on the Cape Fear River where the Cape Fear Public Utility Authority (CFPUA) withdraws water for treatment and potable use distribution. Elevated levels of PFAS have been observed in both the raw source water from the Cape Fear River and finished water at the CFPUA's Water Treatment Plants (WTPs). Traditional water treatment processes do not successfully remove GenX and other PFAS (Hopkins et al., 2018). The effectiveness of currently implemented and proposed PFAS pollution control strategies adopted by Chemours directly impacts the quality of CFPUA's intake water and community exposure to these substances.

Chemours submitted the Cape Fear River PFAS Loading Reduction Plan (Geosyntec, 2019b) in August 2019 and CFPUA engaged Tetra Tech to conduct a technical review of the report (Tetra Tech, 2019). The review evaluated the technical soundness of the modeling, the reasonableness of the assumptions applied in the analyses, the reasonableness of the proposed strategies for reducing PFAS loads, identified critical gaps, and recommended additional studies related to reducing PFAS loads. Comments most pertinent to CFPUA's downstream water intake included the lack of groundwater data, insufficient extents and lack of information about the extent, magnitude, and impact of offsite groundwater and soil contamination, lack of information necessary to characterize PFAS contamination in the sediment of the riverbed and riparian wetlands, and lack of information regarding the effectiveness of the proposed treatment measures.

A technical review of the CAP is presented in this report. The CAP describes site information, recent receptor monitoring details, a numerical hydraulic groundwater model, PFAS signatures source assessment, recent corrective actions summary, human health and ecological exposure and hazard assessments, proposed remediation activities by source pathway, and performance monitoring plans. The appendices relevant to the fate and transport of PFAS in the environment were also reviewed. This includes Appendix A - On and Offsite Assessment Tables; Appendix B - Additional Corrective Action Plan Tables and Figures; Appendix C - K_{ow}, K_{oc} and Mass Distribution Calculations; Appendix D - Southwestern Offsite Seeps Assessment; Appendix E - PFAS Signatures Assessment; and Appendix H - Numerical Groundwater Modeling Report. CFPUA plans to collaborate with expert Dr. Jamie Dewitt for elements related to human exposure and toxicity, as described in Appendix F - Human Health Screening Level Exposure Assessment of Table 3+ PFAS. The ecological assessment, discussed in Appendix G – Ecological Screening Level Exposure Assessment of Table 3+ PFAS, and Appendix I – Detailed Costs were not reviewed as part of the technical assessment described in this report.

3.0 TECHNICAL REVIEW

Key comments from the technical review of the CAP and supporting appendices are discussed in the following sections. The adequacy of the modeling and CAP cannot be evaluated due to the reasons summarized below.

3.1 TECHNICAL SOUNDNESS

This section summarizes concerns regarding the technical soundness of data and analyses cited to support conclusions in the Cape Fear River PFAS CAP and supporting appendices.

1. Information provided in the quarterly reports indicate that monitoring conducted aligns with specifications in the approved monitoring plan. However, results from the PFAS monitoring tests are inconsistently applied in the assessments. On page xii of the CAP, it states “The PFAS that originate from the Site are referred to as Table 3+ PFAS. The Table 3+ analytical method was developed to analyze PFAS specific to the Site that were identified through non-targeted chemical analyses. Currently, the Table 3+ method can quantitate for 20 PFAS compounds including HFPO-DA, i.e., “GenX”. When examining PFAS at the Site, the sum of these compounds, i.e., total Table 3+ PFAS compounds, is often used to evaluate trends and distributions.” However, in some analysis components Table 3+ PFAS are applied, in other components the assessment is limited to HFPO-DA, and sometimes Modified EPA Method 537 compounds are evaluated. This inconsistency hinders comparison between sources and components of the study (i.e., not always apples-to-apples). Example instances and impacts of this are described below.
    - The CO specifies the PFAS to be monitored for public drinking water and private wells (paragraphs 19-21 and 24) in Attachment C. According to paragraph 11 in the CO, ongoing sampling for process and non-process wastewater and stormwater at the facility is to be conducted for “all” PFAS for which test methods and lab standards have been developed, although these are not explicitly listed. The results described in the quarterly reports seem to include the Table 3+ PFAS and Modified EPA 537 PFAS for most sites, which matches specifications in the monitoring plan. Chemours claims that the Modified EPA 537 PFAS (excluding HFPO-DA) did not originate from the site as these were

already present in the intake water. Modified EPA 537 PFAS other than HFPO-DA are assigned a concentration of zero for onsite transportation pathways in the PFAS mass loading model. However, based on analytical results from the April 2019 monitoring event described in Chemours' first quarterly report, other PFAS (e.g., Perfluoropentanoic Acid) were found in process water from the Chemours Monomers IXM Area (site 16, page 3 of Table 4) at much higher concentrations than found in the background/intake water (later monitoring reports do not include samples from process wastewater). This suggests that some of the other Modified EPA 537 PFAS may originate from manufacturing on the site, but Modified EPA 537 PFAS (except for HFPO-DA) are excluded from the mass loading model and assessments discussed in the CAP (e.g., PFAS signatures). Therefore, it is unclear if the approach abides by the CO requirements and if the approach characterizes PFAS loads from the site accurately. Monitoring results, such as those from onsite and offsite groundwater wells, indicate that the relative proportions of PFAS compounds vary spatially, thus, it cannot be assumed that evaluating HFPO-DA in isolation is representative of other/total PFAS as has been assumed for atmospheric deposition modeling.

- Table 3+ and Modified EPA 537 PFAS methods exclude two PFAS listed in Attachment C of the CO, PFMOPrA, and PFMOBA, which are isomers that have the same chemical formulae as PMPA and PEPA, respectively, but have different chemical structures and CASN numbers. PFHpA listed in Attachment C is not included in the Table 3+ method, although it is included in the Modified EPA 537 method. Monitoring and assessments that are limited to Table 3+ PFAS exclude PFMOPrA, PFMOBA, and PFHpA from Attachment C of the CO.

2. Throughout the report and appendices, reduction targets are expressed as a relative percent reduction compared to an undefined baseline period. Appropriate quantification of the reductions achieved with the implementation of treatment technologies requires a clear definition of the baseline period and associated baseline loads for each PFAS transport pathway. In both the CAP and PFAS Loading Reduction Plan, baseline loading rates have not been specified; instead, relative percent contributions from the various onsite transport pathways are described (e.g., 22 percent for onsite groundwater in May 2019 as listed in Table 7 in the CAP). Without a clear definition of the baseline period and loads, results could be interpreted in a manner that misrepresents progress. For example, monitoring data from a single day were extrapolated to generate an annual HFPO-DA load. The river flow that was applied to estimate the load for 2019 was less than one-third of the river flow applied for 2017. This caused an overestimation of the reported reduction in loading to the Cape Fear River that was described in the technical review report for the PFAS Loading Reduction Plan. It is recommended that a) a clear and consistent baseline period is defined and b) for past and future monitoring events, that the flow, PFAS concentration, and load associated with each transport pathway should be presented.
3. Reductions for aerial deposition were estimated for HFPO-DA and the report states there are "expected comparable reductions for other PFAS", although information to justify this important assumption is lacking (e.g., measured pollutant removal efficiencies for other PFAS through the application of air control technologies). Indeed, differences in adsorption and volatility characteristics among PFAS compounds suggests that rates will differ. Previous comments regarding the atmospheric deposition modeling described in the technical review of the PFAS Loading Reduction Plan do not appear to have been addressed and, thus, remain a concern.

4. Although the analysis time period is not specified in the CAP, historical process water releases are estimated to account for 76 to 86 percent of the Table 3+ PFAS detected in the Cape Fear River with the remainder coming almost entirely from historic air emissions (14 to 24 percent). This implies that no significant loading of Table 3+ PFAS to the river originates from other background sources, although information is not presented to justify this assumption. As described in other comments, only the relative percent contributions are listed and actual load estimates are not presented (i.e., in mass of PFAS per time interval). It is also important to determine how both the magnitude and relative contributions of PFAS loads have shifted over time in response to halting releases of process water in 2017 and subsequent implementation of other control measures.
5. Figure 3 in the CAP shows the total Table 3+ PFAS mass distribution in a normalized volume of the unsaturated and saturated soil zones (kg/m^3). For several of the assessed locations (11 of 18), a result is not shown for the unsaturated zone because no Table 3+ compounds were detected (Table C-3); however, the text does not specify the detection limit.
6. The PFAS signatures assessment component of the CAP evaluated the make-up and distribution of PFAS compounds in onsite and offsite groundwater. Two main categories identified included 1) aerial deposition PFAS signature from emissions to air and 2) combined process water PFAS signature from historic releases of process water to soil and groundwater. The latter signature is only detected onsite, affects approximately 1 square mile, exhibits Table 3+ PFAS concentrations of 2,900 to 18,000,000 ng/L onsite, and is estimated to contribute 76 to 86 percent of Table 3+ PFAS loading to the river. The former (aerial) signature is detected on and offsite, affects >70 square miles, exhibits lower Table 3+ PFAS concentrations (15 to 13,000 ng/L onsite and 10 to 4,500 ng/L offsite) and is estimated to contribute 14 to 24 percent of Table 3+ PFAS loading to the river. Comments related to the PFAS signatures assessment are summarized below:
    - Three PFAS signatures were established for aerially deposited PFAS from a hierarchical cluster analysis. These include 1) predominantly PMPA (perfluoromethoxypropyl carboxylic acid); 2) predominantly HFPO-DA (hexafluoropropylene oxide dimer acid); and 3) mixed PMPA and HFPO-DA. Another signature, predominately PFMOAA (perfluoro-1-methoxyacetic acid), is described to be the signature representative of process water contamination. A physical/chemical/geological explanation for the distribution of the signatures is missing and a discussion regarding the interactions and transformations of PFAS (precursors to degradation resistant PFAAs (perfluoroalkyl acids) via abiotic or biotic mechanisms) over time is lacking, although the report generically states that transformation of most PFAS substances in the environment is negligible. For example, why is PFMOAA primarily associated with process waste contamination? Are there atmospheric transport mechanisms that influence the distribution of the aerial signatures? The rate at which rainfall scours a substance from the air will vary according to the Henry's law constant, which varies across the PFOA/PFOS substances in Appendix G, however, the CAP does not describe this phenomenon (note that the Table 2-3 in Appendix G lists the Henry's law constants and includes a footnote stating the estimates originate from the CAP, but that does not appear to be correct). This contradicts previous statements that claim atmospheric deposition modeling of HFPO-DA is directly applicable to other PFAS. What other biogeochemical transformations in the environment influence the distribution of the aerial signatures?

- The thresholds used to differentiate the signatures (e.g., what constitutes an aerial mixture signature versus a predominately PMPA or HFPO-DA aerial signature) is vague and should be explicitly described.
- The signatures assessment did not attempt to distinguish the portion of the PFAS signatures attributed to background, or non-Chemours, sources (e.g., biosolids applications, fire response chemicals, atmospheric deposition from other regional or global sources).
- The report does not describe how the findings from the signature assessment will inform future studies and remediation efforts.
- We suggest that the analysis could be improved and clarified through the application of a fugacity analysis with a model such as QWASI (Mackay et al., 1983) to determine the likely theoretical distribution of compounds of interest between air, soil, and water (e.g., Kong et al., 2018).

7. To simulate groundwater hydraulics, an EVS geologic model (seven hydrostatic and heterogenous units) and a FEFLOW 3D finite element groundwater model were developed for the site. Comments regarding the development and calibration of the numerical groundwater model (Appendix H) include:
   - As noted in the numerical groundwater modeling report, the subsurface hydraulic conductivity (K) values listed in Table 2 for the Surficial and Black Creek aquifers are well outside of the typical range presented in Table 1. Anomalous K values would have implications for the estimation of groundwater discharge and pumping rates. Were calibrations attempted with lower K values and, if so, what were the outcomes? Also, the model sensitivity test ranges for K (±20 percent) appear low given the modeled versus typical range values presented in the report. Were the much higher K values derived from the groundwater model calibration subsequentially incorporated into the contaminant mass loading estimates that were generated separately? If not, the mass loading flux to the river due to groundwater discharge may be significantly underestimated.
   - The numerical groundwater modeling report describes the data source for specifying the upper layer boundary (site precipitation and evapotranspiration estimates for the Mid-Atlantic Coastal Plain from USGS) but does not present the initial rainfall recharge rates used in the model. It is inferred from the wording that these served as initial rates that were adjusted during the model calibration, however, the final calibrated rates are not provided. On page 12 it is stated that the final hydraulic parameters are provided in Table 3, although Table 3 instead lists the final calibration statistics for the three zones (Perched Zone, Surficial Aquifer, and Black Creek Aquifer), not the hydraulic parameters.
   - It is stated that localized anthropogenic stormwater recharge (a second upper layer boundary in addition to rainfall recharge described in the previous bullet) and historic infiltration from previously unlined sedimentation basins is included in the top boundary condition. The sedimentation basins have been lined so it is unclear why the basins are assumed to contribute infiltration water to the Perched Zone for the simulation period of October 2019. In addition, the rate is presented as 80,000 GPD and this should be correspondingly presented as a depth-based rate (e.g., inches per day/month).
   - Bluff seep discharge rates were evaluated but the report lacks presentation of performance metrics. Based on the information provided (Table 6.2), the model underpredicts Cape Fear River bluff seeps by about 88 percent and overestimates Old

Outfall 002 flow by 60 to 140 percent (range provided for measured/estimated flow). Therefore, the model seems to provide a weak correlation of these outflow features although the implications are not discussed.

- It is not clear from the numerical groundwater modeling report and CAP whether the onsite seeps originate from the perched zone, surficial aquifer, or both – this is important information for the development of a groundwater remediation strategy. It is also unclear what groundwater flow unit the offsite seeps described in Section 3.5 of the CAP discharge from.
- There is no quantification of the groundwater flux into the river from each of the groundwater flow units included in the model. Such fluxes should inform the basis for developing groundwater extraction and treatment scenarios.
- The daily median water elevation for the Cape Fear River measured at the W.O. Huske Dam is used to set the hydraulic head for the eastern boundary condition. It is not stated if this is the median water elevation for October 2019 or another period, although the former is preferable for the steady-state application described.
- On page 13 it is stated that an overall error of 10 percent or less is considered acceptable for the intended application (although no reference is provided) and that the groundwater model achieves this target (overall and for the Surficial and Black Creek Aquifers). Contradictorily, the calibration resulted in a Normalized Root Mean Square (NRMS) error of 12.5 percent for the final groundwater model (Table 5). Therefore, the calibration effort did not achieve the target performance metric. Additional information regarding model performance and justification that the calibrated model is acceptable is needed. For example, it would be preferable to report performance metrics (such as NRMS) for each borehole calibration site to assess spatial variability in model performance. NRMS errors are presented for the three vertical zones, and the error for the Perched Zone is quite high, 25.2 percent – it is noted that additional calibration efforts may be required to improve the representation of hydraulics in this zone. It is also stated that the calibrated FEFLOW model meets the requirements of the NCDEQ 2007 Groundwater Modeling Policy, however, these are not presented or discussed. The first step in the guidance (Define Study Objectives) is not addressed – specific and detailed objectives are called for in the guidance but not provided in the modeling report, although these are critical for producing a technically sound and appropriate model.
- The model was calibrated for steady-state conditions in October 2019. It would be preferable to complete a model validation using monitoring and conditions from an alternative period to demonstrate that the calibrated parameters are robust and the model responds correctly to different conditions. This is important because, as discussed in Section 7, the model was run for a forecast period of 1 year for the purpose of evaluating remedy scenarios given that conditions vary throughout the year (e.g., precipitation and recharge, boundary condition hydraulic heads including the Cape Fear River).
- The rationale and logic behind the selection of remedy simulations is missing. The scenario set should be identified based on clear objectives and technical/hydrogeologic analysis. In Section 5.4 of the CAP, it is stated that the hydraulic containment objectives are presented in Table 8, however, the table lists a summary of the six predictive simulations without describing the objectives. For example, no information is provided about:

- The groundwater discharge rates to the river under ambient conditions from each hydrogeologic unit, which would be necessary to establish the minimum required pumping rates for plume capture.
- The expected unit-specific maximum sustainable pumping rates for extraction wells based on hydrogeologic analyses and calculations.
- The hydrogeologic units from which the extraction wells draw water. Is it just the Black Creek Aquifer or are the wells screened across the Surficial Aquifer too?
- Capture zone calculations for wells in the initial well placement scheme.
- The rationale behind groundwater extraction rates being selected for the different scenarios. For example, there is a scenario with 41 wells pumping at 20 gpm each (820 gpm total) and another with 31 wells pumping at 30 gpm (930 gpm total), although the Black Creek Aquifer groundwater discharge for each scenario is presented as 1551 gpm. If the pumping scheme extracts substantially less groundwater compared to the discharge rate, then the entire plume will not be captured.

- There is no information provided regarding the locations of the extraction wells nor the constraints on the placement of the extraction wells in Appendix H or Section 5 of the CAP. Shifting the wells back from the river will alter capture processes and impact the assessment of feasibility. The groundwater units that the extraction wells will capture water from is not clear in the documentation. Comparisons are made for the Black Creek Aquifer. It is unclear if the perched and surficial aquifers are also targeted.
- It is not clear what is represented in column 5 of Table 7, labeled “Black Creek Groundwater Capture Flow into the Cape Fear River – By Simulated Pumping (GPM)”. Manipulating the numbers in the other columns does not shed light on what the value is supposed to represent.
- It is unclear where the flow diverted by the groundwater barrier will go (e.g., will groundwater reemerge downstream of the wall terminus?). This should be described. It remains uncertain if a groundwater barrier to limit interactions between onsite contaminated groundwater and the Cape Fear River would be feasible and effective.

8. Comments related to the measured and calculated partition and mass distribution coefficients (Appendix C and Section 3.7 of the CAP) include:
   - In Section 3.7 it is stated that detailed calculations for the mass estimates are provided in Appendix C, however, Appendix C describes the process but does not include sufficient data/spreadsheets to verify the calculations.
   - In this appendix, Log K_{ow} values were used to derive Log K_{oc} values for various PFAS compounds. Contradictorily, in the 2018 Interstate Regulatory Technology Council (IRTC) guidance document “Naming Conventions and Physical and Chemical Properties of Per- and Polyfluoroalkyl Substances” it specifically states that “It should be noted that although the K_{ow} for some organic contaminants can be used for estimating K_{oc}, this cannot be performed for estimating values for PFAS”. This calls into question the technical approach used in Appendix C and the results obtained.
   - For HFPO-DA, the Table C-2 Log K_{oc} value is 1.1, while in Table 2 of the CAP it is 1.69. Which (if either) of these is correct and used for the calculations?
   - Throughout Table C-2, as the Log K_{ow} increases, the Log K_{oc} increases as well. This is true except when comparing PFBA and PFPeA – what is unique about these compounds? The specific calculations are not provided for review and evaluation.

9. In the monitoring well redevelopment and resampling section, it is stated that 17 wells were redeveloped onsite, and 45 wells were resampled onsite based on recommendations issued in the Onsite and Offsite Assessment Report. The CAP does not provide summary level statistics for the groundwater monitoring effort, which would be very informative (e.g., mean and range of concentrations observed).
10. As described in the updated PFAS characterization sampling plan for process and non-process wastewater and stormwater, the raw intake point onsite is used to characterize background PFAS levels. However, water from the Cape Fear River at the intake point may be influenced by legacy atmospheric emissions and contaminated groundwater attributable to the site. Samples collected further upstream are needed to better characterize background PFAS concentrations.

3.2 CRITICAL GAPS

1. Concerns regarding the planned strategies to meet the cleanup goals described in Table 10 in the CAP include:
    - **Old Outfall 002**. The cleanup goal and proposed capture and treat strategy are solely designed to handle dry weather flows, thus, wet weather flows that may facilitate erosion of contaminated sediment are excluded. Based on the three 2019 monitoring events (May, June, and September), the relative contribution of Old Outfall 002 is estimated to be 26 percent of the total onsite PFAS load to the Cape Fear River. In Table 14, 26 percent of the planned loading reduction to the Cape Fear River is attributed to the capture and treatment of Old Outfall 002. This implies that 100 percent of PFAS will be treated by 2020 for the outfall, which conflicts with only targeting groundwater with the process wastewater signature.
    - **Willis Creek and Georgia Creek**. Indirect air abatement controls and onsite groundwater remedies are listed as strategies, but no creek specific controls are planned (e.g., removal of PFAS elevated sediment, flow capture and treatment).
    - **Onsite Groundwater**. The cleanup goal for groundwater describes mitigation of PFAS with a process water signature, thus, inherently excluding remediation of onsite groundwater exhibiting an aerial deposition signature. As shown in Figure 2, some of the groundwater wells onsite exhibit the latter. Based on the three 2019 monitoring events (May, June, and September), the relative contribution of onsite groundwater is estimated to be 18 percent of the total onsite PFAS load to the Cape Fear River. In Table ES2, 18 percent of the planned loading reduction to the Cape Fear River is attributed to onsite groundwater treatment. This implies that 100 percent of PFAS in groundwater will be treated by 2024, which conflicts with only targeting groundwater with the process wastewater signature.
    - **Offsite Groundwater and Offsite Soils**. It is stated that PFAS contamination has been detected in an area of 70 square miles (or more) surrounding the facility. However, because of the extent of the contamination, lack of scalable remediation technologies, and because no groundwater standards have been issued, it is claimed in the CAP that restoring groundwater conditions to PQLs is not feasible. A lack of management of offsite pollution does not seem to comply with 2L Rules as required in the CO Paragraph 16. It is also stated that PFAS are not expected to degrade in a reasonable time period in the environment. This is a concern because contaminated soils and groundwater will contribute legacy PFAS to the Cape Fear River in the future, continuing to impact the

quality of raw intake water for CFPUA. PFAS loading just downstream of the site and at the CFPUA intake should be quantified and compared to better understand the potential for long-term contamination from offsite sediment erosion, resurfacing groundwater, and releases from sediment in the riverbed and riparian areas. The assessment should compare loading at the two locations under varied conditions (e.g., dry/low flow periods, storm events). Also, the CAP describes several newly identified seeps, labeled E to M, south of the site, although no treatment plans are prescribed.

- **Onsite Soils**. Contamination in onsite soils remains unclear and no remediation strategies have been suggested in the CAP.
- **Outfall 002**. The remediation strategies for Outfall 002 are too vague, stating that compliance with NPDES permit requirements will be completed. Information regarding the PFAS-related requirements that will be included in Chemours' NPDES permit should be requested from DEQ.

2. As discussed in Section 5.1 of the CAP, the groundwater numerical model is only intended to simulate subsurface hydraulic processes, not associated PFAS fate and transport, for the purpose of remedy costing and design. Therefore, in its current state, the model provides limited insight in terms of PFAS loading and potential remediation effectiveness. In addition, the groundwater model covers the limited domain of the site. Thus, groundwater hydraulics are not represented for the surrounding vicinity contaminated by PFAS due to legacy atmospheric deposition. Since offsite seep data is attributed to aerial PFAS deposition, it could be used to estimate groundwater PFAS discharges to the river throughout the area (including upstream and downstream of the site) by using a distance-versus-concentration gradient approach and including discharge from both sides of the river due to airborne transport processes. This analysis would be informative, although it is not discussed.
3. There is a very limited discussion of PFAS transformations in the environment and the implications for ongoing contamination, exposure risk, and remediation activity effectiveness (e.g., presence of precursors that can degrade to PFAS analytes over time). It is noted in Section 3.4, that total Table 3+ concentrations in wells are comparable to prior results (within ± 25 percent), however, temporal monitoring records have not been applied to explore transformations of PFAS, nor has available and relevant information from the literature been summarized.
4. As noted in the previous technical review, a critical gap is that the extent, magnitude, and impact (loading) of PFAS contamination in offsite groundwater and soils are poorly quantified. Releases of contaminated groundwater, diffusion from contaminated sediment, and erosion of contaminated soils may contribute PFAS to the CFPUA's intake water following the implementation of the proposed onsite control strategies. PFAS contamination of sediment in the Cape Fear River bed and riparian wetlands remains uncertain and diffusion from these stores could act as a long-term source of PFAS to the river. A river sediment sampling plan was issued in August 2019 and it is anticipated that monitoring will be conducted at several riverine locations, including near CFPUA's raw water intake site, and a report released in 2020.
5. At this time, a comprehensive flow mass balance that represents all inflow and outflows at the site has not been developed. It is stated in Section 3.4 of Appendix H that the numerical groundwater model will eventually be used to support the development of an initial water budget. However, this is a current information gap.
6. In the CAP, the onsite Willis Creek to the north and Georgia Branch Creek to the south are described as being erosional channels that empty to the Cape Fear River. PFAS accumulated in the creek beds that is eroded during storm events may contribute to ongoing PFAS loading to the

river, yet the report does not attempt to measure bed contamination and model sediment transport (net deposition and scour) for the purpose of characterizing particulate-associated PFAS transport. Note that deeper soil samples (depths of 8.5 to 11 feet) have been collected in the vicinity of Willis Creek at a single location (Figure A7-1). The results for the analytes reported were either flagged as "UJ" (defined as "Analyte not detected. Reporting limit may not be accurate or precise") or flagged as "<" (defined as "Analyte not detected above associated reporting limit").

7. It was noted in the technical review for the PFAS Loading Reduction Plan and the CAP (Section 3.3.3) that discharge of Chemours' process wastewater has been halted and the waste is injected into subsurface storage out-of-state. However, elevated HFPO-DA and PFMOAA concentrations were also observed in Kuraray process wastewater, which continues to be discharged from the onsite WWTP via Outfall 002, as discussed in the PFAS Loading Reduction Plan and previous technical review. Loading from Kuraray process wastewater remains unquantified and untreated.
8. Another gap, although perhaps minor, is related to process wastewater. Before June 21, 2017 process wastewater was discharged to the Cape Fear River and after November 29, 2017 process wastewater was captured, stored, and transported offsite for disposal. The report does not describe what was done with process wastewater in the interim, between June 22 and November 28, 2017.

3.3 OTHER COMMENTS

Other comments related to vulnerabilities pertaining to CFPUA's intake water include:

1. No manufacturing process changes have been required for Chemours to date. Spills or unknown leaks or emissions at the facility remain a risk to CFPUA's source water.
2. All monitoring applied in the assessment appears to have been conducted by Geosyntec and contracted labs for Chemours. DEQ can require split sampling (samples provided to DEQ for parallel testing) per the CO. Split sampling would be beneficial from the perspective of CFPUA for quality assurance and control checking, therefore, CFPUA should inquire about completed split sampling and the findings.

4.0 REFERENCES

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT F
TO AMENDED INTERVENOR COMPLAINT

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Evaluation of Maternal, Embryo, and Placental Effects in CD-1 Mice following Gestational Exposure to Perfluorooctanoic Acid (PFOA) or Hexafluoropropylene Oxide Dimer Acid (HFPO-DA or GenX)

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Background: Perfluorooctanoic acid (PFOA) is a poly- and perfluoroalkyl substance (PFAS) associated with adverse pregnancy outcomes in mice and humans, but little is known regarding one of its replacements, hexafluoropropylene oxide dimer acid (HFPO-DA, referred to here as GenX), both of which have been reported as contaminants in drinking water.

Objectives: We compared the toxicity of PFOA and GenX in pregnant mice and their developing embryo–placenta units, with a specific focus on the placenta as a hypothesized target.

Methods: Pregnant CD-1 mice were exposed daily to PFOA (0, 1, or 5 mg/kg) or GenX (0, 2, or 10 mg/kg) via oral gavage from embryonic day (E) 1.5 to 11.5 or 17.5 to evaluate exposure effects on the dam and embryo–placenta unit. Gestational weight gain (GWG), maternal clinical chemistry, maternal liver histopathology, placental histopathology, embryo weight, placental weight, internal chemical dosimetry, and placental thyroid hormone levels were determined.

Results: Exposure to GenX or PFOA resulted in increased GWG, with increase in weight most prominent and of shortest latency with 10 mg/kg/d GenX exposure. Embryo weight was significantly lower after exposure to 5 mg/kg/d PFOA (9.4% decrease relative to controls). Effect sizes were similar for higher doses (5 mg/kg/d PFOA and 10 mg/kg/d GenX) and lower doses (1 mg/kg/d PFOA and 2 mg/kg/d GenX), including higher maternal liver weights, changes in liver histopathology, higher placental weights and embryo–placenta weight ratios, and greater incidence of placental abnormalities relative to controls. Histopathological features in placentas suggested that PFOA and GenX may exhibit divergent mechanisms of toxicity in the embryo–placenta unit, whereas PFOA- and GenX-exposed livers shared a similar constellation of adverse pathological features.

Conclusions: Gestational exposure to GenX recapitulated many documented effects of PFOA in CD-1 mice, regardless of its much shorter reported half-life; however, adverse effects toward the placenta appear to have compound-specific signatures. https://doi.org/10.1289/EHP6233

Introduction

Perfluorooctanoic acid (PFOA) is a fully fluorinated, eight-carbon synthetic chemical belonging to the class of compounds known as poly- and perfluoroalkyl substances (PFAS). PFAS are used in a wide range of industrial processes and consumer products and are globally ubiquitous, persistent, and detectable in nearly all humans living in industrialized nations (ATSDR 2019; Kato et al. 2011). Although humans are exposed to PFAS through multiple routes, drinking water is one of the most well-understood sources of exposure (Hu et al. 2016).

Within the general U.S. population, serum levels of PFOA have declined from a geometric mean of 5.2 ng/mL in 1999–2000 (CDC 2009) to 1.56 ng/mL in 2015–2016 (CDC 2019). This shift is likely the result of efforts by the U.S. Environmental Protection Agency (U.S. EPA) to reduce environmental emissions and to phase out U.S. production and use of PFOA by 2015 (U.S. EPA 2006). Similarly, in 2017, the European Union placed restrictions on the production and use of PFOA (European Commission 2017). Despite such efforts, exposure to PFOA remains a concern due to its long human half-life ($\sim$3.5 y) (Olsen et al. 2007), environmental persistence (Lindstrom et al. 2011), and the fact that longer-chain/precursor PFAS chemicals can degrade and form PFOA. In response to restrictions on PFOA, manufacturers have increased production on replacement compounds with alternative chemistries aimed at making the compounds less bioaccumulative and with shorter serum half-lives; however, toxicity data for these alternative PFAS are limited (Bao et al. 2018).

Hexafluoropropylene oxide dimer acid (HFPO-DA), referred to herein as GenX, is a PFOA replacement compound. GenX has received intense public scrutiny in North Carolina since its discovery in (Strynar et al. 2015), and contamination of, the Cape Fear River Basin following release from a manufacturing facility (Sun et al. 2016). GenX has also been measured in the environment in other regions of the United States, including the Ohio River (Hopkins et al. 2018), as well as in other countries, including the Xiaoqing River in China (Brandsma et al. 2018) and the Rhine River in Europe (Heydebreck et al. 2015).

PFAS are detectable in the serum of pregnant women and in cord blood, and the ratio of the concentration of PFOA in maternal serum to cord serum is typically $\sim$1:1 (Kim et al. 2011; Monroy et al. 2008). Maternal exposure to PFOA has been associated with multiple adverse health outcomes, including increased gestational weight gain (GWG) (Ashley-Martin et al. 2016), pregnancy-induced hypertension (Darrow et al. 2013), preeclampsia (Savitz et al. 2012; Stein et al. 2009), and reduced birth

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weight (Apelberg et al. 2007; Fei et al. 2007; Johnson et al. 2014; Kobayashi et al. 2017; Lam et al. 2014; Rijs and Bogers 2017). Based on a systematic review of the literature and meta-analysis, the shift in birth weight associated with PFOA exposure has been estimated to be −18.9 g birth weight per 1-ng/mL increase in serum PFOA [95% confidence interval (CI): −29.8, −7.9] (Johnson et al. 2014).

In mice, the reproductive and developmental effects of gestational exposure to PFOA are well documented. Previous studies have shown gestational exposure to PFOA in mice results in maternal liver damage (Lau et al. 2006), maternal hypolipidemia (Yahia et al. 2010), and reduced embryo weight (Koustas et al. 2014). It has been estimated from a meta-analysis of data from eight mouse studies that the shift in mice is −0.023 g pup birth weight per 1-mg/kg body weight (BW)/d increase in PFOA dose to pregnant dams (95% CI: −0.29, −0.016) (Koustas et al. 2014). In contrast, there is a paucity of data regarding the reproductive and developmental effects of GenX. A previous reproductive and developmental toxicity study of GenX in CD-1 mice determined the no observed adverse effect level (NOAEL) for reproductive toxicity and maternal systemic toxicity (microscopic changes in maternal liver) was 5 mg/kg/d HFPO-DA (GenX; DuPont-18,405-1,037). A recent study in rats showed limited gestational exposure to HFPO-DA (GenX) resulted in a lowest observed adverse effect level (LOAEL) for disrupted maternal thyroid hormone (TH) (LOAEL: 30 mg/kg/d) and lipids (LOAEL: 125 mg/kg/d), up-regulated gene expression in peroxisome proliferator–activated receptor (PPAR) signaling pathways in both maternal and embryo liver (LOAEL: 1 mg/kg/d), and lower BWs in gestationally exposed female offspring (LOAEL: 125 mg/kg/d) (Conley et al. 2019). Additional studies examining the reproductive and developmental effects of GenX are needed.

The biological mechanism through which PFOA exerts adverse effects on embryo growth is not known, but the placenta is a suspected target tissue. The placenta is critical for embryo growth and development, and disruptions in placental development or function can lead to adverse outcomes for both maternal and embryo health. Previous animal studies have examined the effect of gestational exposure to PFOA on maternal mammary gland development and embryo growth (Macon et al. 2011; White et al. 2007), but effects on the placenta have yet to be evaluated. The aims of this study were to compare the effects of gestational exposure to PFOA and a replacement, GenX, on GWG, embryo growth, liver pathology, and placental development/morphology.

Methods

Animals

Naïve female CD-1 mice between 7.5 and 15.5 wk of age from the NIEHS colony were bred in-house on a single night, and copulatory plug–positive females were identified on embryonic day (E) 0.5. Pregnant dams were singly housed in ventilated polypropylene cages and received nesting materials, National Institutes of Health (NIH)-31 diet (Zeigler Bros., Inc.) and reverse osmosis deionized (RODI) water *ad libitum*. Animals were housed in humidity- and temperature-controlled rooms with 25°C and 45–60% average humidity and standard 12-h light cycles. All animal procedures were approved by the NIEHS Animal Care and Use Committee (ASP #2017-0022).

Dosing Solutions

PFOA ammonium salt (CAS #3825-26-1) was purchased from Millipore Sigma, and GenX [ammonium 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)propanoate; CAS# 62,037-80-3] was purchased from SynQuest Laboratories. PFOA and GenX dosing solutions were prepared in RODI water and administered to mice once daily via oral gavage. Daily doses were administered between 0700 and 0800 hours and adjusted to the BW of the mouse based on the previous day's weight at a volume of 0.01 mL/g BW. PFOA doses of 5 mg/kg BW/d (high dose) and 1 mg/kg BW/d (low dose) were selected based on previous work that demonstrated a reduction in neonatal weight gain (Lau et al. 2006; White et al. 2007). The dose of 1 mg/kg BW/d PFOA, used in the mouse developmental toxicity study of Lau et al. 2006, provided a lowest effect dose that was used to set the reference dose within the U.S. EPA's drinking water lifetime health advisory level (HAL) of 70 ppt PFOA (U.S. EPA 2016). Given that the state of North Carolina has a provisional health goal of 140 ppt GenX (double the PFOA HAL), we selected doses of GenX (10 mg/kg BW/d, high dose; 2 mg/kg BW/d, low dose) to mirror doses of PFOA previously used in HAL decision-making.

Study Design

This experiment was conducted over two blocks (Block 1 and Block 2) to achieve a total of $n = 11–13$ litters per treatment group and sacrifice time point (E11.5 and E17.5). The experimental design of the second block was identical to the first block of the study, and experimental methods were similar but expanded upon to include more rigorous and detailed measurements. Copulatory plug–positive mice (E0.5) were weighed to obtain a baseline BW and placed into one of five groups. Once all mice were assigned to groups, mean BWs were calculated, and a few animals were reassigned so that mean BWs in each group were similar. This was done to avoid confounding effects of baseline BW. Treatment groups were then randomly assigned a color by using a random sequence generator. Experimenters and dosing technicians were blinded to the treatment group to which the color groups corresponded throughout the duration of the study, including at necropsy. Randomly assigned treatment groups included in each block: vehicle control (deionized water only), 1 mg/kg BW/d PFOA, 5 mg/kg BW/d PFOA, 2 mg/kg BW/d GenX, and 10 mg/kg BW/d GenX. Pregnant dams were dosed via oral gavage from E1.5 to E11.5 or from E1.5 to E17.5. The sacrifice time points were selected *a priori* to examine effects of gestational PFOA or GenX exposure on embryo and placental growth prior to placental maturation (E11.5) as well as after full placental maturation (E17.5) (Watson and Cross 2005). The E11.5 early-gestation time point was selected because it overlaps a critical period of placental development in the mouse where the placenta undergoes vascularization with the uterine wall and chorioallantoic branching of vessels begins (Watson and Cross 2005). The E17.5 late-gestation time point was selected so that embryo weight changes that may be related to treatment would be evident.

Necropsy

On the day of necropsy, dams received daily oral gavage between 0700 and 0800 hours and were weighed and then euthanized humanely by swift decapitation, and serum was collected. In Block 1, necropsies were completed from 0800 to 1600 hours, and in Block 2, necropsies were completed from 0800 to 1200 hours. Serum from dams euthanized in Block 1 was snap frozen for internal dosimetry analyses. Serum and urine from Block 2 dams were reserved for clinical chemistry analyses. In both blocks, the uterus was removed, and total implantation sites were counted based on gross observation of an implantation nodule

along the uterine horn. Viable embryos, nonviable embryos, and sites of resorption were counted based on gross observation. Embryos were considered viable if they were properly formed, were not pale in color, and were of similar size to neighboring embryos. Embryos that were poorly formed and pale in color (without heartbeat) were considered nonviable. Sites of resorption were defined as a dark red–appearing clot-like nodule apparent on gross observation.

From each uterus, first, viable embryos and their matched placentas were collected in succession within a horn and immediately snap frozen ($n = 2$–5 per litter), and subsequent embryos were collected for growth measurements ($n = 2$–11 per litter). Additional placentas were collected and placed in 4% paraformaldehyde (PFA) for histological analysis (Block 2 only). Amniotic fluid was collected by needle aspiration from litters euthanized at E11.5 and snap frozen in liquid nitrogen. Embryo livers were collected from litters euthanized at E17.5 and snap frozen in liquid nitrogen. Dam livers were weighed, a portion of the left lateral lobe was placed in 4% PFA for histology, and another portion of the same lobe was snap frozen in liquid nitrogen. A third liver section was obtained from Block 2 dams and fixed in McDowell and Trump's fixative for electron microscopy (EM). Gross lesions were collected when observed and placed in 4% PFA for histology. Dam kidneys were removed, a cross section was prepared from the right kidney, a longitudinal section was prepared from the left kidney, and both sections were fixed in 4% PFA for histological analysis.

Tissue Preparation/Histology/Clinical Measures

Dam livers, kidneys, and placentas were trimmed and embedded by the NIEHS Mouse Embryo Phenotyping Core. Tissues collected at necropsy were fixed in 4% PFA for 72 h and paraffin embedded, and 5-μm sections were prepared and stained with hematoxylin and eosin (H&E). Pathology was evaluated and a pathology review conducted by S.A.E. Diplomate American College of Veterinary Pathologists (DACVP). Pathology reviews were conducted as an informed approach analysis [e.g., nonblinded analysis; see Sills et al. (2019)]. Select tissue slides were scanned using the AT2 Scanner (Aperio). Images were then captured for publication using the ImageScope software; version 12.3.0.5056 (Aperio). Serum and urine obtained from dams in Block 2 were analyzed using the AU480 clinical chemistry analyzer (Beckman Coulter Inc.). Reagents and calibration standards used to measure alkaline phosphatase (ALP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood urea nitrogen (BUN), serum creatinine, urine creatinine, glucose (Glu), total protein (TP), triglyceride (Trig), high-density lipoprotein (HDL), cholesterol (Chol), and albumin (ALB) were purchased from Beckman Coulter Inc. Reagents for sorbitol dehydrogenase (SDH), total bile acid (TBA), and micro-TP were purchased from Sekisui Diagnostics. The reagent used to measure low-density lipoprotein (LDL) was purchased form Diazyme Laboratories.

Transmission Electron Microscopy

Block 2 dam liver portions stored in McDowell and Trump's fixative (McDowell and Trump 1976) were processed using a Leica EM TP processor. Briefly, samples were rinsed with buffer, postfixed in 1% osmium tetroxide in 0.1-M phosphate buffer, rinsed in distilled water, dehydrated, and embedded in Ply/Bed® 812 (Polysciences, Inc.) epoxide resin. Blocks were trimmed, and semithin sections (~0.5 μm) were stained with 1% toluidine blue (Poly-scientific R&D Corp.) O in 1% sodium borate to ascertain areas of interest. Ultrathin sections (90–110 nM) were cut from areas of interest and placed on 200-mesh copper grids and stained with uranyl acetate and lead citrate, and digital images were captured using an Orius® SC1000 side mount camera (Gatan) attached to a Techani T12 transmission electron microscope (TEM) (FEI Company). In general, peroxisomes were smaller than mitochondria and round with a dark, electron-dense, granular matrix and surrounded by a single membrane. Mitochondria were round to elongated, had a matrix that was less electron dense than peroxisomes and contained crista, and were surrounded by an inner and outer membrane. Samples were analyzed by R.D.K., Ph.D.

Placental Thyroid Hormone Quantification

Thyroid hormones (T3, triiodothyronine; T4, thyroxine; rT3, reverse triiodothyronine) in placenta were analyzed according to the methods described in Leonetti et al. (2016). Briefly, ~300 mg (207– 526 mg) of two to three pooled placental tissues of same-sex embryos was homogenized and digested for 16 h overnight in PRONASE® Protease (*Streptomyces griseus*) solution (EMD Millipore Corp.). Each pooled sample of two to three placentas was considered as one biological replicate and included placentas from the same litter when possible. Three biological replicates were used for each treatment group and each sex. Samples were spiked with an antioxidant solution (containing 37.5 mg/mL each of citric acid, ascorbic acid, and dithiothreitol) and ^{13}C isotopically labeled internal standards (T4, T3, and rT3), and cold acetone was added to stop the digestion reaction. Samples were vortex mixed and centrifuged three times for 2 min at 10,000 relative centrifugal force (rcf), and the supernatants were collected and combined. Sample pH was adjusted with 6 M hydrochloric acid to pH < 2. A liquid–liquid extraction with cyclopentane was performed and the cyclopentane layer discarded; briefly, 1 mL of cyclopentane was added to the supernatant and vortexed before the sample was centrifuged for 3 min at 3,000 rcf and the cyclopentane layer discarded, and this was repeated three times. A liquid–liquid extraction with ethyl acetate was performed; briefly, 3 mL of ethyl acetate was added to the extract and vortexed before being shaken on a plate shaker for 30 min and centrifuged for 3 min at 3,000 rcf, and the ethyl acetate layer collected; this was repeated three times. Ethyl acetate extracts were dried down to 50 μL under a gentle nitrogen stream and resuspended in 1 mL of 0.01 M hydrochloric acid in 10% methanol. Samples were purified by solid-phase extraction using SampliQ Optimized Polymer Technology (OPT) cartridges (3 mL, 50 mg; Agilent Technologies). Final extracts in 400 μL of 1:1 methanol:water were filtered using Whatman® Mini-UniPrep® Syringeless Filters [Polytetrafluoroethylene (PTFE), 0.2 μm; GE Healthcare]. Extracts were analyzed on an Agilent high-performance liquid chromatography (HPLC) 1260 with a Synergi™ 50 mm × 2 mm Polar-RP column (2.5 μm; Phenomenex) coupled to an Agilent model 6460 tandem mass spectrometer with electrospray ionization (HPLC-MS/MS-ESI). Mobile phases consisted of 10 mM formic acid in methanol and 10 mM formic acid in water. Laboratory processing blanks were extracted alongside the placental tissues to monitor background levels. No TH were detected in the lab blanks. Method detection limits (MDLs) were calculated using a signal-to-noise value of 3 for each analyte (T3, T4, and rT3). Values were normalized to the wet weight of placenta extracted for a final value of nanogram hormone/gram placenta. Values below the MDL (T4, 0.84 ng/g; T3, 0.42 ng/g; rT3, 0.67 ng/g) were imputed using the calculation MDL × 0.5, and values lacking a quantifiable peak on mass spectrometry were excluded from the analysis.

Internal Dosimetry

Maternal serum, maternal liver, amniotic fluid, and whole embryos were analyzed for PFOA and GenX concentrations using methods similar to those previously reported (Conley et al. 2019; McCord et al. 2018; Reiner et al. 2009; Rushing et al. 2017). Solid tissues were homogenized in RODI water at a ratio of approximately 1:3 tissue mass (milligrams) to liquid volume (microliters). Maternal serum, amniotic fluid, and tissue homogenates (25 μL) were spiked with internal standard suspended in 0.1 M formic acid in a denaturation step, followed by a subsequent protein crash using ice-cold acetonitrile. Samples were vortex mixed after addition of formic acid and acetonitrile and then centrifuged at $10{,}000 \times g$ for 5 min. Extract supernatants were separated using a Waters ACQUITY UPLC® (Waters Corporation) fitted with a Waters ACQUITY UPLC® BEH C18 Column (130Å; 1.7 μm; 2.1 mm × 50 mm). Detection was performed using a Waters Quattro Premier™ XE tandem quadrupole mass spectrometer in negative ionization mode. Stable isotopes of PFOA ($^{13}C_3$, MPFOA; Wellington Laboratories) or GenX ($^{13}C_3$, M3HFPODA; Wellington Laboratories) were used as internal standards for quantification of vehicle control samples (run against a nine-point calibration curve of 0–100 ng/mL) and experimental samples (run against a nine-point calibration curve of 200–20,000 ng/mL). Vehicle control and dosed animal samples were quantified for both PFOA and GenX using respective isotope-labeled chemicals and calibration curves.

Embryo/Placental Growth Metrics

Gross observations were recorded at necropsy. Embryo sex was determined by polymerase chain reaction (PCR) amplification of the *Sry* gene (forward, 5′-GCTTCAGTAATCTCAGCACCTAGAA-3′, and reverse, 3′-CACATTGGCATGATAGCTCCAAATT-5′) using a snipped portion of tissue (TransnetYX®, Inc.). Embryos and their placentas were weighed separately as wet tissue. Images of embryos were obtained on a Leica Z16 APO imaging scope, and embryo length was measured as snout-to-rump distance using FIJI (Schindelin et al. 2012) and Zen 2 Blue (Zeiss).

Statistical Analysis

Data were analyzed in R (version 1.1.456; R Development Core Team). Sample sizes for each end point are reported in the accompanying figure legends or tables. A threshold of $p < 0.05$ was used for determining statistical significance unless otherwise noted. Analyses combining data from both experimental blocks were performed after verifying the absence of experimental block effects. Single-observation dam outcomes (e.g., liver weight, relative liver weight, implantation sites, resorptions, viable embryos, and internal dose metrics) were analyzed by analysis of variance using the lme4 (Bates et al. 2014) and lmerTest packages (Kuznetsova et al. 2017). Simultaneous tests for general linear hypotheses were corrected for multiple comparisons of means using Tukey contrasts in the package multcomp (Hothorn et al. 2009).

For all statistical tests adjusting for litter size as a fixed effect in the model, litter size was defined as the number of viable embryos. GWG on the day of sacrifice was adjusted for litter size using a general linear model. To compare GWG growth curves, GWG was measured as the percent change in BW compared to E0.5 and analyzed using mixed-effects models controlling for litter size and accounting for repeated measures of dams over time.

Embryo and placental metrics were analyzed using mixed-effect models and included *a priori* fixed effects of treatment group and litter size and a random-effects term for the dam using the lme4 package. Embryo and placental metrics included embryo weight, embryo length, placental weight, and embryo:placenta weight ratios, a meaningful predictor of fetal birth outcomes in humans (Hayward et al. 2016). To account for potential introduction of random effects, the study block (Block 1 or Block 2) and experimenter handling of embryo/placental tissues (Experimenter A or Experimenter B) were included as additional random effects. Models were fit in a stepwise procedure for random effects, and final models included treatment group and litter size as fixed effects using the lmerTest package (Kuznetsova et al. 2017). All final models included dam as a random effect but were allowed to vary in the inclusion of experimenter and experimental block random effects based on likelihood ratio test results. Point estimates and 95% CIs were determined from the final model using the Wald method. The number of individual observations for each outcome (embryo weight, placenta weight, and embryo:placenta weight ratio) and the number of litters evaluated in the mixed-effect models are shown in Table S1.

To document the effects of PFOA and GenX on the placenta, placentas were assessed for histopathological lesions in five to six litters per treatment group for both time points, with an average of seven individual placentas evaluated per litter. Analyses of histopathological data included placentas collected from viable embryos and excluded fused placentas and placentas collected from sites of resorption, which did not occur more frequently than at expected background levels in this strain. Histopathological lesions of evaluated placenta were evaluated using two statistical approaches. The first approach assumed the absence of litter effects and considered each placenta evaluated within a treatment group to be a totally independent observation, regardless of its litter of origin. These data were analyzed as counts using a generalized linear model with a Poisson regression using the package lme4 (Bates et al. 2014). The second approach considered the litter as the biological unit and compared the relative incidence of placental lesions [e.g., percent within normal limits (WNL)] to adjust for differences in the total number of observations across litters within and between treatment groups. These data were analyzed using a linear model. Both approaches were subjected to simultaneous tests for general linear hypotheses to correct for multiple comparisons using Tukey contrasts in the package multcomp (Hothorn et al. 2009).

TH concentrations in the placenta were quantified, and the ratios of T3:T4 and rT3:T4 in E17.5 placentas were assessed to evaluate potential disruption of peripheral TH control (e.g., impacts on thyroid deiodinase activity). Each end point was analyzed for sex × treatment interaction or for an overall effect of sex. Placenta TH were analyzed by analysis of variance using lme4 (Bates et al. 2014). Simultaneous tests for general linear hypotheses were corrected for multiple comparisons of means using Tukey contrasts in the package multcomp (Hothorn et al. 2009). Placental TH and their ratios were initially analyzed with embryo sex as an interaction term in the model, with the dose group as the predictor variable. Inclusion of a sex interaction or sex covariate in the final model was examined in a stepwise fashion. Internal dosimetry data were analyzed by analysis of variance. Simultaneous tests for general linear hypotheses were corrected for multiple comparisons of means using Tukey contrasts in the package multcomp (Hothorn et al. 2009).

Results

Internal Dosimetry

Maternal serum, maternal liver, amniotic fluid (E11.5 only), and whole-embryo dosimetry varied based on compound, dose, and time point. Urine collection was attempted at necropsy of pregnant

dams exposed to GenX but was unable to be consistently collected in sufficient volume for dosimetry analysis. Concentrations of GenX in the serum of dams exposed daily to 10 mg/kg of GenX was equivalent to the concentration of PFOA in serum of dams exposed to 5 mg/kg/d of PFOA at E11.5 (118.1 ± 10.4 μg GenX/mL serum and 117.3 ± 20.6 μg PFOA/mL serum, respectively; Figure 1A,B; Table S2). In contrast, GenX accumulation in the serum of dams exposed to 2 mg/kg/d GenX was 32% higher than the accumulation of PFOA in the serum of dams exposed to 1 mg/kg/d PFOA (33.5 ± 15.7 μg GenX/mL serum and 25.4 ± 3.7 μg PFOA/mL serum, respectively; Figure 1A,B; Table S2). Serum levels of either dose of PFOA or GenX measured at E17.5 were lower from those measured at E11.5 (Figure 1A,B; Tables S2 and S3). This could be explained by a dilution effect caused by blood volume expansion over the course of gestation or may be due to increased transfer to embryos over time.

Accumulation of PFOA in the maternal liver was greater than the accumulation of GenX, regardless of dose level or collection time point (Figure 1C,D; Tables S2 and S3). While maternal serum levels of PFOA or GenX were surprisingly roughly equivalent at E11.5 in dams exposed to PFOA or GenX, respectively, the accumulation of PFOA in the maternal liver was markedly higher in mice exposed to PFOA than the accumulation of GenX in liver of mice exposed to GenX (Figure 1C,D; Tables S2 and S3). It appeared that bioaccumulation of PFOA in the liver had reached a maximum of approximately 160–180 μg PFOA/g liver by E17.5 regardless of PFOA dose group (Figure 1C; Table S3). When comparing across low (1 mg/kg/d PFOA vs. 2 mg/kg/day/GenX) and high (5 mg/kg/d PFOA vs. 10 mg/kg/d GenX) dose groups at each time point, the fold change comparing GenX accumulation in the liver to the PFOA accumulation in the liver was 7.6-fold lower (2 mg/kg GenX vs. 1 mg/kg PFOA; E11.5), 8.9-fold lower (10 mg/kg GenX vs. 5 mg/kg PFOA; E17.5), 11.2-fold lower (10 mg/kg GenX vs. 5 mg/kg PFOA; E17.5), and 39.7-fold lower (2 mg/kg GenX vs. 1 mg/kg PFOA; E17.5) (Figure 1C,D; Tables S2 and S3). Unlike PFOA, GenX did not significantly bioaccumulate further in dam livers between E11.5 and E17.5 (Figure 1D; Tables S2 and S3).

Amniotic fluid concentrations of PFOA and GenX were roughly equivalent when comparing the accumulation in dams exposed at the high (5 mg/kg/d PFOA vs. 10 mg/kg/d GenX) and low doses (1 mg/kg/d PFOA vs. 2 mg/kg/d GenX) (Figure 2A,C; Table S2). Comparing across PFOA and GenX dose groups, embryo accumulation at E11.5 was greatest in mice exposed to 10 mg/kg/d GenX (3.21 ± 0.5 μg/g), followed by mice exposed to 5 mg/kg/d PFOA (2.34 ± 0.3 μg/g), 2 mg/kg/d GenX (0.91 ± 0.2 μg/g), and 1 mg/kg/d PFOA (0.80 ± 0.10 μg/g) (Figure 2B,D; Table S2). At E17.5, embryo accumulation was not different between sexes for either compound at the doses tested (Figure 2B,D; Table S3). Concentrations of PFOA or

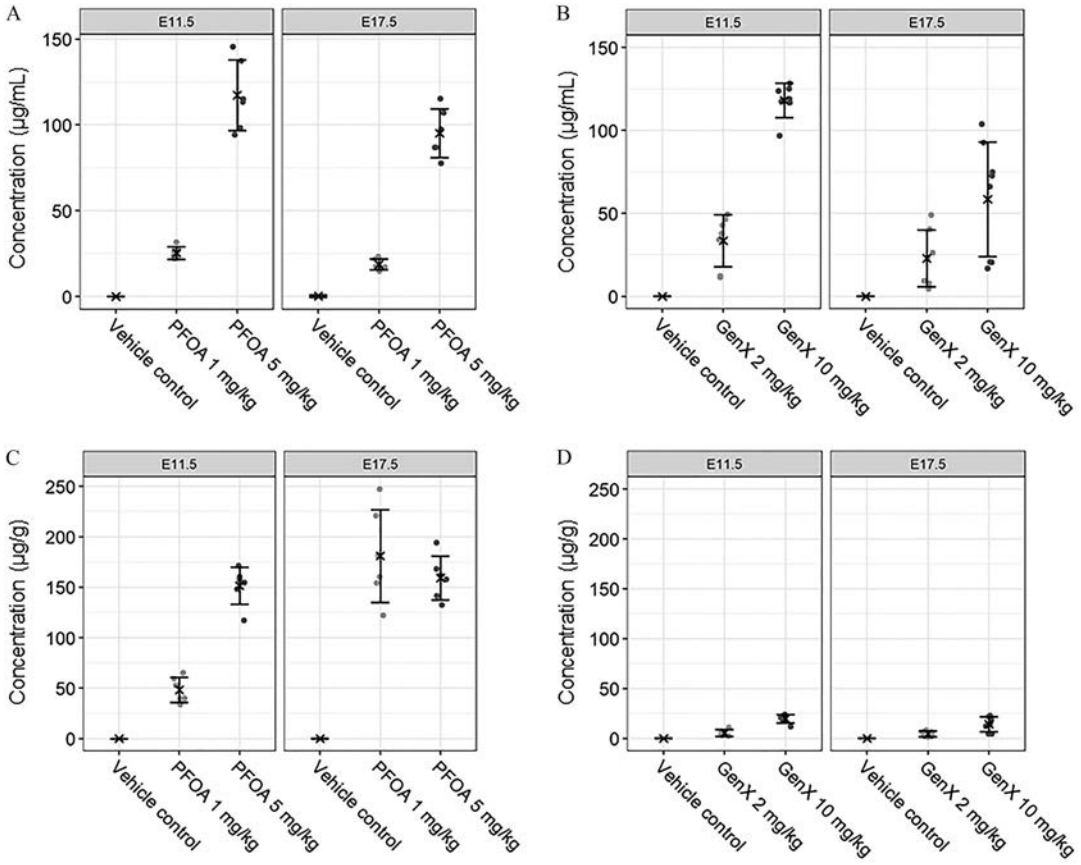

Figure 1. Internal dosimetry of perfluorooctanoic acid (PFOA) and GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] in maternal serum and liver at embryonic day (E) 11.5 and E17.5. (A) Maternal serum concentration (microgram PFOA per milliliter serum) at E11.5 and E17.5, (B) maternal serum concentration (microgram GenX per milliliter serum) at E11.5 and E17.5, (C) maternal liver concentration (microgram PFOA per gram liver) at E11.5 and E17.5, and (D) maternal liver concentration (microgram GenX per gram liver) at E11.5 and E17.5 were determined by high-performance liquid chromatography–tandem mass spectrometry. Treatment group mean values are denoted with an "X" flanked above and below by error bars showing standard deviation, and individual data points are shown as gray circles ($n = 6$–8). Vehicle control (VC) samples were quantified for PFOA and GenX; all VC means were below the limit of detection (LOD) of 10 ng/mL for both PFOA and GenX except for maternal serum (0.211 ± 0.55 μg/mL). Statistical comparisons of internal dosimetry across all treatment groups are shown in Tables S2 and S3.

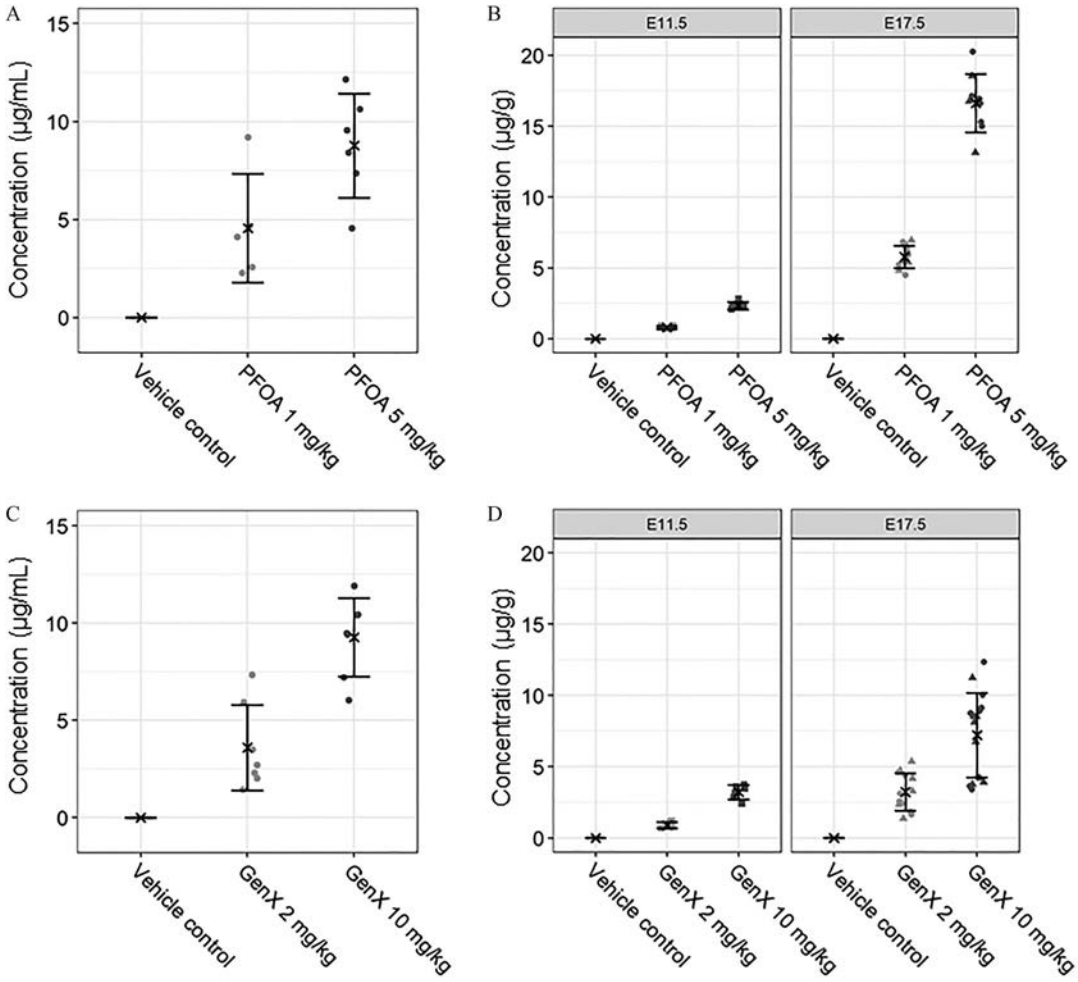

Figure 2. Internal dosimetry of perfluorooctanoic acid (PFOA) and GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] in amniotic fluid and whole embryos. (A) Amniotic fluid concentration (microgram PFOA per milliliter amniotic fluid) at embryonic day (E) 11.5, (B) whole-embryo concentration (microgram PFOA per gram embryo) at E11.5 and E17.5, (C) amniotic fluid concentration (microgram GenX per milliliter amniotic fluid) at E11.5, and (D) whole-embryo concentration (microgram GenX per gram embryo) at E11.5 and E17.5 were determined by high-performance liquid chromatography–tandem mass spectrometry. Treatment group mean values are denoted with an "X" flanked above and below by error bars showing standard deviation, and individual data points are shown as gray squares, circles, or triangles ($n = 6$–8). Triangles, E17.5 male embryos; circles, E17.5 female embryos; squares, pooled E11.5 embryos (B and D). Vehicle control (VC) samples were quantified for PFOA and GenX; all VC means were below the limit of detection (LOD) of 10 ng/mL for both PFOA and GenX. Statistical comparisons of internal dosimetry across all treatment groups are shown in Tables S2 and S3.

GenX in embryos were greater when measured at E17.5 than at E11.5, suggesting accumulation of both compounds over time in the embryo regardless of the shorter half-life of GenX (Figure 2B,D; Tables S2 and S3).

Maternal Outcomes

Gross anomalies were visually evident in some dams upon necropsy; excess abdominal fluid, edematous tissues, clotted placentas, and two fetuses attached to a single placenta were noted. However, these findings were unexpected *a priori* and thus were not looked for in each animal, were not reported by dose group, and require further investigation in future studies.

Mean dam BWs at E0.5 were similar across all treatment groups, including PFOA and GenX, for either sacrifice time point and did not differ from vehicle controls (Table 1). The relative change in dam BW from E0.5 to the time of collection (percent change in weight; GWG) was significantly greater after exposure to 10 mg/kg/d GenX at E11.5 (7.4% greater BW gain at E11.5 relative to vehicle controls; $p < 0.05$; Table 1). The number of implantation sites, viable embryos, nonviable embryos, and resorptions did not significantly differ among treatment groups, including PFOA and GenX, at either time point relative to the vehicle controls, although 10 mg/kg/d GenX-treated dams had fewer implantation sites and viable embryos at E17.5 (Table S4). When controlling for litter size, relative GWG was significantly greater than controls in 10 mg/kg/d Gen-treated mice (E11.5: 7.1% greater compared to controls; E17.5: 19.1% greater compared to controls) and 5 mg/kg/d PFOA-treated mice (E17.5: 14.5% greater compared to controls; Table S5). Effect estimates from mixed-effect models adjusting for repeated measures of relative GWG (dataset shown in Figure 3C), litter size, and gestational/embryonic day showed significantly higher relative GWG in mice exposed to 10 mg/kg/d GenX (E11.5 and E17.5) (Figure 3A,B), 2 mg/kg/d GenX (E17.5) (Figure 3B), and 5 mg/kg/d PFOA (E17.5) (Figure 3B).

Dam liver weights were significantly higher in all treated groups compared to vehicle controls at E11.5 (Table 1). At E17.5, absolute liver weights of dams were significantly higher in the 5 mg/kg/d PFOA, 2 mg/kg/d GenX, and 10 mg/kg/d GenX-treatment groups than in vehicle controls (Table 1). Dam relative liver weight (as a percentage of BW) was significantly higher in both PFOA and GenX treatment groups relative to vehicle controls at E11.5 and E17.5 (Table 1). At E11.5, vehicle

Table 1. Maternal indices at embryonic day 11.5 and 17.5 [mean ± standard deviation (SD); $n = 11–13$].

Embryonic day	Maternal index	Vehicle control	1 mg/kg BW/d (PFOA)	5 mg/kg BW/d (PFOA)	2 mg/kg BW/d GenX (HFPO-DA)	10 mg/kg BW/d GenX (HFPO-DA)
11.5	E0.5 weight (g)	30.6 ± 5.5	31.2 ± 3	31.1 ± 3.2	29.7 ± 2.2	30.7 ± 2.5
11.5	Weight at necropsy (g)	37.9 ± 4.3	38.8 ± 2.4	40.2 ± 3.5	38.3 ± 3.2	40.0 ± 2.5
11.5	Weight at necropsy (% change from E0.5)	24.9 ± 9.2	24.7 ± 6.3	29.6 ± 6.3	28.9 ± 5.4	32.3 ± 9.6*
11.5	Liver weight (g)	2.2 ± 0.3	2.9 ± 0.2*	4.5 ± 0.5*	3.1 ± 0.2*	4.2 ± 0.5*
11.5	Relative liver weight (% BW)	5.9 ± 0.7	7.4 ± 0.5*	11.0 ± 0.9*	8.1 ± 0.5*	10.2 ± 0.7*
11.5	Kidney weight (g)	0.20 ± 0.01	0.20 ± 0.02	0.21 ± 0.03	0.22 ± 0.02	0.23 ± 0.06
11.5	Relative kidney weight (% BW)	0.53 ± 0.01	0.51 ± 0.04	0.51 ± 0.05	0.54 ± 0.04	0.52 ± 0.11
17.5	E0.5 weight (g)	30.5 ± 3.3	28.5 ± 3.8	29.1 ± 3.4	28.2 ± 3.5	28.7 ± 3.6
17.5	Weight at necropsy (g)	56.3 ± 5.6	54.6 ± 5.3	57.4 ± 6.0	55.4 ± 6.5	56.7 ± 5.5
17.5	Weight at necropsy (% change from E0.5)	86.0 ± 22.8	92.6 ± 17.1	98.7 ± 20.2	97.3 ± 15.2	98.5 ± 15.7
17.5	Liver weight (g)	2.7 ± 0.3	3.1 ± 0.4	5.3 ± 0.5*	3.5 ± 0.5*	4.6 ± 0.4*
17.5	Relative liver weight (% BW)	4.8 ± 0.3	5.6 ± 0.5*	9.3 ± 0.7*	6.3 ± 1.0*	8.1 ± 0.5*
17.5	Kidney weight (g)	0.21 ± 0.02	0.22 ± 0.04	0.24 ± 0.03	0.21 ± 0.02	0.25 ± 0.02*
17.5	Relative kidney weight (% BW)	0.37 ± 0.04	0.40 ± 0.04	0.40 ± 0.03	0.37 ± 0.02	0.43 ± 0.03*

Note: BW, body weight. $n = 6–8$ for kidney weight and relative kidney weight. *$p < 0.05$ relative to vehicle control [analysis of variance (ANOVA) with post hoc multiple comparison correction using Tukey contrasts].

control livers exhibited either normal hepatocellular features with uniform hepatocellular size and cytoplasmic glycogen or minimal centrilobular hepatocellular hypertrophy with decreased glycogen, consistent with pregnancy at this stage of gestation. At E17.5, vehicle control livers exhibited hepatocellular changes consistent with pregnancy at this stage of gestation (minimal to mild centrilobular hepatocellular hypertrophy with karyomegaly, increased mitotic figures, decreased glycogen, and increased basophilic granular cytoplasm (Figures 4A and 5A). Compared with their respective controls, all livers (100% incidence) from both PFOA- and GenX-treated dams at E11.5 and E17.5 showed a variety of adverse outcomes (Figure S1), including some degree of cytoplasmic alteration, characterized by varying degrees of hepatocellular hypertrophy with decreased glycogen and intensely eosinophilic granular cytoplasm (Figures 4C,E and 5C,E; Tables S6 and S7). As the severity increased, there was extension of the cytoplasmic alteration into the midzonal and periportal regions. Also, as the cytoplasmic alteration increased in severity, there was an observed decrease in mitoses and increase in apoptotic cell death (Figures 4E and 5E). A few livers from exposed animals also had focal regions of classic necrosis. Incidence of liver lesions and vacuolation are reported in Tables S6 and S7.

Histopathological liver findings from a subset of E17.5 dams, including all dose groups for PFOA, GenX, and vehicle controls for comparison, were further evaluated using TEM. All vehicle control livers exhibited normal ultrastructure for this stage of gestation. In the centrilobular regions with hepatocellular hypertrophy, there was abundant glycogen, prominent rough endoplasmic reticulum (RER) with abundant ribosomes, numerous lysosomes, and minimal vacuolation with vacuoles often containing remnant membrane material as myelin figures (Figures 4B and 5B). Livers from mice exposed to 1 mg/kg/d PFOA exhibited enlarged hepatocytes with increased cytoplasmic organelles consistent with mitochondria and peroxisomes, evenly dispersed glycogen, and small vacuoles in the centrilobular regions (Figure 4D) compared to vehicle controls. Livers from mice exposed to 5 mg/kg/d PFOA exhibited abnormal ultrastructure with abundant organelles consistent with mitochondria and peroxisomes, highly prevalent cytoplasmic vacuolation, reduced RER with fewer ribosomes, and less abundant glycogen (Figure 4F). Livers from mice exposed to 2 mg/kg/d GenX exhibited abnormal ultrastructure with enlarged hepatocytes containing more abundant cytoplasmic organelles consistent with mitochondria and peroxisomes, and vacuolation (Figure 5D). Livers from mice exposed to 10 mg/kg/d GenX exhibited abnormal ultrastructure with enlarged hepatocytes containing abundant organelles consistent with mitochondria and peroxisomes, and prevalent vacuolation often with remnant membrane material as myelin figures, abundant RER with few ribosomes present, and unevenly dispersed glycogen appearing as clustered clumps (Figure 5F). At the level of TEM, PFOA and GenX generally caused a variety of cellular alterations: increased vacuolation, increased numbers of cytoplasmic organelles consistent with mitochondria and peroxisomes, reduced glycogen stores and reduction of RER ribosomes (Figure S2). Marked clumping of glycogen was a unique observation in livers of mice exposed to 10 mg/kg/d GenX, likely a secondary effect due to abundant mitochondria, peroxisomes, and RER.

Kidney weights and relative kidney weights of dams exposed to either dose of PFOA or GenX did not differ from vehicle controls at E11.5 (Table 1). At E17.5, 10 mg/kg/d GenX-exposed mice exhibited higher kidney weight relative to vehicle controls (both absolute kidney weight and relative kidney weight) (Table 1). Kidney cross sections and longitudinal sections were histopathologically evaluated at E11.5 and E17.5 time points, and diagnoses were made with no threshold: cortical glomeruli; cortical and medullary tubules; papillary collecting ducts; parenchyma; and vascular tree including renal artery, interlobar artery, interlobular artery, arcuate artery, and renal veins. Kidneys from vehicle control and treated animals were histologically WNL.

Clinical Chemistry

Dam serum Trig levels were significantly lowered at E11.5 across all treatment groups compared to controls in a dose–response manner (5 mg/kg/d PFOA and 10 mg/kg/d GenX lowered Trigs by 58% and 61%, respectively; 1 mg/kg/d PFOA and 2 mg/kg/d GenX lowered Trigs by 37% and 43%, respectively; Table 2). At E17.5, dam serum Trigs were significantly lower in 5 mg/kg/d PFOA and 10 mg/kg/d GenX-treated mice (66% lower and 74% lower, respectively) (Table 3).

At E11.5, serum Glu levels in dams exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX were lower relative to controls (20% and 18% lower, respectively), but this shift did not reach statistical significance (Table 2; $p = 0.06$ and $p = 0.20$, respectively). By E17.5, serum Glu remained lower in 5 mg/kg/d PFOA-exposed mice and 10 mg/kg/d GenX-exposed mice, but this shift was also not statistically significant (Table 3; $p = 0.41$ and $p = 0.42$, respectively).

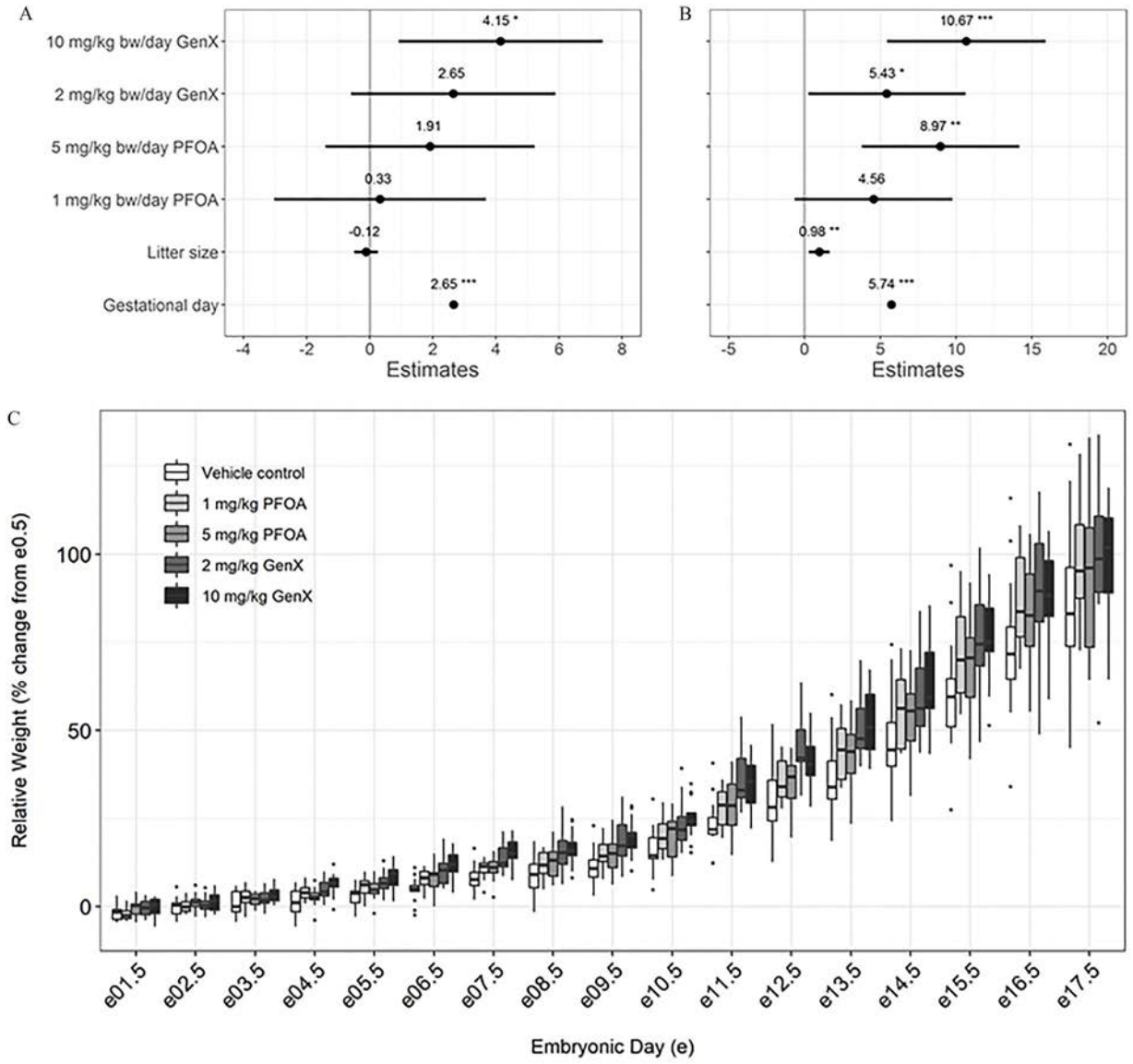

Figure 3. Gestational weight gain (GWG) repeated-measure, mixed-effect model estimates for pregnant dams exposed to perfluorooctanoic acid (PFOA) and GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)]. Effect estimates for pregnant dams exposed through embryonic day 11.5 (A) or 17.5 (B) are centered around the vehicle control group ($y = 0$) and show the point estimate of the relative change in dam weight (percent change from E0.5) with 95% confidence intervals (CIs). (C) Boxplots of relative weight gain over time, with the upper and lower hinges corresponding to the first and third quartiles (25th and 75th percentiles), the middle hinge corresponding to the median, and the upper whisker extending to the highest value that is within 1.5 times the distance between the first and third quartiles [interquartile range (IQR)] of the hinge and the lower whisker extending to the lowest value within 1.5 times the IQR of the hinge. $n = 11$–13 dams per treatment group. $^{*}p < 0.05$. $^{**}p < 0.01$. $^{***}p < 0.001$. Beta estimate 95% confidence intervals do not overlap zero. [Repeated-measures mixed-effect model adjusting *a priori* for litter size and gestational (embryonic) day as fixed effects and the dam as a random effect, vehicle control as reference group].

At E11.5, dams exposed to 2 mg/kg/d GenX exhibited higher Chol and HDL compared with controls (66% and 56% higher, respectively) (Table 2). E11.5 dams exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX similarly exhibited higher Chol and HDL levels relative to controls, but this shift did not reach statistical significance ($p = 0.42$ and $p = 0.42$, respectively) (Table 3). By E17.5, treatment-related effects on Chol and HDL appeared to be generally attenuated (Table 3). At E17.5, mice exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX exhibited lower LDL (50% lower and 31% lower, respectively), but only the shift in PFOA-exposed mice was significant (Table 3).

Dams exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX exhibited higher ALT relative to controls (a 172% increase and a 200% increase, respectively), but these shifts were not statistically significant with post hoc corrections (Table 2). By E17.5, treatment group–related effects on ALT were attenuated. At E17.5, dams exposed to 5 mg/kg/d PFOA exhibited lower serum ALB, increased AST, increased SDH, and lower total serum protein relative to controls (Table 3). Similar shifts occurred in mice exposed to 10 mg/kg/d GenX with respect to AST, SDH, and TP, but were not statistically significant (Table 3). Overall, GenX and PFOA liver pathology was consistent across dose groups and time points (100% incidence of cytoplasmic alteration) (Table S6 and S7), while changes in ALT, AST, and SDH measurements were not statistically significant across all GenX or PFOA dose groups or time points.

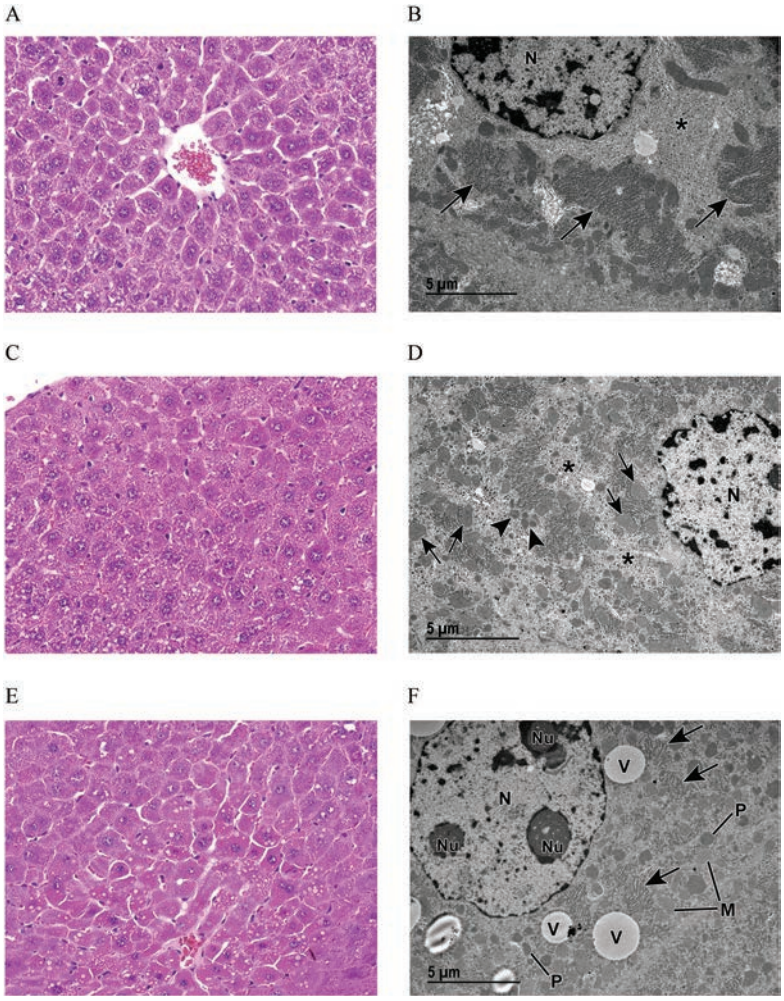

Figure 4. Light and transmission electron microscopy (TEM) of liver from vehicle control (VC) and perfluorooctanoic acid (PFOA)–exposed pregnant dams at embryonic day (E) 17.5. (A) Light microscopic image at 40× magnification of liver from a VC pregnant dam (control) showing centrilobular hepatocellular hypertrophy with karyomegaly, increased basophilic granular cytoplasm, and decreased glycogen. (B) Corresponding TEM magnification shows prominent rough endoplasmic reticulum (arrows) with abundant ribosomes and evenly dispersed, abundant glycogen (asterisk) (see Figure S2A). (C) Light microscopic image at 40× magnification of liver from a pregnant dam at E17.5 and treated with 1 mg/kg/d PFOA. (D) Although this liver appears to be within normal limits when viewed with light microscopy, TEM reveals an increase in scattered vacuoles (see Figure S2B); decreased, evenly dispersed glycogen (asterisks); as well as abundant mitochondria (arrows) and peroxisomes (arrowheads). (E) Light microscopic image at 40× magnification of liver from a pregnant dam at E17.5 and treated with 5 mg/kg/d PFOA. Increased cytoplasmic vacuoles are evident at this light microscopic level. (F) TEM reveals abundant cytoplasmic organelles consistent with mitochondria (M) and peroxisomes (P), extensive vacuoles (V), less prominent rough endoplasmic reticulum (arrows) with fewer ribosomes and less abundant glycogen (see Figure S2C,S2D). Note: N, nucleus; NU, nucleolus; TEM, transmission electron microscopy.

Embryo and Placenta Outcomes

Although the number of implantation sites, viable embryos, nonviable embryos, or resorptions did not significantly differ across treatment groups at E11.5 or E17.5 (Table S4), we evaluated embryos and their placentas for differences in weight. At E11.5, there were no significant differences in viable embryo weight, placental weight, or embryo:placenta weight ratios across treatment groups relative to vehicle controls (Table S8). At E17.5, significantly lower viable embryo weight was observed in 5 mg/kg/d PFOA-treated mice (5 mg/kg/d PFOA embryos were 129 mg lower in BW than vehicle control embryos based on mixed-effect model estimates; Figure 6A and Table S8). At E17.5, placental weight was significantly higher in 5 mg/kg/d PFOA- and 10 mg/kg/d GenX-treated mice relative to vehicle controls (an estimated 21 mg and 15.5 mg increase in placental weight relative to controls, respectively; Figure 6B and Table S8). Embryo:placenta weight ratios (mg:mg) were significantly reduced relative to controls in 5 mg/kg/d PFOA- and 10 mg/kg/d GenX-treated mice at E17.5 (Figure 6C and Table S8).

At E11.5, placental lesions were relatively sparse and mostly included labyrinth atrophy, labyrinth necrosis, or early fibrin clot formation. At E11.5, there were no differences in the incidence of placentas WNL across treatment groups (Table S9). At E17.5, placental abnormalities were observed in all treatment groups and tended to occur as litter-specific effects (e.g., most or all placenta within one litter were affected), and the most common lesions included labyrinth congestion (Figure 7B), labyrinth atrophy (Figure 7C), early fibrin clots (Figure S3A), labyrinth necrosis (Figure 7D), and placental nodules (Figure S3B). Placental nodules were most likely resorption of an adjacent twin. Placentas of mice exposed to 5 mg/kg/d PFOA exhibited labyrinth congestion as the most common lesion, whereas placentas of mice exposed to either 2 mg/kg/d or 10 mg/kg/d GenX primarily exhibited atrophy of the labyrinth (Figure 8 and Table S10). Early fibrin clots were most common in placentas of mice exposed to 10 mg/kg/d GenX (Figure 8 and Table S10). At E17.5, placentas WNL were significantly lower in mice exposed to 5 mg/kg/d PFOA or 10 mg/kg/d GenX when all evaluated

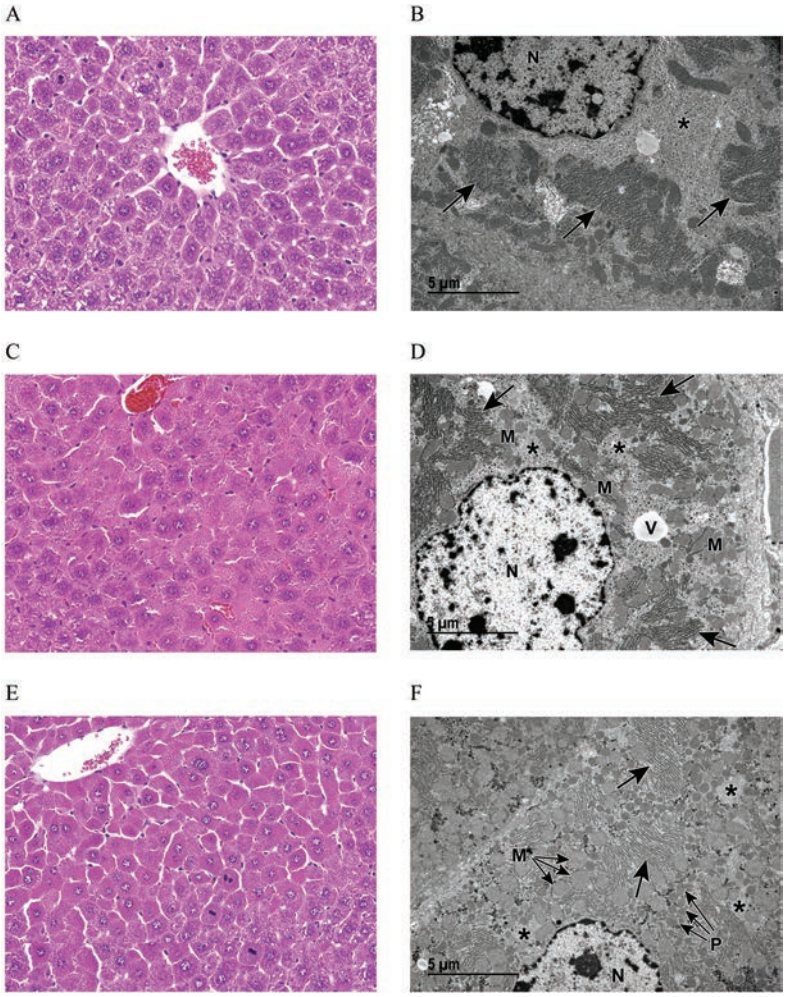

Figure 5. Light and transmission electron microscopy (TEM) of liver from vehicle control (VC) and GenX-exposed pregnant dams at embryonic day (E) 17.5. (A) Light microscopic image at 40× magnification of liver from a VC pregnant dam showing centrilobular hepatocellular hypertrophy with karyomegaly, increased basophilic granular cytoplasm, and decreased glycogen. (B) Corresponding medium TEM magnification shows prominent rough endoplasmic reticulum (arrows) with abundant ribosomes and evenly dispersed, abundant glycogen (asterisk) (see Figure S2A). (C) Light microscopy at 40× magnification, and (D) transmission electron microscopy of liver from a pregnant dam at E17.5 treated with 2 mg/kg/d GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] or 10 mg/kg/d GenX (E and F). Marked cytoplasmic alteration is evident in (C) and (E). TEM (D and F; see Figure S2E and S2F, respectively) reveals an abundance of cytoplasmic organelles, consistent with mitochondria (M) and peroxisomes (P) that increase with increasing dose (D compared to F). Note also the decreased glycogen (asterisks) as well as the vacuole (V) and rough endoplasmic reticulum (arrows). N, nucleus.

placentas were considered as independent observations (regardless of litter of origin) (Table S10). Placental lesions were also evaluated to account for litter effects by using the proportion of placenta within a litter that was WNL (percent WNL). Comparing placenta using this method showed a reduction in placenta WNL in mice exposed to 5 mg/kg/d PFOA, 2 mg/kg/d GenX, and 10 mg/kg/d GenX (Table S10).

Placental Thyroid Hormones

For all placental TH endpoints, sex × treatment interaction and sex as a covariate did not significantly influence model fit and were not incorporated in the final linear model (Table S11). Placentas exposed to 10 mg/kg/d GenX had significantly higher T4 relative to controls (60% increase) (Table 4). This effect occurred in both male and female placentas, but statistical significance was attenuated post hoc in sex-stratified models likely due to low sample sizes. There was a trend towards a significant effect of higher T4 in placentas exposed to 2 mg/kg/d GenX (38% increase; Table 4), but this effect was attenuated after applying post hoc corrections for multiple tests. Similarly, a trend toward a lower T3:T4 ratio was observed in placentas exposed to 10 mg/kg/d GenX, but this effect was attenuated after applying post hoc corrections. There were no other significant effects of sex or treatment on placental rT3, T3, T3:T4 ratio, or rT3:T4 ratio.

Discussion

Our prior work in mice has consistently shown reduced birth weight resulting from gestational exposure to PFOA (Macon et al. 2011; White et al. 2007), but we did not examine effects on the placenta, a critical organ that facilitates embryo growth, nor did we examine the effects of replacement PFAS congeners. Here we present evidence consistent with previous reports of PFOA-reduced embryo growth and provide novel evidence indicating that the pregnant dam liver and placenta are sensitive targets of both PFOA and a replacement PFAS, GenX. Adverse placental and maternal effects were most prominent in late gestation (E17.5) in mice gestationally exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX, but 2 mg/kg/day GenX also exhibited significant effects on maternal liver and placenta. Future studies

Table 2. Clinical chemistry panel of dam serum at embryonic day 11.5.

Measurement	Vehicle control [mean ± SD (*n*)]	1 mg/kg/d PFOA [mean ± SD (*n*)]	5 mg/kg/d PFOA [mean ± SD (*n*)]	2 mg/kg/d GenX [mean ± SD (*n*)]	10 mg/kg/d GenX [mean ± SD (*n*)]
ALB (g/dL)	2.48 ± 0.18 (5)	2.42 ± 0.22 (5)	2.36 ± 0.21 (5)	2.75 ± 0.33 (4)	2.8 ± 0.17 (3)
ALP (U/L)	68.8 ± 13.0 (5)	54.6 ± 4.4 (5)	56.6 ± 35.6 (5)	58.4 ± 9.0 (5)	83.0 ± 25.8 (5)
ALT (U/L)	26.0 ± 5.6 (5)	28.8 ± 11.5 (5)	70.8 ± 16.2 (5)	24.2 ± 13.7 (5)	78.2 ± 62.0 (5)
AST (U/L)	63.6 ± 9.9 (5)	144.6 ± 167.6 (5)	92.6 ± 20.3 (5)	69.0 ± 22.0 (5)	136.8 ± 138.9 (4)
BUN (mg/dL)	16.0 ± 2.1 (5)	15.0 ± 2.7 (5)	15.8 ± 1.3 (5)	18.3 ± 4.6 (4)	13.7 ± 1.5 (3)
Chol (mg/dL)	56.4 ± 4.6 (5)	68.8 ± 18.0 (5)	69.4 ± 9.9 (5)	93.4 ± 27.8* (5)	77.0 ± 16.4 (4)
Cre (mg/dL)	0.21 ± 0.02 (5)	0.2 ± 0.05 (5)	0.18 ± 0.03 (5)	0.2 ± 0.04 (4)	0.18 ± 0.02 (3)
Glu (mg/dL)	275.2 ± 39.5 (5)	278.4 ± 27.8 (5)	220.4 ± 22.1 (5)	249.3 ± 25.8 (4)	226.7 ± 28.9 (3)
HDL (mg/dL)	32.2 ± 1.5 (5)	34.8 ± 10.9 (5)	42.6 ± 4.0 (5)	50.2 ± 15.7* (5)	43.3 ± 6.1 (4)
LDL (mg/dL)	10.8 ± 1.3 (5)	12.2 ± 1.9 (5)	10.6 ± 1.5 (5)	15 ± 4.8 (4)	12.5 ± 1.9 (4)
SDH (U/L)	9.4 ± 7.5 (5)	8.4 ± 7.8 (5)	12.4 ± 8.3 (5)	7.0 ± 6.5 (4)	8.0 ± 3.65 (4)
TBA (μM/L)	2.0 ± 0.71 (5)	1.5 ± 0.58 (4)	2.0 ± 0.0 (5)	1.4 ± 0.55 (5)	35.3 ± 67.8 (4)
TP (g/dL)	4.22 ± 0.18 (5)	4.04 ± 0.3 (5)	3.78 ± 0.22 (5)	4.5 ± 0.48 (4)	4.37 ± 0.29 (3)
Trig (mg/dL)	205.6 ± 56.0 (5)	130.4 ± 16.2* (5)	86.4 ± 15.8* (5)	117.6 ± 33.9* (5)	80.3 ± 14.4* (4)
Ucrea (mg/dL)	54.4 ± NA (1)	92.0 ± 13.1 (4)	50.1 ± 33.8 (4)	53.2 ± 14.0 (3)	82.9 ± 33.2 (5)

Note: ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; Chol, cholesterol; Cre, creatinine; Glu, glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; SDH, sorbitol dehydrogenase; TBA, total bile acids; TP, total protein; Trig, triglycerides; Ucrea, urinary creatinine; U/L, units per liter. *$p < 0.05$ relative to vehicle control [analysis of variance (ANOVA) with post hoc multiple comparison correction using Tukey contrasts].

should investigate adverse effects at doses lower than 2 mg/kg/d GenX to determine more precise percent responses at different lower dose levels using a benchmark dose approach.

It is well documented in humans and animal models that PFAS readily pass from maternal serum to the developing embryo via the placenta (Chen et al. 2017; Yang et al. 2016a, 2016b) and that PFOA transplacentally transfers to the mouse offspring (Fenton et al. 2009). Here, we report transplacental transfer of both PFOA and GenX, higher placenta weight, higher incidence of placental lesions, and lower embryo–placenta weight ratios in mice exposed to 5 mg/kg/d PFOA or 10 mg/kg/d GenX.

In humans, placenta weight and placental-to-fetal (also reported as feto-placental) weight ratios are clinically relevant end points that have been associated with adverse pregnancy outcomes (Hutcheon et al. 2012; Risnes et al. 2009; Thornburg et al. 2010). The placenta is a critical organ that mediates the transport of nutrients, oxygen, waste, and xenobiotics between mother and embryo, and it is rarely evaluated in reproductive toxicity studies. We chose the placenta as a focal end point due to its importance in studies of human pregnancy outcomes (Hutcheon et al. 2012; Risnes et al. 2009), its role as a programming agent of latent health outcomes in both the mother and child (Thornburg et al. 2010), and our own hypothesis that it is a key target tissue of PFAS.

Placental insufficiency (PI) occurs when functional capacity of the placenta is limited or deteriorates, resulting in reduced transplacental transfer of oxygen and nutrients to the fetus (Gagnon 2003). Reduction or impairment of placental blood flow (Chaddha et al. 2004), aberrant fibrin depositions or other thrombo-occlusive damage in the placenta (Chaddha et al. 2004), and disruption of maternal–placental THs (Belet et al. 2003) are all believed to contribute to PI pathogenesis in women. We provide evidence illustrating pathological and physiological features that are concordant with PI in our experimental mouse model. Here we show maternal exposure to PFOA- or GenX-induced atrophy, necrosis, and congestion of the murine placental labyrinth (suggestive of impaired transplacental transfer of nutrients and/or oxygen), aberrant formation of early fibrin clots, and disruption of placental TH (GenX only). These data are suggestive of a PI phenotype induced by maternal exposure to PFAS in mice that deserves further investigation.

In epidemiological studies, disproportionately large placentas increase the risk for adverse health outcomes in neonates (Hutcheon et al. 2012) and adult offspring (Risnes et al. 2009). The placenta influences cardiovascular disease (CVD) risk in the

Table 3. Clinical chemistry panel of dam serum at embryonic day 17.5.

Measurement	Vehicle control [mean ± SD (*n*)]	1 mg/kg/d PFOA [mean ± SD (*n*)]	5 mg/kg/d PFOA [mean ± SD (*n*)]	2 mg/kg/d GenX [mean ± SD (*n*)]	10 mg/kg/d GenX [mean ± SD (*n*)]
ALB (g/dL)	2.23 ± 0.21 (4)	2.04 ± 0.09 (5)	1.53 ± 0.27* (6)	2.32 ± 0.26 (5)	2.26 ± 0.3 (5)
ALP (U/L)	58.0 ± 7.8 (4)	50.2 ± 4.2 (5)	74.8 ± 23.8 (6)	55.4 ± 11.8 (5)	88.8 ± 13.0* (5)
ALT (U/L)	13.0 ± 7.5 (4)	7.0 ± 4.3 (5)	16.8 ± 7.7 (6)	4.4 ± 3.9 (5)	9.6 ± 2.1 (5)
AST (U/L)	81.0 ± 6.5 (4)	73.0 ± 14.0 (5)	172.2 ± 63.1* (6)	65.6 ± 12.1 (5)	113.2 ± 36.6 (5)
BUN (mg/dL)	16.0 ± 2.9 (4)	16.4 ± 1.7 (5)	18.7 ± 5.3 (6)	13.6 ± 1.1 (5)	15.2 ± 1.8 (5)
Chol (mg/dL)	75.5 ± 11.6 (4)	83.8 ± 20.0 (5)	68.5 ± 16.4 (6)	86.6 ± 17.1 (5)	97.4 ± 8.4 (5)
Cre (mg/dL)	0.18 ± 0.04 (4)	0.2 ± 0.01 (5)	0.16 ± 0.06 (6)	0.17 ± 0.03 (5)	0.15 ± 0.06 (5)
Glu (mg/dL)	129.3 ± 11.7 (4)	121.0 ± 17.3 (5)	112.0 ± 15.8 (6)	123.2 ± 13.1 (5)	111.6 ± 15.5 (5)
HDL (mg/dL)	34.0 ± 10.2 (4)	37.2 ± 6.2 (5)	38.8 ± 11.2 (6)	39.4 ± 8.5 (5)	50.0 ± 8.9 (5)
LDL (mg/dL)	22.0 ± 0.8 (4)	24.0 ± 10.7 (5)	11.0 ± 3.0 (5)	20.0 ± 3.9 (5)	15.2 ± 2.9 (5)
SDH (U/L)	5.5 ± 7.9 (4)	3.4 ± 6.1 (5)	24.3 ± 11.2* (6)	1.2 ± 2.2 (5)	11.4 ± 6.8 (5)
TBA (μM/L)	3.8 ± 0.96 (4)	3.0 ± 1.2 (5)	8.0 ± 7.9 (6)	4.8 ± 3.0 (5)	6.2 ± 4.2 (5)
TP (g/dL)	4.2 ± 0.37 (4)	3.9 ± 0.11 (5)	2.8 ± 0.39* (6)	4.1 ± 0.36 (5)	3.9 ± 0.52 (5)
Trig (mg/dL)	472.5 ± 78.9 (4)	364.0 ± 272.9 (5)	159.0 ± 65.5* (6)	257.0 ± 120.3 (5)	120.6 ± 31.7* (5)
Ucrea (mg/dL)	25.8 ± 15.8 (2)	24.7 ± 23.1 (2)	11.5 ± 5.9 (3)	18.6 ± 5.1 (4)	20.2 ± 15.7 (4)

Note: ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; Chol, cholesterol; Cre, creatinine; Glu, glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; SDH, sorbitol dehydrogenase; TBA, total bile acids; TP, total protein; Trig, triglycerides; Ucrea, urinary creatinine. *$p < 0.05$ relative to vehicle control (ANOVA with post hoc multiple comparison correction using Tukey contrasts).

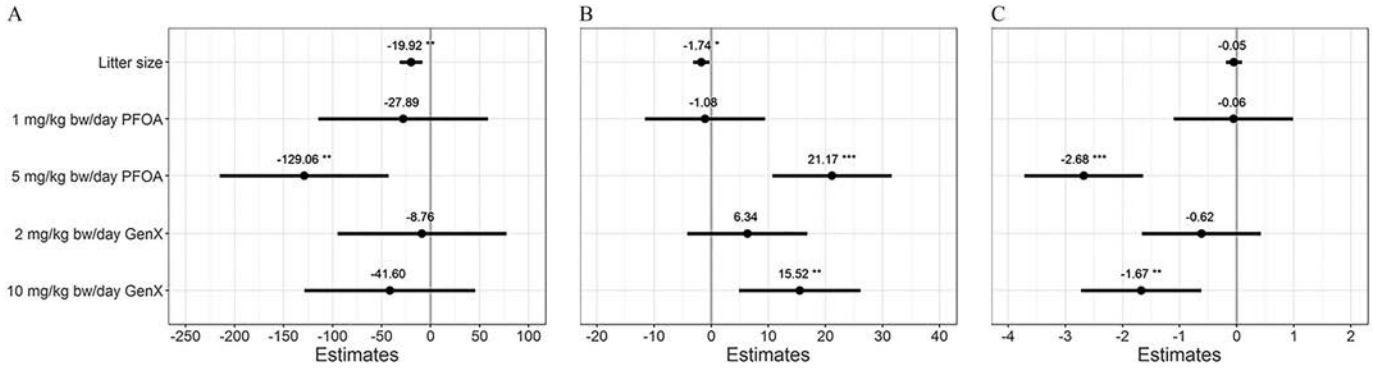

Figure 6. Mixed-effect model estimates for (A) embryo weight (mg), (B) placental weight (mg), and (C) embryo:placenta weight ratios (mg:mg) after exposure *in utero* to perfluorooctanoic acid (PFOA) or GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] at embryonic day (E) 17.5. Effect estimates are centered around the vehicle control group ($y = 0$) and show the point estimate of the relative change in weight (in milligrams; A and B) or weight ratio (mg:mg; C) with 95% confidence intervals (CIs). $^{*}p < 0.05$. $^{**}p < 0.01$. $^{***}p < 0.001$. Beta estimate 95% confidence intervals do not overlap zero (mixed-effect model adjusting *a priori* for litter size as a fixed effect and the dam as a random effect, vehicle control as reference group). Adjusted estimates and 95% CIs are shown in Table S8.

offspring (Risnes et al. 2009), and the functional capacity of the placenta is likely the driver of fetal heart fitness (Thornburg et al. 2010). Placentas that are disproportionately large relative to fetal size tend to exhibit reduced functional capacity with respect to optimal blood flow and vascular resistance (Risnes et al. 2009; Salafia et al. 2006), which could lead to both adverse perinatal (Hutcheon et al. 2012) and adult CVD outcomes (Thornburg et al. 2010). Here we show higher placenta weights that were disproportionate to embryo weights in mice exposed to PFOA and GenX. Whether the increased placental weight is due to pathological changes or is a compensatory mechanism to protect the developing fetus is not known. The extent to which gestational exposure to these environmental contaminants could adversely impact perinatal and adult offspring health outcomes, especially cardiovascular outcomes, should be the focus of future studies.

A previous report has shown dose-dependent necrotic changes in the placenta of mice exposed to 10 mg/kg/d and 25 mg/kg/d PFOA, and pup mortality and gestational weight loss were evident (Suh et al. 2011). Here, placental lesions in mice exposed to 2 mg/kg/d GenX, 10 mg/kg/d GenX, and 5 mg/kg/d PFOA at E17.5 occurred at a significantly higher incidence compared to controls, and the labyrinth was the specific target. This is significant because the maternal–embryo exchange of oxygen, nutrients, and waste occurs in the placental labyrinth. Adverse placental effects of 5 mg/kg/d PFOA and 10 mg/kg/d GenX occurred at both the litter level as well as across all placenta evaluated, regardless of litter, and adverse placental effects of 2 mg/kg/d GenX were significant when considered at the level of the litter as a unit. The lowest doses tested in this study resulting in adverse placental pathology were 2 mg/kg/d GenX and 5 mg/kg/d PFOA. Given that maternal serum accumulation and embryo deposition of PFOA and GenX were similar at the high (5 mg/kg PFOA vs. 10 mg/kg GenX) and low doses (1 mg/kg PFOA vs. 2 mg/kg GenX) and that the placenta is at the interface between these two compartments, the disparate patterns in adverse placenta histopathology suggest that the placenta may be more sensitive to the effects of GenX vs. PFOA. The mechanisms of toxicity towards the placenta may also

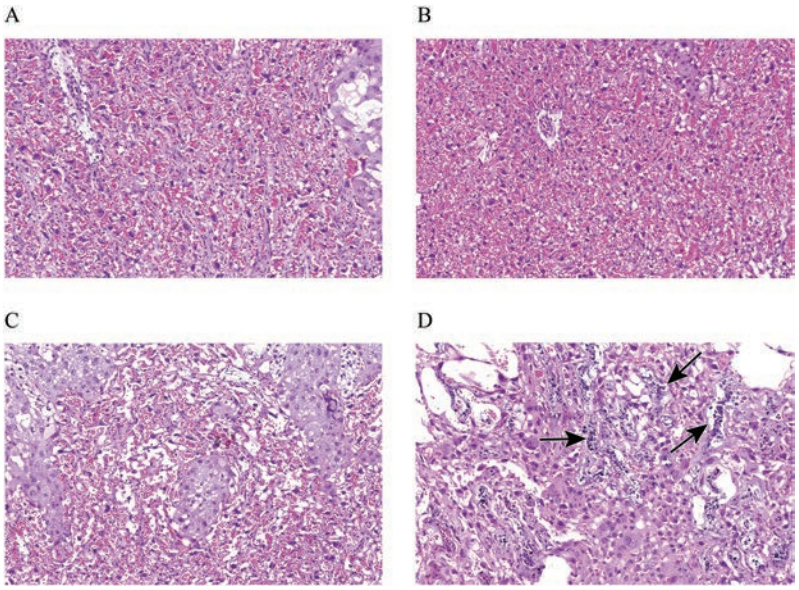

Figure 7. Representative examples of histopathological placenta findings observed in dams at embryonic day (E) 11.5 and E17.5, treated with perfluorooctanoic acid (PFOA) or GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)]. (A) Normal labyrinth from a vehicle control dam at E17.5. (B) Labyrinth congestion in a dam at E17.5 that was treated with 10 mg/kg/d GenX (C) Moderate labyrinth atrophy of the trilaminar trophoblast layer at E17.5 in a dam treated with 10 mg/kg/d GenX. (D) Labyrinth necrosis (arrows) in an E17.5 dam that was treated with 10 mg/kg/d GenX. All images at 20× magnification.

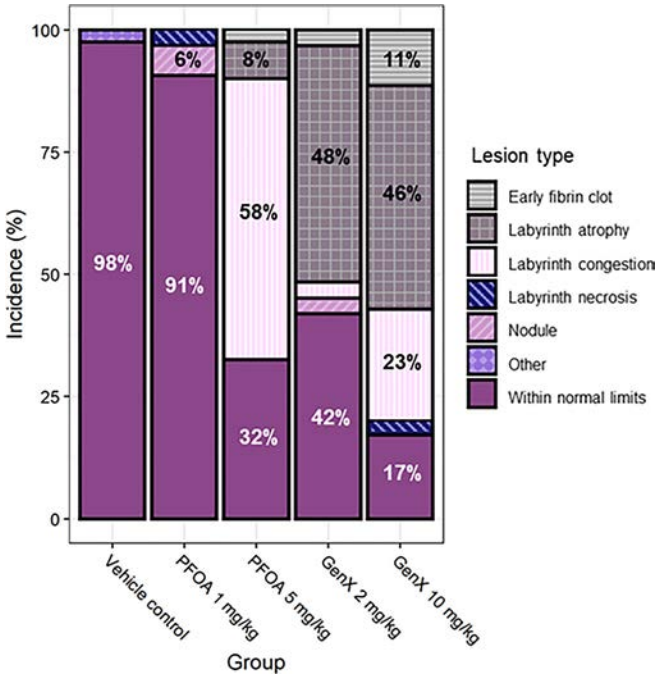

Figure 8. Incidence of placenta lesions across treatment groups at embryonic day 17.5. $n = 5$–6 litters with 31–41 placentas evaluated per treatment group (an average of 6–8 placentas per litter). Incidence values <4% are not numerically indicated, but all values and statistical comparisons of placenta lesion incidences across treatment groups at E17.5 are shown in Table S10.

differ between the two PFAS and will be pursued in ongoing studies.

TH play a critical role in neurodevelopment (de Escobar et al. 2004; Porterfield 1994). PFAS are well-documented thyroid disrupters in humans (Coperchini et al. 2017; Webster et al. 2016), including in pregnant women (Ballesteros et al. 2017; Berg et al. 2015; Wang et al. 2014; Webster et al. 2014). Generally, maternal PFAS levels during pregnancy are associated with shifts in TH levels consistent with hypothyroidism (e.g., elevated thyroid-stimulating hormone), which is associated with increased risk for low birth weight (Alexander et al. 2017). It is possible that PFAS chemicals exert some adverse effects on embryo growth via TH disruption across the maternal–placental–embryo unit. Indeed, Conley et al. (2019) reported maternal serum total triiodothyronine (T3) and thyroxine (T4) were reduced in rats exposed to 125–500 mg/kg/d HFPO-DA (GenX) during gestational days 14–18. Maternal serum TH could not be measured due to volume constraints in our study. As the placenta regulates the degree to which maternal THs pass to the developing fetus, and it maintains the optimal balance of the TH throughout embryo development (Chan et al. 2009), the relationship between PFAS-induced maternal TH changes and placental function requires additional study, especially given the role of TH in fetal neurodevelopment.

In a systematic review and meta-analysis of nonhuman evidence for effects of PFOA on BW, it was estimated that a 1-unit (1 mg/kg BW/d) increase in PFOA is associated with a −0.023 g (95% CI: −0.029, −0.016) shift in pup birth weight (Koustas et al. 2014). Here we report a −0.028 g (95% CI: −0.114, 0.586) shift in embryo weight on E17.5 in mice exposed to 1 mg/kg/d PFOA and a −0.129 g (95% CI: −0.215, −0.043) shift in mice exposed to 5 mg/kg/d PFOA. Effects on embryo weight at E17.5 in this study can be summarized as most severe to least severe: 5 mg/kg/d PFOA (−0.129 g), 10 mg/kg/d GenX (−0.042 g), 1 mg/kg/d PFOA (−0.023 g), and 2 mg/kg/d GenX (−0.009 g). An industry study of CD-1 mice exposed to 5 mg/kg/d HFPO-DA (GenX) from preconception through weaning showed reduced pup weight at postnatal day (PND) 1 that persisted through PND 21 with effects more severe in male offspring (DuPont-18,405-1,037). In rats, mean embryo weights were decreased in rats exposed to 100 mg/kg/d HFPO-DA (GenX) for 15 d of gestation (Edwards 2010a), and in a different study, female birth weights were reduced after 5 d of gestational exposure at 125 mg/kg (Conley et al. 2019). To our knowledge, there are no human data showing associations between maternal GenX exposure and birth weight outcomes.

Several human cohort studies have shown that higher levels of prenatal or early-life PFOA exposure is associated with increased adiposity in childhood (Braun et al. 2016; Fleisch et al. 2017) and metabolic disruption in young adulthood (Domazet et al. 2016). Additionally, it is known that low birth weight is associated with adult diseases, including metabolic syndrome in both humans and animals (Barker 2004). Due to the environmental ubiquity of a mixture of PFAS chemicals, it is difficult to unravel the relative contributions of prenatal and postnatal (e.g., chronic, lifelong) exposure and adverse health outcomes. Animal studies allow for discrete measurement of health outcomes associated with specific critical periods of exposure, and future work should investigate metabolic disruption in offspring exposed *in utero* to provide key insights on the metabolic programming capacity of PFAS.

In the present study, PFOA (5 mg/kg/d) and GenX (2 mg/kg/d or 10 mg/kg/d) exposures resulted in significantly higher GWG in mice, with significant effects emerging at an earlier point in gestation in mice exposed to GenX and occurring at a lower dose than PFOA (2 mg/kg/d GenX vs. 5 mg/kg/d PFOA). In contrast, a decrease in mean maternal weight gain was reported in a recent study of gestational exposure to GenX in rats exposed to 250 or 500 mg/kg/d (Conley et al. 2019). Although these findings are not consistent with the higher GWG reported here, it is possible that statistical methods (absolute change in maternal weight vs. relative change in weight analyzed using repeated measures models), differing windows of exposure (5 d during mid- to late gestation vs. exposure throughout gestation), and interspecies differences in preliminary PFAS elimination

Table 4. Placental thyroid hormone measurements at embryonic day 17.5.

Hormone	Vehicle control {mean ± SD [*n* (a, b)]}	1 mg/kg/d PFOA {mean ± SD [*n* (a, b)]}	5 mg/kg/d PFOA {mean ± SD [*n* (a, b)]}	2 mg/kg/d GenX {mean ± SD [*n* (a, b)]}	10 mg/kg/d GenX {mean ± SD [*n* (a, b)]}
rT3 (ng/g)	1.2 ± 0.7 [5 (4, 1)]	0.7 ± 0.4 [6 (3, 3)]	1.4 ± 0.7 [5 (5, 0)]	1.7 ± 0.8 [6 (6, 0)]	1.6 ± 0.3 [6 (6, 0)]
T3 (ng/g)	0.3 ± 0.2 [6 (1, 5)]	0.2 ± 0 [6 (0, 6)]	0.2 ± 0 [4 (0, 4)]	0.3 ± 0.2 [5 (0, 5)]	0.2 ± 0 [6 (0, 6)]
T4 (ng/g)	3.8 ± 0.6 [6 (6, 0)]	2.5 ± 1.0 [6 (6, 0)]	2.8 ± 1.3 [6 (6, 0)]	5.3 ± 1.7 [6 (6, 0)]	6.1 ± 1.1* [6 (6, 0)]
T3:T4 ratio	0.07 ± 0.04 [6]	0.09 ± 0.03 [6]	0.07 ± 0.02 [4]	0.05 ± 0.01 [5]	0.03 ± 0.01 [6]
rT3:T4 ratio	0.33 ± 0.19 [5]	0.30 ± 0.21 [6]	0.45 ± 0.05 [5]	0.32 ± 0.12 [6]	0.27 ± 0.08 [6]

Note: Sample sizes are expressed as the total number of samples (*n*) as well as the number of samples above the MDL (a) and below the MDL (b). Nonquantifiable samples below the MDL were imputed using the calculation MDL × 0.5. MDL values were: T4, 0.84 ng/g; T3, 0.42 ng/g; rT3, 0.67 ng/g. MDL, method detection limit; rT3, reverse triiodothyronine; SD, standard deviation; T3, triiodothyronine; T4, thyroxine. *$p < 0.05$ relative to vehicle control [analysis of variance (ANOVA) with post hoc multiple comparison correction using Tukey contrasts].

rates [GenX elimination half-life in rats: ~5 h vs. ~20 h in mice, (Gannon et al. 2016)] could explain these disparate results. It is possible that different elimination rates of the compound make the comparison of equivalent or similar external doses a challenge. In fact, dam serum concentrations of rats exposed to 500 mg/kg/d from gestation day (GD) 14-18 reported in Conley et al. (2019) were of similar magnitude to those observed in mice exposed to 10 mg/kg/d throughout gestation in the present study (~100 μg/mL). Similarly, serum concentrations from pregnant mice in the current study exposed to 2 mg/kg/d GenX were roughly equivalent (~33 μg/mL) to serum concentrations obtained from rat dams exposed to 62.5 mg/kg/d GenX in the study by Conley et al. (2019).

Higher GWG observed in our PFOA-exposed mice is consistent with findings reported in humans; interquartile range increases in GWG were associated with elevated cord blood levels of PFOA (odds ratio = 1.33; 95% CI: 1.13,1.56) (Ashley-Martin et al. 2016). Similarly, other legacy PFAS compounds such as perfluorooctanesulfonic acid are positively associated with GWG (Jaacks et al. 2016). However, our data describing the relationship between maternal exposure to GenX and increased GWG in a mouse model are novel. Importantly, higher GWG is associated with adverse outcomes for both mother and infant in humans, including increased risk for pregnancy-associated hypertension (with or without smaller birth weights), gestational diabetes, postpartum weight retention, increased risk for unsuccessful breastfeeding, and increased risk for stillbirth, infant mortality, and preterm birth (Rasmussen and Yaktine 2009). These disorders share many risk factors, but it is not fully understood to what extent their etiologies are interrelated and/or interdependent (Villar et al. 2006) or what mechanisms may be driving them. Our data suggest a need for additional study of the adverse maternal and offspring health outcomes associated with GenX exposure.

Liver toxicity is a consistent finding in animal studies of PFOA (Li et al. 2017) and other PFAS, but studies examining GenX are limited. Here, we report similar histopathological findings in livers of exposed pregnant dams to those previously described by our group (and others) in offspring prenatally exposed to PFOA, including increased extent of hepatocellular hypertrophy, cytoplasmic alteration, and increased mitochondria (Filgo et al. 2015; Lau et al. 2006). We hypothesize that the consistent and persistent hepatic cytoplasmic alterations seen following PFAS exposures lead to increased incidence and/or distribution of cell death, which is consistent with the decrease in mitotic figures compared to control liver sections. This constellation of lesions is considered adverse and is incompatible with long-term normal liver function. The maternal liver responds to estrogen produced by the placenta and produces thyroid-binding globulin, which, in turn, regulates the level of maternal circulating TH (Nader et al. 2009). It is possible that altered maternal liver function due to PFOA or GenX exposure plays an important role in mediating placental and embryo outcomes.

In addition to consistently observed histopathological changes in the liver induced by either PFOA or GenX, maternal clinical chemistry indicated shifts in liver enzymes, including higher ALT (10 mg/kg/d GenX; E11.5), higher ALP (10 mg/kg/d GenX; E17.5), higher AST (5 mg/kg/d PFOA; E17.5), and higher SDH (5 mg/kg/d PFOA; E17.5). Our TEM findings build upon a growing body of evidence demonstrating potential mechanisms of PFAS-induced hepatic toxicity other than PPAR and demonstrate this for the first time with GenX.

In a previous reproductive and developmental toxicity study of HFPO-DA (GenX) in CD-1 mice, 5 mg/kg/d was determined to be the NOAEL for reproductive toxicity and maternal systemic toxicity (based on microscopic changes in maternal liver; DuPont-18,405-1,037) (Edwards 2010b). Here, we are not able to report a NOAEL, as significant adverse effects occurred in the lowest GenX dose group evaluated in this study (2 mg/kg/d). We demonstrate adverse systemic toxicity of dams exposed to 2 mg/kg/d GenX, which include microscopic alterations in the liver, higher GWG, and higher incidence of placental lesions. Dam serum GenX concentrations obtained at E17.5 in the present study were comparable to dam plasma concentrations reported by DuPont-18,405-1,037: 22.9 μg/mL (present study, 2 mg/kg/d on E17.5), 36.4 μg /mL (DuPont-18,405-1,037, 5 mg/kg/d on lactation day 21), and 58.5 μg/mL (present study, 10 mg/kg/d on E17.5; compared in Figure S4). However, it should be noted that in the present study at all tested doses, both PFOA and GenX, maternal serum concentrations were higher at E11.5 than E17.5. This could be explained by maternal off-loading of body burden to developing embryos and other maternal tissues (i.e., liver) and rapid expansion of maternal blood volume throughout the course of pregnancy.

There are several limitations to this study regarding experimental design, sample sizes, and interspecies differences. Due to performing the experiment over two experimental blocks, some end points were only evaluated from one of the two blocks, limiting statistical power. It is possible that some effects would achieve statistical significance with a larger number of observations. The two-block design did not impair the strength of the effect when significant effects were present in end points evaluated at both time points, which was verified by statistical analysis. It is possible that variance in half-life, amount of exposure to these chemicals, and other interspecies differences may limit the human relevance of the findings reported here. Although the mouse and human both have discoid hemochorial placenta, the maternal–placental–embryo unit in mice differs from that in humans in other ways, including the labyrinthine vs. villous structure, the number of offspring carried during each pregnancy (~14 vs. ~1), and gestation length (~20 d vs. ~280 d). Although there are distinct interspecies differences between humans and mice, the outbred CD-1 mouse was selected in the current study due to its genetic diversity. While the CD-1 mouse is sensitive to PFOA, compared to other inbred mouse strains (Tucker et al. 2015), significant treatment-related effects were still detectable despite its greater biologic variability in response. It is not known whether there are strain differences in sensitivity to GenX, which should be investigated in future studies.

Conclusion

In a comparative reproductive and developmental study in mice of PFOA and a replacement, GenX, we report adverse effects of both compounds against the maternal–embryo–placenta unit. Both PFOA and GenX induced elevated GWG, higher maternal liver weights, adverse microscopic pathological changes in the maternal liver, and abnormal histopathological lesions in mature placenta. Importantly, we provide evidence that illustrates GenX (as low as 2 mg/kg/d) significantly affects the maternal–embryo–placenta unit differently than its predecessor PFOA and that this alternative compound may have a unique mechanism(s) of reproductive toxicity in this model system. Lastly, we build a case for the importance of evaluating the placenta as a critical tissue in studies of developmental and reproductive toxicity through utilizing clinically relevant, translational end points to illustrate the unique susceptibility of this organ to the adverse effects of GenX.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT G
TO AMENDED INTERVENOR COMPLAINT

A Section 508–conformant HTML version of this article is available at https://doi.org/10.1289/EHP4372.

Adverse Maternal, Fetal, and Postnatal Effects of Hexafluoropropylene Oxide Dimer Acid (GenX) from Oral Gestational Exposure in Sprague-Dawley Rats

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Background: Hexafluoropropylene oxide dimer acid [(HFPO-DA), GenX] is a member of the per- and polyfluoroalkyl substances (PFAS) chemical class, and elevated levels of HFPO-DA have been detected in surface water, air, and treated drinking water in the United States and Europe.

Objectives: We aimed to characterize the potential maternal and postnatal toxicities of oral HFPO-DA in rats during sexual differentiation. Given that some PFAS activate peroxisome proliferator-activated receptors (PPARs), we sought to assess whether HFPO-DA affects androgen-dependent development or interferes with estrogen, androgen, or glucocorticoid receptor activity.

Methods: Steroid receptor activity was assessed with a suite of *in vitro* transactivation assays, and Sprague-Dawley rats were used to assess maternal, fetal, and postnatal effects of HFPO-DA exposure. Dams were dosed daily via oral gavage during male reproductive development (gestation days 14–18). We evaluated fetal testes, maternal and fetal livers, maternal serum clinical chemistry, and reproductive development of F1 animals.

Results: HFPO-DA exposure resulted in negligible *in vitro* receptor activity and did not impact testosterone production or expression of genes key to male reproductive development in the fetal testis; however, *in vivo* exposure during gestation resulted in higher maternal liver weights (≥62.5 mg/kg), lower maternal serum thyroid hormone and lipid profiles (≥30 mg/kg), and up-regulated gene expression related to PPAR signaling pathways in maternal and fetal livers (≥1 mg/kg). Further, the pilot postnatal study indicated lower female body weight and lower weights of male reproductive tissues in F1 animals.

Conclusions: HFPO-DA exposure produced multiple effects that were similar to prior toxicity evaluations on PFAS, such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), but seen as the result of higher oral doses. The mean dam serum concentration from the lowest dose group was 4-fold greater than the maximum serum concentration detected in a worker in an HFPO-DA manufacturing facility. Research is needed to examine the mechanisms and downstream events linked to the adverse effects of PFAS as are mixture-based studies evaluating multiple PFAS. https://doi.org/10.1289/EHP4372

Introduction

Per- and polyfluoroalkyl substances (PFAS) are a group of high-profile contaminants of emerging concern; the concern is primarily due to extensive research indicating these compounds have extreme environmental persistence (Awad et al. 2011), widespread occurrence (Kaboré et al. 2018; Kannan et al. 2004; Pan et al. 2018), long biological half-lives (Li et al. 2018), and nearly ubiquitous human exposure (Calafat et al. 2007). Further, there is concern for human health effects due to laboratory animal and epidemiological research on both perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). When administered throughout gestation, both PFOS and PFOA have been shown to produce adverse effects in rodent models, including extensive pup mortality and reduced growth rates (Grasty et al. 2003; Lau et al. 2003; Thibodeaux et al. 2003), and their administration is also correlated with increased incidence rates of thyroid dysfunction (Coperchini et al. 2017) and low birth weight (Apelberg et al. 2007) in human populations. Because of the combination of these factors, PFOS was primarily phased out of production by 2002, and subsequently added to Annex B of the Stockholm Convention, and the U.S. EPA has set drinking water health advisories for PFOS and PFOA at 70 parts per trillion (U.S. EPA 2016b). Similarly, beginning in 2006 the major manufacturers of PFOA voluntarily agreed to phase out production by 2015 (U.S. EPA 2006). However, a variety of structural analogs have been developed and utilized as replacement compounds in the production of a range of consumer and industrial products for which fluoropolymers provide desirable characteristics (Wang et al. 2013; Wang et al. 2017b).

Hexafluoropropylene oxide dimer acid [(HFPO-DA), GenX] is a PFAS compound that is used as a polymerization aid in the manufacturing of high-performance fluoropolymers following the phase out of PFOA (Beekman et al. 2016). Recent environmental monitoring studies in North Carolina and the Netherlands have reported elevated levels of HFPO-DA, among other PFAS, in air, groundwater, and surface water sampled within the proximity of manufacturing sites and in drinking water originating from contaminated surface sources (Gebbink et al. 2017; McCord et al. 2018; Strynar et al. 2015; Sun et al. 2016). Despite the extensive *in vivo* toxicity research available for PFOS and PFOA, relatively little peer-reviewed experimental data exist for HFPO-DA or the other PFAS

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Supplemental Material includes complete data tables with means, standard errors, and samples sizes for all data depicted in figures and figures of fetal testis testosterone production and *in vitro* endocrine receptor transactivation assays.

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analogs that have been recently detected. In addition to peer-reviewed studies (Caverly Rae et al. 2015; Gannon et al. 2016; Rushing et al. 2017; Wang et al. 2017a), guideline registration studies from the manufacturer of HFPO-DA are publicly available (https://hero.epa.gov/hero/index.cfm/project/page/project_id/2627); however, even though *in utero* exposure to PFOS and other PFAS induced extensive neonatal mortality and reduced offspring body weights in rats, similar studies have not been conducted with HFPO-DA to our knowledge. Overall, the paucity of data has led to calls for coordinated efforts to screen and assess the toxicity of the myriad PFAS currently detected in environmental matrices (Bruton and Blum 2017; Wang et al. 2017b).

PFOS and PFOA are known activators of peroxisome proliferator-activated receptors (PPARs), primarily alpha (PPARα) and gamma (PPARγ) (Vanden Heuvel et al. 2006). HFPO-DA is hypothesized to activate PPARs based on observed up-regulation of PPAR-signaling pathway genes (Wang et al. 2017a), increased markers of liver peroxisome proliferation (DuPont 2008a, 2008b; Rushing et al. 2017), and increased liver weight in mice and/or rats (Caverly Rae et al. 2015; DuPont 2008a, 2008b; Rushing et al. 2017; Wang et al. 2017a). Some phthalate ester metabolites are also PPAR activators (Lapinskas et al. 2005) and *in utero* exposure reduces gene expression of steroidogenic enzymes and decreases production of testosterone in the testes of male offspring, leading to reproductive tract malformations in rats (Hannas et al. 2011; Mylchreest et al. 2002; Parks et al. 2000; Wilson et al. 2004b). Similarly, Zhao et al. (2014) reported that PFOS reduced testosterone production and impaired fetal rat Leydig cells following *in utero* exposure. The specific molecular initiating event(s) (MIE) by which PFOS and some phthalate esters produce male reproductive toxicity remain(s) elusive; however, it has been proposed that activation of PPAR, specifically PPARα, plays an essential role (Corton and Lapinskas 2005; Gazouli et al. 2002; Nepelska et al. 2015). If this MIE is truly responsible for the anti-androgenic effects of phthalates, then oral exposure to other proposed PPAR agonists, such as HFPO-DA, would be expected to reduce male testis testosterone production *in utero* and cause male rat reproductive tract malformations, similar to the active phthalates.

In regard to the above concerns, there were two goals for the present study. First, we were interested in identifying whether HFPO-DA, like other PFAS, activates PPAR signaling pathways and, if so, does this lead to a reduction in fetal testis testosterone production resulting in the subsequent increase in the incidence/severity of male reproductive defects. Second, we wanted to leverage these experiments to provide additional relevant *in vivo* data on the potential for gestational oral HFPO-DA exposure to produce toxic effects in the mother or offspring. We conducted studies with pregnant rats dosed during the specific gestational window critical to masculinization of the male fetal reproductive tract [gestation days (GD) 14–18] (Carruthers and Foster 2005). We evaluated and report on a range of effects primarily related to the maternal and fetal livers, circulating maternal thyroid hormones and lipids, and a single-dose level pilot study on postnatal development. Further, because of prior conflicting reports on the endocrine receptor activity of PFAS and the potential relevance to mammalian reproductive development, we assessed the estrogen, androgen, and glucocorticoid receptor activity (agonism/antagonism) of HFPO-DA using *in vitro* transcriptional activation assays.

Methods

Dosing Solutions

Dosing solutions were prepared using high-performance liquid chromatography-grade water purchased from Honeywell Research Chemicals and HFPO-DA ammonium salt (CAS: 62037-80-3; Product No.: 2122-3-09; Lot: 00005383) purchased from SynQuest Laboratories. HFPO-DA purity was 100% as determined by the supplier via perchloric acid titration. Dosing was administered once daily via oral gavage at 2.5 mL/kg body weight across a range of 1–500 mg HFPO-DA/kg-body weight per day (specific doses for different studies reported below). Doses were selected based on data from existing developmental toxicity studies on HFPO-DA in Sprague-Dawley rats. A published study by Caverly Rae et al. (2015) reported 1 mg/kg per day was a no observed adverse effect level (NOAEL) and 500 mg/kg per day was an upper dose that was tolerated in the rat. Further, an industry guideline prenatal developmental toxicity study by DuPont (2010) reported a NOAEL of 10 mg/kg per day and that 1,000 mg/kg per day was overtly toxic to the dam. The doses utilized in the present experiments were chosen to evaluate the reported NOAELs and allow for full dose–response assessment while avoiding overt maternal toxicity at highly elevated doses.

Animals

Time-mated Sprague-Dawley rats [Crl:CD(SD)], approximately 90 d of age, were purchased from Charles River Laboratories and shipped to the National Health and Environmental Effects Research Laboratory at the U.S. EPA in Research Triangle Park, North Carolina, on GD2 (GD0 = bred date; GD1 = plug positive date). Dams and their offspring were housed individually in clear polycarbonate cages (20 × 25 × 47 cm) with heat-treated, laboratory-grade pine shavings and fed NIH07 rodent diet and filtered (5 μm) municipal tap water *ad libitum*. Dams were weight-ranked and stratified then randomly assigned to treatment groups to produce similar mean weights and variances. This study was conducted in accordance with a protocol approved by the U.S. EPA National Health and Environmental Effects Research Laboratory's Institutional Animal Care and Use Committee. Animals were housed in a facility accredited by the Association for Assessment and Accreditation of Laboratory Animal Care and maintained at 20–22°C, 45–55% humidity, and a 12:12 h photoperiod (lights off at 1800 hours).

Evaluation of Fetal and Maternal Effects during Gestation

A total of three blocks of 15 dams per block were dosed once daily from GD14–18 with either water vehicle (control) or HFPO-DA to evaluate fetal and maternal effects (Figure 1A). The first block of dams was dosed with control, 62.5, 125, 250, or 500 mg/kg HFPO-DA ($n = 3$ dams for each). The second and third blocks of dams were dosed with control, 1, 3, 10, or 30 mg/kg HFPO-DA ($n = 3$ per dose per block). Total sample sizes were $n = 9$ for control, $n = 6$ for 1, 3, 10, 30 mg/kg, and $n = 3$ for 62.5, 125, 250, and 500 mg/kg HFPO-DA. In the first two blocks, spanning the entire dose range, we evaluated fetal testis testosterone production, fetal testis gene expression, fetal and maternal liver gene expression, fetal body weight, and maternal serum thyroid hormone and lipid concentrations. In the third block, encompassing the lower dose range utilized here, we collected fetal plasma for measuring HFPO-DA concentrations. Across all three blocks we evaluated maternal weight gain during dosing, reproductive output (number of fetuses and resorptions), maternal serum HFPO-DA concentration, and maternal liver weight at necropsy.

For the first two blocks, spanning the full dose range, late gestation (GD18) dams were euthanized by decapitation at ∼2 h after the final oral dose [∼0830–1000 hours Eastern Standard Time (EST)]. Trunk blood was collected and serum isolated via centrifugation (10,000 × *g* for 15 min at 4°C) in vacutainer tubes, transferred to 1.5-mL microcentrifuge tubes and stored at −80°C.

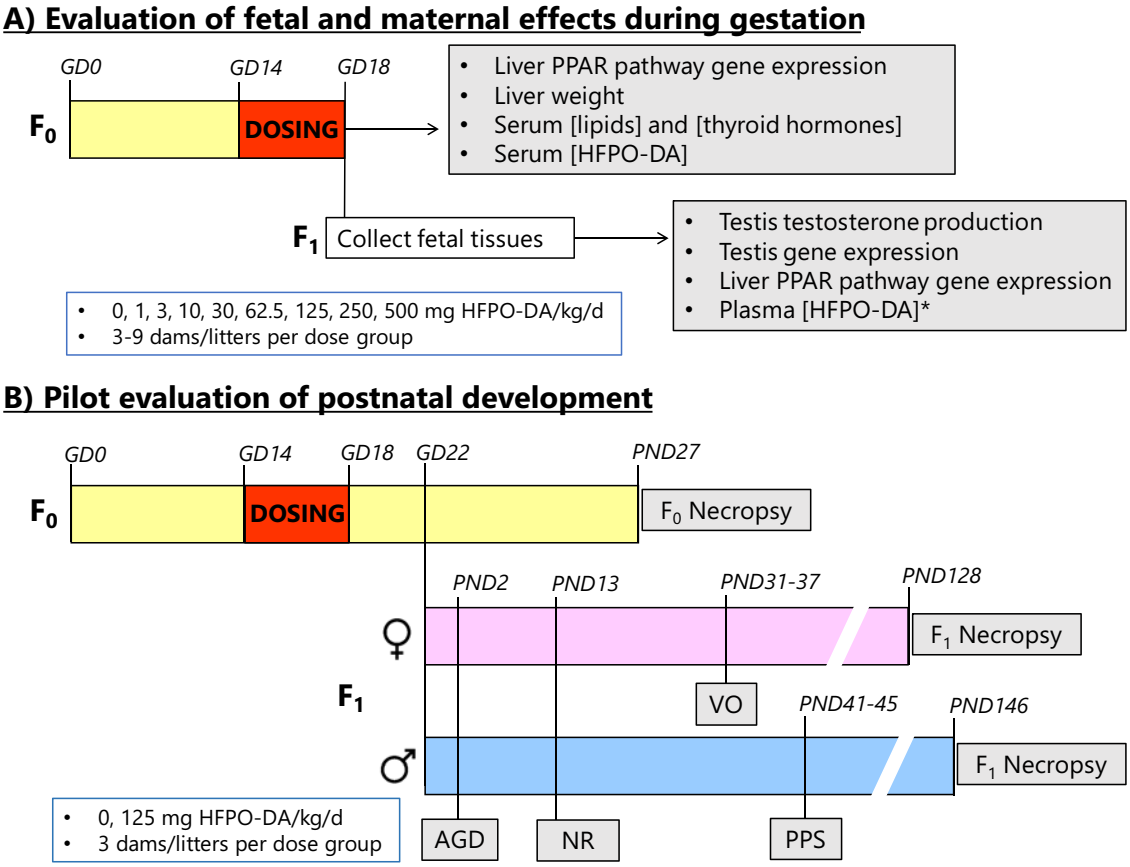

Figure 1. Schematic diagram of study designs for evaluating maternal, fetal, and postnatal effects of oral gestational hexafluoropropylene oxide dimer acid (HFPO-DA) exposure. Both (A) fetal and (B) postnatal study designs used oral gavage dosing from gestation day (GD) 14–18 at the indicated exposure levels. Fetal plasma HFPO-DA concentration (*) was only evaluated at doses of 0–30 mg/kg per day. AGD, anogenital distance; NR, nipple retention; PND, postnatal day; PPAR, peroxisome proliferator-activated receptor; PPS, preputial separation; VO, vaginal opening.

Dam liver weight was recorded and a sample of liver tissue was collected into a polypropylene microcentrifuge tube containing 500 µL TRIzol Reagent (Invitrogen) on ice. Fetuses were removed and two randomly selected fetuses per litter were weighed. Fetal testes were collected from all male pups with a single testis from the first three males used for determination of *ex vivo* testosterone production and the remaining testes were homogenized and preserved in TRIzol Reagent for gene expression analysis. The liver was collected from a single, randomly selected fetus per dam/litter for gene expression analysis and transferred to a polypropylene microcentrifuge tube containing 500 µL TRIzol Reagent (Invitrogen) on ice. Both dam and fetal liver samples were individually homogenized using a Bullet Blender (Next Advance) with 1-mm zirconium oxide beads, transferred to clean tubes, and stored at −80°C prior to RNA extraction (see below). *Ex vivo* fetal testis testosterone production was measured as previously reported (Wilson et al. 2004b) except the radioimmunoassay (RIA) utilized here was supplied by ALPCO (Catalog No. 72-TESTO-CT2, ALPCO). Briefly, one testis was isolated from each of three separate male fetuses in each litter and incubated in a humidified atmosphere at 37°C for 3 h in 500 µL of M-199 media (phenol red–free; Hazelton Biologics, Inc.) supplemented with 10% dextran-coated charcoal-stripped fetal bovine serum (Hyclone Laboratories) in 24-well plates under gentle agitation. After incubation, media were removed and stored in siliconized microcentrifuge tubes at −80°C until RIA analyses, which were performed according to manufacturer specifications.

Gene expression in fetal testes and fetal/maternal livers was assessed using reverse transcriptase real-time PCR of cDNA synthesized from RNA extracted from sample homogenates. RNA extraction was conducted according to TRIzol Reagent manufacturer specifications using chloroform and isopropanol. Following extraction, RNA was purified using the RNeasy Mini Kit (Catalog No. 74104; Qiagen). RNA concentration and purity (260:280 ratio ≥1.8) were determined with a NanoDrop 2000 spectrophotometer (Thermo Scientific). For the fetal testes, a 96-well gene array plate was previously custom designed to contain 89 target genes and 3 housekeeping genes, an intra-assay control, a genomic DNA control, a reverse transcriptase control, and a positive PCR control [see Table S1; SABioscience; (Hannas et al. 2012)]. For the fetal and maternal livers, we utilized the RT^2 Profiler PCR Array for Rat PPAR Targets by Qiagen (Catalog No. 330231 PARN-149Z), which contains 84 target genes relevant to PPARα, -β/δ, and -γ signaling pathways and 5 potential housekeeping genes (see Table S2). PCR reactions were run using RT2 SYBR Green quantitative PCR (qPCR) Master Mix (SABioscience) on an iCycler iQ Real-Time Detection System (Bio-Rad) for fetal testes and on a CFX96 Touch Real-Time Detection System (Bio-Rad) for maternal and fetal livers.

For the third block, dosed with the lower dose range (1–30 mg/kg HFPO-DA), late gestation (GD18) dams were euthanized by decapitation ∼2 h after the final dose, liver weight was recorded, and trunk blood was collected for serum isolation. Serum was isolated from trunk blood via centrifugation (10,000 × *g*; 15 min; 4°C) using Becton Dickinson vacutainer tubes and stored in 1.5-mL siliconized microcentrifuge tubes at −80°C for future analyses. Fetuses were removed and fetal blood was collected from the jugular vein from all fetuses within a litter

using heparinized glass capillary tubes. Blood was expelled from capillary tubes using fine-tip disposable transfer pipets into a microcentrifuge tube forming a single composite sample per litter. Fetal blood was then centrifuged at $10{,}000 \times g$ for 15 min at 4°C and plasma was transferred to clean tubes and frozen at −80°C.

Maternal sera from all three blocks and fetal plasma from the third block were analyzed for HFPO-DA concentrations similar to previously reported methods (McCord et al. 2018; Reiner et al. 2009; Rushing et al. 2017). Serum or plasma samples (25 μL) were denatured using 0.1 M formic acid (FA) followed by a cold (−20°C) acetonitrile (ACN) protein crash. The volumes of FA and ACN varied based on the anticipated concentrations of HFPO-DA in the sample (0–100 ng HFPO-DA/mL = 100 μL FA + 0.5 mL ACN; 100–5,000 ng HFPO-DA/mL = 100 μL FA + 1.0 mL ACN; 5,000–200,000 ng HFPO-DA/mL = 1.0 mL FA added, then 100-μL subsamples removed and crashed with 900 μL cold ACN). Samples were vortex mixed after FA and ACN additions then centrifuged at $10{,}000 \times g$ for 5 min and the supernatant removed. Sample extracts were separated using a Waters ACQUITY ultra performance liquid chromatograph (UPLC) (Waters Corporation) fitted with a Waters ACQUITY UPLC BEH C18 column (2.1 mm × 50 mm; 1.7 μm; 130 Å). Detection was performed using a Waters Quattro Premier XE tandem quadrupole mass spectrometer in negative ionization mode. A stable isotope of HFPO-DA ($^{13}C_3$, Wellington Laboratories) was used as an internal standard for quantitation. Separate calibration curves were prepared for the ranges 0–100 ng/mL, 100–5,000 ng/mL, and 5,000–200,000 ng/mL to account for expected concentration differences between control, offspring (fetus/pup), and dam concentrations across the dose range tested.

Maternal serum samples from the first two blocks were analyzed for thyroid hormones and a standard lipid panel. Total triiodothyronine (T_3) and thyroxine (T_4) were quantified by radioimmunoassay (RIA) according to manufacturer specifications (IVD Technologies). Thyroid hormone samples were run in duplicate (mean intra-assay coefficient of variation 15.5% for T_3, 11.5% for T_4), and two calibration standards were run as unknowns with observed concentrations varying from expected by <15% for T_3 and <20% for T_4. Thyroid hormone RIA values were considered below detection when specific binding (B/B_0) was ≥90% (0.2 ng/mL for T_3 and 2 ng/mL for T_4) (Sui and Gilbert 2003). Serum total cholesterol, high-density lipoproteins (HDL), low-density lipoproteins (LDL), and triglycerides were quantified using a Beckman Coulter AU480 clinical chemistry analyzer (Beckman Coulter, Inc.) as per manufacturer's protocol. All reagents were obtained from the instrument manufacturer except for the LDL assay, which was obtained from Diazyme Laboratories.

Pilot Evaluation of Postnatal Development

A single-dose level pilot study utilizing time-mated SD rats was conducted to examine the potential postnatal effects of *in utero* exposure to HFPO-DA from a similar dosing interval to the fetal studies (Figure 1B). The study consisted of dams exposed to oral daily dosing with either water vehicle or 125 mg/kg HFPO-DA ($n = 3$ for each) from GD14–18. This dose was selected because it was the highest dose level that did not significantly reduce maternal weight gain during dosing from the fetal evaluation studies. Dams gave birth naturally beginning on the morning of GD22 [i.e., postnatal day (PND) 0]. On PND2 all pups were sexed, weighed, and anogenital distance (AGD) was measured using a Leica MZ6 stereomicroscope (Leica Microsystems) fitted with an ocular micrometer. On PND13, the offspring were sexed, weighed, and evaluated for retention of female-like nipples/areolae. On PND27, the dams were euthanized, uterine implantation sites were scored, pups were weaned to two animals per cage by sex and treatment group, and food was changed to NTP2000 rodent diet. Beginning on PND31 for female offspring and PND41 for male offspring, individuals were evaluated daily for markers of pubertal onset, vaginal opening (VO) for females and balano-preputial separation (BPS) for males.

Beginning at PND128, adult F1 females were weighed, euthanized via decapitation, and examined via necropsy for any reproductive tract malformations and tissue weights were collected for uterus, paired ovaries, liver, paired kidneys, and visceral adipose tissue. Similarly, beginning at PND146 adult F1 males were weighed, euthanized, and examined for reproductive tract malformations and weights were collected for all relevant reproductive tissues. Male necropsy included weights of glans penis, ventral prostate, paired seminal vesicles, paired testes, paired epididymides, levator ani–bulbocavernosus (LABC), paired bulbourethral (Cowper's) glands, paired kidneys, visceral adipose tissue, and epididymal adipose tissue. After weighing, the left epididymis was separated into two sections, the cauda and the corpus plus caput, and individually minced in M-199 media. Total sperm counts in epididymal sections were measured using a Multisizer 3 Coulter counter (Beckman Coulter).

In Vitro *Transcriptional Activation Assays*

HFPO-DA was assessed for agonism and antagonism of transcriptional activation for estrogen (ER), androgen (AR), and glucocorticoid receptors (GR). Method details for *in vitro* transactivation assays for ER (Wilson et al. 2004a), AR (Hartig et al. 2002, 2007), and GR (Conley et al. 2017; Medlock Kakaley et al. 2018) have been previously reported. Briefly, for ER activity we utilized the stably transfected T47D-KBluc cell line [publicly available via American Type Culture Collection (ATCC); CRL-2865] according to protocols provided by ATCC with the modification of Dulbecco's Modified Eagle Media (DMEM) as the cell culture media instead of Roswell Park Memorial Institute (RPMI) media. We utilized adenoviral transduction to introduce chimp AR (Ad5chAR-g) (Hartig et al. 2007) or human GR (Ad/GR4) (Shih et al. 1991) and a luciferase-based promoter-reporter construct (MMTV-Luc; Ad/mLuc7) (Shih et al. 1991) into CV-1 cells (ATCC CCL-70) to assess GR and AR activity, respectively. For viral transduction, cells were grown to confluence in 60-mm Petri dishes in 10% dextran-coated charcoal-treated fetal bovine serum RPMI-1640 growth media. Confluent cells were split at a ratio of 1:3 into 60-mm dishes and inoculated on day 7 ($\sim 5 \times 10^6$ cells/dish) with adenoviral vectors at multiplicities of infection of 1 receptor to 50 reporter constructs. After 24 h incubation with adenoviral vectors, cells were rinsed, resuspended in media, and seeded into assay plates. All assays were run in 96-well plates and luminescence was detected using a BMG Fluostar Omega luminometer (BMG Labtech) following 24-h exposure. HFPO-DA was tested for receptor agonism and antagonism at 10-fold concentration intervals from 100 pM to 10 μM (ER) or 100 pM to 100 μM (AR and GR). For ER activity, the reference agonist was 17β-estradiol [(E2) CAS: 50-28-2] and the reference antagonist was ICI-182780 (CAS: 129453-61-8). When assessing ER antagonism, HFPO-DA was competed against 10 pM E2. For AR activity the reference agonist was dihydrotestosterone [(DHT) CAS: 521-18-6] and the reference antagonist was hydroxyflutamide (CAS: 52806-53-8). When assessing AR antagonism, HFPO-DA was competed against 100 pM DHT. For GR activity, the reference agonist was dexamethasone [(Dex) CAS: 50-02-2] and the reference antagonist was mifepristone (CAS: 84,371-65-3). When assessing GR antagonism, HFPO-DA was competed against 1 nM Dex. Cellular cytotoxicity across the dosing range was determined for CV-1 cells utilizing the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) dye

(Mosmann 1983). HFPO-DA was analyzed using $n = 2$–3 biological replicate assay plates (i.e., unique cell passages) with four technical replicates per treatment per plate.

Data Analyses

All values are reported as mean ± standard error (SE) and all statistical comparisons were conducted at $\alpha = 0.05$ significance level except for PPAR pathway gene expression, which utilized $\alpha = 0.0001$ to detect highly significant analysis of variance (ANOVA) results and $\alpha = 0.01$ to determine pairwise differences of treatment as compared with controls for significant genes. Treatment effects as compared with control were identified using ANOVA in SAS (version 9.4; SAS Institute). Fetal and postnatal data were analyzed using PROC MIXED to correct for the nested effects of individuals within litters (fetus/pup data nested within litter, litter as random variable); dam data were analyzed using PROC GLM. Pairwise comparison of significant ANOVA results was performed using the least squares means (LSMEANS) procedure in SAS. GraphPad Prism (version 7.02; GraphPad, Inc.) was used to generate all figures and to conduct dose–response curve analyses.

Fetal testis and maternal/fetal liver gene expression data were analyzed using the comparative cycle threshold (C_T) method. Briefly, delta C_T values were calculated using the equation $2^{-\Delta\Delta C_T}$ and normalized to the mean C_T value of the appropriate housekeeping genes. We selected housekeeping genes for each tissue and gene array that did not display a significant (ANOVA $p > 0.01$) treatment effect of HFPO-DA exposure (fetal liver = *Actb*, *B2m*; maternal liver = *Actb*, *Hprt1*, *Rplp1*; and fetal testis = *Actb*, *Gusb*, *Ldha*). Delta C_T values were then converted to fold-induction by dividing the treated replicate delta C_T by the mean delta C_T of the control replicates for each gene. Fold-induction values were then then $\log_{10}$-transformed prior to ANOVA.

Fetal testis testosterone production was normalized to the mean control concentration within a given block and analyzed as percentage of control values across blocks. Maternal liver weight was analyzed using body weight as a covariate within PROC GLM followed by pairwise comparison using LSMEANS, this analysis produces linear regressions of body weight versus liver weight for each dose group. Mean female AGD was subtracted from individual male AGD measures to calculate percentage reduction as compared with control.

Serum HFPO-DA concentrations in the mother and the fetus were analyzed as a function of oral dose administered to the mother. We utilized nonlinear regression (exponential one-phase association) to describe the increase and saturation of serum HFPO-DA concentrations across the full oral dose range (1–500 mg/kg) for maternal serum. Fetal plasma HFPO-DA concentrations were only analyzed in the low-dose range (1–30 mg/kg), which was better described using a linear uptake model. We compared the slopes of the low-dose linear regressions for maternal serum and fetal plasma HFPO-DA concentrations using GraphPad Prism.

Dose–response analyses for the *in vitro* transactivation assay data and the most sensitive *in vivo* end points and were conducted using four-parameter logistic regression in GraphPad Prism (constraint to bottom = 0%, top = 100%). *In vitro* luminescence data was normalized to background (vehicle control), $\log_{10}$ transformed, and converted to percentage maximum response based on saturating levels of reference agonist. *In vivo* data were modeled as a function of $\log_{10}$-transformed internal dose (i.e., dam serum HFPO-DA concentration from GD18), and response data

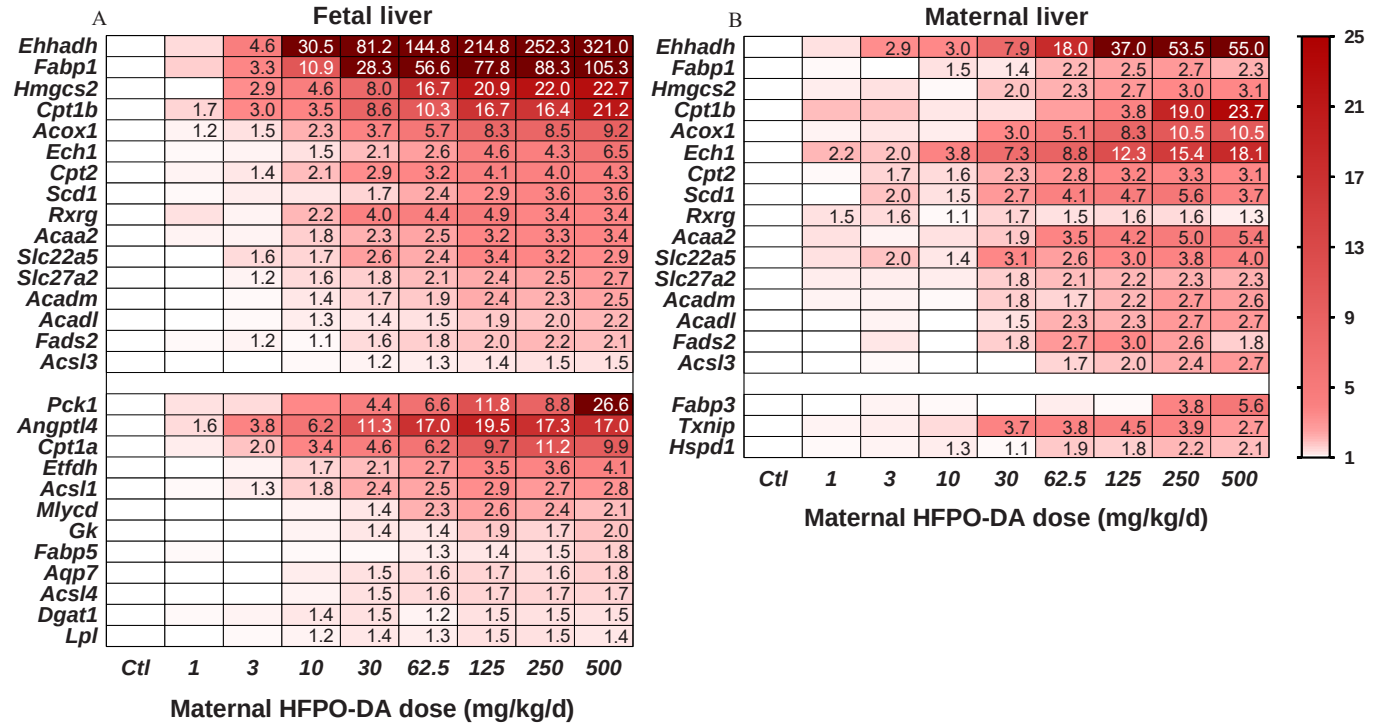

Figure 2. Expression of significantly up-regulated genes (ANOVA, $p < 0.0001$) from peroxisome proliferator-activated receptor (PPAR) signaling pathway gene arrays in (A) fetal ($n = 6$ for control, $n = 3$ for treated) and (B) maternal ($n = 5$ for control, $n = 3$ for treated) livers following gestation day (GD) 14–18 oral maternal exposure to hexafluoropropylene oxide dimer acid (HFPO-DA). Upper portions (above break) display significantly altered genes common to both fetal and maternal livers, lower portions display genes differentially altered between fetal and maternal livers. Cell values represent significant ($p < 0.01$) dose-level fold-induction values relative to control livers [cells with no value were not significantly different from control (see Table S2 for gene descriptions, and Tables S3 and S6 for complete gene expression data)]. Legend indicates fold-induction compared with control with darker shaded genes more highly expressed. Genes with fold-induction >25-fold of control were beyond the scale of the legend. Ctl, control.

was normalized to control and presented as a percentage. We estimated effect concentrations equivalent to a 5% deviation from control (EC_5). Reduction in maternal serum T_3 concentration was modeled by ascribing a concentration of one-half of the detection limit (i.e., 0.1 ng/mL; detection limit of 0.2 ng/mL) for the dose groups that were below the detection limit.

Maternal rat serum concentrations were compared with human plasma concentrations from workers in a HFPO-DA manufacturing facility in Dordrecht, Netherlands (DuPont 2017). Human plasma samples represented workers who volunteered to participate in the study with the goal of determining whether there were measurable quantities of HFPO-DA in their blood. Some of the workers were in areas with potential for exposure and others were not (17/24 participants had detectable HFPO-DA levels). Comparisons were made in order to determine how the doses used in the current study relate to likely "worst case" human concentrations based on internal exposure levels rather than comparing exposures across species based upon estimated external dose levels. We calculated the margin of internal exposure (MOIE) as a ratio of maternal rat serum concentration to human plasma concentration for each of the 17 workers with detectable levels (Bessems et al. 2017). MOIEs were calculated using the mean maternal rat serum HFPO-DA concentration from the 1- and 125-mg/kg dose levels because these represented the lowest oral dose administered and the administered oral dose for the pilot postnatal study.

Results

Fetal Effects from GD14–18 Dosing

Fetal livers from HFPO-DA–exposed litters displayed highly significant (ANOVA $p < 0.0001$), dose–responsive up-regulation of 28 different genes in the PPAR signaling pathway arrays (Figure 2A; see also Table S3). Most affected genes were associated with fatty acid metabolism (*Acaa2, Acadl, Acadm, Acox1, Acsl1, Acsl3, Acsl4, Cpt1a, Cpt1b, Cpt2, Ehhadh, Etfdh, Fads2, Fabp1, Gk, Hmgcs2, Mlycd*, and *Scd1*). Remaining up-regulated genes were associated with lipid transport (*Angptl4, Dgat1, Lpl*), adipogenesis (*Ech1, Lpl*), water transport (*Aqp7*), insulin signaling (*Cpt1a, Dgat1, Pck1*), PPAR transcription factors (*Rxrg*), or PPAR ligand transporters (*Fabp1, Fabp5, Slc22a5, Slc27a2*). The most highly up-regulated genes included *Ehhadh* (321-fold), *Fabp1* (105-fold), *Pck1* (27-fold), *Hmgcs2* (23-fold), *Cpt1b* (21-fold), and *Angptl4* (17-fold). Several genes were significantly ($p < 0.01$) up-regulated even at the lowest dose level tested (1 mg/kg) including *Cpt1b, Angptl4*, and *Acox1*.

In contrast to the observed changes in fetal PPAR liver genes, the results for the expression of genes from our custom array for detecting phthalate-like effects in the fetal testis were not significantly different from controls (see Table S4). Further, fetal testis testosterone production was not significantly different from controls at any dose (see Figure S1, Table S5).

Maternal Effects from GD14–18 Dosing

Similar to fetal livers, maternal livers displayed highly up-regulated expression of PPAR signaling pathway–associated genes (Figure 2B; see also Table S6). Overall, the maternal and fetal livers shared up-regulation of 16 genes. The majority of shared, up-regulated genes were associated with fatty acid metabolism (*Acaa2, Acadl, Acadm, Acox1, Acsl3, Cpt1b, Cpt2, Ehhadh, Fads2, Fabp1, Hmgcs2*, and *Scd1*). Also similar to the fetal liver, the remaining up-regulated maternal genes were associated with adipogenesis (*Ech1*), PPAR transcription factors (*Rxrg*), or PPAR ligand transporters (*Slc22a5, Slc27a2*). In contrast to the fetal liver, the maternal livers of treated rats did not differ significantly from controls in the expression of *Acsl1, Acsl4, Angptl4, Aqp7, Cpt1a, Dgat1, Etfdh, Fabp5, Gk, Lpl, Mlycd*, or *Pck1*; whereas 2 genes associated with cell proliferation (*Hspd1*, *Txnip*) and 1 with fatty acid metabolism (*Fabp3*) were significantly up-regulated in the maternal liver but not the fetal liver. Further, the maternal and fetal livers shared the most highly up-regulated gene (*Ehhadh*; 55-fold in maternal liver) and both had highly up-regulated *Cpt1b* expression (24-fold in maternal liver). Only 1 of the shared genes was noticeably more highly up-regulated in the maternal liver than the fetal liver (*Ech1*; 18-fold vs. 6-fold in maternal and fetal livers, respectively). Overall, the PPAR signaling pathway was up-regulated in both maternal and fetal livers, with both sharing many of the same up-regulated genes; however, the overall profiles of induction were noticeably different between the two life stages, with the fetal liver seemingly displaying greater sensitivity both in terms of the number of genes affected and the degree of up-regulation.

During the GD14–18 dosing window, dams had significantly less body weight gain at the 250- and 500-mg/kg dose levels compared with controls (ANOVA $p = 0.0037$; Figure 3A; see also Table S5). On GD18, dams had significantly higher liver weights in the 62.5-to 500-mg/kg dose groups than controls (ANOVA $p < 0.0001$; Figure 3B; see also Table S5). There were no significant differences in numbers of live pups, resorptions, or fetal body weight compared with controls (see Table S5).

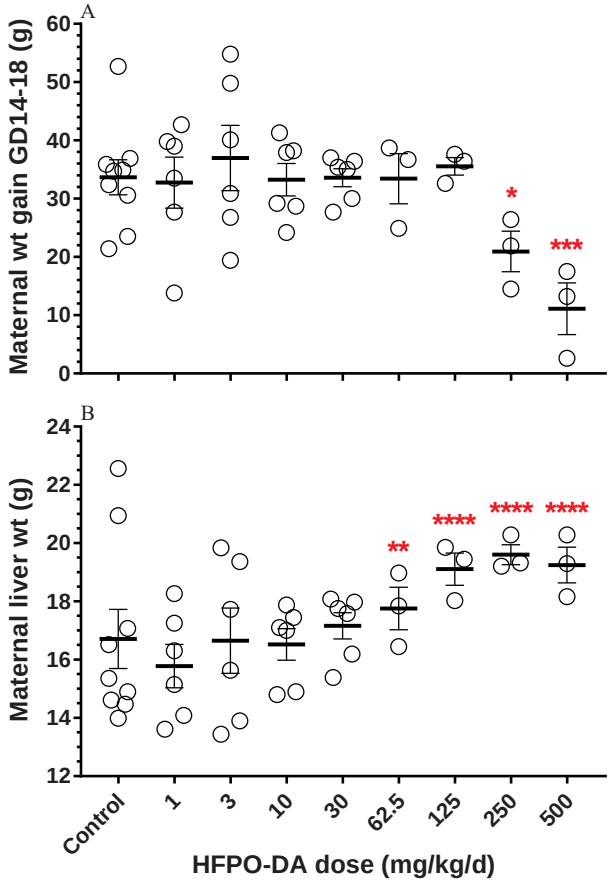

Figure 3. (A) Maternal body weight gain during gestation day (GD)14–18 dosing period and (B) maternal liver weight on GD18. Data points represent individual replicates (control, $n = 9$; 1–30 mg/kg, $n = 6$; 62.5–500 mg/kg, $n = 3$), bars and whiskers represent mean ± standard error, and asterisks represent significant differences compared with control values (*, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$; ****, $p < 0.0001$). Statistical significance was determined using analysis of variance; for liver weight analysis, body weight was included as a covariate.

Maternal serum samples displayed dose–responsive decreases in all measures of thyroid hormones and lipids (Figure 4; see also Table S5). Serum triglycerides were significantly lower at 500 mg/kg, cholesterol and HDL were significantly lower at 250 and 500 mg/kg, and total T_4 and LDL were significantly lower at ≥125 mg/kg. The most sensitive end point was serum total T_3, which was significantly lower at ≥30 mg/kg and below assay detection levels (i.e., <0.2 ng/mL) in the top two dose levels.

Postnatal Effects from GD14–18 Dosing

In the HFPO-DA pilot postnatal study that utilized GD14–18 dosing, one of three control dams was not pregnant, reducing the sample size to $n = 2$ litters. Control dams and dams dosed with 125 mg/kg HFPO-DA gave birth to litters with equal numbers of viable pups. On a litter means basis, there were no significant differences for any end point measured through the onset of puberty (see Table S7). On an individual pup basis (as opposed to litter means), female offspring body weight was significantly lower than controls at multiple time points (PND2, PND27, and at VO), indicating a potential trend in growth deficit to investigate in future studies.

Adult males at necropsy had significantly lower tissue weight of the right epididymis on a litter means basis, but no other tissues were affected as compared with controls (see Table S8). On an individual basis, treated male rats had significantly lower tissue weights of the right testis, left testis, paired testes, right

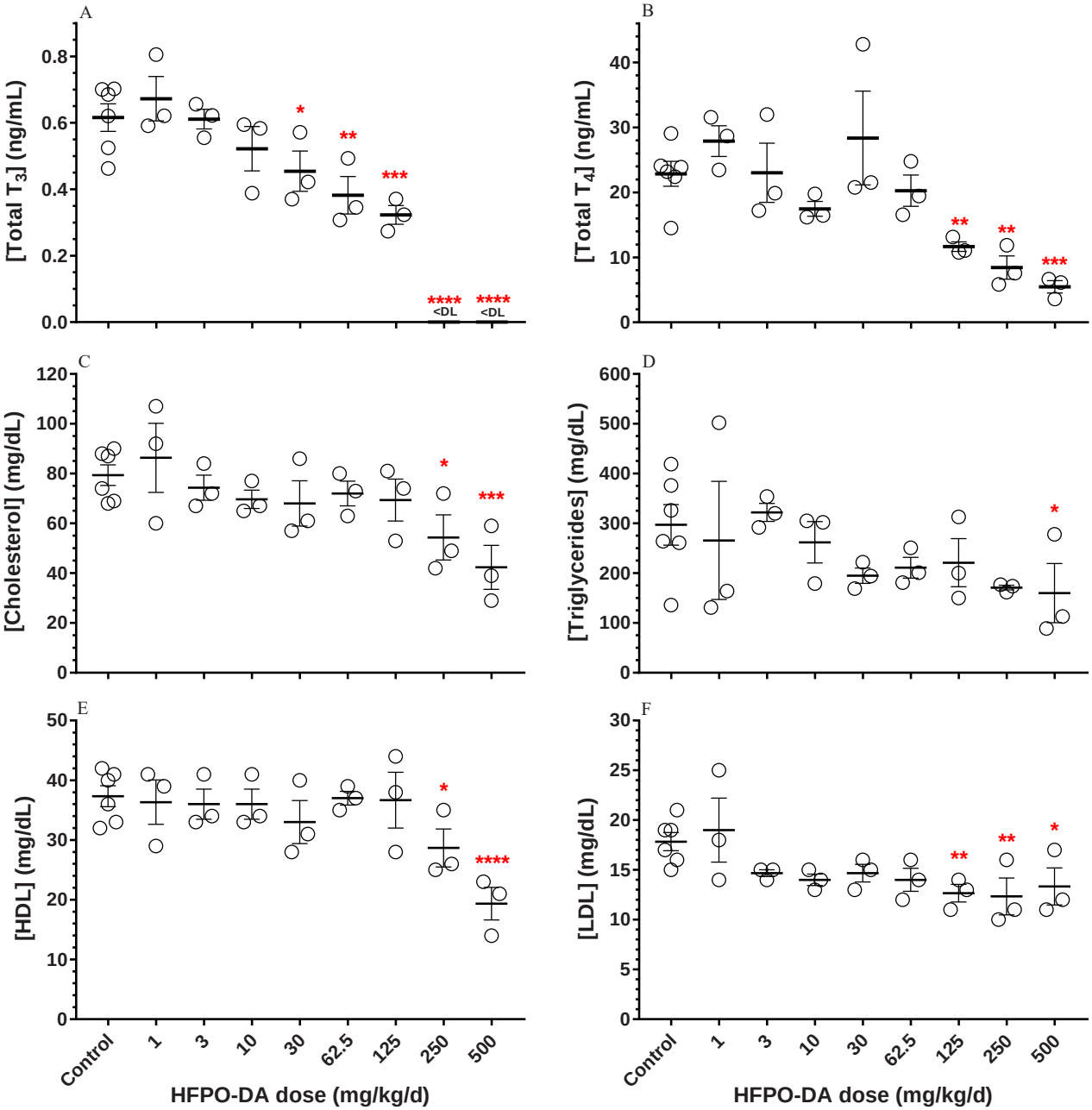

Figure 4. Concentrations of (A) total triiodothyronine (T_3), (B) total thyroxine (T_4), and lipids [(C) cholesterol, (D) triglycerides, (E) high-density lipoproteins (HDL), and (F) low-density lipoproteins (LDL)] in maternal serum following oral hexafluoropropylene oxide dimer acid (HFPO-DA) dosing from gestation days (GD) 14–18. Dam serum was collected on GD18 approximately 2 h after final oral dose. Data points represent individual replicates (control, $n = 6$; treated, $n = 3$), bars and whiskers represent mean ± standard error, and asterisks represent significant differences compared with control values using analysis of variance (*, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$; ****, $p < 0.0001$). <DL, values below radioimmunoassay detection limit.

epididymis, left epididymis, paired epididymides, and epididymal adipose tissue as compared with controls.

Adult females at necropsy displayed no significant differences in any end point as compared with controls on a litter means basis (see Table S9). On an individual basis, treated female rats had significantly smaller AGD and lower liver weight as compared with controls.

HFPO-DA Concentrations in Maternal Serum and Fetal Plasma

Maternal serum and fetal plasma contained increasing concentrations of HFPO-DA as a function of oral dose following dosing during the GD14–18 experimental window (Figure 5; see also Table S10). Over the full maternal dose range (1–500 mg/kg), uptake appeared to saturate at the higher dose levels and was modeled using exponential one-phase association ($R^2 = 0.84$) with a plateau of 112 ± 15 μg/mL (Figure 5A). In the lower dose range (1–30 mg/kg), increases in maternal serum and fetal plasma HFPO-DA concentrations were linear (Figure 5B); however, the maternal slope was significantly greater than the fetal slope with maternal serum HFPO-DA increasing 0.46 μg/mL and fetal plasma HFPO-DA concentration increasing 0.12 μg/mL for each 1-mg/kg increase in oral maternal dose ($p < 0.0001$).

Dose–Response Analyses

Using maternal serum HFPO-DA concentrations, we estimated effect concentrations for an EC_5 for the most sensitive end points: maternal liver weight, maternal liver gene expression, and maternal serum [T_3] and [T_4] (Figure 6). Maternal [T_3] was the most sensitive end point with an EC_5 of 3.8 μg/mL (estimated maternal oral dose of 8.2 mg/kg using the linear equation from Figure 5) followed by liver *Ehhadh* expression ($EC_5 = 14.1$ μg/mL), liver weight ($EC_5 = 17.6$ μg/mL), and [T_4] ($EC_5 = 17.8$ μg/mL).

Comparison of Maternal Rat and Human Internal Exposure Levels

The human worker HFPO-DA plasma concentrations reported by Dupont (2017) ranged from 0.001–0.169 μg/mL, whereas the mean maternal rat serum concentrations reported here ranged from 0.68–100.7 μg/mL following a 5-d exposure. At the lowest dose level tested here (1 mg/kg), the rat:human MOIEs ranged from 4 to 566 (14/17 MOIEs were >100; Figure 7A). Further, at the dose utilized in the postnatal pilot study (125 mg/kg), the rat:human MOIEs ranged from 272 to 38,333 (15/17 MOIEs were >1,000 and 12/17 MOIEs were >10,000; Figure 7B). It is important to note that the maternal rat serum concentrations utilized in this comparison were from short-term (5-d) exposures, whereas the human plasma concentrations were from individuals working in an HFPO-DA manufacturing facility and likely represent chronic exposure levels, but it is unknown whether these concentrations represent a steady state.

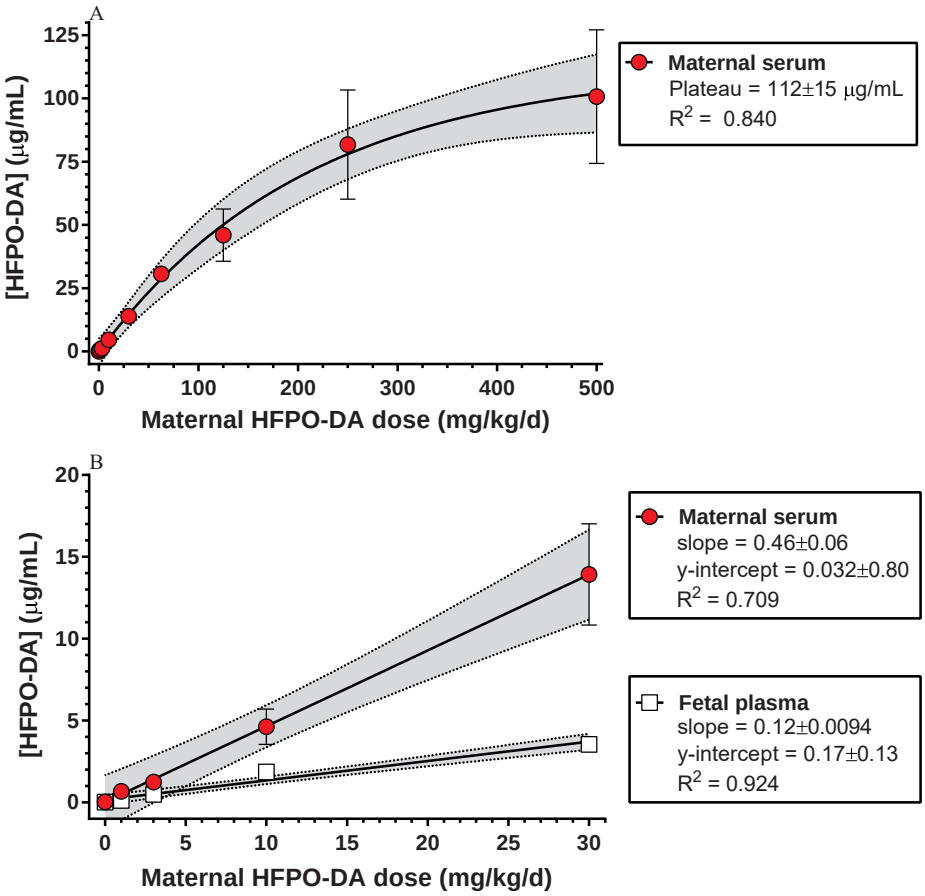

Figure 5. Maternal serum and fetal plasma hexafluoropropylene oxide dimer acid (HFPO-DA) concentrations (mean ± standard error, $n = 3$–9; see Table S10) as a function of oral dose following maternal exposure from gestation day (GD) 14–18. Samples were collected on GD18 approximately 2 h after final oral dose. (A) Full maternal dose range modeled using exponential one-phase association and (B) low dose range modeled using linear regression (95% confidence intervals shaded). Fetal plasma was collected only from the low dose range (1–30 mg/kg per day).

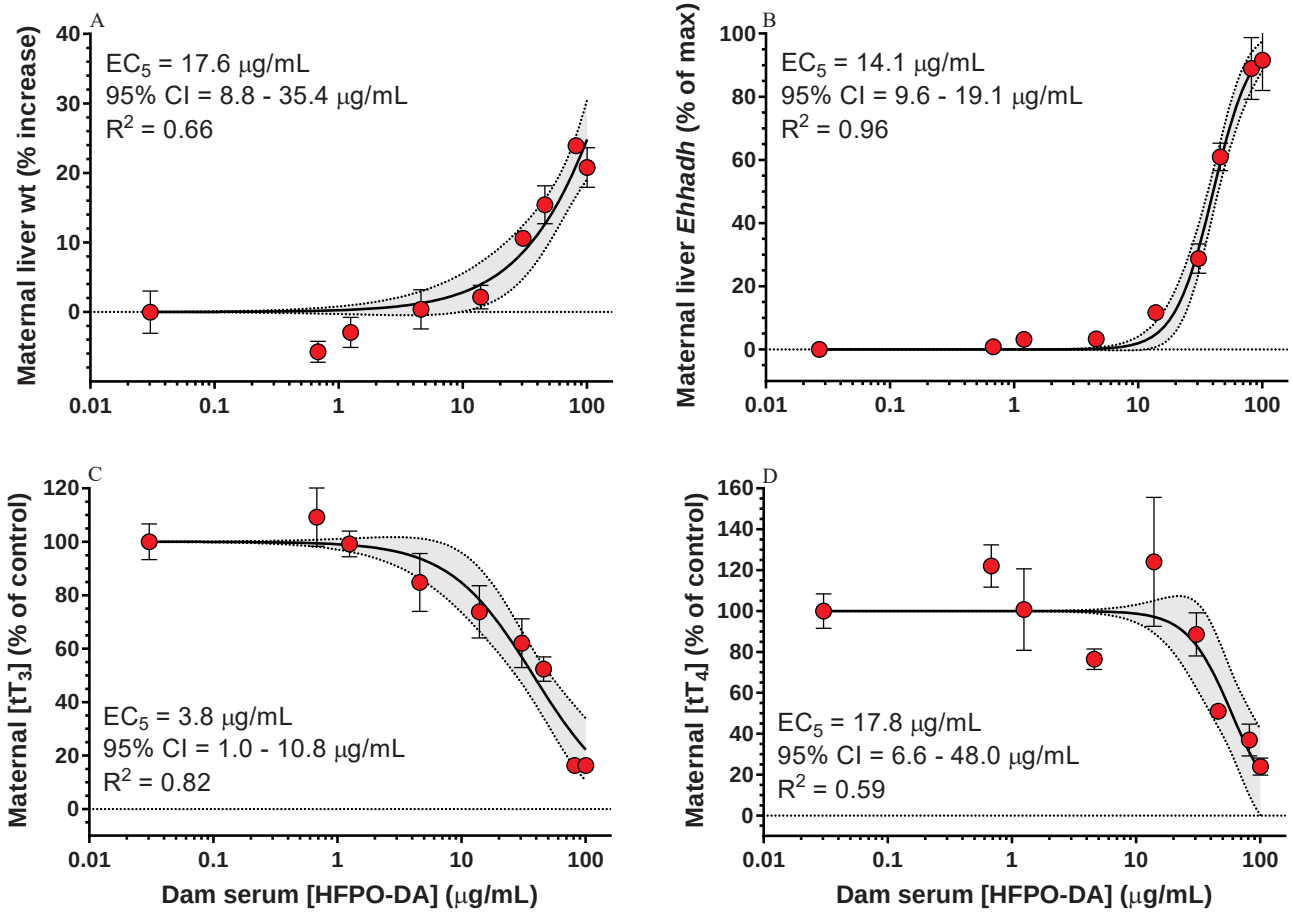

Figure 6. Dose–response curves (four-parameter logistic regression) and 5% effect estimates [EC_5 with 95% confidence intervals (CIs)] for the most sensitive end points [(A) maternal liver weight, (B) maternal liver *Ehhadh* gene expression, (C) maternal serum total triiodothyronine (tT_3), and (D) total thyroxine (tT_4)] as a function of maternal serum hexafluoropropylene oxide dimer acid (HFPO-DA) concentration. Dam serum HFPO-DA concentrations represent those measured on gestation day (GD)18 following GD14–18 dosing. Data points represent mean ± standard error, (A) control $n = 9$, 1–30 mg/kg per day $n = 6$, 62.5–500 mg/kg per day $n = 3$; (B–D) control, $n = 6$; treated, $n = 3$.

In Vitro *Nuclear Receptor Transactivation*

HFPO-DA did not display any estrogenic activity (agonism or antagonism) at concentrations ranging from 100 pM to 10 µM (see Figure S2). Further, there was no androgen or glucocorticoid receptor agonism at concentrations ranging from 100 pM to 100 µM. At the very highest dose tested (100 µM), which approached the cytotoxic dose of 300 µM, HFPO-DA exposure did result in a slight glucocorticoid receptor antagonism ($28 \pm 3\%$ reduction in luciferase expression) and a moderate androgen receptor antagonism ($42 \pm 1\%$ reduction).

Discussion

The range of adverse effects resulting from oral maternal HFPO-DA exposure reported here are consistent with limited data available for HFPO-DA (Caverly Rae et al. 2015; DuPont 2008a, 2010; Gannon et al. 2016; Rushing et al. 2017; Wang et al. 2017a) and the extensive toxicity literature available for other PFAS, notably PFOS and PFOA [reviewed by ATSDR (2018), ECHA (2014), OECD (2002) and U.S. EPA (2016a)]. We observed up-regulation of genes associated with PPAR signaling pathways, maternal hepatomegaly, reductions in maternal serum lipids and thyroid hormones, and indications of reduced body and tissue weights in F1 animals. All of these effects have been observed following maternal exposure to PFOS/PFOA in laboratory animals and several have been previously observed for HFPO-DA. However, despite extensive PPAR pathway up-regulation, HFPO-DA did not produce any effects that are hallmarks of phthalate syndrome, including reduced fetal testis testosterone production, phthalate-specific fetal testis gene expression changes, reduced AGD on PND2, or male reproductive malformations. This lends support to the hypothesis that the effects of phthalates on male reproductive development are not mediated via the PPAR pathway.

The specific dosing interval utilized in developmental toxicity studies with PFAS is a critical factor for the types of effects that have been described. Grasty et al. (2003) reported significantly increased neonatal mortality and reduced pup weight in Sprague-Dawley rats following gestational PFOS exposure at 25 mg/kg across a range of 4-d dosing windows. These effects increased in severity as the dosing window moved later in gestation. Further, it was demonstrated that dosing only on GD19–20 was sufficient to produce these effects. Subsequent studies that included dosing during the full gestational period also reported pup mortality and reduced pup body weight. Lau et al. (2003) examined PFOS exposure in the rat and reported significantly increased neonatal mortality shortly after birth (<24 h) at ≥3 mg/kg. Separate studies in Sprague-Dawley rats confirmed the neonatal mortality following gestational exposure to PFOS at ≥1.6 mg/kg (Luebker et al. 2005a, 2005b). Similar results have been reported with other PFAS, primarily PFOA, and in other species, including mice and cynomolgus monkeys [reviewed by Abbott (2015) and Lau et al. (2007)]. In the pilot postnatal study presented here,

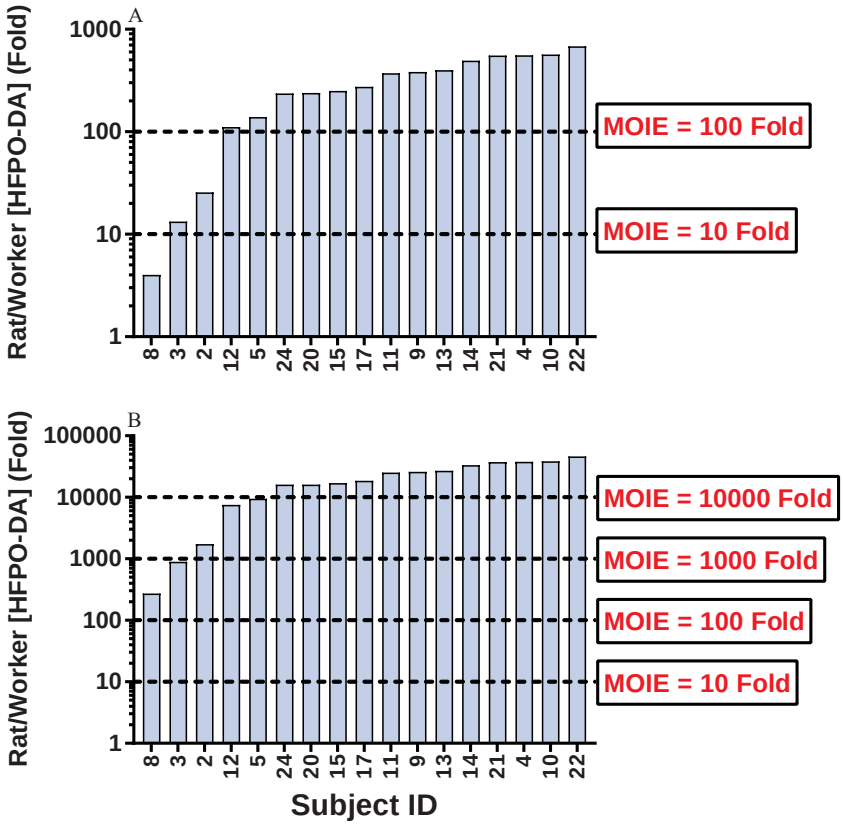

Figure 7. Comparison of mean maternal Sprague-Dawley rat serum hexafluoropropylene oxide dimer acid (HFPO-DA) concentration from (A) 1- and (B) 125-mg/kg per day exposure groups and individual human plasma HFPO-DA concentrations from workers in an HFPO-DA manufacturing facility in the Netherlands (DuPont 2017). Horizontal lines indicate various margins of internal exposure (MOIE) levels as compared with individual worker plasma concentrations.

there was an indication of decreased female pup weight but no effect on pup survival following HFPO-DA exposure from GD14–18 at a relatively high dose (125 mg/kg). However, expanding the dosing timeline to include the entire period of fetal development (i.e., GD8 through parturition) appears to reduce neonatal survival and body weight similar to PFOS exposure but at ~20-fold higher oral maternal doses [J.M. Conley and L.E. Gray (personal communication)].

As mentioned above, female pup body weight in the HFPO-DA dose group was significantly lower, on an individual analysis basis, 2 d after birth compared with control animals. Previous studies with laboratory rats have reported stunted growth of surviving pups following PFOS exposure. Lau et al. (2003) reported that pups exposed *in utero* to PFOS at ≥2 mg/kg displayed lower body weights, and Luebker et al. (2005b) reported the same response in all dose levels tested (i.e., ≥0.4 mg/kg). Overall, reduced pup weight appears to be one of the most sensitive end points in *in utero* PFAS studies. This effect aligns with multiple epidemiological studies, indicating a negative association between human birth weight and concentrations of PFOS/PFOA [reviewed by Bach et al. (2015) and Negri et al. (2017)] and should be more extensively evaluated for HFPO-DA exposure.

PFAS are known to primarily activate PPARα, particularly in the mammalian liver, however other receptors, such as PPARγ, have also been shown to be activated (Vanden Heuvel et al. 2006). Although the biological significance of induction of PPAR pathway gene expression is not known, it was overall the most sensitive end point in the present studies. Even at the lowest dose tested (1 mg/kg), the fetal liver displayed multiple significantly up-regulated genes (*Cpt1b, Acox1, Angptl4*). Bjork et al. (2008) performed a similar experiment with gestational PFOS exposure in the SD rat (exposed to 3 mg/kg from GD2 to GD20) and identified 445 genes via microarray that were significantly altered in the fetal liver. Four genes associated with fatty acid metabolism were individually verified using qPCR, 3 of which were also identified as significantly up-regulated in the present study (*Acox1, Cpt1a, Cpt1b*). Further, maternal PPAR pathway gene expression was almost equally as affected as the fetal livers, however with a notably distinct profile. Wang et al. (2017a) reported up-regulation of PPAR pathway genes in mouse liver following HFPO-DA exposure, whereas Hu et al. (2005) and Martin et al. (2007) performed microarray analyses of adult rat liver gene profiles following oral PFOS and PFOA exposure and reported similar up-regulation of clusters of genes primarily associated with lipid homeostasis. The gene expression profiles reported here indicate that HFPO-DA reached the fetal organs and activated nuclear receptor–mediated cell-signaling pathways and that the profile of expression was different than the maternal gene expression profile. However, the findings are not adequate to definitively conclude that a PPARα mechanism of action is operative for the HFPO-DA effects observed here.

In addition to changes in PPAR-mediated gene expression in the maternal liver, we observed a number of alterations to maternal serum lipid and thyroid hormone profiles similar to previous PFAS studies. Luebker et al. (2005b) reported significantly reduced serum cholesterol in pregnant SD rats following PFOS exposure, and Martin et al. (2007) also reported significantly reduced serum cholesterol in adult male SD rats following both PFOS and PFOA exposure. Disruption of maternal rat cholesterol synthesis with a HMG-CoA reductase inhibitor *in utero* has been

shown to induce fetal and neonatal death and retard growth in the absence of maternal toxicity (Henck et al. 1998). It is believed that the majority, if not all, of the cholesterol utilized in the earliest stages of fetal development is derived from the mother, prior to the onset of fetal cholesterol synthesis (Baardman et al. 2013). Further, Martin et al. (2007), Thibodeaux et al. (2003), and Yu et al. (2009) reported significant reductions in serum total T_3 and T_4 for both PFOS and/or PFOA; however, T_4 appeared to be more greatly reduced, whereas in the present study T_3 was more affected. Maternal thyroid hormones are critical for fetal neurological development because the mother is the primary source of T_4 for the developing brain (Morreale de Escobar et al. 2004) and reduced maternal thyroid hormone concentrations are quantitatively linked to reduced fetal concentrations (O'Shaughnessy et al. 2018). Despite the consistency observed across laboratory rat studies, it is unclear how these results relate to human health effects from PFAS exposure because many epidemiological studies report the opposite patterns or equivocal results (Lau et al. 2007; U.S. EPA 2016a).

Gomis et al. (2018) recently reported on the potential discrepancy in toxicity among a range of PFAS when using orally administered dose as compared with internal dose. By accounting for toxicokinetics in rats across multiple PFAS, the toxicity of some fluorinated alternatives appears to be more equitable to the long-chain PFAS when potency is compared based on internal dose. However, it is important to highlight the substantial toxicokinetic differences between PFOS and HFPO-DA in the rat. In the female rat, HFPO-DA has a reported half-life of $\sim$5 h following oral exposure to 10–30 mg/kg (Gannon et al. 2016) and is not expected to accumulate, whereas PFOS has a reported half-life of $\sim$60–70 days following oral exposure to 2–15 mg/kg (Chang et al. 2012) and does accumulate. Our samples were collected 2 h after the final oral dose, which is just slightly after the peak serum concentration is achieved in the female rat based on the Gomis et al. (2018) model.

In addition to intraspecies differences in PFAS toxicokinetics, it is also important to note that interspecies differences in absorption, distribution, metabolism, and excretion of PFAS are vast, with half-lives and clearance rates of numerous compounds appearing to be significantly longer in humans and nonhuman primates than in rats/mice (Chang et al. 2012; Olsen et al. 2007). The half-life of HFPO-DA in humans is currently unknown; however, similar to the discussion above, internal dosimetry can potentially reduce uncertainty in cross-species hazard assessment. For comparison, we calculated MOIE values for maternal rat serum concentrations versus plasma samples from humans working in a HFPO-DA manufacturing facility in the Dordrecht, Netherlands (DuPont 2017) (Figure 7). Bessems et al. (2017) originally described the use of MOIE as a physiologically based kinetic modeling approach for reducing uncertainty in the safety assessment of human dermal exposures using oral rodent toxicity data. Comparison of MOIE accounts for species- and route-dependent differences in metabolism between humans and research animals. Here, we utilized a similar calculation to reduce the species-to-species variation in PFAS toxicokinetics and to provide context for the oral doses utilized in terms of known human exposure levels. The highest detected plasma concentration from a worker (0.169 μg/mL) was 4-fold lower than the mean maternal rat serum HFPO-DA concentration from the lowest dose level (1 mg/kg per day) reported here; whereas the same worker concentration was 272-fold below the mean maternal serum concentration from the dose level (125 mg/kg per day) used in the pilot postnatal study presented here. Overall, characterizing toxicokinetics and internal dosimetry for PFAS, including HFPO-DA, can facilitate the determination of the relevance of doses in laboratory animals to human exposures, thereby reducing some of the uncertainty in estimating human health risks from exposure.

The HFPO-DA toxicity profile observed here was highly similar to effects observed in peer-reviewed and industry guideline studies for HFPO-DA as well as in studies conducted for PFOS (among other PFAS). PPAR signaling pathways were activated in maternal and fetal livers and may also be activated in other tissues/organs; however, the effects observed are not necessarily exclusive to PPARα, or even PPAR signaling in general (Rosen et al. 2017). The GenX chemicals health assessment is currently undergoing independent, external peer-review in the Office of Water (U.S. EPA). Included in that assessment is a summary of available mode-of-action (MOA) information. Although findings in this study are consistent with other PPARα agonists (e.g., increases in liver weight, up-regulation of PPAR pathway target genes), data gaps exist for key events and other mechanisms that might be involved, particularly in other tissues besides those like the liver with high PPARα levels. Overall, the findings for HFPO-DA are limited and not adequate to support ascribing a PPARα MOA to the multitude of effects seen in this study. Due to the reductions in maternal serum thyroid hormones and lipids observed here, and preliminary studies in our lab, an expanded dosing period that includes the entire period of fetal development may lead to effects on fetal and neonatal development similar to those observed with PFOS and PFOA exposure. Extensive research is needed to investigate the mechanism(s) by which HFPO-DA/PFOS/PFOA produce toxicity, to characterize the toxicokinetics for this and other PFAS in order to better predict toxic effects, and to assess the mixture-based effects of exposure to multiple PFAS compounds given their ubiquitous occurrence.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT H
TO AMENDED INTERVENOR COMPLAINT

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Toxicity of Balb-c mice exposed to recently identified 1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl] oxyethane-1-sulfonic acid (PFESA-BP2)

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ABSTRACT

1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl]oxyethane-1-sulfonic acid (PFESA-BP2) was first detected in 2012 in the Cape Fear River downstream of an industrial manufacturing facility. It was later detected in the finished drinking water of municipalities using the Cape Fear River for their water supply. No toxicology data exist for this contaminant despite known human exposure. To address this data gap, mice were dosed with PFESA-BP2 at 0, 0.04, 0.4, 3, and 6 mg/kg-day for 7 days by oral gavage. As an investigative study, the final dose groups evolved from an original dose of 3 mg/kg which produced liver enlargement and elevated liver enzymes. The dose range was extended to explore a no effect level. PFESA-BP2 was detected in the sera and liver of all treated mice. Treatment with PFESA-BP2 significantly increased the size of the liver for all mice at 3 and 6 mg/kg-day. At the 6 mg/kg-day dose, the liver more than doubled in size compared to the control group. Male mice treated with 3 and 6 mg/kg-day and females treated with 6 mg/kg-day demonstrated significantly elevated serum markers of liver injury including alanine aminotransferase (ALT), glutamate dehydrogenase (GLDH), and liver/body weight percent. The percent of PFESA-BP2 in serum relative to the amount administered was similar in male and female mice, ranged from 9 to 13 %, and was not related to dose. The percent accumulation in the liver of the mice varied by sex (higher in males), ranged from 30 to 65 %, and correlated positively with increasing dose level.

1. Introduction

Perfluoroalkyl substances (PFASs) have been detected in the global environment, including points far from sites of production and/or use. (Giesy and Kannan, 2001) The unique stability of the carbon-fluorine bond results in PFASs having exceedingly long environmental half-lives (Banks et al., 2013). Concerns about PFASs have resulted in establishment of regulations for some PFASs and voluntary advisory levels for others (ITRC Council, 2018). Public concerns and regulatory guidelines have focused on a small number of PFASs. Although there are currently thousands of compounds categorized as PFASs (Wang et al., 2017), there have been only approximately 1223 PFAS historically registered in commerce in the US, with 602 actively in commerce today (USEPA, 2019).

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; ESI, electrospray ionization; GLDH, glutamate dehydrogenase; HDPE, high density polyethylene; IACUC, Institutional Animal Care and Use Committee; NCDEQ, North Carolina Department of Environmental Quality; NRC, National Research Council; NVHOS, 1,1,2,2-tetrafluoro-2-(1,2,2,2-tetrafluoroethoxy)ethanesulfonic acid DTXSID80904754; PCR, polymerase chain reaction; PFASs, perfluoroalkyl substances; PFESA-BP2, 1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl]oxyethane-1-sulfonic acid DTXSID10892352; PFOA, perfluorooctanoic acid DTXSID8031865; PFOS, perfluorooctanoic sulfonic acid DTXSID3031864; HFPO-DA, perfluoro-2-methyl-3-oxahexanoic acid DTXSID70880215; TSCA, Toxic Substance Control Act; USEPA, United States Environmental Protection Agency

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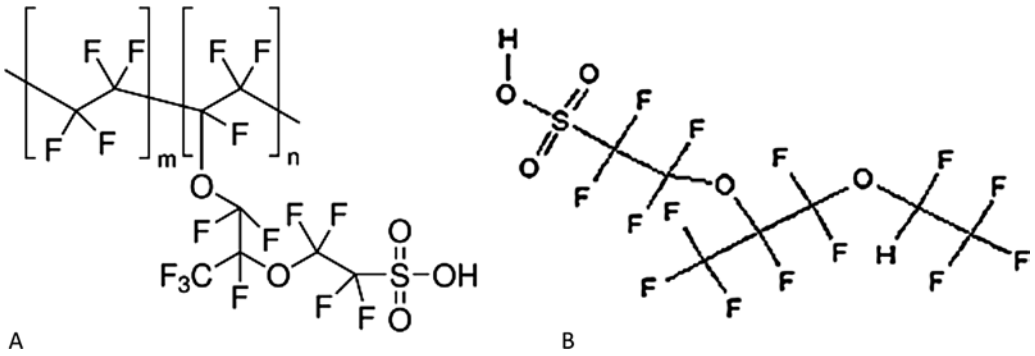

Fig. 1. Structure of the Nafion Polymer (A) and PFESA-BP2 (B).

1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl]oxyethane-1-sulfonic acid (PFESA-BP2 CAS #749836-20-2) is assumed to exist as a by-product of manufacturing Nafion polymer (Fig. 1). PFESA-BP2 has not been the subject of a pre-manufacture notice and review under the US Toxic Substance Control Act (TSCA), which is required only for chemicals intended for a commercial purpose. By-product release into the environment does not follow the same laws as chemicals intended for commerce, therefore there is no toxicology information requirement. PFESA-BP2 is a 7-carbon sulfonate with an monoisotopic mass of 463.93 amu and with two internal ether oxygens, giving it a mass and general structure (length) that is similar to perfluorooctanoic sulfonic acid (PFOS - 498.93 amu). These similarities may infer a longer half-life and possibly similar toxicity. Because the compound is a by-product of Nafion, a sulfonated tetrafluoroethylene-based polymer, it also has been referred to as Nafion by-product 2.

In 2012, two PFESA byproducts (i.e. PFESA-BP2 and perfluoro-3,6-dioxa-4-methyl-7-octene-1-sulfonic acid (PFESA-BP1 CAS #29311-67-9 DTXSID30892354))were detected in North Carolina's Cape Fear River, downstream of an industrial manufacturing facility. (Strynar et al., 2015) In a September 2017 report to the North Carolina Department of Environmental Quality (NCDEQ), the United States Environmental Protection Agency (USEPA) used a non-targeted analytical method to estimate PFESA-BP2 concentrations in Chemours discharge effluent and the Cape Fear River downstream of manufacturing as 45,200 ng/L and 2075 ng/L, respectively. (Buckley, 2017) These reported PFESA-BP2 concentrations were provided as gross estimates because a PFESA-BP2 standard was unavailable at that time. As such, these concentrations assume that the mass spectrometer responded to the non-targeted analyte as if it were Perfluoro-2-methyl-3-oxahexanoic acid, HFPO-DA, CAS #13,252-13-6], for which a standard was available. The report suggests such estimates are accurate to within 10-fold of the estimated value.

In July 2017, North Carolina's Brunswick County drinking water provider (H2Go) began bi-weekly sampling for PFESA-BP2, with concentration estimates ranging from non-detectable (ND) to 134 ng/L in their finished drinking water. (H2GO PFC Sampling, 2020) NCDEQ reported PFESA-BP2 in private wells near the industrial manufacturing facility with concentrations up to 125 ng/L (NCDEQ, 2018). With the availability of an authentic standard provided by the manufacturer, subsequent studies corroborated PFESA-BP2 contamination in finished drinking water (Hopkins et al., 2018), but also in 99 % of serum samples from public volunteers from this same region (Katlorz, 2018). The study demonstrated the presence of PFESA-BP2 is likely isolated to the area downstream of the NC industrial manufacturing facility because serum samples from residents of Raleigh, NC, Chapel Hill, NC, Durham, NC and Dayton, Ohio did not contain this compound. These studies demonstrate the presence of PFESA-BP2 contamination in water sources within the Cape Fear River Basin, as well as the widespread presence of this compound in human serum samples from this same region.

Despite the known presence of PFESA-BP2 in the environment and in human blood, there are no known toxicology studies utilizing PFESA-BP2. Previous studies on perfluorooctanoic acid PFOA and PFOS have demonstrated that these compounds bioaccumulate in the liver and serum of affected animals (rat, mouse, rabbit, monkey), and induce liver toxicity. (Lau et al., 2006; Yang et al., 2014)

Given the potential health effects associated with PFAS compounds and the presence of PFESA-BP2 in human serum, this initial study examined the hepatotoxic effects and bioaccumulation of PFESA-BP2 in adult mice exposed by oral gavage for seven days (0.04–6 mg/kg-day).

2. Material and methods

2.1. Animals

Balb-c mice, an inbred strain we have used to study hepatotoxic algal toxins, 10 − 12-week-old males and females, were obtained from Charles River Laboratories (Raleigh, NC, USA). The animals arrived at the US EPA's National Health and Environmental Effects Research Laboratory (NHEERL) animal facility post-weaning and allowed to acclimate for at least 5 days prior to initiation of the experiments. Animals were randomly selected, but cage groups were corrected to keep the body weight variance < 1. Animals were housed by treatment group in polycarbonate cages on heat-treated pine shaving bedding in animal rooms with a controlled temperature range (22–26 °C) and a 12:12-h light–dark cycle. Animals were fed commercial rodent chow (Purina Prolab) and water ad libitum. All studies were conducted after approval by the USEPA Institutional Animal Care and Use Committee (IACUC) using recommendations of the 2011 National Research Council (NRC) "Guide for the Care and Use of Laboratory Animals" and the Public Health Service Policy on the Humane Care and Use of Laboratory Animals. (Guide for the Care and Use of Laboratory Animals, 2011)

2.2. Experimental design

Animals were dosed with PFESA-BP2 for seven consecutive days by gavage using 20-gauge stainless steel feeding needles. Seven-day exposure was chosen to enable demonstration of bioaccumulation and a dose of 3 mg/kg was used which exhibited effects. Additional dosages were added in later blocks to establish a wide range of responses. The complete experiment was run across five different blocks. Each block included control animals, and each dose group was used in at least two blocks, except for the highest dose (6 mg/kg) which was not repeated. Doses of 0, 0.04, 0.4, 3, and 6 mg/kg-day were administered once daily in the afternoon. The number of animals ranged from 10 to 24 per dose group, divided equally between males and females. Animals were weighed before the dosing was started, every other day during dosing, and at the time of euthanasia. Their appearance was monitored daily. PFESA-BP2 was obtained from Chemours (78.8 % purity - 14 % potassium fluoride (KF) – 6.6 % (1,1,2,2-Tetrafluoro-2-(1,2,2,2-

tetrafluoroethoxy) ethanesulfonic acid (NVHOS CAS #801209-99-4)). A stock dosing solution was prepared by dissolving PFESA-BP2 in ethanol (EtOH) followed by dilution with deionized (DI) water for a final concentration of 1 g/L in 90:10 DI H2O:EtOH. The stock solution was diluted with DI water to establish dosing solution concentrations for each treatment at a dosing volume of 0.2 mL per day. The final PFESA-BP2 concentration in the dosing solutions ranged from 0.002 to 0.8 g/L (data not shown). The control group received the carrier of Picopure water with an ethanol concentration equal to the dosing solution with the highest ethanol concentration which was always the high dose males (did not exceed 7.15 % ethanol).

Approximately 24 h after the seven-day dosing was completed, all animals were anesthetized by CO_2 inhalation, weighed, euthanized by exsanguination (blood collection), and necropsied. Blood was obtained transdermally from the heart with a 25-gauge 5/8 in needle attached to a 1 mL syringe. Whole blood was collected in 0.5 mL serum separator tubes (Becton Dickinson), allowed to clot at room temperature, centrifuged at 13,000 rpm for 1.5 min per manufacturer's instructions (Dickinson, 2011), and serum isolated. Serum samples were stored at −20 °C in 2.5 mL high density polyethylene (HDPE) tubes until analysis. The liver was removed from each animal, weighed, and divided into samples. One sample of the liver was stored in foil at −20 °C for PFESA-BP2 analysis, a sample from the largest liver lobe was fixed in 10 % neutral buffered formalin for 48 h before being transferred to 70 % ethanol for histopathology, and a third sample was placed in RNAlater and stored at −20 °C for polymerase chain reaction (PCR) analysis at a later time.

2.3. Histopathology

Samples of liver from one male and one female mouse from the control, 0.4 mg/kg-day and 3 mg/kg-day treatment groups were viewed microscopically to study the appearance of the cells by Pathogenesis LLC (Gainesville, FL). Limited resources restricted the number of tissues that could be processed and analyzed, but our main goal was to confirm that the increased liver weight was due to hepatocyte hypertrophy as seen with other PFAS (Toxicologic Profile of Perfluoroalkyls, Draft, US Dept. HHS, 2018) versus hepatocyte hyperplasia. Livers from the 0.04 mg/kg-day and 6 mg/kg-day group were not analyzed with histopathology. Each block was sectioned at 5 microns and stained with hematoxylin and eosin according to a previously published methodology. (Chernoff et al., 2018) Tissue sections were evaluated microscopically without the evaluator having prior knowledge of the treatment group. Histologic features were scored using a semi-quantitative scoring scheme with 0 = no change to 4 = severe change (Chernoff et al., 2018). Numbers of individual apoptotic hepatocytes (consistent with apoptosis) and mitotic figures were counted in each of ten 400X fields centered on a central vein.

For computer-aided image analysis of Zone 3 (centrilobular) hepatocytes, multiple photomicrographs at 1000X magnification were collected from at least 5 randomly selected hepatic lobules per mouse. The area of 30 individual hepatocytes from Rappaport Zone 3 of each liver was calculated using the lasso tool in Photoshop (lasso to outline individual hepatocytes > Image > Analysis > Record Measurement), Adobe Photoshop CC 2017.

2.4. Clinical chemistry

All serum clinical chemistry analyses were carried out using the Randox Daytona Plus instrument (Belfast, Northern Ireland). Due to serum volumes < 300 µL, serum chemistries were not performed in duplicates, Hepatic cell and bile duct injury was assessed by determining the serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), glutamate dehydrogenase (GLDH), and bilirubin. Markers for potential renal injury included serum concentrations of blood urea nitrogen (BUN) and creatinine. Serum glucose, total protein, and albumin were measured as markers of general toxicity. All assays were performed using reagents obtained from the instrument manufacturer.

2.5. Extraction and analysis of PFESA-BP2 from tissue and serum

PFESA-BP2 was extracted from serum and tissue samples using methods presented in Reiner et al., 2009. (Reiner et al., 2009) In brief, liver samples were weighed in 15 mL HDPE centrifuge tubes and homogenized at approximately a 3:1 DI:sample wet weight ratio using an Omni-Prep Multi Sample Homogenizer. Liver homogenate and serum samples from mice treated with PFESA-BP2 were diluted at variable ratios with DI water to bring the concentrations within the values of the external calibration curve. Serum and liver homogenates from control mice were analyzed directly without dilution. The diluted samples (50 µL) were pipetted into a fresh 15 mL HDPE centrifuge tube, followed by 100 µL of 0.1 M formic acid. After vortex-mixing, 0.5 mL of cold acetonitrile (ACN) was added to each tube. Samples were vortex-mixed again and then centrifuged at 1000 rpm for 3 min. The supernatant (100 µL) was combined with 300 µL of 2.5 mM ammonium acetate in HDPE vials. Approximately 10 % of the samples were extracted in duplicate.

Samples were analyzed using an Agilent 1100 series HPLC equipped with an Eclipse Plus C8 column (2.1 × 50 mm, 3.5 µm; Agilent) interfaced to an Agilent 6210 series Accurate-Mass MS-TOF system with negative electrospray ionization (ESI). The mobile phase system consisted of 0.4 mM ammonium formate in 95:5 deionized water:methanol (A) and 95:5 methanol:deionized water (B). Quantification of PFESA-BP2 was based on comparison of a single ion peak area in negative mode 462.9326 [M − H]- to the response of an external standard curve created by spiking variable levels of standard into control liver homogenate or serum. The standard used for quantification was provided by the manufacturer as an 1% aqueous solution. Analytical blanks (i.e. ACN and Pico-pure water) were analyzed with every run. When appropriate, isotopically labeled (^{13}C) PFOA purchased from Wellington Laboratories Inc. was used as the internal standard for quantification of the liver and serum concentrations.

2.6. Statistical evaluation

All variables were analyzed separately by sex with two-way main effects ANOVAs, which included factors for dose and block. This allowed testing for changes due to PFESA-BP2 treatment after adjusting for mean differences due to block. If the F-test for treatment effect was significant ($p < 0.05$), each treatment group was compared to vehicle controls with pairwise t-tests, using Dunnett's adjustment for multiple comparisons. The ANOVA assumptions of normality and homogeneity of variance were examined using the Shapiro-Wilk and Levene's tests. ALT, AST, GLDH were transformed to the log10 scale to satisfy these assumptions.

3. Results

3.1. Toxicity

Changes in animals' body weights, liver weights, and liver appearance are summarized in Table 1. No changes in the animals' appearance or overt behavior were observed during the dosing period. Significant increases in body weights during the dosing period occurred in the 6 mg/kg female animals. The relative and absolute liver weights increased significantly in the 3 and 6 mg/kg dose groups for the males and females. At the 6 mg/kg-day dose level, the liver weight was greater than the controls by two-fold. At necropsy, livers of 3 and 6 mg/kg-day mice were enlarged and pale, and the surfaces were reticulated (i.e. pattern of individual liver lobules made visible due to color change of hepatocytes). The control group contained no animals with

Table 1
Effects of PFESA-BP2 on average body weights, liver weights, and clinical serum chemistry in Balb-c mice after 7 days of treatment. The sample sizes for the data presented here are demonstrated in Table S1 for each dose group and variable.

	Males				
	0	0.04 mg/kg-day	0.4 mg/kg-day	3 mg/kg-day	6 mg/kg-day
Number of Mice Dosed	12	10	15	10	5
Body Weight (g)	22.9 ± 0.37	23.2 ± 0.55	23.6 ± 0.42	23.2 ± 0.47	23.9 ± 0.69
Liver Weight (g)	1.29 ± 0.03	1.33 ± 0.05	1.37 ± 0.04	2.02 ± 0.04 ***	2.79 ± 0.06 ***
Liver/Body Weight (%)	5.62 ± 0.08	5.75 ± 0.12	5.83 ± 0.09	8.70 ± 0.10 ***	11.7 ± 0.15 ***
Number of Mice with Visual Liver Reticulation	0	1	2	10	5
ALT (log 10 U/L)	1.81 ± 0.06	1.79 ± 0.09	1.72 ± 0.07	2.04 ± 0.08	2.34 ± 0.11 ***
AST (log 10 U/L)	2.08 ± 0.07	2.07 ± 0.10	1.94 ± 0.08	2.15 ± 0.09	2.24 ± 0.13
GLDH	1.15 ± 0.04	1.19 ± 0.06	1.05 ± 0.05	1.35 ± 0.05 *	1.71 ± 0.08 ***
BUN (mg/dl)	9.07 ± 0.35	9.11 ± 0.48	9.08 ± 0.44	9.09 ± 0.41	9.26 ± 0.62
Albumin (g/dl)	3.30 ± 0.08	3.32 ± 0.11	3.44 ± 0.09	3.56 ± 0.10	3.59 ± 0.14
Globulin (g/dl)	2.07 ± 0.05	2.09 ± 0.07	2.16 ± 0.06	2.25 ± 0.06	2.35 ± 0.09 *
Total Protein (g/dl)	5.37 ± 0.11	5.42 ± 0.17	5.59 ± 0.13	5.81 ± 0.14	5.93 ± 0.21
Glucose (mg/dl)	201 ± 9.52	194 ± 13.9	192 ± 10.8	200 ± 12.0	159 ± 17.57
Tbil (mg/dl)	0.41 ± 0.07	0.31 ± 0.10	0.30 ± 0.08	0.35 ± 0.09	0.30 ± 0.12
Triglycerides (mg/dl)	319 ± 32.6	325 ± 33.0	328 ± 29.5	341 ± 23.8	374 ± 31.9
Cholesterol (mg/dl)	128 ± 9.05	122 ± 9.75	129 ± 8.70	140 ± 7.00	122 ± 9.43
	Females				
	0	**0.04 mg/kg-day**	**0.4 mg/kg-day**	**3 mg/kg-day**	**6 mg/kg-day**
Number of Mice Dosed	12	10	15	10	6
Body Weight (g)	18.9 ± 0.23	18.7 ± 0.34	19.0 ± 0.27	19.5 ± 0.29	20.0 ± 0.43 *
Liver Weight (g)	0.98 ± 0.03	0.94 ± 0.04	0.96 ± 0.03	1.67 ± 0.03 ***	2.38 ± 0.05 ***
Liver/Body Weight (%)	5.20 ± 0.13	5.08 ± 0.19	5.09 ± 0.15	8.29 ± 0.16 ***	11.5 ± 0.24 ***
Number of Mice with Visual Liver Reticulation	0	3	1	7	5
ALT (log 10 U/L)	1.92 ± 0.08	1.85 ± 0.12	1.88 ± 0.09	2.01 ± 0.10	2.49 ± 0.15 **
AST (log 10 U/L)	2.26 ± 0.07	2.22 ± 0.10	2.28 ± 0.08	2.13 ± 0.08	2.54 ± 0.12
GLDH	1.40 ± 0.07	1.31 ± 0.10	1.23 ± 0.08	1.35 ± 0.08	1.75 ± 0.12 *
BUN (mg/dl)	8.31 ± 0.42	8.11 ± 0.53	8.30 ± 0.42	9.07 ± 0.46	8.92 ± 0.69
Albumin (g/dl)	3.33 ± 0.09	3.26 ± 0.14	3.35 ± 0.11	3.53 ± 0.12	3.43 ± 0.17
Globulin (g/dl)	1.79 ± 0.06	1.73 ± 0.09	1.84 ± 0.07	2.09 ± 0.08 *	2.08 ± 0.12
Total Protein (g/dl)	5.12 ± 0.15	4.98 ± 0.22	5.17 ± 0.18	5.62 ± 0.19	5.52 ± 0.28
Glucose (mg/dl)	224 ± 12.1	227 ± 17.7	213 ± 14.1	212 ± 15.2	235 ± 22.4
Tbil (mg/dl)	0.24 ± 0.06	0.32 ± 0.09	0.45 ± 0.07	0.31 ± 0.08	0.35 ± 0.11
Triglycerides (mg/dl)	191 ± 39.5	185 ± 33.8	200 ± 25.5	324 ± 25.0 *	280 ± 36.1
Cholesterol (mg/dl)	113 ± 23.8	114 ± 23.5	134 ± 18.2	99.6 ± 17.8	112 ± 25.7

The statistics for this table are based use F-test p-value from ANOVA; Averages demonstrated for each group with standard error.
* $p \leq 0.05$, ** $p \leq 0.001$, *** $p \leq 0.0001$ relative to control.

reticulated livers.

Samples from the control, 0.4 mg/kg-day and 3 mg/kg-day treatment groups were viewed microscopically to study the appearance of the cells. Livers from the 0.04 mg/kg-day and 6 mg/kg-day dose group were not analyzed with histopathology. Histopathology revealed hepatocyte hypertrophy predominantly in the centrilobular portion of the liver lobule (Rappaport Zone 3) for the 3 mg/kg-day dose group (Fig. 2). The hypertrophy extended to a lesser degree into Zone 2. Mitotic figures were another change observed in the 3 mg/kg-day livers and may indicate a response by the liver not seen in the control and 0.4 mg/kg-day mice. Intracytoplasmic vacuoles (spaces) were present in all treatment groups and are created during tissue processing which washes out lipid and glycogen accumulation within the cytoplasm. Vacuoles were recorded as fine to moderately large, sharp-edged, clear vacuoles consistent with lipid accumulation or as vacuoles with less distinct borders consistent with glycogen accumulation. The vacuoles consistent with glycogen accumulation did not vary between zones of the liver lobule or treatment group, whereas the vacuoles consistent with lipid accumulation were observed in Zones 2 and 3 and had slightly increased numbers in the 3 mg/kg-day livers compared to the control group. When hypertrophy is present, it is common to develop initially around the central vein and spread outward as seen in the 3 mg/kg-day mice. Larger group numbers would need to be evaluated to determine if cell death and intracytoplasmic vacuoles are significant in the higher dose.

Serum liver function markers indicative of hepatotoxicity were detected in both sexes within the 3 and 6 mg/kg-day treatment groups. Elevated ALT concentrations occurred in the 6 mg/kg/day treatment for both sexes and in the 3 mg/kg/day male dose group (Table 1). Increased GLDH was seen in both 3 and 6 mg/kg-day males and the 6 mg/kg-day females. Elevated serum protein levels occurred in males with significant increases in both globulin and total proteins at the 3 and 6 dose levels. For females, only the globulin levels were increased for both the 3 and 6 mg/kg-day dose levels.

3.2. PFESA-BP2 bioaccumulation

All analytical blanks were negative for PFESA-BP2. The coefficient of determination (R2) was greater than 0.98 for all standard curves. Average PFESA-BP2 serum concentrations ranged from 0.47 μg/mL in the 0.04 mg/kg-day dose group to 88 μg/mL in the 6 mg/kg-day dose group (Table 2). The average serum concentration at the lowest dose level was between 100 and 200-fold higher than the average PFESA-BP2 concentrations reported in serum from the residents of Wilmington, NC (Katlorz, 2018). It is notable that bioaccumulation did occur with the presence of two internal ether oxygens, suggesting molecular length (and mass) increase retention in biological systems. The average PFESA-BP2 liver concentrations ranged from 1.4 μg/g in the 0.04 mg/kg-day female mice to 240 μg/g in the 6 mg/kg-day male mice (Table 2). The concentrations of PFESA-BP2 in the serum and liver are in the range of previously reported mouse serum PFOA/PFOS concentrations. (Lau et al., 2006; Thibodeaux et al., 2003; Wolf et al., 2008; Guo et al., 2019) For example, samples collected from WT mice dosed with PFOA at 3 mg/kg-day for seven days demonstrated average

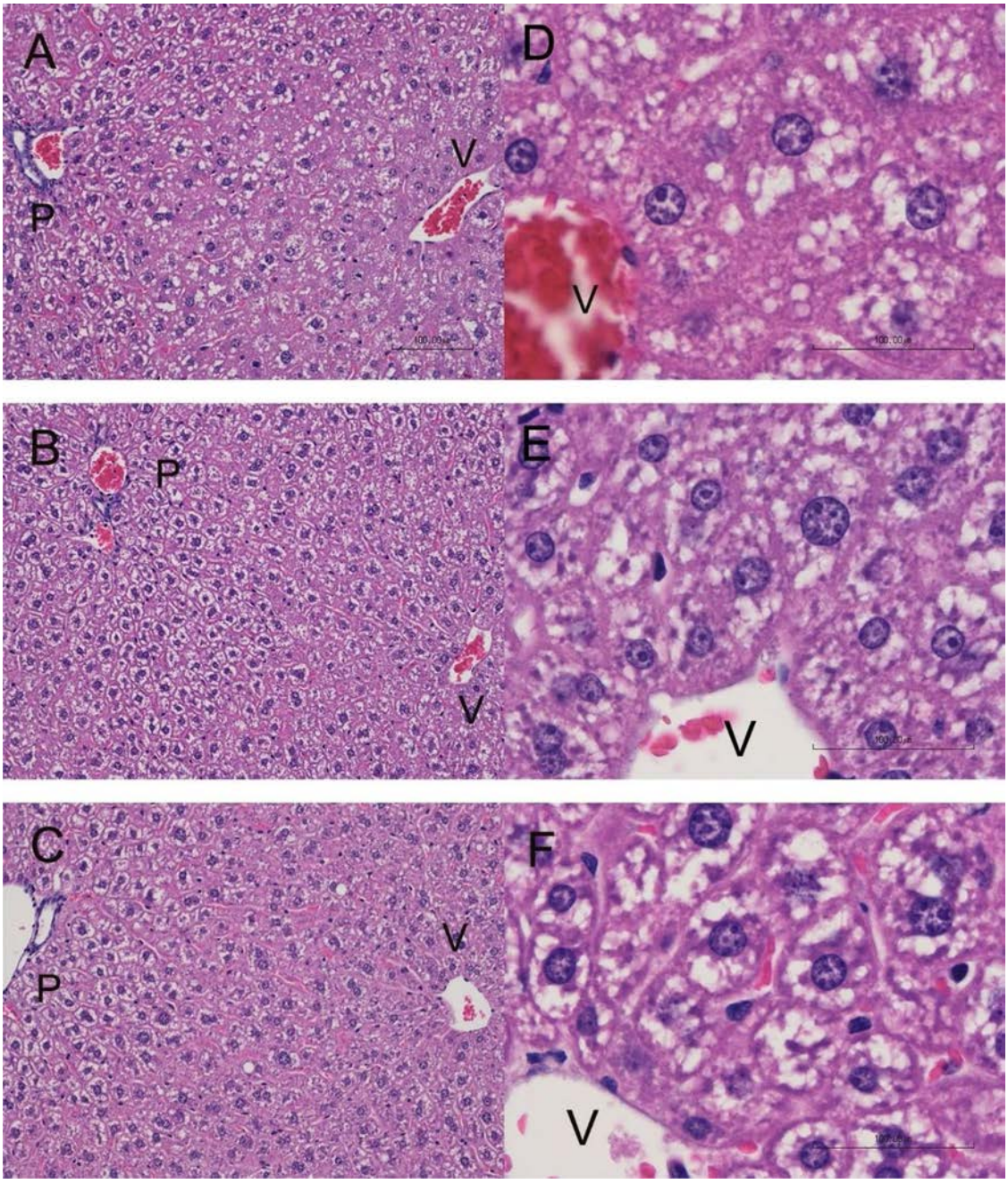

Fig. 2. Liver histopathology for Balb-c mice receiving PFESA-BP2 at 3 mg/kg-day (A, D), 0.4 mg/kg-day (B, E), or vehicle (C, F). Livers from the 3 mg/kg-day dose group demonstrated increased cytoplasmic volume and density of cytoplasmic contents of centrilobular hepatocytes surrounding the central vein (V) compared with hepatocytes closer to the portal region (P), a change which was not observed in liver from the lower concentration of PFESA-BP2 or vehicle mice. Slides A, B, and C are at 100x magnification; slides D, E, and F are at 400x magnification.

serum concentrations of ~33.3 μg/mL. (Wolf et al., 2008) This value is slightly lower than the 3 mg/kg-day serum concentrations reported here (~48 μg/mL), but it is unclear if the lower values are attributed to compound differences or the strain of mouse treated for the experiment.

The percent of PFESA-BP2 in serum relative to the amount administered, ranging from 9 to 13 %, was similar in male and female mice and did not demonstrate a direct relationship with dose (Table 2). The percent accumulation in the liver of the mice, ranging from 30 to 65 %, varied by sex (higher in the males) and correlated positively with increasing dose level (Table 2). Higher accumulations in the liver compared to serum could have implications for the human population in cases where PFESA-BP2 was identified in serum. (Katlorz, 2018)

PFESA-BP2 was detected at low levels (< 0.3 μg/g) in two of the control livers analyzed. The contamination is assumed to be due to reuse of necropsy instruments across animals because it was present in only two of the livers and was not present in the serum for these animals, and dosing protocol would not allow occurrence of cross-contamination with dosing instruments. Since the serum levels are an order of magnitude lower than that in the livers of mice treated with PFESA-BP2, this contamination is not expected to affect the toxicity and bioaccumulation results.

4. Discussion

The results presented here demonstrate that short term (7 day) exposures to PFESA-BP2 significantly increased liver weights in treated

Table 2
Serum and liver PFESA-BP2 concentrations relative to total dose administered.

	Male			
	0.04 mg/kg-day	**0.4 mg/kg-day**	**3 mg/kg-day**	**6 mg/kg-day**
Dosing Solution Concentration (g/L)	4.0	36.8	344	716
Total Administered (μg)	5.6	51.52	481.6	1002.4
Serum Concentration (μg/mL)	0.51 ± 0.07[b]	3.99 ± 0.28	47.0 ± 3.45	83.9 ± 17.1
Liver Concentration (μg/g)	2.41 ± 0.38	20.1 ± 3.23	143 ± 31.2	235 ± 30.9
Serum Accumulation (μg)[a]	0.69	5.51	63.8	117
Liver Accumulation (μg)	3.21	27.5	289	656
% Serum Accumulation	12 %	11 %	13 %	12 %
% Liver Accumulation	57 %	53 %	60 %	65 %
	Female			
	0.04 mg/kg-day	**0.4 mg/kg-day**	**3 mg/kg-day**	**6 mg/kg-day**
Dosing Solution Concentration (g/L)	3.15	30.1	313	624
Total Administered (μg)	4.41	42.14	438.2	873.6
Serum Concentration (μg/mL)	0.44 ± 0.17[b]	3.55 ± 0.98	48.0 ± 14.4	92.9 ± 83.9
Liver Concentration (μg/g)	1.43 ± 0.12	17.5 ± 4.37	129 ± 41.4	208 ± 24.2
Serum Accumulation (μg)[a]	0.48	3.95	54.8	109
Liver Accumulation (μg)	1.34	16.8	215	495
% Serum Accumulation	11 %	9%	12 %	12 %
% Liver Accumulation	30 %	40 %	49 %	57 %

[a] Serum volumes estimated assuming serum accounts for 5.85 % of the total body weight.
[b] Group means with one standard deviation.

mice following doses of 3 and 6 mg/kg-day and created a greater than two-fold increase in liver weight of both male and female mice at the 3 and 6 mg/kg-day. Previous rodent PFAS studies have demonstrated hypertrophy due to peroxisome proliferation. (Wolf et al., 2008; Chappell et al., 2020; Cui et al., 2009; Adinehzadeh et al., 1999; Blake et al., 2020), We propose that the hypertrophy seen with this sulfonated PFAS, similar in mass and length to PFOS, would likely act by similar mechanisms. Elevated serum liver function tests indicate that injury occurs at PFESA-BP2 doses ≥ 3 mg/kg-day in both sexes with males apparently more sensitive than the females. There were no adverse effects detected at the 0.04 and 0.4 mg/kg-day doses compared to the control group. At the lowest dose (0.04 mg/kg-day - ~500 ppb), serum levels were 100- to 200-fold higher than median serum concentration from humans exposed to PFESA-BP2 through drinking water (~3 ppb (Katlorz, 2018)).

5. Conclusions

To our knowledge this is the first toxicology study of PFESA-BP2. Given that this chemical induces hepatic effects comparable to those associated with other PFASs, additional toxicology studies are warranted. A mechanistic study using liver tissue collected in this study is currently in progress. Genomic analysis and more histopathological evaluations can also be explored with tissues collected in this study. Future work should include extended in vivo treatments to simulate a chronic environmental exposure covering different developmental life stages.

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Declaration of Competing Interest

The authors declare that there are no conflicts of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.tox.2020.152529.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant. _____ CAPE FEAR PUBLIC UTILITY AUTHORITY, Intervenor-Plaintiff, v. STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Defendant.	**INTERVENOR COMPLAINT BY CAPE FEAR PUBLIC UTILITY AUTHORITY FOR DECLARATORY AND INJUNCTIVE RELIEF**

COMES NOW Cape Fear Public Utility Authority ("CFPUA"), through counsel, and alleges and says:

BACKGROUND FACTS

1. CFPUA is a public utility authority created by New Hanover County and the City of Wilmington pursuant to North Carolina General Statutes Chapter 162A, and is vested with authority to sue in its own name. N.C. Gen. Stat. § 162A-6. CFPUA exercises public and essential

governmental functions to provide for the public health and welfare of its customers by providing potable water for residents of New Hanover County and the City of Wilmington. CFPUA owns and operates a water intake located on the Cape Fear River, downstream of the Defendant's Fayetteville Works Facility, and a water treatment plant to provide potable water to 200,000 North Carolinians and the schools, hospitals, industry, and other businesses and institutions that serve them.

2. Defendant The Chemours Company FC, LLC ("Chemours") is a corporation organized and existing under the laws of Delaware, and registered to do business as a foreign corporation in the State of North Carolina. Chemours currently owns and operates the Fayetteville Works Facility, located at 22828 NC Highway 87 W., Fayetteville, North Carolina.

3. The State's original Complaint ("State's Original Complaint") in this action was brought on behalf of the Department of Environmental Quality ("DEQ"), an agency of the State of North Carolina, pursuant to its delegated authority under the federal Clean Water Act ("CWA"), 33 U.S.C. §§ 1251 *et seq.*, to administer and enforce the National Pollution Discharge Elimination System ("NPDES") program, 33 U.S.C. § 1342, as specified in Article 21 of Chapter 143 of the North Carolina General Statutes. *See* Original Complaint ¶¶ 6-10.

4. As alleged in the State's Original Complaint in this action, this matter arises out of Defendant's operation of the Fayetteville Works Facility (the "Facility"), a chemical manufacturing facility located adjacent to the Cape Fear River just south of Fayetteville, North Carolina.

5. In the State's Original Complaint, the State alleged (among other things):

a. The surface water into which Defendant's Fayetteville Works Facility discharges wastewater is used as a public water supply source that serves residents and businesses in several counties [Paragraph 48];

b. Chemours and its predecessor knew for years that GenX and related compounds were being discharged from the Facility into surface waters of the State, in violation of North Carolina water quality laws [Paragraphs 56, 88];

c. Water samples collected at various times from the Cape Fear River showed concentrations of GenX were present in the Cape Fear River at levels in excess of the health goal established by the North Carolina Department of Health and Human Services ("DHHS") [Paragraphs 63, 87];

d. GenX and related compounds discharged from the Facility have been and are present in public drinking water supplied to residents and businesses in several counties [Paragraph 55];

e. On information and belief, public water supply treatment plants are ineffective at removing GenX and related compounds from Cape Fear River water [Paragraph 54];

f. From at least the beginning of 2009, Chemours' predecessor was aware of EPA's concern regarding the toxic effects of GenX on human health and the environment [Paragraphs 78-80];

g. Chemours' continuing violations of North Carolina water quality laws adversely affect the public interest [Paragraph 128]; and

h. The State is entitled to injunctive relief against Chemours to prevent and abate Chemours' unpermitted discharges [Paragraph 129].

6. GenX and related compounds are within a family of chemicals known as per- and polyfluoroalkyl substances or "PFAS." These chemicals are commonly used in the manufacture of nonstick coatings, stain- and water-resistant products, in fire-fighting foams, and for other consumer and commercial purposes.

7. Beginning the last week of June 2017, the Cape Fear Public Utility Authority has undertaken periodic sampling and analysis of Cape Fear River water, both the intake "raw" river water and treated "finished" water for distribution. A spreadsheet of the analytical results for samples of raw and finished water is attached as Exhibit A. The spreadsheet reflects that samples of the raw and finished Cape Fear River water have contained at least 23 different specific PFAS compounds in the water samples, The spreadsheet also shows the continuing variability of concentrations of PFAS compounds in the raw water and the finished water.

8. Additionally, graphs charting historic PFAS levels in the Cape Fear River against river flows at the raw water intake is attached as Exhibits B and C. As these exhibits demonstrate, PFAS concentrations are largely a function of river flows. Higher river flows dilute PFAS in the river, leading to lower concentrations. Conversely, lower flows result in higher PFAS concentrations. Accordingly, the levels of PFAS that CFPUA and its customers are exposed to are largely dependent on weather.

9. CFPUA's water treatment plant does not have the capability to treat and remove the PFAS pollutants that currently exist in the Cape Fear River. Although CFPUA can take certain interim measures to reduce PFAS levels in finished water by periodically replacing biofilters designed for other purposes, those measures are not only unsustainably expensive but also reduce the biofilters' capacity to remove contaminants for which they were designed.

10. After conducting pilot testing on treatment options to remove the PFAS pollutants from Cape Fear River water, CFPUA determined that the addition of a granular activated carbon ("GAC") filter system would be its best option for treatment. The cost of designing, constructing, testing, implementing, and operating a GAC system will be at least $70 million over a ten year period.

11. The Cape Fear Public Utility Authority board approved a resolution authorizing CFPUA to proceed with the design, permitting, and construction of a GAC. CFPUA has since completed the GAC designs, sold revenue bonds to finance the cost of the GAC, executed a construction contract, begun charging customers for the amortization of the bonds, and begun construction on the plant. The GAC is expected to be completed and operational in 2022.

State's Actions Following its Original Complaint Have Left CFPUA Unprotected

12. On September 8, 2017 – less than 24 hours after the State filed its Original Complaint – a hearing was held at which a Consent Order was entered ("Original Consent Order"), which recites that it "partially resolves this matter." Original Consent Order at 1.

13. Prior to the State's commencement of this enforcement action, the Cape Fear Public Utility Authority and its counsel were in frequent contact with various representatives of the North Carolina Department of Environmental Quality (DEQ) to provide information, especially emphasizing the vulnerable population served by CFPUA, and urging the State to take prompt and comprehensive enforcement action. Neither CFPUA nor its counsel were informed by the State of the filing of this action, the hearing scheduled for September 8, 2017, or the proposed Original Consent Order. CFPUA learned of the action and the Original Consent Order only after the Original Consent Order had been entered and filed.

14. On October 16, 2017, the Cape Fear Public Utility Authority filed a separate action against Chemours and its predecessor in interest, E. I. du Pont de Nemours ("DuPont") in federal court in the Eastern District of North Carolina. *Cape Fear Public Utility Authority v. The Chemours Company FC, LLC and E.I. du Pont de Nemours and Company*, 7:17-cv-195 ("CFPUA's Federal Suit"). Following a similar action initiated by Brunswick County against Chemours and DuPont, 7:17-cv-209, the two actions were consolidated and a Master Complaint of Public Water Suppliers (the "Master Complaint") was filed, in which Town of Wrightsville Beach and Lower Cape Fear Water & Sewer Authority joined.

15. The claims alleged in the Master Complaint are common law claims arising under State law. As alleged in the Master Complaint and in CFPUA's Notice to Conform to Master Complaint:

a. Chemours and DuPont have discharged PFAS, directly and via the groundwater and air emissions, into the State's groundwater and the Cape Fear River, in violation of federal and state law and applicable permits;

b. CFPUA is a downstream riparian owner that uses water from the Cape Fear River;

c. The quality of the waters of the Cape Fear River water is unreasonably diminished by the past and current discharges and other releases of PFAS by Chemours and DuPont;

d. As a riparian owner, CFPUA has a right to use water from the Cape Fear River whose quality is not unreasonably diminished;

e. PFAS discharged by Chemours and DuPont have accumulated in the sediment of the Cape Fear River, the groundwater that feeds the River, and in deposits in the

watershed from the air emissions from the Facility, and this will continue to unreasonably diminish the quality of the waters of the Cape Fear River;

f. CFPUA's water treatment plant does not have the technical capability to treat and remove the PFAS pollutants that currently exist in the Cape Fear River;

g. The current and prior PFAS discharges have caused and continue to cause harm and damages to CFPUA;

h. CFPUA is entitled to damages for the prior pollution caused by Chemours and its predecessor and to injunctive relief to prevent and abate continuing harm and damages to CFPUA.

16. On or around April 9, 2018, the State of North Carolina filed an Amended Complaint and Motion for Interim Preliminary Relief ("Amended Complaint") in this action. In its Amended Complaint, the State alleged (among other things) many of the same or similar allegations it had alleged in its Original Complaint (as described in Paragraph 5 of this Complaint) regarding Chemours' knowing discharges of GenX and other PFAS into the Cape Fear River, the toxic effects of PFAS on human health and the environment, the use of the river water as a public water supply source that serves residents and businesses in several counties, and the presence of PFAS discharged from the Chemours Facility to the public drinking water. The State also alleged in its Amended Complaint that: (a) it has obtained additional evidence of the extent of contamination caused by Chemours' release of PFAS into the environment [Paragraph 5]; (b) Chemours has identified the migration of groundwater from the Chemours Facility to the Cape Fear River as the most significant current source of contaminant loading in the river [Paragraph 126]; and (c) a major source of groundwater contamination, both onsite and offsite, is Chemours' air emissions [Paragraph 132].

17. On June 11, 2018, the State published a proposed Order for Preliminary Injunctive Relief for public comment. On July 10, 2018, CFPUA (through its counsel) provided written comments in response to the State's proposed order. The comments generally supported the preliminary relief sought by the State, but also requested revisions to the proposed order that would seek additional information and provide additional preliminary relief for the downstream water utilities.

18. On November 21, 2018, the day before Thanksgiving, DEQ announced on its website its proposal to enter into a proposed Consent Order ("PCO") with Chemours and Cape Fear River Watch (an environmental organization that also signed the PCO and that seeks to intervene in this action). *See* https://deq.nc.gov/news/press-releases/2018/11/21/release-state-officials-require-chemours-provide-permanent-drinking. DEQ's announcement states, "The proposed consent order is a comprehensive resolution regarding per- and polyfluoroalkyl substances (PFAS) contamination originating from Chemours' Fayetteville Works facility." The announcement also states that DEQ will accept public comment on the PCO until December 21, 2018.

19. The Cape Fear Public Utility Authority was unaware that the parties to this action had reached a proposed settlement or had agreed to propose a Consent Order until the PCO was published by DEQ on the day before Thanksgiving. CFPUA was not consulted about or notified of the status of the parties' settlement negotiations, the potential terms of a proposed settlement, or the impending publication of the PCO. DEQ did not seek input from CFPUA regarding how the terms of the proposed settlement might (or might not) provide relief to CFPUA and its customers.

20. Paragraph 12 of the PCO was targeted toward reducing PFAS loading to the Cape Fear River, which would theoretically reduce the PFAS entering CFPUA's raw water intake.

However, the PCO allowed for a five year implementation period with limited interim reductions. The PCO also included requirements that seek to reduce future discharges of PFAS pollutants from the Chemours Facility and to prevent current and future consumption of contaminated groundwater by citizens who live around the Facility and obtain potable water from water supply wells in the vicinity of the Facility. But the PCO did not include requirements to prevent the current and ongoing use or consumption of contaminated Cape Fear River water by downstream citizens and other users (including CFPUA) – even though the State acknowledges this harm, acknowledges CFPUA's current inability to remove these pollutants from Cape Fear River water, and requests relief for this harm in the State's complaints in this action.

21. On December 20, 2018, CFPUA filed a Motion to Intervene and calendared it for hearing on January 14, 2019, in light of the deficiencies in the proposed Consent Order. Following discussions with DEQ regarding the terms of the PCO, CFPUA agreed to remove its Motion to Intervene from the calendar but not withdraw the motion itself to allow the parties time to consider further improvements to the PCO. *See* Tr. of Hrg. on Mot. for Entry of Consent Order ("Hrg. Tr.") at 31.

22. Then, on February 20, 2019, counsel for DEQ notified counsel for CFPUA that DEQ, Chemours and the River Watch had agreed upon revised the terms of the PCO and had filed a motion for entry of a proposed Revised Consent Order ("Revised PCO" or "Consent Order"), to be heard five days later. CFPUA had not previously seen or been notified of the revised terms, nor was there time for CFPUA to advise the Board on the revised terms of the PCO being proposed or get board approval or disapproval to pursue its Motion to Intervene and so advised the Court. *See* Hrg. Tr. At 30–31. The Court did not rule on CFPUA's motion at the February 25 Hearing.

23. An improvement over the prior version, the Revised PCO provided for more protections to downstream users, such as interim benchmarks in the reduction of PFAS loading to the river. However, the Revised PCO still had the same fundamental deficiencies described above—it left CFPUA customers exposed to PFAS in their drinking water for years, while ensuring clean water for the citizens of Bladen County.

Deficiencies in the Revised Consent Order

24. One of the most significant aspects of the Revised Consent Order is the requirement for replacement water supplies, set forth in Section F. For fourteen PFAS identified on Attachment C, the Revised Consent Order established drinking water standards of 10 parts per trillion (ppt) for any individual PFAS, and 70 ppt for combined PFAS levels (the "Bladen County Limit"). Revised Consent Order ¶ 20. For persons whose water is contaminated in excess of the Bladen County Limit, Chemours is obligated to provide interim replacement water within three days of being notified, and permanent reverse osmosis systems within six months. Revised Consent Order ¶¶ 20 and 23.

25. Inexplicably, the Bladen County Limit only applies to groundwater users. The result is that Bladen County residents whose groundwater exceeds the Bladen County Limit standard receive near-immediate relief. Conversely, CFPUA and its customers, whose raw and finished water regularly exceed the Bladen County Limit standard, must wait years for clean water. This unequal treatment of North Carolina citizens that have suffered similar harm because of the actions and inactions of Chemours and DuPont is still unexplained and arbitrary and capricious.

26. The Revised Consent Order is based on a flawed premise. As justification for entry of the Revised PCO, DEQ and Chemours both assured this Court that the implementation of the provisions in the Revised PCO had reduced and would continue to reduce downstream PFAS

levels in the Cape Fear River. For instance, counsel for DEQ asserted that "[a]s a result of DEQ requiring cessation of the discharge of process wastewater, there were dramatic reductions in the concentrations of GenX in Chemours' discharge," and "similar reductions" in CFPUA's finished water. Hrg. Tr. at 8. Similarly, counsel for Chemours opined that the cessation of its PFAS-laden wastewater discharges "resulted in truly dramatic reductions in the levels of GenX in the river." Hrg. Tr. at 23. DEQ further emphasized to the Court that Paragraph 12 of the Revised PCO requires Chemours to demonstrate a plan to achieve the maximum feasible reduction in PFAS loading from the facility to the Cape Fear River, and was "of central importance for downstream communities." Hrg. Tr. at 14.

27. In other words, by turning off the PFAS spigot into the Cape Fear River that was Chemours' process wastewater in the first instance, and by requiring Chemours to study and then address PFAS loading from its facility to the Cape Fear River thereafter, DEQ theorized that PFAS levels in the river had dropped and would continue to drop in the immediate near term as it had in the prior 6 months..

28. The reality has not matched the representations made to the Court. As demonstrated by the continued monitoring of PFAS over the past 18 months, PFAS levels in the Cape Fear River have been variable and are largely dependent on river flows. PFAS in groundwater, surface water runoff, and sediment continues to migrate into the river from and around the Facility and from accumulated sediment in the Cape Fear River bed due to decades of contamination.

29. Accordingly, in the months preceding the February 2019 hearing, high river flows were largely responsible for the "dramatic reductions" in PFAS concentrations presented to the Court, rather than merely a matter of Chemours having halted its process wastewater discharges.

Following the hearing, PFAS levels in the Cape Fear River later increased significantly due to drier weather, rather than continuing their decline as was represented to the Court.

30. Chemours and DEQ both theorize that migration of groundwater from the Chemours Facility to the Cape Fear River is the most significant source of PFAS contamination in the river, which Chemours has yet to resolve. Am. Compl. ¶ 126. It is therefore no surprise that, of the 58 raw water sampling events since the hearing on the Revised PCO, 47 exceeded the Bladen County Limit. Of the 44 finished water samples, 32 exceeded this standard.

31. Further, the relief to CFPUA offered by the Revised Consent Order will not be realized for years, unlike the relief provided to Bladen County residents. The Order allows Chemours five years to implement a plan to reduce PFAS loading to the Cape Fear River from groundwater at the Facility. Consent Order ¶ 12.a.

32. As required by the Consent Order, Chemours submitted to DEQ a Cape Fear River PFAS Loading Reduction Plan on August 26, 2019, and the related Corrective Action Plan ("CAP") on December 31, 2019, detailing its proposals to remediate the groundwater at the Facility and reduce PFAS loading to the river. Under Chemours' own estimates (which CFPUA's consultant Tetra Tech has opined is not scientifically supported (*see* Exhibit D and Exhibit E, attached)), it will take through 2022 for them to control just 43% of the PFAS loading from their facility to the Cape Fear River. By the end of 2024, Chemours estimates it will have controlled just 79% of the current PFAS releases from its Facility to the river. The full extent of Chemours' proposed remedial actions are expected to take between 5 and 10 years, if not longer. All the while, the water of the Cape Fear River at CFPUA's intake regularly exceeds the Bladen County Limit.

33. Conversely, the Revised Consent Order requires that Chemours provide temporary replacement water supplies to the citizens of Bladen County within 3 days of becoming aware that an affected user's groundwater exceeds the Bladen County Limit, and a permanent replacement within 6 months. Consent Order ¶¶ 20 and 23.

34. Finally, the Revised Consent Order and Chemours' Loading Reduction Plan and CAP fall short of assuring adequate relief to CFPUA. As an initial matter, even assuming Chemours can meet its projections, its remedial actions would reduce PFAS loading from its Facility by just 79%. But Chemours itself acknowledges that its proposed long-term groundwater remedy is "still highly conceptual," and that "it is not presently possible to conclude with confidence whether this alternative is economically feasible." *See* CAP at 71, 74. Moreover, those plans do nothing to address the extensive soil, groundwater, and sediment contamination in the larger area surrounding the Facility and in the riverbed, which will continue releasing PFAS to the Cape Fear River for decades. Therefore, the PFAS Loading Reduction Plan and CAP represent a future and possible solution for the downstream water utilities.

35. As such, CFPUA and its customers will continue being subjected to river water contaminated with PFAS. And given the limits of the remediation proposed by Chemours, there is no assurance that even after its completion the water of the Cape Fear River will meet the Bladen County Limit. The only way to assure that CFPUA's finished water will meet that standard is to build a treatment system designed to remove PFAS, as CFPUA is doing.

36. Based on the evidence in this action and the studies arising from implementation of the Consent Order and Revised Consent Order, the State is aware that: (a) PFAS pollutants exist in the surface water in the Cape Fear River; (b) even if the Chemours Facility immediately ceases all emissions and discharges of PFAS pollutants into the Cape Fear River, those pollutants will

continue to contaminate the surface water in the Cape Fear River for decades to come (since pollutants in the vegetation, soils, and groundwater in a large and unknown radius around the Chemours Facility and in riverbed sediments will continue to migrate into the river water through groundwater flow and surface run-off); (c) Cape Fear River water is being used downstream from the Chemours Facility by CFPUA, customers of CFPUA, and other citizens; and (d) downstream utilities like CFPUA do not have the ability to consistently remove these pollutants from the drinking water supplied to their customers. Yet the State has left CFPUA to its own devices in dealing with the PFAS contamination in the river.

The Proposed Addendum Does Not Ensure Relief to CFPUA

37. Since the Revised Consent Order was entered, CFPUA has continued to share its monitoring results and concerns with DEQ, including its data showing that the high variability of PFAS concentrations in the Cape Fear River over time are largely dependent on the volume of flow. Chemours has conducted site assessments which indicate that the majority of PFAS loading from the Chemours Facility into the Cape Fear River is due to groundwater contamination. Chemours itself also calculated that it has been and is the primary contributor to PFAS in the Cape Fear River, estimating that Facility has been responsible for between 68% and 84% of PFAS concentrations at CFPUA's water intake in the river.

38. In its discussions with CFPUA, DEQ recognized that the Revised Consent Order in its current form is deficient. DEQ and Chemours now proposed additional modifications, releasing a draft Addendum to the Revised Consent Order for public comment on August 13, 2020 (the "proposed Addendum"). However, consistent with its past practice, DEQ again did not consult with CFPUA on the terms of the proposed Addendum, instead notifying CFPUA of its existence just hours before its public release.

39. The proposed Addendum requires concrete remediation directed toward limiting groundwater migration from the Chemours Facility into the Cape Fear River. Yet, as it relates to CFPUA and other downstream water users, the proposed Addendum still suffers from exactly the same major deficiency as the Revised Consent Order: There is still no assurance, or even reasonable expectation, that PFAS levels in the Cape Fear River will ever consistently reach the Bladen County Limit, and there is still no immediate relief for CFPUA or its customers. CFPUA is left in the same position as it was before the proposed Addendum: wait an indeterminate number of years for an indeterminate level of relief from an indeterminate number of PFAS compounds.

40. As part of the Addendum requirements and as described in the proposed NPDES Permit # NC 0089915 that was sent to public notice on July 10, 2020 ("proposed Permit"), DEQ is requiring Chemours to analyze for 59 distinct PFAS compounds found in Chemours' proposed wastewater discharge (the "Full Suite = Table 3+ Lab SOP +Method 537 Compounds"). This proposed Permit only covers stormwater and groundwater seepage in old outfall 2, and does not govern the main Chemours wastewater treatment plant discharge that is currently being captured and shipped offsite for disposal (which will be covered by a separate, subsequent permit). CFPUA has filed a written objection to DEQ's failure to place any controls over the remaining 56 PFAS compounds and the fact that the proposed Permit would allow a maximum daily discharge of the three regulated compounds (GenX, PFMOAA and PMPA) of 964 ppt.

41. In addition, in its June 30, 2020, PFAS Non-Targeted Analysis and Methods Interim Report on Process and Non-Process Wastewater and Stormwater, Chemours identified 21 new and previously unknown PFAS compounds in its "General Facility Discharge" and 250 new and unknown PFAS compounds in its "Process Wastewater."

42. The Secretaries' Science Advisory Board ("SAB") continues to review studies related to the appropriate health and regulatory standards for GenX and other PFAS compounds. At its August 31, 2020 meeting, the SAB reviewed studies by two groups of scientists (Exhibits F and G) on the probable toxicological impacts of GenX (and PFOA) on mice and rats and studies by another group of scientists (Exhibit H) on the probable toxicological impacts of BFESA-BP2 (Nafion Byproduct 2) on mice. Both of these PFAS compounds are found in significant amounts in Chemours' discharges and releases. And these are but two of the hundreds of PFAS compounds now identified as emanating from the Chemours Facility and likely entrained in the sediments of the riverbed. What health and environmental impacts these compounds will have individually and synergistically is going to take decades to determine.

43. Based on the evidence in this action and the studies undertaken since implementation of the Revised Consent Order, the State is aware that: (a) hundreds of PFAS pollutants exist in the surface water in the Cape Fear River; (b) even if the Chemours Facility immediately ceases all emissions and discharges of PFAS pollutants into the Cape Fear River and completes the remediation improvements now promised, those pollutants will continue to contaminate the surface water in the Cape Fear River for decades to come (because PFAS pollutants in the vegetation, soils, and groundwater in a large and unknown radius around the Chemours Facility will continue to migrate into the river water through groundwater flow and surface run-off and PFAS pollutants entrained in the Cape Fear River sediment will continue to be released into the river water); (c) Cape Fear River water is being used downstream from the Chemours Facility by CFPUA, customers of CFPUA, and other citizens; and (d) downstream utilities like CFPUA do not have the ability to consistently remove PFAS pollutants from the drinking water supplied to their customers without the construction of additional treatment

systems, such as the GAC system currently being constructed by CFPUA. Yet again, the State has left CFPUA to its own devices in dealing with current and reasonably foreseeable PFAS contamination in the river.

Mandatory abatement of violations under N.C. Gen. Stat. § 143-215.6C

44. As alleged in the State's Amended Complaint, the past and ongoing unpermitted discharges and releases of PFAS by Chemours violate the State laws implementing the Clean Water Act. Am. Compl. ¶¶ 145–164.

45. The State further alleged that North Carolina has the authority to take enforcement action against violations of the Clean Water Act and the implementing State laws, which prohibit the discharge of unpermitted pollutants. Am. Compl. ¶ 14.

46. Water from the Cape Fear River is withdrawn by CFPUA and treated in its treatment plant, and the treated water is then distributed to its customers for drinking and other public uses. The relevant stream segment of the Cape Fear River from which the water is withdrawn by CPFUA is classified WS-IV CA.

47. One State water quality standard applicable to all fresh surface waters is: "Oils, deleterious substances, colored, or other wastes: *only* such amounts as *shall not* render the waters injurious to public health, secondary recreation, or to aquatic life and wildlife, or adversely affect the palatability of fish, aesthetic quality, or impair the waters for any designated uses." 15A NCAC 2B .0211(12) (italics added). One designated use of class WS-IV surface water segments is "a source of water supply for drinking." 15A NCAC 2B .0216(1). The PFAS pollutants discharged and released into the Cape Fear River by Chemours and its predecessor: (a) are deleterious substances within the meaning of this water quality standard; (b) are present in the Cape Fear River

in amounts that render the Cape Fear River waters injurious to public health; and (c) are present in the Cape Fear River in amounts that impair the Cape Fear River waters for its designated use.

48. Under North Carolina's water quality laws implementing the Clean Water Act, DEQ is authorized to institute a civil action for injunctive relief to restrain and abate violations of the applicable water quality laws. N.C. Gen. Stat. § 143-215.6C. Upon a determination by the Court that an alleged violation "has occurred or is threatened, the court shall grant the relief necessary to prevent or abate the violation." *Id.* (emphasis added); Am. Compl. ¶ 46.

49. DEQ expressly brought the Amended Complaint under, *inter alia*, N.C. Gen. Stat. § 143-215.6C.

FIRST CLAIM FOR RELIEF
(Declaratory Judgment-Consent Order is Arbitrary and Capricious)

50. The allegations set forth in the preceding paragraphs are realleged and incorporated by reference.

51. Pursuant to the Declaratory Judgments Act, N.C. Gen. Stat. § 1-253 *et seq.*, and for the reasons stated above, CFPUA seeks an order declaring that the State's decision to settle this enforcement action on the terms stated in the Addendum to the Revised Consent Order is arbitrary and capricious and an abuse of discretion under the North Carolina Administrative Procedure Act.

52. *First*, the proposed Addendum to the Revised Consent Order fails to provide effective remedial requirements for off-site PFAS contamination in the Cape Fear River, river sediment, and air depositions in the soil and groundwater, which will continue to impact the waters of the Cape Fear River and the downstream users of the Cape Fear River for decades into the future. Instead, the State has left CFPUA and other downstream users to the uncertainties and expense of private litigation, to vindicate their rights on their own, and has thereby abandoned its

obligations to enforce the State's environmental laws (including the State's water quality standards) on behalf of all citizens of the State.

53. *Second*, the Addendum to the Revised Consent Order implicitly continues the established drinking water remedial requirements (to the Bladen County Limit) for residents in the vicinity of the Fayetteville Works Facility whose groundwater is impacted by PFAS, but does not establish the same requirements for everyone downstream whose drinking water is also impacted by the same PFAS contaminants. The State's decision to resolve this enforcement action in a manner that mandates unequal treatment of North Carolina citizens is arbitrary and capricious, irrational, and an abuse of discretion.

54. An actual controversy exists based on the State's decision not to fully address the immediate and continuing harms to CFPUA and its customers.

55. CFPUA has no adequate or effective administrative remedy against the State or its agency DEQ. The subject of this Complaint is the underlying historic and ongoing releases of PFAS by Chemours, the public health and environmental harms caused by those releases, and the State's efforts to seek relief for the violations of North Carolina water quality laws in this enforcement action. Jurisdiction to consider and determine the outcome of this action lies in Bladen County Superior Court, over which the Office of Administrative Hearings ("OAH") has no authority. Accordingly, there is no adequate administrative remedy available to CFPUA, an administrative claim in OAH would be futile, and this Court has jurisdiction to determine this action.

56. CFPUA seeks an order declaring that the State's decision to resolve this enforcement action pursuant to the terms of the Addendum to the Revised Consent Order is arbitrary and capricious, irrational, and an abuse of discretion under the North Carolina

Administrative Procedure Act since it (a) does not assure that the existing harm to downstream Cape Fear River water users is abated and (b) implicitly establishes differing and irrational levels of PFAS contamination that are safe for human consumption and use depending on whether a user's exposure to PFAS contaminants arises from use of surface water or groundwater.

SECOND CLAIM FOR RELIEF
(Declaratory Judgment–Equal Protection Violation)

57. The allegations set forth in the preceding paragraphs are realleged and incorporated by reference.

58. The Revised Consent Order and the Addendum thereto implicitly establishes two different sets of drinking water safety levels – one set (the Bladen County Limit) for residents in the vicinity of the Fayetteville Works Facility whose groundwater is impacted by PFAS, and a different set with higher or no levels for everyone downstream whose water is also impacted by PFAS, including CFPUA and its customers.

59. With regard to the safety of their drinking water supply, CFPUA and its customers are similarly situated to residents in the vicinity of the Fayetteville Works Facility who rely on potable water from water supply wells that are contaminated with PFAS, in that: (a) both groups of residents reside in the area of PFAS impact from the Fayetteville Works Facility; (b) both groups of residents rely on drinking water supplies contaminated with PFAS; (c) the drinking water used by both groups of residents has been contaminated by PFAS discharges and releases from the same Facility; and (d) without relief, the drinking water of both groups of residents will continue to be contaminated with PFAS for decades into the future.

60. While the Revised Consent Order requires Chemours to remediate or replace the water supply of nearby residents whose groundwater is contaminated with certain PFAS compounds above the Bladen County Limit, the Addendum and the Revised Consent Order

include no similar requirement for downstream users whose water supply is also contaminated with the same PFAS compounds from the same Facility.

61. The Revised Consent Order's (continued with the Addendum) disparate treatment of North Carolinians exposed to PFAS-contaminated drinking water supplies constitutes discrimination in that the Consent Order's protections do not apply equally to all similarly situated persons, do not reflect a rational distinction between such persons, and therefore, violate equal protection as guaranteed by the Equal Protection Clause of Article I, Section 19 of the North Carolina Constitution and the Equal Protection Clause of Section 1 of the Fourteenth Amendment to the United States Constitution.

62. Upon information and belief, the Revised Consent Order's (continued with the Addendum) distinctions between nearby and downstream groups of residents are not related to a legitimate purpose.

63. CFPUA seeks a judgment declaring that the Addendum and the Revised Consent Order constitute a violation of the United States and North Carolina Constitutions.

THIRD CLAIM FOR RELIEF
(Declaratory Judgment-Abatement of Violation)

64. The allegations set forth in the preceding paragraphs are realleged and incorporated by reference.

65. Under North Carolina's statutes and rules implementing the Clean Water Act, DEQ is authorized by N.C. Gen. Stat. § 143-215.6C to request the Attorney General to institute a civil action for injunctive relief to restrain and abate a violation of the State's water quality laws. Pursuant to this statute, the Attorney General instituted this enforcement action on behalf of the State. Upon a determination by the Court that the alleged violation "has occurred or is threatened,

the court shall grant the relief necessary to prevent or abate the violation." N.C. Gen. Stat. § 143-215.6C (emphasis added); Am. Compl. ¶ 46.

66. The Amended Complaint expressly seeks to enforce, and requests relief pursuant to, N.C. Gen. Stat. § 143-215.6C.

67. Although the Amended Complaint and the terms of the Consent Order are premised on violations of North Carolina's water quality laws by Chemours, which resulted in widespread PFAS contamination in the Cape Fear River, the Consent Order does not prevent or abate the violation. In particular, the Consent Order fails to provide effective relief for off-site PFAS contamination in the Cape Fear River, river sediment, air depositions, and possible future surface water discharges which will continue to impact the waters of the Cape Fear River and the downstream users of the Cape Fear River for decades into the future.

68. An actual controversy exists based on the State's failure to seek effective abatement of the violations of Chemours. As a result, the waters of the Cape Fear River will continue to be impacted by PFAS historically released by Chemours, in violation of North Carolina water quality laws, which will reach CFPUA's intake within the river and affect the quality of CFPUA's finished water, and thereby cause current and future harm to CFPUA and its customers.

69. The State's Amended Complaint alleges the basis for the Court's jurisdiction under N.C. Gen. Stat. § 143-215.6C, and the record shows that the facts alleged by the State will be proved by the evidence that will be presented in this case. However, the State's decision to seek to settle this enforcement action on the basis of the Revised Consent Order (continued with the Addendum) irrationally and arbitrarily fails or refuses to seek the "relief necessary to prevent or abate the violation[s]" alleged in the Amended Complaint. The Revised Consent Order (continued with the Addendum) irrationally and arbitrarily and without justification precludes the Court from

entering the "relief necessary" as required by the enforcement statute under which this action was instituted.

70. Pursuant to the Declaratory Judgments Act, N.C. Gen. Stat. § 1-253 *et seq.*, and for the reasons stated above, CFPUA seeks an order of the Court declaring that: (a) the statutory and regulatory violations alleged by the State in this action have occurred or are threatened; and (b) the Revised Consent Order (continued with the Addendum) fails to meet the mandate of N.C. Gen. Stat. § 143-215.6C, to prevent or abate the violations of North Carolina's water quality laws and rules by Chemours; and (c) the State's decision to agree to the Revised Consent Order (continued with the Addendum) is irrational, arbitrary, and unsupported by the record in this case because the Revised Consent Order (continued with the Addendum) does not seek or accomplish the "relief necessary to prevent or abate the violation" and, if allowed by the Court as agreed-to by the State, would prevent the grant of the "relief necessary" as required by N.C. Gen. Stat. § 143-215.6C.

PRAYER FOR RELIEF

WHEREFORE, Intervernor CFPUA respectfully prays the Court for the following relief:

1. A judicial declaration, pursuant to N.C. Gen. Stat. § 1-253 *et seq.*, that the State's decision to agree to the Revised Consent Order (continued with Addendum) was arbitrary and capricious;

2. A judicial declaration, pursuant to N.C. Gen. Stat. § 1-253 *et seq.*, that the Revised Consent Order (continued with Addendum) violates the Equal Protection Clause of Article I, Section 19 of the North Carolina Constitution and the Equal Protection Clause of Section 1 of the Fourteenth Amendment to the United States Constitution to the extent it arbitrarily and irrationally

treats similarly situated citizens differently for purposes of addressing and abating PFAS discharges or releases to drinking water;

3. A judicial declaration and determination, pursuant to N.C. Gen. Stat. § 1-253 *et seq*., that: (a) the statutory and regulatory violations alleged by the State in this action have occurred or are threatened; and (b) the Revised Consent Order (continued with the Addendum) fails to meet the mandate of N.C. Gen. Stat. § 143-215.6C, to prevent and abate the violations of North Carolina's water quality laws and rules by Chemours; and (c) the State's decision to agree to the Revised Consent Order (continued with the Addendum) is irrational, arbitrary, and unsupported by the record in this case because the Revised Consent Order (continued with the Addendum) does not seek or accomplish the "relief necessary to prevent or abate the violation" and, if allowed by the Court as agreed-to by the State, would prevent the grant of the "relief necessary" as required by N.C. Gen. Stat. § 143-215.6C.

4. An order, following the trial of this case and pursuant to N.C. Gen. Stat. § 143-215.6C, granting the relief necessary to prevent and abate Chemours' violations of the water quality laws of this State;

6. Such other and further relief as to the Court may seem just and proper.

Respectfully submitted this the _____ day of _____, 2019.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT A
TO AMENDED INTERVENOR COMPLAINT

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	9/11/2018	ND	ND	ND	17.5	ND	ND	ND	ND	ND	ND	ND	12.8	ND	ND	ND	ND	67.7	167
Sweeney Raw	9/12/2018	ND	ND	ND	16.9	ND	ND	ND	ND	ND	ND	ND	1.7	ND	ND	ND	ND	3.41	10.9
Sweeney Raw	9/14/2018	ND	ND	ND	15.5	ND	ND	ND	ND	ND	ND	ND	3.29	ND	ND	ND	ND	14.6	43.4
Sweeney Raw	9/15/2018	ND	ND	ND	18.8	ND	ND	ND	ND	ND	ND	ND	1.85	ND	ND	ND	ND	5.84	22.4
Sweeney Raw	9/16/2018	ND	ND	ND	15.2	ND	ND	ND	ND	ND	ND	ND	1.97	ND	ND	ND	ND	8.67	28
Sweeney Raw	9/17/2018	ND	ND	ND	33.8	ND	ND	ND	ND	ND	ND	1.33	2.35	ND	ND	ND	ND	3.19	7.35
Sweeney Raw	9/18/2018	ND	ND	ND	18.6	ND	ND	ND	ND	ND	ND	ND	1.34	ND	ND	ND	ND	2.32	5.66
Sweeney Raw	9/19/2018	ND	ND	ND	17.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.77	4.43
Sweeney Finished	9/19/2018	ND	ND	ND	19.8	ND	ND	ND	ND	ND	ND	ND	9.51	ND	ND	ND	ND	51.9	120
Sweeney Raw	9/20/2018	ND	ND	ND	18.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.16	5.14
Sweeney Raw	9/21/2018	ND	ND	ND	12.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.67	5.28
Sweeney Raw	9/22/2018	ND	ND	ND	8.44	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.09	5.9
Sweeney Raw	9/23/2018	ND	ND	ND	5.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.96	3.22
Sweeney Raw	9/24/2018	ND	ND	ND	6.32	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.88
Sweeney Finished	9/24/2018	ND	ND	ND	7.34	ND	ND	ND	ND	ND	ND	ND	6.8	ND	ND	ND	ND	37.9	96.4
Sweeney Raw	9/25/2018	ND	ND	ND	8.54	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.25	3.63
Sweeney Raw	9/26/2018	ND	ND	ND	15.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.66	7.87
Sweeney Raw	9/27/2018	ND	ND	ND	17.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.05	5.14
Sweeney Raw	9/28/2018	ND	ND	ND	27.7	ND	ND	ND	ND	ND	ND	ND	1.68	ND	ND	ND	ND	3.95	9.97
Sweeney Raw	9/29/2018	ND	ND	ND	25.1	ND	ND	ND	ND	ND	ND	ND	1.4	ND	ND	ND	ND	3.46	8.66
Sweeney Raw	9/30/2018	ND	ND	ND	12.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.69	4.6
Sweeney Raw	10/1/2018	ND	ND	ND	10.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.2	4.37
Sweeney Finished	10/1/2018	ND	ND	ND	16.9	ND	ND	ND	ND	ND	ND	ND	10.7	ND	ND	ND	ND	56.1	125
Sweeney Raw	10/2/2018	ND	ND	ND	9.79	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.74	ND
Sweeney Raw	10/3/2018	ND	ND	ND	11.7	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.3	3.66
Sweeney Raw	10/4/2018	ND	ND	ND	10.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.5
Sweeney Raw	10/5/2018	ND	ND	ND	11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.2	3.77
Sweeney Raw	10/6/2018	ND	ND	ND	10.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.56	4.74
Sweeney Raw	10/7/2018	ND	ND	ND	10.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.22	4.63

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	9/11/2018	126	139	9.5	3.76	5.25	11.9	ND	2.53	ND	ND	16.3	ND	6.95	23.3	ND	2.85		ND	20.1	12.2
Sweeney Raw	9/12/2018	13.4	14.3	4.47	7.29	5.44	10.7	ND	1.43	ND	ND	12.5	ND	7.55	22.3	ND	2.28		ND	17.8	9.08
Sweeney Raw	9/14/2018	43.9	51.5	ND	2.83	4.98	11.7	ND	1.57	ND	ND	13.6	ND	7.05	22.9	ND	2.18		ND	17.7	10.5
Sweeney Raw	9/15/2018	25.5	26.3	5.14	5.37	4.77	12.2	ND	1.7	ND	ND	17.9	ND	6.43	28.9	ND	2.53		ND	16.2	12.1
Sweeney Raw	9/16/2018	28.1	33.4	ND	9.7	4.37	9.04	ND	1.44	ND	ND	15.1	ND	5.63	23.5	ND	1.64		ND	13.5	8.81
Sweeney Raw	9/17/2018	11.2	13.9	7.61	13.8	2.54	6.59	ND	1.07	ND	ND	9.27	ND	3.71	14.2	ND	0.831		ND	9.72	5.54
Sweeney Raw	9/18/2018	9.06	10.6	ND	ND	1.96	5.8	ND	0.683	ND	ND	6.48	ND	2.87	9.8	ND	ND		ND	7.83	4.41
Sweeney Raw	9/19/2018	6.94	9.9	ND	ND	1.52	4.73	ND	ND	ND	ND	2.97	ND	2.59	5.2	ND	0.777		ND	6.45	3.35
Sweeney Finished	9/19/2018	93.9	104	11.8	7.62	2.66	6.51	ND	2.23	ND	ND	8.68	ND	4	12.4	ND	1.99		ND	13.4	6.43
Sweeney Raw	9/20/2018	8.44	10.2	ND	ND	1.69	5.1	ND	0.693	ND	ND	2.99	ND	2.62	5.17	ND	0.824		ND	7.03	3.34
Sweeney Raw	9/21/2018	7.05	10.7	10.2	ND	1.99	5.08	ND	0.736	ND	ND	3	ND	3.27	5.14	ND	0.999		ND	7.36	3.2
Sweeney Raw	9/22/2018	6.89	6.82	ND	ND	1.5	4.86	ND	ND	ND	ND	2.48	ND	1.66	3.83	ND	0.973		ND	7.97	3.23
Sweeney Raw	9/23/2018	4.59	5.73	ND	ND	1.43	4.12	ND	ND	ND	ND	1.73	ND	2.16	2.49	ND	0.996		ND	6.42	3.23
Sweeney Raw	9/24/2018	4.96	4.05	ND	8.71	1.73	5.39	ND	ND	ND	ND	2.06	ND	2.07	4.05	ND	0.914		ND	7.31	3.39
Sweeney Finished	9/24/2018	59.5	69.7	8.63	ND	1.75	4.27	ND	1.68	ND	ND	3.52	ND	3.52	3.53	ND	1.84		ND	10.2	5.43
Sweeney Raw	9/25/2018	6.31	6.7	3.35	ND	1.53	6.79	ND	ND	ND	ND	2.1	ND	1.97	5.32	ND	1.07		ND	7.15	3.38
Sweeney Raw	9/26/2018	13.4	13.7	8.6	24.8	1.79	10.3	ND	ND	ND	ND	3.37	ND	1.97	7.1	ND	1.03		ND	7.47	4.28
Sweeney Raw	9/27/2018	12.6	16	2.12	21	1.75	6.93	ND	ND	ND	ND	2.56	ND	2.36	3.84	ND	0.926		ND	7.59	3.95
Sweeney Raw	9/28/2018	18.9	21.2	12.2	ND	2.43	10.2	ND	ND	ND	ND	3	ND	3.96	5.68	ND	0.744		ND	10.4	6.12
Sweeney Raw	9/29/2018	18.6	17.5	9.19	9.31	2.74	9.95	ND	ND	ND	ND	3.34	ND	4.29	6.33	ND	1.02		ND	10.6	6.13
Sweeney Raw	9/30/2018	9.26	7.53	5.32	14.3	2.64	7.49	ND	0.966	ND	ND	5.36	ND	4.81	8.74	ND	1.4		ND	14.6	6.14
Sweeney Raw	10/1/2018	7.05	8.03	4.82	10.3	2.82	7.35	ND	1.15	ND	ND	7.63	ND	4.34	13.6	ND	1.68		ND	16	7.05
Sweeney Finished	10/1/2018	81.6	92.8	17	5.28	2.93	9.12	ND	1.37	ND	ND	6.31	ND	5.26	9.19	ND	2		ND	15.8	8.51
Sweeney Raw	10/2/2018	4.93	7.02	5.32	17.1	2.85	9.1	ND	1.08	ND	ND	7.95	ND	4.25	13.7	ND	1.39		ND	14.2	7.17
Sweeney Raw	10/3/2018	4.93	7.19	4.81	17.3	2.89	10.1	ND	1.37	ND	ND	9.66	ND	3.89	13	ND	1.55		ND	14.9	6.96
Sweeney Raw	10/4/2018	5.45	6.83	4.15	21.7	3.31	9.97	ND	1.46	ND	ND	9.95	ND	4.25	15.4	ND	1.76		ND	14.7	8.17
Sweeney Raw	10/5/2018	4.23	8.27	4.59	25.3	2.61	10.2	ND	1.33	ND	ND	10	ND	3.75	15.2	ND	1.81		ND	14.8	7.43
Sweeney Raw	10/6/2018	5.29	7.77	4.58	23.6	3.34	10.9	ND	1.59	ND	ND	12.2	ND	3.9	18.6	ND	1.83		ND	16	9.59
Sweeney Raw	10/7/2018	5.63	6.17	3.9	15.9	3.54	12	ND	1.63	ND	ND	14.3	ND	4.52	23.2	ND	2.06		ND	16.8	9.89

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	9/11/2018	1.03	25.9	ND	ND	0.665		ND	**672.24**	**559.56**	**83**
Sweeney Raw	9/12/2018	1.33	27.8	ND	ND	0.58		ND	**191.16**	**84.87**	**44**
Sweeney Raw	9/14/2018	1.1	29	ND	ND	ND		ND	**297.30**	**188.62**	**63**
Sweeney Raw	9/15/2018	1.21	32	ND	ND	ND		ND	**247.14**	**129.10**	**52**
Sweeney Raw	9/16/2018	0.833	24.2	ND	ND	ND		ND	**233.10**	**140.14**	**60**
Sweeney Raw	9/17/2018	0.602	15.3	ND	ND	ND		ND	**163.90**	**103.80**	**63**
Sweeney Raw	9/18/2018	ND	9.85	ND	ND	ND		ND	**97.26**	**54.06**	**56**
Sweeney Raw	9/19/2018	ND	5.48	ND	ND	ND		ND	**74.71**	**44.61**	**60**
Sweeney Finished	9/19/2018	0.632	14	ND	ND	ND		ND	**491.46**	**427.21**	**87**
Sweeney Raw	9/20/2018	ND	5.55	ND	ND	ND		ND	**79.85**	**47.83**	**60**
Sweeney Raw	9/21/2018	ND	5.46	ND	ND	ND		ND	**83.74**	**50.50**	**60**
Sweeney Raw	9/22/2018	ND	4.03	ND	ND	ND		ND	**60.67**	**32.62**	**54**
Sweeney Raw	9/23/2018	ND	3.69	ND	ND	ND		ND	**46.88**	**22.34**	**48**
Sweeney Raw	9/24/2018	ND	4.2	ND	ND	ND		ND	**58.03**	**28.98**	**50**
Sweeney Finished	9/24/2018	ND	5.3	ND	ND	ND		ND	**327.31**	**289.79**	**89**
Sweeney Raw	9/25/2018	ND	8.08	ND	ND	ND		ND	**67.17**	**31.88**	**47**
Sweeney Raw	9/26/2018	ND	16.1	ND	ND	ND		ND	**140.34**	**90.30**	**64**
Sweeney Raw	9/27/2018	ND	7.3	ND	ND	ND		ND	**113.62**	**78.97**	**70**
Sweeney Raw	9/28/2018	0.787	9.15	ND	ND	ND		ND	**148.07**	**98.60**	**67**
Sweeney Raw	9/29/2018	ND	6.82	ND	ND	ND		ND	**144.44**	**96.56**	**67**
Sweeney Raw	9/30/2018	0.936	8.08	ND	ND	ND		ND	**116.26**	**60.46**	**52**
Sweeney Raw	10/1/2018	0.661	9.11	ND	ND	ND		ND	**117.46**	**53.70**	**46**
Sweeney Finished	10/1/2018	0.792	9.2	ND	ND	ND		ND	**475.86**	**411.69**	**87**
Sweeney Raw	10/2/2018	0.657	13.6	ND	ND	ND		ND	**121.85**	**53.85**	**44**
Sweeney Raw	10/3/2018	0.596	13.2	ND	ND	ND		ND	**129.01**	**60.55**	**47**
Sweeney Raw	10/4/2018	0.639	15.4	ND	ND	ND		ND	**137.04**	**61.98**	**45**
Sweeney Raw	10/5/2018	0.637	14.7	ND	ND	ND		ND	**141.83**	**69.36**	**49**
Sweeney Raw	10/6/2018	0.686	18.4	ND	ND	ND		ND	**155.18**	**70.34**	**45**
Sweeney Raw	10/7/2018	0.699	20.5	ND	ND	ND		ND	**156.79**	**61.95**	**40**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Raw	10/8/2018	ND	ND	ND	11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.55	ND
Sweeney Finished	10/8/2018	ND	ND	ND	11.2	ND	ND	ND	ND	ND	ND	ND	9.91	ND	ND	ND	ND	52.3	117
Sweeney Raw	10/9/2018	ND	ND	ND	10.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.83	4.49
Sweeney Raw	10/10/2018	ND	ND	ND	9.94	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.33	ND
Sweeney Raw	10/11/2018	ND	ND	ND	9.58	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.02	4.53
Sweeney Raw	10/12/2018	ND	ND	ND	10.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.29	4.47
Sweeney Finished	10/15/2018	ND	ND	ND	19.4	ND	ND	ND	ND	ND	ND	ND	10	ND	ND	ND	ND	61.3	131
Sweeney Finished	10/23/2018	ND	ND	ND	8.39	ND	ND	ND	ND	2.22	ND	ND	6.14	ND	ND	ND	ND	28.7	77.9
Sweeney Finished	10/31/2018	ND	ND	ND	8.7	ND	ND	ND	ND	ND	ND	ND	4.48	ND	ND	ND	ND	23	48.4
Sweeney Finished	11/5/2018	ND	ND	ND	9.68	ND	ND	ND	ND	ND	ND	ND	4.54	ND	ND	ND	ND	20.6	50.4
Sweeney Finished	11/13/2018	ND	ND	ND	9.30	ND	ND	ND	ND	ND	ND	ND	4.65	ND	ND	ND	ND	28.7	55.8
Sweeney Finished	11/19/2018	ND	ND	ND	5.46	ND	ND	ND	ND	ND	ND	ND	3.06	ND	ND	ND	ND	13.8	28.1
Sweeney Raw	11/20/2018	ND	ND	ND	3.96	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.02
Sweeney Finished	11/21/2018	ND	ND	ND	3.57	ND	ND	ND	ND	ND	ND	ND	3.0	ND	ND	ND	ND	10.5	26.8
Sweeney Raw	11/27/2018	ND	ND	ND	12.0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.59
Sweeney Finished	11/28/2018	ND	ND	ND	8.24	ND	ND	ND	ND	ND	ND	ND	2.9	ND	ND	ND	ND	8.69	21.7
Sweeney Raw	12/3/2018	ND	ND	ND	6.93	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	12/4/2018	ND	ND	ND	4.44	ND	ND	ND	ND	ND	ND	ND	2.0	ND	ND	ND	ND	7.24	19.8
Sweeney Raw	12/10/2018	ND	ND	ND	16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.38
Sweeney Finished	12/11/2018	ND	ND	ND	8.71	ND	ND	ND	ND	ND	ND	ND	2.7	ND	ND	ND	ND	10.2	23.5
Sweeney Raw	12/17/2018	ND	ND	ND	25.6	ND	ND	ND	ND	ND	ND	ND	1.39	ND	ND	ND	ND	1.2	ND
Sweeney Finished	12/18/2018	ND	ND	ND	9.53	ND	ND	ND	ND	ND	ND	ND	1.9	ND	ND	ND	ND	6.19	15.1
Sweeney Raw	12/24/2018	ND	ND	ND	5.46	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	12/26/2018	ND	ND	ND	2.82	ND	ND	ND	ND	ND	ND	ND	1.99	ND	ND	ND	ND	7.35	17.5
Sweeney Raw	12/31/2018	ND	ND	ND	12.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	1/2/2019	ND	ND	ND	4.08	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	10.1	22.4
Sweeney Raw	1/7/2019	ND	ND	ND	6.92	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.87
Sweeney Finished	1/8/2019	ND	ND	ND	3.98	ND	ND	ND	ND	ND	ND	ND	2.2	ND	ND	ND	ND	8.07	18.7
Sweeney Raw	1/14/2019	ND	ND	ND	6.83	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.34

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Raw	10/8/2018	5.23	7.69	4.15	ND	3.77	12.9	ND	1.57	ND	ND	16.4	ND	3.88	25.1	ND	2.13		ND	17.2	8.97
Sweeney Finished	10/8/2018	49.4	81.5	5.28	ND	3.63	13.7	ND	2.22	ND	ND	15.2	ND	5.54	23.5	ND	2.51		ND	17	10.8
Sweeney Raw	10/9/2018	5.57	6.15	4.21	ND	3.67	10.6	ND	1.67	ND	ND	16.8	ND	4.35	26.2	ND	1.86		ND	19.1	10.3
Sweeney Raw	10/10/2018	4.32	5.42	4.01	ND	4.02	9.46	ND	1.68	ND	ND	17.3	ND	4.64	27.3	ND	2.15		ND	18.2	11.6
Sweeney Raw	10/11/2018	4.54	6.27	2.95	ND	3.72	10.4	ND	1.8	ND	ND	16.8	ND	5.0	26	ND	2.14		ND	16.8	10.9
Sweeney Raw	10/12/2018	8.25	9.81	4.87	ND	3.73	10.1	ND	1.92	ND	ND	16.8	ND	4.67	25.4	ND	2.27		ND	18	10.4
Sweeney Finished	10/15/2018	61.3	82.4	10.2	ND	4.85	12.7	ND	2.71	ND	ND	19.3	ND	6.44	27.4	ND	3.34		ND	20.9	14.3
Sweeney Finished	10/23/2018	38.6	47.6	5.35	ND	3.93	10.7	ND	1.94	ND	ND	15.1	ND	5.51	22.8	ND	2.8		ND	19.3	11.6
Sweeney Finished	10/31/2018	25.5	30.7	5.2	ND	4.66	14	ND	2.24	ND	ND	24	ND	6.13	34.6	ND	2.73		ND	18.8	13.7
Sweeney Finished	11/5/2018	27	33.8	6.07	ND	3.8	8.83	ND	2.06	ND	ND	15.4	ND	5.12	21.5	ND	2.39		ND	15.4	11.4
Sweeney Finished	11/13/2018	57.7	70	4.58	ND	3.01	6.24	ND	1.49	ND	ND	8.43	ND	3.92	12.7	ND	1.75		ND	12.8	7.81
Sweeney Finished	11/19/2018	31.9	49.2	ND	ND	1.75	9.33	ND	1.03	ND	ND	4.75	ND	2.23	7.7	ND	1.25		ND	7.7	4.56
Sweeney Raw	11/20/2018	4.65	5.21	ND	ND	1.64	ND	ND	ND	ND	ND	2.55	ND	2.05	3.41	ND	0.89		ND	5.45	3.14
Sweeney Finished	11/21/2018	29.8	36.3	3.0	ND	1.35	ND	ND	0.738	ND	ND	2.69	ND	1.85	3.71	ND	1.12		ND	6.64	3.56
Sweeney Raw	11/27/2018	6.14	12.3	5.48	ND	3.0	3.86	ND	0.711	ND	ND	7.53	ND	3.75	9.46	ND	1.13		ND	12.6	7.5
Sweeney Finished	11/28/2018	32.8	39.8	5.2	ND	2.0	3.52	ND	0.799	ND	ND	5.36	ND	3.0	7.88	ND	1.13		ND	8.22	5.18
Sweeney Raw	12/3/2018	4.21	6.34	ND	ND	2.8	5.0	ND	0.854	ND	ND	10.7	ND	3.01	12.9	ND	1.16		ND	12.8	7.36
Sweeney Finished	12/4/2018	18.2	24.5	2.1	ND	1.67	4.11	ND	ND	ND	ND	6.01	ND	2.23	9.16	ND	0.858		ND	7.24	4.94
Sweeney Raw	12/10/2018	11.7	15.3	ND	13.6	2.54	5.36	ND	0.807	ND	ND	8.88	ND	3.19	11.6	ND	1.41		ND	9.44	6.41
Sweeney Finished	12/11/2018	23.3	35.5	7.17	ND	1.7	3.79	ND	0.657	ND	ND	6.23	ND	1.99	9.02	ND	1.22		ND	6.4	4.54
Sweeney Raw	12/17/2018	9.33	15.3	9.99	ND	1.73	4.09	ND	ND	ND	ND	3.17	ND	2.81	4.79	ND	0.749		ND	6.67	4.01
Sweeney Finished	12/18/2018	17.9	24	5.2	ND	1.07	ND	ND	ND	ND	ND	2.09	ND	1.75	3.33	ND	0.617		ND	4.57	2.69
Sweeney Raw	12/24/2018	3.09	5.98	ND	ND	2.29	ND	ND	ND	ND	ND	3.87	ND	3.85	5.38	ND	0.91		ND	11.7	5.73
Sweeney Finished	12/26/2018	22	22.7	2.62	ND	0.717	1.31	ND	ND	ND	ND	1.44	ND	1.35	2.42	ND	ND		ND	3.96	2.08
Sweeney Raw	12/31/2018	6.3	8.87	ND	ND	2.65	3.97	ND	0.736	ND	ND	6.21	ND	3.45	8.56	ND	1.09		ND	13	7.09
Sweeney Finished	1/2/2019	28.9	31.7	8.34	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	5.01	ND	ND		ND	6.47	3.37
Sweeney Raw	1/7/2019	5.58	6.33	3.38	ND	2.55	4.62	ND	ND	ND	ND	7.08	ND	3.73	9.83	ND	0.826		ND	12.6	7.05
Sweeney Finished	1/8/2019	28.8	28.3	3.7	1.23	1.31	3.38	ND	ND	ND	ND	3.93	ND	1.77	5.25	ND	0.962		ND	7.98	4.61
Sweeney Raw	1/14/2019	5.59	8.19	ND	ND	2.64	6.53	ND	ND	ND	ND	7.58	ND	3.71	10.8	ND	1.23		ND	11.8	6.78

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Raw	10/8/2018	0.703	22.1	ND	ND	ND		ND	**144.34**	**46.02**	**32**
Sweeney Finished	10/8/2018	0.902	20.6	ND	ND	ND		ND	**442.19**	**341.79**	**77**
Sweeney Raw	10/9/2018	0.929	24.8	ND	ND	ND		ND	**152.73**	**49.25**	**32**
Sweeney Raw	10/10/2018	0.875	25	ND	ND	ND		ND	**147.25**	**42.32**	**29**
Sweeney Raw	10/11/2018	0.814	22.7	ND	ND	ND		ND	**146.96**	**46.69**	**32**
Sweeney Raw	10/12/2018	0.818	20.5	ND	ND	ND		ND	**155.10**	**57.29**	**37**
Sweeney Finished	10/15/2018	1.09	26.9	ND	ND	ND		ND	**515.53**	**394.90**	**77**
Sweeney Finished	10/23/2018	0.867	20.8	ND	ND	0.679		ND	**330.93**	**227.78**	**69**
Sweeney Finished	10/31/2018	0.919	31.4	ND	ND	ND		ND	**299.16**	**169.98**	**57**
Sweeney Finished	11/5/2018	0.893	21.1	ND	ND	ND		ND	**259.98**	**167.49**	**64**
Sweeney Finished	11/13/2018	0.669	13.5	ND	ND	ND		ND	**303.05**	**239.16**	**79**
Sweeney Finished	11/19/2018	ND	7.97	ND	ND	ND		ND	**179.79**	**136.27**	**76**
Sweeney Raw	11/20/2018	ND	3.98	ND	ND	ND		ND	**39.95**	**19.39**	**49**
Sweeney Finished	11/21/2018	ND	4.7	ND	ND	ND		ND	**139.32**	**115.65**	**83**
Sweeney Raw	11/27/2018	0.696	8.55	ND	ND	ND		ND	**97.30**	**46.04**	**47**
Sweeney Finished	11/28/2018	ND	6.48	ND	ND	ND		ND	**162.84**	**124.67**	**77**
Sweeney Raw	12/3/2018	0.613	11.2	ND	ND	ND		ND	**85.85**	**28.18**	**33**
Sweeney Finished	12/4/2018	ND	7.55	ND	ND	ND		ND	**121.99**	**84.23**	**69**
Sweeney Raw	12/10/2018	ND	12.3	ND	ND	ND		ND	**122.92**	**69.86**	**57**
Sweeney Finished	12/11/2018	ND	8.53	ND	ND	ND		ND	**155.16**	**117.31**	**76**
Sweeney Raw	12/17/2018	ND	4.94	ND	ND	ND		ND	**95.77**	**65.98**	**69**
Sweeney Finished	12/18/2018	ND	3.46	ND	ND	ND		ND	**99.40**	**81.91**	**82**
Sweeney Raw	12/24/2018	0.646	6.13	ND	ND	ND		ND	**55.04**	**18.40**	**33**
Sweeney Finished	12/26/2018	ND	2.06	ND	ND	ND		ND	**92.32**	**78.42**	**85**
Sweeney Raw	12/31/2018	0.632	7.83	ND	ND	ND		ND	**83.19**	**34.18**	**41**
Sweeney Finished	1/2/2019	ND	4.06	ND	ND	ND		ND	**124.43**	**105.52**	**85**
Sweeney Raw	1/7/2019	ND	8.38	ND	ND	ND		ND	**80.75**	**31.16**	**39**
Sweeney Finished	1/8/2019	ND	4.99	ND	ND	ND		ND	**129.16**	**98.91**	**77**
Sweeney Raw	1/14/2019	ND	9.51	ND	ND	ND		ND	**82.53**	**29.53**	**36**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	1/15/2019	ND	ND	ND	4.58	ND	ND	ND	ND	ND	ND	ND	1.83	ND	ND	ND	ND	9.18	17
Sweeney Raw	1/21/2019	ND	ND	ND	9.69	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.03
Sweeney Finished	1/22/2019	ND	ND	ND	6.02	ND	ND	ND	ND	ND	ND	ND	1.43	ND	ND	ND	ND	5.91	15.1
Sweeney Raw	1/28/2019	ND	ND	ND	4.31	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.35
Sweeney Finished	1/29/2019	ND	ND	ND	3.54	ND	ND	ND	ND	ND	ND	ND	1.48	ND	ND	ND	ND	6.45	15.7
Sweeney Raw	2/4/2019	ND	ND	ND	11.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.72	5.46
Sweeney Finished	2/5/2019	ND	ND	ND	7.2	ND	ND	ND	ND	ND	ND	ND	1.86	ND	ND	ND	ND	7.63	18.1
Sweeney Raw	2/11/2019	ND	ND	ND	19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.27	4.75
Sweeney Finished	2/12/2019	ND	ND	ND	11.5	ND	ND	ND	ND	ND	ND	ND	2.34	ND	ND	ND	ND	7.17	21
Sweeney Raw	2/18/2019	ND	ND	ND	11.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.5	3.73
Sweeney Finished	2/19/2019	ND	ND	ND	10.6	ND	ND	ND	ND	ND	ND	ND	2.05	ND	ND	ND	ND	8.24	22.8
Sweeney Raw	2/25/2019	ND	ND	ND	4.08	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	2/26/2019	ND	ND	ND	3.49	ND	ND	ND	ND	ND	ND	ND	1.87	ND	ND	ND	ND	8.36	19.9
Sweeney Raw	3/4/2019	ND	ND	ND	8.59	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.61
Sweeney Finished	3/5/2019	ND	ND	ND	5.28	ND	ND	ND	ND	ND	ND	ND	1.82	ND	ND	ND	ND	7.25	17.2
Sweeney Raw	3/11/2019	ND	ND	ND	6.75	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.63
Sweeney Finished	3/12/2019	ND	ND	ND	5.50	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.32
Sweeney Raw	3/18/2019	ND	ND	ND	7.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.67
Sweeney Finished	3/19/2019	ND	ND	ND	5.25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.17
Sweeney Raw	3/25/2019	ND	ND	ND	3.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	3/26/2019	ND	ND	ND	3.35	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.70
Sweeney Raw	4/1/2019	ND	ND	ND	7.90	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.87
Sweeney Finished	4/2/2019	ND	ND	ND	3.81	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.27
Sweeney Raw	4/8/2019	ND	ND	ND	18.70	ND	ND	ND	ND	ND	ND	ND	4.17	1.46	ND	ND	ND	1.52	4.48
Sweeney Finished	4/9/2019	ND	ND	ND	9.18	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.91
Sweeney Raw	4/15/2019	ND	ND	ND	8.39	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.78
Sweeney Finished	4/16/2019	ND	ND	ND	2.87	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.86
Sweeney Raw	4/22/2019	ND	ND	ND	18.0	ND	ND	ND	ND	ND	ND	ND	ND	1.48	ND	ND	ND	1.78	5.84
Sweeney Finished	4/23/2019	ND	ND	ND	7.22	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.43

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	1/15/2019	24.4	22	5.66	1.64	1.52	4.26	ND	0.638	ND	ND	5.32	ND	1.99	8.64	ND	0.837		ND	6.23	4.72
Sweeney Raw	1/21/2019	8.39	11.1	13.1	ND	2.16	4.61	ND	0.593	ND	ND	5.26	ND	2.73	8.11	ND	0.952		ND	8.92	6.68
Sweeney Finished	1/22/2019	21	24.6	4.6	ND	1.28	3.18	ND	ND	ND	ND	3.21	ND	1.88	5.27	ND	0.813		ND	5.67	3.38
Sweeney Raw	1/28/2019	3.69	5.08	9.74	ND	2.51	3.67	ND	ND	ND	ND	7.91	ND	3.7	10.2	ND	0.953		ND	10.4	6.74
Sweeney Finished	1/29/2019	20.1	24.3	6.88	ND	1.12	2.66	ND	ND	ND	ND	3.46	ND	1.51	5.41	ND	0.697		ND	4.31	3.18
Sweeney Raw	2/4/2019	10.7	16.9	4.25	6.17	2.28	ND	ND	ND	ND	ND	5.6	ND	3.18	7.7	ND	0.972		ND	11.1	6.06
Sweeney Finished	2/5/2019	21.7	26.3	4.81	ND	1.41	3.04	ND	ND	ND	ND	3.54	ND	1.93	5.5	ND	0.759		ND	6.95	4.03
Sweeney Raw	2/11/2019	13.6	17.8	5.96	ND	2.63	ND	ND	ND	ND	ND	4.64	ND	4.28	7.49	ND	0.735		ND	12.6	6.32
Sweeney Finished	2/12/2019	27.6	37.8	7.01	ND	1.56	ND	ND	ND	ND	ND	2.85	ND	2.72	5.16	ND	ND		ND	7.5	4.3
Sweeney Raw	2/18/2019	7.79	12.4	4.79	7.23	2.26	3.68	ND	ND	ND	ND	3.96	ND	4.25	6.24	ND	ND		ND	9.24	4.58
Sweeney Finished	2/19/2019	29.5	33.1	7.5	ND	1.72	4.15	ND	ND	ND	ND	3.39	ND	2.52	5.97	ND	ND		ND	5.85	3.91
Sweeney Raw	2/25/2019	2.78	3.82	1.61	ND	1.6	ND	ND	ND	ND	ND	2.13	ND	2.1	4.02	ND	0.809		ND	9.54	4.41
Sweeney Finished	2/26/2019	26	27.7	3.17	ND	1.25	ND	ND	0.698	ND	ND	2.1	ND	1.61	3.73	ND	0.782		ND	5.34	3.99
Sweeney Raw	3/4/2019	6.54	7.68	5.21	3.57	1.58	ND	ND	ND	ND	ND	2.07	ND	2.26	3.28	ND	0.723		ND	7.93	4.68
Sweeney Finished	3/5/2019	23.6	28.7	4.51	1.94	1.08	1.8	ND	ND	ND	ND	1.37	ND	1.79	2.77	ND	ND		ND	5.14	2.93
Sweeney Raw	3/11/2019	5.36	4.69	10.6	2.31	1.61	4.39	ND	ND	ND	ND	2.22	ND	2.5	3.74	ND	0.629		ND	8.69	4.64
Sweeney Finished	3/12/2019	5.57	8.09	5.93	2.57	0.853	2.12	ND	ND	ND	ND	1.09	ND	0.775	2.66	ND	ND		ND	2.61	1.92
Sweeney Raw	3/18/2019	5.29	7.07	4.26	4.04	2.30	3.76	ND	0.747	ND	ND	3.59	ND	3.51	5.86	ND	0.991		ND	13.6	6.86
Sweeney Finished	3/19/2019	7.25	7.35	5.75	2.10	1.11	2.74	ND	ND	ND	ND	1.80	ND	1.13	3.76	ND	ND		ND	3.43	3.04
Sweeney Raw	3/25/2019	3.08	4.36	1.32	ND	2.03	3.87	ND	ND	ND	ND	3.26	ND	3.03	5.57	ND	0.893		ND	10.5	5.4
Sweeney Finished	3/26/2019	5.31	4.79	4.39	1.57	0.718	2.46	ND	ND	ND	ND	1.32	ND	0.730	2.68	ND	ND		ND	1.67	1.78
Sweeney Raw	4/1/2019	9.98	12.2	4.09	1.72	1.95	ND	ND	0.673	ND	ND	3.14	ND	2.98	5.32	ND	0.857		ND	11.1	6.32
Sweeney Finished	4/2/2019	6.40	5.39	4.31	1.35	0.617	2.27	ND	ND	ND	ND	1.21	ND	ND	2.42	ND	ND		ND	1.18	1.67
Sweeney Raw	4/8/2019	13.0	14.7	10.6	6.02	2.55	3.08	ND	ND	ND	ND	5.86	ND	4.26	6.54	ND	0.929		ND	12.2	5.82
Sweeney Finished	4/9/2019	8.26	8.63	11.8	2.42	1.04	2.70	ND	ND	ND	ND	2.22	ND	0.817	3.75	ND	ND		ND	1.87	1.95
Sweeney Raw	4/15/2019	7.65	7.20	5.05	2.17	2.05	ND	ND	ND	ND	ND	2.81	ND	2.50	3.67	ND	0.830		ND	8.55	3.69
Sweeney Finished	4/16/2019	6.38	6.50	5.15	1.28	0.766	2.06	ND	ND	ND	ND	0.959	ND	ND	2.48	ND	ND		ND	0.708	1.09
Sweeney Raw	4/22/2019	11.3	15.5	12.1	ND	1.96	ND	ND	ND	ND	ND	2.6	ND	2.69	3.59	ND	0.727		ND	9.38	4.63
Sweeney Finished	4/23/2019	8.00	8.81	12.20	ND	0.811	2.46	ND	ND	ND	ND	1.24	ND	ND	2.22	ND	ND		ND	1.06	1.24

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	1/15/2019	ND	6.19	ND	ND	ND		ND	**126.64**	**91.61**	**72**
Sweeney Raw	1/21/2019	ND	6.72	ND	ND	ND		ND	**92.05**	**50.57**	**55**
Sweeney Finished	1/22/2019	ND	5	ND	ND	ND		ND	**108.34**	**81.87**	**76**
Sweeney Raw	1/28/2019	0.658	7.52	ND	ND	ND		ND	**78.43**	**32.08**	**41**
Sweeney Finished	1/29/2019	ND	4.52	ND	ND	ND		ND	**105.32**	**81.91**	**78**
Sweeney Raw	2/4/2019	ND	6.84	ND	ND	ND		ND	**100.73**	**62.60**	**62**
Sweeney Finished	2/5/2019	ND	5.24	ND	ND	ND		ND	**120.00**	**91.14**	**76**
Sweeney Raw	2/11/2019	0.725	7.2	ND	ND	ND		ND	**109.00**	**67.02**	**61**
Sweeney Finished	2/12/2019	ND	4.71	ND	ND	ND		ND	**143.22**	**117.27**	**82**
Sweeney Raw	2/18/2019	0.738	6.2	ND	ND	ND		ND	**90.49**	**53.30**	**59**
Sweeney Finished	2/19/2019	ND	6.03	ND	ND	ND		ND	**147.33**	**117.18**	**80**
Sweeney Raw	2/25/2019	ND	4.09	ND	ND	ND		ND	**40.99**	**14.42**	**35**
Sweeney Finished	2/26/2019	ND	3.91	ND	ND	ND		ND	**113.90**	**92.59**	**81**
Sweeney Raw	3/4/2019	ND	2.73	ND	ND	ND		ND	**58.45**	**35.27**	**60**
Sweeney Finished	3/5/2019	ND	2.55	ND	ND	ND		ND	**109.73**	**91.67**	**84**
Sweeney Raw	3/11/2019	ND	3.53	ND	ND	ND		ND	**63.29**	**33.56**	**53**
Sweeney Finished	3/12/2019	ND	2.75	ND	ND	ND		ND	**44.76**	**31.07**	**69**
Sweeney Raw	3/18/2019	ND	5.19	ND	ND	ND		ND	**75.86**	**33.04**	**44**
Sweeney Finished	3/19/2019	ND	3.60	ND	ND	ND		ND	**50.48**	**31.67**	**63**
Sweeney Raw	3/25/2019	0.663	5.4	ND	ND	ND		ND	**52.52**	**15.16**	**29**
Sweeney Finished	3/26/2019	ND	3.03	ND	ND	ND		ND	**35.50**	**22.43**	**63**
Sweeney Raw	4/1/2019	ND	5.60	ND	ND	ND		ND	**77.70**	**42.90**	**55**
Sweeney Finished	4/2/2019	ND	2.92	ND	ND	ND		ND	**34.82**	**23.74**	**68**
Sweeney Raw	4/8/2019	0.715	6.11	ND	ND	ND		ND	**122.71**	**80.51**	**66**
Sweeney Finished	4/9/2019	ND	3.93	ND	ND	ND		ND	**60.48**	**44.42**	**73**
Sweeney Raw	4/15/2019	ND	3.55	ND	ND	ND		ND	**60.89**	**36.05**	**59**
Sweeney Finished	4/16/2019	ND	2.30	ND	ND	ND		ND	**34.40**	**25.00**	**73**
Sweeney Raw	4/22/2019	ND	4.11	ND	ND	ND		ND	**95.69**	**68.60**	**72**
Sweeney Finished	4/23/2019	ND	2.91	ND	ND	ND		ND	**50.60**	**39.90**	**79**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Raw	4/29/2019	ND	ND	ND	8.64	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.83
Sweeney Finished	4/30/2019	ND	ND	ND	3.27	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.48
Sweeney Raw	5/6/2019	ND	ND	ND	6.79	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.73
Sweeney Finished	5/7/2019	ND	ND	ND	3.67	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.99
Sweeney Raw	5/13/2019	ND	ND	ND	12.0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.90	6.09
Sweeney Finished	5/14/2019	ND	ND	ND	4.88	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.53
Sweeney Raw	5/20/2019	ND	ND	ND	19.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.57	7.27
Sweeney Finished	5/21/2019	ND	ND	ND	7.24	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.65
Sweeney Raw	5/27/2019	ND	ND	ND	23.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.59	8.01
Sweeney Finished	5/28/2019	ND	ND	ND	9.32	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.25	4.62
Sweeney Raw	6/3/2019	ND	ND	ND	31.9	ND	ND	ND	ND	ND	ND	ND	ND	3.51	ND	ND	ND	6.14	16.7
Sweeney Finished	6/4/2019	ND	ND	ND	13.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.58	6.55
Sweeney Raw	6/10/2019	ND	ND	ND	36.9	ND	ND	ND	ND	ND	ND	ND	ND	5.43	ND	ND	ND	7.09	18.4
Sweeney Finished	6/11/2019	ND	ND	ND	15.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.31	6.69
Sweeney Raw	6/17/2019	ND	ND	ND	9.66	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.82
Sweeney Finished	6/18/2019	ND	ND	ND	7.73	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.90
Sweeney Raw	6/24/2019	ND	ND	ND	9.67	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.65	4.13
Sweeney Finished	6/25/2019	ND	ND	ND	8.03	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.76	7.30
Sweeney Raw	7/1/2019	ND	ND	ND	54.8	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	11.3
Sweeney Finished	7/2/2019	ND	ND	ND	8.61	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.41	6.53
Sweeney Raw	7/8/2019	ND	ND	ND	28.6	ND	ND	ND	ND	ND	ND	ND	ND	3.08	ND	ND	ND	3.88	9.71
Sweeney Finished	7/9/2019	ND	ND	ND	14.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.60	8.25
Sweeney Raw	7/15/2019	ND	ND	ND	24.8	ND	ND	ND	ND	ND	ND	ND	ND	2.70	ND	ND	ND	3.31	7.43
Sweeney Finished	7/16/2019	ND	ND	ND	16.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.80	8.75
Sweeney Raw	7/29/2019	ND	ND	ND	24.8	ND	ND	ND	ND	ND	ND	ND	ND	2.10	ND	ND	ND	2.42	8.85
Sweeney Finished	7/30/2019	ND	ND	ND	19.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.25	10.0
Sweeney Raw	8/12/2019	ND	ND	ND	15.7	ND	ND	ND	ND	ND	ND	ND	ND	1.57	ND	ND	ND	2.04	5.10
Sweeney Finished	8/13/2019	ND	ND	ND	13.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.43	13.4
Sweeney Raw	8/26/2019	ND	ND	ND	28.3	ND	ND	ND	ND	ND	ND	ND	ND	2.70	ND	ND	ND	3.15	9.93

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Raw	4/29/2019	8.79	11.8	6.06	4.04	2.77	4.65	ND	0.718	ND	ND	8.29	ND	4.15	9.01	ND	1.42		ND	16.6	8.79
Sweeney Finished	4/30/2019	4.83	6.11	3.81	ND	0.621	2.24	ND	ND	ND	ND	1.47	ND	ND	3.13	ND	ND		ND	1.26	1.41
Sweeney Raw	5/6/2019	5.76	8.2	2.47	3.03	2.77	5.37	ND	0.730	ND	ND	6.82	ND	3.30	9.42	ND	1.17		ND	15.1	7.87
Sweeney Finished	5/7/2019	5.81	5.64	4.14	2.21	0.910	3.39	ND	ND	ND	ND	2.08	ND	ND	3.92	ND	ND		ND	1.18	1.66
Sweeney Raw	5/13/2019	14.5	18.7	4.61	ND	3.05	ND	ND	0.932	ND	ND	7.93	ND	4.13	10.9	ND	1.17		ND	18.2	8.24
Sweeney Finished	5/14/2019	9.26	9.56	5.21	1.92	0.916	3.60	ND	ND	ND	ND	1.85	ND	ND	4.10	ND	ND		ND	1.09	1.60
Sweeney Raw	5/20/2019	19.4	25.2	8.76	3.96	2.64	5.64	ND	ND	ND	ND	4.98	ND	4.70	8.69	ND	1.24		ND	14.0	7.03
Sweeney Finished	5/21/2019	11.3	11.8	10.1	3.73	0.806	3.57	ND	ND	ND	ND	1.57	ND	0.64	3.07	ND	ND		ND	0.852	1.15
Sweeney Raw	5/27/2019	20.3	29.3	6.78	1.82	3.15	6.52	ND	0.750	ND	ND	7.35	ND	4.46	11	ND	0.95		ND	13.8	6.97
Sweeney Finished	5/28/2019	17.3	17.8	12.7	4.47	1.04	4.04	ND	ND	ND	ND	1.83	ND	0.626	3.93	ND	ND		ND	0.874	1.29
Sweeney Raw	6/3/2019	43.5	52.2	14.6	6.34	3.75	6.89	ND	0.775	ND	ND	9.99	ND	5.83	16.5	ND	1.29		ND	17.4	7.97
Sweeney Finished	6/4/2019	24.3	21.7	18.0	7.36	1.15	6.03	ND	ND	ND	ND	2.27	ND	0.628	5.36	ND	ND		ND	0.940	1.26
Sweeney Raw	6/10/2019	46.8	57.0	14.7	8.23	3.72	5.29	ND	0.890	ND	ND	9.47	ND	6.25	14.4	ND	1.02		ND	18.4	8.27
Sweeney Finished	6/11/2019	28.8	25.8	18.2	7.82	1.22	5.99	ND	ND	ND	ND	2.23	ND	0.592	5.73	ND	ND		ND	1.08	1.26
Sweeney Raw	6/17/2019	7.70	8.02	2.07	2.40	3.44	6.07	ND	0.783	ND	ND	9.57	ND	4.52	15.3	ND	1.07		ND	14.0	6.51
Sweeney Finished	6/18/2019	16.2	15.1	7.0	3.39	1.41	5.35	ND	ND	ND	ND	2.70	ND	0.815	6.58	ND	ND		ND	1.28	1.48
Sweeney Raw	6/24/2019	13.7	14.7	5.46	2.41	2.41	6.43	ND	1.25	ND	ND	13.5	ND	3.58	20.3	ND	1.42		ND	14.4	8.61
Sweeney Finished	6/25/2019	20.0	20.6	10.3	3.45	1.37	6.61	ND	ND	ND	ND	4.76	ND	0.903	10.6	ND	ND		ND	0.908	2.25
Sweeney Raw	7/1/2019	57.7	63.0	64.9	22.6	10.3	6.86	ND	ND	ND	ND	2.22	ND	2.08	5.16	ND	ND		ND	1.56	3.36
Sweeney Finished	7/2/2019	22.6	21.1	8.1	2.43	1.84	8.13	ND	ND	ND	ND	4.51	ND	1.070	11.9	ND	ND		ND	1.280	2.39
Sweeney Raw	7/8/2019	32.5	36.2	8.07	9.52	4.45	7.22	ND	1.29	ND	ND	17.7	ND	5.73	25.7	ND	1.48		ND	19.6	9.63
Sweeney Finished	7/9/2019	30.7	34.5	15.1	6.44	2.19	8.64	ND	ND	ND	ND	5.41	ND	1.130	12.70	ND	ND		ND	1.28	2.59
Sweeney Raw	7/15/2019	24.7	34.9	7.58	7.15	5.53	5.20	ND	1.01	ND	ND	10.9	ND	9.39	16.0	ND	1.37		ND	21.4	8.59
Sweeney Finished	7/16/2019	36.5	35.2	16.5	5.92	2.56	8.18	ND	ND	ND	ND	4.47	ND	1.30	11.5	ND	ND		ND	1.21	2.22
Sweeney Raw	7/29/2019	25.3	30.0	6.67	7.90	5.43	7.70	ND	0.97	ND	ND	12.7	ND	8.54	19.3	ND	1.28		ND	19.7	7.20
Sweeney Finished	7/30/2019	35.2	42.5	13.6	5.14	3.07	8.47	ND	ND	ND	ND	5.41	ND	2.18	12.2	ND	ND		ND	2.36	2.60
Sweeney Raw	8/12/2019	15.5	22.2	5.51	5.79	4.84	8.28	ND	0.967	ND	ND	10.5	ND	7.05	14.2	ND	1.13		ND	15.7	6.62
Sweeney Finished	8/13/2019	32.7	29.5	11.6	5.68	3.10	7.80	ND	ND	ND	ND	5.23	ND	1.90	10.8	ND	ND		ND	1.64	2.51
Sweeney Raw	8/26/2019	28.7	38.9	8.30	ND	5.54	11.2	ND	1.42	ND	ND	24.8	ND	6.24	34.7	ND	1.74		ND	18.4	9.95

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Raw	4/29/2019	1.01	8.21	ND	ND	ND		ND	**108.78**	**51.45**	**47**
Sweeney Finished	4/30/2019	ND	2.93	ND	ND	ND		ND	**33.56**	**21.97**	**65**
Sweeney Raw	5/6/2019	0.726	8.21	ND	ND	ND		ND	**90.46**	**35.79**	**40**
Sweeney Finished	5/7/2019	ND	4.55	ND	ND	ND		ND	**41.15**	**25.54**	**62**
Sweeney Raw	5/13/2019	0.644	10.2	ND	ND	ND		ND	**123.20**	**65.73**	**53**
Sweeney Finished	5/14/2019	ND	4.67	ND	ND	ND		ND	**51.19**	**35.21**	**69**
Sweeney Raw	5/20/2019	0.704	10.2	ND	ND	ND		ND	**146.58**	**91.74**	**63**
Sweeney Finished	5/21/2019	ND	4.58	ND	ND	ND		ND	**64.06**	**49.39**	**77**
Sweeney Raw	5/27/2019	0.749	12.2	ND	ND	ND		ND	**160.30**	**99.75**	**62**
Sweeney Finished	5/28/2019	ND	5.85	ND	ND	ND		ND	**86.94**	**69.29**	**80**
Sweeney Raw	6/3/2019	0.888	16.3	ND	ND	ND		ND	**262.47**	**184.88**	**70**
Sweeney Finished	6/4/2019	ND	8.11	ND	ND	ND		ND	**118.84**	**95.36**	**80**
Sweeney Raw	6/10/2019	0.93	18.0	ND	ND	ND		ND	**281.19**	**204.02**	**73**
Sweeney Finished	6/11/2019	ND	8.39	ND	ND	ND		ND	**130.71**	**106.45**	**81**
Sweeney Raw	6/17/2019	0.747	13.2	ND	ND	ND		ND	**107.88**	**42.24**	**39**
Sweeney Finished	6/18/2019	ND	8.45	ND	ND	ND		ND	**82.41**	**57.04**	**69**
Sweeney Raw	6/24/2019	0.656	16.4	ND	ND	ND		ND	**140.68**	**65.22**	**46**
Sweeney Finished	6/25/2019	ND	12.3	ND	ND	ND		ND	**111.14**	**76.20**	**69**
Sweeney Raw	7/1/2019	0.788	5.25	ND	ND	ND		ND	**311.88**	**276.52**	**89**
Sweeney Finished	7/2/2019	ND	14.8	ND	ND	ND		ND	**116.65**	**75.24**	**65**
Sweeney Raw	7/8/2019	1.04	22.2	ND	ND	ND		ND	**247.60**	**149.26**	**60**
Sweeney Finished	7/9/2019	ND	15.5	ND	ND	ND		ND	**160.93**	**116.9**	**73**
Sweeney Raw	7/15/2019	1.29	18.9	ND	ND	ND		ND	**212.15**	**123.47**	**58**
Sweeney Finished	7/16/2019	ND	14.6	ND	ND	ND		ND	**167.11**	**125.54**	**75**
Sweeney Raw	7/29/2019	0.980	21.3	ND	ND	ND		ND	**213.14**	**120.74**	**57**
Sweeney Finished	7/30/2019	ND	16.3	ND	ND	ND		ND	**180.78**	**133.6**	**74**
Sweeney Raw	8/12/2019	1.31	16.6	ND	ND	ND		ND	**160.61**	**83.91**	**52**
Sweeney Finished	8/13/2019	ND	13.9	ND	ND	ND		ND	**156.09**	**114.44**	**73**
Sweeney Raw	8/26/2019	1.19	33.7	ND	ND	ND		ND	**268.86**	**144.78**	**54**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	8/27/2019	ND	ND	ND	18.2	ND	ND	ND	ND	ND	ND	ND	1.29	ND	ND	ND	ND	3.26	12.7
Sweeney Raw	9/4/2019	ND	ND	ND	11.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.75	4.08
Sweeney Raw	9/5/2019	ND	ND	ND	11.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.69	3.91
Sweeney Raw	9/6/2019	ND	ND	ND	11.1	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.69
Sweeney Raw	9/9/2019	ND	ND	ND	76.0	ND	ND	ND	ND	ND	ND	3.78	6.14	ND	ND	ND	ND	7.98	14.3
Sweeney Finished	9/10/2019	ND	ND	ND	25.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.70	12.4
Sweeney Raw	9/23/2019	ND	ND	ND	28.4	ND	ND	ND	ND	ND	ND	ND	2.98	ND	ND	ND	ND	3.20	8.80
Sweeney Finished	9/24/2019	ND	ND	ND	20.6	ND	ND	ND	ND	ND	ND	ND	1.62	ND	ND	ND	ND	2.76	13.2
Sweeney Raw	10/1/2019	ND	ND	ND	39.4	ND	ND	ND	ND	ND	ND	ND	3.41	ND	ND	ND	ND	3.74	14.30
Sweeney Finished	10/2/2019	ND	ND	ND	28.4	ND	ND	ND	ND	ND	ND	ND	2.05	ND	ND	ND	ND	3.99	16.7
Sweeney Raw	10/7/2019	ND	ND	ND	38.8	ND	ND	ND	ND	ND	ND	ND	2.91	ND	ND	ND	ND	5.29	17.5
Sweeney Finished	10/8/2019	ND	ND	ND	34.2	ND	ND	ND	ND	ND	ND	ND	1.96	ND	ND	ND	ND	3.99	18.5
Sweeney Raw	10/21/2019	ND	ND	ND	41.7	ND	ND	ND	ND	ND	ND	ND	3.33	ND	ND	ND	ND	6.13	17.6
Sweeney Finished	10/22/2019	ND	ND	ND	36.3	ND	ND	ND	ND	ND	ND	ND	2.07	ND	ND	ND	ND	4.13	17.4
Sweeney Raw	11/4/2019	ND	ND	ND	24.8	ND	ND	ND	ND	ND	ND	ND	1.68	ND	ND	ND	ND	2.65	9.52
Sweeney Finished	11/5/2019	ND	ND	ND	12.6	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.54	9.16
Sweeney Raw	11/18/2019	ND	ND	ND	35.0	ND	ND	ND	ND	ND	ND	ND	1.62	ND	ND	ND	ND	4.38	9.97
Sweeney Finished	11/19/2019	ND	ND	ND	12.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.38	8.25
Sweeney Raw	12/2/2019	ND	ND	ND	22.50	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.26	6.01
Sweeney Finished	12/3/2019	ND	ND	ND	11.30	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.40	6.54
Sweeney Raw	12/16/2019	ND	ND	ND	19.90	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.46	7.89
Sweeney Finished	12/17/2019	ND	ND	ND	11.70	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.83	5.43
Sweeney Raw	12/30/2019	ND	ND	ND	8.04	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.69
Sweeney Finished	12/31/2019	ND	ND	ND	6.40	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.26	3.92
Sweeney Raw	1/13/2020	ND	ND	ND	6.36	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.30
Sweeney Finished	1/14/2020	ND	ND	ND	6.01	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.79
Sweeney Raw	1/27/2020	ND	ND	ND	12.70	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.33	5.12
Sweeney Finished	1/28/2020	ND	ND	ND	9.77	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.71	5.84
Sweeney Raw	2/10/2020	ND	ND	ND	4.47	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.36

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	8/27/2019	40.3	46.2	14.0	6.62	3.82	13.2	ND	ND	ND	ND	9.84	ND	1.81	21.9	ND	ND		ND	2.05	3.29
Sweeney Raw	9/4/2019	11.5	14.8	5.97	3.24	4.31	10.8	ND	0.97	ND	ND	21.9	ND	4.80	26.4	ND	1.54		ND	14.5	8.44
Sweeney Raw	9/5/2019	12.9	16.5	7.41	6.60	4.7	11.5	ND	1.04	ND	ND	23.1	ND	5.72	28.2	ND	1.60		ND	15.5	8.42
Sweeney Raw	9/6/2019	12.7	15.8	6.77	ND	4.22	11.2	ND	1.07	ND	ND	24.2	ND	4.22	30.9	ND	1.53		ND	15.4	7.70
Sweeney Raw	9/9/2019	36.1	52.3	21.7	16.0	5.00	12.5	ND	0.94	ND	ND	26.0	ND	4.75	35.2	ND	1.45		ND	15.5	8.30
Sweeney Finished	9/10/2019	39.0	44.9	16.7	8.82	3.57	16.4	1.20	ND	ND	ND	12.3	ND	1.84	25.4	ND	ND		ND	1.93	3.44
Sweeney Raw	9/23/2019	24.1	34.2	8.74	15.6	3.39	16.1	ND	ND	ND	ND	7.98	ND	5.03	11.3	ND	0.714		ND	11.4	5.19
Sweeney Finished	9/24/2019	39.7	44.8	15.8	7.30	2.87	9.75	ND	ND	ND	ND	6.51	ND	2.24	10.8	ND	ND		ND	2.52	2.88
Sweeney Raw	10/1/2019	40.3	46.8	10.4	25.7	5.60	9.16	ND	1.01	ND	ND	14.9	ND	6.73	21.1	ND	1.29		ND	16.0	7.58
Sweeney Finished	10/2/2019	52.6	63.4	17.6	8.37	4.05	10.8	ND	ND	ND	ND	10.2	ND	2.57	17.0	ND	ND		ND	3.69	4.18
Sweeney Raw	10/7/2019	50.2	57.4	15.7	11.0	6.73	13.8	ND	0.820	ND	ND	22.1	ND	7.71	33.4	ND	1.40		ND	15.2	9.63
Sweeney Finished	10/8/2019	65.1	60.1	28.6	11.4	6.52	12.6	ND	ND	ND	ND	13.7	ND	2.90	29.6	ND	ND		ND	3.78	5.11
Sweeney Raw	10/21/2019	47.7	55.6	11.4	11.1	6.61	18.3	ND	1.05	ND	ND	30.2	ND	7.41	45.5	ND	1.67		ND	14.9	10.20
Sweeney Finished	10/22/2019	57.4	65.7	18.0	10.5	5.66	18.2	ND	ND	ND	ND	19.5	ND	3.45	33.4	ND	0.646		ND	3.68	5.92
Sweeney Raw	11/4/2019	23.0	26.7	9.21	5.47	6.52	17.1	ND	0.961	ND	ND	22.3	ND	9.18	32.6	ND	1.41		ND	17.1	9.84
Sweeney Finished	11/5/2019	24.5	29.1	6.14	3.19	3.27	8.31	ND	ND	ND	ND	9.52	ND	2.47	16.8	ND	ND		ND	2.50	3.68
Sweeney Raw	11/18/2019	34.2	41.8	17.1	ND	5.99	2.05	ND	0.737	ND	ND	14.3	ND	5.97	24.4	ND	1.61		ND	12.9	7.96
Sweeney Finished	11/19/2019	25.3	24.8	11.3	3.50	2.46	6.18	ND	ND	ND	ND	4.98	ND	1.59	10.9	ND	ND		ND	2.08	2.55
Sweeney Raw	12/2/2019	20.60	24.00	13.00	ND	4.24	5.95	ND	ND	ND	ND	6.43	ND	5.51	12.40	ND	0.78		ND	11.00	5.08
Sweeney Finished	12/3/2019	18.40	18.90	15.10	3.38	1.93	4.41	ND	ND	ND	ND	3.87	ND	1.88	7.67	ND	ND		ND	2.24	2.09
Sweeney Raw	12/16/2019	19.80	21.30	6.72	6.02	6.06	10.60	ND	0.87	ND	ND	15.70	ND	4.26	30.80	ND	1.07	ND	ND	13.00	7.92
Sweeney Finished	12/17/2019	14.50	13.70	7.09	4.23	2.55	7.57	ND	ND	ND	ND	6.24	ND	1.68	13.30	ND	ND	ND	ND	2.41	3.04
Sweeney Raw	12/30/2019	7.98	8.52	ND	2.86	4.02	7.07	ND	ND	ND	ND	11.90	ND	3.78	18.30	ND	1.03	ND	ND	10.10	6.60
Sweeney Finished	12/31/2019	10.30	9.65	4.91	2.84	2.21	6.11	ND	ND	ND	ND	6.50	ND	1.56	12.60	ND	ND	ND	ND	2.50	2.71
Sweeney Raw	1/13/2020	6.86	6.98	3.50	1.49	5.01	5.92	ND	ND	ND	ND	7.95	ND	4.14	15.10	ND	0.97	ND	ND	11.30	8.92
Sweeney Finished	1/14/2020	10.40	10.20	4.81	2.34	2.24	5.61	ND	ND	ND	ND	4.02	ND	1.53	9.44	ND	ND	ND	ND	2.46	3.15
Sweeney Raw	1/27/2020	12.40	16.00	8.50	ND	3.76	5.31	ND	ND	ND	ND	9.10	ND	4.02	12.90	ND	0.97	ND	ND	13.10	7.22
Sweeney Finished	1/28/2020	13.30	15.00	11.10	3.22	2.69	5.99	ND	ND	ND	ND	5.90	ND	2.05	11.90	ND	ND	ND	ND	4.42	3.94
Sweeney Raw	2/10/2020	4.17	5.82	7.71	ND	2.40	3.29	ND	ND	ND	ND	5.34	ND	2.93	5.79	ND	0.71	ND	ND	8.18	4.37

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	8/27/2019	ND	25.9	ND	ND	ND		ND	**224.38**	**152.41**	**68**
Sweeney Raw	9/4/2019	0.79	24.4	ND	ND	ND		ND	**171.49**	**74.54**	**43**
Sweeney Raw	9/5/2019	1.07	25.3	ND	ND	ND		ND	**186.76**	**83.71**	**45**
Sweeney Raw	9/6/2019	0.96	26.7	ND	ND	ND		ND	**178.16**	**74.26**	**42**
Sweeney Raw	9/9/2019	0.71	32.2	ND	ND	ND		ND	**376.85**	**260.3**	**69**
Sweeney Finished	9/10/2019	ND	28.6	ND	ND	ND		ND	**244.40**	**162.02**	**66**
Sweeney Raw	9/23/2019	0.665	15.6	ND	ND	ND		ND	**203.39**	**134**	**66**
Sweeney Finished	9/24/2019	0.640	11.9	ND	ND	ND		ND	**195.89**	**152.29**	**78**
Sweeney Raw	10/1/2019	1.23	21.0	ND	ND	ND		ND	**289.65**	**198.95**	**69**
Sweeney Finished	10/2/2019	0.651	22.8	ND	ND	ND		ND	**269.05**	**203.31**	**76**
Sweeney Raw	10/7/2019	1.24	31.8	ND	ND	ND		ND	**342.63**	**220.9**	**64**
Sweeney Finished	10/8/2019	0.812	31.2	ND	ND	ND		ND	**330.07**	**237.55**	**72**
Sweeney Raw	10/21/2019	1.51	45.1	ND	ND	ND		ND	**377.01**	**224.76**	**60**
Sweeney Finished	10/22/2019	1.04	41.6	ND	ND	ND		ND	**344.60**	**231**	**67**
Sweeney Raw	11/4/2019	1.60	36.5	ND	ND	ND		ND	**258.14**	**125.33**	**49**
Sweeney Finished	11/5/2019	ND	19.7	ND	ND	ND		ND	**153.48**	**96.75**	**63**
Sweeney Raw	11/18/2019	1.13	26.9	ND	ND	ND		ND	**248.02**	**158.37**	**64**
Sweeney Finished	11/19/2019	ND	13.4	ND	ND	ND		ND	**130.97**	**91.81**	**70**
Sweeney Raw	12/2/2019	0.88	13.80	ND	ND	ND		ND	**154.43**	**94.8**	**61**
Sweeney Finished	12/3/2019	ND	8.60	ND	ND	ND		ND	**107.71**	**78.89**	**73**
Sweeney Raw	12/16/2019	1.00	25.40	ND	ND	ND	ND	ND	**200.76**	**99.79**	**50**
Sweeney Finished	12/17/2019	ND	14.30	ND	ND	ND	ND	ND	**109.57**	**64.72**	**59**
Sweeney Raw	12/30/2019	0.91	14.30	ND	ND	ND	ND	ND	**108.10**	**41.99**	**39**
Sweeney Finished	12/31/2019	ND	11.60	ND	ND	ND	ND	ND	**85.07**	**45.78**	**54**
Sweeney Raw	1/13/2020	1.01	11.30	ND	ND	ND	ND	ND	**99.11**	**35.44**	**36**
Sweeney Finished	1/14/2020	ND	8.95	ND	ND	ND	ND	ND	**74.95**	**41.57**	**55**
Sweeney Raw	1/27/2020	0.68	10.80	ND	ND	ND	ND	ND	**123.91**	**65.15**	**53**
Sweeney Finished	1/28/2020	ND	11.20	ND	ND	ND	ND	ND	**108.03**	**65.84**	**61**
Sweeney Raw	2/10/2020	ND	4.70	ND	ND	ND	ND	ND	**61.24**	**28.87**	**47**

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		11-chloroeicosafluoro-3-oxaundecane-1-sulfonate (PF3OUdS)	2-(N-ethylperfluoro-1-octanesulfonamido)-ethanol (N-EtFOSE)	2-(N-methylperfluoro-1-octanesulfonamido)-ethanol (N-MeFOSE)	2,3,3,3-Tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)-propanoic acid (PFPrOPrA) GenX	4-(Heptafluoroisopropoxy)hexafluorobutanoic acid (PFECA-G)*	9-chlorohexadecafluoro-3-oxanonane-1-sulfonate	Fluorotelomer sulfonate 10:2 (10:2 FTS)	Fluorotelomer sulfonate 4:2 (4:2 FTS)	Fluorotelomer sulfonate 6:2 (6:2 FTS)	Fluorotelomer sulfonate 8:2 (8:2 FTS)	Nafion Byproduct 1*	Nafion Byproduct 2*	N-ethylperfluoro-1-octanesulfonamide (N-EtFOSA)	N-ethylperfluoro-1-octanesulfonamidoacetic acid	N-methylperfluoro-1-octanesulfonamide (N-MeFOSA)	N-methylperfluoro-1-octanesulfonamidoacetic acid	Perfluoro(3,5,7,9-tetraoxadecanoic) acid (PFO4DA)*	Perfluoro(3,5,7-trioxaoctanoic) acid (PFO3OA)*
In Consent Order					Y	Y						Y	Y					Y	Y
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L
Sweeney Finished	2/11/2020	ND	ND	ND	4.73	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.11	4.36
Sweeney Raw	2/24/2020	ND	ND	ND	4.61	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	2/25/2020	ND	ND	ND	4.35	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.06
Sweeney Raw	3/9/2020	ND	ND	ND	13.00	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.64	3.61
Sweeney Finished	3/10/2020	ND	ND	ND	8.35	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.27	3.80
Sweeney Raw	4/6/2020	ND	ND	ND	11.30	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.37	4.93
Sweeney Finished	4/7/2020	ND	ND	ND	5.04	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.89
Sweeney Raw	5/4/2020	ND	ND	ND	9.42	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.38	3.58
Sweeney Finished	5/5/2020	ND	ND	ND	4.84	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.22
Sweeney Raw	6/1/2020	ND	ND	ND	4.19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Finished	6/2/2020	ND	ND	ND	3.67	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Sweeney Raw	7/6/2020	ND	ND	ND	17.80	ND	ND	ND	ND	ND	ND	ND	1.37	ND	ND	ND	ND	2.10	7.26
Sweeney Finished	7/7/2020	ND	ND	ND	11.30	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.07
Sweeney Raw	7/22/2020	ND	ND	ND	23.50	ND	ND	ND	ND	ND	ND	ND	1.41	ND	ND	ND	ND	2.25	6.65
Sweeney Raw	7/23/2020	ND	ND	ND	23.90	ND	ND	ND	ND	ND	ND	ND	1.47	ND	ND	ND	ND	2.71	6.45
Sweeney Raw	7/24/2020	ND	ND	ND	24.90	ND	ND	ND	ND	ND	ND	ND	1.69	ND	ND	ND	ND	2.70	8.61
Sweeney Raw	7/25/2020	ND	ND	ND	22.50	ND	ND	ND	ND	ND	ND	ND	1.75	ND	ND	ND	ND	2.63	8.51
Sweeney Raw	8/2/2020	ND	ND	ND	16.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.25	5.36
Sweeney Raw	8/3/2020	ND	ND	ND	15.2	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.50	3.94
Sweeney Raw	8/4/2020	ND	ND	ND	13.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.45	3.70
Sweeney Raw	8/5/2020	ND	ND	ND	13.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.23	3.43
Sweeney Finished	8/5/2020	ND	ND	ND	12.1	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	4.11

Raw and Finished PFAS Data

Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoro(3,5-dioxahexanoic) acid (PFO2HxA)*	Perfluoro-2-methoxyacetic acid (PFMOAA)*	Perfluoro-3-methoxypropanoic acid (PFMOPrA)*	Perfluoro-4-methoxybutanic acid (PFMOBA)*	Perfluorobutanesulfonate (PFBS)	Perfluorobutyric acid (PFBA)	Perfluorodecanesulfonate (PFDS)	Perfluorodecanoic acid (PFDA)	Perfluorododecanoic acid (PFDoA)	Perfluoroheptanesulfonate (PFHpS)	Perfluoroheptanoic acid (PFHpA)	Perfluorohexadecanoic acid (PFHxDA)	Perfluorohexanesulfonate (PFHxS)	Perfluorohexanoic acid (PFHxA)	Perfluorononanesulfonate (PFNS)	Perfluorononanoic acid (PFNA)	Perfluorooctadecanoic acid (PFODA) - added 12-16-19	Perfluorooctanesulfonamide (PFOSA)	**Perfluorooctanesulfonate (PFOS)**	**Perfluorooctanoic acid (PFOA)**
In Consent Order		Y	Y	Y	Y							Y									
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	NG/L	NG/L
Sweeney Finished	2/11/2020	9.69	9.73	6.02	ND	1.78	4.53	ND	ND	ND	ND	4.29	ND	1.60	7.01	ND	ND	ND	ND	3.37	3.17
Sweeney Raw	2/24/2020	4.04	9.89	2.01	1.50	2.61	3.43	ND	ND	ND	ND	5.45	ND	3.11	6.54	ND	0.75	ND	ND	8.93	5.40
Sweeney Finished	2/25/2020	7.79	18.30	2.78	2.04	2.03	4.47	ND	ND	ND	ND	3.43	ND	1.33	7.05	ND	ND	ND	ND	3.12	3.56
Sweeney Raw	3/9/2020	11.60	18.70	5.05	6.75	2.93	ND	ND	ND	ND	ND	4.38	ND	3.61	6.03	ND	0.90	ND	ND	9.48	5.64
Sweeney Finished	3/10/2020	11.40	12.10	6.46	4.55	1.88	4.29	ND	ND	ND	ND	3.37	ND	1.80	6.91	ND	ND	ND	ND	3.04	3.26
Sweeney Raw	4/6/2020	12.70	9.11	6.98	4.49	4.33	5.53	ND	ND	ND	ND	11.30	ND	4.27	13.70	ND	1.42	ND	ND	15.20	8.68
Sweeney Finished	4/7/2020	6.79	4.81	9.41	1.75	1.15	3.71	ND	ND	ND	ND	2.88	ND	ND	4.78	ND	ND	ND	ND	1.23	1.70
Sweeney Raw	5/4/2020	9.68	10.90	11.10	3.54	4.42	5.75	ND	ND	ND	ND	6.69	ND	5.38	12.00	ND	0.82	ND	ND	12.40	7.92
Sweeney Finished	5/5/2020	5.64	8.49	6.42	2.14	1.13	3.60	ND	ND	ND	ND	1.81	ND	0.64	4.85	ND	ND	ND	ND	1.06	1.53
Sweeney Raw	6/1/2020	3.77	3.40	2.67	ND	2.38	3.05	ND	ND	ND	ND	2.69	ND	3.51	4.90	ND	1.02	ND	ND	11.30	4.63
Sweeney Finished	6/2/2020	5.44	7.34	7.03	1.43	1.08	2.92	ND	ND	ND	ND	1.70	ND	0.68	3.93	ND	ND	ND	ND	1.22	1.32
Sweeney Raw	7/6/2020	20.00	23.80	9.82	7.50	3.95	5.88	ND	0.65	ND	ND	3.90	ND	4.83	7.40	ND	0.93	ND	ND	14.80	7.90
Sweeney Finished	7/7/2020	15.00	16.50	12.70	5.44	2.23	6.43	ND	ND	ND	ND	2.23	ND	0.97	6.73	ND	ND	ND	ND	1.41	2.34
Sweeney Raw	7/22/2020	24.10	24.80	12.40	ND	4.18	5.76	ND	0.73	ND	ND	3.96	ND	5.02	7.46	ND	1.12	ND	ND	16.40	6.83
Sweeney Raw	7/23/2020	24.90	22.10	12.30	ND	4.41	6.06	ND	0.75	ND	ND	3.75	ND	5.41	7.82	ND	1.34	ND	ND	17.90	6.51
Sweeney Raw	7/24/2020	26.50	34.60	7.12	9.18	4.14	6.58	ND	0.61	ND	ND	4.65	ND	5.05	7.67	ND	1.31	ND	ND	13.20	6.41
Sweeney Raw	7/25/2020	25.90	30.00	7.41	7.17	4.03	6.42	ND	0.69	ND	ND	3.83	ND	4.43	7.47	ND	0.98	ND	ND	13.80	6.32
Sweeney Raw	8/2/2020	17.3	22.0	10.80	2.45	4.09	4.86	ND	ND	ND	ND	3.31	ND	4.46	6.11	ND	0.834	ND	ND	13.0	6.09
Sweeney Raw	8/3/2020	11.7	19.0	10.30	2.58	4.52	5.66	ND	ND	ND	ND	3.74	ND	4.59	7.65	ND	0.947	ND	ND	12.1	6.59
Sweeney Raw	8/4/2020	13.3	15.8	9.73	ND	5.12	5.63	ND	0.790	ND	ND	4.46	ND	4.67	8.59	ND	1.22	ND	ND	14.7	7.70
Sweeney Raw	8/5/2020	12.1	15.4	9.28	ND	4.42	5.71	ND	0.817	ND	ND	4.49	ND	4.65	8.22	ND	1.28	ND	ND	12.6	7.01
Sweeney Finished	8/5/2020	19.6	24.3	16.6	3.79	3.03	7.04	ND	ND	ND	ND	2.63	ND	1.14	7.91	ND	ND	ND	ND	1.37	2.12

Raw and Finished PFAS Data

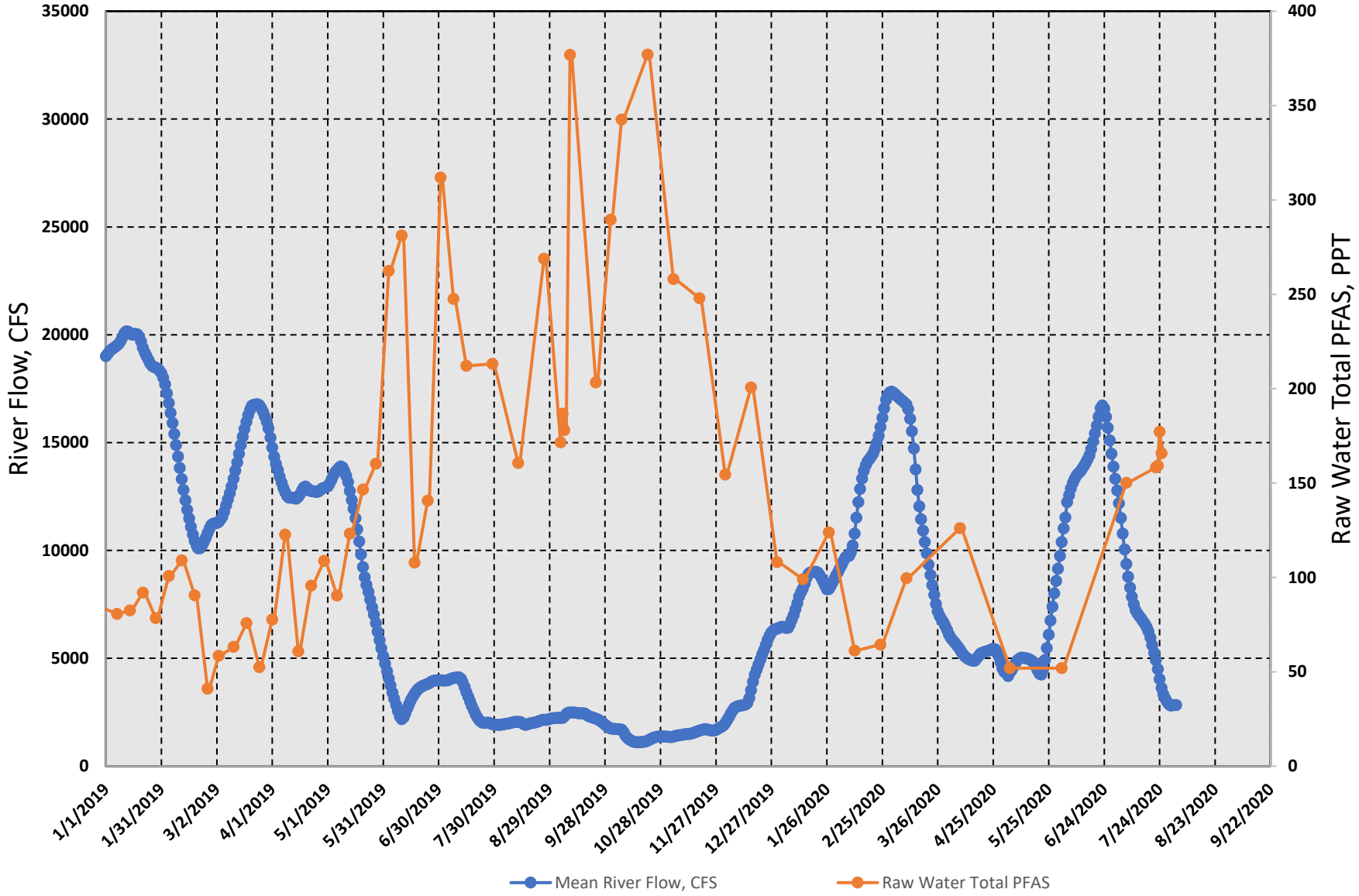
Blue row - compound in Consent Order Beige Rows - Finished Water (Potable) White Rows - Raw Water (River) Red Column - Legacy Compounds		Perfluoropentanesulfonate (PFPeS)	Perfluoropentanoic acid (PFPeA)	Perfluorotetradecanoic acid (PFTeDA)	Perfluorotridecanoic acid (PFTrDA)	Perfluoroundecanoic acid (PFUdA)	Sodium 2,2,4,4,6,6,8,8,10,10,12,12,12-tridecafluoro-3,5,7,9,11-pentaoxadodecanoate - added 12-16-19	Sodium dodecafluoro-3H-4,8-dioxanonanoate (ADONA)	**Total of all Compounds**	**Total of Compounds in Consent Order**	**% of Total in Consent Order**
In Consent Order											
Sample location	Sample date	NG/L	NG/L	NG/L	NG/L	NG/L		NG/L	**Total**	**NG/L**	**%**
Sweeney Finished	2/11/2020	ND	6.90	ND	ND	ND	ND	ND	**69.29**	**40.93**	**59**
Sweeney Raw	2/24/2020	ND	6.13	ND	ND	ND	ND	ND	**64.40**	**27.5**	**43**
Sweeney Finished	2/25/2020	ND	6.62	ND	ND	ND	ND	ND	**69.93**	**41.75**	**60**
Sweeney Raw	3/9/2020	ND	6.32	ND	ND	ND	ND	ND	**99.64**	**64.73**	**65**
Sweeney Finished	3/10/2020	ND	6.84	ND	ND	ND	ND	ND	**79.32**	**51.3**	**65**
Sweeney Raw	4/6/2020	0.66	10.10	ND	ND	ND	ND	ND	**126.07**	**62.18**	**49**
Sweeney Finished	4/7/2020	ND	5.23	ND	ND	ND	ND	ND	**50.37**	**32.57**	**65**
Sweeney Raw	5/4/2020	1.20	11.40	ND	ND	ND	ND	ND	**117.58**	**56.29**	**48**
Sweeney Finished	5/5/2020	ND	5.19	ND	ND	ND	ND	ND	**48.56**	**30.56**	**63**
Sweeney Raw	6/1/2020	ND	4.36	ND	ND	ND	ND	ND	**51.87**	**16.72**	**32**
Sweeney Finished	6/2/2020	ND	3.74	ND	ND	ND	ND	ND	**41.50**	**26.61**	**64**
Sweeney Raw	7/6/2020	0.71	8.35	ND	ND	ND	1.19	ND	**150.15**	**93.55**	**62**
Sweeney Finished	7/7/2020	ND	7.50	ND	ND	ND	ND	ND	**93.85**	**66.24**	**71**
Sweeney Raw	7/22/2020	0.99	9.51	ND	ND	ND	1.25	ND	**158.32**	**99.07**	**63**
Sweeney Raw	7/23/2020	0.77	9.31	ND	ND	ND	1.32	ND	**159.18**	**97.58**	**61**
Sweeney Raw	7/24/2020	0.83	9.87	ND	ND	ND	1.58	ND	**177.20**	**119.95**	**68**
Sweeney Raw	7/25/2020	0.68	9.39	ND	ND	ND	1.91	ND	**165.82**	**109.7**	**66**
Sweeney Raw	8/2/2020	0.675	7.49	ND	ND	ND	ND	ND	**127.48**	**79.87**	**63**
Sweeney Raw	8/3/2020	0.804	8.94	ND	ND	ND	ND	ND	**119.76**	**67.96**	**57**
Sweeney Raw	8/4/2020	0.771	9.51	ND	ND	ND	ND	ND	**120.64**	**61.94**	**51**
Sweeney Raw	8/5/2020	0.593	8.94	ND	ND	ND	ND	ND	**113.47**	**59.23**	**52**
Sweeney Finished	8/5/2020	ND	9.69	ND	ND	ND	ND	ND	**115.43**	**83.13**	**72**

NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT B
TO AMENDED INTERVENOR COMPLAINT

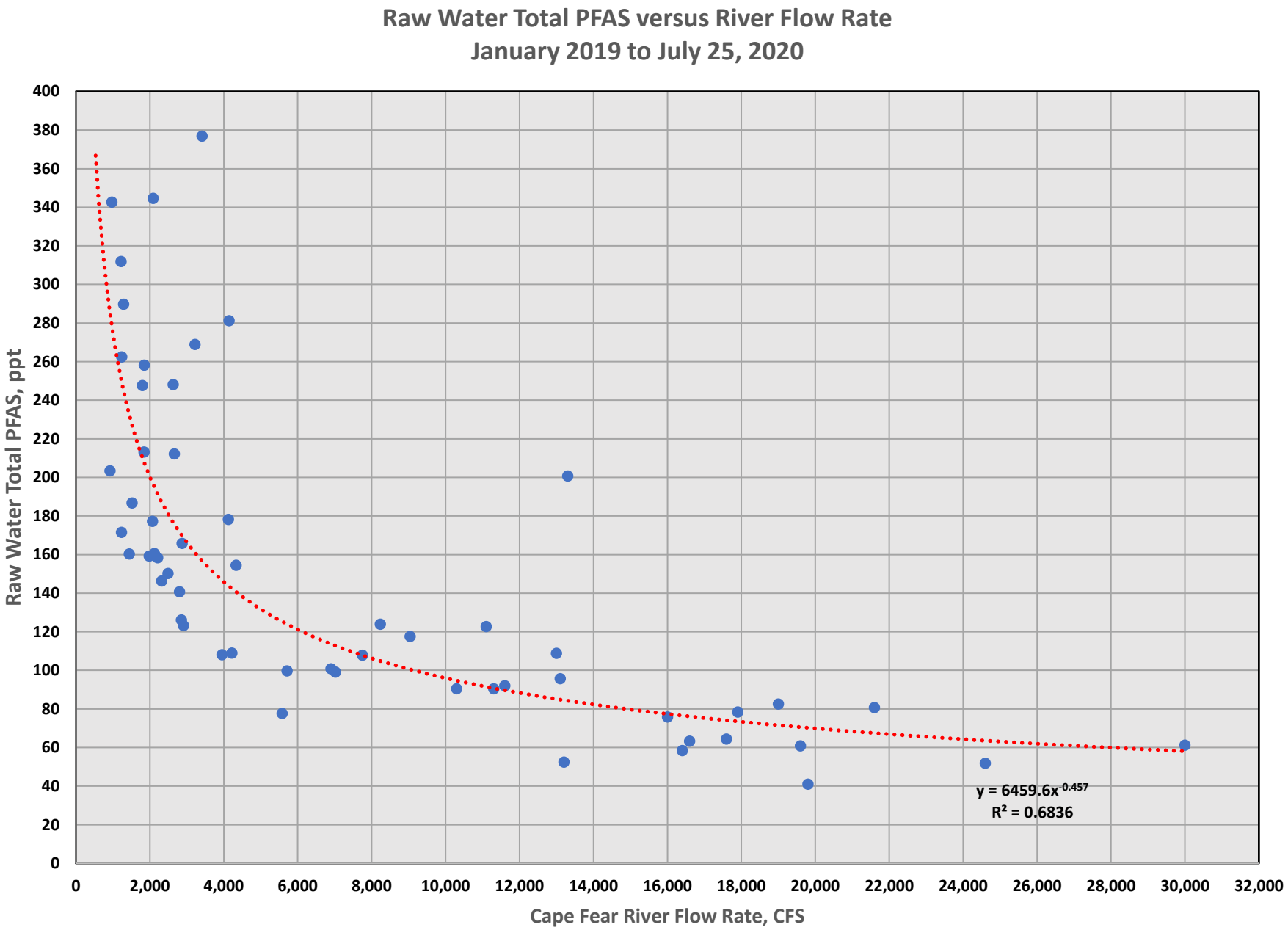
Cape Fear River Flow vs Raw Water Total PFAS

USGS 02105769 Cape Fear River at L&D #1, Kelly, NC



NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT C
TO AMENDED INTERVENOR COMPLAINT


Raw Water Total PFAS versus River Flow Rate
January 2019 to July 25, 2020
Raw Water Total PFAS, ppt
Cape Fear River Flow Rate, CFS
y = 6459.6x^-0.457
R² = 0.6836
0
20
40
60
80
100
120
140
160
180
200
220
240
260
280
300
320
340
360
380
400
0
2,000
4,000
6,000
8,000
10,000
12,000
14,000
16,000
18,000
20,000
22,000
24,000
26,000
28,000
30,000
32,000

NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT D
TO AMENDED INTERVENOR COMPLAINT

Technical Review of Cape Fear River PFAS Loading Reduction Plan for Cape Fear Public Utility Authority (CFPUA)

September 27, 2019

PREPARED FOR

Cape Fear Public Utility Authority
235 Government Center Drive
Wilmington, NC 28403

PREPARED BY

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TETRA TECH

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1.0 BACKGROUND

Chemours Company issued the Cape Fear River PFAS Loading Reduction Plan (Geosyntec, 2019) to the North Carolina Department of Environmental Quality (NCDEQ) and Cape Fear River Watch (CFRW) on August 26, 2016 in response to the Consent Order (CO) entered by the Bladen County Superior Court (paragraphs 12 and 11.1) on February 25, 2019. The CO was issued regarding emissions and discharges of per- and polyfluoroalkyl substances (PFAS), including hexafluoropropylene oxide dimer acid (HFPO-DA; 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)propanoic acid) and the ammonium salt of HFPO-DA, which has the trade name of GenX®, from the Fayetteville Works facility. GenX is used to manufacture high performance fluoropolymers. GenX replaces the ammonium salt of perfluorooctanoic acid (PFOA), which was phased out of production in 2009 because PFOA is persistent in the environment, bioaccumulates, and is toxic. At that time the Fayetteville Works facility was owned and operated by E.I. du Pont de Nemours and Company (DuPont). The Chemours Company was founded in July 2015 as a spin-off from DuPont.

In 2009 EPA authorized the manufacture of GenX; however, EPA also issued an order that required DuPont to capture, at an overall efficiency of 99%, new chemical substances from wastewater effluent and air emissions (premanufacture notice numbers P-08-508 and P-08-509). News broke regarding high levels of GenX and PFAS in the Cape Fear River and downstream potable waters in 2017 – spurring further environmental investigations and facility inspections. Shortly thereafter NCDEQ filed a Complaint alleging violations of the premanufacture order due to evidence in downstream waters of PFAS discharges by Chemours and DuPont, ultimately leading to the August 26, 2016 CO.

The Fayetteville Works facility is located in Bladen County, NC on the west side of the Cape Fear River just upstream of the William O, Huske Lock and Dam (Lock and Dam #3). The facility includes two Chemours manufacturing areas, the Monomers IXM area and the Polymer Processing Aid Area (PPA area), as well as an onsite process Wastewater Treatment Plant (WWTP) and Power Area (Geosyntec, 2019). In addition, manufacturing areas on the facility grounds are leased to Kuraray America Inc. for Butacite® and SentryGlas® production and to DuPont for polyvinyl fluoride (PVF) resin manufacturing.

The Chemours Fayetteville Works facility is located about 55 miles upstream of the Kings Bluff water intake on the Cape Fear River where the Cape Fear Public Utility Authority (CFPUA) withdraws water for treatment and potable use distribution. Elevated levels of PFAS have been observed in both the raw source water from the Cape Fear River and finished water at the CFPUA's Water Treatment Plants (WTPs). Traditional water treatment processes do not successfully remove GenX and other PFAS (Hopkins et al., 2018). The effectiveness of currently implemented and proposed PFAS pollution control strategies adopted by Chemours directly affect the quality of CFPUA's intake water and community exposure to these substances.

In light of these concerns, CFPUA engaged Tetra Tech to conduct a technical review of the PFAS Loading Reduction Plan and associated environmental assessments. Specifically, CFPUA requested input on the technical soundness of the surface and groundwater modeling, reasonableness of the assumptions applied in the analyses, reasonableness of the seven proposed strategies for reducing PFAS loads, identification of critical gaps in the analyses, and recommendations for additional studies related to reducing PFAS loads.

The Cape Fear River PFAS Loading Reduction Plan itself consists of 33 pages plus a cover letter, but is supported by five technical appendices: 1) PFAS Mass Loading Model, 2) Seeps and Creeks

Investigation Report, 3) Outfall 002 Assessment, 4) Terracotta Pipe Grouting Report, and 5) HFPO-DA Loading Reduction Estimates, all of which were completed by Chemours' consultant, Geosyntec Consultants of NC, P.C. The PFAS Loading Reduction Plan includes seven proposed actions aimed to reduce PFAS loading to the Cape Fear River. Findings from the review of the plan and supporting technical reports are discussed in this memorandum.

To better understand the relationship between river flow rate at the Kings Bluff intake and PFAS concentrations, CFPUA has developed a correlation analysis between the variables. CFPUA requested a technical review of the correlation analysis, which is also discussed in this memorandum as are implications related to the loading reduction plan.

2.0 TECHNICAL REVIEW

The PFAS loading reduction plan is informed by the PFAS Mass Loading Model (MLM), which evaluates contributions of PFAS to the Cape Fear River from nine pathways (Figure 1):

- Upstream river water and groundwater
- Willis Creek (north of the facility)
- Direct atmospheric deposition on the river in the vicinity of the facility
- Outfall 002
- Onsite upwelling groundwater
- Four identified onsite channelized seeps
- Old Outfall 002
- Offsite groundwater
- Georgia Branch Creek (south of the facility)

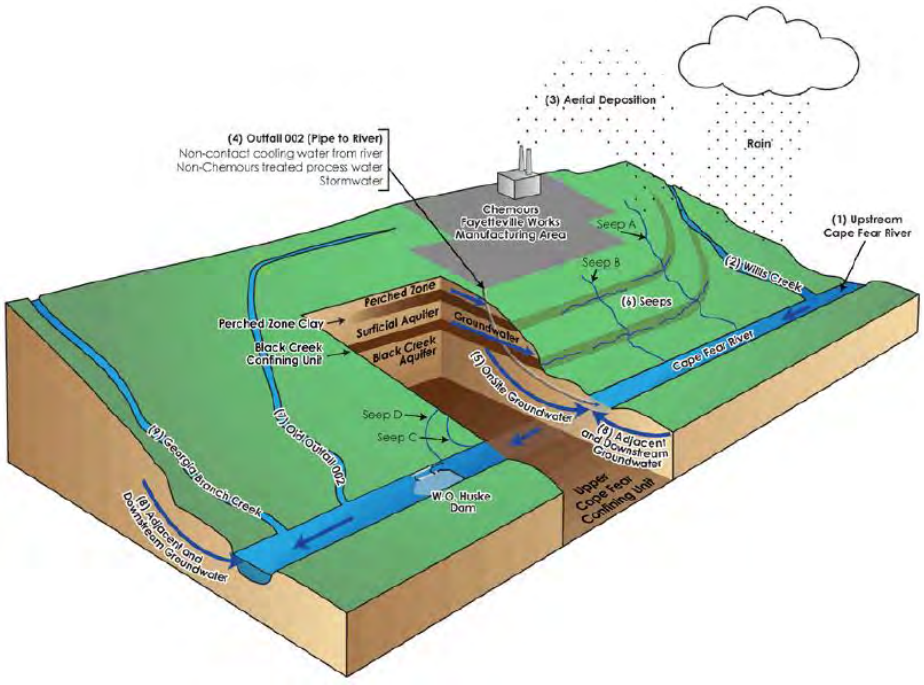

Figure 1. PFAS Transport Pathways (Geosyntec, 2019; Figure 5)

The MLM incorporates analyses and findings from the other appendices, such as the Seeps and Creeks Investigation Report that is used for characterizing groundwater conditions and contributions. Comments on the technical soundness, reasonableness of the assumptions applied, and critical gaps are discussed in the sections below. Key comments are summarized in Table 1.

Table 1. Key Comments from the Technical Review

Brief Description of Comment	Section (Comment Number)
Lack of adequate groundwater monitoring data and application of post-Hurricane Florence data.	2.1 (#1) and 2.2 (#1 and #5)
The modeling applied insufficient extents for resurfacing groundwater, resulting in potentially underestimated loads to the river.	2.2 (#2 and #3)
Limited scope of atmospheric deposition modeling (e.g., only HFPO-DA; seemingly conservative application of October 2018 conditions; limited spatial extent)	2.1 (#4)
Lack of information about the extent, magnitude, and impacts of offsite PFAS groundwater and soil contamination that may continue to contribute PFAS to the river.	2.2 (#4) and 2.3 (#7)
Lack of information to characterize PFAS contamination of sediment in the Cape Fear River bed and riparian wetlands.	2.2 (#6) and 2.3 (#7)
Implementation timing and ongoing risks for untreated sources.	2.3 (#1 and #2)
Lack of information regarding the effectiveness of treatment technologies.	2.3 (#3)
Need for notification requirements regarding spills or other releases since no production related changes have been required to date.	2.3 (#5)
Concerns regarding discharges of Kuraray process wastewater shown to contain elevated PFAS concentrations.	2.3 (#6)

2.1 TECHNICAL SOUNDNESS

This section summarizes our concerns regarding the technical soundness of data that has been assembled and cited to support conclusions in Cape Fear River PFAS Loading Reduction Plan and supporting appendices.

1. Onsite groundwater sampling data used to estimate mass loading to the river is based on a single round of samples collected primarily post Hurricane Florence – four of the five well samples in Appendix A are from late October – early November 2018, while the hurricane occurred in September 2018 with over 12 inches of rain recorded in nearby Fayetteville during the hurricane. This rainfall (and associated infiltration) may have significantly impacted short-term groundwater

sampling data, thus the representativeness of the data used is in question, especially since no other sampling data for the wells were provided for comparison purposes.

2. Onsite and offsite groundwater (transport pathways 5 and 8) PFAS concentrations used for the mass loading model are not provided in Table 3 of the MLM report. Is there a reason why these were specifically excluded while all other transport pathways had concentrations provided? What are the concentrations that were used?
3. It is unclear how groundwater south of the plant between Old Outfall 2 and Georgia Bank Creek was handled. Was groundwater in this area included in the onsite or offsite groundwater mass loading calculations? What parameters were used in the evaluation of contributions to the river from this area?
4. Previously reported deposition contours for air emissions from the Fayetteville Works facility were used to quantify the atmospheric deposition load in the MLM (ERM, 2018). Estimated deposition rates were combined with the average Cape Fear River surface area and estimated residence time to estimate a mass loading from aerial deposition to the river. The deposition load to the river surface was only evaluated for a ~3.5 km segment of the river near the facility. Key concerns regarding the modeling analysis follow, and critical gaps in the overall study related to atmospheric deposition are discussed in the next subsection. Note that some information discussed here is presented in the atmospheric deposition modeling report (ERM, 2018).
    a. The atmospheric deposition modeling focuses solely on HFPO-DA (ERM, 2018). To estimate the atmospheric deposition load of other PFAS compounds (non-HFPO-DA) for the MLM, concentration ratios derived from well monitoring samples are applied. The report, however, lacks proof that ratios from well measurements are directly applicable to air concentrations. Indeed, the ratios are likely to be different as PFAS compounds volatility, airborne transport, and subsurface soil sorption characteristics are not linearly related (ITRC, 2018). Therefore, this is not a reasonable assumption given the lack of evidence. The report also does not describe how the air transport and deposition of other PFAS compounds (non-HFPO-DA) differs from that of HFPO-DA.
    b. The MLM applies expected not actual emissions from the facility for October 2018. The MLM does not thoroughly discuss how factors that influence variability in air transport and deposition (e.g., fluctuations due to weather) are addressed. It is unclear if the results applied represent a single month (i.e., October 2018) extrapolated to represent annual deposition or if annual deposition is characterized by modeling emissions, transport, and deposition over a multi-year period. If it is the former, the application of October 2018 seems to be conservative; simulations of PFAS deposition for May 2018 are more widespread compared to October 2018. According to Table C-1 the same emission rates are applied for both (May and October 2018) scenarios, which means the differences in the extent of deposition are due to atmospheric conditions. Application of conditions for a single month is not reasonable for evaluating the annual load and the MLM should account for variability in conditions that impact the load. If in fact the atmospheric deposition modeling used to inform the MLM simulated a multi-year period, the report should clarify the methods. In addition, it is important that the impacts of intra- and inter-annual variability are discussed, including fluctuating emissions from the facility (i.e., due to operations cycling) and weather (e.g., wind direction and speed).
    c. Dilution factors are applied to estimate resulting concentrations in groundwater wells surrounding the property for various atmospheric deposition scenarios, however, the approach assumes zero concentration in existing aquifer water. Thus, the resulting

groundwater concentrations presented are biased low. [Note this information does not seem to be applied in the MLM.]

5. It is noted in Section 2.1.5. of the "Seeps and Creeks" appendix that samples were collected to avoid inclusion of suspended solids. In the final bullet of Section 3.4 of the Outfall 002 Assessment report it is stated that no relationship between TSS and total or dissolved PFAS was found (although details of the analysis are not provided). However, this conflicts with the fact that elevated PFAS concentrations at Location 22 are attributed to sediment clogging the autosampler (Outfall 002 Assessment report). Sorption of PFAS compounds is complex because the compounds have a lipophilic head and a hydrophobic tail. Thus, a clear relationship to TSS is not expected. A relationship to organic carbon on a PFAS species-by-species basis is likely yet was not examined.
6. The MLM approximates loading rates for each pathway based on PFAS concentration and flow data. The validity of the results for certain pathways is impacted by sparse monitoring records. For example, only a single sample was applied to characterize the upstream load (Section 4.1), even though elevated PFAS levels have been observed in upstream waters such as the Haw River (Barnes, 2019). Using a single sample to estimate the long-term load is not sufficient and additional monitoring should be conducted to characterize the upstream load across various seasons and flow regimes. It is stated in Section 4.5 that all EPA 537 PFAS compounds did not originate from the site as these were present in intake water. Therefore, EPA 537 PFAS compounds were assigned a zero concentration for the MLM. It can be deferred (although it is not explicitly stated) that this finding is based on the single upstream sample. Additional sampling is needed to evaluate the potential contribution of EPA 537 PFAS from the site.
7. No explanation is provided as to why some EPA 537 PFAS sampling method substances are reported as "NS" – defined as compound was not analyzed for in collected sample(s) or sample was not collected. Due to the lack of monitoring for these compounds, the total PFAS concentrations and loads reported in the study may be an underestimate of actual total PFAS concentrations and loads.
8. The DVM Narrative Reports show that many of the collected samples applied in the MLM did not meet sampling protocols (e.g., due to exceeded hold time). In addition, there are several cases where the dissolved concentration exceeds that of the total concentration for a PFAS substance (Table 10 Analytical Results – Stormwater Sampling). These data quality concerns contribute uncertainty to the monitoring and modeling results.
9. Results from TestAmerica were pending from the Outfall 002 monitoring at the time the report was issued. Results presented are from the onsite Chemours lab. The report does not specify if the Chemours lab is approved through the Resource Conservation and Recovery Act (RCRA). The report and modeling should be updated to incorporate the TestAmerica records.
10. HFPO-DA reductions from 2017 and 2019 in the load to the Cape Fear River are presented in the HFPO-DA Loading Reduction Estimates report. For both 2017 and 2019 monitoring from a single day was applied to estimate a typical daily load, which was directly extrapolated to generate an annual load (by multiplying by the number of days per year). The river flow applied to compute the annual load estimate for 2019 was less than one-third of the river flow applied to compute the annual load estimate for 2017, which falsely skews (overestimates) the reported percent reductions in loading to the Cape Fear River. It is not reasonable to assume that monitoring from a single day can be used to compute an accurate annual load. Recent load estimates computed by CFPUA based on more frequent monitoring at Lock and Dam #1 are higher. The analysis

should be redone and samples from multiple monitoring events spanning various seasons and flows should be applied for characterizing baseline and current loads and associated reductions.

2.2 CRITICAL GAPS

1. Overall, there is a significant lack of site-specific data regarding groundwater conditions at the facility. The report indicates that a total of five monitoring wells were available and used in the mass loading evaluation, which is not nearly adequate for delineating site geologic/hydrogeologic conditions and groundwater impacts considering the three groundwater flow systems involved. The report also indicates that additional groundwater characterization work is planned/underway for the site, which should provide data to more accurately portray onsite groundwater impacts to the river and improve the representativeness of the loading model. Hydrogeologic characteristics were in many cases estimated based on literature values and/or empirical evidence – generic ranges for hydraulic conductivity were used from general hydrogeology references, and groundwater flow gradients were estimated from water levels in riverside wells and a river gauging level remote from the site. It is important to collect adequate site-specific data to use in developing a technically sound detailed hydrogeologic conceptual site model that encompasses all three groundwater flow zones identified at the site (perched zone, surficial aquifer, and Black Creek aquifer) for quantifying groundwater flow rates and volumetric discharges/mass loading to the river.
2. Using observed mass loading at Bladen Bluffs, the MLM was calibrated through the adjustment of the following parameters: hydrologic conductivity for the Upper and Lower portions of the Black Creek Aquifer, groundwater discharge length (i.e., area contributing resurfacing groundwater to the river), and an offsite gradient adjustment factor. The rationale for modifying the discharge area for groundwater during model calibration iterations (only 40% to 75% of the total area was used) is unclear – all groundwater in the three flow zones identified (perched zone, surficial aquifer, Black Creek aquifer) should eventually discharge to the Cape Fear River either via direct discharge (Black Creek aquifer) or via seeps and surface water. Clearly the onsite groundwater discharge area length is significantly under-represented as described in Table D-2 of the onsite groundwater flow estimate (2,900 feet), which results in an under-estimation of onsite groundwater discharge from the Chemours site to the river. The calibration process was used as the rationale for this reduced length, however, the calibration process should be constrained to accurately reflect site conditions. Assuming 100% discharge of the Black Creek aquifer to the river would increase discharge/mass loading to the river significantly.
3. Similar to the previous comment, groundwater upwelling to the river is assumed to be less than 100%. Based on a USGS report regarding groundwater flow in the Coastal Plain Aquifer System of North Carolina, some shallow groundwater in the area may resurface as baseflow to the Cape Fear River while some may resurface further downstream (Giese et. al., 1991); however, additional field information is needed to support this parameterization. The assumed aquifer thickness for offsite groundwater discharge to the river is not provided – what was assumed and what is the basis for the assumption? Finally, a hydraulic conductivity value of 2.55 x 10-4 m/s was used for calculating offsite groundwater discharge to the river; however much lower K values were assumed for onsite groundwater (Black Creek aquifer). It is reasonable to assume that offsite shallow groundwater across the river is from the same formation; why the difference in K values? This would underestimate the relative mass loading via onsite groundwater versus

offsite groundwater. In addition, the Black Creek aquifer is likely to be slightly thicker on the other side of the River as it is generally down-dip; was this taken into account?

4. The loading analysis excludes deposition to surrounding land (wet or dry) that is stored in offsite soils, transported to streams via erosion, and leached into groundwater. These mechanisms and associated loadings have yet to be properly quantified. An investigation for the DuPont Washington Works plant near the Ohio-West Virginia border found contamination from atmospheric deposition up to 20 miles from the plant (Zevitas and Zemba, 2018). It is plausible that air emissions at the Fayetteville Works facility were/are transported further than assumed in the loading analysis, deposited, stored in soils, and leached into groundwater that resurfaces as baseflow to the river. Wells exhibiting high levels of PFAS contamination opposite of observed groundwater pathways (e.g., wells on the east side of the river) support this concept (ERM, 2018). This also could explain why concentrations and loads of some PFAS compounds are higher at the Kings Bluff intake compared to Bladen Bluffs, specifically during June 2019 (Table 7-A and Table 7-B), but the MLM was only calibrated at the Bladen Bluffs intake located about five miles downstream of the facility. CFPUA analyzed the relationship between raw water total PFAS and river flow rate using 2019 monitoring records (Figure 2). Elevated PFAS concentrations occur during periods of low flow. Given the halting of the release of process wastewater by Chemours, the elevated concentrations are likely attributable to onsite and offsite groundwater, releases from sediment bed stores, and/or currently unidentified other point sources. Therefore, a critical gap in the current analysis framework is that the extent, magnitude, and impacts of offsite PFAS groundwater and soil contamination has not been evaluated. Releases of contaminated groundwater, diffusion from contaminated sediment, and erosion of contaminated soils may contribute PFAS to the CFPUA's intake water following the implementation of the proposed control strategies (Section 2.3). Additional offsite monitoring and modeling is needed to understand the long-term implications on downstream water quality.

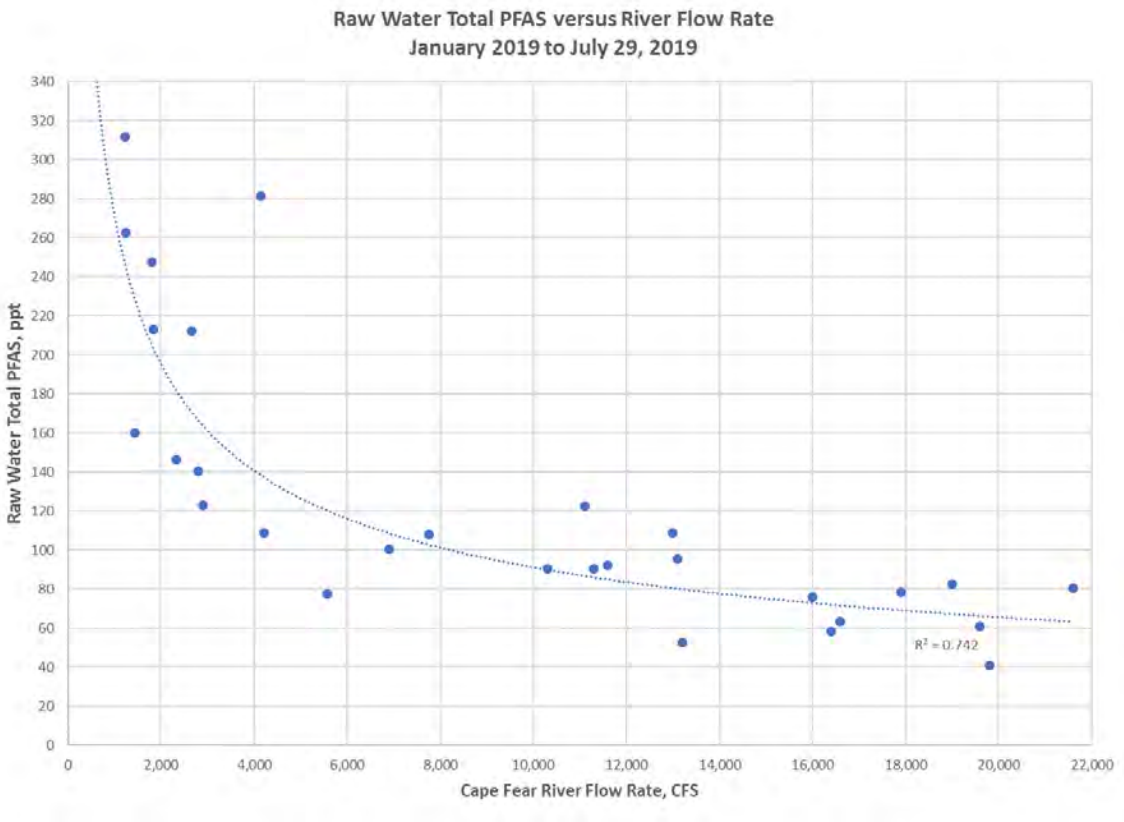

Figure 2. PFAS Concentrations and Cape Fear River Flow (provided by CFPUA)

5. For offsite groundwater where airborne deposition is considered to be the mechanism for PFAS transport to groundwater, prevailing wind directions should be utilized to estimate groundwater concentrations and mass loading to the river through offsite groundwater discharge to the river (see supplemental wind rose). For example, the predominant wind directions measured at nearby Fayetteville are from the southwest and from the northeast, which generally correlates with Figure E-2. For the area east and southeast of the site, however, there is very little data (few residential wells) and a review of Figure E-2 suggests that PFAS loading to groundwater in this area may be underestimated. The sampling data for wells west and northwest of the site (a much larger data set) could, however, be used to project/estimate groundwater concentrations/mass loading due to airborne deposition in the east-southeast area as the proportion of west and northwest winds (from west to east) is similar to/slightly higher than east/southeast winds (1998 – 2019 data). As currently configured, it appears that offsite groundwater mass loading to the river from east/southeast of the site may be underestimated.
6. A critical gap in the technical framework is that no sampling has been reported to characterize PFAS contamination of sediment in the Cape Fear River bed or riparian wetlands. It is anticipated that historic emissions and discharges from the facility have accumulated and caused long-term residual contamination of the river and riparian wetlands. Diffusion from such contaminant stores could provide a long-term source of PFAS contamination to the river. Scouring of contaminated sediment from the river bed or banks during high flow events could also elevate PFAS concentrations in downstream intake water. Sediment sampling along the mainstem should be conducted to characterize the extent and magnitude of sediment bed and riparian wetland contamination and the potential associated risks. Areas prone to excess build-

up of organic matter, such as sluggish riverine swamps and pools behind the locks and dams, face a higher risk of exhibiting elevated sediment PFAS concentrations. A comprehensive study is needed to characterize sediment PFAS contamination in the Cape Fear River bed that includes assessment of potential contamination hot-spots, such as the Kings Bluff intake canal situated near the Cape Fear River Lock and Dam #1. In addition, onsite sediment sampling has been sparse and should be extended to all concentrated surface flow pathways (e.g., open channel to Outfall 002).

7. A flow-based PFAS loading curve prepared by CFPUA for 2019 is shown in Figure 3. Higher PFAS loads are associated with higher flows, which indicates that stormwater and/or sediment bed erosion (as described in the previous comment) contributes PFAS to the river. Yet, these sources are poorly quantified, including both onsite and offsite stormwater contributions.

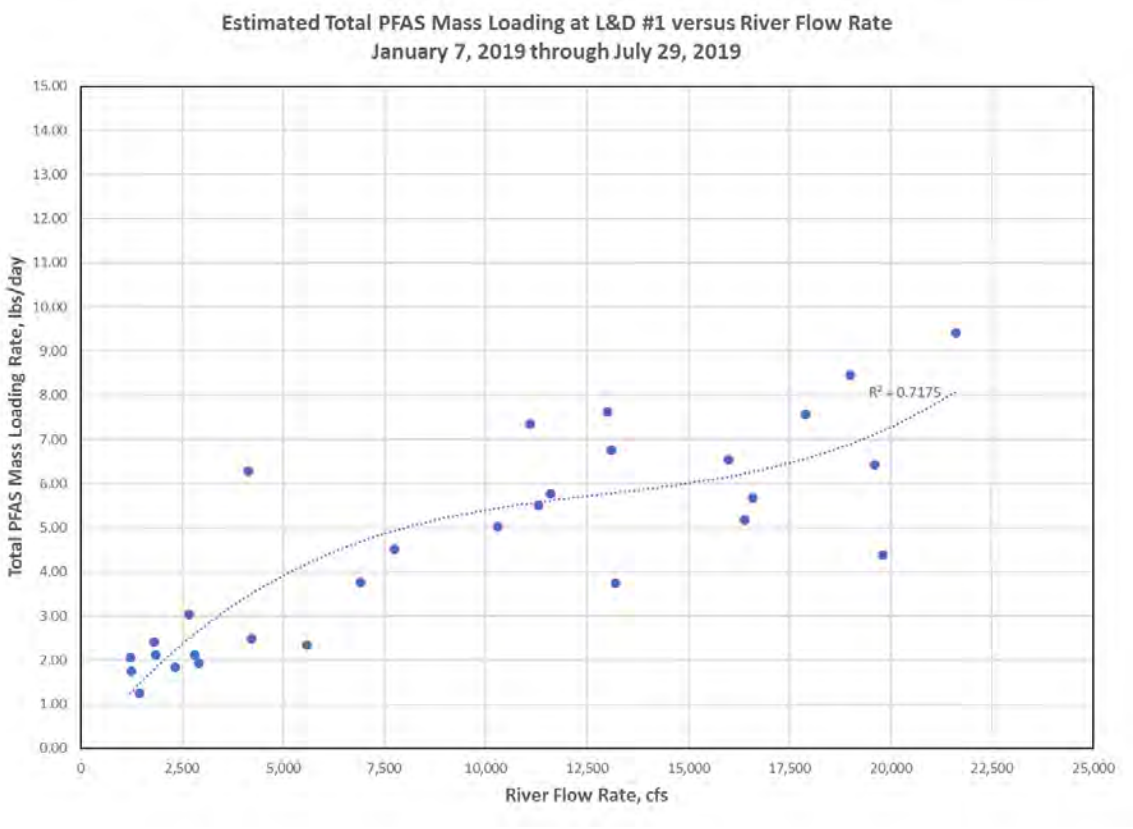

Figure 3. Flow-based PFAS Loading Rate (provided by CFPUA)

8. A mass balance evaluation of flow from the facility to the river is not provided in the Geosyntec (2019) report and is needed to verify the overall annual flow balance applied in the MLM. Such an evaluation should incorporate flow sources, storages, and discharges surface and subsurface discharges from the facility study area.
9. The possibility of additional diffuse discharges from the perched zone/shallow aquifer in other areas along the river should be investigated.

2.3 LOADING REDUCTION PLAN AND STRATEGIES

Chemours has previously implemented PFAS loading control measures: 1) eliminating process wastewater discharges (excluding those from site tenants Kuraray and DuPont), 2) air emission controls, 3) lining the facility's cooling water channel and sediment ponds, and 4) extraction of groundwater discarded offsite.

Seven new control strategies are proposed for the Chemours Fayetteville Works facility in the current plan (Geosyntec, 2019): 1) capture and treat Old Outfall 002 water (within two years), 2) capture and treat groundwater from seeps (within five years), 3) targeted sediment removal from conveyance network (within one year), 4) develop a stormwater pollution prevention plan (within one year), 5) targeted stormwater source control and/or treatment (within four years), 6) decommission and replacement of remaining terracotta piping (that carried industrial process wastewater; within two years), and 7) assessment of potential groundwater intrusion into the conveyance network (within five years). All proposed actions are to be implemented within five years and are onsite controls (on the Fayetteville Works property). Key comments regarding the plan and strategies follow.

1. It is stated on page v. regarding the control strategies that "Four of these actions would be implemented within two years of Consent Order Amendment and three of the actions would be implemented within five years of Consent Order Amendment (assuming all necessary permits and authorizations are provided in a timely manner)." Control actions may not be implemented on schedule due to the ambiguity of this statement, which poses a risk to downstream users.
2. The actions related to groundwater (#2 and #7) are set to take the longest time to implement yet are the top loading sources according to the MLM. Plans to evaluate and address groundwater and stormwater are still being developed, thus, loadings from these sources remain a vulnerability to downstream water supplies.
3. No specific treatment option is listed for captured onsite surface and groundwater, nor is the effectiveness of the proposed treatment methods demonstrated. Without these specifications it is uncertain if the loading reduction plan will effectively mitigate PFAS pollution. An onsite study evaluating the proposed treatment technologies and observed effectiveness (i.e., percent removal, treated concentrations and loads) should be required.
4. The onsite perched zone pumping described in the report (Section 3; Completed Reduction Actions) amounts to <0.1 gpm. Has there been any evaluation to determine whether the pumping rate can be increased via more aggressive pumping or additional groundwater extraction points to enhance capture of this highly impacted groundwater?
5. No manufacturing process changes have been required to date. Spills or unknown leaks or emissions at the facility remain a risk to CFPUA's source water. In paragraph 15 of the CO, Chemours is to provide notification to downstream water utilities in the event of elevated PFAS releases through Outfall 002. However, CFPUA should consider requesting spill (or other contaminant release) notification requirements that are more comprehensive.
6. Discharge of Chemours' process wastewater has been halted and the waste is injected into subsurface storage out-of-state. However, elevated HFPO-DA and PFMOAA concentrations were also observed in Kuraray process wastewater, which continues to be discharged from the onsite WWTP (page 18 of the Outfall 002 Assessment) via Outfall 002. Sources causing contamination of Kuraray process wastewater have not been identified and quantified. Furthermore, control strategies have not been required or proposed for the Kuraray process wastewater.

7. No PFAS loading control strategies are recommended for contaminated offsite soils, offsite groundwater, or river sediment due to the lack of evaluation of these sources (see Section 2.2). Additional strategies may be needed following the evaluation of these sources to ensure protection of downstream water quality.

All monitoring applied in the assessment appears to have been conducted by Geosyntec and contracted labs for Chemours. DEQ can require split sampling (samples provided to DEQ for parallel testing) per the Consent Order. Split sampling would be beneficial from the perspective of CFPUA for quality assurance and control checking, therefore, CFPUA should inquire about completed split sampling and the findings, or the rationale for why split sampling has not occurred to date.

3.0 REFERENCES

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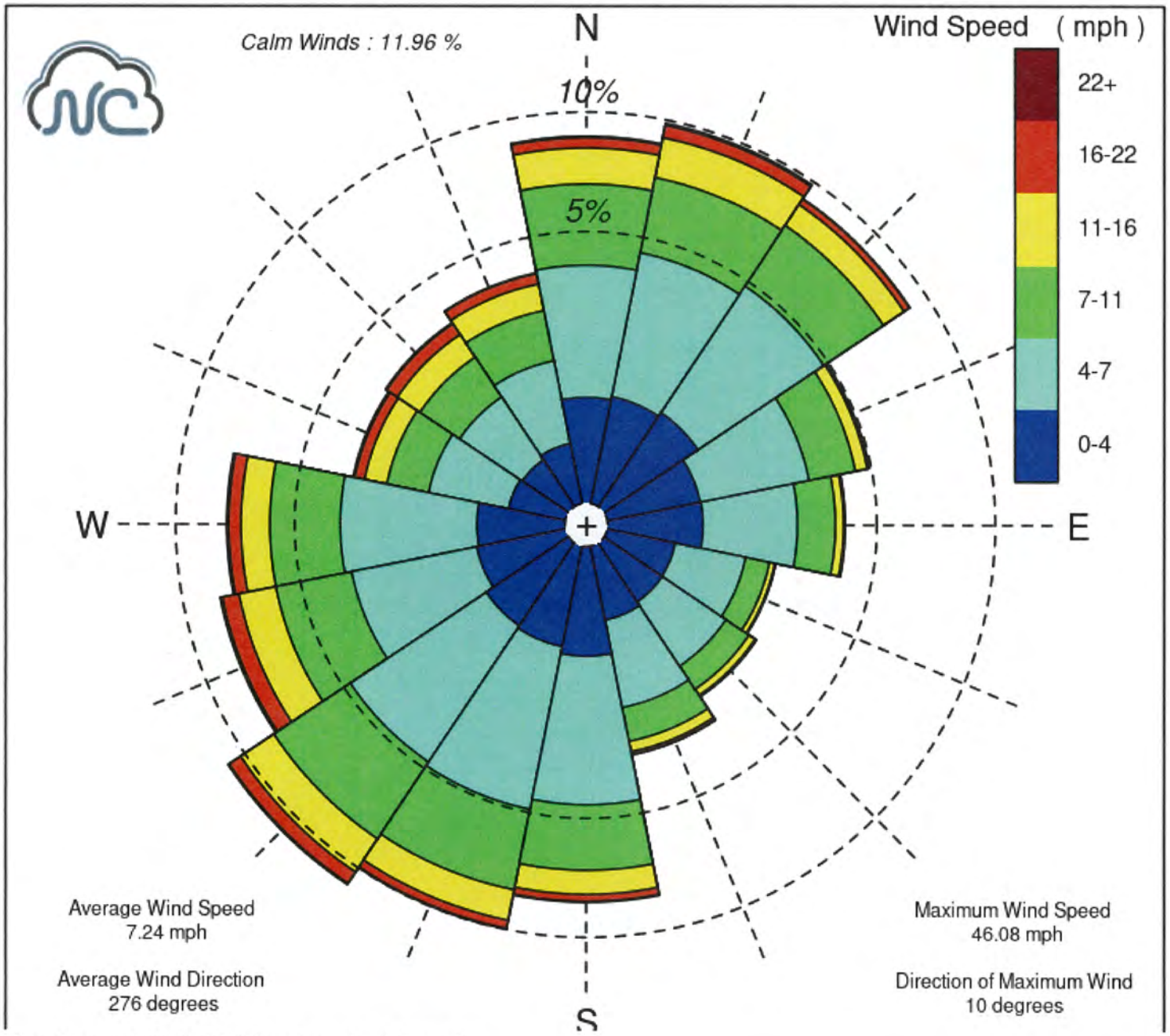
Wind Rose for Fayetteville Airport (KFAY)
Jan. 10, 1998 to Sep. 19, 2019



Shows wind blowing from a particular direction

U.S. Department of Commerce
National Oceanic & Atmospheric Administration
National Environmental Satellite, Data, and Information Service
Current Location: Elev: 192 ft. Lat: 35.1092° N Lon: -78.8944° W
Station: **FAYETTEVILLE 2.6 N, NC US US1NCCM0038**

Record of Climatological Observations

These data are quality controlled and may not be identical to the original observations.
Generated on 09/19/2019

National Centers for Environmental Information
151 Patton Avenue
Asheville, North Carolina 28801

Observation Time Temperature: Unknown Observation Time Precipitation: Unknown

Year	Month	Day	Temperature (F)			Precipitation					Evaporation		Soil Temperature (F)					
			24 Hrs. Ending at Observation Time		At Observation	24 Hour Amounts Ending at Observation Time				At Obs. Time			4 in. Depth			8 in. Depth		
			Max.	Min.		Rain, Melted Snow, Etc. (in)	Flag	Snow, Ice Pellets, Hail (in)	Flag	Snow, Ice Pellets, Hail, Ice on Ground (in)	24 Hour Wind Movement (mi)	Amount of Evap. (in)	Ground Cover (see *)	Max.	Min.	Ground Cover (see *)	Max.	Min.
2018	09	01																
2018	09	02				0.00												
2018	09	03																
2018	09	04				0.00												
2018	09	05				0.00												
2018	09	06				0.00												
2018	09	07				0.00												
2018	09	08				0.00												
2018	09	09																
2018	09	10				0.00												
2018	09	11				0.00												
2018	09	12				0.50												
2018	09	13																
2018	09	14				0.78												
2018	09	15				3.67												
2018	09	16				3.02												
2018	09	17				5.07												
2018	09	18				1.59												
2018	09	19				0.00												
2018	09	20				0.00												
2018	09	21				0.00												
2018	09	22				0.00												
2018	09	23				0.00												
2018	09	24				0.00												
2018	09	25				0.00												
2018	09	26				0.00												
2018	09	27				0.00												
2018	09	28				0.33												
2018	09	29				0.00												
2018	09	30				0.00												
		Summary				14.96		0.0										

Empty, or blank, cells indicate that a data observation was not reported.

*Ground Cover: 1=Grass; 2=Fallow; 3=Bare Ground; 4=Brome grass; 5=Sod; 6=Straw mulch; 7=Grass muck; 8=Bare muck; 0=Unknown

"s" This data value failed one of NCDC's quality control tests.

"T" values in the Precipitation or Snow category above indicate a "trace" value was recorded.

"A" values in the Precipitation Flag or the Snow Flag column indicate a multiday total, accumulated since last measurement, is being used.

Data value inconsistency may be present due to rounding calculations during the conversion process from SI metric units to standard imperial units.

NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT E
TO AMENDED INTERVENOR COMPLAINT

Technical Review of Cape Fear River PFAS Corrective Action Plan for Cape Fear Public Utility Authority (CFPUA)

February 28, 2020

PREPARED FOR

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1.0 EXECUTIVE SUMMARY

This report is a technical review of the Corrective Action Plan (CAP; Geosyntec, 2019a) for remediation of per- and polyfluoroalkyl substances (PFAS) discharged by the Chemours Company Fayetteville Works facility. Comments regarding the technical soundness of the assessments presented in the CAP and critical gaps are discussed in Section 3.0. The main concerns relevant to the Cape Fear Public Utility Authority's (CFPUA) downstream raw water intake are summarized below. Based on the information provided and information lacking, the adequacy of the modeling and CAP cannot be judged.

- The CAP and past reports use an **inconsistent application of PFAS analyte groups** for monitoring, loading analyses, and remediation planning (Section 3.1 #1). It is stated that, except for HFPO-DA, Modified EPA 537 method PFAS do not originate from onsite manufacturing; however, this is inconsistent with some process water samples presented in Characterization of PFAS in Process and Non-process Wastewater and Stormwater Quarterly Report #1 (Table 4, Location ID 16). Loads from the Modified EPA 537 method PFAS are excluded from the mass balance model. As a result, the model may underestimate PFAS loading from the site that impacts downstream water quality.
- The CAP **does not clearly define a baseline period**. The PFAS Loading Reduction Plan and CAP are also missing important information; relative contributions are presented by transport pathway, however, flows, concentrations, and loads to the river (mass of total PFAS per time) are not specified. Without a clear definition of the baseline period and loads, results could be interpreted in a manner that misrepresents progress and the effectiveness of remediation strategies (Section 3.1 #2).
- Multiple **technical issues related to the numerical groundwater model** are discussed in Section 3.1 #7 and Section 3.2 #2 that raise questions about the validity of the model and simulated remediation strategies. The model lacks a validation period to establish the robustness of the calibration. The report does not provide a rationale for the selection of proposed remedies and, based on the limited information provided, it is uncertain if the strategies will effectively capture and treat the PFAS-contaminated groundwater plumes.
- The onsite treatment strategies described in the CAP **neglect components of onsite pathways that may continue to contribute PFAS to the river** (Section 3.2 #1). The strategy specified for Old Outfall 002, for example, targets dry weather flows for treatment and excludes the treatment of wet weather flows that have the potential to transport contaminated sediment to the river. No creek-specific controls are planned for Willis Creek and Georgia Creek and no treatment plans are described for the newly identified seeps (E to M) south of the site. The effectiveness of the proposed treatment measures is uncertain and cannot be evaluated from the material provided in the CAP.
- There is a gap regarding the extent, magnitude, and **loading of PFAS from offsite contaminated soils and groundwater** that could act as long-term sources of PFAS to the river, continuing to impact the quality of raw intake water for CFPUA (Section 3.2 #1 and #4). PFAS contamination from Chemours has been detected in an area of 70 square miles (or more) surrounding the facility. However, because of the extent of the contamination, lack of scalable remediation technologies, and because no groundwater standards have been issued, it is claimed in the CAP that restoring groundwater conditions to PQLs is not feasible, which does not seem to comply with 2L Rules as required by the CO (paragraph 16). PFAS contamination of sediment in the bed and riparian wetlands of the river also remains uncertain. A comparative PFAS loading assessment just downstream of the site and at the CFPUA raw water intake is needed to evaluate offsite loading contributions to the river.

2.0 BACKGROUND

Chemours Company submitted the Cape Fear River PFAS Corrective Action Plan (Geosyntec, 2019a) to the North Carolina Department of Environmental Quality (NCDEQ) and Cape Fear River Watch (CFRW) on December 31, 2019, in response to the Consent Order (CO) entered by the Bladen County Superior Court (paragraphs 11.1 and 12) on February 25, 2019. The CO was issued regarding emissions and discharges of PFAS, including HFPO-DA and the ammonium salt of HFPO-DA, which has the trade name of GenX®, from the Fayetteville Works facility. GenX is used to manufacture high-performance fluoropolymers. GenX replaces the ammonium salt of perfluorooctanoic acid (PFOA), which was phased out of production in 2009 because PFOA is persistent in the environment, bioaccumulates, and is toxic. At that time the Fayetteville Works facility was owned and operated by E.I. du Pont de Nemours and Company (DuPont). The Chemours Company was founded in July 2015 as a spin-off from DuPont.

In 2009 EPA authorized the manufacture of GenX; however, EPA also issued an order that required DuPont to capture new chemical substances from wastewater effluent and air emissions at an overall efficiency of 99 percent (premanufacture notice numbers P-08-508 and P-08-509). News broke regarding elevated levels of GenX and PFAS in the Cape Fear River in 2017 – spurring further environmental investigations and facility inspections. Shortly thereafter, NCDEQ filed a Complaint alleging violations of Title 15A of the North Carolina Administrative Code Subchapter 02L .0202 Groundwater Quality Standards due to evidence of PFAS discharges by Chemours and DuPont, ultimately leading to the CO.

The Fayetteville Works facility is in Bladen County, North Carolina, on the west side of the Cape Fear River just upstream of the William O. Huske Lock and Dam (Lock and Dam #3). The facility includes two Chemours manufacturing areas, the Monomers IXM area and the Polymer Processing Aid Area (PPA area), as well as an onsite process wastewater treatment plant (WWTP) and power area (Geosyntec, 2019b). Manufacturing areas on the facility grounds are leased to Kuraray America Inc. for Butacite® and SentryGlas® production and to DuPont for polyvinyl fluoride (PVF) resin manufacturing.

The Chemours Fayetteville Works facility is located about 55 miles upstream of the Kings Bluff water intake on the Cape Fear River where the Cape Fear Public Utility Authority (CFPUA) withdraws water for treatment and potable use distribution. Elevated levels of PFAS have been observed in both the raw source water from the Cape Fear River and finished water at the CFPUA's Water Treatment Plants (WTPs). Traditional water treatment processes do not successfully remove GenX and other PFAS (Hopkins et al., 2018). The effectiveness of currently implemented and proposed PFAS pollution control strategies adopted by Chemours directly impacts the quality of CFPUA's intake water and community exposure to these substances.

Chemours submitted the Cape Fear River PFAS Loading Reduction Plan (Geosyntec, 2019b) in August 2019 and CFPUA engaged Tetra Tech to conduct a technical review of the report (Tetra Tech, 2019). The review evaluated the technical soundness of the modeling, the reasonableness of the assumptions applied in the analyses, the reasonableness of the proposed strategies for reducing PFAS loads, identified critical gaps, and recommended additional studies related to reducing PFAS loads. Comments most pertinent to CFPUA's downstream water intake included the lack of groundwater data, insufficient extents and lack of information about the extent, magnitude, and impact of offsite groundwater and soil contamination, lack of information necessary to characterize PFAS contamination in the sediment of the riverbed and riparian wetlands, and lack of information regarding the effectiveness of the proposed treatment measures.

A technical review of the CAP is presented in this report. The CAP describes site information, recent receptor monitoring details, a numerical hydraulic groundwater model, PFAS signatures source assessment, recent corrective actions summary, human health and ecological exposure and hazard assessments, proposed remediation activities by source pathway, and performance monitoring plans. The appendices relevant to the fate and transport of PFAS in the environment were also reviewed. This includes Appendix A - On and Offsite Assessment Tables; Appendix B - Additional Corrective Action Plan Tables and Figures; Appendix C - K_{ow}, K_{oc} and Mass Distribution Calculations; Appendix D - Southwestern Offsite Seeps Assessment; Appendix E - PFAS Signatures Assessment; and Appendix H - Numerical Groundwater Modeling Report. CFPUA plans to collaborate with expert Dr. Jamie Dewitt for elements related to human exposure and toxicity, as described in Appendix F - Human Health Screening Level Exposure Assessment of Table 3+ PFAS. The ecological assessment, discussed in Appendix G – Ecological Screening Level Exposure Assessment of Table 3+ PFAS, and Appendix I – Detailed Costs were not reviewed as part of the technical assessment described in this report.

3.0 TECHNICAL REVIEW

Key comments from the technical review of the CAP and supporting appendices are discussed in the following sections. The adequacy of the modeling and CAP cannot be evaluated due to the reasons summarized below.

3.1 TECHNICAL SOUNDNESS

This section summarizes concerns regarding the technical soundness of data and analyses cited to support conclusions in the Cape Fear River PFAS CAP and supporting appendices.

1. Information provided in the quarterly reports indicate that monitoring conducted aligns with specifications in the approved monitoring plan. However, results from the PFAS monitoring tests are inconsistently applied in the assessments. On page xii of the CAP, it states “The PFAS that originate from the Site are referred to as Table 3+ PFAS. The Table 3+ analytical method was developed to analyze PFAS specific to the Site that were identified through non-targeted chemical analyses. Currently, the Table 3+ method can quantitate for 20 PFAS compounds including HFPO-DA, i.e., “GenX”. When examining PFAS at the Site, the sum of these compounds, i.e., total Table 3+ PFAS compounds, is often used to evaluate trends and distributions.” However, in some analysis components Table 3+ PFAS are applied, in other components the assessment is limited to HFPO-DA, and sometimes Modified EPA Method 537 compounds are evaluated. This inconsistency hinders comparison between sources and components of the study (i.e., not always apples-to-apples). Example instances and impacts of this are described below.
    - The CO specifies the PFAS to be monitored for public drinking water and private wells (paragraphs 19-21 and 24) in Attachment C. According to paragraph 11 in the CO, ongoing sampling for process and non-process wastewater and stormwater at the facility is to be conducted for “all” PFAS for which test methods and lab standards have been developed, although these are not explicitly listed. The results described in the quarterly reports seem to include the Table 3+ PFAS and Modified EPA 537 PFAS for most sites, which matches specifications in the monitoring plan. Chemours claims that the Modified EPA 537 PFAS (excluding HFPO-DA) did not originate from the site as these were

already present in the intake water. Modified EPA 537 PFAS other than HFPO-DA are assigned a concentration of zero for onsite transportation pathways in the PFAS mass loading model. However, based on analytical results from the April 2019 monitoring event described in Chemours' first quarterly report, other PFAS (e.g., Perfluoropentanoic Acid) were found in process water from the Chemours Monomers IXM Area (site 16, page 3 of Table 4) at much higher concentrations than found in the background/intake water (later monitoring reports do not include samples from process wastewater). This suggests that some of the other Modified EPA 537 PFAS may originate from manufacturing on the site, but Modified EPA 537 PFAS (except for HFPO-DA) are excluded from the mass loading model and assessments discussed in the CAP (e.g., PFAS signatures). Therefore, it is unclear if the approach abides by the CO requirements and if the approach characterizes PFAS loads from the site accurately. Monitoring results, such as those from onsite and offsite groundwater wells, indicate that the relative proportions of PFAS compounds vary spatially, thus, it cannot be assumed that evaluating HFPO-DA in isolation is representative of other/total PFAS as has been assumed for atmospheric deposition modeling.

- Table 3+ and Modified EPA 537 PFAS methods exclude two PFAS listed in Attachment C of the CO, PFMOPrA, and PFMOBA, which are isomers that have the same chemical formulae as PMPA and PEPA, respectively, but have different chemical structures and CASN numbers. PFHpA listed in Attachment C is not included in the Table 3+ method, although it is included in the Modified EPA 537 method. Monitoring and assessments that are limited to Table 3+ PFAS exclude PFMOPrA, PFMOBA, and PFHpA from Attachment C of the CO.

2. Throughout the report and appendices, reduction targets are expressed as a relative percent reduction compared to an undefined baseline period. Appropriate quantification of the reductions achieved with the implementation of treatment technologies requires a clear definition of the baseline period and associated baseline loads for each PFAS transport pathway. In both the CAP and PFAS Loading Reduction Plan, baseline loading rates have not been specified; instead, relative percent contributions from the various onsite transport pathways are described (e.g., 22 percent for onsite groundwater in May 2019 as listed in Table 7 in the CAP). Without a clear definition of the baseline period and loads, results could be interpreted in a manner that misrepresents progress. For example, monitoring data from a single day were extrapolated to generate an annual HFPO-DA load. The river flow that was applied to estimate the load for 2019 was less than one-third of the river flow applied for 2017. This caused an overestimation of the reported reduction in loading to the Cape Fear River that was described in the technical review report for the PFAS Loading Reduction Plan. It is recommended that a) a clear and consistent baseline period is defined and b) for past and future monitoring events, that the flow, PFAS concentration, and load associated with each transport pathway should be presented.
3. Reductions for aerial deposition were estimated for HFPO-DA and the report states there are "expected comparable reductions for other PFAS", although information to justify this important assumption is lacking (e.g., measured pollutant removal efficiencies for other PFAS through the application of air control technologies). Indeed, differences in adsorption and volatility characteristics among PFAS compounds suggests that rates will differ. Previous comments regarding the atmospheric deposition modeling described in the technical review of the PFAS Loading Reduction Plan do not appear to have been addressed and, thus, remain a concern.

4. Although the analysis time period is not specified in the CAP, historical process water releases are estimated to account for 76 to 86 percent of the Table 3+ PFAS detected in the Cape Fear River with the remainder coming almost entirely from historic air emissions (14 to 24 percent). This implies that no significant loading of Table 3+ PFAS to the river originates from other background sources, although information is not presented to justify this assumption. As described in other comments, only the relative percent contributions are listed and actual load estimates are not presented (i.e., in mass of PFAS per time interval). It is also important to determine how both the magnitude and relative contributions of PFAS loads have shifted over time in response to halting releases of process water in 2017 and subsequent implementation of other control measures.
5. Figure 3 in the CAP shows the total Table 3+ PFAS mass distribution in a normalized volume of the unsaturated and saturated soil zones (kg/m^3). For several of the assessed locations (11 of 18), a result is not shown for the unsaturated zone because no Table 3+ compounds were detected (Table C-3); however, the text does not specify the detection limit.
6. The PFAS signatures assessment component of the CAP evaluated the make-up and distribution of PFAS compounds in onsite and offsite groundwater. Two main categories identified included 1) aerial deposition PFAS signature from emissions to air and 2) combined process water PFAS signature from historic releases of process water to soil and groundwater. The latter signature is only detected onsite, affects approximately 1 square mile, exhibits Table 3+ PFAS concentrations of 2,900 to 18,000,000 ng/L onsite, and is estimated to contribute 76 to 86 percent of Table 3+ PFAS loading to the river. The former (aerial) signature is detected on and offsite, affects >70 square miles, exhibits lower Table 3+ PFAS concentrations (15 to 13,000 ng/L onsite and 10 to 4,500 ng/L offsite) and is estimated to contribute 14 to 24 percent of Table 3+ PFAS loading to the river. Comments related to the PFAS signatures assessment are summarized below:
    - Three PFAS signatures were established for aerially deposited PFAS from a hierarchical cluster analysis. These include 1) predominantly PMPA (perfluoromethoxypropyl carboxylic acid); 2) predominantly HFPO-DA (hexafluoropropylene oxide dimer acid); and 3) mixed PMPA and HFPO-DA. Another signature, predominately PFMOAA (perfluoro-1-methoxyacetic acid), is described to be the signature representative of process water contamination. A physical/chemical/geological explanation for the distribution of the signatures is missing and a discussion regarding the interactions and transformations of PFAS (precursors to degradation resistant PFAAs (perfluoroalkyl acids) via abiotic or biotic mechanisms) over time is lacking, although the report generically states that transformation of most PFAS substances in the environment is negligible. For example, why is PFMOAA primarily associated with process waste contamination? Are there atmospheric transport mechanisms that influence the distribution of the aerial signatures? The rate at which rainfall scours a substance from the air will vary according to the Henry's law constant, which varies across the PFOA/PFOS substances in Appendix G, however, the CAP does not describe this phenomenon (note that the Table 2-3 in Appendix G lists the Henry's law constants and includes a footnote stating the estimates originate from the CAP, but that does not appear to be correct). This contradicts previous statements that claim atmospheric deposition modeling of HFPO-DA is directly applicable to other PFAS. What other biogeochemical transformations in the environment influence the distribution of the aerial signatures?

- The thresholds used to differentiate the signatures (e.g., what constitutes an aerial mixture signature versus a predominately PMPA or HFPO-DA aerial signature) is vague and should be explicitly described.
- The signatures assessment did not attempt to distinguish the portion of the PFAS signatures attributed to background, or non-Chemours, sources (e.g., biosolids applications, fire response chemicals, atmospheric deposition from other regional or global sources).
- The report does not describe how the findings from the signature assessment will inform future studies and remediation efforts.
- We suggest that the analysis could be improved and clarified through the application of a fugacity analysis with a model such as QWASI (Mackay et al., 1983) to determine the likely theoretical distribution of compounds of interest between air, soil, and water (e.g., Kong et al., 2018).

7. To simulate groundwater hydraulics, an EVS geologic model (seven hydrostatic and heterogenous units) and a FEFLOW 3D finite element groundwater model were developed for the site. Comments regarding the development and calibration of the numerical groundwater model (Appendix H) include:
   - As noted in the numerical groundwater modeling report, the subsurface hydraulic conductivity (K) values listed in Table 2 for the Surficial and Black Creek aquifers are well outside of the typical range presented in Table 1. Anomalous K values would have implications for the estimation of groundwater discharge and pumping rates. Were calibrations attempted with lower K values and, if so, what were the outcomes? Also, the model sensitivity test ranges for K (±20 percent) appear low given the modeled versus typical range values presented in the report. Were the much higher K values derived from the groundwater model calibration subsequentially incorporated into the contaminant mass loading estimates that were generated separately? If not, the mass loading flux to the river due to groundwater discharge may be significantly underestimated.
   - The numerical groundwater modeling report describes the data source for specifying the upper layer boundary (site precipitation and evapotranspiration estimates for the Mid-Atlantic Coastal Plain from USGS) but does not present the initial rainfall recharge rates used in the model. It is inferred from the wording that these served as initial rates that were adjusted during the model calibration, however, the final calibrated rates are not provided. On page 12 it is stated that the final hydraulic parameters are provided in Table 3, although Table 3 instead lists the final calibration statistics for the three zones (Perched Zone, Surficial Aquifer, and Black Creek Aquifer), not the hydraulic parameters.
   - It is stated that localized anthropogenic stormwater recharge (a second upper layer boundary in addition to rainfall recharge described in the previous bullet) and historic infiltration from previously unlined sedimentation basins is included in the top boundary condition. The sedimentation basins have been lined so it is unclear why the basins are assumed to contribute infiltration water to the Perched Zone for the simulation period of October 2019. In addition, the rate is presented as 80,000 GPD and this should be correspondingly presented as a depth-based rate (e.g., inches per day/month).
   - Bluff seep discharge rates were evaluated but the report lacks presentation of performance metrics. Based on the information provided (Table 6.2), the model underpredicts Cape Fear River bluff seeps by about 88 percent and overestimates Old

Outfall 002 flow by 60 to 140 percent (range provided for measured/estimated flow). Therefore, the model seems to provide a weak correlation of these outflow features although the implications are not discussed.

- It is not clear from the numerical groundwater modeling report and CAP whether the onsite seeps originate from the perched zone, surficial aquifer, or both – this is important information for the development of a groundwater remediation strategy. It is also unclear what groundwater flow unit the offsite seeps described in Section 3.5 of the CAP discharge from.
- There is no quantification of the groundwater flux into the river from each of the groundwater flow units included in the model. Such fluxes should inform the basis for developing groundwater extraction and treatment scenarios.
- The daily median water elevation for the Cape Fear River measured at the W.O. Huske Dam is used to set the hydraulic head for the eastern boundary condition. It is not stated if this is the median water elevation for October 2019 or another period, although the former is preferable for the steady-state application described.
- On page 13 it is stated that an overall error of 10 percent or less is considered acceptable for the intended application (although no reference is provided) and that the groundwater model achieves this target (overall and for the Surficial and Black Creek Aquifers). Contradictorily, the calibration resulted in a Normalized Root Mean Square (NRMS) error of 12.5 percent for the final groundwater model (Table 5). Therefore, the calibration effort did not achieve the target performance metric. Additional information regarding model performance and justification that the calibrated model is acceptable is needed. For example, it would be preferable to report performance metrics (such as NRMS) for each borehole calibration site to assess spatial variability in model performance. NRMS errors are presented for the three vertical zones, and the error for the Perched Zone is quite high, 25.2 percent – it is noted that additional calibration efforts may be required to improve the representation of hydraulics in this zone. It is also stated that the calibrated FEFLOW model meets the requirements of the NCDEQ 2007 Groundwater Modeling Policy, however, these are not presented or discussed. The first step in the guidance (Define Study Objectives) is not addressed – specific and detailed objectives are called for in the guidance but not provided in the modeling report, although these are critical for producing a technically sound and appropriate model.
- The model was calibrated for steady-state conditions in October 2019. It would be preferable to complete a model validation using monitoring and conditions from an alternative period to demonstrate that the calibrated parameters are robust and the model responds correctly to different conditions. This is important because, as discussed in Section 7, the model was run for a forecast period of 1 year for the purpose of evaluating remedy scenarios given that conditions vary throughout the year (e.g., precipitation and recharge, boundary condition hydraulic heads including the Cape Fear River).
- The rationale and logic behind the selection of remedy simulations is missing. The scenario set should be identified based on clear objectives and technical/hydrogeologic analysis. In Section 5.4 of the CAP, it is stated that the hydraulic containment objectives are presented in Table 8, however, the table lists a summary of the six predictive simulations without describing the objectives. For example, no information is provided about:

- The groundwater discharge rates to the river under ambient conditions from each hydrogeologic unit, which would be necessary to establish the minimum required pumping rates for plume capture.
- The expected unit-specific maximum sustainable pumping rates for extraction wells based on hydrogeologic analyses and calculations.
- The hydrogeologic units from which the extraction wells draw water. Is it just the Black Creek Aquifer or are the wells screened across the Surficial Aquifer too?
- Capture zone calculations for wells in the initial well placement scheme.
- The rationale behind groundwater extraction rates being selected for the different scenarios. For example, there is a scenario with 41 wells pumping at 20 gpm each (820 gpm total) and another with 31 wells pumping at 30 gpm (930 gpm total), although the Black Creek Aquifer groundwater discharge for each scenario is presented as 1551 gpm. If the pumping scheme extracts substantially less groundwater compared to the discharge rate, then the entire plume will not be captured.

- There is no information provided regarding the locations of the extraction wells nor the constraints on the placement of the extraction wells in Appendix H or Section 5 of the CAP. Shifting the wells back from the river will alter capture processes and impact the assessment of feasibility. The groundwater units that the extraction wells will capture water from is not clear in the documentation. Comparisons are made for the Black Creek Aquifer. It is unclear if the perched and surficial aquifers are also targeted.
- It is not clear what is represented in column 5 of Table 7, labeled “Black Creek Groundwater Capture Flow into the Cape Fear River – By Simulated Pumping (GPM)”. Manipulating the numbers in the other columns does not shed light on what the value is supposed to represent.
- It is unclear where the flow diverted by the groundwater barrier will go (e.g., will groundwater reemerge downstream of the wall terminus?). This should be described. It remains uncertain if a groundwater barrier to limit interactions between onsite contaminated groundwater and the Cape Fear River would be feasible and effective.

8. Comments related to the measured and calculated partition and mass distribution coefficients (Appendix C and Section 3.7 of the CAP) include:
   - In Section 3.7 it is stated that detailed calculations for the mass estimates are provided in Appendix C, however, Appendix C describes the process but does not include sufficient data/spreadsheets to verify the calculations.
   - In this appendix, Log K_{ow} values were used to derive Log K_{oc} values for various PFAS compounds. Contradictorily, in the 2018 Interstate Regulatory Technology Council (IRTC) guidance document “Naming Conventions and Physical and Chemical Properties of Per- and Polyfluoroalkyl Substances” it specifically states that “It should be noted that although the K_{ow} for some organic contaminants can be used for estimating K_{oc}, this cannot be performed for estimating values for PFAS”. This calls into question the technical approach used in Appendix C and the results obtained.
   - For HFPO-DA, the Table C-2 Log K_{oc} value is 1.1, while in Table 2 of the CAP it is 1.69. Which (if either) of these is correct and used for the calculations?
   - Throughout Table C-2, as the Log K_{ow} increases, the Log K_{oc} increases as well. This is true except when comparing PFBA and PFPeA – what is unique about these compounds? The specific calculations are not provided for review and evaluation.

9. In the monitoring well redevelopment and resampling section, it is stated that 17 wells were redeveloped onsite, and 45 wells were resampled onsite based on recommendations issued in the Onsite and Offsite Assessment Report. The CAP does not provide summary level statistics for the groundwater monitoring effort, which would be very informative (e.g., mean and range of concentrations observed).
10. As described in the updated PFAS characterization sampling plan for process and non-process wastewater and stormwater, the raw intake point onsite is used to characterize background PFAS levels. However, water from the Cape Fear River at the intake point may be influenced by legacy atmospheric emissions and contaminated groundwater attributable to the site. Samples collected further upstream are needed to better characterize background PFAS concentrations.

3.2 CRITICAL GAPS

1. Concerns regarding the planned strategies to meet the cleanup goals described in Table 10 in the CAP include:
    - **Old Outfall 002**. The cleanup goal and proposed capture and treat strategy are solely designed to handle dry weather flows, thus, wet weather flows that may facilitate erosion of contaminated sediment are excluded. Based on the three 2019 monitoring events (May, June, and September), the relative contribution of Old Outfall 002 is estimated to be 26 percent of the total onsite PFAS load to the Cape Fear River. In Table 14, 26 percent of the planned loading reduction to the Cape Fear River is attributed to the capture and treatment of Old Outfall 002. This implies that 100 percent of PFAS will be treated by 2020 for the outfall, which conflicts with only targeting groundwater with the process wastewater signature.
    - **Willis Creek and Georgia Creek**. Indirect air abatement controls and onsite groundwater remedies are listed as strategies, but no creek specific controls are planned (e.g., removal of PFAS elevated sediment, flow capture and treatment).
    - **Onsite Groundwater**. The cleanup goal for groundwater describes mitigation of PFAS with a process water signature, thus, inherently excluding remediation of onsite groundwater exhibiting an aerial deposition signature. As shown in Figure 2, some of the groundwater wells onsite exhibit the latter. Based on the three 2019 monitoring events (May, June, and September), the relative contribution of onsite groundwater is estimated to be 18 percent of the total onsite PFAS load to the Cape Fear River. In Table ES2, 18 percent of the planned loading reduction to the Cape Fear River is attributed to onsite groundwater treatment. This implies that 100 percent of PFAS in groundwater will be treated by 2024, which conflicts with only targeting groundwater with the process wastewater signature.
    - **Offsite Groundwater and Offsite Soils**. It is stated that PFAS contamination has been detected in an area of 70 square miles (or more) surrounding the facility. However, because of the extent of the contamination, lack of scalable remediation technologies, and because no groundwater standards have been issued, it is claimed in the CAP that restoring groundwater conditions to PQLs is not feasible. A lack of management of offsite pollution does not seem to comply with 2L Rules as required in the CO Paragraph 16. It is also stated that PFAS are not expected to degrade in a reasonable time period in the environment. This is a concern because contaminated soils and groundwater will contribute legacy PFAS to the Cape Fear River in the future, continuing to impact the

quality of raw intake water for CFPUA. PFAS loading just downstream of the site and at the CFPUA intake should be quantified and compared to better understand the potential for long-term contamination from offsite sediment erosion, resurfacing groundwater, and releases from sediment in the riverbed and riparian areas. The assessment should compare loading at the two locations under varied conditions (e.g., dry/low flow periods, storm events). Also, the CAP describes several newly identified seeps, labeled E to M, south of the site, although no treatment plans are prescribed.

- **Onsite Soils**. Contamination in onsite soils remains unclear and no remediation strategies have been suggested in the CAP.
- **Outfall 002**. The remediation strategies for Outfall 002 are too vague, stating that compliance with NPDES permit requirements will be completed. Information regarding the PFAS-related requirements that will be included in Chemours' NPDES permit should be requested from DEQ.

2. As discussed in Section 5.1 of the CAP, the groundwater numerical model is only intended to simulate subsurface hydraulic processes, not associated PFAS fate and transport, for the purpose of remedy costing and design. Therefore, in its current state, the model provides limited insight in terms of PFAS loading and potential remediation effectiveness. In addition, the groundwater model covers the limited domain of the site. Thus, groundwater hydraulics are not represented for the surrounding vicinity contaminated by PFAS due to legacy atmospheric deposition. Since offsite seep data is attributed to aerial PFAS deposition, it could be used to estimate groundwater PFAS discharges to the river throughout the area (including upstream and downstream of the site) by using a distance-versus-concentration gradient approach and including discharge from both sides of the river due to airborne transport processes. This analysis would be informative, although it is not discussed.
3. There is a very limited discussion of PFAS transformations in the environment and the implications for ongoing contamination, exposure risk, and remediation activity effectiveness (e.g., presence of precursors that can degrade to PFAS analytes over time). It is noted in Section 3.4, that total Table 3+ concentrations in wells are comparable to prior results (within ± 25 percent), however, temporal monitoring records have not been applied to explore transformations of PFAS, nor has available and relevant information from the literature been summarized.
4. As noted in the previous technical review, a critical gap is that the extent, magnitude, and impact (loading) of PFAS contamination in offsite groundwater and soils are poorly quantified. Releases of contaminated groundwater, diffusion from contaminated sediment, and erosion of contaminated soils may contribute PFAS to the CFPUA's intake water following the implementation of the proposed onsite control strategies. PFAS contamination of sediment in the Cape Fear River bed and riparian wetlands remains uncertain and diffusion from these stores could act as a long-term source of PFAS to the river. A river sediment sampling plan was issued in August 2019 and it is anticipated that monitoring will be conducted at several riverine locations, including near CFPUA's raw water intake site, and a report released in 2020.
5. At this time, a comprehensive flow mass balance that represents all inflow and outflows at the site has not been developed. It is stated in Section 3.4 of Appendix H that the numerical groundwater model will eventually be used to support the development of an initial water budget. However, this is a current information gap.
6. In the CAP, the onsite Willis Creek to the north and Georgia Branch Creek to the south are described as being erosional channels that empty to the Cape Fear River. PFAS accumulated in the creek beds that is eroded during storm events may contribute to ongoing PFAS loading to the

river, yet the report does not attempt to measure bed contamination and model sediment transport (net deposition and scour) for the purpose of characterizing particulate-associated PFAS transport. Note that deeper soil samples (depths of 8.5 to 11 feet) have been collected in the vicinity of Willis Creek at a single location (Figure A7-1). The results for the analytes reported were either flagged as "UJ" (defined as "Analyte not detected. Reporting limit may not be accurate or precise") or flagged as "<" (defined as "Analyte not detected above associated reporting limit").

7. It was noted in the technical review for the PFAS Loading Reduction Plan and the CAP (Section 3.3.3) that discharge of Chemours' process wastewater has been halted and the waste is injected into subsurface storage out-of-state. However, elevated HFPO-DA and PFMOAA concentrations were also observed in Kuraray process wastewater, which continues to be discharged from the onsite WWTP via Outfall 002, as discussed in the PFAS Loading Reduction Plan and previous technical review. Loading from Kuraray process wastewater remains unquantified and untreated.
8. Another gap, although perhaps minor, is related to process wastewater. Before June 21, 2017 process wastewater was discharged to the Cape Fear River and after November 29, 2017 process wastewater was captured, stored, and transported offsite for disposal. The report does not describe what was done with process wastewater in the interim, between June 22 and November 28, 2017.

3.3 OTHER COMMENTS

Other comments related to vulnerabilities pertaining to CFPUA's intake water include:

1. No manufacturing process changes have been required for Chemours to date. Spills or unknown leaks or emissions at the facility remain a risk to CFPUA's source water.
2. All monitoring applied in the assessment appears to have been conducted by Geosyntec and contracted labs for Chemours. DEQ can require split sampling (samples provided to DEQ for parallel testing) per the CO. Split sampling would be beneficial from the perspective of CFPUA for quality assurance and control checking, therefore, CFPUA should inquire about completed split sampling and the findings.

4.0 REFERENCES

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT F
TO AMENDED INTERVENOR COMPLAINT

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Evaluation of Maternal, Embryo, and Placental Effects in CD-1 Mice following Gestational Exposure to Perfluorooctanoic Acid (PFOA) or Hexafluoropropylene Oxide Dimer Acid (HFPO-DA or GenX)

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Background: Perfluorooctanoic acid (PFOA) is a poly- and perfluoroalkyl substance (PFAS) associated with adverse pregnancy outcomes in mice and humans, but little is known regarding one of its replacements, hexafluoropropylene oxide dimer acid (HFPO-DA, referred to here as GenX), both of which have been reported as contaminants in drinking water.

Objectives: We compared the toxicity of PFOA and GenX in pregnant mice and their developing embryo–placenta units, with a specific focus on the placenta as a hypothesized target.

Methods: Pregnant CD-1 mice were exposed daily to PFOA (0, 1, or 5 mg/kg) or GenX (0, 2, or 10 mg/kg) via oral gavage from embryonic day (E) 1.5 to 11.5 or 17.5 to evaluate exposure effects on the dam and embryo–placenta unit. Gestational weight gain (GWG), maternal clinical chemistry, maternal liver histopathology, placental histopathology, embryo weight, placental weight, internal chemical dosimetry, and placental thyroid hormone levels were determined.

Results: Exposure to GenX or PFOA resulted in increased GWG, with increase in weight most prominent and of shortest latency with 10 mg/kg/d GenX exposure. Embryo weight was significantly lower after exposure to 5 mg/kg/d PFOA (9.4% decrease relative to controls). Effect sizes were similar for higher doses (5 mg/kg/d PFOA and 10 mg/kg/d GenX) and lower doses (1 mg/kg/d PFOA and 2 mg/kg/d GenX), including higher maternal liver weights, changes in liver histopathology, higher placental weights and embryo–placenta weight ratios, and greater incidence of placental abnormalities relative to controls. Histopathological features in placentas suggested that PFOA and GenX may exhibit divergent mechanisms of toxicity in the embryo–placenta unit, whereas PFOA- and GenX-exposed livers shared a similar constellation of adverse pathological features.

Conclusions: Gestational exposure to GenX recapitulated many documented effects of PFOA in CD-1 mice, regardless of its much shorter reported half-life; however, adverse effects toward the placenta appear to have compound-specific signatures. https://doi.org/10.1289/EHP6233

Introduction

Perfluorooctanoic acid (PFOA) is a fully fluorinated, eight-carbon synthetic chemical belonging to the class of compounds known as poly- and perfluoroalkyl substances (PFAS). PFAS are used in a wide range of industrial processes and consumer products and are globally ubiquitous, persistent, and detectable in nearly all humans living in industrialized nations (ATSDR 2019; Kato et al. 2011). Although humans are exposed to PFAS through multiple routes, drinking water is one of the most well-understood sources of exposure (Hu et al. 2016).

Within the general U.S. population, serum levels of PFOA have declined from a geometric mean of 5.2 ng/mL in 1999–2000 (CDC 2009) to 1.56 ng/mL in 2015–2016 (CDC 2019). This shift is likely the result of efforts by the U.S. Environmental Protection Agency (U.S. EPA) to reduce environmental emissions and to phase out U.S. production and use of PFOA by 2015 (U.S. EPA 2006). Similarly, in 2017, the European Union placed restrictions on the production and use of PFOA (European Commission 2017). Despite such efforts, exposure to PFOA remains a concern due to its long human half-life ($\sim$3.5 y) (Olsen et al. 2007), environmental persistence (Lindstrom et al. 2011), and the fact that longer-chain/precursor PFAS chemicals can degrade and form PFOA. In response to restrictions on PFOA, manufacturers have increased production on replacement compounds with alternative chemistries aimed at making the compounds less bioaccumulative and with shorter serum half-lives; however, toxicity data for these alternative PFAS are limited (Bao et al. 2018).

Hexafluoropropylene oxide dimer acid (HFPO-DA), referred to herein as GenX, is a PFOA replacement compound. GenX has received intense public scrutiny in North Carolina since its discovery in (Strynar et al. 2015), and contamination of, the Cape Fear River Basin following release from a manufacturing facility (Sun et al. 2016). GenX has also been measured in the environment in other regions of the United States, including the Ohio River (Hopkins et al. 2018), as well as in other countries, including the Xiaoqing River in China (Brandsma et al. 2018) and the Rhine River in Europe (Heydebreck et al. 2015).

PFAS are detectable in the serum of pregnant women and in cord blood, and the ratio of the concentration of PFOA in maternal serum to cord serum is typically $\sim$1:1 (Kim et al. 2011; Monroy et al. 2008). Maternal exposure to PFOA has been associated with multiple adverse health outcomes, including increased gestational weight gain (GWG) (Ashley-Martin et al. 2016), pregnancy-induced hypertension (Darrow et al. 2013), preeclampsia (Savitz et al. 2012; Stein et al. 2009), and reduced birth

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weight (Apelberg et al. 2007; Fei et al. 2007; Johnson et al. 2014; Kobayashi et al. 2017; Lam et al. 2014; Rijs and Bogers 2017). Based on a systematic review of the literature and meta-analysis, the shift in birth weight associated with PFOA exposure has been estimated to be −18.9 g birth weight per 1-ng/mL increase in serum PFOA [95% confidence interval (CI): −29.8, −7.9] (Johnson et al. 2014).

In mice, the reproductive and developmental effects of gestational exposure to PFOA are well documented. Previous studies have shown gestational exposure to PFOA in mice results in maternal liver damage (Lau et al. 2006), maternal hypolipidemia (Yahia et al. 2010), and reduced embryo weight (Koustas et al. 2014). It has been estimated from a meta-analysis of data from eight mouse studies that the shift in mice is −0.023 g pup birth weight per 1-mg/kg body weight (BW)/d increase in PFOA dose to pregnant dams (95% CI: −0.29, −0.016) (Koustas et al. 2014). In contrast, there is a paucity of data regarding the reproductive and developmental effects of GenX. A previous reproductive and developmental toxicity study of GenX in CD-1 mice determined the no observed adverse effect level (NOAEL) for reproductive toxicity and maternal systemic toxicity (microscopic changes in maternal liver) was 5 mg/kg/d HFPO-DA (GenX; DuPont-18,405-1,037). A recent study in rats showed limited gestational exposure to HFPO-DA (GenX) resulted in a lowest observed adverse effect level (LOAEL) for disrupted maternal thyroid hormone (TH) (LOAEL: 30 mg/kg/d) and lipids (LOAEL: 125 mg/kg/d), up-regulated gene expression in peroxisome proliferator–activated receptor (PPAR) signaling pathways in both maternal and embryo liver (LOAEL: 1 mg/kg/d), and lower BWs in gestationally exposed female offspring (LOAEL: 125 mg/kg/d) (Conley et al. 2019). Additional studies examining the reproductive and developmental effects of GenX are needed.

The biological mechanism through which PFOA exerts adverse effects on embryo growth is not known, but the placenta is a suspected target tissue. The placenta is critical for embryo growth and development, and disruptions in placental development or function can lead to adverse outcomes for both maternal and embryo health. Previous animal studies have examined the effect of gestational exposure to PFOA on maternal mammary gland development and embryo growth (Macon et al. 2011; White et al. 2007), but effects on the placenta have yet to be evaluated. The aims of this study were to compare the effects of gestational exposure to PFOA and a replacement, GenX, on GWG, embryo growth, liver pathology, and placental development/morphology.

Methods

Animals

Naïve female CD-1 mice between 7.5 and 15.5 wk of age from the NIEHS colony were bred in-house on a single night, and copulatory plug–positive females were identified on embryonic day (E) 0.5. Pregnant dams were singly housed in ventilated polypropylene cages and received nesting materials, National Institutes of Health (NIH)-31 diet (Zeigler Bros., Inc.) and reverse osmosis deionized (RODI) water *ad libitum*. Animals were housed in humidity- and temperature-controlled rooms with 25°C and 45–60% average humidity and standard 12-h light cycles. All animal procedures were approved by the NIEHS Animal Care and Use Committee (ASP #2017-0022).

Dosing Solutions

PFOA ammonium salt (CAS #3825-26-1) was purchased from Millipore Sigma, and GenX [ammonium 2,3,3,3-tetrafluoro-2-(heptafluoropropoxy)propanoate; CAS# 62,037-80-3] was purchased from SynQuest Laboratories. PFOA and GenX dosing solutions were prepared in RODI water and administered to mice once daily via oral gavage. Daily doses were administered between 0700 and 0800 hours and adjusted to the BW of the mouse based on the previous day's weight at a volume of 0.01 mL/g BW. PFOA doses of 5 mg/kg BW/d (high dose) and 1 mg/kg BW/d (low dose) were selected based on previous work that demonstrated a reduction in neonatal weight gain (Lau et al. 2006; White et al. 2007). The dose of 1 mg/kg BW/d PFOA, used in the mouse developmental toxicity study of Lau et al. 2006, provided a lowest effect dose that was used to set the reference dose within the U.S. EPA's drinking water lifetime health advisory level (HAL) of 70 ppt PFOA (U.S. EPA 2016). Given that the state of North Carolina has a provisional health goal of 140 ppt GenX (double the PFOA HAL), we selected doses of GenX (10 mg/kg BW/d, high dose; 2 mg/kg BW/d, low dose) to mirror doses of PFOA previously used in HAL decision-making.

Study Design

This experiment was conducted over two blocks (Block 1 and Block 2) to achieve a total of $n = 11–13$ litters per treatment group and sacrifice time point (E11.5 and E17.5). The experimental design of the second block was identical to the first block of the study, and experimental methods were similar but expanded upon to include more rigorous and detailed measurements. Copulatory plug–positive mice (E0.5) were weighed to obtain a baseline BW and placed into one of five groups. Once all mice were assigned to groups, mean BWs were calculated, and a few animals were reassigned so that mean BWs in each group were similar. This was done to avoid confounding effects of baseline BW. Treatment groups were then randomly assigned a color by using a random sequence generator. Experimenters and dosing technicians were blinded to the treatment group to which the color groups corresponded throughout the duration of the study, including at necropsy. Randomly assigned treatment groups included in each block: vehicle control (deionized water only), 1 mg/kg BW/d PFOA, 5 mg/kg BW/d PFOA, 2 mg/kg BW/d GenX, and 10 mg/kg BW/d GenX. Pregnant dams were dosed via oral gavage from E1.5 to E11.5 or from E1.5 to E17.5. The sacrifice time points were selected *a priori* to examine effects of gestational PFOA or GenX exposure on embryo and placental growth prior to placental maturation (E11.5) as well as after full placental maturation (E17.5) (Watson and Cross 2005). The E11.5 early-gestation time point was selected because it overlaps a critical period of placental development in the mouse where the placenta undergoes vascularization with the uterine wall and chorioallantoic branching of vessels begins (Watson and Cross 2005). The E17.5 late-gestation time point was selected so that embryo weight changes that may be related to treatment would be evident.

Necropsy

On the day of necropsy, dams received daily oral gavage between 0700 and 0800 hours and were weighed and then euthanized humanely by swift decapitation, and serum was collected. In Block 1, necropsies were completed from 0800 to 1600 hours, and in Block 2, necropsies were completed from 0800 to 1200 hours. Serum from dams euthanized in Block 1 was snap frozen for internal dosimetry analyses. Serum and urine from Block 2 dams were reserved for clinical chemistry analyses. In both blocks, the uterus was removed, and total implantation sites were counted based on gross observation of an implantation nodule

along the uterine horn. Viable embryos, nonviable embryos, and sites of resorption were counted based on gross observation. Embryos were considered viable if they were properly formed, were not pale in color, and were of similar size to neighboring embryos. Embryos that were poorly formed and pale in color (without heartbeat) were considered nonviable. Sites of resorption were defined as a dark red–appearing clot-like nodule apparent on gross observation.

From each uterus, first, viable embryos and their matched placentas were collected in succession within a horn and immediately snap frozen ($n = 2$–5 per litter), and subsequent embryos were collected for growth measurements ($n = 2$–11 per litter). Additional placentas were collected and placed in 4% paraformaldehyde (PFA) for histological analysis (Block 2 only). Amniotic fluid was collected by needle aspiration from litters euthanized at E11.5 and snap frozen in liquid nitrogen. Embryo livers were collected from litters euthanized at E17.5 and snap frozen in liquid nitrogen. Dam livers were weighed, a portion of the left lateral lobe was placed in 4% PFA for histology, and another portion of the same lobe was snap frozen in liquid nitrogen. A third liver section was obtained from Block 2 dams and fixed in McDowell and Trump's fixative for electron microscopy (EM). Gross lesions were collected when observed and placed in 4% PFA for histology. Dam kidneys were removed, a cross section was prepared from the right kidney, a longitudinal section was prepared from the left kidney, and both sections were fixed in 4% PFA for histological analysis.

Tissue Preparation/Histology/Clinical Measures

Dam livers, kidneys, and placentas were trimmed and embedded by the NIEHS Mouse Embryo Phenotyping Core. Tissues collected at necropsy were fixed in 4% PFA for 72 h and paraffin embedded, and 5-μm sections were prepared and stained with hematoxylin and eosin (H&E). Pathology was evaluated and a pathology review conducted by S.A.E. Diplomate American College of Veterinary Pathologists (DACVP). Pathology reviews were conducted as an informed approach analysis [e.g., nonblinded analysis; see Sills et al. (2019)]. Select tissue slides were scanned using the AT2 Scanner (Aperio). Images were then captured for publication using the ImageScope software; version 12.3.0.5056 (Aperio). Serum and urine obtained from dams in Block 2 were analyzed using the AU480 clinical chemistry analyzer (Beckman Coulter Inc.). Reagents and calibration standards used to measure alkaline phosphatase (ALP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood urea nitrogen (BUN), serum creatinine, urine creatinine, glucose (Glu), total protein (TP), triglyceride (Trig), high-density lipoprotein (HDL), cholesterol (Chol), and albumin (ALB) were purchased from Beckman Coulter Inc. Reagents for sorbitol dehydrogenase (SDH), total bile acid (TBA), and micro-TP were purchased from Sekisui Diagnostics. The reagent used to measure low-density lipoprotein (LDL) was purchased form Diazyme Laboratories.

Transmission Electron Microscopy

Block 2 dam liver portions stored in McDowell and Trump's fixative (McDowell and Trump 1976) were processed using a Leica EM TP processor. Briefly, samples were rinsed with buffer, postfixed in 1% osmium tetroxide in 0.1-M phosphate buffer, rinsed in distilled water, dehydrated, and embedded in Ply/Bed® 812 (Polysciences, Inc.) epoxide resin. Blocks were trimmed, and semithin sections (~0.5 μm) were stained with 1% toluidine blue (Poly-scientific R&D Corp.) O in 1% sodium borate to ascertain areas of interest. Ultrathin sections (90–110 nM) were cut from areas of interest and placed on 200-mesh copper grids and stained with uranyl acetate and lead citrate, and digital images were captured using an Orius® SC1000 side mount camera (Gatan) attached to a Techani T12 transmission electron microscope (TEM) (FEI Company). In general, peroxisomes were smaller than mitochondria and round with a dark, electron-dense, granular matrix and surrounded by a single membrane. Mitochondria were round to elongated, had a matrix that was less electron dense than peroxisomes and contained crista, and were surrounded by an inner and outer membrane. Samples were analyzed by R.D.K., Ph.D.

Placental Thyroid Hormone Quantification

Thyroid hormones (T3, triiodothyronine; T4, thyroxine; rT3, reverse triiodothyronine) in placenta were analyzed according to the methods described in Leonetti et al. (2016). Briefly, ~300 mg (207– 526 mg) of two to three pooled placental tissues of same-sex embryos was homogenized and digested for 16 h overnight in PRONASE® Protease (*Streptomyces griseus*) solution (EMD Millipore Corp.). Each pooled sample of two to three placentas was considered as one biological replicate and included placentas from the same litter when possible. Three biological replicates were used for each treatment group and each sex. Samples were spiked with an antioxidant solution (containing 37.5 mg/mL each of citric acid, ascorbic acid, and dithiothreitol) and ^{13}C isotopically labeled internal standards (T4, T3, and rT3), and cold acetone was added to stop the digestion reaction. Samples were vortex mixed and centrifuged three times for 2 min at 10,000 relative centrifugal force (rcf), and the supernatants were collected and combined. Sample pH was adjusted with 6 M hydrochloric acid to pH < 2. A liquid–liquid extraction with cyclopentane was performed and the cyclopentane layer discarded; briefly, 1 mL of cyclopentane was added to the supernatant and vortexed before the sample was centrifuged for 3 min at 3,000 rcf and the cyclopentane layer discarded, and this was repeated three times. A liquid–liquid extraction with ethyl acetate was performed; briefly, 3 mL of ethyl acetate was added to the extract and vortexed before being shaken on a plate shaker for 30 min and centrifuged for 3 min at 3,000 rcf, and the ethyl acetate layer collected; this was repeated three times. Ethyl acetate extracts were dried down to 50 μL under a gentle nitrogen stream and resuspended in 1 mL of 0.01 M hydrochloric acid in 10% methanol. Samples were purified by solid-phase extraction using SampliQ Optimized Polymer Technology (OPT) cartridges (3 mL, 50 mg; Agilent Technologies). Final extracts in 400 μL of 1:1 methanol:water were filtered using Whatman® Mini-UniPrep® Syringeless Filters [Polytetrafluoroethylene (PTFE), 0.2 μm; GE Healthcare]. Extracts were analyzed on an Agilent high-performance liquid chromatography (HPLC) 1260 with a Synergi™ 50 mm × 2 mm Polar-RP column (2.5 μm; Phenomenex) coupled to an Agilent model 6460 tandem mass spectrometer with electrospray ionization (HPLC-MS/MS-ESI). Mobile phases consisted of 10 mM formic acid in methanol and 10 mM formic acid in water. Laboratory processing blanks were extracted alongside the placental tissues to monitor background levels. No TH were detected in the lab blanks. Method detection limits (MDLs) were calculated using a signal-to-noise value of 3 for each analyte (T3, T4, and rT3). Values were normalized to the wet weight of placenta extracted for a final value of nanogram hormone/gram placenta. Values below the MDL (T4, 0.84 ng/g; T3, 0.42 ng/g; rT3, 0.67 ng/g) were imputed using the calculation MDL × 0.5, and values lacking a quantifiable peak on mass spectrometry were excluded from the analysis.

Internal Dosimetry

Maternal serum, maternal liver, amniotic fluid, and whole embryos were analyzed for PFOA and GenX concentrations using methods similar to those previously reported (Conley et al. 2019; McCord et al. 2018; Reiner et al. 2009; Rushing et al. 2017). Solid tissues were homogenized in RODI water at a ratio of approximately 1:3 tissue mass (milligrams) to liquid volume (microliters). Maternal serum, amniotic fluid, and tissue homogenates (25 μL) were spiked with internal standard suspended in 0.1 M formic acid in a denaturation step, followed by a subsequent protein crash using ice-cold acetonitrile. Samples were vortex mixed after addition of formic acid and acetonitrile and then centrifuged at $10{,}000 \times g$ for 5 min. Extract supernatants were separated using a Waters ACQUITY UPLC® (Waters Corporation) fitted with a Waters ACQUITY UPLC® BEH C18 Column (130Å; 1.7 μm; 2.1 mm × 50 mm). Detection was performed using a Waters Quattro Premier™ XE tandem quadrupole mass spectrometer in negative ionization mode. Stable isotopes of PFOA ($^{13}C_3$, MPFOA; Wellington Laboratories) or GenX ($^{13}C_3$, M3HFPODA; Wellington Laboratories) were used as internal standards for quantification of vehicle control samples (run against a nine-point calibration curve of 0–100 ng/mL) and experimental samples (run against a nine-point calibration curve of 200–20,000 ng/mL). Vehicle control and dosed animal samples were quantified for both PFOA and GenX using respective isotope-labeled chemicals and calibration curves.

Embryo/Placental Growth Metrics

Gross observations were recorded at necropsy. Embryo sex was determined by polymerase chain reaction (PCR) amplification of the *Sry* gene (forward, 5′-GCTTCAGTAATCTCAGCACCTAGAA-3′, and reverse, 3′-CACATTGGCATGATAGCTCCAAATT-5′) using a snipped portion of tissue (TransnetYX®, Inc.). Embryos and their placentas were weighed separately as wet tissue. Images of embryos were obtained on a Leica Z16 APO imaging scope, and embryo length was measured as snout-to-rump distance using FIJI (Schindelin et al. 2012) and Zen 2 Blue (Zeiss).

Statistical Analysis

Data were analyzed in R (version 1.1.456; R Development Core Team). Sample sizes for each end point are reported in the accompanying figure legends or tables. A threshold of $p < 0.05$ was used for determining statistical significance unless otherwise noted. Analyses combining data from both experimental blocks were performed after verifying the absence of experimental block effects. Single-observation dam outcomes (e.g., liver weight, relative liver weight, implantation sites, resorptions, viable embryos, and internal dose metrics) were analyzed by analysis of variance using the lme4 (Bates et al. 2014) and lmerTest packages (Kuznetsova et al. 2017). Simultaneous tests for general linear hypotheses were corrected for multiple comparisons of means using Tukey contrasts in the package multcomp (Hothorn et al. 2009).

For all statistical tests adjusting for litter size as a fixed effect in the model, litter size was defined as the number of viable embryos. GWG on the day of sacrifice was adjusted for litter size using a general linear model. To compare GWG growth curves, GWG was measured as the percent change in BW compared to E0.5 and analyzed using mixed-effects models controlling for litter size and accounting for repeated measures of dams over time.

Embryo and placental metrics were analyzed using mixed-effect models and included *a priori* fixed effects of treatment group and litter size and a random-effects term for the dam using the lme4 package. Embryo and placental metrics included embryo weight, embryo length, placental weight, and embryo:placenta weight ratios, a meaningful predictor of fetal birth outcomes in humans (Hayward et al. 2016). To account for potential introduction of random effects, the study block (Block 1 or Block 2) and experimenter handling of embryo/placental tissues (Experimenter A or Experimenter B) were included as additional random effects. Models were fit in a stepwise procedure for random effects, and final models included treatment group and litter size as fixed effects using the lmerTest package (Kuznetsova et al. 2017). All final models included dam as a random effect but were allowed to vary in the inclusion of experimenter and experimental block random effects based on likelihood ratio test results. Point estimates and 95% CIs were determined from the final model using the Wald method. The number of individual observations for each outcome (embryo weight, placenta weight, and embryo:placenta weight ratio) and the number of litters evaluated in the mixed-effect models are shown in Table S1.

To document the effects of PFOA and GenX on the placenta, placentas were assessed for histopathological lesions in five to six litters per treatment group for both time points, with an average of seven individual placentas evaluated per litter. Analyses of histopathological data included placentas collected from viable embryos and excluded fused placentas and placentas collected from sites of resorption, which did not occur more frequently than at expected background levels in this strain. Histopathological lesions of evaluated placenta were evaluated using two statistical approaches. The first approach assumed the absence of litter effects and considered each placenta evaluated within a treatment group to be a totally independent observation, regardless of its litter of origin. These data were analyzed as counts using a generalized linear model with a Poisson regression using the package lme4 (Bates et al. 2014). The second approach considered the litter as the biological unit and compared the relative incidence of placental lesions [e.g., percent within normal limits (WNL)] to adjust for differences in the total number of observations across litters within and between treatment groups. These data were analyzed using a linear model. Both approaches were subjected to simultaneous tests for general linear hypotheses to correct for multiple comparisons using Tukey contrasts in the package multcomp (Hothorn et al. 2009).

TH concentrations in the placenta were quantified, and the ratios of T3:T4 and rT3:T4 in E17.5 placentas were assessed to evaluate potential disruption of peripheral TH control (e.g., impacts on thyroid deiodinase activity). Each end point was analyzed for sex × treatment interaction or for an overall effect of sex. Placenta TH were analyzed by analysis of variance using lme4 (Bates et al. 2014). Simultaneous tests for general linear hypotheses were corrected for multiple comparisons of means using Tukey contrasts in the package multcomp (Hothorn et al. 2009). Placental TH and their ratios were initially analyzed with embryo sex as an interaction term in the model, with the dose group as the predictor variable. Inclusion of a sex interaction or sex covariate in the final model was examined in a stepwise fashion. Internal dosimetry data were analyzed by analysis of variance. Simultaneous tests for general linear hypotheses were corrected for multiple comparisons of means using Tukey contrasts in the package multcomp (Hothorn et al. 2009).

Results

Internal Dosimetry

Maternal serum, maternal liver, amniotic fluid (E11.5 only), and whole-embryo dosimetry varied based on compound, dose, and time point. Urine collection was attempted at necropsy of pregnant

dams exposed to GenX but was unable to be consistently collected in sufficient volume for dosimetry analysis. Concentrations of GenX in the serum of dams exposed daily to 10 mg/kg of GenX was equivalent to the concentration of PFOA in serum of dams exposed to 5 mg/kg/d of PFOA at E11.5 (118.1 ± 10.4 μg GenX/mL serum and 117.3 ± 20.6 μg PFOA/mL serum, respectively; Figure 1A,B; Table S2). In contrast, GenX accumulation in the serum of dams exposed to 2 mg/kg/d GenX was 32% higher than the accumulation of PFOA in the serum of dams exposed to 1 mg/kg/d PFOA (33.5 ± 15.7 μg GenX/mL serum and 25.4 ± 3.7 μg PFOA/mL serum, respectively; Figure 1A,B; Table S2). Serum levels of either dose of PFOA or GenX measured at E17.5 were lower from those measured at E11.5 (Figure 1A,B; Tables S2 and S3). This could be explained by a dilution effect caused by blood volume expansion over the course of gestation or may be due to increased transfer to embryos over time.

Accumulation of PFOA in the maternal liver was greater than the accumulation of GenX, regardless of dose level or collection time point (Figure 1C,D; Tables S2 and S3). While maternal serum levels of PFOA or GenX were surprisingly roughly equivalent at E11.5 in dams exposed to PFOA or GenX, respectively, the accumulation of PFOA in the maternal liver was markedly higher in mice exposed to PFOA than the accumulation of GenX in liver of mice exposed to GenX (Figure 1C,D; Tables S2 and S3). It appeared that bioaccumulation of PFOA in the liver had reached a maximum of approximately 160–180 μg PFOA/g liver by E17.5 regardless of PFOA dose group (Figure 1C; Table S3). When comparing across low (1 mg/kg/d PFOA vs. 2 mg/kg/day/GenX) and high (5 mg/kg/d PFOA vs. 10 mg/kg/d GenX) dose groups at each time point, the fold change comparing GenX accumulation in the liver to the PFOA accumulation in the liver was 7.6-fold lower (2 mg/kg GenX vs. 1 mg/kg PFOA; E11.5), 8.9-fold lower (10 mg/kg GenX vs. 5 mg/kg PFOA; E17.5), 11.2-fold lower (10 mg/kg GenX vs. 5 mg/kg PFOA; E17.5), and 39.7-fold lower (2 mg/kg GenX vs. 1 mg/kg PFOA; E17.5) (Figure 1C,D; Tables S2 and S3). Unlike PFOA, GenX did not significantly bioaccumulate further in dam livers between E11.5 and E17.5 (Figure 1D; Tables S2 and S3).

Amniotic fluid concentrations of PFOA and GenX were roughly equivalent when comparing the accumulation in dams exposed at the high (5 mg/kg/d PFOA vs. 10 mg/kg/d GenX) and low doses (1 mg/kg/d PFOA vs. 2 mg/kg/d GenX) (Figure 2A,C; Table S2). Comparing across PFOA and GenX dose groups, embryo accumulation at E11.5 was greatest in mice exposed to 10 mg/kg/d GenX (3.21 ± 0.5 μg/g), followed by mice exposed to 5 mg/kg/d PFOA (2.34 ± 0.3 μg/g), 2 mg/kg/d GenX (0.91 ± 0.2 μg/g), and 1 mg/kg/d PFOA (0.80 ± 0.10 μg/g) (Figure 2B,D; Table S2). At E17.5, embryo accumulation was not different between sexes for either compound at the doses tested (Figure 2B,D; Table S3). Concentrations of PFOA or

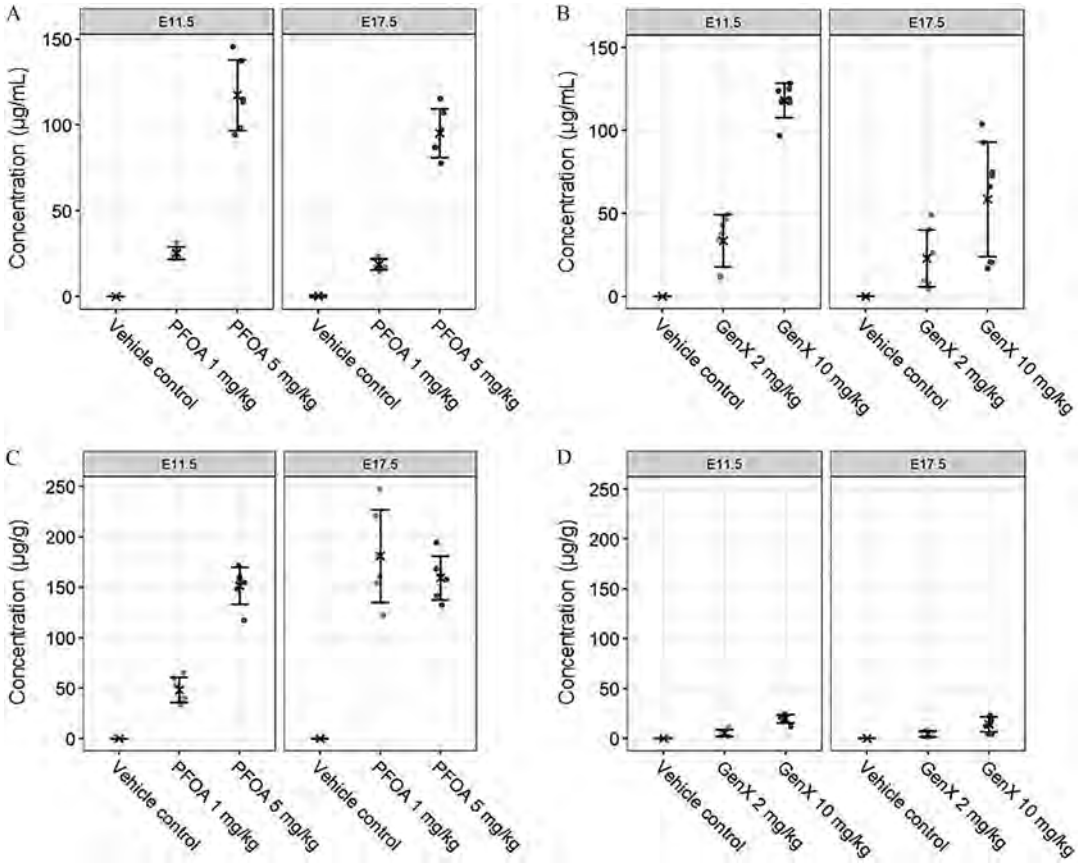

Figure 1. Internal dosimetry of perfluorooctanoic acid (PFOA) and GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] in maternal serum and liver at embryonic day (E) 11.5 and E17.5. (A) Maternal serum concentration (microgram PFOA per milliliter serum) at E11.5 and E17.5, (B) maternal serum concentration (microgram GenX per milliliter serum) at E11.5 and E17.5, (C) maternal liver concentration (microgram PFOA per gram liver) at E11.5 and E17.5, and (D) maternal liver concentration (microgram GenX per gram liver) at E11.5 and E17.5 were determined by high-performance liquid chromatography–tandem mass spectrometry. Treatment group mean values are denoted with an "X" flanked above and below by error bars showing standard deviation, and individual data points are shown as gray circles ($n = 6$–8). Vehicle control (VC) samples were quantified for PFOA and GenX; all VC means were below the limit of detection (LOD) of 10 ng/mL for both PFOA and GenX except for maternal serum (0.211 ± 0.55 μg/mL). Statistical comparisons of internal dosimetry across all treatment groups are shown in Tables S2 and S3.

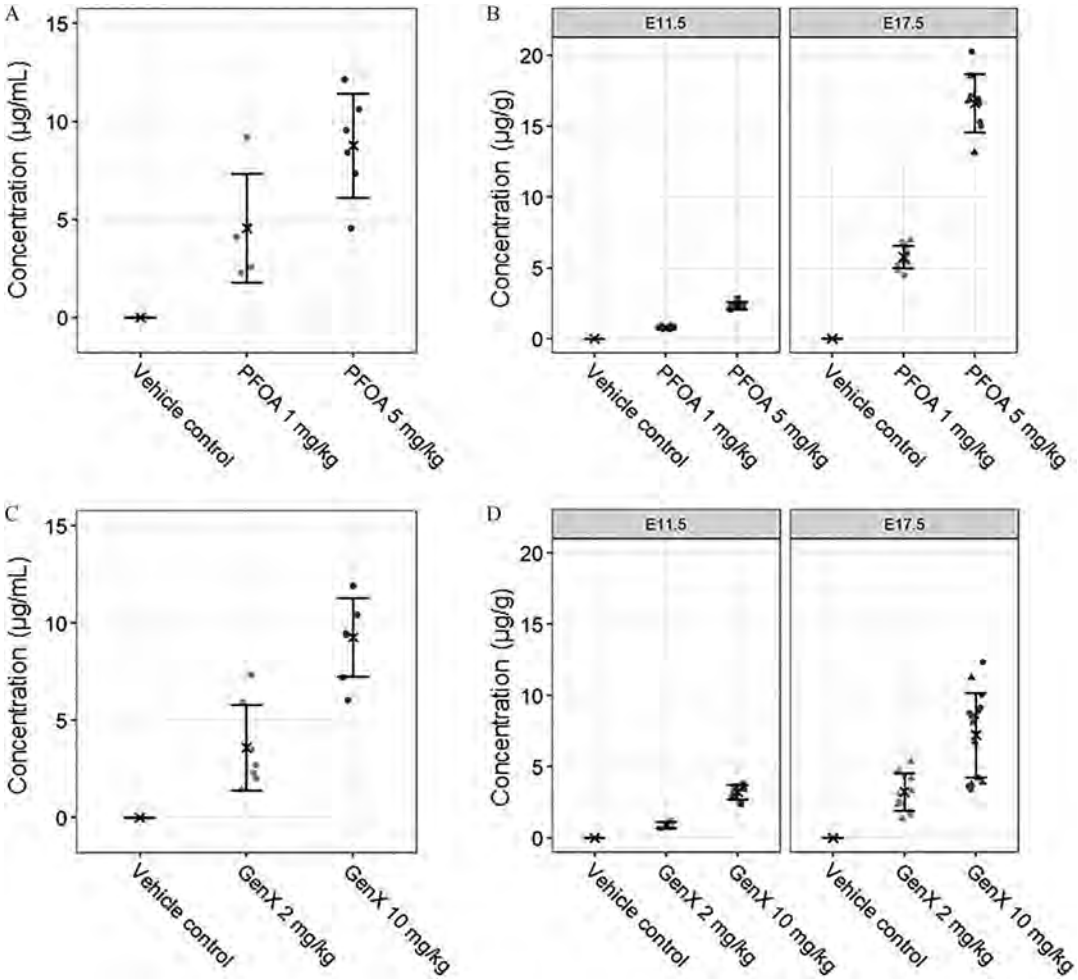

Figure 2. Internal dosimetry of perfluorooctanoic acid (PFOA) and GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] in amniotic fluid and whole embryos. (A) Amniotic fluid concentration (microgram PFOA per milliliter amniotic fluid) at embryonic day (E) 11.5, (B) whole-embryo concentration (microgram PFOA per gram embryo) at E11.5 and E17.5, (C) amniotic fluid concentration (microgram GenX per milliliter amniotic fluid) at E11.5, and (D) whole-embryo concentration (microgram GenX per gram embryo) at E11.5 and E17.5 were determined by high-performance liquid chromatography–tandem mass spectrometry. Treatment group mean values are denoted with an "X" flanked above and below by error bars showing standard deviation, and individual data points are shown as gray squares, circles, or triangles ($n = 6$–8). Triangles, E17.5 male embryos; circles, E17.5 female embryos; squares, pooled E11.5 embryos (B and D). Vehicle control (VC) samples were quantified for PFOA and GenX; all VC means were below the limit of detection (LOD) of 10 ng/mL for both PFOA and GenX. Statistical comparisons of internal dosimetry across all treatment groups are shown in Tables S2 and S3.

GenX in embryos were greater when measured at E17.5 than at E11.5, suggesting accumulation of both compounds over time in the embryo regardless of the shorter half-life of GenX (Figure 2B,D; Tables S2 and S3).

Maternal Outcomes

Gross anomalies were visually evident in some dams upon necropsy; excess abdominal fluid, edematous tissues, clotted placentas, and two fetuses attached to a single placenta were noted. However, these findings were unexpected *a priori* and thus were not looked for in each animal, were not reported by dose group, and require further investigation in future studies.

Mean dam BWs at E0.5 were similar across all treatment groups, including PFOA and GenX, for either sacrifice time point and did not differ from vehicle controls (Table 1). The relative change in dam BW from E0.5 to the time of collection (percent change in weight; GWG) was significantly greater after exposure to 10 mg/kg/d GenX at E11.5 (7.4% greater BW gain at E11.5 relative to vehicle controls; $p < 0.05$; Table 1). The number of implantation sites, viable embryos, nonviable embryos, and resorptions did not significantly differ among treatment groups, including PFOA and GenX, at either time point relative to the vehicle controls, although 10 mg/kg/d GenX-treated dams had fewer implantation sites and viable embryos at E17.5 (Table S4). When controlling for litter size, relative GWG was significantly greater than controls in 10 mg/kg/d Gen-treated mice (E11.5: 7.1% greater compared to controls; E17.5: 19.1% greater compared to controls) and 5 mg/kg/d PFOA-treated mice (E17.5: 14.5% greater compared to controls; Table S5). Effect estimates from mixed-effect models adjusting for repeated measures of relative GWG (dataset shown in Figure 3C), litter size, and gestational/embryonic day showed significantly higher relative GWG in mice exposed to 10 mg/kg/d GenX (E11.5 and E17.5) (Figure 3A,B), 2 mg/kg/d GenX (E17.5) (Figure 3B), and 5 mg/kg/d PFOA (E17.5) (Figure 3B).

Dam liver weights were significantly higher in all treated groups compared to vehicle controls at E11.5 (Table 1). At E17.5, absolute liver weights of dams were significantly higher in the 5 mg/kg/d PFOA, 2 mg/kg/d GenX, and 10 mg/kg/d GenX-treatment groups than in vehicle controls (Table 1). Dam relative liver weight (as a percentage of BW) was significantly higher in both PFOA and GenX treatment groups relative to vehicle controls at E11.5 and E17.5 (Table 1). At E11.5, vehicle

Table 1. Maternal indices at embryonic day 11.5 and 17.5 [mean ± standard deviation (SD); $n = 11–13$].

Embryonic day	Maternal index	Vehicle control	1 mg/kg BW/d (PFOA)	5 mg/kg BW/d (PFOA)	2 mg/kg BW/d GenX (HFPO-DA)	10 mg/kg BW/d GenX (HFPO-DA)
11.5	E0.5 weight (g)	30.6 ± 5.5	31.2 ± 3	31.1 ± 3.2	29.7 ± 2.2	30.7 ± 2.5
11.5	Weight at necropsy (g)	37.9 ± 4.3	38.8 ± 2.4	40.2 ± 3.5	38.3 ± 3.2	40.0 ± 2.5
11.5	Weight at necropsy (% change from E0.5)	24.9 ± 9.2	24.7 ± 6.3	29.6 ± 6.3	28.9 ± 5.4	32.3 ± 9.6*
11.5	Liver weight (g)	2.2 ± 0.3	2.9 ± 0.2*	4.5 ± 0.5*	3.1 ± 0.2*	4.2 ± 0.5*
11.5	Relative liver weight (% BW)	5.9 ± 0.7	7.4 ± 0.5*	11.0 ± 0.9*	8.1 ± 0.5*	10.2 ± 0.7*
11.5	Kidney weight (g)	0.20 ± 0.01	0.20 ± 0.02	0.21 ± 0.03	0.22 ± 0.02	0.23 ± 0.06
11.5	Relative kidney weight (% BW)	0.53 ± 0.01	0.51 ± 0.04	0.51 ± 0.05	0.54 ± 0.04	0.52 ± 0.11
17.5	E0.5 weight (g)	30.5 ± 3.3	28.5 ± 3.8	29.1 ± 3.4	28.2 ± 3.5	28.7 ± 3.6
17.5	Weight at necropsy (g)	56.3 ± 5.6	54.6 ± 5.3	57.4 ± 6.0	55.4 ± 6.5	56.7 ± 5.5
17.5	Weight at necropsy (% change from E0.5)	86.0 ± 22.8	92.6 ± 17.1	98.7 ± 20.2	97.3 ± 15.2	98.5 ± 15.7
17.5	Liver weight (g)	2.7 ± 0.3	3.1 ± 0.4	5.3 ± 0.5*	3.5 ± 0.5*	4.6 ± 0.4*
17.5	Relative liver weight (% BW)	4.8 ± 0.3	5.6 ± 0.5*	9.3 ± 0.7*	6.3 ± 1.0*	8.1 ± 0.5*
17.5	Kidney weight (g)	0.21 ± 0.02	0.22 ± 0.04	0.24 ± 0.03	0.21 ± 0.02	0.25 ± 0.02*
17.5	Relative kidney weight (% BW)	0.37 ± 0.04	0.40 ± 0.04	0.40 ± 0.03	0.37 ± 0.02	0.43 ± 0.03*

Note: BW, body weight. $n = 6–8$ for kidney weight and relative kidney weight. *$p < 0.05$ relative to vehicle control [analysis of variance (ANOVA) with post hoc multiple comparison correction using Tukey contrasts].

control livers exhibited either normal hepatocellular features with uniform hepatocellular size and cytoplasmic glycogen or minimal centrilobular hepatocellular hypertrophy with decreased glycogen, consistent with pregnancy at this stage of gestation. At E17.5, vehicle control livers exhibited hepatocellular changes consistent with pregnancy at this stage of gestation (minimal to mild centrilobular hepatocellular hypertrophy with karyomegaly, increased mitotic figures, decreased glycogen, and increased basophilic granular cytoplasm (Figures 4A and 5A). Compared with their respective controls, all livers (100% incidence) from both PFOA- and GenX-treated dams at E11.5 and E17.5 showed a variety of adverse outcomes (Figure S1), including some degree of cytoplasmic alteration, characterized by varying degrees of hepatocellular hypertrophy with decreased glycogen and intensely eosinophilic granular cytoplasm (Figures 4C,E and 5C,E; Tables S6 and S7). As the severity increased, there was extension of the cytoplasmic alteration into the midzonal and periportal regions. Also, as the cytoplasmic alteration increased in severity, there was an observed decrease in mitoses and increase in apoptotic cell death (Figures 4E and 5E). A few livers from exposed animals also had focal regions of classic necrosis. Incidence of liver lesions and vacuolation are reported in Tables S6 and S7.

Histopathological liver findings from a subset of E17.5 dams, including all dose groups for PFOA, GenX, and vehicle controls for comparison, were further evaluated using TEM. All vehicle control livers exhibited normal ultrastructure for this stage of gestation. In the centrilobular regions with hepatocellular hypertrophy, there was abundant glycogen, prominent rough endoplasmic reticulum (RER) with abundant ribosomes, numerous lysosomes, and minimal vacuolation with vacuoles often containing remnant membrane material as myelin figures (Figures 4B and 5B). Livers from mice exposed to 1 mg/kg/d PFOA exhibited enlarged hepatocytes with increased cytoplasmic organelles consistent with mitochondria and peroxisomes, evenly dispersed glycogen, and small vacuoles in the centrilobular regions (Figure 4D) compared to vehicle controls. Livers from mice exposed to 5 mg/kg/d PFOA exhibited abnormal ultrastructure with abundant organelles consistent with mitochondria and peroxisomes, highly prevalent cytoplasmic vacuolation, reduced RER with fewer ribosomes, and less abundant glycogen (Figure 4F). Livers from mice exposed to 2 mg/kg/d GenX exhibited abnormal ultrastructure with enlarged hepatocytes containing more abundant cytoplasmic organelles consistent with mitochondria and peroxisomes, and vacuolation (Figure 5D). Livers from mice exposed to 10 mg/kg/d GenX exhibited abnormal ultrastructure with enlarged hepatocytes containing abundant organelles consistent with mitochondria and peroxisomes, and prevalent vacuolation often with remnant membrane material as myelin figures, abundant RER with few ribosomes present, and unevenly dispersed glycogen appearing as clustered clumps (Figure 5F). At the level of TEM, PFOA and GenX generally caused a variety of cellular alterations: increased vacuolation, increased numbers of cytoplasmic organelles consistent with mitochondria and peroxisomes, reduced glycogen stores and reduction of RER ribosomes (Figure S2). Marked clumping of glycogen was a unique observation in livers of mice exposed to 10 mg/kg/d GenX, likely a secondary effect due to abundant mitochondria, peroxisomes, and RER.

Kidney weights and relative kidney weights of dams exposed to either dose of PFOA or GenX did not differ from vehicle controls at E11.5 (Table 1). At E17.5, 10 mg/kg/d GenX-exposed mice exhibited higher kidney weight relative to vehicle controls (both absolute kidney weight and relative kidney weight) (Table 1). Kidney cross sections and longitudinal sections were histopathologically evaluated at E11.5 and E17.5 time points, and diagnoses were made with no threshold: cortical glomeruli; cortical and medullary tubules; papillary collecting ducts; parenchyma; and vascular tree including renal artery, interlobar artery, interlobular artery, arcuate artery, and renal veins. Kidneys from vehicle control and treated animals were histologically WNL.

Clinical Chemistry

Dam serum Trig levels were significantly lowered at E11.5 across all treatment groups compared to controls in a dose–response manner (5 mg/kg/d PFOA and 10 mg/kg/d GenX lowered Trigs by 58% and 61%, respectively; 1 mg/kg/d PFOA and 2 mg/kg/d GenX lowered Trigs by 37% and 43%, respectively; Table 2). At E17.5, dam serum Trigs were significantly lower in 5 mg/kg/d PFOA and 10 mg/kg/d GenX-treated mice (66% lower and 74% lower, respectively) (Table 3).

At E11.5, serum Glu levels in dams exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX were lower relative to controls (20% and 18% lower, respectively), but this shift did not reach statistical significance (Table 2; $p = 0.06$ and $p = 0.20$, respectively). By E17.5, serum Glu remained lower in 5 mg/kg/d PFOA-exposed mice and 10 mg/kg/d GenX-exposed mice, but this shift was also not statistically significant (Table 3; $p = 0.41$ and $p = 0.42$, respectively).

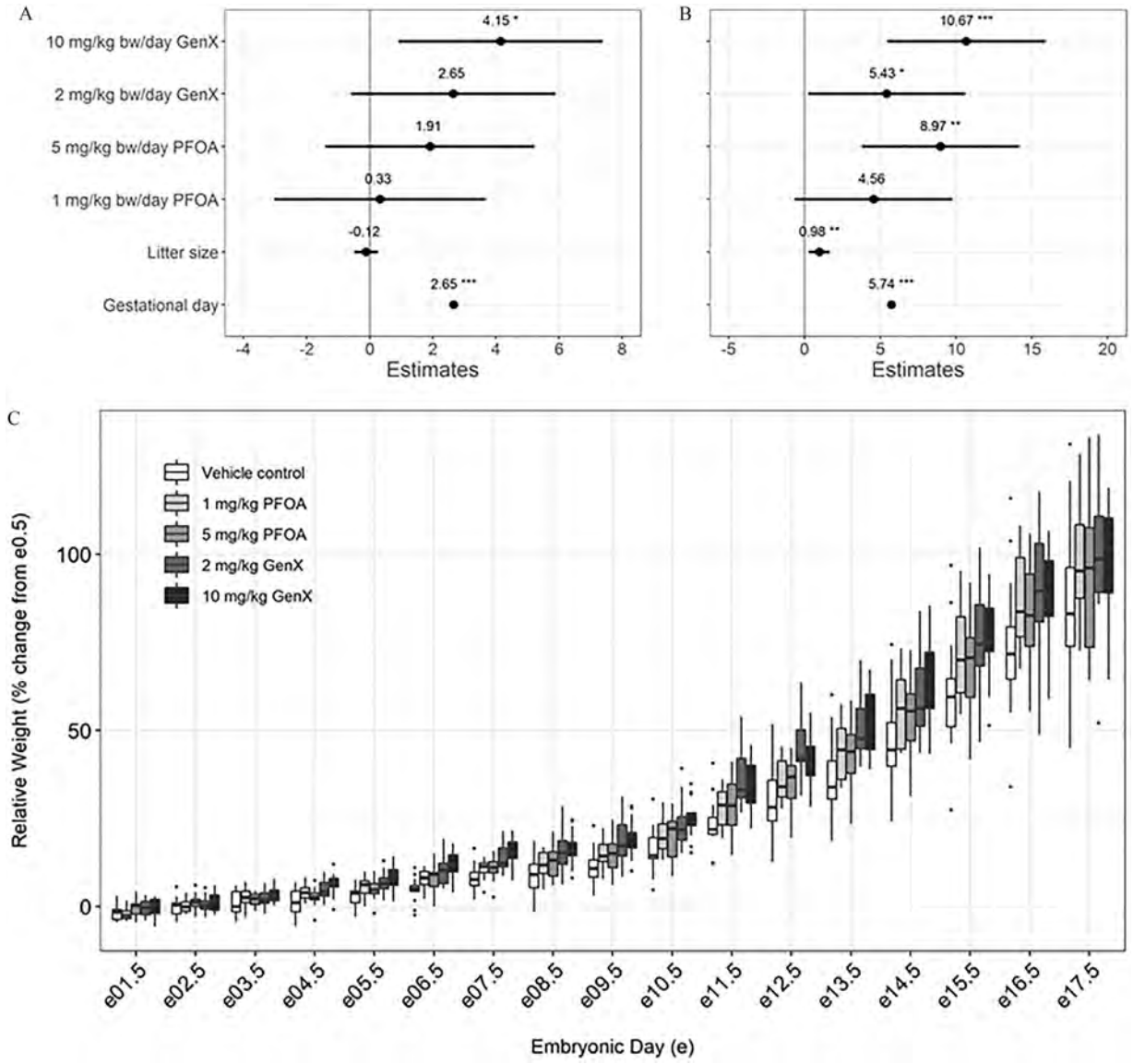

Figure 3. Gestational weight gain (GWG) repeated-measure, mixed-effect model estimates for pregnant dams exposed to perfluorooctanoic acid (PFOA) and GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)]. Effect estimates for pregnant dams exposed through embryonic day 11.5 (A) or 17.5 (B) are centered around the vehicle control group ($y = 0$) and show the point estimate of the relative change in dam weight (percent change from E0.5) with 95% confidence intervals (CIs). (C) Boxplots of relative weight gain over time, with the upper and lower hinges corresponding to the first and third quartiles (25th and 75th percentiles), the middle hinge corresponding to the median, and the upper whisker extending to the highest value that is within 1.5 times the distance between the first and third quartiles [interquartile range (IQR)] of the hinge and the lower whisker extending to the lowest value within 1.5 times the IQR of the hinge. $n = 11$–13 dams per treatment group. $^{*}p < 0.05$. $^{**}p < 0.01$. $^{***}p < 0.001$. Beta estimate 95% confidence intervals do not overlap zero. [Repeated-measures mixed-effect model adjusting *a priori* for litter size and gestational (embryonic) day as fixed effects and the dam as a random effect, vehicle control as reference group].

At E11.5, dams exposed to 2 mg/kg/d GenX exhibited higher Chol and HDL compared with controls (66% and 56% higher, respectively) (Table 2). E11.5 dams exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX similarly exhibited higher Chol and HDL levels relative to controls, but this shift did not reach statistical significance ($p = 0.42$ and $p = 0.42$, respectively) (Table 3). By E17.5, treatment-related effects on Chol and HDL appeared to be generally attenuated (Table 3). At E17.5, mice exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX exhibited lower LDL (50% lower and 31% lower, respectively), but only the shift in PFOA-exposed mice was significant (Table 3).

Dams exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX exhibited higher ALT relative to controls (a 172% increase and a 200% increase, respectively), but these shifts were not statistically significant with post hoc corrections (Table 2). By E17.5, treatment group–related effects on ALT were attenuated. At E17.5, dams exposed to 5 mg/kg/d PFOA exhibited lower serum ALB, increased AST, increased SDH, and lower total serum protein relative to controls (Table 3). Similar shifts occurred in mice exposed to 10 mg/kg/d GenX with respect to AST, SDH, and TP, but were not statistically significant (Table 3). Overall, GenX and PFOA liver pathology was consistent across dose groups and time points (100% incidence of cytoplasmic alteration) (Table S6 and S7), while changes in ALT, AST, and SDH measurements were not statistically significant across all GenX or PFOA dose groups or time points.

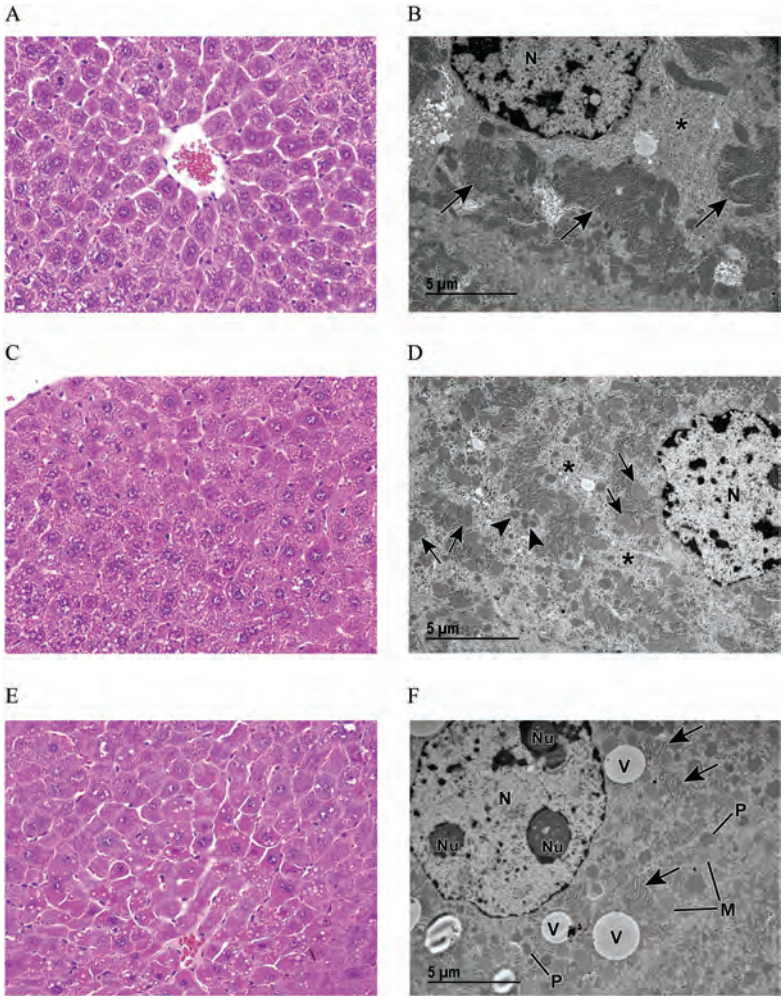

Figure 4. Light and transmission electron microscopy (TEM) of liver from vehicle control (VC) and perfluorooctanoic acid (PFOA)–exposed pregnant dams at embryonic day (E) 17.5. (A) Light microscopic image at 40× magnification of liver from a VC pregnant dam (control) showing centrilobular hepatocellular hypertrophy with karyomegaly, increased basophilic granular cytoplasm, and decreased glycogen. (B) Corresponding TEM magnification shows prominent rough endoplasmic reticulum (arrows) with abundant ribosomes and evenly dispersed, abundant glycogen (asterisk) (see Figure S2A). (C) Light microscopic image at 40× magnification of liver from a pregnant dam at E17.5 and treated with 1 mg/kg/d PFOA. (D) Although this liver appears to be within normal limits when viewed with light microscopy, TEM reveals an increase in scattered vacuoles (see Figure S2B); decreased, evenly dispersed glycogen (asterisks); as well as abundant mitochondria (arrows) and peroxisomes (arrowheads). (E) Light microscopic image at 40× magnification of liver from a pregnant dam at E17.5 and treated with 5 mg/kg/d PFOA. Increased cytoplasmic vacuoles are evident at this light microscopic level. (F) TEM reveals abundant cytoplasmic organelles consistent with mitochondria (M) and peroxisomes (P), extensive vacuoles (V), less prominent rough endoplasmic reticulum (arrows) with fewer ribosomes and less abundant glycogen (see Figure S2C,S2D). Note: N, nucleus; NU, nucleolus; TEM, transmission electron microscopy.

Embryo and Placenta Outcomes

Although the number of implantation sites, viable embryos, nonviable embryos, or resorptions did not significantly differ across treatment groups at E11.5 or E17.5 (Table S4), we evaluated embryos and their placentas for differences in weight. At E11.5, there were no significant differences in viable embryo weight, placental weight, or embryo:placenta weight ratios across treatment groups relative to vehicle controls (Table S8). At E17.5, significantly lower viable embryo weight was observed in 5 mg/kg/d PFOA-treated mice (5 mg/kg/d PFOA embryos were 129 mg lower in BW than vehicle control embryos based on mixed-effect model estimates; Figure 6A and Table S8). At E17.5, placental weight was significantly higher in 5 mg/kg/d PFOA- and 10 mg/kg/d GenX-treated mice relative to vehicle controls (an estimated 21 mg and 15.5 mg increase in placental weight relative to controls, respectively; Figure 6B and Table S8). Embryo:placenta weight ratios (mg:mg) were significantly reduced relative to controls in 5 mg/kg/d PFOA- and 10 mg/kg/d GenX-treated mice at E17.5 (Figure 6C and Table S8).

At E11.5, placental lesions were relatively sparse and mostly included labyrinth atrophy, labyrinth necrosis, or early fibrin clot formation. At E11.5, there were no differences in the incidence of placentas WNL across treatment groups (Table S9). At E17.5, placental abnormalities were observed in all treatment groups and tended to occur as litter-specific effects (e.g., most or all placenta within one litter were affected), and the most common lesions included labyrinth congestion (Figure 7B), labyrinth atrophy (Figure 7C), early fibrin clots (Figure S3A), labyrinth necrosis (Figure 7D), and placental nodules (Figure S3B). Placental nodules were most likely resorption of an adjacent twin. Placentas of mice exposed to 5 mg/kg/d PFOA exhibited labyrinth congestion as the most common lesion, whereas placentas of mice exposed to either 2 mg/kg/d or 10 mg/kg/d GenX primarily exhibited atrophy of the labyrinth (Figure 8 and Table S10). Early fibrin clots were most common in placentas of mice exposed to 10 mg/kg/d GenX (Figure 8 and Table S10). At E17.5, placentas WNL were significantly lower in mice exposed to 5 mg/kg/d PFOA or 10 mg/kg/d GenX when all evaluated

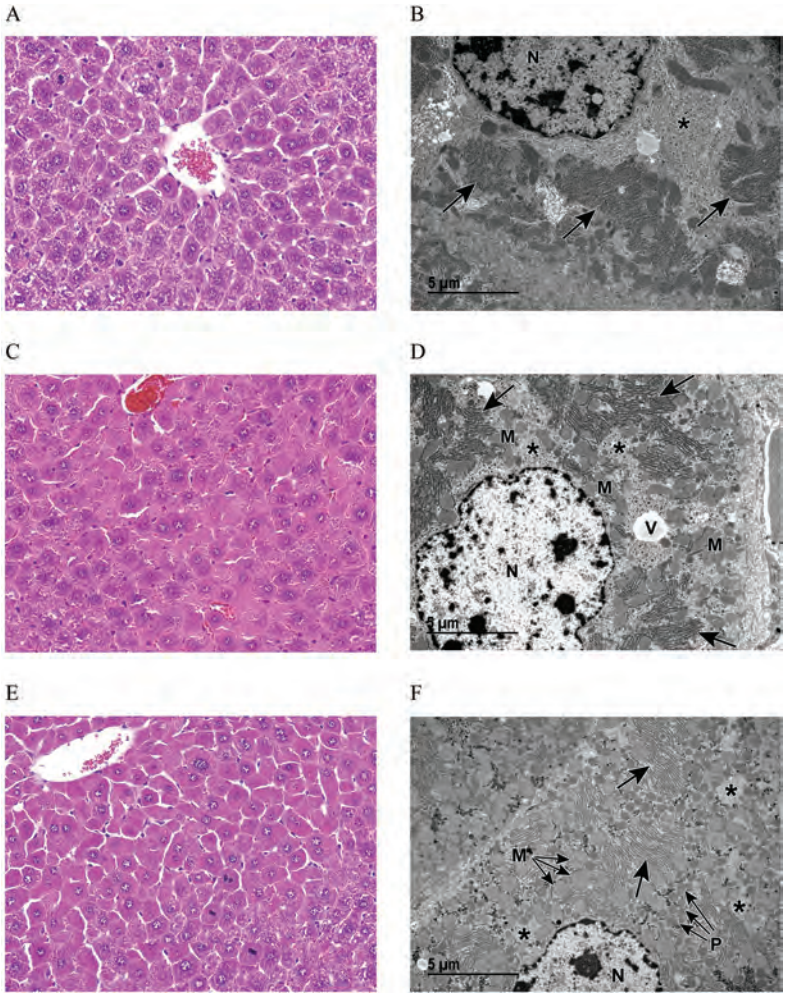

Figure 5. Light and transmission electron microscopy (TEM) of liver from vehicle control (VC) and GenX-exposed pregnant dams at embryonic day (E) 17.5. (A) Light microscopic image at 40× magnification of liver from a VC pregnant dam showing centrilobular hepatocellular hypertrophy with karyomegaly, increased basophilic granular cytoplasm, and decreased glycogen. (B) Corresponding medium TEM magnification shows prominent rough endoplasmic reticulum (arrows) with abundant ribosomes and evenly dispersed, abundant glycogen (asterisk) (see Figure S2A). (C) Light microscopy at 40× magnification, and (D) transmission electron microscopy of liver from a pregnant dam at E17.5 treated with 2 mg/kg/d GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] or 10 mg/kg/d GenX (E and F). Marked cytoplasmic alteration is evident in (C) and (E). TEM (D and F; see Figure S2E and S2F, respectively) reveals an abundance of cytoplasmic organelles, consistent with mitochondria (M) and peroxisomes (P) that increase with increasing dose (D compared to F). Note also the decreased glycogen (asterisks) as well as the vacuole (V) and rough endoplasmic reticulum (arrows). N, nucleus.

placentas were considered as independent observations (regardless of litter of origin) (Table S10). Placental lesions were also evaluated to account for litter effects by using the proportion of placenta within a litter that was WNL (percent WNL). Comparing placenta using this method showed a reduction in placenta WNL in mice exposed to 5 mg/kg/d PFOA, 2 mg/kg/d GenX, and 10 mg/kg/d GenX (Table S10).

Placental Thyroid Hormones

For all placental TH endpoints, sex × treatment interaction and sex as a covariate did not significantly influence model fit and were not incorporated in the final linear model (Table S11). Placentas exposed to 10 mg/kg/d GenX had significantly higher T4 relative to controls (60% increase) (Table 4). This effect occurred in both male and female placentas, but statistical significance was attenuated post hoc in sex-stratified models likely due to low sample sizes. There was a trend towards a significant effect of higher T4 in placentas exposed to 2 mg/kg/d GenX (38% increase; Table 4), but this effect was attenuated after applying post hoc corrections for multiple tests. Similarly, a trend toward a lower T3:T4 ratio was observed in placentas exposed to 10 mg/kg/d GenX, but this effect was attenuated after applying post hoc corrections. There were no other significant effects of sex or treatment on placental rT3, T3, T3:T4 ratio, or rT3:T4 ratio.

Discussion

Our prior work in mice has consistently shown reduced birth weight resulting from gestational exposure to PFOA (Macon et al. 2011; White et al. 2007), but we did not examine effects on the placenta, a critical organ that facilitates embryo growth, nor did we examine the effects of replacement PFAS congeners. Here we present evidence consistent with previous reports of PFOA-reduced embryo growth and provide novel evidence indicating that the pregnant dam liver and placenta are sensitive targets of both PFOA and a replacement PFAS, GenX. Adverse placental and maternal effects were most prominent in late gestation (E17.5) in mice gestationally exposed to 5 mg/kg/d PFOA and 10 mg/kg/d GenX, but 2 mg/kg/day GenX also exhibited significant effects on maternal liver and placenta. Future studies

Table 2. Clinical chemistry panel of dam serum at embryonic day 11.5.

Measurement	Vehicle control [mean ± SD (*n*)]	1 mg/kg/d PFOA [mean ± SD (*n*)]	5 mg/kg/d PFOA [mean ± SD (*n*)]	2 mg/kg/d GenX [mean ± SD (*n*)]	10 mg/kg/d GenX [mean ± SD (*n*)]
ALB (g/dL)	2.48 ± 0.18 (5)	2.42 ± 0.22 (5)	2.36 ± 0.21 (5)	2.75 ± 0.33 (4)	2.8 ± 0.17 (3)
ALP (U/L)	68.8 ± 13.0 (5)	54.6 ± 4.4 (5)	56.6 ± 35.6 (5)	58.4 ± 9.0 (5)	83.0 ± 25.8 (5)
ALT (U/L)	26.0 ± 5.6 (5)	28.8 ± 11.5 (5)	70.8 ± 16.2 (5)	24.2 ± 13.7 (5)	78.2 ± 62.0 (5)
AST (U/L)	63.6 ± 9.9 (5)	144.6 ± 167.6 (5)	92.6 ± 20.3 (5)	69.0 ± 22.0 (5)	136.8 ± 138.9 (4)
BUN (mg/dL)	16.0 ± 2.1 (5)	15.0 ± 2.7 (5)	15.8 ± 1.3 (5)	18.3 ± 4.6 (4)	13.7 ± 1.5 (3)
Chol (mg/dL)	56.4 ± 4.6 (5)	68.8 ± 18.0 (5)	69.4 ± 9.9 (5)	93.4 ± 27.8* (5)	77.0 ± 16.4 (4)
Cre (mg/dL)	0.21 ± 0.02 (5)	0.2 ± 0.05 (5)	0.18 ± 0.03 (5)	0.2 ± 0.04 (4)	0.18 ± 0.02 (3)
Glu (mg/dL)	275.2 ± 39.5 (5)	278.4 ± 27.8 (5)	220.4 ± 22.1 (5)	249.3 ± 25.8 (4)	226.7 ± 28.9 (3)
HDL (mg/dL)	32.2 ± 1.5 (5)	34.8 ± 10.9 (5)	42.6 ± 4.0 (5)	50.2 ± 15.7* (5)	43.3 ± 6.1 (4)
LDL (mg/dL)	10.8 ± 1.3 (5)	12.2 ± 1.9 (5)	10.6 ± 1.5 (5)	15 ± 4.8 (4)	12.5 ± 1.9 (4)
SDH (U/L)	9.4 ± 7.5 (5)	8.4 ± 7.8 (5)	12.4 ± 8.3 (5)	7.0 ± 6.5 (4)	8.0 ± 3.65 (4)
TBA (μM/L)	2.0 ± 0.71 (5)	1.5 ± 0.58 (4)	2.0 ± 0.0 (5)	1.4 ± 0.55 (5)	35.3 ± 67.8 (4)
TP (g/dL)	4.22 ± 0.18 (5)	4.04 ± 0.3 (5)	3.78 ± 0.22 (5)	4.5 ± 0.48 (4)	4.37 ± 0.29 (3)
Trig (mg/dL)	205.6 ± 56.0 (5)	130.4 ± 16.2* (5)	86.4 ± 15.8* (5)	117.6 ± 33.9* (5)	80.3 ± 14.4* (4)
Ucrea (mg/dL)	54.4 ± NA (1)	92.0 ± 13.1 (4)	50.1 ± 33.8 (4)	53.2 ± 14.0 (3)	82.9 ± 33.2 (5)

Note: ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; Chol, cholesterol; Cre, creatinine; Glu, glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; SDH, sorbitol dehydrogenase; TBA, total bile acids; TP, total protein; Trig, triglycerides; Ucrea, urinary creatinine; U/L, units per liter. *$p < 0.05$ relative to vehicle control [analysis of variance (ANOVA) with post hoc multiple comparison correction using Tukey contrasts].

should investigate adverse effects at doses lower than 2 mg/kg/d GenX to determine more precise percent responses at different lower dose levels using a benchmark dose approach.

It is well documented in humans and animal models that PFAS readily pass from maternal serum to the developing embryo via the placenta (Chen et al. 2017; Yang et al. 2016a, 2016b) and that PFOA transplacentally transfers to the mouse offspring (Fenton et al. 2009). Here, we report transplacental transfer of both PFOA and GenX, higher placenta weight, higher incidence of placental lesions, and lower embryo–placenta weight ratios in mice exposed to 5 mg/kg/d PFOA or 10 mg/kg/d GenX.

In humans, placenta weight and placental-to-fetal (also reported as feto-placental) weight ratios are clinically relevant end points that have been associated with adverse pregnancy outcomes (Hutcheon et al. 2012; Risnes et al. 2009; Thornburg et al. 2010). The placenta is a critical organ that mediates the transport of nutrients, oxygen, waste, and xenobiotics between mother and embryo, and it is rarely evaluated in reproductive toxicity studies. We chose the placenta as a focal end point due to its importance in studies of human pregnancy outcomes (Hutcheon et al. 2012; Risnes et al. 2009), its role as a programming agent of latent health outcomes in both the mother and child (Thornburg et al. 2010), and our own hypothesis that it is a key target tissue of PFAS.

Placental insufficiency (PI) occurs when functional capacity of the placenta is limited or deteriorates, resulting in reduced transplacental transfer of oxygen and nutrients to the fetus (Gagnon 2003). Reduction or impairment of placental blood flow (Chaddha et al. 2004), aberrant fibrin depositions or other thrombo-occlusive damage in the placenta (Chaddha et al. 2004), and disruption of maternal–placental THs (Belet et al. 2003) are all believed to contribute to PI pathogenesis in women. We provide evidence illustrating pathological and physiological features that are concordant with PI in our experimental mouse model. Here we show maternal exposure to PFOA- or GenX-induced atrophy, necrosis, and congestion of the murine placental labyrinth (suggestive of impaired transplacental transfer of nutrients and/or oxygen), aberrant formation of early fibrin clots, and disruption of placental TH (GenX only). These data are suggestive of a PI phenotype induced by maternal exposure to PFAS in mice that deserves further investigation.

In epidemiological studies, disproportionately large placentas increase the risk for adverse health outcomes in neonates (Hutcheon et al. 2012) and adult offspring (Risnes et al. 2009). The placenta influences cardiovascular disease (CVD) risk in the

Table 3. Clinical chemistry panel of dam serum at embryonic day 17.5.

Measurement	Vehicle control [mean ± SD (*n*)]	1 mg/kg/d PFOA [mean ± SD (*n*)]	5 mg/kg/d PFOA [mean ± SD (*n*)]	2 mg/kg/d GenX [mean ± SD (*n*)]	10 mg/kg/d GenX [mean ± SD (*n*)]
ALB (g/dL)	2.23 ± 0.21 (4)	2.04 ± 0.09 (5)	1.53 ± 0.27* (6)	2.32 ± 0.26 (5)	2.26 ± 0.3 (5)
ALP (U/L)	58.0 ± 7.8 (4)	50.2 ± 4.2 (5)	74.8 ± 23.8 (6)	55.4 ± 11.8 (5)	88.8 ± 13.0* (5)
ALT (U/L)	13.0 ± 7.5 (4)	7.0 ± 4.3 (5)	16.8 ± 7.7 (6)	4.4 ± 3.9 (5)	9.6 ± 2.1 (5)
AST (U/L)	81.0 ± 6.5 (4)	73.0 ± 14.0 (5)	172.2 ± 63.1* (6)	65.6 ± 12.1 (5)	113.2 ± 36.6 (5)
BUN (mg/dL)	16.0 ± 2.9 (4)	16.4 ± 1.7 (5)	18.7 ± 5.3 (6)	13.6 ± 1.1 (5)	15.2 ± 1.8 (5)
Chol (mg/dL)	75.5 ± 11.6 (4)	83.8 ± 20.0 (5)	68.5 ± 16.4 (6)	86.6 ± 17.1 (5)	97.4 ± 8.4 (5)
Cre (mg/dL)	0.18 ± 0.04 (4)	0.2 ± 0.01 (5)	0.16 ± 0.06 (6)	0.17 ± 0.03 (5)	0.15 ± 0.06 (5)
Glu (mg/dL)	129.3 ± 11.7 (4)	121.0 ± 17.3 (5)	112.0 ± 15.8 (6)	123.2 ± 13.1 (5)	111.6 ± 15.5 (5)
HDL (mg/dL)	34.0 ± 10.2 (4)	37.2 ± 6.2 (5)	38.8 ± 11.2 (6)	39.4 ± 8.5 (5)	50.0 ± 8.9 (5)
LDL (mg/dL)	22.0 ± 0.8 (4)	24.0 ± 10.7 (5)	11.0 ± 3.0 (5)	20.0 ± 3.9 (5)	15.2 ± 2.9 (5)
SDH (U/L)	5.5 ± 7.9 (4)	3.4 ± 6.1 (5)	24.3 ± 11.2* (6)	1.2 ± 2.2 (5)	11.4 ± 6.8 (5)
TBA (μM/L)	3.8 ± 0.96 (4)	3.0 ± 1.2 (5)	8.0 ± 7.9 (6)	4.8 ± 3.0 (5)	6.2 ± 4.2 (5)
TP (g/dL)	4.2 ± 0.37 (4)	3.9 ± 0.11 (5)	2.8 ± 0.39* (6)	4.1 ± 0.36 (5)	3.9 ± 0.52 (5)
Trig (mg/dL)	472.5 ± 78.9 (4)	364.0 ± 272.9 (5)	159.0 ± 65.5* (6)	257.0 ± 120.3 (5)	120.6 ± 31.7* (5)
Ucrea (mg/dL)	25.8 ± 15.8 (2)	24.7 ± 23.1 (2)	11.5 ± 5.9 (3)	18.6 ± 5.1 (4)	20.2 ± 15.7 (4)

Note: ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; Chol, cholesterol; Cre, creatinine; Glu, glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; SDH, sorbitol dehydrogenase; TBA, total bile acids; TP, total protein; Trig, triglycerides; Ucrea, urinary creatinine. *$p < 0.05$ relative to vehicle control (ANOVA with post hoc multiple comparison correction using Tukey contrasts).

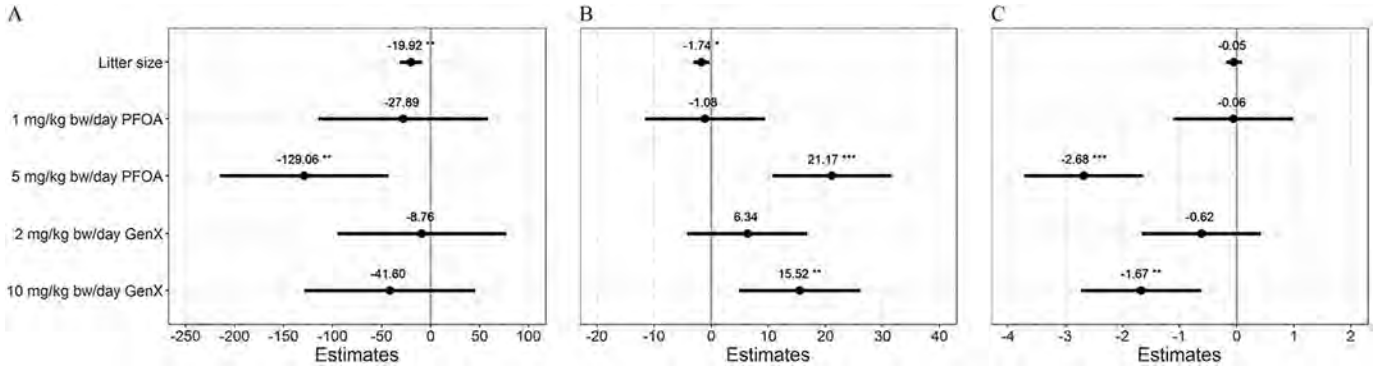

Figure 6. Mixed-effect model estimates for (A) embryo weight (mg), (B) placental weight (mg), and (C) embryo:placenta weight ratios (mg:mg) after exposure *in utero* to perfluorooctanoic acid (PFOA) or GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)] at embryonic day (E) 17.5. Effect estimates are centered around the vehicle control group ($y = 0$) and show the point estimate of the relative change in weight (in milligrams; A and B) or weight ratio (mg:mg; C) with 95% confidence intervals (CIs). $^{*}p < 0.05$. $^{**}p < 0.01$. $^{***}p < 0.001$. Beta estimate 95% confidence intervals do not overlap zero (mixed-effect model adjusting *a priori* for litter size as a fixed effect and the dam as a random effect, vehicle control as reference group). Adjusted estimates and 95% CIs are shown in Table S8.

offspring (Risnes et al. 2009), and the functional capacity of the placenta is likely the driver of fetal heart fitness (Thornburg et al. 2010). Placentas that are disproportionately large relative to fetal size tend to exhibit reduced functional capacity with respect to optimal blood flow and vascular resistance (Risnes et al. 2009; Salafia et al. 2006), which could lead to both adverse perinatal (Hutcheon et al. 2012) and adult CVD outcomes (Thornburg et al. 2010). Here we show higher placenta weights that were disproportionate to embryo weights in mice exposed to PFOA and GenX. Whether the increased placental weight is due to pathological changes or is a compensatory mechanism to protect the developing fetus is not known. The extent to which gestational exposure to these environmental contaminants could adversely impact perinatal and adult offspring health outcomes, especially cardiovascular outcomes, should be the focus of future studies.

A previous report has shown dose-dependent necrotic changes in the placenta of mice exposed to 10 mg/kg/d and 25 mg/kg/d PFOA, and pup mortality and gestational weight loss were evident (Suh et al. 2011). Here, placental lesions in mice exposed to 2 mg/kg/d GenX, 10 mg/kg/d GenX, and 5 mg/kg/d PFOA at E17.5 occurred at a significantly higher incidence compared to controls, and the labyrinth was the specific target. This is significant because the maternal–embryo exchange of oxygen, nutrients, and waste occurs in the placental labyrinth. Adverse placental effects of 5 mg/kg/d PFOA and 10 mg/kg/d GenX occurred at both the litter level as well as across all placenta evaluated, regardless of litter, and adverse placental effects of 2 mg/kg/d GenX were significant when considered at the level of the litter as a unit. The lowest doses tested in this study resulting in adverse placental pathology were 2 mg/kg/d GenX and 5 mg/kg/d PFOA. Given that maternal serum accumulation and embryo deposition of PFOA and GenX were similar at the high (5 mg/kg PFOA vs. 10 mg/kg GenX) and low doses (1 mg/kg PFOA vs. 2 mg/kg GenX) and that the placenta is at the interface between these two compartments, the disparate patterns in adverse placenta histopathology suggest that the placenta may be more sensitive to the effects of GenX vs. PFOA. The mechanisms of toxicity towards the placenta may also

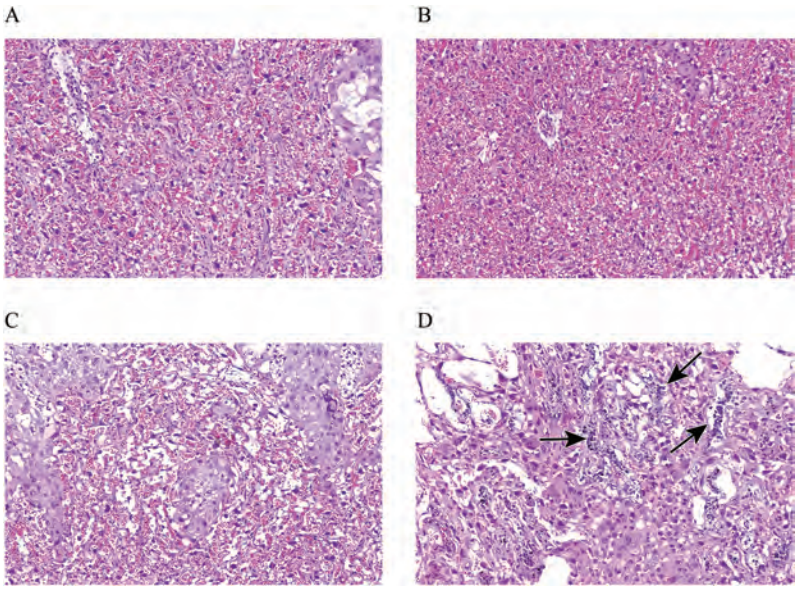

Figure 7. Representative examples of histopathological placenta findings observed in dams at embryonic day (E) 11.5 and E17.5, treated with perfluorooctanoic acid (PFOA) or GenX [hexafluoropropylene oxide dimer acid (HFPO-DA)]. (A) Normal labyrinth from a vehicle control dam at E17.5. (B) Labyrinth congestion in a dam at E17.5 that was treated with 10 mg/kg/d GenX (C) Moderate labyrinth atrophy of the trilaminar trophoblast layer at E17.5 in a dam treated with 10 mg/kg/d GenX. (D) Labyrinth necrosis (arrows) in an E17.5 dam that was treated with 10 mg/kg/d GenX. All images at 20× magnification.

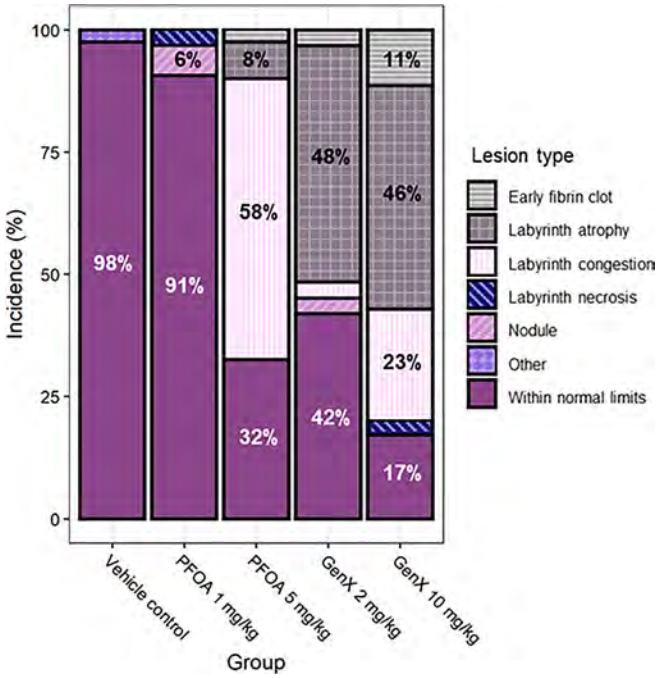

Figure 8. Incidence of placenta lesions across treatment groups at embryonic day 17.5. $n = 5$–6 litters with 31–41 placentas evaluated per treatment group (an average of 6–8 placentas per litter). Incidence values <4% are not numerically indicated, but all values and statistical comparisons of placenta lesion incidences across treatment groups at E17.5 are shown in Table S10.

differ between the two PFAS and will be pursued in ongoing studies.

TH play a critical role in neurodevelopment (de Escobar et al. 2004; Porterfield 1994). PFAS are well-documented thyroid disrupters in humans (Coperchini et al. 2017; Webster et al. 2016), including in pregnant women (Ballesteros et al. 2017; Berg et al. 2015; Wang et al. 2014; Webster et al. 2014). Generally, maternal PFAS levels during pregnancy are associated with shifts in TH levels consistent with hypothyroidism (e.g., elevated thyroid-stimulating hormone), which is associated with increased risk for low birth weight (Alexander et al. 2017). It is possible that PFAS chemicals exert some adverse effects on embryo growth via TH disruption across the maternal–placental–embryo unit. Indeed, Conley et al. (2019) reported maternal serum total triiodothyronine (T3) and thyroxine (T4) were reduced in rats exposed to 125–500 mg/kg/d HFPO-DA (GenX) during gestational days 14–18. Maternal serum TH could not be measured due to volume constraints in our study. As the placenta regulates the degree to which maternal THs pass to the developing fetus, and it maintains the optimal balance of the TH throughout embryo development (Chan et al. 2009), the relationship between PFAS-induced maternal TH changes and placental function requires additional study, especially given the role of TH in fetal neurodevelopment.

In a systematic review and meta-analysis of nonhuman evidence for effects of PFOA on BW, it was estimated that a 1-unit (1 mg/kg BW/d) increase in PFOA is associated with a −0.023 g (95% CI: −0.029, −0.016) shift in pup birth weight (Koustas et al. 2014). Here we report a −0.028 g (95% CI: −0.114, 0.586) shift in embryo weight on E17.5 in mice exposed to 1 mg/kg/d PFOA and a −0.129 g (95% CI: −0.215, −0.043) shift in mice exposed to 5 mg/kg/d PFOA. Effects on embryo weight at E17.5 in this study can be summarized as most severe to least severe: 5 mg/kg/d PFOA (−0.129 g), 10 mg/kg/d GenX (−0.042 g), 1 mg/kg/d PFOA (−0.023 g), and 2 mg/kg/d GenX (−0.009 g). An industry study of CD-1 mice exposed to 5 mg/kg/d HFPO-DA (GenX) from preconception through weaning showed reduced pup weight at postnatal day (PND) 1 that persisted through PND 21 with effects more severe in male offspring (DuPont-18,405-1,037). In rats, mean embryo weights were decreased in rats exposed to 100 mg/kg/d HFPO-DA (GenX) for 15 d of gestation (Edwards 2010a), and in a different study, female birth weights were reduced after 5 d of gestational exposure at 125 mg/kg (Conley et al. 2019). To our knowledge, there are no human data showing associations between maternal GenX exposure and birth weight outcomes.

Several human cohort studies have shown that higher levels of prenatal or early-life PFOA exposure is associated with increased adiposity in childhood (Braun et al. 2016; Fleisch et al. 2017) and metabolic disruption in young adulthood (Domazet et al. 2016). Additionally, it is known that low birth weight is associated with adult diseases, including metabolic syndrome in both humans and animals (Barker 2004). Due to the environmental ubiquity of a mixture of PFAS chemicals, it is difficult to unravel the relative contributions of prenatal and postnatal (e.g., chronic, lifelong) exposure and adverse health outcomes. Animal studies allow for discrete measurement of health outcomes associated with specific critical periods of exposure, and future work should investigate metabolic disruption in offspring exposed *in utero* to provide key insights on the metabolic programming capacity of PFAS.

In the present study, PFOA (5 mg/kg/d) and GenX (2 mg/kg/d or 10 mg/kg/d) exposures resulted in significantly higher GWG in mice, with significant effects emerging at an earlier point in gestation in mice exposed to GenX and occurring at a lower dose than PFOA (2 mg/kg/d GenX vs. 5 mg/kg/d PFOA). In contrast, a decrease in mean maternal weight gain was reported in a recent study of gestational exposure to GenX in rats exposed to 250 or 500 mg/kg/d (Conley et al. 2019). Although these findings are not consistent with the higher GWG reported here, it is possible that statistical methods (absolute change in maternal weight vs. relative change in weight analyzed using repeated measures models), differing windows of exposure (5 d during mid- to late gestation vs. exposure throughout gestation), and interspecies differences in preliminary PFAS elimination

Table 4. Placental thyroid hormone measurements at embryonic day 17.5.

Hormone	Vehicle control {mean ± SD [*n* (a, b)]}	1 mg/kg/d PFOA {mean ± SD [*n* (a, b)]}	5 mg/kg/d PFOA {mean ± SD [*n* (a, b)]}	2 mg/kg/d GenX {mean ± SD [*n* (a, b)]}	10 mg/kg/d GenX {mean ± SD [*n* (a, b)]}
rT3 (ng/g)	1.2 ± 0.7 [5 (4, 1)]	0.7 ± 0.4 [6 (3, 3)]	1.4 ± 0.7 [5 (5, 0)]	1.7 ± 0.8 [6 (6, 0)]	1.6 ± 0.3 [6 (6, 0)]
T3 (ng/g)	0.3 ± 0.2 [6 (1, 5)]	0.2 ± 0 [6 (0, 6)]	0.2 ± 0 [4 (0, 4)]	0.3 ± 0.2 [5 (0, 5)]	0.2 ± 0 [6 (0, 6)]
T4 (ng/g)	3.8 ± 0.6 [6 (6, 0)]	2.5 ± 1.0 [6 (6, 0)]	2.8 ± 1.3 [6 (6, 0)]	5.3 ± 1.7 [6 (6, 0)]	6.1 ± 1.1* [6 (6, 0)]
T3:T4 ratio	0.07 ± 0.04 [6]	0.09 ± 0.03 [6]	0.07 ± 0.02 [4]	0.05 ± 0.01 [5]	0.03 ± 0.01 [6]
rT3:T4 ratio	0.33 ± 0.19 [5]	0.30 ± 0.21 [6]	0.45 ± 0.05 [5]	0.32 ± 0.12 [6]	0.27 ± 0.08 [6]

Note: Sample sizes are expressed as the total number of samples (*n*) as well as the number of samples above the MDL (a) and below the MDL (b). Nonquantifiable samples below the MDL were imputed using the calculation MDL × 0.5. MDL values were: T4, 0.84 ng/g; T3, 0.42 ng/g; rT3, 0.67 ng/g. MDL, method detection limit; rT3, reverse triiodothyronine; SD, standard deviation; T3, triiodothyronine; T4, thyroxine. *$p < 0.05$ relative to vehicle control [analysis of variance (ANOVA) with post hoc multiple comparison correction using Tukey contrasts].

rates [GenX elimination half-life in rats: ~5 h vs. ~20 h in mice, (Gannon et al. 2016)] could explain these disparate results. It is possible that different elimination rates of the compound make the comparison of equivalent or similar external doses a challenge. In fact, dam serum concentrations of rats exposed to 500 mg/kg/d from gestation day (GD) 14-18 reported in Conley et al. (2019) were of similar magnitude to those observed in mice exposed to 10 mg/kg/d throughout gestation in the present study (~100 μg/mL). Similarly, serum concentrations from pregnant mice in the current study exposed to 2 mg/kg/d GenX were roughly equivalent (~33 μg/mL) to serum concentrations obtained from rat dams exposed to 62.5 mg/kg/d GenX in the study by Conley et al. (2019).

Higher GWG observed in our PFOA-exposed mice is consistent with findings reported in humans; interquartile range increases in GWG were associated with elevated cord blood levels of PFOA (odds ratio = 1.33; 95% CI: 1.13,1.56) (Ashley-Martin et al. 2016). Similarly, other legacy PFAS compounds such as perfluorooctanesulfonic acid are positively associated with GWG (Jaacks et al. 2016). However, our data describing the relationship between maternal exposure to GenX and increased GWG in a mouse model are novel. Importantly, higher GWG is associated with adverse outcomes for both mother and infant in humans, including increased risk for pregnancy-associated hypertension (with or without smaller birth weights), gestational diabetes, postpartum weight retention, increased risk for unsuccessful breastfeeding, and increased risk for stillbirth, infant mortality, and preterm birth (Rasmussen and Yaktine 2009). These disorders share many risk factors, but it is not fully understood to what extent their etiologies are interrelated and/or interdependent (Villar et al. 2006) or what mechanisms may be driving them. Our data suggest a need for additional study of the adverse maternal and offspring health outcomes associated with GenX exposure.

Liver toxicity is a consistent finding in animal studies of PFOA (Li et al. 2017) and other PFAS, but studies examining GenX are limited. Here, we report similar histopathological findings in livers of exposed pregnant dams to those previously described by our group (and others) in offspring prenatally exposed to PFOA, including increased extent of hepatocellular hypertrophy, cytoplasmic alteration, and increased mitochondria (Filgo et al. 2015; Lau et al. 2006). We hypothesize that the consistent and persistent hepatic cytoplasmic alterations seen following PFAS exposures lead to increased incidence and/or distribution of cell death, which is consistent with the decrease in mitotic figures compared to control liver sections. This constellation of lesions is considered adverse and is incompatible with long-term normal liver function. The maternal liver responds to estrogen produced by the placenta and produces thyroid-binding globulin, which, in turn, regulates the level of maternal circulating TH (Nader et al. 2009). It is possible that altered maternal liver function due to PFOA or GenX exposure plays an important role in mediating placental and embryo outcomes.

In addition to consistently observed histopathological changes in the liver induced by either PFOA or GenX, maternal clinical chemistry indicated shifts in liver enzymes, including higher ALT (10 mg/kg/d GenX; E11.5), higher ALP (10 mg/kg/d GenX; E17.5), higher AST (5 mg/kg/d PFOA; E17.5), and higher SDH (5 mg/kg/d PFOA; E17.5). Our TEM findings build upon a growing body of evidence demonstrating potential mechanisms of PFAS-induced hepatic toxicity other than PPAR and demonstrate this for the first time with GenX.

In a previous reproductive and developmental toxicity study of HFPO-DA (GenX) in CD-1 mice, 5 mg/kg/d was determined to be the NOAEL for reproductive toxicity and maternal systemic toxicity (based on microscopic changes in maternal liver; DuPont-18,405-1,037) (Edwards 2010b). Here, we are not able to report a NOAEL, as significant adverse effects occurred in the lowest GenX dose group evaluated in this study (2 mg/kg/d). We demonstrate adverse systemic toxicity of dams exposed to 2 mg/kg/d GenX, which include microscopic alterations in the liver, higher GWG, and higher incidence of placental lesions. Dam serum GenX concentrations obtained at E17.5 in the present study were comparable to dam plasma concentrations reported by DuPont-18,405-1,037: 22.9 μg/mL (present study, 2 mg/kg/d on E17.5), 36.4 μg /mL (DuPont-18,405-1,037, 5 mg/kg/d on lactation day 21), and 58.5 μg/mL (present study, 10 mg/kg/d on E17.5; compared in Figure S4). However, it should be noted that in the present study at all tested doses, both PFOA and GenX, maternal serum concentrations were higher at E11.5 than E17.5. This could be explained by maternal off-loading of body burden to developing embryos and other maternal tissues (i.e., liver) and rapid expansion of maternal blood volume throughout the course of pregnancy.

There are several limitations to this study regarding experimental design, sample sizes, and interspecies differences. Due to performing the experiment over two experimental blocks, some end points were only evaluated from one of the two blocks, limiting statistical power. It is possible that some effects would achieve statistical significance with a larger number of observations. The two-block design did not impair the strength of the effect when significant effects were present in end points evaluated at both time points, which was verified by statistical analysis. It is possible that variance in half-life, amount of exposure to these chemicals, and other interspecies differences may limit the human relevance of the findings reported here. Although the mouse and human both have discoid hemochorial placenta, the maternal–placental–embryo unit in mice differs from that in humans in other ways, including the labyrinthine vs. villous structure, the number of offspring carried during each pregnancy (~14 vs. ~1), and gestation length (~20 d vs. ~280 d). Although there are distinct interspecies differences between humans and mice, the outbred CD-1 mouse was selected in the current study due to its genetic diversity. While the CD-1 mouse is sensitive to PFOA, compared to other inbred mouse strains (Tucker et al. 2015), significant treatment-related effects were still detectable despite its greater biologic variability in response. It is not known whether there are strain differences in sensitivity to GenX, which should be investigated in future studies.

Conclusion

In a comparative reproductive and developmental study in mice of PFOA and a replacement, GenX, we report adverse effects of both compounds against the maternal–embryo–placenta unit. Both PFOA and GenX induced elevated GWG, higher maternal liver weights, adverse microscopic pathological changes in the maternal liver, and abnormal histopathological lesions in mature placenta. Importantly, we provide evidence that illustrates GenX (as low as 2 mg/kg/d) significantly affects the maternal–embryo–placenta unit differently than its predecessor PFOA and that this alternative compound may have a unique mechanism(s) of reproductive toxicity in this model system. Lastly, we build a case for the importance of evaluating the placenta as a critical tissue in studies of developmental and reproductive toxicity through utilizing clinically relevant, translational end points to illustrate the unique susceptibility of this organ to the adverse effects of GenX.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT G
TO AMENDED INTERVENOR COMPLAINT

A Section 508–conformant HTML version of this article is available at https://doi.org/10.1289/EHP4372.

Adverse Maternal, Fetal, and Postnatal Effects of Hexafluoropropylene Oxide Dimer Acid (GenX) from Oral Gestational Exposure in Sprague-Dawley Rats

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Background: Hexafluoropropylene oxide dimer acid [(HFPO-DA), GenX] is a member of the per- and polyfluoroalkyl substances (PFAS) chemical class, and elevated levels of HFPO-DA have been detected in surface water, air, and treated drinking water in the United States and Europe.

Objectives: We aimed to characterize the potential maternal and postnatal toxicities of oral HFPO-DA in rats during sexual differentiation. Given that some PFAS activate peroxisome proliferator-activated receptors (PPARs), we sought to assess whether HFPO-DA affects androgen-dependent development or interferes with estrogen, androgen, or glucocorticoid receptor activity.

Methods: Steroid receptor activity was assessed with a suite of *in vitro* transactivation assays, and Sprague-Dawley rats were used to assess maternal, fetal, and postnatal effects of HFPO-DA exposure. Dams were dosed daily via oral gavage during male reproductive development (gestation days 14–18). We evaluated fetal testes, maternal and fetal livers, maternal serum clinical chemistry, and reproductive development of F1 animals.

Results: HFPO-DA exposure resulted in negligible *in vitro* receptor activity and did not impact testosterone production or expression of genes key to male reproductive development in the fetal testis; however, *in vivo* exposure during gestation resulted in higher maternal liver weights (≥62.5 mg/kg), lower maternal serum thyroid hormone and lipid profiles (≥30 mg/kg), and up-regulated gene expression related to PPAR signaling pathways in maternal and fetal livers (≥1 mg/kg). Further, the pilot postnatal study indicated lower female body weight and lower weights of male reproductive tissues in F1 animals.

Conclusions: HFPO-DA exposure produced multiple effects that were similar to prior toxicity evaluations on PFAS, such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), but seen as the result of higher oral doses. The mean dam serum concentration from the lowest dose group was 4-fold greater than the maximum serum concentration detected in a worker in an HFPO-DA manufacturing facility. Research is needed to examine the mechanisms and downstream events linked to the adverse effects of PFAS as are mixture-based studies evaluating multiple PFAS. https://doi.org/10.1289/EHP4372

Introduction

Per- and polyfluoroalkyl substances (PFAS) are a group of high-profile contaminants of emerging concern; the concern is primarily due to extensive research indicating these compounds have extreme environmental persistence (Awad et al. 2011), widespread occurrence (Kaboré et al. 2018; Kannan et al. 2004; Pan et al. 2018), long biological half-lives (Li et al. 2018), and nearly ubiquitous human exposure (Calafat et al. 2007). Further, there is concern for human health effects due to laboratory animal and epidemiological research on both perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). When administered throughout gestation, both PFOS and PFOA have been shown to produce adverse effects in rodent models, including extensive pup mortality and reduced growth rates (Grasty et al. 2003; Lau et al. 2003; Thibodeaux et al. 2003), and their administration is also correlated with increased incidence rates of thyroid dysfunction (Coperchini et al. 2017) and low birth weight (Apelberg et al. 2007) in human populations. Because of the combination of these factors, PFOS was primarily phased out of production by 2002, and subsequently added to Annex B of the Stockholm Convention, and the U.S. EPA has set drinking water health advisories for PFOS and PFOA at 70 parts per trillion (U.S. EPA 2016b). Similarly, beginning in 2006 the major manufacturers of PFOA voluntarily agreed to phase out production by 2015 (U.S. EPA 2006). However, a variety of structural analogs have been developed and utilized as replacement compounds in the production of a range of consumer and industrial products for which fluoropolymers provide desirable characteristics (Wang et al. 2013; Wang et al. 2017b).

Hexafluoropropylene oxide dimer acid [(HFPO-DA), GenX] is a PFAS compound that is used as a polymerization aid in the manufacturing of high-performance fluoropolymers following the phase out of PFOA (Beekman et al. 2016). Recent environmental monitoring studies in North Carolina and the Netherlands have reported elevated levels of HFPO-DA, among other PFAS, in air, groundwater, and surface water sampled within the proximity of manufacturing sites and in drinking water originating from contaminated surface sources (Gebbink et al. 2017; McCord et al. 2018; Strynar et al. 2015; Sun et al. 2016). Despite the extensive *in vivo* toxicity research available for PFOS and PFOA, relatively little peer-reviewed experimental data exist for HFPO-DA or the other PFAS

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Supplemental Material includes complete data tables with means, standard errors, and samples sizes for all data depicted in figures and figures of fetal testis testosterone production and *in vitro* endocrine receptor transactivation assays.

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analogs that have been recently detected. In addition to peer-reviewed studies (Caverly Rae et al. 2015; Gannon et al. 2016; Rushing et al. 2017; Wang et al. 2017a), guideline registration studies from the manufacturer of HFPO-DA are publicly available (https://hero.epa.gov/hero/index.cfm/project/page/project_id/2627); however, even though *in utero* exposure to PFOS and other PFAS induced extensive neonatal mortality and reduced offspring body weights in rats, similar studies have not been conducted with HFPO-DA to our knowledge. Overall, the paucity of data has led to calls for coordinated efforts to screen and assess the toxicity of the myriad PFAS currently detected in environmental matrices (Bruton and Blum 2017; Wang et al. 2017b).

PFOS and PFOA are known activators of peroxisome proliferator-activated receptors (PPARs), primarily alpha (PPARα) and gamma (PPARγ) (Vanden Heuvel et al. 2006). HFPO-DA is hypothesized to activate PPARs based on observed up-regulation of PPAR-signaling pathway genes (Wang et al. 2017a), increased markers of liver peroxisome proliferation (DuPont 2008a, 2008b; Rushing et al. 2017), and increased liver weight in mice and/or rats (Caverly Rae et al. 2015; DuPont 2008a, 2008b; Rushing et al. 2017; Wang et al. 2017a). Some phthalate ester metabolites are also PPAR activators (Lapinskas et al. 2005) and *in utero* exposure reduces gene expression of steroidogenic enzymes and decreases production of testosterone in the testes of male offspring, leading to reproductive tract malformations in rats (Hannas et al. 2011; Mylchreest et al. 2002; Parks et al. 2000; Wilson et al. 2004b). Similarly, Zhao et al. (2014) reported that PFOS reduced testosterone production and impaired fetal rat Leydig cells following *in utero* exposure. The specific molecular initiating event(s) (MIE) by which PFOS and some phthalate esters produce male reproductive toxicity remain(s) elusive; however, it has been proposed that activation of PPAR, specifically PPARα, plays an essential role (Corton and Lapinskas 2005; Gazouli et al. 2002; Nepelska et al. 2015). If this MIE is truly responsible for the anti-androgenic effects of phthalates, then oral exposure to other proposed PPAR agonists, such as HFPO-DA, would be expected to reduce male testis testosterone production *in utero* and cause male rat reproductive tract malformations, similar to the active phthalates.

In regard to the above concerns, there were two goals for the present study. First, we were interested in identifying whether HFPO-DA, like other PFAS, activates PPAR signaling pathways and, if so, does this lead to a reduction in fetal testis testosterone production resulting in the subsequent increase in the incidence/severity of male reproductive defects. Second, we wanted to leverage these experiments to provide additional relevant *in vivo* data on the potential for gestational oral HFPO-DA exposure to produce toxic effects in the mother or offspring. We conducted studies with pregnant rats dosed during the specific gestational window critical to masculinization of the male fetal reproductive tract [gestation days (GD) 14–18] (Carruthers and Foster 2005). We evaluated and report on a range of effects primarily related to the maternal and fetal livers, circulating maternal thyroid hormones and lipids, and a single-dose level pilot study on postnatal development. Further, because of prior conflicting reports on the endocrine receptor activity of PFAS and the potential relevance to mammalian reproductive development, we assessed the estrogen, androgen, and glucocorticoid receptor activity (agonism/antagonism) of HFPO-DA using *in vitro* transcriptional activation assays.

Methods

Dosing Solutions

Dosing solutions were prepared using high-performance liquid chromatography-grade water purchased from Honeywell Research Chemicals and HFPO-DA ammonium salt (CAS: 62037-80-3; Product No.: 2122-3-09; Lot: 00005383) purchased from SynQuest Laboratories. HFPO-DA purity was 100% as determined by the supplier via perchloric acid titration. Dosing was administered once daily via oral gavage at 2.5 mL/kg body weight across a range of 1–500 mg HFPO-DA/kg-body weight per day (specific doses for different studies reported below). Doses were selected based on data from existing developmental toxicity studies on HFPO-DA in Sprague-Dawley rats. A published study by Caverly Rae et al. (2015) reported 1 mg/kg per day was a no observed adverse effect level (NOAEL) and 500 mg/kg per day was an upper dose that was tolerated in the rat. Further, an industry guideline prenatal developmental toxicity study by DuPont (2010) reported a NOAEL of 10 mg/kg per day and that 1,000 mg/kg per day was overtly toxic to the dam. The doses utilized in the present experiments were chosen to evaluate the reported NOAELs and allow for full dose–response assessment while avoiding overt maternal toxicity at highly elevated doses.

Animals

Time-mated Sprague-Dawley rats [Crl:CD(SD)], approximately 90 d of age, were purchased from Charles River Laboratories and shipped to the National Health and Environmental Effects Research Laboratory at the U.S. EPA in Research Triangle Park, North Carolina, on GD2 (GD0 = bred date; GD1 = plug positive date). Dams and their offspring were housed individually in clear polycarbonate cages (20 × 25 × 47 cm) with heat-treated, laboratory-grade pine shavings and fed NIH07 rodent diet and filtered (5 μm) municipal tap water *ad libitum*. Dams were weight-ranked and stratified then randomly assigned to treatment groups to produce similar mean weights and variances. This study was conducted in accordance with a protocol approved by the U.S. EPA National Health and Environmental Effects Research Laboratory's Institutional Animal Care and Use Committee. Animals were housed in a facility accredited by the Association for Assessment and Accreditation of Laboratory Animal Care and maintained at 20–22°C, 45–55% humidity, and a 12:12 h photoperiod (lights off at 1800 hours).

Evaluation of Fetal and Maternal Effects during Gestation

A total of three blocks of 15 dams per block were dosed once daily from GD14–18 with either water vehicle (control) or HFPO-DA to evaluate fetal and maternal effects (Figure 1A). The first block of dams was dosed with control, 62.5, 125, 250, or 500 mg/kg HFPO-DA ($n = 3$ dams for each). The second and third blocks of dams were dosed with control, 1, 3, 10, or 30 mg/kg HFPO-DA ($n = 3$ per dose per block). Total sample sizes were $n = 9$ for control, $n = 6$ for 1, 3, 10, 30 mg/kg, and $n = 3$ for 62.5, 125, 250, and 500 mg/kg HFPO-DA. In the first two blocks, spanning the entire dose range, we evaluated fetal testis testosterone production, fetal testis gene expression, fetal and maternal liver gene expression, fetal body weight, and maternal serum thyroid hormone and lipid concentrations. In the third block, encompassing the lower dose range utilized here, we collected fetal plasma for measuring HFPO-DA concentrations. Across all three blocks we evaluated maternal weight gain during dosing, reproductive output (number of fetuses and resorptions), maternal serum HFPO-DA concentration, and maternal liver weight at necropsy.

For the first two blocks, spanning the full dose range, late gestation (GD18) dams were euthanized by decapitation at ∼2 h after the final oral dose [∼0830–1000 hours Eastern Standard Time (EST)]. Trunk blood was collected and serum isolated via centrifugation (10,000 × *g* for 15 min at 4°C) in vacutainer tubes, transferred to 1.5-mL microcentrifuge tubes and stored at −80°C.

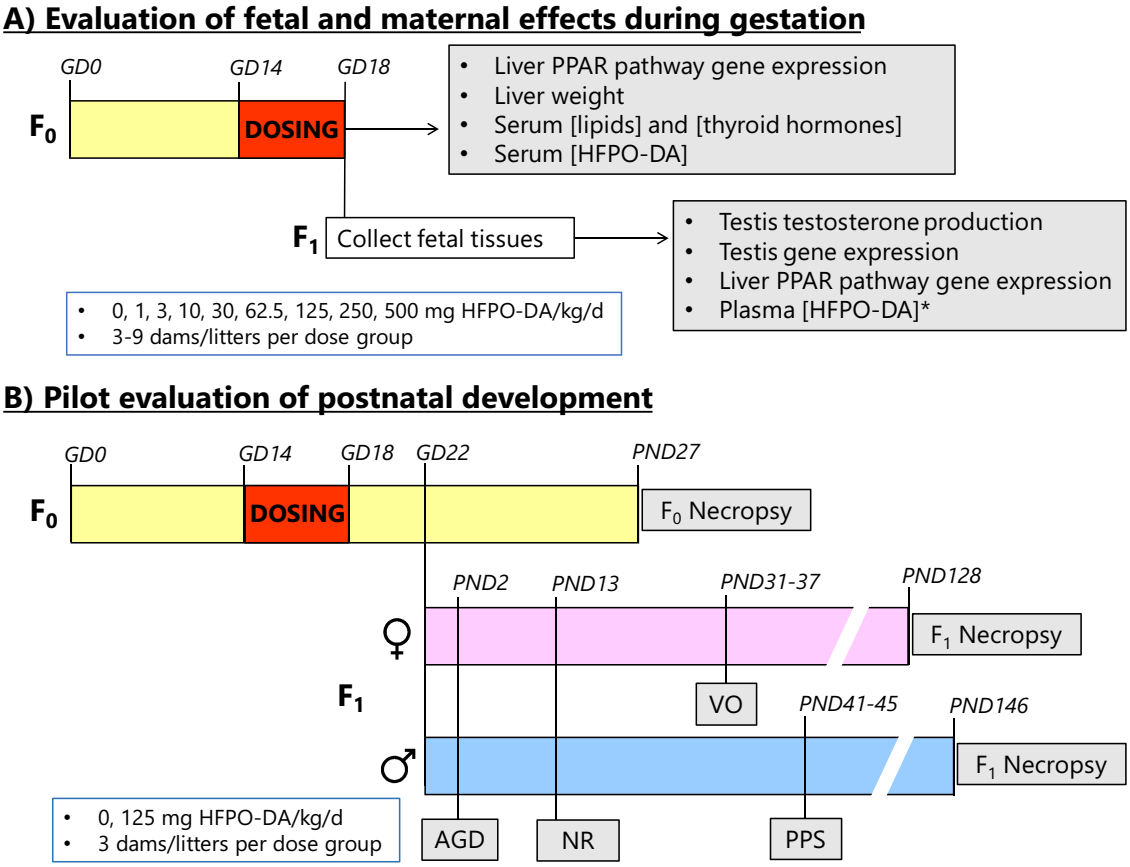

Figure 1. Schematic diagram of study designs for evaluating maternal, fetal, and postnatal effects of oral gestational hexafluoropropylene oxide dimer acid (HFPO-DA) exposure. Both (A) fetal and (B) postnatal study designs used oral gavage dosing from gestation day (GD) 14–18 at the indicated exposure levels. Fetal plasma HFPO-DA concentration (*) was only evaluated at doses of 0–30 mg/kg per day. AGD, anogenital distance; NR, nipple retention; PND, postnatal day; PPAR, peroxisome proliferator-activated receptor; PPS, preputial separation; VO, vaginal opening.

Dam liver weight was recorded and a sample of liver tissue was collected into a polypropylene microcentrifuge tube containing 500 µL TRIzol Reagent (Invitrogen) on ice. Fetuses were removed and two randomly selected fetuses per litter were weighed. Fetal testes were collected from all male pups with a single testis from the first three males used for determination of *ex vivo* testosterone production and the remaining testes were homogenized and preserved in TRIzol Reagent for gene expression analysis. The liver was collected from a single, randomly selected fetus per dam/litter for gene expression analysis and transferred to a polypropylene microcentrifuge tube containing 500 µL TRIzol Reagent (Invitrogen) on ice. Both dam and fetal liver samples were individually homogenized using a Bullet Blender (Next Advance) with 1-mm zirconium oxide beads, transferred to clean tubes, and stored at −80°C prior to RNA extraction (see below). *Ex vivo* fetal testis testosterone production was measured as previously reported (Wilson et al. 2004b) except the radioimmunoassay (RIA) utilized here was supplied by ALPCO (Catalog No. 72-TESTO-CT2, ALPCO). Briefly, one testis was isolated from each of three separate male fetuses in each litter and incubated in a humidified atmosphere at 37°C for 3 h in 500 µL of M-199 media (phenol red–free; Hazelton Biologics, Inc.) supplemented with 10% dextran-coated charcoal-stripped fetal bovine serum (Hyclone Laboratories) in 24-well plates under gentle agitation. After incubation, media were removed and stored in siliconized microcentrifuge tubes at −80°C until RIA analyses, which were performed according to manufacturer specifications.

Gene expression in fetal testes and fetal/maternal livers was assessed using reverse transcriptase real-time PCR of cDNA synthesized from RNA extracted from sample homogenates. RNA extraction was conducted according to TRIzol Reagent manufacturer specifications using chloroform and isopropanol. Following extraction, RNA was purified using the RNeasy Mini Kit (Catalog No. 74104; Qiagen). RNA concentration and purity (260:280 ratio ≥1.8) were determined with a NanoDrop 2000 spectrophotometer (Thermo Scientific). For the fetal testes, a 96-well gene array plate was previously custom designed to contain 89 target genes and 3 housekeeping genes, an intra-assay control, a genomic DNA control, a reverse transcriptase control, and a positive PCR control [see Table S1; SABioscience; (Hannas et al. 2012)]. For the fetal and maternal livers, we utilized the RT^2 Profiler PCR Array for Rat PPAR Targets by Qiagen (Catalog No. 330231 PARN-149Z), which contains 84 target genes relevant to PPARα, -β/δ, and -γ signaling pathways and 5 potential housekeeping genes (see Table S2). PCR reactions were run using RT2 SYBR Green quantitative PCR (qPCR) Master Mix (SABioscience) on an iCycler iQ Real-Time Detection System (Bio-Rad) for fetal testes and on a CFX96 Touch Real-Time Detection System (Bio-Rad) for maternal and fetal livers.

For the third block, dosed with the lower dose range (1–30 mg/kg HFPO-DA), late gestation (GD18) dams were euthanized by decapitation ∼2 h after the final dose, liver weight was recorded, and trunk blood was collected for serum isolation. Serum was isolated from trunk blood via centrifugation (10,000 × *g*; 15 min; 4°C) using Becton Dickinson vacutainer tubes and stored in 1.5-mL siliconized microcentrifuge tubes at −80°C for future analyses. Fetuses were removed and fetal blood was collected from the jugular vein from all fetuses within a litter

using heparinized glass capillary tubes. Blood was expelled from capillary tubes using fine-tip disposable transfer pipets into a microcentrifuge tube forming a single composite sample per litter. Fetal blood was then centrifuged at $10{,}000 \times g$ for 15 min at 4°C and plasma was transferred to clean tubes and frozen at −80°C.

Maternal sera from all three blocks and fetal plasma from the third block were analyzed for HFPO-DA concentrations similar to previously reported methods (McCord et al. 2018; Reiner et al. 2009; Rushing et al. 2017). Serum or plasma samples (25 μL) were denatured using 0.1 M formic acid (FA) followed by a cold (−20°C) acetonitrile (ACN) protein crash. The volumes of FA and ACN varied based on the anticipated concentrations of HFPO-DA in the sample (0–100 ng HFPO-DA/mL = 100 μL FA + 0.5 mL ACN; 100–5,000 ng HFPO-DA/mL = 100 μL FA + 1.0 mL ACN; 5,000–200,000 ng HFPO-DA/mL = 1.0 mL FA added, then 100-μL subsamples removed and crashed with 900 μL cold ACN). Samples were vortex mixed after FA and ACN additions then centrifuged at $10{,}000 \times g$ for 5 min and the supernatant removed. Sample extracts were separated using a Waters ACQUITY ultra performance liquid chromatograph (UPLC) (Waters Corporation) fitted with a Waters ACQUITY UPLC BEH C18 column (2.1 mm × 50 mm; 1.7 μm; 130 Å). Detection was performed using a Waters Quattro Premier XE tandem quadrupole mass spectrometer in negative ionization mode. A stable isotope of HFPO-DA ($^{13}C_3$, Wellington Laboratories) was used as an internal standard for quantitation. Separate calibration curves were prepared for the ranges 0–100 ng/mL, 100–5,000 ng/mL, and 5,000–200,000 ng/mL to account for expected concentration differences between control, offspring (fetus/pup), and dam concentrations across the dose range tested.

Maternal serum samples from the first two blocks were analyzed for thyroid hormones and a standard lipid panel. Total triiodothyronine (T_3) and thyroxine (T_4) were quantified by radioimmunoassay (RIA) according to manufacturer specifications (IVD Technologies). Thyroid hormone samples were run in duplicate (mean intra-assay coefficient of variation 15.5% for T_3, 11.5% for T_4), and two calibration standards were run as unknowns with observed concentrations varying from expected by <15% for T_3 and <20% for T_4. Thyroid hormone RIA values were considered below detection when specific binding (B/B_0) was ≥90% (0.2 ng/mL for T_3 and 2 ng/mL for T_4) (Sui and Gilbert 2003). Serum total cholesterol, high-density lipoproteins (HDL), low-density lipoproteins (LDL), and triglycerides were quantified using a Beckman Coulter AU480 clinical chemistry analyzer (Beckman Coulter, Inc.) as per manufacturer's protocol. All reagents were obtained from the instrument manufacturer except for the LDL assay, which was obtained from Diazyme Laboratories.

Pilot Evaluation of Postnatal Development

A single-dose level pilot study utilizing time-mated SD rats was conducted to examine the potential postnatal effects of *in utero* exposure to HFPO-DA from a similar dosing interval to the fetal studies (Figure 1B). The study consisted of dams exposed to oral daily dosing with either water vehicle or 125 mg/kg HFPO-DA ($n = 3$ for each) from GD14–18. This dose was selected because it was the highest dose level that did not significantly reduce maternal weight gain during dosing from the fetal evaluation studies. Dams gave birth naturally beginning on the morning of GD22 [i.e., postnatal day (PND) 0]. On PND2 all pups were sexed, weighed, and anogenital distance (AGD) was measured using a Leica MZ6 stereomicroscope (Leica Microsystems) fitted with an ocular micrometer. On PND13, the offspring were sexed, weighed, and evaluated for retention of female-like nipples/areolae. On PND27, the dams were euthanized, uterine implantation sites were scored, pups were weaned to two animals per cage by sex and treatment group, and food was changed to NTP2000 rodent diet. Beginning on PND31 for female offspring and PND41 for male offspring, individuals were evaluated daily for markers of pubertal onset, vaginal opening (VO) for females and balano-preputial separation (BPS) for males.

Beginning at PND128, adult F1 females were weighed, euthanized via decapitation, and examined via necropsy for any reproductive tract malformations and tissue weights were collected for uterus, paired ovaries, liver, paired kidneys, and visceral adipose tissue. Similarly, beginning at PND146 adult F1 males were weighed, euthanized, and examined for reproductive tract malformations and weights were collected for all relevant reproductive tissues. Male necropsy included weights of glans penis, ventral prostate, paired seminal vesicles, paired testes, paired epididymides, levator ani–bulbocavernosus (LABC), paired bulbourethral (Cowper's) glands, paired kidneys, visceral adipose tissue, and epididymal adipose tissue. After weighing, the left epididymis was separated into two sections, the cauda and the corpus plus caput, and individually minced in M-199 media. Total sperm counts in epididymal sections were measured using a Multisizer 3 Coulter counter (Beckman Coulter).

In Vitro *Transcriptional Activation Assays*

HFPO-DA was assessed for agonism and antagonism of transcriptional activation for estrogen (ER), androgen (AR), and glucocorticoid receptors (GR). Method details for *in vitro* transactivation assays for ER (Wilson et al. 2004a), AR (Hartig et al. 2002, 2007), and GR (Conley et al. 2017; Medlock Kakaley et al. 2018) have been previously reported. Briefly, for ER activity we utilized the stably transfected T47D-KBluc cell line [publicly available via American Type Culture Collection (ATCC); CRL-2865] according to protocols provided by ATCC with the modification of Dulbecco's Modified Eagle Media (DMEM) as the cell culture media instead of Roswell Park Memorial Institute (RPMI) media. We utilized adenoviral transduction to introduce chimp AR (Ad5chAR-g) (Hartig et al. 2007) or human GR (Ad/GR4) (Shih et al. 1991) and a luciferase-based promoter-reporter construct (MMTV-Luc; Ad/mLuc7) (Shih et al. 1991) into CV-1 cells (ATCC CCL-70) to assess GR and AR activity, respectively. For viral transduction, cells were grown to confluence in 60-mm Petri dishes in 10% dextran-coated charcoal-treated fetal bovine serum RPMI-1640 growth media. Confluent cells were split at a ratio of 1:3 into 60-mm dishes and inoculated on day 7 ($\sim 5 \times 10^6$ cells/dish) with adenoviral vectors at multiplicities of infection of 1 receptor to 50 reporter constructs. After 24 h incubation with adenoviral vectors, cells were rinsed, resuspended in media, and seeded into assay plates. All assays were run in 96-well plates and luminescence was detected using a BMG Fluostar Omega luminometer (BMG Labtech) following 24-h exposure. HFPO-DA was tested for receptor agonism and antagonism at 10-fold concentration intervals from 100 pM to 10 μM (ER) or 100 pM to 100 μM (AR and GR). For ER activity, the reference agonist was 17β-estradiol [(E2) CAS: 50-28-2] and the reference antagonist was ICI-182780 (CAS: 129453-61-8). When assessing ER antagonism, HFPO-DA was competed against 10 pM E2. For AR activity the reference agonist was dihydrotestosterone [(DHT) CAS: 521-18-6] and the reference antagonist was hydroxyflutamide (CAS: 52806-53-8). When assessing AR antagonism, HFPO-DA was competed against 100 pM DHT. For GR activity, the reference agonist was dexamethasone [(Dex) CAS: 50-02-2] and the reference antagonist was mifepristone (CAS: 84,371-65-3). When assessing GR antagonism, HFPO-DA was competed against 1 nM Dex. Cellular cytotoxicity across the dosing range was determined for CV-1 cells utilizing the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) dye

(Mosmann 1983). HFPO-DA was analyzed using $n = 2$–3 biological replicate assay plates (i.e., unique cell passages) with four technical replicates per treatment per plate.

Data Analyses

All values are reported as mean ± standard error (SE) and all statistical comparisons were conducted at $\alpha = 0.05$ significance level except for PPAR pathway gene expression, which utilized $\alpha = 0.0001$ to detect highly significant analysis of variance (ANOVA) results and $\alpha = 0.01$ to determine pairwise differences of treatment as compared with controls for significant genes. Treatment effects as compared with control were identified using ANOVA in SAS (version 9.4; SAS Institute). Fetal and postnatal data were analyzed using PROC MIXED to correct for the nested effects of individuals within litters (fetus/pup data nested within litter, litter as random variable); dam data were analyzed using PROC GLM. Pairwise comparison of significant ANOVA results was performed using the least squares means (LSMEANS) procedure in SAS. GraphPad Prism (version 7.02; GraphPad, Inc.) was used to generate all figures and to conduct dose–response curve analyses.

Fetal testis and maternal/fetal liver gene expression data were analyzed using the comparative cycle threshold (C_T) method. Briefly, delta C_T values were calculated using the equation $2^{-\Delta\Delta C_T}$ and normalized to the mean C_T value of the appropriate housekeeping genes. We selected housekeeping genes for each tissue and gene array that did not display a significant (ANOVA $p > 0.01$) treatment effect of HFPO-DA exposure (fetal liver = *Actb*, *B2m*; maternal liver = *Actb*, *Hprt1*, *Rplp1*; and fetal testis = *Actb*, *Gusb*, *Ldha*). Delta C_T values were then converted to fold-induction by dividing the treated replicate delta C_T by the mean delta C_T of the control replicates for each gene. Fold-induction values were then then $\log_{10}$-transformed prior to ANOVA.

Fetal testis testosterone production was normalized to the mean control concentration within a given block and analyzed as percentage of control values across blocks. Maternal liver weight was analyzed using body weight as a covariate within PROC GLM followed by pairwise comparison using LSMEANS, this analysis produces linear regressions of body weight versus liver weight for each dose group. Mean female AGD was subtracted from individual male AGD measures to calculate percentage reduction as compared with control.

Serum HFPO-DA concentrations in the mother and the fetus were analyzed as a function of oral dose administered to the mother. We utilized nonlinear regression (exponential one-phase association) to describe the increase and saturation of serum HFPO-DA concentrations across the full oral dose range (1–500 mg/kg) for maternal serum. Fetal plasma HFPO-DA concentrations were only analyzed in the low-dose range (1–30 mg/kg), which was better described using a linear uptake model. We compared the slopes of the low-dose linear regressions for maternal serum and fetal plasma HFPO-DA concentrations using GraphPad Prism.

Dose–response analyses for the *in vitro* transactivation assay data and the most sensitive *in vivo* end points and were conducted using four-parameter logistic regression in GraphPad Prism (constraint to bottom = 0%, top = 100%). *In vitro* luminescence data was normalized to background (vehicle control), $\log_{10}$ transformed, and converted to percentage maximum response based on saturating levels of reference agonist. *In vivo* data were modeled as a function of $\log_{10}$-transformed internal dose (i.e., dam serum HFPO-DA concentration from GD18), and response data

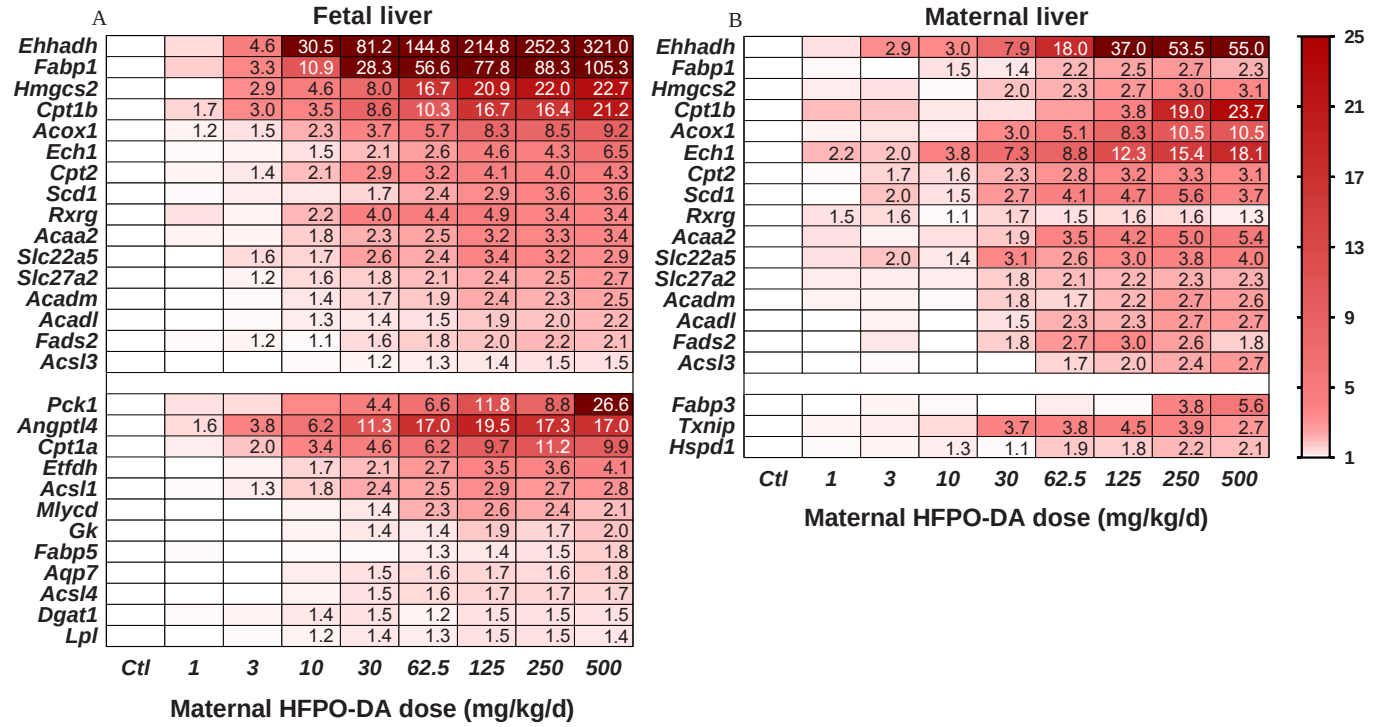

Figure 2. Expression of significantly up-regulated genes (ANOVA, $p < 0.0001$) from peroxisome proliferator-activated receptor (PPAR) signaling pathway gene arrays in (A) fetal ($n = 6$ for control, $n = 3$ for treated) and (B) maternal ($n = 5$ for control, $n = 3$ for treated) livers following gestation day (GD) 14–18 oral maternal exposure to hexafluoropropylene oxide dimer acid (HFPO-DA). Upper portions (above break) display significantly altered genes common to both fetal and maternal livers, lower portions display genes differentially altered between fetal and maternal livers. Cell values represent significant ($p < 0.01$) dose-level fold-induction values relative to control livers [cells with no value were not significantly different from control (see Table S2 for gene descriptions, and Tables S3 and S6 for complete gene expression data)]. Legend indicates fold-induction compared with control with darker shaded genes more highly expressed. Genes with fold-induction >25-fold of control were beyond the scale of the legend. Ctl, control.

was normalized to control and presented as a percentage. We estimated effect concentrations equivalent to a 5% deviation from control (EC_5). Reduction in maternal serum T_3 concentration was modeled by ascribing a concentration of one-half of the detection limit (i.e., 0.1 ng/mL; detection limit of 0.2 ng/mL) for the dose groups that were below the detection limit.

Maternal rat serum concentrations were compared with human plasma concentrations from workers in a HFPO-DA manufacturing facility in Dordrecht, Netherlands (DuPont 2017). Human plasma samples represented workers who volunteered to participate in the study with the goal of determining whether there were measurable quantities of HFPO-DA in their blood. Some of the workers were in areas with potential for exposure and others were not (17/24 participants had detectable HFPO-DA levels). Comparisons were made in order to determine how the doses used in the current study relate to likely "worst case" human concentrations based on internal exposure levels rather than comparing exposures across species based upon estimated external dose levels. We calculated the margin of internal exposure (MOIE) as a ratio of maternal rat serum concentration to human plasma concentration for each of the 17 workers with detectable levels (Bessems et al. 2017). MOIEs were calculated using the mean maternal rat serum HFPO-DA concentration from the 1- and 125-mg/kg dose levels because these represented the lowest oral dose administered and the administered oral dose for the pilot postnatal study.

Results

Fetal Effects from GD14–18 Dosing

Fetal livers from HFPO-DA–exposed litters displayed highly significant (ANOVA $p < 0.0001$), dose–responsive up-regulation of 28 different genes in the PPAR signaling pathway arrays (Figure 2A; see also Table S3). Most affected genes were associated with fatty acid metabolism (*Acaa2, Acadl, Acadm, Acox1, Acsl1, Acsl3, Acsl4, Cpt1a, Cpt1b, Cpt2, Ehhadh, Etfdh, Fads2, Fabp1, Gk, Hmgcs2, Mlycd*, and *Scd1*). Remaining up-regulated genes were associated with lipid transport (*Angptl4, Dgat1, Lpl*), adipogenesis (*Ech1, Lpl*), water transport (*Aqp7*), insulin signaling (*Cpt1a, Dgat1, Pck1*), PPAR transcription factors (*Rxrg*), or PPAR ligand transporters (*Fabp1, Fabp5, Slc22a5, Slc27a2*). The most highly up-regulated genes included *Ehhadh* (321-fold), *Fabp1* (105-fold), *Pck1* (27-fold), *Hmgcs2* (23-fold), *Cpt1b* (21-fold), and *Angptl4* (17-fold). Several genes were significantly ($p < 0.01$) up-regulated even at the lowest dose level tested (1 mg/kg) including *Cpt1b, Angptl4*, and *Acox1*.

In contrast to the observed changes in fetal PPAR liver genes, the results for the expression of genes from our custom array for detecting phthalate-like effects in the fetal testis were not significantly different from controls (see Table S4). Further, fetal testis testosterone production was not significantly different from controls at any dose (see Figure S1, Table S5).

Maternal Effects from GD14–18 Dosing

Similar to fetal livers, maternal livers displayed highly up-regulated expression of PPAR signaling pathway–associated genes (Figure 2B; see also Table S6). Overall, the maternal and fetal livers shared up-regulation of 16 genes. The majority of shared, up-regulated genes were associated with fatty acid metabolism (*Acaa2, Acadl, Acadm, Acox1, Acsl3, Cpt1b, Cpt2, Ehhadh, Fads2, Fabp1, Hmgcs2*, and *Scd1*). Also similar to the fetal liver, the remaining up-regulated maternal genes were associated with adipogenesis (*Ech1*), PPAR transcription factors (*Rxrg*), or PPAR ligand transporters (*Slc22a5, Slc27a2*). In contrast to the fetal liver, the maternal livers of treated rats did not differ significantly from controls in the expression of *Acsl1, Acsl4, Angptl4, Aqp7, Cpt1a, Dgat1, Etfdh, Fabp5, Gk, Lpl, Mlycd*, or *Pck1*; whereas 2 genes associated with cell proliferation (*Hspd1*, *Txnip*) and 1 with fatty acid metabolism (*Fabp3*) were significantly up-regulated in the maternal liver but not the fetal liver. Further, the maternal and fetal livers shared the most highly up-regulated gene (*Ehhadh*; 55-fold in maternal liver) and both had highly up-regulated *Cpt1b* expression (24-fold in maternal liver). Only 1 of the shared genes was noticeably more highly up-regulated in the maternal liver than the fetal liver (*Ech1*; 18-fold vs. 6-fold in maternal and fetal livers, respectively). Overall, the PPAR signaling pathway was up-regulated in both maternal and fetal livers, with both sharing many of the same up-regulated genes; however, the overall profiles of induction were noticeably different between the two life stages, with the fetal liver seemingly displaying greater sensitivity both in terms of the number of genes affected and the degree of up-regulation.

During the GD14–18 dosing window, dams had significantly less body weight gain at the 250- and 500-mg/kg dose levels compared with controls (ANOVA $p = 0.0037$; Figure 3A; see also Table S5). On GD18, dams had significantly higher liver weights in the 62.5-to 500-mg/kg dose groups than controls (ANOVA $p < 0.0001$; Figure 3B; see also Table S5). There were no significant differences in numbers of live pups, resorptions, or fetal body weight compared with controls (see Table S5).

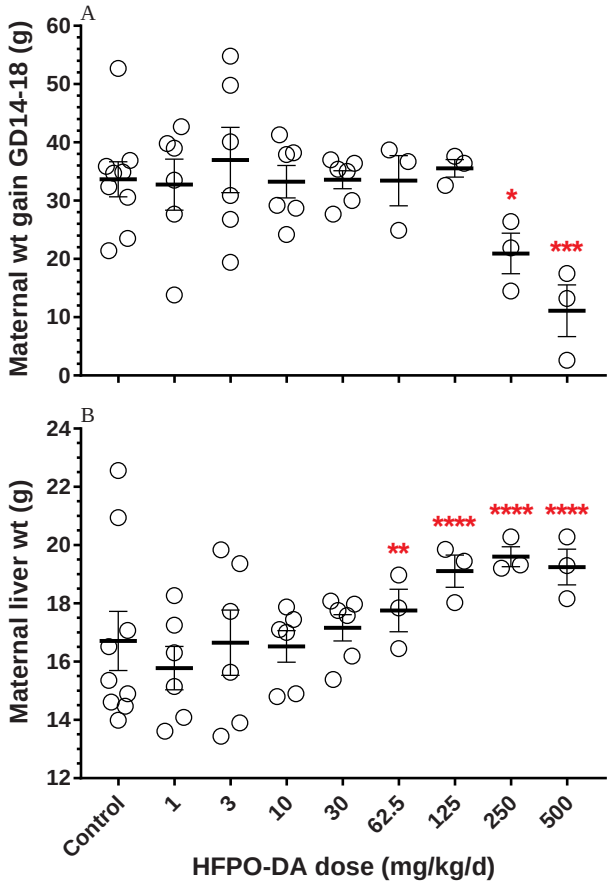

Figure 3. (A) Maternal body weight gain during gestation day (GD)14–18 dosing period and (B) maternal liver weight on GD18. Data points represent individual replicates (control, $n = 9$; 1–30 mg/kg, $n = 6$; 62.5–500 mg/kg, $n = 3$), bars and whiskers represent mean ± standard error, and asterisks represent significant differences compared with control values (*, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$; ****, $p < 0.0001$). Statistical significance was determined using analysis of variance; for liver weight analysis, body weight was included as a covariate.

Maternal serum samples displayed dose–responsive decreases in all measures of thyroid hormones and lipids (Figure 4; see also Table S5). Serum triglycerides were significantly lower at 500 mg/kg, cholesterol and HDL were significantly lower at 250 and 500 mg/kg, and total T_4 and LDL were significantly lower at ≥125 mg/kg. The most sensitive end point was serum total T_3, which was significantly lower at ≥30 mg/kg and below assay detection levels (i.e., <0.2 ng/mL) in the top two dose levels.

Postnatal Effects from GD14–18 Dosing

In the HFPO-DA pilot postnatal study that utilized GD14–18 dosing, one of three control dams was not pregnant, reducing the sample size to $n = 2$ litters. Control dams and dams dosed with 125 mg/kg HFPO-DA gave birth to litters with equal numbers of viable pups. On a litter means basis, there were no significant differences for any end point measured through the onset of puberty (see Table S7). On an individual pup basis (as opposed to litter means), female offspring body weight was significantly lower than controls at multiple time points (PND2, PND27, and at VO), indicating a potential trend in growth deficit to investigate in future studies.

Adult males at necropsy had significantly lower tissue weight of the right epididymis on a litter means basis, but no other tissues were affected as compared with controls (see Table S8). On an individual basis, treated male rats had significantly lower tissue weights of the right testis, left testis, paired testes, right

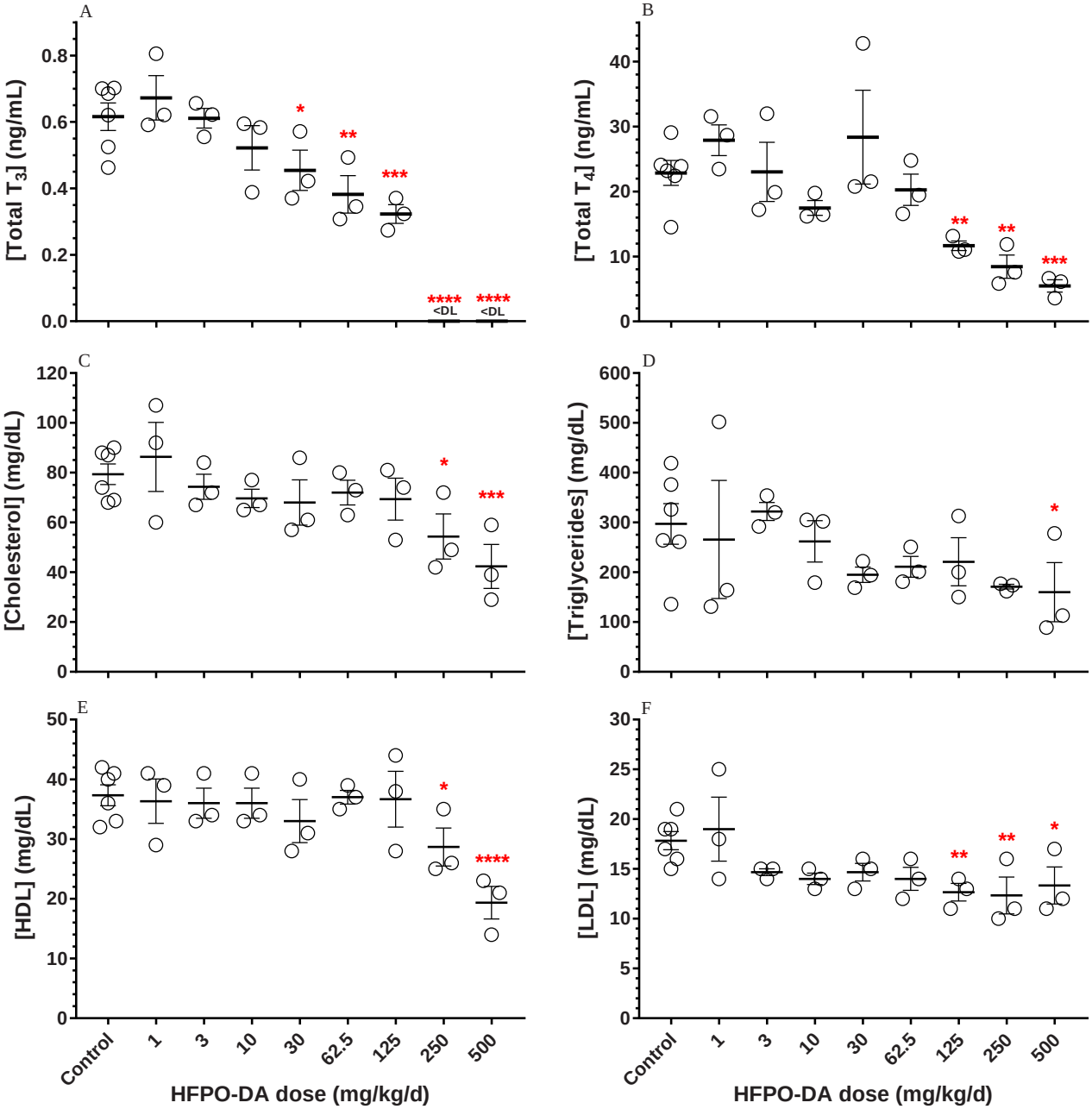

Figure 4. Concentrations of (A) total triiodothyronine (T_3), (B) total thyroxine (T_4), and lipids [(C) cholesterol, (D) triglycerides, (E) high-density lipoproteins (HDL), and (F) low-density lipoproteins (LDL)] in maternal serum following oral hexafluoropropylene oxide dimer acid (HFPO-DA) dosing from gestation days (GD) 14–18. Dam serum was collected on GD18 approximately 2 h after final oral dose. Data points represent individual replicates (control, $n = 6$; treated, $n = 3$), bars and whiskers represent mean ± standard error, and asterisks represent significant differences compared with control values using analysis of variance (*, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$; ****, $p < 0.0001$). <DL, values below radioimmunoassay detection limit.

epididymis, left epididymis, paired epididymides, and epididymal adipose tissue as compared with controls.

Adult females at necropsy displayed no significant differences in any end point as compared with controls on a litter means basis (see Table S9). On an individual basis, treated female rats had significantly smaller AGD and lower liver weight as compared with controls.

HFPO-DA Concentrations in Maternal Serum and Fetal Plasma

Maternal serum and fetal plasma contained increasing concentrations of HFPO-DA as a function of oral dose following dosing during the GD14–18 experimental window (Figure 5; see also Table S10). Over the full maternal dose range (1–500 mg/kg), uptake appeared to saturate at the higher dose levels and was modeled using exponential one-phase association ($R^2 = 0.84$) with a plateau of 112 ± 15 μg/mL (Figure 5A). In the lower dose range (1–30 mg/kg), increases in maternal serum and fetal plasma HFPO-DA concentrations were linear (Figure 5B); however, the maternal slope was significantly greater than the fetal slope with maternal serum HFPO-DA increasing 0.46 μg/mL and fetal plasma HFPO-DA concentration increasing 0.12 μg/mL for each 1-mg/kg increase in oral maternal dose ($p < 0.0001$).

Dose–Response Analyses

Using maternal serum HFPO-DA concentrations, we estimated effect concentrations for an EC_5 for the most sensitive end points: maternal liver weight, maternal liver gene expression, and maternal serum [T_3] and [T_4] (Figure 6). Maternal [T_3] was the most sensitive end point with an EC_5 of 3.8 μg/mL (estimated maternal oral dose of 8.2 mg/kg using the linear equation from Figure 5) followed by liver *Ehhadh* expression ($EC_5 = 14.1$ μg/mL), liver weight ($EC_5 = 17.6$ μg/mL), and [T_4] ($EC_5 = 17.8$ μg/mL).

Comparison of Maternal Rat and Human Internal Exposure Levels

The human worker HFPO-DA plasma concentrations reported by Dupont (2017) ranged from 0.001–0.169 μg/mL, whereas the mean maternal rat serum concentrations reported here ranged from 0.68–100.7 μg/mL following a 5-d exposure. At the lowest dose level tested here (1 mg/kg), the rat:human MOIEs ranged from 4 to 566 (14/17 MOIEs were >100; Figure 7A). Further, at the dose utilized in the postnatal pilot study (125 mg/kg), the rat:human MOIEs ranged from 272 to 38,333 (15/17 MOIEs were >1,000 and 12/17 MOIEs were >10,000; Figure 7B). It is important to note that the maternal rat serum concentrations utilized in this comparison were from short-term (5-d) exposures, whereas the human plasma concentrations were from individuals working in an HFPO-DA manufacturing facility and likely represent chronic exposure levels, but it is unknown whether these concentrations represent a steady state.

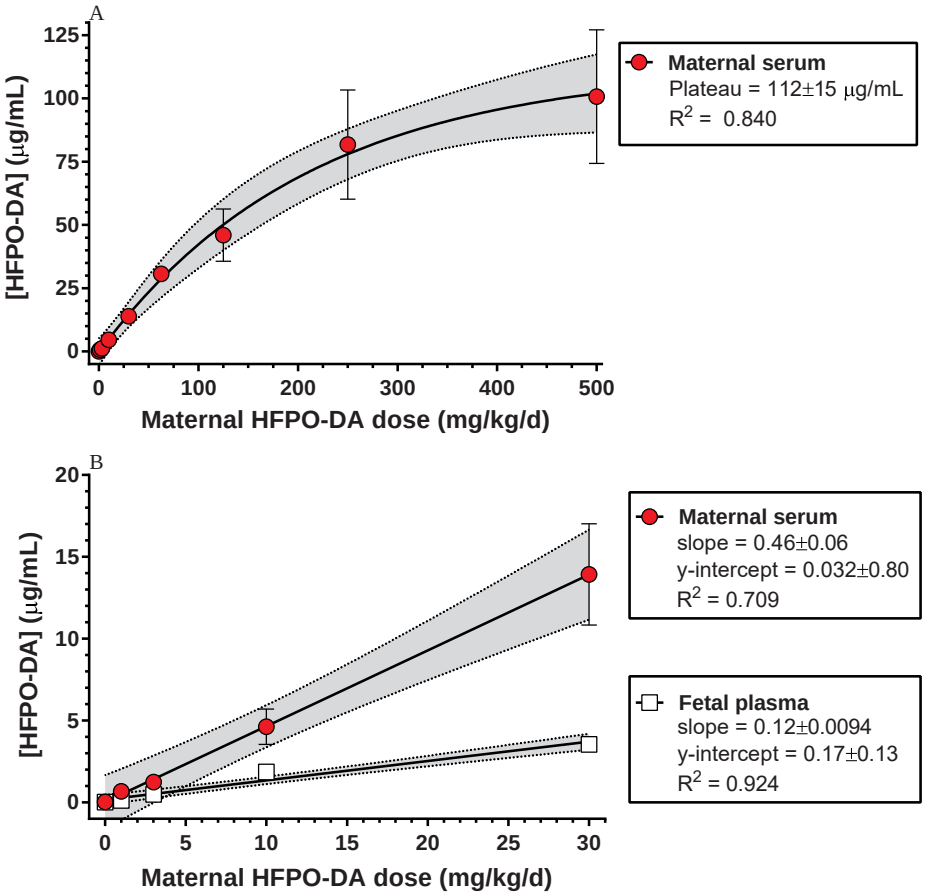

Figure 5. Maternal serum and fetal plasma hexafluoropropylene oxide dimer acid (HFPO-DA) concentrations (mean ± standard error, $n = 3$–9; see Table S10) as a function of oral dose following maternal exposure from gestation day (GD) 14–18. Samples were collected on GD18 approximately 2 h after final oral dose. (A) Full maternal dose range modeled using exponential one-phase association and (B) low dose range modeled using linear regression (95% confidence intervals shaded). Fetal plasma was collected only from the low dose range (1–30 mg/kg per day).

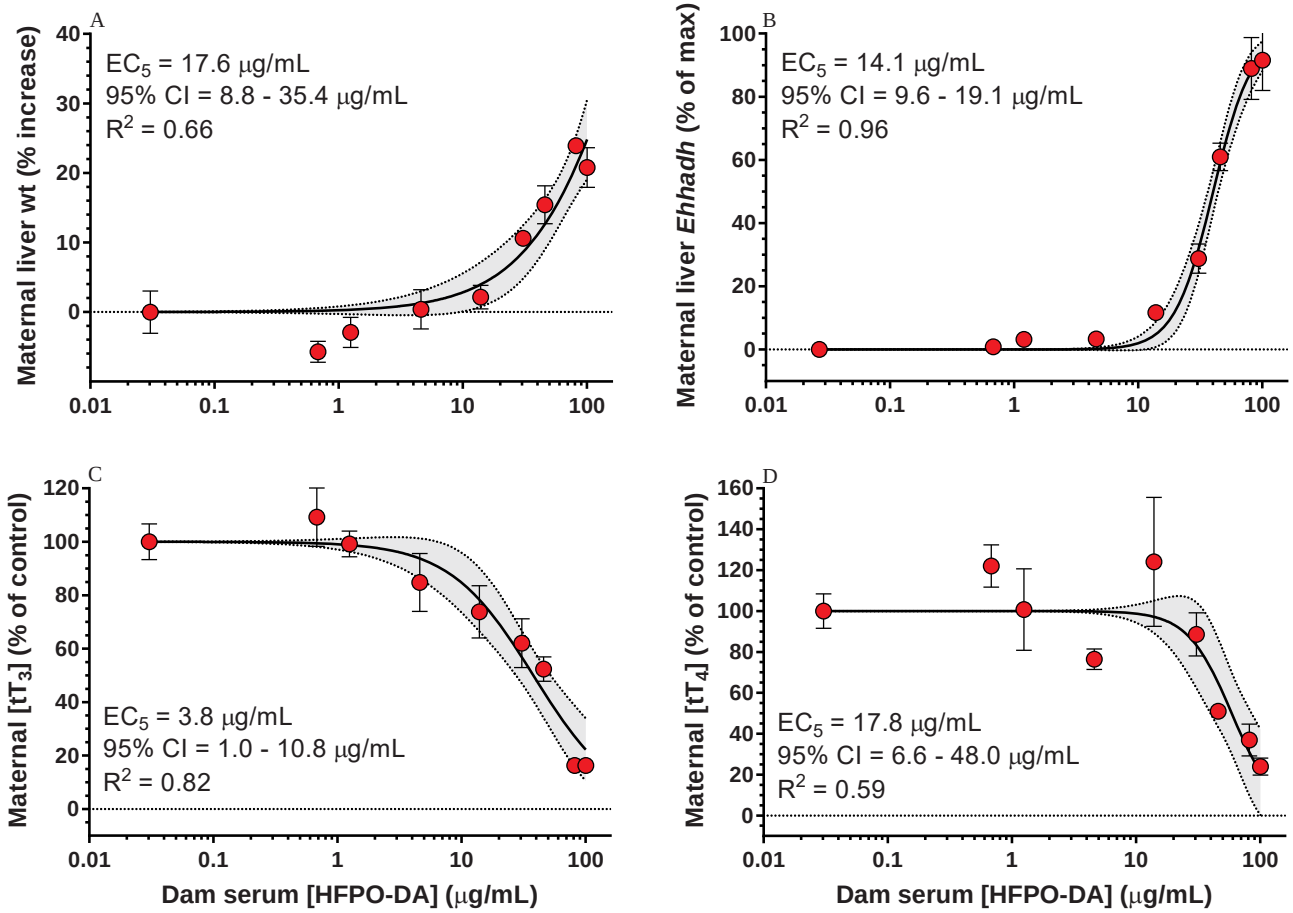

Figure 6. Dose–response curves (four-parameter logistic regression) and 5% effect estimates [EC_5 with 95% confidence intervals (CIs)] for the most sensitive end points [(A) maternal liver weight, (B) maternal liver *Ehhadh* gene expression, (C) maternal serum total triiodothyronine (tT_3), and (D) total thyroxine (tT_4)] as a function of maternal serum hexafluoropropylene oxide dimer acid (HFPO-DA) concentration. Dam serum HFPO-DA concentrations represent those measured on gestation day (GD)18 following GD14–18 dosing. Data points represent mean ± standard error, (A) control $n = 9$, 1–30 mg/kg per day $n = 6$, 62.5–500 mg/kg per day $n = 3$; (B–D) control, $n = 6$; treated, $n = 3$.

In Vitro *Nuclear Receptor Transactivation*

HFPO-DA did not display any estrogenic activity (agonism or antagonism) at concentrations ranging from 100 pM to 10 μM (see Figure S2). Further, there was no androgen or glucocorticoid receptor agonism at concentrations ranging from 100 pM to 100 μM. At the very highest dose tested (100 μM), which approached the cytotoxic dose of 300 μM, HFPO-DA exposure did result in a slight glucocorticoid receptor antagonism ($28 \pm 3\%$ reduction in luciferase expression) and a moderate androgen receptor antagonism ($42 \pm 1\%$ reduction).

Discussion

The range of adverse effects resulting from oral maternal HFPO-DA exposure reported here are consistent with limited data available for HFPO-DA (Caverly Rae et al. 2015; DuPont 2008a, 2010; Gannon et al. 2016; Rushing et al. 2017; Wang et al. 2017a) and the extensive toxicity literature available for other PFAS, notably PFOS and PFOA [reviewed by ATSDR (2018), ECHA (2014), OECD (2002) and U.S. EPA (2016a)]. We observed up-regulation of genes associated with PPAR signaling pathways, maternal hepatomegaly, reductions in maternal serum lipids and thyroid hormones, and indications of reduced body and tissue weights in F1 animals. All of these effects have been observed following maternal exposure to PFOS/PFOA in laboratory animals and several have been previously observed for HFPO-DA. However, despite extensive PPAR pathway up-regulation, HFPO-DA did not produce any effects that are hallmarks of phthalate syndrome, including reduced fetal testis testosterone production, phthalate-specific fetal testis gene expression changes, reduced AGD on PND2, or male reproductive malformations. This lends support to the hypothesis that the effects of phthalates on male reproductive development are not mediated via the PPAR pathway.

The specific dosing interval utilized in developmental toxicity studies with PFAS is a critical factor for the types of effects that have been described. Grasty et al. (2003) reported significantly increased neonatal mortality and reduced pup weight in Sprague-Dawley rats following gestational PFOS exposure at 25 mg/kg across a range of 4-d dosing windows. These effects increased in severity as the dosing window moved later in gestation. Further, it was demonstrated that dosing only on GD19–20 was sufficient to produce these effects. Subsequent studies that included dosing during the full gestational period also reported pup mortality and reduced pup body weight. Lau et al. (2003) examined PFOS exposure in the rat and reported significantly increased neonatal mortality shortly after birth (<24 h) at ≥3 mg/kg. Separate studies in Sprague-Dawley rats confirmed the neonatal mortality following gestational exposure to PFOS at ≥1.6 mg/kg (Luebker et al. 2005a, 2005b). Similar results have been reported with other PFAS, primarily PFOA, and in other species, including mice and cynomolgus monkeys [reviewed by Abbott (2015) and Lau et al. (2007)]. In the pilot postnatal study presented here,

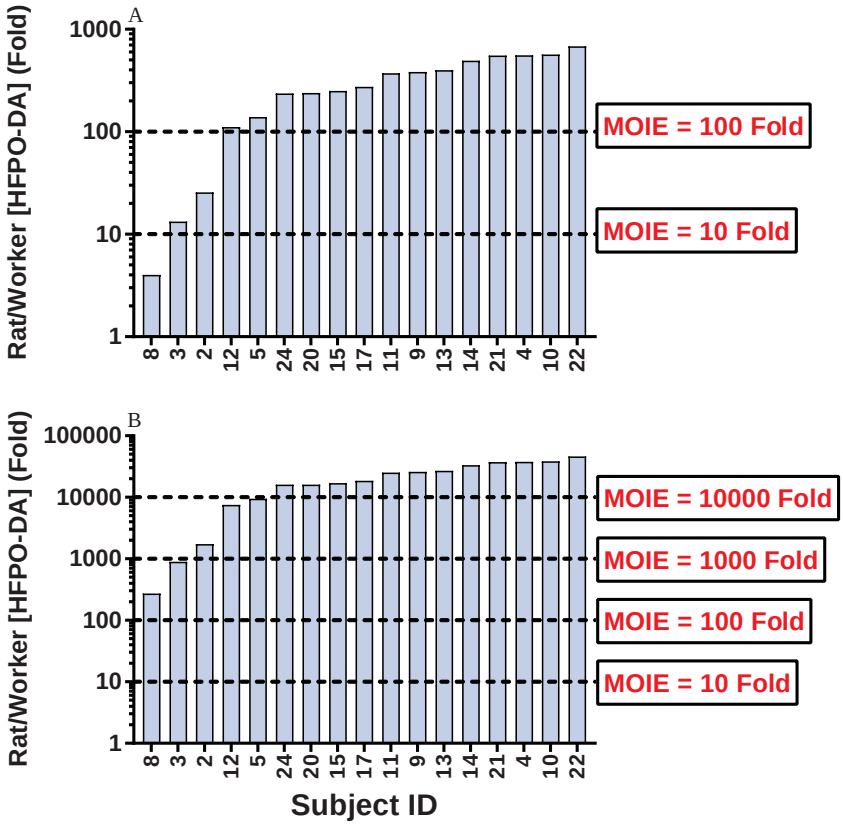

Figure 7. Comparison of mean maternal Sprague-Dawley rat serum hexafluoropropylene oxide dimer acid (HFPO-DA) concentration from (A) 1- and (B) 125-mg/kg per day exposure groups and individual human plasma HFPO-DA concentrations from workers in an HFPO-DA manufacturing facility in the Netherlands (DuPont 2017). Horizontal lines indicate various margins of internal exposure (MOIE) levels as compared with individual worker plasma concentrations.

there was an indication of decreased female pup weight but no effect on pup survival following HFPO-DA exposure from GD14–18 at a relatively high dose (125 mg/kg). However, expanding the dosing timeline to include the entire period of fetal development (i.e., GD8 through parturition) appears to reduce neonatal survival and body weight similar to PFOS exposure but at ~20-fold higher oral maternal doses [J.M. Conley and L.E. Gray (personal communication)].

As mentioned above, female pup body weight in the HFPO-DA dose group was significantly lower, on an individual analysis basis, 2 d after birth compared with control animals. Previous studies with laboratory rats have reported stunted growth of surviving pups following PFOS exposure. Lau et al. (2003) reported that pups exposed *in utero* to PFOS at ≥2 mg/kg displayed lower body weights, and Luebker et al. (2005b) reported the same response in all dose levels tested (i.e., ≥0.4 mg/kg). Overall, reduced pup weight appears to be one of the most sensitive end points in *in utero* PFAS studies. This effect aligns with multiple epidemiological studies, indicating a negative association between human birth weight and concentrations of PFOS/PFOA [reviewed by Bach et al. (2015) and Negri et al. (2017)] and should be more extensively evaluated for HFPO-DA exposure.

PFAS are known to primarily activate PPARα, particularly in the mammalian liver, however other receptors, such as PPARγ, have also been shown to be activated (Vanden Heuvel et al. 2006). Although the biological significance of induction of PPAR pathway gene expression is not known, it was overall the most sensitive end point in the present studies. Even at the lowest dose tested (1 mg/kg), the fetal liver displayed multiple significantly up-regulated genes (*Cpt1b, Acox1, Angptl4*). Bjork et al. (2008) performed a similar experiment with gestational PFOS exposure in the SD rat (exposed to 3 mg/kg from GD2 to GD20) and identified 445 genes via microarray that were significantly altered in the fetal liver. Four genes associated with fatty acid metabolism were individually verified using qPCR, 3 of which were also identified as significantly up-regulated in the present study (*Acox1, Cpt1a, Cpt1b*). Further, maternal PPAR pathway gene expression was almost equally as affected as the fetal livers, however with a notably distinct profile. Wang et al. (2017a) reported up-regulation of PPAR pathway genes in mouse liver following HFPO-DA exposure, whereas Hu et al. (2005) and Martin et al. (2007) performed microarray analyses of adult rat liver gene profiles following oral PFOS and PFOA exposure and reported similar up-regulation of clusters of genes primarily associated with lipid homeostasis. The gene expression profiles reported here indicate that HFPO-DA reached the fetal organs and activated nuclear receptor–mediated cell-signaling pathways and that the profile of expression was different than the maternal gene expression profile. However, the findings are not adequate to definitively conclude that a PPARα mechanism of action is operative for the HFPO-DA effects observed here.

In addition to changes in PPAR-mediated gene expression in the maternal liver, we observed a number of alterations to maternal serum lipid and thyroid hormone profiles similar to previous PFAS studies. Luebker et al. (2005b) reported significantly reduced serum cholesterol in pregnant SD rats following PFOS exposure, and Martin et al. (2007) also reported significantly reduced serum cholesterol in adult male SD rats following both PFOS and PFOA exposure. Disruption of maternal rat cholesterol synthesis with a HMG-CoA reductase inhibitor *in utero* has been

shown to induce fetal and neonatal death and retard growth in the absence of maternal toxicity (Henck et al. 1998). It is believed that the majority, if not all, of the cholesterol utilized in the earliest stages of fetal development is derived from the mother, prior to the onset of fetal cholesterol synthesis (Baardman et al. 2013). Further, Martin et al. (2007), Thibodeaux et al. (2003), and Yu et al. (2009) reported significant reductions in serum total T_3 and T_4 for both PFOS and/or PFOA; however, T_4 appeared to be more greatly reduced, whereas in the present study T_3 was more affected. Maternal thyroid hormones are critical for fetal neurological development because the mother is the primary source of T_4 for the developing brain (Morreale de Escobar et al. 2004) and reduced maternal thyroid hormone concentrations are quantitatively linked to reduced fetal concentrations (O'Shaughnessy et al. 2018). Despite the consistency observed across laboratory rat studies, it is unclear how these results relate to human health effects from PFAS exposure because many epidemiological studies report the opposite patterns or equivocal results (Lau et al. 2007; U.S. EPA 2016a).

Gomis et al. (2018) recently reported on the potential discrepancy in toxicity among a range of PFAS when using orally administered dose as compared with internal dose. By accounting for toxicokinetics in rats across multiple PFAS, the toxicity of some fluorinated alternatives appears to be more equitable to the long-chain PFAS when potency is compared based on internal dose. However, it is important to highlight the substantial toxicokinetic differences between PFOS and HFPO-DA in the rat. In the female rat, HFPO-DA has a reported half-life of $\sim$5 h following oral exposure to 10–30 mg/kg (Gannon et al. 2016) and is not expected to accumulate, whereas PFOS has a reported half-life of $\sim$60–70 days following oral exposure to 2–15 mg/kg (Chang et al. 2012) and does accumulate. Our samples were collected 2 h after the final oral dose, which is just slightly after the peak serum concentration is achieved in the female rat based on the Gomis et al. (2018) model.

In addition to intraspecies differences in PFAS toxicokinetics, it is also important to note that interspecies differences in absorption, distribution, metabolism, and excretion of PFAS are vast, with half-lives and clearance rates of numerous compounds appearing to be significantly longer in humans and nonhuman primates than in rats/mice (Chang et al. 2012; Olsen et al. 2007). The half-life of HFPO-DA in humans is currently unknown; however, similar to the discussion above, internal dosimetry can potentially reduce uncertainty in cross-species hazard assessment. For comparison, we calculated MOIE values for maternal rat serum concentrations versus plasma samples from humans working in a HFPO-DA manufacturing facility in the Dordrecht, Netherlands (DuPont 2017) (Figure 7). Bessems et al. (2017) originally described the use of MOIE as a physiologically based kinetic modeling approach for reducing uncertainty in the safety assessment of human dermal exposures using oral rodent toxicity data. Comparison of MOIE accounts for species- and route-dependent differences in metabolism between humans and research animals. Here, we utilized a similar calculation to reduce the species-to-species variation in PFAS toxicokinetics and to provide context for the oral doses utilized in terms of known human exposure levels. The highest detected plasma concentration from a worker (0.169 μg/mL) was 4-fold lower than the mean maternal rat serum HFPO-DA concentration from the lowest dose level (1 mg/kg per day) reported here; whereas the same worker concentration was 272-fold below the mean maternal serum concentration from the dose level (125 mg/kg per day) used in the pilot postnatal study presented here. Overall, characterizing toxicokinetics and internal dosimetry for PFAS, including HFPO-DA, can facilitate the determination of the relevance of doses in laboratory animals to human exposures, thereby reducing some of the uncertainty in estimating human health risks from exposure.

The HFPO-DA toxicity profile observed here was highly similar to effects observed in peer-reviewed and industry guideline studies for HFPO-DA as well as in studies conducted for PFOS (among other PFAS). PPAR signaling pathways were activated in maternal and fetal livers and may also be activated in other tissues/organs; however, the effects observed are not necessarily exclusive to PPARα, or even PPAR signaling in general (Rosen et al. 2017). The GenX chemicals health assessment is currently undergoing independent, external peer-review in the Office of Water (U.S. EPA). Included in that assessment is a summary of available mode-of-action (MOA) information. Although findings in this study are consistent with other PPARα agonists (e.g., increases in liver weight, up-regulation of PPAR pathway target genes), data gaps exist for key events and other mechanisms that might be involved, particularly in other tissues besides those like the liver with high PPARα levels. Overall, the findings for HFPO-DA are limited and not adequate to support ascribing a PPARα MOA to the multitude of effects seen in this study. Due to the reductions in maternal serum thyroid hormones and lipids observed here, and preliminary studies in our lab, an expanded dosing period that includes the entire period of fetal development may lead to effects on fetal and neonatal development similar to those observed with PFOS and PFOA exposure. Extensive research is needed to investigate the mechanism(s) by which HFPO-DA/PFOS/PFOA produce toxicity, to characterize the toxicokinetics for this and other PFAS in order to better predict toxic effects, and to assess the mixture-based effects of exposure to multiple PFAS compounds given their ubiquitous occurrence.

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NORTH CAROLINA BLADEN COUNTY	IN THE GENERAL COURT OF JUSTICE SUPERIOR COURT DIVISION 17 CvS 580
STATE OF NORTH CAROLINA, *ex rel.*, MICHAEL S. REGAN, SECRETARY, NORTH CAROLINA DEPARTMENT OF ENVIRONMENTAL QUALITY, Plaintiff, v. THE CHEMOURS COMPANY FC, LLC, Defendant.	**RENEWED AND AMENDED MOTION TO INTERVENE BY CAPE FEAR PUBLIC UTILITY AUTHORITY (VERIFIED)**

EXHIBIT H
TO AMENDED INTERVENOR COMPLAINT

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Toxicity of Balb-c mice exposed to recently identified 1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl] oxyethane-1-sulfonic acid (PFESA-BP2)

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ABSTRACT

1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl]oxyethane-1-sulfonic acid (PFESA-BP2) was first detected in 2012 in the Cape Fear River downstream of an industrial manufacturing facility. It was later detected in the finished drinking water of municipalities using the Cape Fear River for their water supply. No toxicology data exist for this contaminant despite known human exposure. To address this data gap, mice were dosed with PFESA-BP2 at 0, 0.04, 0.4, 3, and 6 mg/kg-day for 7 days by oral gavage. As an investigative study, the final dose groups evolved from an original dose of 3 mg/kg which produced liver enlargement and elevated liver enzymes. The dose range was extended to explore a no effect level. PFESA-BP2 was detected in the sera and liver of all treated mice. Treatment with PFESA-BP2 significantly increased the size of the liver for all mice at 3 and 6 mg/kg-day. At the 6 mg/kg-day dose, the liver more than doubled in size compared to the control group. Male mice treated with 3 and 6 mg/kg-day and females treated with 6 mg/kg-day demonstrated significantly elevated serum markers of liver injury including alanine aminotransferase (ALT), glutamate dehydrogenase (GLDH), and liver/body weight percent. The percent of PFESA-BP2 in serum relative to the amount administered was similar in male and female mice, ranged from 9 to 13 %, and was not related to dose. The percent accumulation in the liver of the mice varied by sex (higher in males), ranged from 30 to 65 %, and correlated positively with increasing dose level.

1. Introduction

Perfluoroalkyl substances (PFASs) have been detected in the global environment, including points far from sites of production and/or use. (Giesy and Kannan, 2001) The unique stability of the carbon-fluorine bond results in PFASs having exceedingly long environmental half-lives (Banks et al., 2013). Concerns about PFASs have resulted in establishment of regulations for some PFASs and voluntary advisory levels for others (ITRC Council, 2018). Public concerns and regulatory guidelines have focused on a small number of PFASs. Although there are currently thousands of compounds categorized as PFASs (Wang et al., 2017), there have been only approximately 1223 PFAS historically registered in commerce in the US, with 602 actively in commerce today (USEPA, 2019).

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; ESI, electrospray ionization; GLDH, glutamate dehydrogenase; HDPE, high density polyethylene; IACUC, Institutional Animal Care and Use Committee; NCDEQ, North Carolina Department of Environmental Quality; NRC, National Research Council; NVHOS, 1,1,2,2-tetrafluoro-2-(1,2,2,2-tetrafluoroethoxy)ethanesulfonic acid DTXSID80904754; PCR, polymerase chain reaction; PFASs, perfluoroalkyl substances; PFESA-BP2, 1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl]oxyethane-1-sulfonic acid DTXSID10892352; PFOA, perfluorooctanoic acid DTXSID8031865; PFOS, perfluorooctanoic sulfonic acid DTXSID3031864; HFPO-DA, perfluoro-2-methyl-3-oxahexanoic acid DTXSID70880215; TSCA, Toxic Substance Control Act; USEPA, United States Environmental Protection Agency

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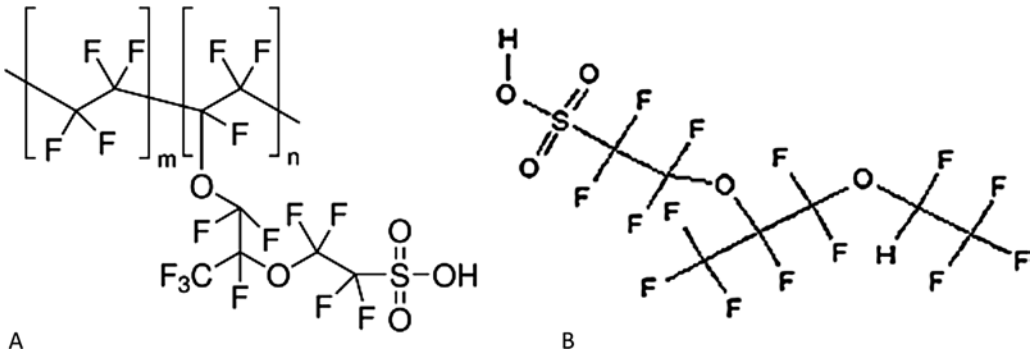

Fig. 1. Structure of the Nafion Polymer (A) and PFESA-BP2 (B).

1,1,2,2-tetrafluoro-2-[1,1,1,2,3,3-hexafluoro-3-(1,1,2,2-tetrafluoroethoxy)propan-2-yl]oxyethane-1-sulfonic acid (PFESA-BP2 CAS #749836-20-2) is assumed to exist as a by-product of manufacturing Nafion polymer (Fig. 1). PFESA-BP2 has not been the subject of a pre-manufacture notice and review under the US Toxic Substance Control Act (TSCA), which is required only for chemicals intended for a commercial purpose. By-product release into the environment does not follow the same laws as chemicals intended for commerce, therefore there is no toxicology information requirement. PFESA-BP2 is a 7-carbon sulfonate with an monoisotopic mass of 463.93 amu and with two internal ether oxygens, giving it a mass and general structure (length) that is similar to perfluorooctanoic sulfonic acid (PFOS - 498.93 amu). These similarities may infer a longer half-life and possibly similar toxicity. Because the compound is a by-product of Nafion, a sulfonated tetrafluoroethylene-based polymer, it also has been referred to as Nafion by-product 2.

In 2012, two PFESA byproducts (i.e. PFESA-BP2 and perfluoro-3,6-dioxa-4-methyl-7-octene-1-sulfonic acid (PFESA-BP1 CAS #29311-67-9 DTXSID30892354))were detected in North Carolina's Cape Fear River, downstream of an industrial manufacturing facility. (Strynar et al., 2015) In a September 2017 report to the North Carolina Department of Environmental Quality (NCDEQ), the United States Environmental Protection Agency (USEPA) used a non-targeted analytical method to estimate PFESA-BP2 concentrations in Chemours discharge effluent and the Cape Fear River downstream of manufacturing as 45,200 ng/L and 2075 ng/L, respectively. (Buckley, 2017) These reported PFESA-BP2 concentrations were provided as gross estimates because a PFESA-BP2 standard was unavailable at that time. As such, these concentrations assume that the mass spectrometer responded to the non-targeted analyte as if it were Perfluoro-2-methyl-3-oxahexanoic acid, HFPO-DA, CAS #13,252-13-6], for which a standard was available. The report suggests such estimates are accurate to within 10-fold of the estimated value.

In July 2017, North Carolina's Brunswick County drinking water provider (H2Go) began bi-weekly sampling for PFESA-BP2, with concentration estimates ranging from non-detectable (ND) to 134 ng/L in their finished drinking water. (H2GO PFC Sampling, 2020) NCDEQ reported PFESA-BP2 in private wells near the industrial manufacturing facility with concentrations up to 125 ng/L (NCDEQ, 2018). With the availability of an authentic standard provided by the manufacturer, subsequent studies corroborated PFESA-BP2 contamination in finished drinking water (Hopkins et al., 2018), but also in 99 % of serum samples from public volunteers from this same region (Katlorz, 2018). The study demonstrated the presence of PFESA-BP2 is likely isolated to the area downstream of the NC industrial manufacturing facility because serum samples from residents of Raleigh, NC, Chapel Hill, NC, Durham, NC and Dayton, Ohio did not contain this compound. These studies demonstrate the presence of PFESA-BP2 contamination in water sources within the Cape Fear River Basin, as well as the widespread presence of this compound in human serum samples from this same region.

Despite the known presence of PFESA-BP2 in the environment and in human blood, there are no known toxicology studies utilizing PFESA-BP2. Previous studies on perfluorooctanoic acid PFOA and PFOS have demonstrated that these compounds bioaccumulate in the liver and serum of affected animals (rat, mouse, rabbit, monkey), and induce liver toxicity. (Lau et al., 2006; Yang et al., 2014)

Given the potential health effects associated with PFAS compounds and the presence of PFESA-BP2 in human serum, this initial study examined the hepatotoxic effects and bioaccumulation of PFESA-BP2 in adult mice exposed by oral gavage for seven days (0.04–6 mg/kg-day).

2. Material and methods

2.1. Animals

Balb-c mice, an inbred strain we have used to study hepatotoxic algal toxins, 10 − 12-week-old males and females, were obtained from Charles River Laboratories (Raleigh, NC, USA). The animals arrived at the US EPA's National Health and Environmental Effects Research Laboratory (NHEERL) animal facility post-weaning and allowed to acclimate for at least 5 days prior to initiation of the experiments. Animals were randomly selected, but cage groups were corrected to keep the body weight variance < 1. Animals were housed by treatment group in polycarbonate cages on heat-treated pine shaving bedding in animal rooms with a controlled temperature range (22–26 °C) and a 12:12-h light–dark cycle. Animals were fed commercial rodent chow (Purina Prolab) and water ad libitum. All studies were conducted after approval by the USEPA Institutional Animal Care and Use Committee (IACUC) using recommendations of the 2011 National Research Council (NRC) "Guide for the Care and Use of Laboratory Animals" and the Public Health Service Policy on the Humane Care and Use of Laboratory Animals. (Guide for the Care and Use of Laboratory Animals, 2011)

2.2. Experimental design

Animals were dosed with PFESA-BP2 for seven consecutive days by gavage using 20-gauge stainless steel feeding needles. Seven-day exposure was chosen to enable demonstration of bioaccumulation and a dose of 3 mg/kg was used which exhibited effects. Additional dosages were added in later blocks to establish a wide range of responses. The complete experiment was run across five different blocks. Each block included control animals, and each dose group was used in at least two blocks, except for the highest dose (6 mg/kg) which was not repeated. Doses of 0, 0.04, 0.4, 3, and 6 mg/kg-day were administered once daily in the afternoon. The number of animals ranged from 10 to 24 per dose group, divided equally between males and females. Animals were weighed before the dosing was started, every other day during dosing, and at the time of euthanasia. Their appearance was monitored daily. PFESA-BP2 was obtained from Chemours (78.8 % purity - 14 % potassium fluoride (KF) – 6.6 % (1,1,2,2-Tetrafluoro-2-(1,2,2,2-

tetrafluoroethoxy) ethanesulfonic acid (NVHOS CAS #801209-99-4)). A stock dosing solution was prepared by dissolving PFESA-BP2 in ethanol (EtOH) followed by dilution with deionized (DI) water for a final concentration of 1 g/L in 90:10 DI H2O:EtOH. The stock solution was diluted with DI water to establish dosing solution concentrations for each treatment at a dosing volume of 0.2 mL per day. The final PFESA-BP2 concentration in the dosing solutions ranged from 0.002 to 0.8 g/L (data not shown). The control group received the carrier of Picopure water with an ethanol concentration equal to the dosing solution with the highest ethanol concentration which was always the high dose males (did not exceed 7.15 % ethanol).

Approximately 24 h after the seven-day dosing was completed, all animals were anesthetized by CO_2 inhalation, weighed, euthanized by exsanguination (blood collection), and necropsied. Blood was obtained transdermally from the heart with a 25-gauge 5/8 in needle attached to a 1 mL syringe. Whole blood was collected in 0.5 mL serum separator tubes (Becton Dickinson), allowed to clot at room temperature, centrifuged at 13,000 rpm for 1.5 min per manufacturer's instructions (Dickinson, 2011), and serum isolated. Serum samples were stored at −20 °C in 2.5 mL high density polyethylene (HDPE) tubes until analysis. The liver was removed from each animal, weighed, and divided into samples. One sample of the liver was stored in foil at −20 °C for PFESA-BP2 analysis, a sample from the largest liver lobe was fixed in 10 % neutral buffered formalin for 48 h before being transferred to 70 % ethanol for histopathology, and a third sample was placed in RNAlater and stored at −20 °C for polymerase chain reaction (PCR) analysis at a later time.

2.3. Histopathology

Samples of liver from one male and one female mouse from the control, 0.4 mg/kg-day and 3 mg/kg-day treatment groups were viewed microscopically to study the appearance of the cells by Pathogenesis LLC (Gainesville, FL). Limited resources restricted the number of tissues that could be processed and analyzed, but our main goal was to confirm that the increased liver weight was due to hepatocyte hypertrophy as seen with other PFAS (Toxicologic Profile of Perfluoroalkyls, Draft, US Dept. HHS, 2018) versus hepatocyte hyperplasia. Livers from the 0.04 mg/kg-day and 6 mg/kg-day group were not analyzed with histopathology. Each block was sectioned at 5 microns and stained with hematoxylin and eosin according to a previously published methodology. (Chernoff et al., 2018) Tissue sections were evaluated microscopically without the evaluator having prior knowledge of the treatment group. Histologic features were scored using a semi-quantitative scoring scheme with 0 = no change to 4 = severe change (Chernoff et al., 2018). Numbers of individual apoptotic hepatocytes (consistent with apoptosis) and mitotic figures were counted in each of ten 400X fields centered on a central vein.

For computer-aided image analysis of Zone 3 (centrilobular) hepatocytes, multiple photomicrographs at 1000X magnification were collected from at least 5 randomly selected hepatic lobules per mouse. The area of 30 individual hepatocytes from Rappaport Zone 3 of each liver was calculated using the lasso tool in Photoshop (lasso to outline individual hepatocytes > Image > Analysis > Record Measurement), Adobe Photoshop CC 2017.

2.4. Clinical chemistry

All serum clinical chemistry analyses were carried out using the Randox Daytona Plus instrument (Belfast, Northern Ireland). Due to serum volumes < 300 µL, serum chemistries were not performed in duplicates, Hepatic cell and bile duct injury was assessed by determining the serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), glutamate dehydrogenase (GLDH), and bilirubin. Markers for potential renal injury included serum concentrations of blood urea nitrogen (BUN) and creatinine. Serum glucose, total protein, and albumin were measured as markers of general toxicity. All assays were performed using reagents obtained from the instrument manufacturer.

2.5. Extraction and analysis of PFESA-BP2 from tissue and serum

PFESA-BP2 was extracted from serum and tissue samples using methods presented in Reiner et al., 2009. (Reiner et al., 2009) In brief, liver samples were weighed in 15 mL HDPE centrifuge tubes and homogenized at approximately a 3:1 DI:sample wet weight ratio using an Omni-Prep Multi Sample Homogenizer. Liver homogenate and serum samples from mice treated with PFESA-BP2 were diluted at variable ratios with DI water to bring the concentrations within the values of the external calibration curve. Serum and liver homogenates from control mice were analyzed directly without dilution. The diluted samples (50 µL) were pipetted into a fresh 15 mL HDPE centrifuge tube, followed by 100 µL of 0.1 M formic acid. After vortex-mixing, 0.5 mL of cold acetonitrile (ACN) was added to each tube. Samples were vortex-mixed again and then centrifuged at 1000 rpm for 3 min. The supernatant (100 µL) was combined with 300 µL of 2.5 mM ammonium acetate in HDPE vials. Approximately 10 % of the samples were extracted in duplicate.

Samples were analyzed using an Agilent 1100 series HPLC equipped with an Eclipse Plus C8 column (2.1 × 50 mm, 3.5 µm; Agilent) interfaced to an Agilent 6210 series Accurate-Mass MS-TOF system with negative electrospray ionization (ESI). The mobile phase system consisted of 0.4 mM ammonium formate in 95:5 deionized water:methanol (A) and 95:5 methanol:deionized water (B). Quantification of PFESA-BP2 was based on comparison of a single ion peak area in negative mode 462.9326 [M − H]- to the response of an external standard curve created by spiking variable levels of standard into control liver homogenate or serum. The standard used for quantification was provided by the manufacturer as an 1% aqueous solution. Analytical blanks (i.e. ACN and Pico-pure water) were analyzed with every run. When appropriate, isotopically labeled (^{13}C) PFOA purchased from Wellington Laboratories Inc. was used as the internal standard for quantification of the liver and serum concentrations.

2.6. Statistical evaluation

All variables were analyzed separately by sex with two-way main effects ANOVAs, which included factors for dose and block. This allowed testing for changes due to PFESA-BP2 treatment after adjusting for mean differences due to block. If the F-test for treatment effect was significant ($p < 0.05$), each treatment group was compared to vehicle controls with pairwise t-tests, using Dunnett's adjustment for multiple comparisons. The ANOVA assumptions of normality and homogeneity of variance were examined using the Shapiro-Wilk and Levene's tests. ALT, AST, GLDH were transformed to the log10 scale to satisfy these assumptions.

3. Results

3.1. Toxicity

Changes in animals' body weights, liver weights, and liver appearance are summarized in Table 1. No changes in the animals' appearance or overt behavior were observed during the dosing period. Significant increases in body weights during the dosing period occurred in the 6 mg/kg female animals. The relative and absolute liver weights increased significantly in the 3 and 6 mg/kg dose groups for the males and females. At the 6 mg/kg-day dose level, the liver weight was greater than the controls by two-fold. At necropsy, livers of 3 and 6 mg/kg-day mice were enlarged and pale, and the surfaces were reticulated (i.e. pattern of individual liver lobules made visible due to color change of hepatocytes). The control group contained no animals with

Table 1
Effects of PFESA-BP2 on average body weights, liver weights, and clinical serum chemistry in Balb-c mice after 7 days of treatment. The sample sizes for the data presented here are demonstrated in Table S1 for each dose group and variable.

	Males				
	0	0.04 mg/kg-day	0.4 mg/kg-day	3 mg/kg-day	6 mg/kg-day
Number of Mice Dosed	12	10	15	10	5
Body Weight (g)	22.9 ± 0.37	23.2 ± 0.55	23.6 ± 0.42	23.2 ± 0.47	23.9 ± 0.69
Liver Weight (g)	1.29 ± 0.03	1.33 ± 0.05	1.37 ± 0.04	2.02 ± 0.04 ***	2.79 ± 0.06 ***
Liver/Body Weight (%)	5.62 ± 0.08	5.75 ± 0.12	5.83 ± 0.09	8.70 ± 0.10 ***	11.7 ± 0.15 ***
Number of Mice with Visual Liver Reticulation	0	1	2	10	5
ALT (log 10 U/L)	1.81 ± 0.06	1.79 ± 0.09	1.72 ± 0.07	2.04 ± 0.08	2.34 ± 0.11 ***
AST (log 10 U/L)	2.08 ± 0.07	2.07 ± 0.10	1.94 ± 0.08	2.15 ± 0.09	2.24 ± 0.13
GLDH	1.15 ± 0.04	1.19 ± 0.06	1.05 ± 0.05	1.35 ± 0.05 *	1.71 ± 0.08 ***
BUN (mg/dl)	9.07 ± 0.35	9.11 ± 0.48	9.08 ± 0.44	9.09 ± 0.41	9.26 ± 0.62
Albumin (g/dl)	3.30 ± 0.08	3.32 ± 0.11	3.44 ± 0.09	3.56 ± 0.10	3.59 ± 0.14
Globulin (g/dl)	2.07 ± 0.05	2.09 ± 0.07	2.16 ± 0.06	2.25 ± 0.06	2.35 ± 0.09 *
Total Protein (g/dl)	5.37 ± 0.11	5.42 ± 0.17	5.59 ± 0.13	5.81 ± 0.14	5.93 ± 0.21
Glucose (mg/dl)	201 ± 9.52	194 ± 13.9	192 ± 10.8	200 ± 12.0	159 ± 17.57
Tbil (mg/dl)	0.41 ± 0.07	0.31 ± 0.10	0.30 ± 0.08	0.35 ± 0.09	0.30 ± 0.12
Triglycerides (mg/dl)	319 ± 32.6	325 ± 33.0	328 ± 29.5	341 ± 23.8	374 ± 31.9
Cholesterol (mg/dl)	128 ± 9.05	122 ± 9.75	129 ± 8.70	140 ± 7.00	122 ± 9.43
	Females				
	0	**0.04 mg/kg-day**	**0.4 mg/kg-day**	**3 mg/kg-day**	**6 mg/kg-day**
Number of Mice Dosed	12	10	15	10	6
Body Weight (g)	18.9 ± 0.23	18.7 ± 0.34	19.0 ± 0.27	19.5 ± 0.29	20.0 ± 0.43 *
Liver Weight (g)	0.98 ± 0.03	0.94 ± 0.04	0.96 ± 0.03	1.67 ± 0.03 ***	2.38 ± 0.05 ***
Liver/Body Weight (%)	5.20 ± 0.13	5.08 ± 0.19	5.09 ± 0.15	8.29 ± 0.16 ***	11.5 ± 0.24 ***
Number of Mice with Visual Liver Reticulation	0	3	1	7	5
ALT (log 10 U/L)	1.92 ± 0.08	1.85 ± 0.12	1.88 ± 0.09	2.01 ± 0.10	2.49 ± 0.15 **
AST (log 10 U/L)	2.26 ± 0.07	2.22 ± 0.10	2.28 ± 0.08	2.13 ± 0.08	2.54 ± 0.12
GLDH	1.40 ± 0.07	1.31 ± 0.10	1.23 ± 0.08	1.35 ± 0.08	1.75 ± 0.12 *
BUN (mg/dl)	8.31 ± 0.42	8.11 ± 0.53	8.30 ± 0.42	9.07 ± 0.46	8.92 ± 0.69
Albumin (g/dl)	3.33 ± 0.09	3.26 ± 0.14	3.35 ± 0.11	3.53 ± 0.12	3.43 ± 0.17
Globulin (g/dl)	1.79 ± 0.06	1.73 ± 0.09	1.84 ± 0.07	2.09 ± 0.08 *	2.08 ± 0.12
Total Protein (g/dl)	5.12 ± 0.15	4.98 ± 0.22	5.17 ± 0.18	5.62 ± 0.19	5.52 ± 0.28
Glucose (mg/dl)	224 ± 12.1	227 ± 17.7	213 ± 14.1	212 ± 15.2	235 ± 22.4
Tbil (mg/dl)	0.24 ± 0.06	0.32 ± 0.09	0.45 ± 0.07	0.31 ± 0.08	0.35 ± 0.11
Triglycerides (mg/dl)	191 ± 39.5	185 ± 33.8	200 ± 25.5	324 ± 25.0 *	280 ± 36.1
Cholesterol (mg/dl)	113 ± 23.8	114 ± 23.5	134 ± 18.2	99.6 ± 17.8	112 ± 25.7

The statistics for this table are based use F-test p-value from ANOVA; Averages demonstrated for each group with standard error.
* $p \leq 0.05$, ** $p \leq 0.001$, *** $p \leq 0.0001$ relative to control.

reticulated livers.

Samples from the control, 0.4 mg/kg-day and 3 mg/kg-day treatment groups were viewed microscopically to study the appearance of the cells. Livers from the 0.04 mg/kg-day and 6 mg/kg-day dose group were not analyzed with histopathology. Histopathology revealed hepatocyte hypertrophy predominantly in the centrilobular portion of the liver lobule (Rappaport Zone 3) for the 3 mg/kg-day dose group (Fig. 2). The hypertrophy extended to a lesser degree into Zone 2. Mitotic figures were another change observed in the 3 mg/kg-day livers and may indicate a response by the liver not seen in the control and 0.4 mg/kg-day mice. Intracytoplasmic vacuoles (spaces) were present in all treatment groups and are created during tissue processing which washes out lipid and glycogen accumulation within the cytoplasm. Vacuoles were recorded as fine to moderately large, sharp-edged, clear vacuoles consistent with lipid accumulation or as vacuoles with less distinct borders consistent with glycogen accumulation. The vacuoles consistent with glycogen accumulation did not vary between zones of the liver lobule or treatment group, whereas the vacuoles consistent with lipid accumulation were observed in Zones 2 and 3 and had slightly increased numbers in the 3 mg/kg-day livers compared to the control group. When hypertrophy is present, it is common to develop initially around the central vein and spread outward as seen in the 3 mg/kg-day mice. Larger group numbers would need to be evaluated to determine if cell death and intracytoplasmic vacuoles are significant in the higher dose.

Serum liver function markers indicative of hepatotoxicity were detected in both sexes within the 3 and 6 mg/kg-day treatment groups. Elevated ALT concentrations occurred in the 6 mg/kg/day treatment for both sexes and in the 3 mg/kg/day male dose group (Table 1). Increased GLDH was seen in both 3 and 6 mg/kg-day males and the 6 mg/kg-day females. Elevated serum protein levels occurred in males with significant increases in both globulin and total proteins at the 3 and 6 dose levels. For females, only the globulin levels were increased for both the 3 and 6 mg/kg-day dose levels.

3.2. PFESA-BP2 bioaccumulation

All analytical blanks were negative for PFESA-BP2. The coefficient of determination (R2) was greater than 0.98 for all standard curves. Average PFESA-BP2 serum concentrations ranged from 0.47 μg/mL in the 0.04 mg/kg-day dose group to 88 μg/mL in the 6 mg/kg-day dose group (Table 2). The average serum concentration at the lowest dose level was between 100 and 200-fold higher than the average PFESA-BP2 concentrations reported in serum from the residents of Wilmington, NC (Katlorz, 2018). It is notable that bioaccumulation did occur with the presence of two internal ether oxygens, suggesting molecular length (and mass) increase retention in biological systems. The average PFESA-BP2 liver concentrations ranged from 1.4 μg/g in the 0.04 mg/kg-day female mice to 240 μg/g in the 6 mg/kg-day male mice (Table 2). The concentrations of PFESA-BP2 in the serum and liver are in the range of previously reported mouse serum PFOA/PFOS concentrations. (Lau et al., 2006; Thibodeaux et al., 2003; Wolf et al., 2008; Guo et al., 2019) For example, samples collected from WT mice dosed with PFOA at 3 mg/kg-day for seven days demonstrated average

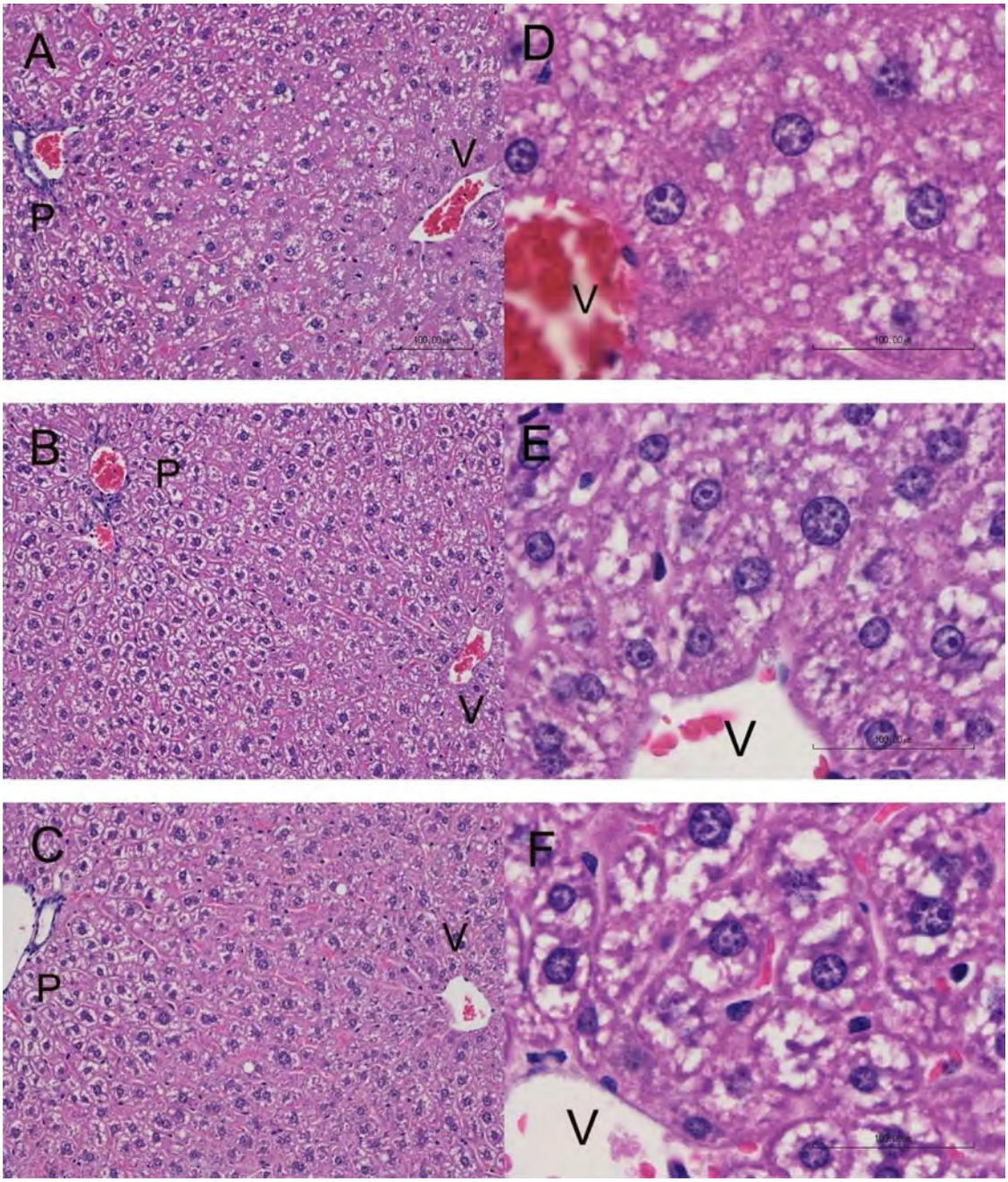

Fig. 2. Liver histopathology for Balb-c mice receiving PFESA-BP2 at 3 mg/kg-day (A, D), 0.4 mg/kg-day (B, E), or vehicle (C, F). Livers from the 3 mg/kg-day dose group demonstrated increased cytoplasmic volume and density of cytoplasmic contents of centrilobular hepatocytes surrounding the central vein (V) compared with hepatocytes closer to the portal region (P), a change which was not observed in liver from the lower concentration of PFESA-BP2 or vehicle mice. Slides A, B, and C are at 100x magnification; slides D, E, and F are at 400x magnification.

serum concentrations of ~33.3 μg/mL. (Wolf et al., 2008) This value is slightly lower than the 3 mg/kg-day serum concentrations reported here (~48 μg/mL), but it is unclear if the lower values are attributed to compound differences or the strain of mouse treated for the experiment.

The percent of PFESA-BP2 in serum relative to the amount administered, ranging from 9 to 13 %, was similar in male and female mice and did not demonstrate a direct relationship with dose (Table 2). The percent accumulation in the liver of the mice, ranging from 30 to 65 %, varied by sex (higher in the males) and correlated positively with increasing dose level (Table 2). Higher accumulations in the liver compared to serum could have implications for the human population in cases where PFESA-BP2 was identified in serum. (Katlorz, 2018)

PFESA-BP2 was detected at low levels (< 0.3 μg/g) in two of the control livers analyzed. The contamination is assumed to be due to reuse of necropsy instruments across animals because it was present in only two of the livers and was not present in the serum for these animals, and dosing protocol would not allow occurrence of cross-contamination with dosing instruments. Since the serum levels are an order of magnitude lower than that in the livers of mice treated with PFESA-BP2, this contamination is not expected to affect the toxicity and bioaccumulation results.

4. Discussion

The results presented here demonstrate that short term (7 day) exposures to PFESA-BP2 significantly increased liver weights in treated

Table 2
Serum and liver PFESA-BP2 concentrations relative to total dose administered.

	Male			
	0.04 mg/kg-day	**0.4 mg/kg-day**	**3 mg/kg-day**	**6 mg/kg-day**
Dosing Solution Concentration (g/L)	4.0	36.8	344	716
Total Administered (μg)	5.6	51.52	481.6	1002.4
Serum Concentration (μg/mL)	0.51 ± 0.07[b]	3.99 ± 0.28	47.0 ± 3.45	83.9 ± 17.1
Liver Concentration (μg/g)	2.41 ± 0.38	20.1 ± 3.23	143 ± 31.2	235 ± 30.9
Serum Accumulation (μg)[a]	0.69	5.51	63.8	117
Liver Accumulation (μg)	3.21	27.5	289	656
% Serum Accumulation	12 %	11 %	13 %	12 %
% Liver Accumulation	57 %	53 %	60 %	65 %
	Female			
	0.04 mg/kg-day	**0.4 mg/kg-day**	**3 mg/kg-day**	**6 mg/kg-day**
Dosing Solution Concentration (g/L)	3.15	30.1	313	624
Total Administered (μg)	4.41	42.14	438.2	873.6
Serum Concentration (μg/mL)	0.44 ± 0.17[b]	3.55 ± 0.98	48.0 ± 14.4	92.9 ± 83.9
Liver Concentration (μg/g)	1.43 ± 0.12	17.5 ± 4.37	129 ± 41.4	208 ± 24.2
Serum Accumulation (μg)[a]	0.48	3.95	54.8	109
Liver Accumulation (μg)	1.34	16.8	215	495
% Serum Accumulation	11 %	9%	12 %	12 %
% Liver Accumulation	30 %	40 %	49 %	57 %

[a] Serum volumes estimated assuming serum accounts for 5.85 % of the total body weight.
[b] Group means with one standard deviation.

mice following doses of 3 and 6 mg/kg-day and created a greater than two-fold increase in liver weight of both male and female mice at the 3 and 6 mg/kg-day. Previous rodent PFAS studies have demonstrated hypertrophy due to peroxisome proliferation. (Wolf et al., 2008; Chappell et al., 2020; Cui et al., 2009; Adinehzadeh et al., 1999; Blake et al., 2020), We propose that the hypertrophy seen with this sulfonated PFAS, similar in mass and length to PFOS, would likely act by similar mechanisms. Elevated serum liver function tests indicate that injury occurs at PFESA-BP2 doses ≥ 3 mg/kg-day in both sexes with males apparently more sensitive than the females. There were no adverse effects detected at the 0.04 and 0.4 mg/kg-day doses compared to the control group. At the lowest dose (0.04 mg/kg-day - ~500 ppb), serum levels were 100- to 200-fold higher than median serum concentration from humans exposed to PFESA-BP2 through drinking water (~3 ppb (Katlorz, 2018)).

5. Conclusions

To our knowledge this is the first toxicology study of PFESA-BP2. Given that this chemical induces hepatic effects comparable to those associated with other PFASs, additional toxicology studies are warranted. A mechanistic study using liver tissue collected in this study is currently in progress. Genomic analysis and more histopathological evaluations can also be explored with tissues collected in this study. Future work should include extended in vivo treatments to simulate a chronic environmental exposure covering different developmental life stages.

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Declaration of Competing Interest

The authors declare that there are no conflicts of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.tox.2020.152529.

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From:	Emily Donovan
To:	comments.chemours; Holman, Sheila; Regan, Michael S
Subject:	[External] Chemours Public Comments -- Addendum to Consent Order re Paragraph 12
Date:	Thursday, September 17, 2020 1:41:31 PM
Attachments:	CCF Letter DEQ Addendum to Chemours Consent Order.pdf

Dear Ms. Holman and Sec. Regan,

I hope this email finds you both well. Clean Cape Fear offers the attached written comments regarding the proposed Addendum to the Chemours Consent Order. Our comments include a petition signed by over 1,000 people who live or vacation downstream of the Chemours Fayetteville Works Site.

If you have any trouble accessing the attached document, please contact me.

With gratitude,

Emily Donovan
Co-Founder
Clean Cape Fear
FB/Twitter: @CleanCapeFear
www.cleancapefear.org
704.491.6635 | cell

"Above all, maintain constant love for one another, for love covers a multitude of sins." 1 Peter 4:8

CLEAN CAPE FEAR

Sheila Holman
Assistant Secretary's Office
1601 Mail Service Center
Raleigh, NC 27699-1601
Via email: comments.chemours@ncdenr.gov

September 17, 2020

RE: Chemours Public Comments

Ms. Holman:

Clean Cape Fear is an alliance of established advocacy groups, community leaders, educators, and professionals working together to restore and protect our water quality, as well as spotlight deficiencies in governmental regulations that adversely impact our right to clean water. I am writing to submit comments on the Addendum to Chemours Consent Order Paragraph 12. We appreciate the opportunity to submit comments on this important aspect of the Consent Order, which seeks to address PFAS loading from the Fayetteville Works Facility (Facility) via groundwater, stormwater, and on-site streams.

We are supportive of this proposed Addendum and the comments submitted by Natural Resources Defense Council (NRDC), our comments are not intended to delay or prevent the progress being made to stop PFAS loading from the Facility via groundwater, stormwater, and on-site streams. Measures taken to stop the continued releases of PFAS into the Cape Fear River at the source are very important to all downstream residents.

Sadly, we feel this Addendum has omitted addressing an inequity established by the Chemours Consent Order which continues to leave residents downstream of the Facility who rely on the Cape Fear River as their primary source of drinking water without immediate relief from continued chronic exposures to DuPont/Chemours' PFAS chemical waste. We strongly encourage you to create a PFAS Community Relief Fund, paid for by Chemours, for impacted residents to immediately access vouchers to cover the cost to purchase and install under-sink reverse osmosis filtration units, as well as yearly filter replacements until all permanent solutions at the Facility are fully operational and adequate independent testing confirms their success.

Please see the attached petition signed by over 1,000 people who live or vacation in our impacted communities requesting you immediately create a PFAS Community Relief Fund for Victims of Chemours/DuPont. Thank you for this opportunity to comment and please feel free to reach out to me if you have any questions or concerns.

With gratitude,

Emily Donovan, co-founder
Clean Cape Fear

Attachment: Petition with Signatures

North Carolina Department of Environmental Quality,

1101 people have signed a petition on Action Network telling you to Create a PFAS Community Relief Fund for Victims of Chemours/DuPont.

Here is the petition they signed:

Dear Secretary Regan and Assistant Secretary Holman:

Downstream residents have been overexposed to DuPont/Chemours' PFAS chemical waste for decades.

We believe DuPont/Chemours used our drinking water supply to cut costs and increase their profits—these actions placed nearly 300,000 residents downstream from their Fayetteville facility in harm's way transferring external costs onto innocent and unassuming North Carolinians.

These PFAS chemicals are associated with multiple serious health effects including suppression of the immune system, developmental disorders, thyroid disease, and cancer.

The financial burden of these chemicals often goes undiscussed. A single cancer treatment can cost a patient nearly $1 million. A study by the Nordic Council found that inaction on these forever chemicals will lead to billions of euros in healthcare costs annually--meaning just the healthcare costs alone of doing nothing will far exceed the cost of taking preventative measures now. A similar analysis has not been performed in the United States.

We believe DuPont/Chemours knew their PFAS chemical waste could cause harm and willfully chose to not invest in preventive manufacturing precautions.

Chemours is a Fortune 500 company with over $5.5 billion in annual sales. In 2019, they returned $486 million to their shareholders through stock buybacks and dividends. Ultimately, they continue to increase shareholder value at the expense of North Carolinians.

In 2019, Chemours legally established a threshold for providing immediate relief to well owners who had 70 ppt for total PFAS, or 10 ppt per individual PFAS, in their drinking water. This "70/10 threshold" for immediate relief was only extended to well owners in Bladen and Cumberland counties and, sadly, excluded the 300,000 impacted residents downstream who have been suffering from similar, if not greater, drinking water contamination on a regular basis. We believe Chemours knew the true extent of their historical contamination into our river but failed to fully disclose this information at the time the consent order was signed.

On average, downstream residents drank over 70 ppt of total PFAS and 10 ppt of Schedule C PFAS for the majority of 2019 and we continue to drink DuPont/Chemours' PFAS chemical waste for most of 2020. This has been well documented by regular testing of tap water provided by Cape Fear Public Utilities and Brunswick County Public Utilities.

Because DuPont/Chemours historically never provided test standards to accurately quantify how much PFAS impacted communities were being exposed to, the true nature of our contamination crisis keeps rising as new test standards are produced.

Per the 2019 consent order, Chemours was legally required to identify all PFAS generated from their manufacturing processes and general operations.

A non-targeted analysis from Chemours, released in June 2020, revealed we were likely exposed to an additional 271 “unknown” PFAS chemicals. Based on our understanding, 250 of those forever chemicals are currently being diverted into tanker trucks and driven to Deer Park, TX for deep well injection until NC DEQ approves Chemours’ new discharge permit.

The remaining 21 PFAS chemicals are likely still leaking into the Cape Fear River--per the non-targeted analysis report. It is reasonable to assume we are currently being exposed to these additional 21 “unknown” PFAS chemicals on top of the already high levels of PFAS our utilities continue to report in their regular samples.

Brunswick, New Hanover and Pender County impacted residents are still drinking some of the highest levels of PFAS in tap water per a recent nationwide tap water study from the Environmental Working Group. Again, these levels do not include the newly disclosed "unknown" PFAS.

A recent news article published in Discover magazine_ is sounding the alarm regarding endocrine disrupting chemicals, like PFAS, increasing our risk of having more severe Covid-19. This troubling news should create an increased sense of urgency for NC DEQ to act on our behalf.

NC DEQ is currently receiving public comments regarding amending paragraph 12 of the consent order which addresses remediation measures for continued PFAS releases into our river.

This amendment is a good step forward, however, it does not address providing immediate relief to the 300,000 impacted residents still chronically exposed to untold amounts of DuPont/Chemours’ toxic PFAS chemical waste by simply using on our own faucets.

The amended consent order seeks to provide permanent pollution control measures by spring of 2023—2.5 years from now.

It's our understanding, interim pollution measures will not be available for at least eight more months and none of these remediation efforts address sediment contamination all along the river which would likely still provide background levels of PFAS into the drinking water of impacted communities already harmed by decades of overexposure.

Brunswick County Public Utilities and Cape Fear Public Utility Authority are both working to upgrade their treatment processes to address this environmental crime. These upgrades are costing ratepayers millions of dollars and will not be fully operational for another 2-3 years.

This inequity and harm must stop now.

We are demanding that NC DEQ require Chemours to immediately establish a PFAS Community Relief Fund specifically for impacted communities who rely on the Cape Fear River as their primary source of drinking water.

Chemours must pay into this revolving fund to cover the cost of vouchers for downstream impacted residents who seek to install under sink reverse osmosis filters. These vouchers should cover the cost of filtration purchase and installation, as well as, yearly filter

replacements until all permanent solutions at the Chemours Fayetteville facility are fully operational and adequate testing confirms their success.

In light of the discovery of the additional 271 unknown PFAS chemicals, NC DEQ should not approve Chemours' process wastewater discharge permit application until:

1. A PFAS Community Relief Fund is established.

2. Chemours creates test standards to enable independent scientists to accurately quantify the levels of all these unknown PFAS..

3. The identity of every new compound is known.

4. Chemours can accurately quantify the levels of these unknown PFAS.

Thank you for your time and attention to this important topic.

You can view each petition signer and the comments they left you below.

Thank you,

Clean Cape Fear

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1. An anonymous signer (*ZIP code:)*

2. Kim Blanchard (*ZIP code: 28412)*

3. Lisa Ferguson (*ZIP code: 28403)*
Lisa Ferguson

4. Allison Lockshier (*ZIP code: 28412)*

5. Sandra Ford (*ZIP code: 28451)*
So tired of this BS. Why is Chemours still in business in NC? Way past time to shut them down, have them pay to clean up their environmental mess.

6. Amelia Monroe (*ZIP code: 28451)*

7. aaron charles (*ZIP code: 28405-2735)*

8. Arthur Bell (*ZIP code: 28348)*

9. Antje Burke (*ZIP code: 18411)*

10. Adele Godino (*ZIP code: 28401)*
Please stop polluting our waters do you not want to leave your children healthy planet!!? Adele Godino

11. Amy Long (*ZIP code: 28451)*

12. ann glossl (*ZIP code: 28451)*

13. Aimee Cook (*ZIP code: 28479)*

14. Laurene Allen (*ZIP code: 03054)*
The cost to communities struggling to stop their exposure to industrial chemicals that do not belong in our water, environment or bodies is immense. Victims should not have to pay for what they did not cause.

15. Aleeze Arthur (*ZIP code: 28411)*

16. Alexis Luckey (*ZIP code: 27701)*
Chemours must be held responsible for polluting our state and providing redress to communities harmed by corporate irresponsibility.

17. Alison Born (*ZIP code: 28403)*

18. Allie Sheffield (*ZIP code: 28445)*

19. Amanda Mayfield (*ZIP code: 28348)*

20. Amanda Bishop (*ZIP code: 28451)*
CLEAN WATER!!!

21. Ashli Gibson (*ZIP code: 28403-2611)*

22. Matt Amrhein (*ZIP code: 28468)*

23. Amanda Fontana (*ZIP code: 28479)*

24. Amy Shands (*ZIP code: 28479)*
Both my dog and myself have had a rare form of cancer. I have a teenager and it makes me sick to think about her become sick just from drinking the water I have provided her for her health. This is ridiculous!

25. Amy Herring (*ZIP code: 28451)*

26. Amy Jones (*ZIP code: 19422)*

27. Amy Stermer (*ZIP code: 29479)*

28. Andrea Carson (*ZIP code: 28411)*
This is outrageous!

29. Angelika Lacer (*ZIP code: 28405)*

30. Angie Fanning (*ZIP code: 28445)*

31. Cissie Brooks (*ZIP code: 28403)*

32. Ann Chatfield (*ZIP code: 28451)*

33. Ann Stephani (*ZIP code: 28493)*

34. Ann Hillman (*ZIP code: 55406)*

35. Alexandra Craig (*ZIP code: 28403)*

36. Stephen Abarno (*ZIP code: 28403)*
Time for politicians to STOP taking money from chemical companies !! Take care of the people you represent, and NOT Yourselfs!! Disgusting .....

37. Arka Shanks (*ZIP code: 28412)*

38. Ericka Hallis (*ZIP code: 28348)*

39. Edward Stellin (*ZIP code: 28451)*

40. Amy Sass (*ZIP code: 28451)*

41. Ashley Winters (*ZIP code: 28315)*

42. Ashley Daniels (*ZIP code: 28412)*
I'm grateful for this initiative. Polluters need to pay!

43. Anthony Snider (*ZIP code: 28403)*

Our bodies are not DuPont's sewer! We have a RIGHT to clean drinking water! It's a fundamental human right. Make them stop dumping in our mouths!

44. Audrey Dunn (*ZIP code: 28403)*

45. Audrey Wright (*ZIP code: 28451)*

46. Audrey Marshall (*ZIP code: 28451)*
I can not fathom that Chemours has gotten away with poisoning us for decades!

47. Deena Delfosse (*ZIP code: 28451)*

48. Arlene Holmes (*ZIP code: 28451)*

49. Melanie Fazio (*ZIP code: 28451)*

50. Rachel Baldwin (*ZIP code: 28401)*

51. Nancy Sdeo (*ZIP code: 28451)*

52. Barb Brostrom (*ZIP code: 28451)*

53. Barry Barsamian (*ZIP code: 28451)*

54. BARBARA SFRAGA (*ZIP code: 28468)*
What DuPont/Chemours has been inflicting on consumers in the Cape Fear region is nothing short of criminal. And to have no there recourse than to pay for tainted water for decades is a bureaucratic nightmare. I am a 2 time cancer survivor in my 60s. Chemours/DuPont needs to get it together NOW and clean up their act or board up and close down! The NC DEQ needs to establish a PFAS Community Relief Fund TO BE PAID FOR BY CHEMOURS/DUPONT, to provide impacted residents with immediate relief from these continued toxic exposures. This is a travesty. And it needs to be reversed NOW!

55. Barbara Smeltzer (*ZIP code: 28479)*

56. David Bristol (*ZIP code: 28409)*

57. Barry Laub (*ZIP code: 28451)*

58. brad creacey (*ZIP code: 37115)*

59. Carleton Waugh (*ZIP code: 28451)*

60. Bill Hodge (*ZIP code: 28451)*

Stop dumping chemicals!

61. Leslie B Sternstein (*ZIP code: 28451)*
I was recently diagnosed with three thyroid nodules. I now must go every six months for biopsies. We drank & cooked with this water & still bathe with it. My health has changed drastically in the past year with no apparent causes. We deserve cleAn, safe water! The polluters need to provide clean water to people and clean up the mess they made. Make THEM come have Their kids drink it daily! When we pay taxes we have every right to expect clean, safe water. Give that to us - Now.

62. Florence Solomine (*ZIP code: 28451)*
This must be addressed by these polluters . They should close down.

63. Brian Beauregard (*ZIP code: 28469)*
IT IS ABHORRENT THAT THESE CORPORATIONS ARE NOT HELD FISCALLY ACCOUNTABLE! THEY, NOT US, SHOULD BE FURNISHING US DRINKING WATER! WHERE IS THE JUSTICE????

64. Becky Workiewicz (*ZIP code: 28479)*

65. Rebecca Wilson (*ZIP code: 27704)*

66. BARBARA BEAUREGARD (*ZIP code: 28469)*

67. Ben Reischer (*ZIP code: 28451)*

68. Beth McDonnell (*ZIP code: 28411)*

69. Beth Bell (*ZIP code: 28451)*

70. Mary Hunt (*ZIP code: 28409)*
I buy bottled water and use it for drinking, cooking etc. With my limited income that is an expense I can ill afford but feel I cannot do without. These chemicals are dangerous.

71. Elizabeth Wroblewski (*ZIP code: 28451)*

72. Betsy Ulman (*ZIP code: 28412)*

73. Elizabeth Fryman (*ZIP code: 28412)*

74. Beverley McGuire (*ZIP code: 28409)*

75. Bianca Glinskas (*ZIP code: 28401)*

76. Elizabeth Gruber (*ZIP code: 28451)*

77. Heidi Rehder (*ZIP code: 28403)*

78. priscilla rebillard (*ZIP code: 28412)*
Dupont/Chemours must pay for the harm they have caused to our communities!

79. Anna Bandlyke (*ZIP code: 38465)*

80. Barbara Pinto (*ZIP code: 28451)*

81. William Johnson (*ZIP code: 28318)*

82. Barbara Rainis (*ZIP code: 28451.)*

83. Beth Walters (*ZIP code: 28405)*
Enough is enough. Shut Chemours down now! They have been proven to be neglectful and untrustworthy when it comes to the lives of those in SE NC.

84. Barbara Melon (*ZIP code: 28451)*

85. Robert Friedman (*ZIP code: 28451)*

86. Bobbi Keller (*ZIP code: 28451)*

87. Uyen Nguyen (*ZIP code: 28462)*

88. Leonard Kiausas (*ZIP code: 28451)*

89. Leonard Kiausas (*ZIP code: 28451)*

90. Betsy Wood (*ZIP code: 28412)*
Yes its time to Pay up! We have been spending HUNDREDS of dollars on clean drinking water and have to budget this into our monthly social security. We are down river in New Hanover County and are suffering from Chemours chemical wastes. We need relief NOW .

91. Debra Corbett (*ZIP code: 28451)*

92. Bruce Piggot (*ZIP code: 28468)*
Corporate pollution is a crime against human & all life. You would think that even those getting rich while polluting would consider the harm to their children but greed is a disease and corporations are not people and to hold them accountable Citizens United (Corporations United in Colluding) must be overturned as they are the Fox in the henhouse & the criminal polluters and charlatans that have becime immune to effective prosecution. Vote for those running to fix our corrupt broken political and economic system and Vote out those colluding against we the people. But VOTE! VOTE! PLEASE VOTE FOR HONEST CHANGE FOR A BETTER FUTURE!

93. D Wagner (*ZIP code: 28443)*
DuPont and Chemours knowingly dumped poison in our waters! They need to be shut down!

94. Lisa Brewster (*ZIP code: 28409)*
Thank you for initiating this petition. I have been resentful that I have to purchase drinking water and continue to pay CFPUA!

95. Brian Fields (*ZIP code: 28303)*

96. Bruan Mortensen (*ZIP code: 28457)*

97. Briana Rainford (*ZIP code: 28451)*

98. Barbara Martin (*ZIP code: 28451)*

99. Bridget Tarrant' (*ZIP code: 28401)*
It is unacceptable that Chemours has not been held responsible to pay for safe drinking water for those in SE North Carolina! Please do the right thing by making them pay for the harm they have caused

100. Brittany Bernardini (*ZIP code: 28451)*

101. Brittany Mowery (*ZIP code: 28461)*

102. Darby Stephens (*ZIP code: 28451)*

103. Glenda Browning (*ZIP code: 28469)*

104. Bruce Holsten (*ZIP code: 28409)*

105. Athena Bryson (*ZIP code: 28409)*

106. Laurie Lindsay (*ZIP code: 28451)*
Approximately 21 of these additional "unknown" PFAS chemicals may be actively releasing into our river and contaminating our tap water--this is on top of the PFAS currently being reported by local utilities. Impacted communities deserve immediate relief.

We have a moral obligation to protect all impacted community members--not just those who can afford personal home filtration systems. Everyone should have access to the relief they need from these toxic forever chemicals--ASAP.

107. Brittney Sanchez (*ZIP code: 28401)*
It's ridiculous how little has been done for residents of the Cape Fear region and other parts of our state (and country)! Corporations must be held accountable. Chemours gets until 2025 to "outline steps" for PFAS Reductions? That's not good enough for me and it shouldn't be for you. Own up. Help

our community. Clean up the Cape Fear River!

108. Bud Abramowitz (*ZIP code: 28451)*

109. Katrina Kuehn (*ZIP code: 28451)*

110. Holly Burch (*ZIP code: 28411)*

111. Jennifer Burns (*ZIP code: 27612)*
I am absolutely livid to hear that additional damage is still being done by the Chemours/DuPont situation and that such minimalistic approaches have been used. How many more times do citizens and their voted representatives need to be conned before it is realized that for every grievance that is discovered, there are 10 more being hidden in the middle of every scandal.
Chemours needs to pay for reverse osmosis filtering all the way down through ALL the districts being impacted by their immoral, unethical business practices. Additionally, not only they, but every company producing synthetic chemicals need to pay for every public utility to enhance their levels of water decontamination, using reverse osmosis WITH remineralization - without increasing taxpayer and utility fees. This is a no-brainer. There is no citizen in any tax bracket or any demographic that should be continue to be exposed to this mass poisoning. There is no longer any excuse for ignorance. Modern technology that finally shows the long term damage that has been created, and that continues to be be created, is not going away. Our public protection agencies need to look these monstrous situations in the face and deal with them now, not later. They have the means to financially correct their problems, and they should be held accountable.

112. Wayne Manzi (*ZIP code: 28451)*

113. Brian Woolgar (*ZIP code: 28451)*

114. Debra Degalis (*ZIP code: 28428)*

115. Lawrence Cahoon (*ZIP code: 28403-1919)*

116. Ann Carbone (*ZIP code: 28451)*

117. Carla Lewin (*ZIP code: 28412)*

118. Carol Szatko (*ZIP code: 28468)*

119. Carolyn Ferrell (*ZIP code: 27517-4915)*

120. Carolyn Lenzen (*ZIP code: 28465)*

121. Carrie Riccardi (*ZIP code: 28443)*

122. Carrie Stewart (*ZIP code: 28451)*

123. Diane Carrigan (*ZIP code: 28451)*

124. Eunice Rowe (*ZIP code: 28467)*

125. Catherine Beaman (*ZIP code: 28409)*

126. Cathleen Anton (*ZIP code: 28451)*

127. Cathy Norton (*ZIP code: 28422)*

128. Catherine Tierney (*ZIP code: 28451)*

129. Carole Surridge (*ZIP code: 28461)*
Chemours has a moral responsibility to provide relief to residents impacted by the release of PFAS chemical waste into the Cape Fear River.

130. Cathie Carpenter (*ZIP code: 28461)*

131. Crystal Clark (*ZIP code: 28479)*

132. Catherine Collins (*ZIP code: 28348)*

133. Carol Simmons (*ZIP code: 28312)*
we need help now. Filters on wells would help, but need them now. Reverse Osmossis on the wells not defacing our homes.

134. Chris Kramer (*ZIP code: 28409)*

135. Cheryl Stanbury (*ZIP code: 28409)*

136. Catherine Docous (*ZIP code: 28403)*
I believe Chemours/Dupont should reimburse residents affected for hone filtration! I have spent much to provide safe water! This should never have happened.

137. Charles Dunmire (*ZIP code: 28451)*
Please hear us !

138. Cheryl Crossman (*ZIP code: 28451)*

139. Courtney Justus (*ZIP code: 28411)*

140. Celeste Zellin (*ZIP code: 28451)*
Poisons in our water is not only dangerous and disgusting but immoral. Those that dump them are assaulting and murdering us and should be so charged.

141. Catherine Parello (*ZIP code: 28451)*

142. Caitlin Sims (*ZIP code: 24060)*

143. Cheryl Friedman (*ZIP code: 28451)*

144. Susan Beavis (*ZIP code: 28451)*

145. Gail Cole (*ZIP code: 28479)*

146. Chamisa Wheeler (*ZIP code: 28403)*

147. Suzanne Taylor (*ZIP code: 28412)*
Please make this a top priority for residents of Wilmington. It really is an urgent situation.

148. Charlotte Webb (*ZIP code: 28403)*

149. Charles Owens (*ZIP code: 28451)*

150. Karen Bearden (*ZIP code: 27612)*

151. Chris McKinley, MPAS, PA-C (*ZIP code: 28451)*

152. Christi Golder (*ZIP code: 28411)*

153. Christina Norvell (*ZIP code: 28409)*

154. Christina Parks (*ZIP code: 28401)*
We need RO treatment for the entire area and studies into the lasting impacts our community faces from being poisoned for decades.

155. Krystal Sacik (*ZIP code: 28479)*

156. Christine Valaika (*ZIP code: 28451)*

157. Claire Reveille (*ZIP code: 28451)*

158. Claire Alley (*ZIP code: 28403)*

159. Clarice Reber (*ZIP code: 28411)*

160. Christina Roth (*ZIP code: 28403)*

161. Carol Whitham (*ZIP code: 28451)*

162. Christopher Bailey (*ZIP code: 28411)*

163. Christina Clay (*ZIP code: 28405)*
I am requesting NC DEQ establish a PFAS Community Relief Fund paid for by Chemours.

164. Cathy McAfee (*ZIP code: 28451)*

165. Carolee Morris (*ZIP code: 28461)*
Unconscionable that anyone else should bear financial burdens for this contamination....of long standing.

166. Christine Zimmermann (*ZIP code: 28451)*
The health of citizens should be prioritized over the profits of business. Companies should be better regulated to ensure that they don’t poison our water, but if they do so, they need to pay mightily to clean it. They should also be very heavily fined as a deterrent.

167. Angela Calabrese (*ZIP code: 28462-2111)*

168. shawn Mullins (*ZIP code: 28428)*

169. Connie and Greg Stiger (*ZIP code: 28451)*

170. Conrad White (*ZIP code: 28451)*

171. Stephen Conroy (*ZIP code: 28451)*
I'm so disappointed in Chemours's lack of responsibility and the actions of our local and state legislators regarding these issues.

172. Anthony DiCroce (*ZIP code: 28451)*
This continued pollution is not except able. It’s unsafe and puts many residents effected by this pollution at a severe Heath risk. It’s time that those responsible are held accountable.

173. Timothy Jacob (*ZIP code: 28451)*

174. Charles Nolan (*ZIP code: 28451)*

175. Carla Bailey (*ZIP code: 28411)*

176. Rachel Benge (*ZIP code: 28412)*
Chemours/Dupont need to be held responsible for releasing chemicals into our water.

177. Ann Mateya (*ZIP code: 28451)*

178. DONNA CRONIN (*ZIP code: 28451)*

179. Claudia Crook (*ZIP code: 28401)*
Clean drinking water is a fundamental right, and public utility; while I can afford additional filters, many cannot, and that should not determine their long term health and exposure to PFAS.

180. Crystal Young (*ZIP code: NC)*

181. Carol Grosbier (*ZIP code: 28479)*

182. Elizabeth Broyles (*ZIP code: 28054)*

183. Connor Bennett (*ZIP code: 28411)*
The public should not have to pay for the pollution of a private company!

184. Coley Pritchett (*ZIP code: 28409)*

185. Cornelia Maxted (*ZIP code: 28451-6028)*
We should ALL have clean water. Since it's now polluted with PFAS, a Community Relief Fund should be established.

186. Colleen Erin (*ZIP code: 60442)*

187. Courtney Younghans (*ZIP code: 28479)*

188. Donna Laflamme (*ZIP code: 28451)*

189. Delphine Fernandez (*ZIP code: 28451)*
Delphine Fernandez

190. Dale Todd (*ZIP code: 28451)*

191. Danielle Eriksson (*ZIP code: 28405)*
MAKE THE POLLUTER PAY!

192. Danielle Dillard (*ZIP code: 28479)*

193. Danielle Richardet (*ZIP code: 28411)*

194. Danny Morrow (*ZIP code: 28405)*

195. Daphene Morris (*ZIP code: 28451)*

196. Debra Potter (*ZIP code: 28409)*

197. Darrell Collins (*ZIP code: 28348)*
Wish I could sell my house but can’t with gen-x in my well!

198. David Gallagher (*ZIP code: 28451)*
It is well past due that the residents of southeastern North Carolina stop being overexposed to Chemours’ PFAS chemical waste. In fact an additional 250+ "unknown" PFAS chemicals not previously disclosed by Chemours, approximately 21 of these additional "unknown" PFAS chemicals may be actively releasing into our river and contaminating our tap water. When will NC-DEQ make this their #1 priority and take immediate action to protect the citizens of North Carolina. PLEASE HELP US!!

199. Patti Ashley (*ZIP code: 28451)*

200. David Smith (*ZIP code: 28411)*
Pay victims!

201. Donna Bennett (*ZIP code: 28451)*

202. Don Bushman (*ZIP code: 28409)*

203. Daniel Weinfeld (*ZIP code: 28451)*

204. Denise Chadurjian (*ZIP code: 28401)*

205. Chris Ferguson (*ZIP code: 28451)*

206. Debra Willis (*ZIP code: 28451)*

207. Deanna Dunshee (*ZIP code: 28401)*

208. Dean Stewart (*ZIP code: 28451)*
The bare minimum that can be done is not charging us for our own demise

209. deb burgess (*ZIP code: 01545)*
Please join my name to you list if complaints to DuPont for their part in contaminating my drinking water, my cooking water and my cleaning water. I feel unsafe using any water from the tap. I think this is appalling in 2020 we can’t get clean drinking water from a faucet in my own home.

210. Deborah Todd (*ZIP code: 28451)*

211. Debbie Waitley (*ZIP code: 28312)*

212. Debra Fontana (*ZIP code: 28409)*

213. Deborah Sottile (*ZIP code: 28451)*

214. Dolores Saulter (*ZIP code: 28451)*

215. Desiree Fuller (*ZIP code: 28451)*

216. Doug Esleeck (*ZIP code: 28409)*

217. Devon fuller (*ZIP code: 28451)*

218. Nancy Dieffenbach (*ZIP code: 28409)*
Close Chemours. It's poisoning our community and all those up the Cape Fear River!!!

219. Gregory Amrhein (*ZIP code: 28467)*

220. Dorothy Cole (*ZIP code: 28451)*

221. Derek Hartman (*ZIP code: 28479)*

222. Helen Crenshaw (*ZIP code: 28451)*
Support this petition

223. Shannon Wright (*ZIP code: 28411)*

224. Diane Upton (*ZIP code: 28429)*

225. Diane Rezek (*ZIP code: 28405)*

226. Richard Hubbard (*ZIP code: 28451)*

227. Diane Cotter (*ZIP code: 03461)*

228. Art Dietrich (*ZIP code: 28451)*
How can the state allow Chemours to continue to operate and poison our water supply?

229. Lori DiFonzo (*ZIP code: 28451)*

230. Gina Andrews (*ZIP code: 28212)*

231. Della Kirkland (*ZIP code: 28479)*

232. Debra Mescal (*ZIP code: 07758)*
DuPont needs to pay and laws need to be changed!

233. Donna Ruby (*ZIP code: 28451)*
This has been happening for far too long!

234. Denea Labajetta (*ZIP code: 27707)*
Shame on the huge corporations, that just want to make a $ and not care about the well-being of our future. We ingest enough poison on a day to day basis, with pesticides, air pollution, car exhaust and our waters even more polluted. We can't exist without our pollinators and animals and trees, stop the insanity!!!!!!!!

235. Deborah Lee (*ZIP code: 28412)*

236. Donna Maher (*ZIP code: 28451)*
Why is clean water being prolonged for years? It has already been poisoning folks long enough. Make Chemours pay now and close them down. Stop worrying about the almighty dollar!

237. Donna Maher (*ZIP code: 28451)*
Totally unacceptable! The US is not a 3rd world country, but we are being treated like one.

238. Donald Florence (*ZIP code: 28461)*

239. Donna Saraga (*ZIP code: 28451)*

240. donna madonna (*ZIP code: 28405)*

241. Donna Maher (*ZIP code: 28451)*
This is criminal. Make them pay for our water until until it’s ? satisfactory and Shut them down! They are a repeat offender

242. Donna Walters (*ZIP code: 28451)*

243. Doris Sharp (*ZIP code: 28405)*

244. Doug Omeara (*ZIP code: 28451)*

245. Darlene Parlett (*ZIP code: 28411)*
I am tired of paying for drinking water every month. I believe Chemours or the Water Department should reimburse me for expenses paid to enable me to have clean water. I have the receipts! People in our area who cannot afford drinkable water are forced to consume water which very likely will cause them multiple medical problems, including several types of cancer. This is totally UNACCEPTABLE!

246. Dorothy Pawlowski (*ZIP code: 28479)*

Please clean up our water! We deserve clean water to use in our homes!

247. David Perry (*ZIP code: 28412)*
Make Chemours pay!

248. Dean Polumbo (*ZIP code: 28384)*

249. David Thomas (*ZIP code: 28411)*
Why should the residents of communities whose water supplies are contaminated by Chemours discharge , have to pay for the expense of filtering out the Chemours product to make the water safe to drink?

250. Drake Phelps (*ZIP code: 27513)*

251. Donna Romano (*ZIP code: 28409)*

252. PT Hogan (*ZIP code: 28451)*

253. Debra Shaw (*ZIP code: 28451)*
Chemours needs to be held responsible for the damage they've done to our water supply. They are clearly not going to do the right thing on their own.

254. Dorene Shirey (*ZIP code: 28451)*
Clean water that is safe to drink should be reasonably expected! Turning a blind eye to the poisons dumped into water sources should be criminal. The companies who contaminated our water must be held responsible to clean up their mess! Not a little cursory fine that costs the company less than proper management of chemicals and waste products—-but, the actual clean up and restoration to water that is safe!

255. Daniel Skrobialowski (*ZIP code: 28451)*
Chemours/DuPont create and introduce these chemicals into the environment without regard to the long term effects to public safety. They are responsible and should be made to be accountable for the cleanup, restitution and interim relief to all affected citizens.

256. Dennis Perler (*ZIP code: 28451)*

257. Donald Taylor (*ZIP code: 28451)*

258. Deborah Warner (*ZIP code: 28409)*

259. Dwight Willis (*ZIP code: 28462)*

260. Debbie Halley (*ZIP code: 28451)*

261. Denise Wright (*ZIP code: 28451-4504)*

262. Emma McLaughlin (*ZIP code: 28409)*

263. Eric Peterson (*ZIP code: 28451)*

264. Diann Driffing (*ZIP code: 28451)*

265. Eleanor DeMeglio (*ZIP code: 28461)*
Clean water is a NECESSARY resource for all citizens. Responsible parties MUST step up & address this situation. Current and future health concerns depend on DuPont/Chemours doing the right thing, doing it ASAP and doing whatever is necessary to ascertain what damage has already been done by these chemicals and the immediate cessation of their use.

266. Eden Avery (*ZIP code: 28409)*
To ensure that our family has safe water, we have been paying almost $100 a month EXTRA for bottled water in addition to what we have to pay for polluted public water! And we are priveleged to be able to do this for our family. Too many are not able to afford this - they should not have to. Clean, safe water should be a RIGHT for all! No more excuses.

267. Esther Murphy (*ZIP code: 28411)*

268. Rachel Williamson (*ZIP code: 28403)*

269. Ernesto Ferreri (*ZIP code: 28409)*
One day people will look back on our times and think: "How could they have allowed this to happen?". The answer: politicians who threw their constituents "under the bus" to make sure their campaign chests were full and favors owed to them were many-- if they were public servants they would not let this go on.

Clean water and air should not be hard to do, the corporations would still be making good money. Nothing wrong with that, but to harm the populace for a little extra is criminal.

270. Edward Stripling (*ZIP code: 28451)*
Chemours/DuPont should immediately close it's plant in North Carolina. They should be made to provide water filtration plants for all residents that use the water they contaminated and they should provide clean uncontaminated water to all persons affected until the filtration plants have been placed into operation.

271. Eileen Kigler (*ZIP code: 28451)*

272. Edith Kurie (*ZIP code: 28412)*

273. Elli Klein (*ZIP code: 28405)*
DuPont/Chemours = EVIL

274. Ellen Weinberg (*ZIP code: 28451)*

275. Elena Mock (*ZIP code: 27502)*

276. Ellen Colwell (*ZIP code: 28401)*
Clean drinking water needs to be a priority for our community! We pay for drinking water from the city and it is negligent for the city to do what is necessary to ensure that water is safe. We need to do better and we need to hold companies (DuPont/Chemours) Financially responsible for the damage they've done to our people.

277. emily grace (*ZIP code: 28412)*
Clean drinking water should be a right, not a privilege. Polluting is unfair for everyone. Do your part and take responsibility

278. Emily Peat (*ZIP code: 28479)*

279. Lynn Montroy (*ZIP code: 28451)*

280. Erik Olson (*ZIP code: 20005)*
On behalf of the Natural Resources Defense Council

281. Erika Ullman (*ZIP code: 27510)*

282. Eileen Lazecko (*ZIP code: 28409)*
As a breastfeeding mother, it is my duty to protect my son and this contamination directly effects us both. Stop this immediately!

283. Eileen Ronci (*ZIP code: 28451)*

284. Emily Donovan (*ZIP code: 28479)*

285. Emily Silverman (*ZIP code: 28451)*

286. Elijah Yetter-Bowman (*ZIP code: 27278)*

287. Evan Folds (*ZIP code: 28403)*

288. Ellen Mote (*ZIP code: 28409)*
My sister and I both contracted cancers within a few years of moving to Wilmington and drinking the tap water here. I survived, she did not. I am now working on the front lines against Covid 19 as a health care worker. What are DuPont and Chemours doing?

289. Faith Lough (*ZIP code: 28451)*
I can't believe this has not been resolved. Safe, Clean water should be the norm.

290. Katherine OBrien (*ZIP code: 28479)*
tired of polluted water.

291. Faye Bledsoe (*ZIP code: 28348)*

292. Frances Manning (*ZIP code: 27612)*

293. Paul Reali (*ZIP code: 28481)*

294. Howard Ferguson (*ZIP code: 28409)*
Please, keep our water and community safe from these silent potentially dangerous chemicals! All we ask is for our water to be safe! Not the trash csn for large companies that seem not to care about those downstream.

295. Fern Bugg (*ZIP code: 28403-6039)*

296. Fred Fiss (*ZIP code: 28461)*
Dupont should provide every household with filtrations systems at their expense. They should help clean the waters in our river. They should stop production now. They should also cover the expense of the expensive R.O. water plants to clean up their mess.

297. Jane Ledington (*ZIP code: 28451)*

298. Mary Holst (*ZIP code: 28403)*

299. Frank Kostek (*ZIP code: 28451)*

300. Frank Williams (*ZIP code: 28451)*
Frank Williams

301. Paula Jenkins (*ZIP code: 28401)*

302. FERNANDO MELON (*ZIP code: 28451)*

303. Brian Jones (*ZIP code: 28412)*
We demand compensation for your industrial pollution in our drinking water for decades.

304. Forrest McFeeters (*ZIP code: 28412)*

305. Amelia Florence (*ZIP code: 28461)*

306. frank volpe (*ZIP code: 28451)*

307. Francine Fiorentino (*ZIP code: 28451)*
Why as a citizen of the US do I have to chose between drinking water filled with chemicals or paying for clean water to be delivered to my home every week.
Are we a third world country and is there any reason why our representatives are not fighting for their

constituents to end this nightmare.

308. Frederick C Campau (*ZIP code: 28409)*
Stop polluting our water!

309. Frank Pinto (*ZIP code: 28451)*

310. Shirley LaRusso (*ZIP code: 28451)*

311. Cheryl Fulton (*ZIP code: 28461)*

312. Zachary Roscoe (*ZIP code: 28403)*
The water sucks! I'd like my my kids to not have to deal with any medical conditions in the future because of contaminated water!

313. Gaeten Lowrie (*ZIP code: 28405)*

314. Gail Haas (*ZIP code: 28451)*

315. Gary Markulic (*ZIP code: 28451)*

316. Sharon Snellgrove (*ZIP code: 28401)*

317. Gar Kramer (*ZIP code: 28451)*

318. Gary Ruta (*ZIP code: 28451)*
Gary Ruta

319. Karin Gately (*ZIP code: 28405)*

320. Gay Hull (*ZIP code: 28451)*

321. Gary Ruezinsky (*ZIP code: 28451)*

322. Gene Lindemann (*ZIP code: 28451)*
Chemours has been doing this for more then 20 years. Paying off local politicians and being allowed to knowingly dumping their pollutions into our rivers. they should be prosecuted for their crimes!
Gene Lindemann

323. Diane Smith (*ZIP code: 28451)*
Consumers deserve relief. Clean up your mess and pay for the RO system for the towns.

324. Geovanna Mckinnon (*ZIP code: 28306)*

Geovanna Best

325. Regina O’Donnell (*ZIP code: 28401)*

326. Virginia Conrad (*ZIP code: 28451)*

327. Gary Kugler (*ZIP code: 28451)*

328. Glenda Howard (*ZIP code: 28457)*

329. Glenn Walker (*ZIP code: 28409)*

330. Glenn Lazenby (*ZIP code: 28451)*

331. gordon johnson (*ZIP code: 28443)*

332. Gloria Shen (*ZIP code: 28805)*

333. Sandra Kesler (*ZIP code: 28468)*

334. Gail Capel (*ZIP code: 28465)*

335. Dawn Williamson (*ZIP code: 28312)*
We need better, long term solutuons to contaminated water and soil. I want to bathe, swim, wash dishes and clothes, etc in,clean water. My garden and livestock deserve to be taken care of with clean, unpolluted water

336. Alfia White (*ZIP code: 28409)*

337. Randi Gonen (*ZIP code: 28451)*

338. Grace Kromke (*ZIP code: 28412)*

339. Gregory Ryan (*ZIP code: 28451)*

340. Greta Bliss (*ZIP code: 28403)*
It is unacceptable to pollute the drinking water of entire communities, anywhere, at any time. The Covid crisis must not be a distraction or excuse. Chemours must clean up its act, NOW.

341. Dora Griffiths (*ZIP code: 28306)*

342. Brendan Martin (*ZIP code: 05468)*

343. Gary Savarese (*ZIP code: 28451)*

344. Ginger Ludwig (*ZIP code: 28479)*

345. Gail Spence (*ZIP code: 28479)*

346. Helen Freifeld (*ZIP code: 28451-6603)*

347. Halyn Blackburn (*ZIP code: 28412)*
Halyn Blackburn

348. Beth Hansen (*ZIP code: 28409)*

349. Gail Hogan (*ZIP code: 28411)*
We need Chemours to provide filters from their discharged PFAS chemicals in our water source in New Hanover county.

350. Jessica Osborne (*ZIP code: 28451)*

351. Barbara Harris (*ZIP code: 28403)*

352. Linda Dorshaw (*ZIP code: 28409)*

353. Bonnie Thiele (*ZIP code: 28405)*

354. Hedi Perotto (*ZIP code: 28401)*
Chemours has to be stopped and be made accountable for what they have done to our comnunity.

355. Henry Ponton (*ZIP code: 28451)*

356. Amy Hermann (*ZIP code: 28411)*

357. Dan George (*ZIP code: 28479)*
Chemours MUST be held accountable for knowingly polluting our river and our water supply. This has gone on way too long with zero accountability for the blatent disregard for the environment and human health.

358. Judith Gilbert (*ZIP code: 28465)*
please make brunswick county water safe!

359. Michael Hillman (*ZIP code: The 55406)*

360. Howard Flicker (*ZIP code: 28451)*

361. Holli Phillips (*ZIP code: 28371)*

362. Howard Hemeon (*ZIP code: 28451-9269)*
Action must be taken to stop this polluting by careless and irresponsible companies whose only concern is their bottom line. They must be stopped and they must pay for their reckless business practices...now!

363. Harry Hull (*ZIP code: 28451)*

364. Jeanne Dresser (*ZIP code: 21231)*

365. Cheryl Villante (*ZIP code: 28469)*

366. Robert Feldman (*ZIP code: 28451)*

367. ingrid lebowitz (*ZIP code: 28451)*

368. Dana Sargent (*ZIP code: 28479)*

369. Linda Bierer (*ZIP code: 28451)*
We deserve clean water now!

370. Iris King (*ZIP code: 28451)*
We all need CLEAN water!

371. Cheryl Godsey (*ZIP code: 28348)*
We need a PERMANENT and COMPLETE remedy to this contamination. RO will only cause leech field and septic system problems down the road and I KNOW chemours isn't going to pay for all those repairs and issues.

3 TAPS?????? What about SHOWERS and swimming in our own Pool and Growing our own FOOD on our OWN land that WE PAY FOR??????? Chemours deliberately contaminated and they need to deliberately correctly and COMPLETELY give us clean water!!!!!!

372. Ivanna Knox (*ZIP code: 28425)*

373. Jessica DeGolyer (*ZIP code: 28451)*

374. Callie & Jack Edmundson (*ZIP code: 28451)*

375. Jade Beavers (*ZIP code: 28401)*

376. Juditg Gooch (*ZIP code: 28461)*

377. Samantha Worrell (*ZIP code: 28425)*

378. Jaime Banta (*ZIP code: 28443)*

379. Jaime Gossin (*ZIP code: 28409)*

380. Jake Abrahamson (*ZIP code: 28401)*

381. JAMES FIORE (*ZIP code: 28451)*

382. James Coakley (*ZIP code: 28451)*
Why is this company still allowed to manufacture and pollute?

383. Jan Abbott (*ZIP code: 28451)*

384. Janet Rodrick (*ZIP code: 28412)*

385. Janet Beal (*ZIP code: 28451)*

386. Jane Palmer (*ZIP code: 28451)*

387. Janet Gorrell (*ZIP code: 28479)*

388. McElligott Janet (*ZIP code: 28451)*
Chemours must take responsibility now for their disgusting behavior. It should never have happened in the first place.

389. JANET FARRELL (*ZIP code: 28411)*
Help ! My OV STAGE 4 Cancer was discovered 2 1/2 years ago. Could be the water

390. Janice Mason (*ZIP code: 28479-5832)*

391. JANICE WOOLRIDGE (*ZIP code: 28479)*

392. Janet Helmers (*ZIP code: 28451-3405)*

393. Jan Wilkerson (*ZIP code: 28412)*

394. jason hudson (*ZIP code: 28403)*

395. Joseph Brown (*ZIP code: 28401)*

396. Julie Geery (*ZIP code: 28451)*

397. Joann Birkenstock (*ZIP code: 28451)*

398. Jennifer Campbell (*ZIP code: 28409)*

399. John Casciato (*ZIP code: 28412)*
I firmly believe that the only way to stop Chemours from polluting our waters is to shut them down. No more fines, regulations, deadlines, or broken promises.
It is time for ACTION!

400. Joyce Chmura (*ZIP code: 98115)*

401. Julia Martinelli (*ZIP code: 28468)*

402. Jean Hamilton (*ZIP code: 28451)*

403. Wendy Levens (*ZIP code: 28479)*
I strongly support this petition.
Wendy Levens

404. Jean Catanzaro (*ZIP code: 28451)*

405. Jean-Marie Whittington (*ZIP code: 28451)*

406. Jeanne Gillespie (*ZIP code: 28479)*

407. Jeannie Lennon (*ZIP code: 28403)*

408. Jeffrey Meuwissen (*ZIP code: 28451)*
Chemours/DuPont should be required to build new Reverse Osmosis water treatment plants for all affected water users. They polluted it, why should they not be responsible for correcting the problem? General Electric in NY was required to dredge the Hudson River to treat PCB contamination. Precedence has been established. Do not let Chemours off the hook!

409. Jeanne Green (*ZIP code: 28451)*

410. Jen Johnson (*ZIP code: 28403)*

411. Jen Mara (*ZIP code: 28443)*

412. Jennifer Kiernan (*ZIP code: 28451)*

413. Jenny Sassman (*ZIP code: 28209)*

414. Jesse Shaw (*ZIP code: 28451)*

Chemors’s behavior is unamerican.

415. Jess Sciuto (*ZIP code: 28451)*

416. JOHN JAMESON (*ZIP code: 28412)*
We need to have these issues fixed at the source

417. Joyce Formy-Duval (*ZIP code: 28409)*

418. Jeff Gray (*ZIP code: 28451)*
Please provide clean water to all residents of Leland. Chemours or any company releasing chemicals into the Cape Fear or other waterways is unacceptable.

419. Jennifer Greene (*ZIP code: 28405)*

420. Jeffrey Hall (*ZIP code: 28411)*
Make dupont pay for an entire, system-wide cleanup solution for all of Cape Fear water customers. Regardless of the price.

421. Jennifer Hudson (*ZIP code: 28403)*

422. Jillian Anderson (*ZIP code: 27701)*

423. Jim Zelenski (*ZIP code: 28451-9734)*
Let's fix this now!

424. Joy gregory (*ZIP code: 28412)*

425. Judy DiMizio (*ZIP code: 28451)*

426. Jean Kohner (*ZIP code: 28451)*

427. Jack Koonce (*ZIP code: 28412)*

428. Judy DiMizio (*ZIP code: 28451)*

429. Lisa Bazinet (*ZIP code: 28306)*

430. Jan Ligas (*ZIP code: 28451)*

431. Joan Zeltmann (*ZIP code: 28451)*

432. JANE MARTIN (*ZIP code: 28461)*

433. Jocelyn McGuinness-Hickey (*ZIP code: 27615)*

434. Jeff Gerhart (*ZIP code: 28401)*

435. Joanne Shy (*ZIP code: 28412)*
It's despicable that this is allowed!! Polluting any water should be punishable.

436. John Myers (*ZIP code: 28348)*

437. Joseph Hennessey (*ZIP code: 28461)*
stop the river/drinking water pollution

438. Joan Eipper (*ZIP code: 28451)*
Be considerate. Have some respect. Do what is right for your fellow man and CHILDREN!

439. Joann Bristol (*ZIP code: 28409)*

440. John Wood (*ZIP code: 28412)*

441. John Thompson (*ZIP code: 28451)*

442. Martha Johnson (*ZIP code: 28461)*

443. John Stipa (*ZIP code: 28451)*

444. John Bays (*ZIP code: 28451)*

445. Joanne Reeves (*ZIP code: 28451)*

446. Joanne Levitan (*ZIP code: 28451)*

447. Jon Beals (*ZIP code: 28451)*

448. Kathleen Jones (*ZIP code: 28403)*

449. Barbara Jordan (*ZIP code: 28451)*

450. Jorge Corzo (*ZIP code: 28269)*
I was force to move out from Wilmington NC because my health issues, now I relocated to Charlotte NC, and my health started back on track

451. Joy Cranidiotis (*ZIP code: 28451)*
Allowing Chemours to poison our children and families for years has to stop. Here in Brunswick

County, most of my family and friends are on thyroid medications and have numerous other maladies that now can be linked to the water supply, the Cape Fear River. And Chemours has and continues to dump GenX and 250 unknown PFAS into the Cape Fear River. Impacted communities need immediate relief NOW!!!

452. Joyce Spencer (*ZIP code: 28451)*

453. Joy DeMeglio (*ZIP code: 28461)*

454. John Finn (*ZIP code: 28451)*

455. Patricia Devine (*ZIP code: 28451)*

456. James Powers (*ZIP code: 28451)*

457. Jessica Middleswarth (*ZIP code: 28451)*

458. Jeremy Middleswarth (*ZIP code: 28451)*

459. Jeffrey Long (*ZIP code: 28451)*
I think they should do something to make up for the fact that we can no longer drink our water.

460. Risa Rodriguez (*ZIP code: 28306)*

461. JAMES SMITH (*ZIP code: 28451)*

462. Joseph Saporta (*ZIP code: 28451)*
It's disgraceful that this water is contaminated.

463. Janet Shorter (*ZIP code: 28451)*

464. Joseph Digirolomo (*ZIP code: 28451)*

465. Jessica Travis (*ZIP code: 28405)*

466. Judith Chandler (*ZIP code: 28412)*

467. Julia McClure (*ZIP code: 28468)*

468. Julia Leimkuhler (*ZIP code: 28409)*

469. Julia Brock (*ZIP code: 40502)*

470. Julie Marie (*ZIP code: 28461)*

471. Justin Thompson (*ZIP code: 28479)*

472. Justin Soponis (*ZIP code: 28451)*

473. Joni King (*ZIP code: 28401)*

474. Alan Just (*ZIP code: 28451)*

475. Joanne Woolgar (*ZIP code: 28451)*

476. J Barbara Bakowycz (*ZIP code: 28409)*
I am a Wilmington, NC resident. My background as a registered nurse is in critical care and community health. NONE of this is acceptable. The recent discovery of 21 ADDITIONAL unknown PFAS chemicals contaminating our tap water...compounded by the lack of disclosure by Chemours...is doubly egregious.

477. Kade Hampton (*ZIP code: 83646)*
They who have spilt and poisoned the watersupply shall be responsible indefinitely.

478. Matthew McCoy (*ZIP code: 28403)*

479. Kathy Chavis (*ZIP code: 28312)*
Absolutely we should be protected from these chemicals. We should be provided clean and chemical free water. In our area of this we should be added on to the water lines that are right near us already.

480. Trish Eberhard (*ZIP code: 28451)*
We need to stop the poisoning NOW

481. kandace williams (*ZIP code: 28465)*

482. Kara Kenan (*ZIP code: 28479)*
It is absolutely absurd that people living downstream from Chemours are STILL at risk. We deserve relief and we demand it NOW.

483. Karen Pappas (*ZIP code: 28443)*

484. Karen Rodenheiser (*ZIP code: 28451)*

485. Kasey Werner (*ZIP code: 28411)*

486. Kate Griffin (*ZIP code: 28412)*
Stop the poison. Make the polluters pay.

487. Katheryn Lozer (*ZIP code: 28479)*

488. Kathie Jordaens (*ZIP code: 28462)*

489. Kathleen Yonce (*ZIP code: 28465)*

490. Kathryn Polk (*ZIP code: 28411)*

491. Kathryn Edwards (*ZIP code: 28451)*

492. Kathy Lambui (*ZIP code: 28401)*

493. Katy Monaghan (*ZIP code: 28403)*

494. Karen Worden (*ZIP code: 28479)*

495. Sharon Stewart (*ZIP code: 28412)*

496. Kayla Fryar (*ZIP code: 28479)*

497. Kristi Simms (*ZIP code: 28403)*
Chemours should pay to clean up their mess.

498. Loribeth Meunier (*ZIP code: 28411)*

499. Kelly Chase (*ZIP code: 28451)*

500. Keri Wray (*ZIP code: 28451)*

501. Kerri Murdock (*ZIP code: 84535)*

502. Daniel Donnellan (*ZIP code: 28451)*

503. Karen Groves (*ZIP code: 28451)*

504. Kathleen Hewes (*ZIP code: 28479)*

505. Kathy Hall (*ZIP code: 28451)*

506. Kim Swinny (*ZIP code: 28405)*

507. Kim Otto (*ZIP code: 28451)*

508. Kimberly Hulon (*ZIP code: 28403)*
Thank you for your efforts in protecting residents and holding violators accountable!

509. Kim Freeman (*ZIP code: 28412)*

510. bryan king (*ZIP code: 28451)*

511. Ericka Marino (*ZIP code: 28451)*
Ericka Marino

512. Kathryn Riss (*ZIP code: 08854-7516)*

513. Kathleen Kulage (*ZIP code: 28451)*

514. Karen Abbott (*ZIP code: 28451)*

515. Kevin Funk (*ZIP code: 28443)*
Does Chemoyr really bring enough money to the region to warrant them getting a pass in basically poisoning our water? I think not.

516. Kristina Campbell (*ZIP code: 28348)*
My daughter and myself have numerous health conditions since Relocating to the grays creek area near chemours 10 years ago. 10 years of constant Unexplainable health problems

517. Kathleen McDonough (*ZIP code: 28452)*

518. Katelyn McKinney (*ZIP code: 28401)*

519. Kenneth Thies (*ZIP code: 28403)*

520. Donna Stokes (*ZIP code: 28348)*

521. kathryn koppel (*ZIP code: 28451)*

522. Gary Krauss (*ZIP code: 28451)*

523. Kathy Rayle (*ZIP code: 28403)*

524. Heather Kreidler (*ZIP code: 28479)*

525. Kristine Bowman (*ZIP code: 28451)*
We're mad and arent going to take it anymore. We need clean water NOW!

526. Krista Jorgensen (*ZIP code: 28405)*
I am requesting NC DEQ establish a PFAS Community Relief Fund paid for by Chemours.

527. Krista Jones (*ZIP code: 28479)*

528. Kristen Grecco (*ZIP code: 28479)*

529. kristina speight (*ZIP code: 28479)*

530. Kristin Barfield (*ZIP code: 28348)*

531. Kristine Hoegh (*ZIP code: 28409)*

532. Kristine Serpa (*ZIP code: 28306)*

533. Kathie Schiller (*ZIP code: 28451)*

534. Douglas Dove (*ZIP code: 28451)*

535. Kent Mickel (*ZIP code: 28451)*
Shut down that plant asap they are incompetent

536. Katie Owen (*ZIP code: 28479)*

537. Kyle Stokes (*ZIP code: 28348)*

538. KAtie Gates (*ZIP code: 28409)*
We have moved to Wilmington this year and have already had unusual health impacts that no one can pinpoint. I am very concerned for all of our health, but especially my 2 teenage children health. I don't want them to develop precancerous condition, have reproductive/fertility issues due to water pollution. We live int he US of A and since the Clean Water Act in the early 1970's I would expect that our water is some of the cleanest int he world. Not so in Wilmington. I'm flabbergasted that Dupont/Chemours can get away with such incredible pollution for so many years with undeniable evidence of toxicity of effluent like GEn X and PFOA forms to downstream communities. This corruption has to go. We need Chemours/Dupont to pay for all the upgrades to our water treatment plants ASAP. We live near Carolina beach and when we swim there, my kids are affected by the toxins in the water that comes out of Snows Cut. We have invested a lot in our move from Colorado and bought a new home. I'm beginning to regret that in doing so I have put my family in harm's way. We love Wilmington, the people, the area and need this pollution atrocity addressed ASAP. We have invested thousands in a whole house water filter system and will try to install a small RO tank under our sink. These are major unforeseen expenses for us and there is still no guarantee that all GEn X and PFOAs are removed as I cant afford the $500 to pay to have a water sample tested. Thanks for holding this industry and company accountable.

539. Kimberli Theophilos (*ZIP code: 28412-3490)*

540. Kathy Weitner (*ZIP code: 28411)*

541. karen tracy (*ZIP code: 28451)*

542. Connie Craddock (*ZIP code: 28451)*

543. Darlene Levine (*ZIP code: 28451)*

544. Lauren Francis (*ZIP code: 28405)*

545. Shannon Mansfield (*ZIP code: 28409)*

546. Larissa Claar (*ZIP code: 28457)*

547. Sherry Mulhollen (*ZIP code: 28451)*

548. Ann Stokes (*ZIP code: 28405)*

549. Laura Goode (*ZIP code: 28753)*

550. Laura Rayman (*ZIP code: 28409)*

551. Laura Trivett (*ZIP code: 28403)*

552. laura ward (*ZIP code: 28469)*

553. Laurene Rapoza (*ZIP code: 28401)*

554. Lauren Quattrucci (*ZIP code: 28412)*

555. Laura Beck (*ZIP code: 28451)*

556. Bernadette Morris (*ZIP code: 28409)*

557. Lusa Bowers (*ZIP code: 28348)*

558. Loraine Buker (*ZIP code: 28409)*

559. Leonard Burdick (*ZIP code: 28451)*

560. Linda Busineau (*ZIP code: 28451)*

561. Leo Van Herpe, M.D. (*ZIP code: 28451)*

562. Lyle Benson (*ZIP code: 28451)*

563. Leah Schenck (*ZIP code: 28409)*

564. David Murray (*ZIP code: 28451)*
DuPont/Chemours should stop discharging forever chemicals into our water. They should fully fund a community relief fund. They should pay for clean water and stop forcing us to drink poison. They should develop and discover new technology to safely dispose of and break down forever chemicals, instead of pushing them into our land and water.

565. Patric LeBeau (*ZIP code: 28409)*
This must stop.

566. Lee Bryant (*ZIP code: 28480)*
We should be reimbursed for having to purchase clean drinking water.

567. Elyse Sherman (*ZIP code: 28451)*
Chemours' negligence in this matter is unconscionable. We have had to buy a reverse osmosis system for our home at our expense. The fact that there were unhealthy contaminants in the Leland water was not revealed to us when we bought our home, so a decision to a) move here and b) install a whole house RO system was not made available to us. This is an outrage! And Leland is not planning to do anything about this until 2023! Chemours should pay for the "adjustments" we have had to make to our homes, now and in the future, for their negligence and continued abuse of the environment.

568. Leslie Christensen (*ZIP code: 28451)*

569. Leslie Antos (*ZIP code: 28480)*
DuPont/Chemours actions and inactions have affected downstream communities. They need to endure clean water for these counties as well. There are consequences to knowingly taking away people's source of clean water.

570. Leslie Stewart (*ZIP code: 27516)*

571. Lisa Menius (*ZIP code: 28409)*

572. Laurie Hoegler (*ZIP code: 28451)*

573. Margaret DeLuca (*ZIP code: 28409)*

574. Liliana Berman (*ZIP code: 28451)*

575. Lili Fiore (*ZIP code: 28451)*

576. Linda Eastman (*ZIP code: 28469)*

577. Linda Shilts (*ZIP code: 28451)*

578. Linda Ronan (*ZIP code: 28412)*

579. Lindsay Lake (*ZIP code: 28403)*

580. Lindsey McCoy (*ZIP code: 28403)*

581. Lin Summers (*ZIP code: 28409)*

582. Lior Vered (*ZIP code: 27516)*

583. Lisa Getz (*ZIP code: 28403)*

584. Lisa Myers (*ZIP code: 28451)*

585. Lisa Wisner (*ZIP code: 28405)*

586. Liz Saller (*ZIP code: 28465)*

587. Leslie Lillo (*ZIP code: 28451)*

588. Lydia Mahoney (*ZIP code: 28451)*

589. Lauren Knowles (*ZIP code: 28479)*

590. Kyle Horton (*ZIP code: 28409-5829)*

591. Lois Lewis (*ZIP code: 28348)*
They need to figure out a better way to take care of the problem they caused. The water filters are not working. We need water city water hook up and they pay for it.

592. Lynn Anderson (*ZIP code: 28405)*
As a downstream resident, I'm concerned for the health of my neighbors who may not be financially positioned to purchase bottled water. I'm concerned for my own property value that will be diminished once buyers are more widely aware of the additional health risk of residing in Hew Hanover County.

593. Linda Mortensen (*ZIP code: 28457)*

594. Marie Lockhart (*ZIP code: 28451)*

595. April Fieno (*ZIP code: 28479)*
Its shameful that this has been allowed for so long.

596. Loraine Carbone (*ZIP code: 28479)*
Loraine Carbone

597. Lori Martin (*ZIP code: 28451)*
Please address our water source. I didn't move here to die. This beautiful state should have a clean water supply. We may move to find that if nothing is done.

598. Lorri Honeycutt (*ZIP code: 28409)*
Lorri Honeycutt

599. Lou Mateya (*ZIP code: 28451)*

600. Linda Allen (*ZIP code: 28411)*
They should pay for this TOXIC water

601. Linda Eiman (*ZIP code: 28451)*
I want clean water for everyone and DuPont should pay for it. This is America!! We should not even be having this discussion!!

602. Glenn Tetterton (*ZIP code: 28401)*
I brought up a child on water from the Lower Cape Fear. We filtered it, but that was not enough to remove these chemical pollutants. My wife and I both have thyroid issues. What else should we expect from our water? Anger does not begin to describe my feelings on this.

603. leland evans jr (*ZIP code: 28451)*

604. Tom Simmons (*ZIP code: 28461)*
The children in our public schools should have clean, safe drinking water.

605. Karen Fleming (*ZIP code: 28451)*

606. Lydia Hall (*ZIP code: 28457)*

607. Lynda Loytty (*ZIP code: 28409)*

608. Lynn Paterson (*ZIP code: 28479)*

609. Lynne Gibbs (*ZIP code: 28479)*

610. Melanie Beightley (*ZIP code: 28403)*

611. Mary Anne McDonald (*ZIP code: 27701)*
DuPont/Chemours has a responsibility to the affected communities who they have exposed to PFAS. DuPont/Chemours must be held responsible and pay for the damage they caused these communities.

612. Marcia Morgan (*ZIP code: 28428)*

613. Anne Ferigo (*ZIP code: 28409)*

614. Tara Smith-Russell (*ZIP code: 28451)*

615. Margaret Mullins (*ZIP code: 28428)*

616. Magdalena Bonk (*ZIP code: 28443)*

617. Patricia Kelley (*ZIP code: 28451)*

618. Marguerite White (*ZIP code: 28451)*

619. Cynthia Mascia (*ZIP code: 28451)*
Stop already! They should be fined for this!!!

620. Mary Morgan (*ZIP code: 28401)*

621. Maralee Demark (*ZIP code: 28451)*

622. Marci Curtis (*ZIP code: 27614)*
Everyone deserves clean water. Rural residents across the state need relief from chemical contamination in their drinking water. I am requesting NC DEQ establish a PFAS Community Relief Fund paid for by Chemours. This fund will provide impacted residents with immediate relief from these continued toxic exposures.

623. Marci Staten (*ZIP code: 28411)*

624. Margaret Walsh (*ZIP code: 28451)*

625. Andrew Marhevsky (*ZIP code: 28409)*

626. Marian Schnitzel (*ZIP code: 28461)*

627. Mariel Kruse (*ZIP code: 28443)*

628. Marilyn Angello (*ZIP code: 28451)*
We deserve clean drinking water. The companies that are responsible for dumping known

contaminants in our water supply need to be held accountable.

629. Marilyn Bergeron (*ZIP code: 28451)*

630. Marina Nielsen (*ZIP code: 28409)*

631. Mark Gorrell (*ZIP code: 28479)*

632. Mark Bromeier (*ZIP code: 28412)*

633. Martin Weinberg (*ZIP code: 28451)*

634. marvin jacobs (*ZIP code: 28479)*
DuPont needs to clean up the environment of the PFAS.

635. Mary Turner-Danylec (*ZIP code: 28405)*

636. Maryann Gherardi (*ZIP code: 28451)*

637. Mary Sturgill (*ZIP code: 28451)*

638. Mary Carroll (*ZIP code: 28451)*

639. Mary Ellen Bell (*ZIP code: 28451)*
Chem ours must pay for the clean up and healthcare needs resulting from dumping over the years.

640. maston howze (*ZIP code: 28391)*

641. Matthew Bland (*ZIP code: 28403)*

642. Matthias Rhein (*ZIP code: 28306)*
We need while house systems for everyone!!!

643. Matthew Spinner (*ZIP code: 28451)*

644. Mary beth Cowper (*ZIP code: 28409)*

645. Madeline Kramer (*ZIP code: 28403)*

646. Scott Mcclung (*ZIP code: 28451)*

647. Melissa Green (*ZIP code: 28462)*

648. Mary Lee McKell (*ZIP code: 28451)*

649. Marilyn Shapiro (*ZIP code: 28470)*

650. Marie Garcia (*ZIP code: 28451)*
Had I known about this issue, I would have never moved to the community a year ago

651. Toni Carroll (*ZIP code: 28451)*

652. Jesse Shaw (*ZIP code: 28451)*

653. Michael Duda (*ZIP code: 28479)*

654. Gloria Thomas (*ZIP code: 28467)*

655. Meghan Phillips (*ZIP code: 28402)*

656. Melissa Philpot (*ZIP code: 28348)*
We need a solution that will help the residents long term. Filters on sinks and whole house filtration isn't enough. It's time to force all involved to stop placing a bandaid on a wound that requires stitches.

657. Melea Stoltenberg (*ZIP code: 28326)*

658. Melissa Huffman (*ZIP code: 28451)*
Clean water should be a priority!

659. Melody Casteen (*ZIP code: 28451)*
I’ve lived here my whole life, surely I’ve consumed more than my fair share of this mess. I’ve now had to spend more money in bottled water for the safety of myself and family.
Something has to be done about this.

660. Mel Rauch (*ZIP code: 28403)*

661. mary petro (*ZIP code: 28451-____)*
It is unconscionable that this company with a history of water pollution has not been made responsible for cleaning up their mess! It is time for government response to force remediation for ALL river water consumers.

662. Meredith Scharton (*ZIP code: 28451)*

663. Merridy Bilodeau (*ZIP code: 28451)*

664. Douglas Helmers (*ZIP code: 28451)*

665. Marie Gordon (*ZIP code: 20902)*

666. SPARY DAUTERMAN (*ZIP code: 28403)*

667. Janice Metz (*ZIP code: 28451)*

668. Marisa Falank (*ZIP code: 28479)*

669. Manuel Fort (*ZIP code: 28348)*

670. Mike Fleming (*ZIP code: 28451)*
This is very serious. Quick, strong assistance ir respectfully requested.

671. Mary Frances McClure (*ZIP code: 28409)*

672. Madeleine Gordon (*ZIP code: 28479)*
My husband lost his life to mantle cell lymphoma , a rare form of lymphoma, in 2015. He drank nothing but water from our taps in Oak Island (zip 28465)

673. Margaret Graff (*ZIP code: 27451)*

674. Marg Benson (*ZIP code: 28451)*

675. Michael DeMeglio (*ZIP code: 28461)*

676. Michelle Soules (*ZIP code: 28451)*

677. MIchael Kirsche (*ZIP code: 28412)*

678. Michael Stambaugh (*ZIP code: 28411-7151)*

679. Mildred Bethea (*ZIP code: 28401)*

680. casey miner (*ZIP code: 28401)*

681. Melissa Moore (*ZIP code: 28451)*
Diagnosed with leukemia 4/2020 after living in Brunswick county for the last 7+ years. I know there are thousands of us that have been diagnosed with terrifying illnesses and diseases Due to our lack of clean drinking water. ITS UNACCEPTABLE!!! It’s 2020 and for AMERICANS to not having quality fresh drinking water is insane!!

682. Martina Jonsson-Boykin (*ZIP code: 28412)*

683. Melissa Juhan (*ZIP code: 28451)*

684. Monica Williamson (*ZIP code: 28409)*

685. Mary Seigfreid (*ZIP code: 28451)*
It is unconscionable that you find it ok for others to drink water that contains many chemicals that are hazardous if not deadly to them.
I live in Leland. It's the fastest growing city in NC. Which translates into a large tax base as well. Many people are finding out about the water issues and are deciding not to come here. If we would've known about how NC, doesn't care about the environment much less its citizens, we would have gone elsewhere as well.
Please do the right thing and make those who are polluting our water, thus the ground and air, clean our water so we can live a healthy life.
Thank you for your time.
Mary Seigfreid

686. Marcia Kosslow (*ZIP code: 28451)*

687. Kathleen Tyler (*ZIP code: 28405)*

688. Merrily Locke (*ZIP code: 28401)*

689. Mike Mckay (*ZIP code: 29401)*

690. Melissa Ross (*ZIP code: 27562)*

691. MELISSA WORRELL (*ZIP code: 28409)*
Paying for water that I can not drink, and I can not afford a reverse osmosis system. Have to get bottled water every week. Also hate showering, etc. in it. Please help!

692. Marilyn O'Brien (*ZIP code: 28451)*
I am OUTRAGED THAT THIS IS ALLOWED TO CONTINUE. It is bad enough that our drinking water is unsafe with so many carcinogens, but I feel it is absolutely unconscionable to allow builders to continue to build and sell homes to unsuspecting buyers (such as myself). It is my understanding that our children are provided bottled water to drink while in school. What about the residents of Brunswick County? Additionally, in this election year, I find it difficult to believe that our elected representatives seemingly are not willing to "go to bat" for their constituents.

693. Molla Donaldson (*ZIP code: 28461-2943)*

694. Molly Stuart (*ZIP code: 28403)*

695. Molly Curnyn (*ZIP code: 28409)*

696. Elizabeth Wittmer (*ZIP code: 28403)*

The very least this company can do until they have eradicated this chemical from our water supply is to provide RO water filtration systems to each and every household affected by their manufacturing.

697. Michele Wuensch (*ZIP code: 28409)*

698. Darla Thomas (*ZIP code: 28306)*

699. S McCourt (*ZIP code: 28451)*

700. Monica Rusko (*ZIP code: 28451)*

701. Monica Rolquin (*ZIP code: 28409)*

702. Sharon Powers (*ZIP code: 28479)*
Every Home should get FREE filter RO systems....

703. Janet Decou (*ZIP code: 28465)*

704. Patricia Martin (*ZIP code: 28451)*

705. Meghan Henderson (*ZIP code: 28405)*
This has gone on for far too long. It's time for change.

706. Stephanie Small (*ZIP code: 28451)*

707. Jeanne Johnson (*ZIP code: 28451)*
This problem just keeps getting worse and, unfortunately, we the people are the ones being hurt. We have been exposed to dangerous chemicals and we are petrified to drink the water. Now is the time for you to do the right thing. You need to ensure that Chemours sets up a relief fund to protect everyone.

708. Mary Schoeler (*ZIP code: 28451)*

709. Marilyn Sakowski (*ZIP code: 28451)*

710. Audrey Mike parker (*ZIP code: 28411)*

711. Matt Stewart (*ZIP code: 28479)*

712. Maria Stone (*ZIP code: 27518)*

713. Miles Murphy (*ZIP code: 28403)*

714. Maria Ange (*ZIP code: 28411)*

715. Michael Workman (*ZIP code: 28451)*

716. myra dotson (*ZIP code: 27516)*

717. Myrlena Lee (*ZIP code: 28451)*

718. Sharon Salz (*ZIP code: 28451)*

719. Neil Gilbert (*ZIP code: 28468)*

720. Nancy Lamb (*ZIP code: 28465)*

721. Nancy Celli (*ZIP code: 28479)*

722. Natalie Hinton-Stalling (*ZIP code: 28411)*

723. Kayla Benton (*ZIP code: 28412)*

724. Joyce Farmer (*ZIP code: 28405)*

725. Nancy Simpson (*ZIP code: 28451)*

726. Patricia Nabors (*ZIP code: 28348)*

727. Sue Patterson (*ZIP code: 28405)*

728. William Taylor (*ZIP code: 28403)*

729. Denise Daniels (*ZIP code: 28451)*
We deserve to have clean water!

730. Carol Felenstein (*ZIP code: 28451)*

731. Nicholas Hiteshew (*ZIP code: 28409)*

732. Nick kipriotis (*ZIP code: 28348)*

733. Nicholas Newell (*ZIP code: 28479)*
Only in America can corporate greed kill Americans legally.

734. Nina Marable (*ZIP code: 28468)*

735. Noel Santorelli (*ZIP code: 28451)*

736. Noelle Powers (*ZIP code: 28401)*

737. Daniel Norkun (*ZIP code: 28451)*

738. Nicole Ratliff (*ZIP code: 28451)*

739. Melanie Nusbaum (*ZIP code: 28479)*
We all deserve clean drinking water!

740. Nicole Wendelbo (*ZIP code: 27278)*

741. Oliver Downey (*ZIP code: 28451)*

742. Olivia Clifton (*ZIP code: 28401)*

743. Olof Preston (*ZIP code: 28405)*
We deserve clean drinking water

744. Kempie Kirkland (*ZIP code: 28412)*

745. Olinka Hollie (*ZIP code: 35758)*

746. Barbara Price (*ZIP code: 28479)*
They have to be stopped!! AND held legally responsible!

747. Melinda McEnroe (*ZIP code: 28411)*

748. Patricia Walpole (*ZIP code: 28451)*

749. Claire ODonnell (*ZIP code: 28401)*
Both my parents got cancer, living in New Hanover County. A PFAS Community Relief Fund is the LEAST Chemours/DuPont can do about the devastation they are causing in our communities. Fund this, clean up our area, get out of here, and stop poisoning people in communities where you are located.

750. Debra Oryszak (*ZIP code: 28462)*

751. Gwendolyn Osborne (*ZIP code: 28451)*
Water in Leland is nasty. If you don't believe me, run your bathtub full and see how dirty it is. Taste is terrible also. I buy bottled water to drink and cook.

752. Pam Sender (*ZIP code: 28451)*

753. Pam Watkins (*ZIP code: 28403)*
Chemours needs to be held accountable for this mess.

754. Patricia Pettinati (*ZIP code: 28451)*

755. Patricia Malusa (*ZIP code: 28451)*
poor water quality. Should not be in the US

756. Patricia Ward (*ZIP code: 28412)*

757. Pat Chisholm (*ZIP code: 28443)*

758. Patricia Moakler (*ZIP code: 28451)*

759. Paula Carson (*ZIP code: 28451)*
Our water has been ruined by Chemours and they need to stop dumping into the rivers and work on cleaning up this mess.
Paula Carson

760. Pamela Bolduc (*ZIP code: 28409)*

761. Philip Bowman (*ZIP code: 28451)*

762. paul DeLong (*ZIP code: 28409)*
Chemours needs to clean up their mess! Its not just about money. remove the chemicals! I blame my son's premature birth and costs associated on Chemours!

763. Peggy Lacey (*ZIP code: 28461)*

764. Marguerite Herga (*ZIP code: 28412)*
I am very concerned about How the contaminated Cape Fear River water is effecting our health , the increased chances of getting Covid virus .. the chances of cancer, our grand children to many things to name .. please have DuPont clean up our river and put in a proper filtration or compensate our County New Hanover, so this problem can be addressed and alleviated for the future of our citizens!
This is so important and this is our only Water Drinking resource!
Please don't leave us out !!
I am a Retired spec Ed teacher in NHCS
Raised both our children here for 26 yrs ~ children grown. But
now have 2 grandchildren .. here
Need this fixed !
I know first hand what environmental impacts do to young children in education ...
it's a beautiful place to live .. please help us get our river clean again DuPont should not be allowed to do this ..
Thank you !

M J Herga
3826 Appleton Way
Wilmington, NC

765. Penny Larason (*ZIP code: 19444)*

766. Rodman Roberts (*ZIP code: 28479)*
It appears that elected officials and government agencies continue to put profits from big companies over peoples health. It also appears that that the negotiation process has not been 100% inclusive. The net result is that the citizens continue to be burdened money and health issues related to toxic drinking water.

767. Chris Gillis (*ZIP code: 28451)*

768. Patricia Foote (*ZIP code: 28451)*

769. Dave Ritter (*ZIP code: 28451)*
Let's get this resolved once and for all! Chemours should be severely fined.

770. Philip Brown (*ZIP code: 28409)*

771. Phoebe Gooding (*ZIP code: 27703)*

772. Pat Walsh (*ZIP code: 28451)*

773. Casey Scott (*ZIP code: 28451)*

774. Peter Muenzen (*ZIP code: 28451)*

775. Paul Healy (*ZIP code: 28451-6511)*
Need refund for RO install and filter costs

776. Steve Roberts (*ZIP code: 28401)*

777. Steve Roberts (*ZIP code: 28401)*

778. Leon Mckay (*ZIP code: 28403)*

779. Jamie Brake (*ZIP code: 28451)*

780. Patricia Paolini (*ZIP code: 28451)*

781. Natalie Labate (*ZIP code: 28412)*
Forcing us to pay for the cleaning of pollution Chemours created is unjust in every way. Polluters

must be held to account for their actions!

782. Pamela Roth (*ZIP code: 28451)*
Pamela R. Roth

783. Paige Riddle (*ZIP code: 28306)*
My family has lived less than 2mi from the Fayetteville Works plant for 30+ years. They would have community communications “informing the neighbors” but apparently all lies.

784. Rosemary Lucas (*ZIP code: 28409)*

785. Margaret Spallek (*ZIP code: 28480)*

786. Patricia Baumann (*ZIP code: 28451)*
Need refund for RO system and filters

787. Sandy Jones (*ZIP code: 28451)*

788. Carlos Lee (*ZIP code: 28451)*

789. Jennifer Henthorn (*ZIP code: 28451)*

790. Candace Waugh (*ZIP code: 28451)*

791. Pam Walker (*ZIP code: 28403)*

792. Rachel Gillilan (*ZIP code: 28405)*

793. Romando Daniels (*ZIP code: 28451)*
Why is Chemours still allowed to dump PFAS into the Cape Fear River? This is a travesty and those responsible should be held accountable and prosecuted.

794. Mimi Kessler (*ZIP code: 28403)*

795. Steven Rauschkolb (*ZIP code: 28451)*

796. Peter Rawitsch (*ZIP code: 28443)*

797. Raymond McAlonan (*ZIP code: 28451)*
Please stop poisoning my family.

798. Rosemary Beals (*ZIP code: 28451)*

799. Kathy Moore (*ZIP code: 28348)*
I can not grow a garden because of the toxic water and soil. I now have kidney disease. Had to pay another company to come and and put a whole house filter system in which I feel Chemour should give every affected house.

800. Rachel Panting (*ZIP code: 28411)*

801. Audrey Napier (*ZIP code: 28348)*
We should not have to accept a band aid such as a R.O system because it is cheaper for Chemours. It is a gunshot wound and a band aid will not work. We need a whole house system. Do you realize the R.O. system does not apply to our hot water. Do you realize alot of us have gone through hot water heaters thinking it is sediments from our wells. No! It is the sediment from PFAS. Hot water does not rid PFAS so why is that Chemours won't protect our hot water?????

802. Uyen Nguyen (*ZIP code: 28462)*

803. ROBERT DE HAAS (*ZIP code: 28412)*

804. Richard Groves (*ZIP code: 28401)*

805. Regina Cicchetti (*ZIP code: 28451)*

806. Regina Murray (*ZIP code: 28451)*

807. Kathryn Reilly (*ZIP code: 28405)*

808. Rena Mclaurin (*ZIP code: 28348)*

809. Bernard Quattrucci (*ZIP code: 28479)*
CLEAN OUR WATER!!

810. Rexann Williams (*ZIP code: 28465)*

811. Rebecca Felton (*ZIP code: 28461)*

812. Erica Grantmyre (*ZIP code: 28461)*

813. Rich McElaney (*ZIP code: 28412)*
Hold Chemours accountable for its continuous toxic assault on public health

814. RICHARD PIZZIMENTi (*ZIP code: 28451)*

815. Sherrill Hewitt (*ZIP code: 28451)*
This can't continue.

816. Rissa Meisner (*ZIP code: 28306)*

817. Cristina Perez (*ZIP code: 28451)*

818. ritch burgess (*ZIP code: 28451)*

819. Gail Ritter (*ZIP code: 28451)*

820. Robert Bailey (*ZIP code: 28451)*

821. Linda Carlson (*ZIP code: 28412)*

822. rachel glenn (*ZIP code: 28451)*

823. Richard Maxted (*ZIP code: 28451)*
We moved to Brunswick County a few weeks after the initial announcement regarding GenX in our drinking water. We would not have moved here if we had known about this early enough! We were fortunate enough to be able to spend over $3000 of our own money to treat water that already is priced 3x higher that what we were paying in NY for very clean water. It is INEXCUSABLE that Chemours has been allowed to continue operating, they should be shut down immediately, and not allowed to operate until they prove that they are not polluting our environment.

824. Russell Reid (*ZIP code: 28451)*

825. Rachel Schroeder (*ZIP code: 28403)*

826. rodney McCoy (*ZIP code: 28451)*

827. R Peiffer (*ZIP code: 28451)*

828. Robert Taylor (*ZIP code: 34481)*
Clean it up now!

829. William Belke (*ZIP code: 28451)*

830. Darlene Robey (*ZIP code: 28451)*

831. Robert Martin (*ZIP code: 28451)*

832. Carol Roberts (*ZIP code: 28451)*

833. Robin Barrington (*ZIP code: 28479)*

834. Alan Etkin (*ZIP code: 28451)*
Chemours' actions are inexcusable! We have already invested in an under sink system for our drinking water and need to buy another system for our refrigerator water and ice dispensers. We were not aware of this situation when we purchased our home! Chemours should pay for the accommodations we have to make to our homes, past, present and future as a result of their negligence!

835. Mary Roland (*ZIP code: 28451)*

836. Ronni Dunmire (*ZIP code: 28451)*
Help us have clean,healthy water !

837. Susan Roscher (*ZIP code: 28451)*
It is outrageous that Brunswick, New Hanover and Pender counties are not inluded in the relief fund by Chemours/Dupont. Clean water is vitally important to our health, well-being and for our children and future generations. Please act now!!

838. Susan Rosenberg (*ZIP code: 28451)*

839. Roxanne Tart (*ZIP code: 28451)*
We've installed a reverse osmosis system and there's so much wasted water. That's money down the drain....literally! This company knew they were poisoning the water for years and should have to pay to clean it up and for the burden too every resident affected.

840. randy glenn (*ZIP code: 28451)*

841. Ruth Syre (*ZIP code: 28412)*

842. Rushell Bongiorno (*ZIP code: 28403)*

843. russell larock (*ZIP code: 28451)*
clean water would nice

844. Russell Croake (*ZIP code: 28451)*
How can you keep poisoning the people that work for you and their neighbors, children and Wildlife. You have no heart. Corporate greed and paying off the politicians.

845. Rylee Sherwood (*ZIP code: 28405)*

846. STEPHEN Malusa (*ZIP code: 11743)*

847. Laurie Hodgson (*ZIP code: 28451)*

848. Stephanie Marulli (*ZIP code: 28451)*
EVERYONE deserves to drink pure, clean water. DuPont/Chemours needs to immediately stop

dumping contaminated water into nearby rivers and streams and be made to remediate all contaminated soils in these areas, no matter what the cost. In addition, DuPont/Chemours should install whole house reverse osmosis water filtration systems, free of charge, to all dwellings that receive their water from the Cape Fear River and any other source contaminated by this company. DuPont/Chemours would also be responsible for maintaining these filtration systems and providing homeowners/renters with additional replacement filters as needed.

849. Sharon Lerner (*ZIP code: 28451)*

850. Sally Buchanan (*ZIP code: 28465)*
The old expression "you break it, you buy it" should apply. Chemours ruined our drinking water, they should pay to give us back healthy water.

851. Susan Moore (*ZIP code: 28451)*
Thank you for working so hard on this issue.

852. Sara Messer (*ZIP code: 28412)*

853. Sandie Bateman (*ZIP code: 28451)*

854. Sandra Mcintosh (*ZIP code: 28312)*
Our waters are polluted. Then you drop off water and leave in our yards that in 100 degree weather are polluted by the plastic. I have showered in this water for 20 years. An under the sink fix (if it will fit under your sink) is not the answer for me!

855. Steven Brodhead (*ZIP code: 28451)*

856. Sheila Burdick (*ZIP code: 28451)*

857. Sherri Schultz (*ZIP code: 28451)*
Sherri Schultz

858. Sara Schulz (*ZIP code: 28451)*

859. Sarah Murphy (*ZIP code: 08840)*

860. Sue VanNote (*ZIP code: 28405)*

861. Steven Dalton (*ZIP code: 28479)*

862. Sallie Minnich (*ZIP code: 28451)*

863. Tamara Walker (*ZIP code: 28409)*

864. Sean Kiernan (*ZIP code: 28451)*

865. Shannon Gentry (*ZIP code: 28405)*

866. laura niewold (*ZIP code: 28451)*

867. Carla Jacobs (*ZIP code: 28479)*

868. Sarah Wall (*ZIP code: 27614)*

869. Susan Zimmer (*ZIP code: 28451)*

870. Sharon Fay (*ZIP code: 28449)*

871. Susan Fenzl (*ZIP code: 28468)*

872. Sheilla Figgins (*ZIP code: 28479)*
They should have to pay for all the bottled water I have had to purchase over the past 8 years. Also, they should have to pay for the 2 precious dogs I lost to cancer 2 years apart! I lost a Black Lab in 2014 and a Standard Poodle in 2016.

873. Stephen Foote (*ZIP code: 28451)*

874. Steve & Kathy Frankel (*ZIP code: 28411)*

875. Steve Harrison (*ZIP code: 28451)*

876. Sharon Pate-Batts (*ZIP code: 28443)*

877. shaun mitchell (*ZIP code: 28401)*
no one on the planet should allow our water to be polluted. make the polluters pay.

878. Shawn Streeter (*ZIP code: 28479)*

879. Shayne Escher (*ZIP code: 28479)*

880. Jennifer Sheargold (*ZIP code: 28412)*

881. Stephen Sheargold, Ph.D. (*ZIP code: 28412)*

882. Shelley Tucker (*ZIP code: 28451)*

883. Sheri Plotkin (*ZIP code: 28451-6032)*

884. Debbie Manes (*ZIP code: 28451)*
I have to buy water from a water delivery company just to drink the water because of all the contaminated
water from Chemours/DuPont in the Cape Fear River. Please take care of water and quit polluting the river.

885. Shirley Dietrich (*ZIP code: 28391)*

886. Samuel Shores (*ZIP code: 28407)*

887. WILLIAM TAYLOR (*ZIP code: 28451)*

888. Manny Mayfield (*ZIP code: 28348)*

889. Susan Roth (*ZIP code: 28451)*

890. Sandra Core (*ZIP code: 28461)*

891. Steven White (*ZIP code: 28451)*

892. Lance Edwards (*ZIP code: 28451)*

893. Sara Hagan (*ZIP code: 28451)*

894. Samantha Smith (*ZIP code: 28401)*

895. Susannah Lukens (*ZIP code: 28401)*

896. Sandra navarro (*ZIP code: 28405)*

897. Steven Ronan (*ZIP code: 28412)*

898. Steve Odee (*ZIP code: 28412)*

899. An anonymous signer (*ZIP code: 28461)*

900. An anonymous signer (*ZIP code: 28461)*

901. Timothy White (*ZIP code: 28411)*

902. Susan Owens (*ZIP code: 28451)*

903. mark spinner (*ZIP code: 28451)*

Are they ever going to stop polluting and are we ever going to do anything about it

904. Carol Huber (*ZIP code: 28451)*
This should not be an issue. Everyone is entited to safe, clean water. Have a conscious, morals and ethics to each other. That's all.

905. Candy Adams (*ZIP code: 28451)*

906. Spring Harkins (*ZIP code: 28470-5626)*

907. Patricia Spuhler (*ZIP code: 28451)*

908. Susan Sabatini (*ZIP code: 28412)*

909. Susan Catania (*ZIP code: 28451)*

910. Stacia Welborn (*ZIP code: 28403)*
Clean water should be a right for everyone and you have the ability to make that happen. Please do what you know is right.

911. Maria Henderson (*ZIP code: 28306)*

912. Rose St. Clair (*ZIP code: 28403)*

913. Stel Bailey (*ZIP code: 32927)*

914. Stephanie Tucker (*ZIP code: 28412)*

915. Stephani Garrett (*ZIP code: 28409)*
Ridiculous that it is taking this long and more is being found!

916. Steve Hosmer (*ZIP code: 28451)*

917. Stephen Lane (*ZIP code: 28451)*

918. Kenneth Cain (*ZIP code: 28306)*

919. Kory Stokes (*ZIP code: 28348)*

920. Holly Mcgee (*ZIP code: 28489)*
Holly and James McGee

921. Melissa Streeter (*ZIP code: 28479)*

922. Corinna Struckholz (*ZIP code: 28412)*
Enough is enough - everyone deserves clean water

923. Stuart Werner (*ZIP code: 28411)*
Thanks

924. Sue-Ann Rush (*ZIP code: 28451)*
Please help us downstream! We have years of residuals to deal with, which will take longer than the immediate surroundings of the Chemours Plant. Downstream residents need immediate financial assistance as well to eliminated additional costs to protect themselves against the contaminants in their drinking water. Please consider helping all impacted communities, Immediately.

925. Suzan Federman (*ZIP code: 28451)*

926. Sue Wiblitzhouser (*ZIP code: 28451)*

927. Sue Hayes (*ZIP code: 28412)*

928. Amanda Hynes (*ZIP code: 29588)*

929. Starr Watson (*ZIP code: 28412)*
Shut Chemours down. They have proven repeatedly that they can not be trusted. Stop letting them poison us!

930. Allen Taylor (*ZIP code: 28144)*

931. Dave Hutchens (*ZIP code: 28112)*

932. Jonathan Ware (*ZIP code: 28480)*

933. Susan Utsey (*ZIP code: 28209)*

934. Susan Denston (*ZIP code: 28451)*

935. Susan Hooton (*ZIP code: 28411)*
It is essential that drinking water be protected. Chemours and DuPont have carelessly polluted our water and must make restitution to all the communities they have damaged. Further, they must be penalized so that they will not profit from polluting.

936. Susan KeysHolman (*ZIP code: 28405)*

937. Susan Sullivan (*ZIP code: 28479)*

938. Suzy Tenenbaum (*ZIP code: 28451)*

939. Suzanne Civale (*ZIP code: 28451)*

940. Sandra Verruso (*ZIP code: 28479)*

941. Tammy Parrella (*ZIP code: 28451)*

942. Tammy Moore (*ZIP code: 28479)*

943. Tanner Dodson (*ZIP code: 28411)*

944. Tanner Brittan (*ZIP code: 28411)*
Hold chemours accountable!!

945. Tanya DeLeon (*ZIP code: 28479)*

946. Tara Ferguson (*ZIP code: 28405)*

947. Tara Colligan (*ZIP code: 28451)*

948. Trish Clark (*ZIP code: 28412)*

949. Thomas Wetherington (*ZIP code: 28479)*

950. Susan Adie (*ZIP code: 28451)*
DuPont needs to take responsibility and pay up!!

951. Zachary Terault (*ZIP code: 28412)*

952. Teresa DiGirolomo (*ZIP code: 28451)*

953. Teresa Prevatte (*ZIP code: 28449)*

954. teresa mann (*ZIP code: 28403)*

955. theresa abruzzo (*ZIP code: 288451451)*

956. Teresa Watkins (*ZIP code: 28451)*

957. Terry Rotas (*ZIP code: 28451)*

958. Christopher Thatcher (*ZIP code: 80306)*

959. Sharon Cox (*ZIP code: 28451)*

960. Sandy Perotto (*ZIP code: 28401)*
Stop. Please just stop putting profits before people. Until then I will "hope and pray" that Chemours higher ups, their families, their children and those in government allowing this to happen, also suffer as much as the people effected buy their greed.
May your children be afflicted with ill bodies and suffer like ours.

961. Theresa Tate (*ZIP code: 28451)*

962. Debbie Sharpe (*ZIP code: 28387)*
I lived in Wilmington for 20 years and drank the water that poisoned me with breast cancer metastasized to the bone. You allow Chemours to continue to have a permit. You are tasked with protecting the citizens of the State of North Carolina. Please do so. Debbie Jocelyn Sharpe

963. Michael Gaghan (*ZIP code: 28451)*

964. Robin Soderena (*ZIP code: 28405)*

965. Michael Sileno (*ZIP code: 27408)*

966. Kyle Thomas (*ZIP code: 28306)*

967. Tom Laakmann (*ZIP code: 28409)*

968. Carolyn Smith (*ZIP code: 28479)*
Something needs to be done to make our drinking water safe!

969. Tiffany Toler (*ZIP code: 28312)*
Our water, soil, and air have been ruined by chemours / Dupont /Dupont De Nemours. Anyone wonder what else they've been dumping directly into the river from the Monsnato plant on Cedar Creek Rd?

970. Tina LUDENA-SASS (*ZIP code: 28451)*

971. Jim Tiner (*ZIP code: 28306)*

972. Tikeysha Tomlin (*ZIP code: 28451)*

973. Tom Kennedy (*ZIP code: 28409)*

974. Toby Davignon (*ZIP code: 28479)*

975. Patricia McDaniel (*ZIP code: 28451)*

We demand clean safe drinking water!

976. THERESA PATEREK (*ZIP code: 28451)*

977. Taylor Smith (*ZIP code: 28451)*

978. Tom Rini (*ZIP code: 28401)*

979. Tom Brimberry (*ZIP code: 28412)*

980. Thomas Geery (*ZIP code: 28451)*
We should be compensated for having to install special filtration systems on our public drinking water!

981. steve matteson (*ZIP code: 28451)*

982. Tracy Baker (*ZIP code: 28451)*

983. Tracy Tritten (*ZIP code: 28401)*

984. Henry Lanier (*ZIP code: 28405)*

985. Terry Volpe (*ZIP code: 28451)*

986. Theodore Janeczko (*ZIP code: 28451)*
Dupont is slowly killing us. They must be stopped!!
Aspirin had to be approved by the FDA, who approved the PFSAs? The system is not working.....at least for the simple homeowner. Please help us!!

987. Joseph Baldwin (*ZIP code: 37082)*
Its clearly obvious that the community that works for them act like they cant stop working there cause its some "livelyhood" i live near that community

988. HARRY STANHOPE (*ZIP code: 28451)*

989. Valerie Akerhielm (*ZIP code: 28429)*

990. Virginia Radcliffe (*ZIP code: 28411)*

991. Cassandra Lintz (*ZIP code: 28401)*
It’s about time to make these corporations pay for the damage they inflict on our communities.

Do the right thing.

992. Victoria Crouse (*ZIP code: 27604)*

993. Virginia Holman (*ZIP code: 28428)*

994. Veronica Munro (*ZIP code: 28451)*

995. Ginny Wrightfrierson (*ZIP code: 28401)*
They must stop poisoning us. Shut them down and get them out of here

996. Denise Quattrucci (*ZIP code: 28479)*

997. Nancy Walker (*ZIP code: 28405)*
CFPUA customers should not have to pay for the filtration system to be constructed. That is clearly the full responsibility of Dupont/Chemours. Those chemicals are in our drinking, cooking and bath water that we are paying for and may pay for in the future with medical bills related to the pollutants in the water.

998. Constance Broughton (*ZIP code: 28451)*

999. Wayne Fluke (*ZIP code: 28451)*

1000. Paul Antsen (*ZIP code: 28451)*

1001. Andrea Baker (*ZIP code: 28451)*

1002. Wanda Ingram (*ZIP code: 28479)*

1003. Melissa Foley (*ZIP code: 28412)*
CLOSE CHEMOURS DOWN!!!

1004. Wendy Carroll (*ZIP code: 28479)*
I buy 8 gallons of purified water weekly! I also Buy 2 cases buy 20 oz Waters weekly! Just so I don’t have to ingest tainted tap water.

1005. Katrina White (*ZIP code: 28451)*
We demand clean water!

1006. Representative Billy Richardson (*ZIP code: 27601-1096)*
NC House District 44 (Cumberland Co./Fayetteville)

1007. William Kramer (*ZIP code: 28451-9486)*

1008. Ashley Wildrick (*ZIP code: 28409)*

1009. Angie Wodrazka (*ZIP code: 28479)*

1010. Francis Wodrazka (*ZIP code: 28479)*

1011. Woody Johnson (*ZIP code: 28461)*

1012. Michael Wroblewski (*ZIP code: 28451)*

1013. Walt Sparrow-Hood (*ZIP code: 28451)*

1014. WENDY JOHNSON (*ZIP code: 27358)*

1015. Wanda Wooten (*ZIP code: 28403)*

1016. Sonia Benitez (*ZIP code: 28451)*

1017. Yvonne Lane (*ZIP code: 28348)*
We need real help with our serious water problem. If the people who created this problem had to drink this water, something would be done !

1018. Brittany LaValley (*ZIP code: 28403)*
Chemours/DuPont needs to help the community and provide a way for all us of to have clean water. Not only can they do that through more safely managing their polluting, but also through funding a community wide Reverse Osmosis systems in all affected counties and restitution for those of us who have been forced to buy bottles water for almost 4 years now. Enough is enough. They are a multi-million dollar corporation and they need to be held responsible.

1019. Sam Rankin (*ZIP code: 28403)*
It is inexcusable that individuals must pay at their homes and with tax dollars for chemours' pollution. And that's not including paying doctors for health problems. Make chemours pay for all wastewater treatment plant upgrades, and pay a fine to individual residents for the expenses they have incurred.

1020. Yvonne Moody (*ZIP code: 28461)*

1021. Linda Zeliznik (*ZIP code: 28451)*
Please help us help our children. They need clean water to survive this earth.

1022. Nukhet Ucin (*ZIP code: 28401)*
Do not play with our life

1023. Heather Caveny (*ZIP code: 28401)*

1024. Joan L Zito (*ZIP code: 28451)*

1025. Jay Zellin (*ZIP code: 28451)*

1026. Kat Malec (*ZIP code: 28405)*
They need to pay!!! My animals are dead and my water isn’t safe!!

From: Tom Vitaglione
To: comments.chemours
Subject: [External] Comments on Chemours Addendum
Date: Thursday, September 17, 2020 8:52:18 AM
Attachments: DEQ letter 9.17.2020.docx

DEQ Assistant Secretary

Please accept the attached comments regarding the Chemours addendum.

Stay well,
Tom vitaglione
Senoir Fellow
NC Child
919-376-7949

NC Child
The Voice for North Carolina's Children

3101 Poplarwood Court
Suite 300
Raleigh, NC 27604-1044

919.834.6623
ncchild.org

September 17, 2020

NC Department of Environmental Quality
Assistant Secretary's Office
217 West Jones Street
Raleigh, NC 27603

Re: Chemours Public Comments

NC Child appreciates the opportunity to provide comments on the 2019 consent order for the cleanup of PFAS pollution coming from Chemours' Fayetteville Works Facility.

NC Child is a non-profit, non-partisan organization advocating for local, state, and federal policies that promote and protect the health and well-being of children and their families.
Children are particularly vulnerable to environmental contaminants as a result of their smaller body masses and lower blood volumes than adults. We therefore applaud the consent agreement, as it will result in positive impacts on child health. NC Child requests that DEQ sign the agreement and present it to the Bladen County Superior Court for approval.

Combined with the original consent order, the addendum will mean that each of the major pathways of contamination (air, process water discharge, Old Outfall 002, groundwater discharge, seeps, and stormwater) must be reduced by at least 99%. Contaminated water from Old Outfall 002, extracted groundwater, and stormwater will all be treated with technology that can reduce PFAS to levels below detection limits. As a result, residents – and especially children – will be protected from PFAS contamination.

We thank DEQ for all its efforts to protect North Carolina's environment, and particularly its youngest and most vulnerable residents.

Sincerely,

Tom Vitaglione
Senior Fellow

From: Claudia Stack
To: comments.chemours
Subject: [External] Comment re: Chemours/PFAs
Date: Wednesday, September 16, 2020 6:47:35 PM

Hello,
I am emailing to express my outrage that a private company, Chemours, has been allowed to contaminate water in southeastern NC for years and has profited from their ability to add toxins to our drinking water and soil.

The discharge of PFAs continued even after the state found out, and we have untold numbers of health effects in the Cape Fear Region because of Chemours' greed and the way that the state and the EPA allowed the situation to continue.

I have lived in Pender County, NC for 27 years. I was just diagnosed with breast cancer. Many friends of mine in the region have experienced cancers and reproductive problems. We have a right to know whether there is an unusually high number of health problems in the region related to the toxins released into the water.

I think that Chemours should be mandated to study the health impacts of the PFAs it has released, provide safe, clean drinking water to all persons who currently get their drinking water from the Cape Fear River and groundwater sources, and set up a compensation fund for persons who experience health impacts.

Sincerely,
Claudia Stack
98 Casha Road
Rocky Point NC 28457
(910) 264-4469

From: Stephanie Schweickert
To: comments.chemours
Subject: [External] RE: Chemours Public Comments
Date: Wednesday, September 16, 2020 4:50:52 PM
Attachments: NC Conservation Network Chemours Public Comment Petition Signatures.pdf

Ms. Martin,

Please see the attached petition signed by over 1,141 North Carolina residents, which urges DEQ to support the addendum to the Chemours Consent Decree. This addendum will largely eliminate PFAS pollution into the Cape Fear River and protect downstream communities. However, contaminated soil and groundwater remain near the facility, putting nearby families and community members at risk. In addition to accepting the addendum, we urge DEQ to require Chemours to address the soil and groundwater contamination in a strong Corrective Action Plan.

Thank you for your time and please let me know if you have any questions or need additional information. Your attention to this matter is appreciated.

Sincerely,

Stephanie Bishop Schweickert, Senior Campaign Organizer
NC Conservation Network
stephanie@ncconservationnetwork.org
919.857.4699 x103 (p)

Like us on Facebook: http://www.facebook.com/NCConservationNetwork
Follow us on Twitter: @NCConservation
Follow us on Instagram: @ncconservationnetwork

North Carolina Conservation Network

234 Fayetteville Street
5th Floor
Raleigh, NC 27601
919.857.4699

September 16, 2020

N.C. Department of Environmental Quality
ATTN: Sharon Martin
217 West Jones St.
Raleigh, NC 27603

Re: Chemours Public Comments

Dear Ms. Martin,

Please see the attached petition signed by over 1,141 North Carolina residents, which urges DEQ to support the addendum to the Chemours Consent Decree. This addendum will largely eliminate PFAS pollution into the Cape Fear River and protect downstream communities. However, contaminated soil and groundwater remain near the facility, putting nearby families and community members at risk. In addition to accepting the addendum, we urge DEQ to require Chemours to address the soil and groundwater contamination in a strong Corrective Action Plan.

Thank you for your time and please let me know if you have any questions or need additional information. Your attention to this matter is appreciated.

Sincerely,

Stephanie Schweickert

Stephanie Schweickert, Senior Campaign Organizer
NC Conservation Network
234 Fayetteville Street, 5th Floor
Raleigh, NC 27601
919-857-4699

Dear Department of Environmental Quality,

We the undersigned, support the addendum to the Chemours Consent Decree, which will largely eliminate PFAS pollution into the Cape Fear River and protect downstream communities. However, contaminated soil and groundwater remain near the facility, putting nearby families and community members at risk. In addition to accepting the addendum, please address soil and groundwater contamination in a strong Corrective Action Plan.

Thank you.

Sincerely,

	First Name	Last Name	Street Address Line 1	City	State	Zip Code
1.	David	Barnes	310 Colony Ave S	Ahoskie	NC	27910
2.	Tripp	Carter	Apt 28 153 Wellingham Ave	Greenville	NC	27834
3.	Michael	Zyvoloski Sr.	514 Blair Shores Rd	Roper	NC	27970
4.	Ken	Lautzenheiser	310 E Baker St	Tarboro	NC	27886-3806
5.	Hunter	Roberson	2180 Valley View Dr	Henderson	NC	27536-3557
6.	Kenneth A.	Morris	2351 Staton Mill Rd	Bethel	NC	27812-9633
7.	Jarrett	Whelan	4008 Colony Woods Dr	Greenville	NC	27834-1082
8.	Tracie	Creta	403 Kempton Drive	Greenville	NC	27834
9.	John	Hinnant	503 Mount Vernon Dr Nw	Wilson	NC	27893-2227
10.	Carole	Reynolds	3611 Crosswinds Dr	Stem	NC	27581-9244
11.	Diane	Reed	7533 Shep Royster Rd	Oxford	NC	27565
12.	Holly	Potthoff	306 N Country Club Dr	Oxford	NC	27565-2820
13.	Lawrence	Adrian	101 Kaitlin Dr	Durham	NC	27713
14.	Clarence Ray	Jones	2613 E Weaver St	Durham	NC	27707-3055
15.	Joel	Herndon	3433 Sheridan Dr	Durham	NC	27707
16.	Kathleen	Malley	318 Brandermill Dr	Durham	NC	27713
17.	Kenneth	Crews	P. O. Box 1062	Durham	NC	27702
18.	Keval	Khalsa	1215 Carroll St	Durham	NC	27707-1311
19.	Louis	Desantis	1118 Hooper Place	Durham	NC	27703
20.	Peter	Schubert	927 Bluestone Rd	Durham	NC	27713
21.	Casey	Therrien	614 Glen Hollow Dr	Durham	NC	27705-5675
22.	Betsy	Bickel	117 W Trinity Ave	Durham	NC	27701
23.	Connie	Raper	2614 Woodmont Dr	Durham	NC	27705-2760
24.	Jude	Casseday	6 Bair Cir	Durham	NC	27704-1552
25.	Hiroshi	Mayomi	1101 Fern St	Durham	NC	27701
26.	Magaretha	Herman	2419 Highland Ave	Durham	NC	27704-4328
27.	Anthony	Madejczyk	2705 Highland Ave	Durham	NC	27704-4307
28.	Angela	Vieth	3009 Bexley Ave	Durham	NC	27707-2843
29.	Diane	Jackson	123 Applecross Ct	Durham	NC	27713-9333
30.	John	Wiles	5205 Langford Ter	Durham	NC	27713
31.	Claudia	Kaplan	4911 Victoria Drive	Durham	NC	27713
32.	Edena	Thomas	6 Sabre Ct	Durham	NC	27713-7114
33.	Amy	Markin	4909 Harwood Ct	Durham	NC	27713-8103

34.	Anthony	Varvoutis	4754 Ridgetop Dr	Morgantown	WV	26508
35.	Vicky	Brandt	3318 Coachmans Way	Durham	NC	27705
36.	Carol	Rist	1 Barratts Chapel Court	Durham	NC	27705
37.	Claiborne	Clark	4200 Livingstone Pl	Durham	NC	27707-5515
38.	Julia Elizabeth	Hoggard	3740 Swarthmore Road	Durham	NC	27707
39.	Charles	Weil Phd Pg	4125 Farrington Road	Durham	NC	27707
40.	Ellen	Bacon	4201 Swarthmore Rd	Durham	NC	27707-5389
41.	Gary	Gartner	6 Scotland Pl	Durham	NC	27705
42.	Jayne	Boyer	4316 Thetford Rd	Durham	NC	27707-5700
43.	Jeffrey	Nicolaisen	2528 Perkins Rd	Durham	NC	27705-1020
44.	Joy	Metelits	411 Cedar Club Cir	Chapel Hill	NC	27517
45.	Candido	Calciolari	622 Morreene Rd	Durham	NC	27705
46.	Maria	Salgado	2123 Fountain Ridge Rd	Chapel Hill	NC	27517-7925
47.	Marian	Dessent	10 Macgregor Ct	Durham	NC	27705
48.	Susan	Saenger	6 Scotland Pl	Durham	NC	27705
49.	Elizabeth	Norman	1013 Demerius St	Durham	NC	27701
50.	John	Compton	404 W Knox St	Durham	NC	27701
51.	Tasha	Pate	923 N Buchanan Blvd	Durham	NC	27701-1543
52.	Polly	Harris	118 West Trinity Av	Durham	NC	27701
53.	Sandra	Ackerman	1025 Dacian Avenue	Durham	NC	27701
54.	Beth	Owls Daughter	1105 Trail End Rd	Durham	NC	27712
55.	Becky	Hayward	316 November Dr # Dirham	Durham	NC	27712-2441
56.	Judy	Teague	2416 Dawn Trl	Durham	NC	27712-2431
57.	Rebecca	Enfiedjian	2706 Saddle Dr	Durham	NC	27712-1824
58.	S L	Jones	-	Durham	NC	27702
59.	Jan E.	Hicks	1324 Gay St	Rocky Mount	NC	27804-4312
60.	Lisa	Lewis	112 Carrington Dr	Garner	NC	27529
61.	Douglas	Van Luvender	606 Blazing Star Ct	Garner	NC	27529
62.	Lynne	C.	6032 Kentworth Dr	Holly Springs	NC	27540
63.	Della	Fitz-Gerald	5146 Quaker Rd	Wilson	NC	27893-8383
64.	Lesia	Mills	Po Box 1183	Clayton	NC	27528
65.	Jessica	Motta	22 Yadkin St	Clayton	NC	27520-3057
66.	Patrice	Hubert	114 Michael Way	Clayton	NC	27520
67.	Tricia	Oakley	101 Greenwood Cir	Smithfield	NC	27577-3631

68.	Andrea	Crook	200 Kelly Road	Sanford	NC	27332
69.	Amanda	Misner	1773 Mcneill Hobbs Rd	Bunnlevel	NC	28323-8977
70.	Fawn	Barker	45 William Bethune Court	Linden	NC	28356
71.	Martha	Smith	510 W Harnett St	Dunn	NC	28334
72.	Nadine	Murray	171 Orchard Falls Dr	Spring Lake	NC	28390-7174
73.	Elisa	Smith	3316 Broughton Rd	Wendell	NC	27591-9753
74.	Richard	Lolley	6828 Woodtrace Dr	Wendell	NC	27591-7025
75.	Jacqueline	Kosnik	1208 Amber Acres Ln	Knightdale	NC	27545-8901
76.	Betty	Lazo	2803 Falls River Ave	Raleigh	NC	27614-7419
77.	Carol	Pelosi	1255 S Main St	Wake Forest	NC	27587-9282
78.	John	Godfrey	709 Montville Ct	Wake Forest	NC	27587
79.	Rosemary	Somich	345 Dimock Way	Raleigh	NC	27615
80.	Scott	Vandiver	2812 Crystal Oaks Ln	Raleigh	NC	27614-9871
81.	Thomas	Cadwallader	404 Dimock Way	Wake Forest	NC	27587
82.	John	Franklin	11504 Hyde Pl	Raleigh	NC	27614
83.	Jackie	Franklin	11504 Hyde Place	Raleigh	NC	27614
84.	Jere	Snyder	6805 Laurdane Rd	Raleigh	NC	27613-5938
85.	Julie	Brooks	1196 Old Still Way	Wake Forest	NC	27587-5904
86.	Lisa	Lambert	1136 Mauldin Cir	Wake Forest	NC	27587-4420
87.	Sean	Dempsey	2327 Mount Vernon Church Rd	Raleigh	NC	27614-9220
88.	Todd	Fields	2413 Pleasant Union Church Rd	Raleigh	NC	27614-7111
89.	Peter	Van Dorsten	7301 Rainwater Rd	Raleigh	NC	27615-5460
90.	Alex	East	207 Marvista Ct	Cary	NC	27518-9197
91.	Barry	Rosett	2419 Tiltonshire Ln	Apex	NC	27539
92.	David	Biesack	3671 Echo Farms Blvd	Wilmington	NC	28412
93.	Farshid	Bondar	128 Castlewood Dr	Cary	NC	27511
94.	Jane Ann	Hughes	7760 Netherlands Dr	Raleigh	NC	27606
95.	Richard	Demarse	100 Schaffer Close	Cary	NC	27518
96.	Robert	Peek	7328 Bedford Ridge Dr	Apex	NC	27539-4151
97.	Jill	Shank	5405 Leopards Bane Ct	Holly Springs	NC	27540
98.	Lindsi	Hines	630 Aiken Pkwy	Fuquay Varina	NC	27526-2064
99.	Miriam	Youngquist-Thurow	6209 Thurlow Ct	Holly Springs	NC	27540
100.	Nel	Hornaday	1006 Newington Way	Apex	NC	27502-4360
101.	Monica	Barriga	1300 Albertson Pl	Apex	NC	27502-6754

102.	Stephen	Boletchek	1106 Elbury Dr	Apex	NC	27502-2250
103.	Julie	Gupton	370 Brewer Rd	Louisburg	NC	27549-8285
104.	Mary Alden	Hanson	7412 Rocky Ridge Rd.	Wake Forest	NC	27587
105.	Philip	Davis	2653 Huntsman Trl	Zebulon	NC	27597-8514
106.	William	Blaine	1209 Litchborough Way	Wake Forest	NC	27587
107.	John	Kinsella	6109 Hollow View Ct.	Fuquay-Varina	NC	27526
108.	Amy	Popp	6905 Pinnacle Ridge Rd	Raleigh	NC	27603-9126
109.	Rachel	Wendel	920 Open Field Dr	Garner	NC	27529
110.	Megan	Burns	1116 Durbin Way	Fuquay Varina	NC	27526-9352
111.	Panchito	Juarez	285 Loblolly Circle	Louisburg	NC	27549
112.	Jennifer	Symonds	110 Windy Hill Ct	Aydlett	NC	27916-9750
113.	Christine	Mills	278 Baxter Ln	Moyock	NC	27958-8613
114.	Mark	Mchugh	1127 Brumsey Ct	Corolla	NC	27927-9602
115.	Roxy	Darling	936 Waterlily Rd	Coinjock	NC	27923-9735
116.	Mary	Haubenreiser	118 S Academy St	Washington	NC	27889-5063
117.	Terri	Krebs	1001 Meadow Dr	Elizabeth City	NC	27909-9392
118.	Vannie	Simmons	805 Boston Ave	Washington	NC	27889-3483
119.	Jonathan	Cole	40305 Williams Rd	Avon	NC	29715-0064
120.	Cathy	Pescevich Kreplin	608 Harbour View Drive	Kill Devil Hills	NC	27948
121.	Greg	Hamby	1206 Harbor Ct	Kitty Hawk	NC	27949-4046
122.	Ginny	Nolan	3204 S Memorial Ave	Nags Head	NC	27959
123.	Amy	Adams	108 Camelot St	Washington	NC	27889
124.	Rosemary	Rawlins	2507 S Bridge Ln	Nags Head	NC	27959-9695
125.	Scott	Bradley	Po Box 402	Ocracoke	NC	27960
126.	Thomas	Warren	30 Quarterdeck	New Bern	NC	28562-3805
127.	William	Cresswell	18 Sassafrass Loop	Arapahoe	NC	28510-8503
128.	Ellen	Beery	905 Osprey Ct	New Bern	NC	28560-8951
129.	Jim	Privette	Po Box 251	Oriental	NC	28571
130.	Deborah	Fox	102 Balboa Court	New Bern	NC	28560
131.	Rollin	Morse	3701 Cerise Circle	New Bern	NC	28562
132.	Terry	Halpern	306 Whittaker Pt Rd	Oriental	NC	28571
133.	Diane	Hannum	1250 Pine Valley Dr	New Bern	NC	28562-2938
134.	April	Hardee	7528 Sound Dr	Emerald Isle	NC	28594
135.	Robert	Austin	135 Williston Creek Road	Williston	NC	28579

136.	Donald	Long	415 Old Swansboro Rd	Newport	NC	28570
137.	Henry	Nehring	393 Norris Landing Rd	Swansboro	NC	28584-7498
138.	Karena	Bond	5936 Shady Grove Cir	Raleigh	NC	27609
139.	Mary	Forsyth	650 Cedar Point Blvd	Cedar Point	NC	28584
140.	Patricia	Rister	323 Winding Woods Way	Beaufort	NC	28516
141.	Teresa	Rice	105 S 28Th St	Morehead City	NC	28557
142.	Barbara	Conrad	6212 N Highland Blvd	Grifton	NC	28530
143.	Brittny	Callender	2159 Wolf Ln	Kinston	NC	28501-9702
144.	Brenda	Johnson	18 Arrowhead Dr	Hubert	NC	28539-4102
145.	Marion	Cowan	1303 Blue Creek Rd.	Jacksonville	NC	28540
146.	Susan	Ballard	1226 Nrir	Ntb	NC	28460
147.	Vickie	Cunningham	237 Marsh Haven Dr	Sneads Ferry	NC	28460
148.	Michelle	Smith	405 Silva Cv	Richlands	NC	28574-6398
149.	Senovia	Vazquez		Hubert	NC	28539
150.	Rachel	Roper	754Bgatewood Dr.	Winterville	NC	28590
151.	Julie	Papp	109 Oakmont Dr. #51	Greenville	NC	27858-5954
152.	Lonnie	Foreman	723 Corbett St	Winterville	NC	28590-8661
153.	Susan	Howell	513 Plymouth Dr.	Greenville	NC	27858
154.	Susan	Snellings	1427 Saddlewood Dr	Greenville	NC	27858-8298
155.	Jessica	Robinson	1116 S State St	Raleigh	NC	27601-2056
156.	Jason	Whitham	1510 Joe Louis Ave	Raleigh	NC	27610
157.	Clifton	Lavenhouse	2539 Crescent Forest Dr	Raleigh	NC	27610-2970
158.	Stephanie	Schweickert	1125 Stoneferry Lane	Raleigh	NC	27606
159.	George Ann	Ricks	1001 Barmkin Pl	Knightdale	NC	27545
160.	Amy	Cox	509 Huron Rd	Raleigh	NC	27610
161.	Andrea	Osborn	111 North King Charles Road	Raleigh	NC	27610
162.	Brandon	Whitesell	408 Culpepper Ln	Raleigh	NC	27610
163.	Brittany	Iery	1116 Holburn Pl	Raleigh	NC	27610
164.	Chris	Conley	4800 Walden Ct Apt B	Raleigh	NC	27604
165.	Thomas	Rudd	5413 Kissimmee Ln	Raleigh	NC	27616-3246
166.	Sterling	Bowen	109 N King Charles Rd	Raleigh	NC	27610
167.	Carolyn	Avera	5505 Buffaloe Rd	Raleigh	NC	27616-6011
168.	George	Lloyd	1007 Crabtree Ct	Knightdale	NC	27545-9294
169.	Anna	Bryant	200 Woods Ream Dr	Raleigh	NC	27615-7228

170.	Angie	Brummitt	7508 Se Tibbetts St.	Portland	OR	97206
171.	Joe	Bearden	1809 Lakepark Dr	Raleigh	NC	27612
172.	Janis	Ramquist	2208 Oxford Hills Dr	Raleigh	NC	27608
173.	Kathryn	Pritchett	6513 Thetford Ct	Raleigh	NC	27615-6332
174.	Joan	Dulberg	555 Pine Ridge Place	Raleigh	NC	27609
175.	Vickie	Penninger	711 Kimbrough St	Raleigh	NC	27608-2723
176.	James	Marsh	6805 Grimaldi Ct	Raleigh	NC	27612
177.	Anne	Tate	1207 Duplin Rd	Raleigh	NC	27607-3718
178.	Dara	Finkelstein	2509 Harptree Ct	Raleigh	NC	27613-1606
179.	Doris	Whitfield	109 Renwick Ct	Raleigh	NC	27615-2946
180.	Emmy	Moore	2110 St. Mary'S Street	Raleigh	NC	27608
181.	Elizabeth	Kearse	2113 Oakcrest Ct	Raleigh	NC	27612
182.	Jean	Miani	4021 Converse Drive	Raleigh	NC	27609
183.	Cindy	Levey	8012 Clear Brook Dr	Raleigh	NC	27615
184.	James	Nutt	2631 Fairview Road	Raleigh	NC	27608
185.	Kevin	Bobal	6904 Ray Rd	Raleigh	NC	27613
186.	Keith	Meyer	8620 Windjammer Dr	Raleigh	NC	27615
187.	Peg	Gjertsen	3347 Ridgecrest Ct	Raleigh	NC	27607
188.	Thurman	Grove	3320 White Oak Road	Raleigh	NC	27609
189.	Timothy	Tew	407 Transylvania Ave	Raleigh	NC	27609-6953
190.	Trisha	Noonan	116 Northbrook Dr Apt 306	Raleigh	NC	27609-7079
191.	Cheryl	Mcgraw	1004 Braxton Ct	Raleigh	NC	27606
192.	Connie	Orander	1004 Wilshire Dr	Cary	NC	27511-3921
193.	Lori	Campbell	105 Woodgrove Ln	Cary	NC	27518
194.	Ivette	Griffin, Jr.	6431 Daybrook Cir Apt 301	Raleigh	NC	27606-2954
195.	Judy	Donders	313 Glenolden Court	Cary	NC	27513
196.	Karyn	Reid	115 Whispering Pines Ct	Cary	NC	27511-4059
197.	Kris	Black	204 Crystal Dr.	Broadway	NC	27505
198.	Lindsey	Jackson	1860 Scholar Cir	Raleigh	NC	27606-5187
199.	Lubana	Lanewala	5028 Simmons Branch Trail	Raleigh	NC	27606
200.	Lynda	Prediger	100 Summey Ct	Cary	NC	27513
201.	Wj	Richardson	3712 Bryn Mawr Ct	Raleigh	NC	27606
202.	Andreas	Batz	1007 Manchester Dr	Cary	NC	27511
203.	Margaret	Vaughn	818 Chatham Lane	Raleigh	NC	27610

204.	Anne	Kepplinger	2844 Wycliff	Raleigh	NC	27607-3035
205.	Audrey	Gastmeyer	3520 Bridgeton Park Dr	Raleigh	NC	27612-4151
206.	Barbara	Gerlach	2737 Rosedale Av.	Raleigh	NC	27607
207.	Rebecca	Burmester	2121 North Hills Dr Apt I	Raleigh	NC	27613
208.	Jeremy	Burnison	1216 Duffy Place	Raleigh	NC	27603
209.	Chris	Gay	7204 Ray Rd	Raleigh	NC	27613-3985
210.	Helen	Gray	1020 W Peace St Apt U8	Raleigh	NC	27605
211.	Doris	Bolt	3340 Harden Rd	Raleigh	NC	27607
212.	Kathleen	Mcquaid	802 Brooklyn St	Raleigh	NC	27605-1421
213.	Lynne	Walter Msw	3228 Glenridge Dr	Raleigh	NC	27604-2443
214.	Harrison	Marshall	504 Greenwood Circle	Cary	NC	27511
215.	Jason	Cashwell	314 Fairfield Ln	Cary	NC	27511-5408
216.	James	Grady	129 Sterlingdaire Dr	Cary	NC	27511-4384
217.	Leonard	Mole	1406 Laughridge Dr	Cary	NC	27511-5240
218.	Susane	Boukamel	200 Fox View Pl	Nc	NC	27511
219.	Olga	Bushel	207 Firetree Ln	Cary	NC	27513
220.	Stavros	Boinodiris	103 Lippershey Ct	Cary	NC	27513-5664
221.	Donald	Fuchs	4609 Wee Burn Trl	Raleigh	NC	27612
222.	Heather	Needham	4902 Carteret Dr	Raleigh	NC	27612-5714
223.	Barbara	Wilkus	5221 Old Powell Rd	Holly Springs	NC	27540
224.	Shirley	Ware-Gully	103 Bellshill Ct	Cary	NC	27513
225.	Deb	Carr	2007 Castleburg Dr	Apex	NC	27523-5154
226.	Karen	Ferguson	402 Greenwood Circle	Cary	NC	27511
227.	Susan	Edelstein	308 Heidinger Drive	Cary	NC	27511
228.	Rick	Savage	101 Bonner Ct	Cary	NC	27511
229.	Charlotte	Speltz	112 Altair Circle	Apex	NC	27502
230.	Laurel	Callis	1206 Wellstone Cir	Apex	NC	27502
231.	Toni	Chester	5606 Stone Point Ct	Granite Falls	NC	28630
232.	Frank	Moore	3301 Carolina Lily St	Cary	NC	27519-6710
233.	Jessica	Boggs Spellman	1816 Creek Oak Circle	Fuquay Varina	NC	27526
234.	Joseph Louis	Mazzitelli	7303 Calire Park Dr Apt 105	Durham	NC	27707
235.	Krissa	Johnson-Sotomayor	106 Spring Needle Court	Cary	NC	27513
236.	Kimberly	Hurtt	2712 Quail Point Dr	Raleigh	NC	27603-8926
237.	Evelyn	Hamilton	108 Emerald Cir	Durham	NC	27713-2413

238.	Carol	Young	5808 Williamsburg Way	Durham	NC	27713-2636
239.	Susan	Ricker	135 Montclair Cir	Durham	NC	27713
240.	E.L.	Flake	3500 Old Greensboro Rd	Chapel Hill	NC	27516-5898
241.	Jesse	Kaufmann	2304 Davis Rd	Hillsborough	NC	27278-7321
242.	Angela	Burnette	3726 Krystle Ct	Hillsborough	NC	27278
243.	Anne	Tooley	4402 Bradford Ridge Rd.	Efland	NC	27243
244.	Burwell	Ware	126 Kingston Drive	Chapel Hill	NC	27514-1630
245.	Carl	Shy	6626 Bradshaw Quarry Rd	Efland	NC	27243-9617
246.	Erin	Kimrey	1011 Bugle Ct	Chapel Hill	NC	27516-8765
247.	Eleanor	Kinnaird	750 Weaver Dairy Rd Apt 123	Chapel Hill	NC	27514-1439
248.	Janine	Tokarczyk	109 N Oakland Dr	Mebane	NC	27302-3301
249.	Jean	Obarr	750 Weaver Dairy Rd	Chapel Hill	NC	27514-1438
250.	Jim	Chambo	2914 Brightside Dr	Chapel Hill	NC	27516-9740
251.	Kaselehlia	Sielken	136 Kingston Dr	Chapel Hill	NC	27514-1644
252.	Linda	Ashman	100 Basswood Ct	Chapel Hill	NC	27514-1610
253.	Maia	Tellier	403 Knob Ct	Chapel Hill	NC	27517-7935
254.	N.	Marrone	102 Ironwood Pl	Chapel Hill	NC	27514-9575
255.	Patty	Daniel	1904 Jo Mac Rd	Chapel Hill	NC	27516
256.	Philip	Johnson	2600 Croasdaile Farm Pkwy C106 Heritage Hall	Durham	NC	27705
257.	Rebecca	Hunter	228 Indian Trail Rd	Chapel Hill	NC	27514-1926
258.	Jane	Norton	5605 Mount Sinai Rd	Durham	NC	27705-8610
259.	Suzy	Lawrence	8622 Ryan Rd	Chapel Hill	NC	27516-4899
260.	Tanya	Taylor	4607 River Run Ln	Rougemont	NC	27572-8498
261.	Melaina	Dyck	112 Meeting St	Chapel Hill	NC	27516-9168
262.	Nathalie	Worthington	1289 Fordham Blvd, 228	Chapel Hill	NC	27514
263.	Amber	Tarter	1008 Maple Ridge Dr	Chapel Hill	NC	27516-4844
264.	Arielle	Schechter	440 Bayberry Dr	Chapel Hill	NC	27517
265.	Barbara	Thornton	7111 Union Grove Church Rd	Chapel Hill	NC	27516-5267
266.	Ben	Thomas	3617 Fox Chase Rd	Trent Woods	NC	28562
267.	Brian	Rosa	1018 Orange High School Rd	Hillsborough	NC	27278-8418
268.	Catherine	Lavau	605 Shady Lawn Rd	Chapel Hill	NC	27514-2005
269.	Marta	Chase	878 Fearrington Post	Pittsboro	NC	27312-5037
270.	Christine	Carlson	101 Copperline Dr Apt L	Chapel Hill	NC	27516
271.	Coleman	Whittier	4901 Boulder Run Rd	Hillsborough	NC	27278-8300

272.	Carolyn	Cole	2120 N Lakeshore Dr	Chapel Hill	NC	27514-2027
273.	Cindy	Taylor	1315 Beechgrove Ln	Chapel Hill	NC	27516-5398
274.	Davenne	Essif	101 Wrenn Pl	Chapel Hill	NC	27516
275.	David	Flora	550 Carolina Meadows Villa	Chapel Hill	NC	27517
276.	Diane	Nelson	244 Sweet Bay Pl	Carrboro	NC	27510
277.	Don & Darlene	Wells	308 Mitchell St	Hillsborough	NC	27278-2130
278.	Eli	Celli	407 Legends Way	Chapel Hill	NC	27516-4371
279.	Emily	O'Hare	302 Copperline Drive	Apt. Q	NC	27516
280.	Marc	Pendergast	203 Glenview Pl	Chapel Hill	NC	27514-1950
281.	Elisabeth	Curtis	112 Circadian Way	Chapel Hill	NC	27516
282.	Herb	Lowrey	1447 Gray Bluff Trl	Chapel Hill	NC	27517-9126
283.	Eric	Horlbeck	405 Simerville Rd	Chapel Hill	NC	27517
284.	Kate D	Torrey	501 Dogwood Dr	Chapel Hill	NC	27516-2807
285.	Katie	Reily	1200 Galilean Trail	Chapel Hill	NC	27516
286.	Kicab	Castaneda-Mendez	878 Fearrington Post	Pittsboro	NC	27312
287.	Larry	Gottschalk	107 Wild Oak Ln	Carrboro	NC	27510-4139
288.	Julie	Bond-Meers	109 Stephens Street	Chapel Hill	NC	27516
289.	Lynn	Weller	211 Wild Oak Ln	Carrboro	NC	27510
290.	George	Phillips	101 Boyd Dr Apt 2D	Flat Rock	NC	28731-8785
291.	Philip	Carl	345 Carolina Meadows Villa	Chapel Hill	NC	27517-7519
292.	Piper	Honigmann	1215A Hillsborough Rd.	Chapel Hill	NC	27516
293.	Margaretha	Richardson	17 N 15Th St	Wilmington	NC	28401
294.	Robert	Reeber, Phd	1722 Lake Valley Trl	Chapel Hill	NC	27517-7733
295.	Sharon	House	1712 Damascus Church Rd	Chapel Hill	NC	27516-8025
296.	Barbara	Stenross	120 Carol St	Carrboro	NC	27510
297.	Stephanie	Rogers	1008 Starfield Circle	Hillsborough	NC	27278
298.	Thomas	Henkel	3 Mount Bolus Rd	Chapel Hill	NC	27514
299.	Catherine	West	1002 Willow. Dr.. Apt.61	Chapel Hill	NC	27514-2938
300.	Samantha	Allen	184 Dublin Ct	Carthage	NC	28327-7136
301.	Rosalyn	Arnold	2055 Bethabara Rd, Apt 41	Winston-Salem	NC	27106
302.	Denis	Obrien	1535 Caraleigh Mills Ct	Raleigh	NC	27603
303.	James	Womble	2700 N Mayview Rd	Raleigh	NC	27607
304.	Andra	Eich	121 Ashley Lane	King	NC	27021
305.	James	Hoots	3455 Mountain View Rd	Germanton	NC	27019-8245

306.	Kathy	Royal	374 Green Mountain Rd	Hendersonville	NC	28792-2024
307.	Iris	Carman	327 Lakewood Dr	Wilkesboro	NC	28697-8459
308.	Judith	Porter	927 Mulberry Mill Rd	North Wilkesboro	NC	28659-7706
309.	Gwen	Shafer	145 Decoy Dr	Wilkesboro	NC	28697
310.	Hannah	Norwood	1229 Rama Rd	Charlotte	NC	28211-4344
311.	Cama	Merritt	1244 Arbor Rd Apt 224	Winston Salem	NC	27104
312.	Lei	Zhang	557 Doe Run Dr	Kernersville	NC	27284-8080
313.	Brittany	Auten	626 Knollwood Dr	Winston-Salem	NC	27103
314.	Hellen	Shore	414 S Main St	Kernersville	NC	27284
315.	Barbara	Sheffield	620 Drumheller Rd	Clemmons	NC	27012-8554
316.	Cindy	Castevens	648 Irving St.	Winston-Salem	NC	27103
317.	Dr. Althea	Taylor-Jones, Phd	1469 Country Meadows Ln	Kernersville	NC	27284-9563
318.	John	Cardarelli	2423 Hoyt St	Winston Salem	NC	27103-4313
319.	Benjamin	Miller	242 Ridge Forest Ct	Winston Salem	NC	27104-3552
320.	Cynthia	Dunn	2411 Wynbrook Square Ct	Winston Salem	NC	27103-8002
321.	Joanne	Heckel	115 Sir Patricks Ct	Clemmons	NC	27012-7413
322.	Charles	Moore	126 Vintage Ave	Winston Salem	NC	27127
323.	Chris	Mclaughlin	221 E Sprague St	Winston Salem	NC	27127-3013
324.	Frank	Peplowski	518 Tanners Park Ct	Winston-Salem	NC	27101
325.	Jeff	Bohan	900 Teague Rd.	Winston Salem	NC	27107
326.	Tom	Adkisson	1398 Hannaford Rd	Winston-Salem	NC	27103
327.	Alice	Stack	5721 Fox Chase Dr	Winston Salem	NC	27105
328.	Donna	Pellett	5578 Pinebrook Ln	Winston Salem	NC	27105
329.	Kenneth	Hoglund	5037 Cobblestone Rd	Winston Salem	NC	27106-9618
330.	David	Sparks	4536 Thacker Hill Dr	Winston Salem	NC	27106-1653
331.	Thomas	Mann	3625 Bechler Ln	Winston Salem	NC	27106-2869
332.	Debi	Engelhaupt	828 B W 7Th St	Winston-Salem	NC	27101
333.	Keith	Davis	4160 Lakewood Glen Dr	Winston Salem	NC	27107-6881
334.	Diane	Arbour	3409 6Th St Dr Nw	Hickory	NC	28601
335.	Angela	Lucena	1005 Hunting Ridge Rd, Ap A	Raleigh	NC	27615
336.	Diane	Blanks	357 Green St	Boone	NC	28607-3490
337.	Heather	Reaves	Po Box 2646	Boone	NC	28607
338.	Frank	Borkowski	303 Daisy Trce	Banner Elk	NC	28604-8099
339.	Robert	Schlagal	18723 Highway 88	Creston	NC	28615

340.	Chelsea	Cannon	219 Rhododendron Ln	Boone	NC	28607-5705
341.	Donna	Carter	631 Queen St	Boone	NC	28607-3452
342.	Eric	Frauman	111 Rivers St.	Boone	NC	28607
343.	John	Anderson	117 E Cove Ln	Boone	NC	28607-9301
344.	Rebecca	Keeter	5706 Laurel Creek Rd	Banner Elk	NC	28604-7372
345.	Dale	Kirkley	180 Maple Ridge Dr	Boone	NC	28607
346.	Maureen	Dintino	201 Colt Creek Rd	Lansing	NC	28643
347.	Wes	Weaver	342 Dogwood Knl	Boone	NC	28607-8134
348.	James D	Mussetter	2035 Walker Rd	Winston-Salem	NC	27106
349.	Bexky	Myers	943 Enterprise Dr	Lexington	NC	27295
350.	Richard	Marter	3250 Midkiff Rd	Winston Salem	NC	27106-3030
351.	Ann	Clack	208 Crystal Drive	Broadway	NC	27505
352.	Sally	Stuckey	67 Shuler Rd	Candler	NC	28715-9225
353.	Margo	Ewing	511 North Horner Blvd	Sanford	NC	27330-1050
354.	Jay	Yager	200 Park Ave	Sanford	NC	27330-4029
355.	Jeffrey	Evensen	102 Elderberry Ln	Rougemont	NC	27572
356.	Terry	Labombard	189 Miranda Ln	Roxboro	NC	27574-6602
357.	Adrian	Smith	Po Box 265(110 Jones St)	Moncure	NC	27559
358.	Susan	Clayton	101 W Smith Rd	Pittsboro	NC	27312
359.	Billie	Hinton	196 Meadow View Dr	Moncure	NC	27559
360.	Donna	Burford	1495 Gum Springs Church Rd.	Moncure	NC	27559
361.	Jeannie	Ambrose	675 Lichen Trail	Pittsboro	NC	27312
362.	Josephine	Corro	43 Bennett Mountain Trce	Chapel Hill	NC	27516-3711
363.	Judy	Hogan	7598 Moncure Pittsboro Rd	Moncure	NC	27559-0253
364.	Kevin	Flynn	258 Canopy	Pittsboro	NC	27312
365.	Eileen	Mccorry	4103 Fearrington Post	Pittsboro	NC	27312-5049
366.	Martha	Girolami	473 Mt. Pisgah Church Rd.	Apex	NC	27523
367.	Margaret	Wainwright	2 Carolina Mdws Apt 107	Chapel Hill	NC	27517
368.	Mj	Copeland	220 Chatham Business Dr	Pittsboro	NC	27312
369.	Alice	Kirkman	455 Stage Coach Rd	Siler City	NC	27344
370.	Johnny	Mayall	86A Willow Way	Chapel Hill	NC	27516-9469
371.	Rick	Mchenry	499 Forest Lake Est	Moncure	NC	27559
372.	Mary	Lindsey	3000 Galloway Rdg	Pittsboro	NC	27312-8639
373.	Teresa	Ladd	601 Jamestown Rd	Pittsboro	NC	27312

374.	Catherine	Andrews	3038 Fieldstone Ln	Mebane	NC	27302
375.	Glenda	Walden	2241 Sandy Ln	Mebane	NC	27302-9187
376.	Lynn	Moseley	1442 Old Coach Rd	Graham	NC	27253
377.	Scott	Ferguson	2043 Meadow Ln	Graham	NC	27253
378.	Alexis	Lamere	3265 Northwest Trce	Elon	NC	27244-9518
379.	Richard	Arrington	686 Isley School Rd	Burlington	NC	27217-8397
380.	Carolyn	Wilson	332 Thompson St Apt A	Burlington	NC	27215-7380
381.	Louisa	Dang	1236 Jamestowne Dr	Elon	NC	27244
382.	Ruby	Lowe	22 Cates Circle Apartment C Lot 43	Graham	NC	27253
383.	John	Freeze	648 Chaney Road	Asheboro	NC	27205
384.	Katherine	Lowrance	930 Hill St	Greensboro	NC	27408-8716
385.	Anne	Jones	2304 Brandt Vlg	Greensboro	NC	27455
386.	Andrew	Meulendyk	7714 Whipple Trl	Greensboro	NC	27455
387.	Judith	Foster	5409 Amberhill Dr	Greensboro	NC	27455-1136
388.	Dale	Weston	48 Milpond Ln	Greensboro	NC	27455-2179
389.	Nancy	Kondracki	5211 Flintrock Ct	Greensboro	NC	27455-1377
390.	Stephanie	Benson	6808 Palomino Ridge Ct	Summerfield	NC	27358
391.	Tim	Stevenson	2615 Oak Ridge Rd	Oak Ridge	NC	27310
392.	Katherine	Williams	2102 Bryant St	Madison	NC	27025
393.	Molly	Follweiler	206 S Lonesome Rd	Madison	NC	27025
394.	Becky	Sims	4171 Old Julian Rd	Julian	NC	27283
395.	Susan	Russell	8003 Wagmont Dr	Browns Summit	NC	27214-9023
396.	Cathy	Way	4133 Old Way Rd	Sophia	NC	27350
397.	Darlene	Nercessian	4330 Jerry St	Trinity	NC	27370
398.	Kristiana	Van Eyk	632 Mountain Rd	Asheboro	NC	27205
399.	Ronald	Clayton	10860 Old Us Highway 70	Cove City	NC	28523
400.	Judith	West	339 Gregg St	Archdale	NC	27263-3303
401.	Leona	Whichard	344 Cedar Club Circle	Chapel Hill	NC	27517
402.	Paula	Stober	3607 Timberoak Dr	Greensboro	NC	27410
403.	Blake	Walker	53516 Bickett	Chapel Hill	NC	27517
404.	Pamela	Johnson	104 W Bradford Way	Pikeville	NC	27863
405.	Anthony	Gordon	132 Headwaters Dr # 132	Hampstead	NC	28443-2086
406.	Jack	Kelly	7715 Blue Heron Dr W Apt 1	Wilmington	NC	28411
407.	Robert	Rossi	94 Nandina Dr	Hampstead	NC	28443-3679

408.	Herman	Dobbs	158 Ne 13Th Street	Oak Island	NC	28465
409.	Brian	Beauregard	7271 Schooners Ct Sw	Ocean Isle Beach	NC	28469
410.	Carol	Kirsche	4523 Old Towne St	Wilmington	NC	28412-5010
411.	Craig	Brown	670 Kings Trail	Sunset Beach	NC	28468
412.	Cheryl	Crossman	423 Hawthorne Loop Rd	Leland	NC	28451
413.	Dawn	Pieper	6149 River Sound Cir	Southport	NC	28461-3141
414.	Elliott	Tepper	5102 Prices Creek Dr	Southport	NC	28461
415.	Daniel	George	9140 Hickory Ln Se	Winnabow	NC	28479
416.	Jack	Balsinger	1312 Taswell Ct	Leland	NC	28451-9493
417.	Bonnie	Westbrook	3795 Ridge Crest Drive	Southport	NC	28461
418.	Michael	Mcconney	1116 Princesa Ct Sw	Ocean Isle Beach	NC	28469
419.	Martin	Hazeltine	7614 Dunbar Dr Sw	Sunset Beach	NC	28468
420.	Miles	Varner	114 Nw 3Rd St	Oak Island	NC	28465-6809
421.	Michael	Esposito	717 Heather Glen Ln	Calabash	NC	28467-1767
422.	Pete	Key	5007 E Yacht Dr	Oak Island	NC	28465
423.	Scott	Brown	890 Stone Chimney Rd Sw	Supply	NC	28462-3282
424.	Richard	Wheeler	1411 Greenfield Rd Nw	Supply	NC	28462
425.	Shirley	Slominski	138 Bellwood Circle,	Sunset Beach	NC	28468
426.	Lynn	Smith	1176 Riverview Dr Sw	Shallotte	NC	28470-4602
427.	Suzanne	May	1246 Lillibridge Dr	Leland	NC	28451-7020
428.	William	Yingst	1042 Putting Ln	Carolina Shores	NC	28467-2247
429.	Janet	Anderson	1514 Grandiflora Dr	Leland	NC	28451-9531
430.	Janet	Stiegler	1412 West Gantry Ct.	Leland	NC	28451
431.	Fredrick	Milano	Po Box 1518	Boone	NC	28607
432.	Richard	Kelly	2266 Compass Pointe South Wynd Ne	Leland	NC	28451
433.	Sheila	Davis	102 Stoney Creek Ln	Leland	NC	28451-7797
434.	Suzan	Fluke	2287 Azalea Pointe Ct	Leland	NC	28451-6456
435.	John	Lapatchka	1002 Chalet Court	Leland	NC	28451
436.	John	Calloway	5006 Hunters Trail	Wilmington	NC	28405
437.	Earla	Pope	149 Chadwick Ave	Wilmington	NC	28401-2609
438.	Esther	Murphy	7235 Darden Rd	Wilmington	NC	28411
439.	James	Zizzo	2304 Wrightsville Ave.	Wilmington	NC	28403
440.	William	Taylor	2012 Creecy Ave	Wilmington	NC	28403
441.	Patricia	Tarr	1806 Wrightsville Ave	Wilmington	NC	28403

442.	Wendie	Schneider	120 Church St	Wilmington	NC	28401-5008
443.	Gayle	Whetzel	3608 Saint Francis Dr	Wilmington	NC	28409-6602
444.	Melissa	Herzog	317 Lewis Drive	Carolina Beach	NC	28428
445.	Ann	Russell	1534 Village Dr	Wilmington	NC	28401-7534
446.	Aimee	Donaton	224 Seawatch Way	Kure Beach	NC	28449
447.	Rick	Hoppe	202 Loder Ave	Wilmington	NC	28409-4312
448.	Dan	Gallagher	7845 Masonboro Sound Rd	Wilmington	NC	28409
449.	Ann	Hood	206 Texas Ave.,	Carolina Beach	NC	28428
450.	Ellen	Minnich	700 Mason Knoll Dr	Wilmington	NC	28409-3024
451.	Fred	Gainey	1521 Cadfel Ct, #103	Wilmington	NC	28412
452.	Marsha	Rand	3350 Club Villas Dr	Southport	NC	28461
453.	Janis	Wootten	3805 Mayfield Ct	Wilmington	NC	28412
454.	Joann	Bristol	5704 Oak Bluff Ln	Wilmington	NC	28409-2365
455.	Karen	Dunn	622 Waynick Blvd Unit 102	Wrightsville Beach	NC	28480-2101
456.	Andrew	Marhevsky	5017 Dockside Dr	Wilmington	NC	28409
457.	Maryleigh	Preston-Mcclure	1515 Village Dr Apt 1	Wilmington	NC	28401
458.	M	Stanley	Central Blvd	Wilmington	NC	28401
459.	Sue	Hayes	213 Quilon Cir	Wilmington	NC	28412-2046
460.	Starr	Watson	3720 Merestone Dr	Wilmington	NC	28412
461.	Susan	Kolesar	4229 Thursley Rd	Wilmington	NC	28412-8200
462.	Katherine	Hill	509 Whiting Cove	Wilmington	NC	28412
463.	Valerie	Tucker	619 Spencer Farlow Dr	Carolina Beach	NC	28428-3917
464.	Brenda	Fong	215 Avant Dr	Wilmington	NC	28411-9008
465.	Elizabeth	Bauereis	416 Black Diamond Dr	Wilmington	NC	28411-8376
466.	Debra	Gillingham	713 Fairlie Ct	Wilmington	NC	28412
467.	Glenn	Meyer	6442 Quail Run Rd	Wilmington	NC	28409-2203
468.	Bill	Harris	330 Tanbridge Road	Wilmington	NC	28405
469.	James	Taylor	410 E Bedford Rd	Wilmington	NC	28411-9515
470.	Shelley	Anthony	3950 Sweetbriar Rd	Wilmington	NC	28403-5439
471.	Danielle	Laborde	6576 Towles Rd	Wilmington	NC	28409-2123
472.	Lloyd	Smith	317 Pages Creek Drive	Wilmington	NC	28411
473.	Ann	Mccray	1712 Signature Pl	Wilmington	NC	28405-4130
474.	Mercedes	Hyman	6832 Main St	Wilmington	NC	28405-4167
475.	Nancy	Savits	217 Stoneybrook Road	Wilmington	NC	28411

476.	Renee	Ertischek	539 Windstar Ln	Wilmington	NC	28411
477.	Rachel	Schroeder	6229 Wrightsville Ave Apt K	Wilmington	NC	28403
478.	Ronald	Leuchs	1813 S Moorings Dr	Wilmington	NC	28405-5336
479.	Tom	Schultz	414 Hiawassee Ave	Black Mountain	NC	28711-2829
480.	Tamara	Agnelli	6428 Old Fort Road	Wilmington	NC	28411
481.	Joseph	Bye	814 1/2 S 4Th St	Wilmington	NC	28401-5132
482.	Darryl	Johnson	2029 Teresa Dr	Castle Hayne	NC	28429
483.	Jeff	Mills	122 Mohawk Trl	Wilmington	NC	28409
484.	Virginia	Lundeen	405 Sabra Dr	Wilmington	NC	28405
485.	Mark	Weber	318 N.23Rd Street	Wilmington	NC	28405
486.	Monica	Rolquin	6321 Towles Rd	Wilmington	NC	28409
487.	Miles	Murphy	5052 Park Ave	Wilmington	NC	28403
488.	William	Brown	1302 Bexley Dr	Wilmington	NC	28412-2091
489.	Theresa	Elias	218 N Duplin St	Wallace	NC	28466
490.	Gail	Sikes	313 E Church St	Rose Hill	NC	28458-1427
491.	Carrie	Kluiter	273 Parrish Farm Ln	Benson	NC	27504-6033
492.	Jessica	Bogue	207 N Pine St	Princeton	NC	27569-7066
493.	Sandy	Cothern	667 Love Mill Road	Whiteville	NC	28472
494.	Jen	Johnson	1720 Orange St	Wilmington	NC	28403-1000
495.	Susan	Hanna	302 Rl Honeycutt Dr	Wilmington	NC	28412-7172
496.	Laura	Faber	6346 Pawling Ct	Fayetteville	NC	28304-5566
497.	Melisa	Eslinger	5242 Surf Scooter Dr	Fayetteville	NC	28311-0662
498.	Bretton	Little	2711 Bennington Rd	Fayetteville	NC	28303
499.	David	Nikkel	1926 N. Pearl St.	Fayetteville	NC	28303
500.	James	Kerchmar	824 Azalea Dr	Fayetteville	NC	28301-4804
501.	Paula	Mcphail	2122 Clinchfield Drive	Fayetteville	NC	28304
502.	Walt	Dietrich	429 Summerlea Dr	Fayetteville	NC	28311-1171
503.	Henry Louis	Rodriguez Cruz Jr	7718 Eunice Dr	Fayetteville	NC	28306-8625
504.	Linda Sue	Barnes	6713 Wade Stedman Road	Wade	NC	28395
505.	Luvi	Valino	3615 Sunchase Dr	Fayetteville	NC	28306-8092
506.	Jacquelyn	Hough	305 Andrews Rd	Red Springs	NC	28377
507.	Wanda	Maitland	388 Sunset Lake Rd	Lumber Bridge	NC	28357
508.	Cliff	Long	118 Linwood Dr	Albemarle	NC	28001-2923
509.	Arthur	Firth	1011 Emerald Bay Dr	Salisbury	NC	28146

510.	Cindy	Shoaf	225 Playground Ln	Salisbury	NC	28146-7534
511.	Glenn	Ahrendt	140 Winged Foot Rd	Pinehurst	NC	28374
512.	Camilla	Vance Shadley	650 Aiken Rd	Vass	NC	28394
513.	Chas	Griffin	1275 7 Lks N	Seven Lakes	NC	27376
514.	Debra	Christner	3123 7 Lks W	West End	NC	27376-9301
515.	Ann	Collins	188 Murray Hill Road Apt A	Southern Pines	NC	28387
516.	William	Carothers	40 Inverrary Rd	Pinehurst	NC	28374
517.	Linda	Konold	315 Burning Tree Rd	Pinehurst	NC	28374
518.	Kathy	Wright	620 Lighthorse Cir	Aberdeen	NC	28315-3774
519.	Cathleen	Pritchard	4 Georgia Ct	Pinehurst	NC	28374-9647
520.	Patricia	Richardson	1642 Aiken Rd	Vass	NC	28394
521.	Patricia	Griffin	1275 Seven Lakes N., 106 Brown	West End	NC	27376
522.	Richard	Chatham	564 Rubicon Rd	West End	NC	27376
523.	Sandra	Burns	Po Box 221	Jackson Springs	NC	27281-0221
524.	Joanne	Thornton	140 Pinyon Circle	Pinehurst	NC	28374
525.	Gaynelle	Brown	136 Pennington Fry	New London	NC	28127
526.	Wanda	Setzer	1400 Central Dr	Kannapolis	NC	28083-3743
527.	Karen	Kaser-Odor	278 Fryling Ave Sw # 26	Concord	NC	28025
528.	Taylor	Conner	8836 Thatcher Place	Harrisburg	NC	28075-6504
529.	Melissa	Young	1263 Boswell Ct.	Concord	NC	28207
530.	Richard	Lewis	512 Worthington Ct Ne	Concord	NC	28025-2576
531.	Vanessa	Loszko	2804 Pennsylvania Avenue	Kannapolis	NC	28083
532.	Amanda	Brewer	735 Ann Rd	Orrum	NC	28369
533.	Janet	Gray	216 Range Rd	Hope Mills	NC	28348-9704
534.	Raymond	Harris	210 Tiffany Ct Apt D	Fayetteville	NC	28301-3799
535.	Helen	Livingston	311 Montrose Ln	Laurinburg	NC	28352-5517
536.	David	Harkin	5817 Stonebridge Ln	Waxhaw	NC	28173
537.	Robert	Hamby	1207 Keswick Pl	Monroe	NC	28112-5854
538.	Jennifer	Barbara	609 Appomatox Dr	Marvin	NC	28173
539.	Karen	Turner	2153 Darian Way	Waxhaw	NC	28173-5204
540.	Adrianne	S	3005 Cameron Woods Dr	Monroe	NC	28110-7864
541.	Frank	Stroupe	329 Raintree Dr	Matthews	NC	28104
542.	Mark	Sullivan	4016 Logan Cir	Indian Trail	NC	28079-6516
543.	Chet	Hayes	5316 Ravenglass Ct	Waxhaw	NC	28173

544.	Nancy	Behrens	7503 Quail Hill Rd	Charlotte	NC	28210-7262
545.	Reid	Leggett	1701 Brandon Rd	Charlotte	NC	28207-2103
546.	Frank	Sanady	6538 Rosemary Lane	Charlotte	NC	28210
547.	April	Gunning	2521 Breuster Dr	Charlotte	NC	28210-5841
548.	Bryan	Gabriel	679 Hyde Park Dr Ne	Concord	NC	28025
549.	Jd	Doliner	127 Circle Ave	Charlotte	NC	28207
550.	Lucie	Laberge	6442 Donnegal Farm Rd	Charlotte	NC	28270
551.	Charlene	Knop	9307 Raintree Ln	Charlotte	NC	28277
552.	Diane	Frederick	9206 Four Mile Creek Rd	Charlotte	NC	28277-9063
553.	Diana	Travis	6904 Alexander Rd	Charlotte	NC	28270-2806
554.	Christina	Brandt	9506 Mitchell Glen Dr	Charlotte	NC	28277
555.	Helen	Kedziora	11917 Kings Castle Ct	Charlotte	NC	28277-2290
556.	Leigh	Yeoman	10501 Moss Mill Ln	Charlotte	NC	28277-1672
557.	Edward	Turner	11226 Coachman Cir	Charlotte	NC	28277-9173
558.	Babs	Austin	4309 Shea Ln	Mint Hill	NC	28227-9280
559.	Carrie	Fawcett	10821 Redgrave Ln	Mint Hill	NC	28227-8996
560.	Dane	Bowen	8740 Blair Rd	Mint Hill	NC	28227
561.	Michael	Korzelius	3003 Duck Point Drive	Monroe	NC	28110
562.	Adele	Schiessle	6910 Hollow Oak Dr	Mint Hill	NC	28227
563.	Sharon	Campbell	1500 Kirkbridge Ct	Matthews	NC	28105
564.	Trendi	Oakley	7909 Jefferson Colony Rd	Mint Hill	NC	28227-7089
565.	Marilyn	Brown	2901 Carding Pl	Matthews	NC	28105-7169
566.	Stephanie	Kenny	6113 Loch Arbor Ln	Charlotte	NC	28227
567.	Deborah	Smith	4881 Leepers Creek Rd	Maiden	NC	28650-8220
568.	Monica	Strom	7217 Morley Ct.	Wilmington	NC	28411
569.	Bruce	Beerbower	551 3Rd St Ne	Hickory	NC	28601
570.	Andrew	Peterson	1756 31St Avenue Ln Ne	Hickory	NC	28601-8592
571.	Richard	Mccrary	1759 Yellowstone Ct Apt I	Gastonia	NC	28054-1772
572.	Garry	Moyers	107 Adrian Cir	Mount Holly	NC	28120
573.	Rose Marie	Tresp	101 Mercy Dr	Belmont	NC	28012-2898
574.	Jim	Mitchem	154 Old Spring Rd	Belmont	NC	28012-9707
575.	Laura	Liska	6018 Thorburn Way	Belmont	NC	28012
576.	Tyler	Baird	4042 Belle Meade Circle	Belmont	NC	28012
577.	Susan	Dameron	1245 N Hill Dr	Lincolnton	NC	28092-9656

578.	Lajla	Duffy	350 Hunting Ridge Lane	Shelby	NC	28150
579.	David	Marshall	930 W Warren St	Shelby	NC	28150
580.	David	Campbell	1007 Brookhaven Dr	Shelby	NC	28152
581.	Carolyn	Clark	1774 Warrior Dr	Tryon	NC	28782-4571
582.	Don	Clapp	567 Greenville St	Saluda	NC	28773-9780
583.	Kari	Dacey	301 N Trade St	Tryon	NC	28782
584.	Janet	Orselli	P.O. Box 211	Columbus	NC	28722
585.	Lewis	Patrie	26 Wesley Drive, Apt H	Asheville	NC	28803
586.	Joan	Battey	9 Knoll Dr	Fletcher	NC	28732
587.	C. Warren	Pope	12 Mountain Site Ln Ext	Asheville	NC	28803-2195
588.	Eric	Everett	38 Whites Lake Blvd	Saluda	NC	28773
589.	Sandra	Roggero	60 Cherry St	Arden	NC	28704-2735
590.	Timothy	Burgin	135 Louisiana Ave	Asheville	NC	28806
591.	Thomas	Atherton	32 Spears Ave	Asheville	NC	28801-1214
592.	Brooke	German	107 Annandale Ave	Asheville	NC	28801-1307
593.	Betty	Lawrence	142 Hillside St	Asheville	NC	28801
594.	Carolyn	Kanter	118 Maple Dr Apt 1A	Asheville	NC	28805-1166
595.	Charles	Jansen	98 Dorchester Ave	Asheville	NC	28806-3525
596.	Claudia	Nix	72 Sherwood Road	Asheville	NC	28803
597.	Joan	Vogt	527 Rose Hill Rd	Asheville	NC	28803
598.	Helen	Hyatt	14 Swindale St	Asheville	NC	28801
599.	Jean	Wheelock	53 Trail Top Dr	Asheville	NC	28805-0049
600.	Linda	Covington	62 Beverly Rd W	Asheville	NC	28806-4507
601.	Edith	Simpson	15 Springdale Rd	Asheville	NC	28805
602.	Maranda	Johns	6 Angus Ln	Asheville	NC	28805-2538
603.	Marcia	Greenstein	15 Oregon Ave Apt A	Asheville	NC	28806-3470
604.	Marilyn	Bollinger	28 Forestdale Dr	Asheville	NC	28803
605.	Marla	West	81 Wild Cherry Rd	Asheville	NC	28804
606.	Mia	Elias	64 Clingman Ave	Asheville	NC	28801-3284
607.	Robert & Karen	Milnes	1 Ridgeview Drive	Asheville	NC	28804
608.	Tracy	Moore	1 Battle Sq Apt 212	Asheville	NC	28801-2739
609.	Robert	Lundquist	63 Forest Lake Dr	Asheville	NC	28803-9000
610.	Amanda	Seta	12 1/2 Wall St Ste G	Asheville	NC	28801-2732
611.	Sarah	Rubin	17 Maywood Rd	Asheville	NC	28804-2532

612.	James Buck	Schall	31 Elizabeth St	Asheville	NC	28801-2267
613.	Terry	Faulkner	160 Chatham Rd	Asheville	NC	28804
614.	Xandria	Birk	44 N Liberty St	Asheville	NC	28801-1831
615.	Robert	Cozart	Po Box 422	Fairview	NC	28730-0422
616.	Julia	Burr	71 Fortune St	Black Mountain	NC	28711-2788
617.	Miriam	Sexton	18 Cedarwood Trl	Asheville	NC	28803
618.	Deborah	Swanson	568 Garren Creek Rd	Fairview	NC	28730
619.	Fiddle	Witch		Swannanoa	NC	28778
620.	Marilyn	Hamer	220 Dye Leaf Rd	Fairview	NC	28730-9651
621.	Irene	Moser	307 Wilson Cove Rd	Swannanoa	NC	28778-2826
622.	Laurie	Roper	37C Elderberry Lane	Asheville	NC	28804-3924
623.	Leslie	Bennis	21 Leannas Way	Asheville	NC	28805
624.	Peter	Lourekas	Po Box 18738	Asheville	NC	28814
625.	Robert	Swett	301 Montreat Rd	Black Mountain	NC	28711-3119
626.	Sally	Woodard	801 Azalea Ave	Black Mountain	NC	28711
627.	Sam	Collingwood	244A Old Fort Rd	Fairview	NC	28730-9518
628.	Kimberly	Hughes	301A Kerlee Heights Rd	Black Mountain	NC	28711-3612
629.	Z. Vijay	Director	27 Hunting Lodge Dr	Black Mountain	NC	28711
630.	Nancy	Brown	48 Elijah Hall Rd.	Black Mountain	NC	28711
631.	Barbara	Barcomb	311 Virginia Street Sw	Lenoir	NC	28645
632.	Emily	Bowman	4951 Burns Rd	Granite Falls	NC	28630-8147
633.	Tina	Khutsuvan	2982 Fred Bentley Road	Granite Falls	NC	28630
634.	Pamela	Little	2817 Wendell St	Lenoir	NC	28645-7626
635.	Barbara	Ward	108 Walker St	Morganton	NC	28655
636.	Carol	Roof Eanes	285 Highlands Drive	Hampstead	NC	28443
637.	Henry	Belada	1971 Sunnyside Dr.	Morganton	NC	28655
638.	Cynthia	Bringle	160 Lucy Morgan Ln	Bakersville	NC	28705-7389
639.	Cody	Jones	262 Hicks Chapel Loop	Marion	NC	28752
640.	Melissa	Bloom	1066 Beans Creek Rd	Bakersville	NC	28705-7841
641.	Sally	Rogers	Po Box 48	Penland	NC	28765-0048
642.	Bernie	Byrne	2363 Crooked Creek Rd	Mars Hill	NC	28754
643.	Laura	Boggess	501 Bailey St.	Mars Hill	NC	28754
644.	Laura	Boggess	501 Bailey St	Mars Hill	NC	28754
645.	Sandra	Byrne	2363 Crooked Creek Rd	Mars Hill	NC	28754-6927

646.	Brett	Rodgers	850 Upper Browns Creek Rd Ste B	Burnsville	NC	28714-7500
647.	Fred	Coppotelli	383 Seldon Emerson Rd.	Cedar Mountain	NC	28718
648.	Heide	Coppotelli	383 Seldon Emerson Rd	Cedar Mountain	NC	28718
649.	Jay	Slusher	34 Rhett Rd	Flat Rock	NC	28731
650.	Joyce	Dye	10 Rivoli Blvd	Hendersonville	NC	28739
651.	Kenneth	Wallston	1200 Appalachian Blvd	Arden	NC	28704
652.	Kristy	Lapidus	1727 Old Ccc Rd	Hendersonville	NC	28739-8540
653.	Liz	Davis	586 Salola Ln	Brevard	NC	28712-8489
654.	Lorraine	Thomas	5 Westbridge Dr	Hendersonville	NC	28739
655.	Marion	Washer	198 Pine Shadow Dr	Hendersonville	NC	28739-7502
656.	Paul	Hawkins	316 Heather Cir	Brevard	NC	28712-7391
657.	Susan	Nabors	175 Tsiya Ct	Brevard	NC	28712-8473
658.	Sidney	Baker	128 Village Greenway	Flat Rock	NC	28731-7603
659.	Marsha	Stopa	94 Arrowhead Ridge Rd	Brevard	NC	28712-7216
660.	Jan Rowland	Swartz Rowland	16 Cameron Drive	Etowah	NC	28729
661.	Linda	Camp	566 Rambling Dr	Hendersonville	NC	28739
662.	Adrienne	Ferriss	27 Pheasant Dr	Asheville	NC	28803
663.	Margaret	Bradford	31 High Ridge Dr	Mills River	NC	28759
664.	Jude	Pasqualini	46 Piney Mountain Church Rd.	Candler	NC	28715
665.	Beth	Pensiero	128 Exeter Ct	Hendersonville	NC	28791-3254
666.	Diotima	Booraem	399 Blossom Branch Dr	Hendersonville	NC	28792-2034
667.	Ian	Howe	1461 5Th Ave W	Hendersonville	NC	28739-4007
668.	Hayden	Fink	150 Brittany Place Dr Apt H	Hendersonville	NC	28792-7173
669.	Austin	Watson	170 Colony Road	Hendersonville	NC	28792
670.	Chris	Mitchell	149 Cold Springs Rd	Hendersonville	NC	28792-9495
671.	Jacqueline	Knable	878 Sandburg Ter	Hendersonville	NC	28791-2992
672.	Rita	Russo	532 Norman Street	Hendersonville	NC	28791
673.	Kathleen	Pevaroff	17 Panther Ridge Rd	Hendersonville	NC	28792-9291
674.	Sara	Green	50 Greenleaf Cir	Asheville	NC	28804-2320
675.	Pat	Cole	6 Galahad Pl	Asheville	NC	28806
676.	Peter	Roda	20 Pine Meadow Dr	Asheville	NC	28804-2235
677.	Shari	Lane	3 Woodfin Ave	Asheville	NC	28804-3033
678.	Terri	Lefler	626 N Graham St	Charlotte	NC	28202
679.	Frances	Kelly	1965 Riverside Dr	Asheville	NC	28804

680.	Barbara	Miller	4 Lancaster Ln	Weaverville	NC	28787
681.	Braethun	Bharathae-Lane	91 Edwin Pl Apt3	Asheville	NC	28801
682.	Saul	Oliansky	124 Ivy Meadows Dr	Weaverville	NC	28787-9021
683.	Herman	Lankford	175 Britten Cove Rd	Weaverville	NC	28787
684.	Marion	Danforth	9 Williams St	Weaverville	NC	28787
685.	Mary	Buttitta	411 Periwinkle Dr	Asheville	NC	28804
686.	Susan	Parr	322 Midland Dr.	Asheville	NC	28804
687.	O.C.	Edwards	170 South Main Street	Mars Hill	NC	28754
688.	Philip	Stigall	320 Ivy Hill Rd	Weaverville	NC	28787
689.	Janice	Rubino	6 Shuford Road	Weaverville	NC	28787
690.	Adi	S	129 Aurora Dr	Asheville	NC	28805
691.	Maura	Clark	93 Old Cathy Rd	Candler	NC	28715-9548
692.	Meriwether	Beatty	3 Forest Road	Asheville	NC	28803
693.	J	Baker	52 Blossom Rdg	Leicester	NC	28748-5201
694.	Rob	Allyn	59 Luther Cove Rd	Candler	NC	28715
695.	David	Mclintock	920 Tumbling Fork Rd.	Waynesville	NC	28785
696.	Keri	Hollifield	591 Reed Cove Rd	Waynesville	NC	28786
697.	Anthony	Scardaci	298 East St	Waynesville	NC	28786
698.	John & Phyllis	Edwards	924 Po	Cashiers	NC	28717
699.	George	Rector	947 Bo Cove Rd	Cullowhee	NC	28723
700.	Doug	Wingeier	266 Merrimon Avenue	Asheville	NC	28801
701.	Joan	Parks	1102 Rockdale Rd	Whittier	NC	28789
702.	Matthew	Martens	498 Owl Branch Road	Cherokee	NC	28719-0877
703.	Robert	Hyatt	1846 Hammond St	Rocky Mount	NC	27803-2315
704.	Anne	Blaine	126 Dillon Dr	Franklin	NC	28734-1402
705.	Chuck	Stiles	40 White Cloud Drive	Murphy	NC	28906
706.	John	Balogh	95 Mac Cove Dr	Franklin	NC	28734-0448
707.	Pamela	Johnston	2015 Coweeta Church Rd	Otto	NC	28763
708.	Barb	Edlen	2260 Weldon Smith	Lawsonville	NC	27022
709.	Tony	Saiz	1408 Nc Hwy 150 W	Summerfield	NC	27358
710.	Blair	Justice	Po Box 8	Naples	NC	28760
711.	Ellyn	Kirschner	326 Tranquil Ave	Charlotte	NC	28209
712.	Elizabeth	Whitt	1116 Scaleybark Rd Apt 116B	Charlotte	NC	28209-4509
713.	Susan	Towl	101 Long Pond Drive	Sneads Ferry	NC	28460

714.	Beth	Henry	3066 Stoneybrook Rd	Charlotte	NC	28205
715.	Christine	Sheil	1514 Mimosa Ave	Charlotte	NC	28205-2908
716.	Shirley	Griffith	7519 Gayle Ave	Charlotte	NC	28212
717.	Eric	Innes	1421 Iris Drive Apt 4113	Charlotte	NC	28205
718.	Jessica	Williams	1937 Olsen Lane	Charlotte	NC	28213
719.	Ashley	Council	307 N Dotger Ave	Charlotte	NC	28204-4357
720.	Diane	Carre	2041 Berkley Hall Way #304	Fort Mill	SC	29708
721.	Michael	Adams	201 Dinadan Dr Apt H	Charlotte	NC	28217-5164
722.	Roxanne	Holt	7800 Browne Road	Charlotte	NC	28269
723.	Conda	Jones	3616 Greenloch Ct	Charlotte	NC	28269
724.	Fred	Martin	1016 West 1St Street	Charlotte	NC	28202
725.	Allen	Smith	3209 Selwyn Farms Lane	Charlotte	NC	28209
726.	Sandy	Deoliveira	1916 Wilmore Dr	Charlotte	NC	28203-4621
727.	Omar	Perez	2529 Dellinger Cir	Charlotte	NC	28269-2761
728.	Briana	Garvin	3806 Old Stoney Creek Ct	Charlotte,Nc	NC	28269
729.	Ann	Rowell	7001 Thermal Rd	Charlotte	NC	28211-6150
730.	Linda	Levy	7058 Burlwood Rd	Charlotte	NC	28211-6108
731.	Jean	Jones	9226 Royal Highlands Ct	Charlotte	NC	28277
732.	Bill	Guiffre	11205 Cedar Walk Ln	Charlotte	NC	28277-4199
733.	Brandon	Williams	7239 Lockmont Dr	Charlotte	NC	28212
734.	Shannon	Caviness	7525 Cedarbrook Dr	Charlotte	NC	28215-4511
735.	Mike	Rodden	7615 Neal Rd	Charlotte	NC	28262
736.	James	Rogerson	9500 Robert Burns Ct	Charlotte	NC	28213
737.	James	Smith	3406 Summerfield Ridge Lane	Matthews	NC	28105
738.	Edith	Kurie	4305 Tillson Rd	Wilmington	NC	28412
739.	Janet	Fortner	10505 Kerns Rd	Huntersville	NC	28078
740.	Catherine	Denham	111 Peters Pl	Davidson	NC	28036
741.	Sue	Hunt	4618 Sierra View Dr	Denver	NC	28037-7304
742.	Gary	Andrew	319 N Downing St	Davidson	NC	28036
743.	John	Butler	20416 Deep Cove Ct	Cornelius	NC	28031-7231
744.	Michelle	Mitchell	17227 Chardonnay Ct	Cornelius	NC	28031
745.	Sharon	Russell	17524 Tuscany Lane	Cornelius	NC	28031
746.	Stephanie	Woelfle	8146 Townley Rd	Huntersville	NC	28078
747.	John	Delaney	14523 Harvington Dr	Huntersville	NC	28078-2215

748.	Deborah	Steiner	10102 Mountain Apple Dr	Mint Hill	NC	28227
749.	Phyllis	Tarrant	3308 Jonesberry Rd	Matthews	NC	28105
750.	Herbert	Baum	2 Sturbridge Ln	Greensboro	NC	27408-3842
751.	Pat	Cross	7 Granville Oaks Ct	Greensboro	NC	27408-5140
752.	Linda	Archer	3512 Sanfords Creek Ct	Colfax	NC	27235
753.	Ann	Steighner	1218 Lakewood Dr	Greensboro	NC	27410-4440
754.	David	Stubbs	3705 Brown Bark Dr	Greensboro	NC	27410-4605
755.	Janetta	Johnson	3901 Walker Avenue	Greensboro	NC	27403
756.	Jean	Hunt	705 Staunton Dr	Greensboro	NC	27410-6006
757.	Carol	Simpson	3000 W Cornwallis Dr	Greensboro	NC	27408-6730
758.	Bill	Jordan	5001 Liberty Rd	Greensboro	NC	27406-8619
759.	Betsabe	S	598 Montrose Dr	Greensboro	NC	27410-5911
760.	Sharron	Hedges	3709 Cameron Ter	High Point	NC	27265-1463
761.	Kay	Warren	627 Fieldale Pl	High Point	NC	27265-1321
762.	Kathryn	Austin	209 Woodmont Rd	Jamestown	NC	27282-8502
763.	George	Neste	4437 Garden Club St	High Point	NC	27265
764.	Daniel	Morris	1712 Mirabeau Ct	High Point	NC	27265
765.	Robert	Henry	3725 Deerfield St	High Point	NC	27265-9442
766.	Scott	Brown	2204 Gordon Rd	High Point	NC	27265-2410
767.	John	Porter	915 Woodbrook Dr	Greensboro	NC	27410
768.	Sandra	Resner	7607 Middle Dr	Greensboro	NC	27409
769.	Mary	Canel	9312 River Road	Wilmington	NC	28412
770.	David	Myers	211 N Park Dr	Greensboro	NC	27401-1535
771.	John	Davis	610 Bellemeade St	Greensboro	NC	27401
772.	Early	Smith	1007 Glenwood Ave	Greensboro	NC	27403-2908
773.	Ellen	Wells	1 Fraternity Dr	Greensboro	NC	27407-1846
774.	Jerald	Leimenstoll	629 S Elm St	Greensboro	NC	27406
775.	Sandra	Koritz	4 Cactus Court Unit B,	Greensboro	NC	27410
776.	Robin	Davis	2403 Battleground Ave Ste 7	Greensboro	NC	27408-4035
777.	Camille	Harris-Wallace	3701 W Gate City Blvd	Greensboro	NC	27407-4627
778.	Mary	Wakeman	2710 Azalea Dr	Greensboro	NC	27407
779.	Sharon	Daugherty	4312 Bramlet Pl	Greensboro	NC	27407
780.	Corinna	Biller	230 Deerfield Ct	Lexington	NC	27295-5854
781.	Dave	Taylor	3326 Chelsea Village Court	Winston-Salem	NC	27103

782.	Jerry	Chambers	464 Forest Creek Dr	Winston Salem	NC	27107-9293
783.	Debbie	Johnson	222 Rockin Horse Ln	Thomasville	NC	27360-7177
784.	Eva	Sadler	1813 Leonard Rd	Lexington	NC	27295-7474
785.	Judith	Williams	16 Vance Cir	Lexington	NC	27292
786.	Steven	Arey	415 W Marsh St	Salisbury	NC	28144-5321
787.	Telisha	Wood	376 Springway Ln	Cleveland	NC	27013-8990
788.	Ron	Barlow	14245 Cool Springs Rd	Cleveland	NC	27013-8138
789.	Betsy	Tucker	1451 Nc Highway 801 N	Advance	NC	27006-6703
790.	Tucker	Bailey	371 Brangus Way	Mocksville	NC	27028-4627
791.	Irene	Radke	150 Hideaway Ln	Mooresville	NC	28117
792.	Sara	Nolan	181 Castaway Trail	Mooreville	NC	28117
793.	Zach	Whitson	182 Normandy Rd	Mooresville	NC	28117-8430
794.	Bill	Rattray	3909 Inkberry Ct	Apex	NC	27539
795.	Elizabeth	Onan	420 Hickory Dr	Chapel Hill	NC	27517
796.	Josh	Kelly	29 N Market St	Asheville	NC	28801
797.	Les	Stradley	6 Blackberry Ln	Asheville	NC	28804
798.	Norman C	Wussow	4 Mayflower Drive	Asheville	NC	28804
799.	Amber	Albritton	20 Kilkenny Dr	Asheville	NC	28806
800.	Sarah	Overholt	Po Box 202	Pantego	NC	27860-0202
801.	Alice	Summey	144 Church Sto	Saluda	NC	28773
802.	Jack	Mcgowan	4654 19Th Street	San Francisco	CA	94114
803.	Elizabeth	Hardy	4626 White St., Apt. 202	Shallotte	NC	28470
804.	Esther	Garvett	1861 Nw South River Drive	Miami	FL	33125-2768
805.	Teresa	Craig	Po Box 311	Clayton	GA	30525
806.	Janice	Banks	14 Maple St.	Center Barnstead	NC	3225
807.	Silvia	Bertano	Corso Rosselli 123/8	Torino	NY	10129
808.	Judith	Shanley	7 Rodgers Pl	Asheville	NC	28806
809.	Carolyn	Funk	Po Box 11101	Youngstown	OH	44511
810.	Deborah	Smith	3044 N.W. 30Th	Oklahoma City	OK	73112
811.	Karen & Kurt	Weidner	2419 Hoods Mill Rd	Commerce	GA	30529
812.	Connie	Holden	15 Walnut Lane	Fort Mill	SC	29715
813.	Loretta	Wall	6021 Highway 701 S	Conway	SC	29527
814.	Kim	Scott	502 N Church St	Jackson	NC	27845
815.	Linda	Voelker	330 Crowell Ln	Salisbury	NC	28146

816.	Ann	Mcmartin	229 Sunset Drive	Asheville	NC	28804
817.	Jarrett	Barnhill	525 Ivy Dr	Hillsborough	NC	27278-9444
818.	Billy	Blackmon Ii	5309 Goshawk Dr	Hope Mills	NC	28348
819.	John	Gerwin	1008 Ravenwood Dr	Raleigh	NC	27606-1638
820.	Stan	Meyer	5404 Ropley Dr	Greensboro	NC	27455-1149
821.	Christine	Fearing	Brittley Way	Apex	NC	27502
822.	Sj	Davis	106 Sea Dunes Dr	Emerald Isle	NC	28594
823.	Cheryl	Lipstreu	7691 Craig Rd	Belews Creek	NC	27009-9176
824.	Dick	Christensen	1213 Areca Way	Durham	NC	27703-4666
825.	Christine	Curto-Kramer	8205 Yaxley Hall Drive	Raleigh	NC	27616
826.	Daniela	Rossi	123 Anystreet	Aberdeen	ID	83210
827.	Debora	Hilton	4701 Carberry Ct	Charlotte	NC	28226
828.	Douglas	Evans	105 Summerwalk Ct	Cary	NC	27518-9146
829.	Karen	Hudson	1605 Kinloch Dr	Winston Salem	NC	27107-8031
830.	Libby	Johnson	2127 Edwin Avenue	Durham	NC	27705
831.	Rashid	Hendricks	1022 Harvest Grove Ct	Hope Mills	NC	28348
832.	Kevin	Gedney	15026 Skypark Dr	Huntersville	NC	28078
833.	Leslie	Singleton	127 Albemarle Rd	Greensboro	NC	27405
834.	Rev. Jay	Leach	234 North Sharon Amity Road	Charlotte	NC	28211
835.	Jeff	Schweickert	1125 Stoneferry Ln	Raleigh	NC	27606-8092
836.	Jen	Almond	908 Queensferry Rd	Cary	NC	27511-6423
837.	Judith	Utley	111 Halls Creek Dr	Swansboro	NC	28584-9675
838.	Jonathan	Rollman	100 Stonehedge Ave	Durham	NC	27707
839.	Kimberly	Geddes	232 Bowman Road	Aberdeen	NC	28315-5673
840.	Kelly	Prelipp	2101 Cloiater Dr	Charlotte	NC	28211
841.	Doug	Franklin	195 Downings Creek Lane	Hayesville	NC	28904
842.	Lori	Tyman	77 Perry's Chapel Church Rd	Franklinton	NC	27525
843.	Marvin	Maddox	103 Caniff Lane	Cary	NC	27519
844.	Mary	Marinucci	47Bungalow Way	Brevard	NC	28713
845.	M	Win	1008 Pine Valley Dr	Durham	NC	27712-2214
846.	Nicholas	Rose	5026 Waldron Meadow Dr	Charlotte	NC	28226-8800
847.	Jennifer	Hill	2811 Watauga Dr	Greensboro	NC	27408
848.	Linda	Taranto	8330 Deerfoot Dr	Linden	NC	28356
849.	Rosalyn	Snyder	3603 Octavia St	Raleigh	NC	27606-3655

850.	Richard	Loeppert	1317 Rand Dr	Raleigh	NC	27608-1941
851.	Ria	Westphal	907 W Rowan St #A	Fayetteville	NC	28301
852.	Randall	Dail, Jr.	495 River Bluff Dr. Unit 3	Shallotte	NC	28470-5849
853.	Ryan	Barclay	281 Jubal Reeves Cir	Wilmington	NC	27306
854.	Sarah	Leehr	109 Rock Nest Court	Morrisville	NC	27560
855.	Susan	Bartlett	4 Lagrange Dr	Asheville	NC	28805
856.	Stephen	Parker	336 Park Ave	Knightdale	NC	27545
857.	Becky	Shepherd	103Walshingham	Cary	NC	27513
858.	Nancy	Davis	4868 Arlington Street	Hope Mills	NC	28348
859.	Sandra A	Sly	3075 Third St	Surf City	NC	28445-0048
860.	Shannon	Ryan	15046 Deshler Court	Charlotte	NC	28273
861.	Rev. Susan	Warren	656 Sand Hill Rd	Asheville	NC	28806-1554
862.	Tommie	Addison	1100 W Thomas Street	Rocky Mount	NC	27804
863.	Tricia	M	11814 Painted Tree Rd	Charlotte	NC	28226
864.	Mary Ann	Witt	2600 Croasdaile Farm Pkwy	Durham	NC	27705-1331
865.	Barbara	Dornbush	41 Fox Falls Ln	Highlands	NC	28741-6661
866.	Mitzi	Childers	3618 Bridle Path Drive	Vale	NC	28168
867.	Jennifer	Metzler-Fiorino	216 Barbee Blvd	Oak Island	NC	28465
868.	Barbara	Pace	260 Greenfield Ct	Lexington	NC	27295
869.	Elizabeth	Gordon	119 Blossom Ridge	Leicester	NC	28748
870.	Laura	Holt	6335 Fox Chase Dr	Davidson	NC	28036-8036
871.	Therese	Duffy	Po Box 36	Zirconia	NC	28790
872.	Julie	Apperson	108 Thornwood Loop	Sanford	NC	27330-1067
873.	Carole	Dupre	500 W Poplar Ave	Carrboro	NC	27510-1622
874.	Michael	Rollins	640 Poplar Dr	Shelby	NC	28152-7620
875.	Jutta	Moore	2900 Rannock Ct	Raleigh	NC	27604
876.	Shelley	Wheeler	2865 Pine Bloom Way	Leland	NC	28451-6041
877.	Nancy	Harrison	4024 Strendal Drive	Cary	NC	27519
878.	Chris	Worrell	505 Cherokee Dr	Jacksonville	NC	28540-6712
879.	Penny	Eustis	330 Nottingham Rd	Jacksonville	NC	28546-5527
880.	Elizabeth	Eitelman	166 Spring Creek Ln	Wilmington	NC	28411
881.	Richard	Ferguson	7184 Seagrass Cir	Denver	NC	28037-5479
882.	Emmy	Grace	2717 Highland Avenue	Durham	NC	27704
883.	Jake	Poysti	4641 Malone Ct	Raleigh	NC	27616

884.	Bunny	Simoneau	10112 Lafoy Dr	Huntersville	NC	28078
885.	April	Ingle	6240 Spurgeon Way	High Point	NC	27265
886.	Caryn	Segal	8431 N Shoreside Way Ne	Leland	NC	28451-6602
887.	Sangeeta	Parakala	8440 Broderick Pl	Cary	NC	27519
888.	Lori	Del Negro	6900 Three Bridges Cir	Raleigh	NC	27613-3551
889.	Ellen	Dowling	3280 Mannington Dr	Charlotte	NC	28270-2270
890.	Taylor	Hill	49 Richmond Road	Jackson Springs	NC	27281
891.	Peyton	Vaughn	2732 University Dr	Durham	NC	27707-2864
892.	Rahul	Chintalapani	2515 Red Maple Ln	Harrisburg	NC	28075-4506
893.	Danielle	Sheets	1791 Friendly Grove Church Rd	Millers Creek	NC	28651-8736
894.	Christine	Grabar	228 S. 181St East Avenue	Tulsa	OK	74108
895.	Judy	Haughee-Bartlett	3003 Eagle Nest Ct.	Summerfield	NC	27358
896.	Alice	Strickland	312 Point Place, Apt.-C	Fayetteville	NC	28301
897.	Barbara	Grady	4927 N Nc Highway 111	Seven Springs	NC	28578
898.	Monica	Warren	10632 Highstream Drive	Raleigh	NC	27614
899.	Heather	Bishop	3918 Sarah Dr	Charlotte	NC	28217
900.	Vickie	Miller	118 Penny Rd	Jamestown	NC	27260
901.	Raymond	Lee	160 Chatham Road	Asheville	NC	28804
902.	Karen	Mendys	323 Chauncey Circle	Chapel Hill	NC	27516
903.	Carolyn	Smith	313 St Kitts Way	Winnabow	NC	28479
904.	Linda	Ricks	112 Willow Street	Beaufort	NC	28516
905.	Donna	Pridgen	940 Burney Rd	Bladenboro	NC	28320
906.	Kim	Yates	141 East Main Street	Ellerbe	NC	28338
907.	Dennis	Letman	1515 Park Summit Blvd	Apex	NC	27523
908.	Margi	Erickson	412 S 3Rd St	Wilmington	NC	28401
909.	Veronica	Noechel	2224 Mariner Cir	Raleigh	NC	27603-2666
910.	Michael	Tart	1516 Smith Level Rd	Chapel Hill	NC	27516-3230
911.	Wendy	Glen	4625 Vienna Dozier Rd	Pfafftown	NC	27040-9671
912.	John	Galligan	4278 Aviemore Run	Burlington	NC	27215
913.	Steven	Matteson	2061 Simmerman Way	Leland	NC	28451-9490
914.	M	S	9051 Strickland Rd Ste 200	Raleigh	NC	27615-2084
915.	Christopher	Madden	2504 Jefferson Dr	Greenville	NC	27858-4013
916.	Victoria	Estes	1884 Brevard Rd	Arden	NC	28704
917.	Bonnie	Wright	2209 Englewood Ave	Durham	NC	27705-4013

918.	Dragica	Zoric	95 Nichols Street	Everett	MA	2149
919.	Brian	Connors	213 Carolina Sands Dr	Carolina Beach	NC	28428-4604
920.	Donna	Bonarrigo	606 Watson Ave	New Bern	NC	28560-3148
921.	Havana	Adams	164 Qr Drive	Ayersville	NC	27027
922.	Sylvia	Sellers	5014 Providence Rd	Charlotte	NC	28226-5850
923.	Kim	Kiser	123 Midwood Ln	Belmont	NC	28012-8750
924.	Lee	Prevost	700 Vista Lake Dr., Apt. 104	Candler	NC	28715
925.	Lorraine	Loren	40 Rocky Springs Rd	Taylorsville	NC	28681
926.	Madison	Bravo	1407 W Florida St	Greensboro	NC	27403-3321
927.	Marylou	Digiorgio	7215 Kidwelly Lane	Matthews	NC	28104
928.	Sonya	Hannah	105 Lasalle Way	Greensboro	NC	27406-8141
929.	Dawn	Clementi	4700 Riverwood Cir Apt 259	Raleigh	NC	27612-5752
930.	Dannie	Ingle	4495 Greenfield Way Dr	Winston Salem	NC	27103-9759
931.	Meredith	Sunstrom	205 W Davie Street, Unit 510	Raleigh	NC	27601
932.	Ann	Green	740 Three Mile Knob Rd	Pisgah Forest	NC	28768-9060
933.	Margaret	Pumphrey	40 Rocky Knls	Chapel Hill	NC	27516-0327
934.	Jack	Sehestedt	184 Indian Springs Ln	Carthage	NC	28327-6860
935.	Ann S.	Thompson	8405 Bells Lake Rd	Apex	NC	27539-8383
936.	Thomas	Dengate	2303 Wachovia Dr	Greensboro	NC	27403
937.	Shelley	Vyas	1409 Kinnesaw St	Wake Forest	NC	27587-8769
938.	Tom	Jackson	3001 Sikes Mill Rd	Monroe	NC	28110-9782
939.	Ken	Wood	7 Glen Valley Dr	Arden	NC	28704-9407
940.	Robert	Hearn	1082 Nichols Dr	Raleigh	NC	27605
941.	Fred	Lampe	1710 Michaux Rd	Chapel Hill	NC	27514-7636
942.	Tricia	Hayes	1139 Woodlawn Cir	Newton	NC	28658-9041
943.	William	Gulley	1313 Woodburn Rd	Durham	NC	27705-5740
944.	Elizabeth	Gulley	1313 Woodburn Rd	Durham	NC	27705-5740
945.	Paula	Marchio	2405 Palisade Ct	Leland	NC	28451
946.	Denise	Mirandola	842 Ocean Blvd W	Holden Beach	NC	28462-1811
947.	Stacey	Cannon	1903 Stokes Ferry Rd.	Salisbury	NC	28146
948.	Vicki	Parker	6113 Amber Bluffs Crescent	Raleigh	NC	27616
949.	Rebecca	Bryant Williams	3880 Whitehaven Rd	Winston Salem	NC	27106-2554
950.	L	Lundgren	258 Mangia Dr	Wake Forest	NC	27587
951.	Connie	Marhevsky	5017 Dockside Drive	Wilmington	NC	28409

952.	Marlene	Goland	4 Lake Forest Court	Greensboro	NC	27408
953.	Darlene	Parlett	1107 Millheim Court	Wilmington	NC	28411
954.	Barbara	Benson	104 Deerfield Ct	Cedar Point	NC	28584-8047
955.	Katie	Tarr	1236 Waterway Ct	Wilmington	NC	28411
956.	Brandi	Alfieri	15 Hidden Creek Dr	Arden	NC	28704
957.	Deborah	Adam	3001 Monterey St	Greensboro	NC	27406-4225
958.	Claris	Castillo	510 Presidents Walk Ln	Cary	NC	27519-6847
959.	Clark	Pearson	1128 Kitchens Branch Rd.	Sylva	NC	28779
960.	Walter	Wood	304 Hedrick St	Beaufort	NC	28516
961.	Laura	Lathan	1312 Gateshead Lane	Matthews	NC	28105
962.	Alex	Wilder	6153 Windover Creek Ln	Claremont	NC	28610-8047
963.	Melissa	Beaver	3825 Cherry Grove Dr	Hickory	NC	28602-9785
964.	Roger	Zuidema	3394 Walters Rd	Creedmoor	NC	27522-8643
965.	Debbie	Nelms	Po Box 896	Murphy	NC	28906
966.	Sally	Bassett	929 Berry Patch Lane	Pittsboro	NC	27312
967.	Keith	Davenport	Po Box 428	Welcome	NC	27374-0428
968.	Patricia	Burgert	516 Walters Dr	Wake Forest	NC	27587-6177
969.	Cathy	Keizer	139 Renwick Ct	Raleigh	NC	27615-2946
970.	Elizabeth	Albright	Po Box 1226	Buxton	NC	27920-1226
971.	Sherri	White-Williamson	528 Mckoy St	Clinton	NC	28328-2517
972.	Barbara	Zumsteg	335 Queens Rd	Sanford	NC	27330-3411
973.	Lisa	Dale	183 James Rd	Advance	NC	27006-7000
974.	Shirley	Mcnair	725 Burgoyne Dr	Fayetteville	NC	28314
975.	Lucy	Roederer	12026 Wicker Dr	Chapel Hill	NC	27517
976.	Christine	Craig	1950 Patton St	Sanford	NC	28303
977.	Chastity	Outlaw	100 Halstead Dr	Moyock	NC	27958-9003
978.	Claire	Middleton	2831 Heather Glen Ln	Charlotte	NC	28208-2581
979.	Barbara	Firestone	4622 Crestwood Dr	Monroe	NC	28112
980.	Jonathan	Barley	2621 Springhill Ave	Raleigh	NC	27603
981.	Walter	Kelley	112 Annas Way	Grandy	NC	27939-9601
982.	Herbert	Dula	13712 Riding Hill Ave	Charlotte	NC	28213-4251
983.	Linda	Deming	110 Elk Mountain Scenic Hwy	Asheville	NC	28804-1706
984.	Daniel	Patterson Md	1901 London Ln	Wilmington	NC	28405-4210
985.	Susan	Ward	45 Laura Cove Lane	Murphy	NC	28906

986.	Franco	Divallerino	303 Smith Level Rd Apt F22	Chapel Hill	NC	27516-8383
987.	Jessica	Wise	507 Yost Rd	Salisbury	NC	28146-6851
988.	Mary	Brzezinski	902 Haymarket Ln	Wilmington	NC	28412-7415
989.	Meri Ann	Worley	1328 Michigan Blvd	Sanford	NC	27332
990.	Barbara	Veliskakis	6205 Morrison Blvd.	Charlotte	NC	28211-5147
991.	Dr. Charles D.	Chambliss, Jr.	1100 Sycamore Street	Rocky Mount	NC	27801
992.	Chelsea	Brooks	1107 Nonya St	Pleasant Garden	NC	27313
993.	Harrington	Drake	1050 Beaver Dam Rd	Chapel Hill	NC	27517
994.	Kristine	Larson	4929 Wilderness Rd	Wilmington	NC	28412-7719
995.	Nicholas	Borisow	121 E Johnson St	Cary	NC	27513
996.	Ela	Madrazo	7020 Epping Forest Drive	Raleigh	NC	27613
997.	Va	Boyle	23A Trillium Court	Asheville	NC	28805
998.	George	Robinson	6010 Dolphin Rd.	Oriental	NC	28571
999.	Margaret	Fouse	101 Forest Hills Dr	Black Mountain	NC	28711
1,000.	Mary	Baldwin	6516 Red Cedar Rd	Wilmington	NC	28411-4730
1,001.	Michael	Andrews	810 W 4Th St	Winston Salem	NC	27101
1,002.	Jessy	Hargis	3602 Edgefield Road	Greensboro	NC	27409
1,003.	Martha	Livingston	6021 Hwy 181, Jonas Ridge	Jonas Ridge	NC	28641
1,004.	Dawn	Allen	224 Triplett St E3	Jonesville	NC	28642
1,005.	George	Czerw	703 Alyssum Avenue	Caswell Beach	NC	28465
1,006.	Richard	Inskeep	P O Box 618	Badin	NC	28009
1,007.	Andrew	Chung	812 Cotton Exchange Court	Raleigh	NC	27608
1,008.	Lyric	Kinard	102 Kilmorack Dr	Cary	NC	27511
1,009.	Yvonne	Sumner	800 Farmcrest Dr	Charlotte	NC	28206-1320
1,010.	Jennifer	Roberts	619 Clement Ave	Charlotte	NC	28204
1,011.	Angela	Baldwin	2886 Hallsboro Rd S	Hallsboro	NC	28442-9214
1,012.	Margaret	Love	145 Bass Ct	Salisbury	NC	28146-9503
1,013.	Shelby	Ward	525 Summerow Rd	Stanley	NC	28164-1384
1,014.	Kelly	Picarsic	4837 Water Oak Road #14	Charlotte	NC	28211
1,015.	Ruth	Stanley	641 Hopscotch Court	Wilmington	NC	28411
1,016.	Diana	Swift	4314 Branch Bend Lane, Unit 232	Charlotte	NC	28273
1,017.	Kurt	Nichols	9204 Four Mile Creek Rd	Charlotte	NC	28277-9063
1,018.	Jill	Twark	2501 East 5Th St.	Greenville	NC	27858
1,019.	Murray	Lanier	231 Diane Ct	Jacksonville	NC	28540

1,020.	Brant	Bottum	1014 South Virginia Dare Trail	Kill Devil Hills	NC	27948
1,021.	Parran	Foster	186 Blue Ridge Trl	Mooresville	NC	28117
1,022.	Betty	Rogers	5132 Governor Scott Rd	Cedar Grove	NC	27231-9023
1,023.	Anjanette	Hughes	259 Cedar Swamp Rd	Newport	NC	28570-4104
1,024.	Katie	Lowe	2515 Alyssa Ln	Charlotte	NC	28208-5280
1,025.	G	Thomas	6380 Cliffdale Rd	Fayetteville	NC	28314-3116
1,026.	Vernon	Gragg	315 Hibriten Dr. Se	Lenoir	NC	28645
1,027.	Barry	Durham	1315 Lovelace Rd	Pelham	NC	27311-8514
1,028.	Boris	Birger	5323 Griffith Park Rd	Raleigh	NC	27613-1444
1,029.	Corinne	Russell	4900 Connell Dr	Raleigh	NC	27612-3006
1,030.	Sherry	Lee	575 Castle Rising Rd	Fayetteville	NC	28314
1,031.	Chris	Carlin	1265 Grace Rd	Southport	NC	28461
1,032.	Clyde	Schell	211 Stony Branch Rd	New Bern	NC	28562
1,033.	Susan	Thurlow	64 Second St	Tryon	NC	28782
1,034.	Kerri	Gardner	1660 Highpoint St	Wake Forest	NC	27587-6506
1,035.	Brent	Bracken	9 Ponderosa Ct	Greensboro	NC	27406
1,036.	Cheryl	Dalton	404 W Decatur St Apt202	Madison	NC	27025
1,037.	Debbie	Watterson	185 Holly Springs Court	Southern Pines	NC	28387
1,038.	Linda	Kappauf	2406 Beechwood Dr	Waxhaw	NC	28173-8356
1,039.	Lena	Gallitano	2907 Hostetler St	Raleigh	NC	27609
1,040.	Donald	Whetzel	263 Twelve Oaks Drive	Linwood	NC	27299
1,041.	Brendan	Smith	2840 Spring Shade Rd	Apex	NC	27523
1,042.	Venkat	Vadla	210 Begen St	Morrisville	NC	27560
1,043.	Elizabeth	Kostova	394 Vanderbilt Rd	Asheville	NC	28803-3036
1,044.	Elmer	Winterfeld	3824 W. Unionville Indian Trail	Indian Trail	NC	28079
1,045.	Kimberly	Coon	78 Hill Row Lane	Clayton	NC	27527
1,046.	Lavera	Parato	407 Ketner Blvd	Havelock	NC	28532
1,047.	Karen	Waltman	Hendersonville	Hendersonville	NC	34481
1,048.	Barbara	Benjamin Sliney	70 Jordan Ln	Spruce Pine	NC	28777-9349
1,049.	David	Sink	Po Box 947	Leland	NC	28451
1,050.	Nancy	Hunter	1819 8Th Street Dr Ne	Hickory	NC	28601-2191
1,051.	Julie	Thomson	729 Lorentello Circle	Hillsborough	NC	27278
1,052.	Brenda	Kent	213 S Halifax Rd	Rocky Mount	NC	27804-3094
1,053.	Jean	Gurkin	1600 Cotton Patch Rd	Chocowinity	NC	27817-9061

1,054.	Jeanie	Ahrens	4108 Kestrel Ct	Lenoir	NC	28645-6870
1,055.	Susan	Moses	102 Dogwood Ct	Morganton	NC	28655-9181
1,056.	Michael	Burnham	1 Saint Croix Place Apt 2H	Greensboro	NC	27410
1,057.	Pamela	Price	1575 Grove Lane	Wilmington	NC	28409
1,058.	George	Sawyer 3D	1301 Queens Rd	Charlotte	NC	28207
1,059.	Angel	Dallas	401 Rose Crest Ct.	Wilmington	NC	28412
1,060.	Lisa	Fisk	325 Tryon St.	Burlington	NC	27217
1,061.	Jorge	Foster	140 Lindsey Ln	Nebo	NC	28761-6715
1,062.	Lester	Wiggins	1375 W Islands Rd	Williamston	NC	27892-8125
1,063.	Preston	Fort	6532 Tampico Court	Fayetteville	NC	28303
1,064.	Isabelle	Chan	646 Coniston Drive	Leland	NC	28451
1,065.	Virginia	Ledford	1155 Marshburn Road	Wendell	NC	27591-9329
1,066.	Robert	Gelblum	500B Oak Ave	Carrboro	NC	27510
1,067.	Noah	Wright	221 Pine Knoll Rd Lot 41	Forest City	NC	28043
1,068.	Donna	Oglesby	37 Clara Lane	Murphy	NC	28906
1,069.	Eli	Hutchins	121 Moore Ave	Randleman	NC	27317-9493
1,070.	Rodney	Lemley	1125 Mashie Lane	Rocky Mount	NC	27804
1,071.	Elissa	Oliver	804 Walden Dr	Wilmington	NC	28401-6894
1,072.	Ariana	Watkins	4000 Sabre Ln	Wilson	NC	27896-8848
1,073.	Linda	Eastman	7048 Sevilleen St Sw	Ocean Isle Beach	NC	28469-5865
1,074.	Grace	Sanders	32058 Clete Rd	Albemarle	NC	28001-7514
1,075.	Carter	Norris	295 Richmond Rd	Bakersville	NC	28705-8210
1,076.	Beth	Davidson	6319 Thermal Rd	Charlotte	NC	28211-5631
1,077.	Jordan	D'Addeo	3010 Moneta Way	Durham	NC	27703-5786
1,078.	Dorey	Welsh	909 Axis Cir	Hope Mills	NC	28348-9605
1,079.	Pamela	Butler	830 Rolling Pines Loop Rd Ne	Leland	NC	28451-7024
1,080.	John	Mclaughlin	228 Dew Drop Ln	Murphy	NC	28906-8686
1,081.	Salma	Said	2416 Trenton Woods Way	Raleigh	NC	27607-6004
1,082.	Brenna	Mccallum	717 Hamilton Road	Raleigh	NC	27604
1,083.	Robert	Bzduch	481 Simpson Rd	Carthage	NC	28327-9342
1,084.	Nora	Martin	1900 Glendale Ave	Durham	NC	27701-1326
1,085.	Mccayne	Miller	Po Box 99516	Raleigh	NC	27624-9516
1,086.	Grace	Hodgkins	6104 Greenville Loop Rd	Wilmington	NC	28409-2325
1,087.	Catherine	Hartofelis	5804 Winthrop Drive,	Raleigh	NC	27612

1,088.	Sean	Bennett	1839 Simonton Dr	Wilmington	NC	28405-6800
1,089.	Georgia	Bernstein	710 Wellingham Dr	Durham	NC	27713-7504
1,090.	Jane	Rubino	5426 7 Lks W	West End	NC	27376-9319
1,091.	Jess	Fox	108 La Mancha Dr Apt H	Asheville	NC	28805-2115
1,092.	Valerie	Allen	532 Woodberry Circle	Raeford	NC	28376
1,093.	James	Southerland	103 Moray Court	Cary	NC	27511
1,094.	Jerry	Stubberfield	2928 John T Holden Rd Sw	Supply	NC	28462
1,095.	Tamara	Dunlap	603 Hickory Nut Lane	China Grove	NC	28023
1,096.	Debby	Mcdonald	1105 Avebury Ct	Winnabow	NC	28479-5694
1,097.	Patricia	Almeida	914 Athens Drive, C	Raleigh	NC	27606
1,098.	Gary	Lockamy	247 Etown Farm Dr	Clarkton	NC	28433-7362
1,099.	Valerie	Harvey	1035 Ryan Ln	Walnut Cove	NC	27052-6921
1,100.	Diana	Deakin	11 Tattle Branch Circle	Asheville	NC	28805
1,101.	Donald	Rhodes	12728 Scenic Dr	Raleigh	NC	27614-9183
1,102.	Amy	Devereaux	512 Contessa Ct	Clayton	NC	27520-7071
1,103.	Valerie	Alfisi	725 Leatherstone Ln	Fuquay Varina	NC	27526-3715
1,104.	Rene	Specht	602 Beechwood Lakes Dr	Hendersonville	NC	28792-7234
1,105.	Jo	Hall	5809 W3Rd Street	Charlotte	NC	28208
1,106.	Elizabeth	Fender	6023 Bentway Dr	Charlotte	NC	28226-8052
1,107.	Rick	Hauser	887 Moyers Rd	Winston Salem	NC	27104
1,108.	William	Reavis	1105 Piney Grove Rd	Kernersville	NC	27284-7216
1,109.	Julie	Byrd	809 Sp	Murfreesboro	NC	27855
1,110.	Charlene	Harris	148 Mohawk Drive	Garner	NC	27529
1,111.	Kristen	Britt	3315 Auburn Dr.	Fayetteville	NC	28306
1,112.	Mary Beth	Lemon	5023 Revelation Way	Monroe	NC	28110
1,113.	Arnold	Levine	2710 White Pines Court	Monroe	NC	28112
1,114.	Kelli	Bee	2463 Sedgefield Dr	Chapel Hill	NC	27514-6812
1,115.	Steven	Henry	1912 Andrews Store Rd	Pittsboro	NC	27312-5819
1,116.	Amelie	Novio	5314 Greyfield Blvd	Durham	NC	27713-8144
1,117.	Barbara	Spencer	704 Watkins St	Greensboro	NC	27407-2248
1,118.	Kayla	Bennett	56 Mcdowell Rd	Mills River	NC	28759-2532
1,119.	Shannon	Armbrust Mulcahey	2421 Playa Way 2444-B	Wilmington	NC	28403
1,120.	Bob	Young	1422 Southpoint Trl, Ste 201	Durham	NC	27713-6757
1,121.	Carolyn	Turner	7307 Huddlestone Rd	Bailey	NC	27807

1,122.	Roger	Babson	1835 Hawthorne Road	Wilmington	NC	28403
1,123.	Elisa	Roels	8200 River Road	Wilmington	NC	28412
1,124.	Sterling	Vaden	130 S. Blue Ridge Rd.	Black Mountain	NC	28711
1,125.	Marcia	Morgan	110 Green Turtle Lane	Carolina Beach	NC	28428
1,126.	Cornelia	Cornils	218 Seminole Ave Se	Concord	NC	28025
1,127.	Sharon	Cherry	1983 Stone Rose Dr	Rocky Mount	NC	27804
1,128.	Theresa	Jacobs	320 Sam Miller Rd	Warsaw	NC	28398
1,129.	Barbara	Harris	1702 Wrightsville Ave	Wilmington	NC	28403
1,130.	Charmaine	Ortiz	514 Raleigh Ave.	Carolina Beach	NC	28428
1,131.	Kay	Warner	115 Horseshoe Ln	Burgaw	NC	28425
1,132.	Thomasina	Williams	143 Kenansville Highway	Warsaw	NC	28398
1,133.	Victoria	Oconnor	30 Candlewood Circle	Waynesville	NC	28786
1,134.	Kimberly	Anderson	818 Elizabeth Dr	Oak Island	NC	28465
1,135.	Michael	Smith	1575 Grove Lane	Wilmington	NC	28409
1,136.	Mary	Mcalonan	6329 Saxon Meadow Dr	Leland	NC	28451
1,137.	Robert	De Haas	306 Stonewall Jackson Dr	Wilmington	NC	28412
1,138.	Heather	Zaknich	21454 Lee Drive	Redwood Estates	NC	95044
1,139.	Linda	Emerick	70 Sean Dr	Swannanoa	NC	28778
1,140.	Kyle	Kramer	5611 Wrightsville Ave	Wilmington	NC	28403
1,141.	Kelly	Atkins	404 Bayfield Dr	Wilmington	NC	28411-8735

From: Daniel george
To: comments.chemours
Subject: [External] Addendum to consent order
Date: Tuesday, September 15, 2020 10:00:32 PM

I am writing you in favor of the addendum to the consent order. It is imperative that the pollutants from the Chemours facility be stopped in every possible way of entry into our river, our ground, and our air regardless of cost. If I had my way they would be in jail for knowingly polluting for the years that they silently got away with it.

From: pcarringtonyoung@gmail.com
To: comments.chemours
Subject: [External] Addendum to consent order
Date: Tuesday, September 15, 2020 10:25:46 AM

CAUTION: External email. Do not click links or open attachments unless you verify. Send all suspicious email as an attachment to report.spam@nc.gov<mailto:report.spam@nc.gov>

Dear Sir or Madam:

One thing that remains missing in an addendum is any relief for the homeowners wells that are contaminated. There needs to be a permanent whole home solution for these homes. People should not be exposed to contamination by having to bathe in chemicals. There should be relief by providing entire home systems or municipal water and deadlines to complete the same. Homeowners property values have suffered and health is questioned by continuous exposure. This relief should be the main focus of the addendum, human safety.

Phillip Young

From: Juliette Pahl
To: Martin, Sharon L.
Subject: [External] Chemours Addendum
Date: Monday, September 14, 2020 2:26:06 PM

I am in complete support of the Chemours Addendum, and believe that it is a necessary part of the order.

I am a permanent resident in Raleigh.

Juliette Pahl
4604 Pleasant Grove Church Rd.
Raleigh, NC 27613
ph: 410.961.0236

Sent from my iPad

From: Martin, Sharon L.
To: comments.chemours
Subject: FW: [External] Re: Reminder: State seeks feedback on Chemours Consent Order Addendum, Comment Period Open Through Thursday
Date: Monday, September 14, 2020 12:52:42 PM

From: Cheryl [mailto:itpsign@yahoo.com]
Sent: Monday, September 14, 2020 12:36 PM
To: Martin, Sharon L. <sharon.martin@ncdenr.gov>
Subject: [External] Re: Reminder: State seeks feedback on Chemours Consent Order Addendum, Comment Period Open Through Thursday

Chemours needs to provide every victim of their contamination with a permanent and complete solution not a bandaid! They need to take all the profits they've made off of poisoning us and provide total restitution to every person affected. That will shut them down, but that's what they deserve.

Sent from Yahoo Mail for iPhone

On Monday, September 14, 2020, 11:49 AM, Sharon Martin <Sharon.martin@ncdenr.gov> wrote:

DEQLogo.png

Roy Cooper, Governor

Michael S. Regan, Secretary

Release: IMMEDIATE	Contact: Sharon Martin
Date: September 14, 2020	Phone: 919-707-8670

Reminder: State seeks feedback on Chemours Consent Order Addendum, Comment Period Open Through Thursday

RALEIGH – The North Carolina Department of Environmental Quality seeks public comment on the Addendum to the Consent Order, which requires significant additional actions by Chemours to prevent PFAS pollution from entering the Cape Fear River via contaminated groundwater from the Fayetteville Works Site. Comments will be accepted through Thursday, September 17.

Since 2017, DEQ actions and the Consent Order have stopped the process wastewater discharge from the facility and drastically reduced air emissions of PFAS by 99.9%. The additional actions in the Addendum to the Consent Order between DEQ, Cape Fear River Watch and Chemours will further reduce the PFAS contamination to the Cape Fear River and improve water quality for downstream communities.

Moving forward, Chemours is required to treat four identified 'seeps' which account for more than half of the contaminated groundwater reaching the river in two phases.

- The interim measures to filter PFAS at an efficiency of at least 80% from the first of the four seeps will go into effect starting by Mid-November – with all four completed by April 2021.
- The permanent measure is the construction of a subsurface barrier wall approximately 1.5 miles long and groundwater extraction system that will remove at least 99% of PFAS to be completed by March 2023.

Chemours is also required to treat on-site stormwater that is adding residual pollution to the river with a capture and treatment system that must remove at least 99% of PFAS.

Failure to meet the schedules or achieve the removal goals will result in financial penalties, including:

- Failure to meet the construction schedule for the interim measures will result in fines of $5,000 per day for the first 14 days and $10,000/day until construction is complete.
- Failure to meet the barrier wall installation schedule results in a $150,000 fine followed by $20,000 per week until installation

is complete.

- Failure to meet the barrier wall's 95% mass loading goal in the initial demonstration results in a $500,000 fine, with a $100,000 fine for failure to meet any of the four subsequent demonstrations.

Comments on the Addendum will be accepted through September 17. Comments can be submitted electronically to comments.chemours@ncdenr.gov or mailed to Assistant Secretary's Office, RE: Chemours Public Comments 1601 Mail Service Center, Raleigh, NC 27699-1601.

DEQ will consider the public comments before the Addendum is presented for entry by the Bladen County Superior Court. The Addendum is available here.

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https://files.nc.gov/ncdeq/Public_Affairs/Footer.png

From: SVC_DENR.publiccomments
To: comments.chemours
Subject: FW: [External] Chemours
Date: Monday, September 14, 2020 9:05:17 AM

From: Fields,Brian M [mailto:brian.fields@louisville.edu]
Sent: Friday, August 21, 2020 11:10 AM
To: SVC_DENR.publiccomments <publiccomments@ncdenr.gov>
Subject: [External] Chemours

To the North Carolina Department of Environmental Quality,

I am writing today as a resident of Fayetteville and as a person who believes we must have accountability for the crisis that Chemours has caused through pollution of Perfluoroalkyl and Polyfluoroalkyl Substances (PFAS) into the Cape Fear River watershed. I have known many people in my community who have died from cancer, heart disease, and other complications in our region. They are frequently in the low-income, predominantly non-white areas of this region adjacent to the Cape Fear River. While I don't know that PFAS caused their deaths, I know that as lifelong residents in a watershed that has been ravaged by the despicable conduct of Chemours' systemic disregard for the health of our environment, I wouldn't be surprised if it did kill them. I know that as someone who has drank this poisoned water for two decades I assuredly have unsafe levels in my blood. Here are some facts about PFAS:

- PFAS has been detected in human blood, semen, and breast milk. PFAS can cross the placenta, exposing unborn children.
- Studies of people exposed to high levels of PFAS have shown links to Thyroid disease, Immune disorders, Abnormal liver function, Abnormal cholesterol levels, Decreased fertility in men and women, Complications of pregnancy and abnormal development of children exposed in utero, Kidney and testicular cancer
- Recent studies have shown that PFAS can mimic human hormones including thyroid, estrogen and testosterone, resulting in low function. One study looking at young men exposed to high levels of PFAS over long periods of time found lower testosterone activity resulting in smaller genitalia and lower sperm counts.
- PFAS have been dubbed "forever chemicals" because they accumulate in living organisms, including people, and persist for an extremely long time in the environment. Some never fully deteriorate.
- Since 1980, Chemours has discharged Gen X, a form of PFAS chemicals, directly into the Cape Fear River poisoning in the drinking water source for 250,000 people who are largely low income and people of color. While they have reduced their pollution levels since complaints were revealed in 2017, at their own admission there is still contamination into both the water and air. This is unconscionable conduct to poison citizens, and has to stop.

Therefore, I want to voice my support for the North Carolina Department of Environmental

Quality Addendum to the Consent Order to stop Chemours from actively polluting PFAS into the Cape Fear River. These include the following guidelines: 1) The interim measures to filter PFAS at an efficiency of at least 80% from the first of the four seeps will go into effect starting by Mid-November, with all four completed by April 2021. 2) The permanent measure is the construction of a subsurface barrier wall approximately 1.5 miles long and groundwater extraction system that will remove at least 99% of PFAS to be completed by March 2023. 3) Chemours is also required to treat on-site stormwater that is adding residual pollution to the river with a capture and treatment system that must remove at least 99% of PFAS.

Additionally, I support punitive measures in place for Chemours failure to achieve remediation. The following punitive measure have been laid out in the addendum: 1) Failure to meet the construction schedule for the interim measures will result in fines of $5,000 per day for the first 14 days and $10,000 per day until construction is complete, 2) Failure to meet the barrier wall installation schedule results in a $150,000 fine followed by $20,000 per week until installation is complete, 3) Failure to meet the barrier wall's 95% mass loading goal in the initial demonstration results in a $500,000 fine, with a $100,000 fine for failure to meet any of the four subsequent demonstrations.

While I think it is important that punitive measures are in place, I believe these amounts are insufficient to convey the severity of this crisis and the urgency in which Chemours needs to address it with. While these punitive measures are important, they are not deterrent. Chemours had 5.6 billion dollars in revenue for 2019. The maximum $500,000 fine would equal approximately .009% of the revenue of Chemours if they failed to meet barrier wall standards. This is not a deterrent. I ask that the North Carolina Department of Environmental Quality increase the punitive amounts to ensure that Chemours addresses this with an urgency reflective of the stakes in this crisis. Although the death of North Carolinians may not be an incentive for their remedial efforts, I am hopeful a dollar amount that sufficiently incentives Chemours will finally be the incentive that proves enough to ensure they stop poisoning our water.

I appreciate the opportunity for the public comment and ask that this be addressed with the utmost urgency. Thousands of people are already suffering from the COVID-19 crisis, paired with preexisting health conditions possibly caused by Chemours' pollution. These communities are disparately low-income, disparately non-white, and disparately underinsured/uninsured. While this addendum will help everyone, it will have a proportionally large positive impact on these communities, who have faced inequitable treatment by our state and country for too long. I ask that you continue to regulate and punish Chemours and any other polluters vigorously with the full authority of the law, so that we can begin to build a better future for those of us living in the Cape Fear River watershed.

Sincerely,

Brian Michael Fields
Fayetteville, North Carolina

From: Gloria Shen
To: comments.chemours
Subject: [External] Public Comment on the PFAS Addendum
Date: Saturday, September 12, 2020 4:53:07 PM

As a resident of North Carolina, I am submitting my comment to the NC Department of Environment and Natural Resources regarding the addendum to the 2019 consent order.
I am in support of all three sections of the amendment that stipulate the methods by which Chemours is required to remove the toxic PFAS that has contaminated our state's natural resources and caused grievous harm to communities with the exception of a portion of Section 2 where it states that "In the interim, the company is required to reduce pollution in the streams by a minimum of 80 percent."

It is my opinion that the most diligent efforts must be put forth by the company to reduce stream pollution and that *a minimum of 90-95 percent pollution reduction*, not 80 percent, would be a reasonable requirement until the in-stream filters are installed.
This disaster never should have occurred in the first place. There shouldn't be any PFAS in streams or the Cape Fear River. The state and its residents have suffered enough already.
Also, has the interim period before in-stream filters are installed been defined clearly? If a higher percentage of pollution reduction cannot be achieved (beyond the proposed minimum of 80 percent), perhaps the installation of in-stream filters needs to be expedited.
There should be no delay in the remediation efforts.

Thank you for taking the time to consider my comment.

Sincerely,
Gloria Shen

From: SVC_DENR.publiccomments
To: comments.chemours
Subject: FW: [External] Chemours
Date: Thursday, September 10, 2020 3:00:21 PM

From: Fields,Brian M [mailto:brian.fields@louisville.edu]
Sent: Friday, August 21, 2020 11:10 AM
To: SVC_DENR.publiccomments <publiccomments@ncdenr.gov>
Subject: [External] Chemours

To the North Carolina Department of Environmental Quality,

I am writing today as a resident of Fayetteville and as a person who believes we must have accountability for the crisis that Chemours has caused through pollution of Perfluoroalkyl and Polyfluoroalkyl Substances (PFAS) into the Cape Fear River watershed. I have known many people in my community who have died from cancer, heart disease, and other complications in our region. They are frequently in the low-income, predominantly non-white areas of this region adjacent to the Cape Fear River. While I don't know that PFAS caused their deaths, I know that as lifelong residents in a watershed that has been ravaged by the despicable conduct of Chemours' systemic disregard for the health of our environment, I wouldn't be surprised if it did kill them. I know that as someone who has drank this poisoned water for two decades I assuredly have unsafe levels in my blood. Here are some facts about PFAS:

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- PFAS have been dubbed "forever chemicals" because they accumulate in living organisms, including people, and persist for an extremely long time in the environment. Some never fully deteriorate.
- Since 1980, Chemours has discharged Gen X, a form of PFAS chemicals, directly into the Cape Fear River poisoning in the drinking water source for 250,000 people who are largely low income and people of color. While they have reduced their pollution levels since complaints were revealed in 2017, at their own admission there is still contamination into both the water and air. This is unconscionable conduct to poison citizens, and has to stop.

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Sincerely,

Brian Michael Fields
Fayetteville, North Carolina

From: Gail Goodman
To: comments.chemours
Subject: [External] Chemours
Date: Thursday, September 3, 2020 10:53:46 AM

The goal of the Addendum is to significantly and permanently reduce PFAS entering the Cape Fear River from the Chemours site and impacting the downstream communities.

Why is it the GOAL to significantly and permanently Reduce pfas.
How about the GOAL is to simply Eliminate permanently pfas.
How about proposing the implementation of reverse osmosis and or technology accomplishing the same principal. Simply develop it or shut down. Further, Chemours and like industries need to shut down till this technology is in place.
Why do we need these products. America is about innovation. Can we dream and create something better or eliminate the need for the product at all.
Why do the children have to deal with this. Shame on us. How little we care about our own sisters and brothers, even.
Nothing is without consequences. God will judge those who harm his little ones, especially.

Concerned USA citizen

Sent from my iPad

From: Gail Goodman
To: comments.chemours
Subject: [External] Chemours
Date: Wednesday, September 2, 2020 6:23:28 PM

". The system will treat groundwater that currently discharges without treatment into the river, and it is not designed for process wastewater from the facility. Since 2017, Chemours has been prohibited from discharging process wastewater into the Cape Fear River. "

First let me say, over the years I've been commenting, I have not once received a reply to any questions.
Second, the above statement that a new system will treat groundwater going into the river but not wastewater from the facility going into the river is clever and misleading. Don't you think you have it backwards? If you treat the wastewater from the facility, wouldn't that eliminate, automatically, the groundwater problem?
And, third, I found the last sentence in the paragraph hysterical!. Keywords, since "2017", Chemours has been "prohibited" from discharging process wastewater into the mouths of our babies!
I rest my case. If Chemours has been "prohibited" since "2017" from dumping process wastewater into the river, why are we here? If they are "prohibited", why is it still happening four years later? Does the problem lie with the enforcer? Oh, wait, the DEA IS the enforcer! Maybe you need help from big brother. Oh, that's another problem! Is there not one righteous lawyer who will stand and fight in the Supreme Court for the people and perhaps his own children?
I'm not wasting my time with these little love letters back and forth over the years anymore. The time has come to demand our voices be heard. People must rise up. People will rise up.

Thank you for the opportunity to reply. My only wish is drastic action is taken, now
Free citizen of the USA

Sent from my iPad

Sent from my iPad

From: Barbara Bakowycz
To: comments.chemours
Subject: [External] Addendum
Date: Thursday, August 20, 2020 1:24:01 AM

Can Chemours truly be adequately monitored for compliance? I have little confidence of such based on past precedent. At the very least, Chemours should be required to provide RO water to Wilmington and Brunswick County residents ASAP. We are TIRED of the ongoing spin.

J Barbara Bakowycz RN

Sent from my iPad

From: Lauren Me
To: comments.chemours
Subject: [External] Chemours Addendum with Cape Fear River Watch
Date: Wednesday, August 19, 2020 11:02:44 PM

I think this addendum (signed August 13) is the least that Chemours can do. They are causing proven harm by releasing and creating these chemicals. This will at least mitigate some harm.

Lauren

From: Kaili Rich
To: comments.chemours
Subject: [External] Chemours public comment
Date: Wednesday, August 19, 2020 5:05:44 PM

Chemours should be responsible for paying for water purification facilities for both New Hanover and Brunswick counties, including the Sweeney water treatment plant, instead of letting the cost responsibilities fall in the people who live in those counties. Our drinking water routinely exceeds the 10/trillion of PFAS, but we are not granted the same protections as people who live near the plant. Chemours should provide whole house reverse osmosis filters for all lower cape fear residents who's water contains more than 10/trillion. CFPUA needs to be included in all future decisions going forward with chemours, as it is irresponsible to let a county that is severely affected by their PFA pollution to not have a voice in how the issue is corrected. If Chemours can't pay for all of these necessary precautions to protect the people that they have poisoned, then they should be shut down and not allowed to operate in our state. It's time for North Carolina to stop protecting big corporations and to actually start caring about its people. Do the right thing, and extend protections to all North Carolinians who have been affected by chemours toxic waste.

From: Philip McHugh
To: comments.chemours
Subject: [External] Chemours Comment
Date: Wednesday, August 19, 2020 2:20:10 PM

Were the people and elected officials of Brunswick County involved in this addendum?
What about Brunswick County Public Utilities?
Why not?

The people of Brunswick County drink PFAS. We bathe in it. We cook with it, wash our dishes in it, and water our gardens with it.

What is being done by Chemours to help Brunswick County NOW, not 2 or 3 years from now?

The addendum needs an addendum. Have Chemours put $$$$$ into Brunswick County **NOW.**

Philip McHugh
919-818-5441

From: Crystal Young
To: comments.chemours
Subject: [External] Chemours comments
Date: Tuesday, August 18, 2020 3:06:33 PM

Dear Sir or Madam:

In reviewing the Addendum to the Consent Order, I see where action is being taken to decrease further pollution but I do not see where any action is being taken to remedy the homeowners that are already affected. The residents whose wells have been contaminated need remedies to this contamination. With Chemours only offering three sink filters to certain well owners, is not a remedy. It is only a minor band aid for blatant disregard to the residents' health. Well owners do not need or deserve any type of health hazard when it is at the hands of Chemours. The affected well owners need either entire full house filtration or municipal water. The affected well owners do not need to have to worry about the health affects of bathing in contaminated water. Dermal absorption is a clear hazard. Why are the well owners not being given any adequate consideration during this addendum? To proceed with this Addendum without properly fixing a permanent remedy for well owners is a complete injustice to the residents surrounding the plant.

Thank you.

Crystal Young

From: Delmar1st
To: comments.chemours
Subject: [External] public feedback
Date: Tuesday, August 18, 2020 1:38:17 AM

In the benefit of being brief, I have a couple thoughts.
1.) The biggest is simple, the potential fines for all violations should be a higher dollar amount. Enough where profit from production causing the pollution is not profitable. i.e. if the chemical profit is $1M a day of production, the fines should $1.25M per day.
2.) The water levels should match or exceed the cleanliness level of measurement as the new Chemours incinerator (99.9% vs. 99%).
3.) Not only should storm water run off be controlled; but the point of transfer of any chemicals (pre or post Chemours usage). Should have a containment system for any possible spills and a means to clean water entering that system. i.e. like any fuel site containment for spills.
4.) Establish a set of standards for what volume of spillage causes a mandatory report.

VR,

J.M. "Mike" Creager