APPENDIX

STATEMENT OF

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Commissioner of Environmental Protection

BEFORE THE

NEW JERSEY GENERAL ASSEMBLY

Environment and Solid Waste Committee

CONCERNING

Per- and Polyfluoroalkyl Substances (PFAS) Affecting New Jersey Water Supplies

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STATE OF NEW JERSEY
DEPARTMENT OF ENVIRONMENTAL PROTECTION

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Introduction

Chairman Kennedy, and Members of the Committee, my colleagues and I at the Department of Environmental Protection appreciate your interest in and attention to the important public health, safety, and environmental concerns posed by the family of chemicals known as per- and polyfluoroalkyl substances, or "PFAS," and we thank you for the invitation to testify before this Committee.

The Department of Environmental Protection has the distinct honor and privilege of serving New Jerseyans as your environmental and public health regulator, and steward of publicly owned lands and natural resources, but also as your science agency. For many years, DEP's Division of Science Research, Water Resource Management, and Hazardous Site Remediation teams have worked to identify PFAS risks by spearheading global cutting-edge science, developing thoughtful policy around PFAS issues, and holding responsible parties accountable for PFAS pollution. New Jersey has, in fact, set the tone for the nation in responding to the risks of PFAS. We should be so very proud of the work our environmental professionals at the Department have done for us.

Today, I will share with you a few brief scientific findings, a summary timeline of our response to PFAS, and highlight some of the actions that your DEP is taking to address this prolific problem.

What is PFAS?

PFAS is shorthand for a class of synthetic, manmade chemicals that have been used for decades to manufacture water-resistant and stain-resistant products (e.g. fabric treatments, repellents, food container lining), in certain types of firefighting foams and for many other commercial and industrial uses. This class of contaminants is extremely persistent in the environment, they accumulate in the body, and they are toxic. Unlike other persistent, bio-accumulative and toxic compounds, PFAS are unique because they easily dissolve in water, which means they can move readily in water and contaminate surface and groundwaters that are drinking water sources. This is what makes PFAS ubiquitous in the environment around the country. Given their unique, harmful, and persistent properties, PFAS are often referred to as "forever chemicals."

PFAS are also one of several chemicals or chemical classes that the environmental regulatory community refers to as a "contaminant of emerging concern." Contaminants of emerging concern are those that we have not historically identified as presenting environmental or public health concern—like asbestos, lead, or PCBs—substances that this Committee and many Americans would easily identify as harmful. Emerging contaminants are those that we are just beginning to see pose concerns, but for which environmental regulators can lag in managing.

Finding PFAS in New Jersey

The Department completed the nation's first statewide occurrence studies of PFAS in public drinking water in 2006 and 2009. In that study, PFAS were found, generally at low levels, in a high percentage of water systems tested. As a result of this work, New Jersey developed a

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drinking water guidance value of 40 parts per trillion (ppt) in 2007. Between 2013 and 2015, the federal government required certain water systems nationwide, including New Jersey systems, to sample for six PFAS. Results from this sampling event showed that New Jersey had a higher percentage of the occurrence of PFAS than other states. Specifically, New Jersey's occurrence of perfluorooctanoic acid (PFOA) was five times higher; perfluorooctanesulfonic acid (PFOS) was two times higher; and perfluorononanoic acid (PFNA) was ten times higher than the occurrence of these chemicals in other states. These higher results in New Jersey are in part owing to our state's history of PFAS manufacturing, particularly in the southern half of the state.

Human Exposure to PFAS

When drinking water is contaminated, it is a major source of exposure to PFAS. Exposure to even low levels of PFAS in drinking water can be higher than exposure from other common sources of PFAS such as food, food packaging, consumer products, house dust, indoor and outdoor air. Exposure to PFAS from drinking water is largely from ingestion, not from inhalation or absorption through the skin. Other household uses such as showering, bathing, laundry, dishwashing, and rinsing produce is not significant.

Long-chain PFAS such as PFOA, PFOS and PFNA have long human half-lives (time for half of the amount in the body to be excreted) of several years. PFOA, PFOS and PFNA build up in the body over time and remain in the body for many years after exposure has ended. For this reason, risk reduction will not be immediate when consumption of contaminated drinking water is stopped.

PFAS are transferred to breastmilk from the mother. Exposure to PFAS from contaminated drinking water may be higher in infants and children than adults because infants and children consume more fluid, including formula, breast milk, and water, per body weight than older individuals.

There are some special exposure considerations for babies, pregnant and nursing women and women considering or planning on having a child which differ from older children and adults. For bottle-fed babies, bottled water should be used. For nursing babies, despite being exposed to PFAS though breastmilk, the extensive information available on the benefits of breastfeeding are believed to outweigh the potential risk of additional PFAS exposure. Therefore, mothers exposed to PFAS are advised that they should continue to nurse.

For pregnant and nursing women, and women considering or planning on having a child, switching to bottled water or using a home water filter for drinking and cooking will reduce PFAS exposure. However, as mentioned, PFAS are slowly excreted from the body so risk reduction will not be immediate.

For older children and adults, because public water utilities must take prompt action when PFAS exceed the state-mandated Maximum Contaminant Levels (MCLs), no specific bottled or filtered recommendation is made. However, individuals who wish to reduce exposure to PFAS while they wait for the utility to reduce levels, can consider switching to bottled or home filtered water for drinking and cooking.

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Health Effects of PFAS

Studies of PFAS have found a number of different health effects in people. Some health effects have been found consistently in many studies, while there are only a few studies for some other health effects.

For PFOA, which is the most well-studied of the PFAS, the most consistently found health effects are increases in serum cholesterol, some liver enzymes, uric acid levels, and decreased antibody response following vaccination.

For PFOS, also well studied, generally consistent associations were found with increased serum cholesterol and uric acid levels, and decreased antibody response following vaccination.

There are fewer studies of PFNA, but health effects that include increased in cholesterol levels and some liver enzymes have been observed in the general population.

Infants and children may be more sensitive to the effects of PFAS. In humans, exposure to PFAS before birth, infancy, or in early childhood may results in health effects including decreased birth weight, decreased response to vaccination, and increased risk of infectious disease. In laboratory animals, PFAS including PFOA, PFOS and PFNA caused development delays.

These health effects are observed at low levels of exposure, including in the general population without exposure from contaminated drinking water.

PFAS and Cancer

PFOA and PFOS have been found to cause tumors in rodents. PFNA has not been tested for this effect.

In a community with substantial exposure to PFOA through drinking water, PFOA exposure was associated with higher incidence of kidney and testicular cancers.

PFOA was also associated with increased risk of kidney cancer in the general population in a recent study conducted by the National Cancer Institute. More detailed information on cancer studies can be found at NCI "PFAS Exposure and Risk of Cancer"

Increased risk of health effects are related to the level of the PFAS in the blood serum and can occur in a less-than-chronic timeframe.

New Jersey's Regulatory Action on PFAS

As PFAS have emerged as a potential class of contaminants of concern over the last two decades, New Jersey has acted to identify and address PFAS concerns in our environment. For example, New Jersey was the first state in the country to establish a drinking water standard for any PFAS, and now has some of the most stringent water standards in the country.

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Following occurrence studies mentioned earlier, in 2014, the Department called on the expertise of New Jersey's Drinking Water Quality Institute (Institute) to recommend enforceable drinking water standards, also known as a maximum contaminant levels or MCLs. The Institute is an advisory body established by the New Jersey Safe Drinking Water Act, comprised of scientists and technical experts from academia, government, and industry, who make recommendations to the Department regarding drinking water. At that time, the US Environmental Protection Agency (USEPA) offered non-regulatory health guidance for PFOA and PFOS, two of the most frequently detected PFAS; New Jersey was already pursuing its own standards. New Jersey has a long, proud, and necessary history of setting standards to protect our environment and public health that exceed the floor set by the federal government.

In 2018, New Jersey became the first state to establish an MCL for any PFAS chemical when the Department set a drinking water limit of 13 ppt for PFNA. The Department followed this in 2020 with limits for PFOA (14 ppt) and PFOS (13 ppt). These MCLs also serve as New Jersey ground water standards that are used in cleanup of contaminated sites. The Department is currently in the process of developing a standard for the cleanup of other PFAS-contaminated environmental media, such as soil.

The Department is also developing a comprehensive PFAS Action Plan to identify sources of PFAS in water, wastewater, air, and even in fish, and to provide technical and financial support for our regulated entities, including New Jersey's many water systems.

Development of PFAS Drinking Water Standards

The Department and the Institute have extensive experience developing drinking water standards for a wide variety of contaminants, and we have been doing so since the early 1980s. The Institute recommends a health-based MCL, which is the same as recommended MCL whenever the technology exists to detect and treat the chemical at that level. This was the case for PFAS, which could be reliably measured and removed by technology to health-based levels.

New Jersey's evaluations of the health effects of PFOA, PFOS, and PFNA are extremely comprehensive and ground-breaking. In fact, the evaluations were subsequently used by several other states as the basis for their own PFAS drinking water standards. The standards developed using USEPA risk assessment guidance and are protective for both non-cancer and cancer health effects. In fact, the New Jersey Safe Drinking Water Act requires standards to be protective for one in one million risk of cancer from lifetime exposure.

These standards are used in two ways: as a benchmark for testing private wells; and for public water systems that deliver water to New Jersey residents. The monitoring of PFAS for required water systems was phased in over time, beginning with PFNA monitoring in 2019. Although not required, many water systems began sampling and reporting information on all three PFAS at that time because the testing method for PFNA also detects other PFAS, like PFOA and PFOS. the Department encouraged systems to voluntarily report all PFAS results to the Department.

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PFAS Cleanup & Enforcement

In 2019, the Department issued the first-in-the-nation Statewide PFAS Directive under the New Jersey Spill Compensation and Control Act and other foundational DEP authorities seeking important technical information, cleanup action, and compensation from the largest manufacturers and users of PFAS in New Jersey—including 3M, DuPont, Chemours and Solvay. Despite numerous, and in some cases years-long, attempts by DEP to work with these companies to resolve the PFAS contamination that they caused or contributed to, a full and amicable resolution remained elusive. Working with the Attorney General, the Department ultimately filed litigation against these companies to hold them accountable for the damage they have done to New Jersey's environment and to address the public health threat they have created, and the costs that they have effectively transferred to the public.

Occurrence of PFAS in New Jersey Drinking Water Systems

Public Drinking Water Systems

Public drinking water systems are now required to conduct routine monitoring for three PFAS. Monitoring for PFNA commenced in 2020, and monitoring for PFOA and PFOS in 2021. Samples are collected before the water enters the distribution system. As of March 7, 2022, seventy-six (76) New Jersey water systems have had MCL violations for PFNA, PFOA, PFOS, or some combination of the three. PFAS sampling results are publicly available on the DEP Drinking Water Watch website at https://www9.state.nj.us/DEP_WaterWatch_public/.

Once a violation is issued, the water system is required to take several actions including public notification within 30 days. Public water systems have one year to return to compliance, which could mean finding alternative source for their water or installing appropriate treatment technologies. Though some systems may elect to take interim measures, such as shutting off the contaminated source, until a permanent solution has been reached, water systems are required to continually issue Public Notice on a frequency determined by the Department. The Department works with water systems to take steps to minimize exposure to the public while the water system works to install permanent solutions.

In some cases, water systems are able to implement temporary measures such as ceasing use of the contaminated source and rely on their other sources to provide water to customers or purchase water from a neighboring water system. For some systems, however, the only option is to install permanent treatment, which takes time for design and construction. The most commonly used treatment for PFAS is granular activated carbon, although there are other treatments that are considered effective for PFAS, such as ion exchange and reverse osmosis. In New Jersey, there are systems that began the time-consuming process of designing and installing appropriate treatment before the regulations went into effect, which is why some systems are already running treatment systems and others have completed design.

Currently, the Department has reviewed approximately two dozen permit applications for treatment and have been in contact with other water systems that are currently designing their treatment.

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Consumers that are concerned can opt to use bottled water or home filtration devices (e.g., filtered pitchers, under-the-sink filters, etc.) that are NSF certified to remove PFAS.

Private Drinking Water Wells

The testing of private wells is important because 13% of New Jerseyans receive their water from private wells. Under the Private Well Testing Act (PWTA), the Department adopted rules that requires private well owners to test for PFNA, PFOA, and PFOS in addition to other parameters at the time of sale or every 5 years for rental units as of December 1, 2021. When a private well sample shows a contaminant value above an MCL, a notification is made to the County Health Agency. In some situations, the local health authority has the discretion to notify the reported presence of a PWTA parameter in a private well to nearby well owners to test for the parameter of concern. Under the PWTA rules, the local health authority may not reveal the address or location of the impacted residence.

Financial Assistance

To assist New Jersey water systems, funding is available for PFAS treatment through the New Jersey Water Bank, a very successful program leg by the Department in partnership with the New Jersey Infrastructure Bank. Through this program, we work to provide low-cost financing for the design, construction, and implementation of projects that help protect and improve water quality and help ensure safe and adequate drinking water.

The Department is currently in the process of preparing its Water Infrastructure Investment Plan (WIIP) for state fiscal year 2023. The WIIP is funded by capitalization grants from the USEPA, state appropriations for water infrastructure appropriations from general fund, and loan repayments received by DEP. Over the next five years, the program will receive additional funding from the historic Bipartisan Infrastructure Law (BIL). Under the BIL, New Jersey will be receiving approximately $65 million dollars in principal forgiveness or grants, specifically for emerging contaminants, such as PFAS over the next 5 years. Water systems eager for funding to address PFAS risks or any other water infrastructure needs, can apply today. Those interested should visit our website at www.nj.gov/dep/wiip.

Conclusion

In conclusion, I want to thank you, Chairman Kennedy, and all members of the Committee for your time and attention to this important issue. PFAS pose a significant risk to public health, safety, and the environment; the cleanup of these forever chemicals works a tremendous cost burden upon the State, forces our local governments and water utilities to invest significant capital on contaminant removal that could be well-spent on necessary water infrastructure improvements, and represents an affordability burden upon our residents and water ratepayers who will ultimately bear the cost of the much-needed cure. It was my pleasure to share DEP's knowledge and actions with you here today, and my colleagues and I stand ready to assist this Committee and our broader Legislature in addressing this challenge for the benefit of the public we serve. Thank you.

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Review article

Review: Evolution of evidence on PFOA and health following the assessments of the C8 Science Panel

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ABSTRACT

Background: The C8 Science Panel was composed of three epidemiologists charged with studying the possible health effects of PFOA in a highly exposed population in the mid-Ohio Valley. The Panel determined in 2012 there was a 'probable link' (i.e., more probable than not based on the weight of the available scientific evidence) between PFOA and high cholesterol, thyroid disease, kidney and testicular cancer, pregnancy-induced hypertension, and ulcerative colitis.

Objective: Here, former C8 Science Panel members and collaborators comment on the PFOA literature regarding thyroid disorders, cancer, immune and auto-immune disorders, liver disease, hypercholesterolemia, reproductive outcomes, neurotoxicity, and kidney disease. We also discuss developments regarding fate and transport, and pharmacokinetic models, and discuss causality assessment in cross-sectional associations among low-exposed populations.

Discussion: For cancer, the epidemiologic evidence remains supportive but not definitive for kidney and testicular cancers. There is consistent evidence of a positive association between PFOA and cholesterol, but no evidence of an association with heart disease. There is evidence for an association with ulcerative colitis, but not for other auto-immune diseases. There is good evidence that PFOA is associated with immune response, but uneven evidence for an association with infectious disease. The evidence for an association between PFOA and thyroid and kidney disease is suggestive but uneven. There is evidence of an association with liver enzymes, but not with liver disease. There is little evidence of an association with neurotoxicity. Suggested reductions in birthweight may be due to reverse causality and/or confounding. Fate and transport models and pharmacokinetic models remain central to estimating past exposure for new cohorts, but are difficult to develop without good historical data on emissions of PFOA into the environment.

Conclusion: Overall, the epidemiologic evidence remains limited. For a few outcomes there has been some replication of our earlier findings. More longitudinal research is needed in large populations with large exposure contrasts. Additional cross-sectional studies of low exposed populations may be less informative.

1. Introduction

From 2004 to 2012, the 3-member C8 Science Panel, consisting of Tony Fletcher, David Savitz, and Kyle Steenland, was tasked by a West Virginia court with determining whether there was a 'probable link' between perfluoroctanoate (PFOA; referred to as C8 due to the 8-carbon structure) and any human disease (C8 Science Panel 2012, Steenland et al., 2014).

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The Science Panel's work was part of a legal settlement between community plaintiffs and DuPont, who had manufactured Teflon for 50 years near Parkersburg, West Virginia in the mid-Ohio Valley. Teflon production utilized PFOA during tetrafluoroethylene polymerization, and in the process of making Teflon, DuPont released PFOA into both the air and the adjacent Ohio River. PFOA eventually entered the groundwater and contaminated community drinking water supplies in the region (Shin et al. 2011a).

In the legal settlement (Leach et al. v. E.I. du Pont de Nemours & Co , 2005) 'probable link' was defined to mean that it was deemed more probable than not that the exposure had contributed to adverse disease outcomes (C8 Science Panel 2012). A disease was determined to have a 'probable link' with PFOA in the local community if the Science Panel (unanimously or by majority decision) considered that "based upon the weight of the available scientific evidence, it is more likely than not that there is a link between exposure to PFOA and a particular Human Disease among Class Members". In reaching their conclusions, the Science Panel considered epidemiological data published by other investigators, and data generated by the Panel in new studies of this population, with the latter given more weight.

The Science Panel conducted 11 different epidemiologic studies, most of which were dependent on estimating historical serum PFOA concentrations in Ohio Valley residents over time, via known plant emissions, a fate and transport model, and known residential history (C8 Science Panel 2012b). The exposure model predictions correlated well ($r = 0.69$) with observed measurements for 69,000 exposed residents in 2005/2006. Longitudinal studies were conducted, both retrospective and prospective, and self-reported disease was validated via medical records. Our population had both very high exposure and also low exposure, an advantage, although we could not explore effects using a non-exposed referent comparable to the general US population. After six years of study, probable links were declared for kidney and testicular cancer, pregnancy-induced hypertension, thyroid disease, high cholesterol, and ulcerative colitis (Table 1) (C8 Science Panel 2012a).

We compare more recent findings to our original findings not because we view our work as a gold standard, but because our probable link assessments provide a notable benchmark in the evolution of epidemiologic research on the health effects of PFOA exposure. The studies that we conducted remain important as the first large scale longitudinal study of a population with high exposure contrasts. Observations on the evolution of methods and findings since the C8 Science Panel's original assessment provides an informative way to organize the research that has followed.

Table 1
Diseases found by the C8 Science Panel to have a probable link with PFOA.

Disease with probable link[a]	Principal supporting publications from C8 Science Panel
Kidney cancer	Barry et al., 2013
Testicular cancer	Barry et al., 2013
Pregnancy-induced hypertension	Savitz et al. 2012a, Savitz et al. 2012b, Darrow et al. 2013
Thyroid disease	Winquist and Steenland 2014b
High cholesterol	Winquist and Steenland, 2014a; Fitz-Simon et al., 2013
Ulcerative colitis	Steenland et al. 2013

Note: A disease was determined to have a Probable Link with PFOA in the local mid-Ohio community studied by the Science Panel, if the Science Panel considered that "given the scientific evidence available, it is more likely than not that a connection exists between C8 exposure and a particular human disease among class members" (C8 Science Panel 2012).

[a] To see the probable link judgments in full, see C8 Science Panel 2012a, to see the list of the 11 studies conducted by the Science Panel, see C8 Science Panel 2012b.

2. Methods

We examined the studies included in three comprehensive recent reviews of the health effects of PFAS, by the International Agency for Research on Cancer (IARC 2018), Agency for Toxic Substances and Disease Registry (ATSDR 2018), and the European Food Safety Authority (EFSA) (EFSA Contamination Panel . 2018). We also examined other reviews devoted to specific aspects of PFOA health effects (Roth and Wilks 2014, Vrijheid et al. 2016, Ballesteros et al. 2017, Coperchini et al., 2017, Rappazzo et al. 2017, Liew et al., 2018a, Sunderland et al. 2019). We performed additional PubMed searches using keywords 'PFAS', and 'PFOA' for the period 2011 onward, after our Science Panel work concluded.

In this commentary we consider the overall strength of epidemiologic evidence for PFOA-disease associations now that some years have elapsed since the C8 Science Panel's work concluded in 2011–2012. We focus on thyroid disorders, cancer, immune and auto-immune disorders, liver disease, hypercholesterolemia, reproductive outcomes, neurotoxicity, and kidney disease. This re-assessment does not revisit the probable link decisions but re-states them, and then considers more recent evidence, including for some outcomes for which the Science Panel did not determine that there was a link to PFOA. We also discuss the Science Panel's exposure estimation, comment on study design issues, and suggest future epidemiologic research priorities for PFOA, and indirectly, other PFAS.

We reviewed the literature though March 1, 2020. Although we did not methodically search for new literature after March 1, 2020, we did come across an important new report on kidney cancer, which supports an association; hence we have included it here.

3. Results

3.1. Cancer

Overall, we found 19 epidemiologic studies of PFOA and cancer, six of which were of occupational cohorts (of which two were updates of the original cohorts) (see Table S1). In 2012, the Science Panel concluded that there was a probable link between PFOA and both testicular and kidney cancers (C8 Science Panel 2012c). The modest evidence that has accumulated since that time does not generally strengthen the conclusion that PFOA is carcinogenic for any given site, also there is somewhat stronger evidence for kidney cancer.

Despite these caveats, we believe the evidence for an association of PFOA with testicular cancer is suggestive overall, with two Science Panel studies, one cohort (Barry et al. 2013), one ecological/case-control (Viera et al. 2013), having found a relatively strong positive exposure–response for this cancer, supported by animal studies (ATSDR 2018). Testicular cancer is rare and generally not fatal (American Cancer Society 2020), and only Science Panel studies have reported on its relation to PFOA, limiting conclusions (Leonard et al. 2008 also reported on testicular cancer mortality but had only 1 death). The evidence for kidney cancer, also implicated by the Science Panel in their community (Barry et al., 2013) and worker (Steenland and Woskie 2012) cohorts, also remains suggestive although not consistent in newer studies. Kidney cancer was not found in a high-exposure occupational cohort of 3 M workers (Raleigh et al. 2014) based on mortality or incidence, although the number of kidney cancers was small (16 exposed incident cases). Mastrantonio et al. (2017), in an ecologic study in the Veneto region of Italy, comparing areas with PFOA-contaminated drinking water (as well as some other PFAS) with areas with non-contaminated water, did not find an excess of kidney cancer overall, although some excess among women (SMR 1.32(95% CI 1.06–1.65)). Finally a recent population-based case-control study by US National Cancer Institute investigators, with 324 renal cancer cases and 324 individually matched controls, found a positive exposure–response trend with renal cancer for several PFAS including PFOA, perfluorooctane sulfonic acid, and

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perfluorohexane sulfonic acid. Only the association with PFOA remained apparent after adjustment for all three chemicals (Shearer et al. 2020). It should be noted that this general population study had much lower exposure contrasts than other studies.

There is little evidence for a relationship of PFOA with either liver or pancreatic cancer, tumors of which are associated with PFOA in animal studies (ATSDR 2018), with the exception of an increased risk for liver cancer , recently seen in Italian workers exposed occupationally to PFOA (Girardi and Merler, 2019). In our view there is some suggestive evidence for prostate cancer (positive Lundin et al. 2009; some suggestion Vieira et al. 2013, Hardell et al. 2014 , Steenland et al. 2015; largely negative Ericksen et al. 2009, Steenland and Woskie 2012, Barry et al., 2013), but results are inconsistent. This inconsistent evidence has led other reviewing bodies such as IARC (IARC, 2018), ATSDR (ATSDR 2018), and EFSA (EFSA Contamination Panel 2018), to similarly conclude the evidence for PFOA carcinogenicity remains suggestive but not conclusive.

To our knowledge there have been no studies of childhood cancer, but the effects of PFOA on the immune system (see below) might suggest these would be appropriate.

3.2. PFOA in relation to cholesterol and heart disease

The Science Panel concluded in 2012 that there was a probable link between PFOA and serum cholesterol (C8 Science Panel, 2012a). In 2018, the EFSA Panel on Contaminants in the Food Chain concluded that human epidemiological studies provide strong support for causal associations between exposure to PFOS and PFOA and increased serum levels of cholesterol (EFSA Contamination Panel 2018).

There have been numerous cross-sectional studies of associations between PFOA and lipid markers, most finding a clear positive association between serum PFOA and total cholesterol (TC) or low-density (LDL) cholesterol and a minority with positive associations with high-density lipoprotein (HDL) and triglycerides (ATSDR 2018). The few studies reporting the TC/HDL ratio do not show a clear pattern (one positive in a community population (Steenland et al. 2009), another study with workers and community (Wang et al. 2012), being negative or null, respectively). Most studies have focused on total cholesterol, LDL and HDL, though one recent study highlighted a possible association with lipoprotein subfractions, apoC-III in particular (Liu et al. 2020), and more work on such subfractions may help in elucidating the mechanisms of action of PFAS on cholesterol.

The cross-sectional studies have been carried out in three contexts, with positive results in all of them: general population samples with mean PFOA less than 5 ng/mL (for example Nelson et al. 2010), occupational studies with most serum levels above 1000 ng/mL (for example Sakr et al. 2007), and polluted community samples such as the C8 studies, with mean serum PFOA approximately 80 ng/mL (eg., Steenland et al. 2009, Frisbee et al. 2010). The lower the range of PFOA that was studied, the greater the change in cholesterol per unit change in PFOA (Steenland et al. 2010). This observation might reflect a non-linear dose–response relationship, with a stronger effect per unit of exposure in the lower exposure range

The positive association could reflect confounding, if for example regulation of serum level of both PFOA and cholesterol was correlated. Inter-individual variation in enterohepatic cycling of both PFAS and bile acids, the latter affecting serum cholesterol levels, has been postulated as a mechanism for such a correlation between PFAS and cholesterol (EFSA Contamination Panel 2018). Some observations lend support to this view. Correlation between PFAS and cholesterol excretion has been shown in patients with high levels of PFOS, another long chain PFAS, who were given cholestyramine, a drug known to reduce cholesterol, and which led to a sharp decrease in PFOS (Genuis et al., 2014).

If cross sectional studies were the only available evidence, major doubts would remain about the association. However, the same association has been seen in other designs. In a study in Sweden with high mixed exposure to several PFAS, principally PFOS, PFHxS and PFOA, associations with cholesterol were evident in both cross sectional analyses and ecological analysis comparing high and low exposure communities (Li et al. 2020). A longitudinal Science Panel study of 560 adults with repeated serum PFOA, PFOS and cholesterol measurements demonstrated an association between the magnitude of the decline in PFOA or PFOS and fall in cholesterol or LDL, suggesting a reversible effect of PFOA on cholesterol levels (Fitz-Simon et al., 2013). In a different design investigating a diagnosis of raised cholesterol in a large, highly exposed cohort, there was a positive association between modeled cumulative serum PFOA concentrations prior to diagnosis and cholesterol levels. The modeled level was based on estimated intake so there was no concern of correlated excretion (similar to other cohort analyses in this population) (Winquist and Steenland, 2014). A longitudinal study over 15 years of hypercholesterolemia and hypertriglyceridemia in relation to baseline PFAS found positive associations of both with PFOA (Lin et al. 2019).

The increase in LDL cholesterol in relation to PFOA might have been expected to manifest with a concomitant increase in cardiovascular disease risk. The only longitudinal study of cardiovascular disease incidence in relation to prior PFAS exposure is the C8 study of (modeled) PFOA and the results were null (Winquist and Steenland, 2014a). The explanation for this apparent anomaly may be explained by the other associations observed. There is some although inconsistent evidence for a positive association of PFOA (Steenland et al, 2009, see Table 4) with HDL, and also for PFOS (Chateau-Degat et al, 2010, Frisbee et al. 2010) and HDL, in certain populations; an increase of HDL has in turn been associated with a reduced risk of cardiovascular disease. Analysis of data from the C8 Health Project indicated a negative association between PFOA and C-reactive protein (Genser et al. 2015), lower levels of which are also associated with reduced risk of heart disease. Thus, it is plausible that there is a positive association of PFOA with raised cholesterol, yet no impact on the risk of cardiovascular disease.

3.3. Non-malignant thyroid disease and thyroid hormones

3.3.1. Thyroid disease

The Science Panel concluded in 2012 that there was a probable link between PFOA and thyroid disease (C8 Science Panel 2012d), primarily based on its own study of 32,000 participants in the mid-Ohio Valley (Winquist and Steenland, 2014b, published after the probable link judgment but used in the decision). Combining hypo- and hyper-thyroid diseases, a significant trend of increasing risk was observed among adult females in relation to both cumulative and serum level at diagnosis. The clearest trend was for female hyperthyroidism in relation to serum PFOA at the time of diagnosis. A parallel study focused on Dupont Plant workers (n = 3713; 80% male), published a few years after the probable link judgment), reported a trend of increasing thyroid disease risk across quartiles of modeled serum PFOA (with a 10 year lag) for males, but no evidence of trends with the log of cumulative exposure for either males or females (Steenland et al. 2015). Another study by the Science Panel found that measured PFOA child serum levels were associated with a higher risk of thyroid disease (mostly hypothyroidism, n = 61). However, serum PFOA was not associated with subclinical hypo- or hyperthyroidism based on cross-sectional analyses of individual hormone levels (Lopez-Espinosa et al. 2012).

Subsequent to the C8 Science Panel studies, thyroid disease was studied in a community in Sweden, with PFAS exposure from fire-fighting foam contamination of drinking water (Andersson et al 2019). Community members in the contaminated area had median serum levels of 257 ng/mL (PFHxS), 280 ng/mL (PFOS) and 15 ng/mL (PFOA), being approximately 300, 60 and 10 times higher respectively, than general population levels in unexposed areas. Individuals were linked to registers providing diagnoses and prescriptions for hypo- and hyperthyroidism. The study period was divided into three periods of increasing PFAS exposure and cases were assigned to the exposure period at time of

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diagnosis. Neither hypo- nor hyper-thyroidism exhibited evidence of a trend of increasing risk across exposure categories.

Three recent reviews noted that the literature is scarce, and the type of thyroid disease-related outcome assessment varied considerably across studies, with few studies of pregnant women or children (Ballesteros et al. 2017, Coperchini et al. 2017, EFSA, 2018). Two of these judged that there was insufficient evidence to draw a conclusion on PFOA and thyroid disease (Ballesteros et al. 2017, EFSA Contamination Panel 2018), while the third one (Coperchini et al. 2017) reported hypothyroidism as the most consistent finding across studies. Overall, since the original Science Panel findings, our view is that the evidence of an association of PFOA with thyroid disease has gotten weaker.

3.3.2. Thyroid hormones

There were two key studies regarding thyroid hormones that were considered in the original Science Panel probable link judgment (C8 Science Panel 2012d), which concluded there was no consistent evidence of an association between PFOA and thyroid function (Knox et al. 2011, Lopez-Espinosa et al. 2012).

According to a systematic review (Ballesteros et al. 2017), three out of seven studies on the association between prenatal PFOA exposure, and either maternal or child hormones, found positive associations (note, Knox et al. (2011) was omitted from this review). The timing of the studies varied (at birth, first days of life, childhood) as did the type of hormones (one study found an association with TSH; two studies with TT4), so there were no consistent patterns across studies. A *meta*-analysis of 11 thyroid hormone studies in adults (including pregnant women) reported a modest inverse association between PFOA and TT4 but not TSH, free T4 (FT4) or total triiodothyronine (TT3) (Kim et al. 2018). EFSA (2018) concluded there was insufficient support for causal associations between exposure to PFOA and changes in thyroid hormones in adults based on 20 epidemiological studies. A similar conclusion of insufficient evidence was also reported in a prior (Coperchini et al. 2017).

Overall, while a number of studies have suggested associations between thyroid hormones and PFOA in cross-sectional analyses, in our view there is little consistency across studies so evidence for a causal impact on thyroid hormones remains weak.

3.4. Immunotoxicity

In 2012, the Science Panel concluded that there was not a probable link between PFOA exposure and common infections in children or adults (C8 Science Panel 2012e, Looker et al., 2014). Despite some evidence from the C8 Health Study (2005/2006) that higher PFOA exposure was associated with lower influenza vaccine efficacy and lower clinical markers of the immune system, there were also indications that higher PFOA exposure was associated with fewer infections in children and adults (Looker et al., 2014). At the time of the probable link report there was insufficient evidence from either Science Panel or other epidemiological studies (Fei et al. 2010, Grandjean et al. 2012, Okada et al., 2012) to infer a probable link between PFOA exposure and immune function.

Since 2012, however, toxicological, animal, and human epidemiological studies have continued to examine links between PFOA exposure and immunotoxicity related to both immunosuppression (e.g., vaccine response, infection) and hypersensitivity (e.g., asthma, allergy). In 2016, the U.S. National Toxicology Program (NTP) concluded "PFOA is presumed to be an immune hazard to humans based on a high level of evidence that PFOA suppressed the antibody response from animal studies and a moderate level of evidence from studies in humans… there is additional, although weaker, evidence that is primarily from epidemiological studies that PFOA reduced infectious disease resistance [and] increased hypersensitivity-related outcomes" (NTP 2016). The U.S. ATSDR's 2018 public comment draft Toxicological Profile for Perfluoroalkyls listed decreased antibody response to vaccines and increased risk of asthma diagnosis among PFOA's suggested human health effects (ATSDR, 2018). Also in 2018, the EFSA Panel on Contaminants in the Food Chain concluded that associations between PFOA exposure and reduced serum antibody vaccine response were likely to be causal, although there was little evidence to suggest that PFOA was associated with asthma and allergies in children or adults (EFSA Contamination Panel 2018). In contrast, a 2016 review determined that there was insufficient evidence of a causal association between PFOA and immune function in humans (Chang et al. 2016). The most recent reviews, however, support the conclusions of the NTP, ATSDR, and EFSA that PFOA has immunosuppressive potential (Rappazzo et al. 2017, Liew et al., 2018a, DeWitt et al. 2019).

Studies published in recent years primarily focus on outcomes related to asthma (Zhou et al. 2017, Zhou et al. 2017, Impinen et al. 2018, Impinen et al. 2019) and atopic dermatitis (Chen et al. 2018, Impinen et al. 2018, Impinen et al. 2019, Wen et al. 2019) with only two studies examining PFOA exposure in relation to infection (Impinen et al. 2018, Impinen, Longnecker et al. 2019) and one study in relation to Rubella antibody titers (Pilkerton et al. 2018). We believe these recent studies provide little support for an association between PFOA exposure and asthma or atopic dermatitis among children. Results of associations between PFOA exposure and childhood infection are mixed, with studies reporting both increased and decreased associations with parent report of infections in their children (Impinen et al. 2018, Impinen et al. 2019). We believe these newest studies do little to alter the conclusions on PFOA and immunotoxicity previously made by several federal agencies.

In summary, a relatively large number of studies consistently report that PFOA impairs immune function; evidence that PFOA increases risk of human infectious disease or asthma is inconsistent.

3.5. Reproductive outcomes

In 2012 the Science Panel concluded that there was not a probable link between PFOA and birth defects, miscarriage, stillbirth, preterm birth or low birth weight, while there was a probable link to pregnancy-induced hypertension (C8 Science Panel, 2012a). Below we review the cumulative evidence for these outcomes, some of which have received substantial attention with respect to potential impact of PFOA (e.g., birthweight) and others very little (e.g., miscarriage).

There were initial suggestions of higher PFOA levels being associated with a slightly prolonged time to pregnancy (Fei et al. 2009, Velez et al. 2015) as well as reports of decreased sperm count and quality with higher PFOA exposure (Joensen et al. 2009, Vested et al. 2013), but subsequent studies did not support an adverse effect on fecundability (Jorgensen et al. 2014, Bach et al. 2015). In our view, cumulatively the evidence on PFOA provides little support for a consistent or substantial effect on fecundability.

Miscarriage has been considered in few studies, including several from the C8 Health Project (Stein et al. 2009, Savitz et al 2012a, Darrow et al, 2014) as well as in other populations (Jensen et al., 2015, Buck-Louis et al. 2016). With minor exceptions, the associations have been consistently null, indicating the absence of a discernible impact of PFOA on risk of miscarriage.

Hypertensive disorders of pregnancy were judged to have a probable link by the C8 Science Panel based solely on a series of analyses conducted in that population that showed modest but reasonably consistent increases in risk with elevated PFOA exposure (Savitz et al. 2012a, Savitz et al. 2012b, Darrow et al. 2013). Subsequent analyses found that those conclusions were relatively insensitive to potential errors in our exposure and toxicokinetic models (Avanasi et al. 2016a, Avanasi et al. 2016b, Avanasi et al. 2016c). We identified two studies since the C8 Science Panel study (Huang et al. 2019, Wikstrom et al., 2019). Wikstrom et al. (2019) provided supportive evidence of an association between PFOA and preeclampsia (adjusted OR per log unit increase = 1.3 (0.9–1.8) whereas Huang et al. (2019) did not identify an association.

The most extensive body of research pertains to PFOA exposure

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during pregnancy and birthweight, with the most recent *meta*-analysis identifying 24 pertinent studies (Steenland et al. 2018), and with 12 additional studies in the short interval that has followed (see Table S2). Most earlier *meta*-analyses (Johnson et al. 2014, Negri et al, 2017), as well as Steenland et al. (2018), identified an association between level of PFOA in maternal serum or cord blood and a modest reduction in infant birthweight, generally on the order of 10 g per ng/mL. While the absolute magnitude of reductions identified would not be of direct clinical significance for an individual neonate, there would likely be an impact at the population level from reduced infant size at birth. However, this association may be in part a reflection of reverse causality (the outcome itself alters the measured exposure, rather than the other way around) or confounding, related to plasma volume expansion which is associated with birthweight and likely with reduced serum PFOA. Reverse causality or confounding would be most likely to affect studies with low exposure contrasts. Including studies with the widest range of exposure from the C8 Health Project (Stein et al. 2009, Savitz et al. 2012a,b, Darrow et al. 2014) would have effectively reduced the prior pooled estimate of effect to the null (Steenland et al. 2018). Furthermore, studies with PFOA measurement later in pregnancy show stronger associations with birth weight than those with measurements earlier in pregnancy, consistent with the possibility that the overall association is distorted by the magnitude of plasma blood volume expansion and glomerular filtration rate (Steenland et al. 2018, Verner et al. 2015).

More recent studies continue to generate mixed findings, some suggesting a reduced birthweight associated with elevated PFOA and others not finding evidence for such an effect.

Preterm birth has not been the focus of as much research. The studies from the C8 Health Project generated null findings (Stein et al. 2009, Savitz et al. 2012a, b, Darrow et al. 2014). A recent report from the Danish National Birth Cohort did identify elevated risk of preterm birth in the highest PFOA exposure ranges (Meng et al., 2018), but with little corroborative support. Similarly, congenital defects have received little attention and the studies that have been done (Stein et al. 2014a, Vesterholm et al. 2014) generated null findings.

In summary, with the exception of reduced birthweight, discussed in detail above, there have been few studies on other reproductive endpoints (subfecundity, miscarriage, preterm birth, and birth defects). What studies do exist provide little indication of an adverse effect of PFOA. For the one outcome for which the Science Panel did find a probable link, pregnancy-induced hypertension, there are few subsequent studies with mixed results.

3.6. Auto-immune disease

The Science Panel concluded that there was a probable link between PFOA and ulcerative colitis, but not with any other auto-immune disease, including Crohn's disease, the other principal inflammatory bowel disease (C8 Science Panel 2012f). The conclusion of the Science Panel was based on their own community cohort study of 32,000 adults (Steenland et al. 2013). The evidence of an association between PFOA and ulcerative colitis in that cohort in internal exposure–response analyses was among the strongest seen in Science Panel studies, and was consistent between the retrospective (extending back to first exposure, prior to original cohort enrollment in 2005/2006) and prospective (beginning in 2005/2006 at time of original cohort enrollment) components of the cohort study. The Science Panel study of PFOA-exposed workers at DuPont also found an association with ulcerative colitis; the workers in that study represented 12% of the larger community cohort (Steenland et al. 2015). There was a positive finding for rheumatoid arthritis in this worker cohort, but this was contradicted by a null finding in the large community cohort. Since the Science Panel studies, there have been two other studies of the PFOA/ulcerative colitis association. One was a case-control study (Steenland et al. 2018a) in which investigators found higher serum PFOA among cases compared to controls, but findings were limited because PFOA was measured after diagnosis. A recent study of inflammatory bowel disease (Xu et al. 2019), including specifically ulcerative colitis and Crohn's disease, was conducted in an area of Sweden where public water had been contaminated from fire-fighting foam). The main contamination was from PFHxS and PFOS, although there was also an increase in PFOA compared to non-exposed areas. Community members in the contaminated area had median serum levels in 2014, shortly after exposure ended, of 257 ng/mL (PFHxS), 280 ng/mL (PFOS) and 15 ng/mL (PFOA) (Andersson et al 2019). Inflammatory bowel disease was ascertained by linking the registered residential population to registries of diagnoses and prescriptions. No increase in ulcerative colitis, Crohn's disease, or any inflammatory bowel disease was seen comparing the exposed population (n = 60,000) to an adjacent non-exposed population, nor was there any trend in increased risk over exposure period (reflecting increasing cumulative PFAS emissions).

Overall, based on the four published studies to date, we believe the evidence still supports an association of PFOA with ulcerative colitis. Both Science Panel studies of PFOA found strong exposure–response trends. On the other hand, the latest study from Sweden did not find a positive association. However, the Swedish study had a different exposure profile: the population was highly exposed to PFHxS and PFOS, while PFOA was moderately elevated, lower on average than in the mid-Ohio valley. Furthermore, exposure was assigned ecologically based on residential location, and exposure response analyses were conducted based only across decade of increasing exposure. Given the sparse literature, more studies are clearly needed to reach more definitive conclusions.

3.7. Non-malignant liver disease and biomarkers of liver function

3.7.1. Liver disease

In 2012, the Science Panel concluded there was not a probable link between PFOA and liver disease (CS Science Panel 2012h), primarily based on its own study in the mid-Ohio Valley (Darrow et al. 2016). Overall in the literature, there have been five epidemiologic studies of PFOA and incident liver disease, liver disease mortality, or biopsy-determined prevalent fatty liver disease conducted to date. Three studies were conducted among DuPont or 3 M workers with small numbers of cases (<=35) (Lundin et al. 2009, Steenland and Woskie 2012, Steenland et al. 2015); one study was conducted in the community living near the DuPont Washington Works plant (Darrow et al, 2016); and one cross-sectional study based on liver biopsies was conducted among gastric bypass patients (n = 105) in Finland (Rantakokko et al. 2015). Outcomes included all chronic liver diseases (Steenland and Woskie 2012, Darrow et al. 2016), non-hepatitis liver diseases (fatty liver disease, enlarged liver and cirrhosis combined) (Darrow et al. 2016, Steenland et al, 2015), cirrhosis (Lundin et al, 2009) and steatosis or non-alcoholic steatohepatitis (NASH) (Rantakokko et al. 2015). The Steenland et al. (2015) study was a study of disease incidence among a cohort of DuPont workers exposed to PFOA (a subset of the community cohort studied in Darrow et al. 2016), and found a suggestion of a positive association between PFOA and non-hepatitis liver disease, but only when using a 10-year lag of PFOA exposure, and estimated rate ratios were highly imprecise because of the small numbers of cases among workers. There was no evidence of an association in the larger community study, which included 427 cases of fatty liver, enlarged liver, and cirrhosis grouped together for analysis. In the cross-sectional study of gastric bypass patients, Rantakokko et al. (2015) examined serum levels of PFAS in relation to prevalence of simple steatosis (fatty liver with inflammation, n = 24) or non-alcoholic steatohepatitis (NASH, n = 35), measured via liver biopsies of obese patients with background levels of exposure; no association was observed (specific estimates not provided). The Rantakokko et al. study (2015) is the only study data that was unavailable to the Science Panel in 2012. In our view, the limited available data continues to support a conclusion of no probable link between PFOA and liver disease.

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3.7.2. Biomarkers of liver function

In 2012, the Panel concluded there was sufficient support for a causal association between PFOA and increased serum levels of the liver enzyme alanine transferase (ALT), a marker of hepatocellular damage, based largely on its own findings (Gallo et al. 2012). This association has been observed in populations with high occupational exposures (Sakr et al. 2007), in populations experiencing background level exposures such as NHANES (Jain and Ducatman 2019, Gleason et al. 2015, Lin et al., 2010), and in the community living near the DuPont Washington Works plant who experienced a wide range of exposures (Gallo et al. 2012, Darrow et al, 2016). Importantly this positive association is still evident when entirely based on external measures of PFOA exposure (i.e., modeled as opposed to measured serum levels), ruling out the possibility that the positive association is attributable to reverse causality or confounding (e.g., poor liver function causing poor PFOA elimination or sharing a pathway affecting PFOA storage or excretion) (Darrow et al. 2016). In the Gallo et al. (2012) and Darrow et al. (2016) studies, PFOA was positively associated with ALT levels above the reference range, although the clinical relevance of this elevation in enzyme levels is not clear. Adding to the evidence that PFOA causes liver cell injury, a recent study of 200 C8 Health Study adult participants showed that PFOA exposure was associated with cytokeratin 18 M30, a marker of hepatocyte apoptosis (Bassler et al. 2019), and a mechanism of disease progression in nonalcoholic fatty liver disease. There is also evidence that effects on ALT are more pronounced among obese subjects, who are at higher risk of nonalcoholic fatty liver disease (Lin et al. 2010, Jain and Ducatman 2019). Two other markers of liver function commonly assessed, γ-glutamyltransferase (GGT, an early marker of cholestatic liver disorders) and bilirubin have shown inconsistent patterns of association with PFOA (Sakr et al. 2007, Lin et al. 2010, Gallo et al. 2012, Gleason et al. 2015, Darrow et al. 2016,).

The EFSA Panel on Contaminants in the Food Chain March 2018 report (EFSA Contamination Panel 2018) provides a thorough overview of the studies of liver function biomarkers in relation to PFOA and PFOS (see Table 24).

Overall, in our view, the limited existing evidence does not support a link between PFOA and diagnosed liver disease. However, the dearth of adequately powered epidemiologic studies of liver disease, the established liver toxicity of PFOA in experimental animal studies (EFSA Contamination Panel 2018), the storage of PFOA in liver tissue in humans, and extensive evidence that PFOA exposure is associated with markers of hepatocyte cell death, warrants additional research on PFAS and liver disease, particularly nonalcoholic fatty liver disease.

3.8. Neurotoxicity

At the time of the Science Panel's probable link findings in 2012 (C8 Science Panel 2012g), there were four published epidemiologic studies examining associations between developmental exposure to PFOA and neurobehavioral outcomes in children (Fei et al. 2008, Hoffman et al. 2010, Fei and Olsen 2011, Gumpet al. 2011), plus three studies conducted in the C8 Health Project (Stein et al. 2011, Stein et al. 2013, Stein et al. 2014b). The Science Panel concluded that there was no probable link between exposure to PFOA and neurodevelopmental disorders in children, including attention deficit disorders and learning disabilities.

Since that time, numerous studies as well as seven reviews (Bellinger 2013, Roth and Wilks 2014, Vrijheid et al. 2016, Rappazzo et al. 2017, EFSA Contamination Panel 2018, Liew et al., 2018a, Sunderland et al. 2019) have reported on associations between prenatal and/or early childhood exposure to PFOA and various markers and measures of neurotoxicity, such as developmental milestones, attention-deficit/hyperactivity disorder (ADHD), Autism Spectrum Disorder and other behaviors in childhood, and neuropsychological functioning. Despite some compelling data from toxicological and animal studies (summarized in EFSA Contamination Panel 2018), these seven reviews concluded that existing human studies provide no consistent evidence for an association between prenatal and/or early childhood PFOA exposure and adverse neurodevelopmental outcomes in children.

To date, there are an additional nine published studies that were not included in at least one of the seven reviews. Four studies were based in populations previously unexamined for PFOA and neurodevelopment (Jeddy et al. 2017, Ghassabian et al. 2018, Harris et al. 2018, Lyall et al. 2018). Five studies reported on previously examined populations in the Health Outcomes and Measures of the Environment (HOME) Study (Vuong et al. 2018a,b, Zhang et al. 2018), Danish National Birth Cohort (Liew et al., 2018b), and HUMIS Norwegian Human Milk Study (Lenters et al. 2019).

We believe the findings from these nine newest investigations continue to support the conclusion that there is no consistent evidence of an association between prenatal and/or early childhood PFOA exposure and neurobehavioral development. PFOA exposure is variably associated with both better and worse neurodevelopmental outcomes, as well as null findings. The magnitude and direction of the associations vary by timing and dose of exposure, as well as by timing and type of endpoint. Analyses conducted within the same cohort can highlight consistency across the course of development, such as the Danish National Birth Cohort's null findings when examining prenatal PFOA exposure in relation to developmental milestones in infancy (Fei et al. 2008), IQ at age 5 (Liew et al., 2018b), behavioral and motor coordination problems at age 7 (Fei and Olsen 2011), and ADHD or autism by approximately age 11 (Liew et al. 2015). Repeated investigations in the same cohort also underscore the need for discussion of results within the context of existing literature. In the HOME study, for example, cross-sectional analyses at mid-childhood indicated that higher PFOA exposure is associated with increased likelihood of impaired executive function (Vuong et al. 2018a,b); however longitudinal analyses of prenatal and early childhood PFOA exposure and mid-childhood executive function in the same population yielded null findings (Vuong et al. 2016, Vuong et al. 2018a,b).

Overall, a relatively large number of studies examining PFOA and neurotoxicity provide no consistent evidence of increased risk.

3.9. Kidney disease

In 2012, the Science Panel concluded there was insufficient evidence to find a probable link with kidney disease, largely based on its own studies (C8 Science Panel, 2012a). Overall in the literature, there are few studies of kidney disease and PFOA, especially longitudinal studies of populations with above-background exposure. In two occupational cohort mortality studies (one of DuPont workers in the mid-Ohio Valley (Steenland et al. 2012), the other of 3 M workers (Raleigh et al., 2014)), one found a positive trend with estimated PFOA and death from kidney disease (based on serum levels, 13 deaths) and the other did not (based on air levels, 27 cases).

A study of disease incidence among the DuPont occupational cohorts, which showed a mortality positive trend, found no association between estimated cumulative PFOA serum levels and chronic kidney disease incidence (43 cases, verified via medical records) (Steenland et al. 2015).

In a large longitudinal study of a highly-exposed community in the mid-Ohio Valley (of which the workers in the incidence study were a subset), estimated cumulative serum levels were not associated with chronic kidney disease incidence, whether the cohort was studied retrospectively or prospectively (397 cases, verified by medical records) (Dhingra et al. 2016). The remaining studies were cross-sectional studies of glomerular function. In two cross-sectional studies of highly exposed children and adults from the mid-Ohio Valley, there were inverse associations between *measured* serum PFOA and estimated glomerular filtration rate (eGFR), but no association when using *modeled* serum levels; the authors suspected reverse causality or confounding was responsible for the inverse association using a biomarker of PFOA (Watkins et al. 2013, Dhingra et al. 2017). Three other cross-sectional

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studies of measured serum PFOA and glomerular function were conducted in low-exposed general populations. In one cross-sectional study in China the authors found no trend of decreased eGFR with increased serum PFOA (Wang et al. 2019), and in two studies of the US NHANES population, the authors found either an inverse association with eGFR (children: Kataria et al. 2015), or an inverted U-shaped relationship (adults: Jain and Ducatman 2019).

In summary, consistent with the C8 Science Panel conclusion, we believe there is little evidence of an association between PFOA and chronic kidney disease, with the most informative study still being the large cohort study conducted by the C8 Science Panel in the mid-Ohio Valley (Dhingra et al. 2016).

3.10. Methodologic considerations

3.10.1. Cross sectional studies of serum PFOA and biomarkers

There have been a large number of studies of serum PFOA and other exposure biomarkers in low-exposed general populations in relation to biomarkers of disease (eg., kidney function), most notably in NHANES. PubMed, for example, lists 70 such NHANES studies addressing PFOA. Many studies with this design find associations between PFOA and clinical biomarkers or prevalent disease. However, we believe that caution should be exercised about such studies because of the potential for reverse causality or uncontrolled confounding, which can be particularly important at low exposure levels where the exposure contrasts are modest, and more strongly influenced by complex behaviors such as diet. In our own work we have observed instances when an association was found using measured serum PFOA, but not found with modeled PFOA (Dhingra et al. 2017, Watkins et al. 2013, Steenland et al. 2018). While one might argue that the modeled exposure is less accurate than measured biomarkers, we believe in fact the reverse may sometimes occur, that modelled exposure may sometimes provide less biased epidemiological effect estimation than measured biomarkers, as has been argued elsewhere (Weisskopf and Webster, 2017, Savitz and Wellenius 2018).

For example, as noted above, both Watkins et al. (2013) and Dhingra et al. (2017) observed an inverse association between measured serum PFOA and glomerular function, but no association using modeled PFOA, perhaps reflecting decreased glomerular function leading to increased serum PFOA. The inverse association between the PFOA in maternal serum and birthweight found for late but not early pregnancy assessment may reflect maternal blood volume expansion decreasing serum PFOA levels, with several studies showing a decrease in serum PFOA during pregnancy (Fromme et al. 2010, Kato et al. 2014). Furthermore, greater maternal blood volume expansion is associated with increased fetal growth (Vricella 2017), hence the possibility that larger babies would have lower maternal (and therefore newborn) serum PFOA. As a final example from our own work, Dhingra et al. (2016) have shown that the association between early menopause and higher PFOA in several cross sectional studies may also be due to reverse causality, whereby decreased excretion of PFOA at menopause leads to higher serum levels, creating a spurious association of early menopause and higher serum PFOA. Dhingra et al. analyzed measured and model-estimated PFOA in relation to self-reported menopause (controlling for age). Being postmenopausal was associated with higher measured (trend tests: $p = 0.013$), but not modeled ($p = 0.50$) serum PFOA. Modeled PFOA is not affected by excretion dynamics.

Another issue with general population studies, with low background exposure, is that serum PFOA is often highly correlated with other PFAS, eg, PFOS, PFNA, and PFHxS. It is difficult to separate the effects of these chemicals on purported effects. This can also at times occur even in high-exposure settings (e.g., Consonni et al. 2013, Li et al. 2018)

We recognize that cross-sectional studies of biomarkers of disease and serum PFOA levels can provide valuable information, particularly when the variation in exposure biomarkers is driven to a significant degree by exogenous exposure. For example, cross-sectional studies associating PFOA with higher cholesterol provided evidence later confirmed in longitudinal studies. However, caution should be exercised regarding positive associations between biomarkers of disease and PFOA in cross-sectional studies of populations with low background exposure, due to the potential for confounding and reverse causality.

3.10.1. Fate and transport models

Fate and transport models (built with adequate data and accompanied by toxicocokinetic models, see next section) are key to any valid reconstruction of past exposures, needed for longitudinal epidemiologic studies.

For the C8 Science Panel studies in West Virginia and Ohio, we built upon the work of Paustenbach et al. (2007) and others, using a combination of several modeling systems to develop a multimedia model of the transport process. Airborne emissions result in particle-bound PFOA/PFOS/PFAS being distributed into the surrounding community in a pattern dependent upon the prevailing winds, followed by deposition on soil surfaces, penetrations through the soil vadose zone, and subsequent impact upon local groundwater sources (Shin et al. 2011a, Shin et al. 2011b, Shin et al. 2012). Additionally, direct release into water supplies has also been noted (Shin et al. 2011a, Shin et al. 2011b, Shin et al. 2012) and was included in our modeling efforts.

To our knowledge, since our work in 2011–2012, there have been no further instances of comprehensive modeling of the fate and transport of PFOA and PFAS through the environment, coupled to health outcomes. However, incremental advances have been made in several areas focusing on improving physicochemical properties of PFOA and related compounds (Brusseau 2018, Hong and Purucker 2018, Brusseau et al. 2019, Lyu et al. 2018, Silva et al. 2019). From an empirical standpoint, several groups have carried out field investigations to validate laboratory and modeling assessments of physical phenomena associated with PFAS environmental transport (Xiao et al. 2020, Shan, et al. (2014), Zhu and Kannan (2019), Ferrey, et al. 2012, Webster and Ellis 2011). Further, new modeling efforts are now underway to estimate PFAS serum levels in Ronneby, Sweden, where large scale contamination took place (Li et al. 2018). Other large-scale efforts are beginning in the PFOA-contaminated Veneto region of Italy (Pitter et al. 2020), at various U.S. sites as part of the CDC/ATSDR Multi-Site PFAS Study (https://www.atsdr.cdc.gov/pfas/Multi-Site-Health-Study.html), and near Wilmington, North Carolina due to water contamination with several PFAS, including GenX, a shorter-chain fluorinated PFOA substitute (https://cleanaircarolina.org/wp-content/uploads/2019/04/4-Hoppin-NC-Breathe-Final.pdf). In our view, while such work is critical in the development of fate and transport modeling systems, a more comprehensive program is needed whereby modelers, laboratory scientists, exposure scientists, and epidemiologists work together to establish a coherent understanding of the efficacy of using source to human receptor/health effects models to address the impact of these compounds on human health. Such work requires transdisciplinary cooperation similar to that accomplished in the C8 Study and should be expanded to evaluate differential fate and transport, as well as health outcomes, associated with the broader PFAS class of toxicants.

3.10.2. Exposure and toxicokinetic modeling

For the C8 Science Panel studies in the mid-Ohio valley, we relied on individual residential and work histories, reconstructed water PFAS concentrations, water use questionnaires, and (in the absence of self-reported water consumption rates) default EPA exposure factors to develop individual year-by-year estimates of the amount of PFOA ingested through water consumption, and inhaled at the residence and workplace since birth for each study participant (Shin et al. 2011b). However, toxicological effects are believed to be a function of internal target tissue dose (Müller et al. 2004a,b), rather than ingested dose, and other exposure pathways for PFOA are common, including diet (IARC, 2016). We therefore applied a one compartment toxicokinetic model to convert ingested doses to year-by-year serum concentrations, which

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serve as surrogates for target tissue doses (Shin et al. 2011b). Our model accounted for likely contributions of other pathways using a time-dependent background serum contribution that was assigned to all participants in common, based on US median serum PFAS concentrations over time obtained from NHANES (Centers for Disease Control, 2019), and added to serum concentration expected from local water consumption and air inhalation according to the superposition principle. Water concentrations changed dramatically over time (Shin et al. 2011a) and few participants remained in the same water district throughout an entire lifetime, making steady-state assumptions unreliable. Instead, we used a time-dependent closed-form forward solution for standard one compartment toxicokinetic models with piecewise constant exposure rates (i.e., we assumed that PFOA ingestion rates were roughly constant within any one calendar year) (Flesch-Janys et al. 1998).

Our toxicokinetic model assumed a serum half-life of 3.5 years (Olsen et al. 2007), a volume of distribution of 0.181 L/kg for males and 0.198 L/kg for females (Butenhoff et al., 2004), and infant:maternal serum PFOA concentration ratios of 0.785 and 1.27 at birth and age 1 year, respectively (Shin et al. 2011b). Towards validation, we evaluated the Spearman's rank correlation between modelled and measured serum PFOA concentrations, finding a coefficient of 0.67 among all participants and 0.82 among those with the highest quality exposure data (residence in one of the six qualifying water districts at the time of blood sample, and had a self-reported water consumption rate) (Shin et al. 2011b).

Since our original toxicokinetic modeling was performed, evidence has emerged that better supports an average human half-life for PFOA in the range of 2 to 3 years (Bartell et al. 2010, Bartell et al. 2012, Zhang et al. 2013, Russell et al. 2015, Gomis et al. 2016, Li et al. 2018,). A simplified version of our one compartment toxicokinetic model for PFOA, using constant values for the dose rate and background contribution and an updated half-life value of 2.3 years, is available in the form of an online calculator (Bartell 2017), recently updated to incorporate several other PFAS chemicals and the effects of menstruation (Lu and Bartell 2019, Wong et al. 2014).

Our modelled serum PFOA values were used in multiple epidemiologic investigations in the C8 Science Panel Studies, and later sensitivity analysis indicated that epidemiological effect estimates for preeclampsia per interquartile range of modelled serum PFOA were not substantively changed by simulated measurement error in the exposure assignments (Avanasi et al. 2016a, Avanasi et al. 2016b). These results using modelled serum PFOA have also been very useful for triangulating epidemiological effects by comparison to epidemiological associations using measured serum PFOA, due to their distinctly different threats to validity (Watkins et al. 2013, Weisskopf and Webster, 2017).

4. Discussion

Clearly, the volume of epidemiologic research on PFOA and health outcomes has grown since the work of the C8 Science Panel was completed, quite considerably for outcomes such as birthweight, neurodevelopment, thyroid function, and immunology, often supported by toxicological evidence. Much of the research has been opportunistic, extending studies conceived for other purposes to take advantage of the relative ease of measuring biomarkers of PFAS and then assessing their relationship to various health outcomes, illustrated most clearly by the use of NHANES data (e.g., Lin et al. 2010, Gleason et al. 2015, Jain and Ducatman 2019), but also in many established cohort or registry populations (e.g., Fei et al. 2008, 2009, 2010, 2011). Exploiting readily available data from general population studies is efficient but when focused on background exposures as reflected in biomarkers, differences may be strongly influenced by variability in uptake and excretion (rendering them vulnerable to confounding and reverse causation) and precluding historical exposure reconstruction which is feasible only if there is historical information on specific dominant exposure sources.

The strengths of the C8 Science Panel studies remain the examination of a large population with a well-defined source of elevated exposure, an ability to reconstruct historical exposure, a wide range of exposure from background to very high levels, and thorough assessment of clinical health endpoints. However, we studied only a single population. Newer studies for specific outcomes would be expected to vary from our findings due to different sample sizes, lower exposures to PFOA, co-exposures to other PFAS, and varying patterns of confounders such as diet and age.

There are now examples of comparable circumstances in the recent literature including Swedish studies at Ronneby (Andersson et al. 2019, and Xu et al. 2019), and Italian studies from Veneto (Mastrantonio et al. 2018, Girardi and Merler 2019, Manea et al. 2020). A multi-site community study of populations with elevated exposure due to contaminated drinking water is in progress in the US through the Agency for Toxic Substances and Disease Registries (https://www.atsdr.cdc.gov/pfas/index.html). With the discovery of multiple locations around the world with well-defined environmental sources of elevated exposure, more opportunities are likely to arise that individually or collectively offer great promise for extending knowledge of the health effects of PFOA. Occupational studies are also of interest because of their high exposure contrasts, but they generally involve relatively small populations.

The charge to the C8 Science Panel required making dichotomous judgments regarding probable links, which had to be based on the available evidence even recognizing that it was (and remains) incomplete. Those assessments were influential in focusing research attention and it is not surprising that for some of outcomes for which probable links were found, the evidence from research has become weaker with the accumulation of additional studies, e.g., thyroid disorders, and ulcerative colities. For other outcomes that were not found to have probable links to PFOA, the evidence continues to corroborate that assessment, e.g., neurodevelopment. Some of the most important rare diseases for which probable links were identified have had little additional research to confirm or refute the original assessment, notably testicular cancer, pregnancy-inducted hypertension, and ulcerative colitis. For kidney cancer the evidence has been somewhat strengthened by a recent positive case-control study. As noted above, the evidence associating PFOA to some clinical biomarkers is increasingly persuasive, e.g., cholesterol, liver enzymes, and immunologic response to vaccines, yet in our view the evidence on increased risk of heart disease, chronic liver disease, and infectious disease related to PFOA is not evident. As the literature matures, the needs for advancing research on the specific different health endpoints becomes more clear, building on past efforts. While it is disappointing that we do not have greater clarity on adverse human health effects associated with PFOA, this still remains a relatively new area of investigation. With ongoing efforts to minimize exposure to PFOA, however, the opportunities for informative new studies may well diminish.

Declaration of Competing Interest

Three authors (Drs. Savitz, Ryan, Fletcher) have served in the past as paid consultants to law firms conducting litigation involving PFAS. Drs. Savitz and Ryan have worked for plaintiffs, while Dr. Fletcher has worked for both plaintiffs and defendants. Dr. Bartell serves as an expert witness for plaintiffs in two PFAS medical monitoring lawsuits, and receives compensation for those services; the terms of this arrangement were reviewed and approved by the University of California Irvine in accordance with its conflict of interest policies. Dr. Savitz is doing current consulting, in one case for plaintiffs and in the other for defendants.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2020.106125.

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Testimony to the New Jersey Assembly Environment and Solid Waste Committee
March 10, 2022

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Good afternoon Chairman Kennedy, Vice Chair Stanley and Members of the Assembly Environment and Solid Waste Committee. I am Dr. Eileen Murphy, Vice President Government Relations at NJ Audubon and previous Director of the science division of NJ Dept of Environmental Protection where I conducted research on the occurrence of chemical contaminants including PFAS in drinking water for 21 years. I also served for six years as a chartered member of the USEPA Science Advisory Board where I provided expert advice to the agency on issues such as chemical contamination. I appreciate the opportunity today to testify before the Committee about the occurrence and health effects of per- and polyfluoroalkyl substances, known as PFAS.

Sitting beside me is Kelly Knutson from my staff who directs the Coalition for the Delaware River Watershed. Under Kelly's direction, the Coalition has included PFAS as a priority issue for the upcoming year as these compounds have been detected in water, sediment and fish samples throughout the watershed. Indeed, PFAS were first discovered in the early 2000's in the Delaware River and continue to be measured in water, air, soils/sediment, wildlife, and humans.

Addressing PFAS in NJ is critical, and we have already made some progress on that front. Before I outline some of the actions that are ongoing in the State, I'll present some basic information about what PFAS are, how they have been released, where they are found in the environment, particularly in surface water, ground water, and drinking water, how humans are exposed, and possible options for addressing them. I've included some fact sheets with my testimony as additional basic information.

Perfluoroalkyl and polyfluoroalkyl substances (PFAS) are molecules consisting of a chain of carbon and fluorine atoms linked together. This carbon-fluorine bond is extremely strong, making the degradation of these chemicals slow and difficult. This unique structure also results in chemical properties that allow materials coated with them to repel both water and oil. Given these highly desirable qualities, they have been used in a variety of industrial and household products such as stain repellant textiles, fire-fighting foams, personal care products, stain- and water-resistant clothing, carpets and furniture, and paper coatings since the 1940s. PFAS do not degrade readily, which is how they got the nickname "forever chemicals," although some of the longer chain chemicals can break down into shorter chain perfluorinated compounds. Those that do break down, do so very slowly. As a result, PFAS can build up in people, animals, and the environment over time. They have also been found in fish, deer and other wildlife, and appear in the blood of 95-97% of US residents tested. In other words, it's everywhere.

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The most studied of the PFAS are those that were first detected in NJ and PA waters in 2004 - Perfluorooctanoic acid (PFOA) and Perfluorooctanesulfonic acid (PFOS). These two long-chained PFAS are no longer manufactured in the US, and levels in human blood for them have been decreasing since the PFOA Stewardship Program, which called for a 95% phase out of PFOA by 2010 and complete elimination of PFOA and PFOS from emissions and products by 2015. Since the phase out of those two PFAS, however, the number of other perfluorinated substances used commercially and found in environmental media has skyrocketed, and that's because they are still being created, used, and released into the environment. With more research in analytical chemistry and more aggressive monitoring, scientists have been able to identify many of these newer PFAS. According to the USEPA, there are over 10,000 individual PFAS in the environment today (there were 9,252 PFAS on EPA's Master list of PFAS in 2021. When I checked today that list had grown to 12,039). As our methods for detecting these chemicals improves, and as more perfluorinated substances are used commercially, we will continue to detect more of them in environmental media.

After the discovery of PFOA and PFOS in NJ, NJDEP initiated the first statewide occurrence study in the country of public water supplies to determine whether these chemicals were occurring in the state's public water systems. From 2006 through 2010, 54 water utilities were sampled. PFOA was detected in 60% of systems and PFOS in 30% with six additional PFAS detected in samples including perfluorononanoic acid (PFNA).

But just because PFAS are detected in water and other environmental media does not necessarily mean that they 1) reach humans or 2) are harmful. Studies conducted in areas where known sources of PFAS contamination have occurred have demonstrated both - that PFAS are in human blood and that they cause detrimental health effects. I'll go over them briefly here.

National studies report the presence of PFAS in human blood. Since 1999, CDC has measured a handful of PFAS in the U.S. population as part of the National Health and Nutrition Examination Survey (NHANES). NHANES is a survey that measures the health and nutritional status of adults and children in the United States. Here, as with water, PFOS and PFOA are the most studied. The good news is that data from the NHANES survey has shown a significant decrease in blood serum levels of PFAS and PFOS since the phase out of these chemicals in industry. Concurrently, though, they report an increase in the number of other PFAS being detected in study subjects. As scientists improve analytical methods for measuring individual PFAS in blood, we are seeing more of them, just as we did with water. It is more difficult and tedious to measure PFAS in biological samples like blood. It's only a matter of time before we see even more PFAS in blood samples. Scientists can measure hundreds of PFAS in water now through advances in analytical chemistry. How long before analytical methods are capable of detecting hundreds and then thousands of PFAS in human blood?

PFAS in blood indicates that human populations are exposed – that is, these chemicals are getting into our bodies through the food we eat, the water we drink, the air we breathe. Toxicological and epidemiological studies can tell us if those exposures lead to health effects. Numerous studies indicate that the two common PFAS—PFOA and PFOS—can cause reproductive and developmental, liver and

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kidney, and immunological effects in laboratory animals. The most consistent findings from human epidemiology studies are increased cholesterol levels, with more limited findings related to infant birth weights, effects on the immune system, cancer (for PFOA), and thyroid hormone disruption (for PFOS). It is rare to have such rich databases on human subjects, but given the widespread exposures of PFAS to humans, scientists have been able to evaluate impacts directly to humans rather than extrapolate the results of animal tests to humans. While much has been studied on health effects of PFOA and PFOS, more needs to be done to assess impacts from other PFAS. According to the CDC, "Human health effects from perfluorinated compounds at low environmental doses ... are unknown."

PFAS are also in wildlife, including birds – from the Double-crested Cormorants in San Francisco Bay, Northern Cardinals in Hawaii, Snow Buntings in the Arctic, and American Flamingos in the Caribbean, PFAS are detected in their eggs, blood, and liver. But data on wildlife is appallingly lacking though the very limited existing data suggests negative impacts to growth, development, reproduction, immunity to seabirds and other birds. Research to date has focused on impacts to humans, but we need to do more to assess impacts to wildlife.

So, PFAS are present in the environment at ubiquitous levels, and they are entering our bodies and causing negative health effects. The next step is to evaluate ways to reduce exposures to prevent the uptake into our bodies and subsequent negative health impacts.

Regulatory attention has mostly been on the development of health advisories and drinking water standards, or maximum contaminant level (MCL) for the most frequently detected and studied PFAS – PFOA, PFOS and PFNA. The process of reviewing the necessary information for standard development is arduous and lengthy. Information on occurrence, toxicity, analytical methods, and treatment capabilities are needed for each individual chemical. NJ began collecting and evaluating information in 2004 with the discovery of PFOA in NJ water. But it wasn't until 2018 that NJ adopted the first MCL for any PFAS in the US – 13 nanograms/liter (parts per trillion) for PFNA. Shortly after, in 2020, NJ finalized MCLs for PFOA at 14 ng/L (ppt) and for PFOS at 13 ng/L (ppt). Other states have developed standards as well (some have standards for multiple PFAS beyond NJ's three) including MA, MI, NH, NY and VT, while other states have proposed standards – AZ, IA, KY, ME and RI. There are no federal drinking water standards for any PFAS (although there are nonenforceable, nonregulatory health advisories for two – PFOA and PFOS).

What's the point of this chronology showing when NJ first detected perfluorinated substances in 2004 from when we set the first drinking water standard in 2018? Of the thousands of PFAS chemicals known to be in the environment, we regulate three in NJ and it took 14 years to regulate one, 16 years to regulated another two. Our current regulatory approach for developing MCLs for drinking water is chemical specific – one chemical at a time. While scientifically valid and effective, this approach is inadequate for the sheer volume of PFAS being detected in water.

When we first learned of the occurrence of PFAS in water in 2004, we thought that these two emerging contaminants were unique. But each year, new studies have identified first hundreds and now thousands – even tens of thousands – of PFAS in our environment (12,039 as I mentioned earlier). Twelve thousand

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emerging perfluorinated contaminants. As we find more in this class, it causes one to wonder about other emerging contaminants. The USEPA data base of chemicals (Comptox) shows a running list of 906,511 chemicals in the environment needing evaluation. For perspective, there are drinking water standards for around 90 substances.

PFAS represents the challenge we face in regulating "emerging contaminants" in general. We cannot rely solely on our current chemical-by-chemical regulatory scheme, especially when so many of the PFAS have little or no toxicity information. Sixteen years to develop 3 MCLs out of a list of over 10,000 is inadequate. We must develop multi-pronged solutions that include restricting the use of toxic chemicals and developing safer alternatives; developing innovative remediation technologies to clean up contaminated sites; and treating drinking water for a wide suite of potential contaminants using a treatment-based or class-based regulatory approach.

These actions won't replace the current paradigm but rather complement it and accelerate protections for the public. Not just for PFAS but for other currently unidentified "emerging contaminants." We already have treatment-based MCLs for some contaminants, and set class-based standards for PCBs, dioxins, PAHs and others. There is precedent. Is there a will?

Thank you for this opportunity to speak. I brought some materials with information for you and am here to answer any questions you might have.

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PFAS WHAT YOU NEED TO KNOW

WHAT ARE PFAS CHEMICALS?

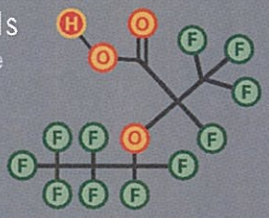
Per- and polyfluoroalkyl substances (PFAS) are a group of man-made chemicals that includes PFOA, PFOS and GenX chemicals. Since the 1940s, PFAS have been manufactured and used in a variety of industries around the globe, including in the United States. PFOA and PFOS have been the most extensively produced and studied of these chemicals. Both are very persistent in the environment and in the human body. Exposure to certain PFAS can lead to adverse human health effects.

PFOA & PFOS

U.S. manufacturers voluntarily phased out PFOA and PFOS, two specific PFAS chemicals.

GenX Chemicals

GenX chemicals are a replacement for PFOA.



WHAT EPA IS DOING

Some of the agency's work includes: development of additional toxicity values, analytical methods for additional PFAS and non-drinking water media as well as treatment options for PFAS in drinking water. EPA is also hosting a National Leadership Summit on PFAS in May 2018.

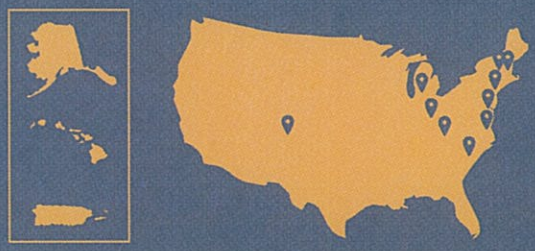
Established methods to measure 14 PFAS compounds in drinking water

Identified five treatment processes for PFOA and PFOS

Identified all PFAS chemicals that are legally available for production and use

Provided national monitoring data for 6 PFAS in drinking water

Issued drinking water health advisories (70 parts per trillion) for PFOA and PFOS in 2016



Provided support for 10 states with site-specific PFAS challenges and problems: NC (Cape Fear River), MI, DE, WV, CO, NY (Hoosick Falls), OH, NH, VT and NJ

Updated website to include tools and information so that states, tribes and local communities can understand, assess and address PFAS incidents and emergencies

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HOW ARE WE EXPOSED TO PFAS?

PFAS include a large number of important chemicals that can be used in some food packaging and can make things grease- and stain-resistant. They are also used in firefighting foams and in a wide range of manufacturing practices. Unfortunately, some of these substances don't break down over time. That means they build up in the environment and in our bodies.

Drinking water can be a source of exposure in communities where these chemicals have contaminated water supplies. Such contamination is typically localized and associated with a specific facility, for example,

- an industrial facility where PFAS were produced or used to manufacture other products, or
- locations where firefighting foam was used such as oil refineries, airfields or other training facilities for firefighters

If you are concerned about the possibility of PFAS in your drinking water, contact your local water supplier and ask for more information about PFAS.

STAIN/GREASE REPELLENT | FIREFIGHTING FOAMS | INDUSTRIAL USES

HEALTH EFFECTS

There is evidence that exposure to PFAS can lead to adverse health outcomes in humans. If humans or animals ingest PFAS (by eating or drinking food or water than contain PFAS), the PFAS are absorbed and can accumulate in the body. PFAS stay in the human body for long periods of time. In some cases, the level of PFAS in the body can increase to the point where people can suffer from adverse health effects.

Studies indicate that high concentrations of PFOA and PFOS can cause reproductive and developmental, liver and kidney, and immunological effects in laboratory animals. Both chemicals have caused tumors in animal studies. The most consistent findings from human studies are increased cholesterol levels among exposed populations, with more limited findings related to:

- infant birth weights
- adverse effects on the immune system
- cancer (for PFOA)
- thyroid hormone effects (for PFOS)

WWW.EPA.GOV/PFAS

SOURCE: U.S.EPA

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Drinking Water Facts:

Updated September 2020

Per- and Polyfluoroalkyl Substances (PFAS) in Drinking Water

*formerly titled PFCs in Drinking Water

- **Per- and polyfluoroalkyl substances (PFAS) are a group of chemicals with many commercial and industrial uses.**
- **PFAS have been associated with a variety of adverse health effects in humans, but it has not been definitively established that PFAS cause these effects.**
- **PFOA, PFNA, and PFOS have drinking water regulations in New Jersey.**

What are PFAS and perfluorinated chemicals (PFCs)?

PFAS are a group of manmade chemicals which include a smaller group of chemicals called PFCs. PFAS repel water and oil and are resistant to heat and chemical reactions. They therefore have important industrial and commercial uses. PFAS are used in production of some non-stick cookware, in waterproof and stain proof coatings, in "leak-proof" coatings on food packaging materials, in fire-fighting foams, and in other uses. PFAS can enter drinking water through industrial release to water, air, or soil; discharges from sewage treatment plants; land application of contaminated sludge; and use of fire-fighting foam.

PFCs are not broken down in the body. Four types of PFCs have been found in the blood (serum) of greater that 98% of the United States population. **These PFCs build up and stay in the human body for many years, and the amount goes down very slowly over time.**

- **PFOS** – perfluorooctane sulfonate
- **PFOA** – perfluorooctanoic acid
- **PFNA** – perfluorononanoic acid
- **PFHxS** – perfluorohexane sulfonate

How can I be exposed to PFAS?

Some PFAS can dissolve in water. Therefore, drinking water may be a major source of exposure to PFAS for people living in communities with contaminated drinking water. Other sources of PFAS exposure include food, food packaging, consumer products, house dust, indoor and outdoor air, and at workplaces where PFAS are made or used.

Exposure to PFAS in drinking water is primarily from ingestion. Exposure to PFAS through other household uses of water such as showering, bathing, laundry and dishwashing is not significant.

Are PFAS harmful to my health?

There is considerable information on the health effects of PFAS in humans and animals, and more information is continually becoming available. In experimental animals, some PFAS have been found to cause developmental, immune, neurobehavioral, liver, endocrine, and metabolic toxicity, generally at levels well above human exposures. Some studies of the general population, communities with drinking water exposures, and exposed workers suggest that PFAS increase the risk of a number of health effects. The most consistent human health effect findings for PFOA – the most well-studied of the PFAS – are increases in serum cholesterol, some liver enzymes, and uric acid levels. For PFOS, the most consistently found human health effects include increased serum cholesterol and uric acid levels. PFOA and PFOS have been associated with decreased antibody response following vaccination.

PFOA and PFOS caused tumors in rodents. In a community with substantial exposure to PFOA through drinking water, PFOA exposure was associated with higher incidence of kidney and testicular cancers.

How can PFAS affect children?

In experimental animals, some PFAS cause developmental effects. In humans, exposure to PFAS before birth or in early childhood may result in decreased birth weight, decreased immune responses, and hormonal effects later in life. More research is needed to understand the role of PFAS in developmental effects.

Infants and children consume more water per body weight than older individuals, so their exposures may be higher than adults in communities with PFAS in drinking water. They may also be more sensitive to the effects of PFAS.

Continue to Page 2

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Continued…

When PFAS are elevated in a drinking water supply, it is advisable to use bottled water to prepare infant formula for bottle-fed babies. Beverages for infants, such as juice made from concentrate, should also be prepared with bottled water. PFAS are present in breast milk. Based on the scientific understanding at this time, since the benefits of breast-feeding are well-established, infants should continue to be breast-fed. Pregnant, nursing, and women considering having children may choose to use home water filters or bottled water for drinking and cooking to reduce exposure to PFAS in your water. However, exposure to fetuses and nursing infants is influenced by past exposures and slow excretion of these substances from the body, so risk reduction will not be immediate.

What levels of PFAS in drinking water are safe?

In 2018, NJ became the first state to establish a drinking water standard for a PFAS chemical when it set a Maximum Contaminant Level (MCL) for **PFNA, at 13 parts per trillion (ppt) [ng/L]**. The New Jersey Department of Environmental Protection (NJDEP) has also established enforceable MCLs for **PFOA (14 ppt) and PFOS (13 ppt)**. These levels are based on current scientific information and are intended to protect for lifetime exposure.

USEPA has issued a lifetime drinking water Health Advisory for **PFOA** and **PFOS** of **70 ppt** individually or when concentrations of PFOA and PFOS are combined. A Health Advisory is non-enforceable guidance that identifies the concentration of a contaminant in drinking water at which USEPA has concluded adverse health effects are not anticipated to occur. NJ's MCLs are more stringent.

How do I know if I have PFAS in my drinking water?

Large public water systems in the U.S. and a subset of smaller water systems were required to test for some PFAS as part of the USEPA Unregulated Contaminant Monitoring program. All of the water systems which tested for PFAS have reported their results in your annual Consumer Confidence Report (CCR) which may be available online or provided by your water provider. The only way to know whether your private well has PFAS is to have it tested. To find a laboratory certified to test, you can contact NJDEP Office of Quality Assurance at 609-292-3950 or at https://www13.state.nj.us/DataMiner

What should I do if I am concerned about PFAS in my drinking water?

PFAS are **not** removed from water by boiling. **If tap or well water is found to contain PFAS, people may choose to use home water filters or bottled water for drinking and cooking to reduce exposure to PFAS in their water.**

Granular activated carbon filters or reverse osmosis water treatment devices are technologies that can reduce the level of PFAS in drinking water. If a treatment is used, it is important to follow the manufacturer's guidelines for maintenance and operation. NSF International, an independent and accredited organization, certifies products proven effective for reducing PFOA and PFOS below the USEPA Health advisory level (70 ppt) (http://info.nsf.org/Certified/DWTU/).

What can blood testing for PFAS tell me?

PFAS can be measured in your blood serum but this is not a routine test. While a blood test may indicate whether you have been exposed to PFAS, results cannot be used to predict your health effects nor can they be linked to specific health problems. Also test results alone cannot be used to specifically identify sources of exposure, and there is no treatment to reduce levels of PFAS in blood. A national program has been measuring PFAS in blood among the U.S. population. This information can be used to determine if the levels of PFAS in your blood are higher than national background levels. For example, if your concentration is higher than the 95th percentile, this means your blood serum concentration is higher than the concentration found in 95% of the U.S. population.

Estimates of four most common PFAS measured in the U.S. general population, 2013-2014 (ng/ml [ppb])

PFAS	Geometric Mean	50th Percentile	95th Percentile
PFOS	4.99	5.20	18.42
PFOA	1.94	2.00	5.51
PFNA	0.67	0.64	1.99
PFHxS	1.35	1.33	5.54

Additional Resources:

http://www.nj.gov/health/ceohs/environmental-occupational/drinking-water-public-health/

National Institute of Environmental Health Sciences
Your Environment. Your Health.

Perfluoroalkyl and Polyfluoroalkyl Substances (PFAS)

These widespread, manmade chemicals have leached into our soil, air, and water. More research is needed to understand if and how they cause health problems.

PFAS are a large, complex, and ever-expanding group of manufactured compounds that are widely used to make everyday products more resistant to stains, grease, and water. For example, they are used to keep food from sticking to cookware, make clothes and carpets resistant to stains, and create firefighting foam that is more effective. PFAS are also used in a variety of other industries, including aerospace, automotive, construction, electronics, and military. Because they take so long to break down in the environment, they remain in air, soil, and water, including sources of drinking water, for a long time.

Why PFAS are receiving so much attention

- Widespread occurrence. Studies are finding PFAS in participants' blood and urine, and people want to know if they may cause health problems.
- Numerous exposures. PFAS are used in hundreds of products for a variety of applications, all over the world, causing multiple opportunities for exposure.
- Growing numbers. More than 4,700 known PFAS chemicals exist, and the numbers are increasing as industry invents new chemicals.[1]
- Persistent. PFAS remain in the environment for an unknown length of time and take many years to leave the body.
- Bioaccumulation. Some PFAS chemicals may accumulate in the body over time, due to more PFAS being absorbed than eliminated.

How people are exposed

Though more research is needed to fully understand all sources of exposure, people are most likely exposed to these chemicals by consuming PFAS-contaminated water or food, using products made with PFAS, or breathing air containing PFAS.

One report by the Centers for Disease Control and Prevention National Health and Nutrition Examination Survey (NHANES) found PFAS in the blood of 97 percent of Americans[2]. A more recent NHANES report suggested a reduction in blood levels of PFOS and PFOA since their removal from consumer products in the early 2000s. However, the number of new PFAS chemicals appear to be growing and exposure is difficult to assess accurately.

What they are

PFAS molecules are made up of a chain of carbon and fluorine atoms linked together. Because the carbon-fluorine bond is one of the strongest ever created, these chemicals do not degrade in the environment. In fact, PFAS products remain in the environment for so long that scientists are unable to estimate an environmental half-life, or the amount of time it takes for 50 percent of the chemical to disappear.

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National Institutes of Health
U.S. Department of Health and Human Services

March 2019

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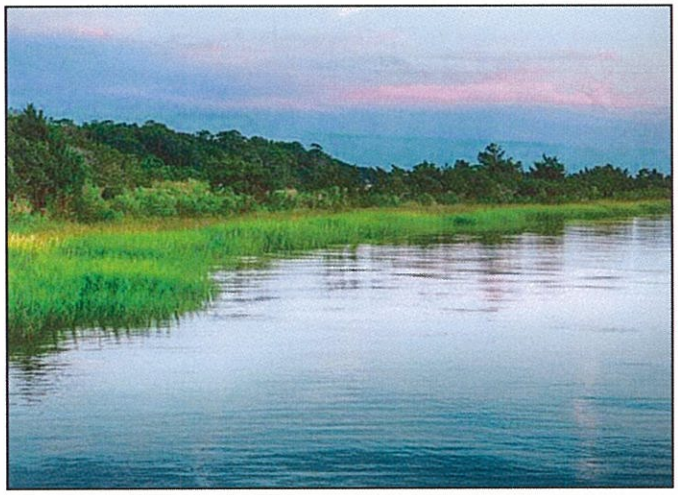
The scientific understanding of PFAS stems almost entirely from research on a select few compounds. Perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) have been manufactured the longest, are the most widespread in the environment, and are the most well-studied. PFOS and PFOA, which are called long-chain PFAS, are no longer manufactured in the United States. However, U.S. chemical manufacturers have replaced these phased-out chemicals with alternative short-chain PFAS, such as GenX.

What we have learned so far

When looking for possible human health effects of chemical compounds that have become intermingled with our natural environment, it is important to understand that they are very hard to study, especially when there are thousands of individual PFAS chemicals. Still, the research conducted to date reveals possible links between human exposures to PFAS and adverse health outcomes. These include potential effects on metabolism,[3] pregnancy,[4] children's cognition and neurobehavioral development,[5] and the immune system.[6]

Much of the research has been supported or led by the National Institute of Environmental Health Sciences (NIEHS) and the National Toxicology Program (NTP), an interagency testing program headquartered at NIEHS. For example, in 2016, NTP concluded that PFOA and PFOS were a hazard to immune system function in humans, based on evidence from prior studies.[7]



Cape Fear River

While knowledge about the potential health effects of PFAS has steadily grown in recent years, many questions remain unanswered. Therefore, NIEHS and NTP continue to fund or conduct research to better understand the effects of PFAS exposure.

What we are doing

NTP is leading multi-faceted toxicology studies to evaluate and identify the adverse effects of PFAS chemicals, such as:

- A systematic literature review of six PFAS chemicals — PFNA, PFHxS, PFHxA, PFDA, PFBA, PFBS — to determine whether they weaken the body's response to vaccinations.
- Animal studies, including a two-year study on PFOA and 28-day studies on seven PFAS chemicals — PFNA, PFHxS, PFHxA, PFDA, PFBA, PFBS, PFOA.

In addition to the NTP effort, NIEHS awards many grants to organizations across the U.S. to conduct their own PFAS studies, including:

- Time-sensitive studies including PFAS exposures in residents near Colorado Springs whose water was contaminated with the PFAS known as perfluorohexane sulfonate (PFHxS), and contamination of the Cape Fear River in North Carolina by GenX.
- Long-term epidemiological studies of health effects of PFAS exposures, some beginning before birth, including one study on more than 300 children in the Faroe Islands.[8]

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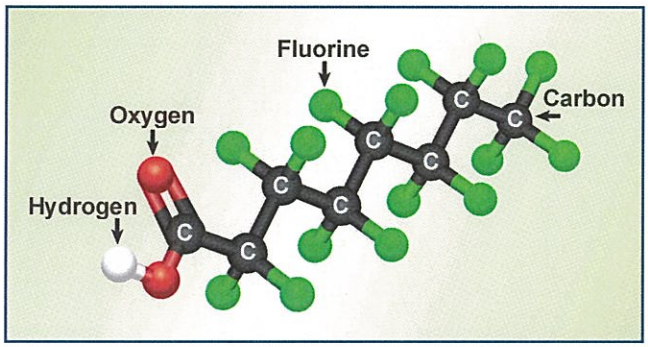
- Children's environmental health research studies. For example, one study found that verbal and non-verbal IQ scores were lower in children with higher prenatal exposure to PFOA and PFOS.[9]
- Long-term studies, including one showing a link between PFAS exposure and an increased risk of Type 2 diabetes in women.[10]



PFOA chemical symbol

The NIEHS Superfund Research Program (SRP) funds the search for practical applications to protect the public from exposures to hazardous substances. Examples include:

- A project called Sources, Transport, Exposure, and Effects of PFASs (STEEP) at the University of Rhode Island is identifying sources of PFAS contamination, assessing human health effects, and educating communities on ways to reduce exposure.[11]
- The Michigan State Superfund Research Center is developing energy-efficient nanoreactors capable of breaking the carbon-fluorine bond that keeps PFAS from degrading.
- Scientists at the University of California, Berkeley are working on options to degrade and destroy aqueous film-forming foams (AFFF) used for firefighting, a major source of PFAS contamination.
- Small Business Innovation Research (SBIR) grantee CycloPure, Inc., is developing new ways to remove hazardous PFAS from water.
- Another SBIR project by EnChem Engineering, Inc. is developing an innovative technology to speed up removal of PFAS at Superfund sites.

Impact on public health

- NIEHS grantees and NTP contribute significantly to the field of alternatives assessment, to ensure harmful chemicals are not replaced by equally harmful, but less well-studied, related compounds.
- NIEHS leadership was part of an international group co-authoring a commentary on PFAS[12] calling for more collaboration among those researching, regulating, and using PFAS. The document is known as the Zurich statement because it grew out of a November 2017 workshop in Zurich, Switzerland.
- Collectively, the work of NIEHS grantees, NTP, and their collaborators will help determine the toxicity of PFAS chemicals and their potential adverse human health effects. Such data will be of value to regulatory agencies and policymakers who will use the information to protect public health and help make informed decisions.
- Some state agencies are reviewing current PFAS research findings from NIEHS grantees, NTP, and others, to help assess and evaluate the impact of these chemicals on human health in their communities.

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The importance of collaboration

PFAS contamination is a complex issue that no single entity or agency will be able to address alone. NIEHS and NTP are actively coordinating efforts with other government agencies to gain a clearer understanding of the ways in which PFAS may affect human health.

- NTP is collaborating with the U.S. Environmental Protection Agency on the Responsive Evaluation and Assessment of Chemical Toxicity (REACT) program, which is developing a new approach to screen more than 100 representative PFAS in search of a common basis for toxicity.
- NIEHS is working with the Agency for Toxic Substances and Disease Registry to initiate exposure assessments and health studies authorized by the National Defense Authorization Act for Fiscal Year 2018.
- NIEHS is working closely with the U.S. Food and Drug Administration on PFAS-related food safety, and the Centers for Disease Control and Prevention on monitoring PFAS exposure levels.

Learn more

- National Toxicology Program
  https://ntp.niehs.nih.gov
- Agency for Toxic Substances and Disease Registry
  www.atsdr.cdc.gov/pfas
- Centers for Disease Control and Prevention
  www.cdc.gov/biomonitoring/PFAS_FactSheet.html
- U.S. Environmental Protection Agency
  www.epa.gov/pfas/epas-pfas-action-plan

For more information on the National Institute of Environmental Health Sciences, go to **www.niehs.nih.gov**.

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State	Drinking Water Action	Compound	Level (ppt)
California	Response Levels	PFOA PFOS	10 40
Connecticut	Action Level	Sum of PFOA, PFOS, PFNA, PFHxS, PFHpA	70
Massachusetts	Adopted Regulation 9/16/20	Sum of PFOA, PFOS, PFNA, PFHxS, PFHpA, PFDA	20
Michigan	Adopted Regulation 8/3/20	PFOA PFOS PFNA PFHxS PFBS PFHxA GenX	8 16 6 51 420 400,000 370
Minnesota	Health Based Guidance Surrogate of PFOS HBV	PFOA PFOS PFHxS	35 27 27
New Hampshire	Adopted Regulation 10/1/19	PFOA PFOS PFHxS PFNA	12 15 18 11
New Jersey	Adopted Regulation Adopted Regulations 6/1/20	PFNA PFOA PFOS	13 14 13
New York	Adopted Regulation 7/30/20	PFOA PFOS	10 10
North Carolina	Health Advisory	GenX	140
Vermont	Health Advisory	Sum of PFOA and PFOS	20

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DELAWARE RIVERKEEPER NETWORK

Testimony March 10 NJ Assembly Environment Committee

What are PFAS? Health risks?

Per- and polyfluoroalkyl substances (PFAS) are compounds with at least one totally fluorinated carbon atom, making an extremely strong bond. They are a class of chemicals used for decades in manufacturing, industrial applications, aqueous film forming foam used in firefighting, and a multitude of consumer products. They are known as "forever chemicals" because they don't break down in the environment. Miniscule doses of PFAS build up in the human body and are linked to disease and adverse health effects, including developmental effects in the fetus and young children, cardiovascular disease, ulcerative colitis, thyroid disease, pregnancy-induced hypertension and cancers, including testicular and kidney cancer.[1] Scientists, health and toxicology professionals, and agencies are extensively studying PFAS compounds since they are persistent and highly mobile in the environment, bioaccumulative in organisms, and toxic at low levels.[2] EPA estimates there are 12,000 individual PFAS chemicals.

How do they enter the State's waterways?

PFAS are discharged directly through their use in manufacturing such as by DuPont and Solvay facilities; and firefighting – such as at Dept. of Defense installations where the foam was used in training and in firefighting; and through many pathways such as sewage and industrial wastewater treatment plants; landfills; application of sewage sludge or "biosolids" on farms and parkland; and degradation of consumer products. They travel by air, through groundwater and surface water, depending on the type of PFAS. They are mobile in water and are found in soils, sediment, vegetation, food, fish, and wildlife. For instance, short-chain PFAS have been found in vegetables such as celery and tomatoes grown in contaminated soils and in milk from cows who ate contaminated grass, the result of PFAS in sewage sludge applied as fertilizer to farm fields.[3] As PFAS have become more understood through research, the remarkable ability of these highly toxic compounds to enter the environment and the human body and stay there has astounded scientists, regulators, health professionals, and the public. In fact, the water crisis created by these compounds has caused worldwide disturbance as people wonder what their exposure to these toxic compounds is doing to themselves and their families.

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[1] http://www.c8sciencepanel.org/newsletter10.html

[2] https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/risk-management-and-polyfluoroalkyl-substances-pfas

[3] A Chem Trust Briefing (2019). PFAS the 'forever chemicals'. Retrieved from https://chemtrust.org/wp-content/uploads/PFAS_Brief_CHEMTrust_2019.pdf. P. 8.

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Extent in New Jersey?

Data shows that New Jersey has higher levels of PFAS contamination than other states. There are many sources of contamination, both legacy and ongoing, with especially high levels of PFNA, PFOA and PFOS. It is very difficult for people to avoid exposure here due to the ubiquitous nature of PFOA and PFOS and the fact we are the most densely populated state in the nation, with so many people living close to the sources of pollution. All Americans have some amount of PFAS in our blood; it's in ground and surface water and is even found in fish and sediment in most places in the state. NJDOH and DEP issued a consumption advisory for PFAS in fish and crabs in 2018. Drinking water in New Jersey showed a wide occurrence of PFAS across the state in both its early occurrence studies in 2006 and 2009 and is now finding even more locations since the full implementation of the MCLs. Because only 224 of NJ's 506 community water systems had been sampled for PFOA and PFOS prior to MCL implementation, many more places in NJ are now finding out they have it in their drinking water and must remove it. The cleanup of the sources of contamination such as DuPont, Solvay, and 3M, is an essential action that NJ is trying to address through regulation, litigation and required remediation of polluted sites. The Dept. of Defense has been sued by NJ for lack of cooperation regarding PFAS. The scale of the contamination is enormous and the struggle to make the polluters pay is still unfolding, pushed by the state's litigation against the offending corporations. But without the cleanup, PFAS will continue to spread and threaten lives and the environment.

Best ways to address this issue in order to protect the public health?

NJDEP has taken many actions to address PFAS compounds, more than most states, and was the first state to adopt a safe drinking water standard, or maximum contaminant level (MCL) for any PFAS compound – it was in 2018 for PFNA, the PFAS compound that shook New Jersey to its core in 2013, resulting in the reconvening of the then-shuttered Drinking Water Quality Institute, empowered with making state-based MCL recommendations for contaminants that foul drinking water. The adoption of MCLs for PFNA, PFOA and PFOS requires their removal from drinking water, a person's major pathway of exposure. This fundamental and desperately needed regulation took almost 15 years to reach fruition but it still puts NJ ahead of the federal government, who has not adopted any federal MCLs for PFAS. NJ's head start is thanks to the groundbreaking work of its nationally recognized scientists and the DWQI, providing unflinching and top notch science-based leadership by DEP and DOH.

And NJ has also adopted other important regulations covering these 3 compounds including adopting groundwater quality standards, which provide minimum remediation standards for the cleanup of contaminated sites and adding these 3 PFAS to the list of hazardous substances under the Spill Compensation and Control Act. Most recently, in January 2022, DEP established an Interim Specific Ground Water Quality Criterion for Constituents in Class II-A Ground Water of 0.002 micrograms per liter (µg/L) for chloroperfluoropolyether carboxylates (ClPFPECAs), the first state to do so, quite an accomplishment.

This was made possible because DEP and EPA scientists performed very difficult forensic analyses to identify and locate this extremely toxic chemical in the environment and local drinking water. This chemical is Solvay's so-called replacement for PFNA and this action is the first step, a unique one, in addressing one of the worst injustices in NJ's PFAS history - it turns out this PFAS chemical is more toxic than the original PFNA that Solvay was supposed be replacing. It's intolerably ironic that the same people who were exposed to one of the most highly toxic of the

51X

PFAS long-chain compounds – PFNA – have also been exposed to this newly discovered dangerous contaminant. And, it's a "so-called" replacement because it turns out this chemical has been used for decades at the Solvay plant. It slipped through EPA's bureaucratic cracks, it was allowed due to lax federal regulations riddled with loopholes that benefit chemical companies and it was hidden from the public because it was shrouded by Solvay's claim of it being a trade secret.

In fact, most of the unjustly burdened affected public still doesn't even know about it. With NJ's GW criterion, there's now a standard for cleanup and a way to further investigate and NJ is way ahead because of that. Further action and public outreach is much needed because the public's questions are begging for answers – where exactly has it migrated to, how does it affect the body, how is it removed from water, are my home grown vegetables safe to eat, is it impacting my and my family's health? This is one stunning episode of the PFAS water crisis that continues to unfold here in NJ and across the world.

Other NJ actions that could be taken:

- Effective cleanup of the Solvay facility - with robust public participation - to remove ALL PFAS from the soil and ground water, including the so-called replacement compound. Investigation into what Solvay is now using at their facility, claiming it to be a less toxic replacement for the replacement compound, to ensure its safety. Most people just don't trust them anymore, and for good reason.

- More blood studies and health assessments of people exposed to PFAS over long periods. The Paulsboro health study is crucially important. We also need studies in the Salem County communities impacted by the DuPont Chambers Works plant, now under Chemours, and in the regions around all NJ's military installations, as well as other locations being investigated by the AG office as part of the ongoing litigation. Training for medical professionals regarding diagnosis and blood sampling and programs that monitor for health effects in impacted communities is needed. People want to know and have a right to know whether PFAS is in their bodies, at what concentration, and how this will affect their health. One thing we know for certain – drinking water that contains PFAS increases a person's exposure and that can increase the risk of developing a disease linked to PFAS. And there are additional exposure routes that contribute to a person's body burden – air, soil, fish and other food, and consumer products. You won't know how much you have unless your blood is tested.

- We urge DEP to regulate PFOA, PFOS, and PFNA under Surface Water Quality regulations to provide restrictions on the discharge of these pollutants. Consideration of air quality standards should be explored. Soil, sediment and air must be monitored, especially if a release is suspected, and releases must be curtailed. PFAS compounds have been moved around the state, sometimes far from the original source of contamination, by discharges from treatment facilities, sewage plants, and sewage sludge and biosolids applications on agricultural fields. Unless the pathways of pollution are found and closed off through monitoring all potential media, these "forever chemicals" that will never go away and will continue to fan out into the environment, threatening all people in New Jersey, even those who may be remote from the original contamination.

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- Potential points of contamination that have not had broad sampling done should be proactively investigated, especially if water sources are not included in public water system monitoring. For instance, private water wells for residents who are within the impact zone of a potential PFAS contamination source.

- Consideration by DWQI should be given to adopting MCL for PFHxS. PFHxS, restricted as a persistent organic pollutant under the Stockholm Convention since 2009, is also found in drinking water here. Studies have found that PFHxS bioaccumulates faster than PFOS and is very persistent; a blood study in PA found higher levels of PFHxS than any other PFAS in those tested.

- Application of sewage sludge, also called "biosolids", to land should be prohibited until it is required that PFAS be tested for and removed from sludge prior to application.

- Consumer product regulations, such as requiring labeling and/or banning of PFAS in cosmetics, personal care products, clothing, food wrappers and containers, are an effective means of reducing exposure through everyday product use.

- Ban the use of PFAS compounds in all but the most essential uses that serve the public interest, and in those instances handle them as hazardous substances in all stages of handling, including waste disposal.

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Dear Catherine,

Thank you for the opportunity to speak tomorrow before the Committee.
Below are links to some documents that we think could be helpful for the Committee in its research on this issue.

1. Rob Bilott, "Exposure", documenting the contamination of communities and water supplies by DuPont: https://www.simonandschuster.com/books/Exposure/Robert-Bilott/9781501172823
2. Landmark paper on PFAS: Gloria B. Post, Judith B. Louis, R. Lee Lippincott, and Nicholas A. Procopio, *Environmental Science & Technology* 2013, 47, 23, 13266-13275 (Article), Publication Date (Web):November 4, 2013DOI: 10.1021/es402884x. https://pubs.acs.org/author/Post%2C+Gloria+B
3. Dr. Gloria Post's published paper on what actions states are taking concerning PFAS: "Recent US State and Federal Drinking Water Guidelines for Per- and Polyfluoroalkyl Substances" https://setac.onlinelibrary.wiley.com/doi/10.1002/etc.4863
4. Results of health studies in the mid-Ohio Valley of PFOA exposure from DuPont's Washington Works plant in Parkersburg, West Virginia.: http://www.c8sciencepanel.org/
5. Review of evidence of PFOA effects on the fetus: Paula I. Johnson, et. al., "The Navigation Guide—Evidence-Based Medicine Meets Environmental Health: Systematic Review of Human Evidence for PFOA Effects on Fetal Growth", 2014. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4181929/pdf/ehp.1307893.pdf
6. "Dark Waters" 2019 major motion picture based on "Exposure" by Attorney Rob Bilott, starring Mark Ruffalo: see https://en.wikipedia.org/wiki/Dark_Waters_(2019_film).
7. Delaware Riverkeeper Network comment on PFNA MCL that was proposed by NJDEP, with Cambridge Environmental Consulting Report: https://www.delawareriverkeeper.org/sites/default/files/DRN%20PFNA%20MCL%20Comment%20to%20NJDEP%20(2017-10-04).pdf
8. Delaware Riverkeeper Network comments on PFOA and PFOS MCLs proposed by NJDEP, with Cambridge Environmental Consulting Report on PFOA: https://bit.ly/2VuRnyZ and on PFOS: https://bit.ly/2C9NMOt
9. Delaware Riverkeeper Network: Virtual Forum on PFAS Contamination in Gloucester County (2021-05-10)
10. Delaware Riverkeeper Network: Highlights from Woodbury, NJ informational meeting on PFAS drinking water threat (2021-07-26)
11. Concerning Solvay Product contamination of the environment and drinking water, exposed 2020 with links to scientific articles: Letter from NJ Groups on PFAS and 2nd letter from groups to NJDEP re Solvay replacement chemical (2020-10-12)
12. Kyle Bagenstose, USA TODAY, "Is EPA putting interests of chemical companies ahead of your health? These experts think so, March 7, 2022. https://www.usatoday.com/story/news/2022/03/07/epa-regulation-dangerous-pfas-chemicals-raises-questions-red-flags/9224137002/?gnt-cfr=1

We are happy to further discuss these issues.
Regards,
Tracy

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NEW JERSEY
AMERICAN WATER

Testimony from Matthew Csik, New Jersey American Water
NJ Assembly Environment and Solid Waste Committee Hearing on PFAS
March 10, 2022 at 2 p.m.

Chairman Kennedy and members of the Assembly Environment and Solid Waste Committee, I am Matthew Csik, Director of Water Quality and Environmental Compliance for New Jersey American Water. I am honored to speak to you today, representing New Jersey American Water, the state's largest water and wastewater services provider.

New Jersey American Water successfully owns, operates and maintains seven surface water treatment plants, 267 wells and 21 wastewater treatment plants, as well as approximately 9,800 miles of water and wastewater pipes, all to serve customers in 190 communities throughout the state. Approximately 2.8 million residents – 1 out of every 3 people living in New Jersey – receive their water and/or wastewater service from us.

New Jersey American Water invests more than $400 million in systems upgrades statewide – more than a million dollars a day – to help ensure quality and reliability. In conjunction with this annual investment in infrastructure, New Jersey American Water's systems continue to meet or surpass federal and state water quality standards.

New Jersey American Water combines the dedication of water quality experts, cutting-edge research, and advanced technology to help recognize potential issues,

such as PFAS, before they impact water quality. We optimize treatment to help reduce the impact, and engineer sustainable solutions to help protect customers. We began making operational changes to achieve compliance with the NJ Department of Environmental Protection's PFAS regulations before they became effective in January 2021, so our customers can feel confident that the water we provide is safe.

PFAS (Per- and Polyfluoroalkyl substances) are a large group of manufactured organic chemicals that are used in a variety of products for their nonstick properties (e.g., Teflon, Scotchgard), as well as in industrial applications such as firefighting. Aqueous Film Forming Foam (AFFF) usage at military bases and airports may be sources of PFAS in drinking water supplies near those locations. From the US EPA's Unregulated Contaminant Monitoring Rule 3 (UCMR3), perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS) were detected in numerous public water systems. PFOA has been phased out of production, but replacement compounds, such as "GenX," have been developed and are increasingly being detected in the environment. There are thousands of PFAS compounds. The compounds have most commonly been detected in groundwater but have also been detected at elevated concentrations in surface waters.

PFAS compounds can be introduced into the environment, thus into drinking water utility source waters, through various ways. Industrial discharges from facilities manufacturing or utilizing PFAS compounds have historically been released into soils and surface waters. Due to the wide use of PFAS compounds and their lack of degradation in the environment, these compounds can be found in the soils, aquifers and surface waters of many areas of New Jersey. Once PFAS compounds

find their way to the aquifers, rivers, streams, and reservoirs of the state, they remain in these waters and create exposure risks to drinking water utility customers unless targeted treatment is installed by the utility.

PFAS have been linked to various toxicological issues and are highly persistent in the environment, which is why they are sometimes referred to as "forever chemicals." The U.S. Environmental Protection Agency (EPA) has set a non-enforceable Health Advisory Level of 70 nanograms per liter (ng/L) or parts per trillion (ppt) for combined PFOA and PFOS. New Jersey was the first state to set a maximum contaminant level (MCL) for PFAS with a regulation of perfluorononanoic acid (PFNA) (13 ng/L) in 2018, followed by PFOA (14 ng/L) and PFOS (13 ng/L) in 2020.

While many drinking water utilities are challenged by PFAS contamination, American Water has a cross-functional team focused on the scientific and regulatory framework related to PFAS detection and emerging technologies for removal. American Water's research group is actively involved in externally funded projects related to the detection, occurrence, and removal of PFAS. Our scientists are members of the technical advisory workgroup for Safe Drinking Water Act Processes and New Contaminants of the American Water Works Association, which has been actively contributing to the fast-paced changes related to detection and regulatory strategies for PFAS. American Water experts frequently collaborate with other leaders, state and federal regulators in departments of environmental protection, EPA, CDC, American Water Works Association, Water Research Foundation, universities and other organizations to better understand issues related to PFAS and public health.

Our Central Laboratory, located in Belleville, IL, is an EPA accredited lab that has the newest EPA approved methods for measuring PFAS, accredited and certified in New Jersey. In addition, our in-house team of research scientists and engineers is actively involved in two major studies being funded by external agencies and are evaluating PFAS method modifications to expand the number of compounds we can effectively measure.

New Jersey American Water leverages our size, diverse locations, and economies of scale to identify and mitigate the risks from PFAS and other chemicals that are not yet widely understood. Selecting the most efficient and cost effective PFAS removal process is strongly dependent on background water matrix composition and targeted PFAS.

New Jersey American Water has successfully piloted cutting-edge treatment strategies to effectively remove PFAS from several groundwater stations in our service territory. These include the use of Granular Activated Carbon and Anion Exchange Technology. In fact, the company's use of these treatments in PFAS removal projects were recognized with three awards in the past year.

Specific examples of our successful treatment of PFAS include the following:

Short Hills Well Station: New Jersey American Water has installed a cutting-edge temporary treatment system that uses anionic exchange resins to remove PFAS from the source water at our Short Hills Well Station. Different than Granular Activated Carbon, these resins are specifically designed to remove PFAS with less maintenance over time. This new technology not only removes PFAS contaminants that are already regulated, but also has shown the ability to remove shorter-chain PFAS more effectively than Granular Activated Carbon. New Jersey American Water was awarded the 2020 Governor's Environmental Excellence

Award and a Commerce and Industry Association of NJ 2021 Environmental Award for the Short Hills Well Station Project.

Green Brook and Charles Street Stations: At times, the best action to take in response to finding PFAS presence is to remove ground water sources from service, though it is not always possible. When confronted with just such an issue with our Green Brook and Charles Street Stations, New Jersey American Water found a different way to address PFAS detections. The Green Brook and Charles Street Ground Water Stations were converted to booster stations, bringing treated surface water from our Canal Road and Raritan-Millstone Water Treatment Plants into service areas that previously received only ground water.

Springfield Station: New Jersey American Water constructed a new treatment system for PFAS removal from the Springfield Well Field. The new treatment system consists of four anion exchange resin vessels housed in the existing treatment building on the site, chemical feed system upgrades including sodium bisulfite, sodium hypochlorite, and ammonium sulfate constructed in the existing chemical rooms, and new low lift pumps installed to accommodate new head conditions through the new treatment system. This proactive and innovative approached earned New Jersey American Water the New Jersey Alliance for Action's Leading Infrastructure Project Award in February 2021.

American Water's engineering and research teams also regularly conduct studies to evaluate new monitoring and treatment technologies so we can remain at the forefront of water quality science.

New Jersey American Water's water treatment technology, combined with its proactive infrastructure investment program and the expertise of more than 800 professionals across the state work together to support the continued provision of high quality and reliable water service to our customers. We stand ready to continue to meet or surpass new water quality standards as they are implemented.