

150 West State Street · Trenton, NJ 08608 · 609.392.4214 · 609.392.4816 (fax) · www.chemistrycouncilnj.org

February 5, 2018

VIA ELECTRONIC MAIL

watersupply@dep.nj.gov Drinking Water Quality Institute

RE: REQUEST FOR PUBLIC INPUT FOR PERFLUOROOCTANE SULFONATE

To Whom It May Concern:

On behalf of our members, the Chemistry Council of New Jersey (CCNJ) and the Site Remediation Industry Network (SRIN) appreciate the opportunity to provide comments to the Drinking Water Quality Institute (DWQI) pursuant to the Institute's request for public input regarding the recently released subcommittee reports on Perfluorooctane Sulfonate (PFOS). CCNJ/SRIN have long advocated for greater transparency and public input with respect to DWQI's activities and we appreciate the steps taken to provide this opportunity.

General Concerns

Based upon available science and data, as further detailed below, we have significant concerns that DWQI's current recommendations related to PFOS are not justified, could not be feasibly implemented by New Jersey water providers, and is not supported by an objective analysis of the available science and data. As such, CCNJ/SRIN strongly recommend that DWQI's current draft Maximum Contaminant Level (MCL) for PFOS be held until such time that scientific evidence can support its recommendation. In the alternative, we urge DWQI to further review the detailed scientific data and literature that was either ignored or missed in its current review of PFOS before submitting a recommendation to the New Jersey Department of Environmental Protection (NJDEP); the following are specific examples:

- Rutgers Environmental Health and Occupational Health Sciences Institute and School of Public Health. 2017. Rutgers Pilot Study of Perfluorochemical Compounds in Paulsboro Residents, Preliminary Study Report. September 13.
- enHealth. 2016. Statement: Interim national guidance on human health reference values for per- and poly-fluoroalkyl substances for use in site investigations in Australia. June. <u>http://www.health.nsw.gov.au/environment/factsheets/Documents/pfas-interimhealth-values-ahppc.pdf</u>



• Chang ET, Adami HO, Boffetta P, Cole P, Starr TB, and Mandel JS. 2014. A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and cancer risk in humans. *Crit Rev Toxicol*. 44(S1):1-81.

We believe that it is in the best interests of public policy and public health in New Jersey to review this science prior to any final PFOS MCL recommendation from DWQI. These resources will provide valuable insight to DWQI and allow for the review of the best information currently available. The Rutgers report is further discussed below.

CCNJ/SRIN and our members continue to advocate for DWQI's and NJDEP's transparent and thorough consideration of the *Rutgers Pilot Study of Perfluorochemical Compounds in Paulsboro Residents, Sept. 13, 2017* (Attachment 1), and the underlying Perfluorinated Compound (PFC) blood sampling data from Paulsboro.

The data gathered by Rutgers is the only available scientifically-gathered evidence of PFC blood serum levels in residents who consumed water from municipal wells affected by Perfluorononanoic Acid (PFNA), and one of the few paired data sets for Perfluorooctanoic Acid (PFOA) or PFOS. To date, DWQI has not acknowledged that these data exist. Ignoring these data undermines the credibility of DWQI's PFC recommendations. The Paulsboro data are readily available, reliable, recent and *local*, and directly relevant to DWQI's recommendations, including for PFOS, which was detected in both the Paulsboro water supply and in residents' blood serum. CCNJ/SRIN urge DWQI to include these data in its regulatory consideration/calculation.

The more than 1,000-page report by the DWQI Health Effects Subcommittee, which includes more than 30 pages of references alone, does not mention Paulsboro or the fact that more than 1,000 residents of Paulsboro had their blood sampled for PFCs, including PFNA, PFOA, and PFOS, in 2016. Nor does it mention that Rutgers enrolled 181 Paulsboro residents in a study and, in cooperation with Rutgers, those residents shared their blood serum results and information about their use of the Paulsboro water system with the Rutgers research team. Additionally, a large subset, 116 residents, answered detailed questions for Rutgers, including water consumption information and health conditions that may be associated with PFCs.

Based on the existence of a questionnaire that was formally developed and approved by Rutgers (Attachment 2), we believe that water consumption data was collected during the Paulsboro study. This questionnaire includes very specific questions about water consumption; please see excerpt below:



"SECTION 3

The next questions are about the time BEFORE you knew about the PFNA in the drinking water and BEFORE you or the borough of Paulsboro took steps to reduce your PFNA exposure.

During the time that you lived in a home served by Paulsboro public water supply, and **BEFORE you knew about the PFNA in the drinking water**, about how many **8 oz cups** of tap water or beverages prepared with tap water did you usually drink per day?

Note: 1 Gallon (128 oz.) = 16 cups; 1 quart (32 oz.) = 4 cups; 1 pint (16 oz.) = 2 cups

_____ Cups per day"

According to the final, published Rutgers report, 116 long form surveys were completed by Paulsboro residents, so it appears that very direct questions were asked of residents and collected by Rutgers about water consumption.

Rutgers published and provided to the residents a report that analyzed those 181 blood serum results. That report, entitled *Rutgers Pilot Study of Perfluorochemical Compounds in Paulsboro Residents, Sept. 13, 2017*, and the underlying data gathered by Rutgers upon which it is based, should be evaluated as part of DWQI and NJDEP regulatory considerations for PFCs, as the data would allow a direct assessment of some of the key assumptions made by DWQI regarding the association between PFC drinking water concentrations and blood serum levels. In addition to the detailed information collected on a subset of the residents, Paulsboro itself has records of when it used its various wells to supply its residents with drinking water and, therefore, there exists a basis for understanding residents' drinking water exposures and associated PFC blood serum levels. However, in the event that Rutgers ultimately did not collect the water consumption data, CCNJ/SRIN would like an explanation as to why not given its direct relevance to the study. In terms of advancing the science, if this data was collected, it would be far more useful for the data's existence to be publicly acknowledged and an explanation given by Rutgers and the State regarding why they are not taking the logical next step to evaluate it.

In addition, we understand that it is possible that there were data quality issues. Yet, this would be puzzling given that Rutgers did use information obtained from other questions in the same survey, for example to help group results by age and sex. If New Jersey is going to be the first in the United States to regulate far and beyond the United States Environmental Protection Agency (USEPA)'s standards, then the data puts researchers in a unique position to support such an action. They can examine available data from the surveys and water sample results to provide



some clarity to assumptions that DWQI and NJDEP are relying upon in their calculations. This can also advance the scientific understanding for PFNA, PFOA, and PFOS, in general.

The Paulsboro study is relevant to DWQI's PFOS MCL recommendation because it includes measurements of multiple PFCs, including PFOA and PFOS. If the assumption is that human health effects of PFNA, PFOA, and PFOS are driven by concentrations in our bodies, the link between external exposure through drinking water and someone's internal dose needs to be calculated with extreme rigor.

There are simply not that many datasets available that provide this information. This is a study of approximately 200 individuals, each of whom provides a direct measure of the same variable that NJDEP is trying to estimate. By comparison, in terms of sample size alone, this study is <u>four times the size</u> of the one and only study in humans NJDEP and DWQI relied upon to support their estimate of the half-life of PFNA in serum; that study only had 50 participants. NJDEP defended its position to use that study in its public response-to-comments, indicating that they were confident such information would support a central tendency estimate of the serum:water ratio. Why not take the next step to evaluate this study?

On the sample design itself, CCNJ/SRIN agree that the data were not collected in a scientifically rigorous way. However, the data still provide important information, not the least of which would be a check on whether the assumptions adopted by DWQI and NJDEP are consistent with data for each of the PFCs (PFNA, PFOA, and PFOS) for this specific sample. For example, Rutgers could examine data on serum and water levels to determine if individuals with elevated serum levels (higher than NHANES) also have higher exposures based on the reported water consumption rates and the concentrations in water (compared to the proposed MCL).

The Paulsboro dataset may prove useful to explore many of the assumptions made for PFNA, PFOA, and PFOS. There should be a transparent discussion of its strengths and weaknesses.

DWQI and NJDEP rely heavily on their assumptions about how PFCs are retained in human blood (versus actual data) to recommend MCLs as extremely low and unprecedented as 13 parts per trillion (ppt) for PFNA and PFOS and 14 ppt for PFOA. These levels are far lower than guidance from USEPA without scientific justification or evidence. Importantly, the levels do not appear defensible when compared to actual empirical data.

For example, for PFNA, the DWQI MCL recommendation is based on the assumption that 4.9 parts per billion (ppb) of PFNA in human blood is an appropriate protective target serum level.



However, according to Rutgers, the measured mean level of PFNA in the blood of 181 Paulsboro residents is 3.6 ppb. In other words, the actual data are below the target level that NJDEP and DWQI have determined is protective. And, yet, Paulsboro drinking water well No. 7 had measured levels of PFNA near 100 ppt or more in August of 2009 and in October 2013 through when the well was taken offline in April 2014. This concentration of PFNA in drinking water is over 7 times higher than the recommended MCL; however, the residential blood serum data shows that serum PFNA levels did not exceed DWQI's target human blood level.

The concentrations of PFNA, PFOA, and PFOS in blood serum of almost 200 residents in Paulsboro have been accurately measured. If 100 ppt in drinking water did not cause the average level in blood serum to exceed the level DWQI and NJDEP used to calculate the MCL, then why would NJDEP and DWQI insist that water suppliers across the state must test for PFNA down to 2 ppt, and install expensive treatment to keep the level of PFNA in their water supplies below 13 ppt?

In addition, we advocate consideration of the study and the underlying data because:

- 1. The data *are* reliable. Phlebotomists were used to gather the samples and a New Jersey certified lab was used to analyze them. Rutgers itself relies on the data in issuing its report.
- 2. They are the *only* available empirical data involving measured quantities of PFCs in drinking water and in human blood serum of New Jersey system users.
- 3. More than 1,000 Paulsboro residents chose to have their blood serum levels sampled for PFCs and 181 of that group chose to make the results available to Rutgers, in response to its request. No one claims that this is a random sampled population necessary for a health study, but it is false to suggest that this data could have no scientific value as to the very assumptions, especially the serum:drinking water ratio, that DWQI has made in their proposed MCLs for several PFCs.

Scientists are trained and able to recognize and evaluate sample size and selection bias, as well as time of exposure versus time of sampling, and use empirical data for appropriate purposes. In this case, *valid, directly relevant data are available* to compare to assumptions being relied on by DWQI and NJDEP to the PFCs actually detected in New Jersey residents using affected water. The residents of Paulsboro and all New Jerseyans deserve a straightforward discussion and consideration of the Paulsboro residents' blood results.

If these unwarranted proposed MCLs are adopted, towns and small public water purveyors will simply be unable to manage testing and treatment to low ppt levels. Consumers will pay the



price for water sampling and treatment costs that are not scientifically justified. Public water providers are not represented on DWQI and may have no choice but to sell their systems to investor-owned water utilities, who will pass the costs through to consumers through rate hikes.

Importantly, we support the use of the best available science; CCNJ/SRIN have always held this position. One recent example is the Site Remediation & Waste Management Program (SRWMP)'s revisions to Soil Remediation Standards (SRS). We submitted a letter of support to then-NJDEP Commissioner Bob Martin because we agreed that the latest USEPA Integrated Risk Information System (IRIS) toxicity values should be incorporated into NJDEP's calculations in determining revised SRS. CCNJ/SRIN stated our support of SRWMP's efforts because we support the use of the best available science, irrespective of whether the numbers ultimately increase or decrease.

Comments on Subcommittee Reports

Please refer to our third attachment for a discussion of specific examples of limitations in DWQI's evaluation of health effects and treatment.

<u>Summary</u>

As discussed above and in Attachment 3, DWQI is proposing an MCL for New Jersey that is far lower than the guideline the federal government recently determined is protective for drinking water. No additional known health protection is achieved, suggesting the DWQI proposal does not overcome the additional cost and reporting/regulatory burden that would unnecessarily hinder economic growth and success in New Jersey.

CCNJ/SRIN strongly urge that the report entitled *Rutgers Pilot Study of Perfluorochemical Compounds in Paulsboro Residents, Sept. 13, 2017*, and the underlying data gathered by Rutgers upon which it is based, be evaluated by DWQI and NJDEP, as the data would allow a direct assessment of some of the key assumptions made by DWQI regarding the association between PFC drinking water concentrations and blood serum levels.

DWQI must be mindful of the science being developed in other states and by the federal government. The works completed by other states/countries and USEPA are also informative to DWQI's PFOS review. It is imperative that the Institute review these works, as they clearly help identify the flaws in New Jersey's current scientific literature regarding PFOS. In situations where urgency is required and federal guidance is available, it is a sound policy for the State to rely on



the federal guidance and allow the scientific process to develop data to support MCLs and other New Jersey environmental standards.

Thank you for the consideration of our comments on this very important issue. We look forward to working with DWQI as it continues its work in recommending drinking water quality standards in New Jersey. If I can be of further assistance, please let me know.

Sincerely,

Dennis Hart Executive Director

Attachments



Chang ET, Adami HO, Boffetta P, Cole P, Starr TB, and Mandel JS. 2014. A critical review of perfluorooctanoate and perfluorooctanesulfonate exposure and cancer risk in humans. *Crit Rev Toxicol*. 44(S1):1-81.

enHealth. 2016. Statement: Interim national guidance on human health reference values for perand poly-fluoroalkyl substances for use in site investigations in Australia. June. <u>http://www.health.nsw.gov.au/environment/factsheets/Documents/pfas-interim-health-</u> values-ahppc.pdf

Rutgers Environmental Health and Occupational Health Sciences Institute and School of Public Health. 2017. Rutgers Pilot Study of Perfluorochemical Compounds in Paulsboro Residents, Preliminary Study Report. September 13.



150 West State Street · Trenton, NJ 08608 · 609.392.4214 · 609.392.4816 (fax) · www.chemistrycouncilnj.org

February 5, 2018

ADDITIONAL PERFLUOROOCTANE SULFONATE (PFOS) COMMENTS

Drinking Water Quality Institute (DWQI) PFOS Subcommittee Reports

Comments on the subcommittee reports are provided below and cover the following key points:

- 1. The federal United States Environmental Protection Agency (USEPA) and other agencies that have comprehensively reviewed the available scientific evidence recognize the uncertainty in the available data and do not share DWQI's perspective on potential health effects of PFOS in drinking water at the proposed Maximum Contaminant Level (MCL).
- 2. DWQI did not consider the effect of exposure duration and route of exposure in their PFOS MCL derivation.
- 3. DWQI's selection of the direct toxicity (immune system) no observed adverse or lowest observed adverse effect levels (NOAEL and LOAEL, respectively) is questionable because of the presence of systemic toxicity (liver).
- 4. DWQI fails to provide any context regarding the proposed Target Human Serum Level.
- 5. DWQI has not evaluated the feasibility of achieving the MCL, nor has it provided an assessment of the potential utility and efficacy of treatment technologies other than granular activated carbon (GAC).

Health Effects Subcommittee Report

USEPA has gathered a great deal of data, nationally, on PFOS and its potential health effects and, very recently, issued a federal protective guideline of 70 parts per trillion (ppt) for Perfluorooctanoic Acid (PFOA) + PFOS in drinking water. DWQI rejects the federal government's careful analysis and replaces it with its own approach which would lead to a far stricter guideline being imposed on the communities and businesses of New Jersey.

USEPA recognizes the lack of scientific evidence and uncertainties associated with the science related to any health effects associated with PFOS. In this regard, the choice of immunological effects as the critical effect is inconsistent with other regulatory agency review (USEPA; ATSDR; Australian Department of Health; Danish EPA) that have concluded that this endpoint requires further study before it can be considered human-relevant at the dose chosen as the Point of Departure (POD). Criticism of this endpoint from the other regulatory reviews included



inconsistent immunosuppressive effects across studies in the database at this dose, questionable human relevance of the observations in mice, and unclear functional changes of in vitro effects at this dose suggesting that the findings may not represent an adverse effect. Further, epidemiological evidence in humans are inconclusive on the potential immunotoxicity of PFOS exposure, casting further doubt on the relevance of this endpoint to humans.

Cancer versus Noncancer Endpoint

At the onset, it is appropriate that the MCL be based on the noncancer endpoints, but not for the reasons provided in DWQI's PFOS Health Effects Subcommittee Report. For the cancer endpoint, a cancer slope factor was derived from the incidence of hepatocellular tumors in **female rats only** as male rat data was "uncertain" because the tumor occurrence was at high dose only (Butenhoff et al., 2012). The importance of this finding was missing in the mode of action (MOA) assessment for PFOS in this document. Based on the Butenhoff et al., 2012 feeding study documentation of tumor formation in high dose female and male rats (20 parts per million (ppm)), other important non-neoplastic, adaptive changes occur in the liver, including hepatocellular hypertrophy with proliferation of endoplasmic reticulum, vacuolation, and increased eosinophilic granulation of the cytoplasm in both males and females at the higher exposure concentrations. These findings are consistent with a threshold MOA due to chronic cellular injury, repair and proliferation. However, the document focused only on the role of peroxisome proliferator activated receptoralpha (PPAR α), which is only one of many potential mechanisms for the histopathological sequela of events leading to tumor formation because of chronic cell injury.

The threshold or noncancer approach is supported by the high dose and one sex/species finding, in addition to the lack of significant tumor formation in the recovery group, indicating that once exposure (and cell injury) is terminated, progression to tumor formation does not occur. Thus, if the noncancer endpoint (liver injury) can be prevented, the cancer endpoint will not develop. In addition, the threshold and, thus, noncancer endpoint risk assessment method application is consistent with the lack of mutagenicity or genotoxicity in PFOS studies. DWQI's document would be much improved by synthesizing the database when assessing the weight of evidence, MOA, and relevance to human exposures consistent with USEPA guidance, including Framework for Determining a Mutagenic Mode of Action for Carcinogenicity and International Program on Chemical Safety (IPCS) Mode of Action Framework (for cancer and noncancer risk assessment).



Indicator of External Exposure

Using serum PFOS levels as an indicator of internal exposure is appropriate since there is published literature demonstrating the dose-response relationship between the internal dose (serum in nanograms per liter (ng/l)) and effects, which is inconsistent with the administered dose (milligrams per kilogram per day) (mg/kg/d)). The latter is the result of many factors, including experimental design such as route of administration (diet versus gavage), as well as species and sex of experimental animals. However, the importance of the differences between the administered and internal dose was not discussed or weighted in DWQI's key study evaluation for quantitative determination of the NOAEL and LOAEL.

Dosing Regimes

The implication of the difference in gavage (or bolus) and dietary dosing regimens is relevant to DWQI and the New Jersey Department of Environmental Protection (NJDEP) in the selection of POD, NOAELs and LOAELs, and, as such, the determination of the reference dose (RfD) used to develop the proposed health-based MCL.

In Table 38 of DWQI's PFOS Health Effects Subcommittee Report, the PODs, NOAELs, and LOAELs based on serum PFOS concentrations from four key studies are provided along with the target endpoint. This is reproduced below for illustration, with the addition of one column for route of administration/duration of exposure and two rows for additional studies (Dong et al., 2011 and Qazi et al., 2010 (2010a reference in the Draft MCL documentation)). While the POD for these two additional studies were not determined for purposes of this review, the NOAEL and LOAEL for immunotoxicity or immunomodulation is provided. These additions better inform the interpretation and selection of the key study for MCL derivation.

In DWQI's MCL support document, the study used to derive the MCL was Dong et al., 2009. As can be gleaned from the table, this study was a 60-day oral gavage study, as were the rest of the key studies identified by the authors of this document except for the Butenhoff et al., 2012 study, which was chronic (up to 104 weeks) dietary administration up to 20 ppm PFOS.



Study	Endpoint	POD	NOAEL	LOAEL	Route/Duration
		(ng/ml)	(ng/ml)	(ng/ml)	of Exposure
Butenhoff	Hepatocellular	4,560.8	2,554ª	11,724ª	Dietary, 20 ppm
et al., 2012	hypertrophy	(BMDL)			for up to 104
	(male rats)				weeks
Dong et al.,	Relative liver	5,585,5	674	7,132	Oral gavage, 60
2009	weight increase	(BMDL)			days
	(male mice)				
Dong et al.,	Relative liver	4,350	4,350	8,210	Oral gavage, 60
2012a	weight increase	(NOAEL)			days
	(male mice)				
Dong et al.,	Decreased	674	674	7,132	Oral gavage, 60
2009	plaque forming	NOAEL)			days
	immune				
	response (male				
	mice)				
Dong et al.,	Decreases IgM		2,360	10,750	Oral gavage, 60
2011	and increases IL-				days
	4 cytokine (male				
	mice)				
Qazi et al,	No adverse		11,600		Dietary, 5.55
2010	immune function				mg/kg, 28 days
	(male mice) ^b				

Table 38 of DWQI's PFOS Health Effects Subcommittee Report

^a Based on AUC

^b Liver toxicity (increased weight liver weight, decreased body weight gain)

Italics – added to DWQI's Draft Document Table 38

It is well established that the route of administration has profound effects on the internal dose, e.g., serum concentrations, as demonstrated in various sources (Marty et al., 2007, Hayes 2007). Daily exposure by oral gavage results in bolus doses is inconsistent with dietary or drinking water exposures, lacking relevance to human exposures such as drinking water. In studying the difference in dosing regimens, Marty et al., 2007 reported that gavage administration resulted in an order of magnitude higher blood levels than the dietary route of exposure. Instead of considering the route of administration (bolus versus dietary), DWQI chose not to use the dietary data because it resulted in less stringent doses than the bolus, which is a flawed assessment. As further support for this critical point, researchers opine that gavage administration should be



abandoned for hazard assessments associated with endocrine disruptors like PFOS (Vandenberg et al. 2014).

Referring to the liver toxicity endpoints in the table above, the NOAEL and LOAEL (a sensitive indicator of liver toxicity – microscopic liver cell hypertrophy) from the chronic dietary administration of PFOS are 2,554 and 11,724 ng/ml, respectively. This indicates that higher levels of PFOS are tolerated without affecting liver hypertrophy when compared to the oral gavage studies producing liver weight increases with NOAEL and LOAEL serum concentrations of 674 to 8,210 ng/ml, respectively. If Butenhoff et al., 2012 study's liver cell hypertrophy was selected as the MCL endpoint, a higher RfD by a factor of approximately 4 would have been developed compared to the less sensitive indicator of liver toxicity (liver weight increase) in the Dong et al., 2009 study. Higher RfD would result in a higher MCL. While the liver toxicity endpoint was not selected for the MCL, this demonstrates the dramatic differences in kinetics and exposure levels producing toxicity from 60-day gavage or bolus versus chronic dietary administration. This important difference was NOT considered in DWQI's document. As noted previously, oral gavage or bolus dosing is not consistent with humans exposed to concentrations in environmental media, including drinking water.

Immunotoxicity

PFOS administration to laboratory animals, including mice and rats, can produce toxicity such as body weight loss and liver enlargement, as well as effects on the immune system. However, for many studies, it is unclear whether PFOS is directly immunotoxic or is a result of general toxicity and stress. As reported in DWQI's document, PFOS exposure results result in suppression of adaptive immunity without toxicity; however, the administrated doses and serum concentrations at which these effects are produced vary widely.

It is key to be able to compare results of studies with the same endpoint and, preferably, the same route of administration. Dong et al., 2011 did not find effects on body, spleen, or thymus weight with oral gavage exposure for 60 days and evaluated functionality of the immune system by measuring antibody and assessed delayed hypersensitivity. The Dong et al., 2011 study serum NOAEL and LOAEL for immunotoxicity were 2,360 and 10,750 ng/l, respectively. This study was not considered in the final study selection for MCL derivation. However, an earlier study by Dong (Dong et al., 2009) was selected in this evaluation for effects on the immune system (decreased plaque forming immune response) as well as liver weight increase (no histology conducted).



The study selected as evidence of direct immunotoxicity (Dong et al., 2009) had signs of liver toxicity as well as immunotoxicity, while the more recent study did not. Dong et al., 2011 produced immunotoxicity without any other signs of toxicity that would confound the interpretation of direct immunotoxicity. The Dong et al., 2009 NOAEL is 3.5 lower than that from the Dong et al., 2011 study that effectively resulted in a lower PFOS MCL than would have been derived from the Dong et al., 2011 study.

The selection of Dong et al., 2009 and the endpoint of immunomodulation (plaque forming cell assay results) is questionable as described above. Lefebvre et al., 2008 assessed the effects of PFOS on the immune system from dietary for 28 days at levels ranging from 2 to 100 mg/kg that are known to alter hepatic function. The authors concluded that "changes in immune parameters in rat did not manifest as functional alterations in response to immune challenge with KLH and may be secondary to hepatic-mediated effects of PFOS in this model" (Lefebvre et al., 2008). Therefore, for derivation of the MCL, hepatic endpoints would be the more sensitive endpoint and should have been considered rather than immune modulation. It does not appear that this study was considered in DWQI's MCL evaluation.

As discussed above, nondietary studies produce liver effects at lower internal exposure levels (serum ng/l). This is supported from immunomodulation studies as well. Dietary exposure for 28 days in rats found no effects on immune tissue weight, cellularity, plaque forming cell assay, or cell activity (i.e. serum IgM and IgG (Qazi et al., Int Immunopharmacol. Nov;10(11):1420-7 (2010b reference in the Draft MCL documentation))). However, there was other evidence of toxicity (i.e. decrease in body weight gain and increase in liver weight). The NOAEL serum concentration for immunotoxicity was 11,600 ng/l but the NOAEL may be higher since this was the only dose studied.

As can be seen in the table above, dietary route of exposure does not produce adverse impacts on the immune system at much higher internal exposure levels compared to the Dong et al gavage studies. Previous studies by Qazi et al evaluated a wider range of exposure doses and concluded that, in contrast to gavage studies, dietary exposure to environmentally relevant doses does not compromise humoral immune response. This finding is supported by Lebevre et al., 2008 (dietary study in male and female rats), where the authors found dietary exposure did not correspond to findings from oral gavage studies.

Apparently, Qazi et al., 2010 negative findings were dismissed from consideration in this evaluation because of positive findings in other studies evaluating plaque forming cells all using



oral gavage (e.g. Dong et al., 2009 and 2011 (Table 44)). This negative finding was explained by "methodological difference" but the finding was dismissed rather than putting the results in context of bolus dosing. This process appears to be biased and not scientifically robust.

Based on Table 42 of DWQI's PFOS Health Effects Subcommittee Report, and using Butenhoff et al., 2012 as the most sensitive noncancer endpoint (hepatocellular hypertrophy) for determination of MCL, the RfD of 12 ng/day was derived by the authors of the draft MCL document. The selection of endpoint and critical study alone would result in an approximately 7-fold higher MCL (i.e. 84 ppt versus 13 ppt). In conclusion, focusing on both factors cited above alone resulted in a scientifically flawed derivation of the PFOS MCL that is overly conservative.

Health-Based MCL Derivation Process

DWQI compares predicted serum PFOA levels to background levels but fails to provide any context regarding the proposed Target Human Serum level. To this point, the health-based MCL derivation process as outlined in DWQI's Figure E-2 is inconsistent with internationally accepted processes to extrapolate hazard information in animals to humans for risk assessment purposes (such as the principles outlined in the IPCS Environmental Health Criteria Monograph no. 104). The process followed by DWQI is non-standard, in that it applies uncertainty factors directly to the animal data prior to adjusting to a human equivalent dose using a clearance factor. The derivation and choice of clearance factor is not well-described, nor is the rationale for choice of adjustment factor clear given the application of adjustment factor to the serum dose versus external dose (i.e. what are the pros and cons for accounting for TK differences under DWQI's process versus internationally accepted processes?).

Treatment Subcommittee Report

Regarding treatment options for PFOS, the Health Effects Subcommittee Report correctly states (first paragraph on page ES-3 and first paragraph on page 9) that, while PFOS and other Perfluorinated Compounds (PFCs) are not effectively removed from drinking water by standard treatment processes, they can be removed from drinking water by GAC or reverse osmosis. However, the report fails to indicate that treatment via anion exchange resin (stand-alone or as a polish to GAC) may also offer significant improvement over stand-alone GAC treatment in terms of both treatment performance and cost effectiveness, particularly for PFOA and PFOS compounds. Since the promise of anion exchange as a treatment option is discussed in the Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water, as well as the Second Addendum to Appendix C, CCNJ/SRIN recommend that



this treatment option be included in the general discussion in the noted places within the Health Effects Subcommittee Report.

In addition, DWQI does not evaluate the feasibility of water suppliers of all kinds and types across the state implementing carbon or other treatment on their water supplies. This failure means that DWQI has not evaluated the feasibility of implementing the MCL it recommends. This will result in water suppliers increasing costs to consumers in the state of New Jersey to treat the PFOS water.

The 2015 Appendix C document states (page 10) that "USEPA notes that "incineration of the concentrated wastes would be needed for the complete destruction of PFCs" (2014)", which is only a best management practice; there is no discussion of regulatory basis for how this waste may be classified under the Resource Conservation and Recovery Act (RCRA). Any discussion of availability and viability of treatment must consider and discuss regulatory disposal requirements (vs. recommendations) of any waste streams.

References

Hayes, AW. 2007. Principles and Methods of Toxicology (Fifth Edition). August 13.

International Program on Chemical Safety (IPCS). 2008. Framework for Analyzing the Relevance of a Cancer Mode of Action for Humans. October 10.

International Program on Chemical Safety (IPCS). 2008. Framework for Analyzing the Relevance of a Noncancer Mode of Action for Humans. October 10.

Marty MS, Domoradzki JY, Hansen SC, Timchalk C, Bartels MJ, Mattsson JL. 2007. The Effect of Route, Vehicle, and Divided Doses on the Pharmacokinetics of Chlorpyrifos and Its Metabolite Trichloropyridinol in Neonatal Sprague-Dawley Rats. *Toxicological Sciences*. 100(2), 360-373.

United States Environmental Protection Agency (USEPA). 2007. Framework for Determining a Mutagenic Mode of Action for Carcinogenicity: Using EPA's 2005 Cancer Guidelines and Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogen (EPA 120/R-07/002-A). September.

Vandenberg LN, Welshons WV, vom Saal FS, Toutain PL, and Myers JP. 2014. Should oral gavage be abandoned in toxicity testing of endocrine disruptors? *Environ Health*. 13:46. June 25.



Rutgers The State University of New Jersey Environmental Occupational Health Institute and School of Public Health Study of Perfluorononanoic Acid (PFNA) Serum Levels in Paulsboro Residents -2016

Section 1. NAME and DOB

What is your name? First:		Last	
What is your date of birth:	/	1	(MM/DD/YYYY)

Section 2. RESIDENTIAL HISTORY

The next part of survey is about the place or places you have lived between 1996 and today. By

places you lived, we mean a house, apartment, or room you lived in for more than 6 months.

What is your current street address?_____

What is the town?

IF TOWN IS PAULSBORO, THE SURVEY WILL SKIP STATE AND ZIP CODE

 What is the State?
 What is the zip code?

What year did you move into this home? _YYYY

What was the source of the tap water for drinking or cooking in this home?

- D Public water supply (town water)
- □ Private well
- Don't know

Have you lived in any other home for more than six months since 1996?

- □ Yes
- □ No (GO TO NEXT SECTION)

Second home





What is the State?	What is the zi	p code?	
	windt is the zi	p couc:	

What year did you move into this home? <u>YYYY</u>

What year did you move out of this home? YYYY

What was the source of the tap water for drinking or cooking in this home?

- Public water supply (town water)
- Private well
- Don't know

Have you lived in any other home for more than six months since 1996?

- □ Yes (CONTINUE)
- □ No (GO TO NEXT SECTION)

CONTINUE UNTIL THE ANSWER IS NO

SECTION 3

The next questions are about the time BEFORE you knew about the PFNA in the drinking water and BEFORE you or the borough of Paulsboro took steps to reduce your PFNA exposure.

During the time that you lived in a home served by Paulsboro public water supply, and **BEFORE you knew about the PFNA in the drinking water**, about how many **8 oz cups** of tap water or beverages prepared with tap water did you usually drink per day?

Note: 1 Gallon (128 oz.) = 16 cups; 1 quart (32 oz.) = 4 cups; 1 pint (16 oz.) = 2 cups

__Cups per day

During the time that you lived in a home **served by Paulsboro public water supply**, did you filter the water?

- Always
- □ Occasionally
- □ Never
- Do not know

During the time that you lived in a home **served by Paulsboro public water supply**, did you drink bottled water at home?





- □ Always
- □ Occasionally
- □ Never
- Do not know

The next questions are about private wells. Since 1996, during the time that you lived in Paulsboro, have you ever lived in a home served by a private well? Check all the apply.

- □ CURRENTLY using a private well in Paulsboro
- PREVIOUSLY used a private well in Paulsboro
- D NEVER lived in a home with a private well in Paulsboro (GO TO NEXT SECTION)
- Do not know/decline to answer (GO TO NEXT SECTION)

During the time that you lived in a home served by a **private well** in Paulsboro, how many 8 oz. cups of water and beverages prepared with well water did you drink per day?

Note: 1 Gallon (128 oz.) = 16 cups; 1 quart (32 oz.) = 4 cups; 1 pint (16 oz.) = 2 cups

____Cups per day

During the time that you lived in a home served by a **private well** in Paulsboro, did you filter the water?

- □ Always
- □ Occasionally
- Never
- Do not know

During the time that you lived in a home served by a **private well** in Paulsboro, did you drink bottled water at home?

- □ Always
- □ Occasionally
- □ Never
- Do not know

SECTION 4

The next questions are about any exposure to PFNA you may have had at your work.

Do you now or have you ever worked at the Solvay facility in West Deptford?





- □ Currently work in the Solvay facility in West Deptford
- □ Previously worked in the Solvay facility in West Deptford
- □ Never worked in the Solvay facility in West Deptford (GO TO NEXT Section)
- Do not know/refused (GO TO NEXT SECTION)

IF YES:

In total, how many years did you work at the West Deptford Solvay facility?

What year did you first work at the West Deptford Solvay facility?

What year did you last work at West Deptford Solvay facility?

What was the job title you had for the longest time while you worked at the West

Deptford Solvay facility?

What were your 3 main job duties at the West Deptford Solvay facility?

1)	
2)	
3)	 _

Did you work with PFNA or Teflon, or work in or near an area where others were using PFNA or Teflon at the West Deptford Solvay facility?

🛛 Yes	🗆 No	Don't know/Not sure
-------	------	---------------------

Do you now or have you ever worked at any other facility where you or others worked with or PFNA or Teflon?

- □ Currently
- □ Previously
- Never
- Do not know/refused (GO TO SECTION 5)

What is/was the name of the facility? _____





What street is/was it on?

In what town?

In what town?

In total, how many years in total did you work at this facility?

What year did you first work at this facility?

What year did you last work at this facility?

What year did you last work at this facility?

What were your 3 main job duties at this facility?

1)

2)

3)

Did you work with PFNA or Teflon, or work in or near an area where others were using PFNA or Teflon at this facility?

□ Yes □ No □ Don't know/Not sure

Do you now or have you ever worked at any **other** facility where you or others worked with or PFNA or Teflon?

□ Yes □ No □ Don't know/Not sure

IF YES WE GO THROUGH THE SAME QUESTIONS AS ABOVE

SECTION 5

Are you now or have you ever been a firefighter, either volunteer or for pay?

- □ Yes If yes, dates (e.g. 1996 to 2016)
- 🗆 No

From	to
From	_to
From	to

Do you now or have you ever worked for a company that installs carpets or that treats carpets for stain protection??





□ Yes If yes, dates (e.g. 1996 to 2016)

🗆 No

From	_to
From	_to
From	_to

During the past year did you eat freshwater fish/shellfish that were caught in streams, lakes or rivers near Paulsboro, West Deptford, Woodbury, Greenwich or the surrounding areas?

□ Yes

- 🗆 No
- Don't know

IF YES,

During the last 12 months, about how many times each month did you eat any kind of fish caught locally?

_____times per month

The next questions are about any tobacco use in your lifetime. This information is very important for us to be able to interpret your survey results.

Have you smoked 100 cigarettes or more in your entire life?

- □ Yes
- □ No (GO TO FIRST QUESTION ON E-CIGARETTES)

During the past 30 days, have you smoked part or all of a cigarette?

- Every Day or Always
- □ Some Days
- □ Not At All (GO TO FIRST QUESTION ON E-CIGARETTES)

During the past 30 days, on days you smoked, how many cigarettes did usually smoke... _____Cigarettes





Have ever used electronic or e-cigarettes even once?

- □ Yes
- □ No (GO TO FIRST QUESTION ON CHEWING TOBACCO)

During the past 30 days, have you used e-cigarettes...

- Every Day or Always
- □ Some Days
- □ Not At All

Have you ever used chewing tobacco, snuff, or snus (a Swedish dry tobacco) even once?

- □ Yes
- □ No (GO TO FIRST QUESTION ON CIGAR USE)

During the past 30 days, have you used chewing tobacco, snuff, or snus (a Swedish dry tobacco)...

- Every Day or Always
- □ Some Days
- □ Not At All

Have you ever used little cigars, cigarillos, or cigars even once?

- □ Yes
- □ No (GO TO FIRST QUESTION ON HOOKAH USE)

During the past 30 days, have you used little cigars, cigarillos, or cigars...

- Every Day or Always
- □ Some Days
- □ Not At All

1. Have you ever smoked tobacco in a hookah, even once?

- □ Yes
- □ No (GO TO SECTION 6)
- 23.1 During the past 30 days have you smoked a hookah, even once?
 - Every Day or Always
 - □ Some Days
 - Not At All





SECTION 6

The blood testing project's goal is to learn about levels of exposure to PFNA. By providing the following information, you are assisting us with learning about health conditions of concern in your community. Providing this information is completely voluntary. Rutgers University will protect the confidentiality of your information and will not share it with any person or entity.

About how much do you weigh without shoes? ____ Weight in pounds



About how tall are you without shoes? __/__ Height (Feet, inches)

Don't know/Prefer not to answer

Have you ever been diagnosed by a doctor with any of the following health conditions?

		CIRCLE THE CORRECT ANSWER		
Circulatory:				
	High blood pressure	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Coronary artery disease	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	High cholesterol	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Stroke	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Other circulatory? Specify other:	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
Autoimmune:				
	Lupus	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Type I diabetes	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Inflammatory bowel disease	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Ulcerative colitis	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Crohn's disease	NO	YES	If yes, what year were you <u>first</u>





				diagnosed? Year
	Multiple sclerosis	NO	YES	If yes, what year were you first
				diagnosed? Year
	Rheumatoid arthritis	NO	YES	If yes, what year were you first
				diagnosed? Year
	Other autoimmune?	NO	YES	If yes, what year were you first
	Specify other:			diagnosed? Year
Liver:				
	Hepatitis	NO	YES	If yes, what year were you first
				diagnosed? Year
	Enlarged liver	NO	YES	If yes, what year were you <u>first</u>
				diagnosed? Year
	Fatty liver disease	NO	YES	If yes, what year were you first
				diagnosed? Year
	Cirrhosis	NO	YES	If yes, what year were you <u>first</u>
				diagnosed? Year
	Other liver disease?	NO	YES	If yes, what year were you first
	Specify other:			diagnosed? Year
Neurological:				
	Alzheimer's disease	NO	YES	If yes, what year were you first
				diagnosed? Year
	Parkinson's disease	NO	YES	If yes, what year were you first
				diagnosed? Year
	AML- Lou Gehrig's	NO	YES	If yes, what year were you first
	disease			diagnosed? Year
	Other neurological	NO	YES	If yes, what year were you <u>first</u>
	disease? Specify other:			diagnosed? Year
	Thyroid:			
	Hypothyroidism	NO	YES	If yes, what year were you first
				diagnosed? Year
	Hyperthyroidism	NO	YES	If yes, what year were you first
				diagnosed? Year
	Other thyroid disease?	NO	YES	If yes, what year were you first
	Specify other:			diagnosed? Year
Kidney:				
	Chronic kidney disease	NO	YES	If yes, what year were you first
				diagnosed? Year
	End-stage renal	NO	YES	If yes, what year were you first
	disease			diagnosed? Year





	Other kidney disease? Specify other:	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
FOR FEMALES O	ONLY: Pregnancy:			
	Pregnancy induced hypertension	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Pre-eclampsia	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
	Other pregnancy problems? Specify other:	NO	YES	If yes, what year were you <u>first</u> diagnosed? Year
Cancer:		NO	YES	
	Specify cancer:			If yes, what year were you <u>first</u> diagnosed? Year
	Specify cancer:			If yes, what year were you <u>first</u> diagnosed? Year
	Specify cancer:			If yes, what year were you <u>first</u> diagnosed? Year
	Other conditions:	NO	YES	
	Specify:			If yes, what year were you <u>first</u> diagnosed? Year
	Specify:			If yes, what year were you <u>first</u> diagnosed? Year
	Specify:			If yes, what year were you <u>first</u> diagnosed? Year

Do you have any other specific health concerns about your PFNA exposure?

- □ Yes (GO TO THE OPEN TEXT FIELD)
- No (GO TO QUESTIONS ABOUT CHILDREN IN YOUR HOUSEHOLD)





Questions about children in your household

- Are you the parent or guardian of any children (under 18 years of age) living with you at your home address?
 □ Yes □ No (IF NO GO TO END OF SURVEY)
- 2. Are you the only parent or guardian of the child or children who is filling out this survey? □ Yes □ No (IF YES GO TO CHILD QUESTIONNAIRE)
- 3. Are you the OLDEST parent or guardian of the child/children who is filling put this survey? □ Yes □ No (IF NO GO TO END OF SURVEY: IF YES GO TO CHILD QUESTIONNAIRE)

For respondents who answered YES to Q1, and Q2 or Yes to Q1, No to Q2 and YES to Q3: Earlier you said you are the parent or guardian of a child or children who are living with you. We have a few questions about each, staring with the oldest child who lives with you at this address... TO CHILD QUESTIONNAIRE

FOR ALL OTHERS AND AFTER CHILD QUESTIONNAIRES ARE COMPLETED: End of Survey

Thank you for participating in the survey. We would like to offer you a \$20 CVS gift card in appreciation. You may either have a card mailed to your address (it will be mailed in 2-3 business days) or sent to your email (it will be sent in 1-2 business days). Please choose an option below to let us know which you would prefer:

- Please send me a card to my mailing address
- Please send the card to my email
- □ I do not want to receive a gift card.

If subject chooses mailing address, he is redirected to a Mail survey:

Please enter the following information to receive your gift card by mail:

Ν	а	n	n	e	:	

Mailing Address:	
(Street/PO.Box)	

___(City,

State, Zip) Thank you again for your participation!





If subject chooses email, he is redirected to an Email survey:

Please enter your email address:

@____

Thank you again for your participation!



Environmental Occupational Health sciences Institute Rutgers, The State University of New Jersey 170 Frelinghuysen Road Piscataway, New Jersey 08854

Rutgers Pilot Study of Perfluorochemical Compounds in Paulsboro Residents

PRELIMINARY STUDY REPORT

September 13, 2017

Environmental Occupational Health sciences Institute Rutgers, The State University of New Jersey 170 Frelinghuysen Road Piscataway, New Jersey 08854

THIS PAGE LEFT BLANK INTENTIONALLY

Environmental Occupational Health sciences Institute Rutgers, The State University of New Jersey 170 Frelinghuysen Road Piscataway, New Jersey 08854

BACKGROUND

Who we are

- A research team from Rutgers University
 - We are: environmental and public heath scientists, doctors, community outreach workers and other researchers.
- We are all members of The Center for Environmental Exposures and Disease (CEED)
 - CEED scientists work in and with NJ communities to understand, detect, prevent and solve environmental health problems.

Why we did the study

- In 2009, a chemical called perfluorononanoic acid (PFNA) was discovered in the Paulsboro public water supply.
 - PFNA is one of a group of chemicals called perfluorochemical compounds (PFC).
- These chemicals are concerning because:
 - They spread easily in the environment
 - They stay in our bodies and the environment for many years.
 - High blood levels of some PFC have been associated with some poor health effects.

What are perfluorochemical (PFC) compounds?

- PFC compounds are used in many manufacturing processes because they are resistant to heat, and repel water, and oil. Industries and jobs they are used in include:
 - Automotive, aerospace electronic, firefighting and others
- PFC are also used in many products we buy and have in our homes, including:
 - Carpeting, upholstery, fire-resistant clothing, food wrapping, non-stick cookware and others

PFNA was in Paulsboro water

- The New Jersey Department of Environmental Protection (NJDEP) recommends levels of no more than 0.01 PPB (parts per billion) in drinking water
- PFNA levels in some Paulsboro water samples were 10 times higher than the recommended level
- Since April 2014, Paulsboro's city water has been filtered to take out PFC from the water that goes to Paulsboro homes and businesses

ABOUT OUR STUDY

<u>The lawsuit</u>

- Because of the PFNA contamination in Paulsboro water, there was a class action lawsuit and settlement
 - The people who lived in Paulsboro and were part of the lawsuit were offered one PFC blood test
- We invited anyone who had a PFC test from the lawsuit to join our study.

What questions did the study try to answer?

- 1. What are the levels of PFNA and other PFC in blood of Paulsboro residents?
- 2. How do PFC levels compare those of all US residents?
 - Does this differ among people of different ages, gender, or race-ethnicity?
- 3. Were there any associations between PFC levels and self-reported health conditions?

Who could be in the study?

• Adults and children who were part of the class action lawsuit and had a copy of their PFC serum level test results.

How did the study work?

- Our research team met with eligible participants at the Independent Oil Workers Union Hall during six sessions between November 2016 and January 2017.
- A copy of the PFC blood test results was scanned onto secured servers, along with a one-page cover sheet that collected information on people's age, race/ethnicity, sex and address.
- Participants had the option to use our laptops to fill out an online survey about their drinking water habits, jobs, and health conditions.
- People who could not attend the in-person meetings but wanted to be in the study could be in the study by mail.

How did we compare PFC blood levels in Paulsboro residents to US residents?

- The US Center for Disease Control and Prevention (CDC) conducts a nationwide biomonitoring program, including blood tests for PFC, that is part of the National Health and Nutrition Examination Survey (NHANES)
- NHANES provides information on levels of chemicals in blood of people in the U.S.
- We compared PFC blood levels in study participants to the PFC blood levels across the U.S. from the most recently available NHANES data 2013 and 2014

WHAT WE FOUND

Who enrolled in the study?

- 194 people enrolled in the study
- Slightly more females than males were in the study
 - The percent of men and woman in the study is similar to that in all of Paulsboro
- People of different race and ethnic groups enrolled in the study.
 - The percent of people of different race and ethnic groups in the study is similar to that in all of Paulsboro.
- There were more older people than younger people enrolled in the study.

TECHNICAL NOTE: Because the people who enrolled in the study were on average older than in the Paulsboro's population, we have adjusted the study results shown in the following tables so they are more like what you would see for all people in Paulsboro.



What PFC were found in study participant's blood?

59% 60% 56% 50% 44% 38% 40% 30% 27% 30% 20% 15% 12% 11% 10% 0% 0-12 12-19 20-39 40-59 60+ Male Female Hispanic NH NH Black White Sex Race Age group

- 4 PFC were found in most study participants' blood
- PFHxS was found in over
 7 out of every 10 people
 tested
- PFOA, PFOS and PFNA were found in more than
 9 out of every 10 people tested
- These are the same PFC found in most people in the United States

Are the levels of these 4 PFC different from other people in the US?

PFOS and PFHxS blood levels in study participants were the same as those in the general US population.

PFOA and PFNA blood levels were higher in study participants than in the general U.S. population.

- The higher PFNA blood levels were expected because the Paulsboro drinking water had been contaminated with PFNA.
- PFOA was also found in Paulsboro drinking water, but at lower levels than PFNA.



TECHNICAL NOTE: The levels are shown as the 50th percentile, meaning half the people had a result below and half had a result above the values shown.

Are the levels of PFNA different in older and younger people or in men and women?

Yes, Overall PFNA levels are higher in older people than younger people in Paulsboro

- \circ $\;$ This pattern is also seen in the US population and other studies of PFC in people's blood
- \circ This is partly because of different water drinking patterns in different age groups.
- PFNA blood levels were higher in children than younger adults (age 20 to 39).
 - This is seen in other studies and is thought to reflect the amount of water people drink and the way people of different ages process of PFC.
- PFNA levels were higher in males than in females.
 - In the US population the levels are about the same in males and females.



HEALTH SURVEY RESULTS

HEALTH SURVEY RESULTS

What do the levels of PFC in blood mean for health?

- We do not know if the levels of PFC in people's blood in Paulsboro may cause an increased risk of any health problems.
- Scientists are working to better understand how different levels of PFC might affect people's health.
- The health effects of PFNA has not been studied as much as other some other PFC. Some, but not all, studies in people have found increased levels of some PFC to be associated with:
 - Effects on the developing fetus and child, including by possible changes in growth, learning, and behavior.
 - Decreased fertility and interference with the body's natural hormones.
 - o Increased cholesterol levels in blood.
 - Effects on the immune system.
 - Increased risk for kidney and testicular cancer.
- It has been not firmly established which, if any, specific health risks occur in people from exposures to PFC or what particular levels of PFC in blood are related to the above conditions.
 - This is because studies have all not found the same results, and
 - Many of these studies have been preliminary studies that just look at a snapshot of people's health and exposure in time.

What we found

- 116 people in the study also completed the longer survey.
- Some questions on the survey asked whether the person had ever been "diagnosed by a doctor with" specific health conditions.
 - Health conditions have been reported in some other studies to occur more frequently in people with higher levels of some PFC. We asked about the following health conditions on the survey:
 - Circulatory conditions like high blood pressure and high cholesterol;
 - Autoimmune diseases like rheumatoid arthritis and ulcerative colitis; and
 - Some kinds of cancer.

- In our study, we did not find any strong associations between PFNA levels in participants' blood and any of the health conditions listed above.
- Our data suggest that participants who said a doctor had diagnosed them with high cholesterol, on average, had higher levels of PFNA
 - Did PFNA cause higher cholesterol levels or other health effects among Paulsboro residents?
 - \circ $\,$ We cannot answer that question at this time. To begin to answer that question we would need:
 - A much larger study with hundreds or even thousands of participants
 - To collect information about people's health directly. One way to do this would be to take blood tests for the health condition ourselves (like blood cholesterol level) and/or get copies of peoples medical records.
 - To follow people over time to see if or how their health changes,
 - To make sure we can account for other factors associated with these health conditions.
- Cholesterol levels vary widely among different people and they can be effected by many things (from https://www.nhlbi.nih.gov/files/docs/public/heart/cholesterol_atglance.pdf)

Here is some information about high cholesterol and actions you can take. We suggest you talk to you doctor about your cholesterol level and if you need to be tested.

Things you cannot do anything about can affect your cholesterol levels:

- Age—As people get older, their cholesterol levels may get higher
- Gender—Men often have higher cholesterol levels then women
- Heredity—High cholesterol can run in families.

These you can do something about:

- Diet—Saturated fat and cholesterol in food may increase your cholesterol level
- Weight—Being overweight tends to increase your cholesterol level
- Physical activity—Regular physical activity can help lower LDL (bad) cholesterol and raise HDL (good) cholesterol.
- In our study we were able to account for differences in people's age, gender, race and smoking (ever, former, never), and body mass index (a combined measure of weight and height).
 - We could not account for factors like heredity, diet or physical activity

Recommendations and next steps

Paulsboro's community water supply is now filtered to remove PFC, including PFNA. The Paulsboro water supply is monitored for PFC by the NJ Department of Environmental Protection

- We recommend you keep a copy of your PFC blood result for you records
- If you have a health care provider, give him or her a copy of your PFC blood result for your medical record and give your doctor a copy of this report
- Screening tests for blood cholesterol are generally recommended for adult men and women at different ages, depending on individual risk levels. Consult with your health care provider about testing your blood cholesterol level.
- Because PFC are slowly excreted from the body, we expect that levels of PFNA among Paulsboro residents will decrease over time.
 - How quickly PFNA is excreted is not known
 - It will most likely take years, so we do not know when levels will return to levels similar to the general population.
- Consider joining a new study conducted by Rutgers (which may shed light on the excretion rates)

This report was prepared by members of the Rutgers Environmental Health and Occupational Health Sciences Institute (EOHSI) and School of Public Health, including:

Principal Investigators

Judith Graber, PhD Assistant Professor, Department of Epidemiology, Rutgers School of Public Health, Rutgers Environmental and Occupational Health Sciences Institute (EOHSI)	Clifford Weisel, PhD Professor, Department of Environmental and Occupational Health, Rutgers School of Public Health. EOHSI
Co-Investigators and Study Staff	
Cora Alexander, MPH Department of Epidemiology, Rutgers School of Public Health. EOHSI	Robert Laumbach, MD, MPH, CIH Assistant Professor, Department of Environmental and Occupational Health, Rutgers School of Public Health. EOHSI
Kathleen Black, PhD Program Manager, Clinical Research and Occupational Medicine, EOHSI	Panos Georgopoulos, PhD Professor, Department of Environmental and Occupational Health, Rutgers School of Public Health. EOHSI
Kerry Butch Program Specialist, EOHSI	Elizabeth Marshall, PhD Associate Professor, Department of Epidemiology, Rutgers School of Public Health
EOHSI research team members without whom this study could not have been conducted: Shahnaz Alimokhtari, MS; Clarimel Cepeda; Jennifer Gilman; Marta Hernandez; Alan Perez.	

This report, and other information about PFC in New Jersey, can be found online at: http://eohsi.rutgers.edu/news-and-events/community-outreach

This study and report are partially funded by the Center for Environmental Exposures and Disease, funded by the National Institute for Environmental Health Science for the last 29 years [ES005022]