Committee Report

REGULAR CALENDAR

February 26, 2019

HOUSE OF REPRESENTATIVES

REPORT OF COMMITTEE

The Committee on Health, Human Services and Elderly Affairs to which was referred HB 691-FN,

AN ACT relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies. Having considered the same, report the same with the following resolution: RESOLVED, that it is INEXPEDIENT TO LEGISLATE.

Rep. Jerry Knirk

FOR THE COMMITTEE

Original: House Clerk
Cc: Committee Bill File

COMMITTEE REPORT

Committee:	**Health, Human Services and Elderly Affairs**
Bill Number:	**HB 691-FN**
Title:	**relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.**
Date:	**February 26, 2019**
Consent Calendar:	**REGULAR**
Recommendation:	**INEXPEDIENT TO LEGISLATE**

STATEMENT OF INTENT

This bill would mandate that the Department of Health and Human Services pay for blood testing for perfluorinated chemicals in a number of situations, including a catch-all situation which would allow anybody in the state to request that the state pay for their testing. This test is a very expensive laboratory research test, not a clinical test. The cost to the department would be prohibitive. Self-selected voluntary testing is not a good method to gather valid epidemiologic data. Although we have data regarding the potential risks of several diseases with pre-exposure, we do not have data as to the direct health implications of specific blood levels. Providing data to individuals when the implications are unknown can lead to unnecessary anxiety and over testing which may be more harmful then the underlying exposure. The issue is not being ignored as the Department of Environmental Services is already investigating the problem statewide.

Vote 16-5.

Rep. Jerry Knirk
FOR THE COMMITTEE

Original: House Clerk
Cc: Committee Bill File

REGULAR CALENDAR

Health, Human Services and Elderly Affairs

HB 691-FN, relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies. **INEXPEDIENT TO LEGISLATE.**

Rep. Jerry Knirk for Health, Human Services and Elderly Affairs. This bill would mandate that the Department of Health and Human Services pay for blood testing for perfluorinated chemicals in a number of situations, including a catch-all situation which would allow anybody in the state to request that the state pay for their testing. This test is a very expensive laboratory research test, not a clinical test. The cost to the department would be prohibitive. Self-selected voluntary testing is not a good method to gather valid epidemiologic data. Although we have data regarding the potential risks of several diseases with pre-exposure, we do not have data as to the direct health implications of specific blood levels. Providing data to individuals when the implications are unknown can lead to unnecessary anxiety and over testing which may be more harmful then the underlying exposure. The issue is not being ignored as the Department of Environmental Services is already investigating the problem statewide. **Vote 16-5.**

Original: House Clerk
Cc: Committee Bill File

COMMITTEE REPORT

COMMITTEE: Health, Human Services and Elderly Affairs

BILL NUMBER: HB 691-FN

TITLE: relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies

DATE: 2-26-19 **CONSENT CALENDAR:** YES ☐ NO ☒

☐ **OUGHT TO PASS**

☐ **OUGHT TO PASS W/ AMENDMENT**

☒ **INEXPEDIENT TO LEGISLATE**

☐ **INTERIM STUDY** (Available only 2nd year of biennium)

Amendment No. _____

STATEMENT OF INTENT:

THIS BILL WOULD MANDATE DHHS TO PAY FOR BLOOD TESTING FOR PERFLUORINATED CHEMICALS IN A NUMBER OF SITUATIONS, INCLUDING A CATCH-ALL SITUATION WHICH WOULD ALLOW ANYBODY IN THE STATE TO REQUEST THAT THE STATE PAY FOR THEIR TESTING. THIS TEST IS A VERY EXPENSIVE LABORATORY RESEARCH TEST, ~~RATHER~~ NOT A CLINICAL TEST. THE COST TO THE DEPARTMENT WOULD BE PROHIBITIVE. SELF-SELECTED VOLUNTARY TESTING IS NOT A GOOD METHOD TO GATHER VALID EPIDEMIOLOGIC DATA. ALTHOUGH WE HAVE DATA REGARDING THE POTENTIAL RISKS OF SEVERAL DISEASES WITH PFC EXPOSURE, WE DO NOT HAVE DATA AS TO THE DIRECT HEALTH IMPLICATIONS OF SPECIFIC BLOOD LEVELS. PROVIDING DATA TO INDIVIDUALS WHEN THE IMPLICATIONS ARE UNKNOWN CAN LEAD TO UNNECESSARY ANXIETY AND OVERTESTING WHICH MAY BE MORE HARMFUL THAN THE UNDERLYING EXPOSURE. THE ISSUE IS NOT BEING IGNORED AS DES IS ALREADY INVESTIGATING THIS PROBLEM STATEWIDE.

COMMITTEE VOTE: 16-5

RESPECTFULLY SUBMITTED,

- Copy to Committee Bill File
- Use Another Report for Minority Report

Rep. JERRY KNIRK
For the Committee

Rev. 02/01/07 - Yellow

Voting Sheets

HOUSE COMMITTEE ON HEALTH, HUMAN SERVICES AND ELDERLY AFFAIRS

EXECUTIVE SESSION on HB 691-FN

BILL TITLE: relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

DATE: February 26, 2019

LOB ROOM: 205

MOTIONS: INEXPEDIENT TO LEGISLATE

Moved by Rep. Knirk | Seconded by Rep. Marsh | Vote: 16-5

CONSENT CALENDAR: NO

Statement of Intent: Refer to Committee Report

Respectfully submitted,

Susan Ticehurst

Rep Susan Ticehurst, Clerk

HOUSE COMMITTEE ON HEALTH, HUMAN SERVICES AND ELDERLY AFFAIRS

EXECUTIVE SESSION on HB 691-FN

BILL TITLE: relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

DATE: 2-26-19

LOB ROOM: 205

MOTION: (Please check one box)

☐ OTP ☑ ITL ☐ Retain (1st year) ☐ Interim Study (2nd year) ☐ Adoption of Amendment # ________ *(if offered)*

Moved by Rep. Knirk Seconded by Rep. Marsh Vote: 16-5

MOTION: (Please check one box)

☐ OTP ☐ OTP/A ☐ ITL ☐ Retain (1st year) ☐ Interim Study (2nd year) ☐ Adoption of Amendment # ________ *(if offered)*

Moved by Rep. ________ Seconded by Rep. ________ Vote: ________

MOTION: (Please check one box)

☐ OTP ☐ OTP/A ☐ ITL ☐ Retain (1st year) ☐ Interim Study (2nd year) ☐ Adoption of Amendment # ________ *(if offered)*

Moved by Rep. ________ Seconded by Rep. ________ Vote: ________

MOTION: (Please check one box)

☐ OTP ☐ OTP/A ☐ ITL ☐ Retain (1st year) ☐ Interim Study (2nd year) ☐ Adoption of Amendment # ________ *(if offered)*

Moved by Rep. ________ Seconded by Rep. ________ Vote: ________

CONSENT CALENDAR: ____ **YES** ✓ **NO**

Minority Report? ~~____~~ Yes ✓ No If yes, author, Rep: ~~________~~ Motion______

Respectfully submitted: Susan Ticehurst

Rep Susan Ticehurst, Clerk

STATE OF NEW HAMPSHIRE
OFFICE OF THE HOUSE CLERK

2019 SESSION

2/7/2019 12:10:51 PM
Roll Call Committee Registers Report

Health, Human Services and Elderly Affairs

Bill #: HB 691 **Motion:** ITL **AM #:** _____ **Exec Session Date:** 2-29-19

Members	YEAS	Nays	NV
Weber, Lucy M. Chairman	✓		
Campion, Polly Kent Vice Chairman	✓		
MacKay, James R.	✓		
Snow, Kendall A.	✓		
Freitas, Mary C.	✓		
Ticehurst, Susan J. Clerk		✓	
Knirk, Jerry L.	✓		
Salloway, Jeffrey C.	✓		
Cannon, Gerri D.	✓		
Nutter-Upham, Frances E.		✓	
Osborne, Richard G.	✓		
Schapiro, Joe	✓		
Woods, Gary L.	✓		
McMahon, Charles E.		✓	
Nelson, Bill G.	✓		
Guthrie, Joseph A.	✓		
Fothergill, John J.	✓		
Marsh, William M.	✓		
Pearson, Mark A.	✓		
Acton, Dennis F.		✓	
DeClercq, Edward			
Stapleton, Walter A.		✓	
TOTAL VOTE:	16	5	

Sub-Committee Actions

HOUSE COMMITTEE ON HEALTH, HUMAN SERVICES AND ELDERLY AFFAIRS

SUBCOMMITTEE WORK SESSION on HB 691-FN

BILL TITLE: relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

DATE: February 26, 2019

Subcommittee Members: Reps. Weber, Campion, Woods, McMahon, M. Pearson, Stapleton, Nelson, Freitas, Knirk, Salloway and R. Osborne

Comments and Recommendations:

MOTIONS: **INEXPEDIENT TO LEGISLATE**

Moved by Rep. Rep. Knirk | Seconded by Rep. Rep. Woods | Vote: 9-2

Respectfully submitted,

Rep. Lucy Weber
Subcommittee Chairman

HOUSE COMMITTEE ON HEALTH, HUMAN SERVICES AND ELDERLY AFFAIRS

SUBCOMMITTEE WORK SESSION on HB 691-FN

BILL TITLE: relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

DATE: 2/26/19

Subcommittee Members: Reps. Weber, Campion, Woods, McMahon, M. Pearson, ~~DeClercq,~~ Stapleton, Nelson, Knirk, Freitas, Salloway and R. Osborne

Comments and Recommendations:

Bill

MOTIONS: OTP, OTP/A, ITL, Retained (1st Yr), Interim Study (2nd Yr)
(Please circle one)

Moved by Rep. Knirk Seconded by Rep. Woods ~~AM~~ Vote: 9-2

Adoption of ~~Amendment~~ # _____

Moved by Rep. _____ Seconded by Rep. _____ Vote: _____

_____ Amendment Adopted _____ Amendment Failed

MOTIONS: OTP, OTP/A, ITL, Retained (1st Yr), Interim Study (2nd Yr)
(Please circle one)

Moved by Rep. _____ Seconded by Rep. _____ AM Vote: _____

Adoption of Amendment # _____

Moved by Rep. _____ Seconded by Rep. _____ Vote: _____

_____ Amendment Adopted _____ Amendment Failed

Respectfully submitted,

Rep. Mary Freitas
Subcommittee Chairman/Clerk

HOUSE COMMITTEE ON HEALTH, HUMAN SERVICES AND ELDERLY AFFAIRS

SUBCOMMITTEE WORK SESSION on HB 691-FN

BILL TITLE: relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

DATE:

Subcommittee Members: Reps. Weber, Campion, Woods, McMahon, M. Pearson, DeClercq, Stapleton, Nelson, Knirk, Freitas, Salloway and R. Osborne

Comments and Recommendations:

~~It~~ Will schedule another work session

MOTIONS: OTP, OTP/A, ITL, Retained (1st Yr), Interim Study (2nd Yr)
(Please circle one)

Moved by Rep. McMahon Seconded by Rep. Salloway AM Vote: _____

Adoption of Amendment # Motion withdrawn, Amendment to be prepared.

Moved by Rep. _____ Seconded by Rep. _____ Vote: _____

_____ Amendment Adopted _____ Amendment Failed

MOTIONS: OTP, OTP/A, ITL, Retained (1st Yr), Interim Study (2nd Yr)
(Please circle one)

Moved by Rep. _____ Seconded by Rep. _____ AM Vote: _____

Adoption of Amendment # _____

Moved by Rep. _____ Seconded by Rep. _____ Vote: _____

_____ Amendment Adopted _____ Amendment Failed

Respectfully submitted,

Rep. L. Weber
Subcommittee Chairman/Clerk

Hearing Minutes

HOUSE COMMITTEE ON HEALTH, HUMAN SERVICES AND ELDERLY AFFAIRS

PUBLIC HEARING ON HB 691-FN

BILL TITLE: **relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.**

DATE: **February 6, 2019**

LOB ROOM: **205** **Time Public Hearing Called to Order: 10:35 AM**

Time Adjourned: 11:45 AM

Committee Members: **Reps. Weber, Campion, Ticehurst, MacKay, Snow, Freitas, Knirk, Salloway, Cannon, Nutter-Upham, R. Osborne, Schapiro, Woods, McMahon, Nelson, Guthrie, Fothergill, Marsh, M. Pearson, Acton, DeClercq and Stapleton**

Bill Sponsors:

Rep. W. Thomas	**Rep. Murphy**	**Rep. Stack**
Rep. Meuse	**Sen. Chandley**	

TESTIMONY

* Use asterisk if written testimony and/or amendments are submitted.

*** 1, 2, 3 Sponsor/Introduced By: Wendy Thomas –**
Attachment #1: (Packet) Agency for Toxic Substances and Disease Registry and additional documents; Attachment #2: Federal Register; Attachment #3: Vermont Sets A Permanent Drinking Water Standard for PFOA. If we had a blood testing program for exposure it would provide those who are contaminated with a baseline of how much is in their bodies. Would show what our contamination area is. Fiscal note should be reevaluated. Questions lab instruments figure. That fee is for water testing, not blood testing.

*** 4, 5 Dr. Benjamin Chan, State Epidemiologist and Christine Bean, Bureau Chief Public Health Laboratory –**
Department takes no position. Dr. Chan: Purpose of PFAS blood testing is to assess exposure, which is best done through random sampling of the population. Gives a general level of exposure. Purpose of blood testing is not to assess environmental contamination. That is done by the Department of Environmental Services. PFAS blood testing is not recommended for eavery individual. It is not a medical test with an established safe/unsafe level. People who have had their blood tested don't know what it means, not because of lack of education but because the tests don't indicate what medical care is recommended. DHHS is now using federal recommendations. Suggests doing a health study, which is currently being done on the federal level in a pilot study, with additional plans for a national study. There are real and valid concerns in the communities. Concerned that PFAS blood testing will not provide the answers that people are looking for. Dr. Bean: The lab is federally accredited, which includes a mandate that a medical provider order the test. Currently working on a Centers for Disease Control funded bio-monitoring program. Have steered some of the work toward PFOAS. Currently working on a statewide surveillance study of 400 randomly selecting participants for chemical of exposure in both humans and the water they drink. Limited number of laboratories that have the capability to do blood testing. Now have ability to do the testing for the 400 in the study but cannot expand to the general population. Do not have the capacity to test 10,000 or more people who have been exposed.

*** 6 George.B. Roberts, American Chemistry Council –**
Opposes because it would require DHS to provide blood tests for anyone who may have been exposed. Overly broad. Could involve up to 500,000 people. DES will have rules by April on allowable parts per million. Not all PFOAs are toxic.

Respectfully submitted,

Susan Ticehurst

Rep. Susan Ticehurst, Clerk

HOUSE COMMITTEE ON HEALTH, HUMAN SERVICES AND ELDERLY AFFAIRS

PUBLIC HEARING ON HB 691-FN

BILL TITLE: **relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.**

DATE:

ROOM: 205 Time Public Hearing Called to Order: _____

Time Adjourned: _____

(please circle if present)

Committee Members: **Reps. Weber, Campion, Ticehurst, MacKay, Snow, Freitas, Knirk, Salloway, Cannon, Nutter-Upham, R. Osborne, Schapiro, Woods, McMahon, Nelson, Guthrie, Fothergill, Marsh, M. Pearson, Acton, DeClercq and Stapleton**

Bill Sponsors:

Rep. W. Thomas	Rep. Murphy	Rep. Stack
Rep. Meuse	Sen. Chandley	

TESTIMONY

* Use asterisk if written testimony and/or amendments are submitted.

House Committee on Health, Human Services & Elderly Affairs
Public Hearing on HB 691-FN

Bill Title:	relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.		
Date:	2/6/19		
Room:	205	Time Public Hearing Called to Order:	10:35
		Time Adjourned:	11:45

Committee Members Present:

X	Shapiro
X	Cannon
X	Stapleton
X	Nutter-Upham
X	Marsh
X	Salloway
X	Fothergill
X	Freitas
X	Snow
X	MacKay
X	Ticehurst
X	Weber

X	DeClerq
X	Osborne
X	Acton
X	Woods
X	Pearson
X	Knirk
X	Guthrie
X	Nelson
X	McMahon
X	Campion

Testimony

* Use asterisk if written testimony and/or amendments are submitted.

*	Attch #	Name	Testimony:
*	1, 2, 3	Sponsor/Introduced By: Wendy Thomas	Attachment #1: (Packet) Agency for Toxic Substances and Disease Registry and additional documents; Attachment #2: Federal Register; Attachment #3: Vermont Sets A Permanent Drinking Water Standard for PFOA. If we had a blood testing program for exposure it would provide those who are contaminated with a baseline of how much is in their bodies. Would show what our contamination area is. Fiscal

			note should be reevaluated. Questions lab instruments figure. That fee is for water testing, not blood testing.
*	4, 5	Dr. Benjamin Chan, State Epidemiologist and Christine Bean, Bureau Chief Public Health Laboratory	Department takes no position. Dr. Chan: Purpose of PFAS blood testing is to assess exposure, which is best done through random sampling of the population. Gives a general level of exposure. Purpose of blood testing is not to assess environmental contamination. That is done by the Department of Environmental Services. PFAS blood testing is not recommended for eavery individual. It is not a medical test with an established safe/unsafe level. People who have had their blood tested don't know what it means, not because of lack of education but because the tests don't indicate what medical care is recommended. DHHS is now using federal recommendations. Suggests doing a health study, which is currently being done on the federal level in a pilot study, with additional plans for a national study. There are real and valid concerns in the communities. Concerned that PFAS blood testing will not provide the answers that people are looking for. Dr. Bean: The lab is federally accredited, which includes a mandate that a medical provider order the test. Currently working on a Centers for Disease Control funded bio-monitoring program. Have steered some of the work toward PFOAS. Currently working on a statewide surveillance study of 400 randomly selecting participants for chemical of exposure in both humans and the water they drink. Limited number of laboratories that have the capability to do blood testing. Now have ability to do the testing for the 400 in the study but cannot expand to the general population. Do not have the capacity to test 10,000 or more people who have been exposed.

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Respectfully submitted,

Rep. Susan Ticehurst, Clerk

SIGN UP SHEET

To Register Opinion If Not Speaking

Bill # HB 691-FN Date 2/6/2019

Committee HHS & EA

** Please Print All Information **

Name	Address	Phone	Representing	(check one) Pro	Con
Senator Sharon Chantley				✓	
Rep Dick Hinch			Hills Dist. 21	✓	
REP. DAVID MEUSE	PORTSMOUTH			✓	
Rep. Nancy Murphy	Hills 21			✓	
Ben Chan			NHDHHS		
Chris Bean			NH DHHS		
Rep Jess Tanner			Rock 37	✓	
Rep Wendy Chase			Strafford Dist 18	✓	
Rep Ellen Read				✓	
Rep Cassandra Levesque			Strafford 4	✓	
Rep Liz McConnell	Brentwood 11			✓	

Testimony

(1)

Agency for Toxic Substances and Disease Registry

Per- and Polyfluoroalkyl Substances (PFAS) and Your Health

The health effects of PFOS, PFOA, PFHxS, and PFNA have been more widely studied than other per- and polyfluoroalkyl substances (PFAS). Some, but not all, studies in humans with PFAS exposure have shown that certain PFAS may:

- affect growth, learning, and behavior of infants and older children
- lower a woman's chance of getting pregnant
- interfere with the body's natural hormones
- increase cholesterol levels
- affect the immune system
- increase the risk of cancer

Scientists are still learning about the health effects of exposures to mixtures of PFAS.

For the most part, laboratory animals exposed to high doses of one or more of these PFAS have shown changes in liver, thyroid, and pancreatic function, as well as some changes in hormone levels. Because animals and humans process these chemicals differently, more research will help scientists fully understand how PFAS affect human health.

Page last reviewed: January 10, 2018

Hello, it has been a while since we last met and I wish to bring it back to March 23, 2016. The original date you met with our worried community. On that date in this very room several concerned residents who have been unknowingly drinking and breathing toxic man-made chemicals requested blood testing.

That is 931 days. That is 930 days too long to wait for answers.

Since this date you have been assuring us that we are safe because we are "under the numbers" your chosen science has deemed safe. We were told blood by YOU that blood testing won't help. Remember, when this news broke the limit for pfoa was 400ppt. It was changed upon pressure from the science community to 100ppt. **You told a concerned man, Dave was his name, that he did not need to take extra precautions in giving his pregnant wife water at that level. I wonder what you would say to Dave today if he were to ask that very same question?**

And then, a few months after making that statement, the limit was dropped to 70ppt, where it still stands. We are constantly being reassured but the reality is- you have no clue what we are dealing with. So why are we not taking more serious precautions with our health? **I assure you, dear experts, adding 2 more chemicals to the permissible 70ppt limit is NOT THE ANSWER!**

We are a contaminated community. We have been dosed and continue to be dosed with well over 30 unnatural, dangerous chemicals in our everyday lives. **This is fact and cannot be disputed.**

We are still denied access to blood testing. Heck, I can't even order the correct blood test and pay out of pocket like I had planned to do this past Spring. After trying to gain access to the testing YOU used for the "Lucky 200" not so random sampling, I only have access to the old, outdated test that will not measure to the sensitivity and will make it look like the result is non-detect. It seems the lab will only deal with the State and will not take individual tests from exposed community members. HOW IS THIS ETHICAL?!?! Isn't this sketchy that they won't allow us blood tests, even if we pay for it ourselves? How long is the half-life for these chemicals again? Is that the motivator to refuse our access?

We need protection Clark, Lisa, Dr. Chan, etc. It is YOUR job to protect our children. As Bill Belichick famously states, "DO - YOUR - JOB!"

Conclusion

Strict liability and medical monitoring are important tools for improving public health, encouraging companies to employ safer practices, and ensuring innocent victims of toxic pollution are made whole. While these recommendations will not resolve every problem, they will create a more fair, just, and modern legal environmental. Additionally, these proposals do not require any additional government programs or regulatory burdens on companies. Instead, they make clear that any risk involved in the use or manufacture of toxic chemicals will be borne by those who profit from them, and who are in the best position to prevent harm in the first place.

Sample Description: MTBE_8279 Grab Potable Water
Saint Gobain
10 Wildcat Falls Rd (Post-treatment)

Project Name: 199712055 - Saint Gobain

Submittal Date/Time: 12/22/2017 11:40
Collection Date/Time: 12/20/2017 09:50

NH Dept of Environmental Svcs
ELLE Sample #: PW 9384169
ELLE Group #: 1890548
Matrix: Potable Water

CAT No.	Analysis Name	CAS Number	Result	Method Detection Limit*	Limit of Quantitation	Dilution Factor
Misc. Organics	**EPA 537 Version 1.1 Modified**		ng/l	ng/l	ng/l	
14473	10:2-fluorotelomersulfonate	120226-60-0	N.D.	3	8	1
14473	4:2 fluorotelomersulfonate	757124-72-4	N.D.	0.9	3	1
14473	6:2 fluorotelomersulfonate	27619-97-2	N.D.	3	8	1
14473	8:2 fluorotelomersulfonate	39108-34-4	N.D.	2	5	1
14473	NEtFOSAA	2991-50-6	N.D.	0.9	3	1
	NEtFOSAA is the acronym for N-ethyl perfluorooctanesulfonamidoacetic Acid.					
14473	NMeFOSAA	2355-31-9	N.D.	0.9	3	1
	NMeFOSAA is the acronym for N-methyl perfluorooctanesulfonamidoacetic Acid.					
14473	Perfluorobutanesulfonate C4	375-73-5	4 PFBS	0.3	0.9	1
14473	Perfluorobutanoic acid C4	375-22-4	5 J PFBA	2	5	1
14473	Perfluorodecanesulfonate	335-77-3	N.D.	0.5	2	1
14473	Perfluorodecanoic acid	335-76-2	N.D.	0.9	2	1
14473	Perfluorododecanesulfonate	79780-39-5	N.D.	0.3	0.9	1
14473	Perfluorododecanoic acid	307-55-1	N.D.	0.3	0.9	1
14473	Perfluoroheptanesulfonate	375-92-8	N.D.	0.4	2	1
14473	Perfluoroheptanoic acid C7	375-85-9	8 PFHpA	0.3	0.9	1
14473	Perfluorohexadecanoic acid	67905-19-5	N.D.	0.3	0.9	1
14473	Perfluorohexanesulfonate C6	355-46-4	4 PFHxS	0.4	2	1
14473	Perfluorohexanoic acid C6	307-24-4	13 PFHxA	0.4	2	1
14473	Perfluorononanesulfonate	474511-07-4	N.D.	0.5	2	1
14473	Perfluorononanoic acid	375-95-1	0.4 J	0.4	2	1
14473	Perfluorooctadecanoic acid C8	16517-11-6	N.D.	0.3	0.9	1
14473	Perfluoro-octanesulfonate C8	1763-23-1	4 PFOS	0.4	2	1
14473	Perfluorooctanoic acid PFOA C8	335-67-1	41 PFOA	0.3	0.9	1
14473	Perfluoropentanesulfonate C5	2706-91-4	0.7 J	0.4	2	1
14473	Perfluoropentanoic acid C5	2706-90-3	9 PFPeA	2	5	1
14473	Perfluorotetradecanoic acid	376-06-7	N.D.	0.3	0.9	1
14473	Perfluorotridecanoic acid	72629-94-8	N.D.	0.3	0.9	1
14473	Perfluoroundecanoic acid	2058-94-8	N.D.	0.4	2	1

The stated QC limits are advisory only until sufficient data points can be obtained to calculate statistical limits.

The recovery for labeled compound used as extraction standard 13C2-PFTeDA is outside of QC acceptance limits as noted on the QC Summary. The data quality is not impacted by this result.

Sample Comments

All QC is compliant unless otherwise noted. Please refer to the Quality Control Summary for overall QC performance data and associated samples.

*=This limit was used in the evaluation of the final result

Why is DHHS doing this blood testing?

The health effects of perfluorochemicals (PFCs) are not clearly understood. In collaboration with the NH Department of Environmental Services drinking water testing program, the NH Department of Health and Human Services is offering PFC blood testing to help determine the full extent of the communities' exposure to these contaminants.

How have I been exposed to PFCs?

PFCs are synthetic chemicals that have been widely used to make a range of household and commercial products including stain resistant furniture, carpeting, and clothing; water-repellant fabrics; and grease-resistant food packaging. Because of this widespread use, most people have been exposed to these chemicals in their everyday lives, and when tested, almost all people have detectable levels of PFCs in their blood. If a person's drinking water has these chemicals, their blood levels are likely higher than the average U.S. resident.

How long do PFCs stay in my body?

Some PFCs remain in a person's blood for a very short amount of time, whereas others can remain for years. Once exposures are removed PFCs, such as perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS), decline naturally in a person's blood by about half every 4-5 years. There is no known medical procedure to remove PFCs from a person's body more quickly than occurs naturally over time.

Are there health effects known to be associated with PFCs?

Some human health studies have found associations between PFC exposure and health effects and others have not, therefore conclusions cannot be made with certainty about any health effects caused by PFCs at this time. Because of this uncertainty, further research is necessary to know how PFCs affect a person's health. A variety of potential health effects in humans are currently being studied, including how PFCs might affect growth and development, liver function, hormone levels, cholesterol levels, and occurrence of some types of cancers.

Summary of the New Hampshire Department of Health and Human Services' Perfluorochemical (PFC) Blood Testing Program, 2016-2017

Beginning in April 2015, the New Hampshire Department of Health and Human Services (DHHS) has conducted blood testing for people in communities where perfluorochemicals (PFCs) have been found in drinking water above lifetime health advisory levels. The DHHS PFC blood testing program measures a person's PFC blood level, or the amount of PFCs in the blood. The DHHS blood testing program was initially launched to test people who may have been exposed to PFCs on the Pease Tradeport. Between April and October 2015, 1,578 members of the Pease Tradeport community had their blood tested for PFC exposure.

In 2016, DHHS expanded the blood testing program to include residents of communities in southern New Hampshire, initially around the Saint-Gobain Performance Plastics facility in Merrimack, where PFCs have been found to contaminate private drinking water wells. DHHS has also conducted a Community Exposure Assessment among the Merrimack Village District (MVD) public water system, a random sample of 217 MVD customers to measure approximate levels of exposure. Below is a summary of 694 blood test results conducted in 2016-2017, including 258 individuals from the Pease Tradeport community, 219 individuals from southern New Hampshire communities on private drinking water wells, and 217 individuals who participated in the MVD Community Exposure Assessment. The results are compared to each other, 2015 blood test results from Pease, other exposed communities and the general U.S. population.

The comparisons below include average and 95th percentile PFC levels found in the communities. The average is the middle level found in the community. The 95th percentile reflects the upper-end of the blood levels that most individuals tested below (95% of individuals in the community tested below this level). The DHHS PFC blood testing program is ongoing and some results may change as additional results become available.

Summary Results for Individuals Who Participated in the MVD Community Exposure Assessment

- People participating in the MVD Community Exposure Assessment had higher levels of PFOA exposure compared with the general U.S. population.
- PFOA levels were lower than levels seen in other exposed communities around the U.S, including Bennington, VT and Hoosick Falls, NY, where residents living near a Saint-Gobain facility received blood testing.
- PFOA blood levels in MVD participants are similar to blood levels in other southern NH residents whose private drinking water wells tested between 40-60 ppt of PFOA.

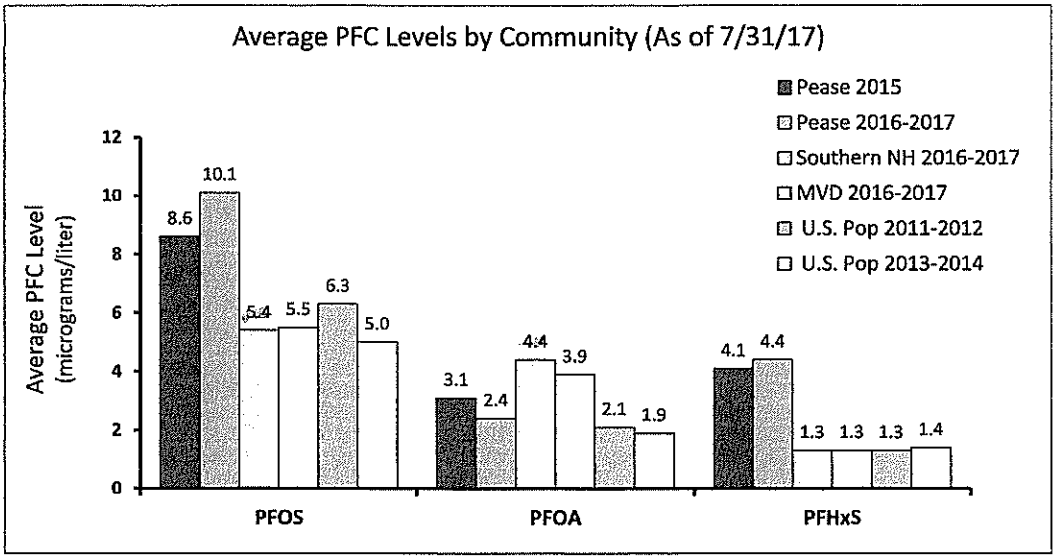
Summary Results for Individuals in Southern NH on Private Wells

- Participants from southern New Hampshire had higher blood levels of perfluorooctanoic acid (PFOA) compared with the general U.S. population.
- Individuals with higher concentrations of PFOA in their private well water have higher blood PFOA levels.
- PFOA levels in southern NH residents were lower than levels seen in other exposed communities around the U.S, including Bennington, VT and Hoosick Falls, NY, where residents living near a Saint-Gobain facility received blood testing.

Summary Result for Individuals Exposed on the Pease Tradeport

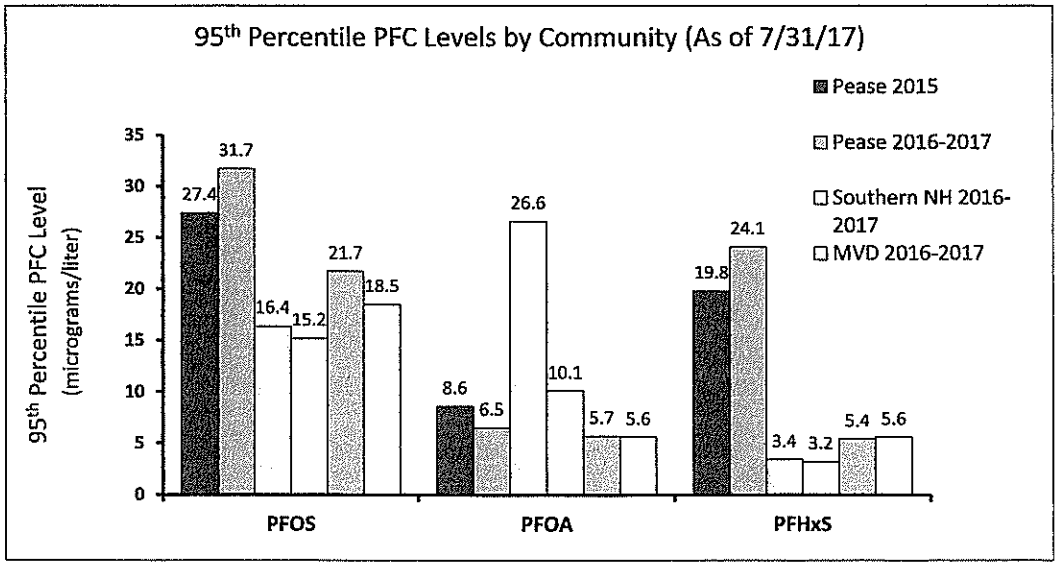
- Individuals from the Pease Tradeport had higher blood levels of PFOA, perfluorooctane sulfonic acid (PFOS), and perfluorohexane sulfonic acid (PFHxS) compared with the general U.S. population. PFOS, PFOA and PF-HxS were detected at elevated levels in a public water supply well tested at Pease in 2014.
- These 2016-2017 results are consistent with the results from PFC blood testing of the Pease community in 2015. PFOS, PFOA and PFHxS were detected at higher levels in one of the drinking water wells in 2014.

Comparing Average Levels for Pease 2015*, Pease 2016-2017, Southern NH 2016-2017, MVD 2016-2017, and the General U.S. Population



*In 2015, the New Hampshire Department of Health and Human Services tested 1,578 individuals exposed to PFC contaminated drinking water at Pease. A full report of those findings can be found at: www.dhhs.nh.gov/dphs/investigation-pease.htm.

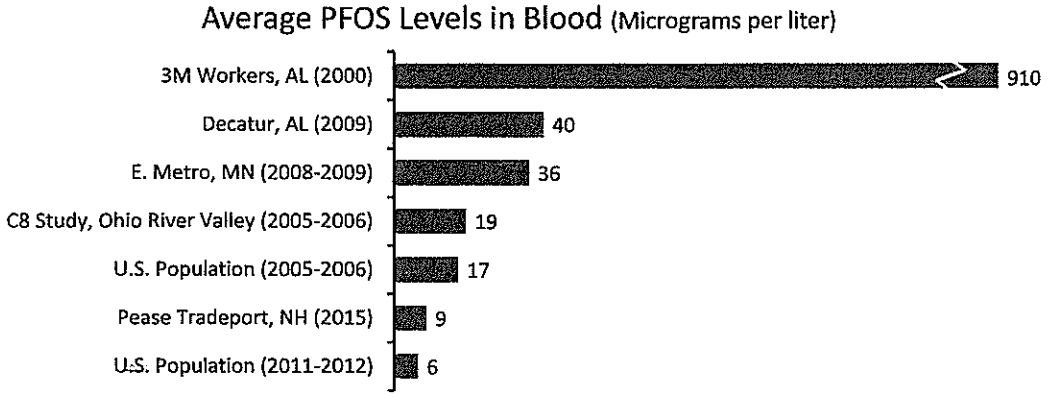
Comparing 95th Percentile Levels for Pease 2015*, Pease 2016-2017, Southern NH 2016-2017, MVD 2016-2017, and the General U.S. Population

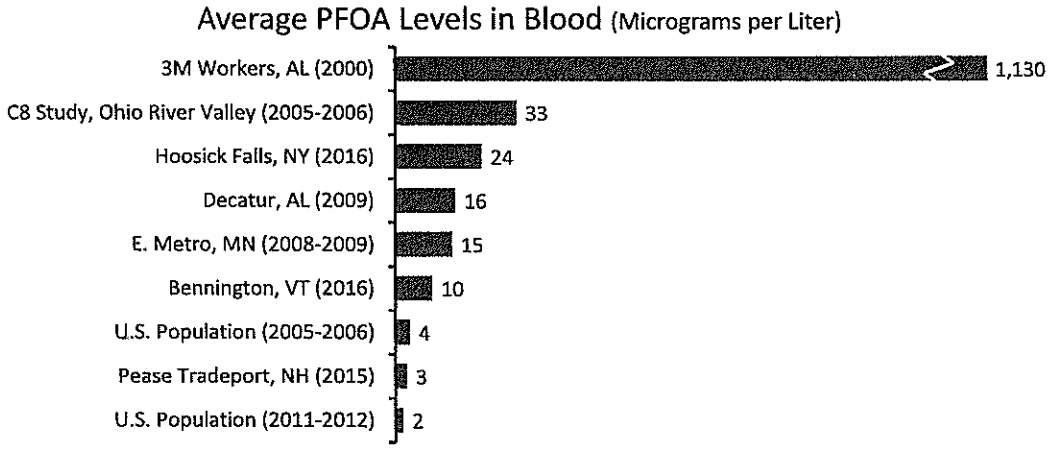


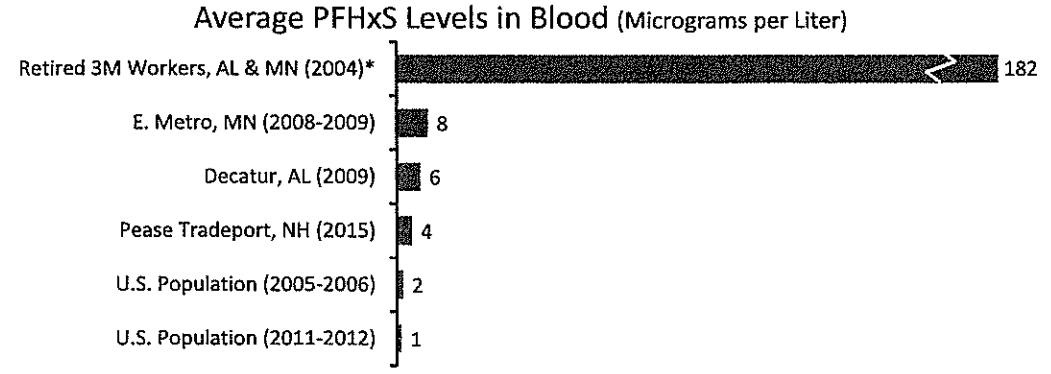
*In 2015, the New Hampshire Department of Health and Human Services tested 1,578 individuals exposed to PFC contaminated drinking water at Pease. A full report of those findings can be found at: www.dhhs.nh.gov/dphs/investigation-pease.htm.

PFC Levels in Other Populations

Below are graphs showing PFOS, PFOA, and PFHxS levels in other populations around the country compared with the New Hampshire communities tested. Comparisons include communities with known exposures to PFCs and the general U.S. population tested as part of a national general health study (the National Health and Nutrition Examination Survey).







Additional Resources

For more information about PFCs and the blood testing program, visit www.dhhs.nh.gov/dphs/pfcs/.

If you or someone you know is experiencing an addiction-related crisis, call 2-1-1 now.

What's New...

DHHS Releases 10-Year Mental Health Plan

DHHS Response to the Office of Child Advocate Annual Report

Report to Governor and Legislature RE Federal Approval of Granite Advantage Health Care Program Waiver

Granite Advantage Health Care Program

State Opioid Response Grant

Medicaid Enrollment Data

DHHS News And Events

NH in Early Stages of Hepatitis A Outbreak
February 5, 2019 - The DHHS Division of Public Health Services is announcing a significant increase in the number of individuals in New Hampshire diagnosed with hepatitis A. Over the past three months, 13 new individuals have been diagnosed with acute hepatitis A infections, including 7 in January, compared to an average of 6 – 7 people annually (range of 1-10 cases annually) over the past five years.

NH DHHS Reschedules January 29th Portsmouth Public Forum for Granite Advantage Health Care Program
January 28, 2019 - DHHS has announced that a public forum in Portsmouth for the Granite Advantage Health Care Program, originally scheduled for Tuesday, January 29, has been rescheduled due to expected inclement weather.

Governor's Press Release: Governor Sununu and NH DHHS Releases 10-Year Mental Health Plan
January 23, 2019 - DHHS is releasing the *10-Year Mental Health Plan*. The Plan provides innovative models to meet the evolving needs of individuals and families and the increasing complexity of New Hampshire's mental health system. It is the culmination of a robust stakeholder engagement process that included input from hundreds of interested parties through focus groups, workgroups, public sessions, and written comments.

DHHS Announces Early Issue of February Food Stamp Benefits
January 15, 2019 - DHHS has announced that food stamp beneficiaries will have access to their February benefits earlier than usual. February benefits will be available to eligible food stamp households beginning January 20, instead of the regular availability date of February 5th.

Assistive Technology Firm ATECH to Wind Down Operations
January 3, 2019 - ATECH Services, an assistive technology company operated by Crotched Mountain Foundation, has informed the NH Department of Health and Human Services (DHHS) that it will discontinue operations by February 28, 2019.

Public Forums Announced to Introduce the Doorway-NH
December 28, 2018 - Today, Governor Chris Sununu and the New Hampshire Department of Health and Human Services (DHHS) released the dates and locations for a series of public forums to introduce "The Doorway-NH," the hub and spoke model that will transform the system serving individuals with a substance use disorder (SUD). Each public forum will be hosted by the Doorway in each of nine regions across the State.

Adobe Acrobat Reader format. You can download a free reader from Adobe (http://get.adobe.com/reader/) .

Granite Advantage Health Care Program
SUD IMD Waiver
Nursing Facility Medicaid Rates and Payments
PAP Section 1115 Demonstration Waiver
Granite Advantage Demonstration Waiver
Medicaid Care Management
Procurement
DSRIP Waiver Program
HB 517 Prior Authorization Implementation
SUD Site Reviews
Community Mental Health Agreement
Operating Statistics Dashboards
Perfluorochemicals (PFCs)
Rules For Public Comment

Apply for Food Stamps?
Apply for Medicaid?
Apply for WIC?
Find my local district office?
Find out about child support?
Foster or adopt a child?
Find a local ServiceLink?
Get health insurance?

Adult Abuse
Child Abuse
Domestic Violence
Foodborne Illness
Homelessness
Infectious Disease
Suicide
Welfare and EBT Fraud

Blood Serum Testing
Vista Analytics Laboratory
1104 Windfield Way
El Dorado Hills, CA. 95762
(916) 673-1520
Jade White
www.vista-analytics.com

Please find attached our serum collection and shipping information as well as a Test Order Form for your perusal.

You will need to identify a party to draw your blood and then centrifuge it to provide only the serum fraction back to us. We unfortunately don't have any relationships with phlebotomists in your area, so who you go to is up to you.

Do you know what PFCs you would like quantified? PFOA/PFOS only? A more comprehensive list? The Serum Test Order form has a list of 9 analytes (PFHxS, PFOA, PFOS, PFNA, PFDA, PFOSA, N-MeFOSAA, PFUdA, N-EtFOSAA) that is fairly similar to what CDC has gathered data on, but we can quantify others. The following website also has a lot of useful information regarding PFAS in blood, so if you are unsure this is a good place to start: https://www.dhhs.nh.gov/dphs/pfcs/blood-testing.htm

We will ship out an insulated shipping container with bio-bags, serum sample tubes, Techni Ice and a return FedEx shipping label. This can be sent directly to you or wherever you designate.

The serum sampling kit is $150 for an individual, and the cost for analysis is below:
1. PFOA/PFOS only - $550 per sample Total cost: $700
2. 9 PFAS - $650 per sample (this test includes PFOA & PFOS listed above) Total cost: $800
    a. PFHxS, PFOA, PFOS, PFNA, PFDA, PFOSA, N-MeFOSAA, PFUdA, N-EtFOSAA
3. EPA list of 24 - ??? Jade White @ Vista will need to get pricing research.

Our turnaround time, from sample receipt, is 45 calendar days.

Blood Draw Facility
VeAr Mobile Phlebotomy
2214 Loma Vista Dr.
Sacramento CA. 95825
(408) 796-9768
Brem

Cost for Blood draw, serum process & boxing samples to send to Vista = $60

State apx #600 per DHHS

Press Release

NH In Early Stages Of Hepatitis A Outbreak

Issued by Bureau of Infectious Disease Control

Publish Date:
February 5, 2019

Contact:
Public Information Office
(603) 271-9389

Concord, NH – The New Hampshire Department of Health and Human Services (DHHS) (http://twitter.com/#!/NHDHHSPIO) Division of Public Health Services (DPHS) is announcing a significant increase in the number of individuals in New Hampshire diagnosed with hepatitis A. Over the past three months, 13 new individuals have been diagnosed with acute hepatitis A infections, including 7 in January, compared to an average of 6 – 7 people annually (range of 1-10 cases annually) over the past five years. This increase is concerning for the beginnings of an outbreak. These new diagnoses have occurred in residents residing across the southern part of our State in the counties of Hillsborough (5), Rockingham (3), Strafford (3), Cheshire (1), and Merrimack (1). Individuals who are at higher risk for hepatitis A are recommended to seek out the vaccine to protect against infection, and anybody wishing to protect themselves from hepatitis A is encouraged to talk with their healthcare provider about obtaining the vaccine, which is very safe and effective.

> "There are large outbreaks of hepatitis A occurring in multiple other states across the country," said Dr. Benjamin Chan, NH State Epidemiologist. "While these outbreaks have often started in individuals experiencing homelessness and those with a substance use disorder, once it is in our communities it can spread very easily even to others without specific risk factors. Thankfully, hepatitis A is a vaccine-preventable disease. We encourage anybody who wishes to protect themselves from hepatitis A to talk with their healthcare provider about obtaining the very effective hepatitis A vaccine."

The hepatitis A virus (HAV) is contagious and is transmitted when a person ingests the virus from objects, food or drinks contaminated by small, undetected amounts of stool from an infected person. The virus can survive for months on surfaces. People at risk of contracting the virus are:

- persons experiencing homelessness
- persons using injection or non-injection recreational drugs, including marijuana
- people experiencing homelessness or with unstable housing (e.g. "couch surfing")
- gay and bisexual men
- people with ongoing, close contact with individuals who use injection and non-injection drugs, or with individuals experiencing homelessness
- close contacts of individuals diagnosed with hepatitis A
- travelers to countries with high rates of the virus

Hepatitis A causes inflammation of the liver; severe infections can result in liver failure and even death. Symptoms include fever, tiredness, loss of appetite, nausea, vomiting, abdominal pain, dark urine, clay-colored bowel movements, joint pain, and jaundice (yellowing of the skin and eyes). These symptoms can last weeks to months and there is no specific treatment for hepatitis A. Hepatitis A is preventable and the vaccination is safe and effective. The vaccine is recommended for:

- all children starting at one year of age and older
- people who are at increased risk of hepatitis A infection (as noted above)
- those with chronic liver diseases (such as hepatitis B and hepatitis C infections)
- anybody wishing to obtain immunity

For more information on hepatitis A, please visit www.dhhs.nh.gov/dphs/cdcs/hepatitisa/.

SICK

SICK **POLLUTERS**

SICK

SICK

We didn't ask for this contamination. This is a complex and overwhelming issue that our community is facing. At this point, companies right here in town continue to pollute with new, shorter chain chemicals said to be as dangerous as PFOA (we have GenX in Merrimack groundwater). We have some control over how we manage the exposure we are faced with daily. **What can we do to protect our loved ones from PFAS exposure?**

We take charge of this issue and get the PFAS out,

What can do?

Some ways to take protective measures for yourself and your family are:

⇒ You are already doing the first thing– **talk about this contamination issue.** Talk to your friends. Talk to your neighbors. Talk to the people in the check out line at the store. Many people still don't know the facts about our water contamination. It can be scary to learn more about our contamination, but it IS possible to work together to overcome this problem. Education is key. The more people know, the better able they are to prevent exposure for themselves and family members.

⇒ **Use filtered or bottled water for cooking and drinking**

This is believed to be one of the best ways to lessen exposure. (*NHDES tests Monadnock Water for PFAS and provides this water to households on bottled water precaution. Note that the local Market Basket brand name water is said to be Monadnock and is 50¢ per gallon jug.)

Filters can be expensive, which is why we believe the polluters must pay to remove it from our water. We didn't put it there. Why should we have to pay to take it out? Talk to us about filters if you are interested in learning more about installing a filter to remove PFAS from your water.

⇒ **Be careful with gardening.** Gardening is believed to be a source of exposure. Plants and vegetables grown using contaminated water and soil have pfas in them.

⇒ Along the same lines, hunting and fishing are of concern as well. **Be careful with fishing and hunting.** Fish and animals (birds, deer, moose, etc.) bio-accumulate PFAS as well. Eating animals with PFAS contamination is a manner of exposure that many don't think about. Recently, Wisconsin and Michigan have instituted health advisories warning citizens to not eat or eat limited fish and animals from PFAS contaminated areas. They have a strong education initiative to protect its residents from this type of exposure.

⇒ **Make phone calls to our Reps and other legislators to promote legislation to protect our public health instead of business profit.**

⇒ **Use PFAS free cookware.** PFAS is also in Teflon pans, you can prevent exposure from your pans by using pots

PFOA IN DRINKING WATER 2016

Photo by Howard Weiss Tisman/VPR

Following news in early 2016 of PFOA-contaminated municipal water wells in Hoosick Falls, New York, and concerns about the former Chemfab property in North Bennington, the Vermont Agency of Natural Resources/Department of Environmental Conservation sampled five private drinking water wells and the No. Bennington municipal water supply for perfluorinated compounds and volatile organic compounds. The five private wells tested showed the presence of perfluorooctanic acid (PFOA) at concentrations ranging from 40 to 2,880 parts per trillion. These levels were above the Vermont Department of Health's drinking water health advisory level of 20 parts per trillion. The Department of Environmental Conservation continued to test residential drinking water wells in North Bennington and Bennington.

In February 2016, the Health Department , and in April began offering PFOA blood testing for affected residents. Results of those tests were announced in July.

On January 26, 2017, the Health Department presented a summary of the results its PFOA blood testing and exposure assessment. The study confirmed that drinking water from contaminated wells was the primary source of exposure to PFOA.

The State of Vermont's investigation and response continues, and included environmental testing in other areas of the state. For more information about the testing, public meetings, and related

non-health aspects of the state's actions:

For more information about agricultural products, read the Agency of Agriculture, Food & Markets

Public Information Line – Dial 2-1-1
This is the call line for residents who have questions about PFOA contamination, or to request a water test.

For questions about the health effects of PFOA:
Call the Vermont Department of Health toll-free at 800-439-8550.

Exposure to Perfluorooctanoic Acid (PFOA) in Bennington and North Bennington, Vermont:

Results of Blood Testing and Exposure Assessment

September 2017

VERMONT
DEPARTMENT OF HEALTH

Table of Contents

List of Figures and Tables

Executive Summary

In 2016, Perfluorooctanoic Acid (PFOA) was found in private drinking water wells in Bennington and North Bennington, Vermont near the former Chemfab property. The Vermont Department of Health did a study looking at blood testing results of people in the Bennington/North Bennington community and how they were exposed to PFOA. The study focused on the following three goals:

1. to better understand how people in the Bennington/North Bennington community were exposed to PFOA,
2. to make sure no additional actions were needed to stop continued exposure to PFOA, and
3. to provide community members with their PFOA blood level and how it compares to background levels in the U.S. population.

Conclusion and Recommendations

The study shows that concentrations of PFOA in blood were linked to concentrations of PFOA in drinking water, which indicates drinking water from contaminated wells was the main way people were exposed to PFOA.

The Health Department recommends people in the Bennington/North Bennington community:

- NOT use water with PFOA concentrations above 20 parts per trillion for drinking, preparing food, cooking, brushing teeth, watering gardens or any other way of taking in water
- Contact their health care provider if they are worried about their health related to their PFOA exposure

The Health Department will update health care providers in the area if there is any new information about PFOA and health.

Background Information

Perfluorooctanoic Acid (PFOA) in Vermont

In early 2016, PFOA-contaminated municipal water wells were discovered in Hoosick Falls, New York. Following this discovery, residents of North Bennington, Vermont raised concerns about the former Chemfab property, which had applied non-stick coatings to fiberglass fabrics from 1970 to 2002. In 2016, the Vermont Department of Environmental Conservation began testing private drinking water wells near the former Chemfab facility for PFOA. The concentrations of PFOA ranged from non-detectable levels to nearly 3,000 parts per trillion. This discovery prompted an investigation by the Health Department, with support from the Southwestern Vermont Medical Center, beginning in April 2016.

What is PFOA?

PFOA is a manufactured chemical that is often used to make household and commercial products that resist heat and chemical reactions, and repel oil, stains, grease and water. PFOA does not break down easily and therefore can stay in the environment and in the body for a long time.

Why is PFOA contamination a health concern?

Prior studies, such as those conducted by the C8 Science Panel in the Mid-Ohio Valley, have shown an association between PFOA in blood and the following adverse health outcomes:

- High cholesterol
- Ulcerative colitis
- Thyroid disease
- Kidney cancer
- Testicular cancer
- High blood pressure during pregnancy

The associations found in these studies are not proof of a cause-and-effect relationship between exposure to PFOA and the above adverse health outcomes. More research is needed before scientists will be able to determine whether there is a definitive cause-and-effect relationship between PFOA and any adverse health outcomes—such as the cause-and-effect relationship between smoking and lung cancer. However, the Health Department does not require such definitive causal relationships to be established in order to take action to protect public health.

Description of Vermont's PFOA Blood Testing and Exposure Assessment Study

Exposure Assessment Description

Each participant in the exposure assessment study was asked to provide a blood sample and complete a questionnaire. The questionnaire was adapted from the questionnaire distributed by New York State to the residents of Hoosick Falls, and focused on sources of PFOA exposure and associated health outcomes that had been identified in previous studies, such as those conducted by the C8 Panel. The purpose of collecting the questionnaire data was to better understand the relationship between consumption of PFOA-contaminated drinking water, the level of PFOA in an individual's blood, and potential adverse health outcomes. Additionally, the Health Department wanted to verify that the consumption of contaminated drinking water was the primary source of exposure to PFOA, and that there was not another, unaccounted for source of exposure in the Bennington area.

Participants were asked a series of questions regarding potential sources of exposure to water contaminated with PFOA, including number of eight-ounce glasses consumed daily of: unfiltered water, filtered water, and bottled water. Participants were also asked to identify other potential sources of exposure to PFOA, such as the consumption of various foods (milk, meat, or eggs) from animals raised in the sampling area, fish caught within the sampling area, or fruits and vegetables grown in the sampling area. Lastly, participants were asked to identify whether they have ever worked or lived at the former Chemfab facility, which was converted to residential, multi-unit housing in after the Chemfab/Saint-Gobain plant closed in 2002 (yes or no). Participants were asked to self-report if they had ever been diagnosed with high cholesterol, chronic kidney disease, increased uric acid levels, altered liver enzymes, ulcerative colitis, pregnancy-induced hypertension, and kidney or testicular cancer.

Who was eligible to have their blood tested?

Individuals were eligible for blood testing if:

1. the Vermont Department of Environmental Conservation (DEC) tested their well water for PFOA, or
2. they lived in a home in the past 8 years that was tested by DEC, or
3. they live or lived, work or worked at the Chemfab/Saint Gobain site.

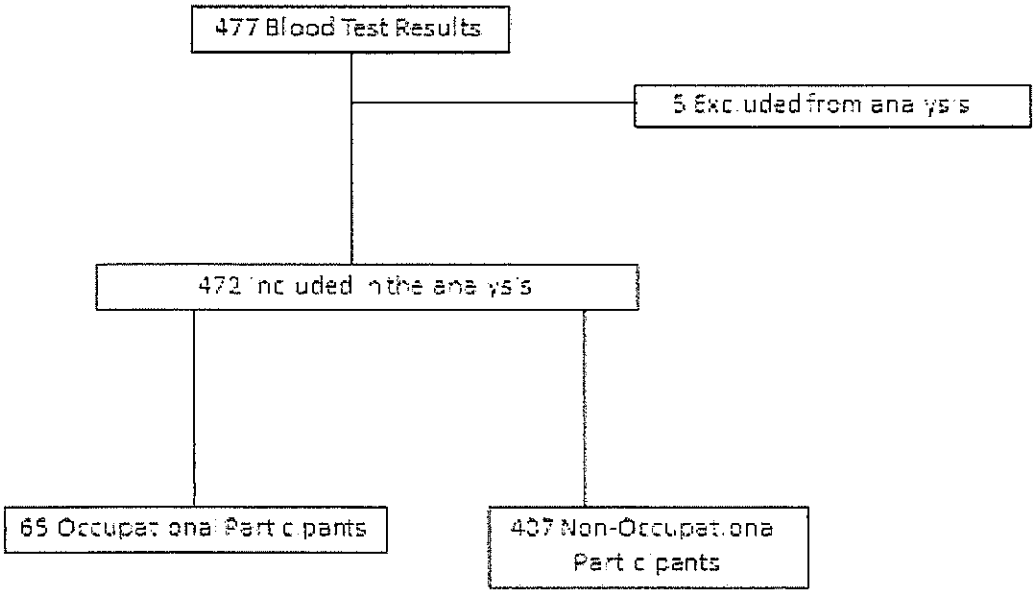
There were 477 blood samples collected as part of the Health Department's blood testing and exposure assessment.

Study Participants

The results of 472 individuals were included in the following analysis. The blood samples of five individuals were not included for various reasons, e.g. not completing the questionnaire. The final group of participants included 65 who were occupationally exposed to PFOA and 407 who were non-occupationally exposed.

Figure 1 illustrates the breakdown of study participants.

Figure 1. Breakdown of Health Department blood testing and exposure assessment participants



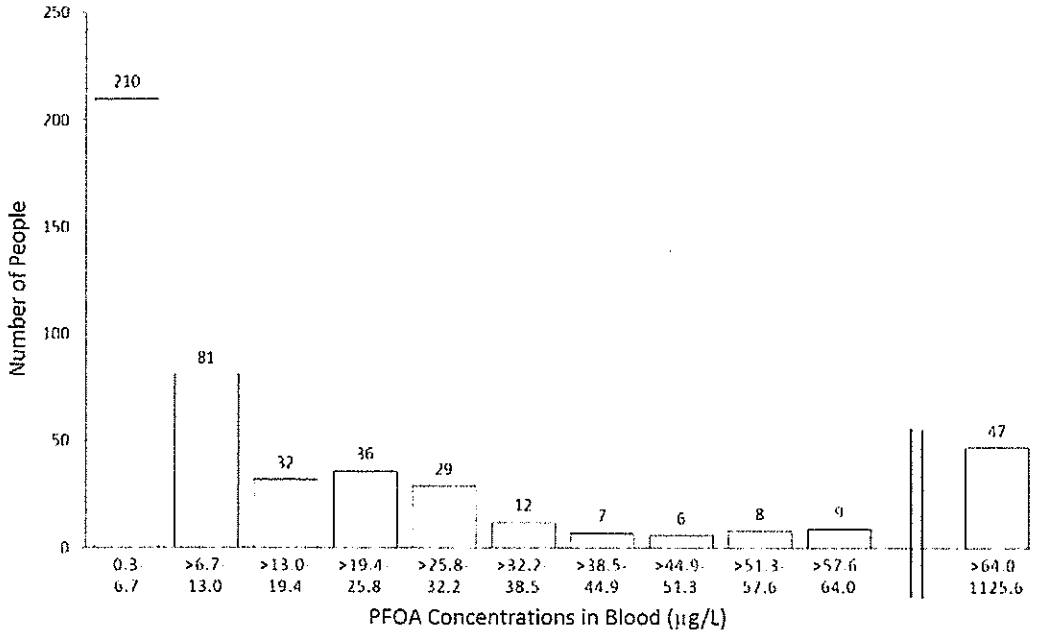
PFOA Concentrations in Blood and Well Water

PFOA Concentrations in Blood

The Bennington/North Bennington exposure assessment analysis included 472 PFOA blood results. The results ranged from 0.3 to 1125.6 µg/L. The geometric mean (a type of average) of these results was 10.1 µg/L compared to a geometric mean of 2.1 µg/L for the entire U.S. population. The 95th percentile was 157.8 µg/L compared to 5.7 µg/L for the entire U.S. population.

Figure 2 illustrates the distribution of these blood test results.

Figure 2. Distribution of PFOA blood results



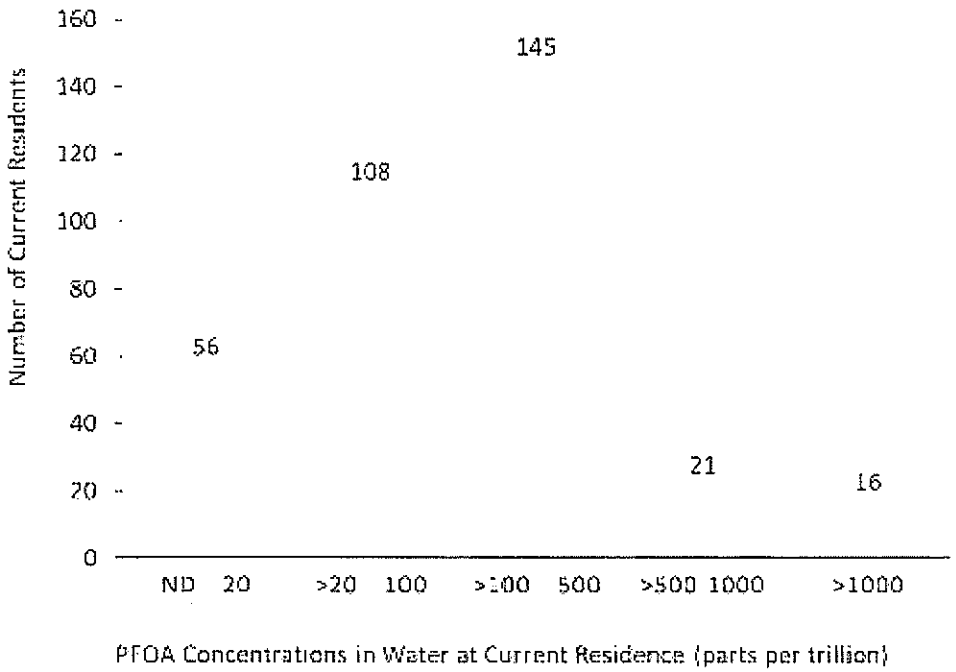
Note: The double black line (||) signifies a change in the PFOA concentration interval width in order to present all of the results on one chart—the range of test results represented in the bar on the far right is greater than the others presented. There is no clinical significance to this distinction.

PFOA Concentrations in Well Water

Drinking water samples were taken from various locations in the Bennington/North Bennington area. There were 345 water samples matched to the blood samples of current residents. When multiple water samples had been taken for a particular household, the maximum concentration was used in this analysis. Of the 345 samples, the geometric mean of PFOA concentrations in well water was 81.4 µg/L, and 291 drinking water results had levels of PFOA that were higher than the Health Department's drinking water health advisory level of 20 parts per trillion.

Figure 3 illustrates the distribution of the water results for the 345 current residents.

Figure 3. Distribution of water results



Note: A result value of ND means that PFOA was not detected.

Association Between PFOA Concentrations in Blood With Measures of Exposure to PFOA

PFOA concentrations in blood were compared to different measures of exposure to PFOA to assess which factors may have influenced the concentration of PFOA in the bodies of study participants. The strength of the association between PFOA in blood and the measures of exposure was assessed using Spearman's rank correlation. The statistical significance of the correlation is reported as the p-value. A description of all the statistical methods used in this analysis can be found in .

Results of blood testing showed that PFOA levels in blood were strongly correlated with PFOA levels in well water (Spearman's rank correlation coefficient = 0.62). The higher the concentration of PFOA in a person's drinking water, the higher the level of PFOA in their blood. Adding further support to this finding, the association with PFOA in blood remained strong (Spearman's rank correlation coefficient = 0.65) when the amount of water an individual drank and how long they drank it for was considered. In other words, the more contaminated water an individual drank and the longer they drank it for, the higher the level of PFOA in their blood.

PFOA levels in blood were weakly correlated with the number of years at current residence (Spearman's rank correlation coefficient = 0.12). Study participants who lived at their current residence longer generally had slighly higher levels of PFOA in their blood. However, it should

be noted that current PFOA concentrations in water may not be equal to historic PFOA concentrations for all years of residence.

PFOA levels in blood were weakly and negatively correlated with the number of glasses of filtered water consumed per day (Spearman's rank correlation coefficient = -0.13). This means that study participants who consumed more filtered water generally had slightly lower PFOA concentrations in blood. This is to be expected, as appropriate filters remove PFOA from water.

PFOA levels in blood were not correlated with the number of glasses of unfiltered water consumed per day at the residence. This means there was no association between consumption of unfiltered water at home and an individual's blood PFOA level. This may be due to individuals being unsure of how much water they consume in a given day. PFOA levels in blood were also not correlated with the number of glasses of bottled water consumed per day. This is to be expected, as presumably, bottled water does not contain PFOA.

Comparison of PFOA Concentration in Blood Across Subgroups

For the purposes of these comparisons, a subgroup was made up of participants who had different demographic or exposure characteristics (e.g. men versus women, workers versus residents, etc.). The comparisons were made using non-parametric statistical methods due to the distribution of the PFOA blood results. The specific tests used were the Wilcoxon rank-sum test (to compare two subgroups) and the Kruskal-Wallis test (to compare three or more subgroups). A more detailed description of these statistical methods can be found in

All the tables in this section of the report include a column entitled "n," which indicates the number of participants in each subgroup. The "geometric mean" column indicates the geometric mean (a type of average) of the blood PFOA concentration for each subgroup. The "p-value" column provides an indication of whether the difference in blood PFOA concentration between the subgroups is statistically significant. For the purposes of this report, a p-value of ≤0.05 was considered to indicate that the PFOA concentrations in blood in one group were significantly different from the PFOA concentrations of the other group.

Comparison of PFOA Concentrations in Blood by Demographic Characteristics

Study results showed higher PFOA blood levels in men compared to women. These data are consistent with other studies, including PFOA biomonitoring in Minnesota and New York. The difference between women and men could be due to sex-specific physiological differences, different occupational histories, consumer product use, or PFOA clearance rates—the time it takes for PFOA to leave the body. Studies have shown that PFOA can leave women's bodies through menstruation, childbirth and breastfeeding. Higher levels of PFOA in blood were seen among women ages 60 and over than among women ages 18 to 59. This may due to less PFOA leaving older women's bodies through menstruation following menopause.

Table 1. PFOA levels in blood (µg/L) by demographic characteristics

	n	Geometric mean	p-value
All participants	472	10.1	N/A
Age groups			
Adults	412	10.7	
Children	60	7.0	p=0.07
Adults			
Male	189	13.0	
Female	213	8.8	p<0.01
Males by age group			
Males, age 18-39 years	30	7.4	
Males, age 40-59 years	75	14.4	
Males, age 60 years and older	81	14.4	p=0.10
Females by age group (3 categories)			
Females, age 18-39 years	39	4.0	
Females, age 40-59 years	89	8.4	
Females, age 60 years and older	83	13.0	p<0.001
Females by age group (2 categories)			
Females, age 18-59 years	128	6.9	
Females, age 60 years and older	83	13.0	p<0.01
Children			
Boys	22	6.5	
Girls	38	7.2	p=0.61
Boys by age group			
Boys, age 12 years and under	13	6.3	
Boys, age 13-17 years	9	6.8	p=0.84
Girls by age group			
Girls, age 12 years and under	20	8.0	
Girls, age 13-17 years	18	6.5	p=0.31
Race/ethnicity			
White	407	10.5	
Other	65	7.9	p=0.16
Household income			
Less than $40,000	93	8.9	
$40,000 to less than $90,000	112	10.8	
$90,000 or more	72	9.0	
Don't know/refused	120	12.6	p=0.40
Highest level of education (adults only)			
High school or less	122	12.6	
Some college	86	11.7	
College graduate	170	8.6	
Don't know/refused	34	14.0	p=0.11

Comparison of PFOA Concentrations in Blood by Work History

As expected, individuals who worked directly with PFOA had statistically higher PFOA levels (geometric mean = 59 µg/L) in their blood compared to those who did not work directly with PFOA (geometric mean = 9.6 µg/L).

Average PFOA blood levels in other populations that worked with PFOA were higher than in the Bennington and North Bennington communities. For example, in a study of workers in Decatur, Alabama, participants had an average level of PFOA in blood of 1130 µg/L. Levels were likely lower among the Bennington and North Bennington worker group, in part, because most of these workers stopped working with PFOA in 2002 or earlier.

Table 2. PFOA levels in blood (µg/L) by work history

	n	Geometric mean	p-value
Potential sources of exposure to PFOA			
Worked directly with PFOA	24	59.0	
Worked indirectly with PFOA prior to 2003	41	10.7	
Worked or lived at Chemfab building after 2002	16	2.8	
Currently live in a home that was tested	351	10.6	
Formerly lived in a home that was tested	27	4.5	
Other	13	2.7	N/A
Work directly with PFOA at job?			
Yes	24	59.0	
No	388	9.6	p<0.001
Ever served in the military?			
Yes	45	13.3	
No	362	10.4	p=0.22
Ever served as a professional/volunteer firefighter?			
Yes	21	10.5	
No	383	10.7	p=0.95
Ever work at power plant?			
Yes	7	8.4	
No	405	10.7	p=0.38
Ever work in wire manufacturing?			
Yes	8	19.6	
No	404	10.6	p=0.20
Ever work in electronics manufacturing?			
Yes	20	15.0	
No	392	10.5	p=0.22
Ever work with fluorocarbons?			
Yes	44	30.5	
No	368	9.4	p<0.001
Ever work in rubber or plastics industry?			
Yes	16	21.6	
No	396	10.4	p=0.05
Ever work with fire-fighting foam?			
Yes	11	10.2	
No	401	10.7	p=0.84

Comparison of PFOA Concentrations in Blood by Diet, Among Non-Workers

Preliminary results showed an association between people who frequently ate fruits and vegetables grown within the sampling area and PFOA in blood. However, this association was only present among those who also consumed contaminated water with high levels of PFOA and was not present among those who consumed water with low levels of PFOA. In other words, consuming contaminated drinking water likely was responsible for the original association. Please see (Supplemental Table 1) for these data.

Table 3. PFOA levels in blood (µg/L) by diet (among non-workers)

	n	Geometric mean	p-value
Fruit/vegetable grown within sampling area			
Daily/Weekly	165	11.8	
Monthly/Never	215	8.3	p=0.04
Milk from animals raised within sampling area			
Yes	19	16.7	
No	236	10.0	p=0.15
Meat from animals raised within sampling area			
Yes	42	7.4	
No	232	10.5	p=0.11
Fish caught within sampling area			
Yes	24	7.8	
No	284	10.3	p=0.30
Eggs from animals raised within sampling area			
Yes	105	12.2	
No	169	9.5	p=0.22

Comparison of PFOA Concentrations in Blood by Medication Use

Statistically significant differences in blood PFOA concentrations were seen among those who reported taking blood pressure or cholesterol-lowering medication. These individuals had a higher geometric mean level of blood PFOA than those who did not report taking such medications.

Table 4. PFOA levels in blood (µg/L) by medication use

	n	Geometric mean	p-value
Cholesterol-lowering medication			
Yes	90	18.1	
No	372	8.9	p<0.001
Blood pressure-lowering medication			
Yes	115	16.2	
No	350	8.9	p<0.001
Thyroid medication			
Yes	44	11.9	
No	416	9.9	p=0.71

Comparison of PFOA Concentrations in Blood by Lifestyle Factors

The results indicate that PFOA concentrations in blood were not statistically different based on lifestyle factors.

Table 5. PFOA levels in blood (µg/L) by smoking, alcohol, exercise and weight status

	n	Geometric mean	p-value
Have you smoked 100 cigarettes in your lifetime?			
Yes	169	12.4	
No	222	9.3	p=0.06
Do you currently smoke?			
Yes	39	15.5	
No	364	10.2	p=0.12
How many drinks do you have in an average			
None	184	11.0	
1 to 3 drinks a week	122	9.3	
4 or more drinks a week	89	13.0	p=0.15
Hours spent doing strenuous exercise			
Less than 3 hours	215	11.3	
3 or more hours	156	9.6	p=0.24
BMI Categories			
Underweight/normal	144	9.2	
Overweight	141	12.9	
Obese	127	10.4	p=0.20

Comparison of PFOA Concentrations in Blood by Women's History and Blood Donation

The results indicate that PFOA concentrations in blood were not statistically different based on number of children, history of breastfeeding, or blood/plasma donation.

Table 6. PFOA levels in blood (µg/L) by women's history and blood donation

	n	Geometric mean	p-value
Women's History			
How many children have you had?			
0	<6	Suppressed	
1	34	9.8	
2	63	9.4	
3 or more	55	10.2	p=0.52
Breastfed at least one child?			
Yes	89	9.5	
No	17	9.4	p=0.76
Blood Donation			
Donate blood or plasma?			
Yes	31	8.5	
No	370	10.8	p=0.17

Note: The Health Department does not report findings (suppresses) when there are less than 6 individuals in a given category. This is to protect confidential health information.

Association Between PFOA Concentrations in Blood and Adverse Health Outcomes

Potential associations between blood PFOA concentration and adverse health outcomes were assessed among adults only. Logistic regression modeling was used, which is a statistical method that can be used to estimate the probability of a given outcome using one or more predictor variables. Further information about this statistical method, how the models were built, and how to interpret the results can be found in . The unadjusted (crude) associations between blood PFOA concentration and the various health outcomes can be found in (Supplemental Table 2). The associations between blood PFOA concentration and various health outcomes, adjusted for the age of participants and lifetime smoking can be found in (Supplemental Table 3).

The results of this exposure assessment **indicated an association** between PFOA concentrations in blood and the following conditions:

- High cholesterol
- Hypertension during pregnancy

The results of this exposure assessment **did not indicate that there is an association** between PFOA concentrations in blood and the following conditions in this population:

- Chronic kidney disease
- Increased uric acid levels
- Altered liver enzymes
- Fatty liver disease
- Hypothyroidism
- Hyperthyroidism
- Ulcerative colitis

Due to sample size, an association between less common health outcomes (such as some of those listed above) and exposure to PFOA, was unlikely to have been detected in this study. The fact that no association was detected with these health outcomes in the Bennington/North Bennington community does not rule out the possibility that an association exists.

The Health Department does not report findings when there are less than 6 individuals with a given health outcome. This is to protect confidential health information, as well as to avoid calculating potentially unstable rates due to small numbers. Due to the limited sample size, we were unable to evaluate the association between PFOA concentrations in blood and the following conditions:

- Testicular cancer
- Kidney cancer

Strengths and Limitations of This Exposure Assessment Study

As with all epidemiologic studies, this exposure assessment is subject to several limitations. First, the Bennington/North Bennington investigation was limited by a small sample size when compared to other PFOA exposure assessment studies. This impacted the Health Department's ability to assess associations between blood PFOA concentration and certain, rare health outcomes.

Additionally, this study was cross-sectional in nature, meaning that it was a "snapshot" of exposure and outcome at one point in time. It does not consider what blood PFOA

concentrations may have been in the past, or health outcomes that participants may develop in the future. Most importantly, it does not allow for temporality to be established between exposure and outcome. With this type of study, it is impossible to determine whether exposure to PFOA occurred before or after health outcomes developed. Therefore, with this type of study, it is impossible to say whether or not exposure to PFOA definitively caused a given health outcome.

Information about exposure to PFOA and various health outcomes was self-reported, and the Health Department did not validate the information via other sources (e.g. medical records).

Lastly, the study population was composed of those who were willing to have their blood tested and share their personal information. The Health Department does not know how many other people were eligible and chose not to participate. Therefore, volunteer/selection bias may be present.

There are several strengths to this study that should also be considered. Response rate for the survey was incredibly high. Only a handful of the 477 surveys that were distributed were not returned. Blood samples were collected for all participants and water samples were collected for all participants who never worked or resided at the former Chemfab building. This allowed for accurate quantification of the concentration of PFOA in both blood and water at the individual level.

Conclusions and Recommendations

Drinking water from contaminated wells was the main, non-occupational source of exposure to PFOA in the Bennington/North Bennington community. The Health Department would have been concerned that there was another undetected and unaddressed exposure pathway if this association between blood PFOA concentration and PFOA concentration in drinking water had not been found.

The Health Department recommends that water with PFOA above 20 parts per trillion NOT be used for drinking, preparing food, cooking, brushing teeth, watering gardens, or any other manner of ingestion. The Health Department recommends that anyone who has concerns about their health related to their exposure to PFOA consult with their health care provider. If new information regarding PFOA and health emerges, the Health Department will update health care providers in the area.

Appendices

Appendix A: Statistical Methodology

Spearman's Rank Correlation

Spearman's rank correlation is the non-parametric version of the commonly used Pearson product-moment correlation. This means that it can be used when data is not normally distributed, and it would be inappropriate to use the Pearson product-moment method. Similar to a Pearson product-moment correlation, Spearman's rank correlation measures the strength and direction of an association between two variables.

A Spearman's rank correlation coefficient of zero indicates that there is no association between the two variables. A Spearman's rank correlation coefficient of 1 indicates that the two variables are perfectly positively correlated (all the data points would fall on the trendline), and that as one variable increases, so does the other. A Spearman's rank correlation coefficient of negative 1 indicates that the two variables are perfectly negatively correlated (all the data points would fall on the trendline), and that as one variable increases, the other decreases.

p-values

In statistics, p-values are used to assess whether the difference seen between two (or more) groups is a true difference or due to chance. These p-values represent the likelihood that an association was found when none truly exists. The smaller the p-value, the stronger the statistical significance of the association, and the more likely there is a true difference between groups. Generally speaking, a p-value of 0.05 is considered to be "statistically significant." As p-values get smaller, for example a p-value of 0.01 or 0.0001, the difference between groups is considered to be more and more significant.

When to Use a Non-Parametric Statistical Test

The decision to use parametric or non-parametric statistics is based upon whether the variables meet the assumptions (rules for appropriate choice) for a statistical test. One of the assumptions for performing a parametric test (e.g. an independent samples t-test or an Analysis of Variance (ANOVA) test) is that the outcome variable is normally distributed (evenly distributed above and below the average). In contrast, non-parametric statistical tests do not make these types of assumptions. In the case of this exposure assessment, the PFOA concentrations in blood were not "normally" distributed. There were far more low concentrations and fewer high concentrations, so the assumption of normality was not met. Therefore, non-parametric statistics were used to compare the mean PFOA concentrations in blood by the different sub-groups.

Wilcoxon Rank-Sum Test

To compare the mean PFOA concentrations in blood across two groups (e.g. adults as compared to children), p-values were generated using a Wilcoxon rank-sum test (the non-parametric equivalent to the independent samples t-test). Instead of comparing mean values, like the independent samples t-test, the Wilcoxon rank-sum compares the order in which the observations from two samples fall when ranked from lowest to highest. This allows the test to assess for statistically significant differences (in this case, of blood PFOA concentration) between two groups, without being affected by the distribution of the data.

Kruskal-Wallis Test

To compare mean PFOA concentrations in blood across three or more groups (e.g. having a BMI considered underweight/normal, a BMI considered overweight, or a BMI considered obese), p-values were generated using a Kruskal-Wallis test (the non-parametric equivalent to an ANOVA test). Rather than comparing the mean values of three or more groups, like the ANOVA test, the Kruskal-Wallis test compares the ranks of three or more groups. This allows the test to assess for a statistically significant difference (in this case, of blood PFOA concentration) between any of the three or more groups.

It is important to remember that a statistically significant p-value generated by a Kruskal-Wallis test is indicative of a difference between any two of the three or more groups. This test does not allow you to identify which two groups are different from each other, or whether all of the groups you are considering are different from each other.

Logistic Regression

Logistic regression is a statistical method used to determine the probability or odds of an outcome occurring. Outcomes modeled in this way must be binary, which means that there are only two alternatives (either you have high cholesterol or you do not). In a logistic regression model, changes in the odds of a given outcome are assessed based on the values of one or more predictor variables. For example, a person's age, smoking status, and weight could be included in a logistic regression model assessing the odds of developing lung cancer.

For this study, two types of logistic regression models were built for each health outcome. The first model, known as a crude model, assessed the odds of each health outcome using only blood PFOA concentration as a predictor variable. These results are presented in Appendix B, Supplemental Table 2. The second model, known as an adjusted model, attempted to control for other variables that may have also influenced likelihood of developing the various health outcomes (confounding variables).

Potential confounders were assessed by adding each variable to the model one at a time. If the crude odds ratio changed by more than 10%, then the variable was considered for adjustment in the final model. Previous studies, biological plausibility, and the 10% change in estimate rule, were considered in determining which confounders to include in the final adjusted model for

each outcome. The final model for each health outcome was adjusted for age and lifetime smoking. These results are presented in Appendix B, Supplemental Table 3.

Odds Ratio Interpretation

An odds ratio is a statistical term that describes the association between an exposure and an outcome. It represents the odds that an outcome will occur given a particular exposure. For example, an odds ratio could be used to describe the odds of getting lung cancer, given exposure to smoking cigarettes.

In the case of this PFOA exposure assessment study, the associated odds of the adverse health outcome increased or decreased by the amount shown in the odds ratio when the PFOA blood concentration increased 10-fold. An odds ratio of 1 indicates no change, an odds ratio of 2 indicates a doubling of the odds of a given outcome, and an odds ratio of 0.5 indicates a halving of the odds of a given outcome.

95% Confidence Interval

The 95% confidence interval is used to estimate the precision of an odds ratio. The narrower a 95% confidence interval is, the more precise the odds ratio estimate. For example, a 95% confidence interval of 1.1 to 1.2 indicates a more precise odds ratio estimate than a 95% confidence interval of 1.1 to 10.0. An odds ratio estimate is considered to be statistically significant if the 95% confidence interval does not contain the "null" value of 1.0. For example, a 95% confidence interval of 0.8 to 1.3 would not be considered statistically significant.

Supplemental Table 3. Adjusted associations between PFOA levels in blood (for each 1-$\log_{10}$ µg/L increase) with various health outcomes

Outcome	n with outcome	n without outcome	Adjusted OR (95% CI)
High Cholesterol	112	269	1.4 (1.1, 2.1)
Chronic Kidney Disease	8	370	0.4 (0.1, 1.3)
Increased Uric Acid Levels	20	353	1.1 (0.5, 2.2)
Altered Liver Enzymes	20	355	1.0 (0.4, 1.9)
Fatty Liver Disease	14	362	0.7 (0.3, 1.7)
Hypothyroidism	44	334	1.0 (0.6, 1.7)
Hyperthyroidism	7	370	0.5 (0.1, 1.8)
Ulcerative Colitis	10	365	1.4 (0.5, 3.5)
Preeclampsia (pregnant women)	13	126	6.2 (1.9, 20.3)

Abbreviations: CI, confidence interval; OR, odds ratio; n, number of participants

DHHS Mission Statement

To join communities and families in providing opportunities for citizens to achieve health and independence

Responsibilities

To meet the health needs of New Hampshire citizens: The Department of Health and Human Services recognizes its responsibility to improve access to health care, to ensure its quality and to control costs through improved purchasing, planning and organization of health care services. The Department will work to prevent disease and to protect and improve the health and safety of all citizens through regulatory and health promotion efforts.

To meet the basic human needs of New Hampshire citizens: The Department has a responsibility to provide financial, medical and emergency assistance and employment support services to those in need, in order to assist individuals in reaching self-sufficiency.

To provide treatment and support services to those who have unique needs including disabilities, mental illness, special health care needs or substance abuse problems: The Department has a responsibility to ensure access to quality community-based services for eligible individuals.

To protect and care for New Hampshire's most vulnerable citizens: The Department has a special responsibility to support those who, due to age, disability or circumstance, are at risk and in need of protection.

AUTHENTICATED U.S. GOVERNMENT INFORMATION GPO

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Tuesday,
March 7, 2006

Part IV

Environmental Protection Agency

40 CFR Part 723

Premanufacture Notification Exemption for Polymers; Amendment of Polymer Exemption Rule to Exclude Certain Perfluorinated Polymers; Proposed Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 723

[EPA–HQ–OPPT–2002–0051; FRL–7735–5]

RIN 2070–AD58

Premanufacture Notification Exemption for Polymers; Amendment of Polymer Exemption Rule to Exclude Certain Perfluorinated Polymers

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA is proposing to amend the polymer exemption rule, which provides an exemption from the premanufacture notification (PMN) requirements of the Toxic Substances Control Act (TSCA), to exclude from eligibility polymers containing as an integral part of their composition, except as impurities, certain perfluoroalkyl moieties consisting of a CF3- or longer chain length. This proposed exclusion includes polymers that contain any one or more of the following: Perfluoroalkyl sulfonates (PFAS); perfluoroalkyl carboxylates (PFAC); fluorotelomers; or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule. If finalized as proposed, any person who intends to manufacture (or import) any of these polymers not already on the TSCA Inventory would have to complete the TSCA premanufacture review process prior to commencing the manufacture or import of such polymers. EPA believes this proposed change to the current regulation is necessary because, based on recent information, EPA can no longer conclude that these polymers "will not present an unreasonable risk to human health or the environment," which is the determination necessary to support an exemption under TSCA, such as the polymer exemption rule.

DATES: Comments must be received on or before May 8, 2006.

ADDRESSES: Submit your comments, identified by docket identification (ID) number EPA–HQ–OPPT–2002–0051, by one of the following methods:

- *http://www.regulations.gov.* Follow the on-line instructions for submitting comments.
- *E-mail: oppt.ncic@epa.gov.*
- *Mail:* Document Control Office (7407M), Office of Pollution Prevention and Toxics (OPPT), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001.
- *Hand Delivery:* OPPT Document Control Office (DCO), EPA East Bldg., Rm. 6428, 1201 Constitution Ave., NW., Washington, DC. Attention: Docket ID number EPA–HQ–OPPT–2002–0051. The DCO is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the DCO is (202) 564–8930. Such deliveries are only accepted during the Docket's normal hours of operation, and special arrangements should be made for deliveries of boxed information.

Instructions: Direct your comments to docket ID number EPA–HQ–OPPT–2002–0051. EPA's policy is that all comments received will be included in the public docket without change and may be made available on-line at *http://www.regulations.gov*, including any personal information provided, unless the comment includes information claimed to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Do not submit information that you consider to be CBI or otherwise protected through regulations.gov or e-mail. The regulations.gov website is an "anonymous access" system, which means EPA will not know your identity or contact information unless you provide it in the body of your comment. If you send an e-mail comment directly to EPA without going through regulations.gov your e-mail address will be automatically captured and included as part of the comment that is placed in the public docket and made available on the Internet. If you submit an electronic comment, EPA recommends that you include your name and other contact information in the body of your comment and with any disk or CD ROM you submit. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment. Electronic files should avoid the use of special characters, any form of encryption, and be free of any defects or viruses.

Docket: All documents in the docket are listed in the regulations.gov index. Although listed in the index, some information is not publicly available, e.g., CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, is not placed on the Internet and will be publicly available only in hard copy form. Publicly available docket materials are available electronically through regulations.gov or in hard copy at the OPPT Docket, EPA Docket Center (EPA/DC), EPA West, Rm. B102, 1301 Constitution Ave., NW., Washington, DC. The EPA Docket Center Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the OPPT Docket is (202) 566–0280.

FOR FURTHER INFORMATION CONTACT: *For general information contact:* Colby Lintner, Regulatory Coordinator, Environmental Assistance Division (7408M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (202) 554–1404; e-mail address: *TSCA-Hotline@epa.gov.*

For technical information contact: Geraldine Hilton, Chemical Control Division (7405M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (202) 564–8986; e-mail address: *hilton.geraldine@epa.gov.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you manufacture or import polymers that contain as an integral part of their composition, except as impurities, certain perfluoroalkyl moieties consisting of a CF3- or longer chain length ("affected polymers"). As specified in the proposed regulatory text (§ 723.250(d)(6)), this includes polymers that contain any one or more of the following: PFAS; PFAC; fluorotelomers; or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule. Persons who import or intend to import polymers that are covered by the final rule would be subject to TSCA section 13 (15 U.S.C. 2612) import certification requirements, and to the regulations codified at 19 CFR 12.118 through 12.127 and 127.28. Those persons must certify that they are in compliance with the PMN requirements. The EPA policy in support of import certification appears at 40 CFR part 707, subpart B. Importers of formulated products that contain a polymer that is a subject of this proposed rule as a component (for example, for use as a water-proof coating for textiles or as a top anti-reflective coating (TARC) used to manufacture integrated circuits) may also be potentially affected. A list of potential monomers and reactants that could be used to manufacture polymers

that would be affected by this rulemaking may be found in the public docket (Ref. 1). Potentially affected entities may include, but are not limited to:

- Chemical manufacturers or importers (NAICS 325), e.g., persons who manufacture (defined by statute to include import) one or more of the subject chemical substances.
- Chemical exporters (NAICS 325), e.g., persons who export, or intend to export, one or more of the subject chemical substances.

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. To determine whether you or your business may be affected by this action, you should carefully examine the applicability provisions in 40 CFR 723.250. If you have any questions regarding the applicability of this action to a particular entity, consult the technical person listed under **FOR FURTHER INFORMATION CONTACT.**

B. What Should I Consider as I Prepare My Comments for EPA?

1. *Submitting CBI.* Do not submit this information to EPA through regulations.gov or e-mail. Clearly mark the part or all of the information that you claim to be CBI. For CBI information in a disk or CD ROM that you mail to EPA, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is claimed as CBI. In addition to one complete version of the comment that includes information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

2. *Tips for preparing your comments.* When submitting comments, remember to:

i. Identify the document by docket number and other identifying information (subject heading, **Federal Register** date, and page number).

ii. Follow directions. The Agency may ask you to respond to specific questions or organize comments by referencing a Code of Federal Regulations (CFR) part or section number.

iii. Explain why you agree or disagree; suggest alternatives and substitute language for your requested changes.

iv. Describe any assumptions and provide any technical information and/or data that you used.

v. If you estimate potential costs or burdens, explain how you arrived at the estimate.

vi. Provide specific examples to illustrate your concerns and suggested alternatives.

vii. Explain your views as clearly as possible, avoiding the use of profanity or personal threats.

viii. Make sure to submit your comments by the comment period deadline identified.

II. Background

A. What Action is the Agency Taking?

The Agency is proposing to exclude from the polymer exemption rule (40 CFR 723.250), which exempts certain chemical substances from TSCA section 5 PMN requirements, polymers containing as an integral part of their composition, except as impurities, certain perfluoroalkyl moieties consisting of a CF3- or longer chain length. This exclusion includes polymers that contain any one or more of the following: PFAS; PFAC; fluorotelomers; or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule. The effective date of the final rule would be one year from the date of publication of the final rule. Manufacture or import of any of these polymers not already on the TSCA Inventory, including polymers currently being produced under the polymer exemption rule, would no longer be eligible for the polymer exemption and, in the case of continued manufacture or import after the effective date of the final rule, would require completion of the premanufacture review requirements under TSCA section 5(a)(1)(A) and 40 CFR part 720 prior to the effective date of the final rule. After expiration of the one year period between the publication date of the final rule and the effective date, the PMN requirement would apply in full to manufacturers and importers of all polymers that are subject to the final rule.

EPA is actively working with industry to develop more complete data on affected polymers. In light of these efforts, certain publicly available and confidential business information regarding the specific chemicals manufactured, current production volumes, uses/applications, environmental fate and effects, and toxicity of the polymeric materials that would be subject to this proposed rule has been made and continues to be made available to EPA on an ongoing basis. Accordingly, EPA may supplement the public docket for this proposed rule with relevant non-confidential business information as it is received by the Agency. Non-confidential information related to this proposed rule may also be found in administrative record number (AR) AR–226, which is the public administrative record that the Agency has established for perfluorinated chemicals generally. Interested parties should consult AR–226 for additional information on PFAS, PFAC, fluorotelomers, or other perfluoroalkyl moieties. To receive an index of AR–226, contact the EPA Docket Center by telephone: (202) 566–0280 or e-mail: *oppt.ncic@epa.gov.*

Additional information may be found in EPA Docket ID No. OPPT–2003–0012, which covers the Agency's enforceable consent agreement (ECA) process for certain of these chemicals. Instructions on accessing an EPA public docket are provided at the beginning of this document under **ADDRESSES.**

B. What is the Agency's Authority for Taking This Action?

Section 5(a)(1)(A) of TSCA requires persons to notify EPA at least 90 days before they manufacture or import a new chemical substance for commercial purposes. Section 3(9) of TSCA defines a "new chemical substance" as any substance that is not on the Inventory of Chemical Substances compiled by EPA under section 8(b) of TSCA. Section 5(h)(4) of TSCA authorizes EPA, upon application and by rule, to exempt the manufacturer or importer of any new chemical substance from part or all of the provisions of section 5 if the Agency determines that the manufacture, processing, distribution in commerce, use, or disposal of such chemical substance, or any combination of such activities will not present an unreasonable risk of injury to human health or the environment. Section 5(h)(4) also authorizes EPA to amend or repeal such rules. EPA is acting under these authorities to amend the polymer exemption rule at 40 CFR 723.250.

C. Why is the Agency Taking This Action?

1. *Polymers containing PFAS or PFAC.* EPA is proposing to amend the polymer exemption rule, last amended in 1995, because the Agency has received information which suggests that polymers containing PFAS or PFAC may degrade and release fluorochemical

residual compounds into the environment. Once released, PFAS or PFAC are expected to persist in the environment, are expected to bioaccumulate, and are expected to be highly toxic. Accordingly, EPA believes that it can no longer make the determination that the manufacturing, processing, distribution in commerce, use, or disposal of polymers containing PFAS or PFAC "will not present an unreasonable risk to human health or the environment" as required under TSCA section 5(h)(4).

PFAS or PFAC are used in a variety of polymeric substances to impart oil and water resistance, stain and soil protection, and reduced flammability. The same features that make the polymeric coatings containing PFAS or PFAC useful, allow the polymeric compound to be stable to the natural environmental conditions that produce degradation. It has been demonstrated that PFAS or PFAC-containing compounds can undergo degradation (chemical, microbial, or photolytic) of the non-fluorinated portion of the molecule leaving the remaining perfluorinated acid untouched (Ref. 2). Further degradation of the perfluoroalkyl residual compounds is extremely difficult. Even under routine conditions of municipal waste incinerators (MWIs), the Agency believes that the PFAS and PFAC produced by oxidative thermal decomposition of the polymers will remain intact (the typical conditions of a MWI are not stringent enough to cleave the carbon-fluorine bonds) to be released into the environment. EPA has evidence that polymers containing PFAS or PFAC may degrade, possibly by incomplete incineration, and release these perfluorinated chemicals into the environment (Ref. 3).

EPA has received data on the PFAS and PFAC chemicals perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), respectively. Biological sampling recently revealed the presence of PFOS and PFOA in fish, birds, and mammals, including humans across the United States and in other countries. The widespread distribution of the chemicals suggests that PFOS and PFOA may bioaccumulate. PFOS and PFOA have a high level of toxicity and have shown liver, developmental, and reproductive toxicity at very low dose levels in exposed laboratory animals (Ref. 4).

Although the Agency has far more data on PFOS and PFOA than on other PFAS and PFAC chemicals, EPA believes that other PFAS and PFAC chemicals of CF3- or longer chain length may share similar toxicity, persistence and bioaccumulation characteristics. Based on currently available information, EPA believes that, while all PFAS and PFAC chemicals are expected to persist, the length of the perfluorinated chain may have an effect on the other areas of concern for these chemicals: Bioaccumulation and toxicity. PFAS and PFAC chemicals with longer carbon chain lengths may be of greater concern (Refs. 5, 6, and 7). EPA has insufficient evidence at this time, however, to definitively establish a lower carbon chain length limit to meet the "will not present an unreasonable risk" finding, which is the determination necessary to support an exemption under section 5(h)(4) of TSCA.

The Agency, working in cooperation with the fluorochemical industry, has been investigating the physicochemical properties, the environmental fate and distribution, and the toxicity of PFAS and PFAC chemicals, including polymers already in production. These data help the Agency to evaluate these polymers to ascertain any potential risks on a case-by-case basis.

2. *Polymers containing fluorotelomers or other perfluoroalkyl moieties.* EPA is also proposing to exclude from the exemption polymers that contain fluorotelomers, or that contain perfluoroalkyl moieties of a CF3- or longer chain length that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule. EPA has received data on various perfluorinated chemical substances that indicate potential concerns and that the Agency should evaluate polymers that contain these perfluoroalkyl moieties through the PMN process. For example, the fluorotelomer alcohol 2-(perfluorooctyl)ethanol [678–39–7], also known as 8–2 alcohol, has been shown to degrade to form PFOA when exposed to activated sludge during accelerated biodegradation studies (Ref. 8).

Initial test data from a study in rats dosed with fluorotelomer alcohol and other preliminary animal studies on various telomeric products containing fluorocarbons structurally similar to PFAC or PFAS have demonstrated a variety of adverse effects including liver, kidney and thyroid effects (Ref. 9).

Preliminary investigations have demonstrated the presence of fluorotelomer alcohols in the air in 6 different cities (Ref. 10). This finding is significant because it is indicative of widespread fluorotelomer alcohol distribution and it further indicates that air may be a route of exposure to these chemicals, which can ultimately become PFOA. Fluorotelomer alcohols are generally incorporated into the polymers via covalent ester linkages, and it is possible that degradation of the polymers may result in release of the fluorotelomer alcohols to the environment.

Based on the presence of fluorotelomer alcohols in the air, the growing data demonstrating that fluorotelomer alcohols metabolize or degrade to generate PFOA (Ref. 11), the preliminary toxicity data on certain compounds containing fluorotelomers (such as the 8–2 alcohol), and the possibility that polymers containing fluorotelomers as an integral part of the polymer composition may degrade in the environment thereby releasing fluorotelomer alcohols or other perfluoroalkyl-containing substances, EPA believes that it can no longer conclude that polymers containing fluorotelomers as an integral part of the polymer composition "will not present an unreasonable risk of injury to health or the environment" as required for an exemption under section 5(h)(4) of TSCA. Therefore, EPA is proposing to exclude polymers that contain such fluorotelomers from the polymer exemption at 40 CFR 723.250.

Although EPA does not have specific data demonstrating that polymers containing perfluoroalkyl moieties other than PFAS, PFAC, or fluorotelomers present the same concerns as those containing PFAS, PFAC, or fluorotelomers, EPA is nevertheless proposing to exclude polymers containing perfluoroalkyl groups, consisting of a CF3- or longer chain length, that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule from the polymer exemption. Based on available data which indicates that compounds containing PFAS or PFAC may degrade in the environment thereby releasing the PFAS or PFAC moiety, and that fluorotelomers may degrade in the environment to form PFAC, EPA believes that it is possible for polymers containing these other types of perfluoroalkyl moieties to also degrade over time in the environment thereby releasing the perfluoroalkyl moiety. EPA also believes that once released, such moieties may potentially degrade to form PFAS or PFAC. EPA does not believe, therefore, that it can continue to make the "will not present an unreasonable risk of injury to health or the environment" finding for such polymers and is proposing to exclude them from the polymer exemption. EPA is specifically requesting comment on this aspect of the proposed rule. Please see Unit VII. of this document for

specific information that EPA is interested in obtaining to evaluate whether continued exemption for polymers containing fluorotelomers or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule is appropriate.

D. Would Manufacturers or Importers of Affected Polymers That Were Previously Manufactured Under the Terms of the Polymer Exemption Rule Need to Complete the PMN Review Process or to Cease Production?

This proposed rule would allow manufacturers or importers of affected polymers, who are in full compliance with the terms of the polymer exemption rule, to continue manufacture or import for a period of one year after the date of publication of the final rule. However, after the one-year period, polymers that are subject to the final rule (including affected polymers made under the polymer exemption rule since promulgation of the 1995 amendment to the rule) would no longer be eligible for exemption under the polymer exemption rule. Therefore, a person who intends to continue manufacturing or importing polymers subject to the final rule without interruption would have to complete the PMN review process before the effective date in order to comply with the final rule. Manufacturers or importers of polymers that are already on the Inventory of Chemical Substances compiled and published under section 8(b) of TSCA (15 U.S.C. 2607(b)) would not be affected by this proposed amendment. The PMN requirements in section 5(a) of TSCA apply only to new chemical substances which are those that are not included on the Inventory of Chemical Substances. However, several of the polymers that are already included on the Inventory of Chemical Substances are subject to control actions under TSCA section 5, including section 5(e) consent orders and section 5(a)(2) Significant New Use Rules (SNURS).

III. Summary of This Proposed Rule

A. Polymers Containing PFAS or PFAC

EPA is proposing to amend the polymer exemption rule (40 CFR 723.250) to exclude polymers containing PFAS or PFAC consisting of a CF3- or longer chain length from eligibility under the polymer exemption. This exclusion would be codified at 40 CFR 723.250(d)(6). EPA has received data on PFOS (a PFAS chemical containing a perfluoroalkyl moiety with eight carbon atoms) and PFOA (a PFAC chemical containing a perfluoroalkyl moiety with seven perfluorinated carbon atoms), that indicate that these chemicals are expected to persist and have the potential to bioaccumulate and be hazardous to human health and the environment. PFOS and PFOA have been found in the blood of workers exposed to the chemicals and in the general populations of the United States and other countries. They have also been found in many terrestrial and aquatic animal species worldwide. PFAS and PFAC chemicals used in the production of polymers may be released into the environment by degradation. It is possible, therefore, that the widespread presence of PFOS and PFOA in the environment may be due, in part, to the degradation of such polymers and the subsequent release of the PFAS and PFAC components into the environment. However, the method of degradation and environmental distribution is uncertain.

Animal test data for PFOS and PFOA have shown liver, developmental, and reproductive toxicity at very low exposure levels. Animal test data indicate that PFOA may cause cancer, and an epidemiologic study reported an increased incidence of bladder cancer mortality in a small number of workers at a plant that manufactures perfluorinated chemicals. The number of carbon atoms on the PFAS/PFAC component may influence the bioaccumulation potential and the toxicity. In particular, there is some evidence that PFAS/PFAC moieties with longer carbon chains may present greater concerns for bioaccumulation potential and toxicity than PFAS/PFAC moieties with shorter carbon chains (Refs. 5, 6, and 7). Although there is insufficient understanding available at present to determine the carbon number below which PFAS and PFAC chemicals "will not present an unreasonable risk," efforts are underway to develop a better understanding of the environmental fate, bioaccumulation potential, and human and environmental toxicity of PFAS and PFAC chemicals with shorter carbon chains. At this time, however, EPA can no longer conclude that polymers containing PFAS or PFAC will not present an unreasonable risk to human health or the environment. Therefore, this proposed amendment would exclude polymers containing PFAS or PFAC from eligibility for exemption from TSCA section 5(a)(1)(A) reporting requirements for new chemical substances.

B. Polymers Containing Fluorotelomers or Other Perfluoroalkyl Moieties

EPA is also proposing to exclude from the polymer exemption rule polymers that contain fluorotelomers, or that contain perfluoroalkyl moieties of a CF3- or longer chain length that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymers molecule. EPA has concerns with respect to the potential health and environmental effects of these substances and the Agency believes that polymers containing such moieties should be subject to the premanufacture review process so that EPA can better evaluate and address these concerns.

As discussed in Unit IV.E., there is a growing body of data demonstrating that fluorotelomer alcohols metabolize or degrade to generate PFOA. Initial studies have also demonstrated toxic effects of certain compounds containing fluorotelomers (derived from the 8–2 alcohol). Preliminary investigations have found that fluorotelomer alcohols were present in the air above several cities, indicating that these substances may be widely distributed and that air may be a route of exposure. EPA believes that polymers containing fluorotelomers or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymers molecule may degrade in the environment thereby releasing fluorotelomer alcohols or other perfluoroalkyl-containing substances. Accordingly, EPA can no longer conclude that polymers containing fluorotelomers and these other perfluoroalkyl moieties "will not present an unreasonable risk of injury to health or the environment" as required for an exemption under section 5(h)(4) of TSCA. Therefore, EPA is proposing to exclude such polymers from the polymer exemption at 40 CFR 723.250.

C. Proposed Implementation

EPA is proposing to delay the implementation of the final rule in order to provide current manufacturers or importers of the affected polymers who are in full compliance with the terms of the existing polymer exemption rule, additional time to come into compliance with the amendment proposed without disrupting their ability to manufacture or import those polymers.

To do this, EPA is proposing to establish an effective date for the final rule that is one year after the date of publication of the final rule. After expiration of the one year implementation period, polymers that

are subject to the final rule (including affected polymers made under the polymer exemption rule) would no longer be eligible for exemption. Therefore, a person who intends to manufacture or import polymers subject to the final rule must complete the TSCA premanufacture review process before the effective date. EPA believes that the one year period between the publication date of the final rule and the effective date of the final rule would provide adequate time for current manufacturers and importers of the polymers subject to the final rule to prepare and submit PMNs for those polymers and for EPA to review the PMNs.

As an alternative to the one year effective date, EPA could establish an effective date of the final rule as 30 days after its publication in the **Federal Register**, the minimum required by section 553(c) of the Administrative Procedure Act, but provide an extended compliance date for those who, prior to the effective date of the final rule, had already initiated the manufacture or import of polymers that are subject to the final rule. Under this approach, the TSCA section 5(a)(1)(A) requirement to submit a PMN for a new chemical substance would be re-established with respect to polymers that are subject to the final rule, beginning 30 days after publication of the final rule in the **Federal Register**. However, those who are manufacturing or importing polymers under the existing exemption would have one year from the effective date to complete the PMN process. EPA is specifically requesting comment on this or other alternatives for implementing the final rule that would achieve the purposes of TSCA section 5 without disrupting ongoing manufacture or import of currently-exempt polymers.

IV. Proposed Rule

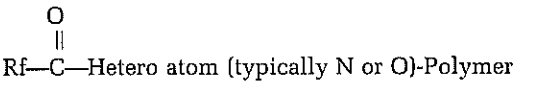
A. History Subsequent to the 1995 Amendment to the Polymer Exemption Rule

The 1995 amendments to the polymer exemption rule expanded the polymer exemption to include polymers made from reactants that contain certain halogen atoms, including fluorine. The best available information in 1995 indicated that most halogen containing compounds, including unreactive polymers containing PFAS and PFAC chemicals, were chemically and environmentally stable and would not present an unreasonable risk to human health and the environment. In 1999, however, the 3M Company (3M) provided the Agency with preliminary reports that indicated widespread distribution of PFOS in humans and animals (Refs. 12, 13, and 14). In addition, on May 16, 2000, 3M announced that it would phase out perfluorooctanyl chemistry in light of the persistence of certain fluorochemicals and their detection at extremely low levels in the blood of the general population and animals. 3M indicated that production of these chemicals would be substantially discontinued by the end of 2000 (Ref. 15). Based on this information from 3M, EPA began to investigate potential risks from PFOS and other perfluorinated chemicals, as well as polymers containing these chemicals. EPA believes that polymers containing PFAS or PFAC chemicals may degrade, releasing these chemicals into the environment where they are expected to persist. The number of carbon atoms on the PFAS or PFAC molecule, whether as a single compound, or as a component of a polymer, may influence bioaccumulation potential and toxicity. EPA also believes that polymers containing fluorotelomers or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule may degrade, releasing these substances into the environment where they may further degrade into PFAS or PFAC.

B. Defining Polymers That Are Subject to This Proposed Rule

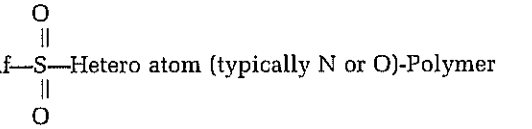
1. *Polymers containing PFAS or PFAC.* This proposed rule applies to a large group of polymers containing one or more fully fluorinated alkyl sulfonate or carboxylate groups. None of these polymers occur naturally. Such polymers are considered "new chemical substances" under TSCA if they have not been included in the Inventory of Chemical Substances compiled and published under section 8(b) of TSCA (15 U.S.C. 2607(b)). For a list of examples of the Ninth Collective Index of chemical names and CAS Registry Numbers (CASRN) of chemical substances used to make polymers that are subject to this proposed rule amendment, see Ref.1. EPA has concerns for the perfluorinated carbon atoms in the Rf substituent, below, when that Rf unit is associated with the polymer through the carbonyl (PFAC) or sulfonyl (PFAS) group. How these materials are incorporated into the polymer is immaterial (they may be counter ions, terminal/end capping agents, or part of the polymer backbone).

PFAC



Rf = Perfluoroalkyl CF3- or greater

PFAS



This proposed rule would specifically exclude from the polymer exemption at 40 CFR 723.250 polymers that contain any PFAS or PFAC group consisting of a CF3- or longer chain length. EPA has increasing concerns as the number of carbon atoms that are perfluorinated in any individual Rf substituent increases. PFOA (perfluorooctanoate) is a PFAC (see top structure) which has 7 carbon atoms in the Rf moiety (CAS nomenclature rules count the carbonyl carbon atom as the eighth carbon for naming purposes, hence the octanoate terminology). PFOS (perfluorooctane sulfonate) is a PFAS (see bottom structure) which has 8 carbon atoms in the Rf moiety. Generally, the longer the chain of perfluorinated C atoms, the greater the persistence and retention time in the body; furthermore, the C8 chain length has been associated with adverse health effects.

Most of the toxicity data currently available on PFAS and PFAC chemicals pertain to the PFOS potassium salt (PFOSK) and the PFOA ammonium salt

(APFO). There is some evidence that PFAS/PFAC moieties with longer carbon chains may present greater concerns than PFAS/PFAC moieties with shorter carbon chains (Refs. 5, 6, and 7). However, EPA has insufficient information at this time to determine a limit for which shorter chain lengths "will not present an unreasonable risk to human health or the environment."

2. *Polymers containing fluorotelomers or other perfluoroalkyl moieties.* EPA is also proposing to exclude polymers that contain fluorotelomers, or that contain perfluoroalkyl moieties of a CF3- or longer chain length that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule.

Fluorotelomers: One method that is commonly used to incorporate perfluorinated compounds into polymers is to use fluorotelomers, such as perfluoroalkyl ethanol. Telomerization is the reaction of a telogen with a polymerizable ethylenic compound to form low molecular weight polymeric compounds, commonly referred to as "telomers." For example, the reaction of pentafluoroethyl iodide (a telogen) with tetrafluoroethylene forms a fluorotelomer iodide intermediate which is then reacted with ethylene and converted into perfluoroalkyl ethanol. This chemical can be further reacted to form a variety of useful materials which may subsequently be incorporated into the polymer (Ref. 16). The fluorochemical group formed by the telomerization process is predominantly straight chain, and depending on the telogen used produces a product having an even number of carbon atoms. However, the chain length of the fluorotelomer varies widely. A representative structure for these compounds is:

F-(CF2-CF2)x-Anything (often CH2-CH2-O-Polymer) $x \geq 1$

Other perfluoroalkyl moieties: Perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule can be attached to the polymers using conventional chemical reactions. A representative structure for these compounds is:

F-(CF2)x-(C,S)-Polymer $x \geq 1$

C. Concerns With Respect to Polymers Containing PFAS, PFAC, Fluorotelomers, or Other Perfluoroalkyl Moieties

EPA is proposing to amend the polymer exemption rule because the Agency has received information which suggests that polymers containing certain perfluoroalkyl moieties consisting of a CF3- or longer chain length (i.e., PFAS, PFAC, fluorotelomers, or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule) may degrade and release fluorochemical residual compounds into the environment. Once released, these substances are expected to persist in the environment, may bioaccumulate, and may be highly toxic. The evidence suggests that fluorotelomers and perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule do persist in the environment, and that they can be metabolically transformed into PFAC, which bioaccumulates and is toxic. The following sections will summarize the concerns the Agency has for PFAS, PFAC, fluorotelomers, or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule.

D. Summary of Data on PFAS and PFAC

1. *Use and production volume data for PFOS.* PFAS chemicals have been in commercial use since the 1950's. There were three main categories of use: Surface treatments, paper protectors (including food contact papers), and performance chemicals (Ref. 3). The various surface treatment and paper protection uses constituted the largest volume of PFOS production and therefore, were believed to present the greatest source of widespread human and environmental exposure to PFOS.

Until the year 2000, 3M was the largest manufacturer of PFAS chemicals in the United States. On May 16, 2000, following discussions with the Agency, 3M issued a press release announcing that it would discontinue the production of perfluorooctanyl chemicals used in the manufacture of some of its repellent and surfactant products. In its statement, 3M committed to "substantially phase out production" by the end of calendar year 2000 (Ref. 17). In subsequent correspondence with the Agency, 3M provided a schedule documenting its complete plan for discontinuing all manufacture of specific PFOS and related chemicals for most surface treatment and paper protection uses (including food contact uses regulated by the Food and Drug Administration (FDA)) by the end of 2000, and for discontinuing all manufacture for any uses by the end of 2002 (Ref. 15).

The 3M phase-out plan eliminated many of these chemicals from further distribution in commerce. The largest production volume (both initially produced and removed from commerce) was for polymers. Other PFAS chemicals, however, continue to be manufactured or imported by other companies and may be of concern. EPA followed the voluntary 3M phase-out with the promulgation of a SNUR under TSCA section 5. The SNUR limits any future manufacture or importation of PFOS before EPA has had an opportunity to review activities and risks associated with the proposed manufacture or importation (Ref. 17a).

PFAS chemicals produced for surface treatment applications provide soil, oil, and water resistance to personal apparel and home furnishings. Specific applications in this use category include protection of apparel and leather, fabric/upholstery, and carpeting. Applications are undertaken in industrial settings such as textile mills, leather tanneries, finishers, fiber producers, and carpet manufacturers. PFAS chemicals are also used in aftermarket treatment of apparel and leather, upholstery, carpet, and automobile interiors, with the application performed by both the general public and professional applicators (Ref. 3). In 2000, the domestic production volume of PFAS chemicals for this use category was estimated to be 2.4 million pounds (Ref. 15).

PFAS chemicals produced for paper protection applications provide grease, oil, and water resistance to paper and paperboard as part of a sizing agent formulation. Specific applications in this use category include food contact applications (plates, food containers, bags, and wraps) regulated by the FDA under 21 CFR 176.170, as well as non-food contact applications (folding cartons, containers, carbonless forms, and masking papers). The application of sizing agents is undertaken mainly by paper mills and, to some extent, converters, who manufacture bags, wraps, and other products from paper and paperboard (Ref. 3). In 2000, the domestic production volume of PFOS chemicals for this use category was estimated to be 2.7 million pounds (Ref. 15).

PFAS chemicals in the performance chemicals category are used in a wide variety of specialized industrial, commercial, and consumer applications. Specific applications include fire fighting foams, mining and oil well surfactants, acid mist suppressants for metal plating and electronic etching baths, alkaline cleaners, floor polishes, photographic film, denture cleaners,

shampoos, chemical intermediates, coating additives, carpet spot cleaners, and as an insecticide in bait stations for ants (Ref. 3). In 2000, the domestic production volume of PFAS chemicals for this use category was estimated to be 1.5 million pounds (Ref. 15).

2. *Use and production volume data for PFOA.* The largest use for PFOA is as a chemical intermediate. Its salts are used in emulsifier and surfactant applications, including as a fluoropolymer polymerization aid in the production of fluoropolymers and fluoroelastomers. This proposed rule does not require PMN notification for polymers where APFO is used exclusively as a polymerization aid and is not incorporated into the polymer structure.

Until the year 2000, 3M was also the largest manufacturer and importer of PFOA and its salts in the United States. Subsequent to its May 16, 2000 announcement (see Unit IV.D.1.), 3M provided clarification that this announcement included PFOA as well as PFOS, indicating that it was phasing out certain FLUORAD Brand specialty materials that contained PFOA and its salts (Ref. 4). Following the phase-out by 3M, DuPont began to manufacture PFOA in the United States, and is currently the sole U.S. producer (Ref. 18). The Fluoropolymer Manufacturers Group has stated that DuPont will not sell APFO outside the fluoropolymer industry (Ref. 18a).

The four principal use categories for salts of PFOA include uses as:

- A fluoropolymer polymerization aid in the industrial synthesis of fluoropolymers and fluoroelastomers such as polytetrafluoroethylene (PTFE) and polyvinylidene fluoride (PVDF), with a variety of industrial and consumer uses (Refs. 19, 20, and 21).
- A post-polymerization processing aid to stabilize suspensions of fluoropolymers and fluoroelastomers prior to further industrial processing (Ref. 19).
- A processing aid for factory-applied fluoropolymer coatings on architectural fabrics, metal surfaces, and fabricated or molded parts (Ref. 20).
- An extraction agent in ion-pair reversed-phased liquid chromatography (Ref. 22).

PTFE and PVDF account for the largest volumes of fluoropolymer production (Ref. 23). PFOA is also used in other fluoropolymer and fluoroelastomer manufacturing and processing. In addition, 3M used PFOA in the industrial synthesis of a fluoroacrylic ester, which is used in an industrial coating application (Ref. 19).

The fluoropolymers manufactured with PFOA as a polymerization aid are used to produce a wide variety of industrial and consumer products. These products include: High performance lubricants; personal care products; architectural fabrics; films; cookware, breathable membranes for apparel; protective industrial coatings; wire and cable insulation; semiconductor chip manufacturing equipment; pump seals, liners and packing; medical tubing; aerospace devices; automotive hoses and tubing; and, a wide variety of electronic products (Ref. 24). The fluoropolymer industry has informed EPA that it does not intend to incorporate PFOA into the polymer structure for these uses (Ref. 24). However, if PFOA were to be incorporated into the structure of a polymer, this proposed rule amendment would require PMN notification.

3. *Exposure data for PFOS and PFOA.* PFOS and PFOA have been detected at low levels in the blood of humans and wildlife throughout the United States, providing clear evidence of widespread exposure to these chemicals (Refs. 4 and 25). Studies are underway to determine the sources of exposure for PFOS and PFOA. Several potential pathways may account for the widespread exposure to these chemicals.

For PFOS, these pathways may have included:

- Dietary intake from the consumption of food wrapped in paper containing PFOS derivatives.
- Inhalation from aerosol applications of PFOS-containing consumer products.
- Inhalation, dietary, or dermal exposures resulting from manufacturing, as well as industrial, commercial, and consumer use and disposal of PFOS-containing chemicals and products.

Because PFOA is not used directly in consumer products, its exposure pathways may result from manufacturing and industrial uses and disposal of PFOA-derived chemicals and products, typically used as processing aids for fluoropolymer manufacturing. EPA has data indicating that PFOA is released into the environment from industrial discharges to air, water, and land (Refs. 19, 20, 26). Canadian research has found that thermolysis of fluoropolymers, e.g., PTFE, can liberate small quantities of perfluorocarboxylic acids, which include PFOA (Ref. 27). However, the extreme conditions needed to produce these PFAC products make this source of PFAC an improbable contributor to the environmental availability of PFAC.

Data indicate that PFOA may also be produced by the degradation or metabolism of fluorotelomer alcohols (Refs. 8 and 48), suggesting exposures to PFOA may result from releases from fluorotelomer manufacturing and processing, and from the use and disposal of fluorotelomer-containing products.

4. *Environmental fate of PFAS and PFAC.* Little information is available on the fate of high molecular weight PFAS and PFAC polymers in the environment. Based on their chemical structures they are expected to be stable, with many derivatives being non-volatile, but few studies are available to allow confirmation.

EPA cannot currently conduct a definitive assessment of the environmental fate and transport of PFOS- and PFOA-derived chemicals. Conventional modeling programs are based on "traditional" organic compounds which contain carbon and hydrogen. These models are not designed to account for the physical-chemical properties and environmental behavior of perfluorinated compounds. Therefore, these models provide results that are not representative of perfluorinated chemicals.

PFOS and PFOA may be expected to be similar in their resistance to hydrolysis, biodegradation and photolysis, however, they may have differences in adsorption/desorption, transport, distribution and bioaccumulation. Based on available data, PFOS and PFOA are expected to persist in the environment.

PFOS and PFOA are stable to hydrolysis. The 3M Environmental Laboratory (Refs. 28 and 29) performed studies of the hydrolysis of PFOS and PFOA. The study procedures were based on EPA's OPPTS Harmonized Test Guideline 835.2110. Results were based on the observed concentrations of PFOS and PFOA in buffered aqueous solutions as a function of time. Based on these studies, it was estimated that the hydrolytic half-lives of PFOS and PFOA at 25°C are greater than 41 and 92 years, respectively.

PFOS and PFOA do not measurably biodegrade in the environment. The biodegradation of PFOA was investigated using acclimated sludge microorganisms and a shake culture study modeled after the Soap and Detergent Association's presumptive test for degradation (Ref. 30). Neither thin-layer nor liquid chromatography detected the presence of any metabolic products over the course of 2 ½ months, indicating that PFOA does not readily undergo biodegradation. In a related study PFOA was not measurably degraded in activated sludge inoculum (Ref. 31). Several other studies conducted between 1977 to 1987 did

not show PFOA biodegradation either; however, the results are questionable due to methodological problems (Refs. 32, 33, 34, and 35). Similar results have been reported for PFOS. No measurable biodegradation of PFOS in activated sludge, sediment, aerobic soil, anaerobic sludge, or pure culture studies were found (Ref. 36).

PFOS and PFOA appear to be stable to photolysis. Direct photolysis of PFOA was examined by Todd (Ref. 37) and photodegradation was not observed. Hatfield (Ref. 38) studied both direct and indirect photolysis utilizing techniques based on EPA and the Organization for Economic Cooperation and Development (OECD) guidance documents. There was no conclusive evidence of direct or indirect photolysis. A PFOA half-life in the environment was estimated to be greater than 349 days.

PFOA appears to be mobile in soils, and there is conflicting data on the mobility of PFOS in soils. The adsorption-desorption of PFOA and PFOS were studied by 3M using 14C-labeled test chemicals in distilled water with a Brill sandy loam soil. The study reported a soil adsorption coefficient (K_{oc}) of 14 for PFOA, and a K_{oc} of 45 for PFOS, indicating that both PFOS and PFOA have high mobility in Brill sandy loam soil. The K_{oc} value for PFOA, and possibly PFOS, however, is questionable due to the lack of accurate information on the purity of the 14C-labeled test substance (Refs. 39 and 40). In another 3M study using OECD method 106 to measure the sorption of PFOS (Ref. 41), it was reported that the chemical strongly adsorbed to all of the soil/sediment/sludge matrices tested. The test substance, once adsorbed, did not desorb readily, even when extracted with an organic solvent. K_{oc} values more than 3 orders of magnitude higher than those reported by Welsh were observed. DuPont evaluated PFOA in a soil absorption/desorption study and found that the average absorption of PFOA in various soils tested at 1:1 soil:solution ratio ranged from 40.8% to 81.8%, and the highest average desorption coefficient (K_d) value, 22.5 mL/g, was found in sludge (Ref. 42). The data from the 3M and DuPont studies, while of high quality, are of limited utility in understanding the movement of PFOA released to soil. Batch sorption studies, because of their limited nature, do not provide all the information needed to understand the behavior of PFOA in the environment. The data raised additional questions, and are not sufficient to understand the behavior of PFOA in soil to allow EPA to determine whether soil is an important pathway for human and environmental exposure to PFOA.

Both substances have low vapor pressures and Henry's Law constants (HLCs), which suggest low potential for volatilization from water. The estimated HLCs for PFOS are 1.4 E-7, 2.4 E-8, 4.7 E-9 , 3 E-9 atm-m^3/mole (atmospheres per meter cubed per mole), utilizing the vapor pressure of 3.3 E-9 atm at 20°C and water solubility values of 12, 25, 370, and 570 (mg/L) in unfiltered seawater, filtered seawater, fresh water and pure water, respectively. For PFOA, the estimated HLCs is < 3.8 x 10E-10 atm-m^3/mole based on a vapor pressure of 9.1 E-8 atm and > 100 g/L solubility in water.

Even though PFOS and PFOA have relatively low vapor pressures, it is possible that they can be adsorbed on suspended particles. This is because PFOS and PFOA are considered semi-volatile organic compounds, i.e., substances with vapor pressures between about 10 E-4 to 10 E-11 atm at ambient temperatures (Ref. 43). The potential adsorption of PFOS and PFOA onto particulate matter might also create an exposure pathway.

EPA believes that PFAS and PFAC chemicals may bioaccumulate, but is uncertain as to the mechanism. Three studies have been conducted that attempted to determine the bioaccumulation potential of PFOS and PFOA. In the first study using the fathead minnow, the calculated bioconcentration factor (BCF) was 1.8 for APFO (Ref. 46). However, questions were raised about the analytical techniques, high test chemical concentration and short test duration of the study. In a Japanese study using carp, the bioaccumulation potential of PFOA was low, with apparent bioaccumulation factors ranging from 3.1–9.1 (Ref. 45). In the final study using bluegill sunfish from the 3M Decatur plant, no fluorochemicals were detected in the river water-exposed fish (Ref. 44). However, interpretation of the study was problematic. For instance, effluent concentrations of subject fluorochemicals were not characterized; the protocol for fish exposure was not found; there was no information on the Tennessee river water or effluent used, whether there was an opportunity for depuration of the fish prior to sacrifice, or the cause of death for the 12 dead fish; and the study did not differentiate between bioaccumulation of the test compound and sorption onto the fish surface. These studies in fish on the bioaccumulation of these chemicals suggest relatively low bioaccumulation potential. However, the detection of PFOS and to a lesser extent PFOA in wild animals indicates the possibility of accumulation of the chemicals in biota. PFOS and PFOA appear to have higher bioaccumulation factors than other PFAS and PFAC chemicals. Thus, the widespread presence of these chemicals in living organisms also suggests that PFOS and PFOA may bioaccumulate.

5. *Health effects of PFAS and PFAC.* Most of the Agency's concerns for the health effects of polymers subject to this proposed rule focus on the perfluoroalkyl moiety, which may be released into the environment. The Agency's non-confidential data for health effects of PFAS and PFAC chemicals are on PFOS (as PFOSK) and PFOA (as APFO). EPA has insufficient evidence to determine that polymers containing PFAS or PFAC with any number of carbons on the perfluoroalkyl moiety "will not present an unreasonable risk to human health or the environment" and is proposing to exclude polymers that contain these chemicals from eligibility for the exemption. Below is a summary of the results of toxicological and epidemiological studies on PFOS and PFOA.

i. *Health effects of PFOS.* All of the data summarized in Unit IV.D.5.i., as well as the primary references, are detailed in the OECD "Hazard Assessment of Perfluorooctane sulfonate (PFOS) and its Salts" (Ref. 25).

Toxicology studies show that PFOS is well absorbed orally and distributes primarily in the serum and liver. PFOS can also be formed as a metabolite of other perfluorinated sulfonates. It does not appear to be further metabolized. Elimination from the body is slow and occurs via both urine and feces. The elimination half-life for an oral dose is 7.5 days in adult rats and approximately 200 days in Cynomolgus monkeys. In humans, the mean elimination half-life of PFOS reported in 9 retired workers appears to be considerably longer, on the order of years (mean = 8.67 years; range = 2.29–21.3 years; standard deviation = 6.12).

PFOS has shown moderate acute toxicity by the oral route with a combined (male and female) rat LD_{50} of 251 mg/kg. The LD_{50} was 233 mg/kg in males and 271 mg/kg in females. A 1-hour LC_{50} of 5.2 mg/L in rats has been reported. PFOS was found to be mildly irritating to the eyes and non-irritating to the skin of rabbits. PFOS does not induce gene mutation in selected strains of *Salmonella typhimurium* or *Escherichia coli* nor does it induce chromosomal aberrations in human lymphocytes in culture when tested *in vitro* either with or without metabolic activation. PFOS does not induce

unscheduled DNA synthesis in primary cultures of rat hepatocytes and is negative when tested *in vivo* in a mouse bone marrow micronucleus assay.

Three 90-day subchronic studies of PFOS have been conducted. One was a dietary study in rats and two were gavage studies in rhesus monkeys. In addition, a four week and a 26 week capsule study in Cynomolgus monkeys and a two-year cancer bioassay in rats, have been conducted . The primary health effects of concern, based on available data, are liver effects, developmental effects, and mortality. Mortality was associated with a steep dose-response across all ages and species.

In the rat subchronic study, CD rats, 5/sex/group, were administered dietary levels of PFOS at 0, 30, 100, 300, 1,000 or 3,000 parts per million (ppm) for 90 days. All of the rats in the 300, 1,000 and 3,000 ppm groups died. Before death, the rats in all groups showed signs of toxicity including emaciation, convulsions following handling, hunched back, red material around the eyes, yellow material around the anogenital region, increased sensitivity to external stimuli, reduced activity, and moist red material around the mouth or nose. Mean body weight and average food consumption were reduced in all groups. Animals in the 100 ppm and 30 ppm dose groups also showed signs of gastrointestinal effects and hematological abnormalities. At necropsy, treatment related gross lesions were present in all treated groups and included varying degrees of discoloration and/or enlargement of the liver and discoloration of the glandular mucosa of the stomach. Histologic examination also showed lesions in all treated groups.

Two 90-day rhesus monkey studies were performed. In the first study, PFOS was administered to male and female rhesus monkeys at doses of 0, 10, 30, 100, or 300 mg/kg/day in distilled water by gavage for 90 days. In the second study, PFOS was administered at doses of 0, 0.5, 1.5, or 4.5 mg/kg/day also in distilled water by gavage for 90 days. None of the monkeys in the first study survived treatment. In the second study, all monkeys in the 4.5 mg/kg/day group died or were sacrificed *in extremis*. Before death all monkeys suffered from similar signs of toxicity including decreased activity, emesis with some diarrhea, body stiffening, general body trembling, twitching, weakness, convulsions, and prostration. At necropsy, several of the monkeys in the 100 and 300 mg/kg/day groups had a yellowish-brown discoloration of the liver; histologic examination showed no microscopic lesions. Congestion, hemorrhage, and lipid depletion of the adrenal cortex was noted in all treated groups in the first study.

In the second study, animals in the 30 mg/kg/day dose group had reduced mean body weight, significant reduction in serum cholesterol and a 50% reduction in serum alkaline phosphatase activity. At necropsy, all males and females had marked diffuse lipid depletion in the adrenals. One male and two females had moderate diffuse atrophy of the pancreatic exocrine cells with decreased cell size and loss of zymogen granules. Two males and one female had moderate diffuse atrophy of the serous alveolar cells characterized by decreased cell size and loss of cytoplasmic granules. Animals in the 1.5 and 0.5 mg/kg/day dose group survived to the end of the study and showed signs of decreased activity and gastrointestinal distress.

Two additional studies were conducted in Cynomolgus monkeys. In the first study, male and female Cynomologus monkeys received doses of 0, 0.02, or 2.0 mg/kg/day PFOS in capsules placed directly into the stomach for 30 days. All animals survived treatment. There were no test-related effects on clinical observations, body weight, food consumption, body temperatures, hematology, enzyme levels, cell proliferation in the liver, testes or pancreas or macroscopic or microscopic pathology findings.

In the second study, PFOS was administered to Cynomolgus monkeys by oral capsule at doses of 0, 0.03, 0.15, or 0.75 mg/kg/day for 26 weeks. Animals from the 0.15 and 0.75 mg/kg/day groups were assigned to a recovery group and were held for observation for an additional 26 weeks after treatment. Two males in the 0.75 mg/kg/day dose group did not survive the 26 weeks of treatment. The first animal died on day 155. In addition to being cold to the touch, clinical signs in the first animal included: Constricted pupils, pale gums, gastrointestinal distress, low food consumption, hypoactivity, labored respiration, dehydration, and recumbent position. An enlarged liver was detected by palpation. Cause of death was determined to be pulmonary necrosis with severe acute inflammation. The second male was sacrificed in a moribund condition on day 179. Clinical signs noted included low food consumption, excessive salivation, labored respiration, hypoactivity and ataxia. The cause of death was not determined. Males and females in the 0.75 mg/kg/day dose-group had lower total cholesterol and males and females in the 0.15 and 0.75 mg/kg/day groups had lower high density lipoprotein cholesterol during treatment. The effect on total cholesterol worsened with time. By day 182, mean total cholesterol for males and females in the high dose group were 68% and 49% lower, respectively, than levels in the control animals. Males in the high dose group also had lower total bilirubin concentrations and higher serum bile acid concentrations than males in either the control or other treatment groups. The effect on total cholesterol was reversed within 5 weeks of recovery and the effect on high density lipoprotein cholesterol was reversed within 9 weeks of recovery.

At terminal sacrifice, females in the 0.75 mg/kg/day dose-group had increased absolute liver weight, liver-to-body weight percentages, and liver-to-brain weight ratios. In males, liver-to body weight percentages were increased in the high-dose group compared to the controls. "Mottled" livers and centrilobular or diffuse hepatocellular hypertrophy and centrilobular or diffuse hepatocellular vacuolation were also observed in high dose males and females. No PFOS related lesions were observed either macroscopically or microscopically at recovery sacrifice indicating that the effects seen at terminal sacrifice may be reversible.

The chronic toxicity and carcinogenicity of PFOS have been studied in rats. The results of the study show that PFOS is hepatotoxic and carcinogenic, inducing tumors of the liver, and thyroid and mammary glands. In this study, groups of 40 to 70 male and female Crl:CD (SD)IGS BR rats were given PFOS in the diets at concentrations of 0, 0.5, 2, 5, or 20 ppm for 104 weeks. A recovery group was given the test material at 20 ppm for 52 weeks and was observed until death. Five animals per sex in the treatment groups were sacrificed during weeks 4, 14, and 53.

At the terminal sacrifice, the livers of animals given 5 or 20 ppm were enlarged, mottled, diffuse darkened, or focally lightened. Hepatotoxicity, characterized by significant increases in centrilobular hypertrophy, centrilobular eosinophilic hepatocytic granules, centrilobular hepatocytic pigment, or centrilobular hepatocytic vacuolation was noted in male and/or female rats given 5 or 20 ppm. A significant increase in hepatocellular centrilobular hypertrophy was also observed in mid-dose (2 ppm) male rats. For neoplastic effects, a significant positive trend was noted in the incidences of hepatocellular adenoma in male rats. A significantly increased incidence was observed for thyroid follicular cell

adenoma in the high-dose recovery group when compared to the control group.

In females, significant positive trends were observed in the incidences of hepatocellular adenoma and combined hepatocellular adenoma and carcinoma. A significant increase for combined thyroid follicular cell adenoma and carcinoma was observed in the mid-high (5.0 ppm) group as compared to the control group. Except for the high-dose group, increases in mammary tumors were observed in all treatment groups when compared to the controls.

Developmental toxicity studies on PFOS have been conducted in rats, mice and rabbits. The first study administered four groups of 22 time-mated Sprague-Dawley rats 0, 1, 5, and 10 mg/kg/day PFOS in corn oil by gavage on gestation days (GD) 6–15. Signs of maternal toxicity consisted of significant reductions in mean body weights during GD 12–20 at the high-dose group of 10 mg/kg/day. No other signs of maternal toxicity were reported. Under the conditions of the study, a no observed adverse effect level (NOAEL) of 5 mg/kg/day and a lowest observed adverse effect level (LOAEL) of 10 mg/kg/day for maternal toxicity were indicated. Developmental toxicity evident at 10 mg/kg/day consisted of reductions in the mean number of implantation sites, corpora lutea, resorption sites, and the mean numbers of viable male, female, and total fetuses, but the differences were not statistically significant. In addition, unusually high incidences of unossified, asymmetrical, bipartite, and missing sternebrae were observed in all dose groups; however, these skeletal variations were also observed in control fetuses at the same rate and therefore these effects were not considered to be treatment-related. A fetal lens finding initially described as a variety of abnormal morphological changes localized to the area of the embryonal nucleus, was later determined to be an artifact of the free-hand sectioning technique and therefore not considered to be treatment-related.

Groups of 25 pregnant Sprague-Dawley rats were administered 1, 5, and 10 mg/kg/day PFOS in corn oil by gavage on gestation days (GD) 6–15. Evidence of maternal toxicity occurred at the 5 and 10 mg/kg/day dose groups both consisted of hunched posture, anorexia, bloody vaginal discharge, uterine stains, alopecia, rough haircoat, and bloody crust. Significant decreases in mean body weight gains during GD 6–8, 6–16, and 0–20 were also observed in the 5 and 10 mg/kg/day dose groups. These reductions were considered to be treatment-related since mean body weight gains were greater than controls during the post-exposure period (GD 16–20). Significant decreases in mean total food consumption were observed on GD 17–20 in the10 mg/kg/day dose group, and on GD 7–16 and 0–20 in both the 5 and 10 mg/kg/day dose groups. The mean gravid uterine weight in the 10 mg/kg/day dose group was significantly lower when compared with controls. The mean terminal body weights minus the gravid uterine weights were lower in all treated groups, with significant decreases at 5 and 10 mg/kg/day. High-dose animals also exhibited an increased incidence in gastrointestinal lesions. No significant differences were observed in pregnancy rates, number of corpora lutea, and number and placement of implantation sites among treated and control groups. Two dams in the 10 mg/kg/day dose group were found dead on GD 17. Under the conditions of the study, a NOAEL of 1 mg/kg/day and a LOAEL of 5 mg/kg/day for maternal toxicity were indicated.

Significant decreases in mean fetal weights for both males and females were observed in the 5 and 10 mg/kg/day dose groups. Statistically significant increases in incomplete closure of the skull were observed in the low- and high-dose groups but not in the mid-dose group. Statistically significant increases in the incidences in the number of litters containing fetuses with visceral anomalies, delayed ossification, and skeletal variations were observed in the high dose group of 10 mg/kg/day. These included external and visceral anomalies of the cleft palate, subcutaneous edema, and cryptorchism as well as delays in skeletal ossification of the skull, pectoral girdle, rib cage, vertebral column, pelvic girdle, and limbs. Skeletal variations in the ribs and sternebrae were also observed. Under the conditions of the study, a NOAEL of 1 mg/kg/day and a LOAEL of 5 mg/kg/day for developmental toxicity were indicated.

In another study, Sprague-Dawley rats and CD-1 mice were administered doses of 0, 1, 5, or 10 mg/kg/day PFOS in 0.5% Tween-20 by gavage beginning on gestation day 2 and continuing until term. Half of the dams were sacrificed on gestation day 21 (rats) or gestation day 17 (mice) and the remaining dams were allowed to deliver. Preliminary results are available. In rats, there was a significant reduction in maternal body weight gain at 5 and 10 mg/kg/day. Maternal serum cholesterol and triglycerides were reduced at 10 mg/kg/day, but liver weights were comparable to control. At 10 mg/kg/day, there was a reduction in fetal body weight and an increase in cleft palate and anasarca. All pups were born alive, but within 4 to 6 hours after birth all the pups in the 10 mg/kg/day group died, and 95% of the pups in the 5 mg/kg/day group died within 24 hours. In mice, maternal body weight was unaffected and liver weights were significantly increased at 5 and 10 mg/kg/day; serum triglycerides were reduced at 5 and 10 mg/kg/day. The incidence of fetal mortality was slightly increased at 10 mg/kg/day and mean fetal body weights were comparable to control. However, neonatal body weights were reduced during the first 3 days of life. Additional studies are underway to further elucidate the dose-response relationships and to examine the mechanism for the neonatal death.

Pregnant New Zealand White rabbits, 22 per group, were administered doses of 0, 0.1, 1.0, 2.5, or 3.75 mg/kg/day PFOS in 0.5% Tween-80 by gavage on gestation days 7–20 in another study. Maternal toxicity was evident at doses of 1.0 mg/kg/day and above. One doe in the 2.5 mg/kg/day group and nine does in the 3.75 mg/kg/day aborted. There was a significant increase in the incidence of scant feces in the 3.75 mg/kg/day group. Scant feces were also noted in one and three does in the 1.0 and 2.5 mg/kg/day groups, respectively. Mean maternal body weight gains were significantly reduced in the 3.75 and 2.5 mg/kg/day group. Mean food consumption (g/kg/day) was significantly reduced in the 2.5 and 3.75 mg/kg/day dose group. The LOAEL for maternal toxicity was 1.0 mg/kg/day and the NOAEL was 0.1 mg/kg/day.

Developmental toxicity was evident at doses of 2.5 mg/kg/day and above. Mean fetal body weight (male, female, and sexes combined) was significantly reduced in the 2.5 and 3.75 mg/kg/day groups. There was also a significant reduction in the ossification of the sternum (litter averages) in the 2.5 and 3.75 mg/kg/day groups, and a significant reduction in the ossification of the hyoid (litter averages), metacarpals (litter averages), and pubis (litter and fetal averages) in the 3.75 mg/kg/day group. The LOAEL for developmental toxicity was 2.5 mg/kg/day and the NOAEL was 1.0 mg/kg/day.

In epidemiological studies, cross-sectional, occupational, and a longitudinal study did not indicate consistent associations between workers' PFOS serum levels and certain hematology and other clinical chemistry parameters. In the cross-sectional analysis, workers with the highest PFOS exposures had significantly higher serum triiodothyronine levels and significantly lower thyroid hormone binding ratio; however, hormonal

parameters were not measured longitudinally. In addition, these studies were conducted on volunteers only, female employees could not be analyzed due to the small number of women employed at these plants, different labs and analytical techniques were used to measure PFOS, and only a small number of employees were common to all of the sampling periods. In a mortality study of workers exposed to PFOS, most of the cancer types and non-malignant causes were not elevated. However, a statistically significant mortality risk of bladder cancer (SMR = 12.77, 95% CI = 2.63–37.35) was reported in 3 male employees. All of the workers had been employed at the plant for more than 20 years and all of them had worked in "high exposure jobs" for at least 5 years. Although it is unlikely that this effect would be due to chance or tobacco smoking, it cannot be ascertained whether fluorochemicals are responsible for the excess of bladder cancer deaths, or whether other carcinogens may be present in the workplace.

In human blood samples, PFOS has been detected in the serum of occupational and general populations in the parts per billion (ppb) to ppm range. In the United States, recent blood serum levels of PFOS in manufacturing employees have been as high as 12.83 ppm, while in the general population, pooled serum collected from the United States blood banks and commercial sources have indicated mean PFOS levels ranging from 29 to 44 ppb. Mean serum PFOS levels from individual samples in adults and children were approximately 43 ppb.

Sampling of several wildlife species from a variety of sites across the United States has shown widespread distribution of PFOS. In recent analyses, PFOS was detected in the ppb range in the plasma of several species of eagles, wild birds, and fish. PFOS has also been detected in the ppb range in the livers of unexposed rats used in toxicity studies, presumably through a dietary source (fishmeal).

Although the PFOS levels detected in the blood of the general population are low, this widespread presence, combined with the persistence, the bioaccumulative potential, and the reproductive and subchronic toxicity of the chemical, raises concerns for potential adverse effects on people and wildlife (wild mammals and birds) over time should the chemical substances continue to be produced, released, and accumulate in the environment.

ii. *Health effects of PFOA.* All of the data presented in Unit IV.D.5.ii. are detailed in an EPA hazard assessment of PFOA (Ref. 4). Primary references can be obtained from that document.

The primary health effects of concern for PFOA, based on available data, are liver toxicity and developmental toxicity. Most of the health effects data for PFOA are on the ammonium salt, APFO. Occupational data indicate that mean serum levels of PFOA in workers range from 0.84 to 6.4 ppm, with the highest reported level of 81.3 ppm. In non-occupational populations, mean pooled blood bank and commercial PFOA samples ranged from 3 to 17 ppb. The mean PFOA level in individual blood samples (in children and adults) was 5.6 ppb.

Animal studies have shown that APFO is well absorbed following oral and inhalation exposure, and to a lesser extent following dermal exposure. Rats show gender differences in the elimination of APFO. APFO distributes primarily to the liver, plasma, and kidney, and to a lesser extent, other tissues of the body including the testis and ovary. It does not partition to the lipid fraction or adipose tissue. APFO is not metabolized and there is evidence of enterohepatic circulation of the compound. Female rats appear to have a secretory mechanism that rapidly eliminates APFO; this secretory mechanism is either lacking or relatively inactive in male rats and is not found in monkeys or humans.

Epidemiological studies on the effects of PFOA in humans have been conducted on workers. Two mortality studies, as well as studies examining effects on the liver, pancreas, endocrine system, and lipid metabolism, have been conducted to date. A longitudinal study of worker surveillance data has also been conducted. A weak association with PFOA exposure and prostate cancer was reported in one study; however, this result was not observed in an update to the study in which the exposure categories were modified. A non-statistically significant increase in estradiol levels in workers with high serum PFOA levels (> 30 ppm) was also reported, but none of the other hormone levels analyzed indicated any adverse effects.

The acute oral toxicity of APFO was tested in male and female rats in three studies. Death occurred at concentrations ≥ 464 mg/kg. Abnormal findings upon necropsy (kidney, stomach, uterus) were observed at 500 mg/kg (higher concentrations were not tested). Clinical signs of toxicity observed in these three studies included: Red-stained face, stained urogenital area, wet urogenital area, hypoactivity, hunched posture, staggered gait, excessive salivation, ptosis, piloerection, decreased limb tone, ataxia, corneal opacity, and hypothermic to touch.

The acute inhalation toxicity of APFO was tested in male and female Sprague-Dawley rats, at a dose level of 18.6 mg/L (nominal concentration), and exposure duration of one hour. Signs of toxicity during and up to 14 days after the exposure period included: excessive salivation, excessive lacrimation, decreased activity, labored breathing, gasping, closed eyes, mucoid nasal discharge, irregular breathing, red nasal discharge, yellow staining of the anogenital fur, dry and moist rales, red material around the eyes, and body tremors. Upon necropsy, lung discoloration was observed in a higher than normal incidence of rats (8/10). Based on the study results, the test substance was not fatal to rats at a nominal exposure concentration of 18.6 mg/L and exposure duration of one hour.

The acute dermal toxicity of APFO was tested in male and female rabbits, at a dose level of 2,000 mg/kg, and a 24-hour exposure period. Dermal irritation consisted of slight to moderate erythema, edema, and atonia; slight desquamation; coriaceousness; and fissuring. No visible lesions were observed upon necropsy. The dermal LD_{50} in rabbits was determined to be greater than 2,000 mg/kg.

APFO did not induce mutation in either *S. typhimurium* or *E. coli* when tested either with or without mammalian activation and did not induce chromosomal aberrations in human lymphocytes also when tested with and without metabolic activation up to cytotoxic concentrations. It was recently reported that APFO did not induce gene mutation when tested with or without metabolic activation in the K-1 line of Chinese hamster ovary (CHO) cells in culture.

APFO was tested twice for its ability to induce chromosomal aberrations in CHO cells. In the first assay, APFO induced both chromosomal aberrations and polyploidy in both the presence and absence of metabolic activation. In the second assay, no significant increases in chromosomal aberrations were observed without activation. However, when tested with metabolic activation, APFO induced significant increases in chromosomal aberrations and in polyploidy.

APFO was tested in a cell transformation and cytotoxicity assay conducted in C_3H $10T_{1/2}$ mouse embryo fibroblasts. The cell transformation was determined as both colony transformation and foci transformation potential. There was no evidence of

transformation at any of the dose levels tested in either the colony or foci assay methods.

Subchronic toxicity studies have been conducted in rats, mice, and Rhesus and Cynomolgus monkeys. A range-finding and a 6-month toxicity study in Cynomolgus monkeys was recently conducted. In all species, the liver is the main target organ. In rats, males had more pronounced hepatotoxicity and histopathologic effects than females, presumably because of the gender difference in elimination of APFO. Subchronic studies in rats and mice with 28 and 90 days of exposure have demonstrated that the liver is the primary target organ and that males are far more sensitive than females due to the gender differences in elimination. In a 90-day study with rhesus monkeys, exposure to doses of 30 mg/kg/day or higher resulted in death, lipid depletion in the adrenals, hypocellularity of the bone marrow, and moderate atrophy of the lymphoid follicles in the spleen and lymph nodes. Chronic dietary exposure of rats to 300 ppm APFO (14.2 and 16.1 mg/kg/day for males and females, respectively) for 2 years resulted in increased liver and kidney weights, hematological effects, and liver lesions in males and females. In addition, testicular masses were observed in males at 300 ppm and ovarian tubular hyperplasia was observed in females after exposure to 30 ppm (1.6 mg/kg/day), the lowest dose tested.

PFOA is immunotoxic in mice. Feeding the mice a diet of 0.02% PFOA resulted in adverse effects to both the thymus and spleen. Other effects included suppression of the specific humoral immune response to horse red blood cells, and suppression of the splenic lymphocyte proliferation in response to lipopolysacccharide (LPS) and concanavalin A (ConA). Studies using transgenic mice indicated that the peroxisome proliferator-activated receptor was involved in causing the adverse effects to the immune system.

Several prenatal developmental toxicity studies of APFO, including two oral studies in rats, one oral study in rabbits, and one inhalation study in rats, have been conducted. In one study, time-mated Sprague-Dawley rats (22 per group) were administered doses of 0, 0.05, 1.5, 5, and 150 mg/kg/day APFO in distilled water by gavage on gestation days (GD) 6–15. Signs of maternal toxicity consisted of statistically significant reductions in mean maternal body weights at the high-dose group of 150 mg/kg/day. Other signs of toxicity that occurred only at the high dose group included ataxia and death in three rat dams. No other effects were reported. Administration of APFO during gestation did not appear to affect the ovaries or reproductive tract of the dams. Under the conditions of the study, a NOAEL of 5 mg/kg/day and a LOAEL of 150 mg/kg/day for maternal toxicity were indicated. No significant differences between treated and control groups were noted for developmental parameters. A fetal lens finding initially described as a variety of abnormal morphological changes localized to the area of the embryonal nucleus, was later determined to be an artifact of the free-hand sectioning technique and therefore not considered to be treatment-related. Under the conditions of the study, a NOAEL for developmental toxicity of 150 mg/kg/day was indicated.

Another developmental study was also conducted on APFO. The study design consisted of an inhalation and an oral portion, each with two trials or experiments. In the first trial the dams were sacrificed on GD 21; while in the second trial, the dams were allowed to litter and the pups were sacrificed on day 35-post partum. For the inhalation portion of the study, the two trials consisted of 12 pregnant Sprague-Dawley rats per group exposed to 0, 0.1, 1, 10, and 25 mg/m^3 APFO for 6 hours/day, on GD 6–15. In the oral portion of the study, 25 and 12 Sprague-Dawley rats for the first and second trials, respectively, were administered 0 and 100 mg/kg/day APFO in corn oil by gavage on GD 6–15.

In trial one of the inhalation study, treatment-related clinical signs of maternal toxicity occurred at 10 and 25 mg/m^3 and consisted of wet abdomens, chromodacryorrhea, chromorhinorrhea, a general unkempt appearance, and lethargy in four dams at the end of the exposure period (high-concentration group only). Three out of 12 dams died during treatment at 25 mg/m^3 (on GD 12, 13, and 17). Food consumption was significantly reduced at both 10 and 25 mg/m^3. Significant reductions in body weight were also observed at these concentrations, with statistical significance at the high-concentration only. Likewise, statistically significant increases in mean liver weights were seen at the high-concentration group. The NOAEL and LOAEL for maternal toxicity were 1 and 10 mg/m^3, respectively. Similar effects were seen in trial two and the NOAEL and LOAEL for maternal toxicity were the same in both trials.

No effects were observed on the maintenance of pregnancy or the incidence of resorptions. Mean fetal body weights were significantly decreased in the 25 mg/m^3 groups and in the control group pair-fed 25 mg/m^3. However, interpretation of the decreased fetal body weight is difficult given the high incidence of mortality in the dams. Under EPA guidance, data at doses exceeding 10% mortality are generally discounted. Under the conditions of the study, a NOAEL and LOAEL for developmental toxicity of 10 and 25 mg/m^3, respectively, were indicated. Similar effects were seen in trial two and the same NOAEL and LOAEL were noted.

In trial one of the oral study, three out of 25 dams died during treatment of 100 mg/kg APFO during gestation (one death on GD 11; two on GD 12). Clinical signs of maternal toxicity in the dams that died were similar to those seen with inhalation exposure. Food consumption and body weights were reduced in treated animals compared to controls. No adverse signs of toxicity were noted for any of the reproductive parameters such as maintenance of pregnancy or incidence of resorptions. Likewise, no significant differences between treated and control groups were noted for fetal weights, or in the incidences of malformations and variations; nor were there any effects noted following microscopic examination of the eyes. In trial two of the oral study, similar observations for clinical signs were noted for the dams as in trial one. Likewise, no adverse effects on reproductive performance or in any of the fetal observations were noted.

An oral two-generation reproductive toxicity study was conducted on APFO. Five groups of 30 Sprague-Dawley rats per sex per dose group were administered APFO by gavage at doses of 0, 1, 3, 10, and 30 mg/kg/day six weeks prior to and during mating. Treatment of the F0 male rats continued until mating was confirmed, and treatment of the F0 female rats continued throughout gestation, parturition, and lactation.

At necropsy, none of the sperm parameters evaluated (sperm number, motility, or morphology) were affected by treatment at any dose level. One F0 male rat in the 30 mg/kg/day dose group was sacrificed on day 45 of the study due to adverse clinical signs (emaciation, cold-to-touch, and decreased motor activity). Necroscopic examination in that animal revealed a pale and tan liver, and red testes. All other F0 generation male rats survived to scheduled sacrifice. Statistically significant increases in clinical signs were also observed in male rats in the high-dose group that included dehydration, urine-stained abdominal fur, and ungroomed coat. No treatment-related effects were reported at any dose

level for any of the mating and fertility parameters assessed. At necropsy, none of the sperm parameters evaluated (sperm number, motility, or morphology) were affected by treatment at any dose level.

At necropsy, statistically significant reductions in terminal body weights were seen at 3, 10, and 30 mg/kg/day. Absolute weights of the left and right epididymides, left cauda epididymis, seminal vesicles (with and without fluid), prostate, pituitary, left and right adrenals, spleen, and thymus were also significantly reduced at 30 mg/kg/day. The absolute weight of the seminal vesicles without fluid was significantly reduced in the 10 mg/kg/day dose group. The absolute weight of the liver was significantly increased in all dose-groups. Kidney weights were significantly increased in the 1, 3, and 10 mg/kg/day dose groups, but significantly decreased in the 30 mg/kg/day group. All organ weight-to-terminal body weight and ratios were significantly increased in all treated groups. Organ weight-to-brain weight ratios were significantly reduced for some organs at the high dose group, and significantly increased for other organs among all treated groups.

No treatment-related effects were seen at necropsy or upon microscopic examination of the reproductive organs, with the exception of increased thickness and prominence of the zona glomerulosa and vacuolation of the cells of the adrenal cortex in the 10 and 30 mg/kg/day dose groups. No treatment-related deaths or adverse clinical signs were reported in parental females at any dose level. No treatment-related effects were reported for body weights, body weight gains, and absolute and relative food consumption values.

There were no treatment-related effects on estrous cyclicity, mating or fertility parameters. None of the natural delivery and litter observations were affected by treatment. Necropsy and histopathological evaluation were also unremarkable. Terminal body weights, organ weights, and organ-to-terminal body weight ratios were comparable to control values for all treated groups, except for kidney and liver weights. The weights of the left and right kidney, and the ratios of these organ weights-to-terminal body weight and of the left kidney weight-to-brain weight were significantly reduced at the highest dose of 30 mg/kg/day. The ratio of liver weights-to-terminal body weight was also significantly reduced at 3 and 10 mg/kg/day.

No effects were reported at any dose level for the viability and lactation indices of F1 pups. No differences between treated and control groups were noted for the numbers of pups surviving per litter, the percentage of male pups, litter size and average pup body weight per litter at birth. Pup body weight on a per litter basis (sexes combined) was reduced in the 30 mg/kg/day group throughout lactation, and statistical significance was achieved on days 1, 5, and 8.

At 30 mg/kg/day, one pup from one dam died prior to weaning on lactation day 1 (LD1). Additionally, on lactation days 6 and 8, statistically significant increases in the numbers of pups found dead were observed at 3 and 30 mg/kg/day. According to the study authors, this was not considered to be treatment related because they did not occur in a dose-related manner and did not appear to affect any other measures of pup viability including numbers of surviving pups per litter and live litter size at weighing. An independent statistical analysis was conducted by EPA. No significant differences were observed between dose groups and the response did not have any trend in dose.

Of the pups necropsied at weaning, no statistically significant, treatment-related differences were observed for the weights of the brain, spleen, and thymus and the ratios of these organ weights to the terminal body weight and brain weight.

No treatment-related adverse clinical signs were observed at any dose level in F2 generation offspring. No treatment-related adverse clinical signs were observed at any dose level. Likewise, no treatment-related effects were reported following necroscopic examination, with the exception of no milk in the stomach of the pups that were found dead. The numbers of pups found either dead or stillborn did not show a dose-response (3/28, 6/28, 10/28, 10/28, and 6/28 in 0, 1, 3, 10, and 30 mg/kg/day dose groups, respectively) and therefore were unlikely related to treatment.

No effects were reported at any dose level for the viability and lactation indices. No differences between treated and control groups were noted for the numbers of pups surviving per litter, the percentage of male pups, litter size, and average pup body weight per litter when measured on LDs 1, 5, 8, 15, or 22. Anogenital distances measured for F2 male and female pups on LDs 1 and 22 were also comparable among the five dosage groups and did not differ significantly. Likewise, no treatment-related effects were reported following necroscopic examination, with the exception of no milk in the stomach of the pups that were found dead. The numbers of pups found either dead or stillborn did not show a dose-response (3/28, 6/28, 10/28, 10/28, and 6/28 in 0, 1, 3, 10, and 30 mg/kg/day dose groups, respectively) and therefore were unlikely related to treatment.

No effects were reported at any dose level for the viability and lactation indices. No differences between treated and control groups were noted for the numbers of pups surviving per litter, the percentage of male pups, litter size, and average pup body weight per litter when measured. Statistically significant increases ($p \leq 0.01$) in the number of pups found dead were observed on lactation day 1 in the 3 and 10 mg/kg/day groups. According to the study authors, this was not considered to be treatment related because they did not occur in a dose-related manner and did not appear to affect any other measures of pup viability including numbers of surviving pups per litter and live litter size at weighing. An independent statistical analysis was conducted by EPA. No significant differences were observed between dose groups and the response did not have any trend in dose. Terminal body weights in F2 pups were not significantly different from controls. Absolute weights of the brain, spleen, and thymus and the ratios of these organ weights-to-terminal body weight and to brain weight were also comparable among treated and control groups.

In summary, under the conditions of the study, the LOAEL for F0 parental males is considered to be 1 mg/kg/day, the lowest dose tested, based on significant increases in the liver and kidney weights-to-terminal body weight and to brain weight ratios. A NOAEL for the F0 parental males could not be determined since treatment-related effects were seen at all doses tested. The NOAEL and LOAEL for F0 parental females are considered to be 10 and 30 mg/kg/day, respectively, based on significant reductions in kidney weight and kidney weight-to-terminal body weight and to brain weight ratios observed at the highest dose.

The LOAEL for F1 generation males is considered to be 1 mg/kg/day, based on significant decreases in body weights and body weight gains, and in terminal body weights; and significant changes in absolute liver and spleen weights and in the ratios of liver, kidney, and spleen weights-to-brain weights; and based on significant, dose-related reductions in body weights and body weight gains observed prior to and during cohabitation and during the entire dosing period. A NOAEL for the F1 males could not be determined since treatment-related effects were seen at all doses tested.

The NOAEL and LOAEL for F1 generation females are considered to be

10 and 30 mg/kg/day, respectively, based on statistically significant increases in postweaning mortality, delays in sexual maturation (time to vaginal patency), decreases in body weight and body weight gains, and decreases in absolute food consumption, all observed at the highest dose tested. The NOAEL for the F2 generation offspring was considered to be 30 mg/kg/day. No treatment-related effects were observed at any doses tested in the study. However, it should be noted that the F2 pups were sacrificed at weaning, and thus it was not possible to ascertain the potential post-weaning effects that were noted in the F1 generation.

Carcinogenicity studies in CD rats show that APFO is weakly carcinogenic, inducing Leydig cell tumors in the male rats and mammary tumors in the females. The compound has also been reported to be carcinogenic to the liver and pancreas of male CD rats. The mechanism(s) of APFO tumorigenesis is not clearly understood. APFO is not mutagenic. Available data indicate that the induction of tumors by APFO is due to a non-genotoxic mechanism, involving activation of receptors and perturbations of the endocrine system. There is sufficient evidence to suggest that APFO is a PPARα-agonist and that the liver carcinogenicity/toxicity of APFO is mediated by binding to PPARα in the liver. The Agency is currently examining the scientific knowledge associated with PPARα-agonist-induced liver tumors in rodents and the relevance to humans. Available data suggest that the induction of Leydig cell tumors (LCT) and mammary gland neoplasms by APFO may be due to hormonal imbalance resulting from activation of the PPARα and induction of the cytochrome P450 enzyme, aromatase. Preliminary data suggest that the pancreatic acinar cell tumors are related to an increase in serum level of the growth factor, cholecystokinin.

There are limited data on PFOA serum levels in workers and the general population. Occupational data from plants in the United States and Belgium that manufacture or use PFOA indicate that mean serum levels in workers range from 0.84 to 6.4 ppm. In non-occupational populations, serum PFOA levels were much lower; in both pooled blood bank samples and in individual samples, mean serum PFOA levels ranged from 3 to 17 ppb. The highest serum PFOA levels were reported in a sample of children from different geographic regions in the United States (range, 1.9 to 56.1 ppb).

Several wildlife species have been sampled to determine levels of PFOA. PFOA has rarely been found in fish or in fish-eating bird samples collected from around the world. PFOA was found in a few mink livers from Massachusetts, but not found in mink from Louisiana, South Carolina, and Illinois. PFOA concentrations in river otter livers from Washington and Oregon were less than the quantification limit of 36 ng/g, wet wt. PFOA was not detected at quantifiable concentrations in oysters collected in the Chesapeake Bay and Gulf of Mexico.

E. Summary of Data on Fluorotelomers and Other Perfluoroalkyl Moieties

EPA has concerns about the potential health and environmental effects of polymers containing fluorotelomers or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule. The Agency believes that polymers containing such substances should be subject to the premanufacture review process so that EPA can better evaluate and address these concerns. In 1981, the first reports of fluorotelomer alcohol metabolism were reported and clearly showed that PFOA was formed from the 8–2 alcohol (Ref. 8). In more recent research published by 3M and in similar tests reported by the Telomer Research Program (TRP), 8–2 alcohol has been shown to degrade to form PFOA when exposed to activated sludge during accelerated biodegradation studies. A single mechanism had been proposed for the conversion of the 8–2 alcohol to form PFOA, whether through metabolic reaction or environmental degradation. Each intermediate in the stepwise sequence of chemical reactions has been identified confirming the proposed mechanism (Ref. 47 and 48).

In addition, initial test data from a study in rats dosed with fluorotelomer alcohol and other preliminary animal studies on various telomeric products containing fluorocarbons structurally similar to PFAC or PFAS have demonstrated a variety of adverse effects including liver, kidney, and thyroid effects (Ref. 9).

Canadian researchers have developed an analytical methodology to measure airborne organo-fluorine compounds (Ref. 49). Using this technique, the researchers monitored air samples in Toronto and were successful in detecting fluoroorganics, including PFOS derivatives and fluorotelomer alcohols. DuPont commissioned a preliminary study in North America by these same researchers and found similar results in six different U.S. and Canadian cities (Ref. 10). While these studies are only preliminary and certainly not conclusive, the fact that the Canadian researchers found fluorotelomer alcohols in the air in six different cities is significant. This finding is indicative of widespread fluorotelomer alcohol distribution, and it further indicates that air may be a route of exposure to these chemicals, which can ultimately become PFOA. The TRP, in developing radiolabeled 8–2 alcohol, noted the volatile nature of this material and the rampant loss of non-radio labeled material attributed to a high vapor pressure (Ref. 50).

Although the source of the fluorotelomer alcohols cannot be determined from the study, most (85% of the production volume) fluorotelomer alcohols produced are used in the manufacture of high molecular weight polymers. These fluorotelomer alcohols are generally incorporated into the polymers via covalent ester linkages, and it is possible that degradation of the polymers may result in release of the fluorotelomer alcohols to the environment. This hypothesis has been posed to TRP, which has begun to investigate whether fluorotelomer-based polymers may be a source of PFOA in the environment (Ref. 51).

Based on the presence of fluorotelomer alcohols in the air, the growing data demonstrating that fluorotelomer alcohols metabolize or degrade to generate PFOA (Ref. 11), the demonstrated toxicity of 8–2 alcohol and certain compounds containing fluorotelomers, and the possibility that polymers containing fluorotelomers could degrade in the environment thereby releasing fluorotelomer alcohols or other perfluoroalkyl-containing substances, EPA can no longer conclude that such polymers "will not present an unreasonable risk of injury to health or the environment" as required for an exemption under section 5(h)(4) of TSCA. Therefore, EPA is proposing to exclude polymers that contain fluorotelomers as an integral part of their composition, except as impurities, from the polymer exemption at 40 CFR 723.250.

Similarly, EPA does not have specific data demonstrating that polymers containing perfluoroalkyl moieties other than PFAS, PFAC, or fluorotelomers present the same concerns as those containing PFAS, PFAC, or fluorotelomers. Nevertheless, EPA is also proposing to exclude polymers containing perfluoroalkyl moieties, consisting of a CF3- or longer chain length, that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule from the polymer exemption. Available data indicate that compounds containing

PFAS or PFAC may degrade in the environment thereby releasing the PFAS or PFAC moiety, and that fluorotelomers may degrade in the environment to form PFAC. Based on these data, EPA believes that it is possible that polymers containing these other types of perfluoroalkyl moieties could also degrade over time in the environment, thereby releasing the perfluoroalkyl moiety. EPA also believes that once released, such moieties may potentially degrade to form PFAS or PFAC. EPA does not believe, therefore, that it can continue to make the "will not present an unreasonable risk of injury to health or the environment" finding for such polymers and is proposing to exclude them from the polymer exemption. EPA is specifically requesting comment on this aspect of the proposed rule. Please see Unit VII. of this document for specific information that EPA is interested in obtaining to evaluate whether continued exemption for polymers containing fluorotelomers or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule is appropriate.

V. Objectives and Rationale for This Proposed Rule

The objective of this proposed rule is to amend the polymer exemption rule to exclude polymers containing as an integral part of the polymer composition, except as impurities, any one or more of certain perfluroalkyl moieties consisting of a CF3- or longer chain length from eligibility for the exemption from TSCA section 5 reporting requirements allowed under the 1995 amendments to the polymer exemption rule. In section 5(a)(1)(A) of TSCA, Congress prohibited persons from manufacturing (including importing) new chemical substances unless such persons submitted a PMN to EPA at least 90 days before such manufacture. Pursuant to section 5(h)(4) of TSCA, EPA is authorized to exempt the manufacturer of any new chemical substance from all or part of the requirements of section 5 if the Agency determines that the manufacture, processing, distribution in commerce, use, or disposal of the substance, or any combination of such activities, will not present an unreasonable risk of injury to health or the environment. Section 5(h)(4) also authorizes EPA to amend or repeal such rules.

While TSCA does not contain a definition of unreasonable risk, the legislative history indicates that the determination of unreasonable risk requires a balancing of the considerations of both the severity and probability that harm will occur against the effect of the final regulatory action on the availability to society of the benefits of the chemical substance. [House Report 1341, 94th Cong. 2nd Session, 14 (1976)]. This analysis can include an estimate of factors such as market potential, the effect of the regulation on promoting or hindering the economic appeal of a substance, environmental effects, and many other factors that are difficult to define and quantify with precision. In making a determination of unreasonable risk, EPA must rely not only on available data, but also on its professional judgment. Congress recognized that the implementation of the unreasonable risk standard "will vary on the specific regulatory authority which the Administrator seeks to exercise."

The polymer exemption rule is intended to exempt from certain section 5 requirements polymers that EPA believes pose a low risk of injury to health or the environment. The exemption criteria are therefore designed to exempt polymers that are of low concern because of their stability, molecular size, and lack of reactivity, among other properties. In contrast, EPA has excluded certain polymers from the exemption where:

- The Agency has insufficient data and review experience to support a finding that they will not present an unreasonable risk. Or
- The Agency has found that under certain conditions, the polymers may present risks which require a closer examination of the conditions of manufacturing, processing, distribution, use, and disposal during a full 90-day PMN review (i.e., the Agency has information suggesting that the conditions for an exemption under section 5(h)(4) are not met).

This approach allows the Agency to maintain full regulatory oversight on potentially higher risk polymers while promoting the manufacture of low-risk polymers.

Based on the data currently available, EPA believes, for the reasons that follow it no longer can make a generally-applicable finding, without additional information, that the manufacture, processing, distribution in commerce, use, and/or disposal of polymers containing certain perfluoroalkyl moieties consisting of a CF3- or longer chain length will not present an unreasonable risk of injury to health or the environment. This exclusion includes polymers that contain any one or more of the following: PFAS; PFAC; fluorotelomers; or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule. To the contrary, EPA believes that the risks presented by such polymers should be evaluated during the 90-day PMN review period that Congress contemplated for new chemicals under section 5(a)(1)(A) of TSCA.

First, PFOS and PFOA, which are members of the PFAS and PFAC category of chemicals as defined in Unit IV.B., have a high level of toxicity and have shown liver, developmental, and reproductive toxicity at very low dose levels in exposed laboratory animals. The primary health effects of concern for PFOS, based on available data, are liver effects, developmental effects, and mortality. The mortality is associated with a steep dose/response across all ages and species. The primary health effects of concern for PFOA are liver toxicity and developmental toxicity. The health effects of PFOS and PFOA are discussed more fully in Unit IV.D.5. With regard to fluorotelomers, it has been demonstrated that the fluorotelomer 8–2 alcohol can be converted to PFOA through metabolic reaction and environmental degradation. Moreover, initial test data from a study in rats dosed with fluorotelomer alcohol and other preliminary animal studies on various telomeric products containing fluorocarbons structurally similar to PFAC or PFAS have demonstrated a variety of toxic effects. With regard to polymers containing perfluoroalkyl moieties other than PFAS, PFAC, or fluorotelomers that would be subject to the rule, EPA does not have specific data demonstrating that such polymers present the same concerns as those containing PFAS, PFAC, or fluorotelomers. Nonetheless, based on available data which indicates that compounds containing PFAS or PFAC may degrade in the environment thereby releasing the PFAS or PFAC moiety, and that fluorotelomers may degrade in the environment to form PFAC, EPA believes that it is possible for polymers containing perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule to also degrade over time in the environment thereby releasing the perfluoroalkyl moiety. EPA also believes that once released, such moieties may potentially degrade to form PFAS or PFAC.

Second, PFOS and PFOA are expected to persist in the environment and they may bioaccumulate. These chemicals are stable to hydrolysis, appear to be stable to photolysis, and do not

measurably biodegrade in the environment. PFOS and PFOA have been found in the blood of workers exposed to the chemicals and in the general population of the United States and other countries. They have also been found in many terrestrial and animal species worldwide. The widespread distribution of the chemicals suggests that PFOS and PFOA may bioaccumulate. Exposure and environmental fate data are discussed more fully in Unit IV.D.3. and Unit IV.D.4. respectively. EPA has also received preliminary data that indicates that certain perfluoroalkyl compounds including fluorotelomer alcohols are present in the air in some large cities. These preliminary data suggest that there may be widespread distribution of fluorotelomer alcohols and that air may be a possible route of exposure to such chemicals.

Third, although the Agency has far more data on PFOS and PFOA than on other PFAS and PFAC chemicals, EPA believes that other PFAS and PFAC chemicals may share similar toxicity, persistence and bioaccumulation characteristics. Based on currently available information, EPA believes that, while all PFAS and PFAC chemicals are expected to persist, the length of the perfluorinated chain may have an effect on the other areas of concern for these chemicals. In particular, there is some evidence that PFAS/PFAC moieties with longer carbon chains may present greater concerns for bioaccumulation potential and toxicity than PFAS/PFAC moieties with shorter carbon chains. (Refs. 5, 6, and 7).

Fourth, EPA has evidence that polymers containing PFAS or PFAC may degrade, possibly by incomplete incineration, and release these perfluorinated chemicals into the environment (Ref. 3). Even under routine conditions of municipal waste incinerators, the Agency believes that the PFAS and PFAC produced by oxidative thermal decomposition of the polymers will remain intact (the typical conditions of a MWI are not stringent enough to cleave the carbon-fluorine bonds) to be released into the environment. It has also been demonstrated that PFAS or PFAC-containing compounds may undergo degradation (chemical, microbial, or photolytic) of the non-fluorinated portion of the molecule leaving the remaining perfluorinated acid untouched (Ref. 2). The Agency further anticipates that a carpet treated with a stain resistant polymer coating containing fluorochemicals would be exposed to conditions over time that could lead to the release of chemical substances which may biodegrade to form PFAC. Further degradation of the PFAC degradation product is extremely difficult. This possibility is consistent with the previously cited degradation studies.

As discussed in Unit II.C.2, EPA does not have specific data demonstrating that perfluoroalkyl moieties other than PFAS, PFAC, or fluorotelomers that would be subject to the rule present the same concerns as PFAS, PFAC, or fluorotelomers. EPA is nevertheless proposing to exclude polymers containing perfluoroalkyl moieties consisting of a CF3- or longer chain length that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule from the polymer exemption. Based on the data summarized in Unit V., EPA believes that it is possible for polymers containing these perfluoroalkyl moieties to degrade in the environment thereby releasing the perfluoroalkyl moiety. EPA also believes that once released, such moieties may potentially degrade to form PFAS or PFAC. EPA believes therefore, that polymers containing these perfluoroalkyl moieties should be evaluated for potential health or environmental concerns through the PMN process.

Efforts are currently underway to develop a better understanding of the environmental fate, bioaccumulation potential, and human and environmental toxicity of PFAS and PFAC chemicals as well as fluorotelomers and other perfluoroalkyl moieties. EPA has insufficient evidence at this time, however, to definitively establish a carbon chain length at which PFAS, PFAC, fluorotelomers, or other perfluoroalkyl moieties that would be subject to the rule will not present an unreasonable risk of injury to health or the environment, which is the determination necessary to support an exemption under section 5(h)(4) of TSCA. Therefore, EPA believes it is reasonable to exclude from the polymer exemption rule polymers containing as an integral part of their composition, except as impurities, certain perfluoroalkyl moieties consisting of a CF3- or longer chain length. This exclusion includes polymers that contain any one or more of the following: PFAS; PFAC; fluorotelomers; or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule.

VI. Other Options Considered

A. Exclude Polymers Containing PFAS, PFAC, Fluorotelomers, or Perfluoroalkyl Moieties That Are Covalently Bound to Either a Carbon or Sulfur Atom Where the Carbon or Sulfur Atom is an Integral Part of the Polymer Molecule, But Only if These Perfluoroalkyl Moieties Contain Greater Than Four Carbon Atoms

This option would allow an exemption for polymers containing PFAS, PFAC, fluorotelomers, or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule, where the perfluoroalkyl moiety contains fewer than five carbon atoms. This option was rejected because, based on available information, EPA cannot continue to find that such polymers "will not present an unreasonable risk to human health and the environment." EPA will continue to evaluate whether exemptions for polymers containing PFAS, PFAC, fluorotelomers, or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule with smaller chain lengths in the perfluoroalkyl moiety are appropriate for future exemption under the polymer exemption rule.

B. Make the Scope of This Proposed Rule Consistent With the SNURs on Perfluorooctyl Sulfonates (67 FR 11007; March 11, 2002 and 67 FR 72854; December 9, 2002)

These two SNURs cover perfluorooctanesulfonic acid (PFOSH) and certain of its salts (PFOSS), perfluorooctanesulfonyl fluoride (POSF), certain higher and lower homologues of PFOSH and POSF, and certain other chemical substances, including polymers, that are derived from PFOSH and its homologues. These chemicals are collectively referred to as perfluoroalkyl sulfonates, or PFAS. Today's proposed rule would exclude from eligibility polymers containing as an integral part of their composition, except as impurities, certain perfluoroalkyl moieties consisting of a CF3- or longer chain length. This exclusion includes polymers that contain any one or more of the following: PFAS; PFAC; fluorotelomers; or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule. Therefore, if the proposed rule were to be made consistent with the SNURs, only PFAS-containing polymers

would be excluded from the polymer exemption rule. This option would have continued to allow exemption under the polymer exemption rule for polymers containing:

- PFAS that are not specifically derived from PFOSH (specifically, the C4 to C10 carbon chain lengths addressed in the SNUR).
- PFAC; fluorotelomers; or other perfluoroalkyl moieties, for which EPA cannot make a "will not present an unreasonable risk to human health or the environment" finding.

C. Exclude From Exemption PFAS (and Not PFAC) Containing Any Number of Carbon Atoms Deemed Appropriate

This option was rejected because although it would remove polymers containing PFAS from exemption under the polymer exemption rule, it would have continued to allow exemption for polymers containing PFAC, for which EPA cannot make a "will not present an unreasonable risk to human health or the environment" finding. This option could also encourage companies to use these chemicals as substitutes for PFOS.

D. Exclude From Exemption All Fluorine-containing Polymers

This option would have excluded from exemption under the polymer exemption rule all fluorine-containing polymers. This option was rejected because EPA does not believe, based on the best available data, that all polymers containing fluorine present concerns that would justify excluding them from the exemption. EPA will continue to evaluate whether exemption for fluorine-containing polymers is appropriate under the polymer exemption rule.

VII. Request for Comment on Specific Issues

EPA is requesting specific responses to the following:

- Is exemption for polymers containing perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule and where the perfluoroalkyl moiety consists of a CF3- or longer chain length appropriate under the polymer exemption rule?

The Agency is looking for information showing whether or not polymers containing such substances degrade and release fluorochemical residual compounds into the environment, and information concerning the toxicity and bioaccumulation potential of such known or possible fluorochemical breakdown products.

In particular, the Agency is also looking for information showing whether such polymers containing perfluoroalkyl moieties with smaller chain lengths (i.e., less than 8 carbons) can degrade and release fluorochemical residual compounds into the environment. If degradation is shown to occur, the Agency would then want information indicating whether once released, these compounds exhibit characteristics similar to PFOS or PFOA in terms of persistence, bioaccumulation, or toxicity, or otherwise exhibit characteristics of potential concern.

- Those who are manufacturing or importing polymers under the existing exemption would have one year from the effective date to complete the PMN process. EPA is specifically requesting comment on this or other alternatives for implementing the final rule that would achieve the purposes of TSCA section 5 without disrupting ongoing manufacture or import of currently-exempt polymers.

VIII. Economic Considerations

EPA has evaluated the potential costs of eliminating the polymer exemption for the chemicals described in this proposal. The results of this evaluation are contained in a document entitled "Economic Analysis of the Amendment of the Polymer Exemption Rule To Exclude Certain Perfluorinated Polymers" (Ref. 54). A copy of this economic analysis is available in the public docket for this action, and is briefly summarized here.

As a result of the elimination of the polymer exemption for the chemicals described in this proposal, any person who intends to manufacture (defined by statute to include import) any of these polymers, which are not already on the TSCA Inventory, would have to first complete the TSCA premanufacture review process prior to commencing the manufacture or import of such polymers. Any person who relied on the exemption in the past and currently manufactures an affected polymer would have to complete the TSCA premanufacture review process to continue the manufacture of such polymers after the effective date of the final rule. In order to provide an opportunity for these existing manufacturers to complete the PMN process without disrupting their manufacture of the affected polymers, the Agency is seeking comment on approaches for structuring a delayed effective date or phase in period for the amendment. For purposes of this analysis, the Agency assumes that existing manufacturers will complete the PMN process within the first year after the effective date of the final rule.

The industry costs for completing and submitting a PMN reporting form are estimated to be $7,267 per chemical. Because the proposed rule would eliminate the cost of complying with the recordkeeping and reporting requirements of the Polymer Exemption Rule, the cost for completing and submitting a PMN as a result of this proposed amendment can be reduced by $308, for a net cost of $6,959 per chemical.

Companies that currently manufacture an affected polymer are estimated to incur a total cost of $6,959 per chemical. Companies that do not currently manufacture an affected polymer, but begin to manufacture such polymers in the future, may also incur potential costs of $19,416 associated with potential delays in commercialization of the new chemical. These companies are estimated to incur a total cost of $26,375 per chemical as a result of this rulemaking (Ref. 52).

The potential number of PMNs that may be submitted each year if the proposed rule is finalized was estimated using the 200 polymer reports received annually under the polymer exemption rule. EPA estimates that this proposal might affect a maximum of six percent of the 200 polymers reported annually, and therefore estimates that a maximum of 12 PMNs may be submitted each year if the proposed rule is finalized. Using the same estimated number of 12 chemicals per year for the 10 years that affected polymers were exempt from PMN requirements under the polymer exemption rule, EPA estimates that a maximum of 120 previously exempt chemicals (12 chemicals x 10 years) could be expected to complete and submit a PMN under the final rule. Thus, the Agency estimates that a maximum of 132 PMNs might be submitted during the first year after the effective date of the final rule, and that a maximum of 12 PMNs might be submitted each subsequent year (Ref. 53).

Using the estimated per chemical costs and the estimated number of PMNs anticipated, EPA estimates the potential impact of this proposal on industry to be a total annual costs for existing manufacturers of $835,080 ($6,959 per chemical costs x 120 chemicals), and a total annual cost for new manufacturers of $316,500 ($26,375 per chemical costs x 12). The total annual potential industry compliance costs of the proposed rule in the first year is estimated to be $1,151,580, which will decrease to an estimated

annual cost of $316,500 in subsequent years.

In addition, as was the case prior to the promulgation of the polymer exemption rule in 1995, the Agency recognizes that the submission of a PMN may lead to other regulatory actions under TSCA, for example consent orders issued under TSCA section 5(e). Any such actions are highly dependent on the circumstances surrounding the individual PMN (e.g., available information and scientific understanding about the chemical and its risks at the time the PMN is being reviewed). Such potential actions and any costs associated with them would not be a direct result of the proposed amendments to the polymer exemption rule. Nevertheless, EPA believes it is informative to provide a brief discussion of the Agency's previous and ongoing regulatory activities with respect to potentially affected polymers.

IX. References

These references have been placed in the public docket that was established under docket ID number EPA–HQ–OPPTS–2002–0051 for this rulemaking as indicated under **ADDRESSES**. The public docket includes information considered by EPA in developing this proposed rule, including the documents listed below, which are physically located in the docket. In addition, interested parties should consult documents that are referenced in the documents that EPA has placed in the docket, regardless of whether these other documents are physically located in the docket. For assistance in locating documents that are referenced in documents that EPA has placed in the docket, but that are not physically located in the docket, please consult the technical person listed in **FOR FURTHER INFORMATION CONTACT**. Reference documents identified with an AR are cross-indexed to non-regulatory, publicly accessible information files maintained in the TSCA Nonconfidential Information Center. Copies of these documents can be obtained as described in **ADDRESSES**.

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X. Statutory and Executive Order Reviews

A. Regulatory Planning and Review

Pursuant to Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993), the Office of Management and Budget (OMB) has designated this proposed rule as a "significant regulatory action" under section 3(f) of the Executive Order because it may raise novel legal or policy issues arising out of legal mandates, the President's priorities, or the principles set forth in the Executive Order. This action was therefore submitted to OMB for review under this Executive Order, and any changes to this document made at the suggestion of OMB have been documented in the public docket for this rulemaking.

EPA has prepared an economic analysis of the potential impacts of this proposed revision to the polymer exemption rule. This economic analysis (Ref. 54) is available in the public docket for this action and is briefly summarized in Unit VIII.

B. Paperwork Reduction Act

The information collection requirements related to the submission of PMNs are already approved by the Office of Management and Budget (OMB) under the Paperwork Reduction Act, 44 U.S.C. 3501 *et seq.* That Information Collection Request (ICR) document has been assigned EPA ICR number 0574.12 and OMB control number 2070–0012. This proposed rule does not impose any new requirements that require additional OMB approval.

Under the PRA, "burden" means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This burden estimate includes the time needed to review instructions, search existing data sources, gather and maintain the data needed, and complete, review, and submit the required PMN, and maintain the required records.

Based on the estimated burden in the existing ICR, if an entity were to submit a PMN to the Agency, the annual reporting burden is estimated to average between 95 and 114 hours per response, with an midpoint respondent burden of 107 hours. This estimate was adjusted to account for the elimination of the existing burden related to the recordkeeping and reporting requirements in the polymer exemption rule, which is estimated to impose a burden on industry of six hours per chemical, i.e., two hours for reporting, and four hours for recordkeeping. The

net paperwork burden for submitting a PMN as a result of this proposed amendment is therefore estimated to be 101 hours per PMN submission. The burden hour cost for this proposed rule is estimated to be $4,459. In addition, PMN submissions must be accompanied by a user fee of $2,500 (set at $100 for small businesses with annuals sales of less than $40 million).

Based on the high-end assumption of 12 PMN submissions annually, the annual burden is estimated to be 1,212 hours (12 × 101 hours). The one-time burden for the companies that submit PMNs for chemicals already in production is estimated to be a maximum of 12,120 hours (120 chemicals x 101 hours per submission).

An agency may not conduct or sponsor, and a person is not required to respond to an information collection request subject to the PRA unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations in 40 CFR, after appearing in the preamble of the final rule, are listed in 40 CFR part 9 and included on any related collection instrument (e.g., on the form or survey).

Submit any comments on the Agency's need for this information, the accuracy of the provided burden estimates, and any suggested methods for minimizing respondent burden, including the use of automated collection techniques, along with your comments on the proposed rule as instructed under **ADDRESSES**. The Agency will consider any comments related to the information collection requirements contained in this proposal as it develops a final rule.

C. Regulatory Flexibility Act

Pursuant to section 605(b) of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*), the Agency hereby certifies that this proposed rule will not have a significant adverse economic impact on a substantial number of small entities.

For purposes of assessing the impacts of today's proposed rule on small entities, small entity is defined as:

- A small business as defined by the Small Business Administration's (SBA) regulations at 13 CFR 121.201 based on the applicable NAICS code for the business sector impacted.
- A small governmental jurisdiction that is a government of a city, county, town, school district or special district with a population of less than 50,000.
- A small organization that is any not-for-profit enterprise which is independently owned and operated and is not dominant in its field.

The regulated community does not include any small governmental jurisdictions or small not-for-profit organizations. For small businesses, the Agency assessed the impacts on small chemical manufacturers in NAICS codes 325 and 324110. The SBA size standards for sectors under NAICS 325 range from 500 to 1,000 employees or fewer in order to be classified as small. The size standard for NAICS code 324110, petroleum refineries, is 1,500 employees.

Based on estimates of the number of PMNs expected to be submitted as a result of this action, it appears that 12 or fewer businesses would be affected per year. The five companies that manufacture the majority of the volume of chemicals that will be affected by the polymer exemption rule belong to either or both of the Fluoropolymer Manufacturers Group, and the Telomer Research Program. These two groups, which have no other members beyond the five companies, are negotiating enforceable consent agreements and other voluntary testing arrangements with the Agency for testing specific chemicals that would be affected by the polymer exemption rule. The two groups have told the Agency that their member companies manufacture the majority of the volume of chemicals that would be affected by the rule. None of these five companies meet the definition of small under the Small Business Administration employee size criteria. The remaining volume of chemicals that could be affected by the rule is low enough so that even if a small company were to be affected, a significant number of businesses would not be affected, nor would any individual small business experience significant impacts. In addition to the estimated impact of having to submit a PMN (see estimates in Unit VIII.), small businesses with less than $40 million in annual sales are entitled to a reduced user fee of $100 for submitting a PMN, rather than the $2,500 user fee, which would further reduce any impacts of the rule on small businesses.

D. Unfunded Mandates Reform Act

Based on EPA's experience with past PMNs, State, local, and tribal governments have not been affected by this reporting requirement, and EPA does not have any reason to believe that any State, local, or tribal government will be affected by this rulemaking. As such, EPA has determined that this regulatory action does not impose any enforceable duty, contain any unfunded mandate, or otherwise have any affect on small governments subject to the requirements of sections 202, 203, 204, or 205 of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4).

E. Federalism

Pursuant to Executive Order 13132, entitled *Federalism* (64 FR 43255, August 10, 1999), EPA has determined that this proposed rule does not have "federalism implications," because it will not have substantial direct effects on the states, on the relationship between the national government and the states, or on the distribution of power and responsibilities among the various levels of government, as specified in the Order. Thus, Executive Order 13132 does not apply to this proposed rule.

F. Consultation and Coordination With Indian Tribal Governments

As required by Executive Order 13175, entitled *Consultation and Coordination with Indian Tribal Governments* (65 FR 67249, November 6, 2000), EPA has determined that this proposed rule does not have tribal implications because it will not have any affect on tribal governments, on the relationship between the Federal government and the Indian tribes, or on the distribution of power and responsibilities between the Federal government and Indian tribes, as specified in the Order. Thus, Executive Order 13175 does not apply to this proposed rule.

G. Protection of Children From Environmental Health and Safety Risks

Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997) does not apply to this proposed rule because this action is not designated as an "economically significant" regulatory action as defined by Executive Order 12866, nor does it establish an environmental standard, or otherwise have a disproportionate effect on children.

H. Actions That Significantly Affect Energy Supply, Distribution, or Use

This proposed rule is not subject to Executive Order 13211, entitled *Actions concerning Regulations that Significantly Affect Energy Supply, Distribution, or Use* (66 FR 28355, May 22, 2001) because it is not designated as an "economically significant" regulatory action as defined by Executive Order 12866, nor is it likely to have any significant adverse effect on the supply, distribution, or use of energy.

I. National Technology Transfer Advancement Act

Section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), 15 U.S.C. 272 note) directs EPA to use voluntary consensus standards in its regulatory activities unless to do so would be inconsistent with applicable law or impractical. Voluntary consensus standards are technical standards (e.g., materials specifications, test methods, sampling procedures, etc.) that are developed or adopted by voluntary consensus standards bodies. This proposed rule does not impose any technical standards that would require EPA to consider any voluntary consensus standards.

J. Environmental Justice

This proposed rule does not have an adverse impact on the environmental and health conditions in low-income and minority communities. Therefore, under Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994), the Agency does not need to consider environmental justice-related issues.

List of Subjects in 40 CFR Part 723

Environmental protection, Chemicals, Hazardous substances, Reporting and recordkeeping requirements.

Dated: February 8, 2006.

Susan B. Hazen,

Acting Assistant Administrator for Prevention, Pesticides and Toxics Substances.

Therefore, it is proposed that 40 CFR part 723 be amended as follows:

PART 723—[AMENDED]

1. The authority citation for part 723 would continue to read as follows:

Authority: 15 U.S.C. 2604.

2. Section 723.250 is amended as follows:

a. By adding several definitions in alphabetical order to paragraph (b).

b. By adding a paragraph (d)(6).

§ 723.250 Polymers.

* * * * *

(b) * * *

Fluorotelomers means the products of telomerization, the reaction of a telogen (such as pentafluoroethyl iodide) with an ethylenic compound (such as tetrafluoroethylene) to form low molecular weight polymeric compounds, which contain an array of saturated carbon atoms covalently bonded to each other (C-C bonds) and to fluorine atoms (C-F bonds). This array is predominantly a straight chain, and depending on the telogen used produces a compound having an even number of carbon atoms. However, the carbon chain length of the fluorotelomer varies widely. The perfluoroalkyl groups formed by this process are usually, but do not have to be, connected to the polymer through a functionalized ethylene group as indicated by the following structural diagram: ($Rf\text{-}CH_2\text{-}CH_2$-Anything).

Perfluororalkyl carboxylate (PFAC) means a group of saturated carbon atoms covalently bonded to each other in a linear, branched, or cyclic array and covalently bonded to a carbonyl moiety and where all carbon-hydrogen (C-H) bonds have been replaced with carbon-fluorine (C-F) bonds. The carbonyl moiety is also covalently bonded to a hetero atom, typically, but not necessarily oxygen (O) or nitrogen (N).

Perfluoroalkyl sulfonate (PFAS) means a group of saturated carbon atoms covalently bonded to each other in a linear, branched, or cyclic array and covalently bonded to a sulfonyl moiety and where all carbon - hydrogen (C-H) bonds have been replaced with carbon - fluorine (C-F) bonds. The sulfonyl moiety is also covalently bonded to a hetero atom, typically, but not necessarily oxygen (O) or nitrogen (N).

* * * * *

(d) * * *

(6) *Polymers which contain certain perfluoroalkyl moieties consisting of a CF3- or longer chain length.* After [*insert date 1 year after date of publication of the final rule in the* **Federal Register**] a polymer cannot be manufactured under this section if the polymer contains as an integral part of its composition, except as impurities, one or more of the following perfluoroalkyl moieties consisting of a CF3- or longer chain length: Perfluoroalkyl sulfonates (PFAS), perfluoroalkyl carboxylates (PFAC), fluorotelomers, or perfluoroalkyl moieties that are covalently bound to either a carbon or sulfur atom where the carbon or sulfur atom is an integral part of the polymer molecule.

(i) Except as provided in paragraph (d)(6)(ii) of this section, any polymer that is subject to paragraph (d)(6) of this section and that has been manufactured prior to [*insert date 1 year after date of publication of the final rule in the* **Federal Register**] may no longer be manufactured after [*insert date 1 year after date of publication of the final rule in the* **Federal Register**] unless that polymer has undergone a premanufacture review in accordance with section 5(a)(1)(A) of TSCA and 40 CFR part 720.

(ii) Paragraph (d)(6) of this section does not apply to polymers which are already on the list of chemical substances manufactured or processed in the United States that EPA compiles and keeps current under section 8(b) of TSCA.

* * * * *

[FR Doc. 06–2152 Filed 3–6–06; 8:45 am]

BILLING CODE 6560–50–S

Vermont Sets A Permanent Drinking Water Standard For PFOA

A legislative committee has permanently set Vermont's safe drinking water standard for the chemicals PFOA and PFOS at 20 parts per trillion.

Vermont's limit is far below the EPA's limit of 70 parts per trillion, and it is now one of the lowest drinking water standards in the country.

PFOA is a dangerous chemical that's been linked to thyroid disease, cancer, high cholesterol and endocrine issues, and it's been detected in drinking water in Bennington County.

It was used to make Teflon and other water-resistant materials.

When PFOA was found in the water in southwestern Vermont in February, very few people in the state had even heard of the chemical.

The state, at the time, set its safe drinking water standard at 20 parts per trillion under an emergency rule.

On Thursday, after months of hearings and a public comment period, the Legislative Committee on Administrative Rules permanently set the safety standard at 20 parts per trillion.

"I think this gives the people in Bennington County who are dealing with concerns related to PFOA a level of comfort," said Department of Environmental Conservation Commissioner Alyssa Schuren. "The rule is now set in stone, and there isn't a question about it any longer."

The contamination in Bennington has been linked to the former Chemfab plant, which was owned by Saint-Gobain before it moved in 2001.

In April, Saint-Gobain brought three law suits against the state challenging its low drinking water standard.

"While Vermont can set a PFOA limit, it is important that the State appropriately evaluates and properly applies the factors that go into setting any such regulatory standard," Saint Gobain spokeswoman Dina Silver Pokedoff said in a prepared statement. "That is why Saint-Gobain Performance Plastics filed in September an appeal of Vermont's emergency rule issued in August that sets the limit for PFOA at 20 ppt."

Two of the lawsuits have already been dismissed.

The other suit challenges the emergency rule and DEC attorney Matt Chapman says the state will look to dismiss those suits now that the standard has been adopted.

He said Saint-Gobain can now challenge the permanent rule if they choose to.

(4)

STATE OF NEW HAMPSHIRE
DEPARTMENT OF HEALTH AND HUMAN SERVICES
DIVISION OF PUBLIC HEALTH SERVICES

Jeffrey A. Meyers
Commissioner

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Testimony for HB 691-FN
Relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies
House Health and Human Services and Elderly Affairs Committee, Legislative Office Building Room 205
February 6, 2019

Good morning Madam Chair Weber and members of the committee. My name is Dr. Benjamin Chan, and I am the State Epidemiologist for the Department of Health and Human Services (DHHS), Division of Public Health Services. With me today is Dr. Chris Bean, Director of the Public Health Laboratories. We are here to provide information on HB 691-FN, relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies. This proposed legislation seeks to require DHHS to make available and cover the cost of Perfluoroalkyl substance (PFAS) chemical blood testing for any individuals potentially exposed to PFAS through drinking water, and to report on the occurrence of dozens of health conditions in affected communities, many of which we don't currently have surveillance systems or databases to monitor. This would, therefore, require DHHS to establish new systems and databases, which would carry substantial cost to the Department.

PFAS are a group of emerging contaminants in the field of environmental science, and most people have been exposed to these chemicals through everyday household products. There have also been several communities in New Hampshire with identified drinking water contamination. Unfortunately, the risk to human health from exposure to this group of chemicals is still unclear, and more research is needed to better understand the health impact of PFAS exposure which has left many New Hampshire residents understandably concerned and looking for answers. To address these concerns, DHHS has provided information about community exposure levels and engaged the Centers for Disease Control and Prevention's Agency for Toxic Substances and Disease Registry (CDC/ATSDR) to study the health effects of PFAS exposure so more answers can be provided to affected individuals and communities. The NH Public Health Laboratories is also conducting a Biomonitoring Surveillance Study funded by CDC. This study will provide a representative assessment of baseline environmental exposures in residents across the state, including PFAS.

Some individuals and communities have wanted PFAS blood testing; however, this blood test has been used primarily to study and evaluate population levels of exposure. The CDC/ATSDR has recommendations for how to conduct blood testing in exposed communities through a limited random sampling and testing approach, which is what we've attempted to do in New Hampshire communities, including our Merrimack Village District Community Exposure Assessment. HB 691, however, opens blood testing for anybody who is potentially exposed and wants a blood test. This process is not consistent with the CDC's recommended science-based approach to PFAS blood testing.

The PFAS blood test is not a medical test and cannot be easily interpreted by healthcare providers or guide healthcare decisions. This is because there is not a known safe vs. unsafe or normal vs. abnormal PFAS blood level. As a result, interpretation of the PFAS blood test is very difficult. We have found that blood testing has caused frustration amongst some residents who have had their blood tested without clear interpretive criteria. While we have attempted to make PFAS blood testing information and resources available to affected individuals and their healthcare providers (https://www.dhhs.nh.gov/dphs/pfcs/documents/pfas-provider-report.pdf), it is not

financially feasible for the NH DHHS to cover the cost of blood testing for anybody exposed to drinking water contamination. Between 2015 and 2018, DHHS performed PFAS blood testing on approximately 2,200 individuals in New Hampshire at an expense of over $300,000 to the State. To open blood testing to thousands more individuals would likely cost the State millions of dollars. Additionally, a requirement to report on health conditions for which we don't already collect data will carry substantial costs as we would need to establish systems for tracking and monitoring the dozens of health conditions.

DHHS understands and shares the concern about exposure to PFAS chemicals, and we are actively working to address these concerns. The most important way to protect people's health from these contaminants is to test drinking water supplies (to identify potential exposures) and eliminate the exposure. This is something that our partner agency, the NH Department of Environmental Services (NHDES) has been continually engaged in over the last few years. We are also working with CDC's ATSDR to advance the science and understanding of health impacts from PFAS. The 2017 federal defense spending bill included funding to conduct a study of PFAS health impacts to humans, which will begin in New Hampshire on the Pease Tradeport. Additional funding is expected from ATSDR in the near future to roll out a national PFAS health study. Hopefully more answers will be forthcoming for affected residents in the future.

We are concerned that HB 691 sets a concerning precedent for future emerging contaminants and circumvents the best science-based recommended practices at the cost of potentially millions of dollars to the State. Thank you for the opportunity to testify. We are happy to address any questions you may have.

Respectfully Submitted,

Lisa Morris, MSSW
Director, Division of Public Health Services

Benjamin Chan, MD, MPH
State Epidemiologist

Christine Bean, PhD, MBA
Bureau Chief, Public Health Laboratory

Venn Diagram

Biomonitoring New Hampshire

Statewide Biomonitoring Surveillance Study - Testing List

Water Testing

-Copper and Lead (stagnant and flushed)
-Iron -Sodium
-Chloride -Fluoride
-Uranium -pH
-Nitrate -Nitrite
-Total coliform bacteria
-Hardness -*E. coli*

PFAS

-8:2 FTS -6:2 FTS -PFBA
-PFDS -PFHpS -PFHxDA
-PFTrDA -PFPeA -PFTeDA

Other

-Many pesticides, herbicides, and insecticides and their environmental breakdown products
-Corrosives (dissolved solids, alkalinity, pH, sulfur)
-Radionuclides (expanded list: gross alpha, gross beta, radium 226, radium 228, uranium, radon)
-Chromium-6 -1,4 dioxane
-Perchlorate -VOCs

As of 11/19/18, Water Testing and Paired Testing lists have not been finalized

Paired Testing!

Metals

-Antimony -Arsenic -Lead
-Barium -Beryllium -Cobalt
-Cadmium -Manganese -Uranium
-Selenium -Strontium -Thallium

PFAS

-PFOA -PFOS -PFHxS -PFBS -PFNA
-PFDA -PFDoA -PFHxA -PFOSA -PFHpA
-PFUnDA -EtFOSAA -MeFOSAA

Pesticides/Herbicides/Insecticides*

-Cypermethrin -Deltamethrin
-Methyl parathion -Malathion
-Chlorpyrifos -Glyphosate
-Permethrin -Cyfluthrin
-2,4,5-T -2,4-D
-Diazinon

Clinical Testing

Metals

-Cesium -Tungsten
-Mercury, Total -Platinum
-Molybdenum -Tin
-Arsenous (III) acid -Arsenic (V) acid
-Arsenobetaine -Arsenocholine
-Dimethylarsinic acid (DMA)
-Monomethylarsonic acid (MMA)

PFAS

-GenX

Pesticides/Herbicides/Insecticides*

-Parathion
-Chlorpyrifos methyl

Tobacco

-Cotinine

* The NH PHL will be looking for the metabolites of the pesticides, herbicides, and insecticides listed here

American Chemistry Council℠

New Hampshire HB 691

The American Chemistry Council (ACC) is a national trade association representing chemicals and plastics manufacturers in the United States, including member companies in New Hampshire. Our members are committed to the safety of their products and to the protection of the public health.

Over 96% of all manufactured goods are directly touched by the business of chemistry, making this industry an essential part of every facet of our nation's economy. Chemistry provides significant economic benefits in every state including New Hampshire. Thanks to chemistry, our lives are healthier, safer, more sustainable and productive than before. More than 2,000 people are employed by the chemistry industry in New Hampshire, and an additional 5,496 are employed by the plastics and rubber industries.

ACC opposes HB 691, a bill that would require the Department of Health and Human Services to offer and pay for blood testing for perfluorinated chemicals (PFCs) for individuals meeting certain criteria.

The bill also requires the Department to report to the public on the prevalence and incidence of indications known or suspected to be associated with exposure to PFAs in municipalities exposed to concentrations of PFAs in an excess of a total of 50 parts per trillion or the current ambient groundwater quality standard found in rule, whichever is lower.

Concerns with HB 691

With regards to the proposed legislation, SB 691, we respectfully oppose this bill because (1) it is overly broad and (2) it is impractical.

1. **The bill is overly broad because it funds blood testing for individuals who have been exposed to PFAS that do not present toxicity concerns.**
    - PFAS include a broad range of products and substances with differing hazard characteristics, structures and intended uses. By some estimates, over 3,000 substances fall within the universe of PFAS chemistry, only some of which present toxicity concerns.
    - Certain PFAS are known to present hazard concerns – namely long-chain perfluoroalkyl acids (PFAAs) such as PFOS, PFOA, and PFHxS. These substances are known PBTs and have been found at elevated levels in several locations in New Hampshire. Of note, major manufacturers, including FluoroCouncil member companies, no longer manufacture, use, or sell long-chain PFAAs or products that can degrade to those substances.
    - By contrast, many other PFAS products do not present the same hazard concerns as those legacy PFAS. For example, the short-chain PFAS used today as replacements for the legacy PFAS are not PBT substances. The universe of PFAS chemistry also

includes a completely different class of substances known as fluoropolymers. Fluoropolymers do not do not present toxicity concerns because they are of such high molecular weight to not be bioavailable.

- As drafted, the bill would fund blood testing for individuals potentially exposed to any PFAS at any level. Because only a limited set of PFAS, notably long-chain PFAAs, present toxicity concerns, we suggest a narrower scope to the bill that would allow funding for blood testing for individuals potentially exposed to those higher hazard substances above an established level of concern.

2. **The bill is also impractical because it could potentially encompass an unnecessarily large percentage of the State's population.**

   - By defining eligibility for blood testing to include anyone potentially exposed to any PFAS at any level, the universe of individuals potentially eligible is significant.
   - PFAS substances are persistent, meaning they take a very long time to degrade. Therefore, it is likely that very low, background levels of some PFAS may be present in waters throughout New Hampshire, often at levels that do not present a significant risk.
   - This overly broad approach is impractical and could detract resources from those individuals potentially exposed to elevated levels of the PFAS known to present hazard concerns.

For additional information or questions, please contact Margaret Gorman, Senior Director, Northeast Region, American Chemistry Council at (518)432-7835 ormargaret_gorman@americanchemistry.com.

H691

State of New Jersey

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CATHERINE R. McCABE
Commissioner

November 8, 2018

Sarah Pillsbury, Administrator
Drinking Water and Groundwater Bureau
New Hampshire Department of Environmental Services
Sarah.Pillsbury@des.nh.gov
RE: 10/3/18 NHDES request for input on setting MCLs for PFOA, PFOS, PFNA and PFHxS

Dear Ms. Pillsbury,

On October 3, 2018, NHDES requested technical input on setting Maximum Contaminants Levels (MCLs) for four long-chain perfluoroalkyl acids (PFAAs) - PFOA, PFOS, PFNA and PFHxS. Information was requested on approaches, data, and studies to be considered in MCL development; health effects studies and data not considered in the ATSDR (2018) Draft Toxicological Profile for Perfluoroalkyls or the USEPA (2016) PFOA and PFOS Health Advisories; and data and methodologies relevant to costs and benefits for MCLs.

The New Jersey Department of Environmental Protection (NJDEP) and the New Jersey Drinking Water Quality Institute, a legislatively-established advisory body to NJDEP, have extensively evaluated the scientific information relevant to development of MCLs for PFOA, PFOS, and PFNA, including detailed reviews of the ATSDR Draft Toxicological Profile and USEPA Health Advisories mentioned by NHDES. Citations for NJDEP and DWQI documents and peer-reviewed publications on these topics are listed at the end of this letter. The major NJDEP conclusions are shown in the attached PowerPoint presentation, which was excerpted from recent longer NJDEP presentations. These conclusions are summarized below, with citations of publications not considered by ATSDR (2018) or USEPA (2016) marked in **bold**.

General approach and conclusions about risk assessment and MCL development for long-chain PFAAs

NJDEP's general approach and overall conclusions about human health risk assessment and MCL development for long-chain PFAAs such as PFOA, PFOS, and PFNA are summarized in *Slides 3 - 14* of the attached PowerPoint presentation. These conclusions are discussed in more detail in a recent publication by **Post et al. (2017).**

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In summary, NJDEP concludes that there is a need for caution about exposure to long-chain PFAAs from drinking water. Unlike other well-known persistent, bioaccumulative and toxic (PBT) chemicals such as PCBs and dioxins, long-chain PFAAs are water soluble and drinking water is an important exposure route. Ongoing ingestion of even low drinking water concentrations of these PFAAs (e.g. well below the USEPA Health Advisory of 70 ng/L) overwhelms exposures from other sources (primarily food and consumer products) prevalent in the general population. Infants, a sensitive subpopulation for developmental effects of PFAAs, receive higher exposures from breast milk or prepared formula than adults using the same contaminated drinking water source. Because these PFAAs have long human half-lives (several years), body burdens remain elevated for many years after exposure to contaminated drinking water ends.

The substantial increases in blood serum levels from low drinking water concentrations are of concern because long-chain PFAAs are associated with human health effects at blood serum levels prevalent in the general population, even without additional drinking water exposure. Although limitations in the epidemiological data preclude their use as the basis for quantitative risk assessment, these human data provide support for a public health protective approach in development of MCLs based on animal data. Long-chain PFAAs cause multiple toxicological effects in laboratory animals, including some at low doses. Evaluation of mode of action data indicates that these toxicological effects are adverse and relevant to humans. In risk assessment of long-chain PFAAs, animal-to-human extrapolations must be based on internal doses (e.g. blood serum levels), not administered doses, because of the same administered dose results in a much higher internal dose in humans than in animal species.

Factors considered in Development of New Jersey Recommended MCLs for PFOA, PFOS and PFNA

The NJ DWQI developed MCL recommendations of 13 ng/ml for PFNA in 2015, 14 ng/L for PFOA in 2017, 13 ng/L for PFOS in 2018. The MCL for PFNA was adopted by NJDEP in September 2018, and the MCL recommendations for PFOA and PFOS are currently used by NJDEP as guidance for public water systems with detections of these contaminants.

As shown in *Slide 3* of the attached PowerPoint presentation, the DWQI MCL recommendations considered three factors: health effects (Health-based MCL), analytical limitations (Practical Quantitation Level; PQL), and availability of drinking water removal treatment methods. For all three of these PFAAs, achievement of the Health-based MCL was not limited by analytical or treatment removal factors, and the Health-based MCL was therefore recommended as the MCL. Links to DWQI MCL recommendations and technical reports on the health effects, analytical and treatment removal considerations for PFOA, PFOS, and PFNA are provided in the citation list below.

Human Health Risk Assessment for PFOA, PFOS, PFNA and PFHxS

PFOA

The basis for the New Jersey Health-based MCL for PFOA is presented in the **NJ DWQI (2017)** document, "Health-Based Maximum Contaminant Level Support Document: Perfluorooctanoic Acid (PFOA)." A detailed review of the basis of the USEPA Health Advisory for PFOA is found in Appendix 2 of DWQI (2017). NJDEP comments on the ATSDR (2018) Draft Toxicological Profile include comments on PFOA. Links to these NJDEP and DWQI documents are provided in the citation list below. The NJDEP approach and conclusions for PFOA risk assessment are summarized on *Slides 16 - 23* of the attached PowerPoint presentation.

Two Reference Doses (RfDs) were developed for PFOA by DWQI (2017). The first RfD is based on delayed mammary gland development from developmental exposure in mice. This was the most sensitive endpoint for PFOA that provided dose-response data needed for RfD development. This effect is well established, as it was observed in nine separate studies and in two strains of mice (reviewed in detail on p. 130-136 of DWQI, 2017). It is considered to be adverse because structural changes in the mammary gland persisted until adulthood, and there is no reason to discount its human relevance. Furthermore, three human studies report that PFOA is associated with decreased duration of breastfeeding. As noted in NJDEP comments to ATSDR, one of these studies, **Timmermann et al. (2016)** was not cited by ATSDR. NJDEP disagrees with the USEPA and ATSDR rationales for dismissal of this endpoint from consideration for risk assessment; see DWQI (2017 - Appendix 2, p. 9-10) and p. 6-7 of NJDEP comments on the ATSDR Draft Toxicological Profile.

As presented in a peer-reviewed publication (**Post et al., 2012**), BMDLs were developed for 10% decreases in mammary gland developmental score and number of terminal endbuds (a quantitative parameter) in gestationally exposed offspring from Macon et al. (2011). The RfD based on these BMDLs is 0.11 ng/kg/day, and the Health-based MCL based on this RfD would be 0.77 ng/L. Although this endpoint and RfD were judged to be scientifically valid, this Health-based MCL was not recommended because there is no precedent for use of this endpoint as the primary basis for risk assessment.

A second RfD of 2 ng/kg/day was based on increased liver weight in mice, with an uncertainty factor of 10 for more sensitive developmental effects including delayed mammary gland development and persistent liver toxicity. A detailed review of PFOA's hepatic effects (beginning on p. 111 of DWQI, 2017) concludes that increased liver weight caused by PFOA is adverse because it co-occurs with and/or progresses to more severe types of hepatic toxicity. For this reason, NJDEP does not agree with the USEPA and ATSDR conclusion that increased liver weight from PFOA is reversible and not adverse based on the criteria of Hall et al. (2012). Additionally, a detailed review of mode of action data concludes that these hepatic effects of PFOA are relevant to humans. The NJDEP conclusions about mode of action and adversity are discussed on p. 10-14 of

the NJDEP comments on ATSDR (2018) Draft Toxicological Profile and on p. 181-190 of DWQI (2017). The Health-based MCL based on the RfD of 2 ng/kg/day is 14 ng/L.

As shown on *Slide 22* of the attached PowerPoint presentation, PFOA was classified as having suggestive evidence of carcinogenicity by NJDEP and USEPA. NJDEP developed a cancer slope factor of 0.021 $(mg/kg/day)^{-1}$ for testicular tumors in male rats from Butenhoff et al. (2012a). The Health-based MCL based on this slope factor and the one-in-one million (1×10^{-6}) risk level specified in the NJ Safe Drinking Water Act (N.J.S.A. 58: 12A) is 14 ng/L, identical to the value based on non-carcinogenic effects.

Blood serum PFOA levels are expected in increase by about 5-fold from the median U.S. level from exposure to the USEPA Health Advisory of 70 ng/L and about 2-fold from exposure to the NJ MCL of 14 ng/L; see Appendix 2, p. 8 and 13 of DWQI (2017) and *Slide 23* of attached PowerPoint presentation. DWQI (2017) concluded that since "several health effects, some with evidence supporting multiple criteria for causality, are associated with PFOA exposures at serum levels well below those that would result from exposure to 70 ng/L in drinking water," "elevations in serum PFOA levels of the magnitude expected from ongoing exposure to 70 ng/L (the USEPA Health Advisory) in drinking water are not desirable and may not be protective of public health."

PFOS

The basis for the New Jersey Health-based MCL for PFOS is presented in the **NJ DWQI (2018)** document, "Health-Based Maximum Contaminant Level Support Document: Perfluorooctane Sulfonate (PFOS)." A detailed review of the basis of the USEPA Health Advisory for PFOS is found in Appendix 2 of DWQI (2018). NJDEP comments on the recent ATSDR PFOS risk assessment are included in NJDEP comments on the ATSDR (2018) Draft Toxicological Profile for Perfluoroalkyls. The NJDEP approach and conclusions for PFOS risk assessment are summarized on *Slides 25 - 27* of the attached PowerPoint presentation.

Suppression of immune response to a foreign antibody, as indicated by decreased plaque forming cell response, was identified by DWQI (2018) and NJDEP (**Pachkowski, 2018**) as the most sensitive toxicological endpoint for PFOS. The NJDEP RfD for PFOS of 1.8 ng/kg/day is based on suppression decreased plaque forming cell response in mice in Dong et al. (2009), and the basis for this RfD is presented in a recent peer-reviewed publication (Pachkowski et al., 2018). Decreased plaque forming cell response in mice has been reported in four studies of PFOS. It is well-established as an endpoint for risk assessment was used as the basis for RfDs developed by USEPA IRIS. As discussed in DWQI (2018) and Pachkowski et al. (2018), associations of PFOS with decreased vaccine response (the analogous human effect) and increased incidence of infectious disease support the human relevance of this effect.

Suppression of immune response has also been identified as a sensitive effect of PFOS by several other federal, state and international agencies and academic researchers including **NTP (2016)**, ATSDR (2018), **MDH (2017), EFSA (2018- draft), Dong et al. (2017)**, and **Lilienthal et al.**

(2017). DWQI (2017 – Appendix 2, p. 305-306) and NJDEP (Pachkowski et al., 2018) conclude that USEPA did not provide a supportable rationale for use of a less sensitive endpoint, decreased rat offspring body weight (Luebker et al., 2005), as the basis for their PFOS RfD.

The NJDEP comments on ATSDR (2018) note that **Keil et al. (2008)** was not included in the discussion of studies decreased plaque forming cell response in mice caused by PFOS, two recent studies, **Impinen et al. (2018)** and **Goudarzi et al. (2017)** that reported epidemiological associations of infectious disease with PFAAs, including PFOS, were not cited.

PFOS was classified as having suggestive evidence of carcinogenicity by NJDEP and USEPA. DWQI (2018) developed a slope factor based on the incidence of hepatocellular tumors in female rats in Butenhoff (2012b). Although the slope factor was judged too uncertain to use as the basis for the MCL, the cancer risk at the MCL of 13 ng/L based on the RfD for immune system suppression was estimated as 3 in one million, which is close to the New Jersey cancer risk goal of one-in-one million.

Blood serum PFOS levels are expected in increase by about 3.7-fold from the median U.S. level from exposure to the USEPA Health Advisory of 70 ng/L and about 1.5-fold from exposure to the NJ MCL of 13 ng/L; see DWQI (2018) - Appendix 2, p. 304 and p. 311, and *Slide 27* of attached PowerPoint presentation. DWQI (2018) concluded that "there is substantial evidence from epidemiology studies that decreased vaccine response occurs at levels of serum PFOS prevalent in the general population," and that "exposure to PFOS in drinking water at the USEPA Health Advisory of 70 ng/L is predicted to increase serum PFOS concentrations to the upper end of this range and higher. Therefore, the magnitude of elevations in serum PFOS levels expected from ongoing exposure to PFOS in drinking water at the USEPA Health Advisory level are not desirable and may not be protective of public health."

PFNA

The basis for the New Jersey Health-based MCL for PFOA is presented in the **NJ DWQI (2015)** document, "Health-Based Maximum Contaminant Level Support Document: Perfluorononanoic Acid (PFNA)," and in the NJDEP PFNA MCL rule proposal (**NJDEP, 2017**) and adoption (**NJDEP, 2018**) documents. NJDEP approach and conclusions for PFNA risk assessment are summarized on *Slides 29 - 39* of the attached PowerPoint presentation.

In summary, toxicological database for PFNA is considerable and is sufficient for development of a Health-based MCL. PFNA causes hepatic, immune system, developmental, renal, and male reproductive toxicity; chronic carcinogenicity studies have not been conducted. The toxicological effects and mode of action of PFNA are generally similar as PFOA. However, as compared to PFOA, PFNA is more biologically persistent (longer half-life), and its effects occur at lower doses and are more severe, in some cases.

Based on rodent half-life studies and limited human half-life data based on urinary excretion, the human half-life of PFNA was estimated to be twice that of PFOA. As is the case for PFOA

(discussed above), mode of action studies indicate that increased liver weight caused by PFNA is adverse and relevant to humans. The Health-based MCL is based on increased liver weight in pregnant mice (Das et al., 2015), the only study providing the numerical serum PFNA data needed for dose-response analysis. Hepatic necrosis occurred at much lower doses and serum PFNA levels, below those that increased liver weight, in another study (**Stump et al., 2008**), but this study could not be used for quantitative risk assessment because numerical serum PFNA data were not provided. As such, an uncertainty factor of 3 was applied to protect for more sensitive effects at lower doses.

Two rat studies that provide important toxicological data for PFNA (**Stump et al., 2008; Mertens et al., 2010**) were not cited by ATSDR (2018), and NJDEP commented to ATSDR that these studies should be considered. These are the longest duration toxicological studies of PFNA (13 weeks and 18-21 weeks), and they report toxicity at a much lower administered dose and serum PFNA levels shorter duration studies. In these studies, rats were dosed with a technical mixture of PFAS (Surflon S-111) consisting primarily of PFNA (74%), with smaller percentages of other perfluorocarboxylic acids (perfluoroundecanoic acid (C11), 20%; perfluorotridecanoic acid (C13), 5%; PFOA, perfluorodecanoic acid, and perfluorododecanoic acid (C12), <1%). A detailed evaluation of the data from these studies concluded that toxicological effects were primarily caused by PFNA; see p. 39-41 of DWQI (2015).

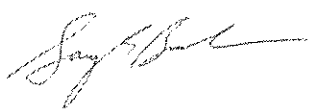
An additional recent study of male reproductive toxicity of PFNA (**Singh and Singh, 2018**) was not available to DWQI (2015) or ATSDR (2015) and should be considered. The citation is provided below.

PFHxS

Although New Jersey has not developed a formal risk assessment for PFHxS, NJDEP scientists have reviewed the current toxicological literature for this compound. The following key studies on toxicity of PFAS were not cited by ATSDR (2018) and should be considered: **Das et al. (2017), Chang et al. (2018)** and **Ramhøj et al. (2018).** Citations are provided below.

We hope that this information is helpful to NHDES in its development of MCLs for PFOA, PFOS, PFNA, and PFHxS. If you have questions or need additional information, please feel free to contact Dr. Gloria Post of the NJDEP Division of Science and Research at gloria.post@dep.nj.gov.

Sincerely,



Gary A. Buchanan, Ph.D.
Director

Attachments

Citations (Those not cited by ATSDR, 2018 are in **bold.**)

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NJ Drinking Water Quality Institute (DWQI) MCL Recommendations

(Documents hotlinked below and posted at https://www.state.nj.us/dep/watersupply/g_boards_dwqi.html)

Perfluorooctane Sulfonate (PFOS), June 2018 – Recommendation Document.

Appendix A – Health-Based Maximum Contaminant Level Support Document for PFOS

Appendix B – Report on the Development of a Practical Quantitation Level for PFOS in Drinking Water

Appendix C – Second Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water

Appendix D – Responses to Comments on DWQI Health Effects Subcommittee Report: "Public Review Draft - Health-Based Maximum Contaminant Level Support Document: PFOS"

Perfluorooctanoic Acid (PFOA), March 2017 – Recommendation Document.

Appendix A – Health-Based Maximum Contaminant Level Support Document" PFOA

Appendix B – Report on the Development of a Practical Quantitation Level for PFOA in Drinking Water

Appendix C – Addendum to Appendix C: Recommendation on Perfluorinated Compound Treatment Options for Drinking Water

Appendix D – Responses to Comments on DWQI Health Effects Subcommittee Report: "Public Review Draft-Health-Based Maximum Contaminant Level Support Document: PFOA"

Perfluorononanoic Acid (PFNA), July 2015 – Recommendation Document.

Appendix A – Health-Based Maximum Contaminant Level Support Document: PFNA

Appendix B – Report on the development of a Practical Quantitation Level for PFNA

Appendix C – Recommendation on Perfluorinated Compound Treatment Options for Drinking Water

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Fiscal Notes

LBAO
19-0619
1/16/19

HB 691-FN- FISCAL NOTE
AS INTRODUCED

AN ACT relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

FISCAL IMPACT: **[X] State** **[ ] County** **[ ] Local** **[ ] None**

	Estimated Increase / (Decrease)			
STATE:	**FY 2020**	**FY 2021**	**FY 2022**	**FY 2023**
Appropriation	$0	$0	$0	$0
Revenue	$0	$0	$0	$0
Expenditures	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase	Indeterminable Increase
Funding Source:	[X] General	[ ] Education	[ ] Highway	[ ] Other

METHODOLOGY:

This bill requires the Department of Health and Human Services to offer and pay for blood testing for perfluorinated chemicals (PFCs) for individuals meeting at least one of several criteria established by the bill. Additionally, whenever a municipality has been exposed to PFCs beyond a certain level defined by the bill, the Department would be required to assess and report the prevalence of a variety of conditions associated with exposure to PFCs.

The Department estimates it would take nine to 12 months to develop the program by hiring and training staff, and the bill would require the addition of four new staff members, as follows:

	FY20	FY21	FY22	FY 23
Program Specialist IV (LG 25, Step 5)				
Salary and Benefits	$90,000	$91,000	$96,000	$97,000
Related Expenses	$14,531	$13,531	$13,531	$13,531
Business Systems Analyst (LG 28, Step 5)				
Salary and Benefits	$100,000	$101,000	$106,000	$107,000
Related Expenses	$12,200	$14,400	$14,400	$14,400
Toxicologist III (LG 26, Step 5)				
Salary and Benefits	$93,000	$94,000	$99,000	$100,000
Related Expenses	$14,531	$13,531	$13,531	$13,531
Lab Assistant III (LG 14, Step 5)				

Salary and Benefits	$64,000	$65,000	$68,000	$69,000
Related Expenses	$14,531	$13,531	$13,531	$13,531
Total Position Costs:	**$402,793**	**$405,993**	**$423,993**	**$427,993**

The Department also anticipates the following additional expenses:

	FY20	FY21	FY22	FY23
Lab Instruments	$485,000	$0	$0	$0
Contract to receive calls from citizens regarding test results	$50,000	$50,000	$50,000	$50,000
Contract for routine specimen transport	$100,000	$100,000	$100,000	$100,000
Instrument Service Contract	$50,000	$50,000	$50,000	$50,000
Expand Qualtrics Contract	$25,000	$25,000	$25,000	$25,000
Access to Chronic Disease Data Contract	$50,000	$50,000	$50,000	$50,000
Dry Ice contract expansion	$25,000	$25,000	$25,000	$25,000
Testing Costs	$150 per test (all years)			
Contract to manage volume of testing	$200 per test (all years)			
Phlebotomy Services	$50.00 per participant (all years)			

The Department estimates the costs for printing and mailing lab reports would range between $25,000 and $50,000 per year. To implement this bill and fulfill the reporting requirements, the Department would procure access to the New Hampshire Comprehensive Health Care Information System, which contains data on chronic disease. However, the Department reports multiple data limitations exist within this data system. Further, many of the incidences in the state required to be reported and analyzed by this bill are located in decentralized databases or not currently captured. The Department estimates the cost of setting up a chronic disease registry to accurately capture incidences of certain chronic diseases is potentially as high as $500,000.

Finally, the Department states the overall cost of implementing the bill's reporting requirement is indeterminable, as the Department is currently unaware of how many municipalities have been exposed to PFC levels higher than the threshold contained in the bill, nor does the Department currently track the prevalence of the various conditions the bill would require it to monitor.

Department of Environmental Services reported this bill will have no impact on their expenditures.

AGENCIES CONTACTED:

Department of Health and Human Services and Department of Environmental Services

Bill as Introduced

HB 691-FN - AS INTRODUCED

2019 SESSION

19-0619
08/05

HOUSE BILL ***691-FN***

AN ACT relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

SPONSORS: Rep. W. Thomas, Hills. 21; Rep. Murphy, Hills. 21; Rep. Stack, Hills. 21; Rep. Meuse, Rock. 29; Sen. Chandley, Dist 11

COMMITTEE: Health, Human Services and Elderly Affairs

ANALYSIS

This bill requires blood testing for certain individuals exposed to perfluorinated chemicals in private or public water supplies.

Explanation: Matter added to current law appears in ***bold italics.***
Matter removed from current law appears [~~in brackets and struckthrough.~~]
Matter which is either (a) all new or (b) repealed and reenacted appears in regular type.

19-0619
08/05

STATE OF NEW HAMPSHIRE

In the Year of Our Lord Two Thousand Nineteen

AN ACT relative to blood testing for individuals exposed to perfluorinated chemicals in private or public water supplies.

Be it Enacted by the Senate and House of Representatives in General Court convened:

1 New Chapter; Perfluorinated Chemicals Testing. Amend RSA by inserting after chapter 130-A the following new chapter:

CHAPTER 130-B

PERFLUORINATED CHEMICALS TESTING

130-B:1 Blood Testing for Perfluorinated chemicals.

I. The department shall develop and implement a program to provide blood testing for persons exposed to perfluorinated chemicals (PFAs) through private or public water supplies at the reduced analytical laboratory price available to state contract holders. The cost of such blood tests shall be covered by the state if:

(a) There is reason to believe or laboratory data demonstrating that the person has been exposed to a drinking water supply in excess of the current applicable groundwater or drinking water criteria;

(b) The department has previously denied the person's request for blood testing for PFAs because his or her public water supply or private water supply does not exceed the current applicable groundwater or drinking water criteria;

(c) The person lives, works, or attends school in an area where there has been a suspected release of PFAs into the air or into the groundwater or drinking water;

(d) The person is a minor who attends daycare or school in an area where there has been a suspected release of PFAs into the air or into the groundwater or drinking water; or

(e) The person lives in a municipality where PFAs have been detected in the drinking water supply.

II. In this chapter, "department" means the department of health and human services.

III. If a municipality has been exposed to concentrations of PFAs in an excess of a total of 50 parts per trillion or the current ambient groundwater quality standard found in rule, whichever is lower, in relation to a known or suspected release, the department shall assess and report to the public the prevalence and incidence of indications known or suspected to be associated with exposure to PFAs within that exposed population including, but not limited to, kidney, liver, or testicular cancer; low birth weight; miscarriages; ulcerative colitis; and thyroid disease or cancer.

2 Effective Date. This act shall take effect 60 days after its passage.