For 20-Plus Years, EPA Has Failed To Regulate 'Forever Chemicals'

The Environmental Protection Agency was first alerted to the health hazards of toxic fluorinated chemicals, known as PFAS, in 1998. In the decades since, the agency has failed to set enforceable regulations on PFAS in drinking water, food, food packaging and a wide array of other everyday consumer goods.

In 2009 and 2019, the EPA announced toothless PFAS "action plans" that fall far short of what is needed to protect Americans, even as reams of studies have linked some PFAS to cancer, reproductive and immune system harm, thyroid disease and other serious health impacts.

ewg

Know your environment.
Protect your health.

1998 – 3M alerts EPA that PFOS, the PFAS chemical in Scotchgard, builds up in blood.
1998 – 3M sends rat studies to EPA, showing liver damage from PFAS exposure.
1999 – EPA begins audit of 3M studies. EPA Alert. 3M Response.
2001 – Attorney Rob Bilott provides EPA with secret DuPont documents on PFOA, the PFAS chemical in Teflon.
2001 – 3M submits PFOS toxicity studies to EPA.
2002 – EPA initiates a "priority review" of PFOA.
2005 – EPA fines DuPont $10.25 million for failing to report "substantial risk of injury to human health" from PFOA.
2006 – EPA brokers a voluntary agreement with DuPont, 3M and other companies to phase out the use of PFOS and PFOA. Announcing the agreement, EPA says "to date, EPA is not aware of any studies specifically relating current levels of PFOA exposure to human health effects."
2006 – Following requests from DuPont, EPA tells consumers that it is safe to use products made with PFAS.
2006 – 3M shares hundreds of secret documents with EPA, resulting in more than $1.5 million in penalties.
2006 – EPA Science Advisory Board draft report finds PFOA to be a "likely human carcinogen."
2009 – EPA publishes a "provisional health advisory" for PFOA and PFOS.
2009 – EPA publishes first PFC action plan.
2012 – EPA requires one-time monitoring by public water systems for some PFAS chemicals.
2015 - EPA proposes a Significant New Use Rule (SNUR) for long-chain PFAS chemicals – as of February 2020, yet to be implemented.
2016 - EPA proposes a Significant New Use Rule (SNUR) for long-chain PFAS chemicals – as of February 2020, yet to be implemented.
2019 - EPA issues second PFAS Action Plan.
2019 - EPA misses self-assigned deadline to issue a plan to set an enforceable legal limit for PFOA and PFOS in drinking water by the end of 2019.

3M General Offices

3M Center
St. Paul, MN 55144-1000
612 733 1110

3M May 15, 1998

BY CERTIFIED MAIL

Document Processing Center (7407)
Attn: Section 8(e) Coordinator
Office Of Toxic Substances
United States Environmental Protection Agency
401 M Street, Southwest
Washington, D. C. 20460

Re: **TSCA Section 8(e) -- Perfluorooctane Sulfonate -- Docket Numbers 8EHQ-1180-373; 8EHQ-1180-374; 8EHQ-0381-0394**

Dear Sir/Madam:

With this letter, 3M Company is submitting information to the EPA Administrator pursuant to Section 8(e) of the Toxic Substances Control Act ("TSCA"). As detailed below, this information relates to fluorochemicals -- specifically, perfluorooctane sulfonate ("PFOS") [CAS No. 2795-39-3] -- and consists of analysis of blood sera samples showing PFOS at very low (*i.e.*, parts per billion ("ppb")) levels. The presence of organic fluorochemicals in the blood of the general population and subpopulations, such as workers, has been known dating back to the 1970's,[1] and 3M's epidemiological study of its own workers indicates no adverse effects at parts per million levels. 3M does not believe that any reasonable basis exists to conclude that PFOS "presents a substantial risk of injury to health or the environment." Nevertheless, as a precautionary measure, 3M is submitting this information to the TSCA Section 8(e) docket at this time.

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[1] See, e.g., Taves, D.R.; *Comparison of "Organic" Fluoride in Human and Nonhuman Serums*, 50 J. Dent. Res. 783 (1971); Guy, W.S., et al.; *Biochemistry Involving Carbon Fluorine Bonds*, American Chemical Society, 117-34 (1976); Ubel, F.A.,et al.; *Health Status Of Plant Workers Exposed to Fluorochemicals -- A Preliminary Report*, 4 American Indus. Hygiene J. 584 (Aug. 1980). 3M has submitted PFOS-related medical surveillance and epidemiological information on its own production workers as well as animal toxicology data previously to the TSCA Section 8(e) docket. See Docket Numbers 8EHQ-1180-373; 8EHQ-1180-374; 8EHQ-0381-0394

Tab 355

Exhibit
2602
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

3M_MN02456909

In the process of validating analytical methodology for measuring PFOS, a product of the electrochemical fluorination process, an outside laboratory detected PFOS at ppb levels in blood samples from individuals with no known occupational exposure to fluorochemicals. Subsequent analyses of commercially pooled sera from human blood bank samples in different regions of the United States measured PFOS levels between 9 ppb and 56 ppb.[2] Analyses of limited historical blood samples from 1969 and 1976 showed mean PFOS levels of 28 ppb and 33 ppb, respectively. Analyses of limited animal sera samples found comparable PFOS levels. 3M also has conducted qualitative *in vitro* and *in vivo* metabolism studies, which suggest the possibility that non-occupational presence of PFOS could result from the metabolic conversion of other fluorochemicals to PFOS.

3M would welcome the opportunity to discuss our findings and our plans. We are sending a copy of this letter to Charles Auer, Director of the Chemical Control Division, and will be contacting him shortly to arrange a meeting for this purpose. In the meantime, please do not hesitate to contact William Weppner at (612) 733-6374 with any questions.

Sincerely,

Dr. Charles Reich
Group Vice President
Chemical Markets Group

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2 3M also analyzed these sera samples for another fluorochemical -- perfluorooctanoate ("PFOA") [CAS No. 3825-26-1] -- but detected the presence of PFOA at quantifiable levels of 12 and 22 ppb in only two of the samples.

Tab 355

REDACTED

SCIENCE PUBLICATION STRATEGY

DEC 10 1998

Publication of scientific and technical information on the FC issue should follow a strategic plan so that key findings can be understood in the context of the published scientific literature. Under this strategy, the science needed to evaluate the safety of PFOS (i.e. the available occupational and toxicology studies) will be published -- or in press -- and thus available to be cited when the publication on serum levels in the general population is published. This will allow the serum level findings to be placed in an understandable, credible context which demonstrates that there is no medical or scientific basis to attribute any adverse health effects to 3M products. In this strategy, the analytical methodology will be published concurrently with the serum level findings.

The strategy is described as a series of steps with a timeline for each activity. The strategy begins with a brief summary of the scientific and technical studies published or publically available:

Key Studies and Reports Available

Ubel, F.A., and others, "Health status of plant workers exposed to fluorochemicals - a preliminary report," *American Industrial Hygiene Association Journal*, vol. 41, pages 584-589, 1980. (*Published study of 3M workers showing no ill health effects of occupational exposure to fluorochemicals.*)

Gilliland, F.D. and Mandel, J.S., "Mortality among employees in a PFOA production plant," *Journal of Occupational Medicine*, vol. 35, pages 950-954, 1993 (*Published study of 3M employees showing no increased mortality due to occupational exposure to PFOA.*).

Gilliland, F.D. and Mandel, J.S., "Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins and cholesterol: a study of occupationally exposed men," *American Journal of Industrial Medicine*, vol. 29, pages 560-568, 1996 (*Published study of 115 3M employees showing no toxicity to the liver due to occupational exposure to PFOA.*).

Key B.D., and others, "Critical review: Fluorinated organics in the biosphere," *Environmental Science and Technology*, vol. 31, pages 2445-2454, 1997. (*PFOS is described as "important commercially as a surfactant and as a precursor of other fluorinated surfactants," as "resistant to biological attack," and as an inhibitor of "gap junction intercellular communication (GJIC) in rat liver epithelial cells cultured in vitro." The paper reports that "inhibition of GJIC has been implicated in tumor promotion during carcinogenesis, teratogenesis and reproductive dysfunction."*)

Reich, C., "Re: TSCA Section 8(e) — Perfluorooctane Sulfonate — Docket Numbers 8EHQ-1180-373; 8EHQ-1180-374; 8EHQ-0381-0394," 3M letter to Office of Toxic Substances, United States Environmental Protection Agency, May 15, 1998. (*This document, which will soon*

become publicly available through the TSCA 8e Office, reports the presence of very low (part per billion) levels of PFOS in blood sera samples for individuals with no known occupational exposure to fluorochemicals.)

Olsen, G.W., "An epidemiologic investigation of reproductive hormones in men with occupational exposure to perfluorooctanoic acid," *Journal of Occupational and Environmental Medicine*, vol. 40, pages 614-622, 1998. (*Study by 3M Medical Department showing no significant hormonal changes in 191 men occupational exposed to PFOA.*).

Reich, C., "Re: TSCA 8(E) SUBSTANTIAL RISK NOTICE ON: N-Ethyl Perfluorooctyl sulfonamido ethanol and Perfluorooctane Sulfonate, Docket Numbers 8EHQ-1180-373; 8EHQ-1180-374; 8EHQ-0381-0394," 3M letter to Office of Toxic Substances, United States Environmental Protection Agency, September 14, 1998. (*This document, which will become publicly available through the TSCA 8e Office, reported that PFOS, when administered to female rats at oral doses of 1.6 or 3.2 milligrams per kilogram body weight per day during pregnancy, significantly reduced pup survival. PFOS also reduced the average gain in body weight of the female rats during pregnancy, with the weight gain at the 3.2 milligrams per kilogram dose of only 87% of the control (no PFOS) rats.*)

Strategy for Publication of Key Studies

1. The PFOS worker study, prepared by Dr. Jeff Mandel and others in the 3M Medical Department, is in final review before submission to an occupationally-focused medical journal. (*This paper will report no adverse biological health effects from exposure to PFOS, based on medical monitoring of workers.*) Comment: publication of this paper is key to demonstrating there is no medical or scientific basis to attribute any adverse health effects to exposure to PFOS.
   Recommendations:
   1) The journal should be selected on the basis of interest in the paper and ability to ensure peer review as quickly as possible.
   2) Target submission of the paper by December 15, 1998; acceptance for publication within three months of submission.
   3) With this plan, this key study could be cited as early as March 15, 1999.

2. PFOS mitochondria study, by Dr. Ken Wallace of the University of Minnesota School of Medicine in Duluth, is being prepared for submission to a peer-reviewed science journal. (*Paper will demonstrate PFOS's mechanism of action on energy metabolism in a test tube (in vitro) system.*) Comment: this paper will be useful for demonstrating a possible mechanism of toxicity of PFOS. However, without the toxicology studies discussed below, the findings are of limited utility for a safety assessment.

Recommendations:
1) The journal should be selected on the basis of interest in the paper and ability to ensure peer review in a timely manner.
2) Target: submission of the paper by March 1999; acceptance for publication within six months of submission.
3) With this plan, this study could be cited by September 1999.

3. The PFOS teratology study, conducted by the 3M Toxicology Department, has been completed. A manuscript of the results, possibly including blood level measurements, could be prepared for publication or presentation at a science conference. (*Paper will demonstrate that exposure to high doses of PFOS to pregnant animals does not cause birth defects in the offspring. The blood level measurements will allow correlations between doses administered in this study and blood levels in animals and humans.*)
Comment: since the study reports largely negative findings (no birth defects), it may be difficult to publish even with the blood level measurements. Consideration should be given to combining this study with the results of the 3M and published subchronic toxicity studies discussed below.
Recommendations:
1) Dr. Chris Wilkinson, a well respected toxicologist with Jellinek, Schwartz & Connolly, Inc., in Washington, D.C., who has been briefed on the FC issue, should be hired to review the study and provide a recommendation on publication of a paper on the teratology study and blood level measurements.
2) Assuming that Dr. Wilkinson and the 3M Toxicology Department agree to submit a paper on the teratology study, Dr. Wilkinson should draft the paper, make final revisions based on 3M review and comments, and submit the paper for publication in a peer reviewed toxicology journal.
3) Target: one month for the recommendation and decision on publication. If the decision is made to proceed with publication, target is three months for completion of the draft paper, one month for 3M review and comment, one month for final revision and submission to a peer-reviewed toxicology journal and three months for acceptance.
4) With this plan, the teratology study could be cited as early as August 1999.

4. PFOS subchronic toxicology studies, conducted by 3M or reported in the scientific literature, could be summarized and a manuscript prepared for publication. (*The paper would review what is known about the toxicity of PFOS from animal studies, prior to conduct of the current studies by 3M.*)
Recommendations:
1) This paper should review, or at least cite, other published toxicity studies on PFOS in addition to the subchronic studies, i.e. all of the published toxicity studies discussed under "Summary of Toxicology Studies" in the "Current Summary" document.

2) Dr. Wilkinson should be hired to review the studies and draft a paper for publication.
3) Target: six months for completion of the draft paper, one month for 3M review and comment, one month for final revision and submission to a peer-reviewed toxicology journal and three months for acceptance.

5. The analytical methods developed to allow specific detection of PFOS in serum levels with a low part per billion detection limit should be written up for publication in a peer reviewed analytical chemistry journal. (*This paper would need to contain data on PFOS levels in serum to document the utility and accuracy of the analytical method.*)
Recommendations:
1) Dr. Wilkinson should be asked to recommend an analytical chemist to prepare a paper for publication on the analytical methods.
2) Assuming that Dr. Wilkinson's recommendation is acceptable to the Analytical Department, the analytical chemist consultant should draft the paper with Dr. Wilkinson's assistance, make final revisions based on 3M review and comments, and submit the paper for publication in a peer reviewed analytical chemistry journal.
3) Target: one month for the recommendation and decision on the consultant. Once a decision is made on the consultant, the target is three months for completion of the draft paper, one month for 3M review and comment, one month for final revision and submission to a peer-reviewed toxicology journal and three to six months for acceptance.
4) With this plan, the analytical study could be cited as early as August 1999.

6. Additional serum level data is needed to document blood levels of PFOS for publication of a peer reviewed science publication. (*This paper would document what is known about PFOS levels in serum and assess the safety of current exposure levels based on the worker study [paper #1 above], and the toxicology studies [papers 2-4 above] that have been completed. The paper would need to reference the analytical methods cited in paper #5*).
Recommendations:
1) A decisions should be made by the 3M Medical Department with the advise of the Legal Department and the Core Team as to what additional data is needed and a plan developed to generate the needed data.
2) The 3M Medical Department should supervise the collection of serum samples with analysis of PFOS levels by the Analytical Department or a contract laboratory approved by them.
3) Dr. Wilkinson should be hired to review the serum data and draft a paper for publication.
4) Target: finalize plans by January 1, 1999, three months to collect samples and analyze the data, three months for completion of the draft paper, one month for 3M review and comment, one month for final revision and submission to a peer-

reviewed toxicology journal and three months for acceptance.
5) With this plan, the serum study could be cited as early as November 1999.

3M General Offices

3M Center
St. Paul, MN 55144-1000
651 733 1110

3M

June 13, 2001

CONFIDENTIAL – FOR SETTLEMENT PURPOSES ONLY

Ms. Ann Pontius
Acting Director, Toxics & Pesticides Enforcement Division
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N. W.
Suite 4109
Washington, D. C. 20044

Re: **3M Company TSCA Section 8(e) Compliance Audit -- Disclosure Of Phase Two Findings**

Dear Ms. Pontius:

3M Company ("3M") has been engaged in communications with your office regarding disclosure of potential violations of TSCA Section 8(e)'s "substantial risk" reporting requirements pursuant to EPA's Self-Audit Policy, 65 Fed. Reg. 19618 (Apr. 11, 2000). These communications have included an August 21, 2000 disclosure of 30 potential violations identified by 3M during Phase One of its Section 8(e) Compliance Audit; a September 22, 2000 letter addressing the relationship of the Compliance Audit to the "Agreement For TSCA Compliance Audit" entered into by 3M and EPA in June 1999; and a December 1, 2000 letter reviewing the facts and circumstances supporting application of the EPA Self-Audit Policy to the Compliance Audit.

3M understands from Kathy Clark and Tony Ellis of your office that EPA has been evaluating the situation and has reached a preliminary decision which will be communicated in writing to 3M within the next few weeks. 3M looks forward to receiving EPA's written preliminary decision. In the meantime, 3M submits this letter to disclose potential violations identified during Phase Two of its Compliance Audit.

I. REVIEW OF AUDIT SCOPE, BACKGROUND AND CONTEXT

By way of brief review of the background and context, Phases One and Two of 3M's 8(e) Compliance Audit are focused on studies and other information that 3M has voluntarily submitted on various fluorochemicals (FCs) in response to two e-mails from Mr. Charles Auer of the Office Of Pollution

Prevention And Toxics ("OPPT") requesting information on various forms of perfluorooctane sulfonate ("PFOS"); on eleven compounds related to PFOS; and on perfluorooctanoic acid ("PFOA"). OPPT subsequently placed these FC studies and information in the TSCA "For Your Information" docket AR-226 (FYI No. 1378). As a shorthand reference, we will refer to the FC studies and information in this letter as the "FYI Submissions."

Phase One of the Compliance Audit included the FYI Submissions made through May of 2000. From the over 600 studies in these FYI submissions, 3M had identified 30 studies that appeared potentially to meet EPA's current TSCA Section 8(e) reporting criteria and that are not already contained in the TSCA Section 8(e) docket, published or otherwise "known to the Administrator." 3M first disclosed and then provided further details regarding these Phase One findings to EPA in the communications identified above.

Phase Two of the Compliance Audit reviewed the FYI submissions made from May 30 through December 31, 2000. As with Phase One of the Compliance Audit, 3M assembled an audit team for Phase Two led by legal counsel from 3M and Latham & Watkins and also comprised of Company scientists and other technical experts. The audit team employed the same two-tier process. Latham & Watkins conducted an independent initial review of the studies. Following this initial review, Latham & Watkins then worked with 3M scientists and technical experts to examine the studies requiring further consideration. Specifically, this further consideration involved (i) consulting with 3M scientists to resolve toxicological and other technical questions as to certain studies; (ii) receiving information from 3M experts relevant to the potential exposure profile of the various compounds; and (iii) examining prior 8(e) filings, FIFRA filings and other sources, including publications, which would make information "known to the Administrator", and hence not 8(e) reportable.

Phase Two covered more studies than Phase One -- over 700 studies -- and the majority of these studies were performed on various formulations dating back to the 1970's of 3M's aqueous fire fighting foam (AFFF) products, which are chemical mixtures comprised primarily of non-fluorochemical components, but containing 0.5 to 6.6 percent PFOS in the formulation. The auditing of the AFFF mixture studies added several additional complexities to Phase Two as compared to Phase One of the Compliance Audit.

First, EPA's current 8(e) reporting guidance does not contain any specific analytical framework for evaluating data on mixtures. For Phase Two, 3M developed a rigorous approach based on the general principles from EPA's current guidance. Under this approach, 3M evaluated the studies based on the reporting triggers for severity of effects and potential for exposures that apply under the guidance to studies on individual chemicals. To assess whether any of

the mixture studies that would otherwise meet these reporting triggers were "corroborative" of information already submitted to the 8(e) docket, published or otherwise "known" to the EPA Administrator, 3M examined whether the effects in any study were reasonably attributable to a particular component of the mixture, and if so, whether the effects of such component are "known" to occur at the levels of the component present in the mixture.

Second, to apply this rigorous approach for evaluating the potential reportability of studies on mixtures, 3M had to compile precise formulation information from historical records. To put this task in perspective, Phase Two involved hundreds of mixture studies, and it was necessary in each case to verify the identities and levels of each mixture component.

Third, for those mixture studies requiring further consideration under the two-tier auditing process, it was necessary for 3M to assess the results of the studies from the standpoint of each component of the formulation. This assessment entailed conducting a toxicological evaluation and literature review of each non-fluorochemical component of each particular mixture formulation. Over 50 mixture studies were identified for further consideration, and thus, required such an assessment.

II. DISCLOSURE OF PHASE TWO AUDIT RESULTS

Phase Two of the Compliance Audit was completed in May of 2001. Based on the audit findings and recommendations, 3M has identified three studies that appear potentially to meet EPA's current reporting guidance. 3M also identified one additional study that would potentially have triggered reporting under the current guidance at the time received by 3M, but for which no present reporting obligation exists due to subsequent publications and 8(e) docket submissions. As to these three studies, 3M has followed the same procedure as recommended by EPA for the Phase One studies identified as potentially reportable. On June 13, 2001, 3M submitted a request that EPA redesignate these three studies now contained in AR-226 (FYI Docket Number 1378) as a supplement to the TSCA Section 8(e) dockets for PFOS and related FCs -- Docket Numbers 373/374. (See Attachment A).

As discussed with the Agency in the context of Phase One of the Compliance Audit, 3M has submitted a substantial body of data on FCs to the TSCA Section 8(e) docket over the years. These submissions reflect the seriousness with which 3M regards its reporting obligation. We have voluntarily

augmented these data through the January 1999 Health Effects White Paper[1], the March 2000 Environmental White Paper[2] and the extensive FYI Submissions. In all cases, the three studies identified as potentially reportable in Phase Two are consistent with prior 8(e) submissions and information in the published literature, but it appears that these studies may not qualify, strictly speaking, as "corroborative" under current EPA guidance, and for this reason, may qualify as potentially reportable under the guidance. Further details regarding these three studies follow below.

⇒ ***Range Finding Rat Teratology Study***: One of the three studies is a range finding rat teratology study on N-EtFOSE which was completed in 1983. Although 3M did submit to the 8(e) docket the results of the definitive study which was completed the following year, the definitive study did not involve the high end dose of 75 mg/kg/day of the range finding study and some of the fetal effects observed at this dose (*e.g.*, cleft palates; incompletely descended testes) do not appear, strictly speaking, corroborative of the results from the definitive study.

⇒ ***Eye Irritation Studies***: Two of the three studies are eye irritation studies on different formulations of AFFF products containing di-ethyl glycol butyl ether (DEGBE) -- a 1991 study with 10 percent DEGBE and a 1975 study with 12 percent DEGBE. The eye irritation observed in these studies -- significant corneal opacity effects -- would appear attributable to DEGBE. Although DEGBE has been reported in the published literature to cause such effects, the lowest level that 3M could locate in the published literature involving significant corneal opacity effects for DEGBE was 25 percent in solution. These two studies showed the same effects, but at lower DEGBE concentrations, and thus, do not appear, strictly speaking, corroborative of the studies in the published literature.

One final noteworthy aspect of Phase Two of the Compliance Audit relates to environmental monitoring data. 3M has been conducting a multi-faceted environmental monitoring program for PFOS and other FCs. This program is ongoing and will not be completed until early in 2002. Phase Two encompassed interim data from one facet of this monitoring program -- measurement of PFOS and other FCs in limited surface water samples at very low part per billion levels -- which had been provided to OPPT through the August

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1 "Perfluorooctane Sulfonate: Current Summary Of Human Serum Health & Toxicology Data" (January 1999) (contained in TSCA 8(e) docket number 8EHQ-0299-373).

2 "Sulfonated Perfluorochemicals In The Environment: Sources, Dispersion, Fate And Effects" (March 2000) (contained in 8(e) docket number 8EHQ-0300-0373).

31, 2000 FYI Submission . EPA's 8(e) reporting guidance for environmental monitoring data is quite limited and has been a continuing source of industry uncertainty. 3M conducted Phase Two applying EPA's existing guidance in a rigorous manner and determined that these interim surface water data should not trigger 8(e) reporting. Nevertheless, in the spirit of full disclosure, we wanted to make the Agency aware of the inclusion of these data in Phase Two of the Compliance Audit and would be willing to answer any questions with regard to our reporting determination.

* * *

Again, 3M looks forward to receiving EPA's written preliminary decision regarding its 8(e) Compliance Audit and to working cooperatively towards a successful resolution of this matter. In the meantime, please do not hesitate to contact Mr. Thomas DiPasquale of 3M's Office Of General Counsel if you have any questions regarding this Phase Two Compliance Audit disclosure.

Very truly yours,

Katherine E. Reed

Katherine E. Reed, Ph.D
Executive Director
Environmental Technology and Safety
Services

Enclosure

cc: Gerald B. Stubbs, EPA Toxics and Pesticide Enforcement Division, Case Development, Policy And Enforcement Branch
Kathy M. Clark, Esq., EPA Toxics and Pesticide Enforcement Division, Office of Regulatory Enforcement
Tony Ellis, EPA Toxics and Pesticide Enforcement Division, Case Development, Policy And Enforcement Branch
Julia A. Hatcher, Esq., Latham & Watkins
Thomas J. DiPasquale, Esq., 3M Office Of General Counsel

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ENFORCEMENT
SENSITIVE

REVISED DRAFT

November 28, 2001

Summary of Disclosures made under the 3M/EPA Audit Agreement

35 Disclosures Made:

- 12 No actions warranted
- 12 Audit Policy met (No Penalties)
- 8 Economic Benefit determinations for $ 109,608
  - $ 10,700 (I-98-60)
  - $ 19,682 (L99-235)
  - $ 14,863 (P99-1002)
  - $ 9,520 (P99-1229)
  - $ 2,542 (L99-456)
  - $ 24,949 (CSA #13) Avoidance
  - $ 13,430 (CSA # 14) Avoidance
  - $ 13,922 (L00-248)
- 3 Stipulated penalties for $ 242,000
  - $ 20,000 (NOC violations)
  - $204,000 (8(e) Phase 1)
  - $ 18,000 (8(e) Phase 2)
- Total Penalty Assessment $ 351,608

ENFORCEMENT
SENSITIVE

REVISED DRAFT: Working Papers
Updated November 28, 2001

DRAFT: Working Papers

3M Company

Disclosure Type	Date Disclosure made	Type of Violation	Proposed Penalty	Violation corrected?	Audit Policy Conditions Met?	Economic benefit?	Disposition or Status
SMMD	2/20/98	§ 5 PMN	$ 40,000	Yes - Company ceased commercial mfg. and submitted a mock PMN for review (I-98-60)	Yes	Yes - $10,700 (See Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Although the company did submit a "mock" PMN, the company is subject to the delayed cost of submitting a PMN.
SMMD	4/8/99	§ 5 LVEA	$186,000	Yes - Company submitted a LVEA, L-99-235.	Yes	Yes - $19,682 (See Ben report)	Company did submit a LVEA but is subject to the delayed costs of submitting the LVEA.
SMMD	10/27/98	§ 8 IUR	$ 0 No action warranted	Company omitted two chemicals to their 1994 IUR submission (Decatur, AL facility and Cordova, IL facility)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.
CSA #1 and CSA #2	11/6/98	§ 8 IUR/ § 8 PAIR	$ 0 Previous NOD Issued	Company submitted their 1994 IUR form and PAIR form for carbon disulfide (Tonawanda, NY facility)	Yes	No	This disclosure was forwarded to Region II for action on 12/1/98. The Region issued a NOD for the violations on 3/17/99.
CSA #3 and CSA #4	11/24/98	§ 8 IUR	$ 0 No action warranted	Company omitted one chemical to their 1994 IUR submission (Bedford Park, IL, and St. Paul, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.

CSA #5	12/10/98	§ 13 Improper cert. for a R&D product	$ 1,430	Company corrected negative certification with a positive certification.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.
CSA #6	12/22/98	§ 5 illegal use	$ 62,700	Company stopped illegal use. A PMN was subsequently submitted by another company.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
CSA #7	1/6/99	§ 5 SNUN	$ 215,600	Company now complying with SNUR requirements.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
CSA # 8	4/26/99	§ 13 False cert.	N/A	No violation occurred.	N/A	N/A	Company submitted a negative certification when none was needed.
CSA # 9	4/29/99	§ 5 PMN	N/A	No violation occurred.	N/A	N/A	Chemical is on the TSCA Inventory as of 1994.
SMMD	5/6/99	§ 8 IUR	$ 18,700	Company failed to submit the 1994 and 1998 IUR form for one chemical at the Decatur, AL site)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
CSA #10	5/11/99	§ 13 False cert.	N/A	No Violation occurred.	N/A	N/A	Company submitted a negative certification when none was needed.
CSA #11	5/20/99	§ 5 SNUN	$ 495,000	Failed to comply with R&D requirements under 40 C.F.R. 721.47.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus

ENFORCEMENT
SENSITIVE

CSA #12	6/4/99	§ 12(b)	N/A	Company disclosed a potential 12(b) violation for an export that occurred on May 26, 1999 for Cas # 74-87-3	N/A	N/A	No Violation occurred. The 12(b) export notification requirement for this chemical was sunset on 7/30/94.
SMMD	6/8/99	§ 8 IUR	$18,700	Company omitted one chemical to their 1998 IUR submission (Cottage Grove, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.
SMMD	6/28/99	§ 5 PMN	$4,059,000	Yes - 3M submitted a PMN (P-99-1002).	Yes	Yes - $14,863 (See Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Although the company did submit a PMN, the company is subject to the delayed cost of submitting the PMN.
SMMD	7/22/99	§ 8 (NOC)	$20,000	Company reported two late NOCs.	No* *Repeat violator $20,000 Stip	N/A	Company had a previous TSCA violation (see TSCA 97-H-34). Company subject to stipulated penalties per the Audit Agreement Section 3(a)(vi).
SMMD	7/22/99	§ 5 PMN	$ 33,000	Yes - 3M submitted a PMN (P-99-1229)	Yes	Yes - $9,520 (See Ben report)	Company did submit a PMN and but is subject to delayed costs.
SMMD	9/21/99	§ 5 LVEA	$ 8,800	Yes - 3M submitted a LVEA L99-456 for this chemical.	Yes	Yes - $ 2,542 (see Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Although the company did submit a LVEA, the company is subject to the delayed cost of submitting the LVEA.

CSA #13	9/29/99	§ 5 PMN	$ 480,000	Yes – The chemical was placed on the TSCA Inventory by another company (deleted) See P-(deleted) (NOC submitted by (deleted) on 6/17/99)	Yes	Yes - $24,949 (see Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Company avoided costs of submitting a PMN.
CSA #14 and CSA #15	11/4/99	§ 5 LVEA	$ 14,300	Company stated that no further manufacture occurred (Final report)	Yes	Yes - $13,430 (See Ben report)	Company avoided cost of submitting an LVEA. No LVEA was filed. All products containing the chemical substance was treated as waste and disposed of by 3M.
SMMD	12/17/99	§ 5 PMN	N/A	Company submitted a LVEA but the Agency determined that the chemical was on the TSCA Inventory (according to company)	N/A	N/A	No Violation occurred.
SMMD	2/10/00	§ 8 IUR	$18,700	Company incorrectly reported the wrong CAS# for a chemical substance to their 1998 IUR (Cottage Grove, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.
SMMD	4/24/00	§ 5	N/A	No determination has been made that a violation occurred.	N/A	N/A	3M has requested a correction of inventory listings to reflect intended chemical species (IC-5854).
SMMD	5/12/00	§ 5 LVEA	$ 11,000	Yes - Company submitted a LVEA, L-00-248.	Yes	Yes - $13,922 (See Ben report)	Company did submit a LVEA but is subject to the delayed costs of submitting the LVEA.

CSA #16	6/2/00	§ 13 Failure to certify for R&D products	Company was unable to reasonably obtain records to determine penalty.	Company imported numerous R&D products without providing the necessary TSCA certifications to Customs	Yes	No	3M has provided the necessary guidance to personnel for future R&D imports requiring TSCA certifications. No past corrections is deemed necessary.
SMMD	6/12/00	§ 8 (IUR)	$ 56,100	Company incorrectly reported the volume amounts of three chemicals for the 1998 IUR report (Cottage Grove, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
CSA # 17	7/7/00 3/30/01	§ 8(c)	$1,804,000	Company reported 164 8(c) allegations that were not contained in the central file.	Yes	No	Economic gains from non-compliance is unknown.
POST FINAL REPORT	11/20/00	§ 5 (polymer exemption)	$ TBD	Company failed to submit an exemption notification requirement.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
POST FINAL REPORT	12/26/00	§ 5 (polymer exemption)	$ TBD	Company failed to submit an exemption notification requirement.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus

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TSCA 8(e) PHASE I	8/21/00	§ 8 (e)	$ TBD To be calculated	29 animal studies ($6,000) 2 human health ($15,000)	No $ 204,000* *Stipulated penalties	N/A	Company did not meet the terms of the audit policy and are subject to the stipulated penalties of the 3M audit agreement.
TSCA 8(e) PHASE II	6/13/01	§ 8 (e)	S TBD To be calculated	3 animal studies ($6,000)	No $ 18,000* *Stipulated penalties	N/A	Company did not meet the terms of the audit policy and are subject to the stipulated penalties of the 3M audit agreement.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OCT - 9 2001

OFFICE OF
ENFORCEMENT AND
COMPLIANCE ASSURANCE

VIA FACSIMILE

Ms. Julia Hatcher, Esq.
Latham & Watkins
Attorneys at Law
1001 Pennsylvania Ave., N.W.
Washington, D.C. 20004-2505

ENFORCEMENT
SENSITIVE

Dear Ms. Hatcher:

As an aide in facilitating our discussion this afternoon, I am sending this letter (which includes some background information) and attachments. EPA is looking forward to resolving this matter with 3M in a timely manner.

Agreement for Toxic Substances Control Act (TSCA) Compliance Audit ("Audit Agreement")

In June 1999, 3M entered into a comprehensive Audit Agreement with EPA to be conducted under the auspices of the Agency's Self-disclosure Policy ("SDP"), 60 Fed. Reg. 66706 (1995) and the terms of the 3M/EPA negotiated Audit Agreement, committing to a comprehensive audit that included TSCA §§ 4, 5, 8, 12 and 13. This Audit Agreement included two concurrent TSCA Audits ("Audit") at 3M's major manufacturing facilities. The Audit included: 1) a comprehensive compliance management systems review of all 3M business units subject to TSCA jurisdiction, which was to cover approximately 24-28 separate business units and facilities (with representative sampling) and 2) a review of the TSCA nomenclature of all chemical reactions and polymerizations between January 1, 1994 - December 31, 1998.

The Audit was scheduled to begin April 24, 1999 and end April 24, 2000. The Audit Agreement included a clause for re-negotiating at the beginning of the 10th month for additional needed time, not to exceed 15 months for an Audit completion date and Final Report due date. 3M requested additional time to complete the Audit, which was extended until July 24, 2000. The Final Report due date was extended until September 24, 2000.

Within 30 days of discovery, 3M was to submit to EPA a report of any potential or actual violation and the action taken to mitigate it. A six-month status report was to provide a list of the products and business units reviewed for TSCA compliance, a summary of all discovered

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violations, and the actions taken to mitigate the violations. The Final Report was to be cumulative, including the information from the six-month report and the same type of information for the latter six months. The Final Report was submitted to the Agency on September 24, 2000.

Penalties

It was agreed, as discussed in the SDP, that in the event EPA took enforcement action, EPA would not seek gravity-based (i.e., non-economic benefit) penalties from eligible facilities that met the conditions outlined in the SDP. If there was an actual or perceived conflict between the SDP and the terms of the Audit Agreement, the terms of the negotiated Agreement would prevail. Notwithstanding the Agreement, EPA reserved the right to take any action pursuant to any applicable authority.

3M also agreed to pay stipulated penalties for certain violations reported by 3M during the Audit that failed to meet the applicable conditions of the SDP and the terms of the Audit Agreement. Under the stipulated penalties provisions, penalties for violations were to be calculated generally as "per chemical" and as "one-day" rather than "per day" violations.

Economic benefit

"EPA retains its full discretion to recover any economic benefit gained as a result of noncompliance." 65 Fed. Reg. 19618, 19626 (Audit Policy). The Audit Agreement further included the provision that "EPA may require 3M to pay an 'economic-benefits' penalty, provided that such penalty is calculated in accordance with then-established EPA policies and procedures for calculating the economic benefits of the type of TSCA violation involved."

Disclosures- See **Summary of Disclosures** and **DRAFT: Working Papers**.

Pursuant to the negotiated Agreement, 3M submitted a total of 35 disclosures, including eleven voluntary disclosures EPA allowed to be included within the scope of the Audit for purposes of penalty mitigation (these self-disclosures were not deemed to be "prior violations" for the purposes of the Audit) and 3M's §8(e) Compliance Audit. EPA has determined that ten disclosures warranted no action; that in 11 disclosures the SDP/Audit Agreement terms were met and no gravity-based penalty is to be assessed; that in seven disclosures, no gravity-based penalties are to be assessed, but $131,976 of economic benefit is to be recovered. (See BEN Runs). Economic benefit from two disclosures are still to be determined based on information necessary from 3M. Stipulated penalties total $242,000 - $20,000 NOC violations; Phase 1 - $204,000 and Phase 2 - $18,000.

On seven disclosures, EPA is seeking additional information concerning the illegal activity, dates of productions and amounts. Two disclosures lack sufficient information to make an assessment as to whether SDP terms have been met. EPA requests that 3M respond to each SDP term as it pertains to each individual self-disclosure so that a determination can be made as

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to whether the conditions have been met.

These assessments will be discussed more fully in following sections of this letter and in the Summary of Disclosures and DRAFT: Working Papers.

TSCA Section 8(e) Audit

Within the last 3-4 months of the Audit Agreement time period, 3M began a separate 3M TSCA §8(e) Compliance Audit ("§8(e) Audit") after OPPTS requested all 3M's information and studies concerning FCs and related compounds ("FCs"). Before the Final Report was due on September 24, 2000, 3M submitted thirty-one §8(e) FC violations (one disclosure) on August 21, 2000. 3M also expressed its intent to conduct two more phases of its §8(e) Audit. Phase 2 would continue to focus on FCs while Phase 3 would include non-FC related chemicals.

In June 2001, 3M submitted three additional FC violations under Phase 2 (one disclosure). It is EPA's current understanding that Phase 3 has been canceled.

These §8(e) disclosures do not meet all of the terms of the Audit policy because there was an EPA information request concerning these chemicals and these disclosures were not contemplated within the scope of the original Audit Agreement. As noted earlier, the Audit Agreement contained "stipulated penalties" for TSCA §8(e) violations disclosed during the Audit that did not meet the terms of the SDP or the Audit Agreement. ($15,000 per human study; $6,000 for other studies).

Since the 8(e) Audit was begun and violations were disclosed to TPED before the Final Report was due, EPA agrees to include these §8(e) disclosures related to this particular chemical and its compounds within the scope of the Audit Agreement under the 8(e) stipulated penalties provision. The Office of Pollution Prevention and Toxics has determined that the following self-disclosed studies are human studies:

1) Flurochemical Exposure Assessment of Decatur Chemical and Film Plant Employees, study date 8/11/1999

2) Analysis of FCs in Samples of Children's Sera, study date 05/21/1999

Phase One stipulated penalties include $30,000 for the these two human studies and $174,000 for the remaining 29 studies, for a total of $204,000. Phase 2 stipulated penalties for three "other studies" are $18,000. Total stipulated penalties for the self-disclosed TSCA §8(e) studies are $222,000.

Next Steps

Upon receipt of the additional information necessary to determine whether conditions

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were met for the designated violations, EPA will determine economic benefit, if any, for those violations. EPA also requests the necessary information concerning production dates and amounts, as noted on the Working Papers chart. Again, EPA does appreciate 3M's willingness to self-disclose and to correct its violations. If you have any questions concerning this matter, please call me at (202) 564-4164 or Tony Ellis at (202) 564-4167.

Sincerely,

Kathy M. Clark

Kathy M. Clark

Enclosures

cc: Michael Nash, Esq.
Tony Ellis
Gerald Stubbs

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DRAFT

October 9, 2001

Summary of Disclosures made under the 3M/EPA Audit Agreement

35 Disclosures Made:

- 10 No actions warranted
- 11 Audit Policy met (No Penalties)
- 9 Economic Benefit determinations for $ 131,976 plus TBD
  - $ 14,785
  - $ 27,567
  - $ 19,855
  - $ 12,887
  - $ 3,505
  - $ 34,315
  - $ TBD (two disclosures, one penalty)
  - $ 19,062
- 3 Stipulated penalties for $ 242,000
  - $ 20,000 (NOC violations)
  - $204,000 (8(e) Phase 1)
  - $ 18,000 (8(e) Phase 2)
- 2 Additional information needed to support audit policy

Additionally, there are 7 disclosures that are captured above that the Agency is seeking additional information on concerning the illegal activity, dates of productions and amounts.

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DRAFT: Working Papers
Updated October 9 2001

DRAFT: Working Papers

3M Company

Disclosure Type	Date Disclosure made	Type of Violation	Proposed Penalty	Violation corrected?	Audit Policy Conditions Met?	Economic benefit?	Disposition or Status
SMMD	2/20/98	§ 5 PMN	$ 40,000	Yes - Company ceased commercial mfg. and submitted a mock PMN for review (I-98-60)	Yes	Yes - $14,785 (See Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Although the company did submit a "mock" PMN, the company is subject to the delayed cost of submitting a PMN.
SMMD	4/8/99	§ 5 LVEA	**Insufficient information to determine penalty (Need dates/ amounts from 1996 to 1999.)**	Yes - Company submitted a LVEA, L-99-235.	Yes	Yes - $27,567 (See Ben report)	Company did submit a LVEA but is subject to the delayed costs of submitting the LVEA.
SMMD	10/27/98	§ 8 IUR	$ 0 No action warranted	Company omitted two chemicals to their 1994 IUR submission (Decatur, AL facility and Cordova, IL facility)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.
CSA #1 and CSA #2	11/6/98	§ 8 IUR/ § 8 PAIR	$ 0 Previous NOD Issued	Company submitted their 1994 IUR form and PAIR form for carbon disulfide (Tonawanda, NY facility)	Yes	No	This disclosure was forwarded to Region II for action on 12/1/98. The Region issued a NOD for the violations on 3/17/99.
CSA #3 and CSA #4	11/24/98	§ 8 IUR	$ 0 No action warranted	Company omitted one chemical to their 1994 IUR submission (Bedford Park, IL, and St. Paul, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.

CSA #5	12/10/98	§ 13 Improper cert. for a R&D product	$ 1,430	Company corrected negative certification with a positive certification.	Insufficient information was provided to support audit policy.	No	The Agency considers the economic benefit from non-compliance to be de-minimus.
CSA #6	12/22/98	§ 5 illegal use	$ 62,700	Company stopped illegal use. A PMN was subsequently submitted by another company.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
CSA #7	1/6/99	§ 5 SNUN	$ 215,600	Company now complying with SNUR requirements.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
CSA # 8	4/26/99	§ 13 False cert.	N/A	No violation occurred.	N/A	N/A	Company submitted a negative certification when none was needed.
CSA # 9	4/29/99	§ 5 PMN	N/A	No violation occurred.	N/A	N/A	Chemical is on the TSCA Inventory as of 1994.
SMMD	5/6/99	§ 8 IUR	$ 18,700	Company failed to submit the 1994 and 1998 IUR form for one chemical at the Decatur, AL site)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
CSA #10	5/11/99	§ 13 False cert.	N/A	No Violation occurred.	N/A	N/A	Company submitted a negative certification when none was needed.
CSA #11	5/20/99	§ 5 SNUN	$ 495,000	Failed to comply with R&D requirements under 40 C.F.R. 721.47.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus

CSA #12	6/4/99	§ 12(b)	N/A	Company disclosed a potential 12(b) violation for an export that occurred on May 26, 1999 for Cas # 74-87-3	N/A	N/A	No Violation occurred. The 12(b) export notification requirement for this chemical was sunset on 7/30/94.
SMMD	6/8/99	§ 8 IUR	$18,700	Company omitted one chemical to their 1998 IUR submission (Cottage Grove, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.
SMMD	6/28/99	§ 5 PMN	**Insufficient information to determine penalty. Needs batch dates and amounts from 1996 to 1999.**	Yes - 3M submitted a PMN (P-99-1002).	Yes	Yes - $19,855 (See Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Although the company did submit a PMN, the company is subject to the delayed cost of submitting the PMN.
SMMD	7/22/99	§ 8 (NOC)	$20,000	Company reported two late NOCs.	No* *Repeat violator $20,000 Stip	N/A	Company had a previous TSCA violation (see TSCA 97-H-34). Company subject to stipulated penalties per the Audit Agreement Section 3(a)(vi).
SMMD	7/22/99	§ 5 PMN	**Insufficient information to determine penalty. Need batch dates and amounts from 1996 to 1999.**	Yes - 3M submitted a PMN (P-99-1229)	Yes	Yes - $12,887 (See Ben report)	Company did submit a PMN and but is subject to delayed costs.

SMMD	9/21/99	§ 5 LVEA	Insufficient information to determine penalty. Need batch amounts and dates from 1996 to 1999.	Yes - 3M submitted a LVEA L99-456 for this chemical.	Yes	Yes - $ 3,505 (see Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Although the company did submit a LVEA, the company is subject to the delayed cost of submitting the LVEA.
CSA #13	9/29/99	§ 5 PMN	$ 480,000	Yes – The chemical was placed on the TSCA Inventory by another company (deleted) See P-(deleted) (NOC submitted by (deleted) on 6/17/99)	Yes	Yes - $34,315 (see Ben report)	Company requested and was granted enforcement discretion to distribute existing stocks. Company avoided costs of submitting a PMN.
CSA #14 and CSA #15	11/4/99	§ 5 PMN or LVEA	$ 14,300 Need to determine if LVEA or PMN was submitted.	Company stated that no further manufacture occurred (Final report)	Yes	Yes - Add't info needed	Unable to verify if a LVEA was submitted by the company. Need to check with company and OPPT. Avoidance or delayed costs.
SMMD	12/17/99	§ 5 PMN	N/A	Company submitted a LVEA but the Agency determined that the chemical was on the TSCA Inventory (according to company)	N/A	N/A	No Violation occurred.
SMMD	2/10/00	§ 8 IUR	$18,700	Company incorrectly reported the wrong CAS# for a chemical substance to their 1998 IUR (Cottage Grove, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus.

SMMD	4/24/00	§ 5		Nomenclature issue of several PMNs; unable to determine if violation has occurred.			3M has requested a correction of inventory listings to reflect intended chemical species (IC-5854). Need to check with company and OPPT on status of request.
SMMD	5/12/00	§ 5 LVEA	Insufficient information to determine penalty. Need batch amounts and dates from 1996 to 2000.	Yes - Company submitted a LVEA, L-00248.	Yes	Yes - $19,062 (See Ben report)	Company did submit a LVEA but is subject to the delayed costs of submitting the LVEA.
CSA #16	6/2/00	§ 13 Failure to certify for R&D products	Insufficient information to determine penalty. Need approx number of imports from 1996 to 2000.	Company imported numerous R&D products without providing the necessary TSCA certifications to Customs	Yes	No	3M has provided the necessary guidance to personnel for future R&D imports requiring TSCA certifications. No past corrections is deemed necessary.
SMMD	6/12/00	§ 8 (IUR)	$ 56,100	Company incorrectly reported the volume amounts of three chemicals for the 1998 IUR report (Cottage Grove, MN)	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus

CSA # 17	7/7/00 3/30/01	§ 8(c)	$1,804,000	Company reported 164 8(c) allegations that were not contained in the central file.	Yes	No	Economic gains from non-compliance is unknown.
POST FINAL REPORT	11/20/00	§ 5 (polymer exemption)	$ TBD	Company failed to submit an exemption notification requirement.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
POST FINAL REPORT	12/26/00	§ 5 (polymer exemption)	$ TBD	Company failed to submit an exemption notification requirement.	Yes	No	The Agency considers the economic benefit from non-compliance to be de-minimus
TSCA 8(e) PHASE I	8/21/00	§ 8 (e)	$ TBD To be calculated	29 animal studies ($6,000) 2 human health ($15,000)	No $ 204,000* *Stipulated penalties	N/A	Company did not meet the terms of the audit policy and are subject to the stipulated penalties of the 3M audit agreement.
TSCA 8(e) PHASE II	6/13/01	§ 8 (e)	$ TBD To be calculated	3 animal studies ($6,000)	No $ 18,000* *Stipulated penalties	N/A	Company did not meet the terms of the audit policy and are subject to the stipulated penalties of the 3M audit agreement.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
ENFORCEMENT AND
COMPLIANCE ASSURANCE

December 14, 2005

MEMORANDUM

SUBJECT: Consent Agreement and Proposed Final Order to Resolve DuPont's Alleged Failure to Submit Substantial Risk Information Under the Toxic Substances Control Act (TSCA) and Failure to Submit Data Requested Under the Resource Conservation and Recovery Act (RCRA)

FROM: Granta Y. Nakayama
Assistant Administrator

TO: Environmental Appeals Board

The Office of Enforcement and Compliance Assurance requests that the Environmental Appeals Board (Board) approve the accompanying Consent Agreement and proposed Final Order executed by E.I. du Pont de Nemours and Company (DuPont) and the Environmental Protection Agency (EPA) that settles this matter for $10.25 million in penalties plus an additional $6.25 million expenditure for Supplemental Environmental Projects (SEPs).[1] This memorandum conforms to the Board's Consent Order Review Procedures dated January 5, 1993.

The Consent Agreement resolves violations of the Toxic Substances Control Act (TSCA), 15 U.S.C. §§ 2601 et seq., and the Resource Conservation and Recovery Act (RCRA), 42 U.S.C. §§ 6901 et seq., as alleged in two administrative complaints filed on July 8, 2004 (subsequently amended on October 13, 2004), and December 6, 2004, copies of which are included with this transmittal package as attachments A and B.[2] The Consent Agreement also

[1]The term "EPA" is used throughout this memorandum to refer to EPA's Enforcement program, other programs or the agency as a whole. The Environmental Appeals Board holds the delegated authority to issue the Final Order in this matter.

[2] By Order of Administrative Law Judge Barbara Gunning dated December 7, 2004, the two administrative actions were consolidated. See attachment C. The allegations in the first Complaint are discussed in this memorandum as Counts 1, 2 and 3. The allegation in the

simultaneously commences and concludes four additional alleged violations of TSCA, as discussed below. All eight alleged violations are collectively referred to in this memorandum as EPA's Action.

The Consent Agreement complies with Section 22.18(b) of the Consolidated Rules of Practice Governing the Administrative Assessment of Civil Penalties and the Revocation/Termination or Suspension of Permits (Rules of Practice), 40 C.F.R. § 22.18(b). I have reviewed the Consent Agreement and determined that it is consistent with the statutes authorizing the Agency's action and that the civil penalty is appropriate.

I. Background

A. TSCA Substantial Risk Reporting Requirement

TSCA § 8(e), 15 U.S.C. § 2607(e), provides that a chemical manufacturer, processor, or distributor who obtains information which reasonably supports the conclusion that a substance or mixture presents a substantial risk of injury to human health or the environment shall immediately inform the Administrator. The requirement to inform the Administrator continues until either the person submits the information or has actual knowledge that the Administrator has been adequately informed through another source. EPA relies upon TSCA § 8(e) information to be made aware of potential risks to human health and the environment posed by chemicals. Congress established the TSCA § 8(e) reporting requirement to ensure that EPA would be informed about potential risks so that it could be able to take any appropriate action to protect the public or the environment. Failure to receive TSCA § 8(e) substantial risk information deprives EPA of being fully apprised of potential risks about chemicals and impairs EPA's ability to take those actions necessary to address potential risks to human health or the environment.

B. The Chemical at Issue

EPA's enforcement action against DuPont involves the synthetic chemical Amonium Perfluorooctanoate (APFO), also known as C-8 and sometimes called PFOA (Perfluorooctanoic Acid) because APFO disassociates to PFOA in water. PFOA is a perfluorinated detergent/surfactant which has been used by DuPont since 1951 in connection with Teflon®-related products at its Washington Works facility outside Parkersburg, West Virginia. PFOA is produced synthetically and formed through the degradation or metabolism of other fluorochemical products, such as fluorinated telomers that are used in non-stick coatings on carpets, clothing, and food wrappers.

December 6, 2004 Complaint is discussed in this memorandum as Count 4. There are four additional allegations raised and resolved in the Consent Agreement that are discussed in this memorandum as Counts 5, 6, 7 and 8.

C. Importance of Timely TSCA § 8(e) Reporting for PFOA

EPA has placed a high priority on understanding the impacts of PFOA. EPA has determined that PFOA is biopersistent in certain animals and associated with developmental effects in animals. As noted in the "Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid and Its Salts," U.S. EPA, Office of Pollution Prevention and Toxics, Risk Assessment Division at 6; 11 (Jan. 4, 2005) (http://www.epa.gov/opptintr/pfoa/pfoarisk.htm), PFOA is considered to be bioaccumulative in humans with a long half-life of about 4.37 years and has the potential for developmental/reproductive toxicity and immunotoxicity in humans. The average human serum background level of PFOA in the general population of the U.S. is estimated to be approximately 5 parts per billion (ppb) and EPA expects this to be true worldwide. PFOA is not naturally occurring, thus all PFOA in human blood is attributable to human activity. EPA is seeking to identify the pathway or pathways (air, water, food, etc.) that result in human exposure to PFOA.[3]

D. EPA's Receipt of TSCA § 8(e) Information Regarding PFOA

On March 6, 2001, Robert A. Bilott, Esq., of Taft, Stettinius & Hollister LLP, sent copies of documents to EPA that he had obtained as part of class action litigation against DuPont. The class action had been looking into claims of PFOA drinking water contamination in West Virginia and Ohio around the DuPont facility. Bilott's documents indicated that DuPont had studied PFOA in pregnant workers and their offspring as early as May, 1981 and thus had obtained the first direct human evidence of PFOA crossing the placenta in humans. Bilott's documents also indicated that DuPont had performed substantial sampling of drinking water in the homes and businesses near its facility, and that DuPont understood in 1987, and confirmed repeatedly in 1988 and 1991, that the drinking water in the homes near its Washington Works facility in West Virginia exceeded DuPont's community exposure guideline for PFOA exposure.

On September 15, 2004, Bilott sent EPA the results of blood sampling not submitted by DuPont that showed elevated levels of PFOA in the blood of twelve people in the community near DuPont's Washington Works facility. The samples showed levels of PFOA ranging from 15.7 ppb to 128 ppb.

On December 20, 2004, DuPont provided EPA with blood sampling results for persons that were not employed at the facility that had been performed sometime in 2002. These ten individuals lived in the vicinity of DuPont's Washington Works Plant in West Virginia and reportedly drank water from private wells located near one or more DuPont landfills at which DuPont disposed PFOA.

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[3]On January 12, 2005, EPA submitted a Draft Risk Assessment for PFOA to the Science Advisory Board for peer review.

While the parties were in negotiations to resolve Counts 1-4 (discussed in detail below), DuPont advised EPA that it had additional materials that it intended to submit to EPA, without conceding that the information was subject to the requirements of § 8(e). In December 2004 and January 2005, DuPont submitted forty-one boxes of information related to PFOA to EPA. EPA reviewed these documents to see if any of the information had not been submitted to EPA as required by TSCA § 8(e). Most of the information had been submitted previously to the Agency. Of the information that had not been previously submitted, EPA determined that three studies should have been submitted under TSCA. This information included two toxicity studies performed on July 11, 1997. One was an inhalation study that exposed male rats to an aerosol form of a perfluorinated chemical. The other was also an inhalation study and involved a different perfluorinated chemical sprayed on rats. DuPont has claimed the identity of these chemicals as Confidential Business Information (CBI). A third study involved an August 29, 1997 inhalation study on rats of a third perfluorinated chemical the identity of which has also been claimed as CBI.

E. Background of the RCRA Claim

The DuPont Washington Works facility operates under a permit pursuant to Section 3005(a) of the Resource Conservation and Recovery Act (RCRA), 42 U.S.C. § 6925(a), and 40 C.F.R. Part 270. In 1989, EPA issued the portion of DuPont's hazardous waste permit ("Permit") that addresses the provisions of the Hazardous and Solid Waste Amendments of 1984. Pub. L. 98-616, Title II, Nov. 8, 1984. The Permit included provisions implementing, inter alia, RCRA § 3004(u), 42 U.S.C. § 6924(u), and 40 C.F.R. § 264.101. Section 3004(u) of RCRA and 40 C.F.R. § 264.101 require "corrective action for all releases of hazardous waste or constituents from any solid waste management unit at a treatment, storage, or disposal facility seeking a permit under [Subchapter C], regardless of the time at which waste was placed in such unit." RCRA § 3004(u); 40 C.F.R. § 264.101.

Under Part I, § I.7 of DuPont's Permit, EPA may request any relevant information to determine whether cause exists to modify the Permit, revoke and reissue the Permit, terminate the Permit, or to determine compliance with the Permit. On May 5, 1997, EPA requested that DuPont provide "known toxicological information" about PFOA in EPA's conditional approval of DuPont's Verification Investigation Report, a report required under the terms of the permit used to describe whether there has been a release of a hazardous waste from a solid waste management unit. On June 6, 1997, DuPont responded to EPA's request for known toxicological information about PFOA but did not include the human blood sampling information concerning the transplacental movement of PFOA that DuPont obtained in 1981. Upon a review of the records associated with DuPont's permit in early 2004, EPA confirmed that DuPont had failed to submit the 1981 data to EPA pursuant to the terms of the RCRA permit.

II. Summary of the Violations

Count 1 alleges that DuPont failed to comply with TSCA § 8(e) when it failed to submit to EPA the information from 1981 that demonstrated transplacental movement of PFOA in humans. This data was substantial risk information concerning PFOA.

Count 2 alleges that DuPont failed to comply with TSCA § 8(e) when it failed to submit to EPA the information concerning PFOA contamination of the drinking water inside people's homes. This data was substantial risk information concerning PFOA.

Count 3 alleges that DuPont violated RCRA § 3005(a) when DuPont failed to comply with the EPA request for "known toxicological information" by failing to submit the 1981 toxicity data concerning PFOA.

Count 4 alleges that DuPont failed to comply with TSCA § 8(e) when it failed to submit the information from 2004 concerning the elevated PFOA blood levels in twelve individuals living in the vicinity of the Washington Works facility. This data was substantial risk information concerning PFOA.

Count 5 alleges that DuPont failed to comply with TSCA § 8(e) when it failed to report data concerning blood test results of ten individuals living near the Washington Works facility with elevated levels of PFOA. This data was substantial risk information concerning PFOA.

Counts 6, 7 and 8 allege that DuPont failed to comply with TSCA § 8(e) on three occasions when it failed to report toxicity data about the three different rat inhalation studies performed on July 11, 1997 and August 29, 1997. Each of the three studies was substantial risk information concerning the aerosol form of a perfluorinated chemical.

III. Penalty Policy

EPA uses its Enforcement Response Policy for Reporting and Recordkeeping Rules and Requirements for TSCA §§ 8, 12 and 13 (March 31, 1999) (TSCA Penalty Policy) and the RCRA Civil Penalty Policy (June 23, 2003) to help interpret penalty factors contained in each statute and to be consistent in penalty assessment for similarly situated violators committing similar violations. The policies are not binding and are used on a case-by-case basis. TSCA § 16(a)(2)(B) requires EPA to take into account the statutory factors of "Nature," "Circumstances," "Extent," and "Gravity." RCRA § 3008 requires EPA to consider the seriousness of the violation and the violator's good faith efforts to comply. EPA also considers the violator's ability to pay, effect on ability to continue to do business, economic benefit, history of violations and other matters as justice may require.

The TSCA Penalty Policy addresses the potential seriousness of the failure to report under TSCA § 8(e) by providing for, under the proper circumstances, penalty assessments for each day of violation. The TSCA Penalty Policy provides that the full statutory maximum penalty for each day of violation may be appropriate if the new information that was not reported would have had a bearing on the Agency's risk assessment and chemical control efforts. EPA considers human exposure data to be more important than animal data. EPA also considers whether the failure to report directly interfered with the Agency's ability to address potentially unreasonable risks to human health. The TSCA Penalty Policy reflects the seriousness EPA attaches to violations of TSCA § 8(e) by not placing caps on the penalties assessed for these violations. Accordingly, for a violation that EPA determines to have directly disrupted EPA's ability to address situations involving potentially imminent hazards, unreasonable risks, or substantial endangerment to health or the environment, the TSCA Penalty Policy provides that the penalty will be the statutory per day maximum authorized under TSCA for the full period of noncompliance. For those violations of TSCA § 8(e) where the failure to report would not have directly interfered with the Agency's ability to address imminent hazards, unreasonable risks, or substantial endangerment, the Penalty Policy generally provides for penalties based on each month of violation (the statutory maximum for each day of violation divided by 30).

IV. The Settlement

EPA settled this case in two phases. The first phase resolved the first four Counts that had been alleged in the two complaints. The second phase resolved Counts five through eight that arose from information DuPont provided to EPA after the two complaints were filed.

A. Phase 1: The First Four Counts

Count 1 involves information that DuPont obtained in 1981 regarding human data demonstrating the rate of movement of PFOA from a mother to her fetus. EPA was not aware of this information until Bilott sent it to EPA in 2001. EPA considers the data to be highly significant because the Agency did not previously have any data from humans showing movement of PFOA from mother to fetus, only data from lab animals. The TSCA Penalty Policy notes that violations involving TSCA § 8(e) information that directly disrupt EPA's ability to address situations involving potentially unreasonable risk or substantial endangerment to human health should be assessed the maximum penalty for each day of the violation. The policy further notes that "failure to comply with the TSCA § 8(e) reporting requirements can be the most serious violations of TSCA § 8. These reports alert the Agency to new information which may have a bearing on the Agency's chemical hazard/risk assessment and chemical control efforts."

For a violation such as Count I, the Penalty Policy provides for the statutory maximum penalty on a per-day basis. The statutory maximum for nearly twenty years of daily penalties for

Count 1 is $183,837,500.[4] EPA believed that DuPont's failure to provide the information regarding the transfer of PFOA across the placenta was significant human data and should be assessed under the circumstances factor of the statute with the highest penalty because of its potential harm to EPA's ability to assess risk to human health. However, after calculating the theoretical maximum penalty, the Agency had to assess other factors in determining the appropriate penalty, particularly the risk that the theoretical maximum could not be obtained in litigation (i.e., the "litigation risk").

There were several potential litigation risks that could have prevented EPA from obtaining the theoretical maximum. The first is whether the Administrative Law Judge (ALJ) would have found it appropriate to assess a penalty at the higher rate as information that "directly disrupts" the Agency's risk management activities under TSCA. DuPont was prepared to argue that the information was not of such great significance. DuPont has asserted that it had submitted similar data in lab animals and that the data from 1981 was merely confirmatory and not conclusive of substantial risk. Moreover, DuPont would have noted that EPA has never obtained an ALJ assessment of a penalty under TSCA § 8(e) for per day assessment of the statutory maximum penalty. EPA believes it would have prevailed on this issue, but there is no certainty in litigation. If the ALJ determined that EPA did not prove that the failure to submit the information "directly disrupted" EPA's risk assessment then, under the Penalty Policy, the maximum penalty would be divided by 30 to $6,127,917 for Count 1.

Second, the theoretical maximum assumes that EPA would succeed in obtaining penalties for each day between DuPont obtaining the information in 1981 and EPA receiving the information in 2001. However, there is case law on the statute of limitations that could significantly reduce the penalty that EPA could obtain. DuPont could have asserted that the five year statute of limitations for civil penalties, 28 U.S.C. § 2462, would prevent EPA from bringing Counts 1, 2, or 3, at all, as the action was filed more than five years after DuPont originally failed to submit the information. EPA would have responded that DuPont's failure to submit the information constituted a continuing violation for each day the information remained unsubmitted. The Board's decisions support EPA's argument here and EPA believes it would have prevailed. (See, e.g., In re Lazarus Inc., 7 E.A.D. 318 (EAB 1997) and Newell Recycling, 8 E.A.D. 598 (EAB 1999)) Yet, even if EPA had prevailed on the continuing violations issue, DuPont could have further argued that the penalties should be limited to those violations which occurred within five years prior to the date of the Complaint. If DuPont prevailed on such a

[4] This value assumes a penalty starting on June 15, 1981, the date the information became available to DuPont, and continuing until March 6, 2001, the date EPA learned of the information. The calculation involves two statutory maximum penalties because of the inflation adjustment rule. One portion of Count 1 would be for the time period prior to January 30, 1997 and includes 5,709 days at $25,000 which equals $142,725,000. For the days after January 31, 1997, the higher daily penalty of $27,500 for 1,495 days totals $ 41,112,500. Adding these two amounts together results in a hypothetical statutory maximum of $183,837,500 for Count 1.

theory for limiting penalties, the statutory maximum for Count 1 would have been $16,582,500.[5]

EPA also faced significant litigation risk that could have prevented any recovery of penalties under Count 2. Count 2 involved the contamination of drinking water in people's homes well above the internal standard of 1ppb that DuPont had set as part of its community exposure guidelines for PFOA in water. There is evidence that DuPont became aware of levels of PFOA exceeding 1 ppb coming out of the tap in homes in the 1980's but did not report those data to EPA as required under TSCA § 8(e). Prosecution of this Count carried a litigation risk, however, because EPA took a series of administrative actions contemporaneous with DuPont's testing that may have altered the reporting obligations under TSCA. Starting in February of 1991, the Agency announced its desire to bring the chemical industrial sector into better compliance with TSCA § 8(e), and offered companies the chance to participate in the TSCA § 8(e) Compliance Audit Program, or CAP, and to settle past instances of noncompliance. While the program was designed to be a backward-looking audit of past unreported data, the series of Agency statements by which EPA announced and developed the CAP[6] seem to have left some ambiguity regarding the reporting requirements in place during the time the CAP was being developed and eventually executed with DuPont, between February 1991 and June 27, 1996.

Judge Gunning recognized this litigation risk at the hearing on the motions for summary judgment on Count 2, noting in her Order Denying Motions for Accelerated Decision on Counts II and III, "quite frankly, I am having great difficulty making sense of the Revised Addendum with the four corners of the Consent Agreement, the CAP Agreement, and the Revised Addendum." She indicated that she was unable to discern a clear meaning of the enforcement waiver that DuPont claimed had been given to all environmental contamination reporting under TSCA § 8(e) as part of EPA's CAP. This language from the Judge raises the possibility that EPA would have recovered no penalty for Count 2 because EPA waived its enforcement authority as part of the settlement under the CAP. Even if the Judge were to have found EPA had not waived its statutory authority to take an action, there were questions about fair notice issues that may have prevented a penalty against DuPont under TSCA for its environmental contamination.

Therefore, as part of defending Count 2, EPA has agreed that it would limit the penalties for failure to provide data related to the drinking water contamination to the time period prior to the 1996 settlement under the CAP. The penalties for Count 2 would only be calculated from 1992 until 1996. The TSCA Penalty Policy assigns daily penalties where the alleged violations do not directly disrupt the EPA's ability to address substantial risk by using the statutory

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[5]Using the time period of July 8, 1999 (five years before the filing date of July 7, 2004) and March 6, 2001 (the date EPA received the data) multiplied by $27,500.

[6]These communications included Federal Register notices, letters to and agreements with individual participating companies, "enforcement waivers" granted during the audit period, as well as various amendments and addenda issued over the span of five years.

maximum amount and dividing by thirty. Thus, the unmitigated (gravity) penalty under the TSCA Penalty Policy for Count 2 is $1,036,433. As with Count 1, EPA would have asserted that collection of penalties is not prevented by the statute of limitations under a continuing violation theory. If, as with Count 1, DuPont prevailed on limiting collection of penalties for continuing violations to those which occurred within five years of the complaint, EPA would have recovered no penalties for Count 2.

Count 3 is a RCRA violation and, under that Penalty Policy, the gravity-based penalty could be $312,300. This gravity-based penalty is derived by treating the "potential for harm" as moderate and the "extent of deviation" as moderate, resulting in a penalty of $8,000 (which is within the range of $5,500 to $8,799). EPA selected the moderate category for the "potential for harm" axis of the matrix because the toxicological information that EPA requested would be used, inter alia, to develop a risk-based comparison level for PFOA to be used in the Health Assessment that DuPont was performing as part of corrective action at the facility. Because there was no health-based criteria available for PFOA, DuPont was required to propose to EPA a provisional risk-based comparison level based, conservatively, on toxicity data. Without having all toxicological information about PFOA, EPA could not completely assess whether the risk-based comparison level that DuPont proposed was appropriate. EPA also recognizes that the RCRA Penalty Policy expressly identifies failure to respond to a formal information request, the violation at issue in Count 3, may have serious implications and merit substantial penalties where the violation undermines the statutory or regulatory purposes or procedures implementing the RCRA program. EPA selected the moderate category for the "extent of deviation" axis of the matrix because while DuPont did provide some toxicological information, and therefore partially responded to the information request, it withheld rare and important human health data -- data that fits squarely within the category of requested information, i.e., "toxicological information."

Under the penalty policy, it is presumed that multi-day penalties are appropriate for days 2-180 of violations with a moderate-moderate gravity-based designation. Because this violation could be designated as moderate-moderate in the gravity-based penalty matrix, and because the violation continued from June 11, 1997 to at least March 7, 2001, the date that EPA received the transplacental movement information, it is appropriate to treat this violation as a multi-day violation. Accordingly, the multi-day penalty component, under the multi-day matrix, would be a per day penalty of $1700 (which is within the range of $1,760 to $275) for 179 days. To calculate the $312,300 penalty, the multi-day penalty component, $304,300 would be added to the $8,000. As with Count 1, EPA would have asserted that collection of penalties is not prevented by the statute of limitations under a continuing violation theory.

Count 4 is another TSCA violation, but it is only a few days long in duration and it is not of the nature that directly disrupted EPA's ability to address an unreasonable risk situation. Thus the unmitigated (gravity) penalty under the policy is $42,250.[7] [8]

All four Counts were considered in settlement collectively since they all pertained to the Counts in the filed complaints. These first four Counts were settled in principle for a penalty of $10 million plus an additional $5 million to be spent on SEPs.

B. Phase 2: The Last Four Counts

DuPont provided information concerning PFOA blood levels in individuals who did not work at the Washington Works facility that gave rise to the violation in Count 5. EPA's review of the boxes of documents submitted by DuPont after the complaints had been filed resulted in three additional alleged violations of TSCA § 8(e) in Counts 6, 7 and 8.

Since all four of the additional alleged violations involved TSCA § 8(e) violations for PFOA or other perfluorinated chemicals, they were collectively settled with the initial four violations. The failure to provide the blood level data on the residents involved less than three months of failure to report. EPA considered this violation to be a major violation for which per day penalties applied, but did not directly disrupt EPA's ability to address situations involving unreasonable risk or substantial endangerment, and thus the Penalty Policy would assess one day at the statutory maximum and the remaining days would each have a penalty of the statutory maximum divided thirty. The proposed penalty for the three alleged violations for failure to report the three aerosol applications of the perfluorinated chemicals likewise would have been

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[7] $\$32{,}500 + \frac{(10 \text{ days} - 1) \times \$32{,}500}{30} = \$42{,}250$

This equation uses September 5, 2004 until September 14, 2004 for dates of penalty.

[8] EPA determined that no additional penalty was necessary to recover the economic benefit of the violations contained in Counts 1-4 because, under the existing methods for determining economic benefit for reporting obligations under TSCA § 8(e) or RCRA corrective action permits, the economic benefit was much less than the penalty collected. EPA also decided that DuPont is such a large company that the ability to pay and the ability to continue to do business were not a problem for this company. Lastly, EPA noted that DuPont has prior violations under TSCA.

divided by thirty under the Penalty Policy. These violations were resolved for an additional $250,000 penalty and $1.25 million in SEPs.[9]

These three violations again posed significant statute of limitations risk since DuPont obtained the information in 1997. It was possible that EPA would not have been able to recover any penalty had DuPont prevailed on that issue. There were also additional issues involving the clarity of the guidance with respect to inhalation exposure. These issues would have been issues of first impression.

C. Appropriateness of the Penalty as a Whole

EPA believes that the penalty it received for the eight counts in this action is appropriate under the statutory penalty factors of TSCA and RCRA. Since the theoretical maximum penalty for Count 1 is so much larger than for the other seven counts, EPA's determination as to the appropriate penalty for the case was based largely on its evaluation of the seriousness of the violation and the other factors, particularly litigation risk, associated with Count 1. There was significant risk under Count 1 that EPA would not be able to prove successfully 1) that the violation directly disrupted EPA's risk assessment activities under TSCA, and 2) that the violation was of a continuing nature and therefore not partly or totally barred by the statute of limitations. Thus, the Judge could have been weighing these issues in deciding whether it would be appropriate to assess nearly twenty years of penalties. EPA took all of these risks into consideration when determining an acceptable penalty for settlement. EPA faced similar litigation risks associated with the statute of limitations for Counts 2, 3, and 6-8. EPA also faced the risk of no recovery under Count 2 due to the lack of clarity surrounding the effect of the 1991 TSCA Compliance Audit Program. In light of the substantial litigation risk, EPA determined that a variance from the TSCA and RCRA penalty policies would be appropriate in this matter. EPA also considered the deterrent effect that a $10,250,000 penalty plus $6,250,000 expenditure for SEPs would have on the regulated TSCA community generally and DuPont in particular.

The $10.25 million penalty is the largest administrative penalty under any statute ever obtained by EPA. It is also more than ten times greater than the largest TSCA § 8(e) penalty EPA has ever obtained.[10] Therefore, although the penalty is a significant reduction from the theoretical maximum penalty under the statute and the TSCA and RCRA penalty policies, EPA

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[9]Counts 6, 7 and 8 dealt with information obtained by DuPont in 1997 and submitted to the Agency in December 2004. The aggregate unadjusted gravity based penalty for these violations is approximately $4.5 million.

[10]It is worth noting that the highest TSCA § 8(e) settlements prior to this action were the $1,000,000 payments several companies made as part of the TSCA § 8(e) Compliance Audit Program.

believes it will have a significant deterrent effect on the regulated community. In fact, since filing the initial complaint in July 2004, there has been a significant increase in TSCA § 8(e) and useful information sent into EPA by industry that does not rise to the level of substantial risk under TSCA § 8(e), but has been submitted to EPA as "For Your Information" (FYI).[11]

This settlement also establishes a commitment by DuPont to spend $6.25 million to perform two voluntary SEPs. The first SEP is a Fluorotelomer-based Product Biodegradration SEP (Biodegradation SEP). Pursuant to this SEP, DuPont will investigate the biodegradation potential of certain chemicals to breakdown to form PFOA. The SEP, valued at $5 million and to be completed in three years, will evaluate nine of DuPont's commercial fluorotelomer-based products in commerce prior to the settlement. Using two types of biodegradation studies, the SEP will help the public to better understand the inherent degradation potential of fluorotelomer-based products to form PFOA and the behavior of such products when released to the environment.[12] DuPont will use independent laboratories to perform all work associated with the Biodegradation SEP and will hire an independent third party to serve as a Panel Administrator for a Peer Consultation Panel. The Peer Consultation Panel will address specific charges related to the biodegradation studies. The public will have the opportunity to nominate Peer Consultation Panel members.

The scientific community, including EPA, does not have a full understanding of how people are exposed to PFOA. In 2003, EPA released a preliminary risk assessment for PFOA and started a public process, involving industry, stakeholders, and others, to identify and generate additional information to better understand the sources of PFOA and the pathways of human exposure. This Biodegradation SEP will help industry, scientists, the public, and EPA

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[11]FYI submissions often come from trade associations and industry consortia that submit TSCA § 8(e) notices on behalf of member companies covered under the reporting requirement. EPA has received FYI submissions covering a wide variety of chemical substances and mixtures from chemical companies, trade associations, unions, public interest groups, civic associations, private citizens, academic institutions, state and other federal agencies, as well as similar organizations/agencies in foreign countries. These notices contain information on human exposure, epidemiology, toxicity test results, monitoring studies, environmental fate, and other information that may be pertinent to risk assessment.

[12]OECD Guideline 303A, one of the two methodologies that will be followed for the biodegradation studies to be performed under the Biodegradation SEP, is subject to copyright. EPA has purchased a copy of OECD 303A and has included it in the CBI version of the settlement package. See CBI settlement package, Appendix A, Attachment C1. In the non-CBI version of this settlement package, EPA has not included a copy of OECD Guideline 303A but has prepared a document explaining where and how it can be purchased and where it can be viewed. See Attachment D to this memorandum. See also non-CBI settlement package, Appendix A, Attachment C1.

examine the potential sources of PFOA in the environment and potential routes of human exposure to PFOA. For instance, one of the biodegradation studies will help determine if commercial fluorotelomer-based polymer products breakdown to form PFOA, which could explain a source of PFOA in the environment. The other biodegradation study will examine the behavior of commercial fluorotelomer-based polymer products in a simulated waste water treatment plant, which could explain both a source of PFOA in the environment and a route of human exposure to PFOA. The results of these studies will assist EPA in determining a more accurate assessment of the potential risks posed by PFOA and by chemicals that may degrade to form PFOA, and to identify what voluntary or regulatory actions, if any, would be appropriate. In implementing the SEP, DuPont has agreed to require the laboratories it contracts with to follow the Agency's Good Laboratory Practices regulations as well as prepare and follow a Quality Assurance Project Plan.

The Second SEP is a Microscale Chemistry and Green Chemistry SEP in Junior High Schools and High Schools in Wood County, West Virginia. Pursuant to this SEP, DuPont will spend $1.25 million in five junior high schools and three high schools. The goals of this SEP include reducing the adverse impact to public health by minimizing the potential exposure to chemicals in schools, reducing the adverse impact to the environment in and around Wood County, West Virginia by minimizing hazardous waste generated at schools, and enhancing science safety in all of the schools involved in the SEP. The implementation of this SEP will involve close coordination with teachers and administrators in the participating schools. The SEP is expected to be completed over a three year period beginning on the date that the settlement is approved by the Board.

V. Human Health and Environmental Concerns

This administrative action involves information about the movement of PFOA from pregnant women to their babies, the contamination of public drinking water supplies in the vicinity of DuPont's Washington Works Facility, additional substantial risk information related to PFOA and a request for PFOA toxicity information as part of RCRA corrective action. The Agency regards this information as potentially useful in its ongoing priority review to understand the potential risks that PFOA may pose to human health or the environment. TSCA § 8(e) information is extremely important to alert the Agency to potential risks so that EPA may prioritize its assessment of chemicals so that the most hazardous chemicals are studied immediately.

VI. Past or Pending Actions

DuPont has three prior TSCA § 8 reporting violations. On October 3, 1996, a Consent Order was signed resolving TSCA § 8(e) violations as part of the CAP. On December 2, 1997, a Consent Order was signed resolving TSCA § 8(a) violations concerning Notices of Commencement of production of a new chemical. On September 29, 2003, a Consent Order was

Consent Order was signed resolving TSCA § 8(a) violations concerning Notices of Commencement of production of a new chemical. On September 29, 2003, a Consent Order was signed resolving TSCA § 8(a) violations concerning Inventory Update Rule violations.

VII. Conclusion

For the foregoing reasons, I recommend that the EAB approve the Consent Agreement and sign the Proposed Final Order.

Attachments

cc: Peter Robertson, DuPont Counsel

UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY

IN THE MATTER OF: **E. I. du Pont de Nemours and Company** Wilmington, DE Respondent Washington Works Facility Route 892 South DuPont Road Washington, Wood County, WV	**Docket No. TSCA-HQ-2004-0016** **Docket No. RCRA-HQ-2004-0016** **FIRST AMENDED COMPLAINT AND NOTICE OF OPPORTUNITY FOR HEARING**

INTRODUCTION

This First Amended Complaint and Notice of Opportunity for Hearing ("Complaint") is filed pursuant to the Toxic Substances Control Act ("TSCA") § 16(a), 15 U.S.C. § 2615(a), and the Resource Conservation and Recovery Act ("RCRA") §§ 3008(a) and (g), as amended by the Hazardous and Solid Waste Amendments ("HSWA"), 42 U.S.C. §§ 6928(a) and (g), and the Consolidated Rules of Practice Governing the Administrative Assessment of Civil Penalties and the Revocation/Termination or Suspension of Permits ("Consolidated Rules of Practice"), 40 C.F.R. Part 22. A copy of which was enclosed with the original Complaint filed July 7, 2004. See original Complaint, Enclosure A. The Complainant is Walker B. Smith, Director, Office of Regulatory Enforcement, Office of Enforcement and Compliance Assurance, United States Environmental Protection Agency ("EPA" or the "Agency"), who has been duly delegated the authority to institute this action. The Respondent, E. I. du Pont de Nemours and Company ("DuPont" or "Respondent"), with its Headquarters Office located at 1007 Market Street,

Wilmington, Delaware, is the owner and operator of a treatment, storage, or disposal facility, and manufacturer, processor or distributor of chemical substances and mixtures found in commerce.

This Complaint serves as notice that Complainant has reason to believe the Respondent failed to immediately submit information as required by TSCA § 8(e), 15 U.S.C. § 2607(e), thereby committing an unlawful act under TSCA § 15, 15 U.S.C. § 2614. This Amended Complaint further serves as notice that Complainant has reason to believe the Respondent has violated RCRA Subtitle C, 42 U.S.C. §§ 6921-6939e, West Virginia hazardous waste management regulations, the federal hazardous waste corrective action regulations in effect at the time of the violation, and Respondent's RCRA Permit Number WVD 04 587 5291. Sections 15 and 16 of TSCA authorize EPA to take an enforcement action against any person that commits a prohibited action under TSCA. Sections 3008(a) and (g) of RCRA authorize EPA to take an enforcement action whenever it is determined that a person is in violation of any requirement of RCRA Subtitle C, EPA's regulations thereunder, or any regulation of a state hazardous waste program that has been authorized by EPA.

On May 29, 1986, pursuant to Section 3006(b) of RCRA, 42 U.S.C. § 6926(b), and 40 C.F.R. Part 271, Subpart A, the State of West Virginia ("West Virginia") was granted final authorization to administer its base hazardous waste management program in lieu of the federal base hazardous waste management program established under RCRA Subtitle C, 42 U.S.C. §§ 6921-6939e. Through this final authorization, the provisions of the West Virginia hazardous waste management program ("Original Authorized Program") became requirements of RCRA Subtitle C and are, accordingly, enforceable by EPA pursuant to RCRA § 3008(a), 42 U.S.C. § 6928(a). A revised West Virginia hazardous waste management program, set forth at West

Virginia Code of State Rules, West Virginia Hazardous Waste Management Rule (WVHWMR), Title 33, Dep't of Envtl. Protection, Div. of Waste Management, Series 20, Sections 33-20-1 through 33-20-15 ("Revised Authorized Program"), was authorized by EPA on July 10, 2000, and accordingly, the provisions of the Revised Authorized Program are enforceable by EPA on and after July 10, 2000, pursuant to § 3008(a) of RCRA, 42 U.S.C. § 6928(a).

On December 15, 2003, pursuant to RCRA § 3006(b), and 40 C.F.R. Part 271, EPA authorized revisions to the West Virginia hazardous waste management program. In particular, West Virginia was authorized to administer the Federal Corrective Action Program, Section 3004(u) of RCRA, 42 U.S.C. § 6924(u), created under the Hazardous and Solid Waste Amendments ("HSWA"), enacted on November 8, 1984 (Pub. L. No. 98-616), which amended Subtitle C of RCRA. See also 40 C.F.R. §§ 264.100 - 264.101. At all relevant times, for purposes of the violation of RCRA § 3008(a) at issue in this administrative Complaint, West Virginia was not authorized to implement the Federal Corrective Action Program. Sections 3008(a) and (g) of RCRA, 42 U.S.C. § 6928(a) and (g), authorize EPA to assess a civil penalty against any person who violates any requirement of Subtitle C of RCRA or a regulation or permit issued thereunder.

In accordance with RCRA § 3008(a)(2), 42 U.S.C. § 6928(a)(2), EPA has notified the State of West Virginia through the West Virginia Division of Environmental Protection ("DEP"), of EPA's intent to issue a Complaint to Respondent for the violation of RCRA, as alleged herein. In support of this Complaint, Complainant hereby makes the following allegations:

COMPLAINT

GENERAL ALLEGATIONS FOR COUNTS I-II

1. DuPont owns and operates a manufacturing facility, known as Washington Works, located at Route 892 South DuPont Road, Washington, Wood County, West Virginia 26181 ("Washington Works Facility"). DuPont was the owner and operator of this facility at all times relevant to this Complaint.

2. DuPont manufactures, processes, or distributes in commerce a chemical substance or mixture as those terms are defined in TSCA §§ 3 and 8(f), 15 U.S.C. §§ 2602 and 2607(f), respectively.

3. DuPont is a person subject to the requirements of TSCA § 8(e), 15 U.S.C. § 2607(e).

4. The Ammonium Perfluorooctanoate ("APFO") product, with CAS No. 3825-26-1 (Octanoic acid, pentadecafluoro-, ammonium salt), was marketed by the 3M Company under the tradename FC-143. At all times relevant to this Complaint, DuPont purchased FC-143, commonly referred to by DuPont as C-8 or C8, from 3M.[1]

5. APFO is comprised of an ammonium cation and a perfluorooctanoic acid ("PFOA") anion. The "oate" suffix of APFO is the nomenclature tool used to signify the anionic form of a carboxylic acid. The suffix "oic" of pure PFOA is used to signify the neutral protonated form of a carboxylic acid. The pure form of PFOA, CAS number 335-67-1, consists of the PFOA anion and its associated cation which is a proton (H+), which is

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[1] The 3M Company manufactured APFO and sold it to DuPont since 1951. In May 2000, 3M announced that it was discontinuing certain perfluorinated chemistries. DuPont began production of APFO between 2000 and 2002 after its supplier, the 3M Company, discontinued manufacturing APFO.

thus different from the PFOA anion alone. In water or biologic media, APFO quickly dissociates to the ammonium cation and the PFOA anion.

6. When APFO is measured in humans or the environment, it is measured by its PFOA anion presence and not by the intact APFO. Because there cannot be APFO without the PFOA anion, and because APFO measured in humans or the environment is measured by the PFOA anion, a short-hand for discussing APFO is "PFOA." Consequently, reference to APFO or C-8 is a reference to this form of PFOA and not the protonated form of PFOA with CAS No. 335-67-1.

7. EPA consistently uses APFO, C-8, and PFOA interchangeably as evidenced in the 2003 fact sheet, available to the public at www.epa.gov/opptintr/pfoa/pfoafcts.pdf, in which EPA stated that "[t]he 'PFOA' acronym is used to indicate not only perfluorooctanoic acid itself, but also its principal salts. The most commonly used chemical in this grouping is the ammonium salt, ammonium perfluorooctanoate or APFO, which is sometimes called 'C8'."

8. While most major toxicological studies and industrial exposures involve APFO, the toxicological effects are likely related to the dissociated anionic form of the acid, i.e., PFOA.

9. Most animal toxicity studies have been conducted with APFO.

10. EPA has identified potential human health concerns from exposure to PFOA.

11. PFOA is a perfluorinated detergent/surfactant manufactured, processed or distributed in the United States by DuPont, in connection with Teflon®-related products.

12. Since 1951, DuPont has manufactured, processed or distributed PFOA at the Washington Works Facility.

13. At all times relevant to this Complaint, Respondent manufactured, processed, or distributed APFO, and consequently, Respondent manufactured, processed, or distributed the PFOA anion of APFO, also referred to as PFOA.

14. The DuPont Washington Works Facility has vented PFOA into the air, treated waste containing PFOA in anaerobic digestion ponds, disposed of waste containing PFOA into landfills, and discharged PFOA into the Ohio River.

15. PFOA is hepatotoxic (liver toxin) to animals.

16. PFOA is biopersistent in animals and humans.

17. PFOA is bioaccumulative in humans.

18. PFOA is associated with developmental effects in animals.

19. PFOA is in the blood of the general population in all geographic regions of the United States.

20. PFOA is not naturally occurring, thus all PFOA in human blood is attributable to human activity. PFOA is produced synthetically and formed through the degradation or metabolism of other fluorochemical products, such as fluorinated telomers.

21. DuPont and other researchers have studied PFOA in lab animals. There are gender differences in the elimination of PFOA in rats.

22. There are substantial differences in the half-life of PFOA in rats and humans. There are considerable differences among species in the kinetics of PFOA.

23. In September 2002, the Director of the Office of Pollution Prevention and Toxics ("OPPT") initiated a priority review on PFOA. EPA published Federal Register Notice, 68 Fed. Reg. 18626 (April 16, 2003), to collect additional information. The Agency determined from recent studies that PFOA causes developmental toxicity and other effects in laboratory animals.

24. On April 10, 2003, EPA released a preliminary assessment indicating there was potential exposure to PFOA at very low levels to the U.S. general population. However, this risk assessment also reflected considerable scientific uncertainty regarding the potential risks.

25. At TSCA § 2(a)(2), 15 U.S.C. § 2601(a)(2), it states,

> "Findings - The Congress finds that - (2) among the many chemical substances and mixtures which are constantly being developed and produced, there are some whose manufacture, processing, distribution in commerce, use or disposal may present an unreasonable risk of injury to health or the environment."

26. At TSCA §§ 2(b)(2)and 2(b)(3), 15 U.S.C. §§ 2601(b)(2) and 2601(b)(3), respectively, it states as follows,

> "Policy - It is the policy of the United States that - (2) adequate authority should exist to regulate chemical substances and mixtures which present an unreasonable risk of injury to health or the environment, and to take action with respect to chemical substances and mixtures which are imminent hazards; and (3) authority over chemical substances and mixtures should be exercised in such a manner as to not impede unduly or create unnecessary economic barriers to technological innovation while fulfilling the primary purpose of this Act to assure that such innovation and commerce in such chemical substances and mixtures do not present an unreasonable risk of injury to health or the environment."

27. Section § 8(e) of TSCA, 15 U.S.C. § 2607(e), provides that,

> "Any person who manufactures, processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment shall immediately inform the Administrator of such information unless such person has actual knowledge that the Administrator has been adequately informed of such information."

GENERAL ALLEGATIONS FOR COUNT III

28. Respondent is a corporation incorporated in the State of Delaware and is a "person" as defined by WVHWMR § 33-20-2, RCRA § 1004(15), 42 U.S.C. § 6903(15), and 40 C.F.R. § 260.10. At all relevant times, for purposes of this Complaint, DuPont was a corporation organized under the laws of the State of Delaware.

29. DuPont owns and operates the Washington Works Facility located at Route 892 South DuPont Road, Washington, Wood County, West Virginia 26181.

30. DuPont is, and has been, at all times relevant to this Complaint, the "owner" and "operator" of the Washington Works Facility as those terms are defined by WVHWMR § 33-20-2 and 40 C.F.R. § 260.10.

31. DuPont's Washington Works Facility is a "facility," as that term is defined by WVHWMR § 33-20-2 and 40 C.F.R. § 260.10.

32. Section 3004(u) of RCRA, and regulations promulgated thereunder, codified at 40 C.F.R. §§ 264.100 - 264.101, require corrective action as necessary to protect human health and the environment for all releases of hazardous waste or constituents from any solid waste management unit at a treatment, storage, or disposal facility, regardless of the time at which waste was placed in such unit, for all permits issued after November 8, 1984.

33. PFOA is in the soil and groundwater at, and within the vicinity of, DuPont's Washington Works Facility.

34. PFOA, as described above, is a discarded material and a "solid waste" as defined under RCRA § 1004(27), 42 U.S.C. § 6903(27) and a "hazardous waste" as defined under RCRA § 1004(5), 42 U.S.C. § 6903(5).

35. On or about January 5, 1987, West Virginia issued to DuPont a RCRA "base" permit for the treatment, storage, or disposal of hazardous waste at its Washington Works Facility.

36. In March 1985, EPA requested DuPont provide information on the Solid Waste Management Units ("SWMUs") at the Washington Works Facility.

37. On December 13, 1989, EPA issued to DuPont the corrective action portion of DuPont's's full RCRA Permit, EPA ID No. WVD 04 587 5291 ("Corrective Action Permit"), for the Washington Works Facility, pursuant to Sections 3005(c) and 3004(u) of RCRA, 42 U.S.C. §§ 6925(c), 6924(u). The Corrective Action Permit was based upon information EPA received in response to the March 1985 request for information.

38. On December 16, 1999, EPA extended the term of the corrective action portion of DuPont's RCRA permit until the effective date of a new corrective action permit for the Washington Works Facility.

39. The corrective action portion of DuPont's RCRA Permit, as extended, remains fully effective as of the filing of this Complaint.

COUNT I - Transplacental Movement of PFOA

40. Complainant re-alleges paragraphs 1-27, above, as if fully set forth below.

41. On or about March 20, 1981, the 3M Company, DuPont's supplier of PFOA, advised DuPont about the potential for PFOA to cause birth defects in rats. Specifically, 3M advised that researchers observed what appeared to be treatment related damage to the eye lenses of some rat pups.[2]

42. On or about May 14, 1981, DuPont revised an existing document that described the results of a blood sampling of eight pregnant employees at the Washington Works Facility.[3] The following August 1981, DuPont revised this document again with handwritten notations. This 'blood sampling' document identifies the levels of PFOA measured in the blood of certain pregnant employees at the Washington Works Facility along with a description of the status of a child.

43. DuPont's's human blood sampling was conducted to monitor these pregnant employees for their exposure to PFOA, to monitor umbilical cord blood for PFOA on at least one occasion, and to test babies' blood for PFOA on at least two occasions.

44. The May 14, 1981 document provides certain details on one pregnant woman with 0.078 parts per million ("ppm") C-8 in her blood. She is described as having a "Normal child - born April 1981. Umbilical cord blood 0.055 ppm."

45. The existence of the "0.055 ppm" of PFOA in the umbilical cord blood demonstrates PFOA movement in humans, and specifically, that PFOA moved from the mother, through the placenta, to the fetus.

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[2]Later studies could not reproduce the fetal lens defect.

[3]The document does not indicate the date it was created.

46. DuPont did not immediately submit, nor has it ever submitted, this human blood sampling information concerning the transplacental movement of PFOA, a chemical known then to be persistent, to demonstrate liver toxicity in animals and that DuPont was reviewing for possible birth defects.

47. On or about December 18, 1981, by memorandum, DuPont discussed an inquiry from a mother with a child with 0.4 ppm C-8 in its blood, asking whether a baby's liver was more susceptible to damage by PFOA than that of an adult. The memorandum from J. F. Doughty, DuPont's Washington Works Facility, to R.D. Ingalls, DuPont's Wilmington, Delaware office, also inquired as to whether the 3M studies on C-8 showed any malformations other than eye defects.

48. On or about March 16, 1982, DuPont reported data to EPA, that EPA subsequently regarded as substantial risk data under TSCA § 8(e), concerning the transplacental movement of PFOA in rats. DuPont continued to fail or refuse to disclose that it had obtained human blood sampling data in 1981confirming the transplacental movement of PFOA in humans.

49. DuPont reviewed the human blood sampling information as part of its litigation preparation involving the Washington Works Facility in federal district court, Southern District of West Virginia, when it produced the human blood sampling document to opposing counsel on September 24, 2000.

50. DuPont continued to fail or refuse to submit to EPA the data concerning human blood sampling confirming the transplacental movement of PFOA after it had included the document in production for litigation September 24, 2000.

51. The human blood sampling information confirming the transplacental movement of PFOA is information that reasonably supports the conclusion that PFOA presents a substantial risk of injury to human health that the Administrator was not already adequately informed about at the time the information was obtained by DuPont or at any time prior to the date EPA finally received the data.

52. The 1981 data indicating that PFOA moves across the placental barrier between PFOA-exposed mothers and their fetuses suggest that such fetuses could experience toxic effects associated with PFOA, including persistence/bioaccumulation, and, as observed in animal tests, developmental toxicity and liver toxicity. The human data are more indicative of such possibility in humans than the data submitted to EPA by DuPont in 1982, which demonstrated that PFOA moved across the placental barrier in rats used in laboratory experiments. EPA's efforts to characterize effects of PFOA might have been more expeditious had the data on transplacental movement of the chemical in humans been submitted immediately by DuPont when DuPont obtained the information in 1981.

53. DuPont's failure to immediately inform EPA about the information concerning the human blood sampling constitutes a violation of TSCA § 8(e), 15 U.S.C. § 2607(e).

54. The Agency considers the human blood sampling information confirming transplacental movement of PFOA in humans to reasonably support the conclusion of a substantial risk of injury to health or the environment. The Administrator was not adequately informed

about this risk at the time the information was obtained by DuPont in 1981, and was not informed until March 6, 2001.[4]

55. DuPont was required to immediately inform the EPA about the human blood sampling information confirming transplacental movement of PFOA under TSCA § 8(e), 15 U.S.C. § 2607(e), as information which reasonably supports the conclusion that such substance or mixture presents a substantial risk to health.

56. DuPont was required under TSCA § 8(e) to inform the Administrator every day between June 15, 1981 and March 6, 2001, about the human blood sampling information confirming transplacental movement of PFOA.

57. DuPont failed or refused to immediately inform the Administrator about the human blood sampling information confirming transplacental movement of PFOA.

58. Section § 15(3)(B) of TSCA, 15 U.S.C. § 2614(3)(B), provides that it is unlawful for any person "to fail or refuse to submit reports, notices, or other information" as required.

59. DuPont's failure or refusal to submit the human blood sampling information as required under TSCA § 8(e) is an unlawful act under TSCA § 15(3)(B).

COUNT II - Public Water Supply Contamination

60. Complainant re-alleges paragraphs 1-27, above, as if fully set forth below.

[4]Mr. Robert A. Bilott, Esq. of Taft, Stettinius & Hollister, LLP first supplied the human blood sampling document to EPA on March 6, 2001.

61. On or about June 14, 1984, DuPont compiled sampling results which determined that PFOA was present in the public water supply in communities in the vicinity of the Washington Works Facility.

62. On or about August 29, 1984, DuPont summarized the results from water samples collected on March 15, 1984, and a second set of water samples taken on June 4, 1984, in a letter to J. A. Schmid, from J. F. Doughty (signed John Doughty) entitled, SUMMARY OF C-8 IN WATER SAMPLING PROGRAM. This letter includes a table showing the location of samples of drinking water from such sites as an employee's home, a public drinking fountain, a private well, and other sites collected on March 15, 1984. These sampling sites were located in West Virginia and Ohio. The above-described 1984 drinking water sampling results detected PFOA in the public water supply for Lubeck, West Virginia and in Little Hocking, Ohio.

63. On or about March 13, 1987, DuPont recorded C-8 at 1.9 parts per billion ("ppb") in two water samples described as "Lubeck Business Tap." On or about May 12, 1988, November 2, 1988, May 7, 1989, May 23, 1991, May 29, 1991, and August 8, 1991, DuPont obtained "LPSD Home Tap" sampling showing PFOA at the respective levels of 2.2 ppb, 1.4 ppb, 0.7 ppb, 3.8 ppb, 3.8 ppb and 3.9 ppb in home tap water.

64. The two samples in paragraph 63 from "Lubeck Business Tap[s]" appear to be among the five samples discussed in a DuPont Interoffice Memorandum dated May 12, 1987, from Tony Playtis to Roger Zipfel. Sample number 2 and sample number 3 are identified as "taken on" March 13, 1987, and the analytical report showed C-8 at 1.9 ppb for both samples. Sample number 2 is identified as drinking water coming from Powell's General

Store, Washington, WV and sample number 3 is identified as drinking water from the Lubeck Pennzoil, Lubeck, WV. Both samples were collected by C. L. Hill.

65. On or about August 29, 1988, a DuPont interoffice memorandum from Anthony J. (Tony) Playtis to Roger J. Zipfel with the subject: "Test Results - C8 in Groundwater," contained information on the level of PFOA detected in six water samples. Four of the six samples indicate PFOA over 1 ppb at the locations sampled. One sample, among the six listed, is described as follows:

Sample Description	C8 Level
Lubeck Water - Playtis Home 5/12/88, 17:00	2.2 ppb

66. On or about January 30, 1989, in a DuPont Interoffice memorandum from Anthony J. (Tony) Playtis to Roger J. Zipfel with the subject: "Test Results - C8 in Water," there are results for four local water sources sampled. The results from two of the four samples indicate PFOA in an amount greater than 1ppb. One sample, among four listed, is described as follows:

Sample Description	ppb C8
Lubeck Water - Playtis Home 11/2/88, 17:00	1.4

67. As of 1991, DuPont described its CEG as follows: Community Exposure Guidelines ("CEGs") are DuPont's exposure guidelines that are expected to be without any effect to members of the community during continuous 24-hour a day exposure to a chemical or physical agent. CEGs are based on the best available information from industrial experience, animal toxicity studies, controlled human exposure studies, and epidemiological findings.

68. On or about June 6, 1991, DuPont set a Community Exposure Guideline for drinking water ("CEGw") at 1 microgram per liter ("1 μg/L" or "1 ppb") for PFOA. In June of 1991, DuPont's Washington Works Facility was aware of the 1 ppb CEGw that had been established for PFOA.

69. At the time DuPont adopted a CEGw at 1 ppb, it had collected results from drinking water samples as discussed above, and had information regarding the level of PFOA detected in such samples. DuPont took many drinking water samples and tested them for PFOA during the years 1984 through 1991. In a June 14, 1984, Personal and Confidential Update, titled "UPDATE ON C-8 IN WATER SAMPLES," some of the following sampling results were provided

- Washington 3/25/84 ..... 1.2 ppb C-8
- Washington 6/4/84 ..... 1.0 ppb C-8
- Lubeck 6/4/84 ..... 1.5 ppb C-8
- Little Hocking 3/15/84 ..... 0.8 ppb C-8

70. In a July 11, 2003, letter to Rich Hefter of EPA, DuPont provided some but not all of the analytical results that it had obtained for PFOA in drinking water. Some of the analytical results provided to EPA in July 2003, are listed below:

C-8 OFF SITE SAMPLING	C-8 PPB
• LUBECK BUSINESS TAP (2) 3/13/87	1.9, 1.9
• LPSD HOME TAP -P 5/12/88	2.2
• LPSD HOME TAP -P 11/2/88	1.4
• LPSD HOME TAP -P 5/7/89	0.7
• LPSD HOME TAP -M 5/23/91	3.8
• LPSD HOME TAP - C 5/29/91	3.8

The C-8 OFF SITE SAMPLING table also includes a sampling result dated after DuPont had adopted the CEGw standard of 1 ppb. It is listed on the chart as follows:

- LPSD HOME TAP -M 8/8/91 ..... 3.9

71. These results, discussed in paragraphs 69-70, above, indicate a substantial risk of widespread exposure to a chemical at a level of concern that requires informing the Administrator immediately.

72. DuPont purchased the drinking water supply wells from the Lubeck Public Service District (LPSD) during the 1986 to 1990 time period. New drinking water supply wells were established 2.7 miles away from the Washington Works Facility for the LPSD.

73. On or about June 23, 1991, after DuPont had purchased the drinking water supply wells from LPSD, DuPont detected PFOA at 2.4 ppb in a new well in the new Lubeck well field (2.7 miles south - southwest of Washington Works), as discussed in a September 19, 1991 memorandum to Walt Stewart from Terry Vandell with the subject: "Meeting Minutes Of The On-Site Washington Works Meeting (September 11, 1991, 9:00 AM-11:00 AM) Regarding The September 4, 1991 Proposed C-8 Sampling Program."

74. DuPont failed or refused to submit the information it had obtained on the widespread contamination of PFOA in public drinking water at levels exceeding its CEGw, even after reviewing it specifically to decide whether it should be submitted to EPA under TSCA § 8(e). On or about January 12, 2000, in a letter from DuPont's Senior Counsel Andrea V. Malinowski, Esq. to Douglas Johns, Esquire, Legal, General Electric Plastics ("GE"), DuPont replied to an e-mail concerning FC-143. The letter states, "Regarding item 1, DuPont did not submit a TSCA 8(e) notification to EPA concerning the presence of FC-143 in environmental media. The 8(e)-reportability of the presence of FC-143 in environmental media was reviewed within DuPont and determined to not be reportable."

The letter provides four bullets as the reasons for that decision. Those bullets are summarized as follows:

- Toxicology studies were submitted to EPA by the 3M Company for FC-143,
- Discharge of FC-143 in an outfall to the Ohio River had been reported to EPA,
- FC-143 was detected in the aquifer underlying a solid waste management unit identified as B-4, at the Washington Works Facility,
- The presence of "detectable levels" of C-8 in the Lubeck public supply wells had been reported to EPA's waste program in a February 9, 1990 letter.

75. The four bullets listed by DuPont in its January 12, 2000, letter to GE are misleading as follows:

- The toxicology studies provided by 3M do not include DuPont's human blood sampling data concerning the transplacental movement of PFOA.
- DuPont's statement that FC-143 is present in outfall 005 does not provide information about widespread contamination of home tap water with PFOA above DuPont's CEGw of 1 ppb.
- DuPont's detection of PFOA under the DuPont Local Landfill does not give the Administrator notice that any of the PFOA had migrated off site. The statement was made as part of a statement of "Releases, Spills, etc." that dealt with leakage of surfactant when "the third basin was constructed . . ."
- DuPont's statement to EPA in the Verification Investigation Workplan for Six Solid Waste Management Units on February 9, 1990, that C-8 was in the public water supply begins with the statement, "Releases have occurred in the past." After discussing in the

Verification Investigation Workplan the construction of a dam in 1964 and the re-lined impoundments in 1973-74, DuPont states, "The Lubeck public supply wells have perfluorooctanoate (also called C-8). Washington Works is in the process of purchasing these wells from Lubeck Water supply." This statement gives the impression that the C-8 release has ceased, was confined to a small area, and that DuPont purchased the contaminated area to prevent public exposure.

76. In DuPont's January 12, 2000 letter to GE, discussed in paragraphs 74-75, above, DuPont fails or refuses to recognize that its C-8 contamination in public drinking water is ongoing, that C-8 contamination extends into people's homes, and that DuPont had never informed the Administrator of levels of C-8 contamination of drinking water greater than three times higher than DuPont's own CEGw set more than 8 years before DuPont wrote its letter to GE.

77. DuPont's January 12, 2000 letter to GE does not discuss the 1 ppb CEGw for PFOA established by DuPont on June 6, 1991, and the subsequent samples obtained from one of the new public water supply wells showing over twice that level at 2.4 ppb PFOA on June 23, 1991.

78. DuPont reviewed the PFOA contamination of the public water supply information as part of its preparation for litigation in federal district court, Southern District of West Virginia, concerning PFOA contamination originating from the Washington Works facility. DuPont failed or refused to submit to EPA the substantial risk information concerning PFOA contamination in public drinking water that it provided to opposing counsel on or about October 18, 2000.

79. TSCA § 8(e), 15 U.S.C. § 2607(e), provides that "Any person who manufactures, processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment shall immediately inform the Administrator of such information unless such person has actual knowledge that the Administrator has been adequately informed of such information."

80. The Agency considers the information concerning the contamination of the public water supply to reasonably support the conclusion of a substantial risk of injury to health or the environment.[5] The Administrator was not informed at the time DuPont obtained monitoring data showing contamination of the public water supply prior to 1991, and subsequent to that time.

81. DuPont was required under TSCA § 8(e), 15 U.S.C. § 2607(e), to immediately report the information concerning DuPont's monitoring data of the contamination of the public water supply for the communities in the vicinity of its Washington Works Facility and this obligation continued as DuPont learned more about the contamination.

82. DuPont was required under TSCA § 8(e) to inform the Administrator every day between July 24, 1991 and March 6, 2001 about the information it had obtained on the widespread contamination of public drinking water at a level greater than its CEGw.

83. DuPont was required to inform the Administrator immediately about information concerning the PFOA contamination of public drinking water that DuPont obtained in

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[5]Robert A. Bilott, Esq. of Taft, Stettinius & Hollister, LLP, submitted the information as discussed in footnote 4.

1984. DuPont continued to fail or refuse to submit this information to the Administrator as it increased its understanding that the PFOA contamination extended into people's homes and was more than twice DuPont's own Community Exposure Guideline for water.

84. TSCA § 15(3)(B) of TSCA, 15 U.S.C. § 2614(3)(B), provides that it is unlawful for anyone "to fail or refuse to submit reports, notices, or other information" required by TSCA.

85. DuPont's failure or refusal to immediately submit to the Administrator its understanding that the PFOA contamination extended into people's homes at levels approaching and exceeding its Community Exposure Guideline for water as required under TSCA § 8(e) is an unlawful act under TSCA § 15(3)(B).

COUNT III - RCRA Permit Violation

86. Complainant re-alleges paragraphs 28-39, above, as if fully set forth below.

87. Section 3005(a) of RCRA, 42 U.S.C. § 6925(a), provides, in pertinent part, that each person owning or operating an existing facility or planning to construct a new facility for the treatment, storage, or disposal of hazardous waste is required to obtain a permit and comply with the regulations promulgated by EPA concerning permitting requirements. In addition, the treatment, storage, or disposal of hazardous waste or the construction of a new facility is prohibited unless in compliance with all applicable permitting requirements.

88. Section 3005(c) of RCRA, 42 U.S.C. § 6925(c), provides, in pertinent part, that upon a determination by EPA (or a state, if applicable), of compliance by a facility for which a

permit is applied for pursuant to RCRA §§ 3004 and 3005, 42 U.S.C. §§ 6924 and 6925, EPA (or the state) shall issue a permit for such facilities; and that each permit issued under RCRA § 3005 shall contain such terms and conditions necessary to protect human health and the environment.

89. Section 3004(u) of RCRA, 42 U.S.C. § 6924(u), requires, in pertinent part, that each permit issued after November 8, 1984, by EPA or an authorized state, shall require corrective action from any solid waste management unit at the treatment, storage, or disposal facility seeking such permit, regardless of the time at which waste was placed in such unit.

90. Section 3004(v) of RCRA, 42 U.S.C. § 6924(v), requires, in pertinent part, that corrective action be taken beyond the facility boundary at a treatment, storage or disposal facility where necessary to protect human health and the environment.

91. 40 C.F.R. § 270.32(b)(2) and WVHWMR § 33-20-11.1, provide, in pertinent part, that each permit issued under RCRA § 3005 shall contain terms and conditions as EPA determines necessary to protect human health and the environment.

92. Part I, Section C of DuPont's's Corrective Action Permit states, in pertinent part, that pursuant to RCRA § 3005(c)(3), the permit contains those terms and conditions determined necessary to protect human health and the environment.

93. 40 C.F.R. § 270.30(a) and WVHWMR § 33-20-11.1, provide, in pertinent part, that a RCRA permittee must comply with all conditions of its permit, and that any permit noncompliance, except under the terms of an emergency permit, constitutes a violation of

RCRA and is grounds for an enforcement action; for permit termination, revocation and reissuance, or modification; or for denial of a permit renewal application.

94. Part 1, Section 1.1 of DuPont's's Corrective Action Permit requires Respondent to comply with all conditions of the permit, except to the extent and for the duration such noncompliance is authorized by an emergency permit. Any other permit noncompliance constitutes a violation of RCRA and is grounds for enforcement action, permit termination, revocation and reissuance, or modification, or for denial of a permit renewal application.

95. 40 C.F.R. § 270.30(f) and WVHWMR § 33-20-11.1, provide, in pertinent part, that the permittee shall furnish within a reasonable time, any relevant information that EPA may request to determine whether cause exists for modifying, revoking and reissuing, or terminating the permit, or to determine compliance with the permit.

96. Part 1, Section 1.7 of DuPont's's Corrective Action Permit requires, in pertinent part, that Respondent shall furnish, within the specified time, any relevant information that EPA may request to determine whether cause exists for modifying, revoking and reissuing, or terminating the permit, or to determine compliance with the permit.

97. The Corrective Action Permit for the Washington Works Facility generally requires DuPont's to perform the following: 1) a Verification Investigation (VI), including a VI Workplan and VI Report, to evaluate to what extent hazardous constituents have been released to the soil, surface water, and groundwater as a result of historic plant operations at Solid Waste Management Units (SWMUs) and to define SWMU areas of concern where additional data are needed to determine the extent of constituent migration; 2) a

RCRA Facility Investigation (RFI), including an RFI Workplan and RFI Report, for suspected releases from specific SWMUs at the Washington Works Facility, and 3) a Corrective Measure Study. All plans, reports, schedules, and other submissions required by the terms of EPA's portion of DuPont's's Corrective Action Permit are, upon approval by EPA, incorporated into the permit.

98. On or about December 14, 1990, DuPont submitted to EPA a revised VI Workplan. As required by Part II.B.1 of DuPont's's Corrective Action Permit, the VI Workplan is designed to, among other things, describe how the permittee will investigate the release of hazardous waste or hazardous constituents from all SWMUs and determine the need for further investigation and/or implementation of interim measures at the Washington Works Facility.

99. DuPont's's Verification Investigation was conducted in the winter of 1991 for the SWMUs at the Washington Works Facility. C-8 is one of the constituents that DuPont was required to investigate as part of the Verification Investigation.

100. On or about April 3, 1992, DuPont submitted to EPA a VI Report. As required by Part II.B.2 of DuPont's's Corrective Action Permit, the VI Report was to contain all data organized in a logical sequence and include, among other things, summaries of all findings, problems encountered during the investigation, actions taken to correct the problems, and copies of all daily reports, inspections reports, and laboratory/monitoring data. DuPont was also required to include in the VI Report, conclusions and recommendations.

101. C-8 (also referred to as PFOA or FC-143 as described in paragraph 6, above) is one of the constituents that DuPont detected as part of the Verification Investigation it performed at the Washington Works Facility, and included in its VI Report to EPA in April of 1992.

102. On or about May 5, 1997, EPA issued a Notice of Deficiency (Notice) to DuPont for the VI Report. In the Notice, EPA requested that DuPont provide a response to EPA, within 30 days of receipt, for all deficiencies identified in the Notice.

103. In the Groundwater portion of the Notice, EPA requested that DuPont provide to EPA "known toxicological information" regarding C-8.

104. In DuPont's Response to the Notice of Deficiency (Response to the Notice) on or about June 6, 1997, and in its specific response to EPA's request for "known toxicological information," DuPont directed EPA to information that was included in the VI Report, and provided "[a]dditional C-8 toxicological information" at Attachment 2 of the Response to Notice, titled "Toxicological Information on C-8."

105. The "Toxicological Information on C-8" included certain "Health Hazardous Data."

106. In the section regarding "Health Hazardous Data," DuPont did not provide EPA the human blood sampling information concerning the transplacental movement of PFOA that DuPont obtained in 1981 when performing blood sampling of pregnant workers at the Washington Works Facility.

107. Information regarding the transplacental movement of C-8 in humans is, and, at the time of EPA's Notice and DuPont's Response to the Notice was, "known toxicological information" about C-8.

108. Neither in its Response to the Notice in June 1997, nor at any other time, did DuPont provide to EPA the information regarding the transplacental movement of C-8 in humans.

109. In its Response to the Notice in June 1997, DuPont did not provide all "known toxicological information" it had regarding C-8 because it did not provide to EPA the information regarding the transplacental movement of C-8 in humans.

110. All known toxicological information about C-8 is "relevant information" that EPA might request "to determine whether cause exists for modifying, revoking and reissuing or terminating [DuPont's Corrective Action Permit,] or to determine compliance with this permit. Part I, Section I.7; 40 C.F.R. § 270.30(h); WVHWMR § 33-20-11.

111. DuPont's failure to provide this known toxicological information constitutes noncompliance with DuPont's duty to provide information, as required by Part 1, Section I.7 of DuPont's Corrective Action Permit, 40 C.F.R. § 270.30(h) and WVHWMR § 33-20-11.1

112. Because DuPont did not comply with this provision of its Corrective Action Permit to provide known toxicological information, DuPont did not comply with all conditions of its permit, as required by Part 1, Section I.1. of DuPont's Corrective Action Permit, 40 C.F.R. § 270.30(a), and WVHWMR § 33-20-11.1.

113. From at least June 6, 1997, until at least March 6, 2001, DuPont violated RCRA § 3005(a), 42 U.S.C. § 6925(a), Part 1, Section I.7 of DuPont's Corrective Action Permit, 40 C.F.R. § 270.30(h), and WVHWMR § 33-20-11.1, by failing to provide the known toxicological information on C-8 described above.

CIVIL PENALTY ASSESSMENT FOR COUNTS I-II

Section § 16 of TSCA, 15 U.S.C. § 2615, authorizes the assessment of a civil penalty for the violations described herein of $25,000 for each day of violation occurring and continuing before January 30, 1997, and up to $27,500 for each day of violation occurring and continuing after January 30, 1997,[6] to March 6, 2001, the date EPA received the TSCA § 8(e) information from a third-party.

Pursuant to 40 C.F.R. § 22.14(a)(4)(ii), Complainant is not proposing a specific penalty at this time, but will do so at a later date. See 40 C.F.R. § 22.19(a)(4). In determining the amount of a civil penalty for violations of TSCA, Complainant shall take into account the nature, circumstances, extent, and gravity of the violations alleged, as well as DuPont's ability to pay, effect on ability to continue to do business, any history of prior such violations, the degree of culpability, and such other matters as justice may require. See also Enclosure B to the original Complaint.

CIVIL PENALTY ASSESSMENT FOR COUNT III

Sections §§ 3008(a)(3) and (g) of RCRA, 42 U.S.C. §§ 6928(a)(3) and (g), authorize the assessment of a civil penalty for violations described herein of $25,000 for each day of violation occurring and continuing before January 30, 1997, and up to $27,500 for each day of violation

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[6] The Federal Civil Penalties Inflation Adjustment Act of 1990, as amended by the Debt Collection Improvement Act of 1996, requires EPA to periodically adjust penalties to account for inflation. EPA's Civil Monetary Penalty Inflation Adjustment Rule establishes $27,500 as the maximum civil penalty that may be assessed under TSCA § 16(a), per violation, between January 31, 1997, through May 15, 2004, and $32,500 for violations occurring thereafter. See 40 C.F.R. § 19, 61 Fed Reg. 69,360 (Dec. 31, 1996); 69 Fed. Reg. 7121 (Feb. 13, 2004).

occurring and continuing after January 30, 1997,[7] at least to March 6, 2001, the date EPA received the information regarding the transplacental movement of PFOA from a third-party.

Pursuant to 40 C.F.R. § 22.14(a)(4)(ii), Complainant is not proposing a specific penalty at this time, but will do so at a later date. See 40 C.F.R. § 22.19(a)(4). In determining the amount of a civil penalty for violations of RCRA pursuant to RCRA §§ 3008(a)(3) and (g), 42 U.S.C. §§ 6928(a)(3) and (g), Complainant shall take into account the seriousness of the violation and any good faith efforts by DuPont to comply with the applicable requirements. See also Enclosure C to the original Complaint

NOTICE OF OPPORTUNITY TO REQUEST A HEARING

As provided in TSCA § 16(a)(2)(A), 15 U.S.C. § 2615(a)(2)(A), and RCRA § 3008(b), 42 U.S.C. § 6928(b), you have the right to request a formal hearing to contest any material fact set forth in this Complaint or to contest the appropriateness of the penalty. To avoid being found in default, which constitutes an admission of all facts alleged in the Complaint and a waiver of the right to a hearing and having a penalty assessed without further proceedings, you must file a written Answer within thirty (30) days of receiving this Complaint.

Pursuant to the Consolidated Rules of Practice, your Answer must clearly and directly admit, deny, and/or explain each of the factual allegations contained in this Complaint with regard to which you have any knowledge. If you have no knowledge of a particular fact and so

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[7] The Federal Civil Penalties Inflation Adjustment Act of 1990, as amended by the Debt Collection Improvement Act of 1996, requires EPA to periodically adjust penalties to account for inflation. EPA's Civil Monetary Penalty Inflation Adjustment Rule establishes $27,500 as the maximum civil penalty that may be assessed under RCRA §§ 3008(a) and (g), per violation, between January 31, 1997, through May 15, 2004, and $32,500 for violations occurring thereafter. See 40 C.F.R. § 19, 61 Fed Reg. 69,360 (Dec. 31, 1996); 69 Fed. Reg. 7121 (Feb. 13, 2004).

state, the allegation is denied. Failure to deny any of the allegations in this Complaint will constitute an admission of the undenied allegation.

The Answer shall also state the circumstances and arguments, if any, which are alleged to constitute the grounds of defense and the basis for opposing any proposed penalty, and shall specifically request an administrative hearing if desired. EPA will consider, among other factors, DuPont's "ability to pay" to adjust the civil penalty to be assessed in this proceeding. For purposes of Count III, the burden of raising and demonstrating an inability to pay rests with DuPont. If you deny any material fact or raise any affirmative defense, you will be considered to have requested a hearing. The Answer must be filed with the:

Headquarters Hearing Clerk (1900L)
United States Environmental Protection Agency
1200 Pennsylvania Ave. N.W.
Washington, DC 20460

Please send a copy of the Answer and all other documents that you file in this action to the following attorneys assigned to represent EPA in this matter:

Mark Garvey, Attorney
Toxics and Pesticides Enforcement Division (2245A)
Office of Regulatory Enforcement
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460-0001
(202) 564-4168

Ilana Saltzbart, Attorney
Toxics and Pesticides Enforcement Division (2245A)
Office of Regulatory Enforcement
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460-0001

Any hearing requested will be conducted in accordance with the Administrative Procedures Act, 5 U.S.C. § 551 *et seq.*, and the Consolidated Rules of Practice. See Enclosure A to the original Complaint.

INFORMAL SETTLEMENT CONFERENCE

Whether or not you request a hearing, you may confer informally with EPA to discuss the facts of this case, or amount of the penalty, and the possibility of settlement. An informal settlement conference does not, however, affect your obligation to file a written Answer to the Complaint.

EPA has the authority, where appropriate, to modify the amount of the penalty to reflect any settlement reached with you in an informal conference. The terms of such an agreement would be embodied in a Consent Agreement and Final Order ("CAFO"). A CAFO signed by EPA and you would be binding as to all terms and conditions specified therein upon signature by the Environmental Appeals Board.

Please be advised that the Consolidated Rules of Practice prohibit any *ex parte* (unilateral) discussion of the merits of any action with the Administrator, Environmental Appeals Board Judge, Administrative Law Judge, or any person likely to advise these officials in the decision of the case, after the Complaint is issued.

By:

Date: 10-13-04

Walker B. Smith, Director
Office of Regulatory Enforcement
Office of Enforcement And Compliance Assurance
U.S. Environmental Protection Agency

UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY

IN THE MATTER OF:	
E. I. du Pont de Nemours and Company	Docket No. TSCA-HQ-2005-5001
Wilmington, DE	COMPLAINT AND NOTICE OF OPPORTUNITY FOR HEARING
Respondent	
Washington Works Facility Route 892 South DuPont Road Washington, Wood County, WV	

INTRODUCTION

This Complaint and Notice of Opportunity for Hearing ("Complaint") is filed pursuant to the Toxic Substances Control Act § 16(a), 15 U.S.C. § 2615(a), ("TSCA"), and the Consolidated Rules of Practice Governing the Administrative Assessment of Civil Penalties and the Revocation/Termination or Suspension of Permits ("Consolidated Rules of Practice"), 40 C.F.R. Part 22, a copy of which is enclosed with this Complaint. See Enclosure A. The Complainant is Ann M. Pontius, Director, Toxics & Pesticides Enforcement Division, Office of Regulatory Enforcement, Office of Enforcement and Compliance Assurance, United States Environmental Protection Agency ("EPA" or the "Agency"), who has been duly delegated the authority to institute this action. The Respondent is E. I. du Pont de Nemours and Company ("DuPont" or "Respondent"), 1007 Market Street, Wilmington, Delaware, a manufacturer, processor or distributor of chemical substances and mixtures in commerce.

This Complaint serves as notice that Complainant has reason to believe that Respondent failed to immediately submit information as required by TSCA § 8(e), 15 U.S.C. § 2607(e), thereby committing an unlawful act under TSCA § 15, 15 U.S.C. § 2614. Section 16 of TSCA authorizes EPA to take an enforcement action against any person that commits a prohibited action under TSCA.

In support of this Complaint, Complainant hereby makes the following allegations:

COMPLAINT

GENERAL ALLEGATIONS

1. Respondent owns and operates a manufacturing facility, known as Washington Works ("Washington Works Facility"), located at Route 892 South DuPont Road, Washington, Wood County, West Virginia, 26181. Respondent was the owner and operator of this facility at all times relevant to this Complaint.

2. Respondent "manufactures," "processes," or "distributes in commerce" a "chemical substance" or "mixture" as those terms are defined in TSCA § 3, 15 U.S.C. § 2602, and TSCA § 8(f), 15 U.S.C. § 2607(f).

3. Respondent is a person subject to the requirements of TSCA § 8(e), 15 U.S.C. § 2607(e).

4. At all times relevant to this Complaint, DuPont manufactured Ammonium Perfluorooctanoate ("APFO"), CAS No. 3825-26-1 (Octanoic acid, pentadecafluoro-, ammonium salt).[1]

[1] The 3M Company manufactured APFO and sold it to DuPont from 1951until 2002 under the tradename FC-143.

5. APFO is comprised of an ammonium cation and a perfluorooctanoic acid ("PFOA") anion.[2] The "oate" suffix of APFO is the nomenclature tool used to signify the anionic form of a carboxylic acid. The suffix "oic" of pure PFOA is used to signify the neutral protonated form of a carboxylic acid. The pure form of PFOA, CAS number 335-67-1, consists of the PFOA anion and its associated cation which is a proton (H+), which is thus different from the PFOA anion alone. In water or biologic media, APFO quickly dissociates to the ammonium cation and the PFOA anion.

6. When APFO is measured in humans or the environment, it is measured by its PFOA anion presence and not by the intact APFO. Because there cannot be APFO without the PFOA anion, and because APFO measured in humans or the environment is measured by the PFOA anion, a short-hand for discussing APFO is "PFOA." Consequently, reference to APFO, C-8, C8 or PFOA is a reference to the dissociated (anionic) form of PFOA and not the protonated form of PFOA with CAS No. 335-67-1.

7. EPA consistently uses APFO, C-8, and PFOA interchangeably as evidenced in the 2003 fact sheet, available to the public at www.epa.gov/opptintr/pfoa/pfoafcts.pdf, in which EPA stated that "[t]he 'PFOA' acronym is used to indicate not only perfluorooctanoic acid itself, but also its principal salts. The most commonly used chemical in this grouping is the ammonium salt, ammonium perfluorooctanoate or APFO, which is sometimes called 'C8'. "

8. While most major toxicological studies and industrial exposures involve APFO, the toxicological effects are likely related to the PFOA anion.

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[2] A synonym for this PFOA anion is "perfluorooctanoate."

9. Most animal toxicity studies have been conducted with APFO.

10. EPA has identified potential human health concerns from exposure to PFOA.

11. APFO is a perfluorinated detergent/surfactant manufactured, processed, or distributed in commerce in the United States by DuPont, in connection with its Teflon®-related products.

12. At all times relevant to this Complaint, Respondent manufactured, processed, or distributed in commerce APFO, and consequently, Respondent manufactured, processed, or distributed the PFOA anion associated with APFO.

13. Thus, at all times relevant to this Complaint, Respondent has manufactured, processed or distributed PFOA (i.e., the PFOA anion) at its Washington Works Facility.

14. PFOA is in the soil, groundwater, and drinking water at, and/or within the vicinity of, DuPont's Washington Works Facility.

15. PFOA is hepatotoxic (liver toxin) to animals.

16. PFOA is persistent in the environment.

17. PFOA is bioaccumulative in humans in that it has a half-life estimated at 4.4 years.

18. PFOA is associated with developmental effects in animals.

19. PFOA is believed to be present in the blood of the general population in all geographic regions of the U.S. As stated in the Agency's April 2003 Preliminary Risk Assessment, "[t]he highest serum PFOA levels of the general public were reported in a sample of children from different geographic regions in the U.S. (mean, 5.6 ppb [parts per billion]; range, 1.9 – 56.1 ppb)."

20. PFOA is not naturally occurring, thus all PFOA in human blood is attributable to human

activity. PFOA is produced synthetically and can be formed through the degradation or metabolism of other fluorochemical products, such as fluorinated telomers.

21. DuPont and other researchers have studied PFOA in lab animals.

22. There are gender differences in the elimination of PFOA in rats.

23. There are substantial differences in the half-life of PFOA in rats and humans.

24. There are considerable differences among species in the kinetics of PFOA.

25. In September 2002, the Director of the Office of Pollution Prevention and Toxics (OPPT) initiated a priority review of PFOA in all its forms. EPA published a Federal Register Notice, 68 Fed. Reg. 18,626 (April 16, 2003), as part of its effort to collect additional information. The Agency is interested in collecting information because certain studies indicated that PFOA causes developmental toxicity and other effects in laboratory animals. EPA's preliminary assessment, released April 10, 2003, indicates potential exposure of the U.S. general population to PFOA at very low levels. However, this risk assessment also reflects considerable scientific uncertainty regarding the potential risks.

26. TSCA § 2(a)(2), 15 U.S.C. § 2601(a)(2) states, "Findings - The Congress finds that - (2) among the many chemical substances and mixtures which are constantly being developed and produced, there are some whose manufacture, processing, distribution in commerce, use or disposal may present an unreasonable risk of injury to health or the environment."

27. TSCA § 2(b)(2), 15 U.S.C. § 2601(b)(2) and TSCA § 2(b)(3), 15 U.S.C. § 2601(b)(3) state, "Policy - It is the policy of the United States that - (2) adequate authority should exist to regulate chemical substances and mixtures which present an unreasonable risk of injury to health or the environment, and to take action with respect to chemical substances

and mixtures which are imminent hazards; and (3) authority over chemical substances and mixtures should be exercised in such a manner as to not impede unduly or create unnecessary economic barriers to technological innovation while fulfilling the primary purpose of this Act to assure that such innovation and commerce in such chemical substances and mixtures do not present an unreasonable risk of injury to health or the environment."

28. TSCA § 8(e), 15 U.S.C. § 2607(e), provides that, "Any person who manufactures, processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment shall immediately inform the Administrator of such information unless such person has actual knowledge that the Administrator has been adequately informed of such information."

Count I - Results of PFOA Serum Testing

29. Complainant re-alleges paragraphs 1 through 28, above, as if fully set forth below.

30. On or about September 15, 2004, Robert A. Bilott, an attorney representing plaintiffs in litigation against DuPont for APFO/PFOA contamination of drinking water in West Virginia and Ohio, submitted a letter to EPA containing "the results of PFOA exposed community serum sampling" performed by DuPont and its contractor, Exygen.

31. Mr. Bilott first received the results of this community serum sampling from DuPont, or an agent for DuPont, on or around August 5, 2004.

32. Specifically, the letter describes the results of a DuPont serum sampling of twelve members of the general population living near the Washington Works Facility. The letter

claims that all twelve of the individuals tested were exposed to PFOA through drinking water provided by the Lubeck Public Service District (LPSD), where according to DuPont, the level of PFOA in the drinking water has averaged approximately 0.5 parts per billion (ppb) over the last several years.

33. The letter from Mr. Bilott states that all twelve of the individuals tested claim to have stopped using the contaminated public drinking water as their primary source of drinking water approximately three years ago.

34. The serum sampling consisted of five females and seven males, of which only one, a seventy-year old male, had previously worked at the Washington Works Facility.

35. Human serum sample levels of PFOA for these 12 individuals were reported to range from 15.7 ppb to 128 ppb, with a mean of 67 ppb. The median value is in the range of 60 ppb PFOA. As stated above, the average background serum level of PFOA in individuals residing in the United States is estimated to be approximately 5 ppb.

36. These human serum sample levels of PFOA for these 12 individuals represent the first human serum sampling results the Agency has seen concerning individuals exposed in a community setting.

37. DuPont failed or refused to submit to EPA the data concerning human serum sampling of twelve members of the general population living near the Washington Works Facility after it had obtained this information from its contractor, Exygen.

38. The human serum sampling data are particularly useful because they represent an attempt to associate body burden in the general population with a specific exposure pathway and a source of exposure. This data is information that reasonably supports the conclusion that

PFOA presents a substantial risk of injury to human health that the Administrator was not already adequately informed about at the time the information was obtained by DuPont or at any time prior to the date EPA received the data.

39. The Agency considers the human serum sampling information to reasonably support the conclusion of a substantial risk of injury to health or the environment. The Administrator was not adequately informed about this risk at the time the information was obtained by DuPont.

40. DuPont obtained this information on or after July 29, 2004 but no later than August 5, 2004, the date at which DuPont transmitted this information to Mr. Bilott, as described in Paragraph 31, above.

41. DuPont was required to immediately inform EPA about the human serum sampling data under TSCA § 8(e), 15 U.S.C. § 2607(e), as information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health unless DuPont had actual knowledge that the Administrator had been adequately informed of the serum data.

42. DuPont failed or refused to immediately inform the Administrator about the community human serum sampling information.

43. DuPont became aware on or around October 12, 2004, that the Administrator had been informed about this human serum sampling data.

44. TSCA § 15(3)(B), 15 U.S.C. § 2614(3)(B), provides that it is unlawful for any person "to fail or refuse to submit reports, notices, or other information" required by TSCA.

45. DuPont's failure to immediately inform EPA about the information concerning human

serum sampling from individuals exposed to PFOA in a community setting constitutes a violation of TSCA § 8(e), 15 U.S.C. § 2607(e).

46. DuPont's failure or refusal to submit the human serum sampling information as required under TSCA § 8(e) is an unlawful act under TSCA § 15(3)(B).

CIVIL PENALTY ASSESSMENT

Section 16 of TSCA, 15 U.S.C. § 2615, authorizes the assessment of a civil penalty for the violations described herein of $32,500 for each day of violation.[3] In determining the amount of a civil penalty for violations of TSCA, Complainant shall take into account the nature, circumstances, extent, and gravity of the violations alleged, as well as Respondent's ability to pay, effect on ability to continue to do business, any history of prior such violations, the degree of culpability, and such other matters as justice may require. See also Enclosure B. Pursuant to 40 C.F.R. § 22.14(a)(4)(ii), Complainant is not proposing a specific penalty at this time, but will do so at a later date. See 40 C.F.R. § 22.19(a)(4).

NOTICE OF OPPORTUNITY TO REQUEST A HEARING

As provided in TSCA § 16(a)(2)(A), 15 U.S.C. § 2615(a)(2)(A), you have the right to request a formal hearing to contest any material fact set forth in this Complaint or to contest the

[3] The Federal Civil Penalties Inflation Adjustment Act of 1990, as amended by the Debt Collection Improvement Act of 1996, requires EPA to periodically adjust penalties to account for inflation. EPA's Civil Monetary Penalty Inflation Adjustment Rule establishes $32,500 for violations occurring after March 15, 2004. See 69 Fed. Reg. 7121 (Feb. 13, 2004).

appropriateness of the penalty. To avoid being found in default, which constitutes an admission of all facts alleged in the Complaint and a waiver of the right to a hearing and having a penalty assessed without further proceedings, you must file a written Answer within thirty (30) days of receiving this Complaint.

Pursuant to the Consolidated Rules of Practice, your Answer must clearly and directly admit, deny, and/or explain each of the factual allegations contained in this Complaint with regard to which you have any knowledge. If you have no knowledge of a particular fact and so state, the allegation is denied. Failure to deny any of the allegations in this Complaint will constitute an admission of the undenied allegation.

The Answer shall also state the circumstances and arguments, if any, which are alleged to constitute the grounds of defense and the basis for opposing any proposed penalty, and shall specifically request an administrative hearing if desired. EPA will consider, among other factors, Respondent's "ability to pay" to adjust the civil penalty to be assessed in this proceeding. If you deny any material fact or raise any affirmative defense, you will be considered to have requested a hearing. The Answer must be filed with the:

Headquarters Hearing Clerk (1900L)
United States Environmental Protection Agency
1200 Pennsylvania Ave. N.W.
Washington, DC 20460

Please send a copy of the Answer and all other documents that you file in this action to the following attorneys assigned to represent EPA in this matter:

Mark Garvey, Attorney
Toxics and Pesticides Enforcement Division (2245A)
Office of Regulatory Enforcement

U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460
(202) 564-4168

Ilana Saltzbart, Attorney
Toxics and Pesticides Enforcement Division (2245A)
Office of Regulatory Enforcement
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460
(202) 564-9935

Any hearing requested will be conducted in accordance with the Administrative Procedures Act, 5 U.S.C. § 551 *et seq.*, and the Consolidated Rules of Practice. See Enclosure A.

INFORMAL SETTLEMENT CONFERENCE

Whether or not you request a hearing, you may confer informally with EPA to discuss the facts of this case, or amount of the penalty, and the possibility of settlement. An informal settlement conference does not, however, affect your obligation to file a written Answer to the Complaint.

EPA has the authority, where appropriate, to modify the amount of the penalty to reflect any settlement reached with you in an informal conference. The terms of such an agreement would be embodied in a Consent Agreement and Final Order ("CAFO"). A CAFO signed by EPA and you would be binding as to all terms and conditions specified therein upon signature by the Environmental Appeals Board.

Please be advised that the Consolidated Rules of Practice prohibit any *ex parte* (unilateral) discussion of the merits of any action with the Administrator, Environmental Appeals

Board Judge, Administrative Law Judge, or any person likely to advise these officials in the decision of the case, after the Complaint is issued.

By:

Ann M Pontius

Date: Dec. 6, 2004

Ann M. Pontius, Director
Toxics & Pesticides Enforcement Division
Office of Regulatory Enforcement
Office of Enforcement And Compliance Assurance
U.S. Environmental Protection Agency

ENCLOSURE

A - Consolidated Rules of Practice - 40 C.F.R. Part 22

B - TSCA Enforcement Response Policies

C - Notice of Securities and Exchange Commission Registrants' Duty to Disclose Environmental Legal Proceedings

CERTIFICATION

I hereby certify that the original of the foregoing Complaint and Notice of Opportunity for Hearing, Docket Nos. TSCA-HQ-2005-5001 has been filed with the Headquarters Hearing Clerk and that copies were sent:

by certified mail, return receipt requested to both parties below

and by fax without enclosures to:

Stacey J. Mobley
Senior Vice President, General Counsel, and Chief Administrative Officer
DuPont
1007 Market Street
Room D-7038
Wilmington, Delaware 19898
fax: 302 773-4679

Peter D. Roberston
Patton Boggs, LLP
2550 M Street, NW
Washington, DC 20037
fax: 202 457-6315

Brenda F. Mosley

Brenda F. Mosley, Ph.D. (~~2245A~~)
Toxics and Pesticides Enforcement Division
Office of Regulatory Enforcement
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W..
Washington, DC 20460

12-6-04
Date

UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY

BEFORE THE ADMINISTRATOR

IN THE MATTER OF	)	
	)	
E. I. du Pont de Nemours	)	**DOCKET NOS. TSCA-HQ-2004-0016**
and Company,	)	**RCRA-HQ-2004-0016**
	)	**TSCA-HQ-2005-5001**
RESPONDENT	)	

ORDER DENYING MOTIONS FOR ACCELERATED DECISION ON COUNTS II AND III
ORDER SETTING PREHEARING EXCHANGE SCHEDULE FOR COUNTS II, III, AND IV

Procedural Background

The complainant in this matter is the Office of Civil Enforcement[1] ("OCE" or "Complainant") of the United States Environmental Protection Agency ("the EPA"). OCE contends that Respondent, E.I. du Pont de Nemours and Company ("DuPont" or "Respondent"), committed violations of the Toxic Substances Control Act ("TSCA") and Resource Conservation and Recovery Act ("RCRA"). On July 8, 2004, OCE filed its first complaint in this matter, the Complaint and Notice of Opportunity for Hearing ("Complaint"), under docket numbers TSCA-HQ-2004-0016 and RCRA-HQ-2004-0016, to which DuPont filed its Answer and Request for Hearing ("Answer").

OCE alleges, in Counts I and II, that DuPont violated Section 8(e) of TSCA, which provides that:

> Any person who manufactures, processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment shall immediately inform the [EPA]

[1] The Office of Civil Enforcement is the new name for the Office of Regulatory Enforcement. Notice of Office Name Change (Feb. 17, 2005).

> Administrator of such information unless such person has actual knowledge that the [EPA] Administrator has been adequately informed of such information.

15 U.S.C. § 2607(e). Specifically, OCE alleges in Count I failure to provide blood sampling information regarding transplacental movement of perflurooctanoic acid ("PFOA") in humans, and alleges in Count II failure to report PFOA contamination of the public water supply. In Count III, brought pursuant to Section 3008 the Resource Conservation and Recovery Act ("RCRA"), 42 U.S.C. § 6928, OCE alleges that DuPont violated its RCRA permit by failing to provide blood sampling information concerning the transplacental movement of PFOA (also referred to as "C-8" or ammonium perfluorooctanoate ("APFO")) in humans.

On September 8, 2004, DuPont filed a Motion for Accelerated Decision on Counts II and III ("DuPont's Motion for Acc. Dec.") and requested oral argument on that motion. Shortly after DuPont moved for accelerated decision, OCE moved to amend its Complaint, to replace it with the First Amended Complaint and Notice of Opportunity for Hearing ("Amended Complaint"), and I granted that motion. DuPont filed its Answer to First Amended Complaint and Request for Hearing ("Amended Answer"). On October 8, 2004, OCE filed a response to DuPont's Motion for Accelerated Decision, and OCE also moved for accelerated decision, only as to Count III. *See* Complainant's Mem. of Law in Support of Its Response to Respondent's Motion for Accelerated Decision on Count II ("OCE's Count II Response"); Complainant's Mem. of Law in Support of: Response to Respondent's Motion for Accelerated Decision, and Motion for Accelerated Decision on Liability for Count III ("OCE's Count III Response"). DuPont filed reply briefs as to both Counts II and III. *See* DuPont's Reply Brief in Support of Its Motion for Accelerated Decision on Count II (Oct. 19, 2004) ("DuPont's Count II Reply"); DuPont's Reply Mem. in Support of Its Motion for Accelerated Decision on Count III and Mem. in Opposition to EPA's Motion for Accelerated Decision on Count III (Nov. 16, 2004) ("DuPont's Count III Reply"). Thereafter, OCE filed its reply brief as to Count III. *See* Complainant's Reply in Support of Complainant's Motion for Accelerated Decision on Count III (Dec. 13, 2004) ("OCE's Count III Reply").

On December 6, 2004, the EPA filed an additional Complaint against DuPont, under Docket Number TSCA-HQ-2005-5001, which brought a TSCA count titled "Results of PFOA Serum Testing." In the latter count, OCE alleges failure or refusal to submit to the EPA data concerning human serum sampling of twelve members of the general population living near the Washington Works Facility, which DuPont obtained on or after July 29, 2004 but no later than August 5, 2004. DuPont filed an answer to the latter count. OCE moved to consolidate the new Complaint with the pending action, and I granted consolidation.[2]

[2] Now that the two complaints have been consolidated, the TSCA count titled "Results of PFOA Serum Testing" shall be referred to as Count IV.

On December 16, 2004, I heard oral arguments on the parties' motions for accelerated decision on Counts II and III.[3] Thereafter, I issued an order directing post-argument briefs to be submitted no later than February 4, 2005, that post-argument briefs should focus on issues raised at the oral argument, and that reply briefs would not be accepted.[4] Order Setting Briefing Schedule (Dec. 28, 2004). The parties filed their post-oral argument briefs on February 4, 2005. *See* DuPont's Post-Argument Brief on Pending Motions for Accelerated Decision ("DuPont's Post-Argument Br."); Complainant's Post-Argument Briefs on Counts II and III ("OCE's Post-Argument Br.").

Standard for Adjudicating a Motion for Accelerated Decision

Section 22.20(a) of the Rules of Practice[5] authorizes the Administrative Law Judge to "render an accelerated decision in favor of a party as to any or all parts of the proceeding, without further hearing or upon such limited additional evidence, such as affidavits, as he may require, if no genuine issue of material fact exists and a party is entitled to judgment as a matter of law." 40 C.F.R. § 22.20(a).

Motions for accelerated decision under 40 C.F.R. § 22.20(a) are akin to motions for summary judgment under Rule 56 of the Federal Rules of Civil Procedure ("FRCP"). *See, e.g., BWX Technologies, Inc.*, RCRA (3008) Appeal No. 97-5, 9 E.A.D. 61, 74-75 (EAB 2000); *In the Matter of Belmont Plating Works*, Docket No. RCRA-5-2001-0013, 2002 EPA ALJ LEXIS 65 at *8 (ALJ, Sept. 11, 2002). Rule 56(c) of the FRCP provides that summary judgment "shall be rendered forthwith if the pleadings, depositions, answers to interrogatories, and admissions on file, together with the affidavits, if any, show that there is no genuine issue of any material fact and that the moving party is entitled to a judgment as a matter of law." Therefore, federal court decisions interpreting Rule 56 provide guidance for adjudicating motions for accelerated decision. *See CWM Chemical Service*, TSCA Appeal 93-1, 6 E.A.D. 1 (EAB 1995).

The United States Supreme Court has held that the burden of showing that no genuine issue of material fact exists is on the party moving for summary judgment. *Adickes v. S. H. Kress & Co.*, 398 U.S. 144, 157 (1970). In considering such a motion, the Tribunal must construe the evidentiary material and reasonable inferences drawn therefrom in the light most favorable to the non-moving party. *See Anderson v. Liberty Lobby, Inc.*, 477 U.S. 242, 255 (1985); *Adickes*, 398 U.S. at 158-59; *see also Cone v. Longmont United Hospital Assoc.*, 14 F.3d 526, 528 (10th Cir. 1994). Summary judgment on a matter is inappropriate when

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[3] The oral arguments took place in Washington, D.C., in the EPA Administrative Courtroom.

[4] Accordingly, I need not consider reply briefs or similar filings, such as motions for clarification, filed after February 4, 2005, in response to the post-argument briefs.

[5] Consolidated Rules of Practice Governing the Administrative Assessment of Civil Penalties and the Revocation/Termination or Suspension of Permits.

contradictory inferences may be drawn from the evidence. *Rogers Corp. v. EPA*, 275 F.3d 1096, 1103 (D.C. Cir. 2002).

In assessing materiality for summary judgment purposes, the Supreme Court has determined that a factual dispute is material where, under the governing law, it might affect the outcome of the proceeding. *Anderson*, 477 U.S. at 248; *Adickes*, 398 U.S. at 158-159. The substantive law involved in the proceeding identifies which facts are material. *Id.*

The Supreme Court has found that a factual dispute is genuine if the evidence is such that a reasonable finder of fact could return a verdict in favor of the non-moving party. *Id.* In determining whether a genuine issue of fact exists, the judge must decide whether a finder of fact could reasonably find for the non-moving party under the evidentiary standards in a particular proceeding. *Anderson*, 477 U.S. at 252.

Once the party moving for summary judgment meets its burden of showing the absence of genuine issues of material fact, Rule 56(e) requires the opposing party to offer countering evidentiary material or to file a Rule 56(f) affidavit. Under Rule 56(e), "When a motion for summary judgment is made and supported as provided in this rule, an adverse party may not rest upon the mere allegations or denials of his pleading, but must set forth specific facts showing there is a genuine issue for trial." The Supreme Court has found that the non-moving party must present "affirmative evidence" and that it cannot defeat the motion without offering "any significant probative evidence tending to support" its pleadings. *Anderson*, 477 U.S. at 256 (quoting *First Nat'l Bank of Arizona v. Cities Service Co.*, 391 U.S. 253, 290 (1968)).

More specifically, the Court has ruled that the mere allegation of a factual dispute will not defeat a properly supported motion for summary judgment, as Rule 56(e) requires the opposing party to go beyond the pleadings. *Celotex Corp. v. Catrett*, 477 U.S. 317 at 322 (1986); *Adickes*, 398 U.S. at 160. Similarly, a simple denial of liability is inadequate to demonstrate that an issue of fact does indeed exist in a matter. *In the Matter of Strong Steel Products*, Docket Nos. RCRA-05-2001-0016, CAA-05-2001-0020, and MM-05-2001-0006, 2002 EPA ALJ LEXIS 57 at *22 (ALJ, September 9, 2002). A party responding to a motion for accelerated decision must produce some evidence which places the moving party's evidence in question and raises a question of fact for an adjudicatory hearing. *Id.* at 22-23; *see In re Bickford, Inc.*, Docket No. TSCA-V-C-052-92, 1994 TSCA LEXIS 90 (ALJ, November 28, 1994).

The Supreme Court has noted, however, that there is no requirement that the moving party support its motion with affidavits negating the opposing party's claim or that the opposing party produce evidence in a form that would be admissible at trial in order to avoid summary judgment. *Celotex*, 477 U.S. at 323-324. The parties may move for summary judgment or successfully defeat summary judgment without supporting affidavits provided that other evidence referenced in Rule 56(c) adequately supports its position. Of course, if the moving party fails to carry its burden to show that it is entitled to summary judgment under established principles, then no defense is required. *Adickes*, 398 U.S. at 156.

The evidentiary standard of proof in the matter before me, as in all other cases of administrative assessment of civil penalties governed by the Rules of Practice, is a "preponderance of the evidence." 40 C.F.R. § 22.24. In determining whether or not there is a genuine factual dispute, I, as the judge and finder of fact, must consider whether I could reasonably find for the non-moving party under the "preponderance of the evidence" standard.

Accordingly, a party moving for accelerated decision must establish through the pleadings, depositions, answers to interrogatories, and admissions on file, together with any affidavits, the absence of genuine issues of material fact and that it is entitled to judgment as a matter of law by the preponderance of the evidence. On the other hand, a party opposing a properly supported motion for accelerated decision must demonstrate the existence of a genuine issue of material fact by proffering significant probative evidence from which a reasonable presiding officer could find in that party's favor by a preponderance of the evidence. Even if a judge believes that summary judgment is technically proper upon review of the evidence in a case, sound judicial policy and the exercise of judicial discretion permit a denial of such a motion for the case to be developed fully at trial. *See Roberts v. Browning*, 610 F.2d 528, 536 (8th Cir. 1979).

DISCUSSION

I. Count II

A. The Alleged Groundwater Notification Violation

DuPont admits that it has owned and operated a manufacturing facility, known as Washington Works in Washington, West Virginia at all times relevant to this matter. Amended Answer ¶ 1. DuPont further admits that it manufactured, processed, or distributed in commerce a chemical substance or mixture as those terms are defined in Section 3 of TSCA, 15 U.S.C. § 2602, and Section 8(f) of TSCA, 15 U.S.C. § 2607(f). Amended Answer ¶ 2. DuPont admits that it used ammonium perfluorooctanoate ("APFO") as a processing aid at its Washington Works Facility. *Id.* ¶ 13. It is undisputed that APFO is composed of an ammonium cation and a perfluorooctanoate acid ("PFOA") anion. *Id.* ¶ 5. Furthermore, when in contact with water, APFO disassociates to: (1) the PFOA anion; and (2) the ammonium cation. *Id.* ¶ 13. DuPont refers to APFO as "C-8." *Id.* ¶ 4.

DuPont admits that when analytical chemists test blood or environmental media for APFO, they generally estimate the level of APFO present by testing for the concentration of the anion, PFOA. *Id.* ¶ 6. Therefore, test results may purport to measure levels of APFO, C-8, or PFOA in blood or water, but actually measure only PFOA. *Id.* DuPont admits that the Washington Works facility has released PFOA into the air, treated water containing PFOA in anaerobic digestion ponds, disposed of water containing PFOA into landfills, and discharged PFOA into the Ohio River. *Id.* ¶ 14.

DuPont admits that at high enough doses and durations of exposure, PFOA has been shown to produce liver toxicity in some test animals, and that at lower doses can produce such toxicity through a process known as induction of peroxisome proliferation. *Id.* ¶ 15. However, DuPont states that humans are not susceptible to peroxisome proliferation. *Id.* DuPont admits that PFOA is "biopersistent" in animals and humans, as well as "bioaccumulative" in humans, based on DuPont's understanding of those terms. *Id.* ¶¶ 16-17. DuPont further admits that, based on current knowledge, PFOA is not naturally occurring, that all PFOA present in human blood is attributable in some sense to human activity, and that PFOA is produced synthetically.[6] *Id.* ¶ 20.

Under Count II, titled "Public Water Supply Contamination," OCE alleges that on or about June 6, 1991, DuPont set a Community Exposure Guideline for drinking water ("CEGw") at 1 microgram per liter ("1 µg/L" or "1 ppb")[7] for PFOA, and that in June of 1991, DuPont's Washington Works Facility was aware of the 1 ppb CEGw that had been established for PFOA. Amended Complaint ¶ 68. In contrast, DuPont contends that on or about June 6, 1991, DuPont's acceptable exposure level committee set a provisional CEGw for PFOA at 1 microgram per liter, and that DuPont did not adopt the provisional CEGw for PFOA in water until on or about February 7, 1992.[8] Amended Answer ¶ 68.

OCE alleges that at the time DuPont adopted a CEGw at 1 ppb, it had collected results from drinking water samples, documented in various memorandums, and had information regarding the level of PFOA detected in such samples. Amended Complaint ¶ 69. In response, DuPont states that the documents to which OCE refers are the best evidence of their contents, and to the extent that OCE's allegations do not accurately state the contents of that document, those allegations are denied. Amended Answer ¶ 69.

OCE alleges that the EPA was not informed at the time DuPont obtained monitoring data showing "contamination" of the public water supply prior to 1991, and subsequent to that time. Amended Complaint ¶ 80. OCE alleges that DuPont was required under Section 8(e) of TSCA to immediately report the information concerning DuPont's monitoring data of the "contamination" of the public water supply for the communities in the vicinity of its Washington Works Facility and this obligation continued as DuPont learned more about the contamination. *Id.* ¶ 81. Finally, OCE alleges that DuPont was required under Section 8(e) of TSCA to inform the EPA every day between July 24, 1991 and March 6, 2001 (when the EPA received

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[6] In response to my question at the oral argument, "[I]s the EPA alleging human health effects, or is it strictly an environmental media," OCE stated, "Count II is strictly the environmental contamination data that DuPont became aware of in mid to late 1991 . . . ." Oral Arg. Tr. at 62.

[7] The acronym "ppb" means "parts per billion," and "µg/L" means micrograms per liter.

[8] The dispute of fact about the CEGw is not determinative for purposes of this order on DuPont's motion for accelerated decision.

information about the alleged contamination) about the information that it had obtained on the "widespread contamination" of public drinking water at a level greater than its CEGw, and that DuPont was required to inform the EPA immediately about information concerning the PFOA "contamination" of public drinking water that DuPont obtained in 1984. *Id.* ¶¶ 82-83. DuPont denies that the information in question reasonably supports any conclusion of substantial risk, and moreover, denies that the *EPA* considers the information at issue to reasonably support the conclusion of a substantial risk of injury to health or the environment. Amended Answer ¶¶ 78, 80.

As alleged in Count II, on or about June 6, 1991, DuPont set its community exposure guideline for drinking water at 1 part per billion ("ppb"). Oral Arg. Tr. at 70. OCE further alleges that on June 23, 1991, DuPont detected PFOA in a new well in Lubeck, which was approximately 2.7 miles from DuPont's Washington Works Facility. *Id.* According to OCE, "on June 26, 1991, DuPont began analyzing its water contamination data collected, admittedly, from '84 until '91 to decide whether or not to report to the [EPA] under TSCA 8(e)." *Id.* at 70-71. DuPont allegedly found that there had been levels of PFOA in wells, with one of the samples reading 3.9 ppb. *Id.* at 71; *see* OCE's Count II Response, Ex. 23. However, according to OCE, DuPont decided that no Section 8(e) notification was warranted. Oral Arg. Tr. at 71. OCE submits that "Where EPA's Count II comes into play is in two more dates, September 11, 1991, and November [19], 1991."[9] *Id.*; *see also id.* at 62.[10] On September 11, 1991, DuPont allegedly had a meeting and discussed all prior water sampling events in the context with what was going in mid to late 1991 terms of DuPont's dealing with the Lubeck Water Authority. *Id.* at 71 (referring to OCE's Count II Response, Ex. 23); *see also* OCE's Count II Response at 11. In its pre-argument brief, OCE contends that DuPont took additional water samples on November 19, 1991, with levels above the alleged CEGw level of 1 ppb and that a November memorandum reports these results. OCE's Count II Response at 12 (citing OCE's Count II Response, Ex. 24).

DuPont moves for accelerated decision and for dismissal of Count II on the ground that OCE is barred from bringing such an enforcement action as a matter of law by the parties' prior consent agreement and a consent order entered into as part of the TSCA § 8(e) Compliance Audit Program. DuPont's Motion for Acc. Dec. at 2.

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[9] *See* OCE's Count II Response, Exs. 23 and 24.

[10] In response to my question, "[I]s the EPA alleging human health effects, or is it strictly an environmental media," OCE stated, "Count II is strictly the environmental contamination data that DuPont became aware of in mid to late 1991 and withheld from the [EPA], Your Honor. It does build on prior data, some data points that may have preceded 1991." Oral Arg. Tr. at 62.

B. Introduction to Section 8(e) of TSCA and the TSCA Section 8(e) Compliance Audit Program

Section 8(e) of TSCA became effective on January 1, 1977. DuPont points out that Congress did not grant the EPA any rulemaking authority with respect to Section 8(e), nor did it grant the EPA any general rulemaking authority under TSCA. *Id.* at 7; *see* TSCA § 8(e), 42 U.S.C. § 2607(e). Thus, in 1977 the EPA proposed guidance on its interpretation of and policy concerning the provisions of Section 8(e) and solicited and received comments. 43 Fed. Reg. 11,110 (Mar. 16, 1978). On March 16, 1978 the EPA published a Statement of Interpretation of Enforcement Policy for Notification of Substantial Risk Under Section 8(e) ("1978 Enforcement Policy"), which the EPA published in the Federal Register. *Id.*

The 1978 Enforcement Policy provides that "A 'substantial risk of injury to health or the environment' is a risk of considerable concern because of (a) the seriousness of the effect [see Subparts (a), (b), and (c) below for an illustrative list of effects of concern], and (b) the fact or probability of its occurrence." *Id.* at 11,111. 1978 Enforcement Policy, Part V (brackets in original). For purposes of determining what constitutes substantial risks, Part V of the 1978 Enforcement Policy categorizes effects for which substantial-risk information must be reported under three main categories: (a) "human health effects," (b) "environmental effects," and (c) "emergency incidents of environmental contamination." *Id.* at 11,112. The 1978 Enforcement Policy further subcategorizes those effects. *Id.* Subcategory (b)(1) is "widespread and previously unsuspected distribution in environmental media, as indicated in studies (excluding materials contained within appropriate disposal facilities)." *Id.* Subcategories (b)(2)-(5) include the following environmental effects: (b)(2) "Pronounced bioaccumulation. Measurements of indicators of pronounced bioaccumulation heretofore unknown to the [EPA] Administrator . . . should be reported when coupled with potential for widespread exposure and any non-trivial adverse effect"; (b)(3) "Any non-trivial adverse effect, heretofore unknown to the [EPA] Administrator, associated with a chemical known to have bioaccumulated to a pronounced degree or to be widespread in environmental media"; (b)(4) "Ecologically

significant changes in species' interrelationships . . . ," and; (b)(5) "Facile transformation or degradation to a chemical having an unacceptable risk . . . ." *Id.*

On February 1, 1991, the EPA announced the opportunity to register for the TSCA Section 8(e) Compliance Audit Program ("CAP"). 56 Fed. Reg. 4,127, 4,128. The CAP called for registrants to audit and report for Section 8(e) information, provided for stipulated penalties for each study or report submitted pursuant to the CAP, and set an overall limit on penalties to be assessed pursuant to the CAP.

On June 20, 1991, the EPA announced suspension of Part V(b)(1) ("widespread and previously unsuspected distribution in environmental media, as indicated in studies (excluding materials contained within appropriate disposal facilities)") and Part V(c) ("emergency incidents of environmental contamination") of the 1978 Enforcement Policy. 56 Fed. Reg. 28,458, 28,459. The EPA stated that, despite the suspension of V(b)(1) and V(c) of the 1978 Enforcement Policy, "regulatees auditing their files for reportable environmental risk information under the TSCA Section 8(e) Compliance Audit Program should be guided by the statutory language of section 8(e) and Part V(b)(2) through (b)(5) of the [1978 Enforcement Policy]." *Id.* Moreover, "In assessing whether information or studies involving widespread and previous unsuspected environmental distribution, emergency incidents of environmental contamination, or other previously unknown situations involving significant environmental contamination should be submitted under the TSCA Section 8(e) Compliance Audit Program, or under section 8(e) in general, regulatees should make a reasonable judgement whether such information meets the statutory standards of TSCA section 8(e) instead of relying on Parts V(b)(1) or V(c) of the [1978 Enforcement Policy]." *Id.* EPA's June 1991 Federal Register notice concluded, "Even though EPA is suspending the applicability of Parts V(b)(1) and V(c) of the [1978 Enforcement Policy], persons are still responsible under TSCA section 8(e) to report information that reasonably supports a conclusion of substantial risk of injury to the environment. This is a continuing statutory obligation." *Id.*

On or about July 5, 1991. DuPont registered for the TSCA Section 8(e) CAP by signing the Registration and Agreement for TSCA Section 8(e) Compliance Audit Program ("CAP Agreement") *See* DuPont's Motion for Acc. Dec., Ex. 12, Attach. A.

On September 30, 1991, the EPA split the CAP into two phases. 56 Fed. Reg. 49,478, 49,479. It announced, "Because refinement of guidance on reportability of information on chemical release/detection in environmental media is underway, EPA is extending the reporting deadline for reporting such information under the TSCA Section 8(e) CAP to 6 months after publication of final reporting guidance." *Id.* According to the parties' Consent Agreement, on or about January 31, 1992, the EPA mailed an "Addendum" to DuPont to modify the CAP Agreement "only regarding the reporting of information on the release of chemical substances to and detection of chemical substances in all environmental media." DuPont's Motion for Acc. Dec., Ex. 12 (Consent Agreement, Docket No. TSCA-96-H-47 (Oct. 1, 1996) ("Consent Agreement"), Part I.C.

DuPont and the EPA subsequently agreed to a Revised Addendum to the TSCA Section 8(e) CAP Agreement ("Revised Addendum"), dated June 27, 1996, which sets forth the waiver of enforcement action at issue in this matter.[11] *See* Consent Agreement, Attach. B. Part IV.A of the Revised Addendum reads:

> Information on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, that predates the effective date of the final revised guidance will not be the subject of an EPA TSCA section 8(e) penalty enforcement action.

On October 1, 1996, the parties signed a Consent Agreement, which incorporates the terms of the CAP Agreement and the Revised Addendum. The EPA Environmental Appeals Board ("EAB") then executed a Consent Order, approving the Consent Agreement. *Id.*, Ex. 13 ("Consent Order," Docket Number TSCA-96-H-47 (Oct. 3, 1996)).

C. Introduction to the Parties' Arguments

In summary, DuPont argues that the charges in Count II are barred by the CAP Agreement entered into by DuPont and the EPA, as amended by the Revised Addendum, which were incorporated into the Consent Agreement signed by the parties and approved by the EAB in the Consent Order. Specifically, DuPont argues that under the Revised Addendum, dated June 27, 1996, the EPA clearly and unambiguously promised not to bring a Section 8(e) enforcement action based on information that existed prior to the effective date of the final revised guidance on the reportability of Section 8(e) information, which was published in the Federal Register on June 3, 2003.[12] DuPont asserts that the information on which Count II is based existed prior to the final revised guidance. Therefore, DuPont argues, the EPA is barred from enforcing the alleged violations under Count II.

OCE counters that DuPont oversimplifies the matter by highlighting only limited language of the Revised Addendum that supports its argument, and that when the language of the Revised Addendum and the CAP is viewed in whole it is apparent that DuPont's assertions are false. OCE contends that the CAP instituted a backwards-looking audit of limited duration to resolve past compliance. Specifically, OCE variously contends that the EPA waived its ability to

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[11] The Revised Addendum states that the Revised Addendum supersedes the original Addendum to the CAP Agreement ("Addendum"). According to the parties' Consent Agreement, on or about January 31, 1992, the EPA mailed the Addendum to DuPont to modify the CAP Agreement "only regarding the reporting of information on the release of chemical substances to and detection of chemical substances in all environmental media." Consent Agreement, Part I.C.

[12] TSCA Section 8(e); Notification of Substantial Risk; Policy Clarification and Reporting Guidance, 68 Fed. Reg. 33,129 (June 3, 2003).

press enforcement actions as to information on the release of chemical substances to and detection and chemical substances in environmental media "generated" prior to the announcement of the CAP on February 1, 1991 (or alternatively, prior to the CAP commencement date of July 1, 1991, or; prior to DuPont's registration for the CAP, on or about July 5, 1991), and prospectively, from June 27, 1996 forward.

OCE contends that, under Section 8(e) of TSCA, DuPont was subject to an ongoing statutory obligation from 1991 through 1996 to report information on the release of chemical substances to and detection and chemical substances in environmental media, and that this obligation was not affected or eliminated by the CAP or the CAP Agreement. OCE argues that Paragraph IV.A of the Revised Addendum does not bar Count II, as advanced by DuPont. Admitting that the Revised Addendum waived enforcement, OCE asserts that such waiver of enforcement does not apply to the period from the beginning of the CAP in 1991 to 1996, when the EPA eliminated "Phase 2" of the CAP, via the Revised Addendum.

In the alternative, DuPont submits that even if the CAP were a "lookback" audit, then it was a lookback from February 28, *1992* backwards, which was the original deadline for reporting data under the CAP. As noted, OCE contends that Count II "comes into play" on September 1991 and November 1991. Accordingly, DuPont argues that even under OCE's "lookback" theory, the EPA waived enforcement of the matters alleged in Count II.

As another basis for accelerated decision, DuPont argues that Count II is barred by the EAB's Consent Order, by virtue of res judicata. DuPont contends, *inter alia*, that the instant matter arises out of the same nucleus of facts as the 1996 complaint the EPA filed against DuPont pursuant to the CAP Agreement and by the EAB's Consent Order on that matter, which incorporated the Revised Addendum. DuPont further contends that the EPA could have asserted the current Count II in the 1996 complaint but did not. OCE counters that the Revised Addendum did not waive enforcement over the September and November 1991 dates that allegedly form the basis for Count II, and that the Consent Order, incorporating the parties' Consent Agreement, specifically permits matters of non-compliance to be litigated.

D. Contract Law and Parol Evidence (Extrinsic Evidence)

Consent agreements have many of the attributes of ordinary contracts and as such they should be construed, basically, as contracts. *United States v. ITT Cont'l Banking Co.*, 420 U.S. 233, 237-38 (1975); *accord Village of Kaktovic v. Watt*, 689 F.2d 222, 230 (D.C. Cir. 1982); *United States v. N. Colo. Water Conservancy District*, 608 F.2d 422, 430 (10th Cir. 1979). This type of settlement contract may not be unilaterally rescinded. *Village of Kaktovic*, 689 F.2d at 230. Consent agreements in settlement of EPA administrative enforcement actions are "enforceable like any other agreement; the fact that the subject matter of the agreement does not limit itself to the assessment of a civil penalty is irrelevant to its enforceability." *In re Chem. Waste Management, Inc.*, 1 E.A.D. 851, 857 n.11 (JO 1984) (citing *Village of Kaktovic*, 689 F.2d at 230). Therefore, I turn to contract law in examining the enforcement waiver contained in the Revised Addendum, which was incorporated into the parties' Consent Agreement.

Language within a contract must be read "in the context of the entire agreement" and must be construed "so as not to render portions of it meaningless." *Dalton v. Cessna Aircraft*, 98 F.3d 1298, 1305 (Fed. Cir. 1996); *Murphy v. Keystone Steel & Wire Co.*, 61 F.3d 560, 565 (7th Cir. 1995); *accord In re Julie's Limousine & Coachworks, Inc.*, CAA Appeal No. 03-06, 2004 EPA App. LEXIS 23, slip op. at 21-22 & n.31 (EAB, July 23, 2004), 11 E.A.D. ___ (fundamental principles of textual interpretation dictate that the adjudicator must interpret the text so as to give each word meaning and to avoid creating surplusage). When a contract term is unambiguous, the courts determine its meaning as a matter of law at the summary judgment stage. *LeJune v. Bliss-Salem, Inc.*, 85 F.3d 1069, 1073 (3rd Cir. 1996) (applying federal common law); *accord Murphy*, 61 F.3d at 564-65; *NRM Corp. v. Hercules, Inc*, 758 F.2d 676, 681-82 (D.C. Cir. 1985). "Determining whether contract language is ambiguous is also a question of law, and contract language is ambiguous if the terms are inconsistent on their face, or if the terms allow reasonable but differing interpretations of their meaning." *Rodrigues-Abreu v. Chase Manhattan Bank*, 986 F.2d 580, 586 (1st Cir. 1993) (citing cases).

"If the language of the contract is ambiguous, we turn to surrounding circumstances, undisputed extrinsic evidence, to divine the parties' intent." *Id.* (citing, *inter alia, Lumpkin v. Envirodyne Industries*, 933 F.2d 449, 456 (7th Cir. 1991)); *accord NRM*, 758 F.2d at 682 ("Only if the court determines as a matter of law that the agreement is ambiguous will it look to extrinsic evidence of intent to guide the interpretive process."). "Summary judgment based upon the construction of contract language is appropriate only if the meaning of the language is clear, considering all the surrounding circumstances and undisputed evidence of intent, and there is no genuine issue as to the inferences which might reasonably be drawn from the language." *Rodrigues-Abreu*, 986 F.2d at 586 (citing cases); *accord NRM*, 758 F.2d at 682 ("When, however, the language is unclear and the search for intent extends beyond the four corners of the agreement, the intended meaning of the contract is a disputed and, necessarily, material question of fact and summary judgment is improper.").

As discussed previously, the burden for summary judgment is on the movant. For Count II, DuPont is the only party moving for summary judgment. Therefore, the narrow issue before me is whether the contractual provision at issue – the waiver of enforcement – is unambiguous in favor of the movant, DuPont, when taking into account that the movant has the burden on this count and that all reasonable inferences of material fact are drawn in favor of the non-moving party, OCE.

E. Description of the Consent Agreement, Including the CAP Agreement and the Revised Addendum

As discussed, the starting point for contractual interpretation is to look within the four corners of the contract, to determine whether the contract is unambiguous. The settlement agreement (i.e., contract) in this matter consists of the "Consent Agreement," Docket No. TSCA-96-H-47, executed by the "Regulatee" (DuPont) and the EPA, and filed on October 1, 1996 with

the following attachments: Attachment A – the CAP Agreement,[13] and; Attachment B – the Revised Addendum to the CAP Agreement. DuPont's Motion for Acc. Dec., Ex. 12. On October 3, 1996, the EAB executed a "Consent Order," under Docket Number TSCA 96-H-47, which approved the Consent Agreement. *Id.*, Ex. 13. The Consent Order consists of a brief recitation of the penalty amount and payment procedures, and expressly incorporates the Consent Agreement by reference. *Id.* The Consent Agreement is attached to the Consent Order. *See id.*

The Consent Agreement provides, "All of the terms and conditions of this Consent Agreement together comprise one agreement, and each of the terms and conditions is in consideration of all of the other terms and conditions."[14] Consent Agreement, Part VI.H. Accordingly, the Consent Agreement and its attachments are an integrated contract and the parol evidence rule applies.

DuPont contends that the plain language of the Revised Addendum waived enforcement over all the allegedly reportable information OCE cited as the basis for Count II. In particular, DuPont focuses on the language in Part IV.A of the Revised Addendum, which reads:

> *Information* on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, *that predates the effective date of the final revised guidance* will not be the subject of an EPA TSCA section 8(e) penalty enforcement action.

Revised Addendum, Part IV.A (emphasis added). DuPont emphasizes that Part IV.A states plainly that the EPA waived all Section 8(e) claims based on "environmental data" that existed before the revised guidance, published in 2003. DuPont's Count II Reply at 3. Further, DuPont argues that if the EPA had intended to qualify "predates" it could have easily done so. *Id.*

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[13] The CAP Agreement is undated, as are the date(s) of the signatures to the CAP Agreement. However, Unit (i.e., Part or Section) I.D. of the CAP Agreement provides, "the TSCA Section 8(e) Compliance Audit Program shall commence no later than July 1, 1991." The Consent Agreement states that on or about July 5, 1991, DuPont registered for the TSCA section 8(e) CAP by signing the CAP Agreement. Consent Agreement at 1. However, OCE asserts that DuPont signed the CAP Agreement on June 28, 1991. OCE's Count II Response at 8 (citing OCE's Count II Response, Ex. 26)); Oral Arg. Tr. at 77 (citing to DuPont's Motion for Acc. Dec., Ex. 8); OCE's Post-Argument Br. on Count II at 5. A review of the cited exhibits as well as the rest of the record currently before this Tribunal does not indicate the purported June 28, 1991 registration date.

[14] *See also* CAP Agreement, Unit II.D.5: "All of the terms and conditions of this CAP Agreement together comprise one agreement, and each of the terms and conditions is in consideration for all of the other terms and conditions."

The Consent Agreement recounts that on February 1, 1991, the EPA published a Federal Register notice (56 Fed. Reg. 4,128) that set forth the TSCA Section 8(e) CAP and announced the opportunity for all regulated parties to register for and participate in the CAP. Consent Agreement, Part I.A. Reportedly, 122 companies registered for the CAP. On April 26, 1991 and June 20, 1991, the EPA published Federal Register notices (56 Fed. Reg. 19,514 and 56 Fed. Reg. 28,458) that modified certain terms of the TSCA Section 8(e) CAP. Consent Agreement, Part I.A. The Consent Agreement states that "on or about July 5, 1991," DuPont registered for the TSCA Section 8(e) CAP by signing the CAP Agreement. Consent Agreement, Parts I.B and II.B.

The CAP Agreement provides, "The Regulatee [DuPont] agrees to conduct a TSCA Section 8(e) Compliance Audit Program to determine its compliance status with TSCA section 8(e)." CAP Agreement, "Unit" (i.e., Part or Section) I.A. Thus, the CAP Agreement provides for DuPont to audit its records to find Section 8(e) violations and to report such to the EPA. As originally written, the CAP was to commence no later than July 1, 1991 and terminate on February 28, 1992,[15] and all submissions under the CAP would have to be delivered to the EPA no later than February 28, 1992. CAP Agreement, Unit I.D-E. The parties agreed, "This CAP Agreement and the Consent Agreement and Consent Order in this matter shall be a complete settlement of all civil and administrative claims and causes of action which arose or could have arisen under TSCA section 8(e) in connection with any study or report listed or submitted pursuant to the terms of this CAP Agreement." CAP Agreement, Unit II.A.1.

The CAP Agreement provides, "In conducting the TSCA Section 8(e) Compliance Audit Program, the Regulatee [DuPont] shall follow the statutory language of TSCA section 8(e) and [the 1978 Enforcement Policy], with the exception of Parts V(b)(1) and V(c) of the [1978 Enforcement Policy] to determine whether the reviewed study or report is:" (a) not reportable, (b) reportable, or (c) data that would have been reportable under Section 8(e) when initially obtained by the Regulatee, and that subsequent to the Section 8(e) reporting deadline (and before June 18, 1991), were previously submitted. CAP Agreement, Unit II.B.1. However, Footnote 1 of the CAP Agreement qualifies, "In determining whether the kind of information or studies referenced in Parts V(b)(1) and V(c) (i.e., widespread and previously unsuspected distribution in environmental media and emergency incidents of environmental contamination) should be submitted under the TSCA Section 8(e) Compliance Audit Program, the Regulatee [DuPont] should make a reasonable judgement whether such information meets the statutory standards of TSCA section 8(e) instead of relying on the guidance in Parts V(b)(1) and V(c) of the [1978 Enforcement Policy]." CAP Agreement at 3 n.1.

Pursuant to the CAP Agreement, DuPont agreed to pay stipulated civil penalties for all studies or reports submitted under the CAP as Section 8(e) data. CAP Agreement, Unit II.B.2. The stipulated penalty amounts were "$15,000 per study for any submitted study or report involving effects in humans" and "$6,000 per study for any other submitted study or report

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[15] However, the CAP Agreement provided that the EPA could grant extensions to the termination date. CAP Agreement, Unit I.E.

submitted as TSCA section 8(e) data," and $5,000 for each late-submitted study or report that was received by the EPA prior to June 18, 1991. CAP Agreement, Unit II.B.2-3. The parties agreed to a $1,000,000 cap on the total civil penalty for each Regulatee.[16] CAP Agreement, Unit II.B.3.

Upon termination of the CAP, the Regulatee was to provide the EPA with a Final Report certifying that the CAP has been completed. CAP Agreement, Unit II.B.5. As provided in the CAP Agreement, following termination of the audit, the EPA agreed to present the Regulatee with a Consent Agreement and Consent Order summarizing the results of the CAP and specifying the terms of payment of stipulated civil penalties. CAP Agreement, Unit II.B.6.

Under "Other Matters," the CAP Agreement provides that "Nothing in this CAP Agreement shall relieve the Regulatee from complying with all applicable TSCA regulations or other applicable environmental statutes." CAP Agreement, Unit II.D.

On or about January 31, 1992, the EPA mailed the "Addendum to the CAP Agreement" ("Addendum") to DuPont to modify (as stated in the Consent Agreement) the CAP Agreement "only regarding the reporting of information on the release of chemical substances to and detection of chemical substances in all environmental media." Consent Agreement, Part I.C. "The deadline for reporting all other information under the CAP remained unchanged at February 28, 1992 unless otherwise extended."[17] Consent Agreement, I.C.

On or about June 27, 1996, DuPont entered into an agreement, referred to as the Revised Addendum, to supersede the Addendum and to modify the CAP Agreement to specify that DuPont (referred to as the Regulatee in the Revised Addendum) "[i]s no longer required to conduct a file search for information on the release of chemical substances to and detection of chemical substances in environmental media, or for environmental toxicity on plant effluents; and that a second Final Report is no longer necessary." Consent Agreement, Part I.D. According to the Consent Agreement, DuPont timely submitted the Final Report on or about October 26, 1992. Consent Agreement, Part II.D.

Therefore, the first Final Report, which DuPont submitted on or about October 26, 1992, became the only Final Report. The Final Report indicated that a total of 1,380 studies were listed or submitted as Section 8(e) data pursuant to the CAP Agreement, with: 24 human health effects studies, at $15,000 per study; 1,287 studies listed under the category for "any other study

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[16] For instance, DuPont's overall penalty under the Section 8(e) CAP was $1,000,000. Consent Agreement, Part V.E. However, DuPont's penalty would have been $8,427,000 without the $1,000,000 limit. DuPont's Motion for Acc. Dec., Ex. 11 (Docket No. TSCA-96-H-47, Complaint, Sept. 30, 1996) at 6.

[17] According to the Consent Agreement, "[DuPont] submitted the Addendum to EPA on September 26, 1992; however, EPA presently has no record of an Addendum for [DuPont]." Consent Agreement, I.C.

or report submitted as TSCA Section 8(e) data" (i.e., for studies that were not human health effects studies), at $6,000 per study, and; 69 late-submitted studies given to the EPA prior to June 18, 1991, at $5,000 per study. Consent Agreement, Parts II.D and IV. Pursuant to the limitation on overall penalties under the CAP Agreement, DuPont's total civil penalty was $1,000,000. Consent Agreement, Part IV. The Consent Agreement provided, under "Other Matters," that "Nothing in this Consent Agreement and Consent Order shall relieve [DuPont] of the duty to comply with all applicable provisions of TSCA and other environmental statutes." Consent Agreement, Part VI.

Turning to the Revised Addendum to the CAP Agreement, Paragraph I of the Revised Addendum provides:

> The TSCA Section 8(e) Compliance Audit Program, which the Regulatee agreed to conduct in the Registration requirement I.A. does not include: information on the release of chemical substances to and detection of chemical substances in environmental media; or environmental toxicity data on plant effluents. The Regulatee, therefore, is no longer required to conduct a file search for this information. Further, footnote 1 of the [CAP] Agreement pertains solely to chemical release and detection information and therefore, is no longer applicable to the administration of the TSCA Section 8(e) Compliance Audit Program.

Paragraph II of the Revised Addendum provides that the first Final Report shall be considered the Final Report and controlling document for purposes of determining the information listed or submitted under the CAP. The Revised Addendum, at Paragraph III, states that "EPA intends to publish final revised guidance in the Federal Register on reporting information on the release of chemical substances to and detection of chemical substances in environmental media." Furthermore, "EPA also intends to publish a question and answer document to illustrate application of the guidance. The final revised guidance will not be effective prior to EPA's publication of the question and answer document." Revised Addendum, Paragraph III.

Paragraph IV of the Revised Addendum reads as follows:

> IV. Impact of the final revised guidance on:
>
> A. Information on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, that predates the effective date of the final revised guidance will not be the subject of an EPA TSCA section 8(e) penalty enforcement action.

> B. Information on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, that may have been submitted under Phase 1 of the CAP Program will not result in the assessment of penalties for such studies or reports submitted under this TSCA Section 8(e) Compliance Audit Program.

The Revised Addendum, at Paragraph V, provides that "Information generated after the effective date of the new final revised guidance on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, will be submitted prospectively pursuant to TSCA Section 8(e) and the new final revised guidance, not the CAP Agreement. Therefore, no penalty will accrue under the CAP Agreement for the submission of such information."

F. The Parties' Arguments Regarding the Duration of the Waiver of Enforcement

1. DuPont's Arguments As to the Waiver of Enforcement

DuPont contends that in the Revised Addendum (in Part I), the EPA stated explicitly that DuPont need not search its files for data regarding detection of chemicals in environmental media, and that the EPA then promised (in Part IV.A) that the EPA would not bring a Section 8(e) enforcement action based on information in DuPont's files prior to the effective date of the final reporting guidance, which was published in 2003. DuPont's Motion for Acc. Dec. at 24-25. Under DuPont's view, the contract language that is embodied in the Revised Addendum clearly and unambiguously states that DuPont need not search its files for preexisting data regarding detection of chemicals in water samples, and that the EPA would not bring a Section 8(e) enforcement action for any failure to report information prior to EPA's final guidance for that reporting. *Id.* at 25. DuPont points out that the water samples at issue in Count II are data that existed before the 2003 guidance. *Id.* According to DuPont's argument, due to the "plain language" of the Revised Addendum, the EPA promised not to assert, and waived any right to pursue, the enforcement action that the EPA now pursues in Count II. *Id.*

DuPont emphasizes that the word "predates" in Paragraph IV.A of the Revised Addendum "means what it says." DuPont's Count II Reply at 3. DuPont contends that the term predates "is not qualified by anything suggesting that it really means . . . 'predates, but only if it is after June 27, 1996.'" *Id.* DuPont argues that if the EPA intended to qualify 'predates,' it could have easily done so. *Id.* Furthermore, DuPont submits, "There is a strong presumption against reading into contracts provisions that easily could have been included but were not." *Id.* (quoting *Fix v. Quantum Indus. Partners LDC*, 374 F.3d 549, 553 (7th Cir. 2004)).

Regarding Paragraph IV.B of the Revised Addendum, DuPont argues that IV.A and IV.B actually address two different topics. *Id.* at 4. DuPont argues that IV.A tells DuPont and the other CAP registrants that were each asked to sign the Revised Agreement that they need not

submit "environmental data" that existed before the EPA issues its final revised guidance, and that the EPA would not bring any Section 8(e) enforcement action based on "environmental data" that existed before the EPA issues its final guidance. *Id.* Paragraph IV.B on the other hand, assures DuPont and the other CAP registrants that, if they already had submitted "environmental data" to the EPA under the CAP, they would not be fined under the original CAP for such submissions. *Id.* Paragraph IV.B was an effort to level the playing field between such submitters and those who had not made such a submission. *Id.* at 5; Oral Arg. Tr. at 18-20.

Furthermore, DuPont interprets Paragraph IV.A as stating that those persons who had not submitted environmental data would not be subject to Section 8(e) enforcement actions, but that IV.A does not address the fine status of those companies who had already submitted environmental data under the CAP and were facing automatic stipulated fines of $6,000 per study submitted. DuPont's Count II Reply at 5*;* Oral Arg. Tr. at 18-20. According to DuPont, the EPA added Paragraph IV.B to clarify that those who had already submitted "environmental data" would be placed on the same footing as those who had not submitted the data, by adding that those who had submitted such data would not be penalized for having reported the data. DuPont's Count II Reply at 5; Oral Arg. Tr. at 18-20. DuPont contends that Paragraph I of the Revised Addendum, only eliminates the requirement to *audit* for "environmental data", and that it does not address penalties or enforcement actions. Oral Arg. Tr. at 20. Therefore, according to DuPont's argument, Paragraph IV.B would be necessary to remove the threat of stipulated automatic penalties for "Phase 2" information submitted during "Phase 1." *Id.* at 20-21.

Finally, DuPont raises an argument as to the cutoff date for the CAP. DuPont submits, *in arguendo,* that even if the CAP were a "lookback" audit, then it was a lookback from February 28, *1992* backwards, which was the original deadline for reporting data under the CAP.[18] *Id.* at 108-09; DuPont's Post-Argument Br. at 8-9. February 28, 1992 comes after the September 1991 and November 1991 dates on which Count II allegedly "comes into play." Oral Arg. Tr. at 71; *see also id.* at 62. DuPont's argument is, "Thus, even if we assume, *arguendo,* that [OCE] is correct when it asserts that EPA only waived enforcement for data that existed prior to the original cut-off date for including data in the CAP, EPA still waived enforcement of Count II because all of the data in question in Count II existed prior to February 28, 1992. Thus even under [OCE's] 'look back' theory, EPA waived enforcement of the matters alleged in Count II." DuPont's Post-Argument Br. at 9.

[18] DuPont's deadline for submitting Phase 1-type information appears to have been extended beyond February 28, 1992, as the Consent Agreement states that DuPont timely submitted the its Final Report for audited information on or about October 26, 1992. *See* Consent Agreement, Part II.D.

2. OCE's Arguments As to the Waiver of Enforcement

In contrast to DuPont's position, OCE argues that DuPont was subject to an ongoing statutory obligation under Section 8(e) of TSCA to report information on the release of chemical substances to and detection of chemical substances in environmental media that ran from 1991 through 1996, and that the EPA never eliminated this obligation through the CAP or the Revised Addendum.

First, OCE indicated that the language "Phase 2" of the CAP refers to "information on the release of chemical substances to and detection and chemical substances in environmental media." OCE's Count II Response at 15. Later, OCE clarified its position to mean that "Phase 2" of the CAP requires the submission of environmental contamination data not just under Part V(b)(1) of the 1978 Enforcement Policy, but also under Parts V(b)(2)-(5), even though the guidance for V(b)(2)-(5) had never been called into question. OCE's Post-Argument Br. on Count II at 3; *see also* Oral Arg. Tr. at 69-70.[19] Count II, which mentions bioaccumulation and biopersistence, among other effects, (Amended Complaint ¶¶ 15-20), may be interpreted as alleging not just V(b)(1)-type violations, but also other environmental effects-type violations that would fall under V(b)(2)-(5).

Initially, OCE posited that the plain language of Paragraph I of the Revised Addendum removed the CAP's applicability to Phase 2 data generated after June 27, 1996 (the date of the Revised Addendum), in effect voiding the Phase 2 portion of the CAP program. OCE's Count II Response at 18. Furthermore, OCE stated that Paragraph IV.A of the Revised Addendum is consistent with EPA's interpretation of Paragraph I of the Revised Addendum. *Id.* According to OCE, Paragraph IV.A of the Revised Addendum operates as a prospective waiver of OCE's right to enforce Section 8(e) claims from June 27, 1996 until the issuance of a final "Phase 2" reporting deadline, thereby temporarily relieving regulatees of their obligations until promulgation of final reporting guidelines.[20] *Id.* OCE stated that it is for the latter reason that OCE is not seeking additional penalties from DuPont at this time for the period from 1996 until today. *Id.* at 18 n.15.

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[19] At oral argument, I asked OCE whether Paragraph IV.A of the Revised Addendum, using the language "release of chemical substances to and detection of chemical substances in environmental media," is the same as Part V(b)(1) of the 1978 Enforcement Policy, which uses the language "widespread and previously unsuspected distribution in environmental media." Oral Arg. Tr. at 68-69. OCE responded, "I believe that the terminology used by EPA in the 1996 Addendum is subsumed within the broader category of V(b) environmental contamination." *Id.* at 69. Then, in response to my question: "So, it's not limited to V(b)(1), the charges that you're alleging in Count II," OCE responded, "In Count II, it is environmental contamination, so it is V(b)." *Id.* at 69-70.

[20] However, at oral argument, OCE submitted that the EPA may have actually granted a prospective waiver as early as May 15, 1996, which is the date of the Cover Letter to the Revised Addendum. Oral Arg. Tr. at 79.

Regarding Paragraph IV.B, OCE contends that the EPA reached back to waive its right to enforce penalty actions against regulatees who may have submitted "Phase 2" data at any time prior to the issuance of the Revised Addendum on June 27, 1996. OCE's Count II Response at 19. As argued by OCE, to give Paragraph IV.A of the Revised Addendum the reading advocated by DuPont – that the EPA waived the right to enforce Section 8(e) for Phase 2 data generated at *any* time prior to finalization of the guidance – is incorrect, because that reading would render Part IV.B. meaningless. *Id.*

OCE further argues, "If Respondent's reading of IV.A were correct, there would be *no need* for an explicit waiver for Phase 2 data that had been submitted during the Phase 1 reporting period, because under Respondent's interpretation of the Addendum, regulatees would be off the hook for *all* Phase 2 data generated at any time before issuance of the final guidance in 2003, including that submitted under Phase 1." *Id.* (footnote omitted). Therefore, "Respondent's reading would render IV.B of the Revised Addendum meaningless, violating well-established principles of contract interpretation." *Id.*

In its post-argument brief, OCE argues that Paragraph I of the Revised Addendum eliminated the Phase 2 reporting requirement, which OCE interprets as meaning "[t]here could be no *CAP* penalties for Phase 2 information." OCE's Post-Argument Br. on Count II at 10-11 (emphasis added). In doing so, OCE points out the language of Paragraph I stating that the "TSCA Section 8(e) Compliance Audit Program . . . does not include: information on the release of chemical substances to and detection of chemical substances in environmental media; or environmental toxicity data on plant effluents." *Id.* at 10 & n.4. OCE states that Paragraph I of the Revised Addendum "eliminated the Phase 2 reporting requirement, meaning that by definition, there could be no CAP penalties for Phase 2 information."[21] *Id.* at 11. "However, industry was then subject to potential penalties for pre-1991 information that was no longer covered by the CAP." *Id.* "(In essence, elimination of the Phase 2 CAP removed the protection industry would have received for pre-1991 violations.)" *Id.* "[Paragraph] IV(B) was therefore added to address this unintended exposure for Phase 2 information submitted pursuant to the CAP, and ensure that all parties were treated the same regarding their historic violations of TSCA § 8(e)." *Id.* As noted, OCE contends that reading Paragraph IV.A as a retroactive waiver would render Paragraph IV.B superfluous. *See id.* at 10.

Regarding the cutoff date for information falling under the CAP, OCE indicates a cutoff date as early as February 1, 1991, when the EPA first announced the CAP in the Federal Register, and as late as July of 1991.[22] OCE asserts that, "It was made extremely clear, like

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[21] *See also* OCE's Count II Response at 19 n.16.

[22] In its pre-oral argument brief, OCE stated that "the purpose of the CAP in 1991 was to allow companies that signed up to conduct an audit of their compliance status under TSCA § 8(e) *as of that point in time*." OCE's Count II Response at 14. The latter statement would indicate that the CAP covered information generated prior to the date when DuPont signed the

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many EPA enforcement initiatives, that the purpose was to address past noncompliance, to allow defendants to pay stipulated penalties and then move on." Oral Arg. Tr. at 77. Furthermore, OCE argues that the CAP refers only to violations committed prior to February (or July) 1991 backward, by pointing to the CAP Agreement's "Other Matters" provision, which states: "Nothing in this CAP Agreement shall relieve the Regulatee from complying with all applicable TSCA regulations or other applicable environmental statutes."[23] Oral Arg. Tr. at 78 (quoting CAP Agreement, Unit II.D). In sum, according to OCE, the EPA would have a window of opportunity to enforce Section 8(e) violations for information generated after February (or July) 1991 up to the June 27, 1996 Revised Addendum.

3. Arguments Regarding the Extrinsic Documents

In support of their positions concerning the duration of the enforcement waiver in Part IV.A of the Revised Addendum, the parties have submitted various documents outside the four corners of the Consent Agreement. Principally, these documents include the cover letter to the Revised Addendum and various Federal Register notices concerning the CAP, and a comment and response document.

The parties refer to the following Federal Register notices: (1) Registration and Agreement for TSCA section 8(e) Compliance Audit Program; Notice, 56 Fed. Reg. 4,127 (Feb. 1, 1991) ("February 1991 notice"); (2) Registration and Agreement for TSCA section 8(e) Compliance Audit Program Modification, 56 Fed. Reg. 19,514 (Apr. 26, 1991) ("April 1991 notice"); (3) Registration and Agreement for TSCA section 8(e) Compliance Audit Program Modification; Notice, 56 Fed. Reg. 28,458 (June 20, 1991) ("June 1991 notice"), and; (4) Registration and Agreement for TSCA Section 8(e) Compliance Audit Program Modification, 56 Fed. Reg. 49,478 (Sept. 30, 1991) ("September 1991 notice"). The cover letter to the Revised

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[22](...continued)
CAP Agreement. Furthermore, in its post-argument brief, OCE contends that the CAP audit period was designed to review information generated up to the date DuPont signed to participate in the CAP. OCE's Post-Argument Br. on Count II at 5. OCE asserts that DuPont signed the CAP Agreement on June 28, 1991. (However, the Consent Agreement, at 1, states that DuPont signed the CAP Agreement "on or about July 5, 1991," and there is not yet support in the record before me for a specific date of June 28, 1991.) OCE also indicates a cutoff date of July 1, 1991 in its pre-argument briefs, where OCE states that DuPont had an ongoing obligation to report between July 1, 1991 and June 27, 1996. OCE's Count II Response at 17, 20. Finally, at the oral argument, OCE put forth a cutoff date of February 1, 1991: "During what I'm calling the entire CAP development period, which was from announcement of the CAP in February of 1991 through the closing of the CAP in July of 1996, DuPont was obligated to stay in ongoing compliance with TSCA Section 8(e)." Oral Arg. Tr. at 59. Nevertheless, all three dates are prior to the September and November 1991 dates that OCE put forth to support Count II.

[23] DuPont signed the CAP Agreement "on or about July 5, 1991." Consent Agreement at 1.

Addendum, which is dated May 15, 1996, was sent from Jesse Baskerville, Director of the Toxics and Pesticides Enforcement Division, EPA, and addressed to DuPont. DuPont's Motion for Acc. Dec., Ex. 9 ("Cover Letter to Revised Addendum"). Finally, there is the February 20, 2003 Comment and Response Document for Revised Policy Statement of Section 8(e) of TSCA. DuPont's Motion for Acc. Dec., Ex. 14 ("2003 Comment and Response Document").

DuPont argues that Count II is barred not only by the "plain meaning" of the Revised Addendum, but also when taking into account the context in which the contract was executed and common sense. Oral Arg. Tr. at 10, 15, and 21.

DuPont contends that the 1978 Enforcement Policy speaks in general terms and does not set clearly defined standards. DuPont's Motion for Acc. Dec. at 7. As a result, states DuPont, each company subject to Section 8(e) was required to exercise individual subjective judgment to determine what information must be reported, and that the lack of guidance led to a number of disagreements between the EPA and regulated entities. *Id.* at 7-8. For example, in 1984, 1989, and 1990, respectively, the EPA filed enforcement actions against Union Carbide Corporation, Monsanto Company, and Halocarbon Products Corporation, in each case for allegedly failing to submit a single study or piece of information. *Id.* at 8. In settling these matters, the EPA and the respondent took what DuPont describes as the "unusual step" of setting forth in the respective consent agreements a detailed discussion of their continuing substantial differences of opinion regarding the clarity of the reporting standards, the scope of reporting obligations under Section 8(e), and whether the information in question actually triggers Section 8(e)'s mandatory reporting obligations. *Id.* (citing DuPont's Motion for Acc. Dec., Exs. 5, 6, and 7).

DuPont notes that on February 1, 1991, the EPA announced a one-time voluntary Section 8(e) CAP, February 1991 notice, 56 Fed. Reg. 4,127, "to avoid similar disputes." DuPont's Motion for Acc. Dec. at 8. Under the CAP Agreement that the EPA had developed, any company that registered for the CAP pledged to audit its files for reportable information not previously submitted to the EPA, report any information that the EPA might consider reportable, and pay a stipulated penalty of $6,000 to $15,000 for each previously unreported study or report. *Id.* In return, the EPA agreed, among other things, that each company's total liability would be limited to $1,000,000, regardless of how many previously unreported studies the company submitted. *Id.* (citing February 1991 notice, 56 Fed. Reg. at 4,130).

Shortly after announcing the CAP, the EPA announced modifications to the CAP program. *Id.* at 9 (citing April 1991 notice, 56 Fed. Reg. at 19,514). The EPA was concerned about a so-called "data dump"; that without further guidance on what information must be submitted under TSCA Section 8(e), companies would give the EPA too much information. *Id.* (citing 56 Fed. Reg. at 19,514)). Therefore, states DuPont, the EPA pledged to issue, prior to the July 1, 1991 deadline for the CAP registration, an 8(e) Reporting Guide that would include a record of all previous initial submissions made under 8(e), a compilation of Question and Answer ("Q&A") documents EPA had recently prepared, and a written review of several hypothetical "case histories" prepared by the Chemical Manufacturers Association, each of

which illustrated various issues for which guidance was lacking. *Id.* (citing 56 Fed. Reg. at 19,515)).

On June 20, 1991, the EPA issued the "TSCA Section 8(e) Reporting Guide" ("1991 Reporting Guide") and announced its availability.[24] *Id.* (citing June 1991 notice, 56 Fed. Reg. at 28,458). In the June 1991 notice, the EPA acknowledged that the 1978 Enforcement Policy needed "additional clarification" and that "possible misinterpretation" likely would lead to "over-reporting." *Id.* (quoting 56 Fed. Reg. at 28,458). Accordingly, the EPA formally "suspended" Parts V(b)(1) and V(c) of the 1978 Enforcement Policy and declared that it would prepare new guidance on reporting standards. *Id.* (citing 56 Fed. Reg. at 28,459). DuPont points out that, according to the June 1991 notice, CAP participants were to be guided solely by the statutory language when auditing company records of "detection of chemicals in environmental media." *Id.* at 9. Shortly after this announcement, DuPont registered for the CAP by signing the standard form CAP Agreement. *Id.* at 10. Under the (original) terms of the CAP Agreement, each participant was to complete its audit and submit a final report to the EPA no later than February 28, 1992. *Id.*

DuPont notes, "On September 30, 1991, however, EPA extended indefinitely the CAP reporting deadline for information on the detection of chemicals in environmental media, instructing companies that such information need not be audited and reported until six months after EPA published its final revised guidance on reporting for such information." *Id.* (citing September 1991 notice, 56 Fed. Reg. at 49,478). The September 1991 notice predicted that the EPA would issue the final revised guidance in Spring 1992. *Id.* (citing 56 Fed. Reg. at 49,479). In issuing the September 1991 notice, the EPA split the CAP into two phases, which DuPont interprets as follows: "'[P]hase I' of the CAP would be limited to auditing for reportable toxicology studies, with final reports still due to EPA by February 28, 1992, while 'Phase II' (regarding information on detection of chemicals in environmental media) would involve a six-month auditing period triggered by publication of EPA's revised guidance." *Id.* (citing 56 Fed. Reg. at 49,479).

DuPont emphasizes the importance of the September 1991 notice. DuPont's Count II Reply at 8. DuPont points out that the September 1991 notice was EPA's final Federal Register statement on the reporting deadline for "environmental data" until the EPA circulated its Revised Addendum five years later. *Id.* DuPont points out that the September 1991 notice "states clearly" that the deadline for all CAP participants, which includes DuPont, to report environmental data was extended until six months after publication of final reporting guidance. *Id.* DuPont argues that the September 1991 notice is a "clear statement" of EPA's intent to waive enforcement during that period, which "strongly corroborates" DuPont's interpretation of

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[24] DuPont asserts that the 1991 Reporting Guide did not include any EPA standards for "reporting detection of chemicals in environmental media." DuPont's Motion for Acc. Dec. at 9. Neither DuPont nor OCE, to date, have provided this Tribunal with a copy of the 1991 Reporting Guide. (Although its availability was announced in the Federal Register, it does not appear to have been published in the Federal Register.)

the Revised Addendum. *Id.* As for the Addendum to the CAP Agreement, DuPont asserts that the Addendum it signed gave the same assurance that the EPA had given in the September 1991 notice.[25] *Id.* at 9.

DuPont contends that EPA's 1993 notice confirms that EPA's September 1991 notice waived any Section 8(e) penalty enforcement action. Oral Arg. Tr. at 14 (referring to 1993 notice, 58 Fed. Reg. at 37,736). DuPont goes on to argue, "then comes the Revised Addendum . . . and at no time did EPA ever say to any of the CAP participants well, now, you have to hurry up and report." *Id.* Instead, argues DuPont, in 1991 the EPA extended the time for reporting and in 1993 the EPA confirmed that, and in the Revised Addendum the EPA states that it is waiving any Section 8(e) penalty enforcement action. *Id.*

DuPont argues that the Cover Letter to the Revised Addendum assured CAP participants that the EPA would not bring an enforcement action based on *any* environmental data that existed before the effective date of the final guidance. DuPont's Count II Reply at 9 (citing Cover Letter to the Revised Addendum at 2). In particular, DuPont quotes two sentences from the Cover Letter to the Revised Addendum, which read as follows:

> [E]PA has decided that it is reasonable and equitable to enforce the final revised reporting guidance on a prospective basis only. *Therefore,* information on the release of chemical substances to and detection of chemical substances in environmental media; . . . that predate the effective date of the guidance will not be the subject of an EPA TSCA Section 8(e) enforcement action.

Id. (quoting Cover Letter to Revised Addendum at 2) (emphasis added). DuPont quotes the dictionary definition of "therefore," meaning "for that reason, consequently." *Id.* at 9 (quoting *Webster's New Collegiate Dictionary,* G.&C. Merriam Co. 1201 (1979)). Accordingly, DuPont argues that "the only reasonable reading of these two sentences is that EPA had concluded that 'it is reasonable and equitable to enforce the final guidance on a prospective basis only' and, *for that reason,* environmental data that 'predate the effective date of the guidance will not be the subject of an EPA TSCA Section 8(e) enforcement action.'" *Id.* at 9-10.

DuPont further argues that in the 2003 Comment and Response Document, regarding the proposed final revised guidance, the EPA again expressed that any data that existed before the final guidance would not form the basis of any EPA enforcement action. *Id.* at 10. In particular, DuPont points to the follow exchange:

> COMMENT: Once EPA finalizes its new section 8(e) guidance, it should only be applied prospectively. The Agency [EPA] itself has admitted that the nature and scope of section 8(e) reporting

[25] The parties have not provided this Tribunal with a copy of the Addendum that DuPont signed.

> requirements for environmental information have not been clear, and it took the unusual step of suspending its prior guidance. Moreover, many additional Federal and state reporting requirements have been enacted since TSCA became effective in 1976,[26] further muddying the regulatory waters.
>
> The confusion associated with the scope of environmental reporting under section 8(e), and the absence of Agency attention to the issue, contrasts sharply with the long history of health-related section 8(e) guidance and reporting. Given this history, and the continuing questions raised about the Agency's proposal [sic] new guidance, it would be inappropriate to apply the guidance retroactively.
>
> RESPONSE: Given the circumstances noted by the commenter, the suspension of the previous guidance, the emphasis on health and environmental effects reporting, the length of time required to propose revised guidance, and the greater specificity of the revised guidance, EPA has concluded that the revised guidance will be enforced prospectively. This means that companies will not have to review preexisting files for information that may be subject to section 8(e) reporting. *These preexisting files would only come into "play" if data obtained by a company after the effective date of the guidance triggered a review* of such data and in doing so the combination of data met the section 8(e) reporting criteria.

Id. (emphasis added). DuPont interprets EPA's response to the comment as expressly stating that the preexisting files would trigger potentially enforceable reporting obligations *only* if new data caused the company to go back and review its old data, and the combination of the new and old data met the Section 8(e) reporting criteria. *Id.* at 10.

OCE, on the other hand, sees two separate tracks: one track for information generated prior to February 1, 1991 (or prior to July 1991) and a separate track for information generated after those dates up to 1996. Oral Arg. Tr. at 59, 65. To support this argument, OCE points to the Federal Register notices. Regarding the period from early to mid 1991 through 1996, OCE contends there was an ongoing statutory obligation to report information on the release of chemical substances to and detection of chemical substances in environmental media. *Id.* at 65; OCE's Count II Response at 14-15. Furthermore, OCE contends that the September 1991 notice suspended "Phase 2" under the *CAP* program's lookback audit, but that it did *not* suspend the reporting obligation for ongoing compliance with the statute. Oral Arg. Tr. at 64-66.

Regarding the Cover Letter to the Revised Addendum, OCE argues that instead of promising to not bring any Section 8(e) claims for information generated at any time prior to the

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[26] TSCA became effective on January 1, 1977.

2003 guidance, OCE promised to not bring such claims for information generated *prospectively* – from June 27, 1996, forward – until the final guidance.[27] EPA's Count II Response at 20 (citing Cover Letter at 2: "EPA has decided that it is reasonable and equitable to enforce the final revised reporting guidance *on a prospective basis only*" (emphasis added)). OCE asserts, "There is a very big difference between the CAP audit program, which was an enforcement initiative undertaken in 1991, and then the [1996 Revised Addendum and its Cover Letter],[28] which arguably affected more than just the CAP program." Oral Arg. Tr. at 65; *see also* OCE's Count II Response at 14-15. As for the 2003 Comment and Response Document, OCE submits that DuPont ignores the requirement that still existed between February 1, 1991, and June 27, 1996, to comply with the statutory provisions of Section 8(e) of TSCA, irrespective of the guidance. Oral Arg. Tr. at 86.

G. Discussion of the Waiver of Enforcement and the Cutoff Period for the CAP

1. Analysis Within the Four Corners of the Consent Agreement

As discussed, when a party moves for accelerated decision on the ground that a consent agreement bars enforcement, summary judgment is inappropriate unless the consent agreement unambiguously bars enforcement in favor of the movant. Furthermore, only if the language of the consent agreement is ambiguous, does the adjudicator turn to surrounding circumstances, undisputed extrinsic evidence, to divine the parties' intent. The Consent Agreement expressly incorporates, and therefore includes within its four corners, the CAP Agreement and the Revised Addendum.

Quite frankly, I am having great difficulty making sense of the Revised Addendum within the four corners of the Consent Agreement, the CAP Agreement, and the Revised Addendum. Not helping matters, as discussed *supra*, OCE has adjusted its interpretation throughout these proceedings as to many key aspects of the Revised Addendum, which may suggest that the EPA – who drafted the Revised Addendum – does not have a clear vision of the meaning of the Revised Addendum. Nevertheless, the burden at this juncture is on DuPont to prove that the language of the Consent Agreement is unambiguous.

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[27] In response to my question, "What happened in 1996 that caused EPA to change its position?," EPA counsel made the bald assertion that "EPA was facing potential statute of limitations problems with the closeout of Phase 1." Oral Arg. Tr. at 73; *see also* Complainant's Post-Argument Br. on Count II at 3 (no citation of support provided). I place no reliance on factual assertions unsupported by the record presently before me.

[28] By "the letter that came out in 1996," OCE appears to be referring to the Cover Letter to the Revised Addendum, which included the Revised Addendum as an attachment.

I note that some of the key terms, or potentially key terms, used in the Revised Addendum are not defined or not clearly defined within the four corners of the Consent Agreement, the CAP Agreement, and the Revised Addendum, or within the Consent Order. Looking solely within the four corners, the undefined or not clearly defined terms include: "Phase I," "environmental toxicity data for plant effluents," "information on the release of chemical substances to and detection of chemical substances in environmental media," and "final revised guidance."[29] As these terms are not defined within the four corners of the Consent Agreement, CAP Agreement, and Revised Addendum, I cannot discern a clear meaning of the enforcement waiver at issue, and therefore cannot interpret such waiver unambiguously in favor of the movant.[30] For this reason alone, a denial of DuPont's motion for accelerated decision is warranted.

Additionally, DuPont has not sustained its burden under the accelerated decision standard because OCE's arguments concerning the language of the Consent Agreement, CAP Agreement, and Revised Addendum are adequate to defeat DuPont's motion. Within their four corners, the Consent Agreement, CAP Agreement, and Revised Addendum, may be read as creating a lookback audit, for information existing prior to early to mid-1991, separate from ongoing statutory obligations to comply with TSCA. For instance, these three documents may be read as indicating that the EPA announced the CAP on February 1, 1991,[31] that the CAP was to commence no later than July 1, 1991,[32] that DuPont registered on or about July 5, 1991,[33] and

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[29] The absence of definitions for "information on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents" is particularly troublesome. The parties agree that Paragraph IV.A of the Revised Addendum waives enforcement over such information, but disagree as to whether the waiver is retroactive or prospective. However, without a definition of these terms, within the confines of the Consent Agreement, it is not clear whether such waiver affects all of the types of information alleged in Count II. For instance, in Count II OCE suggests a very wide range of effects, by entitling Count II as "Public Water Supply Contamination," and alleging that PFOA has been shown to produce liver toxicity in test animals, that PFOA is biopersistent in animals and humans, as well as bioaccumulative in humans.

[30] Although the Consent Agreement, at Part I.A., references the February, April, and June 1991 Federal Register notices, it does not expressly incorporate such notices as part of the Consent Agreement, and therefore such notices do not become part of the Consent Agreement. Morever, such notices do not readily clarify the meanings of these terms introduced by the Revised Addendum.

[31] Consent Agreement, Part I.A.

[32] CAP Agreement, Part I.B and I.D.

[33] Consent Agreement, Part I.B.

that the CAP did not relieve DuPont of the duty to comply with TSCA,[34] which suggests a lookback audit separate from statutory compliance. Moreover, all three documents make reference to the Compliance Audit Program. Within the context of the CAP being a lookback audit, Paragraph I of the Revised Addendum may be read as terminating the audit as to the so-called "Phase 2" information dated prior to 1991, but then exposing CAP registrants to penalties and/or enforcement actions as to such information already reported pursuant to the CAP under the so-called "Phase 1." Paragraph IV.B of the Revised Addendum may be read as eliminating the assessment of penalties for Phase 2 reports and studies submitted under Phase 1. Accordingly, one may read Paragraph IV.B as providing some protection against the assessment of penalties for information submitted prior to the termination of the CAP in Paragraph I. DuPont argues that Paragraph IV.A creates a retroactive waiver of enforcement, but the protection against the assessment of penalties in Paragraph IV.B would arguably render such a retroactive waiver superfluous, in violation of contract law principles.[35] Moreover, the CAP Agreement itself provides: "All of the terms and conditions of this CAP Agreement together comprise one agreement, and each of the terms and conditions is in consideration for all of the other terms and conditions."[36] Finally, Paragraph IV.A may be read as a prospective waiver of enforcement action, commencing in 1996, when reading it within the context of there being a lookback audit and that ongoing statutory compliance was required from early to mid-1991 through 1996.

With regards to the language in Paragraph IV.A, DuPont quotes *Fix v. Quantum Industrial Partners, LDC*, 374 F.3d 549, 553 (7th Cir. 2004), for the following principle: "There is a strong presumption against reading into contracts provisions that easily could have been included but were not."[37] OCE's Count II Reply at 3. In the latter case, however, the court held that the contract's terms were unambiguous on the face of the contract, rendering summary judgment appropriate, and thus the court excluded extrinsic evidence that contradicted the language of the contract; in particular, the parties had expressly adopted a term from a separate document, but chose *not* to adopt language from that very same document that was at odds with the terms of the contract. *Id.* Clearly, based on the facts that are presently before me, the factual situation in the *Fix* case is distinguishable from the instant matter.

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[34] Consent Agreement, Part VI.A ("Other Matters"); *see also* CAP Agreement, Part II.D.1 ("Other Matters"). *But see infra* note 38.

[35] It is an axiom in contract law that language within a contract must be read "in the context of the entire agreement" and must be construed "so as not to render portions of it meaningless." *Dalton v. Cessna Aircraft*, 98 F.3d 1298, 1305 (Fed. Cir. 1996); *Murphy v. Keystone Steel & Wire Co.*, 61 F.3d 560, 565 (7th Cir. 1995).

[36] CAP Agreement, Unit II.D.5.

[37] I would point out that *Fix* is a diversity case in which *state* law was the controlling law rather than federal law.

In conclusion, DuPont has not sustained its burden on summary judgment. I emphasize to the parties that my determination that an evidentiary hearing is warranted and that summary judgment is inappropriate does not suggest that I have developed or adopted a particular interpretation of the Consent Agreement and Consent Order, the CAP Agreement, or the Revised Addendum to the CAP Agreement. It simply means that the language is susceptible to interpretation contrary to the interpretation put forth by the movant. Furthermore, I note that I may deny a motion for accelerated decision (i.e., summary judgment) as a matter of discretion in order to fully develop the evidence concerning the disputed language, particularly in light of the potential ramifications such a determination may have on the other CAP registrants. *See Roberts v. Browning*, 610 F.2d 528, 536 (8th Cir. 1979).

2. Analysis Taking Into Account Extrinsic Documents

As discussed *supra*, summary judgment on Count II is not appropriate. Nevertheless, I examine the extrinsic evidence proferred by the parties, which primarily consists of several Federal Register notices, the Cover Letter to the Revised Addendum, and the 2003 Comment and Response Document. Both parties argue that the proferred extrinsic evidence supports their respective interpretations of the language of the Consent Agreement, the CAP Agreement, and the Revised Addendum.

With the February 1991 Federal Register notice, the EPA announced the opportunity to register for EPA's TSCA Section 8(e) Compliance Audit Program ("CAP"). 56 Fed. Reg. at 4,128. The CAP was originally set to commence February 1, 1991, and close on May 2, 1991, *id.*, but the commencement and closing dates were later amended. The CAP was a "*one-time* voluntary" program, designed to strongly encourage companies to voluntarily audit their files for studies reportable under Section 8(e). *Id.* at 4,129 (emphasis added). Persons interested in registering for the CAP were required to request a CAP Agreement and submit a signed CAP Agreement to the EPA no later than May 2, 1991. *Id.* at 4,128.

The February 1991 notice stated that "Up-to-date information on hazard and exposure is vital in supporting EPA efforts to protect human health and the environment from risks from toxic chemicals," and that the "EPA has the responsibility under TSCA to perform needed risk assessments on chemicals." *Id.* "Companies that do not report vital information are undermining the effectiveness of the early warning system intended under section 8(e)." *Id.* EPA recognized that there was, at the very least, a perception of significant disincentives to dissuade companies from auditing "*past* studies" and reporting them to EPA, due to high monetary penalties. *Id.* at 4128 (emphasis added). Furthermore, in evaluating some enforcement cases, the EPA found that some companies may have been misinterpreting Section 8(e) of TSCA and the 1978 Enforcement Policy. *Id.* The EPA emphasized that it had not changed its interpretation. *Id.* at 4,128-29. However, the EPA clarified that if serious health effects are discovered, then companies must submit the information without further evaluation (i.e., without using a weight-of-the-evidence method of discounting the significance of the information). *Id.* at 4,128; *see also* Oral Arg. Tr. at 72. The February 1991 notice stated that the CAP "has been developed" to encourage industry reporting by setting forth guidelines that identify in advance

EPA's enforcement response and allow companies to assess liability prior to electing to participate. 56 Fed. Reg. at 4,129.

Following that announcement were the initially proposed terms of the CAP Agreement. *Id.* at 4,129-31. Under "Other Matters" under the proposed terms for the CAP Agreement was the following provision: "Nothing in this CAP Agreement shall relieve the Regulatee from complying with all applicable TSCA regulations or other applicable environmental statutes." *Id.* at 4,130. The latter provision also exists in the CAP Agreement DuPont signed. CAP Agreement, Unit II.D; *see also* Oral Arg. Tr. at 78.

The February 1991 notice can be reasonably read as providing some support for OCE's position that the CAP was designed as a "lookback" auditing program. The February 1991 notice first announced OCE's disagreement with companies' use of the weight-of-the-evidence method for health effects and then the notice set forth limitations for that method. Once that clarification had been made, EPA announced a "one-time" auditing program for "past studies." Moreover, the requirement under the "Other Matters" provision that CAP registrants continue to follow the law is written in the present tense.[38] *See* Oral Arg. Tr. at 74, 78.

The April 1991 Federal Register notice announced modifications to the CAP and the CAP Agreement. 56 Fed. Reg. at 19,514. The April 1991 notice states that the CAP is a "one-time voluntary audit program developed in order to achieve EPA's goal of obtaining any *outstanding* TSCA section 8(e) data." *Id.* (emphasis added).

Principally, the April 1991 notice expressed concern about an overflow, or "data dump," of information resulting from the audits.[39] *Id.* The EPA recognized that proper application of

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[38] On the other hand, the precise wording of the "Other Matters" provision at issue states that "Nothing in this CAP Agreement shall relieve the Regulatee from complying with all applicable TSCA *regulations* or *other* applicable environmental *statutes*." There is no *regulation* that implements Section 8(e) of TSCA, and as correctly observed by DuPont, Congress did not confer any rulemaking authority on the EPA as to Section 8(e). *See* DuPont's Motion for Acc. Dec. at 7. Rather, the EPA implements TSCA by way of policies, such as the 1978 Enforcement Policy and the 2003 guidance. In contrast, the Consent Agreement, which was executed in 1996, has its own "Other Matters" provision, which reads: "Nothing in this Consent Agreement and Consent Order shall relieve Respondent of the duty to comply with all applicable *provisions* of TSCA and other environmental statutes." Consent Agreement, Part VI.A (emphasis added).

[39] Indeed, the CAP program's $1,000,000 limitation on overall penalties may have acted as an incentive for companies to overreport. A person regulated by Section 8(e) might submit as many studies as possible in order to shield the company from enforcement actions involving those studies. If such a person had already reached the $1,000,000 limit, there would no longer be the threat of stipulated penalties for the extra studies submitted under the CAP program. For (continued...)

Section 8(e) requires the exercise of scientific judgment. *Id.* The April 1991 notice announced EPA's plans to disseminate a Section 8(e) reporting guide, comprised of status reports, a compilation of question and answer ("Q&A") documents, and a written review of several hypothetical 'case histories' prepared by the Chemical Manufacturers Association. *Id.* at 19,515. The latter review of case histories was in response to a written request from the Chemical Manufacturers Association for additional guidance in the areas of neurotoxic effects and environmental effects/releases. *Id.* The April 1991 notice stated that EPA would make every effort to complete the reporting guide in early June 1991 and release it prior to the revised registration deadline/audit commencement date of June 18, 1991. *Id.* "However, if necessary because of a delay in completion of the guidance on the environmental effects/release information, reporting of this information under the TSCA Section 8(e) Compliance Audit Program will be put on a specific schedule . . . " *Id.* at 19,514.

The April 1991 notice extended the CAP registration deadline/audit commencement date for 45 days, to June 18, 1991. *Id.* Furthermore, it extended the CAP audit termination date/deadline date for approximately 90 days, to February 28, 1992 (which is the same termination date used in DuPont's CAP Agreement). *Id.*

The April 1991 notice can be reasonably read as indicating that there was a lookback audit under the CAP for prior studies, consistent with OCE's argument. In particular, the April 1991 notice reiterated that this "one-time" audit program was developed in order to obtain "any *outstanding"* TSCA Section 8(e) data. *Id.* (emphasis added).

The June 20, 1991 notice announced the availability of a Section 8(e) reporting guide and announced modifications to the CAP program and to the CAP Agreement. June 1991 notice, 56 Fed. Reg. at 28,458. The June 1991 notice stated that the "TSCA Section 8(e) Compliance Audit Program is a one-time voluntary compliance audit program developed to obtain outstanding TSCA section 8(e) data *and* foster compliance with the statutory obligations of TSCA section 8(e)." *Id.* (emphasis added). The CAP modifications again extended the registration deadline, this time to July 1, 1991 (which became the final registration deadline),[40] and modified EPA's guidance for reporting information concerning "widespread and previously unsuspected

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[39](...continued)
instance, in absence of the $1,000,000 limit on stipulated penalties under the CAP, DuPont would have owed $8,427,000 for the over 1,380 studies it submitted to the EPA. DuPont's Motion for Acc. Dec., Ex. 11 (Docket No. TSCA-96-H-47, Complaint, Sept. 30, 1996 ("1996 Complaint")) at 6.

[40] As noted previously within this decision, the Consent Order states that DuPont registered for the CAP on or about July 5, 1991. However, the latter date would make the registration untimely due to the deadline of July 1, 1991, unless the EPA granted a registration extension to DuPont. Nevertheless, OCE does not raise an argument as to the timeliness of DuPont's registration. Moreover, OCE makes the (unsupported) assertion in its briefs that DuPont registered on or about June 28, 1991, which would render DuPont's registration timely.

distribution in environmental media" and "emergency incidents of environmental contamination" under Section 8(e). *Id.* Moreover, the June 1991 notice added a stipulated penalties provision, at $5,000 each, regarding studies or reports that were received by the EPA prior to June 18, 1991, but were late in meeting the 15-day reporting deadline under the 1978 Enforcement Policy. *See id.* at 28,458-59. The CAP Agreement DuPont signed reflects the modifications from the April 1991 and June 1991 Federal Register notices. Furthermore, the CAP Agreement DuPont signed on or about July 5, 1991 provides that the CAP "shall commence no later than July 1, 1991." CAP Agreement, I.D.

With the June 1991 notice, the EPA suspended Parts V(b)(1) and V(c) of the 1978 Enforcement Policy, which concern "widespread and previously unsuspected distribution in environmental media, as indicated in studies (excluding materials contained within appropriate disposal facilities)" and "emergency incidents of environmental contamination," respectively. 56 Fed. Reg. at 28,459. The June 1991 notice states that in reviewing the 1978 Enforcement Policy "in connection with the TSCA Section 8(e) Compliance Audit Program," the EPA has determined that Part V(b)(1) and Part V(c) of the 1978 Enforcement Policy need additional clarification and that possible misinterpretation with regard to the guidance in these sections could lead to overreporting *under the TSCA Section 8(e) Compliance Audit Program*." *Id.* (emphasis added). Therefore, the EPA announced plans to review the reporting of information in order to determine what information of these types should "continue to be considered for submittal" under Section 8(e), and that interested persons would be allowed the opportunity to comment on proposed revisions to Parts V(b)(1) and V(c). *Id.*

The June 1991 notice stated that, despite the suspension of V(b)(1) and V(c) of the 1978 Enforcement Policy, "regulatees auditing their files for reportable environmental risk information under the TSCA Section 8(e) Compliance Audit Program should be guided by the statutory language of section 8(e) and Part V(b)(2) through (b)(5) of the [1978 Enforcement Policy]." *Id.* Moreover, "In assessing whether information or studies involving widespread and previous unsuspected environmental distribution, emergency incidents of environmental contamination, or other previously unknown situations involving significant environmental contamination should be submitted under the TSCA Section 8(e) Compliance Audit Program, *or under section 8(e) in general*, regulatees should make a reasonable judgement whether such information meets the statutory standards of TSCA section 8(e) instead of relying on Parts V(b)(1) or V(c) of the [1978 Enforcement Policy]." *Id.* (emphasis added). The June 1991 notice concluded with the admonition that, "Even though EPA is suspending the applicability of Parts V(b)(1) and V(c) of the [1978 Enforcement Policy], persons are still responsible under TSCA section 8(e) to report information that reasonably supports a conclusion of substantial risk of injury to the environment. This is a continuing statutory obligation." *Id.*

Thus, the June 1991 notice can reasonably be read as having been addressed towards two groups: (1) regulatees auditing for information pursuant to the CAP ("under the Compliance

Audit Program") *and* (2) persons acting "under Section 8(e) in general."[41] *See id.* Furthermore, the second group, for persons acting "under Section 8(e) in general," would appear to address ongoing compliance. Despite the notice's announcement of considering revisions to Parts V(b)(1) and V(c) of the 1978 Enforcement Policy, it only announced an extension for registration *under the CAP* and an extension for submitting audited information *under the CAP*. Moreover, the June 1991 notice did *not* announce an extension of the normal 15-day reporting deadline information that was not gathered pursuant to the CAP.

I observe that there is a dispute as to the cutoff date for defining the latest time included in the CAP audit period, with DuPont asserting, *in arguendo*, that even if the CAP constituted a lookback audit, then it was a lookback from 1992, the original reporting deadline, rather than July or February 1991.[42] As noted in the above discussion, the CAP was a one-time program involving "outstanding" data, and the June 1991 notice can be read as requiring ongoing compliance as of June 1991; the CAP Agreement was signed shortly after the June 1991 notice. Therefore, it is possible to draw an inference in favor of one or more of the cutoff dates submitted by OCE.

The September 1991 notice described the June 1991 notice as follows: "With regard to Parts V(b)(1) and V(c) of the [1978 Enforcement Policy], the regulated community was informed that until such time as EPA refined its guidance regarding the types of information on the release of chemical substances to and the detection of chemical substances in environmental media that are reportable under section 8(e) of TSCA, regulatees should focus on the statutory language of TSCA section 8(e) and make a reasonable judgment whether such information is reportable for purposes of TSCA Section 8(e) CAP *as well as ongoing compliance with section 8(e).*" September 1991 notice, 56 Fed. Reg. at 49,478 (emphasis added).

The September 1991 notice split the CAP into two phases. *Id.* at 49,479. It announced, "Because refinement of guidance on reportability of information on chemical release/detection in environmental media is underway, EPA is extending the reporting deadline for reporting such information *under the* TSCA Section 8(e) *CAP* to 6 months after publication of final reporting guidance." *Id.* (emphasis added). At the time of the September 1991 notice, the EPA reportedly anticipated publishing the final guidance in Spring 1992. *Id.*

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[41] The CAP Agreement DuPont signed, on or about July 5, 1991, reflects the CAP modifications announced in the February, April, and June 1991 Federal Register notices. Additionally, the Consent Agreement references those Federal Register notices. Consent Agreement, Part I.A.

[42] As discussed previously, *supra* note 22 and accompanying text, OCE has put forth cutoff dates of February 1, 1991, June 28, 1991, and July 1, 1991. The September 1991 and November 1991 dates that OCE contends "come[] into play" in Count II postdate even the latest of the cutoff dates suggested by OCE.

To reflect the September 1991 modification to the CAP program, an Addendum, entitled "Addendum to CAP Agreement," was to be sent to all persons registered for the CAP and to be added to all CAP Agreements. *Id.* The September 1991 notice includes an Addendum, providing that the CAP for the reporting of "information on the release of chemical substances to and detection of chemical substances in environmental media" shall terminate six months after the EPA publishes final refined guidance on such reporting.[43] *Id.* Furthermore, "This modification applies only to reporting of information on the release of chemical substances to and detection of chemical substances in environmental media. The deadline for reporting all other information *under the* TSCA Section 8(e) *CAP* remains unchanged at February 28, 1992." *Id.* (emphasis added). The Addendum further provided, "All TSCA Section 8(e) *Compliance Audit Program* submissions regarding information on the release of chemical substances to and detection of chemical substances in environmental media must be delivered to EPA no later than 6 months after EPA publishes final guidance refining the [1978 Enforcement Policy] as it pertains to such reporting." *Id.*

The Addendum provided for there to be two Final Reports, with the first Final Report listing all studies or reports listed or submitted to the EPA by the Regulatee other than those regarding information on the release of chemical substances to and detection of chemical substances in environmental media, and was to be submitted no later than February 28, 1992. *Id.* The second Final Report was to list each study or report listed or submitted to the EPA by the Regulatee regarding information on the release of chemical substances to and detection of chemical substances in environmental media, and was to be submitted no later than six months after the EPA published final refined guidance on the reporting of such information. *Id.* The Addendum further provided that one Consent Agreement and Consent Order would be presented to the Regulatee, and that the Consent Agreement and Consent Order would be presented after EPA's receipt of the second Final Report, regarding information on the release of chemical substances to and detection of chemical substances in environmental media, and would cover all information submitted by the Regulatee *under the CAP*. *Id.*

The September 1991 notice may be reasonably read as supporting OCE's position. Although the September 1991 notice extends the deadline for reporting "information on the release of chemical substances to and detection of chemical substances in environmental media," the notice repeatedly states that it is extending the deadline for "reporting information *under the CAP*." At no point does the September 1991 notice extend the deadline for *ongoing compliance* with Section 8(e).

With the 1993 Federal Register notice, the EPA published proposed revisions to the 1978 Enforcement Policy in regards to "mandatory reporting of information on the release of chemical substances to, and the detection of chemical substances in, environmental media," and other

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[43] The parties have not provided this Tribunal with a copy of the Addendum that DuPont signed. The Consent Agreement states, "Respondent submitted the Addendum to EPA on September 26, 1992; however, EPA presently has no record of an Addendum for Respondent." Consent Agreement, I.C.

matters, and it solicited public comment on that proposal. TSCA Section 8(e); Notice of Clarification and Solicitation of Public Comment, 58 Fed. Reg. 37,735 (July 13, 1993) ("1993 notice"). The 1993 notice recounted the history of the CAP, stating that on February 1, 1991 EPA announced a "one-time" voluntary compliance audit program designed primarily to: (1) achieve the EPA's goal of obtaining any "outstanding" Section 8(e) information, and (2) encourage companies to voluntarily audit their files for Section 8(e)-reportable data. *Id.* at 37,736. In exchange, the CAP incorporated stipulated monetary penalties and an overall monetary penalty ceiling. *Id.* In reviewing existing guidance as the result of questions raised by companies considering participating in the CAP, the EPA suspended the applicability of Parts V(b)(1) and V(c) of the 1978 Enforcement Policy. *Id.* The regulated community was informed that the EPA would modify the Section 8(e) policy to provide greater specificity regarding the types of information that should be submitted under Section 8(e). *Id.* In the interim, the "regulated community" was directed by EPA to focus on the statutory language of Section 8(e) as the standard by which to determine the reportability of such information "for purposes of the Section 8(e) CAP *as well as ongoing compliance with section 8(e).*" *Id.* (emphasis added). On September 30, 1991, the "EPA announced an extension of the section 8(e) *CAP* reporting deadline for information relating to the release of chemical substances to and detection of chemical substances in environmental media until such time as [the EPA] develops final refined section 8(e) reporting guidance on this point." *Id.* (emphasis added). The September 1991 notice "addresses only the reportability of information concerning non-emergency situations on 'widespread and previously unsuspected distribution in environmental media.'" *Id.* The 1993 notice announced that the EPA was deferring publishing refined and/or amending the Section 8(e) guidance regarding emergency incidents of environmental contamination information, considering that such guidance should be developed as part of the EPA's over-all policy concerning Federal chemical emergency/accident prevention, reporting, response, and/or remediation. *Id.*

The 1993 notice announced that the EPA was in the process of resolving enforcement and compliance issues concerning reporting of "section 8(e) 'environmental' information under 'Phase 2' *of the CAP,* and under section 8(e) more generally." *Id.* (emphasis added). It further stated that after the EPA considers comments in response to the 1993 notice, the EPA would issue in the Federal Register final refined guidance for reporting information concerning non-emergency situations regarding "environmental contamination." *Id.* Following was Section 8(e) policy changes, including proposed changes to Part V(b)(1) ("widespread and previously unsuspected distribution in environmental media") of the 1978 Enforcement Policy, but it did not propose changes to Part V(b)(2)-(5). *Id.* at 37,741.

Again, the 1993 notice may be read as supporting OCE's view. As with the June 1991 and September 1991 notices, it suggests reporting "under Phase 2 of the CAP" and reporting "under Section 8(e) more generally" were separate. It describes the September 1991 notice as extending the *CAP* deadline. There is no mention of an extension of the deadline for ongoing compliance.

Through a 1995 Federal Register notice, the EPA solicited additional public comment on revisions to Part V(b)(1) of the 1978 Enforcement Policy. TSCA Section 8(e); Notice of Availability of Draft Policy and Reopening of Comment Period, 60 Fed. Reg. 14,756 (Mar. 20, 1995) ("1995 notice"). The 1995 notice stated that the EPA had used the comments received in response to the 1993 proposed revisions to draft revised policy text that the EPA believed responded to the main comments. *Id.* Further, the 1995 notice announced that the EPA was making available for public comment the draft guidance text in the public docket.[44] *Id.* Comments were to be submitted and received by the EPA no later than May 4, 1995. *Id.*

The Cover Letter to the Revised Addendum, dated May 15, 1996, as well as the Revised Addendum, was signed by the EPA's Mr. Jesse Baskerville, who was Director of the Toxics and Pesticides Enforcement Division. The Cover Letter was addressed to DuPont, and below DuPont's address the salutation reads "Dear CAP Participant:" and then states that the September 1991 notice "[a]nnounced . . . an extension of the TSCA Section 8(e) *CAP* reporting deadline for submission of information regarding release of chemical substances to and detection of chemical substances in environmental media." Cover Letter at 1 (emphasis added). The Cover Letter states that the September 1991 "[a]nnouncement established a Phase Two of the *CAP* for section 8(e) information on the release of chemical substances to and the detection of chemical substances in environmental media and environmental toxicity data for plant effluents." *Id.* (emphasis added). The Cover Letter provides, "All TSCA Section 8(e) *CAP* submissions under Phase 2 were to be delivered to EPA no later than six months after EPA publishes final revised environmental guidance ('guidance')," and "The exact date would appear in the Federal Register notice announcing the revised guidance." *Id.* (emphasis added). The Cover Letter further states:

> On January 30, 1992, EPA provided CAP participants with an "Addendum to CAP Agreement" and policy statements that formally established the Two Phases to the CAP, and permitted the submission of the following information during Phase Two:
>
> > information on the release of chemical substances to and detection of chemical substances in environmental media, and
> >
> > environmental toxicity testing performed on plant effluents.

Id. The Cover Letter advised CAP participants: "The deadline for reporting all other information under the TSCA section 8(e) Compliance Audit Program remained unchanged at February 28, 1992 unless otherwise extended," and "The Addendum was to be executed by the Regulatee and returned to EPA for ratification and entry." *Id.*

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[44] Neither party has submitted to this Tribunal the 1995 draft revisions.

The Cover Letter recounts, "Since ratification of the Addendum, EPA has twice issued, for notice and comment, revised draft reporting guidance." *Id.* at 2. The Cover Letter states, "After review of extensive comments, EPA has decided that it is reasonable and equitable to enforce the final revised reporting guidance *on a prospective basis only*." *Id.* (emphasis added). "Therefore, information on the release of chemical substances to and detection of chemical substances in environmental media; or environmental toxicity data on plant effluents *that predate the effective date of the guidance will not be the subject of an EPA TSCA Section 8(e) enforcement action*." *Id.* (emphasis added). Next, the Cover Letter states, "We are aware that some CAP participants may have submitted this data under Phase 1 of the CAP program. Accordingly, *penalties will not be assessed* for any Phase 2 type studies or reports submitted under the TSCA Section 8(e) CAP as TSCA Section 8(e) data." *Id.* (emphasis added).

Mr. Baskerville states in the Cover Letter, "To effectuate this decision it is necessary to revise the previously ratified Addendum, and modify the [CAP Agreement]. *Id.* Accordingly, he states, "The attached Revised Addendum to the CAP Agreement supersedes the previous Addendum and specifies the following:

> The Regulatee no longer is required to conduct a file search for information on the release of chemical substances to and detection of chemical substances in environmental media, or for environmental toxicity data on plant effluents.
>
> A second Final Report is no longer necessary. Therefore, the first Final Report becomes the controlling document described in Unit II.A.8. of the CAP Agreement.

Id.

The Cover Letter may reasonably be read as OCE argues: as indicating a prospective waiver of enforcement, because the Cover Letter states that the EPA has decided to enforce the final revised reporting guidance "on a *prospective* basis only." *Id.* (emphasis added). The Cover Letter (and Revised Addendum) is to be read within the overall context, which includes the Federal Register notices. As discussed, the Federal Register notices indicate that up to the date of the Cover Letter and Revised Addendum, the EPA had been requiring persons to make a reasonable judgment whether V(b)(1)-type information ("widespread and previous unsuspected environmental distribution"), V(c)-type information ("emergency incidents of environmental contamination"), "or other previously unknown situations involving significant environmental contamination" should be submitted under the CAP *or* under Section 8(e) in general. *See* June 1991 notice, 56 Fed. Reg. at 28,458. EPA's September 1991 extension of the reporting deadline for information on the release of chemical substances to and detection of chemical substances in environmental media *only* applied for reporting information "under the TSCA Section 8(e)

CAP," and it did not extend the deadline for *ongoing* compliance.[45] *See* September 1991 notice, 56 Fed. Reg. at 49,478 (emphasis added).

The 2003 Comment and Response Document, dated February 20, 2003, comes before the final revised guidance and comments on the proposal to finalize that guidance. The 2003 Comment and Response Document states that companies will not have to review "*preexisting* files" for information that may be subject to Section 8(e) reporting, and "These *preexisting* files would only come into 'play' if data obtained by a company after the effective date of the guidance triggered a review of such data and in doing so the combination of data met the section 8(e) reporting criteria." 2003 Comment and Response Document at 1. Nevertheless, this document may be read within the context of OCE's theory that ongoing compliance with the statute was required from February or July 1991 through 1996, which can be seen from the Federal Register notices, and that Paragraph IV.A of the Revised Addendum only prospectively waived enforcement up to the date of the final revised guidance.

In sum, the extrinsic evidence proferred by the parties does not render the Consent Agreement, including the CAP Agreement and Revised Addendum, unambiguous in favor of the movant. Taking into account the extrinsic evidence, as argued by OCE one may reasonably view the CAP as a "lookback" audit that includes information generated before February or July 1991; that ongoing compliance was required for information generated on or after February or July 1991, and was required from February or July 1991 up to June 27, 1996 – the date of the Revised Addendum, and; that the EPA never suspended reporting for ongoing compliance but *only* suspended the auditing and reporting for information under the *CAP* (i.e., only information generated before February or July 1991). Therefore, one may reasonably interpret Paragraph IV.A of the Revised Addendum as a *prospective* waiver, meaning it applies only as to the information generated from June 27, 1996 forward up to the effective date of the final revised guidance, which was issued in 2003. In essence, one may reasonably view the Compliance Audit Program as not applying the alleged Count II violations, as argued by OCE.[46]

Pursuant to the summary judgment standard, DuPont has not proven that the waiver of enforcement in Paragraph IV.A of the Revised Addendum waives enforcement over the September and November 1991 dates that OCE contends "comes into play" in Count II. As

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[45] I recognize, however, that even if reporting of information obtained from February or July 1991 through May or June of 1996 was required, the EPA *may* have decided to change course in 1996, to completely waive enforcement even for pre-1996 violations. After all, as discussed *supra*, even prior to the 1996 Cover Letter and Revised Addendum, the EPA had recognized, as indicated in the Federal Register notices, that there were problems with the 1978 Enforcement Policy that could result in overreporting. *E.g.*, June 1991 notice, 56 Fed. Reg. at 28,459.

[46] *See* Oral Arg. Tr. at 65; OCE's Count II Response at 14-15.

such, summary judgment is not appropriate on this issue and an evidentiary hearing is warranted.[47]

H. Genuine Issues of Material Fact

In addition to finding that accelerated decision is not warranted as a matter of law, I further find that genuine issues of material fact exist. For example, there is a genuine dispute of material fact as to whether the levels of PFOA allegedly detected in DuPont's wells at levels as high as 3.9 ppb reasonably support a conclusion of substantial risk of injury to health or the environment. In particular**,** DuPont contends that there is "overwhelming scientific evidence" that levels of 3.9 ppb or less PFOA in drinking water pose no risk, and that a multi-agency scientific panel, which includes EPA scientists, has determined that a lifetime of daily exposure to PFOA concentrations of up to 150 ppb in all drinking water that a person ingests would not be expected to result in any deleterious effects. DuPont's Motion for Acc. Dec. at 3; *but see* OCE's Count II Response at 4; *see also, e.g.*, EPA's Count II Response, Ex. 1 (discussing standards set at 1 ppb versus 150 ppb). A prerequisite to TSCA Section 8(e) liability is that the information obtained by the respondent must reasonably support the conclusion that a substance or mixture presents a substantial risk of injury to health or the environment. 15 U.S.C. § 2607(e).

I. Res Judicata

DuPont argues that, regardless of whether the EPA waived enforcement over the Count II claim, the doctrine of res judicata bars the EPA from bringing those claims due to the EAB's Consent Order that approved the parties' Consent Agreement. DuPont's Motion for Acc. Dec. at 27.

The doctrine of res judicata, also known as claim preclusion, applies both to judicial consent decrees and to administrative consent agreements. *In re Int'l Paper Co.,* RCRA (3008) Appeal No. 90-3, 3 E.A.D. 562, 567 (CJO 1991). Typically, when a court enters a final judgment on the merits in an action, the doctrine of res judicata bars the parties from re-litigating the same cause of action in a subsequent suit. *In re Wego Chem. & Mineral Corp.,* TSCA Appeal No. 92-4, 4 E.A.D. 513, 520 (EAB 1993); *Int'l Paper*, 3 E.A.D. at 567; *accord Nevada v. United States*, 463 U.S. 110, 129-30 (1983). Under the doctrine of res judicata, the moving party bears the burden to show the following requirements: (1) there was a final judgment on the

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[47] Although not necessarily determinative, it would be helpful to be informed about the actions of the parties and other CAP participants with regards to "Phase 2" information generated from February 1, 1991 through June 27, 1996. In particular, prior to the instant case being filed, has the EPA ever brought any Section 8(e) enforcement action(s) against any CAP participant for failure to report any Section 8(e) information generated from February 1, 1991 through June 27, 1996? From February 1, 1991 up to the current date, did DuPont, or any other CAP participant, report to the EPA any "Phase 2" Section 8(e) information generated from February 1, 1991 through June 27, 1996? Arguably, such information may establish the parties' course of performance.

merits in a prior action, (2) involving the same parties, and (3) the subsequent proceeding is based on the same cause of action. *Wego,* 4 E.A.D. at 520. The parties disagree as to whether the subsequent proceeding is based on the same nucleus of operative facts. Oral Arg. Tr. at 80-81. At oral argument, OCE also asserted that there was not a final judgment on the merits in regards to the violations alleged in Count II such that is has preclusive impact over the instant action. *Id.* at 81-84. It is not necessary to reach the latter issue at this time because, as discussed below, I conclude that DuPont has not proven that there is no genuine issue of material fact regarding whether the instant proceeding is based on the same cause of action as in the prior action.

"Whether two cases implicate the same cause of action turns on whether they share the same 'nucleus of facts.'" *Apotex, Inc. v. FDA,* 393 F.3d 210, 217 (D.C. Cir. 2004); *accord Int'l Paper,* 3 E.A.D. at 568 (barring a claim on the ground of res judicata where the issues arose out of the "same nucleus of operative facts" as those raised and settled previously and therefore involved "the same cause of action"). "In pursuing this inquiry, the court will consider 'whether the facts are related in time, space, origin, or motivation, whether they form a convenient trial unit, and whether their treatment as a unit conforms to the parties' expectations or business understanding or usage.'" *Apotex,* 393 F.3d at 217 (quoting *I.A.M. Nat'l Pension Fund v. Indus. Gear Mfg. Co.,* 723 F.2d 944, 949 n. 5 (D.C. Cir.1983), quoting 1B J. Moore, Moore's Fed. Practice ¶ 0.410[1] (2d ed. 1983)); *see also Wego,* 4 E.A.D. at 520 (whether or not a cause of action in a judgment and a case are considered the same also hinges, among other things, on: (1) whether the acts complained of are the same; (2) whether the material facts are the same; and (3) whether the proof required is the same (citing *United States v. Athlone Inds.,* 746 F.2d 977, 984 (3rd Cir. 1984)).

On October 3, 1996, the EAB executed a Consent Order, which approved the Consent Agreement. DuPont's Motion for Acc. Dec., Ex. 13. The Consent Order provides that "Respondent shall comply with all terms of the Consent Agreement, incorporated herein by reference."[48] *Id.* Attached to the Consent Order is the Consent Agreement, and the attachments thereto: the CAP Agreement and the Revised Addendum. *See id.*

DuPont argues that Count II arises out of the same nucleus of operative facts resolved by the Consent Order, and that res judicata therefore bars Count II. DuPont's Motion for Acc. Dec. at 27. Specifically, DuPont argues, "Here, the Consent Order and Count II do not just arise out of the same nucleus of operative facts[;] they involve the very same issue: whether TSCA § 8(e) required DuPont to report information on the detection of chemical substances in environmental media received by or known to DuPont before EPA issued its final guidance for such reporting." *Id.* at 28. DuPont contends that the Consent Order, by incorporating by reference the Revised

[48] It may be that the EAB received correspondence or other communications from the Office of Regulatory Enforcement or other EPA offices before or contemporaneous with the EAB's approval of the Consent Order. *See* Preamble to Rules of Practice, 64 Fed. Reg. 40,138, 40,149 (July 23, 1999) (discussing the TSCA Section 8(e) CAP within the context of ex parte communications). To date, this Tribunal has not been provided such information.

Addendum, specifically addresses DuPont's obligation to report the detection of chemical substances in drinking water and finally resolves that DuPont need not report such information prior to the EPA issuing its final guidance. *Id.* at 28-29. DuPont argues, "Because the Consent Order addresses DuPont's obligation under TSCA § 8(e) to report detection of any chemical in water samples if the detection occurred before EPA issued its final guidance, and because Count II alleges that DuPont obtained and failed to report detection of PFOA in such samples before EPA issued its final guidance, Count II arises out of the same nucleus of operative facts . . . resolved in the Consent Order." *Id.* at 29.

Furthermore, DuPont argues that res judicata bars OCE from asserting Count II on the ground that OCE could have asserted but did not assert the current Count II in the prior litigation. *Id.* (citing *Nevada,* 463 U.S. at 129-30); DuPont's Post-Argument Br. at 10. DuPont argues, "Not only do Count II and the Consent Order arise from the same nucleus of operative facts, but it is readily apparent that EPA could have brought the current Count II in the [1996] Complaint that led to the Consent Order." DuPont's Motion for Acc. Dec. at 29 (referring to DuPont's Motion for Acc. Dec., Ex. 10: Complaint, Docket No. TSCA-96-H-47 ("1996 Complaint")). DuPont points out that in the 1996 Complaint, the EPA noted DuPont's obligation to report the presence of chemical substances in environmental media. *Id.* (citing 1996 Complaint at 2, 7). DuPont states that, in 1996, the EPA could have alleged that DuPont was liable for failing to make such reports, but rather than allege that DuPont was liable for failing to report such information, the EPA instead stated that DuPont was no longer required to conduct a file search for such information. *Id.* (citing 1996 Complaint at 7). Finally, DuPont points to a February 9, 1990 Verification Investigation Workplan ("VIW") addressed to the EPA, "[t]elling the [EPA] that DuPont in 1990 had detected C8 or PFOA in the Lubeck wells. These are the same water samples that form the basis of Count 2. EPA was aware of that. Clearly they could have brought those claims." Oral Arg. Tr. at 28 (citing OCE's Count II Response, Ex. 20 at 18).

OCE counters that the Revised Addendum does not cover the September and November 1991 dates on which Count II is based. Oral Arg. Tr. at 81. OCE also quotes the Consent Agreement/Order: "This Consent Agreement and Consent Order shall be a complete settlement of all administrative claims and civil causes of action alleged in the Complaint." OCE's Count II Response at 22 (quoting Consent Order at 1). OCE argues that the Complaint in the instant case is limited to the studies that were submitted by DuPont, and does not encompass studies withheld by regulatees. *Id.* According to OCE, it is that alleged withholding that has given rise to this action, the question being whether DuPont violated Section 8(e) when it failed to provide certain data, and that OCE's case in chief turns on DuPont's violations of Section 8(e) notwithstanding the existence of the CAP. *Id.* at 22-23. OCE argues that the instant case concerns DuPont's failure to report studies to the EPA between 1991 and 1996, which is the time period OCE contends was not covered by a waiver of enforcement and during which ongoing compliance with Section 8(e) was required. Oral Arg. Tr. at 81. Therefore, so argues OCE, the Consent Order in the instant case did not arise from the same claims and does not act as a bar. *Id.* Furthermore, OCE points out that the Consent Agreement/Order specifically permits matters of non-compliance to be litigated. *Id.* (referring to Consent Agreement, Part VI.A, and; CAP Agreement, Unit II.D.1). As for the February 1990 VIW that purportedly put the EPA on notice

of the alleged violation prior to the announcement of the CAP, OCE contends that the EPA rejected this submission *in toto* as deficient, and when DuPont resubmitted its revised VIW in December 1990, it had omitted the statement regarding Lubeck public supply well contamination. OCE's Post-Argument Br. on Count II at 13 (citing OCE's Count III Response, Ex. 6 (Dec. 14, 1990 VIW), at 26).

As discussed *supra*, the Revised Addendum could be read as not covering the September and November 1991 dates that allegedly form the basis for Count II. Accordingly, there is a genuine dispute of material fact regarding whether the Consent Order and Count II are based on the same nucleus of operative facts. In addition, looking at the Consent Order, I observe that the EAB expressly incorporates the terms of the parties' Consent Agreement, which includes the CAP Agreement and the Revised Addendum. As correctly observed by OCE, the Consent Agreement does provide that matters of non-compliance may be litigated. Consent Agreement, Part VI.A; CAP Agreement, Unit II.D.1.

Finally, there may be a genuine dispute of material fact as to whether the information on which Count II is based is pre-February or pre-July 1991 information.[49] OCE submits that Count II is based on environmental contamination data that Dupont allegedly became aware of in mid to late 1991, more specifically September 11, 1991 and November 19, 1991. Oral Arg. Tr. at 62, 71. OCE points out that the TSCA reporting obligation accrues when a company becomes aware of information that indicates substantial risk to health or the environment. *Id.* at 62. However, OCE admits that the environmental contamination data at issue "[d]oes build on prior data, some data points that may have preceded 1991," *id.*, and OCE has stated that DuPont became aware of the environmental contamination prior to signing the CAP Agreement in 1991.[50] *Id.* at 72.

Accordingly, DuPont has not sustained its burden on summary judgment. Therefore, I **DENY** DuPont's Motion for Accelerated Decision on Count II.

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[49] However, one could argue that the Consent Agreement and CAP Agreement reserve EPA's enforcement authority to litigate pre-1991 violations of the CAP. *See* Oral Arg. Tr. at 81; Consent Agreement, Part VI.A; CAP Agreement, Unit II.D.1.

[50] One of OCE's exhibits, the minutes from a meeting on September 11, 1991, indicates that in 1984, C-8 (i.e., PFOA or APFO) was found at levels less than 1.5 ppb, downgradient from DuPont's Washington Works facility. OCE's Count II Response, Ex. 23. The minutes further state, "However, data do not indicate large increases in C-8 concentration since 1987, (from 2.0 to 5.9 ppb)." *Id.* The minutes continue that "Off-site water samples from home taps (i.e. from the existing Lubeck wellfield) indicate C-8 from .7 to 3.9 ppb, with the 3.9 ppb measured from a sample taken on 8/8/91." *Id.* "C-8 was detected in a new well in the new Lubeck wellfield (2.7 miles south-southwest of Washington Works), at 2.4 ppb on 6/23/91." *Id.* Considering that OCE has stated that the mid to late 1991 information forming the basis for Count II "does build on prior data, some data points that may have preceded 1991," there is a genuine dispute of material fact as to whether the data on which Count II is based should be viewed as pre or post 1991 information.

II. Count III

The parties have filed cross-motions for accelerated decision as to Count III. For the reasons discussed herein, resolution of this matter is more appropriate for an evidentiary hearing, and therefore, the parties' motions for accelerated decision are denied.

A. The Allegations Forming the Basis of Count III

DuPont admits that on or about January 5, 1987, the West Virginia Department of Natural Resources, Division of Waste Management issued to DuPont a RCRA permit for the treatment, storage, or disposal of hazardous waste at DuPont's Washington Works facility. Amended Answer ¶ 35. DuPont admits that on December 13, 1989, the EPA issued to DuPont the corrective action portion of DuPont's permit for the Washington Works facility. *Id.* ¶ 37. Moreover, on December 16, 1999, the EPA extended the term of the corrective action portion of DuPont's RCRA permit for the Washington Works facility until the effective date of a new corrective action permit for the Washington Works facility. *Id.* ¶ 38.

OCE alleges that in 1981, when performing blood sampling of pregnant workers at the Washington Works Facility, DuPont obtained human blood sampling information concerning the transplacental movement of PFOA (i.e., "C-8" or "APFO"). Amended Complaint ¶¶ 42, 106 (referring to OCE's Count III Response, Ex. 3 (titled "C-8 Blood Sampling Results"). DuPont admits that the document at issue contains numbers that purport to be levels of PFOA detected in the blood of DuPont employees. Amended Answer ¶ 42.

In Count III, OCE alleges that DuPont committed a RCRA permit violation by failing to comply with its duty to provide information, as provided by Part One, Section I.7, of DuPont's RCRA Corrective Action Permit for the Washington Works facility. Amended Complaint ¶¶ 111-113; *see* OCE's Count III Response, Ex. 1 ("DuPont's Corrective Action Permit"), Part One, § I.7. Specifically, OCE alleges that on or about May 5, 1997, the EPA issued a Notice of Deficiency to DuPont for a Verification Investigation Report ("VI Report"). Amended Complaint ¶ 102. In the Notice of Deficiency, the EPA reportedly requested that DuPont provide a response to the EPA, within 30 days of receipt, for all deficiencies identified in the Notice. *Id.* In the "Groundwater" section of the Notice of Deficiency, the EPA allegedly requested that DuPont provide to the EPA "known toxicological information" regarding C-8.[51] *Id.* ¶ 103. In DuPont's "Response to Notice of Deficiency," DuPont allegedly directed the EPA to information that was included in the VI Report and provided "[a]dditional C-8 toxicological

[51] Specifically, the request for information states: "Section 7.2 [of DuPont's VI Report] discusses that C-8 and TRITON®, found in wells at the Riverbank Landfill, the Anaerobic Digestion Ponds, and the Burning Grounds, are not 40 CFR Part 264, Appendix IX constituents and PALs ["Proposed Action Levels"] or MCLs ["Maximum Contaminant Levels"] assigned to them. Please provide known toxicological information." EPA's Count III Response, Ex. 9 at 2-3.

information" as Attachment 2 of the Response to Notice of Deficiency, titled "Toxicological Information on C-8." *Id.* ¶ 104. OCE alleges that DuPont's "Toxicological Information on C-8" document included a section regarding "Health Hazardous Data." *Id.* ¶ 105. Furthermore, OCE alleges that in the Response to the Notice in June 1997, DuPont did not provide "all 'known toxicological information'" it had regarding C-8 because it did not provide to the EPA the information regarding the transplacental movement of C-8 in humans, and that DuPont has not provided such information to the EPA. *Id.* ¶¶ 108-109. OCE alleges that all known toxicological information about C-8 is "relevant information" that the EPA might request to determine whether cause exists for modifying, revoking and reissuing, or terminating DuPont's Corrective Action Permit, or to determine compliance with this permit. *Id.* ¶ 110 (citing, *inter alia*, EPA's Count III Response, Ex. 1 (DuPont's Corrective Action Permit), Part One, § I.7; 40 C.F.R. § 270.30(h)).

OCE further alleges that DuPont's failure to provide "known toxicological information" constitutes noncompliance with DuPont's duty to provide information as required by Part One, Section I.7, of DuPont's Corrective Action Permit, and therefore DuPont did not comply with all conditions of its permit. *Id.* ¶¶ 111-112 (citing 40 C.F.R. § 270.30(h) and West Virginia Hazardous Waste Management Rule ("WVHWMR") § 33-20-11.1).[52] In conclusion, OCE alleges that from at least June 6, 1997, until at least March 6, 2001, DuPont was in violation of: Section 3005(a) of RCRA, 42 U.S.C. § 6925(a); Part One, Section I.7 of DuPont's Corrective Action Permit, and; 40 C.F.R § 270.30(h), and WVHWMR § 33-20-11.1, by failing to provide the information requested by EPA. Amended Complaint ¶ 113.

DuPont admits that its June 1997 Response to Notice did not expressly inform the EPA about the 1981 document mentioning the umbilical cord blood sample. Amended Answer ¶ 108. However, DuPont denies that such information is "known toxicological information." *Id.* ¶¶ 107, 108.

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[52] OCE points out that on May 29, 1986, the EPA granted the State of West Virginia final authorization to administer its base hazardous waste management program in lieu of the federal base hazardous waste management program, and that the provisions of the West Virginia hazardous waste management program became requirements of RCRA and are enforceable by the EPA pursuant to Section 3008(a) of RCRA, 42 U.S.C. § 6928(a). Amended Complaint at 2. OCE further points out that on July 10, 2000, the EPA authorized revisions to West Virginia's base hazardous waste program, and that the provisions of the revised program are enforceable by the EPA. *Id.* at 2-3. However, OCE also points out that at all relevant times for purposes of the Count III violation, West Virginia was not authorized to implement the Federal Corrective Action Program. *Id.* at 3. *See In re Pyramid Chem. Co.*, Docket No. RCRA-HQ-2003-0001, 2004 EPA App. LEXIS 32, at *34 (EAB, Sept. 16, 2004), 11 E.A.D. ___ (State regulations only become the operative standards in lieu of the Federal program as to "[t]hose aspects of RCRA for which the state program is authorized."). I would point out that the West Virginia regulation cited in the Amended Complaint, WVHWMR § 33-20-11.1, adopts and incorporates by reference 40 C.F.R. part 270, except as to provisions that do not appear to be determinative for Count III.

B. EPA's Authority to Request Information

In its initial Complaint (and Amended Complaint), OCE alleged that C-8 is a "hazardous constituent," and then amended its Complaint in response to DuPont's Motion for Accelerated Decision, to add an allegation that PFOA (i.e., "C-8" or "APFO") "is a discarded material and a 'solid waste' as defined under RCRA § 1004(27), 42 U.S.C. § 6903(27) and a 'hazardous waste' as defined under RCRA § 1004(5), 42 U.S.C. § 6903(5)." Amended Complaint ¶ 34; *see also id.* ¶¶ 99, 101. In seeking amendment of the Complaint, OCE stated that the allegation was added for the purpose of responding to DuPont's legal arguments about EPA's authority to address PFOA under Section 3004(u) of RCRA and to address the factual issue raised by DuPont regarding whether PFOA is a hazardous waste, but that OCE need not establish that PFOA is a hazardous waste, and that therefore the allegation is not necessary in order to prevail on Count III. Motion for Leave to File First Amended Complaint (Oct. 13, 2004) at 2.

In essence, DuPont argues that the EPA did not have the authority to request "known toxicological information" about C-8 under the statutory provisions of RCRA because C-8 is neither a hazardous constituent nor a hazardous waste listed or identified under EPA's regulations. DuPont's Count III Reply at 2-3. DuPont argues that Congress expressly limited EPA's authority to require corrective action under Section 3004(u) of RCRA, 42 U.S.C. § 6924(u), to hazardous wastes and hazardous constituents identified or listed by EPA in its regulations, rather than the statutory definition in Section 1004(5) of RCRA, 42 U.S.C. § 6903(5).[53] DuPont's Count III Reply at 1-3; *see also* DuPont's Post-Argument Br. at 20-27. Furthermore, DuPont argues that its corrective action permit expressly incorporated EPA's regulatory definition of hazardous waste rather than the statutory definition and that the Permit did not "expand" EPA's statutory authority. *Id.* at 1-3.

Section 3001 of RCRA, titled "Identification and listing of hazardous waste," provides, *inter alia*:

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[53] Section 3004(u), 42 U.S.C. § 6924(u), titled "Continuing releases at permitted facilities," of RCRA provides:

> Standards promulgated under this section shall require, and a permit issued after November 8, 1984, by the [EPA] Administrator or a State shall require, corrective action for all releases of hazardous waste or constituents from any solid waste management unit at a treatment, storage, or disposal facility seeking a permit under this subchapter, regardless of the time at which waste was placed in such unit. Permits issued under section 6925 of this title [i.e., Section 3005 of RCRA] shall contain schedules of compliance for such corrective action (where such corrective action cannot be completed prior to issuance of the permit) and assurances of financial responsibility for completing such corrective action.

> [t]he [EPA] Administrator shall, after notice and opportunity for public hearing, and after consultation with appropriate Federal and State agencies, develop and promulgate criteria for identifying the characteristics of hazardous waste, and for listing hazardous waste, which should be subject to the provisions of this subchapter, taking into account toxicity, persistence, and degradability in nature, potential for accumulation in tissue, and other related factors such as flammability, corrosiveness, and other hazardous characteristics. Such criteria shall be revised from time to time as may be appropriate.

42 U.S.C. § 6921(a). In contrast, Section 1004(5) of RCRA, 42 U.S.C. § 6903(5), defines the term "hazardous waste" as:

> a solid waste, or combination of solid wastes, which because of its quantity, concentration, or physical, chemical, or infectious characteristics may –
>
> (A) cause, or significantly contribute to an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness; or
>
> (B) pose a substantial present or potential hazard to human health or the environment when improperly treated, stored, transported, or disposed of, or otherwise managed.

DuPont responded in part to EPA's request for "known toxicological information" regarding C-8, but did not provide the EPA with the 1981 blood sample results, which DuPont contends are not "known toxicological information." Oral Arg. Tr. at 50-52; *see* Response to Notice of Deficiency (OCE's Count III Response, Ex. 10). DuPont contends that in providing some information about C-8 but omitting the 1981 blood sample results it was not concealing the results but rather trying to respond in good faith, albeit "voluntarily." Oral Arg. Tr. at 52. Moreover, DuPont posits that because the information request did not specifically ask for "all" toxicological information, that DuPont did not have to provide the 1981 blood sample results. *See id.* at 50. DuPont suggests that its response to the information request explained to the EPA that its response was limited. *See id.* at 52 (referring to Response to Notice of Deficiency, Attach. 2 (titled "Toxicological Information on C-8"), at 1: "The following information pertains to the Environmental and Human Health Effects of Ammonium Perfluoroctanoate."). Furthermore, DuPont contends that its correspondence with the EPA indicates that it "[w]as very clear in saying that [the EPA] should understand that this substance is not a hazardous constituent." *Id.* at 52-53.

OCE alleges that the information that the EPA requested from DuPont concerning the C-8 is "relevant information" that the EPA may request to determine whether cause exists for modifying, revoking and reissuing or terminating DuPont's Corrective Action Permit, or to

determine compliance with that permit. Amended Complaint ¶ 110 (citing DuPont's Corrective Action Permit, Part One, § I.7; 40 C.F.R. § 270.30(h)). OCE argues, "The *only* limitation on [EPA's] information request authority is that the Request for Information is relevant to determining whether cause exists to modify, revoke and re-issue, terminate, or to determine compliance," and that such authority is not limited to requesting information regarding substances known to be regulatory hazardous wastes or hazardous constituents. Oral Arg. Tr. at 89 (emphasis added); *see also* OCE's Count III Response at 8-13; OCE's Count III Reply at 17-18; OCE's Post-Argument Br. on Count III at 2, 5-14. Therefore, OCE argues that EPA's authority to request information regarding a substance, such as C-8, is not defeated even if such substance is not a regulatory hazardous waste or a hazardous constituent. OCE's Count III Response at 8-9; OCE's Post-Argument Br. on Count III at 8-14.

OCE points out that, generally, an administrative agency's request for information will be enforced where: (1) the investigation is within the agency's authority, (2) the request is not too indefinite, and (3) the information requested is reasonably relevant. OCE's Post-Argument Br. on Count III at 7-8. Furthermore, OCE states that the EPA Chief Judicial Officer ("CJO") adopted this three part test in the case: *In re Environmental Protection Corp. (East Side) Disposal Facility*, RCRA (3008) Appeal No. 90-1, 3 E.A.D. 318 (CJO 1990), *adopting*, Docket No. RCRA-09-86-0001, 1987 EPA ALJ LEXIS 22 (ALJ, Apr. 8, 1987), *aff'd but rev'd and remanded in part on other grounds*, *Environmental Protection Corp. v. Thomas*, No. CV F-87-447-EDP (E.D. Cal, July 13, 1988) (unpublished mem.), *decision on remand*, 1989 EPA ALJ LEXIS 24 (ALJ, Oct. 24, 1989).

I observe that, pursuant to Section 3007(a) of RCRA, 42 U.S.C. § 6927(a), Congress conferred upon the EPA broad authority to request information. *National-Standard Co. v. Adamkus*, 881 F.2d 352, 360-61 (7th Cir. 1989). I further observe that the Duty to Provide Information section of the Permit reads as follows:

> The Permittee shall furnish, within the specified time, any relevant information which the [EPA] . . . may request to determine whether cause exists for modifying, revoking and reissuing, or terminating this permit, or to determine compliance with this permit. The Permittee shall also furnish to the [EPA], upon request, copies of records required to be kept by this permit. (40 C.F.R. §§ 270.30(h) and 264.74(a))[.]

DuPont's Corrective Action Permit, Part One, § I.7. Furthermore, under the regulation titled "Conditions applicable to all permits," there is a "Duty to provide information," which states:

> The permittee shall furnish to the [EPA], within a reasonable time, any relevant information which the [EPA] may request to determine whether cause exists for modifying, revoking and reissuing, or terminating this permit, or to determine compliance with this permit. The permittee shall also furnish to the [EPA], upon request, copies of records required to be kept by this permit.

40 C.F.R. § 270.30(h).

To borrow language from the ALJ in *Environmental Protection Corp.*, I too observe,

> "It would show a startling suspension of common sense and be a strange and ineffectual enforcement policy if respondents and possible violators were given the discretion and authority to determine what is and is not . . . " relevant to a hazardous waste information request. "To accede to such an argument smacks of relying upon a fox to be completely objective concerning the number of hens in a chicken house."

Environmental Protection Corp., 3 E.A.D. at 320-21 (quoting the ALJ).[54] Furthermore, I read *Environmental Protection Corp.* as indicating that, at the time that the respondent in that case partially responded to the information request, the respondent offered its rational for not submitting other documents requested. *See* 1989 EPA ALJ LEXIS 24, at *18. An argument that the recipient of an EPA information request may unilaterally make the relevancy determination and withhold information without notifying, or without sufficiently notifying, the EPA of such withholding is untenable.

I am persuaded by OCE's arguments concerning its broad authority under DuPont's Corrective Action Permit to request information that is reasonably relevant in determining whether cause exists for modifying, revoking and reissuing, terminating, or determining compliance with the Permit, and that EPA's 1997 request for "known toxicological information" regarding C-8 was not precluded simply because C-8 is not or may not be a regulatory hazardous waste or a hazardous constituent.[55] I point out that the text of neither the duty to provide

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[54] *Accord In re Montco Research Products, Inc.*, Docket No. RCRA-83-165-R-KMC, 1986 EPA ALJ LEXIS 20, at *16 (ALJ, Mar. 4, 1986) ("[T]he purpose of [RCRA] would be thwarted if the decision whether to respond to a § 3007 information request was left to the discretion of the person from whom the information was requested.").

[55] I have considered the Declaration of Marcia E. Williams, which was proffered by DuPont in support of its motion for accelerated decision. DuPont's Count III Reply, Ex. A (Nov. 14, 2004) ("Williams's Declaration"). Ms. Williams was a former long-term official at the EPA from 1970 to February 1988. *Id.* at ¶ 1. Ms. Williams was the Director of EPA's Office of Solid Waste from mid-1985 through February 1988, where she reportedly directed the implementation of RCRA and the Hazardous and Solid Waste Act Amendments of 1984. *Id.* I note that DuPont's Corrective Action Permit was issued *after* Ms. Williams's tenure at the EPA, as it was issued in 1989 and then amended in 1999. Amended Answer ¶¶ 37, 38.

Nevertheless, Ms. Williams opines that the types of information that were intended to be covered under 40 C.F.R. § 270.30(h) included the types of information that formed the basis for

(continued...)

information provision of the permit, nor the duty to provide information regulation, contains any proviso that the EPA can only request information about a substance if it is a regulatory hazardous waste or hazardous constituent. *See* DuPont's Corrective Action Permit, Part One, § I.7; 40 C.F.R. § 270.30(h). Instead, the language of the permit and the regulation provides for information requests relevant to whether cause exists for modifying, revoking and reissuing, or terminating the permit, or to determine compliance with the permit. *Id.* Accordingly, I reject DuPont's attempt to interject limitations to EPA's information request authority that do not exist within the text of either DuPont's Corrective Action Permit or the information request regulation.

Nonetheless, I find that both parties have raised genuine issues of material fact. An evidentiary hearing will afford the parties the opportunity to develop the facts with regard to whether EPA's information request was reasonably relevant to determining whether cause existed for modifying, revoking and reissuing, terminating, or determining compliance with DuPont's Corrective Action Permit. For instance, OCE contends that there are numerous examples of how a request for toxicological information about PFOA (C-8) is reasonably relevant to a determination of whether cause exists to modify, revoke and reissue, or terminate DuPont's Corrective Action Permit, or to determine compliance with the Permit. OCE's Post-Argument Br. on Count III at 10. OCE contends that such examples include: understanding the potential interactions with other contaminants at the site, determining whether to use the omnibus authority to include any terms or conditions in the Permit necessary to protect human health or the environment, ascertaining whether PFOA contains hazardous constituents, developing a risk-based comparison level for PFOA, and that the EPA can request information out of concern for the safety of EPA inspectors. *Id.* at 10-11; OCE's Count III Reply at 22-23.

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[55](...continued)
the Permit. *Id.* Ms. Williams further opines that these standard information submission requirements were not intended to address the submission of health-related information about a compound that was not an Appendix VIII hazardous constituent or a RCRA hazardous waste under 40 C.F.R. § 261.3. *Id.* DuPont's arguments expressed through Ms. Williams's opinions are rejected, as they contradict the broad information request authority within the text of the Permit and the text of the regulation, and contradict the caselaw.

I note, however, elsewhere within her declaration, Ms. Williams lends support to OCE's position that the EPA may request information reasonably relevant to modification of a corrective action permit, regardless of whether the EPA is requesting information about a substance that would be subject to corrective action as a hazardous waste or hazardous constituent. Specifically, after asserting that PFOA (i.e., C-8) is not a hazardous waste or constituent, Ms. Williams admits that PFOA "[c]ould be selected as a monitoring parameter in a RCRA operating or corrective action permit . . . ," even though Ms. Williams states that PFOA would not be subject to corrective action release provisions of RCRA that require cleanup of hazardous waste. *Id.* ¶ 12.

Another factual issue not resolved at this juncture is whether DuPont unilaterally made the relevancy determination and withheld information without notifying, or without sufficiently notifying, the EPA of such withholding.[56] Furthermore, as discussed in the following sections, DuPont raises factual questions concerning whether the blood sampling results constituted "known toxicological information" at the time of the 1997 information request and whether the statute of limitations bars Count III.

Accordingly, the parties' respective motions for accelerated decision on Count III are **DENIED**. *See Roberts v. Browning*, 610 F.2d 528, 536 (8th Cir. 1979).

C. Question As to Whether the Blood Sampling Results Constituted "Known Toxicological Information"

The issue of whether the blood sampling results in the 1981 document constituted "known toxicological information" at the time of the 1997 information request is better addressed in an evidentiary hearing.

The blood sampling document at issue in Count III is an undated one-page document titled "C-8 BLOOD SAMPLING RESULTS" and contains a subheading, "Births and Pregnancies." OCE's Count III Response, Ex. 3. Most of the document is typed, but there are also some handwritten notes on the document. The blood sampling document indicates that the blood of all eight of the employees tested contain C-8, and it indicates the level of C-8 present within the blood of each employee. In a corresponding column, the document indicates whether the child born to each employee is "normal" or has birth defects, it states the birth date of each child with birth dates from 1980 to 1981, and it indicates levels of C-8 in two of the children. For instance, under the column titled "PPM C-8 in Blood," the document indicates .078 ppm of C-8 in one of the employee's blood samples, and the corresponding reference under the "Status" column states: "Normal child – born April 1981. Umbilical cord blood 0.055 ppm."

In support of OCE's motion for accelerated decision on Count III, OCE proffers the affidavit of Oscar Hernandez, Ph.D. OCE's Count III Response, Ex. 2 (Oct. 6, 2004) ("Dr. Hernandez's Affidavit"). Dr. Hernandez states that the blood sampling results "[c]onstitute toxicological information, since the data provide insights into the biological disposition of a chemical in humans." *Id.* at 1. Dr. Hernandez further states:

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[56] At this time, it is unnecessary to address OCE's argument that DuPont waived its right to challenge EPA's authority to request toxicological information about C-8 because DuPont allegedly chose not to take advantage of the dispute resolution provision in its permit. *See* OCE's Post-Argument Br. on Count III at 14-15.

Regarding OCE's contentions that DuPont is challenging the terms of its Permit, *see* OCE's Count III Response at 17-19, one may read DuPont's arguments as challenging OCE's *interpretation* of the Permit rather than being solely confined to challenging the terms of the Permit as written. At this time, it is premature to reach a determination on OCE's argument.

> Because human data are not readily available, toxicologists most frequently rely on animal data to draw conclusions and develop assumptions about the biological behavior of chemicals. The latter conclusions and assumptions become the basis for the evaluation of comparable effects in humans, an extrapolation that introduces uncertainty in the analysis. Availability of human data reduces the uncertainty associated with extrapolation of animal data.

Id. Based on the principles stated above, Dr. Hernandez opines that the language below "adequately characterizes the nature of the 1981 information":

> The 1981 data indicating that PFOA moves across the placental barrier between PFOA-exposed mothers and their fetuses suggest that such fetuses could experience toxic effects associated with PFOA, including persistence/bioaccumulation, and, as observed in animal tests, developmental toxicity and liver toxicity. The human data are more indicative of such possibility in humans than the data submitted to EPA by DuPont in 1982, which demonstrated that PFOA moved across the placental barrier in rats used in laboratory experiments. EPA's efforts to characterize effects of PFOA might have been more expeditious had the data on transplacental movement of the chemical in humans been submitted immediately by DuPont when DuPont obtained the information in 1981.

Id. at 1-2.

DuPont contends that there is a factual dispute as to OCE's contention that the 1981 blood sample results were "known toxicological information." DuPont's Count III Reply at 33-34. In particular, DuPont contends that the 1981 observation that C-8 was present in the blood supplied to a fetus was not "toxicological" information as that term is ordinarily defined. *Id.* In support of this position, DuPont proffers the declaration of Dr. Jonathan Borak, M.D., D.A.B.T., who states:

> "a reasonably knowledgeable toxicologist would have expected that PFOA crossed the human placenta" and "[t]he datum [cited by Dr. Hernandez] essentially only restates that which would have been obvious to a reasonably knowledgeable toxicologist, that PFOA can cross the placenta. In other words, it is essentially neither toxicological nor informative."

DuPont's Count III Reply, Ex. C ("Dr. Borak's First Declaration"), ¶¶ 12, 20 (Nov. 15, 2004). Furthermore, Dr. Borak opines that

> the analysis of the cord blood sample "[s]erved only as an indication that there had been exposure to PFOA, a fact that was known in advance and was the specific reason that the sample had been obtained" and "It is my professional opinion that because the cord blood datum contained no information regarding the potential hazards of exposure, the mechanisms of action, the adverse effects anticipated or known about PFOA and because it provided no information useful for evaluating the adequacy of proposed exposure standards . . . , it was neither toxicological nor informative and, therefore, does not represent 'toxicological information.'"

Id. ¶ 24.

Dr. Borak states that he assumes that Dr. Hernandez does not rely on the "First Law of Toxicology," which provides, "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy," but rather that he relies on a more restrictive definition. *Id.* ¶ 18. Accordingly, Dr. Borak focuses more narrowly on adverse effects or outcomes: "Most other definitions of 'toxicology' explicitly include the concept of *adverse* effects or outcomes." *Id.* ¶ 19 (emphasis added). Dr. Borak offers a definition of "toxicology" as follows:

> Toxicology is the scientific study of the mechanisms of action and effects of exposure caused by chemical agents in living organisms. The objectives of toxicology are to characterize the potential hazards of exposure to specific agents and to estimate the probability that such effects will follow anticipated types and levels of exposure.

Id.

Accordingly, Dr. Borak opines that the analysis of the "single blood cord datum" served only as an indication that there had been exposure to PFOA, which he states is a fact that was known in advance and was the specific reason why the sample was obtained. *Id.* ¶¶ 20, 24. Moreover, Dr. Borak contends that "toxicological information" must be informative: "[b]ecause the cord blood datum contained no information regarding the potential hazards of exposure, the mechanisms of action, the adverse effects anticipated or known about PFOA and because it provided no information useful for evaluating the adequacy of proposed exposure standards . . . it was neither toxicological nor informative and, therefore, does not represent 'toxicological information.'" *Id.* ¶ 24. DuPont has contended that at the very least the conflicting testimony of Dr. Borak and Dr. Hernandez demonstrates that there is a disputed issue of material fact about whether the blood sample observation was "toxicological information." DuPont's Count III Response at 34.

In reply, OCE quotes a definition of "toxicology" as a "science that deals with poisons and their effects" and "toxicological" as "of or relating to toxicology." OCE's Count III Reply at 19 (quoting Merriam Webster's Collegiate Dictionary (10th ed. 1997)). OCE also proffers the affidavit of David Gray, Ph.D., who opines that "[t]oxicological information is generally accepted to mean information that relates to toxic (poisonous) substances, their detection, their avoidance, their chemistry and pharmacological actions, and their antidotes and treatments." OCE's Count III Reply, Ex. 1 (Dec. 13, 2004) ("Dr. Gray's Affidavit"), at ¶ 5 (citing Tabor's Medical Dictionary (2001)). Regarding whether the blood test data contains "toxicological *information*," the EPA cites a dictionary definition of "information" as "the communication of knowledge," "facts," or "data." OCE's Count III Reply at 20.

Furthermore, OCE summarizes Dr. Gray's opinion as stating that the particular human PFOA transplacental movement information at issue, as distinguished from rat transplacental movement information, demonstrates not only that this chemical (PFOA) actually (not just theoretically) crosses the human placenta, but that it readily passes the human placenta; the concentrations at which it was detected in the human fetus through transfer from the mother, and; that it acted differently than what toxicologically *theoretically* would have expected. OCE's Count III Reply at 20 (citing Dr. Gray's Affidavit at ¶¶ 9, 10, 13, 14). OCE points out that Dr. Gray explains that it is of great importance whether or nor the rate of transfer is sufficient to result in significant concentrations within the fetus and that PFOA readily passing the placenta would not be anticipated by a toxicologist since PFOA is a large ionized molecule. Dr. Gray's Affidavit ¶¶ 9, 13.

In response to the opinions expressed by Dr. Gray, DuPont proffers a second declaration of Dr. Borak, and contends, *inter alia,* that as a matter of logic the 1981 observation was incapable of providing any information about the rate of movement [of C-8] from mother to fetus because it was a single observation at a single point in time, and that no benchmark existed when the observation was made, or at any subsequent time, against which to measure how "readily" C-8 had crossed the placenta "in that single instance in 1981." DuPont's Post-Argument Br. at 25-26 (citing DuPont's Post-Argument Br., Ex. EE, ¶¶ 7-13).

On review of the competing affidavits and declarations and the parties' arguments, I conclude that the issue of whether the blood sampling results constituted "known toxicological information" at the time of the information request warrants an evidentiary hearing. *See Roberts v. Browning*, 610 F.2d 528, 536 (8th Cir. 1979).

D. Statute of Limitations

The statute of limitations is an affirmative defense, and therefore the burden is on the respondent to prove such defense. *In re Britton Constr. Co.,* CWA Appeal Nos 97-5 & 97-8, 8 E.A.D. 261, 275 (EAB 1999). Although RCRA does not contain a statute of limitations, RCRA civil penalty actions such as the action in the instant case are subject to the general federal statute of limitations set forth at 28 U.S.C. § 2462. *In re Mayes,* RCRA (9006) Appeal No. 04-01, 2005 WL 528542, slip op. at 12-13 (EAB, Mar. 3, 2005), 12 E.A.D. ___ (citing cases); *see In re Harmon Electronics, Inc.,* RCRA (3008) Appeal No. 94-4, 7 E.A.D. 1, 16-23

(EAB 1997), *rev'd on other grounds sub nom. Harmon Indus. v. Browner,* 19 F. Supp. 2d 988 (W.D. Mo. 1988), *aff'd,* 191 F.3d 894 (8th Cir. 1999)). The general federal statute of limitations provides that an action to enforce a civil penalty must be commenced within five years from the date when the claim "first accrued." *Mayes,* slip op. at 13 (citing 28 U.S.C. § 2462).

DuPont argues that Count III is barred by the five-year statute of limitations. DuPont's Count III Reply at 28. DuPont points out that OCE's penalty claim on Count III is based on the Notice of Deficiency, dated May 5, 1997, that required DuPont to submit "known toxicological information" regarding C-8 to the EPA within 30 days of receipt of the Notice, or by June 11, 1997, whichever is later. DuPont's Count III Reply at 28.; *see* Notice of Deficiency at 2-3, 5. DuPont also points out that it provided a Response to Notice of Deficiency, dated June 6, 1997. DuPont's Count III Reply at 28. DuPont argues that if any violation occurred regarding Count III, it accrued on June 1997, and that OCE was required to file its claim prior to June 2002. *Id.* In response, OCE argues that the statute of limitations does not bar Count III due to the "three distinct doctrines" of continuing violation, fraudulent concealment, and equitable tolling. OCE's Count III Reply at 3-4. As discussed below, dismissal of Count III on statute of limitations grounds is not appropriate at this juncture.

DuPont contends, "Based on the plain language of [28 U.S.C.] section 2462, the statute of limitations for DuPont's alleged failure to comply with EPA's Notice of Deficiency began to run when the EPA's claim first accrued on June 12, 1997 at the latest." DuPont's Count III Reply at 30. In making this argument, DuPont relies on the D.C. Circuit case *3M Co. v. Browner,* 17 F.3d 1453, 1461-62 (D.C. Cir. 1994).[57] In the *3M* case, the EPA sought civil penalties under TSCA for failure to file premanufacture notices and for submitting inaccurate customs certifications. In that case, the EPA contended that the limitations period should begin when the EPA discovered the violation, not when the violation occurred. The D.C. Circuit held that in light of the meaning of the word "accrued" within the general federal statute of limitations, "[a]n action, suit or proceeding to assess or impose a civil penalty must be commenced within five years of the date giving rise to the penalty" and rejected the discovery of the violation rule. *Id.* at 1462-63. However, in *3M* the D.C. Circuit recognized fraudulent concealment as an exception to its general ban on the discovery rule. *Id.* at 1461 n.15.

One of OCE's defenses to the statute of limitations argument is that the alleged violation in Count III constitutes a continuing violation. The continuing violation doctrine does not hinge upon when the complainant discovered a violation. Rather the doctrine of continuing violations is an exception to the general rule of accrual, and the doctrine of continuing violations provides that limitations periods for violations deemed to be continuing in nature do not begin to run until

[57] I note that the law of the D.C. Circuit is not necessarily controlling for purposes of the instant case. There is more than one route for appealing this matter to the courts, although an appeal within the D.C. Circuit is one of those potential avenues.

the unlawful course of conduct is completed.[58] *Mayes,* slip op. at 16; *Harmon,* 7 E.A.D. at 21-22; *In re Lazarus, Inc.*, TSCA Appeal No. 95-2, 7 E.A.D 318, 364 (EAB 1997). If a violation is a continuing violation, the complainant must bring an action for civil penalties either during the period of violation or within five years after the violation ceased. *Lazarus,* 7 E.A.D. at 364-65 (citing, *inter alia, Harmon,* 7 E.A.D. at 22). OCE contends that the violation continued *at least* through March 6, 2001, which is the date when OCE allegedly received the information at issue in Count III from a third party. Amended Complaint ¶ 113. OCE filed its initial Complaint in this matter on July 8, 2004.

OCE submits that in determining whether requirements are continuing in nature, the adjudicator looks to the language establishing the legal obligation for words or phrases connoting continuity or descriptions of activities that are typically ongoing. OCE's Count III Reply at 5 (citing *Lazarus,* 7 E.A.D. at 366). OCE further submits that this methodology to determine the nature of a violation would include the statute and regulations, but should begin with DuPont's Corrective Action Permit "[f]or it establishes the required course of conduct in the case at bar." *Id.* (citing Section 3005(a) of RCRA, 42 U.S.C. § 6925(a)); *see also* Oral Arg. Tr. at 102. OCE contends that Section 3005(a) of RCRA, titled "Permit Requirements," establishes a continuing obligation to operate in compliance with the Permit. Oral Arg. Tr. at 102.

OCE further argues that several provisions within the DuPont's Corrective Action Permit establish a continuing violation. OCE's Count III Reply at 5-7; Oral Arg. Tr. at 100-02. Under the heading "Duty to Provide Information," Part One, Section I.7 of DuPont's Corrective Action Permit provides:

> The Permittee shall furnish, *within the specified time*, any relevant information which the [EPA] Regional Administrator . . . may

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[58] In *3M* the D.C. Circuit did not make a holding as to whether the particular violation was a continuing violation. *3M,* 17 F.3d at 1455 n.2 (dicta discussing whether there was a continuing violation). Elimination of the discovery rule does not eliminate the continuing violation doctrine. *See United States v. Reaves*, 923 F. Supp. 1530, 1534 n.1 (M.D. Fla. 1996); *accord Amer. Canoe Ass'n v. D.C. Water and Sewer Auth.,* 306 F. Supp. 2d 30, 40 (D.D.C. 2004), *dismissed upon stipulation of the parties,* No. 04-7129, 2004 WL 2091485 (D.C. Cir., Sept. 17, 2004) (per curiam mem.); *CityFed Financial Corp. v. Office of Thrift Supervision,* 919 F. Supp. 1, 6 (D.D.C. 1994), (applying the doctrine of continuing violations to the general federal statute of limitations; decided several months after *3M* was decided), *aff'd,* 58 F.3d 738 (D.C. Cir. 1995).

Furthermore, the EAB has noted that it would be "fundamentally absurd" to limit RCRA civil penalty enforcement actions o five years, despite a violator's continuing violation of the law; such a limitation would allow a violator to "[b]e free to repeat its violations of the permitting requirements of RCRA indefinitely, safely beyond the reach of the law's pecuniary sanctions." *Harmon,* 7 E.A.D. at 29-30 n.34.

> request to determine whether cause exists for modifying, revoking and reissuing, or terminating this permit, or to determine compliance with this permit.

DuPont's Corrective Action Permit, Part One, § I.7 (citing 40 C.F.R. § 270.30(h) (emphasis added).

OCE argues that although Section I.7 of the Permit, at first blush, may seem to specify action within a particular time-frame, which would arguably make it akin to a one-time violation, other "key provisions" of the Permit mandate a continuous course of conduct rather than a discrete act. OCE's Count III Reply at 5-6. For instance, OCE quotes Part One, Section I.1 of the Permit, which states, "The Permittee shall comply with all conditions of this Permit . . . ." *Id.* at 6. OCE contends that the word "comply" contemplates a continuous course of conduct rather than a discrete act, and that each day that DuPont was not in compliance with its Permit, DuPont was in violation of Section 3005(a) of RCRA, which requires a permitted treatment, storage, and disposal facility to operate in compliance with its permit. *Id.* Moreover, OCE contends that DuPont's obligation to "comply" with its Permit is fundamentally akin to the obligation construed as being continuing in *Harmon,* that the owner or operator of a facility "have" a hazardous waste permit pursuant to Section 3005(a) of RCRA. *Id.* at 6-7 (citing *Harmon,* 7 E.A.D. at 24). OCE points out the EAB's conclusion that the term "have" supported a continuing obligation giving rise to a continuing violation. *Id.* at 6 (citing *Harmon,* 7 E.A.D. at 24). Furthermore, OCE suggests that Section I.7 of the Permit carries no temporal limitation on DuPont's obligation to submit information, but rather that the request for information within 30 days was, in essence, a beginning date for the obligation to provide the information. Oral Arg. Tr. at 100-01; OCE's Count III Reply at 7-8.

OCE further contends that other Sections of DuPont's Corrective Action Permit also establish that the obligation forming the basis for Count III is continuing in nature: Part One, Section I.14 (providing, "The Permittee shall report all other instances of noncompliance not otherwise required to be reported above, at the time monitoring reports are submitted"), and; Part One, Section I.15 (providing, "Whenever the Permittee becomes aware that it failed to submit any relevant facts in the permit application or in any report to [the EPA], the Permittee shall notify [the EPA] of such failure within 7 days. The Permittee shall submit the correct or additional information to [the EPA] no later than 14 days of becoming aware of the deficiency"). OCE's Count III Reply at 7.

DuPont cites several non-RCRA cases in an effort to show that the violation alleged in Count III is not a continuing violation.[59] DuPont's Count III Response at 32. With regards to

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[59] DuPont cites: *Toussie v. United States*, 397 U.S. 112 (1970) (failure to register for the draft under the Universal Military Training and Service Act)*; United States v. Trident Seafoods Corp.,* 60 F.3d 556 (9th Cir. 1995) (Clean Air Act, failure to provide notice of asbestos removal); *United States v. Del Percio,* 870 F.2d 1090 (6th Cir. 1989) (Atomic Energy Act); *New* (continued...)

the Permit, DuPont challenges Sections I.14 and I.15 of the Permit as being inapposite to Count III. DuPont's Post-Argument Br. at 28-29. For instance, DuPont contends, with regards to Section I.14, that it was not required to submit monitoring reports to the EPA. *Id.* at 28. With regards to Section I.15, DuPont suggests that it was not aware that the 1981 blood sample results should have been reported to the EPA. *See id.* at 29. The latter suggestion appears to tie in with DuPont's arguments that the blood sampling information was not "toxicological information" or was not relevant to its corrective action permit. DuPont also contends that OCE is not alleging violations of these provisions of the Permit. *Id.* at 28-29.

With regards to the many non-RCRA cases DuPont cites, these cases are not dispositive to the extent they rely on non-RCRA statutes and regulations for their reasoning, because a determination of whether the nature of a violation is continuing first looks to the statutory language that serves as the basis for the specific violation at issue, and if necessary the legislative history, and then looks to language of the implementing regulation. *Lazarus*, 7 E.A.D. at 366-67; *see Mayes, supra*, slip op. at 16-27; *Harmon*, 7 E.A.D. at 22-40. OCE makes persuasive arguments with regards to the language used in Section 3005(a) of RCRA. I would add that RCRA's "cradle to grave" permitting requirements are intended to impose continuing obligations on owners and operators in order to protect human health and the environment. *Harmon*, 7 E.A.D. at 29. I also note that the Permit's duty to provide information requirement, at I.7, cites to 40 C.F.R. § 270.30(h), which requires information requested to be furnished "within a reasonable time."

However, I recognize that DuPont challenges the relevancy of Sections I.14 and I.15 of the Permit, and that the specific language employed in Section I.7 of the Permit refers to compliance within a "specified time," albeit when read in isolation from the statute, the regulation, the factual context of the request for information, and the remainder of the Permit. An evidentiary hearing is a more appropriate forum for resolving this matter.[60] For instance, an

---

[59](...continued)
York v. Niagara Mohawk Power Corp., 263 F. Supp. 2d 650, 660-63 (W.D.N.Y. 2003) (Clean Air Act, failure to obtain preconstruction permit); *Lazarus, Inc.*, 7 E.A.D at 379 (TSCA); *In re Rhone-Poulenc Basic Chems. Div.*, Docket No. 5-EPCRA-97-053, 1998 WL 289239 (ALJ, Apr. 27, 1998) (Emergency Planning and Community Right-to-Know Act); *In re Frontier Stone, Inc.*, CAA Docket No. II-95-0105, 1997 EPA ALJ LEXIS 131 (ALJ, Mar. 10, 1997) (Clean Air Act, failure to conduct performance test within period proscribed).

[60] DuPont submits that, in determining whether a violation is continuous in nature, the language of a requirement at issue must clearly state that there is a continuous duty, and it cites a Ninth Circuit case as authority. OCE's Count III Response at 31 (citing *Trident Seafoods Corp.*, 60 F.3d 556, 559 (9th Cir. 1995). Cases from the Ninth Circuit are not binding on the instant matter, which arises within West Virginia. *See In re Bil-Dry Corp.*, RCRA (3008) Appeal No. 98-4, 9 E.A.D. 575, 590 (EAB 2001). Moreover, the specific holding in *Trident* was on whether a failure to provide notice under the Clean Air Act subjected the defendant to a per-day penalty (continued...)

evidentiary hearing will allow for a closer examination of the context in which the EPA made the request for information. At this time, I need not consider OCE's two other defenses to the statute of limitations argument: fraudulent concealment and equitable tolling. However, I observe that the allegations of fraudulent concealment clearly raise a genuine issue of material fact requiring an evidentiary hearing. *See, e.g.*, Oral Arg. at 50-52 (DuPont admitting that it responded to part of EPA's 1997 request for "known toxicological information" regarding C-8, but contending that it did not furnish the 1981 blood sampling results because it was responding "voluntarily").

III. Conclusion and Prehearing Schedule

For the reasons stated herein, I deny the parties' motions for accelerated decision. However, I emphasize that an order denying accelerated decision, such as the instant order, does not decide the ultimate truth of the matter, but represents a threshold determination that an evidentiary hearing is necessary.

The parties shall conduct prehearing exchanges, as delineated in my Prehearing Order (Sept. 16, 2004) and my Order Clarifying Prehearing Order (Oct. 21, 2004), on Counts II and III and on the recently added Count IV (titled "Results of PFOA Serum Testing"),[61] which shall be filed *in seriatim* manner, according to the following schedule:

July 1, 2005	–	Complainant's Initial Prehearing Exchange
August 1, 2005	–	Respondent's Prehearing Exchange, including any direct and/or rebuttal evidence
August 15, 2005	–	Complainant's Rebuttal Prehearing Exchange (if necessary)

[60](...continued)
under the Clean Air Act. *Trident*, 60 F.3d at 559.

[61] OCE has already filed its initial prehearing exchange on Count I. As specified in a previous order, DuPont's prehearing exchange on Count I is due April 8, 2005, and OCE's rebuttal prehearing exchange on Count I is due April 22, 2005.

I remind the parties that if they cannot settle this matter, an evidentiary hearing will be held in accordance with Section 556 of the Administrative Procedure Act, 5 U.S.C. § 556.

Finally, I instruct the parties that all future pleadings, including exhibits, shall be submitted in binders. Furthermore, I instruct the parties that all future briefs, memoranda, and motions greater than 15 pages in length (excluding attachments) shall contain a table of contents and a table of authorities with page references.[62]

So ordered.

Dated: March 29, 2005
Washington, D.C.

Barbara A. Gunning
Administrative Law Judge

[62] *See* 40 C.F.R. § 22.4(c)(10).

OECD GUIDELINE FOR THE TESTING OF CHEMICALS

Simulation Test – Aerobic Sewage Treatment

303 A: Activated Sludge Units

OECD Guideline 303 is subject to copyright and is not included in this Attachment C to Appendix A to the Consent Agreement and Final Order, In the Matter of: E. I. du Pont de Nemours and Company, Docket Nos. TSCA-HQ-2004-0016, RCRA-HQ-2004-0016, TSCA-HQ-2005-5001.

To purchase a copy of OECD Guideline 303, visit: www.oecdbookshop.org (ISBN # 9264070427).

To view a read-only copy of OECD Guideline 303, visit the EPA reading room located in EPA's Docket Center, Rm. B102–Reading Room, EPA West Building, 1301 Constitution Ave., NW, Washington, DC. Request to view OPPT-2003-0012-0169.

The EPA Docket Center is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The EPA Docket Center Reading Room telephone number is (202) 566–1744 and the telephone number for the OPPT Docket, which is located in the EPA Docket Center, is (202) 566–0280.

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September 5, 2001

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Document Processing Center (7407)
Office of Toxic Substances
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460
Attn: TSCA Section 8(e) Coordinator

Dear Section 8(e) Docket Coordinator:

Re: TSCA 8(e) Supplemental Notice on Sulfonate-based Fluorochemicals

With this letter, 3M is providing final reports and other supplemental information related to previous TSCA Section 8(e) notifications. Many of the enclosed items are analytical reports providing blood serum and liver levels of test materials for which the in-life report referring to administered doses has already been submitted to the 8(e) docket. In other cases where the 8(e) notification consisted of preliminary data, we are submitting a final study report.

All of the enclosed items are already in EPA's possession and available in TSCA Docket AR-226. We believe, however, that placing these items in the 8(e) docket may allow for more convenient access to information directly related to previous 8(e) notifications by 3M.

The table below lists the enclosed items and references the study or data which already has been the subject of an 8(e) notification by 3M:

Attached Submission	Related Study/Data Already Filed Under 8(e)
1. Amended Analytical Study, 2(N-Ethylperfluorooctane sulfonamido)-ethanol in Two Generation Rat Reproduction, Determination of the Presence and Concentration of PFOS, M556, PFOSAA, and PFOSA in the Liver and PFOS, M556, PFOSAA, PFOSA and EtFOSE-OH in the Sera of Crl:CDBR VAF/Plus Rats Exposed to EtFOSE-OH, 3M Reference No. T-6316.5, Analytical Report TOX-013, LRN-U2095, June 11, 2001.	Combined Oral (Gavage) Fertility, Developmental and Perinatal/Postnatal Reproduction Toxicity Study of N-EtFOSE in Rats, 3M Reference No. T-6316.5, June 30, 1999, full report submitted February 15, 2000 to supplement earlier filing

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Exhibit
2789
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

MET 0438638

2789.0001

Attached Submission	Related Study/Data Already Filed Under 8(e)
2. Analytical Laboratory Report, Determination of the Presence and Concentration of Potassium Perfluorooctanesulfonate (CAS Number: 2759-39-3) in the Serum and Liver of Sprague-Dawley® Rats Exposed to PFOS via Gavage, Laboratory Report No. U2006, Requestor Project No. 3M TOX 6295.9, October 27, 1999. 3. Report Amendment 1, Combined Oral (Gavage) Fertility, Developmental and Perinatal/Postnatal Reproduction Toxicity Study of PFOS in Rats, Argus Research Laboratories, Inc., Protocol 418-008, Sponsor's Study No. 6295.9, April 13, 2000.	Combined Oral (Gavage) Fertility, Developmental and Perinatal/Postnatal Reproduction Toxicity Study of PFOS in Rats, Argus Research Laboratories, Inc., Sponsor's Study No. 6295.9, June 10, 1999, full report submitted February 15, 2000 supplementing earlier filing
4. Analytical Report, Determination of the Presence and Concentration of Perfluorooctanesulfonate, Perfluorooctanesulfonylamide, M556, and M570 in the Liver and Sera Samples, 3M Environmental Laboratory Ref. No. U2636, TOX-028, February 23, 2001	13-Week Dietary Study of N-Methyl Perfluorooctanesulfonamido Ethanol (N-MeFOSE) in Rats, 3M Ref. No. T-6314.1, Covance Study No. 6329-225, dated June 30, 2000, Section 8(e) filing July 24, 2000
5. Analytical Laboratory Report, Determination of the Concentration of PFOS, PFOSA, PFOSAA, and EtFOSE-OH in the Sera and Liver of Crl:CDBR VAF/Plus Rats Exposed to N-EtFOSE, 3M Environmental Laboratory Report No. TOX-098, Laboratory Request No. U2402, 3M Ref. No. T-6316.7, February 6, 2001.	Final Report, Oral (Gavage) Developmental Toxicity Study of 2(N-Ethylperfluorooctanesulfonamido)-ethanol in Rats, 3M Reference No. T-6316.7, December 17, 1998, submitted to Section 8(e) docket per letter of August 21, 2000
6. Analytical Laboratory Report on the Determination of the Presence and Concentration of Potassium Perfluorooctanesulfonate (PFOS) or another metabolite of 2(N-ethylperfluorooctanesulfonamido)-ethanol (N-EtFOSE) in Liver and Serum Specimens, 3M Environmental Laboratory Report No. TOX-097, Laboratory Request No. U2452, 3M Ref. No. T-6316.8, February 8, 2001	Final Report, Oral (Stomach Tube) Developmental Toxicity Study of N-EtFOSE in Rabbits, 3M Reference No. T-6316.8, January 11, 1999, submitted to Section 8(e) docket per letter of August 21, 2000
7. Final Report, Alexander, B., Mortality Studies of Workers Employed at the 3M Decatur Facility, University of Minnesota, April 26, 2001.	Preliminary data submitted to Section 8(e) docket in letter of December 15, 2000

Attached Submission	Related Study/Data Already Filed Under 8(e)
8. Final Report, Acute Oral Toxicity Screen with T-3290CoC in Albino Rats, Safety Evaluation Laboratory, Riker Laboratories, Inc., Project No. 0882AR0362, 3M Reference No. T-3290 (40 % K^+PFOSAA in 3 % EtOH, 17 % IPA and 40 % H_20, L-6778, F-6873, Lot 501), November 5, 1982 *[Bibliography entry in Docket AR-226, final report was to be moved to TSCA 8(e) docket]*	Acute Oral Toxicity Screen with T-3290CoC in Albino Rats, Safety Evaluation Laboratory, Riker Laboratories, Inc., Project No. 0882AR0362, 3M Reference No. T-3290 (40 % K^+PFOSAA in 3 % EtOH, 17 % IPA and 40 % H_20, L-6778, F-6873, Lot 501), November 5, 1982, submitted to Section 8(e) docket in August 21, 2000 self-audit letter (which erroneously refers to rabbits rather than rats)
9. Giesy, J.P., and K. Kannan, Accumulation of Perfluorooctanesulfonate and Related Fluorochemicals in Fish Tissue, Michigan State University, June 20, 2001. 10. Giesy, J.P., and K. Kannan, Accumulation of Perfluorooctanesulfonate and Related Fluorochemicals in Mink and River Otters, Michigan State University, June 20, 2001. 11. Giesy, J.P., and K. Kannan, Perfluorooctanesulfonate and Related Fluorochemicals in Oyster, Crassostrea Virginica, From the Gulf of Mexico and Chesapeake Bay, Michigan State University, June 20, 2001. 12. Giesy, J.P. and K. Kannan, Perfluorooctanesulfonate and Related Fluorochemicals in Fish-Eating Water Birds, Michigan State University, June 20, 2001. 13. Giesy, J.P. and K. Kannan, Accumulation of Perfluorooctanesulfonate and Related Fluorochemicals in Marine Mammals, Michigan State University, June 20, 2001.	Preliminary data submitted to Section 8(e) docket May 26, 1999

If you have any questions about this submission, please contact me at (651)737-4795.

Sincerely,

Georjean Adams

Georjean Adams
Manager, 3M Corporate Product Responsibility

Enclosures

L-6778, F-6873

MR 51674

Acute Oral Toxicity Screen with T-3290CoC in Albino Rats

Experiment No.: 0882AR0362

Conducted At: Safety Evaluation Laboratory
Riker Laboratories, Inc.
St. Paul, Minnesota

Dates Conducted: July 30, 1982 to August 27, 1982

Conducted By: H. S. Rhodes 10/28/82
H. S. Rhodes, Jr. Laboratory Technician, Acute Toxicology — Date

8EHQ-80-373

D. M. Markoe, Jr. 10/28/82
D. M. Markoe, Jr., BS, Toxicologist, Study Director — Date

Reviewed By: Karen D O'Malley 11/5/82
K. D. O'Malley, BS, Senior Toxicologist, Acute Toxicology — Date

dc: M. T. Case
K. L. Ebbens
F. D. Griffith
W. C. McCormick

Confi[illegible]

Summary

An acute oral toxicity screen with T-3290CoC was conducted from July 30, 1982 to August 27, 1982 at Riker Laboratories, Inc., St. Paul, Minnesota using male and female albino rats ranging in body weight from 209-269 grams. The test article was administered by gastric intubation at a dose level equivalent to 200 mg/kg and 1250 mg/kg body weight with 0/10 mortalities noted at the 200 mg/kg dose level and 9/10 mortalities noted at the 1250 mg/kg dose level. The untoward behavioral reactions which occurred during the 28 day observation period at the 200 mg/kg dose level consisted of hypoactivity from 60-120 minutes post dose administration. The 1250 mg/kg dose level exhibited hypoactivity, ataxia, diarrhea and, in the females, unkempt appearance. Recovery was generally precluded by death. Body weight gains were noted in all animals which survived the study. Necropsies performed at termination of the study revealed no visible lesions, however, autolysis precluded evaluation of three 1250 mg/kg dose level animals which died prior to the end of the study. The approximate oral LD50 of T-3290CoC is greater than 200 mg/kg and less than 1250 mg/kg in fasted male and female albino rats.

Introduction

The objective of this study was to approximate the acute oral LD50 of T-3290CoC in fasted albino rats. This study was conducted for research and development purposes and is, therefore, not regulated by the Food and Drug Administration's Good Laboratory Practice Regulation of 1978, although the standard operating procedures of this laboratory adhere to the general principles of this regulation. The raw data generated by the Study Director and the final report are stored in the conducting laboratory's archives.

Method and Results

Young albino rats[a] were used in this test. All animals were held under quarantine for several days prior to testing with only animals which appeared to be in good health and suitable as test animals at the initiation of the study used. The rats were housed in suspended, wire-mesh cages in temperature and humidity controlled rooms and permitted a standard laboratory diet[b] plus water ad libitum except during the 16-20 hour period immediately prior to gastric intubation when food was withheld.

The rats were administered the test material[c] at 200 mg/kg and 1250 mg/kg body weight. All doses were administered undiluted directly into the stomachs of the rats using a hypodermic syringe equipped with a ball-tipped intubating needle[d].

After gastric administration of the test article, the rats were returned to their cages and observed for the following 28 days. Initial, 14 day and final body weights, mortalities (Table 1) and adverse reactions (Table 1) were recorded. A necropsy was conducted on all animals that died during the study as well as those euthanatized and the end of the 28 day observation period (Table 1). The protocol, principal personnel involved in the study, composition characteristics and Quality Assurance statement are contained in Appendices I - IV.

[a] King Labs, Oregon, WI
[b] Ralston Purina Laboratory Chow, Ralston Purina, St. Louis, MO
[c] The test article is 40% solids and was dosed at 200 mg/kg and 1250 mg/kg of solids which is equivalent to an undiluted dose of 500 mg/kg and 3125 mg/kg "as is", respectively.
[d] Popper and Sons, New Hyde Park, NY

TABLE 1

ACUTE ORAL TOXICITY SCREEN - ALBINO RATS

with T-3290CoC

Mortality, Necropsy, Adverse Reactions and Body Weight Data

Dose [a] (mg/kg)	Sex	Animal Number	Individual Body Weights (g) Test Day Number: 0	14	28	Number Dead / Number Tested	Percent Dead
200	M	2R3587	253	293	328	0/5	0
		2R3588	251	306	351		
		2R3589	268	328	369		
		2R3590	263	309	346		
		2R3591	262	310	369		
200	F	2R3607	225	263	269	0/5	0
		2R3608	216	263	260		
		2R3609	219	236	252		
		2R3610	222	241	257		
		2R3611	219	235	249		
1250	M	2R3592	265	(Day 7)		4/5	80
		2R3593	260	(Day 6)			
		2R3594	263	(Day 5)			
		2R3595	269	303	367		
		2R3596	262	(Day 6)			
1250	F	2R3612	217	(Day 5)		5/5	100
		2R3613	222	(Day 4)			
		2R3614	216	(Day 5)			
		2R3615	214	(Day 5)			
		2R3616	209	(Day 3)			

[a] The test article was administered undiluted, however, the dose represents mgs of solids, of which the test article consists of 40% solids.

The acute oral LD50 is greater than 200 mg/kg and less than 1250 mg/kg in fasted male and female rats.

Note: Figures in parenthesis indicate time of death.

Necropsy

Necropsies performed upon termination revealed no visible lesions at the 200 mg/kg dose level. The 1250 mg/kg dose level produced no visible lesions upon necropsy, however, autolysis precluded evaluation of three animals.

TABLE 2

ACUTE ORAL TOXICITY SCREEN - ALBINO RATS

with T-3290CoC

Summary of Reactions

Dose mg/kg	Sex	Reactions	Observation Periods: Number Affected/Number Dosed — Minutes: 1-30	60	120	Days: 1	2	3	4	5	6	7	8	9	10	11	12	13	14*
200	M	Hypoactivity		5/5	5/5	0/5													
200	F	Hypoactivity		5/5	5/5	0/5													
1250	M	Hypoactivity		5/5	0/5	5/5	5/5	5/5	5/5	4/4	3/3	1/1	1/1	1/1	1/1	0/1			
		Ataxia			5/5	0/5					1/3	0/1							
		Diarrhea				4/5	2/5	0/5											
1250	F	Hypoactivity		5/5	0/5	5/5	5/5	4/4	3/3	0/0									
		Ataxia			5/5	0/5													
		Diarrhea			1/5	5/5	2/5	4/4	2/3	0/0									
		Unkempt						4/4	3/3	0/0									

*Observations continued to day 28, however, no significant reactions were noted beyond day 11.

A blank space indicates no significant reactions.

Riker Experiment No.: 0862AR0E362

APPENDIX I

PROTOCOL

TEST: Acute Oral Toxicity

SPONSOR: 3M Commercial Chemicals Division

CONDUCTED BY: **Safety Evaluation Laboratory, Riker Laboratories, Inc., St. Paul, Minnesota**

TEST ARTICLE: T-3290CoC

CONTROL ARTICLE: NONE

PROPOSED STARTING/COMPLETION DATE OF TEST: 7/82 - 10/82

TEST SYSTEM: ALBINO RAT, SD STRAIN

SOURCE: KING LABS, OREGON, WI

Sex: M, F
Number: 5/5
Weight Range: 200-300 grams

OBJECTIVE: The objective of this test will be to characterize the acute oral toxicity of the test article in albino rats. Rats were selected as a test system for reproducibility of response, historical use, ease in handling and general availability.

METHOD: The animals will be housed in stainless steel suspended wire mesh cages in temperature and humidity controlled rooms during both the quarantine and test periods, with food[a] and water offered *ad libitum*[b]. Each animal will be identified by color coding, according to the laboratory's standard operating procedure, which will correspond to the animal numbers on a card affixed to the outside of the cage. A single dosage of _____* mg/kg will be administered each animal, however, if this dosage level does not adequately characterize the toxicity of the test article, additional animals will be administered the test article at supplemental dosage levels. Any additional dosage levels will be documented and filed with this protocol. The test article will be administered to the animals in the form received from the sponsor, after which the animals will be returned to their cages and observed for any untoward behavioral reactions for the following 14 days. Initial and final body weights will be recorded. A gross necropsy which will include, but not be limited to; heart, lungs, liver, kidneys and general gastrointestinal tract will be conducted on all animals which die during the conduct of the test as well as the animals surviving the test period. Any gross abnormalities which are observed during the conduct of the necropsy will be recorded with specific mention to the organ and/or site observed. All raw data generated by the study director and the final report will be stored in the Riker Laboratories' Archive, St. Paul, Minnesota.

[a] Purina Laboratory Chow, Ralston Purina, St. Louis, Missouri
[b] Purina Chow FOOD WILL BE WITHHELD FOR A [illegible] PERIOD [illegible] TO DOSING
*dose at two levels: 1250 mg/kg & 200 mg/kg (as solids) solution is 50 soli

William T. Lynch 7.29.82
Sponsor Date

D. L. [illegible] 7/30/82
Study Director Date

APPENDIX I (concluded)
Deviations and/or Amendments to Protocol

1. The test article was supplied as a solution with 40% solids not 50%.

D. M. MacRae 7/30/82
Study Director Date

2. _____

Study Director Date

3. _____

Study Director Date

4. _____

Study Director Date

5. _____

Study Director Date

APPENDIX II

Principal Participating Personnel Involved in the Study

Name	Function
H. S. Rhodes	Jr. Laboratory Technician Acute Toxicology
D. M. Markoe, Jr., BS	Toxicologist Study Director
K. L. Ebbens, BS	Supervisor Toxicology Testing
K. D. O'Malley, BS	Senior Toxicologist Acute Toxicology
G. C. Pecore	Supervisor Animal Laboratory

APPENDIX III

Composition Characteristics

This study is not regulated by the Good Laboratory Practice Act of 1978 and therefore information pertaining to composition characteristics is not applicable for inclusion in this study.

APPENDIX IV

Quality Assurance Statement

This study is not officially regulated by the Good Laboratory Practice Regulation of 1978, and therefore a statement signed and prepared by the Compliance Audit department is not applicable.

The standard operating procedures of this laboratory does adhere to the general principles of this regulation. The Compliance Audit department does inspect different significant phases for studies underway in the Acute Toxicology Laboratory on a recurring cycle, and the facilities are examined on a three month schedule. In addition a select number of Research & Development studies are routinely picked at random from the Archives by the Compliance Audit department for review.

From: Douglas, David
Sent: Tuesday, August 05, 2003 2:30 PM
To: Brott, Bruce
Cc: Rys, Mark; Williams, Alan ; White, Dann
Subject: EPA Risk Assessment for PFOS/PFOA

Bruce, following up on the DOD article that you showed me this morning, I spoke to Jim Kelly this morning and Jim told me that Helen is following the EPA risk assessment development for the Scotchguard chemicals. He said that she participates in EPA risk assessment development conference call updates. Additionally MDH is developing HRLs for PFOA and PFOS. Jim said that he is moving forward with re-attempting to set up a meeting between 3M and MDH regarding what 3M knows about the extend of ground water contamination on the Cottage Grove facility. Dave

David N. Douglas
Minnesota Pollution Control Agency
Superfund Unit 2/Superfund Section
Majors and Remediation Division
Office: (651) 296-7818
Fax: (651) 296-9707
Email: david.douglas@pca.state.mn.us

Reference News Release: EPA Settles PFOA Case Against DuPont for Largest Environmental Administrative Penalty in Agency History

Release Date: 12/14/2005
Contact Information:

Contact: Dave Ryan, 202-564-4355 / ryan.dave@epa.gov

(Washington, D.C.-Dec. 14, 2005) DuPont will pay $10.25 million -- the largest civil administrative penalty EPA has ever obtained under any federal environmental statute -- to settle violations alleged by EPA over the company's failure to comply with federal law. Under the settlement, filed with the Agency's Environmental Appeals Board, Dupont is also committing to $6.25 million for Supplemental Environmental Projects (SEPs).

The settlement, which still must be approved by the EAB, would resolve DuPont's violations related to the synthetic chemical Perfluorooctanoic Acid (PFOA) under provisions of both the Toxic Substances Control Act (TSCA) and the Resource Conservation and Recovery Act (RCRA).

The settlement resolves the four violations alleged in the Agency's two complaints filed against DuPont in July and December 2004, and settles four additional counts involving information about PFOA that EPA obtained after initiating its action against DuPont. Seven of the eight counts involve violations of TSCA Section 8(e) -- the requirement that companies report to EPA substantial risk information about chemicals they manufacture, process or distribute in commerce.

"This is the largest civil administrative penalty EPA has ever obtained under any environmental statute. Not by a little, by a lot," said Granta Y. Nakayama, assistant administrator for the Office of Enforcement and Compliance Assurance. "EPA takes violations of toxic substances laws seriously and is committed to enforcing those laws. This settlement sends a strong message that companies are responsible for promptly informing EPA about risk information associated with their chemicals."

PFOA (also known as C8 or Ammonium Perfluorooctanoate [APFO]), is used in the manufacturing process of fluoropolymers, including some Teflon® products, at DuPont's Washington Works facility in Washington, W.Va. Fluoropolymers impart desirable properties, including fire resistance and oil, stain, grease, and water repellency. They are used to provide non-stick surfaces on cookware and waterproof, breathable membranes for clothing.

As part of this settlement, DuPont has voluntarily agreed to undertake two Supplemental Environmental Projects (SEPs) valued at $6.25 million. A SEP is an environmentally beneficial project that the violator agrees to undertake in exchange for mitigation of the penalty to be paid. SEPs are related to the environmental violation and further EPA's goal of protecting and enhancing public health and the environment.

The first SEP, valued at $5 million and to be completed in three years, is a project designed to investigate the potential of nine of DuPont's fluorotelomer-based products to breakdown to form PFOA. This SEP will help industry, scientists, the public and EPA examine the potential sources of PFOA in the environment and potential routes of human exposure to PFOA. The public will have an opportunity to nominate members to a Peer Consultation Panel, an independent group of scientists that will address specific charges identified in the SEP. DuPont has agreed to require the laboratories that it contracts with to perform work under the SEP to follow the agency's Good Laboratory Practices standards as well as prepare and follow a Quality Assurance Project Plan.

For the second SEP, DuPont will spend $1.25 million to implement over an expected three year period, the Microscale and Green Chemistry Project at schools in Wood County, West Virginia. This SEP will foster science laboratory curriculum changes to reduce risks posed by chemicals in schools. Using microscale chemistry, which reduces exposure to chemicals, and green chemistry, an approach that uses safer chemicals, the project will reduce risks to children's health and enhance science safety in all of the participating schools.

"We are pleased that as a direct result of this settlement with DuPont, valuable information will be produced for the scientific community to better understand the presence of PFOA in the environment and any potential risks it poses to the public," said Susan Hazen, EPA's principal deputy assistant administrator for the Office of Prevention, Pesticides, and Toxic Substances. "We are hopeful that today's action will serve as an important reminder of the importance of timely

industry reporting of substantial risk information to EPA."

The violations resolved in this settlement consist of multiple failures to report information to EPA about substantial risk of injury to human health or the environment that DuPont obtained about PFOA from as early as 1981 and as recently as 2004. The seven TSCA Section 8(e) counts fall within three types of categories: human health information, environmental contamination, and animal toxicity studies. More information on the violations is available at: http://yosemite.epa.gov/opa/admpress.nsf/blab9f485b098972852562e7004dc686/826fe743d67d744685256f620074c136!OpenDocument

The Consent Agreement and SEPs can be viewed at: https://www.epa.gov/enforcement/ei-dupont-de-nemours-and-company-pfoa-settlements

The full record of EPA's case against DuPont is available to the public through EPA's Headquarters Hearing Clerk who is located in EPA's Office of Administrative Law Judges at 1099 14th St. N.W., Washington, D.C. and can be reached at 202-564-6263. Copies of the settlement are available to the public through the Board's Clerk who is located in the Colorado building, 1341 G St. N.W., Suite 600, Washington, D.C. 20005 and can be reached at 202-233-0122.

Contact Us to ask a question, provide feedback, or report a problem.

https://www.epa.gov/enforcement/reference-news-release-epa-settles-pfoa-case-against-dupont-largest-environmental

Accessed February 7, 2020

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

JAN 25 2006

THE ADMINISTRATOR

Mr. Charles O. Holliday, Jr.
Chairman and Chief Executive Officer
Dupont
1007 Market Street
Wilmington, DE 19898

Dear Mr. Holliday:

As you are aware, Dupont and other proactive companies have been working collaboratively with the Environmental Protection Agency (EPA) to better understand the sources and pathways of exposure to perfluorooctanoic acid (PFOA) and related chemicals. Considerable progress has been made by putting in place a comprehensive testing and research program that will fill in many of the critical information gaps that exist around our understanding of potential exposures and risks. We all recognize that PFOA is persistent in the environment, that it has been detected in human blood, and that animal studies indicate effects of concern. The data from the research and testing programs will allow the Agency and others to make informed decisions about any potential risk management actions that are warranted.

In the meantime, absent the certainty that these data will provide, I am asking you to join with EPA and other stakeholders to commit to a global stewardship program whose goal is to work toward essentially eliminating emissions and product content levels of PFOA and related chemicals.

Participation in the stewardship program requires voluntary corporate commitment to two goals:

1) To commit to achieve, no later than 2010, a 95% reduction, measured from a year 2000 baseline, in *both:*
   - facility emissions to all media of PFOA, precursor chemicals that can break down to PFOA, and related higher homologue chemicals, *and*
   - product content levels of PFOA, precursor chemicals that can break down to PFOA, and related higher homologue chemicals.

2) To commit to working toward the elimination of PFOA, PFOA precursors, and related higher homologue chemicals from emissions and products by five years thereafter, or no later than 2015.

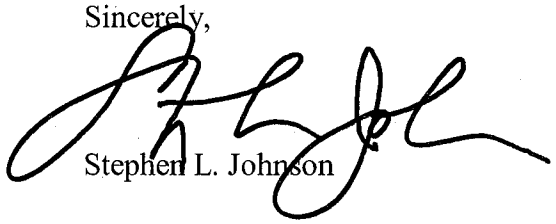
While these program goals are ambitious, some participating companies may attain or even surpass some aspects of the goals before achieving others, some companies may have achieved portions of these goals already, and some may wish to commit to a more aggressive timeline. I encourage participating companies to identify specific individual commitments that go beyond the overall program goals, such as achieving the emissions and/or product content reductions before the 2010/15 goal years. The Agency also recognizes that technological and cost issues may preclude eliminating PFOA and related chemicals entirely from emissions and products by 2015. Annual reporting should help to identify and focus attention on these areas to encourage progress toward that ultimate goal.

Many activities are underway concerning PFOA and related chemicals, including additional research by companies, government agencies, and universities. Participation in the stewardship program will be in addition to a company's existing commitments to the Agency which may include research efforts, enforceable consent agreements, and memoranda of understanding. These ongoing efforts will combine with the 2010/15 PFOA Stewardship Program to further our understanding of this family of persistent, bioaccumulative, and toxic chemicals, and to achieve true long-term environmental and public health benefits. Although our risk assessment activities are not yet complete and new data may change the current picture, to date EPA is not aware of any studies specifically relating current levels of PFOA exposure to human health effects. This may offer us a window of opportunity now to ensure, through the 2010/15 PFOA Stewardship Program and other related efforts, that potential concern levels are never reached.

I hope that you will accept this invitation to step forward into environmental leadership. Please respond by letter with your commitment to the 2010/15 PFOA Stewardship Program by March 1, 2006. Additional information on the details of program commitments is enclosed with this letter. If you have questions concerning this program and your participation in it, please contact Mary Dominiak in the U.S. EPA Office of Pollution Prevention and Toxics, Chemical Control Division, by telephone at 202-564-8104, or by email at dominiak.mary@epa.gov.

I look forward to working with you and achieving these goals.

Sincerely,



Stephen L. Johnson

Enclosure

cc: S. Hazen
C. Auer

2010/15 PFOA Stewardship Program Commitments

Corporate commitment letters should include commitments to both goals of the Stewardship Program:

1) To commit to achieve, no later than 2010, a 95% reduction, measured from a year 2000 baseline, in *both:*
   - facility emissions to all media of PFOA, precursor chemicals that can break down to PFOA, and related higher homologue chemicals, *and*
   - product content levels of PFOA, precursor chemicals that can break down to PFOA, and related higher homologue chemicals.

2) To commit to working toward the elimination of PFOA, PFOA precursors, and related higher homologue chemicals from emissions and products by five years thereafter, or no later than 2015.

Companies participating in this 2010/15 PFOA Stewardship Program will be asked to submit their year 2000 baseline numbers for emissions and product content to EPA by October 31, 2006. To ensure transparency, companies will submit annual public reports on their progress toward the goals in October of each successive year, expressing their progress in terms of company-wide percentage achievements both for U.S. operations and for the company's global business. Companies will also provide to EPA detailed information on their progress in support of their public reports. By participating in the Program, companies grant permission to EPA to share information submitted under the Program with its contractors, including information contained within detailed progress reports that may be claimed as confidential.

These chemicals present considerable scientific challenges in ensuring accurate and reproducible results in chemical analyses. To ensure that the results reported under the 2010/15 PFOA Stewardship Program are both comparable and reliable, each participating company will also commit to work with EPA, other PFOA Stewardship Program participants, and others in order to establish scientifically credible analytical standards and laboratory methods for measuring the chemicals in the program by 2010, the first goal attainment year. Participants will also make a general commitment to continue research to better understand the sources, pathways of exposure, and potential risks of these chemicals.

Corporate commitment letters should be submitted by March 1, 2006, and should be addressed to:

Stephen L. Johnson, Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W. (1101A)
Washington, DC 20460

With a courtesy copy to:

Charles M. Auer, Director
U.S. EPA Office of Pollution Prevention and Toxics
1200 Pennsylvania Avenue, N.W. (7401M)
Washington, DC 20460

Susan M Stalnecker/AE/DuPont
02/16/2006 05:21 PM

To CN=David W Boothe/OU=AE/O=DuPont@DuPont
cc
bcc
Subject Fw: URGENT:Script

History: This message has been replied to.

----- Forwarded by Susan M Stalnecker/AE/DuPont on 02/16/2006 05:21 PM -----

Susan M Stalnecker/AE/DuPont
02/16/2006 04:58 PM

To michaelmccabe1@earthlink.net
cc Gary W Spitzer/AE/DuPont@DuPont, Cynthia C Green/AE/DuPont@DuPont, Carolmarie C Brown/Contractor/AE/DuPont@DuPont, Kathleen H Forte/AE/DuPont@DuPont, R Clifton Webb/AE/DuPont@DuPont, Daniel A Turner/AE/DuPont@DuPont
Subject URGENT:Script

Mike and the cc's, please review the attached. I plan to send to Chad and ask him to call Steve Johnson.

ask.02.16.06.doc

[redacted]

006-0133-0088917

Situation analysis: Publicity around SAB report has linked the Teflon brand to cancer. Coverage has been broad in print and network media. Significant disruptions in our markets and are consumers are very, very concerned.

The "Ask": In our opinion, the only voice that can cut through the negative stories, is the voice of EPA. We need to EPA to quickly (like first thing tomorrow) say the following:

1. Consumer products sold under the Teflon® brand are safe. These include the non-stick cookware in your kitchen, the stain resistant carpet in your family room, and the waterproof jackets in your closes, among other products which are valued by consumers and offer unique and important benefits

2. Further, to date, there are no human health effects known to be caused by PFOA

Another clarifying aspect is where the EPA is with respect to the SAB report. The SAB report is a recommendation to EPA. The EPA will consider the report, along with the new information and studies not considered by the SAB. The current classification of "suggestive" is the standing assessment, until the EPA completes it risk assessment process.

[redacted]

100 percent Participation and Commitment in EPA's PFOA Stewardship Program

Release Date: 03/02/2006

Contact Information: Enesta Jones, (202) 564-4355 / jones.enesta@epa.gov

(Washington, D.C. – March 2, 2006) EPA has received 100 percent participation and commitment in its Global Stewardship Program that will dramatically reduce perfluorooctanoic acid (PFOA) in the environment. Under EPA's leadership, eight companies are voluntarily agreeing to take action now to reduce PFOA releases and product content levels.

"Today we have 100 percent participation and 100 percent commitment. And it's 100 percent the right thing to do," said Susan B. Hazen, EPA's acting assistant administrator for Prevention, Pesticides, and Toxic Substances. "We applaud these companies for their commitment to reducing the amount of PFOA getting into our environment."

The eight companies are: Arkema, Asahi, Ciba, Clariant, Daikin, DuPont, 3M/Dyneon, and Solvay Solexis. They are agreeing to reduce PFOA releases and levels in products by 95 percent by no later than 2010, and to work toward elimination of these sources of PFOA exposure five years after that, but no later than 2015. Companies are being asked to meet these commitments in the United States as well as in their global operations.

PFOA is a processing aid used in the manufacture of other non-stick and stain-resistant surfaces and products. PFOA may also be produced by the breakdown of fluorotelomers, which are used to impart water, stain, and grease resistance to carpets, paper and textile. PFOA is also persistent in the environment. It has been detected in low levels in wildlife and humans, and animal studies conducted have indicated effects of concern.

The use of PFOA in the manufacturing process does not mean that people using these products would be exposed to PFOA. The agency does not believe that consumers need to stop using their cookware, clothing, or other stick-resistant, stain-resistant products.

Specifically, the participating companies have committed to reduce by 95 percent facility emissions and product content levels of PFOA, PFOA precursors, and higher homologue chemicals, by no later than 2010. The year 2000 will serve as the baseline for measuring reductions.

The companies have been asked to submit their year 2000 baseline numbers for emissions and product content to EPA by Oct. 31, 2006. Annual public reports on their progress toward the goals will be due in October of each successive year. To ensure comparable reporting of reductions, participating companies must commit to work with EPA and others to develop and agree upon analytical standards and laboratory methods for these chemicals. EPA is also initiating efforts to add PFOA and related chemicals to the Toxics Release Inventory (TRI) to help monitor the results of the stewardship program.

Copies of the company letters of commitment and details on the PFOA Stewardship Program,

as well as additional information on PFOA:
epa.gov/opptintr/pfoa/commitments.htm

https://archive.epa.gov/epapages/newsroom_archive/newsreleases/95de36c6115a523a8525712500693772.html

Accessed February 7, 2020

Thomas J. DiPasquale/US-Corporate/3M/US
04/25/2006 10:28 PM

To Thomas A Boardman/LA-Legal/3M/US@3M-Corporate
Richard F. Ziegler/US-Corporate/3M/US@3M-Corporate
Katherine E. Reed/US-Corporate/3M/US@3M-Corporate
Fred J. Palensky/US-Corporate/3M/US@3M-Corporate
Dan E. Gahlon/US-Corporate/3M/US@3M-Corporate
cc Jo E. Cernohous/LA-Legal/3M/US@3M-Corporate
bcc
Subject Fw: 3M and EPA Press Release

EPA Press Release--Jo Cernohous is trying to get us together at 5:00 today.
Tom
----- Forwarded by Thomas J. DiPasquale/US-Corporate/3M/US on 04/25/2006 04:22 PM -----

JULIA.HATCHER@LW.com
04/25/2006 04:21 PM

To tjdipasquale@mmm.com
cc
Subject Fw: 3M and EPA Press Release

From Blackberry Wireless
Julia A. Hatcher
LATHAM & WATKINS
555 11th Street, N. W.
Washington, D. C. 20004
(202) 637-2238

-----Original Message-----
From: Ellis.Tony@epamail.epa.gov <Ellis.Tony@epamail.epa.gov>
To: Hatcher, Julia (DC) <JULIA.HATCHER@LW.com>
CC: Ziegel.Dean@epamail.epa.gov <Ziegel.Dean@epamail.epa.gov>
Sent: Tue Apr 25 17:17:12 2006
Subject: 3M and EPA Press Release

Dear Julia,

Below is the Press Release that was just released on the 3M/EPA settlement.

Tony R. Ellis, Case Development Officer
Toxics and Pesticides Enforcement Division (2245A)
Office of Civil Enforcement
1200 Pennsylvania Avenue, N.W.
Washington, DC 20460
Phone (202) 564-4167
Fax (202 564-0035

News for Release: Tuesday, April 25, 2006

U.S. Environmental Protection Agency (EPA)

EPA Settles Case Involving 3M Voluntary Disclosures of Toxic

Exhibit 2099
State of Minnesota v. 3M Co., Court File No. 27-CV-10-28862

Substances
Violations

Contact: Dave Ryan, (202) 564-4355 / ryan.dave@epa.gov

(Washington, D.C. - April 25, 2006) EPA and the 3M Company reached a $1.5 million settlement to resolve reporting violations under the Toxics Substances Control Act (TSCA) that the company voluntarily disclosed to EPA. EPA filed the settlement with the agency's Environmental Appeals Board today, for their review.

3M voluntarily disclosed all of the violations covered by this settlement under the terms of a TSCA corporate-wide audit agreement. 3M has agreed to perform a comprehensive management systems review of 28 separate business units and facilities and to determine the compliance status of all TSCA-regulated chemicals and processes. 3M agreed to pay a $1,521,481 penalty for 244 TSCA violations. As a result of this settlement, 3M has corrected a number of violations, including failures to notify EPA on new chemicals, late reporting on substantial risk information, and other reporting violations. During the course of the audit, 3M produced valuable, previously unreported information that will help the scientific community to better understand the presence of toxic substances in the environment.

"EPA takes violations of toxic substances laws seriously and is committed to enforcing those laws," said Granta Y. Nakayama, assistant administrator for EPA's Office of Enforcement and Compliance Assurance. "We are hopeful that today's action will serve as a reminder of the importance of timely industry reporting of substantial risk information to EPA."

Several of the violations concerned reporting on perfluorinated compounds, including perfluorooctyl sulfonates (PFOS) and perfluorooctanoic acid (PFOA). PFOS-related compounds were the active ingredients used for decades in the original formulation of 3M's Scotchgard stain and water repellents. 3M voluntarily stopped manufacturing PFOS in the United States in 2000, and phased out all of these chemistries on a global basis by the end of 2002. Data submitted by 3M and others led EPA to begin an investigation of these compounds in 2000.

EPA followed up the phase out of PFOS by taking action to implement significant new use rules to restrict the return of PFOS-related chemicals to the U.S. market.

Earlier this year, the agency launched a global PFOA stewardship program. The eight major companies that use or manufacture PFOA have committed to reduce facility emissions and product content of PFOA and related chemicals by 95 percent by no later than 2010, and to work

toward eliminating emissions and product content by 2015. Additional information on this program, and on all the agency's activities with regard to PFOA and related chemicals: http://www.epa.gov/oppt/pfoa/

More information on the settlement:
http://www.epa.gov/compliance/resources/cases/civil/tsca/3m.html

EPA's mission is to protect our nation's land, air and water.
Citizens
can help by reporting potential environmental violations:
http://www.epa.gov/compliance/complaints/

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

May 30, 2006

EPA-SAB-06-006

The Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Subject: SAB Review of EPA's Draft Risk Assessment of Potential Human Health Effects Associated with PFOA and Its Salts

Dear Administrator Johnson:

In response to a request from EPA's Office of Pollution Prevention and Toxics (OPPT), the Science Advisory Board (SAB) convened an expert panel to conduct a peer review of EPA's Draft Risk Assessment of Potential Human Health Effects Associated with Perfluorooctonoic Acid (PFOA) and Its Salts (dated January 4, 2005). PFOA is a synthetic (man-made) chemical used in the manufacture of several commercially important products. PFOA has been detected in the blood of the general U.S. population although it is not fully understood how individuals are exposed to the chemical. To determine whether environmental exposure to PFOA might pose a risk to human health, EPA's draft assessment provided an evaluation of available information on the health effects and human exposure to PFOA. The draft assessment also compared measured human blood levels with the estimated PFOA blood levels that are not anticipated to produce (or can produce minimal) toxicities based on data in tested laboratory animals.

The SAB was asked to comment on: (a) EPA's analysis of how PFOA causes tumors in rats and its relevance for human health and the weight-of- evidence conclusion about the potential for PFOA to cause cancer in humans; (b) the selection of health effects endpoints for risk assessment; (c) the adequacy of available data to provide information on exposure of the general population to PFOA; and (d) EPA's risk assessment approach including the use of kinetic models to estimate PFOA blood levels in available laboratory animals studies.

In general, the SAB Panel endorsed EPA's risk assessment approach, particularly, the inclusion of multiple non-cancer health endpoints for risk assessment, and the use of PFOA blood levels as a measure of estimated dose in place of the administered dose in toxicologic studies. The Panel recommended the inclusion of additional non-cancer health endpoints for risk assessment, and the use of the Benchmark Dose method to better estimate the lowest observed

effect levels and no observed effect levels for risk assessment. Three-quarters of the Panel judged that the weight-of-evidence conclusion for the potential of PFOA to cause cancer in humans was more aligned and consistent with the hazard descriptor of "likely to be carcinogenic" as described in the Agency's cancer guidelines (i.e., 2003 EPA Guidelines for Carcinogen Risk Assessment). They also recommended that a risk assessment be conducted for carcinogenic effects. About one-quarter of the Panel agreed with EPA's conclusion regarding the potential cancer hazard of PFOA to humans and the designation of the cancer descriptor of "suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential". Three quarters of the Panel considered the available human biomonitoring studies adequate to characterize environmental risk to PFOA for the general population. However, about one-quarter of the Panel believed that the available studies are inadequate for risk assessment of subpopulations possibly more highly exposed to PFOA. The scientific rationales for these viewpoints along with specific recommendations on these issues are detailed in the Panel's report.

The SAB strongly urges the Agency to strengthen its risk assessment by considering verified and peer reviewed new information found to be relevant and critical to the assessment. We look forward to receiving your response to this review and appreciate the opportunity to provide EPA with advice on this important subject. We stand ready to assist the Agency in any future efforts in updating the draft risk assessment.

Sincerely,

/signed/

Dr. M. Granger Morgan
Chair
EPA Science Advisory Board

/signed/

Dr. Deborah Cory-Slechta
Chair
PFOA Risk Assessment Review Panel
EPA Science Advisory Board

NOTICE

This report has been written as part of the activities of the EPA Science Advisory Board (SAB), a public advisory group providing extramural scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The SAB is structured to provide balanced, expert assessment of scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the Environmental Protection Agency, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names of commercial products constitute a recommendation for use. Reports of the SAB are posted on the EPA website at http://www.epa.gov/sab.

U.S. Environmental Protection Agency
Science Advisory Board
Perfluorooctanoic Acid Review Panel

CHAIR

Dr. Deborah Cory-Slechta, Director, Environmental and Occupational Health Sciences Institute, Robert Wood Johnson Medical School, University of Medicine and Dentistry of New Jersey and Rutgers State University, Piscataway, NJ

MEMBERS

Dr. Ernest Abel, Professor of Obstetrics and Gynecology; Professor of Psychology; Director, Reproductive Toxicology, Obstetrics and Gynecology Department, School of Medicine / C.S. Mott Center for Human Growth and Development, Wayne State University, Detroit, MI

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Dr. Norman Drinkwater, Professor and Chair of Oncology, McArdle Laboratory for Cancer Research, University of Wisconsin Medical School, Madison, WI

Dr. William L. Hayton, Professor and Associate Dean, Division of Environmental Health Science, College of Pharmacy, School of Medicine and Public Health, Ohio State University, Columbus, OH

Dr. Michael A. Kamrin, Professor Emeritus, Institute of Environmental Toxicology, Michigan State University, Haslett, MI

Dr. James Kehrer, Head, Division of Pharmacology and Toxicology, College of Pharmacy, University of Texas at Austin, Austin, TX

Dr. James E. Klaunig, Professor and Director, Department of Pharmacology and Toxicology, School of Medicine, Indiana University, Indianapolis, IN

Dr. Matthew P. Longnecker, MD ScD, Division of Intramural Research National Institute of Environmental Health Sciences, Epidemiology Branch, National Institute of Environmental Health Sciences, Research Triangle Park, NC

Dr. Ronald Melnick, Director of Special Programs, Environmental Toxicology Program, National Institute of Environmental Health Sciences, Research Triangle Park, NC

Dr. Franklin L. Mink, President, MAI, Lake Orion, MI

Dr. David M. Ozonoff, Professor, Department of Environmental Health, School of Public Health, Boston University, Boston, MA

Dr. Stephen Roberts, Professor, Center for Environmental and Human Toxicology, University of Florida, Gainesville, FL

Dr. Anne Sweeney, Associate Professor, Department of Epidemiology and Biostatistics, School of Rural Public Health, Texas A&M University, Bryan, TX

Dr. Thomas T. Zoeller, Professor, Biology Dept., Morrill Science Center, University of Massachusetts at Amherst, Amherst, MA

SCIENCE ADVISORY BOARD STAFF

Dr. Sue Shallal, Designated Federal Official, Science Advisory Board Staff Office, Washington, DC

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EXECUTIVE SUMMARY

EPA's Office of Pollution Prevention and Toxics (OPPT) requested that the Science Advisory Board review the "Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid (PFOA) and its Salts" (hereafter referred to as the "draft PFOA risk assessment document") which was made available publicly in January 2006. The PFOA Review Panel of the EPA Science Advisory Board met in February 2005 at which time nine charge questions raised by OPPT were deliberated. These questions focused on four issues including, a peroxisome proliferator-activated receptor α (PPAR-alpha) mode of action (MOA) for rodent liver tumors, carcinogenicity descriptors, useful models for evaluation of health effects, toxicokinetic considerations and reliance on currently available human biomonitoring exposure data for calculation of margins of exposure (MOEs). Further discussions of the entire Panel were held during a conference call in July 2005.

This Executive Summary highlights the outcome of the Panel's deliberations. It includes the context for the charge questions and issues raised for consideration by EPA, and the conclusions reached by the SAB Review Panel. The Panel reviewed and discussed the draft risk assessment and the data referenced therein with the understanding that further risk assessment will proceed as more data on PFOA health effects become available. In instances where the views of Panel members diverged, the summary and charge question responses focus to a greater extent on the view expressed by about three quarters of the Panel members since the view of about one-quarter of Panel members coincided with that already expressed in EPA's Draft Risk Assessment. During the review period, new information[1] was presented to the Panel for their consideration. The Panel encourages EPA to consider new information that has been verified and peer-reviewed prior to use in their revision of the Draft Risk Assessment.

Issue 1: Rodent PPAR-alpha Mode of Action for Hepatocarcinogenesis

In rats, PFOA induces liver adenomas, Leydig cell tumors (LCT) and pancreatic acinar cell tumors (PACT). The draft document concludes that these tumors constitute a triad and are the result of a PPAR-alpha agonism MOA. In this MOA, activation of PPAR-alpha leads to cell proliferation and decreased apoptosis, clonal expansion of preneoplastic foci and subsequent tumors. The draft document premises its conclusions about this MOA on studies showing that PFOA is a potent peroxisome proliferator in liver of rats and mice and, like other peroxisome proliferators, induces hepatomegaly in rats. In addition, requisite dose-response and temporal associations for some key events for this MOA have been reported.

Comment on the Weight of Evidence and Adequacy of the Data Available to Identify the Key Events for the PPAR-alpha agonist-induced Rodent Liver Toxicity and Hepatocarcinogenesis for PFOA.

[1] This information included, for example: A) a report entitled, "Pathology Peer Review and Pathology Working Group Review of Mammary Glands from a Chronic Feeding Study in Rats with PFOA Report" conducted by Experimental Pathology Laboratories, Inc. and submitted to the SAB by Dr. Larry Zobel of 3M Medical Department and B) data and documents submitted to the SAB by Mr. Robert Bilott of Taft, Stettinius & Hollister LLP.

The Panel's charge was to determine whether it agreed with the weight of evidence supporting a PPAR-alpha MOA for rodent liver toxicity and hepatocarcinogenesis. Panel members agreed that, considered collectively, evidence to date was consistent with an interpretation that liver tumor induction likely results from a PPAR-alpha MOA. This is based on the observations that PFOA activates the receptor, results in peroxisome proliferation, increases beta-oxidation and produces hepatomegaly, with dose and temporal responses consistent with the PPAR-alpha MOA. These events, moreover, depend upon a functional PPAR-alpha receptor, and no other known MOA, e.g., DNA reactivity or mutagenicity, has been identified.

However, with respect to uncertainties and limitations related to concluding that PPAR-alpha is the ***sole*** MOA for rodent liver tumor induction and toxicity, Panel views diverged.

About three quarters of the Panel members believed that at the current time, sufficient uncertainties and limitations of the data still exist with respect to reaching such a conclusion, given that: 1) In contrast to what would be predicted, administration of PFOA, but not the prototype PPAR-alpha agonist WY-14,643, increased liver weights in PPAR-alpha receptor knockout mice, i.e., in mice where PPAR-alpha activation was precluded, raising the possibility that PFOA-induced liver tumors could occur by PPAR-alpha independent effects. The significance of this finding currently remains uncertain in the absence of a corresponding assessment of histopathology or replication by another laboratory. 2) There is as yet no published evidence that the induction of PPAR-alpha by PFOA results in clonal expansion of pre-neoplastic foci which is considered a critical step in the proposed MOA. 3) There are no data demonstrating increased cell proliferation and/or decreased apoptosis in the liver of PFOA-treated rats, key causative events in the proposed MOA.

These Panel members also viewed two additional issues as requiring further consideration. One is the relevance of the PPAR-alpha MOA to humans. Given that human exposures to PFOA and related chemicals appear ubiquitous, uncertainties and limitations of the data for children have not been adequately characterized to be able to conclude that the PPAR-alpha MOA is not operative in this young age group. A secondary issue thought to require additional characterization in the PFOA response was the potential role of Kupffer cells, resident macrophages in the liver that do not express PPAR-alpha, but are activated by peroxisome proliferators.

A different view expressed by the remaining one quarter of the Panel members was that the observation in PPAR-alpha knockout mice of increased liver weights in response to PFOA, but not to the prototype PPAR-alpha agonist WY-14,643, was not sufficiently significant to undermine the view that PPAR-alpha agonism is the sole MOA for PFOA-induced rodent liver tumors.

In summary, Panel members agreed that collectively the weight of evidence supports the hypothesis that liver tumor induction in rodents by PFOA is mediated by a PPAR-alpha agonism MOA. Most Panel members, however, also felt, based on current evidence, that it is possible that PPAR-alpha agonism may not be the sole MOA for PFOA, that not all steps in the pathway of PPAR-alpha activation- induced liver tumors have been demonstrated, that other

hepatoproliferative lesions require clarification, and that extrapolation of this MOA across the age range in humans is not supported. A few panel members did not share these reservations about a PPAR-alpha agonism MOA for PFOA-induced rodent liver tumors.

Issue 2: Descriptor for Carcinogenic Potential

The draft document reaches the conclusion of 'suggestive' evidence of carcinogenicity but not sufficient to assess human carcinogenic potential of PFOA. This conclusion was based upon: 1) a PPAR-alpha MOA for liver tumors in rodents that was considered not relevant to humans because of their decreased sensitivity to PPAR-alpha agonism when compared to rodents, 2) the absence of hepatic cell proliferation in a 6 month study of PFOA administration in cynomologous monkeys, the species considered closest in physiology to humans; 3) the absence of a strong association between PFOA exposure and tumors in human studies as interpreted in the draft document; 4) the belief that the LCT and PACT tumors produced by PFOA in rats were probably not relevant to humans based on the lower levels of expression of the mediators leutinizing hormone (LCT) and choleocystokinin growth factor receptors (PACT) in humans, as well as differences in quantitative toxicodynamics between rats and humans; and 5) the view that mammary fibroadenomas reported in female rats are equivocal based on their comparable rates of occurrence relative to a historical control group.

Comment on the Proposed Descriptor for the Carcinogenic Potential of PFOA

About three quarters of the Panel members concluded that the experimental weight of evidence with respect to the carcinogenicity of PFOA was stronger than proposed in the draft document, and suggested that PFOA cancer data are consistent with the EPA guidelines descriptor 'likely to be carcinogenic to humans'. According to EPA's Guidelines for Carcinogen Risk Assessment[2] (also known as EPA's Cancer Guidelines), this descriptor is typically applied to agents that have tested positive in more than one species, sex, strain, site or exposure route, with or without evidence of carcinogenicity in humans. Conclusions of these Panel members were based on the following:

- While human data are ambiguous, two separate feeding studies in rats demonstrate that PFOA is a multi-site carcinogen.
- Uncertainties still exist (see Issue 1 comments) as to whether PPAR-alpha agonism constitutes the ***sole*** MOA for PFOA effects on liver. This was based on the fact that PFOA, but not the prototypical PPAR-alpha agonist, WY-14,643, increases liver weights in PPAR-alpha knockout mice, a finding of uncertain significance in the absence of liver histopathology and replication of this finding. Further, mitochondrial proliferation was suggested in the document as a basis of liver toxicity in monkeys exposed to PFOA.
- The exclusion of mammary tumors in the draft document based on comparisons to historical control levels from other laboratories was deemed inappropriate, since the most appropriate control group is a concurrent control group. Using that comparison,

[2] In March 2005, EPA published final Cancer Guidelines and Supplemental Guidance which can be found at the following URL: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=116283.

increases in both fibroadenomas (22%, 42% and 48% for rats treated with 0, 30 and 300 ppm APFO (ammonium perfluorooctanoate or C8, the ammonium salt of PFOA), respectively) and adenocarcinomas (15, 31% and 11%, respectively) were seen in the Sibinski *et al.* (1987) 2 yr PFOA feeding study.

- Insufficient data are currently available to determine the MOA for the observed Leydig cell tumors, pancreatic acinar cell tumors and mammary gland tumors. In the absence of a defined MOA for these tumor types, they must be presumed to be relevant to humans, as suggested by EPA's Cancer Guidelines.

Given the current data base, these Panel members were not willing to ascribe an associated probability value to the potential for PFOA-induced carcinogencity. Nevertheless, based on available evidence to date, most Panel members believed that risk assessments for each of the PFOA-induced tumors are appropriate at the current time.

A different view expressed by the remaining one-quarter of the Panel members was that currently available evidence does not exceed the descriptor "suggestive" of carcinogenicity, based on the belief that PPAR-alpha agonism does serve as the sole MOA for PFOA-induced rodent liver tumors (Issue 1) and that mammary tumors were not demonstrated in animals when compared to historical controls. Thus, these members did not believe the evidence exceeded the draft document descriptor of "suggestive".

Issue 3: Selection of Endpoints

The draft document proposes the use of multiple endpoints from several life stages, species and gender for risk assessment. No specific recommendations on the most appropriate parameters are stipulated at the current time.

Comment on the:

Selection of Toxicity Endpoints for the Risk Assessment
The Most Appropriate Lifestage/Gender/Species for Assessing Human Risk
The Appropriateness of the Available Animal Models

The Panel agreed with the current approach of inclusivity, given the current uncertainties noted above with respect to carcinogenicity, as well as the paucity of information on potential PFOA effects on non-cancer endpoints. Similarly, no exclusion of species should be considered at present, and differences between genders as demonstrated in rat studies again suggest multiple MOAs for PFOA. The use of multiple animal models is appropriate particularly in light of the reported differences in toxicokinetics in rats, non-human primates and humans. Resolution of most appropriate parameters must await additional research, but the process will be facilitated by the ability to measure internal dose.

Panel members did not reach full agreement as to endpoints that should be included for risk assessment and the significance of occupational biomonitoring data. About three quarters of the Panel members supported the inclusion of multiple cancer endpoints and liver histopathology as well as consideration of the data from occupational and epidemiological studies. While the draft document notes that the occupational studies suffer from the fact that they involve

multiplicity of exposures, other studies have shown a high correlation among fluorinated compounds in biological samples from the general population and occupational cohorts. Therefore, these human studies could be advantageous for assessing potential interactions among these compounds that may be associated with adverse human health effects. These Panel members also believed that epidemiologic and occupational studies could not be disqualified without disqualifying virtually all such studies in the risk assessment process. Moreover, it is clear that occupationally-exposed populations have experienced the highest levels of exposure and therefore reported health effects in these studies merit consideration.

A contrasting view expressed by the remaining one-quarter of the Panel members was that the outcomes from studies of human health effects of PFOA were equivocal, and thus these endpoints should not be incorporated into the risk assessment process.

Panel members agreed on the need for additional research, including PPAR-alpha mediated and independent effects of PFOA. Non-carcinogenicity endpoints merit additional attention for several reasons. It is not yet known whether carcinogenicity will represent the most sensitive endpoint for PFOAs. Immunotoxicity has been reported, and derivations of MOEs for such effects were encouraged by many Panel members. Given the prevalence in brain of PPAR receptors, including PPAR-alpha, effects on nervous system structure and function warrant attention. Moreover, no information currently exists with respect to critical periods; therefore, it is important to evaluate effects across age groups. The observations of hormonal alterations in treated animals also deserve further study to assess their importance.

Issue 4: Risk Assessment Approach

Issue 4a: Pharmacokinetic Modeling and Use of AUC as a Measure of Internal Dose

The draft document compares internal dose metrics from animal toxicology studies and human biomonitoring studies for purposes of ultimately generating margin of exposure (MOE) information. Area under the concentration curve (AUC) was calculated from PFOA serum levels in human biomonitoring studies assuming a steady state. In some of the rat studies, serum PFOA concentrations were available, or it was considered that sufficient pharmacokinetic information was available to estimate serum levels. For this purpose, AUC was estimated from a pharmacokinetic model. Specifically, compartmental modeling of serum concentrations using single dose rat oral exposure studies were used to estimate internal dosimetry for the longer term dosing studies based upon the premise that pharmacokinetic information for rats and humans is sufficient for this purpose and that this approach does not exceed the limits of the available data.

Comment on the Use of the One Compartment Pharmacokinetic Model

The Panel concluded that the empirical model used in the draft document was adequate for predicting blood levels resulting from repeated dosing, but that this fitting procedure is specific to this limited data set and this particular application. Concern was expressed, therefore, that use of the descriptor "one compartment" to describe PFOA pharmacokinetics in the draft document is misleading, given the actual complexities in many of the available datasets, and the term should be removed or replaced unless carefully qualified.

Comment on the use of the AUC as a Measure of Internal Dose for Rats and Humans for Calculation of the MOE

The Panel observed that while calculating blood AUC may be an appropriate method to estimate internal dose, it is important to note that at the current time information on PFOA health effects is limited. As additional data become available, other measures may also be appropriate, such as the C_{max}, the integrated dose above a minimum concentration, etc. Regardless of the choice for the measure of internal dose, a clearer rationale needs to be presented for the approach taken, and, importantly, for any choice adopted, the impact of the internal dose measure on the magnitude of the MOE should be described. The Panel also believes that caution should be exercised in assuming that the form of PFOA in blood, i.e., free compound or PFOA bound to various proteins or lipids is constant in serum across the period of observation, given the current information on metabolism.

Issue 4b: Cross Species Extrapolation

In extrapolating data from animal experiments to humans, a default value of 10 is typically applied, with a factor of 3 for differences in toxicodynamics and a value of 3 for toxicokinetic differences. In the PFOA draft risk assessment document, internal doses from animal toxicology studies and human biomonitoring studies were compared. Derivation of data from animal toxicology studies included both measured PFOA serum levels from non-human primates and derived values from pharmacokinetic modeling from rat studies. The reliance on internal dose metrics was considered by OPPT to be sufficient to reduce uncertainties and therefore raised the question of the ability to either eliminate or reduce the default values for cross species extrapolation.

Comment on the Need to Use or Modify the Default Value of 10 for Cross Species Extrapolation Given the Pharmacokinetic Analysis

The use of internal dose metrics in this analysis was considered by the Panel to be a significant step toward reducing uncertainty related to the toxicokinetic uncertainty associated with interspecies extrapolation. Nevertheless, it did not believe that the direct use of blood concentration in the assessment sufficiently reduced the overall uncertainly to eliminate or modify the current default value. Significant uncertainties still remain, including the measured internal dose that best predicts adverse effects in human and other species, the bias inherent in measurement/modeling errors, the lack of information about non-cancer endpoints, developmental vulnerability and the impact of gender, and the multiple PFOA environmental exposures that occur in humans vs. animals, among others. The assumption that PFOA serum levels are at steady state in children 2-12 years of age has not been tested and may not be valid. The Panel likewise stressed that bench mark dose methodologies would be preferable to the reliance in the draft document on LOAEL-driven MOE calculations.

Issue 4c: Human Biomonitoring Data

Currently available data on PFOA levels in humans includes occupational biomonitoring studies as well as three population studies within the U.S. The measurements from the population studies come from: 1) samples from 6 American Red Cross blood banks; 2) a study of Streptococcal A infection in children; and 3) elderly volunteers in a cognitive study in Seattle. The draft EPA document only utilizes data derived from 1 and 2 above in its calculation of the MOE. Occupational biomonitoring data were excluded from the calculation because it was stated that sample sizes were small, data on gender were not available, and that blood monitoring data obtained from 2000 would overestimate current serum levels, since PFOA exposure of this group ceased in 2002. Measured levels from the elderly population were not utilized because values were considerably lower, for unknown reasons, than those reported in the other population studies for adults and children. From the other two population studies utilized in the draft document, geometric means and 90^{th} percentiles were calculated across genders for calculation of MOEs.

Comment on the Adequacy of the Human Exposure Data for Use in Calculating a MOE

Panel members were not in full agreement as to the adequacy of the human exposure data for inclusion in the MOE calculation. Many Panel members shared concerns about the approach adopted in the draft document. One concern related to the generality of the populations currently included in the MOE calculation. It was noted, for example, that use of the blood donor and pediatric biomonitoring data may be acceptable if the purpose is to assess whether there is a potential health effect to the "general" population, although there is some question as to the size of other non-occupational populations that might be more highly exposed and the assumption that PFOA serum levels are at steady state may not be valid for children or fetuses. About three quarters of Panel members agreed that existing subpopulations of the general public are likely to be more highly exposed than those previously reported and results from occupational studies should be included in the MOE calculation. A differing view expressed by the remaining one quarter of the Panel members was that the human biomonitoring data are equivocal and thus not useful to MOE calculation.

Three different summary statistics are presented in the draft document and used in the calculation of the MOE. Of these, the Panel questioned the use of mean values, particularly geometric means in the calculations. Additionally, no rationale was provided for the choice of the 90^{th} percentile as a summary statistic, rather than the use of a higher value. Whatever the approach adopted, justification must be provided for the chosen summary measure and an explicit objective for the MOE analysis described.

INTRODUCTION

Background

This report was prepared by the Science Advisory Board (SAB) PFOA Risk Assessment Review Panel (the "Panel") in response to a request by EPA's Office of Pollution Prevention and Toxics (OPPT) to review their *Draft Risk Assessment of the Potential Human Health Effects Associated With Exposure to Perfluorooctanoic Acid (PFOA) and Its Salts*. According to the document, OPPT has been investigating PFOA and its salts to try to understand the health and environmental issues presented by fluorochemicals, in the wake of unexpected toxicological and bioaccumulation discoveries with respect to perfluorooctane sulfonates (PFOS). PFOA and its salts are fully fluorinated organic compounds that can be produced synthetically or through the degradation or metabolism of other fluorochemical products. PFOA is primarily used as a reactive intermediate, while its salts are used as processing aids in the production of fluoropolymers and fluoroelastomers and in other surfactant uses. PFOA and its salts are persistent in the environment.

OPPT identified 4 issues where they were seeking the SAB's advice and recommendations. These included the proposed mode of action, carcinogenicity descriptors, toxicological endpoints selected and the pharmacokinetic modeling methods used in the risk assessment. OPPT's assessment focused on the potential human health effects associated with exposure to PFOA and its salts. Several toxicological endpoints and hypothesized modes of action were considered. Internal dose metrics were estimated for animal toxicology studies with pharmacokinetic modeling, and were obtained from human biomonitoring studies, assuming steady state. Margin of Exposure (MOE) values were calculated from the internal dose metrics. The SAB PFOA Review Panel was asked to comment on the scientific soundness of this risk assessment.

The Panel deliberated on the charge questions during their February 22-23, 2005 face-to-face meeting and during a conference call on July 6, 2005. The responses that follow represent the views of the Panel. In all cases, there was agreement by a majority of the panel members as to a particular recommendation. In some cases, there were one or more panel members that had a differing point of view; these instances have been noted throughout the report. The specific charge questions to the Panel are as follows:

Charge Questions

Issue 1: Rodent PPAR-alpha Mode of Action for Hepatocarcinogenesis

The postulated mode of action (MOA) of PPARα-agonist induced liver toxicity and liver tumors in rodents involves four causal key events. The first key event is activation of PPARα (which regulates the transcription of genes involved in peroxisome proliferation, cell cycle control, apoptosis, and lipid metabolism). Activation of PPARα leads to an increase in cell proliferation and a decrease in apoptosis, which in turn leads to preneoplastic cells and further

clonal expansion and formation of liver tumors. Of these key events, only PPARα activation is highly specific for this MOA while cell proliferation/apoptosis and clonal expansion are common to other modes of action. There are also several "associative" events that are markers of PPARα agonism but are not directly involved in the etiology of liver tumors. These include peroxisome proliferation (a highly specific indicator that this MOA is operative) and peroxisomal gene expression.

Information that provides evidence that any specific chemical is inducing liver toxicity and tumors via a PPARα agonist MOA includes *in vitro* evidence of PPARα agonism (i.e., evidence from an in vitro receptor assay), *in vivo* evidence of an increase in number and size of peroxisomes, increases in the activity of acyl CoA oxidase, and hepatic cell proliferation. The *in vivo* evidence should demonstrate dose-response and temporal concordance between precursor events and liver tumor formation. Other information that is desirable and may strengthen the weight of evidence for demonstrating that a PPARα agonist MOA is operative includes data on hepatic CYP4A1 induction, palmitoyl CoA activity, hepatocyte hypertrophy, increase in liver weights, decrease in the incidence of apoptosis, increase in microsomal fatty acid oxidation, and enhanced formation of hydrogen peroxide.

OPPT has proposed that there is sufficient weight of evidence to establish that the mode of action for the liver tumors (and precursor effects) observed in rats following exposure to PFOA is PPARα agonism.

Question 1 - Please comment on the weight of evidence and adequacy of the data available to identify the key events for the PPARα agonist-induced rodent liver toxicity and hepatocarcinogenesis for PFOA. Discuss whether the uncertainties and limitations of these data have been adequately characterized.

Issue 2: Descriptor for Carcinogenic Potential

Carcinogenicity studies in Sprague-Dawley rats show that PFOA induces a "tumor triad" similar to a number of other PPARα agonists. This "tumor triad" includes liver tumors, Leydig cell tumors (LCT), and pancreatic acinar cell tumors (PACT). OPPT has proposed that there is sufficient evidence to conclude that the liver tumors are due to PPARα-agonist MOA, and that this MOA is unlikely to occur in humans based on quantitative differences between rats and humans. In addition, the LCT and PACT induced in the rat by PFOA probably do not represent a significant cancer hazard for humans because of quantitative toxicodynamic differences between the rat and the human. Overall, based on no adequate human studies and uncertain human relevance of the tumor triad (liver, Leydig cell and pancreatic acinar cell tumors) from the rat studies, OPPT has proposed that the PFOA cancer data may be best described as providing "*suggestive evidence of carcinogenicty, but not sufficient to assess human carcinogenic potential*" under the interim 1999 EPA Guidelines for Carcinogen Risk Assessment, as well as the 2003 draft EPA Guidelines for Carcinogen Risk Assessment.

Question 2 - Please comment on the proposed descriptor for the carcinogenic potential of PFOA.

Issue 3: Selection of Endpoints

OPPT has proposed the use of several endpoints from several life stages, species and gender for the risk assessment. For this draft assessment, OPPT has not made specific recommendations on the most appropriate endpoint/lifestage/species/gender. Rather, all have been presented to provide transparency.

For adults, endpoints were selected from the non-human primate and rat studies; the endpoints included liver toxicity and possibly mortality for the non-human primates and decreased body weight for rats.

For developmental endpoints, OPPT relied upon the definition of developmental toxicity outlined in the Agency's Developmental Toxicity Risk Assessment Guidelines. These guidelines state that the period of exposure for developmental toxicity is prior to conception to either parent, through prenatal development and continuing until sexual maturation. (In contrast, the period during which a developmental effect may be manifested includes the entire lifespan of the organism). Based on this definition of developmental exposure, OPPT considered developmental effects in the rat two-generation reproductive toxicity study to include reductions in F1 mean pup body weight (sexes combined) on lactation days 1, 5 and 8, an increase in mortality during the first few days after weaning (both sexes), a delay in the timing of sexual maturation (both sexes), and a reduction in mean body weight postweaning (F1 males only).

Question 3 - Please comment on the selection of these toxicity endpoints for the risk assessment.

Question 4 - Given the available data to date, please comment on the most appropriate lifestage/gender/species for assessing human risk.

Question 5 - Please comment on the appropriateness of the available animal models. Please comment on whether additional animal models should be investigated, and if so, what information would better enable us to ascertain potential human risks.

Issue 4: Risk Assessment Approach

A margin of exposure (MOE) approach can be used to describe the potential for human health effects associated with exposure to a chemical. The MOE is calculated as the ratio of the NOAEL or LOAEL for a specific endpoint to the estimated human exposure level. The MOE does not provide an estimate of population risk, but simply describes the relative "distance" between the exposure level and the NOAEL or LOAEL. In this risk assessment there is no information on the sources or pathways of human exposure. However, serum levels of PFOA, which are indicative of cumulative exposure, were available from human biomonitoring studies. In addition, serum levels of PFOA were available for many of the animal toxicology studies or there was sufficient pharmacokinetic information to estimate serum levels. Thus, in this

assessment internal doses from animal and human studies were compared; this is analogous to a MOE approach which uses external exposure estimates.

Issue 4a: Pharmacokinetic Modeling and Use of AUC as a Measure of Internal Dose

As noted above, internal dose metrics from animal toxicology studies and human biomonitoring studies were compared in this draft assessment. For humans, the area under the concentration curve (AUC) was calculated from measured PFOA serum levels in human biomonitoring studies, assuming steady state. For the rat toxicology studies, the area under the concentration curve (AUC) and C_{max} were estimated from a pharmacokinetic model. The pharmacokinetic analysis could be done using a number of approaches including non-parametric analysis, physiologically based pharmacokinetic (PBPK) modeling, and classical compartmental modeling. Each has strengths and limitations given the available data. Non-parametric analyses provide a description of the data that have been collected, but have fairly limited ability to make predictions across species or to account for variations in exposures. PBPK modeling is perhaps the ideal approach for addressing PFOA for purposes of cross-species extrapolation. Extensive pharmacokinetic studies have been undertaken in rats demonstrating complex phenomena including high tissue concentrations in liver, kidney and serum and enterohepatic recirculation of the parent compound. These could be addressed using PBPK modeling for the rats, but the more limited information in monkeys and humans would either require substantial assumptions or preclude use of this approach. Classical compartmental modeling can be used to analyze the existing data on blood concentrations in rats, monkeys, and humans. Currently, the available pharmacokinetic information for rats and humans is sufficient to support compartmental modeling. Comparisons of serum protein binding across species indicated a high degree of binding in all species but also interspecies differences in the percentage of unbound PFOA in plasma. In light of the documented differences in clearance of PFOA across sexes in rats and across species, compartmental modeling of serum concentrations provides a sound approach for estimating internal dosimetry without exceeding the limits of the available data, so this approach was selected for this risk assessment.

Question 6 - Please comment on the use of the one compartment pharmacokinetic model.

Question 7 - Please comment on the use of the AUC as a measure of internal dose for rats and humans for calculation of the MOE.

Issue 4b: Cross Species Extrapolation

Judgments about the "adequacy" of a MOE are based on many considerations including uncertainty associated with cross species extrapolation. Typically, a value of 10 is considered which consists of a value of 3 for toxicodynamics and a value of 3 for toxicokinetics. Each of these can be decreased or increased if there are data to warrant it. In this draft assessment, internal doses from animal toxicology studies and human biomonitoring studies were compared. For humans, the internal doses were based on measured PFOA serum levels in human biomonitoring studies. For the non-human primate toxicology studies, internal doses associated with the NOAEL and/or LOAEL were based on measured PFOA serum levels. For the rat

toxicology studies, pharmacokinetic modeling was used to estimate an internal dose metric associated with a NOAEL or LOAEL.

Question 8 - Please comment on the need to use or modify the default value of 10 for cross species extrapolation given the pharmacokinetic analysis.

Issue 4c: Human Biomonitoring Data

For this draft assessment, human biomonitoring data of PFOA serum levels were available for adults and children. Similar analytical methods were used to measure the PFOA levels in both sets of blood samples. The adult data included 645 U.S. adult blood donors (332 males, 313 females) from 2000-2001, ages 20-69, obtained from six American Red Cross blood banks located in: Los Angeles, CA; Minneapolis/St. Paul, MN; Charlotte, NC; Boston, MA; Portland, OR, and Hagerstown, MD. Each blood bank provided approximately 10 samples per 10-year age interval (20-29, 30-39, etc.) for each sex.

The children's data included a sample of 598 children, ages 2-12 years old, who had participated in a study of group A streptococcal infections. The samples collected in 1994-1995 from children residing in 23 states and the District of Columbia were analyzed for PFOA in 2002.

Question 9 - Please comment on the adequacy of the human exposure data for use in calculating a MOE.

RESPONSES TO THE CHARGE QUESTIONS

Issue 1: Rodent PPAR-alpha Mode of Action for Hepatocarcinogenesis and Liver Toxicity

Question 1. *Please comment on the weight of evidence and adequacy of the data available to identify the key events for the PPAR alpha agonist induced rodent liver toxicity and hepatocarcinogenesis for PFOA. Discuss whether the uncertainties and limitations of these data have been adequately characterized.*

As discussed in the EPA Draft Risk Assessment of the Potential Human Health Effects Associated with Exposure to Perfluorooctanoic Acid and its Salts, a sequence of four key events define the mode of action (MOA) by which PPAR-alpha agonists induce rodent liver tumors. According to this MOA, the initial causal event is (1) activation of PPAR-alpha, which regulates the expression of genes involved in peroxisome proliferation, cell cycle control, apoptosis, and lipid metabolism. These transcriptional events lead to (2) increased cell proliferation and/or decreased cell death. The chronic increase in cell growth occurs primarily in the preneoplastic focal lesions in the liver resulting (3) in the clonal expansion of the preneoplastic lesions, which ultimately results (4) in the development of hepatocellular neoplasms. In addition, there are "associative" events that may or may not be causally linked to the PPAR-alpha MOA for hepatocarcinogenesis which include blockage of cell to cell communication, an increase in peroxisomes, an increase in peroxisomal enzymes, and liver and hepatocyte hypertrophy.

The Panel agreed that, considered collectively, the weight of evidence to date is consistent with the assertion that PFOA is a PPAR-alpha agonist and can induce liver changes in adult rats that have been associated with PPAR-alpha activation. As discussed in the draft PFOA risk assessment, some of the key elements to establish this MOA have been demonstrated by appropriate experiments. In vitro studies demonstrate that PFOA is a PPAR-alpha agonist, and treatment of rats and/or mice results in peroxisome proliferation, increased beta-oxidation, and hepatomegaly, with dose and temporal responses consistent with this MOA for liver tumor induction. Studies comparing PPAR-alpha null and wild-type mice showed that PFOA-induced peroxisome proliferation, beta-oxidation, and immunotoxicity depend on the presence of a functional receptor. Further, no other established modes of action of liver cancer-induction have been reported for PFOA. PFOA is neither DNA reactive nor mutagenic, and thus not involved in a genotoxic mode of action; nor is the liver neoplastic effect due to the induction of repeated hepatocyte death and compensatory regeneration (a cytotoxic mode of action). No PPAR-alpha independent MOA for the rat liver tumor induction has been proposed.

With respect to the weight of evidence and the adequacy of consideration of uncertainties and limitations, however, the Panel did not reach full agreement. About three quarters of the Panel members believed that data gaps still exist and not all of the causal events in the PPAR-alpha MOA have been demonstrated for PFOA. These include the induction of cell proliferation in the liver at early times following PFOA treatment and/or modulation of apoptosis in hepatocytes. They also shared the belief that while the PFOA Draft Risk Assessment in general appropriately discusses the uncertainties and limitations of the data that support a PPAR-alpha MOA for PFOA-induced liver tumors in adult rats, it fails to consider three issues contrary to this MOA in sufficient detail.

First, in a study by Yang *et al.* (2002) cited in the report in the context of the receptor dependence of PFOA immunotoxicity, PPAR-alpha null mice exhibited >2-fold increases in liver weight but no peroxisomal acyl CoA oxidase induction in response to PFOA. No increase in liver weight was observed in PPAR-alpha null mice treated with the well-characterized prototype PPAR-alpha agonist, WY-14,643. While this finding is of uncertain significance, due to the lack of histopathology and the absence of a second study showing such an effect, it nevertheless raises the possibility that PFOA may induce some of its effects in mouse liver by a PPAR-alpha-independent pathway. This observation and the associated uncertainty were not mentioned in the context of liver tumor induction in the draft PFOA risk assessment. Secondly, uncertainties exist with respect to the relevance to exposed fetuses, infants and children of the PPAR-alpha agonist MOA for induction of liver tumors in adults. Humans are refractory to some, but not all, PPAR-alpha activation effects. Data from studies using PPAR-alpha receptor knockout mice have shown that these receptors are essential for the rapid induction of liver neoplasms after exposure to WY-14,643. However, humans do have functional PPAR-alpha receptors, leaving unanswered the question as to why they respond so differently from rats and mice to PPAR-alpha agonists. Available data suggest that the difference between humans and rats or mice may be a consequence of a lower number of PPAR-alpha receptors such that the PPAR-alpha MOA is not considered likely to yield a similar hepatic cancer response in adult humans. However, exposures of fetuses, neonates and children to PFOA remain a potential concern. Rat studies suggest similar PPAR-alpha receptor levels in neonates and adults, but because adult humans have so few receptors, and information in fetuses, neonates and children is minimal, this same extrapolation cannot be made in humans. Given that human exposures to PFOA and related chemicals appear ubiquitous, uncertainties and limitations of the data for children have not been adequately characterized to be able to conclude that the PPAR-alpha MOA is not operative in this young age group.

Second, the current draft PFOA risk assessment states (page 76 lines 15-16) that the "[a]ctivation of PPAR-alpha leads to an increase in cell proliferation and a decrease in apoptosis, which in turn leads to preneoplastic cells …" Questions were raised as to whether there is available experimental evidence that the induction of PPAR-alpha results in an increase in the number of preneoplastic foci. The effect of the PPAR-alpha activation appears to be at the level of focal lesion clonal expansion (Klaunig *et al.*, 2003), however clonal expansion of focal lesions, which is not unique to a PPAR-alpha MOA, has not been shown in rats treated with PFOA.

Thirdly, some Panel members felt that the role of Kupffer cells (shown in Figure 1, page 78 of the draft document) should be discussed in the text of the draft PFOA risk assessment. There is an extensive literature on the essential role of Kupffer cells in signaling peroxisome proliferator-induced hepatocyte proliferation. Studies have shown that hepatocyte proliferation and peroxisome proliferation occur by different mechanisms. Parzefall *et al.* (2001) and Hasmall *et al.* (2001) demonstrated that peroxisome proliferators had no effect on DNA synthesis but still induced peroxisomal acyl CoA oxidase activity in cultured rat and mouse hepatocytes that had been purified to remove contaminating Kupffer cells. Kupffer cells, which are resident macrophages in the liver, are a major source of growth factors (tumor necrosis factor alpha, interleukins) that induce DNA synthesis or suppress apoptosis in purified hepatocytes. A key

finding relevant to the proposed MOA is that Kupffer cells do not express PPAR-alpha (Peters *et al.*, 2000), but are activated by peroxisome proliferators. Prevention of Kupffer cell activation by glycine inhibited, although not completely, the development of liver tumors by the potent peroxisome proliferator, WY-14,643 (Rose *et al.*, 1999). There are no data available on the effects of peroxisome proliferators on human Kupffer cells. Recognizing the role of Kupffer cell activation in the induction of DNA synthesis and subsequent neoplastic development by PPAR-alpha agonists, some members of the FIFRA Science Advisory Panel (2003) [SAP Minutes No. 2003-05] noted that the interplay between PPAR-alpha agonism and Kupffer cells has not been characterized. Thus, the results from the PPAR-alpha null mouse are not directly applicable to the human situation in which PPAR-alpha is present and can be activated.

A different conclusion was reached by the remaining one quarter of the Panel members who found that the weight of evidence was adequate to support a PPAR-alpha agonism mode of action for PFOA-induced rodent liver tumors. In this view, the observation of increased liver weights in response to PFOA but not to the prototype PPAR-alpha agonist WY-14,643 in PPAR-alpha knock-out mice as reported in Yang *et al.* (2002) did not merit significance because the study was not designed to evaluate liver toxicity, and the observation represents a single replication without corresponding histopathology at the current time. Nor was the possible role of Kupffer cells considered to be significant. Based on these considerations, these Panel members believed that PPAR-alpha agonism can be considered the sole MOA for PFOA-induced rodent liver tumors.

Issue 2: Descriptor for Carcinogenic Potential

Question 2. *Please comment on the proposed descriptor for the carcinogenic potential of PFOA.*

The draft PFOA risk assessment proposes that the PFOA cancer data may be best described as providing "*suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential*" under the interim 1999 EPA Guidelines for Carcinogen Risk Assessment (US EPA, 1999), as well as the 2003 draft EPA Guidelines for Carcinogen Risk Assessment (US EPA, 2003). This opinion is based on the view that human studies on PFOA do not provide adequate support of carcinogenicity, as well as on the quantitative differences between rats and humans that OPPT believes raises uncertainties about the human relevance of the "tumor triad" response (liver tumors, Leydig cell tumors, and pancreatic acinar cell tumors) of PPAR-alpha agonist activation in rats.

The determination of an appropriate descriptor for the carcinogenic potential of PFOA was discussed by the Panel in the context of the available carcinogenicity data, an evaluation of mechanistic or MOA data, and guidance on how EPA applies various descriptors for summarizing weight of evidence data. Panel members did not achieve full agreement on the appropriate descriptor. Based on the above considerations, the view of about three quarters of the Panel members was that the descriptor "likely to be carcinogenic" was more consistent with currently available data, while the remaining one quarter of the Panel members reached the

conclusion that the current evidence fails to exceed the descriptor "suggestive" of carcinogenicity.

Cancer studies on PFOA

Carcinogenicity studies in Sprague-Dawley rats have shown that PFOA induces neoplasms at multiple sites. In male rats exposed to 0 or 300 ppm ammonium perfluorooctanoate (APFO) in the feed for 2 years, increased incidences of testicular Leydig cell tumors (LCT) (0% vs. 11%), pancreatic acinar cell tumors (PACT) (0% vs. 11%), and liver adenomas (3% vs. 13%) were observed in treated animals compared to controls (Biegel *et al.*, 2001). In a 2-year study in which male and female Sprague-Dawley rats were fed diets containing 0, 30 or 300 ppm APFO, a dose-related increase in LCT was observed (0% in controls, 4% at 30 ppm, 14% at 300 ppm) (Sibinski *et al.*, 1987). The draft PFOA risk assessment does not address the effects in the liver observed in the Sibinski *et al.* (1987) study. In that study, the incidences of hepatocellular carcinoma in male rats were 6%, 2%, and 10%, and although no adenomas were diagnosed, the incidences of hyperplastic nodules in the liver were 0%, 0%, and 6%. Hyperplastic nodules may be part of the continuum of proliferative lesions in the liver carcinogenic process.

In female rats, a dose-related increase in mammary gland fibroadenomas was reported (22% in controls, 42% at 30 ppm, and 48% at 300 ppm) by Sibinski *et al.* (1987). In addition, the incidence of mammary gland adenocarcinomas was greater in the low dose group than in controls (15% in controls, 31% at 30 ppm, and 11% at 300 ppm). The draft PFOA risk assessment did not consider the mammary gland neoplasms to represent a compound-related effect because of high background rates reported for fibroadenomas in Sprague-Dawley rats in historical control data (37%) reported for female rats in the Haskell Laboratory in 1987 (Sykes). Three-quarters of the Panel members did not believe that historical control comparisons are as reliable as concurrent controls. A number of parameters may contribute to inter-laboratory differences in tumor response including differences in diet, animal age at the start and termination of studies, animal supply sources and breeding practices, environmental conditions, vehicles and routes of administration, animal care procedures that may affect weight gain and survival, and the use of different substrains. Thus, in their view, the concurrent control group is the most appropriate group for evaluations of chemical-related effects. Moreover, in the historical database of Chandra *et al.* (1992), the incidence in controls of mammary gland fibroadenomas was 19.0% and the incidence of adenocarcinomas was 8.8% in female Sprague-Dawley rats. Therefore, a neoplastic effect in the mammary gland is apparent in the Sibinski study in comparison to Chandra *et al.* (1992). Those Panel members therefore believe that the elevated tumor rates observed in female rats in the Sibinski *et al.* (1987) study raise concerns for neoplastic effects induced by PFOA in the mammary gland that should not be dismissed. In addition, while new information[3] was submitted to the panel questioning the findings in the Sibinski study, about three quarters of the Panel members urged that an independent, appropriately-designed histopathology review of the male rat livers and the female mammary glands from the Sibinski study be conducted to re-analyze the resulting tumor incidence data.

[3] a report entitled, "Pathology Peer Review and Pathology Working Group Review of Mammary Glands from a Chronic Feeding Study in Rats with PFOA Report" conducted by Experimental Pathology Laboratories, Inc. and submitted to the SAB by Dr. Larry Zobel of 3M Medical Department

The remaining Panel members believed that the comparison of the Sibinski *et al.* (1987) mammary tumor data to the historical control data (Sykes, 1987) in the draft risk assessment document was valid.

Mode-of-action analysis, uncertainties, and human relevance

The PFOA draft risk assessment proposes that there is sufficient evidence to conclude that liver tumors induced by PFOA are due to a proposed PPAR-alpha agonist MOA (Klaunig *et al.*, 2003), and that this MOA is unlikely to occur in humans based on quantitative differences in the numbers of PPAR-alpha receptors between rats and humans. In addition, the PFOA draft risk assessment proposes that the Leydig cell tumors (LCT) and pancreatic acinar cell tumors (PACT) induced in the rat by PFOA probably do not represent a significant cancer hazard for humans because of quantitative toxicodynamic differences between the rat and the human. Thus, the panel examined issues related to our understanding of the MOA for the multiple tumor types induced by PFOA in rats and the impact of available information on determining the human relevance of the animal tumor responses.

Liver adenomas.

As noted under Issue 1, the Panel concurred that the collective evidence is consistent with the hypothesis of a PPAR-alpha agonist MOA for PFOA with associated peroxisomal β-oxidation activity, increases in absolute and relative liver weight, and liver tumors in Sprague-Dawley rats. Issues on which the Panel members opinions diverged related to whether a PPAR-alpha agonist MOA for liver tumor induction in rats might occur in humans and/or whether additional MOAs might be involved.

Key events in the PPAR-alpha agonist MOA.

The PFOA risk assessment did not identify dose-response data showing increases in hepatocyte proliferation and suppression of apoptosis in rats exposed to PFOA. Many Panel members believed this to be a critical deficiency, because these are key events in the proposed MOA linking activation of PPAR-alpha to the liver tumor response.

Another observation that influenced most Panel members with respect to potential human relevance of the response in rats is the observation that the same early effects actually occur in monkeys exposed to PFOA. These effects include the induction of peroxisomal β-oxidation activity (2.6 fold), significant increases and positive dose-response trends for absolute and relative liver weights (1.6 fold), and the return of relative liver weight to control levels after a 13-week recovery period. Cell proliferation was evaluated in monkeys but only after 6 months of exposure. Unfortunately, neither the rat nor the monkey studies provided data on hepatocyte proliferation during the first 1-2 weeks of exposure, or direct measurements of apoptotic cells during or after exposure to PFOA was stopped. The lack of data on cell proliferation and apoptosis in animals exposed to PFOA makes it impossible to analyze dose-response concordance between these key events and tumor induction for PFOA in relation to other PPAR-alpha agonists. Because the available data for PFOA in rats and monkeys indicate similar responses in the livers of rodents and primates (increased liver weight and induction of hepatic peroxisomal enzyme activity), about three quarters of the Panel members shared the view that human relevance for liver effects induced by PFOA by a PPAR-alpha agonism MOA cannot be discounted.

The remaining panel members, however, considered the increase in liver weight in rats exposed to PFOA and the return to control levels following an 8-week recovery period (Palazzolo, 1993) to be consistent with an increase in cell proliferation and suppression of apoptosis by PFOA during the exposure period. In addition, the lack of an increase in hepatic cell proliferation in rats after 1 month or more exposure to PFOA (Biegel *et al.*, 2001) was considered consistent with observations of a transient increase in hepatocyte proliferation with other peroxisome proliferators.

PPAR-alpha -independent liver effects.

As noted in response to Issue 1, about three quarters of the members of the Panel shared the view that significant uncertainties still exist with respect to the predictability of a PPAR-alpha agonist MOA for human cancer risk associated with exposure to PFOA. In a comparative study of PFOA and the prototype PPAR-alpha agonist Wy-14,643, at doses of each chemical that produced increases in liver weight and peroxisomal fatty acid acyl-CoA oxidase activity in wild-type mice, only PFOA caused a similar 2-fold increase in liver weight (but no increase in acyl-CoA oxidase activity) in PPAR-alpha null mice (Yang *et al.*, 2002). While this study was not designed to assess liver toxicity, it confirms that PFOA is a PPAR-alpha agonist for peroxisomal enzyme induction, and also indicates that liver changes induced by PFOA in rodents can occur by a mechanism that is independent of PPAR-alpha activation. The lack of liver enlargement or tumor response in PPAR-alpha null mice exposed to Wy-14,643 for 11 months has been cited frequently as evidence that liver cancer induction by peroxisome proliferators is mediated by PPAR-alpha activation (Peters *et al.*, 1997). The study of Yang *et al.* (2002) needs to be replicated, but appears to suggest that results with Wy-14,643 in PPAR-alpha null mice do not predict all of the potential liver effects of PFOA.

Further, while not diminishing the conclusion that a PPAR-alpha MOA is operative in the rodent liver carcinogenesis induced by PFOA, about three quarters of the Panel members expressed concern over the as yet incomplete understanding of the role of Kupffer cells in the carcinogenic process. PPAR-alpha independent stimulation of hepatocyte growth factor production in Kupffer cells appear to be essential to the mechanism of hepatocyte replicative DNA synthesis, suppression of apoptosis, and liver tumor development by peroxisome proliferators. Until the interplay between PPAR-alpha agonism and Kupffer cell activation is characterized, negative results from the PPAR-alpha null mouse may not be relevant to the human situation in which Kupffer cells and hepatocellular PPAR-alpha are present and can be activated.

The remaining members of the Panel believed that the finding of increased liver weights produced by PFOA in PPAR-alpha knockout mice, as noted for Issue 1, were not significant enough to undermine the PPAR-alpha agonism MOA, nor did they consider the absence of information about Kupffer cell activation to be relevant to a PPAR-alpha agonism MOA for PFOA-induced rodent liver tumors.

LCTs, PACTs, and mammary neoplasms.

Panel members did not consider the consolidation of liver tumors, LCTs, and PACTs into a triad MOA to be justified. They believed that available evidence is inadequate to support a

PPAR-alpha agonist MOA for the induction of LCTs and PACTs (Klaunig *et al.*, 2003), and, at present, available data are insufficient to characterize the MOA for PFOA-induced LCTs and PACTs. As such, a specific MOA needs to be worked out for each tumor type. In addition, about three quarters of the Panel members felt that the appropriate comparison for mammary neoplasms was to concurrent not to historical controls, and in that view subscribe to the interpretation that PFOA does increase mammary gland neoplasms, and no MOA data are available for the mammary tumor response. As discussed in EPA's Cancer Guidelines, in the absence of sufficient data to establish a MOA, the animal tumor responses are presumed to be relevant to humans.

The remaining Panel members believed, in contrast to the above view, that the comparison of PFOA-induced mammary tumor levels to historical controls was valid, and thus deemed the evidence for mammary neoplasms to be insufficient to demonstrate such tumors in response to PFOA. This served to support the view of these members that PPAR-alpha agonism represented the sole MOA for PFOA-induced rodent liver tumors.

Application of cancer descriptors

The meaning of terms such as "suggestive evidence of carcinogenic potential" or "likely to be carcinogenic to humans" may differ among some in the general public and the EPA because of differences in perception and intent. Hence, EPA recommends a weight-of-evidence narrative that explains the complexity of issues influencing an agent's carcinogenic potential in humans. Descriptors are applied to provide consistency across agents that are evaluated for their carcinogenic potential. In developing their cancer risk assessment guidelines (US EPA 1999, 2003), EPA has not provided definitive criteria for choosing a descriptor; however, examples of the types of evidence that would be covered by a descriptor are listed. EPA also cautions that terms such as "likely," when used as a weight-of-evidence descriptor, do not correspond to a quantifiable probability.

About three quarters of the Panel members shared the view that while human cancer data on PFOA are inadequate to support a definitive conclusion of the presence or absence of a causal association, the animal data are much stronger than the examples summarized in the EPA's Cancer Guidelines under the descriptor "suggestive evidence of carcinogenic potential." The descriptor "suggestive" is typically applied to agents that show a marginal increase in tumors only in a single animal study or a slight increase in a tumor response at a site with a high background rate. The animal data for PFOA are consistent with the examples listed by EPA under the descriptor "likely to be carcinogenic to humans" applied to agents that tested positive in more than one species, sex, strain, site, or exposure route, with or without evidence of carcinogenicity in humans; or a positive study that indicates a highly significant result and where the response is assumed to be relevant to humans. These members concluded, as described above, that data from two separate feeding studies demonstrate that PFOA is a multi-site carcinogen in rats. Significant increases in tumor incidence and dose-response trends were observed in male and female rats. Some of the tumor responses were observed at sites with low background rates; the incidence of PACTs and LCTs in control rats was 0% at both sites. Because available data are insufficient to characterize the MOA for PFOA-induced LCTS, PACTs, or mammary tumors, the responses at these sites are presumed to be relevant to humans. Uncertainties also still exist for the MOA(s) for liver tumors induced by PFOA.

While opting for the descriptor "likely to be carcinogenic to humans" these Panel members were not willing to state an associated probability value for PFOA-induced carcinogenicity; nor do the EPA guidelines require a quantifiable probability. This group also encouraged a cancer risk assessment for each of the PFOA-induced tumors where data permit. The risk characterization narrative should address the state of knowledge and uncertainties in the MOA for each tumor site and a range of approaches should be considered in the cancer dose-response assessment.

The remaining one quarter of the Panel members did not find the weight of evidence strong enough to exceed the descriptor "suggestive". These Panel members agreed with the EPA's risk assessment which proposed that the PFOA cancer data may be best described as providing "*suggestive evidence of carcinogenicty, but not sufficient to assess human carcinogenic potential*" This view was based upon their conclusions that: 1) a mechanism for formation of liver tumors in rats considered not relevant to humans, 2) liver cell proliferation was absent in monkeys, 3) a strong association between PFOA exposure and tumors was not demonstrated in human studies, 4) the belief that the testicular and pancreatic acinar tumors in rats were probably not relevant to humans, and 5) the view that mammary tumors reported in female rats are equivocal. Further these panel members also believed that the ***sole*** MOA for the rodent-induced liver tumors was through PPAR-alpha agonism which they believe was not relevant to humans.

Issue 3: Selection of Endpoints

Question 3. *Please comment on the selection of these toxicity endpoints for the risk assessment.*

The Panel generally agreed with the Agency approach of considering multiple endpoints and developing multiple margin of exposure (MOE) values at this stage in the assessment of potential human health effects associated with PFOA. With regard to the selection of endpoints, the initial overall philosophy should be one of inclusivity. That is, endpoints should be considered unless evidence for an effect by PFOA is equivocal or the dose associated with the effect is sufficiently high that other effects will clearly be of greater concern. The reason for being inclusive is not to generate an exhaustive catalog of PFOA effects, but rather to insure that sensitive effects (i.e., effects occurring at relatively low doses) are not overlooked or prematurely excluded from the assessment.

The Panel agreed with inclusion of all of the endpoints in the current draft of the risk assessment. None were recommended for deletion. However, caveats regarding the use of organ and body weights as endpoints were offered. Organ and body weights are often among the least sensitive endpoints for chemicals that exert specific effects on physiological or developmental systems. Nevertheless, in the absence of information with which to select more specific endpoints (e.g., biochemical or histological changes), body and organ weight changes are likely to be indicative of toxicity.

Many members of the Panel also believed that additional endpoints should be included in the risk assessment, although recognizing that this may not be possible for some endpoints

because of the absence of sufficient information:

- Based on discussion in response to Question 2, PFOA has the potential to produce carcinogenic effects in humans and therefore additional cancer endpoints (liver, testicular, pancreatic acinar, and mammary) should be included in the risk assessment.
- Liver histopathology, other than cancer, should be included as an endpoint since it could not be concluded with confidence that all liver effects are mediated through PPAR-alpha agonism (see response to Question 1), and therefore liver histopathology from PFOA may be relevant to humans. The Panel recognized that interpretation of some liver changes as adverse effects may not always be apparent (e.g., liver enlargement with no other pathology), and this should be discussed in the risk assessment.
- Immunotoxicity should be considered as an endpoint addressed quantitatively in the risk assessment. The Panel recognizes that in order to be incorporated into the risk assessment, immunotoxicity data will need to be derived in rats, or approaches developed for the estimation of serum PFOA concentrations in mice.
- Consideration should be given to addition of endpoints related to lipid metabolism (see comments under response to Issue 3, question #5).

In the view of a few Panel members who believed that data for PFOA was consistent with the descriptor "suggestive" rather than "likely" to be carcinogenic in humans, the cancer endpoints noted above were not recommended for inclusion in the risk assessment as they were deemed not to be independent PFOA effects.

The Panel agreed that additional research on PFOA was needed, and encouraged studies in the areas noted below; the Panel also encouraged exploration of methods to identify critical targets for PFOA beyond a PPAR-alpha MOA:

- Other than ataxia, no data on neurotoxicity endpoints for PFOA are available. Neurotoxicity endpoints, including behavioral measures, should be added to the risk assessment. PPAR-alpha receptors, as well as other PPAR receptors, are found in both neurons and glia, and are found in multiple brain regions (frontal cortex, basal ganglia, reticular formation). It has been proposed that, in addition to their roles common to other tissues, these receptors in brain may have specific functions in the regulation of genes involved in neurotransmission (Moreno *et al.*, 2004). This would further suggest their importance in behavioral function.
- The two-generation rat study (Butenhoff *et al.*, 2004) involved both perinatal PFOA exposure and direct PFOA dosing of the F1 offspring beginning at weaning. The Panel recognized that this approach is consistent with U.S. EPA guidance regarding developmental studies. However, consideration should be given to using developmental endpoints in F1 generation animals prior to initiation of direct dosing so that potential effects associated with perinatal exposure can be more clearly identified.
- Current data suggest that PFOA might produce hormonal effects that would be important to consider, but in most cases the significance of the observations are unclear. For example, in a 26-week study of PFOA administration to cynomolgus monkeys, serum TSH was slightly but significantly elevated in all treatment groups on the final day of the

experiment, and serum thyroxin was slightly but significantly reduced (Butenhoff, 2002). It is not clear whether these observations are physiologically meaningful or whether they were strictly dependent upon treatment per se, since hormone levels appeared to change in the control animals during the course of the experiment as well. The analysis of Butenhoff data did not include a repeated measures ANOVA, so interactions were never pursued. Even that, however, would not have revealed why hormone levels changed over the course of the experiment in control animals. One study reported PFOA-induced decreases in pituitary weight in the F1 generation female rats, but the functional significance of this observation is unclear. Overall, the Panel thought that Margins of Exposure (MOEs) should not be calculated for hormonal endpoints at this time, but that additional research to clarify the hormonal effects of PFOA should be encouraged.

- Adult male rats exhibited a much slower elimination of the ammonium salt of PFOA, i.e., ammonium perfluorooctanoate (APFO or C8), than did females. This appears to be due to gonadal hormones inasmuch as castration increased APFO elimination and testosterone replacement returned the elimination rate toward normal levels. Importantly, renal elimination was blocked by probenecid, a selective antagonist of organic anion transporters (OATPs) (Shitara, 2004). Thus, gender differences in renal OATPs may account for the gender differences in renal clearance of APFO. Likewise, the slower clearance of APFO in males may account for the observation that lower doses of APFO produced adverse effects in males compared to females. For example, the NOAEL for APFO in a 13-week study of male CD rats was 0.56 mg/kg-day whereas females exhibited a NOAEL of 22.4 mg/kg-day. These results suggest that specific organs (e.g., liver, kidney, and perhaps adrenals) are targets of APFO because of the pattern of expression of the OATPs that transport it across the cells (OATP1-4 in rat). Research to identify the relationship between OATP and PFOA toxicity may offer insight into the most important targets for PFOA effects and the best endpoints for evaluation.

Question 4. *Given the available data to date, please comment on the most appropriate lifestage/gender/species for assessing human risk.*

In general, there was consensus that at this stage in the risk assessment process, no lifestage/gender/species should be excluded from consideration in predicting human risk. Moreover, absence of information identifying a "critical period" in development during which PFOA may exert adverse effects on development requires inclusion of all life stages, including fetal development. Biomonitoring data indicate children and adults alike exhibit measurable levels of PFOA in serum, and the half-life of PFOA appears to be around 4 years. Therefore, there is no reason to exclude any developmental period from examination. Finally, the inclusion of data on internal dose is an important element of the dataset for PFOA which should enlighten concerns about the use of female rats, discussed below.

Two considerations arose in evaluating the current dataset for use in assessing human risk. In general, the EPA provides a margin of safety by using exposure values that produce the lowest margin of exposure based on observed effect levels, usually NOAELS or LOAELs, including those in animal models. There was general agreement that the most appropriate criterion for assessing human risk is one that produces the lowest margin of exposure (e.g., 90^{th}, 95^{th}, or 99^{th} percentile) based on a LOAEL in animal models. The second consideration related

to the appropriateness of the non-human primate as a model, generally considered to be most comparable to humans.

With respect to the first, the emphasis is on having data based on the internal dose relationships (i.e., serum PFOA levels) in some of the animal studies so that interspecies differences in metabolism and clearance are taken into account. In addition, these data also allow using both males and females despite a dramatic difference in clearance rate. Also, considering empirical measures of exposures in children and adults, this view emphasizes a concern that both developmental and adult endpoints be captured, and these endpoints have not been evaluated in non-human primates. Therefore, the findings from adult male rats including the 13 week study by Goldenthal (1978) in which liver weight was significantly increased, and the F1 males in the two-generation reproductive toxicity study (Butenhoff *et al.*, 2004) in which body weight was reduced should be considered in further analysis of human health risk.

The second view emphasizes the biological similarities between nonhuman primates and humans for risk assessment. This is particularly important in the case of PFOA because there are a number of issues with a rodent model for PFOA exposure; e.g., sexual dimorphism with respect to elimination of PFOA, and differences in sensitivity to PPAR-alpha signaling between rat and human. However, monkeys also exhibit a different half-life of PFOA than do humans, and information about the potential toxicity of PFOA on non-human primates are derived primarily from adults.

Question 5. *Please comment on the appropriateness of the available animal models. Please comment on whether additional animal models should be investigated, and if so, what information would better enable us to ascertain potential human risks.*

The available animal models are useful, but all are considered uncertain matches for humans with respect to PFOA toxicity. Thus, most Panel members supported continued use of multiple animal models and the need for additional models. As previously noted, some responses to PFOA may occur via modes of action not related to PPAR-alpha agonism. Without knowing how these PPAR-alpha independent effects are mediated, the ability to identify the specific animal models that would be most useful is limited. Some Panel members suggested the development and use of additional animal models without PPAR-alpha, such as transgenic or siRNA rats. Use of these animal models would be of assistance for more clearly identifying PPAR-alpha independent effects of PFOA.

Overall, the Panel thought that results obtained in models using female rats were informative because they currently provide the only indication of potential effects on endpoints specific to females (e.g., reproduction and developmental effects, mammary tumors). However, some concerns were noted regarding the difference in toxicokinetics of PFOA in female versus male rats and monkeys, with females exhibiting more rapid excretion.

As part of a discussion of additional sources of information and animal models to help ascertain potential human risks, the Panel considered observations from studies in humans. The following specific observations in regard to inclusion of the epidemiologic data as informative regarding endpoints were shared by most Panel members:

- The PFOA Draft Risk Assessment did not use the occupational biomonitoring data because "data are not available for specific occupational exposures." The Panel points out that neither are data available for "specific environmental exposures." The further claim that information on "critical factors" like gender, sampling methods and occupation are not available for the worker populations does not seem relevant. Gender differences are not considered in the PFOA Draft Risk Assessment document's MOE calculations (combined male and female values are used) because, unlike in rats, there are no apparent gender differences in PFOA elimination in humans, at least in the sparse published data available at the time of this review.
- In the PFOA Draft Risk Assessment document, limitations of epidemiological studies are emphasized, while some associations (cerebrovascular disease, triglycerides and cholesterol) are deemed less convincing, based on small numbers or inconsistencies in the results. It is undeniable that the epidemiology studies, like the toxicological ones, have some limitations, not the least of which are uncertainties regarding exposure. However, there is little doubt that these workers are more highly exposed than the general population. A special strength of epidemiological studies is that no cross-species extrapolation is needed; humans are the model. It is also true that there may be multiple exposures in the occupational studies, but this fact alone cannot disqualify them without simultaneously disqualifying virtually all epidemiological studies, which doesn't seem appropriate. If the question addressed by an MOE analysis is "how far" are actual human exposures from exposures that are associated with a health effect, any health effect in the epidemiological studies imply the answer is "zero distance," *regardless of the actual serum values.*

While conceding the small numbers and short follow-up in the available epidemiological studies make the positive results less than compelling, they are not, conversely, reassuring. The evidence showing increases in cholesterol and triglyceride values in worker cohorts suggest a possibility of increased risk of cerebrovascular disease mortality.

In responding to charge question #3, therefore, many Panel members shared the view that human cancer and alterations in lipid metabolism data be included in the relevant endpoints for consideration. This implies that the rich data base of occupational exposures be added to the occupational biomonitoring data to be considered. They are not now included in the PFOA Draft Risk Assessment document because the worker epidemiological studies were not considered suitable for quantitative risk assessment.

A contrasting conclusion reached by some Panel members was that the peer-reviewed human data on health effects of PFOA were equivocal, thus there was not consensus that endpoints suggested by some epidemiologic studies should be used as endpoints in the risk assessment.

Issue 4: Risk Assessment Approach

Issue 4a: Pharmacokinetic Modeling and Use of AUC as a Measure of Internal Dose

Question 6. *Please comment on use of the one-compartment pharmacokinetic model.*

The purpose of developing a mathematical model to fit the serum PFOA time course data from the single dose rat oral dosing studies in the PFOA Draft Risk Assessment document was to estimate the AUC and C_{max} values during the longer term toxicology studies with daily dosing. The internal dose metrics calculated with this model were then compared with human serum concentrations to establish an MOE. The equations used to describe these data sets are the same as those usually employed in one-compartment models for uptake and elimination and were referred to throughout the draft document as a one-compartment model.

However, the Panel was concerned that using the "one-compartment" nomenclature without caveats and qualifications will give readers of the Draft Risk Assessment Document the impression that PFOA pharmacokinetics follow a one-compartment description when in fact they are much more complex. In a one-compartment model, the chemical distributes evenly throughout a volume of distribution that is itself in rapid equilibrium with blood. Elimination kinetics are first-order and do not change with dose level or with time. However, the data indicate that it is clearly inappropriate to describe the observed kinetics of PFOA in rats or monkeys as following a simple one-compartment model. The relatively complex pharmacokinetic behavior of PFOA is reflected in several of the pharmacokinetic data sets. For example, elimination from blood after iv dosing and tissue distribution kinetics after oral dosing are poorly characterized by the one-compartment model. In both rats and monkeys, blood levels are related in a complex manner to dosage and the duration of treatment.

Although the one-compartment model is not appropriate, the empirical model used in the document and referred to as a 'one compartment model' is adequate for predicting blood levels resulting from repeated dosing. However, the document needs to make it clear that the fitting procedure is specific to this limited data set and useful for this one application. It is strongly recommended that the terminology 'one-compartment' model should be stricken from the document unless carefully qualified.

Question 7. *Please comment on the use of the AUC as a measure of internal dose for rats and humans for calculation of the MOE.*

Calculating the 'blood' AUC (as a measure of average daily concentration of PFOA) is an appropriate method of estimating the internal dose, although it is not the only possible measure. In the absence of clear understanding of modes of action (MOA), it is also possible that the C_{max}, the integrated dose above a minimum concentration, or some other quantity may be a more plausible measure of internal dose. For example, if the MOA was receptor-based, as might be expected for interactions of PFOA with PPAR or other receptor proteins, one of these other measures of dose might also be appropriate. These alternatives include receptor occupancy or the concentration above some minimum concentration (C_{min}) where C_{min} is the concentration required to initiate activation of the receptor-mediated signaling pathway. In this latter case, the MOE would be based on the integral of (C_t-C_{min}) rather than just the integral of concentration (C_t).

In light of these other possible internal dose measures, the EPA document would be strengthened if a clear rationale for the choice of the AUC were included. Since the inclusion of this explanation may involve a detailed discussion of toxicokinetic and toxicodynamic issues,

such a discussion would best be included as an appendix. While the report does provide an example of how the MOE differs when based on the C_{max} as compared to the AUC, it would be helpful if the impact on the magnitude of the MOE of using each of these other internal dose measures was explored in more detail. Calculations of MOEs based on these other measures would provide a better idea of the extent of possible variability introduced by different internal dose measures that may reflect a variety of possible MOAs.

When estimating an AUC, it is important to note the sample that is being analyzed in the various studies. AUCs can be calculated for serum, plasma or whole blood. These are very different biological matrices. The document should clearly specify the biological media measured in each study in which AUCs are reported.

Another issue to be considered is that the analyses of serum time course in the document are based on the assumption that the analyte in serum is in the same form and the proportion of free compound in blood is constant throughout the period of observation. This assumption does not always hold true. For example, with some siloxanes, the blood concentrations during and after inhalation exposure are primarily free siloxanes that are available for exhalation and metabolism. After a period of time in the body, the siloxanes in blood appear to reside in the lipid pool within the blood and although they are easily analyzed are no longer available for these other clearance processes (see Andersen *et al.*, 2001; Reddy *et al.*, 2003). A situation where the PFOA in blood at much longer times after exposure is in a distinctly different biological pool would lead to difficulties in comparing rat AUC and human AUC values to obtain a MOE. Interspecies differences in PFOA free fraction in plasma may also complicate the comparison of AUC values to obtain a MOE.

The direct use of internal measures of dose by US EPA in this document represents a promising and relatively innovative approach for risk assessments of environmental compounds compared to the more usual practice based on comparing daily dose rates by various routes of administration. This new approach reduces the need to include uncertainties introduced by the use of administered or ambient doses as measures of exposure. This type of risk assessment methodology is likely to become much more widespread due to advances in analytical chemistry and the rapid expansion of human biomonitoring activities throughout the world. Because this risk assessment is likely to serve as a prototype for future tissue-dose based risk assessments, some important issues raised by this tissue-dose based approach need to be more fully considered and adequately contrasted with the more common assessments based on comparisons of administered doses.

To address these issues, the EPA should be encouraged to develop documentation explaining their rationale guiding the use of these tissue-dose based risk assessment approaches. Such documentation should compare current methods based on daily intakes with these alternative, 'tissue-based' approaches to more explicitly address the risk characterization issues that arise in moving to this new approach. Such a document might include discussion of (1) the choices of tissue dose measures based on serum concentrations and the risk implications of each choice; (2) the impacts of utilizing direct measures of tissue dose on the magnitudes of interspecies and interindividual uncertainty factors; (3) the implications of different metrics for characterizing distributions of human tissue dose measures on estimates of MOEs; and (4) the

importance of routine analysis of appropriate blood concentrations; e.g., serum, plasma, etc. in providing the information for most appropriately applying the tissue dose approach.

Issue 4b: Cross Species Extrapolation

Question 8. *Please comment on the need to use or modify the default value of 10 for cross species extrapolation given the pharmacokinetic analysis.*

The internal dose analysis used in this document is considered by the Panel to be a significant step toward reducing uncertainty related to cross species extrapolation. Although reduced, however, cross species toxicokinetic uncertainty is not eliminated. Sources of uncertainty remain, including the lack of information about the measured internal dose that best predicts adverse effect in human and other species, and the bias inherent in measurement/modeling error. While it is difficult to assign a quantitative value to the magnitude of this uncertainty reduction, it can be stated that the toxicokinetic uncertainty value for PFOA would fall within the range of one to three, based on the customary scale of a value of 3 for each aspect of cross species extrapolation, pharmacokinetics and pharmacodynamics. Pharmacodynamics aspects of PFOA cross species scaling are not addressed in a sufficient manner to alleviate the application of some type of uncertainty factor/s (addressing toxicodynamic equivalence across species). The assumption that PFOA serum levels are at steady state in children 2-12 years of age has not been tested and may not be valid. The additional complexity of multiple C-8 environmental exposures in humans versus animal experiments involving exposures to PFOA specifically, further clouds the overall uncertainty analysis.

While the pharmacokinetic modeling that is presented in the PFOA risk assessment is useful, a more comprehensive way to account for biological processes that determine internal dose is with the development of a physiologically based toxicokinetic model. The Panel encourages EPA to continue to develop toxicokinetic models as they can improve dose-response assessment by revealing and describing nonlinear relationships between applied and internal dose.

A discussion of confidence should always accompany the presentation of model results and include consideration of model validation and sensitivity analysis, stressing the predictive performance of the model. Toxicokinetic modeling results may be presented as the preferred method of estimating equivalent human doses or in parallel with default procedures (see Section 3.1.3), depending on the confidence in the modeling.

Standard cross-species scaling procedures are available when the data are not sufficient to support a toxicokinetic model or when the purpose of the assessment does not warrant developing one. The aim is to define dose levels for humans and animals that are expected to produce the same degree of effect (U.S. EPA, 1992b), taking into account differences in scale between test animals and humans in size and in lifespan. It is useful to recognize two components of this equivalence: toxicokinetic equivalence, which determines administered doses in animals, and humans that yield equal tissue doses, and toxicodynamic equivalence, which determines tissue doses in animals and humans that yield equal lifetime risks (U.S. EPA, 1992b).

It is equally important to note that pharmacodynamics aspects of PFOA cross species scaling are not addressed in a sufficient manner to alleviate the application of some type of uncertainty factor/s (addressing toxicodynamic equivalence across species). These factors may be different for each species extrapolated. By the language used in the U.S. EPA Cancer Guidelines, it seems evident that standard default values were never intended to act as complex scaling factors when internal doses in human serum are compared to animal internal doses across multiple pathways, genders, steady-state serum levels with long human half-lives and/or different life stages.

In the case of PFOA the strong reliance on LOAEL-driven MOE calculations instead of more appropriate Bench Mark Dose methodologies, and the absence of probabilistic approaches to assessing human exposure and risk, was considered by most Panel members as another source of dynamic uncertainty.

The use of an uncertainty factor/s based on data variability may be an alternative to the traditional scaling factors given the kinetics analysis strength and in light of the larger concerns of overall uncertainties related to dynamic analysis (as reflected in the MOE approach). This may prove more productive when comparing relatively robust toxicokinetic dose response models involving serum concentrations and/or their surrogates.

In conclusion, whereas toxicokinetic uncertainty is possibly reduced in this analysis, care must be exercised in the estimation of the overall cross species uncertainty, which further dynamic analyses may show falls below or above 10.

Issue 4c: Human Biomonitoring Data

Question 9. *Please comment on the adequacy of the human exposure data for use in calculating a MOE.*

Full agreement was not reached by Panel members with respect to the utility of the human biomonitoring data for the calculation of the MOE. Most Panel members expressed the view that the human exposure information should be utilized in these calculations while a few Panel members believed that these data were equivocal and thus not appropriate for the MOE calculations.

Populations used for MOE calculations

In addition to the occupational biomonitoring data, the PFOA Draft Risk Assessment document described three separate study populations from the United States with available individual serum PFOA levels. One consists of samples from six American Red Cross blood banks, another from a study of Streptococcal A infection in children, and a third from elderly volunteers from Seattle who participated in a study of cognitive function. Only the first two study populations were used in calculating the MOE for the risk assessment.

A question was raised about reliance on the female blood bank donor population for calculating prenatal MOEs, because the influence of pregnancy on serum PFOA levels is not known. Likewise, use

of the samples obtained from the children for the age span of 2-12 years for the postweaning period MOE may not be appropriate because the assumption of steady state used in the MOE analyses may not be valid for children. Half-life issues in humans, especially when considering the impact of age at exposure (or the critical windows of exposure model), contribute to the questions about adequacy of using these samples (Pryor *et al.*, 2000; Selevan *et al.*, 2000; Sweeney *et al.*, 2001). Thus, there are a variety of possible problems with using these data to represent the general population, but the Panel agreed that they were likely to be reasonably representative and are better than data often available for exercises of this nature.

It was suggested that biomonitoring data in highly exposed groups (occupational and environmental) be included in the MOE analyses. It was noted that the existence, size and levels of exposures of populations which may differ from those studied has yet to be fully determined. Until this has been determined, it is not clear what percent of the general population is covered by the MOEs that have been calculated. Thus, the appropriateness of relying solely on the blood bank and pediatric samples for MOE calculations depends strongly on the purpose of the MOE exercise, i.e., whether it is to assess the likelihood that any people could be suffering health effects from PFOA or only the "general population." If the latter case, the biomonitoring data that were used may be appropriate, but the sizes of more highly exposed populations remains unknown and this should be acknowledged.

A few members of the Panel held the view that the human data were equivocal, based on the likely multiplicity of exposures of occupational groups, and thus should not be included in the draft PFOA risk assessment for MOE calculations for the general population.

Depiction of the biomonitoring data

The tables and summary statistics that were used in the draft PFOA risk assessment are somewhat uninformative and unsatisfactory. It is difficult to determine the distribution of population exposures from these given the method of data presentation. A preferable approach would be to use a non-parametric data-driven method to display the data (including the occupational data), using, for example, some density estimation procedure or smoother. Inclusion of the worker data in these displays would allow a clearer understanding of the relationships. Even side-by-side box plots would have been preferable to what was provided. This requires having access to the raw data, however. Because such a request is easy to satisfy, the Panel recommends that EPA provide more informative displays of the biomonitoring data.

Appropriate summary measures for MOE calculations

At least three summary statistics are mentioned in the Draft, the geometric mean, the arithmetic mean, and the 90^{th} percentile.

The rationale for the use of "means" should be explained, especially the use of the geometric means which seems the least satisfactory, since it is about 25% lower than the arithmetic mean in these data. Use of a geometric mean for population inference (to transform a lognormal to a normal distribution, for example) might be justified, but not for the purpose of calculating an MOE. Moreover, the distribution does not even seem to be lognormal, as judged by the Shapiro-Wilk test. The idea that a few censored data points are responsible for failing this test seems highly unlikely, and could have been accounted for in the test itself.

Means of any kind don't seem appropriate for a ubiquitous exposure. Of the three choices, the 90th percentile seems the most appropriate in that case. At least one Panel member wondered why some even higher percentile, say 95th or even a maximum value wouldn't be better. The maximum value in any of the samples is still an underestimate of the maximum value in the population. Even the upper 99.99th percentile represents 30,000 people in the US.

In summary, the Panel finds that:

- Use of the blood donor and pediatric biomonitoring data may be acceptable if the purpose is to assess whether there is a potential health effect to the "general" population, although there is some question as to the size of other non-occupational populations that might be more highly exposed and the assumption that PFOA serum levels are at steady state may not be valid for fetuses, neonates or children;
- Most Panel members believed that occupational biomonitoring data should be included in the MOE calculations, especially regarding additional endpoints such as alterations in lipid metabolism;
- A few members did not favor this inclusion based on the equivocal findings;
- The biomonitoring data should be presented in a more informative manner, for example, through side-by-side box plots or some other method that would better depict the range of values and distributions; and
- Thought should be given to what appropriate summary statistic for the biomonitoring datasets used in MOE calculations should be. Some panelists believe that 90th percentiles or higher, perhaps even maximum values might be most appropriate. In any event, justification for use of the chosen summary measure should be made and related to the explicit objective of the MOE analysis.

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Provisional Health Advisories for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

1. Introduction

EPA recently concluded limited testing of agricultural sites in Alabama where sewage sludge was applied from a local wastewater treatment plant that receives wastewater from numerous industrial sources, including facilities that manufacture and use perfluorooctanoic acid (PFOA) and other perfluorinated chemicals (PFCs). The results from this limited testing indicated elevated levels of PFCs in the sludge and the soil that received the sludge. As a result, EPA has conducted sampling of public drinking water. The levels of PFOA and perfluorooctane sulfonate (PFOS) recently analyzed in community water systems in Lawrence and Morgan Counties are all lower than 0.04 ppb. Based on its current understanding, EPA believes these levels are not of concern and residents may rely upon public water systems. EPA will soon begin groundwater and surface water sampling to determine if PFOA or PFOS has migrated into any private drinking water supplies and ponds in the affected area.

The Office of Water (OW) has developed Provisional Health Advisory values[1] for PFOA and PFOS to assess potential risk from exposure to these chemicals through drinking water. Other PFCs have been found at this site. However, information on the toxicity of PFCs other than PFOS and PFOA is limited and therefore no attempt is made at the present time to develop Provisional Health Advisory values for these other PFCs.

2. Summary of Data for PFOA

Epidemiological studies of exposure to PFOA and adverse health outcomes in humans are inconclusive at present.

Several animal toxicological studies have been conducted using PFOA. These include subchronic, developmental/reproductive, and chronic toxicity/carcinogenicity studies in several animal species, in both sexes. An evaluation of these studies was conducted by the European Food Safety Authority (EFSA) and no-observed-adverse-effect level (NOAEL), lowest-observed-adverse-effect level (LOAEL), and critical endpoints identified (EFSA, 2008).

Among these studies, a recent and well conducted developmental toxicity study in mice was selected by the Office of Water (OW) as the critical study for the derivation of the

[1] Provisional Health Advisory values are developed to provide information in response to an urgent or rapidly developing situation. They reflect reasonable, health-based hazard concentrations above which action should be taken to reduce exposure to unregulated contaminants in drinking water. They will be updated as additional information becomes available and can be evaluated.

Provisional Health Advisory for PFOA (Lau et al., 2006). In this study, CD-1 mice were given the ammonium salt of PFOA by oral gavage from gestational day (GD) 1 to 17 at doses of 0, 1, 3, 5, 10, 20 or 40 mg/kg/day. Significant increase in the incidence of full-litter resorption occurred at 5 mg/kg/day and higher doses. Weight gain in dams that carried pregnancy to term was significantly lower in the 20-mg/kg/day group. At GD 18, some dams were sacrificed for maternal and fetal examinations (group A), and the rest were treated once more with PFOA and allowed to give birth (group B). Postnatal survival, growth, and development of the offspring were monitored. PFOA induced enlarged liver in group A dams at all dosages, but did not alter the number of implantations. The percent of live fetuses was lower only in the 20-mg/kg/day group (74 vs. 94% in controls), and fetal weight was also significantly lower in this group. However, no significant increase in malformations was noted in any treatment group. The incidence of live birth in group B mice was significantly lowered by PFOA: ca. 70% for the 10- and 20-mg/kg/day groups compared to 96% for controls. Postnatal survival was severely compromised at 10 or 20 mg/kg/day, and moderately so at 5 mg/kg/day. Dose-dependent growth deficits were detected in all PFOA-treated litters except the 1-mg/kg/day group. Significant delays in eye-opening (up to 2–3 days) were noted at 5 mg/kg/day and higher dosages. Accelerated sexual maturation was observed in male offspring, but not in females. These data indicate maternal and developmental toxicity of PFOA in the mouse, leading to early pregnancy loss, compromised postnatal survival, delays in general growth and development, and sex-specific alterations in pubertal maturation (Lau et al., 2006).

Toxicity endpoints identified in the Lau et al. (2006) study included a number of developmental landmarks: neonatal eye opening, neonatal survival and body weight at weaning, reduced phalangeal ossification at term, live fetus weight at term, maternal liver weight at term, and maternal weight gains during pregnancy. The most sensitive endpoint was for increased maternal liver weight at term. This endpoint for liver effects was identified in a number of other studies described in EFSA (2008).

Benchmark dose (BMD_{10}) and the 95% lower bound on the BMD ($BMDL_{10}$) were calculated for these toxicity endpoints by the EFSA on the basis of raw data provided by the principal author (Lau, personal communication, November 18, 2008). The lowest $BMDL_{10}$ in the Lau et al. (2006) study was 0.46 mg/kg/day for increase in maternal liver weight at term. This value was used as the point of departure for the derivation of the Provisional Health Advisory value for PFOA. It should be noted that liver effects were also reported in studies in rats and monkeys. $BMDL_{10}$ values for increased liver weight in studies in mice and rats ranged from 0.29 to 0.74 mg/kg/day (EFSA, 2008). The $BMDL_{10}$ for Lau et al. (2006) was in the middle of this range.

3. Summary of Data for PFOS

Epidemiological studies of exposure to PFOS and adverse health outcomes in humans are inconclusive at present.

Several animal toxicological studies have been conducted with PFOS. These include subchronic, developmental/reproductive, and chronic toxicity/carcinogenicity studies in several animal species, in both sexes. An evaluation of these studies was conducted by the EFSA (2008) and NOAEL, LOAEL and critical endpoints identified.

The subchronic toxicity study in Cynomolgus monkeys (Seacat et al., 2002) was selected by the OW as the critical study for the derivation of the Provisional Health Advisory value for PFOS. In the study by Seacat et al. (2002), groups of male and female monkeys received orally potassium PFOS at doses of 0, 0.03, 0.15 or 0.75 mg/kg/day for 183 days. Compound-related mortality in 2 of 6 male monkeys, decreased body weights, increased liver weights, lowered serum total cholesterol, lowered triiodothyronine (T_3) concentration, and lowered estradiol levels were seen at the highest dose tested. At 0.15 mg/kg/day, increased levels of thyroid-stimulating hormone (TSH) in males, reduced total T_3 levels in males and females, and reduced levels of high-density lipoproteins (HDL) in females were seen. A NOAEL of 0.03 mg/kg/day was identified in this study.

4. Calculation of Provisional Health Advisories for PFOA and PFOS

The general equation for the derivation of a Provisional Health Advisory is:

$$\frac{(\text{NOAEL or } BMDL_{10}) \times BW \times RSC}{UF \times \text{Extrapolation Factor} \times \text{Water intake}}$$

Where BW = body weight; RSC = relative source contribution; UF = uncertainty factors

The OW is using the exposure scenario of a 10-kg child consuming 1 L/day of drinking water to calculate the Provisional Health Advisories for PFOA and PFOS. This population subgroup was used because children, who consume more drinking water on a body weight basis than adults, have a higher exposure on a body weight basis than adults. The selection of children's exposure parameters will help to ensure that this Provisional Health Advisory is protective of sensitive populations potentially exposed. A default relative source contribution (RSC) of 20% was used to allow for exposure from other sources such as food, dust and soil. The relevant period of exposure for the Health Advisory is a short-term exposure. This time period is consistent with the toxicity data used for PFOA and PFOS, both of which rely upon subchronic data. The value should be protective of all population subgroup and lifestages.

Data derived extrapolation factors for toxicokinetics were developed to better approximate internal doses for PFOA and PFOS. This step was deemed important because of the marked differences in retention time among humans and the test species in which toxicological data were collected. Available data for PFOA from female mice indicate a half-life of 17 days and from humans, a half-life of 3.8 years (1387 days). Critically, measures of internal exposure should be used as the basis for interspecies extrapolation; the assessment is somewhat complicated by the lack of area under the curve (AUC) or clearance (CL) data. However, the one-compartment model foundation

is useful to convert half-life data to clearance data, assuming steady-state has been reached (Equation 1).

$$\text{Half-life} = (\ln 2 \text{ or } 0.693) \times \text{Volume of Distribution} / \text{CL} \quad (1)$$

The volume of distribution of 198 $\pm$ 69 ml/kg has been estimated in female monkeys (Butenhoff et al., 2004). Olsen et al. (2007) summarized other findings on PFOS and PFOA as indicating primarily an extracellular distribution volume. Olsen et al. (2007) also cited other reports that these agents were highly bound to plasma proteins in rats, monkeys and humans. Together, these data support using the same volume of distribution for rodents and humans, based on the findings (198 ml/kg) in monkeys.

The mouse half-life of 17 days converts:
CL = (0.693 x 198 ml/kg) / 17 days = 8.07 ml/kg/day

The human half-life of 1387 days converts:
CL = (0.693 x 198 ml/kg) / 1387 days = 0.10 ml/kg/day

Calculating the toxicokinetic portion of the interspecies on the basis of plasma CL would be:

CL animal / CL human = 8.07 ml/kg/day / 0.10 ml/kg/day = 80.7

The total interspecies correction derived from using a 3X for toxicodynamics and 81X for toxicokinetics is 243X.

To calculate the Provisional Health Advisory for PFOA, a default intraspecies uncertainty factor of 10 was applied to the $BMDL_{10}$ of 0.46 mg/kg/day to account for variation in susceptibility within the human population. A default uncertainty factor of 3 was used for toxicodynamic differences between animals and humans.

The following Provisional Health Advisory is obtained:

$$\text{PFOA Provisional Health Advisory} = \frac{0.46 \times 1000 \times 10 \times 0.2}{10 \times 3 \times 81 \times 1} = 0.4 \text{ µg/L}$$

Similarly, a data-derived extrapolation factor was developed for PFOS. The half-lives of PFOS in humans and in male and female monkeys were estimated by Lau et al., (2007) to be 5.4 years and 150 days, respectively.

The monkey half-life of 150 days converts:
CL = (0.693 x 198 ml/kg) / 150 days = 0.915 ml/kg/day

The human half-life of 1971 days converts:
CL = (0.693 x 198 ml/kg) / 1971 days = 0.07 ml/kg/day

Calculating the toxicokinetic portion of the interspecies on the basis of plasma clearance would be:

CL animal / CL human = 0.915 ml/kg/day / 0.07 ml/kg/day = 13.1

The total interspecies correction derived from using a 3X for toxicodynamics and 13X for toxicokinetics is 39X.

To calculate the Provisional Health Advisory for PFOS, a default intraspecies uncertainty factor of 10 was applied to the NOAEL of 0.03 mg/kg/day to account for variation in susceptibility within the human population. A default uncertainty factor of 3 was used for toxicodynamic differences between animals and humans.

The following value is obtained:

$$\text{PFOS Provisional Health Advisory} = \frac{0.03 \times 1000 \times 10 \times 0.2}{10 \times 3 \times 13 \times 1} = 0.2\ \mu g/L$$

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Long-Chain Perfluorinated Chemicals (PFCs) Action Plan

I. Overview

Long-chain perfluorinated chemicals (PFCs)[1] are found world-wide in the environment, wildlife, and humans. They are bioaccumulative in wildlife and humans, and are persistent in the environment. To date, significant adverse effects have not been found in the general human population; however, significant adverse effects have been identified in laboratory animals and wildlife. Given the long half-life of these chemicals in humans (years), it can reasonably be anticipated that continued exposure could increase body burdens to levels that would result in adverse outcomes.

Since 2000, the Agency has taken various actions to help minimize the potential impact of PFCs on human health and the environment, including the publication of three Significant New Use Rules on perfluoroalkyl sulfonate (PFAS) chemicals and the review of substitutes for long-chain PFCs as part of its review process for new chemicals under EPA's New Chemicals Program. Although such actions are important steps to reducing exposure to these chemicals, EPA continues to be concerned with long-chain PFCs. Consequently, EPA intends to propose actions in 2012 under the Toxic Substances Control Act (TSCA) to address the potential risks from long-chain PFCs.

EPA intends to consider initiating TSCA section 6 rulemaking for managing long-chain PFCs. If EPA can make certain findings with respect to these chemicals (further analysis of the information will be performed as part of TSCA section 6 rulemaking), TSCA section 6 provides authority for EPA to ban or restrict the manufacture (including import), processing, and use of these chemicals. A rule addressing the PFAS sub-category could expand beyond the reach of the SNURs that the Agency has promulgated over the past decade. For example, the rule could address PFAS-containing articles. A rule addressing the perfluoroalkyl carboxylate (PFAC) sub-category could expand the reach of the 2010/15 PFOA Stewardship Program beyond the eight participating companies and further address the concerns for potential PFAC exposure through the use of PFAC-containing articles. EPA will develop more detailed assessments to support the TSCA section 6(a) "presents or will present an unreasonable risk" findings. If these more detailed assessments indicate that a different approach to risk management is appropriate, EPA will consider additional approaches.

Long-chain PFCs are a concern for children's health. Studies in laboratory animals have demonstrated developmental toxicity, including neonatal mortality. Children's exposures are greater than adults due to increased intakes of food, water, and air per pound of body weight, as well as child-specific exposure pathways such as breast milk consumption, mouthing and ingestion of non-food items, and increased contact with the floor. Biomonitoring studies have found PFCs in cord blood and breast milk, and have reported that children have higher levels of

[1] The terms long-chain PFCs, long-chain perfluoroalkyl sulfonate (PFAS), and long-chain perfluoroalkyl carboxylate (PFAC) chemicals in this document refer only to chemicals described in the chemical identity section, including certain polymers that contain perfluorinated moieties. They do not include other PFCs, particularly those having shorter chain lengths.

some PFCs compared to adults. Thus, given the pervasive exposure to PFCs, the persistence of PFCs in the environment, and studies finding deleterious health effects, EPA will examine the potential risks to fetuses and children.

II. Introduction

As part of EPA's efforts to enhance the existing chemicals program under the Toxic Substances Control Act (TSCA)[2], the Agency identified an initial list of widely recognized chemicals, including PFCs, for action plan development based on their presence in human blood; persistent, bioaccumulative, and toxic (PBT)[3] characteristics; use in consumer products; production volume; and other similar factors. This Action Plan is based on EPA's initial review of readily available use, exposure, and hazard information[4] on PFCs. EPA considered which of the various authorities provided under TSCA and other statutes might be appropriate to address potential concerns with PFCs in developing the Action Plan. The Action Plan is intended to describe the courses of action the Agency plans to pursue in the near term to address its concerns. The Action Plan does not constitute a final Agency determination or other final Agency action. Regulatory proceedings indicated by the Action Plan will include appropriate opportunities for public and stakeholder input, including through notice and comment rulemaking processes.

III. Scope of Review

Continuing contributions of PFAS/PFAC to the environmental/human reservoir are best addressed using a category approach.

The PFAS/PFAC precursors may be polymers that are coated on a specific substrate. This action is considering only the contribution of precursors as a source of PFAS/PFAC, and not the inherent toxic effects of the polymer or exposure to dust that contains fluorinated polymers.

Long-Chain Perfluoroalkyl Sulfonate (PFAS) Sub-Category

The PFAS sub-category includes perfluorohexane sulfonic acid (PFHxS)[5], perfluorooctane sulfonic acid (PFOS)[6], and other higher homologues. The category also includes the acid salts and precursors.

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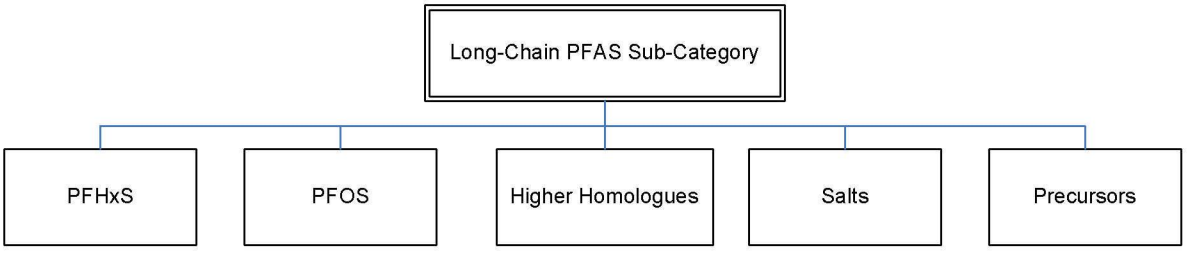
[2] 15 U.S.C. §2601 *et seq.*

[3] Information on PBT chemicals can be found on the EPA website at http://www.epa.gov/pbt/.

[4] Information sources customarily employed include Inventory Update Reporting (IUR) submissions; Toxic Release Inventory (TRI) reporting; data submitted to the HPV Challenge Program; existing hazard and risk assessments performed by domestic and international authorities including but not limited to U.S. Federal government agencies, the Organization for Economic Cooperation and Development, the Stockholm Convention on Persistent Organic Pollutants, Health and Environment Canada, the European Union; and others. Action plans will reference specific sources used.

[5] CF_3-$(CF_2)_5$-SO_3H; CAS RN: [355-46-4].

[6] CF_3-$(CF_2)_7$-SO_3H; CAS RN: [1763-23-1].



The similarities of the chemicals within the PFAS sub-category can be established when reviewing representative structures of the different category member compounds:

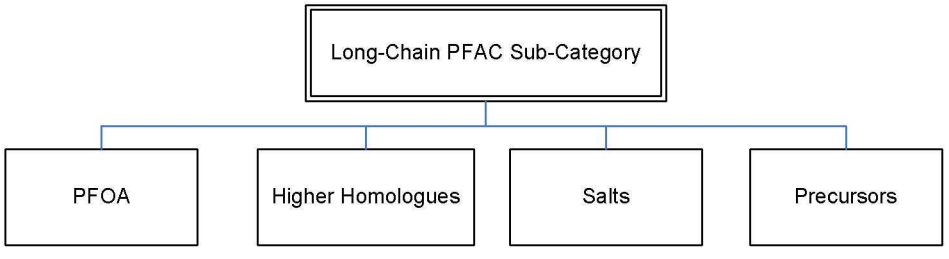
a. $CF_3(CF_2)_n$-SO_3^-M where M = H^+ or any other group where a formal dissociation can be made; and

b. $CF_3(CF_2)_n$-$S(=O)_y$-X where y = 0 – 2 and X is any chemical moiety.

where n > 4.

Long-Chain Perfluoroalkyl Carboxylate (PFAC) Sub-Category

The PFAC sub-category includes perfluorooctanoic acid (PFOA) [7] and other higher homologues. The category also includes the acid salts and precursors.



These similarities within the PFAC sub-category can be established by reviewing representative structures of the different category member compounds:

a. $CF_3(CF_2)_n$-COO^-M where M = H^+ or any other group where a formal dissociation can be made;

b. $CF_3(CF_2)_n$-$CH=CH_2$;

c. $CF_3(CF_2)_n$-C(=O)-X where X is any chemical moiety;

d. $CF_3(CF_2)_m$-CH_2-X where X is any chemical moiety; and

e. $CF_3(CF_2)_m$-Y-X where Y = non-S, non-N hetero atom and where X is any chemical moiety.

[7] CF_3-$(CF_2)_6$-COOH; CAS RN: [335-67-1].

where n > 5 or m > 6.

IV. Uses and Substitutes Summary

Production Volume

PFAS Chemicals

Commercial production of PFAS chemicals began over half a century ago. Total production from 1970 to 2002 was estimated to be about 100,000 tons (Paul A.G., 2009). By 2003, PFOS chemicals were no longer manufactured by 3M, the principal U.S. producer. However, production of PFOS-related chemicals is still ongoing in other countries, though to a much smaller extent than before 2003 (POPRC, 2007). As PFOS-based products became more strictly regulated in developed countries, production shifted to other countries. For example, manufacturers in China began large scale production in 2003 at the advent of 3M's 2002 global PFOS phase-out. China had an annual production in 2004 of less than 50 tons, but has increased production dramatically in recent years, with an estimated production of more than 200 tons in 2006. Approximately 100 tons of that amount is designated for export (POPs, 2008).

PFAC Chemicals

World-wide production of fluorotelomers was estimated at 20 million pounds in 2006. The United States accounts for more than 50 percent of world-wide fluorotelomer production. Textiles and apparel account for approximately 50 percent of the volume, with carpet and carpet care products accounting for the next largest share in consumer product uses. Coatings, including those for paper products, are the third largest category of consumer product uses.

Fluorotelomer release sources, and consequent exposure to fluorotelomers, can be explained through the examination of the life cycle of this category of chemicals:

Manufacture of Monomers ➔ Manufacture of Polymers ➔ Processing and Use ➔ Product Life

The manufacture of non-polymeric chemicals (surfactants, wetting agents, cleansers, etc.) is included in the manufacture of monomers. Some residual monomers are present in the various raw materials and final products of the different steps of manufacturing. Because each intermediate contains the same R_f moiety, the polymers also contain this moiety. The 2010/15 PFOA Stewardship Program encourages the elimination of PFAC precursors in product content. Companies reporting under PFOA Stewardship Program differentiate between the amounts of PFAC precursors present in the final polymer product as residuals and the amount present in the polymer as R_f moities. The availability of PFAC precursor from the content of residuals in fluorotelomer based polymer products (FTBP) would be small in comparison to the amount released should polymeric materials biodegrade in the environment. Potentially all monomeric, not just the small amounts of residual monomers and other monomer raw material and intermediates released at each of the four steps in the sequence above, could be PFAC precursors.

Uses

PFCs are substances with special properties that have thousands of important manufacturing and industrial applications. They impart valuable properties, including fire resistance and oil, stain, grease, and water repellency. For example, they are used to provide non-stick surfaces on cookware and waterproof, breathable membranes for clothing, and are used in many industry segments, including the aerospace, automotive, building/construction, chemical processing, electronics, semiconductors, and textile industries.

PFAS Chemicals

PFAS are synthetic chemicals that do not occur naturally in the environment. Long-chain PFAS chemicals, as defined in this action plan, are no longer manufactured in United States. However, there is a limited set of existing uses for which alternatives are not yet available, and which are characterized by low volume, low exposure potential, and low releases.

The existing SNUR regulations on PFAS chemicals do not affect the continued use of existing stocks of the listed chemicals that had been manufactured or imported into the United States prior to the effective date of the SNURs. Existing products and formulations already in the United States containing these chemicals – for example, PFOS-based fire fighting foams produced before the rules took effect in 2002 – can also still be used without providing notice to the Agency. Because the PFAS SNURs exempt articles, PFOS may be imported or processed as part of an article without the Agency receiving prior notice.

PFAC Chemicals

PFAC are synthetic chemicals that do not occur naturally in the environment. PFOA is manufactured for use primarily as an aqueous dispersion agent [as the ammonium salt] in the manufacture of fluoropolymers, which are substances with special properties that have thousands of important manufacturing and industrial applications.

PFOA also be produced unintentionally by the degradation of some fluorotelomers, which are not manufactured using PFOA but could degrade to PFOA. Fluorotelomers are used to make polymers that impart soil, stain, grease, and water resistance to coated articles. Some fluorotelomer based products are also used as high performance surfactants in products where an even flow is essential, such as paints, coatings, cleaning products, and fire-fighting foams for use on liquid fuel fires. Fluorotelomer-based products can be applied to articles both at the factory and by consumers and commercial applicators in after-market uses such as carpet treatments and water repellent sprays for apparel and footwear.

Fluoropolymers, such as polytetrafluoroethylene (PTFE), which may contain some PFAC contamination, or that use PFOA as an emulsion stabilizer in aqueous dispersions, have a large U.S. market. The wire and cable industry is one of the largest segments of the fluoropolymer market, accounting for more than 35 percent of total U.S. fluoropolymer use. Apparel makes up about 10 percent of total fluoropolymer use, based on total reported production volume. Fluoropolymers are used in a wide variety of mechanical and industrial components, such as

plastic gears, gaskets and sealants, pipes and tubing, O-rings, and many other products. Total U.S. demand for fluoropolymers in 2004 was between 50,000 and 100,000 metric tons. The United States accounted for less than 25 percent of the world consumption of PTFE in 2007, and between 25 and 50 percent of the world consumption of other fluoropolymers. PTFE is the most commonly used fluoropolymer, and the United States consumed less than 50,000 metric tons of PTFE in 2008.

Substitutes

EPA is reviewing substitutes for PFOS, PFOA, and other long-chain PFCs under the New Chemicals Program. EPA established the program under section 5 of TSCA to help manage the potential risk from chemicals new to the marketplace.

EPA's review of alternatives to long-chain PFCs has been ongoing since 2000 and is consistent with the approaches to alternatives encouraged under the PFOA Stewardship Program. Through 2009, EPA has received and reviewed over 100 perfluorinated alternatives of various types. EPA reviews the new substances against the range of toxicity, fate, and bioaccumulation issues that have caused past concerns with perfluorinated substances, as well as any issues that may be raised by new chemistries (EPA, 2009b).

V. Hazard Identification Summary

The information used by EPA for this Action Plan includes the Organisation for Economic Co-operation and Development's (OECD) assessments of PFOS (OECD, 2002) and PFOA (OECD, 2006), EPA's Office of Pollution Prevention and Toxics' (OPPT) draft risk assessment of PFOA (EPA, 2009d), Environment Canada's assessment (Canada, 2006), the assessment of PFOS by the Stockholm Convention on Persistent Organic Pollutants (POPs, 2009), and other sources. The summary of the toxicity information is based on these previous assessments, and where appropriate, additional information on short- and long-chain lengths is provided.

World-Wide Distribution of PFAS and PFAC

Presence in Humans

PFAS and PFAC have been detected in human blood samples throughout the world. Blood samples have been collected in countries world-wide including the United States, Japan, Canada, Peru, Colombia, Brazil, Italy, Poland, Germany, Belgium, Sweden, India, Malaysia, Korea, China, and Australia. In addition, PFAS and PFAC have been detected in breast milk, liver, umbilical cord blood, and seminal plasma. In most cases, the analytes most often detected in human matrices, and usually in the highest concentrations, were PFOS, PFOA, and PFHxS. Other PFAS and PFAC detected in human tissue include perfluorooctane sulfonamide (PFOSA), 2-(N-methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH), 2-(N-ethylperfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH or PFOSAA), perfluoroheptanoic acid (PFHpA), perfluorononanoate (PFNA), perfluorodecanoic acid (PFDeA or PFDA), perfluoroundecanoic acid (PFUA), perfluorododecanoic acid (PFDoA),

perfluoropentanoic acid (PFPeA), perfluorohexanoic acid (PFHxA), and perfluorobutane sulfonate (PFBS).

National Health and Nutrition Examination Survey (NHANES) data show that mean levels of PFOS, PFOA and PFHxS in the general U.S. population older than 12 years declined between the sampling period of 1999-2000 and 2003-2004 (Calafat, 2007). In addition, 3M reported a decline of the same chemicals from 2000 to 2006 in a group of 600 adult American Red Cross (ARC) blood donors (G. W. Olsen, Mari DC, Church TR, Ellefson ME, Reagen WK, Boyd TM, Herron RM, Medhdizadehkashi Z, Nobiletti JB, Rios JA, Butenhoff JL, Zobel LR 2008). The biggest drop reported in both surveys was in PFOS (~30% in NHANES and ~60% in the ARC study). Both reported ~25% decline in PFOA. NHANES reported a 10% decrease in PFHxS while the ARC study reported a 30% drop. Conversely, PFNA increased by approximately 50% over 4 years in NHANES and by 100% over 6 years in the ARC study. 3M also reported a 100% increase in PFDeA, while the increase in NHANES was 60%. 3M reported an 80% increase in PFUA.

It appears that most of PFAS and PFAC do not vary much across adolescents participating in NHANES; however, pooled data from 2001-2002 indicate that most of the levels of perfluorinated compounds are higher in children ages 3-11 years compared to adults (individual samples 2001-2002), especially for PFHxS (Kato, 2009). More recent data on children are not available.

It is clear that there are individuals who have been exposed to perfluorinated compounds at levels much higher than the majority of the population. Recent data indicate that individuals living near a U.S. facility that uses PFOA may have much higher PFOA serum concentrations than those currently reported for the general population (Calafat, 2007; Emmett, 2006).

Presence in the Environment and Wildlife

Water

Log K_{ow} values for PFOA, PFOS and other commercially available ammonium salts range from -0.52 to > 6.8 (De Silva, 2008; Tomlin, 2005) and have water solubilities that range from 0.10 to > 500,000 (Hekster, 2003; Kissa, 2001). Long-chain PFAC have been measured in surface waters of remote areas such as the north shore of Lake Superior, the Hudson Bay region of Northeastern Canada, tributaries of the Pearl River in Guangzhou, China and the Yangtze River. Ice surface samples in the Canadian Arctic (Northwest Territories and Nunavut) had levels of that ranged from 5-246 pg/L for C9-C11 compounds.

Multiple studies have reported a global distribution of PFAC and PFAS that have been reported in wildlife tissue and blood samples. PFAS have also been found in a variety of aquatic organisms. Most recently, four perfluorinated analytes (PFOS and PFAS: C10, C11, and C12) were found in fillets from bluegill in selected rivers in Minnesota and North Carolina (Delinsky, 2009). In general, the highest concentrations in wildlife have been found in the livers of fish-eating animals close to industrialized areas.

Soil and Sediment

PFOA and PFOS are considered to be resistant to degradation in soil. Levels of C9-C11 PFAC have been found in remote Arctic region sediment ranging from 0.68 µg/kg – 2.58 µg/kg. PFAC are known to increase over time in sediment as observed in a 22-year study (1980-2002) of the Niagara River discharge. Sediment dwelling invertebrates such as amphipods, zebra mussels, and crayfish have also been found to have PFOA concentrations ranging from 2.5 – 90 ng/g ww in the Raisin, St. Clair, and Calumet Rivers (MI)(Kannan, 2005). At the 3M Decatur, AL site, PFOA concentrations in Asiatic clams ranged from 0.51 ng/g to 1.01 ng/g. Mussels and oysters in Tokyo Bay were found to contain PFOA concentrations 0.660 ng/g ww and worms from the Ariake Sea in western Japan had concentrations of PFOA of 82 ng/g ww.

PFAS and PFAC are Persistent, Bioaccumulative, and Toxic

Persistence and Bioaccumulation in Humans and Laboratory Animals

Animal studies of the straight-chain PFAS and PFAC have shown that these compounds are well absorbed orally, but poorly eliminated; they are not metabolized, and they undergo extensive uptake from enterohepatic circulation. Studies of PFOS and PFOA have shown that these compounds are distributed mainly to the serum, kidney, and liver, with liver concentrations being several times higher than serum concentrations; the distribution is mainly extracellular. Both compounds have a high affinity for binding to B-lipoproteins, albumin, and liver fatty acid-binding protein. Studies have reported PFOS, PFOA, and several other PFAS and PFAC in umbilical cord blood indicating these chemicals cross the placenta.

The elimination half-lives of several PFAS and PFAC are summarized in Table 1. In general, the rate of elimination decreases with increasing chain length, although the half-life of PFHxS (C6) is longer than the half-life of PFOS (C8) in humans. There is a tremendous species difference in elimination, and elimination is greatly reduced in humans. Thus, the half-life of PFOS is 7 days in rats, 150 days in monkeys, and 5.4 years in humans. There is a gender difference in the elimination of PFOA and other PFAC in laboratory animals. Studies of PFOA in rats have shown that the gender difference is developmentally regulated, and the adult pattern is achieved by sexual maturation. The reason for the species and gender differences in elimination are not well understood. These differences are hormonally controlled, and may also be due to the actions of organic anion transporters. A gender difference has not been found in humans, although uncertainty exists due to the small sample size.

Table 1. Comparative Rates of Elimination*

Serum Half-life	PFHxS (C6)	PFOS (C8)	PFOA (C8)	PFNA (C9)	PFDA (C10)
Rat		7 days	2-4 hours 6-7 days	2 days 31 days	59 days 40 days
Mouse			16 days 22 days	41 days 64 days	
Monkey	87 days 141 days	150 days	30 days 21 days		

Human	8.5 years	5.4 years	2.3-3.8 years		

***Red – females; blue - males**

Regardless of chain length, it is critical to note that the half-lives of these compounds are measured in hours to days to months in rats, mice and monkeys, but years in humans. This means that these compounds will persist and bioaccumulate in humans, and comparatively low exposures can result in large body burdens. The gender and species differences in elimination also indicate that comparisons of toxicological effects must utilize some measure of body burden rather than administered dose.

Persistence and Bioaccumulation in the Environment

PFOS and longer chain PFAC (> C8) bioaccumulate and persist in protein-rich compartments of fish, birds, and marine mammals such as carcass, blood, and liver (Conder, 2008). Studies have found fish bioconcentration factor (BCF) values for C8 to C14 PFAC ranging from 4 – 40,000 in rainbow trout (Martin, 2003). Fish BCF values for C8-C11 PFAS are relatively lower (4-4900). There are two BCF study results for long chain PFAC with BCF values from 4,7000 to 4,800 for perfluorohexadecanic acid (C16) in carp and BCF values from 320 to 430 for perfluorooctadecanoic acid (C18) in carp (Martin, 2003). Available evidence shows the likely potential for bioaccumulation or biomagnifications in marine or terrestrial species. This is due to conformational changes into a helical structure in the molecule resulting in a smaller cross-sectional diameter as chain length increases which can lead to the ability to accumulate in organisms (NITE, 2002a, 2002b). Additional evidence that C14 and C15 PFAC bioaccumulate and are bioavailable is their presence in fish, invertebrates, and polar bears. The bioaccumulation of PFOS and PFAC (C8 through C14) in air-breathing animals (e.g., birds and mammals) is thought to represent biomagnification due to high gastrointestinal uptake and slow respiratory elimination (B. Kelly, MG Ikonomou, JD Blair, B Surridge, F Hoover, R Grace, APC Gobas 2009; B. C. Kelly, Ikonomou MG, Blair JD, Morin AE, Gobas APC, 2007). In addition, Conder et al. state that the bioaccumulation and bioconcentration potential of PFAC are directly related to the length of the perfluorinated chain, and PFAS are more bioaccumulative than PFAC of the same chain length (Conder, 2008).

Within the PFAC and PFAS categories, the perfluorinated carboxylic and sulfonic acids (R_f from C5 to C20) are persistent chemicals that are resistant to degradation under environmental conditions. Even the reaction of PFAS/PFAC precursors with hydroxyl radicals in the atmosphere are considered to be so slow that long range transport is considered a viable exposure pathway (Hurley, 2004; G. W. Olsen, DC Mari, WK Reagen, ME Ellefson, DJ Ehresman, JL Butenhoff, LR Zobel, 2007).

Toxicity in Humans

Until recently, epidemiological and medical surveillance studies have been conducted primarily in the United States on workers occupationally exposed to POSF-based fluorochemicals. These studies specifically examined PFOS or PFOA exposures and possible adverse outcomes. One occupational study of exposures to a PFNA surfactant blend was

undertaken. The studies on PFOS and PFOA include mortality and cancer incidence studies, a study examining potential endocrine effects, an ''episodes-of-care'' study evaluating worker insurance claims data, and worker surveillance studies examining associations between primarily PFOS and/or PFOA serum concentrations and hematology, hormonal and clinical chemistry parameters. The PFNA study examined liver enzymes and blood lipid levels. In general, no consistent association between serum fluorochemical levels and adverse health effects has been observed.

Toxicity in Laboratory Animals

PFOA

The toxicity of PFOA has been extensively studied. Repeated-dose studies in rats have shown reduced body weight, hepatotoxicity, reduced cholesterol, and a steep dose-response curve for mortality. Due to gender differences in elimination, adult male rats exhibit effects at lower administered doses than adult female rats. Thus, dietary exposure for 90 days resulted in significant increases in liver weight and hepatocellular hypertrophy in female rats at 1000 ppm (76.5 mg/kg-day) and in male rats at doses as low as 100 ppm (5 mg/kg-day). Studies in nonhuman primates have shown similar effects at doses as low as 3 mg/kg-day, although the reduction in cholesterol has not been observed.

The carcinogenic potential of PFOA has been investigated in two dietary carcinogenicity studies in Sprague-Dawley rats, and has been shown to induce hepatocellular adenomas, Leydig cell tumors, and pancreatic acinar tumors. It has not been shown to be mutagenic in a variety of assays. There is sufficient evidence to indicate that PFOA is a PPARα-agonist and that the liver carcinogenicity (and toxicity) of PFOA is mediated by PPARα in the liver in rats. There is no evidence that the liver toxicity in nonhuman primates is due to PPARα-agonism. There is controversy over the relevance of this particular mode of action for humans. The mode of action for the Leydig cell tumors and pancreatic acinar tumors has not been established, and therefore these are assumed to be relevant for humans.

Several studies have shown that PFOA is immunotoxic in mice. PFOA causes thymic and splenic atrophy, and has been shown to be immunosuppressive in both *in vivo* and *ex vivo* systems. Studies using transgenic mice showed that the PPARα was involved in causing the adverse effects to the immune system.

Standard prenatal developmental toxicity studies in rats and rabbits in which pregnant animals are exposed only during gestation and sacrificed prior to the birth of the pups have not shown many effects. Thus, there was no evidence of developmental toxicity after exposure to doses as high as 150 mg/kg-day in an oral prenatal developmental toxicity study in rats. In a rat inhalation prenatal developmental toxicity study, the NOAEL and LOAEL for developmental toxicity were 10 and 25 mg/m3, respectively. In a rabbit oral prenatal developmental toxicity study there was a significant increase in skeletal variations after exposure to 5 mg/kg-day, and the NOAEL was 1.5 mg/kg-day.

However, the potential developmental toxicity of PFOA is evident when the pups are evaluated during the postnatal period. Thus, a two-generation reproductive toxicity study in rats

showed a reduction in F1 pup mean body weight during lactation at 30 mg/kg-day group and during the post-weaning period at 10 mg/kg-day. In addition, there was a significant increase in mortality mainly during the first few days after weaning, and a significant delay in the timing of sexual maturation for F1 male and female pups at 30 mg/kg-day.

Due to the rapid elimination of PFOA in female rats, many researchers have examined the developmental toxicity of PFOA in mice. These studies have shown a pattern of developmental effects similar to those observed with PFOS. Full liter resorptions were noted at 40 mg/kg-day and the percent of live fetuses and fetal body weight were reduced at 20 mg/kg-day. The most notable effect of prenatal exposure to PFOA was the severe compromise of postnatal survival at doses as low as 5 mg/kg-day, and the postnatal growth impairment and developmental delays noted among the survivors; the BMD_5 and $BMDL_5$ for neonatal survival were estimated at 2.84 and 1.09 mg/kg-day, respectively. Additional studies in mice have shown that PFOA exposure causes a significant reduction in mammary gland differentiation in the dams and stunted mammary gland development in the female pups.

Several studies have examined the mode of action for the developmental effects. These have shown that exposure to a dose of 20 mg/kg-day for 2 days late in gestation is sufficient to cause the neonatal mortality in mice. Studies with PPARα knockout mice have shown that the PPARα is required for the neonatal mortality and expression of one copy of this gene is sufficient. This is in contrast to the studies showing that PPARα is not involved in the neonatal mortality associated with PFOS exposure. Although there is controversy over the human relevance of the PPARα-agonist hepatotoxicity observed in rodents, the role of PPARα in development and particularly in the PFOA-induced neonatal mortality observed in mice is unknown; therefore this mode of action is assumed to be relevant for humans.

Other PFAC Chemicals

Although there is an extensive database for PFOA, few studies have examined the toxicity of the shorter or longer chained PFAC. However, the data suggest that the toxicity profile is quite similar to that of PFOA, albeit at different dose levels presumably due to the differences in elimination half-life.

Although standard repeated-dose toxicity studies have not been conducted on the PFAC with chain lengths greater than PFOA, many studies have been conducted examining the potential for hepatomegaly and peroxisome proliferation (a marker for the activation of PPARα). Kudo et. al. found that PFOA, PFNA, and PFDA induced the activity of peroxisomal B-oxidation in male rats (2000). Kudo et al. showed that all PFAC with six- to nine-carbon length chains induced hepatomegaly and peroxisomal B-oxidase activity in mice, and the potency was in the order of PFNA > PFOA > perfluoroheptanoic acid (2006). Permadi et al. also showed that PFDA induces hepatomegaly and hepatic peroxisomal palmitoyl-CoA oxidase (1993). Thus, these studies indicate that the PFAC with a carbon chain length of eight and greater activate PPARα. The differences in potency probably reflect the differences in the half-life of the varying chain lengths. Despite the lack of traditional toxicity studies, it is reasonable to conclude that these compounds would likely produce similar effects as those observed with PFOA.

With respect to the potential developmental effects of PFAC with carbon chain lengths greater than C8, EPA is completing a developmental toxicity study of PFNA in mice (C. Lau, personal communication, 2009). Maternal body weight gain was reduced at 3 mg/kg-day, and severe toxicity was observed at 10 mg/kg-day. Neonatal survival was compromised at 5 mg/kg-day, and significant lags in neonatal growth were observed at 3 mg/kg-day. Thus, this study shows a pattern of effects very similar to those observed with PFOA. It is likely that PFAC with carbon chain lengths greater than nine would also result in similar effects, and that the potency would be dependent on the half-life of the compound.

PFOS

The toxicity of PFOS has also been extensively studied and was summarized in OECD report (2002) and by Lau et al. (2006). Repeated-dose studies in rats and nonhuman primates have shown reduced body weight, hepatotoxicity, reduced cholesterol, and a steep dose-response curve for mortality. These effects occur in nonhuman primates at doses as low as 0.75 mg/kg-day, and in rats at 2 mg/kg-day.

The carcinogenic potential of PFOS has been investigated in a dietary carcinogenicity study in Sprague-Dawley rats, and has been shown to induce hepatocellular adenomas at 20 ppm. In addition, thyroid follicular cell adenomas were observed in male rats that had been allowed to "recover" for a year following treatment for one year; the reason for this is unclear. However, thyroid follicular tumors have also been observed in rats exposed to N-EtFOSE, a major precursor of PFOS. PFOS has not been shown to be mutagenic in a variety of assays. Although PFOS can activate PPARα, the data are not sufficient to establish a PPARα-agonist mode of action for the liver tumors.

A standard prenatal developmental toxicity study in rats has shown a significant decrease in fetal body weight and significant increase in external and visceral anomalies, delayed ossification, and skeletal variations; a NOAEL of 1 mg/kg-day and a LOAEL of 5 mg/kg-day for developmental toxicity were indicated. In rabbits, significant reductions in fetal body weight and significant increases in delayed ossification were observed; a NOAEL of 1.0 mg/kg-day and a LOAEL of 2.5 mg/kg-day for developmental toxicity were indicated.

A two-generation reproductive toxicity study in rats showed neonatal mortality. All F1 pups at the highest dose of 3.2 mg/kg-day died within a day after birth, while close to 30% of the F1 pups at 1.6 mg/kg-day died within 4 days after birth. As a result of the pup mortality in the two top dose groups, only the two lowest dose groups, 0.1 and 0.4 mg/kg-day, were continued into the second generation. The NOAEL and LOAEL for the F2 pups were 0.1 mg/kg-day and 0.4 mg/kg-day, respectively, based on reductions in pup body weight.

The results of this study prompted additional research. Studies in which pregnant rats and mice were dosed during gestation and the pups were followed postnatally provided a BMD_5 and $BMDL_5$ for neonatal survival of 1.07 and 0.58 mg/kg-day in rats, respectively, and 7.02 and 3.88 mg/kg-day in mice, respectively. Studies have shown that the critical period of exposure is during late gestation. Mode of action studies initially focused on the lung and found significant histological and morphometric differences in the lungs of pups treated with PFOS. However,

subsequent studies did not find any effect on lung phospholipids and rescuing agents failed to mitigate the neonatal mortality. Thus, the mortality does not appear to be related to lung immaturity. In contrast to PFOA, studies with PPARα knockout mice have shown that the PPARα is not involved in the neonatal mortality. Current research is focusing on the possibility that the physical properties of PFOS may interfere with the normal function of pulmonary surfactant, leading to neonatal mortality.

Other PFAS Chemicals

A combined reproductive/developmental toxicity study of PFHxS has been conducted in rats. In the parental males there was a significant reduction in cholesterol at doses as low as 0.3 mg/kg-day, and hepatotoxicity at doses as low as 3 mg/kg-day. There was no evidence of developmental or reproductive toxicity at doses as high as 10 mg/kg-day.

Toxicity to Wildlife

Adverse effects on exposed populations of organisms have been observed with exposure to perfluorinated compounds in the parts per million range. Studies have shown a reduction in hatchability of chickens when they were exposed *in ovo* to PFOS, and a reduction in survival in 14-day old Northern bobwhite quail from hens exposed to 10 ppm of PFOS in the diet. In addition, a delay in growth and metamorphosis in the Northern leopard frog exposed to 3 mg/L of PFOS has been reported, as well as reduced cumulative fecundity and fertility effects in fathead minnows exposed to 0.1 mg/L PFOS. Further evidence of potential reproductive effects has been observed with exposure to C9-C11 PFAC. A significant induction of vitellogenin in rainbow trout was observed in a dose-dependent manner at concentrations of C10 PFAC 0.0256-2000 μg/g in the diet as well as a weak affinity demonstrated for the hepatic estrogen receptor from C9-C12 PFAC.

Mortality in sediment dwelling organisms such as the nematode, *Caenorhabditis elegans* has been observed with concentrations of C9 up to 0.66 mM and subsequent effects in offspring generations were found at concentrations up to 1nM as evidence by a 70 % decline in fecundity.

VI. Fate Characterization Summary

The PFAS and PFAC acids are strong acids that exist in equilibrium between the neutral form and the anionic form. Both the anionic and neutral forms of PFOA are soluble in water. While the Henry's law constant values suggests partitioning to air for the neutral, protonated form, predicting the amount that partitions into air is complicated because there is uncertainty over the degree to which carboxylic and sulfonic acids partition from the water to atmosphere. The uncertainty arises with regard to the value of the acid dissociation constant (i.e., pK_a), or the fraction of the acid form present at environmentally relevant pH. PFAC and PFAS have been detected in air, water, and soil samples collected throughout the world. The oceans have been suggested as the final sink and route of transport for perfluorinated carboxylic and sulfonic acids, where they have been detected on the surface and at depths > 1,000 meters (Yamashita, 2005).

Some PFAS/PFAC have the potential for long-range transport. They are transported over

long distances (i.e., long-range transport) by a combination of dissolved-phase ocean and gas-phase atmospheric transport; however, determining which is the predominant transport pathway is complicated by the uncertainty over water to atmosphere partitioning. Furthermore, there is evidence that transport and subsequent oxidation of volatile alcohol PFAS/PFAC precursors may contribute to the levels of PFAS / PFAC in the environment.

Studies by industry and academic researchers have shown that fluorotelomer alcohols (FTOH) can be degraded by microorganisms and by abiotic processes. 8-2 FTOH and FTOH of other chain lengths, and related chemicals in mixed microbial cultures, activated sludge and soil systems have been shown to be easily degraded to form PFOA and related perfluorinated acids. Some studies have also shown that $-CF_2-$ groups can be mineralized, forming shorter chain perfluoro acids. If FTOH are absorbed from ingestion, inhalation, dermal or ocular exposure or formed in vivo by from other compounds they can be metabolized by mammals and other organisms to form perfluorinated acids and other fluorinated compounds. FTOH can be degraded by abiotic processes in water and air to produce PFAC and various intermediates. FTOH are fairly volatile. Based on atmospheric half-lives determined in chamber studies, FTOH can be transported globally. Deposition or degradation in areas far from the source can result in PFAC contamination in high latitudes and other remote locations and contribute to global background levels of PFAC and PFAS.

Data submitted by industry and in the open literature show that perfluorooctane sulfonyl fluoride (POSF) and its derivatives can be degraded under environmental conditions to form perfluoroalkyl sulfonates and carboxylic acids. Reaction of POSF ($CF_3(CF_2)_n-SO_2F$) with methyl or ethyl amines is used to produce N-ethyl or N-methyl perfluorooctane sulfonamidoethanols (FOSE). Similar reactions are used to make shorter and longer chain analogs to POSF and POSF derivatives. FOSE compounds, (or $CF_3(CF_2)_n-SO_2N(R1)(R2)$, where R1 and R2 can be hydrogen, methyl or longer alcohols or other organic chains), such as N-methyl and N-ethyl FOSEs can be degraded though a series of intermediates to form both perfluoro carboxylic acids and perfluoroalkyl sulfonates. Data on the degradation of individual intermediates has been used to identify these pathways and has confirmed that these compounds can be degraded by a number of microbial and abiotic mechanisms. Reaction with other chemical intermediates produces other FOSA derivatives, including phosphate esters, fatty acids esters, silanes, carboxylates, and polymers with acrylate, urethane and other linkages. Longer and shorter chain perfluoro sulfonyl derivatives have also been produced intentionally and as unintended reaction products. Based on existing data from the open literature and CBI data, it is expected that that most, if not all, of these POSF and other chain length sulfonyl fluorides and their derivatives will be degraded to carboxylic acids and/or sulfonate over time. Most of these compounds will have environmental and metabolism half-lives of weeks to months. Some will be degraded faster and some will degrade more slowly, but all will eventually be degraded.

Very little data is available on the behavior of other perfluorochemicals in the environment and in vivo but the existing data suggest that they will also be degraded to form PFAC. For example, recent studies have shown that ingested mono and di polyfluoroalkyl phosphates (PAPs) can be degraded in rats to form PFOA and other PFAC in the body. They can also be degraded by microbial processes in soil and wastewater to form perfluorinated acids (D'eon, 2007).

A limited number of studies on the degradation of fluorotelomer-based polymers have been submitted in support of PMN submissions and existing chemicals, and published in the open literature. Based on studies, some fluorotelomer-based polymers are subject to hydrolysis, photolysis and biodegradation to some extent. Studies have shown half-lives of a few days to hundreds of years.

In addition, preliminary research on degradation of fluorotelomers has shown that some urethanes and acrylates biodegrade; however, half-lives and kinetics of the fluorotelomers are not yet well-defined. Ongoing research by EPA's Office of Research and Development (ORD) research is designed to generate high quality data that will help the Agency address some key uncertainties in pathways of exposure and potential risks from PFOA (Washington, 2009).

These studies have shown that the perfluorinated portion of some polymers is released as the polymer is degraded by microbial or abiotic processes to form telomer alcohols or other intermediates and that they eventually form PFAC. Polymers based on POSF and other chain length chemistries show similar degradation rates and release intermediates that further degrade to form perfluorinated acids and sulfonates. Studies have shown that some polymers can undergo indirect photolysis in soil and in aquatic systems and be degraded with half-lives of days to several years.

VII. Exposure Characterization Summary

The pattern of PFAS and PFAC contamination varies with location and among species, which suggests multiple sources of emission and patterns of migration into environmental media from the sources of emission. Major pathways that enable PFOA and PFOS to get into human blood in small quantities are not yet fully understood. Manufacturing releases are known to have contaminated local drinking water supplies in the immediate vicinity of some industrial plants, leading to localized elevated blood levels. The widespread presence of PFOA and PFOS precursors in human blood samples nationwide suggests other pathways of exposure, possibly including long range air transport, and the release of PFOA and PFOS from treated articles.

Summary of Exposure to Consumers and Children from PFCs in Indoor Environments

PFCs in Articles of Commerce

EPA's ORD has conducted research on 116 articles of commerce documenting that PFCs contained in articles of commerce have the potential to be released from those articles. Articles tested and found to contain the highest levels of PFAC were carpet and carpet treatment products, various types of apparel, home textiles, thread sealant tape, floor wax and other sealants, and food contact paper and paper coatings. Carpet and carpet treatment products contained individual PFAC in levels from 0.04-14100 ng/g; food contact paper and paper coatings: 0.05-160,000 ng/g; thread sealant tape and apparel: ND (non-detect)-3488 ng/g and ND-4640ng/g respectively; floor wax and sealer: 0.03-3720 ng/g; and home textiles: ND-519 ng/g. Some of the more commonly found PFAC measured in these articles were PFHxA, PFHpA, PFNA, PFDA, PFUnDA, PFOA and PFOS. Inhalation levels of PFOA and total PFCs

measured in carpet were 5385 pg/cm^3 and 32500 pg/cm^3 respectively (Guo, 2009).

Children are particularly susceptible to exposure from inhalation of PFC off-gassing from carpet and carpet protectants during their earliest years when they are lying, crawling and spending large amounts of time playing on the carpet. The significantly high levels of PFC found by ORD in carpet and carpet protectants pose an exposure concern for children through this pathway. Adults can also be exposed to PFCs in carpets through inhalation and dermal contact. Consumers and children may also be exposed to PFCs in apparel, home textiles, thread sealant tape, floor wax, contact paper and paper coatings. Some of these articles such as paper coatings for foods cannot be ruled out for the ingestion exposure pathways for children and adults depending upon how the PFCs in the paper contacts the food and subsequently humans.

PFCs in Indoor Air

Another source of PFCs to the indoor environment is dust containing not only PFAC and PFAS but also fluorotelomer alcohols. Maximum indoor dust air measurements of 6:2 FTOH were found at 804 ng/g in the house dust of eastern United States (Strynar, 2008). The PFAS (ET-FOSA, Et-FOSE, MeFOSE) chemicals were measured at 646 ng/g, 75440 ng/g, and 8860 ng/g respectively in indoor air in Canada (Shoeib, 2005). PFOA was found at 3700 ng/g in Japanese household vacuum cleaner dust (Moriwaki, 2003).

Summary of Exposure to the General Population

PFCs in Groundwater, Freshwater, Saltwater, and Rainwater

PFAC and PFAS have been found in many countries as well as in Unites States in untreated groundwater, rivers, streams, bays, estuaries, oceans and rain water. Levels of PFAC in groundwater near the 3M Cottage Grove, MN industrial site have been measured as high as 846,000 ng/l (PFOA) and in freshwater as high as 178,000 ng/l (PFBA) (Department of Health and Human Services, 2005). PFOS has been found near Cottage Grove, MN in groundwater at levels of 371,000 ng/l and in freshwater at 18,200 ng/l. PFAC in rainwater has been measured in the United States between 0.1 and 1006 ng/l (PFHpA) (Scott BF, 2006).

Saltwater levels of PFOS have been measured in the Pacific Ocean at 57,700 ng/l and in precipitation from snow and rain in China at 545 ng/l (Liu W, 2009; Yamashita, 2005). While the general population may not directly ingest these groundwater, freshwater and saltwater levels as drinking water, the ground water and freshwater containing PFCs may discharge to surface waters from which municipalities withdraw drinking water. The general population may also experience dermal, ingestion and inhalation exposures when coming into contact with freshwater containing PFCs. Rainwater containing PFCs may contribute PFCs to vegetables and fruits in home gardens, crops grown on commercial crop lands, drinking water reservoirs, and surface waters from which drinking water is withdrawn.

PFCs in Freshwater and Saltwater Fish

Freshwater fish have been found to contain levels of PFAS and PFAC. The highest levels

of PFAS measured in the United States to date were near the 3M Cottage Grove, MN site (Oliaei F, 2006). Liver samples of bass, walleye and carp ranged from 130-6350 ng/g PFOS wet weight. Blood samples of these same fish ranged from PFOS levels of 136-29600 ng/ml in serum. Total PFCs for the blood of freshwater fish in the same area was measured at 32248 ng/ml serum. The highest levels of PFAC for freshwater fish were found near the 3M Cottage Grove, MN site and were measured for blood samples of bass, walleye, and carp in the range of 2.53-210 ng/ml serum. For comparison, saltwater fish in Danish seas had measured levels of PFOS up to 156 ng/g and saltwater fish in Charleston Harbor South Carolina were found with PFOS levels up to 101 ng/g (Bossi R, 2005; Houde M, 2006).

VIII. Risk Management Considerations

Current Risk Management Summary

PFAS Chemicals

Following the voluntary 3M phase-out of PFAS chemicals in the United States in 2002, EPA issued SNURs to control the reintroduction of these chemicals into the U.S. market. Final rules were published on March 11, 2002 (EPA, 2002b) and December 9, 2002 (EPA, 2002a), to limit any future manufacture or importation of 88 PFAS chemicals specifically included in that phase-out. On October 9, 2007, EPA published another SNUR on 183 additional PFAS chemicals (EPA, 2007). Those actions were necessary because data showed that certain alkyl chain lengths of the PFAS chemicals are toxic to human health, bioaccumulate, and are persistent in the environment. PFAS chemicals are no longer manufactured in United States. However a limited set of existing uses was excluded from the SNURs because alternatives were not yet available.

Similar to the PFAS SNURs in United States, PFOS has also been restricted in the European Union, Canada, Australia and other countries, and has been nominated for inclusion in the Stockholm Convention and the Convention on Long-Range Transboundary Air Pollution (LRTAP) Persistent Organic Pollutants (POPs) protocol. At the fourth Conference of the Parties (COP) to the Stockholm Convention on POPs, held in May 2009, delegates agreed to add PFOS, its salts, and perfluorooctane sulfonyl fluoride (PFOSF) to Annex B, subjecting it to restrictions on production and use. Parties agreed that while the ultimate goal is the elimination of PFOS, production of the chemical may continue for limited purposes, including coatings for semiconductors, firefighting foam, photo imaging, aviation hydraulic fluids, metal plating, and certain medical devices. Countries must notify the Convention Secretariat whether they intend to continue production for acceptable purposes. Countries can also ask for specific exemptions allowing the production of PFOS for use in the production of chemical substances used in goods such as carpets, leather and apparel, textiles, paper and packaging, coatings, and rubber and plastics (POPs, 2009).

PFAC Chemicals

OPPT's core strategy for working towards the elimination of PFAC chemicals has been through the PFOA Stewardship Program. Under the program, eight major companies operating

in the United States committed to reduce global facility emissions and product content of PFAC chemicals by 95 percent by 2010, and to work toward eliminating emissions and product content by 2015 (EPA, 2009a). Companies provide annual progress reports, and most companies have reported significant progress in meeting program goals.

On March 7, 2006, EPA published a proposal to amend the polymer exemption rule to exclude polymers containing certain perfluoroalkyl moieties from eligibility for the exemption (EPA, 2006). Under this proposal, polymers containing these perfluoroalkyl moieties would need to go through the pre-manufacture notification (PMN) review process so that EPA can better evaluate these polymers for potential effects on human health and the environment. This change to the current regulation is necessary because, based on current information, EPA can no longer conclude that these polymers "will not present an unreasonable risk of injury to health or the environment" under the terms of the polymer exemption rule, which is the determination necessary to support an exemption under section 5(h)(4) of TSCA. This amendment to the polymer exemption rule is a necessary complement to the PFOA Stewardship Program and will give EPA the necessary tools to review and control risk of PFC-based and related polymers, including those PFAS and PFAC containing polymers.

In January 2009, EPA's Office of Water (OW) developed Provisional Health Advisory (PHA) values for PFOA and PFOS to mitigate potential risk from exposure to these chemicals through drinking water (EPA, 2009c). Due to limited information on the toxicity of PFCs other than PFOA and PFOS, no attempt was made by OW at that time to develop PHA values for the other PFCs. OPPT and OW are working together to determine whether revised health advisory values are needed for PFOA and PFOS.

In October 2009, EPA's Office of Solid Waste and Emergency Response (OSWER) used OW's PHA's to derive sub-chronic R_fD values for PFOA and PFOS. These values may be used in the Superfund program's risk-based equations to derive Removal Action Levels and/or Screening Levels for water and other media, as appropriate.

EPA has taken the leadership role in raising the profile of PFCs at an international level stemming from Agency concerns about the role of long range transport in the environmental distribution of PFCs, and U.S. importation of products containing these chemicals (UNEP, 2009b). As a result of these activities, in May 2009, during the International Conference on Chemicals Management (ICCM2), delegates to the Strategic Approach to International Chemicals Management (SAICM) agreed to consider the development of stewardship programs and regulatory approaches to reduce emissions and content of PFAC and PFAS chemicals in products and to work towards their elimination, where feasible (UNEP, 2009a).

Remaining Issues and Concerns

PFAS Chemicals

PFAS chemicals are no longer manufactured in the United States but continue to be manufactured outside of the United States. Although the PFAS SNURs are an important step toward controlling any future manufacture or import of PFAS chemicals, these chemicals may

continue to be imported into United States in articles, such as carpets, leather and apparel, textiles, paper and packaging, coatings, and rubber and plastics.

Possible scenarios of concern:
- Direct releases to the environment from U.S. facilities as a result of few existing uses.
- Direct releases to the environment from non-U.S. facilities, resulting in transboundary environmental transport to United States.
- Articles containing PFAS chemicals. Recent research by EPA's ORD has shown that consumer articles could release PFCs, significantly increasing the magnitude and duration of exposure to humans and the environment to these chemicals.

PFAC Chemicals

Although the 2010/15 PFOA Stewardship Program is expected to eliminate the production of C8-based fluorotelomers by the eight participating companies by 2015, the potential remains for continued environmental and human loading of PFAC in the United States. This is in part because companies not participating in the PFOA Stewardship Program may follow the market opportunity presented when the eight PFOA Stewardship Program companies leave the PFAC market by 2015. This occurred with PFAS production in some Asian countries after the 3M 2002 phase-out of PFAS chemicals in United States (Wenya, 2008).

Possible scenarios of concern:
- Direct releases to the environment from U.S. facilities not participating in PFOA Stewardship Program.
- Direct releases to the environment from non-U.S. facilities not participating in PFOA Stewardship Program, resulting in transboundary environmental transport to United States.
- Articles, including imports, containing PFAC chemicals. These articles could release PFAC as a result of their residual content in fluorotelomer-based products and/or as the fluorotelomers-based polymers in articles biodegrade.

IX. Next Steps

To date, significant adverse effects have not been found in general human population; however, significant adverse effects have been identified in laboratory animals and wildlife. Given the long half-life of these chemicals in humans (years), it can reasonably be anticipated that continued exposure could increase body burdens to levels that would result in adverse outcomes. Consequently, EPA intends to propose actions in 2012 under TSCA to address the potential risks from long-chain PFCs.

EPA intends to consider initiating TSCA section 6 rulemaking for managing long-chain PFCs. If EPA can make certain findings with respect to these chemicals (further analysis of the information will be performed as part of TSCA section 6 rulemaking), TSCA section 6 provides authority for EPA to ban or restrict the manufacture (including import), processing, and use of these chemicals. A rule addressing the PFAS sub-category could expand beyond the reach of the SNURs that the Agency has promulgated over the past decade. For example, the rule could address PFAS-containing articles. A rule addressing the PFAC sub-category could expand the

reach of the 2010/15 PFOA Stewardship Program beyond the eight participating companies and further address the concerns for potential PFAC exposure through the use of PFAC-containing articles. EPA will develop more detailed assessments to support the TSCA section 6(a) "presents or will present an unreasonable risk" findings. If these more detailed assessments indicate that a different approach to risk management is appropriate, EPA will consider additional approaches.

EPA will continue with the 2010/15 PFOA Stewardship Program to work with companies toward the elimination of long-chain PFCs from emissions and products. EPA will also continue to evaluate alternatives under EPA's New Chemicals Program and collaborate with other countries on managing PFCs.

As part of the Agency's efforts to address these chemicals, EPA also intends to evaluate the potential for disproportionate impact on children and other sub-populations.

X. References

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Parts 141 and 142

[Docket No. EPA–HQ–OW–2009–0090; FRL–9660–4]

RIN 2040–AF10

Revisions to the Unregulated Contaminant Monitoring Regulation (UCMR 3) for Public Water Systems

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: The 1996 amendments to the Safe Drinking Water Act (SDWA) require that the United States Environmental Protection Agency (EPA or the agency) establish criteria for a program to monitor unregulated contaminants and publish a list of up to 30 contaminants to be monitored every five years. This final rule meets the SDWA requirement by publishing the third Unregulated Contaminant Monitoring Regulation (*i.e.,* UCMR 3), listing the unregulated contaminants to be monitored and addressing the requirements for such monitoring. This final rule describes analytical methods to monitor for 28 chemical contaminants and describes the monitoring for two viruses. UCMR 3 provides EPA and other interested parties with scientifically valid data on the occurrence of these contaminants in drinking water, permitting the assessment of the number of people potentially being exposed and the levels of that exposure. These data are one of the primary sources of occurrence and exposure information the agency uses to develop regulatory decisions for these contaminants. In addition, as part of an Expedited Methods Update, this rule finalizes amendatory language for a drinking water inorganic analysis table (''Inorganic chemical sampling and analytical requirements'') in the Code of Federal Regulations (CFR). This minor editorial correction to the table does not affect the UCMR program.

DATES: This final rule is effective on June 1, 2012. For purposes of judicial review, this rule is promulgated as of 1 p.m. Eastern time on May 16, 2012 as provided in 40 CFR 23.7. The incorporation by reference of certain publications listed in this rule is approved by the Director of the Federal Register as of June 1, 2012.

ADDRESSES: EPA has established a docket for this action under Docket ID No. EPA–HQ–OW–2009–0090. All documents in the docket are listed in the index at *www.regulations.gov.* Although listed in the index, some information is not publicly available, *e.g.,* confidential business information (CBI) or other information, the disclosure of which 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 either electronically at *www.regulations.gov* or in hard copy at the Water Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave. NW., Washington, DC. This 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 this Public Reading Room is (202) 566–1744, and the telephone number for the Water Docket is (202) 566–2426.

FOR FURTHER INFORMATION CONTACT: Brenda D. Parris, Technical Support Center, Standards and Risk Management Division, Office of Ground Water and Drinking Water, United States Environmental Protection Agency, Office of Water, 26 West Martin Luther King Drive (MS 140), Cincinnati, Ohio 45268; telephone (513) 569–7961; or email at *parris.brenda@epa.gov*. For general information, contact the Safe Drinking Water Hotline. Callers within the United States may reach the Hotline at (800) 426–4791. The Hotline is open Monday through Friday, excluding legal holidays, from 10:00 a.m. to 4:00 p.m., Eastern time. The Safe Drinking Water Hotline may also be found on the Internet at *http://water.epa.gov/drink/contact.cfm.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

Entities regulated by this action are public water systems (PWSs). All large community and non-transient non-community water systems serving more than 10,000 people are required to monitor. A community water system (CWS) means a PWS, which has at least 15 service connections used by year-round residents or regularly serves an average of at least 25 year-round residents. A non-transient non-community water system (NTNCWS) means a PWS that is not a CWS and regularly serves at least 25 of the same people over six months per year. Only a nationally representative sample of ''small'' community and non-transient non-community systems serving 10,000 or fewer people are required to monitor for the chemical analytes (see USEPA, 2001 for a description of the statistical approach for the nationally representative sample). EPA will pay for the analysis of samples collected by these small systems. Transient non-community water systems (TNCWS) (*i.e.,* systems that do not regularly serve at least 25 of the same people over six months per year) are not required to monitor for the chemical analytes. However, transient ground water systems serving 1,000 or fewer people may be selected for virus monitoring. If selected, these systems are required to permit EPA to sample and analyze for List 3 contaminants and pathogen indicators. EPA will pay for all sampling and analysis costs associated with virus monitoring at these small systems. Exhibit 1 summarizes UCMR 3 applicability by system type and size.

EXHIBIT 1—APPLICABILITY OF UCMR 3 TO WATER UTILITIES BY SYSTEM TYPE AND SIZE

System type	System size [1]	
	Serving >10,000	Serving ≤10,000
UCMR 3 Assessment Monitoring		
CWS & NTNCWS	Requires all systems to monitor for List 1 chemicals	Requires 800 randomly selected systems to monitor for List 1 chemicals. EPA will pay for the analysis of samples.
TNCWS	No requirements	No requirements.

EXHIBIT 1—APPLICABILITY OF UCMR 3 TO WATER UTILITIES BY SYSTEM TYPE AND SIZE—Continued

System type	System size [1]	
	Serving >10,000	Serving ≤10,000
UCMR 3 Screening Survey		
CWS & NTNCWS	Requires all systems serving more than 100,000, and 320 randomly selected systems serving 10,001 to 100,000 to monitor for List 2 chemicals.	Requires 480 randomly selected systems to monitor for List 2 chemicals. EPA will pay for the analysis of samples.
TNCWS	No requirements	No requirements.
UCMR 3 Pre-Screen Testing		
CWS, TNCWS & NTNCWS	No requirements	Requires 800 randomly selected systems to permit EPA to sample and analyze List 3 microbes. The selected systems will be served by non-disinfecting ground water wells in vulnerable areas. EPA will pay for the analysis of samples.

[1] Based on the retail population, as indicated by SDWIS/Fed on December 31, 2010.

States, Territories, and Tribes with primary enforcement responsibility (primacy) to administer the regulatory program for PWSs under SDWA may participate in the implementation of UCMR 3 through Partnership Agreements (PAs). These primacy agencies may choose to perform the required analysis of samples collected for UCMR 3; however, the PWS remains responsible for compliance with this rule. Regulated categories and entities are identified in the following exhibit.

Category	Examples of potentially regulated entities	NAICS [a]
State, Local, & Tribal Governments.	States, local and Tribal governments that analyze water samples on behalf of public water systems required to conduct such analysis; States, local and Tribal governments that directly operate community, transient and non-transient non-community water systems required to monitor.	924110
Industry	Private operators of community and non-transient non-community water systems required to monitor	221310
Municipalities	Municipal operators of community and non-transient non-community water systems required to monitor	924110

[a] NAICS = North American Industry Classification System.

This exhibit is not exhaustive, but rather provides a guide for readers regarding entities that may be regulated by this action. This exhibit lists the types of entities that EPA is now aware may potentially be regulated by this action. Other types of entities not listed in the exhibit could also be regulated. To determine whether your facility is regulated by this action, you should carefully examine the definition of PWS in § 141.2 of Title 40 of the Code of Federal Regulations, and applicability criteria in § 141.40(a)(1) and (2) of this action. If you have questions regarding the applicability of this action to a particular entity, consult the persons listed in the preceding **FOR FURTHER INFORMATION CONTACT** Section.

B. Copies of This Document and Other Related Information

This document is available for download at: *www.regulations.gov*. For other related information, see preceding discussion on docket.

Abbreviations and Acronyms

μg/L Microgram(s) per Liter
ASDWA Association of State Drinking Water Administrators
ATSDR Agency for Toxic Substances and Disease Registry
AGI Acute Gastrointestinal Illness
CCL Contaminant Candidate List
CFR Code of Federal Regulations
CWS Community Water System
DQO Data Quality Objectives
DSMRT Distribution System Maximum Residence Time
EO Executive Order
ELISA Enzyme-linked Immunosorbent Assay
EPA United States Environmental Protection Agency
EPTDS Entry Point to the Distribution System
FR **Federal Register**
GC/MS Gas Chromatography/Mass Spectrometry
GWUDI Ground Water Under the Direct Influence of Surface Water
HCF–22 Chlorodifluoromethane
HPLC/MS/MS High-Performance Liquid Chromatography/Tandem Mass Spectrometry
HRL Health Reference Level
IC/MS Ion Chromatography/Mass Spectrometry
ICR Information Collection Request
IDC Initial Demonstration of Capability
IHS Indian Health Service
LCMRL Lowest Concentration Minimum Reporting Level
LC/MS/MS Liquid Chromatography/Tandem Mass Spectrometry
LFSM Laboratory Fortified Sample Matrix
LFSMD Laboratory Fortified Sample Matrix Duplicate
MDL Method Detection Limit
MRL Minimum Reporting Level
NAICS North American Industry Classification System
NCOD National Drinking Water Contaminant Occurrence Database
ND Not Detected
NTNCWS Non-Transient Non-Community Water System
NTTAA National Technology Transfer and Advancement Act
NWQL National Water Quality Laboratory
OMB Office of Management and Budget
PA Partnership Agreement
PFBS Perfluorobutanesulfonic Acid
PFC Perfluorinated Compounds
PFHpA Perfluoroheptanoic Acid
PFHxS Perfluorohexanesulfonic Acid
PFNA Perfluorononanoic Acid
PFOA Perfluorooctanoic Acid
PFOS Perfluorooctanesulfonic Acid
PT Proficiency Testing
PWS Public Water System
qPCR Quantitative Polymerase Chain Reaction
RFA Regulatory Flexibility Act
RfD Reference Dose
SDWARS Safe Drinking Water Accession and Review System
SM Standard Methods
SRF State Revolving Fund
SBA Small Business Administration

SDWA Safe Drinking Water Act
SDWIS/Fed Federal Safe Drinking Water Information System
TNCWS Transient Non-Community Water System
TTHM Total Trihalomethanes
UCMR Unregulated Contaminant Monitoring Regulation
UMRA Unfunded Mandates Reform Act of 1995
VOC Volatile Organic Compound

Table of Contents

II. Statutory Authority and Background

A. What is the statutory authority for UCMR?

Section 1445(a)(2) of SDWA, as amended in 1996, requires that once every five years, the United States Environmental Protection Agency (EPA) issue a new list of no more than 30 unregulated contaminants to be monitored by public water systems (PWSs). It also requires that EPA enter the monitoring data into the Agency's National Drinking Water Contaminant Occurrence Database (NCOD). EPA must ensure that only a nationally representative sample of PWSs serving 10,000 or fewer people is required to monitor. EPA must also vary the frequency and schedule for monitoring based on the number of persons served, the source of supply, and the contaminants likely to be found.

Section 1445(a)(1)(A) of SDWA, as amended in 1996, requires that every person who is subject to any SDWA requirements establish and maintain such records, make such reports, conduct such monitoring, and provide such information as the Administrator may reasonably require by regulation to assist the Administrator in establishing SDWA regulations. Pursuant to this authority, EPA is requiring the monitoring of total chromium under this final rule.

B. How does EPA meet these statutory requirements?

This final rule fulfills EPA's obligation under SDWA by identifying 29 unregulated contaminants for monitoring during the third UCMR, referred to as ''UCMR 3.'' These contaminants include: 27 chemicals measured using up to seven analytical methods and/or four equivalent consensus organization-developed methods, and two viruses measured using one sample collection and two detection methods. In conjunction with UCMR 3 Assessment Monitoring, monitoring for total chromium is also required. Total chromium monitoring is required under the authority provided in Section 1445(a)(1)(A) of SDWA. EPA has developed the contaminant list (Exhibit 2a and 2b) and sampling design for UCMR 3 (2012–2016) with input from both stakeholders and an EPA–State working group.

Exhibit 2a—UCMR 3 Final Contaminant Lists

List 1, Assessment Monitoring	
1,4-dioxane	vanadium.
molybdenum	strontium.
cobalt	chromium-6 (hexavalent chromium)[1].

1,2,3-trichloropropane	chlorate.
1,3-butadiene	perfluorooctanesulfonic acid (PFOS).
chloromethane (methyl chloride)	perfluorooctanoic acid (PFOA).
1,1-dichloroethane	perfluorononanoic acid (PFNA).
bromochloromethane (Halon 1011)	perfluorohexanesulfonic acid (PFHxS).
bromomethane (methyl bromide)	perfluoroheptanoic acid (PFHpA).
chlorodifluoromethane (HCFC–22)	perfluorobutanesulfonic acid (PFBS).

List 2, Screening Survey

17-β-estradiol	estriol.
17-α-ethynylestradiol (ethinyl estradiol)	equilin.
estrone	testosterone.
4-androstene-3,17-dione.	

List 3, Pre-Screen Testing[2]

enteroviruses	noroviruses.

Exhibit 2b—Total Chromium Monitoring[3]

total chromium

[1] Chromium-6 will be measured as soluble chromate (ion).

[2] Monitoring for microbial indicators—in conjunction with UCMR 3 Pre-Screen Testing—is also required. This monitoring includes sampling for pathogen indicators (*i.e.,* total coliforms, E. coli, bacteriophage, Enterococci and aerobic spores). It is not subject to the stipulation in Section 1445(a)(2)(B)(i) of SDWA that restricts UCMR contaminants to not more than 30. List 3 monitoring, including monitoring of microbial indicators, is only required at selected small systems. EPA will collect the samples from List 3 sampling locations, and will pay for all sampling and analysis costs associated with virus and indicator monitoring at these small systems.

[3] Monitoring for total chromium—in conjunction with UCMR 3 Assessment Monitoring—is required under the authority provided in Section 1445(a)(1)(A) of SDWA.

This list differs from that provided in the March 3, 2011, proposed rule (76 FR 11713, (USEPA, 2011a)) as follows: chromium-6 (hexavalent chromium) and total chromium have been added; *sec*-butylbenzene and *n*-propylbenzene have been deleted; and monitoring of hormones was moved from Assessment Monitoring (List 1) to Screening Survey (List 2).

III. Summary of This Rule

Public water systems (PWS) or EPA will conduct sampling and analysis for Assessment Monitoring (List 1), Screening Survey (List 2), and Pre-Screen Testing (List 3) contaminants, as applicable, at each PWS subject to this rule during a 12 month period within the 2013 to 2015 time frame.

Preparations prior to 2013 include coordination of laboratory approval, selection of representative samples of small systems, development of State Monitoring Plans, establishment of monitoring schedules, and notification of participating PWSs. Exhibit 3 illustrates the major activities that will take place during implementation of UCMR 3.

Exhibit 3: Timeline of UCMR 3 Activities

2012	2013	2014	2015	2016
After proposed rule publication: Lab approval program begins *After applicability date:* EPA/State partnership agreements and State monitoring plans developed (inc. national representative sample) *After final rule publication*: Inform PWSs/establish monitoring plans	**Assessment Monitoring** List 1 Contaminants + Total Chromium All systems serving more than 10,000; 800 systems serving 10,000 or fewer			Complete reporting and analysis of data
	Screening Survey List 2 Contaminants All systems serving more than 100,000; 320 systems serving 10,001 through 100,000; 480 systems serving 10,000 or fewer			
	Pre-Screen Testing List 3 Contaminants + Indicator Organisms 800 non-disinfecting ground water systems in vulnerable areas serving 1,000 or fewer			

EPA generally divides unregulated contaminant monitoring into three types of monitoring, or ''lists.'' ''Assessment Monitoring'' is the largest in scope of the three UCMR monitoring lists or tiers. Under UCMR 3 Assessment Monitoring, 20 ''List 1'' contaminants will be monitored to assess national occurrence in drinking water; total chromium will be monitored in conjunction with Assessment Monitoring. These are the contaminants for which analytical method technologies are well established.

The second tier of UCMR is referred to as ''List 2'' or ''Screening Survey'' monitoring. List 2 contaminants are those with analytical methods that have generally been more recently developed and employ technologies that are not as widely used or laboratory capacity may be insufficient to conduct the larger scale Assessment Monitoring. Under the UCMR 3 Screening Survey, seven ''List 2'' contaminants will be monitored by certain systems (see Exhibit 3).

''Pre-Screen Testing,'' the third tier of UCMR monitoring is generally designed for ''List 3'' contaminants with very new or specialized analytical methods. Under UCMR 3, a selected set of 800 systems that serve fewer than 1,000 retail customers and that do not disinfect are required to assist EPA in sampling their system for two viruses on ''List 3'' and the associated pathogen indicators (*i.e.,* total coliforms, *E. coli,* bacteriophage, *Enterococci* and aerobic spores). This requirement includes community and non-transient, non-community water systems and transient systems.

EPA will pay for the sample kit preparation, sample shipping fees, and analysis costs to minimize the impact of the rule on small systems (those serving 10,000 or fewer people). In addition, no small system will be required to monitor for more than one ''List'' of contaminants. Large systems (those serving more than 10,000 people) will pay for the cost of shipping and laboratory testing for their List 1 and, as applicable, List 2 analyses.

The data collected through the UCMR program are being stored in NCOD to facilitate analysis and review of contaminant occurrence, guide the conduct of the Contaminant Candidate List (CCL) process and support the Administrator in making regulatory decisions for contaminants in the interest of protecting public health, as required under SDWA Section 1412(b)(1). Results of UCMR 1 and 2 monitoring can be viewed by the public at EPA's UCMR Web site: *http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/data.cfm.*

A. What are the major changes between the proposed and final UCMR 3 rule?

EPA published ''Revisions to the Unregulated Contaminant Monitoring Regulation (UCMR 3) for Public Water Systems;'' Proposed Rule, on March 3, 2011 (76 FR 11713, (USEPA, 2011a)). EPA received input from 53 public commenters. After considering the comments, EPA added chromium-6 to the list of unregulated contaminants to be monitored; removed *sec*-butylbenzene and *n*-propylbenzene; and moved monitoring of hormones from Assessment Monitoring to the Screening Survey. EPA is also requiring PWSs to monitor for total chromium concurrent with all chromium-6 monitoring. EPA revised or clarified requirements pertaining to UCMR applicability criteria, reporting, monitoring and quality control. Exhibit 4 provides a summary of these changes and a listing of the corresponding preamble section that provides a more detailed discussion of the revisions and related public comments. Sections III.B–G summarize the different aspects of this rule and the associated major comments received in response to the proposed rule. EPA has compiled a more detailed document containing all public comments and EPA's responses entitled: ''Response to Comments Document for the Unregulated Contaminant Monitoring Regulation (UCMR 3),'' (USEPA, 2012b), which can be obtained by going to *http://www.regulations.gov*, and searching for Docket ID No. EPA–HQ–OW–2009–0090.

EXHIBIT 4—CHANGES TO UCMR 3 BETWEEN PROPOSED AND FINAL RULE

Rule section		Description of change	Corresponding preamble section
Number	Title/description		
141.35(c)(1) and (d)(1)	Data elements	Revise zip code reporting to include only the zip codes for all customers served, rather than those associated with each sampling point.	III.G.2 Sample location and inventory information (zip codes).
141.35(c)(6)(ii) and 141.40(a)(5)(vi)	Reporting schedule	Change laboratory reporting time to 120 days, rather than 60 days; change PWS reporting time to 60 days after laboratory posting, rather than 30 days.	III.G.4 Reporting schedule.
141.40(a)(2)(i)(A) and (a)(2)(ii)(A); and 141.40(a)(3) Table 1.	Analytes to be monitored and related specifications.	Add chromium-6; remove requirement to monitor for *sec*-butylbenzene and *n*-propylbenzene; require total chromium monitoring under SDWA Section 1445 (a)(1)(A); move hormone monitoring to Screening Survey.	III.D.4 Chromium-6 and total chromium, and related methods. III.D.1 List compilation. III.D.2 Hormones and related methods.
141.35(c)(2)	Sample location and inventory information.	Large systems must provide sample location and inventory information to EPA by October 1, 2012.	III.G.4 Reporting schedule.
141.40(a)(3) Table 1, footnote c and 141.40 (a)(4)(i)(C).	Distribution system maximum residence time (DSMRT) sample location.	Revise definition of DSMRT sample required for specific List 1 contaminants.	III.C Where are samples collected? III.D.3 Metals, chlorate, and related methods.
141.35(c)(5)(i) and 141.40 (a)(4)(i)	General rescheduling notification	Large systems may independently change List 1 or List 2 monitoring schedule by October 1, 2012.	III.G.4 Reporting schedule.
141.35(c)(3)	Ground water representative sampling locations.	Large systems may submit representative sampling plan proposals or changes to existing plans by August 1, 2012.	III.C Where are samples collected? III.G.4 Reporting schedule.
141.40(a)(3) Table 1 footnote c	Representative intake	Systems that purchase water from the same wholesaler may sample from a representative intake.	III.C Where are samples collected?

B. Which Water Systems Must Monitor

1. Applicability Based on Population Served

a. This Rule

This rule requires that Assessment Monitoring (for List 1 contaminants) be conducted by all large community and non-transient non-community water systems serving more than 10,000 people, and a nationally representative sample of 800 small water systems serving 10,000 or fewer people; and that the Screening Survey (for List 2 contaminants) be conducted by all large community and non-transient non-community water systems serving more than 100,000 people, a nationally representative sample of 320 large systems serving 10,001 to 100,000 people, and a nationally representative sample of 480 small water systems serving 10,000 or fewer people (as indicated by Federal Safe Drinking Water Information System (SDWIS/Fed) on December 31, 2010). Transient non-community water systems are excluded from Assessment Monitoring and the Screening Survey. In contrast to implementation of UCMR 1 and 2 monitoring, those systems that purchase all of their finished water from another system are not excluded from the requirements of UCMR 3.

b. Summary of Major Comments

EPA received six (6) comments concerning UCMR monitoring based on retail population served. The commenters all agreed that applicability should be based on retail population, although some wanted to exclude those who purchase their water from that applicability. In UCMR 1 and 2, systems that purchased 100% of their water were excluded from monitoring, making estimates of exposure more difficult because many of these purchasing systems represented high-population areas. For UCMR 3, systems that purchase 100% of their water and serve greater than 10,000 people are subject to this rule. Wholesalers that serve a retail population of 10,000 or fewer customers are only required to monitor if they are selected as part of the nationally representative sample of small systems for any list of UCMR contaminants. This should greatly improve exposure estimates for UCMR 3 since exposure estimates will be based on the monitoring data collected from where the water is consumed rather than where it is sold. Between the wholesaler and the purchasing system, contaminant levels may increase (*e.g.,* DBPs or metals) or decrease (*e.g.,* through blending various sources or degradation/chemical reactions).

Some commenters also expressed concern that this applicability change could add an estimated 1,250 systems to the list of those that need to monitor and suggested that this would represent a substantial increase in burden to the drinking water industry. To help mitigate the burden, EPA is allowing those systems that purchase water with multiple connections from the same wholesaler to select a representative connection for sampling. See Section III.C.1.a for further discussion. In addition, EPA notes that approximately 450 wholesale systems will no longer be subject to monitoring; the net increase is approximately 800 systems.

2. Applicability for Transient Systems

a. This Rule

Under UCMR 1 and 2, transient non-community water systems were specifically exempted from monitoring. UCMR 3 now requires participation by transient systems that are selected for Pre-Screen Testing for List 3 contaminants. Under UCMR 3, EPA is conducting Pre-Screen Testing for enterovirus and norovirus, as well as related pathogen indicators, at selected undisinfected ground water systems that serve 1,000 or fewer customers. EPA is including transient systems among the candidate systems—and focusing on viruses at those systems—since viruses are acute pathogens and exposure through a one-time ingestion (*e.g.,* at a transient system) is of potential health concern.

Under 141.40(a)(1) and 141.40(a)(2)(ii)(C), if any system (including transient systems) is notified by EPA or its State that it has been selected for Pre-Screen Testing, the system must permit EPA (at EPA's expense) to sample and analyze for List 3 contaminants and pathogen indicators (*i.e.,* total coliforms, *E. coli,* bacteriophage, *Enterococci* and aerobic spores).

b. Summary of Major Comments

EPA received two (2) comments on including transient non-community systems for List 3 monitoring. One fully supported their inclusion, and the other expressed concern that EPA would not be able to adequately fund the collection and processing of these samples. EPA is confident that it has budgeted sufficient funds to support these activities. As the second commenter noted, transient systems represent a substantial number of the systems serving less than 1,000 customers; therefore, the sampling of these potentially vulnerable systems for these acute pathogens is considered important.

C. Where are samples collected?

1. Entry Point to the Distribution System

a. This Rule

As was the case under UCMR 2, UCMR 3 samples will be collected at entry points to the distribution system (EPTDS). PWSs may perform sampling at representative sampling locations in two cases:

- Demonstrating Representative Ground Water Sampling Locations: Under this rule, large systems that use ground water sources and have multiple EPTDSs can, with prior approval, conduct monitoring at representative sampling locations rather than at each EPTDS. To monitor at representative EPTDSs, large systems must meet the criteria specified in § 141.35(c)(3) and receive approval from EPA or the State. Changes to the rule language clarify that when identifying a representative well, the well must be representative of the highest producing (based on annual volume) and most consistently active wells. In addition, the representative well must be in use at the scheduled sampling time. An alternative location must be sampled if the representative EPTDS is not available at the time of scheduled sampling. This rule establishes a deadline of August 1, 2012 for submission of new proposals or updates to existing plans. See Section III.G.4 for further discussion.
- Representative Intakes from Wholesaler: As specified in § 141.40(a)(3) Table 1, footnote c, systems that purchase water with multiple connections from the same wholesaler may select one representative connection from that wholesaler for UCMR sampling. If a PWS chooses to select a representative intake, each representative intake must receive water from the same source. Additionally, if a PWS chooses to select a representative intake, it must choose a sampling location that represents the highest volume EPTDS connection and is in use at the time of scheduled sampling. If the connection initially selected as the representative EPTDS is not available at the time of scheduled sampling, an alternate representative connection must be sampled.

b. Summary of Major Comments

Five (5) commenters expressed support for EPA's proposal regarding representative sampling points, and representative intakes for PWSs with multiple connections from the same wholesaler; commenters cited cost savings as a benefit of this approach. One commenter also suggested that EPA's approach to representative sampling locations should provide additional flexibility in cases where multiple water systems are receiving water from the same wholesale provider. EPA acknowledges that there are many unique situations with the purchase and sale of drinking water at the wholesale level. In this final rule, EPA has provided clarifying language in § 141.40(a)(3) Table 1, footnote c, specifying that a PWS may select a representative intake from a given wholesaler. EPA is available to advise PWSs regarding choosing the most appropriate sampling site, based on their purchasing situation. However, EPA is requiring all systems that purchase 100% of their water to monitor, for the reasons described in Section III.B.1 of this preamble. Based on the experience of UCMR 1 and UCMR 2, EPA believes it is more appropriate to measure at each purchasing system to more accurately assess exposure. This approach relies on each purchasing system to monitor, thus ensuring the monitoring results reflect any potential water quality changes between the wholesaler and each purchasing system.

2. Distribution System Maximum Residence Time Location

a. This Rule

This rule requires systems that participate in Assessment Monitoring to also sample for total chromium, chromium-6, cobalt, molybdenum, strontium, vanadium, and chlorate both at EPTDSs and in the distribution system. This rule requires systems to collect the samples for these analytes at their distribution system maximum residence time (DSMRT) location(s), (§§ 141.40(a)(3) Table 1, footnote c and 141.40(a)(4)(i)(C)). For clarity, EPA deleted the UCMR reference to the DSMRT specifications under the Stage 1 Disinfection Byproducts Rule at § 141.132(b)(1)(i). EPA now defines DSMRT under UCMR as an active point (*i.e.,* a location that currently provides water to customers) in the distribution system where the water has been in the system the longest relative to the EPTDS. Systems that are subject to the Stage 2 Disinfection By-Products Rule should use their total trihalomethanes (TTHM) highest concentration sampling site(s) as their DSMRT sampling site(s) (USEPA, 2003).

b. Summary of Major Comments

As described in greater detail in Section III.D.3., ''Metals, chlorate, and related methods,'' several commenters suggested that EPA had provided insufficient rationale for requiring DSMRT sampling for cobalt, molybdenum, strontium, vanadium, and chlorate. As elements that may occur in water both naturally, or through industrial activities, cobalt, molybdenum, strontium, and vanadium are expected to be commonly detected in drinking water. EPA believes these metals may be incorporated into pipe deposits and subsequent erosion and/or dissolution may result in waterborne concentrations that differ between the DSMRT and the EPTDS. Regarding chlorate, the use of disinfectants, including use of hypochlorite, chloramines, chlorine dioxide, and ozone can result in chlorate formation. The presence of residual disinfectant in the distribution system and chlorine boosters within the distribution system may result in increases in chlorate concentrations at the DSMRT relative to the EPTDS.

D. What are the UCMR 3 contaminants and associated methods?

1. List Compilation

a. This Rule

EPA is maintaining the list of unregulated contaminants and methods proposed for monitoring with the exception of adding chromium-6, and removing *sec*-butylbenzene and *n*-propylbenzene (see Exhibit 5a). EPA is also requiring PWSs to monitor for total chromium concurrent with all chromium-6 monitoring (Exhibit 5b). The additional data generated by side-by-side measurements of chromium-6 and total chromium will provide valuable information on relative occurrence and the utility of monitoring for total chromium as a surrogate for chromium-6.

Exhibit 5a: 29 Unregulated Analytes and Associated Methods

Assessment Monitoring	
7 Volatile Organic Compounds (VOC) using EPA Method 524.3 (GC/MS): [1]	
1,2,3-trichloropropane	bromomethane (methyl bromide).
1,3-butadiene	bromochloromethane (Halon 1011).
chloromethane (methyl chloride)	chlorodifluoromethane (HCFC–22).
1,1-dichloroethane.	
Synthetic Organic Compound using EPA Method 522 (GC/MS): [2]	
1,4-dioxane.	
4 Metals using EPA Method 200.8 (ICP/MS) [3] **or alternate SM** [4] **or ASTM Methods:** [5]	
cobalt	strontium.
molybdenum	vanadium.
Oxyhalide Anion using EPA Method 300.1 (IC/Conductivity) [6] **or alternate SM** [7] **or ASTM Methods:** [8]	
chlorate.	
6 Perfluorinated Chemicals using EPA Method 537 (LC/MS/MS): [9]	
perfluorooctanesulfonic acid (PFOS)	perfluorohexanesulfonic acid (PFHxS).
perfluorooctanoic acid (PFOA)	perfluoroheptanoic acid (PFHpA).
perfluorononanoic acid (PFNA)	perfluorobutanesulfonic acid (PFBS).
Chromium-6 using EPA Method 218.7 (IC/UV–VIS): [10]	
chromium-6.	
Screening Survey	
7 Hormones using EPA Method 539 (LC/MS/MS): [11]	
17-β-estradiol	estrone.
17-α-ethynylestradiol (ethinyl estradiol)	testosterone.
estriol (16-α-hydroxy-17-β-estradiol)	4-androstene-3,17-dione.
equilin.	

Pre-Screen Testing	
2 Viruses (see Section III.D.5 for methods discussion): [12]	
enterovirus	norovirus.
Exhibit 5b—Total Chromium Monitoring	
Total Chromium using EPA Method 200.8 (ICP/MS) [4] or alternate SM [5] or ASTM Methods: [6]	
total chromium.	

[1] EPA Method 524.3 (GC/MS) (USEPA, 2009a).
[2] EPA Method 522 (GC/MS) (USEPA, 2008).
[3] EPA Method 200.8 (ICP/MS) (USEPA, 1994).
[4] SM 3125 (SM, 21st Ed., 2005).
[5] ASTM D5673–10 (ASTM, 2010).
[6] EPA Method 300.1 (IC/Conductivity) (USEPA, 1997).
[7] SM 4110D (SM, 21st Ed., 2005).
[8] ASTM D6581–08 (ASTM, 2008).
[9] EPA Method 537 (LC/MS/MS) (USEPA, 2009b).
[10] EPA Method 218.7 (IC/UV–VIS) (USEPA, 2011b).
[11] EPA Method 539 (LC/MS/MS) (USEPA, 2010e).
[12] Monitoring also includes sampling for pathogen indicators (*i.e.,* total coliforms, E. coli, bacteriophage, Enterococci and aerobic spores). EPA will pay for all sampling and analysis costs associated with monitoring at these small systems.

b. Summary of Major Comments

Commenters who expressed an opinion about the proposed UCMR 3 analytes were generally supportive. Several commenters suggested that cyanobacterial toxins be added to the list of analytes. EPA agrees that cyanobacterial toxins are of significant interest for future drinking water monitoring. However, EPA currently does not have an available drinking water method for analysis of cyanobacterial toxins. While enzyme-linked immunosorbent assays (ELISA) and high-performance liquid chromatography with UV detection (HPLC/UV) methods have been published (Howard and Boyer, 2007), they do not provide the level of specificity needed for UCMR monitoring. The high-performance liquid chromatography/tandem mass spectrometry (HPLC/MS/MS) methods for cyanobacterial toxins that have been published (Oehrle *et al.,* 2010), do not provide suitable accuracy and precision. EPA has conducted and will continue to conduct methods development research for cyanobacterial toxins both in-house and in cooperation with other laboratories.

2. Hormones and Related Methods

a. This Rule

EPA is revising the requirement for monitoring of the hormones (17-β-estradiol; 17-α-ethynylestradiol; estriol; equilin; estrone; testosterone; and, 4-androstene-3,17-dione), by moving the monitoring from Assessment Monitoring to the Screening Survey.

b. Summary of Major Comments

Three major issues concerning the hormones were raised by commenters. The first was a concern that other than 17-α-ethynylestradiol, the hormones all occur naturally. Based on the low minimum reporting levels (MRLs) specified in this rule, these commenters were concerned that there may be issues with false positives due to background levels of these compounds from samplers.

The ranges of blank results observed during the determination of MRLs are contained in Exhibit 6. In all cases the laboratories easily met the requirement that the concentration of the analytes observed in the blank must be less than one-third of the MRL. In the ''worst case'' the observed blank level equaled one-eighth the MRL. EPA is requiring the collection of field blank samples for UCMR 3 and, to minimize the potential issue of field blank and sample contamination, will provide instructions to both the samplers and the laboratory personnel to wear nitrile gloves when collecting or handling samples for the hormones. These details are specified in EPA's technical manual titled: ''UCMR 3 Laboratory Approval Requirements and Information Document'' (USEPA, 2012d).

EXHIBIT 6—OBSERVED BACKGROUND LEVELS DURING MRL DETERMINATION

Analyte	UCMR MRL (μg/L)	Laboratory 1 (μg/L)	Laboratory 2 (μg/L)	Laboratory 3 (μg/L)
17-β-estradiol	0.0004	ND—0.00006	ND	ND—0.00005
17-α-ethynylestradiol	0.0009	ND—0.00007	ND—0.00008	ND—0.0002
estriol	0.0008	ND—0.00007	ND	ND—0.00006
equilin	0.004	ND—0.00002	ND	ND—0.0005
estrone	0.002	ND—0.0001	0.00001—0.00003	0.02—0.0002
testosterone	0.0001	ND	ND	ND—0.00001
4-androstene-3,17-dione	0.0003	ND	ND	ND—0.000008

ND = Not Detected.

EPA also stipulated in the rule that it will evaluate the situation after six months of monitoring. If at that time, the data indicate that excessive resampling is occurring, EPA will establish alternative MRLs and will notify all affected PWSs and laboratories.

The second issue concerned whether all of the proposed hormones should be monitored (versus a subset of them). There was no consensus among the commenters as to what the ''subset'' should be. Some commenters suggested that monitoring be limited to the five (5)

proposed hormones that are also listed on the final CCL 3 (17-β-estradiol, 17-α-ethynylestradiol, estriol, equilin and estrone). EPA believes that monitoring for testosterone and 4-androstene-3,17-dione is also justified. A number of articles have been published that show the occurrence of testosterone and 4-androstene-3,17-dione in surface waters:

- National Surface Water Reconnaissance (1999–2000): detects of testosterone in 2 (2.8%) of 70 samples at a median concentration of 0.116 µg/L and a maximum concentration of 0.214 µg/L (Kolpin *et al.,* 2002).
- California, Rivers, Irrigation Canals, and Tile Drains (2003–2005): detects of testosterone in 2 (18%) of 11 river samples at a maximum concentration of 0.0006 µg/L; detects in 4 (27%) of 15 irrigation canal samples at a maximum concentration of 0.0019 µg/L; detects in 2 (33%) of 6 tile drain samples at a maximum concentration of <0.0003 µg/L (Kolodziej *et al.,* 2004).
- California Surface Waters (2005–2006): detects of 4-androstene-3,17-dione in 16 (18%) of 89 grazing rangeland surface water samples at a maximum concentration of 0.044 µg/L (Kolodziej and Sedlak, 2007).

In addition, testosterone and 4-androstene-3,17-dione have been shown to be relatively resistant to oxidation (Mash *et al.,* 2010).

The third issue concerned the potential for insufficient laboratory capacity for the monitoring of hormones. Since EPA has moved the hormone monitoring requirement from Assessment Monitoring (List 1) to Screening Survey (List 2), this will substantially reduce the number of PWSs required to monitor for hormones and mitigate any concerns regarding laboratory capacity.

3. Metals, Chlorate, and Related Methods

a. This Rule

This rule requires that samples for the metals—chromium-6, total chromium, cobalt, molybdenum, strontium, and vanadium—as well as chlorate, be collected at one distribution system sampling point per treatment plant (*i.e.,* at the DSMRT) in addition to sampling at the EPTDS. DSMRT samples must be collected at a location that represents the maximum residence time in the distribution system (§§ 141.40(a)(3) Table 1, footnote c and 141.40(a)(4)(i)(C)). (As noted in Section III.C.2.a of this preamble, EPA clarified the DSMRT specifications and deleted the direct DSMRT reference under the Stage 1 Disinfection Byproducts Rule at § 141.132(b)(1)(i).)

EPA is requiring that chlorate samples be collected at both the EPTDS and DSMRT locations to permit the agency to evaluate if chlorate occurs as an oxyhalide disinfection by-product.

b. Summary of Major Comments

Eight (8) commenters suggested that further justification was needed to support monitoring cobalt, molybdenum, strontium, and vanadium at the DSMRT. Three commenters also made similar comments regarding chlorate. Research indicates that vanadium can become incorporated in the corrosion products in iron pipes used for drinking water distribution. As a result, vanadium may be released via dissolution and/or erosion of the mineral deposits that form inside many iron distribution pipes. Gerke *et al.,* (2010) cite research that indicates that relatively minor scouring of these deposits can result in water concentrations of vanadium in excess of 15 µg/L. Similar findings were published by the Water Research Foundation (Friedman *et al.,* 2009). The authors reported vanadium in scaling from several different distribution systems. As a reference point, the Agency for Toxic Substances and Disease Registry (ATSDR) has established an Interim Minimal Risk Level of 0.003 mg/kg/day; a 70 kg adult drinking two liters of water per day would exceed the RfD through water consumption alone if the concentration in the water was greater than 21 µg/L (ATSDR, 2009).

Molybdenum has been identified as being among the heavy metals that can be mobilized from reservoir sediments containing iron and aluminum oxides and hydroxides. Fluctuations in pH of approximately 0.2 pH units were sufficient to considerably affect the release of previously adsorbed molybdenum (Friedman *et al.,* 2009).

Although such findings for cobalt and strontium are not available in the scientific literature, these two elements commonly occur in drinking water. As a result, EPA believes that incorporation of cobalt and/or strontium into pipe deposits within a distribution system could result in mobilization of these metals into drinking water within the distribution system via dissolution and/or erosion. Strontium has been found in greatest amounts in calcium-rich minerals and sediments due to similarities in atomic radii (Fairbridge, 1972). In addition, Friedman *et al.,* (2009) report calcium to be the fourth most concentrated element found in pipe deposit samples. Thus, erosion and/or dissolution of pipe deposits within the distribution system may affect human exposure levels for cobalt, molybdenum, strontium, and vanadium.

The presence of residual disinfectant in the distribution system may result in increases in chlorate concentrations at the DSMRT relative to the EPTDS. The following studies on chlorate formation have linked its presence in treated drinking water to the use of several disinfection processes:

- The generation of chlorine dioxide from chlorite and free chlorine (Gordon *et al.,* 1990; Bolyard *et al.,* 1993; Gallagher *et al.,* 1994);
- The generation of chlorine dioxide from chlorite and hypochlorite (Gallagher *et al.,* 1994);
- Chlorine dioxide oxidation by residual free chlorine (Gordon and Tachiyashiki, 1991; Bolyard *et al.,* 1993);
- Transition metal-catalyzed free chlorine decomposition during disinfection (Gordon *et al.,* 1995);
- Base-catalyzed disproportionation of chlorine dioxide (USEPA, 1999a; Gallagher *et al.,* 1994);
- Photodecomposition of chlorine dioxide (Rice and Gomez-Taylor, 1986; Bolyard *et al.,* 1993; Gallagher *et al.,* 1994; Bergmann and Koparal, 2005);
- Use of chlorate-contaminated hypochlorite solutions—chlorate can come from either the impurity of the original stock solution or decomposition during storage (Bolyard *et al.,* 1992; Bolyard *et al.,* 1993; Gordon *et al.,* 1993; Gordon *et al.,* 1995; Gordon *et al.,* 1997; USEPA, 1999a; WHO, 2005; Snyder *et al.,* 2009; Stanford *et al.,* 2011);
- Use of ozone with residual chlorine (Siddiqui, 1996; von Gunten, 2003); and
- Use of electrochemical disinfection processes (Czarnetzki and Janssen, 1992; Bergmann and Koparal, 2005).

4. Chromium-6 and Total Chromium, and Related Methods

a. This Rule

While EPA did not include chromium-6 in the proposed list of chemicals for UCMR 3 monitoring, EPA did request comment on whether the agency should include it in the final rule due to the concerns about its potential occurrence in public water supplies. EPA also requested comments on whether total chromium should be measured concurrent with chromium-6. Commenters strongly supported requiring monitoring for both chromium-6 and total chromium.

EPA agrees with these commenters and has added chromium-6 to the list of unregulated contaminants to be monitored. EPA is also requiring PWSs to monitor for total chromium concurrent with all chromium-6

monitoring. EPA completed the development and validation of a revised analytical method for the determination of chromium-6 in drinking water, *EPA Method 218.7: Determination of Hexavalent Chromium in Drinking Water by Ion Chromatography with Post-Column Derivatization and UV-Visible Spectroscopic Detection.* This revised method has been extensively studied both within EPA and ion chromatography manufacturers' laboratories as well as through external laboratory validation (USEPA, 2011b).

EPA is using the authority provided in SDWA Section 1445(a)(1)(A) to require monitoring for total chromium in conjunction with the UCMR 3 monitoring of chromium-6. EPA has removed *sec*-butylbenzene and *n*-propylbenzene from UCMR 3. More specifically, the agency has removed *sec*-butylbenzene and *n*-propylbenzene from the UCMR 3 Assessment Monitoring list.

b. Summary of Major Comments

EPA received 30 comments regarding the inclusion of chromium-6 in UCMR 3. Twenty-eight of the 30 commenters supported inclusion. The other two suggested that a health risk from drinking water exposure had not been conclusively established, that regional levels of total chromium in drinking water are very low and that speciation would not be beneficial. The agency believes that the ongoing studies of chromium-6 toxicity warrant UCMR monitoring at this time. EPA believes that collecting national occurrence data will provide beneficial information to the agency regarding how best to protect human health. EPA's second Six-Year Review of National Primary Drinking Water Regulations (USEPA, 2010d) indicated that the levels of total chromium warrant further investigation of chromium-6 occurrence. Chromium can enter the environment from both natural and industrial sources; thus the distribution of both total chromium and chromium-6 may vary based on regional geology and regional industrial activity. Part of the goal of UCMR is to assess the national distribution of the contaminants selected.

Commenters who supported the inclusion of chromium-6 cited two primary reasons for its inclusion in UCMR 3:

- Generating national occurrence data in UCMR 3 will avoid potential delays in any possible regulatory action;
- Monitoring for both total chromium and chromium-6 may allow for determining a relationship between the two species, allowing for possible use of total chromium monitoring, which is less costly and has better holding time requirements, as a surrogate for chromium-6 monitoring.

While generally supporting chromium-6 monitoring in UCMR 3, some commenters expressed concern about the current analytical method. The concerns included procedural issues (*e.g.,* field filtration, preservation and holding time compliance), interferences concerns (*e.g.,* sensitivity and species interconversion prior to sample analysis), the need for round-robin testing of the method laboratory capacity, and the need to determine a lowest concentration minimum reporting level (LCMRL) and MRL for chromium-6. Extensive research by EPA, with support from instrument manufacturers and commercial laboratories, addressed the issues of interferences, sensitivity and analyte preservation. EPA Method 218.7 has undergone peer review, and multi-laboratory LCMRL and MRL determinations have been completed (USEPA, 2011b; USEPA, 2006).

Because UCMR is limited by statute to 30 unregulated contaminants, commenters offered a variety of suggestions for which analyte to remove to accommodate chromium-6. Suggestions included dropping one of the metals, hormones, PFCs, or VOCs. Other suggestions included removing ''the contaminant with the least chance of being detected during monitoring.'' EPA selected *sec*-butylbenzene and *n*-propylbenzene, non-carcinogenic VOCs, for removal after considering data submitted by States that indicated very low occurrence rates. EPA also considered the fact that the currently available health reference levels, 10.3 µg/L and 5.83 µg/L, respectively, are well above the reported levels of occurrence in these data (USEPA, 2012c).

5. Viruses and Related Methods

a. This Rule

EPA is finalizing the requirement for monitoring of the viruses as proposed. This rule requires monitoring for enterovirus and norovirus in UCMR 3 via Pre-Screen Testing of selected undisinfected ground water systems located in karst or fractured bedrock. The monitoring will include 800 PWSs serving 1,000 or fewer customers, including CWSs, and non-transient and transient non-community water systems. Monitoring will also include sampling for pathogen indicators (*i.e.,* total coliforms, *E. coli,* bacteriophage, *Enterococci* and aerobic spores). This monitoring will obtain information concerning the occurrence of enterovirus and norovirus for further evaluation and provide EPA with a better understanding of the co-occurrence of pathogen indicators and viruses.

Enteroviruses will be monitored using one method that has two detection assays. The first is a cell culture assay also used in the Information Collection Rule survey conducted by EPA (61 FR 24353, May 14, 1996 (USEPA, 1996)), with one change; the Virosorb 1–MDS filter will be replaced by the NanoCeram® filter, which will significantly reduce sampling cost. The NanoCeram® filter has proven to be as effective as Virosorb 1–MDS filter for the recovery of enteroviruses (Karim *et al.,* 2009) and noroviruses (Gibbons *et al.,* 2010). The second assay is quantitative polymerase chain reaction (qPCR) based, and detects the viral nucleic acid. Noroviruses will only be monitored using qPCR, as there is no cell culture method available.

Both norovirus and enterovirus qPCR will be performed per the protocol in Lambertini *et al.,* (2008). The qPCR primers and probe for genogroup I norovirus will be as referenced in Jothikumar *et al.,* (2005), while genogroup II Norovirus primers and probe will be as referenced in Ando *et al.,* (1995). Primers and probe referenced in De Leon *et al.,* (1990) and Monpoeho *et al.,* (2000) will be used for enterovirus qPCR.

b. Summary of Major Comments

Several commenters expressed concern about using Method 1615 for monitoring viruses because it has not undergone multi-laboratory validation. EPA notes, however, that individual elements of the method have been used by many researchers worldwide, and the culture assay is, with the exception of a new filter, identical to the Information Collection Rule validated method (FR 24353, May 14, 1996 (USEPA, 1996)). The complete method is published and has undergone thorough peer review as per protocols established by EPA's National Exposure Research Laboratory and consistent with ''The Handbook for Preparing ORD Reports'' (USEPA, 1995). The method has undergone validation at EPA's laboratory, has built in quality controls for PCR inhibition and has positive and negative controls to identify false negative and positive assays. Results from the analysis of initial and ongoing positive and negative proficiency testing (PT) samples will ensure the ability of analysts to perform the method.

Several commenters questioned EPA's use of Borchardt's (2008) data as the basis for including viruses in UCMR 3,

since that work has not been published or undergone peer review. In his study, Borchardt sampled wells from 14 communities in Wisconsin for the presence of enteroviruses and noroviruses. The initial enteric virus RT-qPCR assay results are published in a peer reviewed journal (Hunt *et al.*, 2010). Borchardt's work showed a statistically significant correlation between viral qPCR and self-reported AGI (acute gastrointestinal illness) in the population served. Borchardt's work is also one of the very few studies to assess presence of enteric viruses in undisinfected ground water systems. EPA expects that complete results from Borchardt's work will be published in a peer reviewed journal in the near future. The study results have also been presented at numerous scientific conferences as well as in testimony to the Wisconsin State Senate. A project advisory committee comprised of epidemiologists from the University of California, Berkeley, Michigan State University and the University of Washington provided additional peer review comments during the study planning and data analysis stages.

A few commenters expressed concerns as to whether a survey of 800 undisinfected ground water systems in a sensitive hydrogeology would be nationally representative, noting that only specific geologic regions within the country would be included in the survey. While EPA acknowledges that the 800 undisinfected ground water systems are only a small subset of the total number of systems in the country, the selection of 800 PWSs was statistically derived to be nationally representative of those with sensitive hydrogeology.

EPA also received comments regarding how the agency would use data obtained from a focused and limited occurrence survey, at highly vulnerable and susceptible systems, to provide meaningful data to judge nationwide occurrence and to support regulatory determination. EPA notes that results will provide an understanding of the exposure risks in populations potentially served by a large number of undisinfected systems in karst aquifers nationally. Lastly, some comments addressed the current information on virus-indicator correlation, suggesting that the correlations are weak. EPA notes that most virus-indicator correlation studies have been performed in disinfected systems, not undisinfected ground water systems. EPA also notes that the use of multiple indicators in looking at the correlation will make this monitoring more useful.

6. Perfluorinated Compounds and Related Methods

a. This Rule

EPA is finalizing the requirement for monitoring the perfluorinated compounds (PFCs) as proposed: PFOS, PFOA, PFNA, PFHxS, PFHpA, and PFBS.

b. Summary of Major Comments

EPA received public comments related to several issues with EPA Method 537, used to measure PFCs. These included: The potential for laboratory contamination; concerns that the MRLs developed for the PFCs may be too low or too high; and concerns about the media used to extract the contaminants. EPA successfully tested this method via a multi-laboratory validation and conducted a thorough peer-review process prior to the UCMR 3 proposal. Since then, the method has also been effectively used at additional laboratories. Contamination was not an issue at these laboratories, and they were able to meet the proposed MRLs. While particular laboratories may be able to meet MRLs lower than those proposed, the selected MRLs reflect those achievable by the national array of laboratories that support the program. Regarding the extraction media, the method relies on a very common sorbent (styrene divinylbenzene) that is available from a number of vendors and yields high-quality data.

E. How are laboratories approved for UCMR 3 monitoring?

1. This Rule

All laboratories conducting analyses for UCMR 3 List 1 and List 2 contaminants must receive EPA approval to perform those analyses. Laboratories seeking approval are required to provide EPA with data that demonstrate their successful completion of an initial demonstration of capability (IDC) as outlined in each method, verify successful method performance at the MRLs as specified in this action, and successfully participate in an EPA Proficiency Testing (PT) program for the analytes of interest. On-site audits of candidate laboratories may be conducted. Details of the EPA laboratory approval program are contained in the technical manual titled: ''UCMR 3 Laboratory Approval Requirements and Information Document'' (USEPA, 2012d). This document will be available on the electronic docket at *www.regulations.gov* and will be provided to laboratories that register for the laboratory approval program. In addition, EPA may supply analytical reference standards of known concentrations for select analytes to participating/approved laboratories, where such standards are not readily available through commercial sources.

Pre-Screen Testing (List 3) analyses for viruses and related pathogen indicators (*i.e.,* total coliforms, *E. coli,* bacteriophage, *Enterococci,* and aerobic spores) are organized and paid for by EPA through direct contracts with microbial laboratories. These laboratories are not required to go through the same formal laboratory approval process as the Assessment Monitoring and Screening Survey laboratories; however, they are subject to an analogous laboratory approval process as part of their direct contracts with EPA.

a. Laboratory Approval Process for UCMR 3

The UCMR 3 laboratory approval program is similar to the approval program under UCMR 1 and 2. It is designed to assess and confirm the capability of laboratories to perform analyses using the methods listed in § 141.40(a)(3), Table 1, of this final rule. It will assess whether laboratories meet the required equipment, laboratory performance and data reporting criteria described in this action. This evaluation program is voluntary in that it only applies to laboratories intending to analyze UCMR 3 samples. However, EPA requires water systems to use UCMR 3 approved laboratories when conducting monitoring for those analytes listed in Table 1 of § 141.40(a)(3) of this final rule. A list of laboratories approved for UCMR 3 monitoring is posted to EPA's UCMR Web site: *http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/laboratories.cfm.* Laboratories are encouraged to apply for UCMR 3 approvals as early as possible, as schedules for large PWS sampling will be completed soon after the final rule is promulgated. The steps for the laboratory approval process are listed in the following paragraphs, b through f.

b. Request To Participate

Laboratories must contact EPA and request to participate in the UCMR 3 laboratory approval program. Laboratories must send their request to: UCMR 3 Laboratory Approval Coordinator, USEPA, Technical Support Center, 26 West Martin Luther King Drive (MS 140), Cincinnati, OH 45268; or email at: *UCMR_Sampling_Coordinator@epa.gov.* EPA began accepting requests for registration for the List 1 (Assessment Monitoring) and List 2 (Screening Survey) methods on March 03, 2011.

The final opportunity for a laboratory to request the necessary registration forms is August 1, 2012.

c. Registration

Each laboratory that wishes to participate in UCMR 3 monitoring must complete a registration form. Registration information includes the following: laboratory name, mailing address, shipping address, contact name, phone number, email address and a list of the UCMR 3 methods for which the laboratory is seeking approval. The registration step provides EPA with the necessary contact information and ensures that each laboratory receives a customized application package of materials and instructions for the methods that it plans to use.

d. Application Package

When EPA receives the registration information, a customized application package will be emailed to the laboratory for completion. Information requested in the application includes the following: IDC data, including precision, accuracy and results of MRL studies; information regarding analytical equipment; proof of current drinking water laboratory certification (for any currently regulated chemical); and example chromatograms for each method under review.

The laboratory must post UCMR 3 monitoring results (on behalf of its PWS clients) to EPA's UCMR electronic data reporting system as a condition of maintaining EPA approval.

e. EPA Review of Application Package

EPA will review the application package and, if necessary, request follow-up information. The laboratory must satisfactorily complete this portion of the process before they can participate in the UCMR 3 PT program.

f. Proficiency Testing (PT)

A PT sample is a synthetic sample containing a concentration of an analyte that is known to EPA, but unknown to the laboratory being tested. To complete the initial laboratory approval process, a laboratory must meet specific acceptance criteria for the analysis of a UCMR 3 PT sample(s) for each method for which the laboratory is seeking approval. Initial laboratory approval is contingent upon successful completion of a PT study. EPA will offer two to four opportunities for a laboratory to successfully analyze UCMR 3 PT samples. Two of these studies were conducted prior to the publication of this final rule and at least one study will be conducted after publication of the final rule. Under this approach laboratories could complete their portion of the laboratory approval process prior to publication of this final rule, and therefore receive their approval immediately following the publication of this final rule. Alternatively, laboratories could wait until this final rule is published before completing the required laboratory approval analyses. A laboratory must pass one of the PT studies for each analytical method for which they are requesting approval. Laboratories applying for UCMR 3 approval and laboratories conducting UCMR 3 analyses may be subject to on-site laboratory audits. No PT studies will be conducted after the start of monitoring; however, laboratory audits will be ongoing throughout the entire monitoring period of 2013–2015. Continued laboratory approval is contingent upon successful participation in any audits conducted by EPA.

g. Written EPA Approval

After laboratories successfully complete steps ''b'' through ''f'' of the laboratory approval process, EPA will send the laboratory a letter listing the method(s) for which approval is granted.

2. Summary of Major Comments

Three (3) commenters suggested that EPA modify the requirements for PT samples in UCMR 3 by including a round of PT samples during the UCMR 3 monitoring period in addition to the initial round of PT samples conducted prior to monitoring. Instead of requiring laboratories to conduct ongoing PT samples, EPA will conduct ongoing laboratory audits similar to the process under UCMR 2. Ongoing laboratory audits will allow EPA to evaluate each laboratory's analytical processes for all aspects of sample receipt, storage, processing, analysis and reporting of routine samples. This will provide a better mechanism, compared to an additional PT study, for uncovering any potential data issues and ensuring that laboratories meet the quality requirements.

F. How were minimum reporting levels determined?

1. This Rule

Lowest Concentration Minimum Reporting Levels (LCMRLs) and Minimum Reporting Levels (MRLs) for each analyte were determined through an EPA LCMRL study assessing the data from multiple laboratories prior to publication of the UCMR 3 proposal. The LCMRL is defined as the lowest spiking concentration at which recovery of between 50 and 150% is expected 99% of the time by a single analyst.

The LCMRL is estimated using advanced statistical procedures that have been incorporated into an LCMRL calculator tool that is available on EPA's Web site (*http://water.epa.gov/scitech/drinkingwater/labcert/analyticalmethods_ogwdw.cfm*). The tool estimates a probability distribution for spike recovery as a function of spiking concentration.

MRL

EPA revised the definition of the MRL used in UCMR 2 (72 FR 367, January 4, 2007 (USEPA, 2007)). The revised definition reflects improvements in the statistical procedures for determining the LCMRL and MRL. These improvements were implemented by EPA to make the models more robust, *i.e.,* so that the models can accommodate a wider range of observed LCMRL data sets (USEPA, 2010f). The MRL for an analyte measured by a specified analytical method is designed to be an estimate of an LCMRL that is achievable, with 95% confidence, by a capable analyst/laboratory at least 75% of the time. Such a demonstration of ability to reliably make quality measurements at the MRL is intended to achieve high quality measurements across the nation's laboratories.

In UCMR 3, EPA estimated the MRL for an analyte/method by obtaining data from several laboratories performing corresponding LCMRL studies. These data were used to construct an approximation to the distribution that would result from picking at random a laboratory/analyst proficient in performing the analytical method and having them perform an LCMRL study and compute an LCMRL estimate. The strategy for computing the MRL is two-fold. First, for each LCMRL data set, a distribution for repeated LCMRL determinations by the same laboratory/analyst is estimated by generating a large number of simulated values. Second, these values are combined to create an estimated overall distribution. If a result from one of the laboratories is significantly higher than that of other laboratories, this value would be down-weighted using a robust weight function. The resulting weighted values are used to construct a probability distribution from which the MRL is computed as the 95th percentile.

2. Summary of Major Comments

Several commenters remarked on the complexity of the procedures for determining the LCMRL and the MRL. These commenters were concerned

about the amount of time and effort needed to calculate LCMRLs and MRLs. Some suggested that as an alternative, EPA use the procedure developed for consideration by the Clean Water Act as part of the Federal Advisory Committee on Detection and Quantitation. As a point of clarification, EPA notes that laboratories that participate in UCMR 3 do not need to use the LCMRL and MRL procedures. Instead, laboratories that participate in UCMR 3 will be required to demonstrate their ability to meet the already-established UCMR 3 analyte MRLs by analyzing reagent water samples spiked at or below the established UCMR 3 MRLs. This initial demonstration of capability (IDC) requirement, as described in EPA's ''UCMR 3 Laboratory Approval Requirements and Information Document,'' is no more complex than determining a Method Detection Limit (MDL) (USEPA, 2012d).

A diverse selection of laboratories representing different sizes, experience and business status were selected to participate in the EPA LCMRL studies (as described previously in this section). For transparency, EPA will provide summary tables showing all LCMRL results for UCMR 3 in the docket (USEPA, 2012d).

With regard to comments that the MRLs are being set well below health reference levels (HRLs) in certain cases, EPA believes that this is appropriate because new health effects data may become available in the future that result in lower HRLs.

G. What are the UCMR 3 reporting requirements?

1. General Reporting Requirements/ SDWARS

a. This Rule

Under this rule, EPA is committed to pre-populating the inventory and monitoring data in the reporting system (Safe Drinking Water Accession and Review System (SDWARS)), using data from UCMR 2 and SDWIS/Fed information. For PWSs subject to UCMR 3 that have data in SDWARS from UCMR 2, EPA will transfer data to ''SDWARS 3'' (*i.e.,* the SDWARS update associated with UCMR 3). For water systems that are new to UCMR, EPA will pull the available information from SDWIS/Fed and coordinate with States and EPA Regions for their input where possible. EPA has loaded the available information into SDWARS 3 prior to the publication of this final rule. PWSs will have until October 1, 2012, to update, edit, or change their information or monitoring schedule in SDWARS 3 (see Section III.G.4 for further discussion of reporting deadlines).

b. Summary of Major Comments

Several commenters expressed concern over possible inefficiencies related to data entry into SDWARS, including concern over duplication of past efforts (*e.g.,* having to re-enter information for each sample point for each sampling event) and time spent identifying representative sampling locations at both the EPTDS and DSMRT for UCMR 2. Commenters further noted it would be very helpful if elements that are duplicated for each sample would be automatically pre-filled in each field once the information was entered the first time. As noted, for UCMR 3, EPA plans to preload as much inventory to SDWARS as possible and is taking commenter suggestions into consideration in its design updates to SDWARS. The pre-loaded data will include representative sampling locations previously identified as the EPTDS and DSMRT locations. PWSs will be asked to verify their inventory in SDWARS and large systems may be required to revise this information once their ground water representative monitoring plan has been approved, depending on the level of their State's involvement. See Section III.G.4 for discussion of reporting deadlines.

2. Sample Location and Inventory Information (Zip Codes)

a. This Rule

This final rule establishes a requirement for reporting zip codes associated with all PWS customers. EPA had proposed the reporting of sampling point U.S. Postal Service Zip Codes and the zip codes of all customers served by a given sampling point (as part of the reporting associated with Data Element 4—Sampling Point Identification Code). Obtaining the zip code of the sampling point was intended to assist with future vulnerability assessments. Zip codes that tie populations served to each sampling point were intended to assist with future occurrence and exposure analyses. However, based on stakeholder concerns about the burden associated with reporting this information and concerns about the usefulness of having the zip code of the sampling point, EPA revised the rule language to establish a requirement of only reporting zip codes for customers served by the PWS. These reporting specifications are now established in §§ 141.35(c)(1) and (d)(1) for large and small systems, respectively. EPA believes that required reporting of customer zip codes will provide EPA with useful information for future occurrence analyses.

b. Summary of Major Comments

Eight (8) comments were received regarding the proposed zip code reporting requirements. Most commenters believed that reporting the zip code for each sampling point location would not provide EPA with the information necessary to make future correlations between water quality and the areas served by the water being distributed. After considering public comments, EPA has revised the reporting requirement to only include the zip codes served by the PWS.

3. Disinfectant Type Specifications

a. This Rule

EPA is changing Data Element 6, in Table 1 of 141.35(e). Under UCMR 2, this data element was established to provide information on ''Disinfectant Residual Type'' as it related to monitoring for nitrosamines (part of UCMR 2 Screening Survey monitoring). EPA is modifying the definition of this data element to account for changes to the analyte and monitoring specifications between UCMR 2 and UCMR 3. This revised definition lists additional disinfectant types to provide more specific information on the sources and types of disinfectant schemes that may lead to chlorate formation/occurrence in drinking water.

b. Summary of Major Comments

While commenters were supportive of the collection of these data, several commenters noted that the requirement for reporting this data element was unclear. Some commenters noted that PWSs frequently use multiple disinfectants and reporting only one of those would provide an inaccurate assessment of disinfectants being used. Others noted that EPA needed to make sure that PWSs indicate whether their hypochlorite solution was generated on or off site (onsite: Essentially no storage of stock solution will be needed; offsite: The storage of stock solution will be needed).

EPA agrees that the presentation of the requirements warranted clarification and has revised the list of disinfectants. EPA will clearly indicate in the data reporting system (SDWARS) that PWSs should identify all of the disinfectants used to treat the water.

4. Reporting Schedule

a. This Rule

To help ensure that monitoring and reporting are conducted as scheduled,

UCMR 3 specifies several deadlines related to initial reporting of inventory and scheduling information, as well as reporting of monitoring data. Several deadlines were newly proposed for UCMR 3 (*i.e.,* not used for UCMR 1 or UCMR 2) and finalized in this rule, and some are revised in this final rule to ensure that UCMR 3 is implemented as scheduled. These deadlines are being established to allow EPA enough time to review and process the information, and complete the planning process for UCMR 3 monitoring to begin on January 1, 2013. Changes in deadlines only affect large systems. There are no changes to small system reporting schedules. The schedule changes that are finalized in this rule include:

- Inventory and Scheduling: Large systems that are subject to UCMR 3 must report their inventory and sampling location information (141.35(c)(2)), and any proposed changes to their monitoring schedule (141.35(c)(5)(i) and 141.40(a)(4)(i)) no later than October 1, 2012. As noted, EPA has loaded existing information into SDWARS 3 prior to the publication of this final rule. PWSs will have until October 1, 2012, to update, edit or change their inventory and sample location information or monitoring schedule in SDWARS 3.
- Ground water representative monitoring plans: As described in 141.35(c)(3), large systems that use ground water sources and that have multiple EPTDSs can, with prior approval, conduct monitoring at representative sampling locations rather than at each EPTDS. For systems that have existing approved representative monitoring plans, their approved sampling location information will be pre-loaded into SDWARS and systems must review and confirm, or update this information by October 1, 2012. This rule establishes a deadline of August 1, 2012, for submitting a new ground water representative plan to be reviewed by the State or EPA.
- Monitoring data: This rule re-establishes two deadlines related to reporting of monitoring data: Large systems must require their laboratories to post data to SDWARS within 120 days of sample collection; and large systems must review, approve and submit the data to their State and EPA within 60 days of when the laboratory posts the data. These time frames are specified in 141.35(c)(6)(ii) and 141.40(a)(5)(vi).

b. Summary of Major Comments

Five (5) comments were received on the reduced laboratory reporting time frame. Most commenters did not support the 60-day proposed time frame for laboratories to post data to SDWARS and expressed several concerns: that laboratories may see increased workload due to additional monitoring; that UCMR 3 methods are not in common use and are very sensitive, so greater validation of results may be required; and that field blank analysis may be required for some methods, resulting in longer turnaround times for sampling results. Commenters did not believe that the reduced reporting time frame would increase compliance with monitoring schedules. Seven comments were also received regarding the 30-day proposed time frame for large PWSs to review and approve their data. The majority of the commenters requested the time frame be returned to the 60-day period used under UCMR 1 and 2. Commenters believe the shortened time frame would not give PWSs sufficient time to conduct a full data review and that schedule coordination among multiple staff would be difficult. After considering the public comments, EPA returned the laboratory reporting time frame to 120 days after sample collection (same as earlier UCMRs) and returned the PWS reporting time frame to 60 days after laboratory posting data (same as earlier UCMRs).

IV. State and Tribal Participation

A. Partnership Agreements

1. This Rule

Under UCMR 3, States may continue to have a role in rule implementation through Partnership Agreements (PAs). Because specific activities for individual States are identified and established through the PAs, not through rule language, this rule does not contain reference to PAs.

2. Summary of Major Comments

EPA received no comments regarding State participation in UCMR 3.

B. Governors' Petition and State-Wide Waivers

1. This Rule

This rule retains the UCMR 1 and 2 language that, consistent with SDWA, allows a minimum of seven State Governors to petition EPA to add contaminants to the UCMR Contaminant list. This rule also retains the UCMR 1 and 2 language that allows States to waive monitoring requirements with EPA approval and under very limited conditions.

2. Summary of Major Comments

EPA received no comments regarding the governor's petition or state-wide waiver allowances of UCMR 3.

V. Cost and Benefits of This Rule

In this rule, EPA finalizes a new set of contaminants for monitoring in the third five-year UCMR monitoring period. UCMR 3 also incorporates modifications to improve the rule design. UCMR 3 Assessment Monitoring (for List 1 contaminants) will be conducted from January 2013 through December 2015 by 800 systems serving 10,000 or fewer people, and by all systems serving more than 10,000 people. The 800 small systems will be randomly selected for List 1 monitoring. The UCMR 3 Screening Survey (for List 2 contaminants) will be conducted from January 2013 through December 2015 by all systems serving a population of greater than 100,000 people, a nationally representative set of 320 systems serving between 10,001 and 100,000 people, and a nationally representative set of 480 systems serving fewer than 10,000 people. The nationally representative sets of 320 and 480 systems will both be randomly selected for List 2 monitoring. The Pre-Screen Testing for List 3 contaminants will also be conducted from January 2013 through December 2015 in 800 undisinfected ground water systems serving 1,000 or fewer persons. No small system will be selected for more than one UCMR 3 monitoring list.

It is assumed for this cost estimate that one-third of systems will monitor during each of the three monitoring years. Labor costs pertain to systems, States, and EPA. They include activities such as reading the regulation, notifying systems selected to participate, training water system staff on sample collection procedures, sample collection, including travel time to collect samples, data review, reporting, and record keeping. Non-labor costs will be incurred primarily by EPA and by large PWSs. They include the cost of shipping samples to laboratories for testing and the cost of the actual laboratory analyses.

In this rule, EPA specifies seven EPA-developed analytical methods and four equivalent consensus organization developed methods to monitor for 27 unregulated chemical contaminants, two viruses, and total chromium. While this preamble also describes the analytical methods that will be used for virus monitoring, the rule does not address these methods. Laboratory approval for virus monitoring is not addressed since all of the analyses for the two viruses will be conducted in laboratories under EPA contract and at EPA's expense. Estimated system and EPA costs are based on the analytical costs for all UCMR 3 methods. With the

exception of Methods 200.8 and 300.1, these methods are comparatively new and will not coincide with other compliance monitoring (*i.e.,* no cost savings for concurrent monitoring can be realized).

Laboratory analysis and shipping of samples account for approximately 82% of the total national cost for UCMR 3 implementation. These costs are calculated as follows: the number of systems, multiplied by the number of sampling locations, multiplied by the sampling frequency, multiplied by the unit cost of laboratory analysis. Under UCMR 3, for List 1 Assessment Monitoring and List 2 Screening Survey, surface water (and ground water under the direct influence of surface water (GWUDI)) sampling points will be monitored four times during the applicable year of monitoring, and ground water sample points will be monitored twice during the applicable year of monitoring. Systems will monitor for the metals—cobalt, molybdenum, vanadium, strontium, chromium-6, and total chromium—as well as chlorate, at their EPTDS sampling locations and at one distribution system sampling point per treatment plant (*i.e.,* at the DSMRT). Pre-Screen Testing systems will monitor two times during the three year monitoring period (2013 through 2015) at their EPTDS.

Following publication of the proposed rule and EPA's initial cost and burden estimates, EPA received several cost-related public comments. Several suggested that EPA's estimates of cost and burden (*e.g.,* laboratory and estimated labor burden) to PWSs were too low. EPA estimates of laboratory fees are based on consultations with commercial drinking water laboratories and a review of the costs of similar analytical methods. In response to comments, EPA revisited the analytical method cost estimates. EPA approached four commercial drinking water laboratories and requested pricing estimates for UCMR 3 methods, including the cost of field blanks for methods 524.3 (VOCs), 537 (PFCs), and 539 (hormones). EPA averaged the estimates from the four laboratories and updated the cost figures, which resulted in increased cost estimates for some methods.

With respect to per-system burden estimates, EPA notes that all estimates represent average burden hours, which include surface water systems that may have very few sampling points, and thus lower sampling burden, as well as those systems with higher numbers of sampling points that would have greater labor burden. Moreover, a system's burden is primarily incurred during its one year of required UCMR monitoring (between January 2013 and December 2015). However, in compliance with the requirements of the Paperwork Reduction Act (44 U.S.C. 3501 et seq.), these cost and burden estimates are presented as an average over the applicable three-year information collection request (ICR) period (2012–2014). Small systems (those serving 10,000 or fewer people) will have the lowest burden not only because they generally have fewer sampling locations, but also because these systems will receive substantial direct assistance from EPA and/or their State.

The total cost of Assessment Monitoring analyses is estimated at $1,085 per sample set. The total cost of the single Screening Survey method is estimated at $418 per sample set. Field blank analyses costs are further described in ''Information Collection Request for the Unregulated Contaminant Monitoring Regulation (UCMR 3)'' (USEPA, 2012a). The cost to EPA of the Pre-Screen analyses for viruses and related pathogen indicators (*i.e.,* total coliforms, *E. coli,* bacteriophage, *Enterococci,* and aerobic spores) is estimated at $1,880 per sample set. Shipping estimates are added to the calculated costs to derive the total direct analytical non-labor costs. Estimated shipping costs were based on the average cost of shipping a 25-pound package.

In preparing the UCMR 3 ICR, EPA relied on standard assumptions and data sources used in the preparation of other drinking water program ICRs. These include the PWS inventory, number of sampling points per system, and labor rates. EPA expects that States will incur only labor costs associated with voluntary assistance with UCMR 3 implementation. State costs were estimated using the relevant modules of the State Resource Model that was developed by the Association of State Drinking Water Administrators (ASDWA) in conjunction with EPA (ASDWA, 2003) to help States forecast resource needs. Model estimates were adjusted to account for actual levels of State participation under UCMR. Because State participation is voluntary, level of effort will vary across States and depend on their individual agreements with EPA.

Over the UCMR implementation period of 2012–2016, EPA estimates that nationwide, the annual cost of UCMR 3 is approximately $17.45 million, of which water systems and States will pay approximately $13.3 million; and EPA will pay $4.14 million (most of which is associated with small system monitoring). These total estimated annual costs (labor and non-labor) are incurred as follows:

Respondent	Avg. annual cost. all respondents (2012–2016)
Small Systems (25–10,000), including labor only, non-labor costs paid for by EPA	$0.066 m
Large Systems (10,001–100,000), including labor and non-labor costs	9.55 m
Very Large Systems (100,001 and greater), including labor and non-labor costs	2.94 m
States, including labor costs related to implementation coordination	0.75 m
EPA, including labor for implementation, non-labor for small system testing	4.14 m
Average Annual National Total [1]	17.45 m

[1] Average Annual National Total of $17.45 million is based on rounding.

Over the period of 2012–2016, EPA estimates that nationwide, the total cost of UCMR 3 is approximately $87 million, of which water systems and States will pay approximately $66 million and EPA will pay $21 million.

Additional details regarding EPA's cost assumptions and estimates can be found in the ICR amendment prepared for this final rule (Office of Management and Budget (OMB) number 2040—NEW), which presents estimated cost and burden for the 2012–2014 period (USEPA, 2012a). Estimates of costs over the entire five-year UCMR 3 period of 2012–2016 are attached as an appendix to the ICR. Copies of the ICR and its amendment may be obtained from the EPA public docket for this final rule

under Docket ID Number EPA–HQ–OW–2009–0090.

VI. Statutory and Executive Order Reviews

A. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563: Improving Regulation and Regulatory Review

Under Executive Order 12866 (58 FR 51735, October 4, 1993) and Executive Order 13563 (76 FR. 3821, January 21, 2011, this action is a ''significant regulatory action.'' Accordingly, EPA submitted this action to the Office of Management and Budget (OMB) for review under Executive Orders 12866 and 13563 (76 FR 3821, January 21, 2011) and any changes made in response to OMB recommendations have been documented in the docket for this action.

In addition, EPA prepared an analysis of the potential costs and benefits associated with this action. This analysis is contained in the ''Information Collection Request for the Unregulated Contaminant Monitoring Regulation (UCMR 3)'' (USEPA, 2012a). A copy of the analysis is available in the docket for this action and the analysis is briefly summarized in Section V of the preamble of this final rule.

B. Paperwork Reduction Act

The information collection requirements in this rule have been submitted for approval to the Office of Management and Budget (OMB) under the *Paperwork Reduction Act,* 44 U.S.C. 3501 *et seq.* The information collection requirements are not enforceable until OMB approves them.

The information collected under this final rule fulfills the statutory requirements of Section 1445(a)(2) of SDWA, as amended in 1996. The data collected will describe the source of the water, location, and test results for samples taken from PWSs. The concentrations of any identified UCMR contaminants will be evaluated in conjunction with health effects information and will be considered for future regulation accordingly. Reporting is mandatory. The data are not subject to confidentiality protection.

The annual burden and cost estimates described in this section are for the implementation assumptions described in Section V. Cost and Benefits of the Rule. Respondents to the UCMR 3 will include 2,080 small water systems (800 for Assessment Monitoring, 480 for Screening Survey, and 800 for Pre-Screen Testing), the 4,215 large PWSs (those serving more than 10,000 people), and the 56 States and Primacy agencies (6,351 total respondents). The frequency of response varies across respondents and years. System costs (particularly laboratory analytical costs) vary depending on the number of sampling locations. For cost estimates, it is assumed that systems will conduct sampling evenly across January 2013 through December 2015 (*i.e.,* one-third of systems in each of the 3 consecutive 12-month periods). Because the applicable ICR period is 2012–2014, the third year of monitoring activity (*i.e.,* January through December of 2015) is not captured in the current ICR estimates.

The burden and cost estimates presented in this section represent average costs. In some cases, the costs are presented as an annual average. Average burden or cost per system was derived by calculating total costs, and dividing by the total number of systems expected to monitor during the ICR years of 2012–2014. Average annual burden or cost per system was derived by summing total costs (or burden), dividing by the number of systems expected to monitor during the ICR years of 2012–2014, and then dividing by three years. The total costs and the annual average costs over the ICR years of 2012–2014 are presented in Exhibit 7. Total and annual average costs for the entire 5-year UCMR 3 period can be found in the ICR for UCMR 3, available in the docket for this final rule.

Small systems (those serving 10,000 or fewer) that are selected for UCMR 3 monitoring will sample an average of 1.8 times per system (*i.e.,* number of responses per system) across the three-year ICR period of 2012–2014. The average burden per response for small systems is estimated to be 3.8 hours. Large systems (those serving 10,001 to 100,000 people) and very large systems (those serving more than 100,000 people) will sample and report an average of 2.7 and 3.7 times per system, respectively, across the three-year ICR period of 2012–2014. The average burden per response for large and very large systems is estimated to be 9.2 and 10.2 hours, respectively. States are assumed to have an average of 1.0 response per year (3.0 responses per State across the three-year ICR period of 2012–2014), related to coordination with EPA and systems, with an average burden per response of 233 hours. In aggregate, during the ICR period of 2012–2014, the average response (*e.g.,* responses from systems and States) is associated with a burden of 11.6 hours, with a labor plus non-labor cost of $4,218 per response. Exhibit 7 presents respondent burden and cost estimates for the ICR period of 2012–2014.

EXHIBIT 7—UCMR 3 PER RESPONDENT BURDEN AND COST SUMMARY FOR THE ICR PERIOD

[2012–2014]

Burden (hours)/cost (dollars)	Small systems	Large systems	Very large systems	States	National average
Three-Year Total per Respondent					
Total # of Responses per Respondent	1.8	2.7	3.7	3.0	2.5
Labor Cost per Respondent	$160	$775	$1,437	$41,975	$1,160
Non-Labor Cost per Respondent	$0	$11,785	$34,181	$0	$9,237
Total Cost (Labor plus Non-Labor)	$160	$12,560	$35,619	$41,975	$10,397
Total Cost per Response	$89	$4,677	$9,704	$13,992	$4,218
Total Burden per Respondent (hr)	6.9	24.8	37.5	700.1	28.7
Total Burden per Response (hr)	3.8	9.24	10.2	233.4	11.6
Average Annual per Respondent					
Avg. # of Responses per Respondent	0.6	0.9	1.2	1.0	0.8
Labor Cost per Respondent	$53	$258	$479	$13,992	$387
Non-Labor Cost per Respondent	$0	$3,928	$11,394	$0	$3,079
Avg. Cost (Labor plus Non-Labor)	$53	$4,187	$11,873	$13,992	$3,466
Avg. Cost per Response	$30	$1,559	$3,235	$4,664	$1,406
Avg. Burden per Respondent (hr)	2.3	8.3	12.5	233.4	9.6

EXHIBIT 7—UCMR 3 PER RESPONDENT BURDEN AND COST SUMMARY FOR THE ICR PERIOD—Continued

[2012–2014]

Burden (hours)/cost (dollars)	Small systems	Large systems	Very large systems	States	National average
Avg. Burden per Response (hr)	1.3	3.1	3.4	61.3	3.9

The average per respondent burden hours and costs per year for the ICR period of 2012–2014 are: small systems—2.3 hour burden at $53 for labor; large systems—8.3 hours at $258 for labor, and $3,928 for analytical costs; very large systems—12.5 hours at $479 for labor, and $11,394 for analytical costs; and States—233.4 hours at $13,992 for labor. Burden is defined at 5 CFR 1320.3(b).

Exhibit 8 shows the annual and total national cost and burden for UCMR 3 implementation over the ICR period of 2012–2014.

EXHIBIT 8—UCMR 3 ANNUAL NATIONAL COST AND BURDEN

[2012–2014]

Cost (in millions)	2012	2013	2014	Total
Small System Costs	$0	$0.11	$0.11	$0.22
Large System Costs	0	15.92	15.92	31.84
Very Large System Costs	0	4.90	4.90	9.81
State Costs	0.33	1.0	1.0	2.4
EPA Costs	0.92	6.63	6.57	14.12
Total Cost	1.26	28.55	28.53	58.34

Total Burden (thousands of hours) for All Responses	2012	2013	2014	Total
Small Systems	0	4.8	4.8	9.5
Large Systems	0	31.5	31.5	62.9
Very Large Systems	0	5.2	5.2	10.3
States	13.3	13.6	12.2	39.2
EPA	5.7	11.4	11.4	28.6
Total Burden	19.1	66.5	65.1	150.6

An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations in 40 CFR are listed in 40 CFR part 9. When this ICR is approved by OMB, the agency will publish a technical amendment to 40 CFR part 9 in the **Federal Register** to display the OMB control number for the approved information collection requirements contained in this final rule.

C. Regulatory Flexibility Act

The Regulatory Flexibility Act (RFA) generally requires an agency to prepare a regulatory flexibility analysis of any rule subject to notice and comment rulemaking requirements under the Administrative Procedure Act or any other statute unless the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities. Small entities include small businesses, small organizations, and small governmental jurisdictions.

The RFA provides default definitions for each type of small entity. Small entities are defined as: (1) A small business as defined by the Small Business Administration's (SBA) regulations at 13 CFR 121.201; (2) 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; and (3) a small organization that is any ''not-for-profit enterprise which is independently owned and operated and is not dominant in its field.'' However, the RFA also authorizes an agency to use alternative definitions for each category of small entity, ''which are appropriate to the activities of the agency'' after proposing the alternative definition(s) in the **Federal Register** and taking comment (5 U.S.C. 601(3)–(5)). In addition, to establish an alternative small business definition, agencies must consult with SBA's Chief Counsel for Advocacy.

For purposes of assessing the impacts of this rule on small entities, EPA considered small entities to be PWSs serving 10,000 or fewer people, because this is the system size specified in SDWA as requiring special consideration with respect to small system flexibility. As required by the RFA, EPA proposed using this alternative definition in the **Federal Register** (63 FR 7606, February 13, 1998 (USEPA, 1998a)), requested public comment, consulted with the SBA, and finalized the alternative definition in the Consumer Confidence Reports rulemaking (63 FR 44512, August 19, 1998 (USEPA, 1998b)). Consistent with that Final Rule, the alternative definition has been applied to this regulation.

After considering the economic impacts of this rule on small entities, I certify that this action will not have a significant economic impact on a substantial number of small entities. The small entities directly regulated by this rule are PWSs serving 10,000 or fewer people. EPA has determined that the small entities subject to the requirements of this rule are a subset of the small PWSs (those serving 10,000 or fewer people). The agency has determined that 2,080 small PWSs (across Assessment Monitoring, Screening Survey, and Pre-Screen

Testing), or approximately 3% of small systems, will experience an impact of no more than 0.4% of revenues; the remainder of small systems will not be impacted.

Although this final rule will not have a significant economic impact on a substantial number of small entities, EPA has tried to reduce the impact of this rule on small entities. To ensure that this rule will not have a significant economic impact on a substantial number of small entities, EPA will assume all costs for analyses of the samples and for shipping the samples from these systems to the laboratories contracted by EPA to analyze UCMR 3 samples. EPA has set aside $2.0 million each year from the State Revolving Fund (SRF) with its authority to use SRF monies for the purposes of implementing this provision of SDWA. Thus, the costs to these small systems will be limited to the labor hours associated with 2,080 small systems assisting EPA in collecting UCMR samples and preparing them for shipping.

The evaluation of the overall impact on small systems, summarized in the preceding discussion, is further described as follows. EPA analyzed the impacts for privately-owned and publicly-owned water systems separately due to the different economic characteristics of these ownership types, such as different rate structures and profit goals. For both publicly- and privately-owned systems, EPA used the ''revenue test,'' which compares annual system costs attributed to the rule to the system's annual revenues. Median revenue data from the 2006 Community Water System Survey Volume II: Detailed Tables and Survey Methodology (*http://water.epa.gov/aboutow/ogwdw/upload/cwssreportvolumeII2006.pdf*) were used for public and private water systems. EPA assumes that the distribution of the sample of participating small systems will reflect the proportions of publicly- and privately-owned systems in the national inventory. The estimated distribution of the representative sample, categorized by ownership type, source water, and system size, is presented in Exhibit 9.

EXHIBIT 9—NUMBER OF PUBLICLY- AND PRIVATELY-OWNED SMALL SYSTEMS SUBJECT TO UCMR 3

System size (number of people served)	Publicly-owned	Privately-owned	Total
Ground Water			
500 and under	134	402	536
501 to 3,300	548	208	757
3,301 to 10,000	286	66	352
Subtotal GW	968	677	1,645
Surface Water (and GWUDI)			
500 and under	7	9	16
501 to 3,300	98	35	133
3,301 to 10,000	222	64	286
Subtotal SW	327	108	435
Total of Small Water Systems	1,295	785	2,080

The basis for the UCMR 3 RFA certification for this final rule is as follows: for the 2,080 small water systems that will be affected, the average annual costs for complying with this rule represent 0.4% of system revenues (the highest estimated percentage is for ground water systems serving 500 or fewer people, at 0.40% of its median revenue). Exhibit 10 presents the annual costs to small systems and to EPA for the small system sampling program, along with an illustration of system participation for each year of the UCMR 3 program.

EXHIBIT 10—EPA AND SYSTEMS COSTS FOR IMPLEMENTATION OF UCMR 3 AT SMALL SYSTEMS

Cost description	2012	2013	2014	2015	2016	Total
Costs to EPA for Small System Program (including Assessment Monitoring, Screening Survey, and Pre-Screen Testing).	$0	$5,407,233	$5,407,233	$5,407,233	$0	$16,221,698
Costs to Small Systems including Assessment Monitoring, Screening Survey, and Pre-Screen Testing.	0	$110,720	110,720	110,720	0	332,160
Total Costs to EPA and Small Systems for UCMR 3:.	0	$5,517,953	5,517,953	5,517,953	0	16,553,858
System Monitoring Activity Timeline: [1]						
Assessment Monitoring		⅓ PWSs Sample.	⅓ PWSs Sample.	⅓ PWSs Sample.		800
Screening Survey		⅓ PWSs Sample.	⅓ PWSs Sample.	⅓ PWSs Sample.		480

EXHIBIT 10—EPA AND SYSTEMS COSTS FOR IMPLEMENTATION OF UCMR 3 AT SMALL SYSTEMS—Continued

Cost description	2012	2013	2014	2015	2016	Total
Pre-Screen Testing		⅓ PWSs Sample.	⅓ PWSs Sample.	⅓ PWSs Sample.		800

[1] Total number of systems is 2,080. No small system conducts more than one type of monitoring study.

System costs are attributed to the labor required for reading about their requirements, training staff on requirements, monitoring, including travel time needed to collect samples, reporting, and record keeping. The estimated average annual burden across the five-year UCMR 3 implementation period of 2012–2016 is estimated to be 1.4 hours at $32 per small system. Average annual cost, in all cases, is less than or equal to 0.40% of system revenues. As required by SDWA, the agency specifically structured the rule to avoid significantly affecting small entities by assuming all costs for laboratory analyses, shipping, and quality control for small entities. As a result, EPA incurs the entirety of the non-labor costs associated with UCMR 3 small system monitoring, or 98% of total small system testing costs. Exhibits 11 and 12 present the estimated economic impacts in the form of a revenue test for publicly- and privately-owned systems.

EXHIBIT 11—UCMR 3 RELATIVE COST ANALYSIS FOR SMALL PUBLICLY-OWNED SYSTEMS (2012–2016)

System size (number of people served)	Annual number of systems impacted	Average annual hours per system (2012–2016)	Average annual cost per system (2012–2016)	Revenue test [1] (%)
Ground Water Systems				
500 and under	27	1.14	$24.16	0.08
501 to 3,300	110	1.24	27.67	0.02
3,301 to 10,000	57	1.57	39.71	0.01
Surface Water (and GWUDI) Systems				
500 and under	1	1.63	34.71	0.06
501 to 3,300	20	1.69	37.74	0.02
3,301 to 10,000	44	1.79	45.35	0.005

[1] The ''Revenue Test'' was used to evaluate the economic impact of an information collection on small government entities (*e.g.,* publicly-owned systems); costs are presented as a percentage of median annual revenue in each size category.

EXHIBIT 12—UCMR 3 RELATIVE COST ANALYSIS FOR SMALL PRIVATELY-OWNED SYSTEMS (2012–2016)

System size (number of people served)	Annual number of systems impacted	Average annual hours per system (2012–2016)	Average annual cost per system (2012–2016)	Revenue Test[1] (%)
Ground Water Systems				
500 and under	80	1.14	$24.16	0.40
501 to 3,300	42	1.24	27.67	0.02
3,301 to 10,000	13	1.57	39.74	0.004
Surface Water (and GWUDI) Systems				
500 and under	2	1.63	34.71	0.10
501 to 3,300	7	1.69	37.74	0.01
3,301 to 10,000	13	1.79	45.35	0.005

[1] The ''Revenue Test'' was used to evaluate the economic impact of an information collection on small private entities (*e.g.,* privately-owned systems); costs are presented as a percentage of median annual revenue in each size category.

EPA specifically solicited additional comment on the proposed action on small systems. No comments were received.

D. Unfunded Mandates Reform Act (UMRA)

This rule does not contain a Federal mandate that may result in expenditures of $100 million or more for State, local, and tribal governments, in the aggregate, or the private sector in any one year. Total annual costs of this final rule (across the implementation period of 2012–2016), for State, local, and Tribal governments and the private sector, are estimated to be $17.45 million, of which EPA will pay $4.14 million, or approximately 24%. Thus, this rule is not subject to the requirements of Sections 202 or 205 of UMRA.

This rule is also not subject to the requirements of Section 203 of UMRA because it contains no regulatory requirements that might significantly or uniquely affect small governments. As noted previously, the agency expects to pay for the reasonable costs of sample analysis for the small PWSs required to monitor for unregulated contaminants under this final rule, including those owned and operated by small governments. The only costs that small systems will incur are labor costs attributed to collecting the UCMR samples and packing them for shipment

to the laboratory (EPA will pay for shipping). These costs are minimal. They are not significant or unique. Thus, this rule is not subject to the requirements of UMRA Section 203.

E. Executive Order 13132: Federalism

This action does not have federalism implications. 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 Executive Order 13132. The cost to State and local governments is minimal and the rule does not preempt State law. Thus, Executive Order 13132 does not apply to this action. In the spirit of Executive Order 13132, and consistent with EPA policy to promote communications between EPA and State and local governments, EPA specifically solicited comment on the proposed action from State and local officials.

F. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments

Subject to the Executive Order 13175 (65 FR 67249, November 9, 2000) EPA may not issue a regulation that has tribal implications, that imposes substantial direct compliance costs, and that is not required by statute, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by tribal governments, or EPA consults with tribal officials early in the process of developing the proposed regulation and develops a tribal summary impact statement.

EPA has concluded that this action will have tribal implications. However, it will neither impose substantial direct compliance costs on tribal governments, nor preempt Tribal law. As described previously, this final rule requires monitoring by all large systems (*i.e.,* those serving 10,001 to 100,000 people) and all very large systems (*i.e.,* those serving greater than 100,000 people); 17 Tribal water systems have been identified as large systems based on information in the SDWIS/Fed water system inventory. EPA estimates the average annual cost to each of these large systems, over the five-year rule period, to be less than $2,512 (total cost of about $12,560 per system during the five-year rule period). This cost is based on a labor component (associated with the collection of samples) and a non-labor component (associated with shipping and laboratory fees) and represents less than 0.09% of average revenue/sales for large systems. UCMR also requires monitoring by a nationally representative sample of small systems (*i.e.,* those serving 10,000 or fewer people). EPA estimates that approximately one percent of small Tribal systems will be selected as part of a nationally representative sample for Assessment Monitoring, Screening Survey or Pre-Screen Testing. EPA estimates the average annual cost to the small Tribal systems, over the five year rule period to be $32 (total cost of about $160 per system over the five-year rule period). Such cost is based on the labor associated with collecting a sample and preparing it for shipping and represents 0.4% or less of average revenue/sales for small systems. All other small system expenses (associated with shipping and laboratory fees) are paid by EPA.

EPA consulted with tribal officials early in the process of developing UCMR to permit them to have meaningful and timely input into its development. In developing the original UCMR rule, EPA held stakeholder meetings and prepared background information for stakeholder review. EPA sent requests for review of stakeholder documents to nearly 400 Tribes, Tribal organizations, and small systems organizations to obtain their input. Representatives from the Indian Health Service (IHS) Sanitary Deficiency System and Tribes were consulted regarding decisions on rule design, the design for the statistical selection of small systems, and potential costs. Tribes raised issues concerning the selection of the nationally representative sample of small systems, particularly the manner in which Tribal systems would be considered under the sample selection process. EPA developed the sample frame for Tribal systems and Alaska Native water systems in response to those concerns. EPA worked with the Tribes, Alaska Natives, the IHS, and the States to determine how to classify each Tribal system for consideration in the statistically-based selection of the nationally representative sample of small systems. As a result of those discussions, small PWSs located in Indian country in each of the EPA Regions containing Indian country were evaluated as part of a Tribal category that receives selection consideration comparable to that of small systems outside of Indian country. Thus, Tribal systems have the same probability of being selected as other water systems in the stratified selection process that weighs systems by water source and size class by population served. This final rule maintains the basic program design of UCMR 1 and 2, and continues to build upon the structure of this cyclical program. As part of the development of this rule, EPA held a public stakeholder meeting on April 7, 2010. This meeting was announced to the public in a **Federal Register** notice dated February 23, 2010 (75 FR 8063 (USEPA, 2010a)). Prior to the meeting, background materials and rule development information were sent to specific stakeholders, including representatives from the IHS and the Native American Water Association.

EPA specifically solicited additional comment on the proposed action from tribal officials. EPA received no comments.

G. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks

This action is not subject to EO 13045 because it is not an economically significant regulation pursuant to EO 12866.

H. Executive Order 13211: Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use

This action is not a ''significant energy action'' as defined in Executive Order 13211 (66 FR 28355 (May 22, 2001)), because it is not likely to have a significant adverse effect on the supply, distribution, or use of energy. None of the final UCMR requirements involve actions that use a significant amount of energy.

I. National Technology Transfer and Advancement Act

Section 12(d) of the National Technology Transfer and Advancement Act of 1995 (''NTTAA''), Public Law 104–113, 12(d) (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 otherwise impractical. Voluntary consensus standards are technical standards (*e.g.,* materials specifications, test methods, sampling procedures, and business practices) that are developed or adopted by voluntary consensus standards bodies. NTTAA directs EPA to provide Congress, through OMB, explanations when the agency decides not to use available and applicable voluntary consensus standards.

This rulemaking involves technical standards. EPA has decided to use the methods developed by the agency as well as voluntary consensus standards for the analysis of UCMR 3 contaminants. The agency conducted a search of potentially applicable voluntary consensus standards and identified two major organizations

whose methods are acceptable for determinations under UCMR. These organizations are Standard Methods (SM) and ASTM International. For many of the parameters included in this final action, EPA was unable to identify methods from voluntary consensus method organizations that were appropriate for the monitoring required. However, EPA identified acceptable consensus method organization standards for the analysis of total chromium, vanadium, molybdenum, cobalt, strontium and chlorate. Therefore, EPA is approving analytical methods published by EPA, SM, and ASTM International for these analytes.

J. Executive Order 12898: Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations

Executive Order (EO) 12898 (59 FR 7629 (Feb. 16, 1994)) establishes federal executive policy on environmental justice. Its main provision directs federal agencies, to the greatest extent practicable and permitted by law, to make environmental justice part of their mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of their programs, policies, and activities on minority populations and low-income populations in the United States.

EPA has determined that this final rule will not have disproportionately high and adverse human health or environmental effects on minority or low-income populations. By seeking to identify unregulated contaminants that may pose health risks via drinking water from all PWSs, UCMR furthers the protection of public health for all citizens, including minority and low-income populations using public water supplies. UCMR uses a statistically-derived set of systems for the nationally representative sample that is population-weighted within each system size and source water category so that any PWS within a category has an equivalent likelihood of selection. Additionally, EPA is requiring that PWSs report all U.S. Postal Service Zip Codes in their service area. This additional data element will be used in the evaluation of UCMR 3 occurrence data and could potentially identify areas that have disproportionately high and adverse human health or environmental effects on minority or low-income populations.

K. Congressional Review Act

The Congressional Review Act, 5 U.S.C. 801 et seq., as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of the rule in the **Federal Register**. A Major rule cannot take effect until 60 days after it is published in the **Federal Register**. This action is not a ''major rule'' as defined by 5 U.S.C. 804(2). This rule will be effective June 1, 2012.

VII. Public Involvement in Regulation Development

EPA's Office of Ground Water and Drinking Water routinely engages stakeholders in its regulatory activities for the purpose of providing early input to regulation development. When designing and developing the UCMR program in the late 1990s, EPA held meetings for developing the CCL, establishing the information requirements of the NCOD, and selecting priority contaminants for UCMR monitoring. During the initial development of the UCMR program, stakeholders including PWSs, States, industry, and other organizations attended meetings to discuss the UCMR. Seventeen other meetings were held specifically concerning UCMR development. For a description of public involvement activities related to the first UCMR (UCMR 1), please see the discussion in the September 17, 1999 UCMR Final Rule **Federal Register** at 64 FR 50556 (USEPA, 1999b).

Specific to the development of UCMR 3, a stakeholder meeting was held on April 7, 2010, in Washington, DC. There were 22 attendees, representing State agencies, laboratories, PWSs, environmental groups, and drinking water associations. The topics of presentations and discussions included: Status of UCMR 2; rationale for developing the new list of potential contaminants; analytical methods that could be used in measuring these contaminants; sampling design; procedure for determining LCMRLs; laboratory approval; and other potential revisions based on lessons learned during implementation of UCMR 1 and UCMR 2 (see USEPA, 2010b for presentation materials, and USEPA, 2010c for meeting notes).

EPA requested public comment on the proposed rule (76 FR 11713, March 3, 2011 (USEPA, 2011a)), and established a public docket, under Docket ID No. EPA–HQ–OW–2009–0090. Each set of comments received in response to this request was assigned an EPA Document ID (EPA–HQ–OW–2009–0090+unique four digit extension) and posted for public access on regulations.gov. To view comments, search for the docket ID on the regulations.gov homepage, then click the link to public submissions.

EPA received feedback on UCMR 3 from 53 commenters. Commenters included: private citizens; local and State governments as well as U.S. territories; industry and industry groups; drinking water systems and organizations; and, non-governmental organizations, such as environmental and health advocacy groups. An overview of key comments received is included in Section III of this rule, and the complete report of comments and full EPA responses can be found in the docket on regulations.gov (USEPA, 2012b).

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List of Subjects

40 CFR Part 141

Environmental protection, Chemicals, Incorporation by reference, Indian-lands, Intergovernmental relations, Radiation protection, Reporting and recordkeeping requirements, Water supply.

40 CFR Part 142

Environmental protection, Administrative practices and procedures, Chemicals, Indian lands, Radiation protection, Reporting and recordkeeping requirements, Water supply.

Dated: April 16, 2012.

Lisa P. Jackson,

Administrator.

For the reasons set out in the preamble, Title 40, chapter I of the Code of Federal Regulations is amended as follows:

PART 141—NATIONAL PRIMARY DRINKING WATER REGULATIONS

■ 1. The authority citation for Part 141 continues to read as follows:

Authority: 42 U.S.C. 300f, 300g–1, 300g–2, 300g–3, 300g–4, 300g–5, 300g–6, 300j–4, 300j–9, and 300j–11.

Subpart C—Monitoring and Analytical Requirements

■ 2. Section 141.23 is amended in the table to paragraph (k)(1) by revising entries 18, 19, and 20; by revising footnotes 3, 4, 5, 6, 7, 8, 13, 19, and 22; and by removing footnote 23.

The revisions read as follows:

§ 141.23 Inorganic chemical sampling and analytical requirements.

* * * * *

(k) * * *

(1) * * *

Contaminant	Methodology [13]	EPA method	ASTM [3]	SM [4] (18th, 19th ed.)	SM [4] (20th ed.)	SM online [22]	Other
*	*	*	*	*	*	*	
18. Nitrate	Ion Chromatography	300.0 [6], 300.1 [19]	D4327–97, 03	4110 B	4110 B	4110 B–00	B–1011 [8]
	Automated Cadmium Reduction	353.2 [6]	D3867–90 A	4500–NO_3 F	4500–NO_3 F	4500–NO_3 F–00	
	Ion Selective Electrode			4500–NO_3 D	4500–NO_3 D	4500–NO_3 D–00	601 [7]
	Manual Cadmium Reduction		D3867–90 B	4500–NO_3 E	4500–NO_3 E	4500–NO_3 E–00	
	Capillary Ion Electrophoresis		D6508–00.				
19. Nitrite	Ion Chromatography	300.0 [6], 300.1 [19]	D4327–97, 03	4110 B	4110 B	4110 B–00	B–1011 [8]
	Automated Cadmium Reduction	353.2 [6]	D3867–90 A	4500–NO_3 F	4500–NO_3 F	4500–NO_3 F–00	
	Manual Cadmium Reduction		D3867–90 B	4500–NO_3 E	4500–NO_3 E	4500–NO_3 E–00	
	Spectrophotometric			4500–NO_2 B	4500–NO_2 B	4500–NO_2 B–00	
	Capillary Ion Electrophoresis		D6508–00				

Contaminant	Methodology [13]	EPA method	ASTM [3]	SM [4] (18th, 19th ed.)	SM [4] (20th ed.)	SM online [22]	Other
20. Ortho-phosphate	Colorimetric, Automated, Ascorbic Acid	365.1 [6]		4500–P F	4500–P F		
	Colorimetric, ascorbic acid, single reagent.		D515–88 A	4500–P E	4500–P E		
	Colorimetric Phosphomolybdate; Automated-segmented flow; Automated Discrete.						I–1601–85 [5] I–2601–90 [5] I–2598–85 [5]
	Ion Chromatography	300.0 [6], 300.1 [19]	D4327–97, 03	4110 B	4110 B	4110 B–00	
	Capillary Ion Electrophoresis		D6508–00				

* * * * *

[3] *Annual Book of ASTM Standards,* ASTM International, 100 Barr Harbor Drive, West Conshohocken, PA 19428, *http://www.astm.org.;* Annual Book of ASTM Standards 1994, Vols. 11.01 and 11.02; Annual Book of ASTM Standards 1996, Vols. 11.01 and 11.02; Annual Book of ASTM Standards 1999, Vols. 11.01 and 11.02; Annual Book of ASTM Standards 2003, Vols. 11.01 and 11.02.

[4] *Standard Methods for the Examination of Water and Wastewater,* American Public Health Association, 800 I Street NW., Washington, DC 20001–3710; *Standard Methods for the Examination of Water and Wastewater,* 18th edition (1992); *Standard Methods for the Examination of Water and Wastewater,* 19th edition (1995); *Standard Methods for the Examination of Water and Wastewater,* 20th edition (1998).The following methods from this edition cannot be used: 3111 B, 3111 D, 3113 B, and 3114 B.

[4] *Standard Methods for the Examination of Water and Wastewater,* American Public Health Association, 800 I Street NW., Washington, DC 20001–3710; *Standard Methods for the Examination of Water and Wastewater,* 18th edition (1992); *Standard Methods for the Examination of Water and Wastewater,* 19th edition (1995); *Standard Methods for the Examination of Water and Wastewater,* 20th edition (1998).The following methods from this edition cannot be used: 3111 B, 3111 D, 3113 B, and 3114 B.

[5] U.S. Geological Survey, Federal Center, Box 25286, Denver, CO 80225–0425; Methods for Analysis by the U.S. Geological Survey National Water Quality Laboratory—Determination of Inorganic and Organic Constituents in Water and Fluvial Sediment, Open File Report 93–125, 1993; Techniques of Water Resources Investigation of the U.S. Geological Survey, Book 5, Chapter A–1, 3rd edition, 1989.

[6] ''Methods for the Determination of Inorganic Substances in Environmental Samples,'' EPA/600/R–93/100, August 1993. Available as Technical Report PB94–120821 at National Technical Information Service (NTIS), 5301 Shawnee Road, Alexandria, VA 22312. *http://www.ntis.gov.*

[7] The procedure shall be done in accordance with the Technical Bulletin 601 ''Standard Method of Test for Nitrate in Drinking Water,'' July 1994, PN 221890–001, Analytical Technology, Inc. Copies may be obtained from ATI Orion, 529 Main Street, Boston, MA 02129.

[8] Method B–1011. ''Waters Test Method for Determination of Nitrite/Nitrate in Water Using Single Column Ion Chromatography,'' August, 1987. Copies may be obtained from Waters Corporation, Technical Services Division, 34 Maple Street, Milford, MA 01757, Telephone: 508/482–2963, Fax: 508/482–4056.

* * * * *

[13] Because MDLs reported in EPA Methods 200.7 and 200.9 were determined using a 2x preconcentration step during sample digestion, MDLs determined when samples are analyzed by direct analysis (*i.e.,* no sample digestion) will be higher. For direct analysis of cadmium and arsenic by Method 200.7, and arsenic by Method 3120 B, sample preconcentration using pneumatic nebulization may be required to achieve lower detection limits. Preconcentration may also be required for direct analysis of antimony, lead, and thallium by Method 200.9; antimony and lead by Method 3113 B; and lead by Method D3559–90D, unless multiple in-furnace depositions are made.

* * * * *

[19] ''Methods for the Determination of Organic and Inorganic Compounds in Drinking Water,'' Vol. 1, EPA 815–R–00–014, August 2000. Available as Technical Report PB2000–106981 at National Technical Information Service (NTIS), 5301 Shawnee Road, Alexandria, VA 22312. *http://www.ntis.gov.*

* * * * *

[22] Standard Methods Online, American Public Health Association, 800 I Street NW., Washington, DC 20001, available at *http://www.standardmethods.org.* The year in which each method was approved by the Standard Methods Committee is designated by the last two digits in the method number. The methods listed are the only online versions that may be used.

■ 3. Section 141.35 is amended as follows:

■ a. In paragraph (a) by revising the third sentence,

■ b. By revising paragraph (b) introductory text,

■ c. By revising paragraph (b)(1),

■ d. In paragraph (b)(2) by revising the first sentence,

■ e. By revising paragraph (c)(1),

■ f. By revising paragraph (c)(2),

■ g. In paragraph (c)(3)(i) by removing ''May 4, 2007'' and adding in its place, ''August 1, 2012,''

■ h. In paragraph (c)(3)(ii) by adding a new second and third sentence,

■ i. In paragraph (c)(4) by removing ''June 4, 2007'' and adding in its place, ''October 1, 2012,''

■ j. By revising paragraph (c)(5)(i),

■ k. By revising paragraph (c)(6) introductory text,

■ l. By revising paragraph (c)(6)(ii),

■ m. By revising paragraph (d)(1),

■ n. By revising paragraph (d)(2), and

■ o. In the table to paragraph (e) by revising entry 6.

The revisions and additions read as follows:

§ 141.35 Reporting for unregulated contaminant monitoring results.

(a) * * * For the purposes of this section, PWS ''population served'' is the retail population served directly by the PWS as reported to the Federal Safe Drinking Water Information System (SDWIS/Fed); wholesale or consecutive populations are not included. * * *

(b) *Reporting by all systems.* You must meet the reporting requirements of this paragraph if you meet the applicability criteria in § 141.40(a)(1) and (2).

(1) *Where to submit UCMR reporting requirement information.* Some of your reporting requirements are to be fulfilled electronically and others by mail. Information that must be submitted using EPA's electronic data reporting system must be submitted through: *http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/reporting.cfm.* Documentation that is required to be mailed can be submitted either: To UCMR Sampling Coordinator, USEPA, Technical Support Center, 26 West Martin Luther King Drive (MS 140), Cincinnati, OH 45268; or by email at *UCMR_Sampling_Coordinator@epa.gov*. In addition, you must notify the public of the availability of unregulated contaminant monitoring data as provided in Subpart Q (Public Notification) of this part (40 CFR 141.207). Community Water Systems that detect unregulated contaminants under this monitoring must also address such detections as part of their Consumer Confidence Reports, as provided in Subpart O of this part (40 CFR 141.151).

(2) * * * If you have received a letter from EPA concerning your required monitoring and your system does not meet the applicability criteria for UCMR established in § 141.40(a)(1) or (2), or if a change occurs at your system that may affect your requirements under UCMR as defined in § 141.40(a)(3) through (5), you must mail or email a letter to EPA, as specified in paragraph (b)(1) of this section. * * *

* * * * *

(c) * * *

(1) *Contact and zip code information.* You must provide contact information by October 1, 2012, and provide updates within 30 days if this information changes. The contact information must be submitted using EPA's electronic data reporting system, as specified in paragraph (b)(1) of this section, and include the name, affiliation, mailing address, phone number, and email address for your PWS Technical Contact and your PWS Official. In addition, as a one-time reporting requirement, you must report the U.S. Postal Service Zip Code(s) for all areas being served water by your PWS.

(2) *Sampling location and inventory information.* You must provide your sampling location and inventory information by October 1, 2012, using EPA's electronic data reporting system. You must submit, verify or update the following information for each sampling location, or for each approved representative sampling location (as specified in paragraph (c)(3) of this section regarding representative sampling locations): PWS identification (PWSID) code; PWS facility identification code; water source type, sampling point identification code; and

sampling point type code; (as defined in Table 1 of paragraph (e) of this section). If this information changes, you must report updates, including new sources and sampling locations that are put in use before or during the PWS' UCMR sampling period, to EPA's electronic data reporting system within 30 days of the change.

* * * * *

(3) * * *

(ii) * * * The proposed well must be representative of the highest annual volume producing and most consistently active wells in the representative array. If that representative well is not in use at the scheduled sampling time, you must select and sample an alternative representative well. * * *

* * * * *

(5) * * *

(i) *General rescheduling notification requirements.* Large systems may change their Assessment Monitoring (List 1) or Screening Survey (List 2) schedules up to October 1, 2012, using EPA's electronic data reporting system, as specified in paragraph (b)(1) of this section. After these dates have passed, if your PWS cannot sample according to your assigned sampling schedule (*e.g.,* because of budget constraints, or if a sampling location will be closed during the scheduled month of monitoring), you must mail or email a letter to EPA, as specified in paragraph (b)(1) of this section, prior to the scheduled sampling date. You must include an explanation of why the samples cannot be taken according to the assigned schedule, and you must provide the alternative schedule you are requesting. You are subject to your assigned UCMR sampling schedule or the schedule that you revised on or before October 1, 2012, unless and until you receive a letter from EPA specifying a new schedule.

* * * * *

(6) *Reporting monitoring results.* For each sample, you must report all data elements specified in Table 1 of paragraph (e) of this section, using EPA's electronic data reporting system. You also must report any changes, relative to what is currently posted, made to data elements 1 through 6 to EPA, in writing, explaining the nature and purpose of the proposed change, as specified in paragraph (b)(1) of this section.

* * * * *

(ii) *Reporting schedule.* You must ensure that your laboratory posts the data to EPA's electronic data reporting system within 120 days from the sample collection date (sample collection must occur as specified in § 141.40(a)(4)). You have 60 days from when the laboratory posts the data in EPA's electronic data reporting system to review, approve, and submit the data to the State and EPA, at the Web address specified in paragraph (b)(1) of this section. If you do not electronically approve and submit the laboratory data to EPA within 60 days of the laboratory's posting data to EPA's electronic reporting system, the data will be considered approved by you and available for State and EPA review.

* * * * *

(d) * * *

(1) *Contact and zip code information.* EPA will send you a notice requesting contact information for key individuals at your system, including name, affiliation, mailing address, phone number and email address. These individuals include your PWS Technical Contact and your PWS Official. You are required to provide this contact information within 90 days of receiving the notice from EPA as specified in paragraph (b)(1) of this section. If this contact information changes, you also must provide updates within 30 days of the change, as specified in paragraph (b)(1) of this section. In addition, as a one-time reporting requirement, you must report the U.S. Postal Service Zip Code(s) for all areas being served water by your PWS.

(2) *Reporting sampling information.* You must record all data elements listed in Table 1 of paragraph (e) of this section on each sample form and sample bottle provided to you by the UCMR Sampling Coordinator. You must send this information as specified in the instructions of your sampling kit, which will include the due date and return address. You must report any changes made in data elements 1 through 6 by mailing or emailing an explanation of the nature and purpose of the proposed change to EPA, as specified in paragraph (b)(1) of this section.

(e) * * *

TABLE 1—UNREGULATED CONTAMINANT MONITORING REPORTING REQUIREMENTS

Data Element	Definition
* * * * * * *	
6. Disinfectant Type	All of the disinfectants that have been added to the water being sampled. To be reported by systems for each sampling point, with possible choices being: CLGA= Gaseous chlorine. CLOF = Offsite Generated Hypochlorite (stored as a liquid form). CLON = Onsite Generated Hypochlorite (no storage). CAGC = Chloramine (formed from gaseous chlorine). CAOF = Chloramine (formed from offsite hypochlorite). CAON = Chloramine (formed from onsite hypochlorite). CLDO = Chlorine dioxide. OZON = Ozone. ULVL = Ultraviolet Light. OTHD = All Other Types of Disinfectant. NODU = No Disinfectant Used.
* * * * * * *	

Subpart E—Special Regulations, Including Monitoring Regulations and Prohibition on Lead Use

■ 4. Section 141.40 is amended as follows:
■ a. By revising paragraph (a) introductory text,
■ b. By revising paragraph (a)(1),
■ c. By revising paragraph (a)(2)(i) introductory text,
■ d. By revising the first sentence of paragraph (a)(2)(i)(A),
■ e. By revising paragraph (a)(2)(ii) introductory text,
■ f. By revising paragraph (a)(2)(ii)(A),
■ g. By revising paragraph (a)(2)(ii)(C),
■ h. By revising paragraph (a)(3),
■ i. In paragraph (a)(4)(i) introductory text by removing ''August 2, 2007'' and adding in its place, ''October 1, 2012'',
■ j. By revising paragraph (a)(4)(i)(B),
■ k. By revising paragraph (a)(4)(i)(C),
■ l. In paragraph (a)(4)(i)(D) by removing the last sentence,
■ m. By revising paragraph (a)(4)(ii)(G),
■ n. In paragraph (a)(5)(ii) by removing ''April 4, 2007'' and adding in its place, ''August 1, 2012'' and by revising the last sentence,
■ o. By revising paragraph (a)(5)(iii) introductory text,
■ p. By revising paragraph (a)(5)(iii)(A)(*1*),
■ q. By revising paragraph (a)(5)(iv),
■ r. By revising paragraph (a)(5)(vi), and
■ s. By adding paragraph (c).

The revisions and addition read as follows:

§ 141.40 Monitoring requirements for unregulated contaminants.

(a) *General applicability.* This section specifies the monitoring and quality control requirements that must be followed if you own or operate a public water system (PWS) that is subject to the Unregulated Contaminant Monitoring Regulation (UCMR), as specified in paragraphs (a)(1) and (2) of this section. In addition, this section specifies the UCMR requirements for State and Tribal participation. For the purposes of this section, PWS ''population served,'' ''State,'' '' PWS Official,'' ''PWS Technical Contact,'' and ''finished water'' apply as defined in § 141.35(a). The determination of whether a PWS is required to monitor under this rule is based on the type of system (*e.g.,* community water system, non-transient non-community water system, etc.), and its retail population, as indicated by SDWIS/Fed on December 31, 2010.

(1) *Applicability to transient non-community systems.* If you own or operate a transient non-community water system, and you are notified by your State or EPA, you must permit the State, EPA or their contractors to collect samples for the contaminants specified on List 3 of Table 1, in paragraph (a)(3) of this section.

(2) * * *

(i) *Large systems.* If you own or operate a retail PWS (other than a transient non-community system) that serves more than 10,000 people, you must monitor according to the specifications in this paragraph (a)(2)(i). If you believe that your applicability status is different than EPA has specified in the notification letter that you received, or if you are subject to UCMR requirements and you have not been notified by either EPA or your State, you must report to EPA, as specified in § 141.35(b)(2) or (c)(4).

(A) * * * You must monitor for the unregulated contaminants on List 1 and Total Chromium per Table 1, UCMR Contaminant List, in paragraph (a)(3) of this section. * * *

* * * * *

(ii) *Small systems.* Small PWSs, as defined in this paragraph, will not be selected to monitor for any more than one of the three monitoring lists provided in Table 1, UCMR Contaminant List, in paragraph (a)(3) of this section. EPA will provide sample containers, provide pre-paid air bills for shipping the sampling materials, conduct the laboratory analysis, and report and review monitoring results for all small systems selected to conduct monitoring under paragraphs (a)(2)(ii)(A) through (C) of this section. If you own or operate a PWS that serves 10,000 or fewer people you must monitor as follows:

(A) *Assessment Monitoring.* You must monitor for the unregulated contaminants on List 1 and Total Chromium per Table 1, in paragraph (a)(3) of this section, if you are notified by your State or EPA that you are part of the State Monitoring Plan for Assessment Monitoring.

* * * * *

(C) *Pre-Screen Testing.* You must allow EPA or its representative to collect samples to support monitoring for the unregulated contaminants on List 3 of Table 1, in paragraph (a)(3) of this section, if you are notified by your State or EPA that you are part of the State Monitoring plan for Pre-Screen Testing. In addition, you must permit the collection of samples as necessary for EPA to perform analysis for total coliforms, *E. coli,* bacteriophage, *Enterococci* and aerobic spores.

(3) *Analytes to be monitored.* Lists 1, 2, and 3 of unregulated contaminants and total chromium monitoring are provided in the following table:

TABLE 1—UCMR CONTAMINANT LIST

1-Contaminant	2-CAS Registry No.	3-Analytical methods [a]	4-Minimum reporting level [b]	5-Sampling location [c]	6-Period during which monitoring to be completed
List 1: Assessment Monitoring Chemical Contaminants					
Volatile Organic Compounds					
1,2,3-trichloropropane	96–18–4	EPA 524.3	0.03 µg/L	EPTDS	1/1/2013–12/31/2015
1,3-butadiene	106–99–0	EPA 524.3	0.1 µg/L	EPTDS	1/1/2013–12/31/2015
chloromethane	74–87–3	EPA 524.3	0.2 µg/L	EPTDS	1/1/2013–12/31/2015
1,1-dichloroethane	75–34–3	EPA 524.3	0.03 µg/L	EPTDS	1/1/2013–12/31/2015
bromomethane	74–83–9	EPA 524.3	0.2 µg/L	EPTDS	1/1/2013–12/31/2015
chlorodifluoromethane (HCFC–22).	75–45–6	EPA 524.3	0.08 µg/L	EPTDS	1/1/2013–12/31/2015
bromochloromethane (Halon 1011).	74–97–5	EPA 524.3	0.06 µg/L	EPTDS	1/1/2013–12/31/2015

TABLE 1—UCMR CONTAMINANT LIST—Continued

1-Contaminant	2-CAS Registry No.	3-Analytical methods [a]	4-Minimum reporting level [b]	5-Sampling location [c]	6-Period during which monitoring to be completed
Synthetic Organic Compound					
1,4-dioxane	123–91–1	EPA 522	0.07 µg/L	EPTDS	1/1/2013–12/31/2015
Metals					
vanadium	7440–62–2	EPA 200.8, ASTM D5673–10, SM 3125.	0.2 µg/L	EPTDS and DSMRT.	1/1/2013–12/31/2015
molybdenum	7439–98–7	EPA 200.8, ASTM D5673–10, SM 3125.	1. µg/L	EPTDS and DSMRT.	1/1/2013–12/31/2015
cobalt	7440–48–4	EPA 200.8, ASTM D5673–10, SM 3125.	1. µg/L	EPTDS and DSMRT.	1/1/2013–12/31/2015
strontium	7440–24–6	EPA 200.8, ASTM D5673–10, SM 3125.	0.3 µg/L	EPTDS and DSMRT.	1/1/2013–12/31/2015
Chromium-6					
chromium-6 [d]	18540–29–9	EPA 218.7	0.03 µg/L	EPTDS and DSMRT.	1/1/2013–12/31/2015
Oxyhalide Anion					
chlorate	14866–68–3	EPA 300.1, ASTM D 6581–08, SM 4110D.	20 µg/L	EPTDS and DSMRT.	1/1/2013–12/31/2015
Perfluorinated Compounds					
perfluorooctanesulfonic acid (PFOS).	1763–23–1	EPA 537	0.04 µg/L	EPTDS	1/1/2013–12/31/2015
perfluorooctanoic acid (PFOA).	335–67–1	EPA 537	0.02 µg/L	EPTDS	1/1/2013–12/31/2015
perfluorononanoic acid (PFNA).	375–95–1	EPA 537	0.02 µg/L	EPTDS	1/1/2013–12/31/2015
perfluorohexanesulfonic acid (PFHxS).	355–46–4	EPA 537	0.03 µg/L	EPTDS	1/1/2013–12/31/2015
perfluoroheptanoic acid (PFHpA).	375–85–9	EPA 537	0.01 µg/L	EPTDS	1/1/2013–12/31/2015
perfluorobutanesulfonic acid (PFBS).	375–73–5	EPA 537	0.09 µg/L	EPTDS	1/1/2013–12/31/2015
List 2: Screening Survey					
Hormones					
17-β-estradiol	50–28–2	EPA 539	0.0004 µg/L	EPTDS	1/1/2013–12/31/2015
17-α-ethynylestradiol	57–63–6	EPA 539	0.0009 µg/L	EPTDS	1/1/2013–12/31/2015
estriol	50–27–1	EPA 539	0.0008 µg/L	EPTDS	1/1/2013–12/31/2015
equilin	474–86–2	EPA 539	0.004 µg/L	EPTDS	1/1/2013–12/31/2015
estrone	53–16–7	EPA 539	0.002 µg/L	EPTDS	1/1/2013–12/31/2015
testosterone	58–22–0	EPA 539	0.0001 µg/L	EPTDS	1/1/2013–12/31/2015
4-androstene-3,17-dione	63–05–8	EPA 539	0.0003 µg/L	EPTDS	1/1/2013–12/31/2015
List 3: Pre-Screen Testing [e] Microbiological Contaminants					
enteroviruses	N/A	N/A	N/A	EPTDS	1/1/2013–12/31/2015
noroviruses	N/A	N/A	N/A	EPTDS	1/1/2013–12/31/2015
Total Chromium Monitoring					
total chromium	N/A	EPA 200.8, ASTM D5673–10, SM 3125.	0.2 µg/L	EPTDS and DSMRT.	1/1/2013–12/31/2015

Column headings are:
1—Contaminant: The name of the contaminant to be analyzed.
2—CAS (Chemical Abstract Service) Registry Number or Identification Number: A unique number identifying the chemical contaminants.
3—Analytical Methods: Method numbers identifying the methods that must be used to test the contaminants. For List 3, analyses will only be performed by laboratories under contract to EPA.
4—Minimum Reporting Level: The value and unit of measure at or above which the concentration of the contaminant must be measured using the approved analytical methods. If EPA determines, after the first six months of monitoring, that the MRLs specified in UCMR 3 result in excessive resampling, EPA will establish alternate MRLs and will notify affected PWSs and laboratories of the new MRLs. For List 3, minimum reporting level is based on volume of water filtered and PCR amplification level.

5—Sampling Location: The locations within a PWS at which samples must be collected.
6—Period During Which Monitoring to be Completed: The time period during which the sampling and testing will occur for the indicated contaminant.
[a] The analytical procedures shall be performed in accordance with the documents associated with each method, see paragraph (c) of this section.
[b] The minimum reporting level (MRL) is the minimum concentration of each analyte that must be reported to EPA.
[c] Sampling must occur at entry points to the distribution system (EPTDSs) after treatment is applied that represent each non-emergency water source in routine use over the 12-month period of monitoring. Systems that purchase water with multiple connections from the same wholesaler may select one representative connection from that wholesaler. This EPTDS sampling location must be representative of the highest annual volume connections. If the connection selected as the representative EPTDS is not available for sampling, an alternate highest volume representative connection must be sampled. See 40 CFR 141.35(c)(3) for an explanation of the requirements related to use of representative ground water EPTDSs. Sampling for total chromium, chromium-6, cobalt, molybdenum, strontium, vanadium, and chlorate must be conducted at distribution system maximum residence time (DSMRT) sampling locations. DSMRT is defined as an active point (*i.e.,* a location that currently provides water to customers) in the distribution system where the water has been in the system the longest relative to the EPTDS.
[d] Chromium-6 will be measured as soluble chromate ion (CAS Registry Number 13907–45–4).
[e] EPA will collect the samples from List 3 Pre-Screen Testing sampling locations.

* * * * *

(4) * * *

(i) * * *

(B) *Frequency.* You must collect the samples within the time frame and according to the frequency specified by contaminant type and water source type for each sampling location, as specified in Table 2, in this paragraph. For the second or subsequent round of sampling, if a sample location is non-operational for more than one month before and one month after the scheduled sampling month (*i.e.,* it is not possible for you to sample within the window specified in Table 2, in this paragraph), you must notify EPA as specified in § 141.35(c)(5) to reschedule your sampling.

TABLE 2—MONITORING FREQUENCY BY CONTAMINANT AND WATER SOURCE TYPES

Contaminant type	Water source type	Time frame	Frequency
Chemical	Surface water or ground water under the direct influence of surface water (GWUDI) (includes all sampling locations for which some or all of the water comes from a surface water or GWUDI source at any time during the 12 month monitoring period).	12 months	You must monitor for 4 consecutive quarters. Sample events must occur 3 months apart. (Example: If first monitoring is in January, the second monitoring must occur any time in April, the third any time in July and the fourth any time in October.)
	Ground water	12 months	You must monitor twice in a consecutive 12-month period. Sample events must occur 5–7 months apart.
Microbiological	Ground water	12 months	You must monitor twice in a consecutive 12-month period. Sample events must occur 5–7 months apart.

(C) *Location.* You must collect samples for each List 1 Assessment Monitoring contaminant, and, if applicable, for each List 2 Screening Survey, or List 3 Pre-Screen Testing contaminant, as specified in Table 1, in paragraph (a)(3) of this section. Samples must be collected at each sample point that is specified in column 5 and footnote c of Table 1, in paragraph (a)(3) of this section. If you are a ground water system with multiple EPTDSs, and you request and receive approval from EPA or the State for sampling at representative EPTDS(s), as specified in § 141.35(c)(3), you must collect your samples from the approved representative sampling location(s). Systems conducting Assessment Monitoring must also sample for total chromium, chromium-6, cobalt, molybdenum, strontium, vanadium, and chlorate at the location that represents the maximum residence time in the distribution system (DSMRT). DSMRT is defined as an active point (*i.e.,* a location that currently provides water to customers) in the distribution system where the water has been in the system the longest relative to the EPTDS.

(ii) * * *

(G) *Sampling forms.* You must completely fill out each of the sampling forms and bottles sent to you by the UCMR Sampling Coordinator, including data elements listed in § 141.35(e) for each sample, as specified in § 141.35(d)(2). You must sign and date the sampling forms.

* * * * *

(5) * * *

(ii) * * * Correspondence must be addressed to: UCMR Laboratory Approval Coordinator, USEPA, Technical Support Center, 26 West Martin Luther King Drive, (MS 140), Cincinnati, OH 45268; or emailed to EPA at: *UCMR_Sampling_Coordinator@epa.gov*.

(iii) *Minimum Reporting Level.* The MRL is an estimate of the quantitation limit. Assuming good instrumentation and experienced analysts, an MRL is achievable, with 95% confidence, by 75% of laboratories nationwide.

(A) * * *

(1) All laboratories performing analysis under UCMR must demonstrate that they are capable of meeting data quality objectives at or below the MRL listed in Table 1, column 4, in paragraph (a)(3) of this section.

* * * * *

(iv) *Laboratory fortified sample matrix and laboratory fortified sample matrix duplicate.* You must ensure that your laboratory prepares and analyzes the Laboratory Fortified Sample Matrix (LFSM) sample for accuracy and Laboratory Fortified Sample Matrix Duplicate (LFSMD) samples for precision to determine method accuracy and precision for all contaminants in Table 1, in paragraph (a)(3) of this section. LFSM/LFSMD samples must be prepared using a sample collected and analyzed in accordance with UCMR requirements and analyzed at a frequency of 5% (or 1 LFSM/LFSMD set per every 20 samples) or with each sample batch, whichever is more frequent. In addition, the LFSM/LFSMD fortification concentrations must be alternated between a low-level fortification and mid-level fortification

approximately 50% of the time. (For example: A set of 40 samples will require preparation and analysis of 2 LFSM/LFSMD paired samples. The first LFSM/LFSMD paired sample set must be fortified at either the low-level or mid-level, and the second LFSM/LFSMD paired sample set must be fortified with the other standard, either the low-level or mid-level, whichever was not used for the initial LFSM/LFSMD paired sample set.) The low-level LFSM/LFSMD fortification concentration must be within ±50% of the MRL for each contaminant (*e.g.,* for an MRL of 1 µg/L the acceptable fortification levels must be between 0.5 µg/L and 1.5 µg/L). The mid-level LFSM/LFSMD fortification concentration must be within ±20% of the mid-level calibration standard for each contaminant, and is to represent, where possible and where the laboratory has data from previously analyzed samples, an approximate average concentration observed in previous analyses of that analyte. There are no UCMR contaminant recovery acceptance criteria specified for LFSM/LFSMD analyses. All LFSM/LFSMD data are to be reported.

* * * * *

(vi) *Reporting.* You must require your laboratory to submit these data electronically to the State and EPA using EPA's electronic data reporting system, accessible at (*http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/reporting.cfm*), within 120 days from the sample collection date. You then have 60 days from when the laboratory posts the data to review, approve and submit the data to the State and EPA, via EPA's electronic data reporting system. If you do not electronically approve and submit the laboratory data to EPA within 60 days of the laboratory posting data to EPA's electronic reporting system, the data will be considered approved and available for State and EPA review.

* * * * *

(c) *Incorporation by reference.* These standards are incorporated by reference into this section with the approval of the Director of the Federal Register under 5 U.S.C. 552(a) and 1 CFR part 51. All approved material is available for inspection either electronically at *www.regulations.gov*, in hard copy at the Water Docket, EPA/DC, and from the sources below. The Public Reading Room (EPA West, Room 3334, 1301 Constitution Ave. NW., Washington, DC) is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for this Public Reading Room is (202) 566–1744, and the telephone number for the Water Docket is (202) 566–2426. The material is also available for inspection at the National Archives and Records Administration (NARA). For information on the availability of this material at NARA, call (202) 741–6030 or go to *http://www.archives.gov/federal_register/code_0f_federal_regulations/ibr_locations.html*.

(1) The following methods from the U.S. Environmental Protection Agency, Water Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave. NW., Washington, DC 20004.

(i) EPA Method 200.8 ''Determination of Trace Elements in Waters and Wastes by Inductively Coupled Plasma—Mass Spectrometry,'' Revision 5.4, 1994, available at *https://www.NEMI.gov*.

(ii) EPA Method 218.7 ''Determination of Hexavalent Chromium in Drinking Water by Ion Chromatography with Post-Column Derivatization and UV-Visible Spectroscopic Detection,'' Version 1.0, November 2011, EPA 815–R–11–005, available at *http://water.epa.gov/scitech/drinkingwater/labcert/analyticalmethods_ogwdw.cfm*.

(iii) EPA Method 300.1 ''Determination of Inorganic Anions in Drinking Water by Ion Chromatography,'' Revision 1.0, 1997, available at *http://water.epa.gov/scitech/drinkingwater/labcert/analyticalmethods_ogwdw.cfm*.

(iv) EPA Method 522 ''Determination of 1,4-Dioxane in Drinking Water by Solid Phase Extraction (SPE) and Gas Chromatography/Mass Spectrometry (GC/MS) with Selected Ion Monitoring (SIM),'' Version 1.0, September 2008, EPA/600/R–08/101, available at *http://www.epa.gov/nerlcwww/ordmeth.htm*.

(v) EPA Method 524.3 ''Measurement of Purgeable Organic Compounds in Water by Capillary Column Gas Chromatography/Mass Spectrometry,'' Version 1.0, June 2009, EPA 815–B–09–009, available at *http://water.epa.gov/scitech/drinkingwater/labcert/analyticalmethods_ogwdw.cfm*.

(vi) EPA Method 537 ''Determination of Selected Perfluorinated Alkyl Acids in Drinking Water by Solid Phase Extraction and Liquid Chromatography/Tandem Mass Spectrometry (LC/MS/MS),'' Version 1.1, September 2009, EPA/600/R–08/092, available at *http://www.epa.gov/nerlcwww/ordmeth.htm*.

(vii) EPA Method 539 ''Determination of Hormones in Drinking Water by Solid Phase Extraction (SPE) and Liquid Chromatography Electrospray Ionization Tandem Mass Spectrometry (LC–ESI–MS/MS),'' Version 1.0, November 2010, EPA 815–B–10–001, available at *http://water.epa.gov/scitech/drinkingwater/labcert/analyticalmethods_ogwdw.cfm*.

(2) The following methods from ''ASTM International,'' 100 Barr Harbor Drive, West Conshohocken, PA 19428.

(i) ASTM D5673–10 ''Standard Test Method for Elements in Water by Inductively Coupled Plasma-Mass Spectrometry,'' approved August 1, 2010. Available for purchase at *http://www.astm.org/Standards/D5673.htm*.

(ii) ASTM D6581–08 ''Standard Test Methods for Bromate, Bromide, Chlorate, and Chlorite in Drinking Water by Suppressed Ion Chromatography,'' approved August 15, 2008. Available for purchase at *http://www.astm.org/Standards/D6581.htm*.

(3) The following methods from ''Standard Methods for the Examination of Water & Wastewater,'' 21st edition (2005), American Public Health Association, 800 I Street NW., Washington, DC 20001–3710.

(i) SM 3125 ''Metals by Inductively Coupled Plasma/Mass Spectrometry.''

(ii) SM 4110D ''Determination of Anions by Ion Chromatography, Part D, Ion Chromatography Determination of Oxyhalides and Bromide.''

PART 142—NATIONAL PRIMARY DRINKING WATER REGULATIONS IMPLEMENTATION

■ 5. The authority citation for part 142 continues to read as follows:

Authority: 42 U.S.C. 300f, 300g–1, 300g–2, 300g–3, 300g–4, 300g–5, 300g–6, 300j–4, 300j–9, and 300j–11.

Subpart B—Primary Enforcement Responsibility

■ 6. Section 142.16 is amended as follows:

■ a. In paragraph (j) introductory text by removing ''141.40,''.

■ b. In paragraph (j)(1) by revising the first sentence.

§ 142.16 Special primacy requirements.

* * * * *

(j) * * *

(1) If a State chooses to issue waivers from the monitoring requirements in §§ 141.23 and 141.24, the State shall describe the procedures and criteria, that it will use to review waiver applications and issue waiver determinations. * * *

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[FR Doc. 2012–9978 Filed 5–1–12; 8:45 am]

BILLING CODE 6560–50–P

TABLE 2 TO SUBPART JJJJJJ OF PART 63—WORK PRACTICE STANDARDS, EMISSION REDUCTION MEASURES, AND MANAGEMENT PRACTICES—Continued

If your boiler is in this subcategory . . .	You must meet the following . . .
	(6) A list of the energy savings potential of the energy conservation measures identified, and (7) A comprehensive report detailing the ways to improve efficiency, the cost of specific improvements, benefits, and the time frame for recouping those investments.

■ 14. Table 6 to subpart JJJJJJ is amended by revising the entry for ''2.'' to read as follows:

TABLE 6 TO SUBPART JJJJJJ OF PART 63—ESTABLISHING OPERATING LIMITS

If you have an applicable emission limit for . . .	And your operating limits are based on . . .	You must . . .	Using . . .	According to the following requirements
2. Mercury	Dry sorbent or activated carbon injection rate operating parameters.	Establish a site-specific minimum sorbent or activated carbon injection rate operating limit according to § 63.11211(b).	Data from the sorbent or activated carbon injection rate monitors and the mercury performance stack tests.	(a) You must collect sorbent or activated carbon injection rate data every 15 minutes during the entire period of the performance stack tests; (b) Determine the average sorbent or activated carbon injection rate for each individual test run in the three-run performance stack test by computing the average of all the 15-minute readings taken during each test run. (c) When your unit operates at lower loads, multiply your sorbent or activated carbon injection rate by the load fraction, as defined in § 63.11237, to determine the required injection rate.

[FR Doc. 2014–30388 Filed 1–20–15; 8:45 am]
BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 721

[EPA–HQ–OPPT–2013–0225; FRL–9915–63]

RIN 2070–AJ99

Long-Chain Perfluoroalkyl Carboxylate and Perfluoroalkyl Sulfonate Chemical Substances; Significant New Use Rule

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: Under the Toxic Substances Control Act (TSCA), EPA is proposing to amend a significant new use rule (SNUR) for long-chain perfluoroalkyl carboxylate (LCPFAC) chemical substances by designating as a significant new use manufacturing (including importing) or processing of an identified subset of LCPFAC chemical substances for any use that will not be ongoing after December 31, 2015, and all other LCPFAC chemicals substances for which there are currently no ongoing uses. For this SNUR, EPA is also proposing to make inapplicable the exemption for persons who import LCPFAC chemical substances as part of articles. In addition, EPA is also proposing to amend a SNUR for perfluoroalkyl sulfonate (PFAS) chemical substances that would make inapplicable the exemption for persons who import PFAS chemical substances as part of carpets. Persons subject to these SNURs would be required to notify EPA at least 90 days before commencing such manufacture or processing. The required notifications would provide EPA with the opportunity to evaluate the intended use and, if necessary, an opportunity to protect against potential unreasonable risks from that activity before it occurs.

DATES: Comments must be received on or before March 23, 2015.

ADDRESSES: Submit your comments, identified by docket identification (ID) number EPA–HQ–OPPT–2013–0225, by one of the following methods:

- *Federal eRulemaking Portal: http://www.regulations.gov.* Follow the online instructions for submitting comments. Do not submit electronically any information you consider to be

Confidential Business Information (CBI) or other information whose disclosure is restricted by statute.

- *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:* To make special arrangements for hand delivery or delivery of boxed information, please follow the instructions at *http://www.epa.gov/dockets/contacts.html.*

Additional instructions on commenting or visiting the docket, along with more information about dockets generally, is available at *http://www.epa.gov/dockets.*

FOR FURTHER INFORMATION CONTACT:

For technical information contact: Nicholas Nairn-Birch, Chemical Control Division (7405M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001; telephone number: (202) 564–3668; email address: *nairn-birch.nicholas@epa.gov.*

For general information contact: The TSCA-Hotline, ABVI-Goodwill, 422 South Clinton Ave., Rochester, NY 14620; telephone number: (202) 554–1404; email address: *TSCA-Hotline@epa.gov.*

SUPPLEMENTARY INFORMATION:

I. Executive Summary

A. Does this action apply to me?

You may be potentially affected by this action if you manufacture (including import) or process any of the chemical substances covered by this proposed SNUR. The North American Industrial Classification System (NAICS) codes that are identified in this unit are not intended to be exhaustive, but rather provides a guide to help readers determine whether this rule applies to them. Potentially affected entities may include:

- Manufacturers (including importers) of one or more of subject chemical substances (NAICS codes 325 and 324110); *e.g.*, chemical manufacturing and petroleum refineries.
- Fiber, yarn, and thread mills (NAICS code 31311).
- Carpet and rug mills (NAICS code 314110).
- Home furnishing merchant wholesalers (NAICS code 423220).
- Carpet and upholstery cleaning services (NAICS code 561740).

This action may also affect certain entities through pre-existing import certification and export notification rules under TSCA. Persons who import any chemical substance governed by a final SNUR are subject to the TSCA section 13 (15 U.S.C. 2612) import certification requirements and the corresponding regulations at 19 CFR 12.118 through 12.127; see also 19 CFR 127.28. Those persons must certify that the shipment of the chemical substance complies with all applicable rules and orders under TSCA, including any SNUR requirements. The EPA policy in support of import certification appears at 40 CFR part 707, subpart B. In addition, any persons who export or intend to export a chemical substance that is the subject of this proposed rule on or after February 20, 2015 are subject to the export notification provisions of TSCA section 12(b) (15 U.S.C. 2611(b)), (see 40 CFR 721.20), and must comply with the export notification requirements in 40 CFR part 707, subpart D.

To determine whether you or your business may be affected by this action, you should carefully examine the applicability provisions in 40 CFR 721.5 and 40 CFR 721.9582. 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 is the agency's authority for taking this action?

Section 5(a)(2) of TSCA (15 U.S.C. 2604(a)(2)) authorizes EPA to determine that a use of a chemical substance is a ''significant new use.'' EPA must make this determination by rule after considering all relevant factors, including those listed in TSCA section 5(a)(2). Once EPA determines that a use of a chemical substance is a significant new use, TSCA section 5(a)(1)(B) requires persons to submit a significant new use notice (SNUN) to EPA at least 90 days before they manufacture or process the chemical substance for that use (15 U.S.C. 2604(a)(1)(B)). As described in Unit V., the general SNUR provisions are found at 40 CFR part 721, subpart A.

C. What action is the Agency taking?

EPA is proposing to amend a SNUR at 40 CFR 721.10536 for LCPFAC chemical substances by designating manufacturing (including importing) or processing of LCPFAC chemical substances listed in Table 1 of this unit for any use that is no longer ongoing after December 31, 2015, as a significant new use; designating manufacturing (including importing) or processing of PFOA or its salts for any use as a significant new use; and designating manufacturing (including importing) or processing of all other LCPFAC chemical substances for any use not ongoing as of the date on which this proposed rule is published as a significant new use. For this SNUR, EPA is also proposing to make the exemption at 40 CFR 721.45(f) inapplicable for persons who import LCPFAC chemical substances listed in Table 1 of this unit and PFOA or its salts as part of articles because exposure would increase if in the future LCPFAC chemical substances, including PFOA, are incorporated in articles and then imported. EPA is also proposing to amend a SNUR at 40 CFR 721.9582 for PFAS chemical substances to make the exemption at 40 CFR 721.45(f) inapplicable for persons who import of PFAS chemical substances as part of carpets. This action is consistent with the purpose of the ''Long-Chain Perfluorinated Chemicals Action Plan'' (2009 Action Plan) published on December 30, 2009 (Ref. 1). EPA is continuing to assess these chemical substances to determine what other actions would be warranted. Before promulgating a final SNUR with respect to uses of LCPFAC chemical substances listed in Table 1 of this unit that are now ongoing, but are expected to be phased out by December 31, 2015, EPA will verify through comments on this action, or by other means, that the proposed significant new uses have indeed ceased. Similarly, before promulgating a final SNUR on LCPFAC chemical substances other than those listed in Table 1 of this unit, EPA will determine based on comments on this action and other means what if any uses are ongoing in making significant new use determinations in the final rule. Persons would be required to notify EPA at least 90 days before commencing manufacture or processing of LCPFAC chemical substances for the designated significant new uses. This proposed SNUR is intended to follow and codify an existing voluntary industry commitment to phase out LCPFAC chemical substances by the end of 2015 (Ref. 2). The objectives and rationale for this proposed SNUR are explained in more detail in Unit III.

TABLE 1—LCPFAC CHEMICAL SUBSTANCES SUBJECT TO REPORTING AFTER DECEMBER 31, 2015

CAS registry No. (CASRN)	Accession CAS No.	Chemical name
507–63–1	No Accession Number ...	Octane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8-heptadecafluoro-8-iodo-.
678–39–7	No Accession Number ...	1-Decanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluoro-.
865–86–1	No Accession Number ...	1-Dodecanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,12-heneicosafluoro-.
2043–53–0	No Accession Number ...	Decane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8-heptadecafluoro-10-iodo-.
2043–54–1	No Accession Number ...	Dodecane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10-heneicosafluoro-12-iodo-.
17741–60–5 ...	No Accession Number ...	2-Propenoic acid, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11, 12,12,12-heneicosafluorododecyl ester.
27905–45–9 ...	No Accession Number ...	2-Propenoic acid, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluorodecyl ester.
30046–31–2 ...	No Accession Number ...	Tetradecane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12-pentacosafluoro-14-iodo-.
39239–77–5 ...	No Accession Number ...	1-Tetradecanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14,14-pentacosafluoro-.
60699–51–6 ...	No Accession Number ...	1-Hexadecanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14,15,15,16,16,16-nonacosafluoro-.
65510–55–6 ...	No Accession Number ...	Hexadecane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14-nonacosafluoro-16-iodo-.
68187–47–3 ...	No Accession Number ...	1-Propanesulfonic acid, 2-methyl-, 2-[[1-oxo-3-[(.gamma.-.omega.-perfluoro- C4-16-alkyl)thio]propyl]amino] derivs., sodium salts.
68391–08–2 ...	No Accession Number ...	Alcohols, C8-14, .gamma.-.omega.-perfluoro.
70969–47–0 ...	No Accession Number ...	Thiols, C8-20, .gamma.-.omega.-perfluoro, telomers with acrylamide.
125476–71–3	No Accession Number ...	Silicic acid (H_4SiO_4), sodium salt (1:2), reaction products with chlorotrimethylsilane and 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluoro-1-decanol.
1078712–88–5	No Accession Number ...	Thiols, C4-20, .gamma.-.omega.-perfluoro, telomers with acrylamide and acrylic acid, sodium salts.
1078715–61–3	No Accession Number ...	1-Propanaminium, 3-amino-N-(carboxymethyl)-N,N-dimethyl-, N-[2-[(.gamma.-.omega.-perfluoro-C4-20-a lkyl)thio]acetyl] derivs., inner salts.
CBI	71217	Polyfluoroalkyl betaine.
CBI	89419	Modified fluoroalkyl urethane.
CBI	274147	Perfluorinated polyamine.

CBI = Confidential Business Information. CAS or CASRN = Chemical Abstracts Service Registry Number.

In this proposed rule, the term LCPFAC refers to the long-chain category of perfluorinated carboxylate chemical substances with perfluorinated carbon chain lengths equal to or greater than seven carbons and less than or equal to 20 carbons. The category of LCPFAC chemical substances also includes the salts and precursors of these perfluorinated carboxylates. See Unit II.A. for the specific definition of the LCPFAC category.

PFOA and its salts are subject to this proposed rule. PFOA and examples of PFOA salts with Chemical Abstract Service Registry Numbers (CASRN) and chemical names are shown in Table 2 of this unit. PFOA and its salts are considered LCPFAC chemical substances. EPA believes all uses of PFOA and its salts were phased out by December 31, 2013.

TABLE 2—PFOA AND EXAMPLES OF ITS SALTS

CAS registry No. (CASRN)	Chemical name
335–66–0	Octanoyl fluoride, pentadecafluoro-.
335–67–1	Octanoic acid, pentadecafluoro- (PFOA).
335–93–3	Octanoic acid, pentadecafluoro-, silver salt.
335–95–5	Octanoic acid, pentadecafluoro-, sodium salt.
2395–00–8 ...	Octanoic acid, pentadecafluoro-, potassium salt.
3825–26–1 ...	Octanoic acid, pentadecafluoro-, ammonium salt (APFO).

CAS or CASRN = Chemical Abstracts Service Registry Number.

The PFAS chemical substances for which EPA is modifying an existing SNUR are currently listed in 40 CFR 721.9582(a)(1). All of these chemical substances are collectively referred to in this rule as perfluoroalkyl sulfonates, or PFAS chemical substances. In this proposal, the term PFAS refers to a category of perfluorinated sulfonate chemical substances of any chain length.

EPA will not designate ongoing uses as significant new uses when the final rule is promulgated, except for uses that will be phased out by the end of 2015. Persons who manufacture (including importers) or process any of the chemical substances included in the proposed SNUR for an ongoing use at the time this proposed rule is published would be free to continue without submitting a SNUN. Note, however, that uses not already ongoing as of the publication date of this proposed rule, and ongoing uses that will be phased out by the end of 2015, would not be considered ongoing uses if they later arise, even if they are in existence upon the issuance of a final rule. Furthermore, uses that are ongoing as of the publication date of this proposed rule would not be considered ongoing uses if they have ceased by the date of issuance of a final rule (see Units IV. and VI. for further discussion of what constitutes an ongoing use). Persons who intend to begin or resume commercial manufacture or processing of the chemical substance(s) for a significant new use would have to comply with all applicable SNUN requirements.

The LCPFAC chemical substances identified in Table 1 of this unit are known to have current or recent ongoing uses on the basis of their inclusion in reports submitted to the Agency under the 2012 Chemical Data Reporting (CDR) rule. EPA particularly requests comment on whether any of the current uses of any of the specific chemical substances identified in Table 1 of this unit will continue to be ongoing after December 31, 2015. EPA also requests comment on whether there are currently any ongoing uses, including use as part of articles, of any of the remaining LCPFAC chemical substances that were not identified during the 2012 CDR. Furthermore, EPA requests comment on whether there are any ongoing uses of PFOA or its salts, and whether PFAS chemical substances

are currently imported as part of carpets. EPA would welcome specific documentation of any such ongoing use.

D. Why is the Agency taking this action?

These SNURs are necessary to ensure that EPA receives timely advance notice of any future manufacturing (including importing) and processing of these LCPFAC chemical substances for new uses that may produce changes in human and environmental exposures. The rationale and objectives for this SNUR are explained in Unit III.

E. What are the estimated incremental impacts of this action?

EPA has evaluated the potential costs of establishing SNUR reporting requirements for potential manufacturers (including importers) and processors of the chemical substances included in this proposed rule. The economic analysis, which is available in the docket, is discussed in Unit IX., and is briefly summarized here.

In the event that a SNUN is submitted, costs are estimated to be less than $8,589 per SNUN submission for large business submitters and $6,189 for small business submitters. These estimates include the cost to prepare and submit the SNUN and the payment of a user fee. In addition, for persons exporting a chemical substance that is the subject of a SNUR, a one-time notice must be provided for the first export or intended export to a particular country, which is estimated to cost less than $100 on average per notification. The proposed rule may also affect firms that plan to import articles that contain LCFPAC chemical substances, because, while not required by the SNUR, these parties may take additional steps to determine whether LCPFAC chemical substances are part of the articles that they are considering to import. In the accompanying Economic Analysis for this proposed SNUR, example steps (and their respective costs) that an importer might take to identify LCPFAC chemicals in articles are provided. These can include gathering information through agreements with suppliers, declarations through databases or surveys, or use of a third party certification system. Additionally, importers may require suppliers to provide certificates of testing analysis of the products or perform their own laboratory testing of certain articles. EPA is unable to predict, however, what, if any, particular steps an importer might take; potential total costs were not estimated.

II. Chemical Substances Subject to This Proposed Rule

A. What LCPFAC chemical substances are subject to this proposed SNUR?

LCPFAC chemical substances are synthetic chemicals that do not occur naturally in the environment. The LCPFAC chemical substances identified in this unit, where $5 < n < 21$ or $6 < m < 21$:

1. $CF_3(CF_2)_n$-COO-M where M = H^+ or any other group where a formal dissociation can be made.;
2. $CF_3(CF_2)_n$-CH=CH_2.
3. $CF_3(CF_2)_n$-C(=O)-X where X is any chemical moiety.
4. $CF_3(CF_2)_m$-CH_2-X where X is any chemical moiety.
5. $CF_3(CF_2)_m$-Y–X where Y = non-S, non-N heteroatom and where X is any chemical moiety.

This category definition of LCPFAC chemical substances, based on the chemical structures in this unit, refers to a group of chemical substances containing PFOA and its higher homologues. The category also includes the salts and precursors of these chemical substances. The precursors may be simple derivatives of PFOA and higher homologues or polymers that contain or may degrade to PFOA or higher homologues. These precursors include long-chain fluorotelomers. LCPFAC chemical substances with greater than 20 perfluorinated carbons can be considered polymers within the polymer exemption under 40 CFR 723.250 because they exceed a molecular weight of 1,000 daltons and contain at least 3 monomer units. As it is not EPA's intent to regulate fluoropolymers in this proposed rule, the LCPFAC category in this proposed rule includes a perfluorinated carbon chain length upper limit of 20.

In this proposed rule, PFOA and its salts includes the chemical substances listed in Table 2 of Unit II. PFOA and its salts are considered LCPFAC chemical substances.

Under this proposed rule, any LCPFAC chemical substance identified by 40 CFR 721.10536(b)(1)(i) through (b)(1)(v) that is intentionally used during fluoropolymer formulation, such as an emulsion stabilizer in aqueous dispersions, would be subject to reporting for the significant new uses described in 40 CFR 721.10536(b)(4)(i) through (b)(4)(iv). For example, ammonium perfluorooctanoate (APFO)—when used as an aqueous dispersion agent in fluoropolymer production—is subject to this SNUR if the final fluoropolymer product is used for a significant new use described in 40 CFR 721.10536(b)(4)(i) through (b)(4)(iv).

B. What PFAS chemical substances are subject to this proposed SNUR?

PFAS refers to a category of perfluorinated sulfonate chemical substances of any chain length. The PFAS chemical substances for which EPA is proposing to modify an existing SNUR are currently listed in 40 CFR 721.9582(a)(1).

C. What are the uses and production levels of LCPFAC chemical substances?

PFOA, a member of the LCPFAC category, is a synthetic (man-made) chemical that does not occur naturally in the environment. PFOA is manufactured for use primarily as an aqueous dispersion agent as the ammonium salt in the manufacture of fluoropolymers. PFOA can also be produced unintentionally by the degradation of some fluorotelomers, which are not manufactured using PFOA but could degrade to PFOA. DuPont, which was the last company to manufacture (including import) PFOA and its salts in the United States, ceased all production (including import) of PFOA and its salts in 2013 (Ref. 3).

Fluoropolymers provide nonstick surfaces for cookware and other products, are used as molded automotive parts, and have many other applications. Polytetrafluoroethylene (PTFE) is the dominant fluoropolymer, accounting for 58% (by weight) of world fluoropolymer consumption in 2012 (Ref. 4). The United States accounted for 20% of the world consumption of PTFE in 2012 and 40% of the world consumption of other fluoropolymers.

Fluorotelomers, oligomers of tetrafluoroethylene, are relatively small functionalized molecules used to make polymers and surfactants. World-wide production of fluorotelomer-based polymers (FTBP) was estimated at 20 million pounds in 2006. Fluorotelomer monomers and FTBP are included in the LCPFAC category definition as potential LCPFAC precursors (Ref. 5). The United States accounts for more than 50% of world-wide fluorotelomer/FTPB production. Textiles and apparel account for approximately 50% of the volume used (Ref. 1).

In January 2006, EPA launched the 2010/2015 PFOA Stewardship Program (PFOA Stewardship Program) in partnership with eight companies: DuPont, Solvay Solexis, Asahi Glass Company, Daikin America, Inc., Clariant International Ltd., 3M/Dyneon, Arkema Inc., and BASF (formerly Ciba Specialty Chemicals Corporation) (Ref. 2). These companies represent a majority of global

manufacture of LCPFAC chemical substances (Ref. 6). The program set a goal of reducing facility emissions and product content of LCPFAC chemical substances on a global basis by 95%, no later than 2010, and to eliminate emissions and product content of these chemical substances by 2015. With the exception of one manufacturer who has not participated in the PFOA Stewardship Program, these companies accounted for the total volume of LCPFAC chemical substances reported on the 2012 CDR (see Table 2 of Unit I.). Since these chemical substances are proprietary chemicals, they are not expected to be manufactured by any other company. The eight participating companies have informed EPA that they are on track to phase out LCPFAC chemical substances by the end of 2015 (Ref. 7).

Based on the 2012 CDR, there was one additional manufacturer of certain LCPFAC chemical substances who has not participated in the PFOA Stewardship Program. This company manufactures a small volume of LCPFAC chemical substances, compared to the volume of LCPFAC chemical substances manufactured by PFOA Stewardship Program companies, and those chemicals are primarily used in firefighting foams. This company has expressed an interest in participating in the phase out goal of the PFOA Stewardship Program and has already submitted premanufacture notices (PMNs) for chemical substitutes of their current LCPFAC chemical substances. Other than the PFOA Stewardship Program companies and this one company, there were no other companies that reported manufacture (including import) of LCPFAC chemical substances in the 2012 CDR. Any domestic companies still manufacturing LCPFAC chemical substances are most likely obtaining the feedstocks for that manufacturing process from companies participating in the PFOA Stewardship Program. For these companies to continue manufacturing LCPFAC chemical substances, they would need the feedstock and finished LCPFAC chemical substances currently supplied by companies participating in the PFOA Stewardship Program. As the PFOA Stewardship Program member companies phase out their manufacture of those substances and customer demand continues to shift from LCPFAC chemical substances to alternatives, EPA believes that the manufacture of LCPFAC chemical substances by companies not participating in the PFOA Stewardship Programs are likely to cease by December 31, 2015. EPA would like to receive comments addressing the extent to which companies manufacturing specific LCPFAC chemical substances for particular uses are utilizing existing sources that are not dependent on the PFOA Stewardship Program member companies and that are expected to continue after December 31, 2015. Because specific uses of those specific chemical substances would be considered ongoing, they would be outside the scope of the significant new use when finalized.

D. What are the uses and production levels of PFAS chemical substances?

The Agency previously determined that the 271 PFAS chemical substances identified in 40 CFR 721.9582(a)(1) were no longer being manufactured for any use in the United States, other than for the uses listed under 40 CFR 721.9582(a)(3), (a)(4), and (a)(5) (Refs. 8 and 9). PFAS chemical substances included in 40 CFR 721.9582 were previously used in a variety of products, which can be divided into three main use categories: Surface treatments, paper protection, and performance chemicals. In the past, PFAS chemical substances in the performance chemicals category were used in a wide variety of specialized industrial, commercial, and consumer applications. Specific applications included firefighting foams, mining and oil well surfactants, acid mist suppressants for metal plating and electronic etching baths, alkaline cleaners, floor polishes, inks, photographic film, denture cleaners, shampoos, chemical intermediates, coating additives, carpet spot cleaners, and as an insecticide in bait stations for ants (Ref. 10). In some instances, PFAS chemical substances are no longer used for the uses listed in 40 CFR 721.9582(a)(3), (a)(4), and (a)(5) as a result of new substitutes developed and production and processing changes implemented by companies to eliminate the need for use of PFAS chemical substances. In addition, since those chemicals are no longer manufactured (including imported) other than for the listed uses, EPA believes that those chemical substances are also no longer processed other than for those listed uses.

E. What are the potential health and environmental effects of LCPFAC chemical substances?

The following brief summary of chemistry, environmental fate, exposure pathways, and health and environmental effects of LCPFAC chemical substances is based on the 2009 Action Plan (Ref. 1), references cited in the 2009 Action Plan, and additional selected references published after the 2009 Action Plan.

PFOA is persistent, widely present in humans and the environment, has long half-lives in humans, and can cause adverse effects in laboratory animals, including cancer and developmental and systemic toxicity (Refs. 11, 12, 13, 14, and 15). PFOA precursors, chemicals which degrade or may degrade to PFOA, are also present worldwide in humans and the environment and, in some cases, might be present at higher concentrations than PFOA and be more toxic (Refs. 16, 17, 18, 19, and 20). PFOA higher homologues are chemicals with carbon chain lengths longer than PFOA. Available evidence suggests that toxicity and bioaccumulation appear to be higher for chemical substances with longer carbon chain lengths compared to those with shorter chain lengths (Refs. 21, 22, 23, and 24).

LCPFAC chemical substances have been detected in biota, air, water, dust, and soil samples collected throughout the world. Some LCPFAC chemical substances have the potential for long-range transport. They are transported over long distances by a combination of dissolved-phase ocean and gas-phase atmospheric transport; however, determining which is the predominant transport pathway is complicated by many factors, including the uncertainty over water to atmosphere partitioning. Furthermore, there is evidence that transport and subsequent oxidation of volatile alcohol LCPFAC chemical substance precursors contribute to the levels of LCPFAC chemical substances in the environment.

For a more detailed summary of background information (*e.g.,* chemistry, environmental fate, exposure pathways, and health and environmental effects), as well as references pertaining to LCPFAC chemical substances, please refer to Unit IV. of EPA's initial proposed SNUR on LCPFAC chemical substances published in the **Federal Register** of August 15, 2012 (Ref. 10).

F. What are the potential health and environmental effects of PFAS chemical substances?

PFAS chemical substances degrade ultimately to perfluoroalkylsulfonic acid (PFASA), which can exist in the anionic form under environmental conditions. Further degradation of PFASA is not observed under normal environmental conditions. PFASA is highly persistent in the environment and has a tendency to bioaccumulate (Ref. 25). PFASA can continue to be formed by any PFAS

containing chemical substances introduced into the environment.

Studies have found PFAS chemical substances containing 5 to 14 carbons (C5–C14) in the blood of the general human population as well as in wildlife, indicating that exposure to these chemical substances is widespread (Refs. 1, 4, 26, 27, 28, and 29). The widespread presence of PFAS chemical substances in human blood samples nationwide suggests other pathways of exposure, possibly including the release of PFAS from treated articles.

Biological sampling has shown the presence of certain perfluoroalkyl compounds in fish and in fish-eating birds across the United States and in locations in Canada, Sweden, and the South Pacific (Refs. 26 and 27). The wide distribution of the chemical substances in high trophic levels is strongly suggestive of the potential for bioaccumulation and/or bioconcentration.

Based on currently available information, EPA believes that while all PFAS chemical substances are expected to persist, the length of the perfluorinated chain may also have an effect on bioaccumulation and toxicity, which are also characteristics of concern for these chemical substances. PFAS chemical substances with longer carbon chain lengths may be of greater concern than those with shorter chain lengths (Refs. 4, 21, and 22).

The hazard assessment published by the Organization for Economic Cooperation and Development (Ref. 10) concluded that perfluorooctyl sulfonates (PFOS) are persistent, bioaccumulative and toxic to mammalian species. While most studies to date have focused primarily on PFOS, structure-activity relationship analysis indicates that the results of those studies are applicable to the entire category of PFAS chemical substances, which includes PFOS. Available test data have raised concerns about their potential developmental, reproductive, and systemic toxicity (Refs. 1, 16, 26, and 27).

For a more detailed summary of background information (*e.g.,* chemistry, environmental fate, exposure pathways, and health and environmental effects), as well as references pertaining to PFAS chemical substances, please refer to EPA's proposed SNURs on PFAS chemical substances published in the **Federal Register** of October 18, 2000 (Ref. 30), March 11, 2002, and March 10, 2006 (Refs. 26 and 31). Also, refer to the 2009 Action Plan (Ref. 1).

III. Rationale and Objectives

A. Rationale

EPA is concerned about the effects LCPFAC and PFAS chemical substances may have on human health and the environment. As discussed in Unit II., LCPFAC and PFAS chemical substances are found world-wide in the environment, wildlife, and humans. They are bioaccumulative in wildlife and humans, and are persistent in the environment. They are toxic to laboratory animals, producing reproductive, developmental, and systemic effects in laboratory tests. The exact sources and pathways by which these chemicals move into and through the environment and allow humans and wildlife to become exposed are not fully understood, but are likely to include releases from manufacturing of the chemicals, processing of these chemicals into products, and aging, wear, and disposal of products containing them.

Since the manufacture and processing of LCPFAC chemical substances listed in Table 1 of Unit I. will be discontinued after December 31, 2015, as committed by the principal manufacturers of LCPFAC chemical substances participating in the PFOA Stewardship Program, EPA expects the presence of LCPFAC chemical substances in humans and the environment to decline over time as has been observed in the past when production and use of other persistent chemicals has ceased (Ref. 32). Similarly, EPA expects other LCPFAC chemicals substances to decline as well since the manufacture and processing of those has ceased, as observed by the absence of reporting in the CDR 2012 reporting period. In addition, EPA expects the presence of PFAS chemical substances to decline in humans and the environment since PFAS is no longer imported as part of carpets. EPA is concerned that the manufacturing or processing of these chemical substances for the proposed significant new uses could be reinitiated in the future. If reinitiated, EPA believes that such use would significantly increase the magnitude and duration of exposure to humans and the environment to these chemical substances.

Accordingly, EPA wants the opportunity to evaluate and control, where appropriate, activities associated with those uses, if such manufacturing (including importing) or processing were to start or resume. The required notification provided by a SNUN would provide EPA with the opportunity to evaluate activities associated with a significant new use and an opportunity to protect against unreasonable risks, if any, from exposure to LCPFAC chemical substances.

Consistent with EPA's past practice for issuing SNURs under TSCA section 5(a)(2), EPA's decision to propose a SNUR for a particular chemical use need not be based on an extensive evaluation of the hazard, exposure, or potential risk associated with that use. Rather, the Agency's action is based on EPA's determination that if the use begins or resumes, it may present a risk that EPA should evaluate under TSCA before the manufacturing or processing for that use begins. Since the new use does not currently exist, deferring a detailed consideration of potential risks or hazards related to that use is an effective use of resources. If a person decides to begin manufacturing or processing the chemical for the use, the notice to EPA allows EPA to evaluate the use according to the specific parameters and circumstances surrounding that intended use.

B. Objectives

Based on the considerations in Unit III.A., EPA wants to achieve the following objectives with regard to the significant new use(s) that are designated in this proposed rule:

1. EPA would receive notice of any person's intent to manufacture or process LCPFAC chemical substances, PFOA or its salts, or PFAS chemical substances for the described significant new use before that activity begins.

2. EPA would have an opportunity to review and evaluate data submitted in a SNUN before the notice submitter begins manufacturing or processing these chemical substances for the described significant new use.

3. EPA would be able to regulate prospective manufacturers or processors of these chemical substances before the described significant new use of the chemical substance occurs, provided that regulation is warranted pursuant to TSCA sections 5(e), 5(f), 6, or 7.

IV. Significant New Use Determination

Section 5(a)(2) of TSCA states that EPA's determination that a use of a chemical substance is a significant new use must be made after consideration of all relevant factors including:

- The projected volume of manufacturing and processing of a chemical substance.
- The extent to which a use changes the type or form of exposure of human beings or the environment to a chemical substance.
- The extent to which a use increases the magnitude and duration of exposure

of human beings or the environment to a chemical substance.

- The reasonably anticipated manner and methods of manufacturing, processing, distribution in commerce, and disposal of a chemical substance.

In addition to these factors enumerated in TSCA section 5(a)(2), the statute authorizes EPA to consider any other relevant factors.

To determine what would constitute a significant new use of the LCPFAC and PFAS chemical substances subject to this proposed rule, as discussed in this unit. EPA considered relevant information about the toxicity of these substances, trends in blood levels, likely human exposures and environmental releases associated with possible uses, and the four factors listed in TSCA section 5(a)(2).

As discussed in Unit III.A., once the manufacture (including import) and processing of LCPFAC chemical substances for these uses discontinue in the United States, exposure will decrease over time. EPA expects their presence in humans and the environment to concomitantly decline over time. If any of the new use of LCPFAC chemical substances were to begin after phasing out, EPA believes that such use could both change the type and form and increase the magnitude and duration of human and environmental exposure to the substances, constituting a significant new use. Based on consideration of the statutory factors discussed herein, EPA has preliminary determined the following uses as significant new uses:

- Manufacturing (including importing) or processing of LCPFAC chemical substances listed in Table 1 of Unit I. for any uses that are no longer ongoing after December 31, 2015.
- Manufacturing (including importing) or processing of PFOA or its salts for any use.
- Manufacturing (including importing) or processing of all other LCPFAC chemical substances for any use not ongoing as of the date on which this proposed rule is published.

EPA's Office of Research and Development has conducted research demonstrating that perfluorinated chemicals contained in articles of commerce can be released from those articles. For instance, one study observed the removal of perfluorinated chemicals from treated carpet as a result of carpet cleaning and showed that perfluorinated chemicals contained in treated carpet could be released to the environment (Ref. 33). A second study indicated that perfluorinated chemicals could be released from treated medical garments with water alone (Ref. 34). LCPFAC chemical substances may be similarly released from related articles. EPA believes that once manufacturing of LCPFAC chemical substances have been phased out, there will be fewer articles containing the chemicals substances in the public domain over time and thus, exposure through articles will decrease over time. EPA believes any new use of LCPFAC chemical substances as part of articles would increase the duration and magnitude of human and environmental exposure to the substances. Based on these considerations, EPA has preliminarily determined that importing LCPFAC chemical substances listed in Table 1 of Unit I. and PFOA or its salts as part of articles both constitutes a significant new use and warrants making the exemption at 40 CFR 721.45(f) inapplicable to importers of articles. However, import of fluoropolymer dispersions and emulsions, and fluoropolymers as part of articles, containing PFOA or its salts was not determined to be a significant new use because this use is currently ongoing and EPA is not making inapplicable any of the standard exemptions at 40 CFR 721.45 for PFOA.

In a previous rule EPA designated all uses of the PFAS chemicals identified in 40 CFR 721.9582 as significant new uses, except the ongoing uses specified in 40 CFR 721.9582 (a)(3) through (a)(5), the Agency believes the manufacture (including import) and processing of any of the PFAS chemical substances subject to this rule has been discontinued, including the importing of these chemical substances as part of carpets. Based on EPA's Office of Research and Development's research and the considerations in the preceding paragraphs (see, *e.g.,* Ref. 30), EPA believes that if the import of carpets containing these chemical substances were to resume, people and the environment could be exposed to these chemical substances in articles. The existing regulation at 40 CFR 721.9582 broadly defined the significant use in a way that encompassed import of these chemical substances as part of carpets, but for clarity EPA is proposing to expressly list import as part of carpets as a significant new use for the chemicals covered by 40 CFR 721.9582, and in light of the referenced considerations, EPA is now proposing to make inapplicable the exemption at 40 CFR 721.45 to importers of these chemical substances as part of articles.

As noted in Unit V., EPA is proposing that the exemption at 40 CFR 721.45(f) remain in effect for persons who process chemical substances as part of articles because existing stocks of articles may still contain LCPFAC or PFAS chemical substances.

Table 3 of this unit is a summary of the dates relevant to EPA's preliminary determinations.

TABLE 3—SIGNIFICANT NEW USES FOR LCPFAC CHEMICAL SUBSTANCES, PFOA AND ITS SALTS, OTHER LCPFAC CHEMICAL SUBSTANCES, AND PFAS CHEMICAL SUBSTANCES

New use	LCPFAC in Table 1 of Unit I.	PFOA and its salts	Other LCPFAC	PFAS
Manufacture or processing for any use	After 12/31/2015	1/21/2015	1/21/2015	In effect (see 40 CFR 721.9582).

LCPFAC = Long-chain perfluoroalkyl carboxylate. PFAS = Perfluoroalkyl sulfonate. PFOA = Perfluorooctanoic acid.

V. Importers and Processors of These Chemical Substances as Part of Articles

Once the determination of a significant new use under TSCA section 5(a)(2) has been made, EPA may separately determine whether it would be appropriate to make the regulatory exemption for some or all persons who import or process a chemical substance as part of an article (40 CFR 721.45(f)) inapplicable to a SNUR. In this case, EPA believes that the assumption underpinning this exemption, that people and the environment will generally not be exposed to chemical substances as part of articles, does not hold true. See Unit IV. for a discussion of why EPA believes this assumption is incorrect. Thus EPA is proposing to make this exemption inapplicable to importers of the PFAS chemicals identified in 40 CFR 721.9582 as part of carpets and importers of the chemical substances listed in Table 1 and Table 2 of Unit I.C. as part of an article for the corresponding significant new uses. EPA is requesting comment on the

potential for exposure to these chemical substances via these articles and for comments on the ongoing uses of these chemical substances as part of an article. EPA is not proposing to make this exemption inapplicable to processors of these chemical substances as part of an article. EPA previously determined in a prior rulemaking and is not reopening its determination to make this exemption inapplicable to importers of the LCPFAC chemical substances identified in 40 CFR 721.10536(b)(1) as part of carpets.

VI. Applicability of General Provisions

General provisions for SNURs appear under 40 CFR part 721, subpart A. These provisions describe persons subject to the rule, recordkeeping requirements, exemptions to reporting requirements, and applicability of the rule to uses occurring before the effective date of the final rule. However, EPA is proposing that the exemption at 40 CFR 721.45(f) not apply to persons who import LCPFAC chemicals substances listed in Table 1 of Unit I., PFOA or its salts (See Table 2 of Unit I. for examples of PFOA salts), and PFAS chemicals substances listed in 40 CFR 721.9582. As a result, persons subject to the provisions of this proposed rule would not be exempt from significant new use reporting if they import those LCPFAC chemical substances or PFOA or its salts as part of articles or if they import PFAS chemical substances as part of carpets. However, EPA is also proposing that the exemption at 40 CFR 721.45(f) remain in effect for persons who process chemical substances as part of an article because existing stocks of articles may still contain LCPFAC or PFAS chemical substances. Provisions relating to user fees appear at 40 CFR part 700. According to 40 CFR 721.1(c), persons subject to SNURs must comply with the same notice requirements and EPA regulatory procedures as submitters of PMNs under TSCA section 5(a)(1)(A). In particular, these requirements include the information submissions requirements of TSCA section 5(b) and 5(d)(1), the exemptions authorized by TSCA section 5(h)(1), (h)(2), (h)(3), and (h)(5), and the regulations at 40 CFR part 720. Once EPA receives a SNUN, EPA may take regulatory action under TSCA section 5(e), 5(f), 6, or 7 to control the activities on which it has received the SNUN. If EPA does not take action, EPA is required under TSCA section 5(g) to explain in the **Federal Register** its reasons for not taking action.

Persons who export or intend to export a chemical substance identified in the proposed or final SNUR are subject to the export notification provisions of TSCA section 12(b). The regulations that interpret TSCA section 12(b) appear at 40 CFR part 707, subpart D. In accordance with 40 CFR 707.60(b), this proposed SNUR does not trigger notice of export for articles. Persons who import a chemical substance identified in a final SNUR are subject to the TSCA section 13 import certification requirements, codified at 19 CFR 12.118 through 12.127; see also 19 CFR 127.28. Such persons must certify that the shipment of the chemical substance complies with all applicable rules and orders under TSCA, including any SNUR requirements. The TSCA section 13 import certification requirement applies to articles containing a chemical substance or mixture if so required by the Administrator by a specific rule under TSCA. At this time EPA is not proposing to require import certification for these chemical substances as part of articles. The EPA policy in support of import certification appears at 40 CFR part 707, subpart B.

VII. Applicability of Rule to Uses Occurring Before Effective Date of the Final Rule

As discussed in the **Federal Register** of April 24, 1990 (55 FR 17376), EPA has decided that the intent of TSCA section 5(a)(1)(B) is best served by designating a use as a significant new use as of the date of publication of the proposed rule rather than as of the effective date of the final rule. If uses begun after publication of the proposed rule were considered ongoing rather than new, it would be difficult for EPA to establish SNUR notice requirements, because a person could defeat the SNUR by initiating the proposed significant new use before the document became final, and then argue that the use was ongoing as of the effective date of the final rule. Thus, persons who begin commercial manufacture or processing of the chemical substance(s) that would be regulated through this proposed rule, if finalized, would have to cease any such activity before the effective date of the rule if and when finalized. To resume their activities, these persons would have to comply with all applicable SNUR notice requirements and wait until the notice review period, including all extensions, expires. Uses arising after the publication of the proposed rule are distinguished from uses that exist at publication of the proposed rule. The former would be new uses, the latter ongoing uses, except that uses that are ongoing as of the publication of the proposed rule would not be considered ongoing uses if they have ceased by the date of issuance of a final rule (as EPA expects for the LCPFAC chemical substances listed in Table 1 of Unit I. and PFOA or its salts). To the extent that additional ongoing uses are found in the course of rulemaking, EPA would exclude those specific chemical substances for those specific uses from the final SNUR. EPA has promulgated provisions to allow persons to comply with the final SNUR before the effective date. If a person were to meet the conditions of advance compliance under 40 CFR 721.45(h), that person would be considered to have met the requirements of the final SNUR for those activities.

VIII. Test Data and Other Information

EPA recognizes that TSCA section 5 does not usually require developing any particular test data before submission of a SNUN. There are two exceptions:

- Development of test data is required where the chemical substance subject to the SNUR is also subject to a test rule under TSCA section 4 (see TSCA section 5(b)(1)).
- Development of test data may be necessary where the chemical substance has been listed under TSCA section 5(b)(4) (see TSCA section 5(b)(2)).

In the absence of a TSCA section 4 test rule or a TSCA section 5(b)(4) listing covering the chemical substance, persons are required only to submit test data in their possession or control and to describe any other data known to or reasonably ascertainable by them (15 U.S.C. 2604(d); 40 CFR 721.25; and 40 CFR 720.50). However, as a general matter, EPA recommends that SNUN submitters include data that would permit a reasoned evaluation of risks posed by the chemical substance during its manufacture, processing, use, distribution in commerce, or disposal. EPA encourages persons to consult with the Agency before submitting a SNUN. As part of this optional pre-notice consultation, EPA would discuss specific data it believes may be useful in evaluating a significant new use. SNUNs submitted for significant new uses without any test data may increase the likelihood that EPA will take action under TSCA section 5(e) to prohibit or limit activities associated with this chemical.

SNUN submitters should be aware that EPA will be better able to evaluate SNUNs that provide detailed information on:

1. Human exposure and environmental releases that may result from the significant new uses of the chemical substance.

2. Potential benefits of the chemical substance.

3. Information on risks posed by the chemical substances compared to risks posed by potential substitutes.

IX. SNUN Submissions

EPA recommends that submitters consult with the Agency prior to submitting a SNUN to discuss what data may be useful in evaluating a significant new use. Discussions with the Agency prior to submission can afford ample time to conduct any tests that might be helpful in evaluating risks posed by the substance. According to 40 CFR 721.1(c), persons submitting a SNUN must comply with the same notice requirements and EPA regulatory procedures as persons submitting a PMN, including submission of test data on health and environmental effects as described in 40 CFR 720.50. SNUNs must be submitted on EPA Form No. 7710–25, generated using e-PMN software, and submitted to the Agency in accordance with the procedures set forth in 40 CFR 721.25 and 40 CFR 720.40. e-PMN software is available electronically at *http://www.epa.gov/opptintr/newchems.*

X. Economic Analysis

A. SNUNs

EPA has evaluated the potential costs of establishing SNUR reporting requirements for potential manufacturers and processors of the chemical substance included in this proposed rule (Ref. 35). In the event that a SNUN is submitted, costs are estimated at approximately $8,589 per SNUN submission for large business submitters and $6,189 for small business submitters. These estimates include the cost to prepare and submit the SNUN, and the payment of a user fee. Businesses that submit a SNUN would be subject to either a $2,500 user fee required by 40 CFR 700.45(b)(2)(iii), or, if they are a small business with annual sales of less than $40 million when combined with those of the parent company (if any), a reduced user fee of $100 (40 CFR 700.45(b)(1)). The costs of submission of SNUNs will not be incurred by any company unless a company decides to pursue a significant new use as defined in this proposed SNUR.

The proposed SNUR would require notification to EPA before the importation of articles containing LCPFAC chemical substances listed in Table 1 of Unit I. or PFOA and its salts. While not required by the proposed SNUR, companies importing articles containing these chemical substances may take additional steps to determine whether these chemical substances are part of the articles they are considering to import. Companies typically have an understanding of the contents of the articles they import or process; however, there may be instances when companies decide to gather additional information about these articles from suppliers if not currently available. EPA believes that the costs associated with such information gathering activities would be minimal for this proposed SNUR because these chemical substances are unlikely to be available for use in articles after December 31, 2015. EPA's complete economic analysis is available in the public docket for this proposed rule (Ref. 35).

B. Export Notification

Under TSCA section 12(b) and the implementing regulations at 40 CFR part 707, subpart D, exporters must notify EPA if they export or intend to export a chemical substance or mixture for which, among other things, a rule has been proposed or promulgated under TSCA section 5. For persons exporting a chemical substance that is the subject of a SNUR, a one-time notice must be provided for the first export or intended export to a particular country. The total costs of export notification will vary by chemical, depending on the number of required notifications (*i.e.,* the number of countries to which the chemical substance is exported). While EPA is unable to make any estimate of the likely number of export notifications for the chemical substance covered in this proposed rule SNUR, as stated in the accompanying EA of this proposed SNUR, the estimated cost of the export notification requirement on a per unit basis is $81.04.

C. Import Chemical Substances as Part of an Article

In proposing to make inapplicable the exemption relating to persons that import certain chemical substances as part of an article, this action may affect firms that plan to import types of articles that may contain the subject chemical substance. Some firms have an understanding of the contents of the articles they import. However, EPA acknowledges that importers of articles may have varying levels of knowledge about the chemical content of the articles that they import. These parties may need to become familiar with the requirements of the proposed rule. And, while not required by the SNUR, these parties may take additional steps to determine whether the subject chemical substances are part of the articles that they are considering to import. This determination may involve activities such as gathering information from suppliers along the supply chain, and/or testing samples of the article itself. Costs vary across the activities chosen and the extent of familiarity a firm has regarding the articles it imports. Cost ranges are presented in the Agency's Economic Analysis for this proposed rule (Ref. 35). Based on available information, EPA believes that article importers that choose to investigate their products would incur costs at the lower end of the ranges presented in the Economic Analysis. For those companies choosing to undertake actions to assess the composition of the articles they import, EPA expects that importers would take actions that are commensurate with the company's perceived likelihood that a chemical substance might be a part of an article, and the resources it has available. Example activities and their costs are provided in the accompanying Economic Analysis of this proposed rule (Ref. 32).

XI. Alternatives

Before proposing this SNUR, EPA considered the following alternative regulatory actions:

A. Promulgate a TSCA Section 8(a) Reporting Rule

Under a TSCA section 8(a) rule, EPA could, among other things, generally require persons to report information to the Agency when they intend to manufacture or process a listed chemical for a specific use or any use. However, for LCPFAC and PFAS chemical substances, the use of TSCA section 8(a) rather than SNUR authority would have several limitations. First, if EPA were to require reporting under TSCA section 8(a) instead of TSCA section 5(a), EPA would not have the opportunity to review human and environmental hazards and exposures associated with the proposed significant new use and, if necessary, take immediate follow-up regulatory action under TSCA section 5(e) or 5(f) to prohibit or limit the activity before it begins. In addition, EPA may not receive important information from small businesses, because such firms generally are exempt from TSCA section 8(a) reporting requirements. In view of the level of health and environmental concerns about LCPFAC and PFAS chemical substances if used for the proposed significant new use, EPA believes that a TSCA section 8(a) rule for this chemical substance would not meet EPA's regulatory objectives.

B. Regulate LCPFAC Chemical Substances Under TSCA Section 6

EPA may regulate under TSCA section 6 if ''the Administrator finds that there is a reasonable basis to conclude that the manufacture, processing, distribution in commerce, use or disposal of a chemical substance or mixture . . . presents or will present an unreasonable risk of injury to health or the environment.'' (TSCA section 6(a)). Given that these chemical substances are believed to be phasing out, EPA concluded that risk management action under TSCA section 6 is not necessary at this time. However, if EPA determines that there are persons who intend to manufacture or process these chemicals, EPA may decide to regulate LCPFAC chemical substances under TSCA section 6. This proposed SNUR would allow the Agency to address the potential risks associated with the proposed significant new use.

XII. Request for Comment

A. Do you have comments or information about ongoing uses?

EPA welcomes comments on any aspect of this proposed SNUR. EPA particularly requests comment on whether any of the current uses of any of the specific LCPFAC chemical substances identified in Table 1 of Unit I. will continue to be ongoing after December 31, 2015, or whether there are any ongoing uses of those identified in Table 2 of Unit I. EPA also requests comment on whether there are currently any ongoing uses, including use as part of articles, of any of the remaining LCPFAC chemical substances that were not identified in the 2012 CDR. EPA would welcome specific documentation of any such ongoing use.

B. What should I consider as I prepare my comments for EPA?

1. *Submitting CBI.* It is EPA's policy to include all comments received in the public docket without change or further notice to the commenter and to make the comments available online at *http://www.regulations.gov,* including any personal information provided, unless a comment includes information claimed to be CBI or other information whose disclosure is restricted by statute. Do not submit this information to EPA through regulations.gov or email. 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, subpart B.

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

i. Identify the document by docket ID 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 your estimate in sufficient detail to allow for it to be reproduced.

vi. Provide specific examples to illustrate your concerns and suggest 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.

XIII. References

The following is a listing of the documents that are specifically referenced in this document. The docket includes these documents and other information considered by EPA, including documents that are referenced within the documents that are in the docket, even if the referenced document is not physically located in the docket. For assistance in locating these other documents, please consult the technical person listed under **FOR FURTHER INFORMATION CONTACT**.

1. EPA. Long-Chain Perfluorinated Chemicals Action Plan. December 30, 2009.
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3. Rizzuto, Pat. DuPont Ceases PFOA Manufacture, Is on Track to Stop All Uses By End of 2014. *Bloomberg BNA Daily Environmental Report.* December 5, 2013.
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5. Washington, J.W., et al. Degradability of an Acrylate-Linked Fluorotelomer Polymer in Soil. *Environmental Science and Technology.* 43: 6617–6623. 2009.
6. EPA. PFOA Stewardship Program Baseline Year Summary Report. 2007. *http://epa.gov/oppt/pfoa/pubs/stewardship/sumrpt.html#background.*
7. EPA. Industry Progressing in Voluntary Effort to Reduce Toxic Chemicals. February 10, 2012.
8. EPA. Perfluoroalkyl Sulfonates; Significant New Use Rule. Final Rule. **Federal Register** (67 FR 72854, December 9, 2002) (FRL–7279–1).
9. EPA. Perfluoroalkyl Sulfonates; Significant New Use Rule. Final Rule. **Federal Register** (72 FR 57222, October 9, 2007) (FRL–8150–4).
10. EPA. Perfluoroalkyl Sulfonates and Long-Chain Perfluoroalkyl Carboxylate Chemical Substances; Proposed Significant New Use Rule; Proposed Rule. **Federal Register** (77 FR 48924, August 15, 2012) (FRL–9358–7).
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17. Sturm R., et al. Trends of Polyfluoroalkyl Compounds in Marine Biota and in Humans. *Environmental Chemistry.* 7: 457–484. 2010.
18. Lau, C. Perfluorinated Compounds. *Molecular, Clinical and Environmental Toxicology Experientia Supplementum.* Volume 101, pp. 47–86. 2012.
19. Yoo, H., et al. Concentrations, Distribution and Persistence of Fluorotelomer Alcohols in Sludge-applied Soils Near Decatur, Alabama, USA. *Environmental Science & Technology.* 44: 8397–8402. 2010.
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21. Kudo, N., et.al. Comparison of the Elimination Between Perfluorinated Fatty Acids with Different Carbon Chain

Lengths in Rats. *Chemico-Biological Interactions.* Volume 134(2), pp. 203–216. 2001.
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23. Lasier, P.J., et al. Perfluorinated Chemicals in Surface Waters and Sediments from Northwest Georgia, USA, and Their Bioaccumulation in Lumbriculus Variegates. *Environmental Toxicology andChemistry.* 30: 2194–2201. 2011.
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26. EPA. Perfluoroalkyl Sulfonates; Proposed Significant New Use Rule; Proposed Rule. **Federal Register** (67 FR 11014, March 11, 2002) (FRL–6823–7).
27. EPA. Perfluoroalkyl Sulfonates; Significant New Use Rule; Final Rule. **Federal Register** (67 FR 11008, March 11, 2002) (FRL–6823–6).
28. 3M Company. The Science of Organic Fluorochemistry. St. Paul, Minnesota. February 5, 1999.
29. Centers for Disease Control and Prevention. Fourth National Report on Human Exposure to Environmental Chemicals. Updated Tables. March 2013.
30. EPA. Perfluoroalkyl Sulfonates; Significant New Use Rule; Proposed Rule. **Federal Register** (65 FR 62319, October 18, 2000) (FRL–6745–5).
31. EPA. Perfluoroalkyl Sulfonates; Proposed Significant New Use Rule; Proposed Rule. **Federal Register** (71 FR 12311, March 10, 2006) (FRL–7740–6).
32. Kato, K. et al. Trends in Exposure to Polyfluoroalkyl Chemicals in the U.S. Population: 1999–2008. *Environmental Science and Technology.* 45: 8037–8045. 2011.
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34. Liu, X. et al. Trends of Perfluoroalkyl Acid Content in Articles of Commerce. EPA, Report EPA/600/R–12/585. 2012.
35. EPA. Economic Analysis of the Significant New use Rule for Long-Chain Perfluoroalkyl Carboxylate Chemical Substances. August 20, 2013.
36. EPA. Modification of Significant New Use Rules for Certain Substances; Final Rule. **Federal Register** (62 FR 42690, August 8, 1997) (FRL–5735–4).

XIV. Statutory and Executive Order Reviews

A. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563: Improving Regulation and Regulatory Review

This proposed SNUR has been designated by the Office of Management and Budget (OMB) as a ''significant regulatory action'' under section 3(f) of Executive Order 12866 (58 FR 51735, October 4, 1993). Accordingly, EPA submitted this proposed action to OMB for review under Executive Order 12866 and 13563 (76 FR 3821, January 21, 2011), and any changes made in response to OMB recommendations are documented in the docket.

B. Paperwork Reduction Act (PRA)

This action would not impose any new information collection burden under PRA, 44 U.S.C. 3501 *et seq.* Burden is defined in 5 CFR 1320.3(b). The information collection activities associated with existing chemical SNURs are already approved by OMB under OMB control number 2070–0038 (EPA ICR No. 1188); and the information collection activities associated with export notifications are already approved by OMB under OMB control number 2070–0030 (EPA ICR No. 0795). If an entity were to submit a SNUN to the Agency, the annual burden is estimated to be less than 100 hours per response, and the estimated burden for an export notifications is less than 1.5 hours per notification. In both cases, burden is estimated to be reduced for submitters who have already registered to use the electronic submission system. Additional burden, estimated to be less than 10 hours, could be incurred where additional recordkeeping requirements are specified under 40 CFR 721.125(a), (b), and (c).

An Agency may not conduct or sponsor, and a person is not required to respond to a collection of information that requires OMB approval under PRA, unless it has been approved by OMB and displays a currently valid OMB control number. The OMB control numbers for EPA's regulations in title 40 of the CFR, after appearing in the **Federal Register**, are listed in 40 CFR part 9, and included on the related collection instrument, or form, if applicable.

C. Regulatory Flexibility Act (RFA)

Pursuant to RFA section 605(b), 5 U.S.C. 601 *et seq.,* I hereby certify that promulgation of this proposed SNUR would not have a significant economic impact on a substantial number of small entities. The rationale supporting this conclusion is as follows.

EPA generally finds that proposed and final SNURs are not expected to have a significant economic impact on a substantial number of small entities (See, *e.g.,* Ref. 36). Since these proposed SNURs would require a person who intends to engage in such activity in the future to first notify EPA by submitting a SNUN, no economic impact would occur unless someone files a SNUN to pursue a significant new use in the future or forgoes profits by avoiding or delaying the significant new use. Although some small entities may decide to engage in such activities in the future, EPA cannot presently determine how many, if any, there may be. However, EPA's experience to date is that, in response to the promulgation of SNURs covering over 1,000 chemical substances, the Agency receives only a handful of notices per year. During the six year period from 2005–2011, only three submitters self-identified as small in their SNUN submission (Ref. 35). EPA believes the cost of submitting a SNUN is relatively small compared to the cost of developing and marketing a chemical new to a firm and that the requirement to submit a SNUN generally does not have a significant economic impact.

A SNUR applies to any person (including small or large entities) who intends to engage in any activity described in the rule as a ''significant new use.'' EPA has preliminarily determined, based in part, on the Agency's market research, that these chemical substances are not being manufactured (including imported) or processed for a significant new use. This preliminary determination also includes importation of these chemical substances as part of articles for the significant new use (Unit IV.).

In addition, given existing regulatory limitations both internationally and within the U.S., industry-wide processes, resources that support companies in understanding and managing their supply chains, and the evidence showing minimal worldwide availability of the LCPFACs regulated under the SNUR, EPA believes that there will be minimal impact to importers of these chemical substances as part of articles from this proposed SNUR. Therefore, based on current knowledge, EPA has preliminarily determined that these uses, including the importation of these chemical substances as part of articles, are not ongoing, and that no small entities presently manufacture for the significant new uses addressed in this proposed rule. EPA will consider

information received during the comment period that might indicate that this preliminary determination is incorrect. The SNUR does not require importers of articles to conduct specific activities to ascertain if they are importing an article that uses a chemical subject to the proposed rule. EPA expects importers would take actions that are commensurate with their perceived likelihood of a chemical substance subject to the SNUR being part of an article, and the resources it has available. EPA has no reason to believe that a firm would voluntarily incur substantial costs to comply with the SNUR, but rather each firm will choose the most efficient route to identify whether it is importing the subject chemical substances in articles.

Therefore, EPA believes that the potential economic impact of complying with this proposed SNUR is not expected to be significant or adversely impact a substantial number of small entities.

D. Unfunded Mandates Reform Act (UMRA)

Based on EPA's experience with proposing and finalizing SNURs, State, local, and Tribal governments have not been impacted by these rulemakings, and EPA does not have any reason to believe that any State, local, or Tribal government would be impacted by this proposed rulemaking. As such, the requirements of UMRA sections 202, 203, 204, or 205, 2 U.S.C. 1531–1538, do not apply to this proposed action.

E. Executive Order 13132: Federalism

This action would not have a substantial direct effect on 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 Executive Order 13132 (64 FR 43255, August 10, 1999).

F. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments

This proposed rule does not have Tribal implications because it is not expected to have any effect (*i.e.,* there would be no increase or decrease in authority or jurisdiction) 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. Thus, Executive Order 13175 (65 FR 67249, November 9, 2000) does not apply to this proposed rule.

G. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks

This proposed action is not subject to Executive Order 13045 (62 FR 19885, April 23, 1997), because this proposed action is not intended to address environmental health or safety risks for children.

H. Executive Order 13211: Actions That Significantly Affect Energy Supply, Distribution, or Use

This proposed rule is not subject to Executive Order 13211 (66 FR 28355, May 22, 2001), because this action is not expected to affect energy supply, distribution, or use.

I. National Technology Transfer Advancement Act (NTTAA)

Since this proposed action does not involve any technical standards, section 12(d) of the NTTAA, 15 U.S.C. 272 note, does not apply to this proposed action.

J. Executive Order 12898: Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations

This proposed action does not entail special considerations of environmental justice related issues as delineated by Executive Order 12898 (59 FR 7629, February 16, 1994), because EPA has determined that this proposed action would not have disproportionately high and adverse human health or environmental effects on minority or low-income populations. This proposed action would not affect the level of protection provided to human health or the environment.

List of Subjects in 40 CFR Part 721

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

Dated: December 18, 2014.

Wendy C. Hamnett,

Director, Office of Pollution Prevention and Toxics.

Therefore, it is proposed that 40 CFR chapter I be amended as follows:

PART 721—[AMENDED]

■ 1. The authority citation for part 721 continues to read as follows:

Authority: 15 U.S.C. 2604, 2607, and 2625(c).

■ 2. In § 721.9582:

■ a. Redesignate paragraph (a) as (b).

■ b. Add new paragraph (a).

■ c. Revise newly designated paragraph (b)(2)(iv).

■ d. Add paragraph (c).

The amendments read as follows:

§ 721.9582 Certain perfluoroalkyl sulfonates.

(a) *Definitions.* The definitions in § 721.3 apply to this section. In addition, the following definition applies:

Carpet means a finished fabric or similar product intended to be used as a floor covering. This definition excludes resilient floor coverings such as linoleum and vinyl tile.

(b) * * *

(2) * * *

(iv) Import as part of carpets.

* * * * *

(c) *Specific requirements.* The provisions of subpart A of this part apply to this section except as modified by this paragraph (c).

(1) *Revocation of certain notification exemptions.* With respect to imports of carpets, the provisions of § 721.45(f) do not apply to this section. A person who imports a chemical substance identified in this section as part of a carpet is not exempt from submitting a significant new use notice. The other provision of § 721.45(f), respecting processing a chemical substance as part of an article, remains applicable.

(2) [Reserved]

■ 3. Revise § 721.10536 to read as follows:

§ 721.10536 Long-chain perfluoroalkyl carboxylate chemical substances.

(a) *Definitions.* The definitions in § 721.3 apply to this section. In addition, the following definition applies:

Carpet means a finished fabric or similar product intended to be used as a floor covering. This definition excludes resilient floor coverings such as linoleum and vinyl tile.

(b) *Chemical substances and significant new uses subject to reporting.* (1) The chemical substances identified below, where $5 < n < 21$ or $6 < m < 21$, are subject to reporting under this section for the significant new uses described in paragraph (b)(4)(i) and (b)(4)(iv) of this section.

(i) $CF_3(CF_2)_n$-COO M where M = H^+ or any other group where a formal dissociation can be made.

(ii) $CF_3(CF_2)_n$-CH=CH_2.

(iii) $CF_3(CF_2)_n$-C(=O)-X where X is any chemical moiety.

(iv) $CF_3(CF_2)_m$-CH_2-X where X is any chemical moiety.

(v) $CF_3(CF_2)_m$-Y–X where Y = non-S, non-N heteroatom and where X is any chemical moiety.

(2) The chemical substances listed in Table 1 of this paragraph are subject to reporting under this section for the significant new uses described in paragraph (b)(4)(ii) of this section.

TABLE 1—LCPFAC CHEMICAL SUBSTANCES SUBJECT TO REPORTING AFTER DECEMBER 31, 2015

CAS registry no. (CASRN)	Accession no.	Chemical name
507–63–1	No Accession Number	Octane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8-heptadecafluoro-8-iodo-
678–39–7	No Accession Number	1-Decanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluoro-
865–86–1	No Accession Number	1-Dodecanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,12-heneicosafluoro-
2043–53–0	No Accession Number	Decane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8-heptadecafluoro-10-iodo-
2043–54–1	No Accession Number	Dodecane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10 ,10-heneicosafluoro-12-iodo-
17741–60–5	No Accession Number	2-Propenoic acid, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11, 12,12,12-heneicosafluorododecyl ester
27905–45–9	No Accession Number	2-Propenoic acid, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluorodecyl ester
30046–31–2	No Accession Number	Tetradecane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12-pentacosafluoro-14-iodo-
39239–77–5	No Accession Number	1-Tetradecanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14,14-pentacosafluoro-
60699–51–6	No Accession Number	1-Hexadecanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14,15,15,16,16,16-nonacosafluoro-
65510–55–6	No Accession Number	Hexadecane, 1,1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,11,11,12,12,13,13,14,14-nonacosafluoro-16-iodo-
68187–47–3	No Accession Number	1-Propanesulfonic acid, 2-methyl-, 2-[[1-oxo-3-[(.gamma.-.omega.-perfluoro- C4–16-alkyl)thio]propyl]amino] derivs., sodium salts
68391–08–2	No Accession Number	Alcohols, C8–14, .gamma.-.omega.-perfluoro
70969–47–0	No Accession Number	Thiols, C8–20, .gamma.-.omega.-perfluoro, telomers with acrylamide
125476–71–3	No Accession Number	Silicic acid (H_4SiO4), sodium salt (1:2), reaction products with chlorotrimethylsilane and 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluoro-1-decanol
1078712–88–5	No Accession Number	Thiols, C4–20, .gamma.-.omega.-perfluoro, telomers with acrylamide and acrylic acid, sodium salts
1078715–61–3	No Accession Number	1-Propanaminium, 3-amino-N-(carboxymethyl)-N,N-dimethyl-, N-[2-[(.gamma.-.omega.-perfluoro-C4–20-a lkyl)thio]acetyl] derivs., inner salts
CBI	71217	Polyfluoroalkyl betaine
CBI	89419	Modified fluoroalkyl urethane
CBI	274147	Perfluorinated polyamine

CBI = Confidential Business Information. CAS or CASRN = Chemical Abstracts Service Registry Number.

(3) The chemical substances identified as perfluorooctanoic acid (PFOA) and its salts, including those listed in Table 2 of this paragraph, are subject to reporting under this section for the significant new uses described in paragraph (b)(4)(iii) of this section.

TABLE 2—PFOA AND EXAMPLES OF ITS SALTS

CAS registry no. (CASRN)	Chemical name
335–66–0	Octanoyl fluoride, pentadecafluoro-
335–67–1	Octanoic acid, pentadecafluoro- (PFOA)
335–93–3	Octanoic acid, pentadecafluoro-, silver salt
335–95–5	Octanoic acid, pentadecafluoro-, sodium salt
2395–00–8	Octanoic acid, pentadecafluoro-, potassium salt
3825–26–1	Octanoic acid, pentadecafluoro-, ammonium salt (APFO)

CAS or CASRN = Chemical Abstracts Service Registry Number.

(4) Significant new uses. (i) The significant new use for chemical substances identified in paragraph (b)(1) of this section are: Manufacture (including import) or processing for use as part of carpets or to treat carpets (*e.g.,* for use in the carpet aftercare market).

(ii) The significant new use for chemical substances identified in paragraph (b)(2) of this section are: Manufacture (including import) or processing for any use after December 31, 2015.

(iii) The significant new use for chemical substances identified in paragraph (b)(3) of this section are: Manufacture (including import) or processing for any use. Import of fluoropolymer dispersions and emulsions, and fluoropolymers as part of articles, containing chemical substances identified in paragraph (b)(3) of this section shall not be considered as a significant new use subject to reporting.

(iv) The significant new use for chemical substances identified in paragraph (b)(1) of this section, except for those chemicals identified in Table 1 of paragraph (b)(2) of this section are: Manufacture (including import) or processing for any use other than that use already covered by paragraph (b)(4)(i) of this section.

(c) *Specific requirements.* The provisions of subpart A of this part apply to this section except as modified by this paragraph (c).

(1) *Revocation of certain notification exemptions.* With respect to imports of carpets, the provisions of § 721.45(f) do not apply to this section. With respect to imports of articles, the provisions of § 721.45(f) also do not apply to a chemical substance identified in paragraphs (b)(2) or (b)(3) of this section. A person who imports a chemical substance identified in paragraph (b)(1) of this section as part of a carpet or who imports a chemical substance identified in paragraphs (b)(2) or (b)(3) of this section as part of an article is not exempt from submitting a significant new use notice. The other

provision of § 721.45(f), respecting processing a chemical substance as part of an article, remains applicable.

(2) [Reserved]

[FR Doc. 2015–00636 Filed 1–20–15; 8:45 am]

BILLING CODE 6560–50–P

FEDERAL COMMUNICATIONS COMMISSION

47 CFR Part 73

[MB Docket No. 14–253; RM–11741; DA 15–11]

Radio Broadcasting Services; Sagaponack, New York

AGENCY: Federal Communications Commission.

ACTION: Proposed rule.

SUMMARY: This document requests comments on a Petition for Rule Making filed by Red Wolf Broadcasting Corporation, proposing to amend the FM Table of Allotments, Section 73.202(b) of the Commission's Rules, by allotting Channel 233A at Sagaponack, New York, as a first local service. A staff engineering analysis indicates that Channel 233A can be allotted to Sagaponack consistent with the minimum distance separation requirements of the Commission's Rules with a site restriction located 3.2 kilometers (2 miles) northwest of the community. The reference coordinates are 40–56–01 NL and 72–18–55 WL.

DATES: Comments must be filed on or before March 2, 2015, and reply comments on or before March 17, 2015.

ADDRESSES: Secretary, Federal Communications Commission, 445 12th Street SW., Washington, DC 20554. In addition to filing comments with the FCC, interested parties should serve the petitioner as follows: Scott Woodworth, Esq., Edinger Associates PLLC, 1875 I Street NW., Suite 500, Washington, DC 20006.

FOR FURTHER INFORMATION CONTACT: Rolanda F. Smith, Media Bureau, (202) 418–2700.

SUPPLEMENTARY INFORMATION: This is a synopsis of the Commission's *Notice of Proposed Rule Making,* MB Docket No. 14–253, adopted January 8, 2015, and released January 9, 2015. The full text of this Commission decision is available for inspection and copying during normal business hours in the FCC's Reference Information Center at Portals II, CY–A257, 445 12th Street SW., Washington, DC 20554. This document may also be purchased from the Commission's duplicating contractors, Best Copy and Printing, Inc., 445 12th Street SW., Room CY–B402, Washington, DC 20554, telephone 1–800–378–3160 or via email *www.BCPIWEB.com.* This document does not contain proposed information collection requirements subject to the Paperwork Reduction Act of 1995, Public Law 104–13. In addition, therefore, it does not contain any proposed information collection burden ''for small business concerns with fewer than 25 employees,'' pursuant to the Small Business Paperwork Relief Act of 2002, Public Law 107–198, *see* 44 U.S.C. 3506(c)(4).

Provisions of the Regulatory Flexibility Act of 1980 do not apply to this proceeding.

Members of the public should note that from the time a Notice of Proposed Rule Making is issued until the matter is no longer subject to Commission consideration or court review, all *ex parte* contacts are prohibited in Commission proceedings, such as this one, which involve channel allotments. See 47 CFR 1.1204(b) for rules governing permissible *ex parte* contacts.

For information regarding proper filing procedures for comments, see 47 CFR 1.415 and 1.420.

List of Subjects in 47 CFR Part 73

Radio, Radio broadcasting.

Federal Communications Commission.

Nazifa Sawez,

Assistant Chief, Audio Division, Media Bureau.

For the reasons discussed in the preamble, the Federal Communications Commission proposes to amend 47 CFR part 73 as follows:

PART 73—RADIO BROADCAST SERVICES

■ 1. The authority citation for part 73 continues to read as follows:

Authority: 47 U.S.C. 154, 303, 334, 336 and 339.

§ 73.202 [Amended]

■ 2. Section 73.202(b), the Table of FM Allotments under New York, is amended by adding Sagaponack, Channel 233A.

[FR Doc. 2015–00799 Filed 1–20–15; 8:45 am]

BILLING CODE 6712–01–P

DEPARTMENT OF COMMERCE

National Oceanic and Atmospheric Administration

50 CFR Part 648

[Docket No. 140904749–4999–01]

RIN 0648–BE50

Magnuson-Stevens Fishery Conservation and Management Act Provisions; Fisheries of the Northeastern United States; Standardized Bycatch Reporting Methodology Omnibus Amendment

AGENCY: National Marine Fisheries Service (NMFS), National Oceanic and Atmospheric Administration (NOAA), Commerce.

ACTION: Proposed rule; request for comments.

SUMMARY: NMFS proposes regulations to implement the Standardized Bycatch Reporting Methodology Omnibus Amendment developed by the Mid-Atlantic and New England Fishery Management Councils. This amendment was developed, in part, to respond to a remand by the U.S. District of Columbia Court of Appeals decision in *Oceana* v. *Locke.* The amendment also adds various measures to improve and expand on the Standardized Bycatch Reporting Methodology previously in place. The proposed measures include: A new prioritization process for allocation of observers if agency funding is insufficient; bycatch reporting and monitoring mechanisms; analytical techniques and allocation of at-sea fisheries observers; a performance standard; a review and reporting process; framework adjustment and annual specifications provisions; and provisions for industry-funded observers and observer set-aside programs. In addition to responding to the DC Court of Appeals remand, this action is necessary to re-establish and improve the Standardized Bycatch Reporting Methodology for all 13 Greater Atlantic Region Fishery Management Plans, as required under the Magnuson-Stevens Fishery Conservation and Management Act, after the previous methodology was vacated by the 2011 Court order.

DATES: Comments must be received on or before February 20, 2015.

ADDRESSES: You may submit comments, identified by NOAA–NMFS–2014–0114, by any one of the following methods:

• Electronic Submissions: Submit all electronic public comments via the Federal e-Rulemaking Portal. Go to *www.regulations.gov/*

United States Environmental Protection Agency

FACT SHEET
PFOA & PFOS Drinking Water Health Advisories

Overview

EPA has established health advisories for PFOA and PFOS based on the agency's assessment of the latest peer-reviewed science to provide drinking water system operators, and state, tribal and local officials who have the primary responsibility for overseeing these systems, with information on the health risks of these chemicals, so they can take the appropriate actions to protect their residents. EPA is committed to supporting states and public water systems as they determine the appropriate steps to reduce exposure to PFOA and PFOS in drinking water. As science on health effects of these chemicals evolves, EPA will continue to evaluate new evidence.

Background on PFOA and PFOS

PFOA and PFOS are fluorinated organic chemicals that are part of a larger group of chemicals referred to as perfluoroalkyl substances (PFASs). PFOA and PFOS have been the most extensively produced and studied of these chemicals. They have been used to make carpets, clothing, fabrics for furniture, paper packaging for food and other materials (e.g., cookware) that are resistant to water, grease or stains. They are also used for firefighting at airfields and in a number of industrial processes.

Because these chemicals have been used in an array of consumer products, most people have been exposed to them. Between 2000 and 2002, PFOS was voluntarily phased out of production in the U.S. by its primary manufacturer. In 2006, eight major companies voluntarily agreed to phase out their global production of PFOA and PFOA-related chemicals, although there are a limited number of ongoing uses. Scientists have found PFOA and PFOS in the blood of nearly all the people they tested, but these studies show that the levels of PFOA and PFOS in blood have been decreasing. While consumer products and food are a large source of exposure to these chemicals for most people, drinking water can be an additional source in the small percentage of communities where these chemicals have contaminated water supplies. Such contamination is typically localized and associated with a specific facility, for example, an industrial facility where these chemicals were produced or used to manufacture other products or an airfield at which they were used for firefighting.

EPA's 2016 Lifetime Health Advisories

EPA develops health advisories to provide information on contaminants that can cause human health effects and are known or anticipated to occur in drinking water. EPA's health advisories are non-enforceable and non-regulatory and provide technical information to states agencies and other public health officials on health effects, analytical methodologies, and treatment technologies associated with drinking water contamination. In 2009, EPA published provisional health advisories for PFOA and PFOS based on the evidence available at that time. The science has evolved since then and EPA is now replacing the 2009 provisional advisories with new, lifetime health advisories.

FACT SHEET
PFOA & PFOS Drinking Water Health Advisories

EPA's 2016 Lifetime Health Advisories, continued

To provide Americans, including the most sensitive populations, with a margin of protection from a lifetime of exposure to PFOA and PFOS from drinking water, EPA established the health advisory levels at 70 parts per trillion. When both PFOA and PFOS are found in drinking water, the combined concentrations of PFOA and PFOS should be compared with the 70 parts per trillion health advisory level. This health advisory level offers a margin of protection for all Americans throughout their life from adverse health effects resulting from exposure to PFOA and PFOS in drinking water.

How the Health Advisories were developed

EPA's health advisories are based on the best available peer-reviewed studies of the effects of PFOA and PFOS on laboratory animals (rats and mice) and were also informed by epidemiological studies of human populations that have been exposed to PFASs. These studies indicate that exposure to PFOA and PFOS over certain levels may result in adverse health effects, including developmental effects to fetuses during pregnancy or to breastfed infants (e.g., low birth weight, accelerated puberty, skeletal variations), cancer (e.g., testicular, kidney), liver effects (e.g., tissue damage), immune effects (e.g., antibody production and immunity), thyroid effects and other effects (e.g., cholesterol changes).

EPA's health advisory levels were calculated to offer a margin of protection against adverse health effects to the most sensitive populations: fetuses during pregnancy and breastfed infants. The health advisory levels are calculated based on the drinking water intake of lactating women, who drink more water than other people and can pass these chemicals along to nursing infants through breastmilk.

Recommended Actions for Drinking Water Systems

Steps to Assess Contamination

If water sampling results confirm that drinking water contains PFOA and PFOS at individual or combined concentrations greater than 70 parts per trillion, water systems should quickly undertake additional sampling to assess the level, scope and localized source of contamination to inform next steps

Steps to Inform

If water sampling results confirm that drinking water contains PFOA and PFOS at individual or combined concentrations greater than 70 parts per trillion, water systems should promptly notify their State drinking water safety agency (or with EPA in jurisdictions for which EPA is the primary drinking water safety agency) and consult with the relevant agency on the best approach to conduct additional sampling.

Drinking water systems and public health officials should also promptly provide consumers with information about the levels of PFOA and PFOS in their drinking water. This notice should include specific information on the risks to fetuses during pregnancy and breastfed and formula-fed infants from exposure to drinking water with an individual or combined concentration of PFOA and PFOS above EPA's health advisory level of 70 parts per trillion. In addition, the notification should include actions they are taking and identify options that consumers may consider to reduce risk such as seeking an alternative drinking water source, or in the case of parents of formula-fed infants, using formula that does not require adding water.

FACT SHEET
PFOA & PFOS Drinking Water Health Advisories

Recommended Actions for Drinking Water Systems, continued

Steps to Limit Exposure

A number of options are available to drinking water systems to lower concentrations of PFOA and PFOS in their drinking water supply. In some cases, drinking water systems can reduce concentrations of perfluoraklyl substances, including PFOA and PFOS, by closing contaminated wells or changing rates of blending of water sources. Alternatively, public water systems can treat source water with activated carbon or high pressure membrane systems (e.g., reverse osmosis) to remove PFOA and PFOS from drinking water. These treatment systems are used by some public water systems today, but should be carefully designed and maintained to ensure that they are effective for treating PFOA and PFOS. In some communities, entities have provided bottled water to consumers while steps to reduce or remove PFOA or PFOS from drinking water or to establish a new water supply are completed.

Home drinking water treatment units are typically certified by independent third party organizations against American National Standards Institute (ANSI) standards to verify their contaminant removal claims. Some home filters remove impurities using activated carbon and reverse osmosis, which are the same technologies utilized by public water supply systems to remove PFOA and PFOS. However, there currently are no ANSI protocols for testing home treatment systems to verify that these devices effectively remove PFOA and PFOS or how frequently the filters should be changed in order to maintain removal efficiency. NSF International is currently developing such protocols.

Other Actions Relating to PFOA and PFOS

Between 2000 and 2002, PFOS was voluntarily phased out of production in the U.S. by its primary manufacturer, 3M. EPA also issued regulations to limit future manufacturing, including importation, of PFOS and its precursors, without first having EPA review the new use. A limited set of existing uses for PFOS (fire resistant aviation hydraulic fluids, photography and film products, photomicrolithography process to produce semiconductors, metal finishing and plating baths, component of an etchant) was excluded from these regulations because these uses were ongoing and alternatives were not available.

In 2006, EPA asked eight major companies to commit to working toward the elimination of their production and use of PFOA, and chemicals that degrade to PFOA, from emissions and products by the end of 2015. All eight companies have indicated that they have phased out PFOA, and chemicals that degrade to PFOA, from emissions and products by the end of 2015. Additionally, PFOA is included in EPA's proposed Toxic Substance Control Act's Significant New Use Rule (SNUR) issued in January 2015 which will ensure that EPA has an opportunity to review any efforts to reintroduce the chemical into the marketplace and take action, as necessary, to address potential concerns.

EPA has not established national primary drinking water regulations for PFOA and PFOS. EPA is evaluating PFOA and PFOS as drinking water contaminants in accordance with the process required by the Safe Drinking Water Act (SDWA). To regulate a contaminant under SDWA, EPA must find that it: (1) may have adverse health effects; (2) occurs frequently (or there is a substantial likelihood that it occurs frequently) at levels of public health concern; and (3) there is a meaningful opportunity for health risk reduction for people served by public water systems.

FACT SHEET
PFOA & PFOS Drinking Water Health Advisories

Other Actions Relating to PFOA and PFOS, continued

EPA included PFOA and PFOS among the list of contaminants that water systems are required to monitor under the third Unregulated Contaminant Monitoring Rule (UCMR 3) in 2012. Results of this monitoring effort are updated regularly and can be found on the publicly-available National Contaminant Occurrence Database (NCOD) (https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule#3). In accordance with SDWA, EPA will consider the occurrence data from UCMR 3, along with the peer reviewed health effects assessments supporting the PFOA and PFOS Health Advisories, to make a regulatory determination on whether to initiate the process to develop a national primary drinking water regulation.

In addition, EPA plans to begin a separate effort to determine the range of PFAS for which an Integrated Risk Information System (IRIS) assessment is needed. The IRIS Program identifies and characterizes the health hazards of chemicals found in the environment. IRIS assessments inform the first two steps of the risk assessment process: hazard identification, and dose-response. As indicated in the 2015 IRIS Multi-Year Agenda, the IRIS Program will be working with other EPA offices to determine the range of PFAS compounds and the scope of assessment required to best meet Agency needs. More about this effort can be found at https://www.epa.gov/iris/iris-agenda.

Where Can I Learn More?

- EPA's Drinking Water Health Advisories for PFOA and PFOS can be found at: https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos
- PFOA and PFOS data collected under EPA's Unregulated Contaminant Monitoring Rule are available: https://www.epa.gov/dwucmr/occurrence-data-unregulated-contaminant-monitoring-rule
- EPA's stewardship program for PFAS related to TSCA: https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/and-polyfluoroalkyl-substances-pfass-under-tsca
- EPA's research activities on PFASs can be found at: http://www.epa.gov/chemical-research/perfluorinated-chemical-pfc-research
- The Centers for Disease Control and Prevention's Public Health Statement for PFASs can be found at: http://www.atsdr.cdc.gov/phs/phs.asp?id=1115&tid=237

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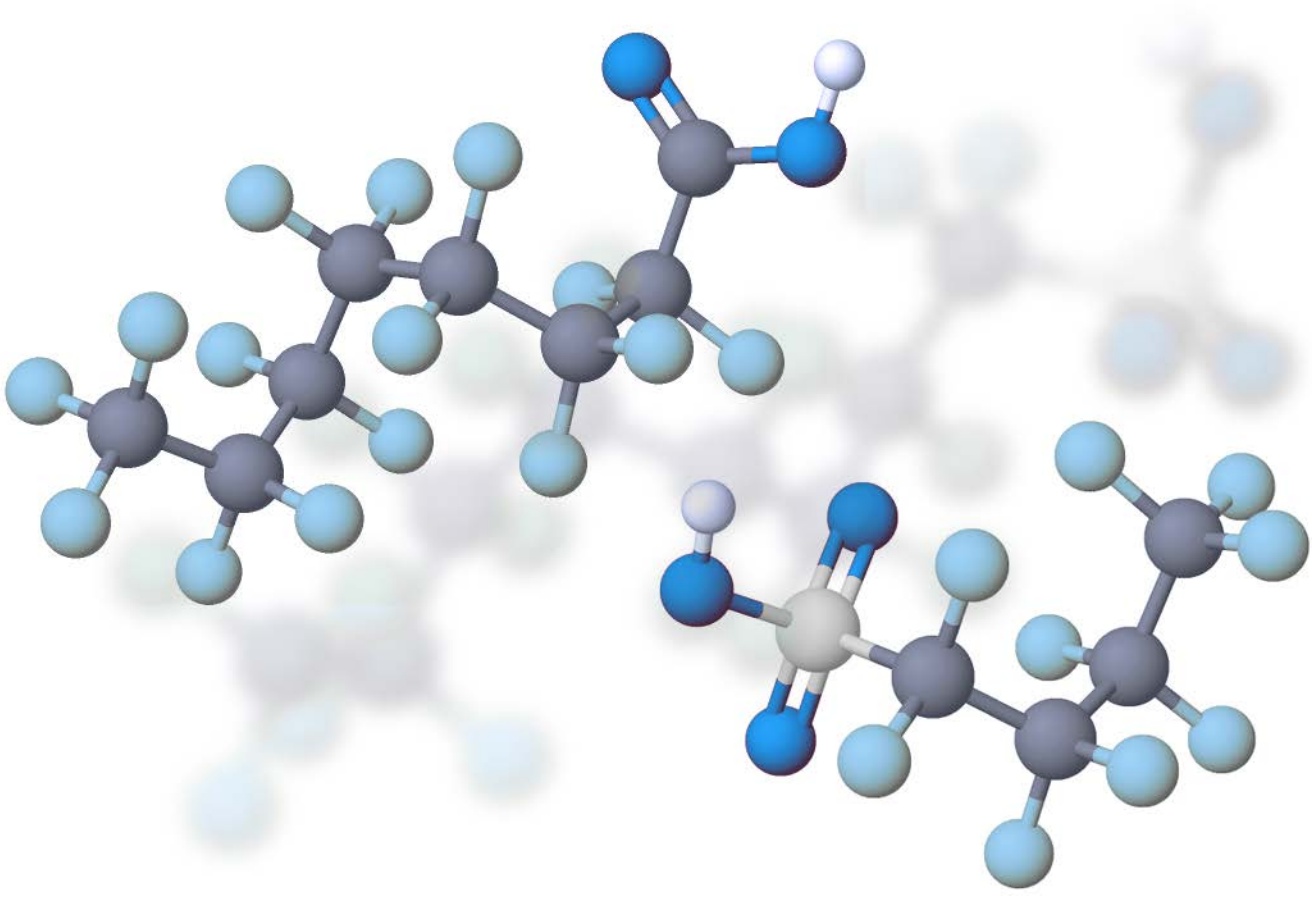
EPA's Per- and Polyfluoroalkyl Substances (PFAS) Action Plan



U.S. Environmental Protection Agency

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List of Acronyms

ACRONYM	FULL PHRASE
ASDWA	Association of State Drinking Water Administrators
ASTHO	Association of State and Territorial Health Officials
ASTSWMO	Association of State and Territorial Solid Waste Management Officials
ATSDR	Agency for Toxic Substances and Disease Registry
CAA	Clean Air Act
CCL	Contaminant Candidate List
CDC	Centers for Disease Control and Prevention
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CWA	Clean Water Act
CWS	Community Water System
DoD	Department of Defense
DWSRF	Drinking Water State Revolving Fund
ECOS	Environmental Council of States
ELGs	Effluent Limitations Guidelines
EPA	Environmental Protection Agency
FDA	Food and Drug Administration
GenX	Gen X Chemicals (i.e., HFPO dimer acid and its ammonium salt), also known as (2,3,3,3-tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)propanoic acid (CASRN 13252-13-6) or hexafluoropropylene oxide (HFPO) dimer acid and 2,3,3,3-tetrafluoro-2-(1,1,2,2,3,3,3-heptafluoropropoxy)propanoate (CASRN 62037-80-3) or HFPO dimer acid ammonium salt)
HA	Health Advisory
HERO	Health and Environmental Research Online
HFPO	Hexafluoropropylene Oxide
HTT	High Throughput Toxicity Testing
HTTK	High Throughput Toxicokinetic
HUD	Department of Housing and Urban Development
ITRC	Interstate Technology and Regulatory Council
KDHE	Kansas Department of Health and Environment
LCPFAC	Long-Chain Perfluoroalkyl Carboxylate
LGAC	Local Government Advisory Committee
MCL	Maximum Contaminant Level
MDEQ	Michigan Department of Environmental Quality
NIST	National Institute of Technology

NPDES	National Pollutant Discharge Elimination System
NTP	National Toxicology Program
OECD	Organization for Economic Cooperation and Development
PFCA	Perfluoroalkyl Carboxylic Acid
PFAS	Per- and Polyfluoroalkyl Substances
PFBS	Perfluorobutane Sulfonic Acid
PFBA	Perfluorobutanoic Acid
PFHpA	Perfluoroheptanoic Acid
PFHxS	Perfluorohexane Sulfonic Acid
PFNA	Perfluorononanoic Acid
PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctane Sulfonate
PMN	Premanufacture notice
ppt	Parts per Trillion
PWSs	Public Water Systems
RCRA	Resource Conservation and Recovery Act
SDWA	Safe Drinking Water Act
SNUN	Significant New Use Notice
SNURs	Significant New Use Rules
TSCA	Toxic Substances Control Act
UCMR	Unregulated Contaminant Monitoring Rule
USACE	United States Army Corps of Engineers
USDA	United States Department of Agriculture
USGS	United States Geological Survey
WCIT	Water Contaminant Information Tool

I. Executive Summary

Per- and polyfluoroalkyl substances (PFAS) are a group of synthetic chemicals that have been in use since the 1940s. PFAS are found in a wide array of consumer and industrial products. PFAS manufacturing and processing facilities, facilities using PFAS in production of other products, airports, and military installations are some of the contributors of PFAS releases into the air, soil, and water. Due to their widespread use and persistence in the environment, most people in the United States have been exposed to PFAS. There is evidence that continued exposure above specific levels to certain PFAS may lead to adverse health effects (USEPA 2016a, 2016b, ATSDR 2018a).

The EPA will continue to partner with other federal agencies, states, tribes, and local communities to protect human health and, where necessary and appropriate, to limit human exposure to potentially harmful levels of PFAS in the environment. The EPA is leading the national effort to understand PFAS and reduce PFAS risks to the public through implementation of this Action Plan and through active engagement and partnership with other federal agencies, states, tribes, industry groups, associations, local communities, and the public.

Key EPA Actions Addressing PFAS-Related Challenges

- **Expand toxicity information for PFAS**
- **Develop new tools to characterize PFAS in the environment**
- **Evaluate cleanup approaches**
- **Develop guidance to facilitate cleanup of contaminated groundwater**
- **Use enforcement tools to address PFAS exposure in the environment and assist states in enforcement activities**
- **Use legal tools such as those in TSCA to prevent future PFAS contamination**
- **Address PFAS in drinking water using regulatory and other tools**
- **Develop new tools and materials to communicate about PFAS**

Throughout recent engagements, the EPA heard clearly the public's desire for immediate action to address potential human health and economic impacts from PFAS in the environment.

This Action Plan describes the EPA's approach to identifying and understanding PFAS, approaches to addressing current PFAS contamination, preventing future contamination, and effectively communicating with the public about PFAS. The Action Plan describes the broad actions the EPA has underway to address challenges with PFAS in the environment, including next steps on the four PFAS management actions the EPA announced at the May 2018 National Leadership Summit. The four actions announced at the Summit were:

- Initiating steps to evaluate the need for a maximum contaminant level (MCL) for perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS);
- Beginning the necessary steps to propose designating PFOA and PFOS as "hazardous substances" through one of the available federal statutory mechanisms[1];
- Developing groundwater cleanup recommendations for PFOA and PFOS at contaminated sites;
- Developing toxicity values or oral reference doses (RfDs)[2] for GenX chemicals[3] and perfluorobutane sulfonic acid (PFBS).

In addition to these significant actions, the EPA's PFAS Action Plan identifies more short-term and long-term actions that are currently being implemented to understand and address PFAS. Short-term actions include:

- Developing new analytical methods and tools for understanding and managing PFAS risk;
- Promulgating Significant New Use Rules (SNURs) that require EPA notification before chemicals are used in new ways that may create human health and ecological concerns; and
- Using enforcement actions to help manage PFAS risk, where appropriate.

Short-term actions are generally taking place or expected to be completed within two years. The Action Plan also sets out long-term regulatory and research approaches the EPA will pursue to reduce exposures and to understand the potential human health and environmental risks associated with PFAS. Actions classified as long-term, such as multi-step research initiatives or regulatory actions, are generally expected to take more than two years. Some long-term actions may result in intermediate steps and products that can help to reduce PFAS exposures and protect public health.

Ecological risks are of great concern to many stakeholders due to the widespread distribution and persistence of PFAS in the environment and the wide variety of PFAS chemicals for which environmental fate and transport is currently uncharacterized. While this Action Plan focuses mainly on human health, characterizing potential ecological impacts and risks are important areas of work for the EPA.

Table 1 below summarizes the key actions the EPA is taking to assist states, tribes, and communities in addressing PFAS. These activities are intended to address challenges identified though stakeholder input

[1] There are multiple statutory mechanisms available to designate PFAS as CERCLA hazardous substances, including CERCLA, RCRA, TSCA, CWA, and CAA.
[2] A reference dose is an estimate of the amount of a chemical a person can ingest daily over a lifetime (chronic RfD) or less (subchronic RfD) that is unlikely to lead to adverse health effects.
[3] hexafluoropropylene oxide (HFPO) dimer acid and its ammonium salt

during the PFAS National Leadership Summit, multiple community engagements, and through the public docket (see Appendices B and C for summaries of stakeholder input).

In addition to the highlighted action items in Table 1, the EPA continues to make progress on developing tools and expanding the body of scientific knowledge needed to understand and effectively manage risk from PFAS, including developing PFAS analytical methods, evaluating treatment and remediation techniques for PFAS, understanding the exposure from various environmental media, and evaluating human health impacts of additional PFAS. These activities are described in more detail in Appendix A.

Table 1. Key PFAS-Related Challenges and Planned and Ongoing EPA Actions

Stakeholder Concern or Challenge	EPA Action(s)	Purpose	Anticipated Timeframe
EPA Priority Actions			
Regulatory uncertainty (e.g., MCL) for PFAS in drinking water	Propose a national drinking water regulatory determination for PFOA and PFOS, highlighting key information gathered by the Agency and our partners to date and additional data needs.	Provide the opportunity for the public to comment on and contribute to the information the EPA may consider related to the regulation of PFAS in drinking water.	2019
Hold responsible parties accountable for PFAS releases into the environment	The EPA has initiated the regulatory development process for listing PFOA and PFOS as CERCLA hazardous substances.	Listing PFOA and PFOS as CERCLA hazardous substances would provide additional authority to address PFOA and PFOS, including the ability to require responsible parties to carry out and/or pay for response actions.	Ongoing Started 2018
Provide guidance for groundwater cleanup actions at contaminated sites	Develop interim cleanup recommendations to address groundwater contaminated with PFOA and PFOS.	Recommendations will provide a starting point for making site-specific cleanup decisions. These recommendations may be considered for federal facility and private-party cleanup under CERCLA, RCRA corrective action programs, and state cleanup programs, where appropriate.	Anticipated 2019
Increase understanding about potential human health impacts of additional PFAS	Finalize draft toxicity assessments for GenX chemicals and PFBS; develop additional PFAS toxicity values for PFBA, PFHxA, PFHxS, PFNA, and PFDA.	Finalized toxicity assessments can be combined with specific exposure information by government and private entities to help characterize potential public health risks associated with exposure to these chemicals.	Final toxicity assessments for PFBS and GenX chemicals in 2019; Draft toxicity assessments for five additional PFAS in 2020

Stakeholder Concern or Challenge	EPA Action(s)	Purpose	Anticipated Timeframe
Expand knowledge about whether new PFAS chemicals entering commerce are safe	Use new statutory requirements added by the Frank R. Lautenberg Chemical Safety for the 21st Century Act to review new PFAS and issue supplemental proposed Significant New Use Rules (SNUR on PFAS).	New chemical reviews under TSCA ensure that unreasonable risks are addressed prior to commercialization. The issuance of SNURs for existing PFAS chemicals prohibits new uses for these chemicals until the EPA determines whether the significant new use presents an unreasonable risk and takes appropriate actions as required by TSCA to address any unreasonable risk.	Ongoing Started in 2016
Short-Term Actions			
Understanding and Addressing PFAS Toxicity and Occurrence			
Establish and curate a clearinghouse of chemical information for PFAS	The EPA's CompTox Chemistry Dashboard has been updated to include several curated lists of PFAS chemicals with links to known chemical, physical, and other properties.	Provide simple access to a comprehensive array of up-to-date information for PFAS of interest.	Ongoing
Expand analytical methods to accurately test for additional PFAS in drinking water	Expand the current drinking water Method 537 to include GenX chemicals and additional PFAS; develop a new drinking water method for additional short-chain PFAS not measured by Method 537.	Improved and/or additional methods would help stakeholders and the EPA accurately test, analyze, and quantify a broader suite of PFAS in their drinking water, including GenX chemicals and other short-chain PFAS.	Method 537.1 completed November 2018; additional methods in 2019
Test for PFAS and PFAS precursors in media other than drinking water	Develop and validate methods for other water matrices (wastewater, surface waters, groundwater), solids (soil, sediment, biosolids, fish tissue), and air (ambient, stack emission, off-gases).	Provide additional methods for stakeholders and the EPA to identify the presence of PFAS in concentrations of concern for media other than drinking water.	2019 – 2021
Coordination across federal agencies with common interests in PFAS toxicity	Participate in a cross-federal-agency working group on PFAS information gathering and sharing.	Better leverage federal investments and reduce redundancies. Provide states, tribes, and communities with consistent cross-federal information for making decisions.	2019

Stakeholder Concern or Challenge	EPA Action(s)	Purpose	Anticipated Timeframe
Identifying and Addressing PFAS Exposures			
Additional robust treatment and remediation technologies for PFAS in the environment	Conduct additional research to identify performance and costs associated with treatment and remediation approaches to address PFAS in the environment, along with any potential unintended consequences associated with specific technologies.	Identify new/additional treatment and remediation options that can be used to address PFAS contamination.	2019
Information about drinking water treatment effectiveness and costs for different PFAS	Incorporate the latest research results for additional PFAS into the EPA's online drinking water treatability database.	Support stakeholders in selecting the most effective drinking water treatment approaches to address concerns with PFAS in the environment.	Ongoing
Hold responsible parties accountable for PFAS releases into the environment	Employ an enforcement strategy that relies first on state and local authorities and utilizes federal authorities as appropriate where, for example, state and local authorities are not available or responsible parties do not address PFAS voluntarily.	Support communities that have PFAS releases by using federal enforcement authorities, where relevant and appropriate.	Ongoing
Understand sources and concentrations of PFAS in the environment	Partner with ECOS to build an interactive map to provide users with easy access to publicly available data on potential PFAS sources and occurrence.	Enable states, tribes, and communities to use the best available data to guide PFAS management decisions.	2019
Risk Communication and Engagement			
Coordinated messaging on PFAS across the federal government	Participate in and coordinate with an interagency PFAS risk communication workgroup to develop consistent communication materials that can be used across the federal government and are informed by the best available science.	Ensure coordinated messaging from the federal government is provided to the states, tribes, and local communities.	Ongoing Start 2019
Communication materials that can be used to inform the public of concerns related to PFAS	Work with other federal agencies, states, and tribes to develop a risk communication toolbox that includes materials and messaging for federal, state, tribal, and local partners to use with the public.	Provide states, tribes, local officials, and utilities with communication tools that convey clear and consistent messages to the public.	2019

Stakeholder Concern or Challenge	EPA Action(s)	Purpose	Anticipated Timeframe
Long-Term Actions			
Increase knowledge about PFAS releases	Explore data availability for listing PFAS chemicals to the Toxics Release Inventory (Section 313 of the Emergency Planning and Community Right-to-Know Act).	Make information about PFAS releases reported by industrial and federal facilities available. This information may be helpful to inform decision-making by communities, government agencies, companies and others.	Start 2019
Reduce PFAS releases into ambient waters and sources of drinking water	Determine if available data and research support the development of Clean Water Act Section 304(a) ambient water quality criteria for human health for PFAS.	When adopted by states and tribes as water quality standards, criteria can be used to set permit limits on discharges to a waterbody and to determine if a waterbody requires cleanup to protect human health and aquatic life.	2021
Hold responsible parties accountable for PFAS releases into the environment	Examine available information and beginning in 2019 seek additional information from industry to explore identification of industrial sources that may warrant potential regulation through national ELGs to be described in preliminary ELG plan 14 (2019).	ELGs require that a technology-based, minimum level of control be applied to any NPDES permit for direct discharge to waters or be directly applicable for indirect dischargers.	Start 2019
Characterize potential health impacts from a broader set of PFAS	Generate PFAS toxicology data through new approaches such as high throughput screening, computational toxicology tools, and chemical informatics for chemical prioritization, screening, and risk assessment.	Inform a more complete understanding of PFAS toxicity for the large set of PFAS chemicals without conventional toxicity data and allow prioritization of actions to potentially address groups of PFAS.	Ongoing
Develop more drinking water occurrence data for a broader group of PFAS	The EPA will propose nationwide drinking water monitoring for PFAS under the next UCMR monitoring cycle utilizing newer methods available to detect more PFAS chemicals and at lower minimum reporting levels (MRLs) than previously possible in earlier monitoring.	Monitoring results will improve understanding of the frequency and concentration of PFAS occurrence in finished U.S. drinking water.	Anticipated 2020
Develop a PFAS data inventory and best practices for contributing data	Develop a data standards best practice that allows sharing of soil, air, water, fish tissue, and other PFAS monitoring data.	Provide a way to share PFAS testing results for media other than drinking water that facilitates integration and easy access and use of PFAS data.	Start 2019

Stakeholder Concern or Challenge	EPA Action(s)	Purpose	Anticipated Timeframe
Access ecological risk information to protect ecosystems	Identify sensitive and susceptible species; synthesize information on bioaccumulation in organisms and food chains; where appropriate develop benchmarks and thresholds for ecological toxicity.	Enable action to protect aquatic ecosystems; establish cleanup levels for contaminated sites; protect recreational and cultural values, such as hunting and fishing.	2022
Understand potential for atmospheric transport of PFAS	Incorporate PFAS information into the EPA atmospheric models to understand the potential for atmospheric fate and transport of PFAS.	Enable risk managers to understand the full range of potential PFAS exposure pathways so that they can prioritize appropriate action.	2022

II. Introduction

Many Americans are concerned about potential health impacts from exposure to per- and polyfluoroalkyl substances (PFAS) in the environment. Over the last decade, there has been a move to the manufacture and use of PFAS that may be less bioaccumulative and may be less likely to cause adverse health effects in humans and the environment. However, contamination from legacy PFAS and uncertainty regarding the safety of newer, alternative, PFAS compounds in the environment are a continuing concern for the federal government, states, tribes, and local communities. The EPA is leading efforts with our federal, state, tribal, and community partners to better characterize and mitigate risks related to the presence of PFAS in the environment. The Agency will work with partners to accomplish these goals through pollution prevention, characterization and remediation of contamination in the environment, evaluation of human health and ecological risks, reducing exposures, development of treatment and remediation technologies, dissemination of risk communication materials, identification of safer alternatives, and use of enforcement authorities and regulatory approaches as appropriate.

This PFAS Action Plan identifies EPA-led short-term actions, longer-term research, and potential regulatory approaches designed to reduce the risks associated with PFAS in the environment. In carrying out this Action Plan, the EPA intends to work closely with its federal partners, states, tribes, and local communities. The challenges associated with PFAS cross multiple environmental media and many potential sources. Effective collaboration among all stakeholders is key to successful characterization, communication, and mitigation of concerns associated with PFAS in the environment. The EPA has heard the concerns expressed by the public through a recent series of EPA-sponsored community engagement meetings and through public comments submitted to the EPA through an open docket. The EPA will work with states, tribes, communities, and other federal agencies to take appropriate steps to protect human health and limit risks from PFAS in the environment. Through implementation of this Action Plan and active engagement with other federal agencies, international organizations, states, tribes, industry groups, associations, local governments, communities, and the public, the EPA will lead the national effort to understand and reduce PFAS risks to the American people. As the EPA learns more about PFAS and the risks they may pose, the Agency may update this Action Plan to reflect that new information.

III. PFAS Identification and Actions Previously Taken by the EPA

The term PFAS refers to per- and polyfluoroalkyl substances. PFAS are a very large group of synthetic chemicals that includes PFOA, PFOS, PFBS, perfluorononanoic acid (PFNA), hexafluoropropylene oxide (HFPO) dimer acid and its ammonium salt (referred to as GenX chemicals), and thousands of other compounds (USEPA 2018a). Due to their strong carbon-fluorine bonds, many PFAS can be very persistent in the environment with degradation periods of years, decades, or longer under natural conditions (Beškoski et al. 2018, Kallenborn 2004, Luo et al. 2015, Parsons et al. 2008, Frömel and Knepper 2010). Differences associated with chain length, chemical structure, and chemical functional groups incorporated into individual PFAS have important implications for mobility, fate, and degradation within the environment, as well as uptake, metabolism, clearance, and toxicity in humans, plants, and other animals. There is evidence that exposure to certain PFAS in the environment can lead to adverse human health effects (ATSDR 2018a, USEPA 2016a, USEPA 2016b). PFOA and PFOS, two of the most widely studied PFAS, have been detected in the blood serum of up to 99% of samples collected between 1999 and 2012 in a population that is representative for the U.S. More recent studies suggest blood levels of PFOA and PFOS have been decreasing since some U.S. manufacturers voluntarily phased out production beginning in 2000[4](ATSDR 2018a, USEPA 2016a, USEPA 2016b, CDC 2018). Measured body concentrations of other PFAS, including replacement PFAS, are showing different patterns (Kato et al. 2011, Olsen et al. 2008, USEPA 2018b). For example, PFNA in women of child-bearing age increased between 1999-2000 and 2007-2008, while perfluorohexane sulfonic acid (PFHxS) was relatively constant (USEPA 2013). However, because these results are based on a broad national survey, they do not depict the exposure distribution for those who live near PFAS-contaminated sites or people who work in

[4] The PFOA Stewardship Program began in 2006. PFOS was phased out by 3M between 2000 and 2002.

occupations that use PFAS. There are many PFAS in wide use for which more information regarding their presence, toxicity and mobility in a variety of environmental media is needed.

Stakeholder Concerns

At the PFAS National Leadership Summit, at community engagement events across the country, and through comments submitted to the docket, the EPA has heard about the many challenges communities are facing with PFAS. The EPA heard that effective collaboration is needed at the federal and state levels to compile and reconcile different information sources, better understand exposure impacts, enhance monitoring approaches, and to develop additional information on PFAS. Stakeholders and decision makers have emphasized the need to accelerate the understanding of PFAS toxicity and the impacts of PFAS to ecosystems as well as the need to expand the availability of analytical methods to detect and characterize exposures of concern.

At these events, the EPA also heard many challenges associated with addressing PFAS including:

- Cost burden and affordability concerns for PFAS-impacted communities and utilities, especially for the cost and operating requirements associated with treatment and remediation technologies;
- Lack of hazardous substance listings, precluding the use of Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) cleanup orders and cost recovery for PFAS;
- Lack of enforceable numeric standards;
- Lack of multi-media sampling methods;
- Confusion about different health values from various authorities; and
- Information gaps on how to safely handle PFAS-containing waste byproducts, biosolids, treatment plant residuals, and materials containing PFAS.

Overarching Challenges for PFAS Management

Understanding the scope of PFAS exposure including sources, pathways, populations exposed, and levels of exposure is critical to effectively characterizing the potential human health and environmental risks associated with these compounds. Other unknown and undiscovered PFAS likely exist within the environment as impurities or byproducts of chemical production or as a result of environmental degradation and transformation processes. Health and occurrence data and validated analytical methods are available for certain PFAS (e.g., PFOA and PFOS). However, for most PFAS there is limited or no toxicity information. While validated EPA drinking water measurement methods are available for 18 PFAS today, including PFOA and PFOS, and more are in development, we lack validated analytical methods for national environmental measurements and assessment of exposure for hundreds of other PFAS. Additional challenges to remediation and cleanup include PFAS occurrence as mixtures with other contaminants. There are continuing research needs related to the development of PFAS destruction technologies. Additional tools and information would improve risk characterization, cleanup options, and management decisions. Knowledge of PFAS impacts on human health and the environment is advancing, and the EPA and other organizations are collaborating to generate research and consider new scientific information as it becomes available. To effectively manage PFAS-related exposures and

human health risks when they have been identified, decision makers must consider the potential sources, available technology and if necessary, the regulatory authorities and enforcement tools that may allow federal agencies, states, tribes, and local governments to address PFAS exposure in the environment.

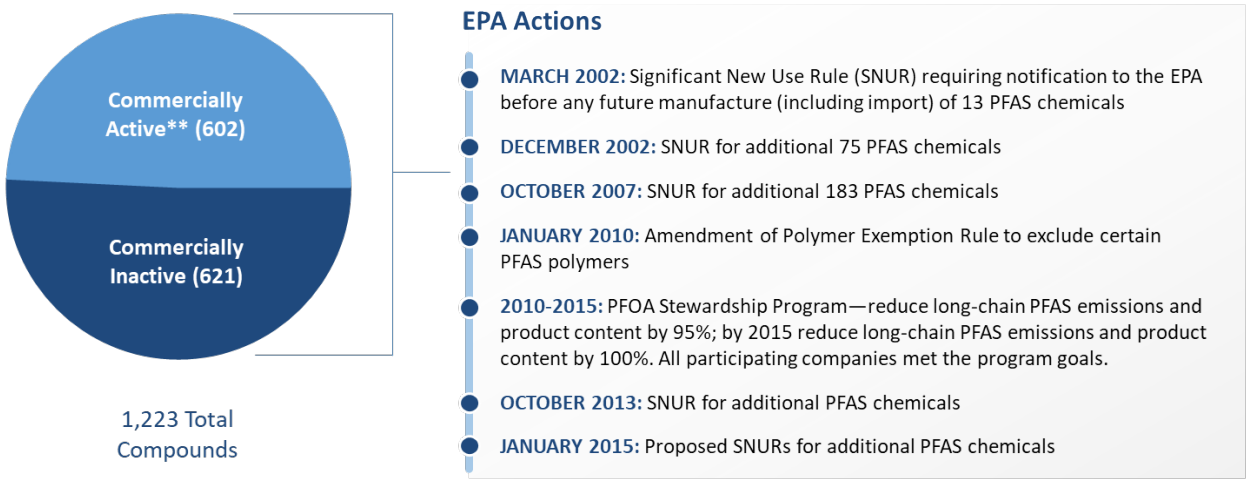
Federal, state, tribal, and local government, communities, and public and private entities will need to partner on developing and implementing management approaches, policies, and solutions to efficiently use limited resources to address PFAS-related risks. While better understanding and reducing the risks posed by PFAS is an important EPA priority, it is not the only public health or environmental challenge faced by our communities. Leveraging resources and partnering is important to ensure the availability of resources to address other priority environmental and public health issues.

While the EPA is evaluating options for development of the most appropriate regulatory programs and tools to address PFAS risks, the EPA also plans to actively lead and support PFAS management efforts using nonregulatory means and enforcement, where appropriate, in partnership with many stakeholders, to protect public health and the environment.

PFAS Use

Over 4,000 PFAS may have been manufactured and used in a variety of industries worldwide since the 1940s (OECD 2018, Guelfo et al. 2018). The EPA's Toxic Substances Control Act (TSCA) Chemical Substance Inventory lists over one thousand PFAS, of which approximately half are known to be commercially active within the last decade. Many PFAS are chemically and thermally stable and demonstrate resistance to heat, water, and oil (Rahman et al. 2014). These properties have made PFAS useful in a variety of consumer products and industrial processes, including firefighting foams, chemical processing, building/construction, aerospace, electronics, semiconductor and automotive industries, stain- and water-resistant coatings (e.g., carpets and rain repellent clothing), food packaging, and in waxes and cleaners (USEPA 2009). Due to their desirable chemical properties for consumer goods, PFAS are widely used in commercial products and can be found in almost every U.S. home and business. All eight companies participating in the EPA's PFOA Stewardship Program voluntarily phased out long-chain PFAS in favor of shorter-chain replacements, which are generally less bioaccumulative and potentially less toxic (Ritter 2010). Previously produced items and imported items may still contain longer-chain PFAS such as PFOA or PFOS (USEPA 2018b). Some replacement PFAS are capable of degrading to PFOA or other long-chain PFAS. Recent research suggests that additional factors aside from chain length may affect the bioaccumulation potential and toxicity of individual PFAS (ITRC 2018a, Ng et al. 2014).

PFAS on the TSCA Inventory*



* The TSCA Inventory is a list of chemical substances approved for U.S. commerce. The original Inventory was compiled from substances reported under the 1978 TSCA Inventory Reporting Rule, and substances have been added since via a commenced Premanufacture Notice.

** Substances on the TSCA Inventory currently designated as commercially active are those reported under the retrospective reporting requirements of the TSCA Inventory Notification (Active/Inactive) rule. These substances were in U.S. commerce at some point between June 2006 and June 2016.

Routes of Exposure

People are exposed to PFAS through the use of consumer products, through occupational exposure, and/or through consuming contaminated food or contaminated drinking water (Fromme et al. 2009). Potential pathways of significant human PFAS exposure include (USEPA 2018a, ATSDR 2018b, Fromme et al. 2009, Ghisi et al. 2018, McGoldrick and Murphy 2016, Stahl et al. 2014, Franko et al. 2012):

- Drinking water from public water and private water systems, typically localized and associated with a release from a specific facility (e.g., manufacturer, processor, landfill, wastewater treatment, or facilities using PFAS-containing firefighting foams);
- Consumption of plants and meat from animals, including fish that have accumulated PFAS;
- Consumption of food that came into contact with PFAS-containing products (e.g., some microwaveable popcorn bags and grease-resistant papers);
- Use of, living with, or otherwise being exposed to commercial household products and indoor dust containing PFAS, including stain- and water-repellent textiles (including carpet, clothing and footwear), nonstick products (e.g., cookware), polishes, waxes, paints, and cleaning products;
- Employment in a workplace that produces or uses PFAS, including chemical production facilities or utilizing industries (e.g., chromium electroplating, electronics manufacturing, or oil recovery); and
- In utero fetal exposure and early childhood exposure via breastmilk from mothers exposed to PFAS.

Potential Human Health Impacts

The majority of research on the potential human health risks of PFAS are associated with oral (ingestion) exposure. Limited data exist on health effects associated with inhalation or dermal exposure to PFAS. Most available toxicity data are based on laboratory animal studies. There are also several human epidemiological studies of PFOA and PFOS. Exposure to some PFAS above certain levels may increase risk of adverse health effects. While many of the same effects are observed for the family of PFAS chemicals, it appears that different adverse effects may be dominant in different PFAS. Depending on the PFAS, increased risks observed in some animal studies include developmental effects to fetuses during pregnancy and infants (e.g., low birth weight, altered puberty, skeletal variations), cancer (e.g., testicular, kidney), liver effects (e.g., tissue damage), immune effects (e.g., changes in antibody production and immunity), thyroid effects related to developmental outcomes, and other effects (e.g., cholesterol changes) (USEPA 2016a, USEPA 2016b). The EPA plans to continue evaluating toxicity information for PFAS; critical information may come from investigating whether exposure to structurally similar PFAS results in similar health effects. Currently, long-chain PFAS are generally thought to present greater toxicity in humans than shorter-chain PFAS (Ritter 2010, Eschauzier et al. 2012), though the toxicities of short-chain PFAS have generally been less thoroughly studied (Danish EPA 2015). Additionally, short-chain PFAS are as persistent in the environment as their longer-chain analogues and are highly mobile in soil and water (Bergström 2014). Due to increasing global production and use, environmental and human exposure to short-chain PFAS is expected to increase over time (Wang et al. 2013). Differences in mobility, fate and persistence in the environment, as well as treatability in environmental media across the complex family of PFAS are expected to contribute to differences in potential exposures and resulting health risks in humans.

History of the EPA's PFAS Actions

The EPA has been actively engaged in preventing risks associated with PFAS. Several statutes provide the EPA with the authority to address PFAS, including TSCA, the Safe Drinking Water Act (SDWA), and CERCLA. This section provides an overview of previous actions the EPA has taken to address PFAS.

Toxic Substances Control Act (TSCA)

Under TSCA, the EPA has broad authority to issue regulations designed to gather health/safety and exposure information on, require testing of, and control exposure to chemical substances and mixtures. TSCA gives the EPA authority to require reporting, record-keeping, and testing of chemical substances and mixtures, and protect against unreasonable risks to human health and the environment from existing chemicals. Among other things, section 5 of TSCA allows the EPA to issue SNURs that require notice to the Agency before chemical substances and mixtures are manufactured (including imported) or processed for significant new uses.

The EPA has used various strategies under TSCA to better understand and reduce exposures to PFAS. For example, in early 2000, the EPA worked with the 3M Company to support the company's voluntary phase-out and elimination of PFOS production and use. As a result of the EPA's 2010/2015 PFOA Stewardship Program, eight major chemical manufacturers and processors agreed to phase out the use

of PFOA and PFOA-related chemicals in their products and emissions from their facilities. All companies met the PFOA Stewardship Program goals by 2015. Through the EPA's work under TSCA, the Agency has also issued various SNURs to require manufacturers (including importers) and processors of certain PFAS chemicals to notify the EPA at least 90 days before starting or resuming significant new uses of these chemicals. This notification would require the EPA to review the significant new use, make a risk determination under section 5, and take appropriate regulatory action based on that risk determination. In 2015, the EPA proposed the most recent SNUR on PFAS to complement the long-chain PFAS phaseout under the 2010/2015 PFOA Stewardship Program by requiring manufacturers (including importers) of PFOA and certain PFOA-related chemicals, including as part of articles, and processors of these chemicals to notify the EPA at least 90 days before starting or resuming new uses of these chemicals. Upon receipt of the notice and prior to any "significant new use" activity commencing, TSCA mandates that the EPA review the potential health and environmental effects, make an affirmative determination on the risks, and take actions necessary to eliminate those risks, as appropriate. The EPA is considering the public comments received on the 2015 proposed SNUR as well as the new statutory requirements added by the Frank R. Lautenberg Chemical Safety for the 21st Century Act as it works to issue a supplemental proposed SNUR on PFAS for the manufacture (including import) of certain long-chain perfluoroalkyl carboxylate (LCPFAC) chemical substances, including as part of categories of certain articles, and the processing of these chemicals.

Safe Drinking Water Act (SDWA)

Section 1412 of the SDWA requires the EPA to publish a list of contaminants known or anticipated to occur in public water systems which may require regulation under the Safe Drinking Water Act (the Contaminant Candidate List). The EPA included PFOA and PFOS on the fourth Contaminant Candidate List (USEPA 2018c). The EPA worked with states and public water systems to characterize the occurrence of six PFAS in the nation's drinking water by including them in the third Unregulated Contaminant Monitoring Rule (UCMR), published in 2012 under the SDWA. The EPA uses the UCMR to collect data for contaminants that are suspected to be present in drinking water and do not have standards set under the SDWA. The EPA collected data for six PFAS in the UCMR: PFOA, PFOS, PFBS, PFNA, PFHxS, and perfluoroheptanoic acid (PFHpA). From 2013-2015, drinking water samples were collected and analyzed in nearly 5,000 public water systems across the nation, accounting for approximately 80% of the U.S. population served by public water systems (USEPA 2016c). The EPA plans to use these monitoring results and other information in the next step in the SDWA regulatory determination process as described below. In addition to the regulatory process, the SDWA provides authority for the Agency to publish drinking water Health Advisories (HAs) which are non-enforceable, health-based drinking water levels. In 2016, the EPA released lifetime Health Advisories for two PFAS (PFOA and PFOS). These Health Advisories provide the public, including the most sensitive populations, with a margin of protection from a lifetime of exposure to PFOA and PFOS from drinking water. Health Advisories are non-enforceable and non-regulatory and provide technical information to state agencies and other public health officials on health effects, analytical methodologies, and treatment technologies associated with drinking water contamination (USEPA 2016a, USEPA 2016b).

Furthermore, pursuant to section 1431(a) of the SDWA, the EPA has authority to take actions the Agency deems necessary to protect public health when a contaminant, whether regulated or not, is

present in or likely to enter a public water system or an underground source of drinking water, and "may present an imminent and substantial endangerment to the health of persons." This authority enables the EPA to respond to emergency conditions and conditions where contamination threatens public health. This section 1431 authority is distinct from the process to establish National Primary Drinking Water Regulations under section 1412 of the SDWA. The EPA has used its authority under section 1431 to issue orders that require persons who have caused or contributed to PFAS contamination to take actions as may be necessary to protect the health of persons, including actions that reduce or prevent exposures.

Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)

CERCLA, commonly known as Superfund, provides the federal government with authority to respond to releases and threatened releases of hazardous substances, and, if they may present an imminent and substantial endangerment, pollutants and contaminants. CERCLA section 104(e) also provides authority to investigate a site to determine whether hazardous substances, pollutants or contaminants have been or may be released. If there is a release of a hazardous substance, parties responsible for the release may be ordered to respond under CERCLA and/or may be liable under CERCLA for the costs of responding to those releases. PFOA and PFOS are considered CERCLA pollutants or contaminants, not hazardous substances. Thus, federal response/cleanup authority exists where the federal agency with CERCLA authority has made a determination that the PFOA or PFOS release may present an imminent and substantial danger to public health or welfare. In addition, the EPA has initiated the regulatory development process to designate PFOA and PFOS as CERCLA "hazardous substances", which would extend CERCLA order and cost recovery authorities to address communities affected by PFOA and PFOS contamination.

The EPA supports federal agencies, states, tribes, and local communities by coordinating with others to identify exposures, developing methods in order to measure PFAS in the environment, and supporting cleanup efforts where PFAS has been identified as a risk to human health, including working with other federal partners and using enforcement tools where necessary. Where the EPA finds that there may be an imminent and substantial endangerment to public health or welfare related to PFAS contamination, the Agency will consider using its response authority under CERCLA section 104 or utilizing its enforcement authorities such as the SDWA section 1431 or Resource Conservation and Recovery Act (RCRA) section 7003.

Consistent with CERCLA, the Agency for Toxic Substances and Disease Registry (ATSDR) recently released draft toxicological profiles for multiple PFAS, which included Minimal Risk Levels (MRLs). ATSDR's MRLs for four PFAS substances (i.e., PFOA, PFOS, PFHxS, and PFNA), when finalized, are intended to serve as screening tools to help public health professionals to determine areas and populations potentially at risk for exposure and can be used as a mechanism to identify hazardous waste sites that are not expected to cause adverse health effects (ATSDR 2018a). The EPA will continue to partner with ATSDR to better understand and communicate risks to human health from PFAS.

IV. Reducing PFAS Exposures: What the EPA Is Doing to Ensure the Problem Is Not Exacerbated

Understanding PFAS in Commerce

Risk Management for PFAS under TSCA

The EPA has the responsibility for reviewing new chemical substances before they enter commerce. The EPA's TSCA New Chemicals program functions as a "gatekeeper" to help manage the potential risk to human health and the environment from chemicals new to the marketplace. TSCA requires the EPA to make risk determinations on new industrial chemicals and provides the EPA with a range of regulatory options to address risks. The EPA has reviewed hundreds of new chemical substitutes for PFOA, PFOS, and other long-chain PFAS under TSCA since 2000. In many cases, the EPA has used its authority under TSCA to impose restrictions on these substances—as well as requiring companies to generate data on physical and chemical properties, environmental fate, toxicokinetics, acute toxicity, irritation and sensitization, repeated dose toxicity,

EPA Priority Action

ACTION: New SNUR on PFAS chemicals.

PURPOSE: In 2015 the EPA proposed the most recent SNUR on PFAS chemicals to complement the long-chain PFAS-phaseout under the 2010/2015 PFOA Stewardship Program.

NEXT STEPS: The EPA is considering the public comments received as well as the new statutory requirements added by the Frank R. Lautenberg Chemical Safety for the 21st Century Act as it works to issue a supplemental proposed SNUR on PFAS.

genotoxicity, reproductive/developmental toxicity, and cancer—as conditions for allowing the substances on the market.

Anyone who plans to manufacture or import a new PFAS chemical substance for a non-exempt[5] commercial purpose must first provide the EPA with notice, known as a premanufacture notice (PMN). The EPA must review and make an affirmative determination on the PMN. For purposes of TSCA, if a chemical is on the TSCA Inventory, the substance is considered an existing chemical substance in U.S. commerce. Any chemical that is not on the Inventory is considered a new chemical substance.

The EPA is required under TSCA to review PMNs in a 90-day period with the goal of identifying whether there are unreasonable risks and applying appropriate controls to mitigate risks where identified. The EPA uses an integrated approach that draws on knowledge and experience across disciplinary and organizational lines to identify releases and exposures and evaluate concerns regarding health and environmental effects. The EPA evaluation includes an assessment of occupational exposures and facility releases to land, water, and air. The EPA then evaluates the impacts of these releases on environmental receptors (primarily aquatic) as well as to the general population, including susceptible populations. The EPA also conducts, when relevant, an assessment of non-workplace exposures such as those experienced by persons using a specific commercial or consumer product containing a chemical (e.g., paints, cleaners). Product use scenarios used to assess risk may include, as appropriate, assessment of 'bystanders' (i.e., persons not actually using the product, but within the exposure vicinity) and subsequent impacts on environmental receptors. As required by TSCA, these evaluations are risk based and consider both hazard and exposure.

By the end of the review period, the EPA must make one of five determinations under TSCA:

1. Insufficient information to perform a reasoned evaluation;
2. Insufficient information and may present unreasonable risk;
3. Not likely to present an unreasonable risk;
4. Presents an unreasonable risk; or
5. Potential for substantial release/exposure.

More information on the EPA's review and decision-making processes is available on the EPA's website at: https://www.epa.gov/reviewing-new-chemicals-under-toxic-substances-control-act-tsca.

The EPA can designate through rulemaking certain new uses of a chemical as significant new uses. Anyone who plans to manufacture or import a chemical substance for a use that has been designated by the EPA as a significant new use must first provide the EPA with notice, known as a significant new use notice (SNUN). The EPA must review and make an affirmative determination on the notice before that new use can commence, if at all. The EPA has already designated significant new uses for more than 400 PFAS chemicals, including for certain PFAS substances that have been through the new chemical review

[5] Certain manufacture of chemical substances is excluded or exempt from full PMN notification requirements, including small quantities of substances manufactured solely for research and development, substances manufactured for test marketing, substances manufactured in low-volumes, and substances manufactured with low releases or low exposures. Some of these exemptions (e.g., the Low Volume Exemption) require submission of an application to the EPA for review and potential action.

process but have not yet been commercialized, and for certain PFAS substances used in manufacturing (including importing) and processing of carpets or for treating carpet.

The Agency proposed in 2015 a Significant New Use Rulemaking for Long-Chain Perfluoroalkyl Carboxylate and Perfluoroalkyl Sulfonate Chemical Substances that would require manufacturers (including importers) of PFOA and certain PFOA-related chemicals, including as part of articles, and processors of these chemicals to notify the EPA at least 90 days before starting or resuming new uses of these chemicals in any products. The Agency plans to follow up on the 2015 SNUR.

Depending on the outcome of its review and determination, under TSCA the EPA may take actions on a new PFAS or significant new PFAS use, ranging from imposing restrictions or limitations (e.g., use restrictions, production volume cap, limitation on releases to water, etc.) to an outright prohibition on manufacture to ensure that the substance does not present an unreasonable risk. For example, if the EPA determines that there is insufficient information to perform a reasoned evaluation or that the chemical may present an unreasonable risk, the EPA may issue an order under TSCA that eliminates the potential for unreasonable risk. The EPA can also require the submitter to conduct testing to better understand whether or to what extent the chemical presents risks. Nearly all TSCA new chemicals orders issued by the EPA are consent orders negotiated with the submitter of the notice. Because these orders are binding only on the original PMN submitter for that substance, the EPA typically also issues a Significant New Use Rule that requires notice to the EPA by any manufacturer or processor who wishes to manufacture or process the chemical in a way other than described in the terms and conditions contained in the order.

Over the decades, and in particular since the beginning of the phase-out of long-chain PFAS in 2006 under the PFOA Stewardship Program, the EPA's new chemicals program has developed significant experience in reviewing PFAS substances before they enter the market. More than 300 PMN or SNUN submissions for PFAS substances have been reviewed by the EPA since the beginning of the PFOA Stewardship Program, of which about 200 were regulated by the EPA, typically under a section 5(e) Order. Similarly, more than 300 Low Volume Exemption Applications have been reviewed by the EPA during this period, most of which were granted based on restrictions/controls in the original or amended submissions.

With the restrictions the EPA has imposed on many of these chemicals, together with the data the EPA required to be generated, the TSCA new chemicals program is an important contributor to helping ensure the safe use of PFAS in commerce.

PFAS and the Toxics Release Inventory

Section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) created the TRI Program. The TRI Program's mission is to provide the public with information about TRI chemicals, including releases, other waste management (e.g., recycling), and pollution prevention from TRI-reporting facilities. The TRI Program is another tool the EPA may use to understand the releases of PFAS by industrial and federal facilities. TRI tracks the management of certain toxic chemicals that may pose a threat to human health and the environment. U.S. facilities in different industry sectors must report annually how much of each chemical is released to the environment and/or managed through recycling,

energy recovery and treatment. A "release" of a chemical means that it is emitted to the air or water or placed in some type of land disposal. The information submitted by facilities is compiled in the Toxics Release Inventory. TRI helps support informed decision-making by companies, government agencies, non-governmental organizations, and the public.

Currently, no PFAS chemicals are included on the list of chemicals required to report to TRI; however, the EPA is considering whether to add PFAS chemicals. In considering listing, the EPA must determine whether data and information are available to fulfill the listing criteria and the extent and utility of the data that would be gathered. For example, hazard data required for TRI listing may be readily available for certain PFAS chemicals, but not others. In addition, in considering if TRI will provide useful information to stakeholders, the EPA also will consider if those PFAS are still active in commerce. The process for listing includes notice and comment rulemaking to list PFAS chemicals for reporting prior to adding these chemicals to the TRI for annual reporting.

V. Understanding PFAS Toxicity to Develop Recommendations and Standards

The EPA is working to understand and address PFAS toxicity through development of human health toxicity assessments on long- and short-chain PFAS. This and other research using advanced toxicological methods will provide a better understanding of PFAS toxicity, including methods for assessing groups of PFAS with similar toxicities and exposures. Toxicity information can be used to provide health protective recommendations and standards for cleanup of environmental media.

The EPA's Actions to Develop Human Health Toxicity Information on PFAS

In 2016, the EPA issued a non-regulatory lifetime Health Advisory (HA) of 70 parts per trillion (ppt) for individual and combined PFOA and PFOS in drinking water. Additional information on the Health Advisories for PFOA and PFOS can be found at https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos. The EPA has made it a priority to produce a new toxicity assessment for GenX chemicals and an updated toxicity assessment for PFBS to facilitate hazard characterization and future risk management decisions. The EPA made

EPA Priority Action

ACTION: The EPA is developing toxicity values for GenX chemicals and PFBS.

PURPOSE: Industry has phased out the use of PFOS and PFOA in favor of shorter-chain PFAS such as GenX chemicals and PFBS. Toxicity values for these replacement chemicals will help inform risk management decisions of federal agencies, states, and tribes to protect human health.

NEXT STEPS: The EPA plans to release final toxicity values for GenX chemicals and PFBS in 2019. Toxicity values for five other PFAS are under development.

draft toxicity assessments for GenX chemicals and PFBS available for public comment in 2018 and expects to issue final toxicity assessments for these two compounds in 2019. Concurrently, the EPA plans to generate additional PFAS toxicity data through *in vitro* high throughput toxicity testing (HTT) and high throughput toxicokinetic (HTTK) assays to inform hazard effects characterization and promote prioritization of chemicals for further *in vivo* testing (Judson et al. 2009, Kavlock and Dix 2010). Generating HTT and HTTK data will improve our understanding of PFAS toxicity and potential human health effects for PFAS compounds for which there is currently limited health-related information and can help to inform Agency and stakeholder decision-making regarding human health risk and remediation levels across the broad landscape of PFAS compounds. In the near term, the EPA intends to also continue to use public peer-reviewed available toxicity information to work towards the development of additional PFAS toxicity assessments for perfluorobutanoic acid (PFBA), perfluorohexanoic acid (PFHxA), PFHxS, PFNA, and perfluorodecanoic acid (PFDA).

Groundwater Cleanup Recommendations for PFOA and PFOS

The EPA is developing Interim Recommendations for Addressing Groundwater Contaminated with PFOA and PFOS to support site-specific cleanup efforts. When finalized, the guidance will provide interim recommendations at sites being evaluated and remediated under the EPA's CERCLA federal cleanup program or at federal-led RCRA corrective action sites. The information and recommendations in this guidance may also be useful for other federal agencies, states, tribes, or other regulatory authorities (e.g., approved state RCRA corrective action programs).

Addressing PFAS in Drinking Water through Standards

The EPA is committed to following the Safe Drinking Water Act process for evaluating drinking water standards for PFAS, including an MCL for PFOA and PFOS. That process involves determining: (1) whether a contaminant may have adverse health effects; (2) whether a contaminant is found in public water systems with a frequency and at levels of concern; and (3) whether, in the sole judgment of the Administrator, there is a meaningful opportunity for health risk reduction through a national drinking water regulation. This process includes a formal rulemaking, engagement with the EPA's National Drinking Water Advisory Council, and extensive public participation. These requirements are expressly prescribed under the Safe Drinking Water Act to ensure scientific integrity and transparency for the regulation of contaminants in public water systems.

EPA Priority Action

ACTION: The EPA is developing interim recommendations for addressing groundwater contaminated by PFOA and PFOS.

PURPOSE: These recommendations will assist the EPA, other federal agencies, states, and tribes in developing and implementing cleanup goals for PFOA and PFOS under CERCLA.

NEXT STEPS: The groundwater cleanup recommendations will be released for public comments prior to finalization.

Certain PFAS have been shown to cause adverse health effects at sufficient exposures, and the EPA is continuing to gather and analyze data regarding the frequency and levels of occurrence of the sampled PFAS. Under the third Unregulated Contaminant Monitoring Rule (UCMR3) program the EPA collected data for six PFAS. From January 2013 through December 2015, samples were collected nationally by all public water systems (PWSs) serving more than 10,000 people, as well as from 800 representative PWSs serving 10,000 or fewer people. Additional information can be found at the EPA's UCMR3 website https://www.epa.gov/dwucmr/third-unregulated-contaminant-monitoring-rule (USEPA 2016c). The EPA found that 1.3 percent of the PWSs monitored under UCMR3 had measured concentrations of PFOA and PFOS that were greater than the EPA's lifetime HA (lifetime HA limit of 70 ppt or 0.07µg/L) (USEPA 2016a, USEPA 2016b).

Using the occurrence information from UCMR3 and other relevant information, the EPA will propose a regulatory determination for PFOA and PFOS in 2019 for public comment. A regulatory determination is the next step in the SDWA process for developing a national primary drinking water regulation. The Agency also recognizes that there is additional information that the EPA should evaluate regarding PFAS other than PFOA and PFOS, including new monitoring and occurrence data, recent health effects data, and additional information to be solicited from the public, which will inform the development of a national drinking water regulation for a broader class of PFAS in the future.

EPA Priority Action

ACTION: The EPA is committed to proposing a regulatory determination for PFOA and PFOS. In addition, the EPA is committed to proposing additional PFAS for the next round of unregulated contaminant monitoring.

PURPOSE: This is the next step in the SDWA process and will enable the EPA to obtain additional information on PFOA, PFOS, and other PFAS compounds to inform regulatory action.

NEXT STEPS: In 2019, propose a regulatory determination for PFOA and PFOS highlighting key information gathered by the Agency to date. The EPA will invite the public to comment on the Agency's efforts to date, including recommending additional information the Agency should consider in its regulatory determination.

The EPA also intends to propose nationwide drinking water monitoring for PFAS under the next UCMR monitoring cycle utilizing newer methods available to detect different PFAS and at lower minimum reporting levels (MRLs) than previously possible in earlier monitoring. As part of this process, the EPA intends to solicit pre-proposal stakeholder input in 2019 and issue a proposed drinking water monitoring rule (UCMR5) in 2020.

In addition to the available UCMR data, the EPA plans to evaluate the extensive occurrence information for PFAS in source and drinking waters recently collected by some states, and which other states intend to collect in the future. The Agency has also heard extensive concerns from the public about PFAS that were not monitored as a part of the UCMR3 effort. Within the proposed regulatory determination federal register notice for PFOA and PFOS, the EPA plans to highlight the information that is known by

the Agency and invite the public to provide additional information that the EPA can consider, including information from additional data sources related to sampling of additional water systems and for a broader suite of PFAS. Based on this and other information (including UCMR finished water data), the EPA will make a final determination for PFOA and PFOS, and as appropriate, other PFAS and take the appropriate next regulatory steps under the SDWA. In the interim, the Agency intends to prioritize prevention and remediation programs to support local communities currently facing PFAS challenges and will exercise its SDWA authorities where necessary and appropriate.

VI. Identifying PFAS and Addressing PFAS Exposures in Affected Communities

The EPA is focused on identifying and addressing PFAS exposures in order to protect people and communities from exposures to PFAS that present an adverse health risk, especially for the most vulnerable members of the exposed population. Additionally, the EPA is focused on providing tools and information to support federal agencies, states, tribes, and local communities to address PFAS in the environment. This work involves coordinating with others to identify exposures, developing methods in order to measure PFAS in the environment, and supporting cleanup efforts where PFAS has been identified as a risk to human health, including working with other federal partners and using enforcement tools where necessary. Where the EPA finds that there may be an imminent and substantial endangerment to public health related to PFAS contamination, the Agency will consider using its response authority under CERCLA section 104 or utilizing its enforcement authorities such as the SDWA section 1431 or RCRA section 7003.

Work with States, Tribes, and Local Governments on Identifying Exposures

Identifying PFAS is the first step in understanding if PFAS exposure may be of concern to a community. PFAS exposure in the general population occurs primarily through consumption of food that has been stored or cooked in materials containing PFAS, eating contaminated food grown in or collected from contaminated soil or water (Ghisi et al. 2018), eating contaminated meat from animals (e.g., fish), contact with household products contact through contaminated soil and dust (Shoeib et al. 2005), or drinking water that has been contaminated with PFAS. Drinking water contamination is typically localized and associated with a specific source of PFAS (for example, an industrial facility where these chemicals were produced or used to manufacture other products; or an airfield, military base, or petroleum or chemical facility at which PFAS containing foams were used for firefighting or training

(USEPA 2018a, Hu et al. 2016, Guelfo et al. 2018)). In addition to the monitoring conducted by the EPA and states as part of the UCMR program (monitored for six PFAS), some states have taken additional steps to understand the occurrence of PFAS contamination in communities with potential PFAS exposures from current or historical activities. In addition, some states have conducted sampling and monitoring more broadly to identify locations with PFAS contamination. These steps include sampling drinking water—either in large water systems that serve multiple communities, private potable wells potentially impacted by releases, or sites where PFAS-containing materials are known to have been used—to gather important baseline data on the presence of PFAS in the environment. A number of environmental monitoring activities are also ongoing to measure and assess trends of PFAS in air, water, fish, wildlife, and sediment. In addition, some states are conducting biomonitoring studies to measure the levels of PFAS in people (ASTHO 2018). States can also consider updating their source water assessments to account for potential PFAS risks based on monitoring results or known sources of contamination. The EPA is working with our partners to develop and disseminate sampling, measurement, and treatment tools to help stakeholders concerned about PFAS in their communities to implement actions to prevent and mitigate harmful human exposures to PFAS.

Many stakeholders have questioned the extent and magnitude of PFAS contamination across the United States. To help fill these information gaps, the EPA intends to compile baseline, publicly available, PFAS environmental data into a visual map. Mapping tools can be used to show known or potential PFAS contamination sources and related information. The EPA may also specify sites of interest to environmental monitoring, such as wildlife refuges and fisheries, as well as additional impacted environmental media (for example, air or soil). These efforts can be used to help assess environmental trends in PFAS concentrations and serve as one source of information for local and regional authorities.

The EPA is also exploring how to coordinate sampling, data sharing, and data evaluation across environmental media and biota to provide online tools that can provide information about PFAS detections for government and public users. The EPA plans to work with state partners to develop data sharing standards so that testing results (either government sampling results or public testing) can be shared in a way that is accessible and useful. The EPA will explore development of a PFAS inventory and data plan. The EPA intends to play a lead role in distributing tools that provide the public with an integrated look at what is known about PFAS detections.

Development of Field and Laboratory Methods to Measure PFAS in the Environment

When available, validated analytical methods for measuring PFAS and PFAS precursors in multiple environmental media enable a more accurate understanding of PFAS occurrence and exposures. This information in turn helps the EPA's effort to focus toxicity studies on the most prevalent PFAS exposures in the environment. With the information produced using validated analytical methods, decision makers can also understand the extent of PFAS contamination and better design and execute remediation and treatment. The EPA recently released an expanded drinking water Method 537.1 to include additional PFAS, including GenX chemicals. Longer-term efforts include the development and multi-lab validation of methods (e.g., SW-846, 40 CFR Part 136) for complex water matrices (e.g., wastewater, surface waters, groundwaters), solids (e.g., soil, sediment, biosolids, fish tissues), air (e.g., ambient, stack emission, off-gases), and other PFAS in drinking water not currently captured by Method 537. In

addition, the EPA continues to collaborate with others to refine and apply high resolution mass spectrometry (HRMS) analytical methods for discovery and identification of additional PFAS in environmental media (McCord et al. 2018, Newton et al. 2017, Strynar et al. 2015). These efforts will support federal partners, states, tribes, and other stakeholders in site assessment and remediation and help characterize the broader environmental occurrence and potential exposure to PFAS compounds in drinking water and other impacted environmental media. For more information on the EPA research plans related to PFAS, please see Section VII.

Risk Assessment Definitions

RESEARCH: The EPA conducts laboratory and field observations, compiles and synthesizes information, and develops models and tools in order to understand toxicity, exposure, treatment, and remediation.

HAZARD IDENTIFICATION & DOSE-RESPONSE ASSESSMENTS: The EPA determines whether exposure to a contaminant (e.g., PFAS) has the potential to cause harm to humans and/or ecological systems, and if so, under what circumstances.

EXPOSURE ASSESSMENTS: The EPA models or measures contamination (e.g., in drinking water) and predicts how people and ecological systems can come in contact with a contaminant, along with the size and characteristics of the population exposed (including the most vulnerable) to estimate exposure.

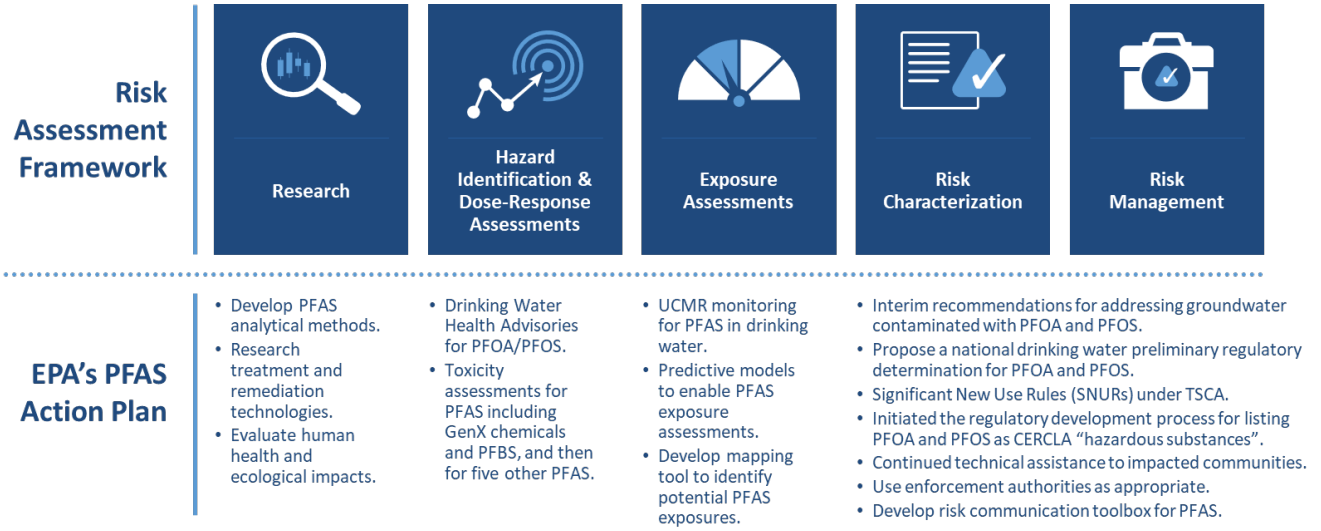
RISK CHARACTERIZATION: The EPA works to integrate the previous steps to create a comprehensive picture of potential PFAS risks, considering hazard, dose-response, and exposure information.

RISK MANAGEMENT: The EPA applies information attained in the previous steps to develop, analyze, and compare options and identify the most appropriate treatment, remediation, or policy response, including how to best exchange information about health or environmental risks among various stakeholders.

Utility of Additional Exposure Information on PFAS

Applying new analytical methods for discovering and measuring PFAS in the environment would enable a better understanding of the sources, types of PFAS, and the exposure pathways which bring PFAS into contact with people and ecosystems. This information could be used to prioritize PFAS for toxicity testing and to facilitate assessment of the relative importance of different pathways (how much PFAS exposure is via food, water, dust, or other media/pathways). This information, combined with more knowledge about PFAS toxicity, could enable stakeholders to identify the PFAS exposures which are of greatest relevance and potential impact to humans and ecosystems, enabling them to prioritize their management efforts and allocate their resources to achieve the maximum reduction in risk. For more information on the EPA's research efforts related to risk assessment, please see Section VII.

EPA Actions and the Risk Assessment Framework



Mitigating PFAS Exposures

To prevent adverse effects to human health and the environment both now and in the future, the EPA is prioritizing short-term exposure prevention and long-term cleanup goals. The EPA will work with federal, state, tribal, and local agencies to employ appropriate authorities, when necessary, to address or prevent PFAS contamination. Potential federal enforcement, regulatory, and response authorities include, for example, the SDWA; RCRA sections 3004(u) and (v); 3005; 3008(h); 3013; and TSCA sections 5, 6, 7, and 8. Additionally, the EPA will continue to develop tools and provide information to support decision-making on mitigating PFAS exposures.

Hazardous Substance Listing for PFAS

EPA Priority Action

ACTION: The EPA has initiated the regulatory development process for listing PFOA and PFOS as CERCLA "hazardous substances" using available statutory mechanisms.

PURPOSE: A "hazardous substance" designation under CERCLA provides more options for the federal government to facilitate use of response and enforcement authorities.

In addition to short-term exposure prevention, the EPA will continue to provide technical assistance on site-specific PFAS challenges across the country, including using CERCLA and other authorities, as appropriate, to investigate sites when needed. The EPA is also developing Interim Recommendations for Addressing Groundwater Contaminated with PFOA and PFOS to support site-specific cleanup efforts (see section V). An important long-term action for federal agencies, states, tribes, communities, and the public is the development of additional tools to facilitate cleanup of PFAS-contaminated sites and recover cleanup costs from responsible parties. In order to augment the EPA's ability to use its CERCLA federal response authority, the EPA is moving forward with how best to designate PFOA and PFOS as CERCLA "hazardous substances" using one of the available statutory mechanisms. Following the PFAS Summit in May 2018, the EPA began an intensive effort to examine the statutory options that could be used to designate PFOA and PFOS as CERCLA hazardous substances. This effort included consideration of the benefits and challenges, as well as the timing and criteria for each available option. There are several statutory authorities available to define PFOA and PFOS as CERCLA hazardous substances, including CERCLA, RCRA, TSCA, Clean Water Act (CWA), and the Clean Air Act (CAA). The EPA is initiating the regulatory development process for listing PFOA and PFOS as CERCLA hazardous substances.

Tools to Mitigate PFAS in Our Nation's Waters

The EPA will continue to work towards providing impacted communities with the tools they need to mitigate risks from PFAS. To further support communities in making decisions about mitigating exposures from drinking water, the EPA intends to continue to update the Drinking Water Treatability Database for PFAS, including treatability and cost information for different technologies and additional PFAS of concern. The treatability database presents information on the control of contaminants in drinking water through treatment processes such as activated carbon, ion exchange, and high-pressure membranes. The treatability database allows utilities, emergency responders, regulators, and other stakeholders access to comprehensive information gathered in a single location. The EPA is also conducting bench-, pilot-, and full-scale experiments to identify performance and cost of treatment (both capital and operations and maintenance), along with potential unintended consequences of employing specific technologies. Better understanding the capabilities of available treatment technologies will further enable the removal of PFAS in drinking water.

Several states are taking actions related to PFAS, including product labeling and consumer products laws, chemical action plans, listing select PFAS as hazardous wastes or designating select PFAS as hazardous substances through state-specific authorities, and developing standards and guidance values

to limit concentrations of PFAS in groundwater or drinking water (ITRC 2018b). PFAS can be considered pollutants under the Clean Water Act, and states can use National Pollutant Discharge Elimination System (NPDES) permits to control discharges from point sources containing PFAS into receiving waters, including sources of drinking water. To support states in managing their water quality, the EPA will evaluate development of ambient water quality criteria under section 304(a) of the Clean Water Act to facilitate state permitting efforts, if adequate data are available.

Parties responsible for PFAS releases, states, and utilities have acted to reduce exposure to PFAS in drinking water from community water systems and private wells through the installation of treatment systems, providing connections to public water systems, point-of-use filters, point-of-entry treatment systems, or through the provision of bottled water. Conventional drinking water treatment technologies (coagulation, flocculation, clarification, filtration, and disinfection) have not been found to be effective in removing PFAS. Technologies have been found to remove longer-chain PFAS, such as PFOA and PFOS, from drinking water including activated carbon adsorption, ion exchange resins, and high-pressure membranes (Rahman et al. 2014, Eschauzier et al. 2012, Flores et al. 2013). These technologies can be used in drinking water treatment facilities, in point-of-entry systems to treat all the potable water that enters a home or other building, or at the point-of-use of potable water, such as in a kitchen sink (USEPA 2018d). The EPA is currently working to better understand the efficacy of commercially available point-of-use and point-of-entry treatment applications for PFAS. In some cases, these treatment technologies can result in considerable cost to utilities or homeowners within communities that have been impacted by PFAS. Concerns continue to be expressed by communities regarding the potential for ongoing exposure to PFAS that are less well characterized or are less amenable to measurement and/or removal using existing treatment technologies.

Each state administers the Drinking Water State Revolving Fund (DWSRF) to provide low-interest loans for drinking water infrastructure and technical assistance to publicly-owned community water systems (CWSs), privately-owned CWSs, and non-profit non-CWSs to facilitate compliance with national primary drinking water regulations or to significantly further the health protection objectives of the SDWA (USEPA 2018d, USEPA 2018e). Under the SDWA, states may set aside up to up to 31% of their DWSRF capitalization grant to fund state programs and third parties to provide assistance and build the capacity of drinking water systems. DWSRF set-asides can fund laboratory or testing equipment for research or contamination prevention. In addition, states with a synthetic organic chemical monitoring waiver program can use the DWSRF to assist with special-purpose monitoring, including PFAS, at local systems that have not yet tested for PFAS (USEPA 2017).

A detailed understanding of the sources of PFAS contamination can help communities impacted by PFAS with the development of long-term solutions. Common sources of PFAS include groundwater plumes associated with areas where fire-fighting foam was used, wastewater effluent or air emissions from industrial facilities where PFAS are manufactured or used, and landfills, including leachate, where materials with high levels of PFAS have been disposed. If a source (or sources) can be identified, then actions can be taken to remediate, reduce or divert the source, or address exposure. As part of the EPA's statutorily-required Effluent Guidelines planning process, the EPA has reviewed readily-available information about PFAS surface water discharges to identify industrial sources that may warrant further study for potential regulation through national Effluent Limitation Guidelines and Standards (ELGs).

Based on the very limited amount of data available, the EPA has identified several industries that are likely to be discharging PFAS in their wastewater and will begin a more detailed study to evaluate the potential for PFAS presence in their wastewater discharges. As part of this study, the EPA plans to gather more detailed information for the following point-source categories: organic chemicals, plastics, synthetic fibers, pulp and paper, textiles, and airports.

Work with Federal Partners

The EPA continues to collaborate with federal agencies to address challenges associated with PFAS. As part of interagency cross-coordination efforts, additional actions may be taken by other agencies to mitigate existing PFAS exposures. The EPA is working with other federal partners, through outreach on EPA PFAS products such as the GenX chemicals and PFBS toxicity assessments as well as the Interim Recommendations for Addressing Groundwater Contaminated with PFOA and PFOS. The EPA plans to collaborate with other agencies on PFAS-related research, for example on toxicology studies of a broad number of PFAS with the National Institute of Environmental Health Sciences (NIEHS) National Toxicology Program (NTP). Additionally, the EPA will also work with other federal agencies such as the Food and Drug Administration (FDA), as appropriate, to support efforts regarding PFAS-related food safety issues. The EPA plans to continue coordinating with other federal agencies, such as ATSDR, FDA, and the United States Department of Agriculture (USDA), to ensure we are providing clear and consistent risk communications. The EPA also plans to work with federal partners, such as the Department of Defense (DoD) at military sites or USDA with respect to agriculture, to reduce PFAS exposures. DoD activities at military sites have included, for example, identifying the extent of PFAS contamination of drinking water sources as a result of releases from DoD facilities, ensuring that, where such contamination has occurred, communities at or near DoD facilities are not reliant on drinking water above the EPA's Health Advisory value for PFOA or PFOS.

VII. Research, Development and Technical Assistance for Addressing PFAS-Related Public Health Questions

Research, Development, and Technical Assistance

Problem Scoping and Formulation

The science needed to protect public health and the environment from PFAS exposure cuts across many applications and disciplines. The risk assessment/risk management paradigm provides a useful means to assess the state of the science available for informing decisions, and to identify gaps in knowledge needed to address the highest priority issues. *Risk assessment,* the integration of PFAS exposure and toxicity information, helps to determine if, when, and where risk exists (probability of harm) to human health or the environment from PFAS, considering both toxicity and exposure. *Risk management* involves solving a PFAS problem once it has been properly identified and characterized, considering available scientific tools and data, as well as economic, legal, social, technological, and policy factors.

The EPA's initial scoping of information available to decision makers for assessing and managing PFAS risks revealed deficiencies in all key areas of the risk paradigm:

- **Hazard and Toxicity:** There are many PFAS of potential concern to the public that may be found in the environment. Most of these PFAS lack sufficient toxicity data to inform our understanding of the potential for adverse human or ecological effects.
- **Exposure:** Information for many PFAS sources, fate and transport, and human and ecological exposure is sparse, both spatially and temporally.

- **Treatment and Remediation:** There is little information on effective methods and costs for treating or removing PFAS from drinking water, groundwater, wastewater, air, soils, and sediments.
- **Science Communication:** Stakeholders lack easy access to the growing body of technical information that can assist them in applying PFAS science to their specific problems and communicating to their constituents.

The EPA's research program will focus on an integrated set of research activities aimed at filling gaps in our current ability to conduct sound risk assessment and risk management activities. This research program is designed to address these data gaps and enable stakeholders to begin making effective decisions for identifying and mitigating risk from PFAS in the environment, as mentioned in Section VI.

The EPA's PFAS research plan consists of ***near term*** (<2 years) and ***long term*** (>2 years) research activities in four areas:

- What are the human health and ecological effects of exposure to PFAS?
- What are the significant sources, fate and transport pathways, and exposures to humans and ecosystems?
- What are the costs and effectiveness of different methods for removing and remediating PFAS in the natural and built environment?
- How does the EPA support stakeholders in using science to protect public health and the environment?

While the activities highlighted in this section are planned to be completed on a longer-term time horizon, many of these efforts will have visible interim milestones and may produce shorter-term products. Many different entities have an interest in—and are actively conducting—research to address PFAS, and so there is a substantial opportunity to advance PFAS science by effective coordination and collaboration amongst these entities. The EPA is committed to leading federal action to protect human health and the environment and to coordinating and cooperating with state and other federal agencies, academia, industry, and non-government organizations to build a body of best available science in the areas described below and to support policy and management decisions and actions by all stakeholders.

Research Area 1: *What are the human health and ecological effects of exposure to PFAS?*

One of the main research needs is a better understanding of the potential human health and ecological hazards from exposure to PFAS. Characterizing hazards through the development of hazard and dose-response assessments capitalizes on existing scientific information where available. For data-poor PFAS, an integrated approach to testing and assessment includes the use of existing hazard information, where available, coupled with data and information generated from new advances in computational and high throughput toxicology and ecotoxicology. These efforts will help the Agency develop toxicity values for additional PFAS, as discussed in Section V.

Research to advance our understanding of human health and ecological effects of PFAS will consist of three complementary lines of work:

- **Development of human health toxicity values where suitable data are available.** The EPA plans to develop cancer and noncancer toxicity values for PFAS where sufficient health effects data currently exist, are publicly available, and adequately support human health toxicity value derivation. The EPA will use established risk assessment guidelines and methods to develop standard toxicity values, such as oral reference doses (RfDs), inhalation reference concentrations (RfCs), oral cancer slope factors (CSFs), and cancer inhalation unit risks (IURs). These assessments will undergo interagency consultation, public comment, and independent external peer-review prior to finalization. The EPA currently has published toxicity assessments for PFOA and PFOS. ***In the near term*** the EPA plans to complete toxicity assessments for GenX chemicals and PFBS. The Agency has begun work on assessments for PFBA, PFHxA, PFHxS, PFDA, and PFNA. The EPA intends to coordinate with federal partners, including ATSDR, on prioritizing and conducting future PFAS toxicity assessments. The EPA will build on work by universities, industry, and other government agencies who are conducting and publishing the peer-reviewed toxicological and epidemiological studies needed to support toxicity assessment.

- **Using computational toxicology approaches to fill in gaps.** For the many PFAS for which published peer-reviewed data are not currently available, the EPA plans to use new approaches such as high throughput and computational approaches to explore different chemical categories of PFAS, to inform hazard effects characterization, and to promote prioritization of chemicals for further testing. These data will be useful for filling gaps in understanding the toxicity of those PFAS with little to no available data. ***In the near term***, the EPA intends to complete assays for a representative set of 150 PFAS chemicals, load the data into the CompTox Chemicals Dashboard for access, and provide peer-reviewed guidance for stakeholders on the use and application of the information. ***In the long term,*** the EPA will continue research on methods for using these data to support risk assessments using New Approach Methods (NAMs) such as read-across and transcriptomics, and to make inferences about the toxicity of PFAS mixtures which commonly occur in real world exposures. The EPA plans to collaborate with NIEHS and universities to lead the science in this area and work with universities, industry, and other government agencies to develop the technology and chemical standards needed to conduct this research.

- **Ecological toxicity.** Ecological toxicity information is also needed by stakeholders to inform risk assessment and management to protect ecosystems, animals, and plant resources they support, and ultimately the human benefits that stem from these resources, including, for example, the prevention of potential PFAS risks associated with consuming game animals and fish. ***In the long term***, the EPA plans to work to identify species which are sensitive or susceptible to PFAS exposure; gather and synthesize information on bioaccumulation of PFAS in organisms and food chains; and, where indicated, develop benchmarks and thresholds for ecological toxicity. The EPA plans to collaborate with the United States Geological Survey (USGS), United States Army Corps of Engineers (USACE), and universities to lead the science in this area.

Research Area 2: *What are the sources, fate and transport pathways, and exposures to humans and ecosystems?*

The diversity of the PFAS family of chemicals enables the use of PFAS for many diverse industrial processes and end use products, which in turn means there are numerous potential sources and pathways by which PFAS can move from a source through the environment. Understanding this complexity is necessary to understand PFAS exposure. The EPA plans to address this complexity through two lines of research and development:

- **New analytical methods.** Developing, validating, and applying new analytical methods for discovering and measuring PFAS in air, water, and soil will enable a better understanding of the specific subsets of PFAS that exist in the environment, as well as the exposure pathways that potentially bring those PFAS into contact with people and ecosystems. This will enable the creation of datasets to better understand fate and transport pathways and to identify cases where exposures exceed thresholds of concern. ***In the near term***, the EPA plans to develop, validate, and publish reliable sampling and laboratory analytical methods to detect, identify, and quantify PFAS in different environmental media (including drinking water, groundwater, wastewater, air, and soil) and in other kinds of samples (e.g., plant and animal tissue), as needed. This includes analytical methods for known PFAS of concern, as well as methods to identify and detect new, currently unknown, PFAS in the environment. ***In the long term,*** the EPA will continue to prioritize, develop, and validate analytical methods for emerging PFAS of concern. The EPA plans to collaborate with USGS, DoD, National Institute of Standards and Technology (NIST), FDA, and private industry to lead the science in this area and rely on universities and industry to develop the technology needed to enable new analytical methods.

- **Exposure assessment.** Exposure information enables decision makers to prioritize the PFAS exposures that are of greatest relevance and impact to human health and the environment, enabling them to prioritize management actions and allocate resources to achieve the maximum reduction in risk. ***In the near term***, the EPA plans to develop a mapping tool to house public datasets of known PFAS source and occurrence data, and tools to analyze PFAS exposure through multiple routes (via water, food, inhalation, or dermal contact). ***In the long term***, the EPA intends to build predictive models to enable PFAS exposure assessment from site-specific to national in scope, to better understand where and how PFAS move through the environment to impact people and ecosystems, and to estimate how much PFAS reaches people via air, water, food, and other pathways. The EPA plans to collaborate with the Department of Housing and Urban Development (HUD), ATSDR, and other federal agencies, as appropriate, to lead the science in this area.

Research Area 3: *What are the costs and effectiveness of different methods for removing and remediating PFAS in the natural and built environment?*

Current technology and approaches for treating or removing chemical contaminants from air, water, and soil are not always effective for PFAS. Better information is needed on the costs and effectiveness of different treatment systems for different PFAS of concern, as well as the development of new treatment technologies that are less expensive, easier to operate, and more sustainable than existing technologies. The EPA is addressing this information need through two related lines of research:

- **Drinking water treatment.** The EPA is evaluating treatment technologies for removal of PFAS from drinking water. States, public water utilities, communities, and federal facilities will benefit by having treatment technology guidance and accurate cost numbers for the treatment of PFAS in drinking water. ***In the near term***, the EPA plans to evaluate performance, cost, and potential unintended consequences of drinking water treatment technologies for different PFAS in small, medium, and large systems. The Agency plans to place data in the EPA's online Drinking Water Treatability Database and associated cost models. The EPA plans to collaborate with states, federal agencies, public water utilities, and private industry to lead the science in this area and will work closely with universities and industry who are developing the treatment technology advances needed to support this research.

- **Contaminated site cleanup.** The complexity of PFAS sources and uses means there are multiple ways that specific sites can become contaminated by PFAS. Examples include improper dumping or disposing of PFAS-contaminated waste, accidental or intentional spills of PFAS-containing products such as firefighting foam, or leaking of PFAS in leachate from landfills. This can result in the contamination of soils, sediments, groundwaters, and surface waters. ***In the near term***, the EPA plans to evaluate the effectiveness and cost of existing treatment and remediation technologies for a variety of PFAS-contaminated sites and develop and test new technologies and approaches for cleaning up PFAS contamination. The EPA plans to collaborate with DoD, states, industry, and non-government organizations to lead the science in this area and work closely with universities and industries developing the treatment technology advances needed to support this research.

Research Area 4: *How does the EPA support stakeholders in using science to protect public health and the environment?*

Stakeholders have varying levels of knowledge and expertise for using the science products that will result from the EPA's research. Part of the research process therefore involves communication of the Agency's research in multiple ways to make the science usable to all stakeholders. This communication needs to include the proper context and any applicable limitations inherent in the work. This may also include applying tools in collaboration with stakeholders through technical assistance. The EPA plans to conduct two lines of work in support of stakeholders.

- **Science communication.** PFAS are of interest to a variety of stakeholder groups. It is important that the EPA maintain suitable communication with each of these groups and facilitate access to new research products as they become available. ***In the near and long term***, the EPA plans to facilitate access to the research products described in this plan via multiple avenues, including publications, reports, online tools and databases, fact sheets, workshops, webinars, and summaries describing our science. The EPA plans to make this information readily available using the EPA PFAS website as the main point of access. The EPA intends to collaborate with states, tribes, and communities to lead work in this activity.

- **Technical assistance.** In certain cases, the EPA provides technical advice, assistance, and collaboration to state, tribal, federal, and community partners in a manner consistent with the Agency's goal of Cooperative Federalism. These technical assistance activities inform cost-efficient and cost-effective risk management decisions by the EPA and its partners, as well as help to advance the science through applied research. ***In the near and long term***, the EPA plans to continue to prioritize engagement in these activities.

VIII. Risk Communication and Engagement

Risk communication and engagement are critical for the EPA to effectively support communities across the country that are addressing PFAS issues. The EPA is actively working to enhance the way in which agencies communicate about potential human health risks that may be associated with these chemicals. PFAS are a complex group of chemicals that can differ in terms of how they are used, how people are exposed, and how they potentially impact public health and ecosystems. There is a lack of definitive scientific information about many chemicals in the PFAS family, making it challenging to communicate with the public about their associated health risks. The EPA also supports the efforts of other federal partners to develop information related to PFAS. Other agencies may issue different values based on factors such as their own statutory, regulatory, or case-specific analyses and exposure assumptions. The EPA continues to take concrete steps, in cooperation with our federal, state, and tribal partners, to communicate how the efforts of the EPA and other federal, state, and tribal agencies help to protect public health and the environment from risks related to PFAS.

Importance of Effectively Communicating PFAS Information to the Public

At the National Leadership Summit and throughout the community engagements, the EPA heard how important it is to communicate effectively with the public and to be transparent in sharing what is known and unknown in a timely manner. The EPA heard that speaking with one voice and providing consistent messaging across federal, state, tribal, and local authorities helps to build trust and ensures that the public has a clear understanding of any PFAS issues that need to be addressed. The EPA also heard that it is important to clearly explain the actions the Agency is taking, as well as the specific concerns that those actions are intended to address. Other comments submitted to the EPA highlighted how important it is to provide information to stakeholders as quickly as possible, while also taking into account the high levels of uncertainty that surround these chemicals. Appendix B provides additional

discussion about feedback from the community engagements and information submitted to the PFAS docket.

The EPA's Goals and Actions on PFAS Risk Communication

PFAS are of significant interest to a diverse set of stakeholders. Clear and consistent communication from all information sources will help stakeholders determine the most appropriate PFAS risk management approach and help the public understand the response. Through this Action Plan, the EPA's goal is to work with other agencies to:

1. Enhance the public's understanding of PFAS by providing clear and consistent information;
2. Enhance the public's understanding of the regulatory processes available to address PFAS and the different standards established for PFAS;
3. Build trust with the public as we work together to address these chemicals; and
4. Provide the public with an understanding of the uncertainties associated with PFAS measurement, exposure, and toxicity, and the importance of considering these uncertainties when identifying effective risk management actions.

For communities directly impacted by PFAS, the EPA plans to:

1. Work in coordination with other federal agencies and local, state, and tribal governments on clearly communicating PFAS information;
2. In support of responses to PFAS found in communities, work with the community to identify the lead agency and explain the role of each agency involved. Establish contact points responsible for managing community questions;
3. Communicate pathways of exposure and what is being done to mitigate exposure through those pathways;
4. Enhance the public's understanding of the potential human health effects associated with PFAS exposure; and
5. Provide information on tangible steps individuals can take on their own to manage risk.

To best support and leverage the efforts of other federal partners, the EPA is committing in the short-term to convene a federal interagency PFAS risk communication workgroup to ensure, as appropriate, collaborative interagency action and consistent messaging on PFAS toxicity that is informed by the best available science. In addition, the EPA plans to enhance communications with the public on PFAS through the following actions:

1. In 2019, develop a risk communication toolbox that includes materials and messaging for federal, state, tribal and local partners to use to inform the public, as they deem appropriate.
2. Continue to listen to and engage with the public; and

3. Continue to support states, tribes, and local officials who have purview in protecting the environment and public health, including the Environmental Council of States (ECOS), the Association of State and Territorial Health Officials (ASTHO), the Association of Clean Water Administrators (ACWA), the Association of State Drinking Water Administrators (ASDWA), the National Tribal Toxic Council, and the Association of State and Territorial Solid Waste Management Officials (ASTSWMO).

Information Needed by Stakeholders to Effectively Communicate About PFAS

Effective communication at the federal, state, tribal, and local level begins by clearly summarizing what is known and unknown about PFAS, with a focus on the key questions with which the public is most concerned. The EPA will help to advance these efforts by continuing its work with other agencies to develop a risk communication toolbox that will include the following:

- Key messages
- Questions and answers
- Infographics
- Fact sheets
- Sample language/template for potential notifications
- Sample communication materials
- Links to available data sources and tools

The EPA will make available materials and informational fact sheets on the EPA's PFAS webpage as part of the risk communication toolbox and, as necessary, will continuously update the information as the science around PFAS evolves. To find the complete set of tools, visit: https://www.epa.gov/pfas/pfas-communication-and-outreach-tools.

Stakeholder Engagement on PFAS

The EPA conducted extensive public outreach in the development of the PFAS Action Plan, including gathering diverse perspectives through the May 2018 National Leadership Summit, direct engagement with the public in impacted communities in five states, engagement with tribal partners, and roundtables conducted with community leaders near impacted sites (USEPA 2018f). The EPA also obtained recommendations from the Local Government Advisory Committee (LGAC), a chartered policy committee comprised of elected and appointed local officials. In addition, the Agency reviewed approximately 120,000 comments in the public docket that was specifically established to gather input for the Action Plan.

Through these engagements, a broad range of stakeholders provided input to the EPA about ongoing PFAS challenges facing states, tribes, and local communities, as well as specific actions needed from the EPA and state regulators in order to protect the public from PFAS in the environment. Key public priorities include the need for identification and remediation of known sources of contamination; source water protection for drinking water supplies; resources to support effective communication with the

public; long-term policy solutions; reliable, enforceable, and actionable standards and risk information; validated and cost effective analytical and sampling methods and tools; treatment solutions; enforcement strategies to reduce the cost burden on citizens; and coordination among all parties involved in mitigation and response. The Agency received comments identifying the importance of developing and relying on the best available science even if that means not rushing to implement regulatory actions in the near term. Stakeholders also emphasized the need to balance the potential cost and burden associated with managing PFAS with the costs and benefits of addressing other competing public health and environmental protection priorities such as the presence of lead in community water systems. Among other things, the LGAC recommended using existing funding tools, such as the State Revolving Funds to address PFAS, prioritizing PFAS-related risk communication activities, developing new methods and certification programs, and using risk-based approaches to address PFAS contamination issues, being mindful that clean and safe water are valued by every American citizen. The EPA plans to continue to seek feedback from stakeholders on actions to address PFAS.

National Leadership Summit

Over 220 participants, including senior officials from 40 states, 3 tribes, Guam, Northern Marianas Islands, 13 federal agencies, congressional staff, and dozens of associations, industry groups, and non-governmental organizations.

Community Outreach

Over 1,000 participants at 7 locations, including community engagements in New Hampshire, Pennsylvania, Colorado, North Carolina, and Kansas; engagement with tribes at the Tribal Lands and Environment Forum and the Saginaw Chippewa Tribe; and a roundtable in Michigan.

Public Docket

Approximately 120,000 comments received.

Information for Individuals Concerned about PFAS

Individuals in communities that are served by a public water system can contact their local water supplier to ask for information on any PFAS monitoring the utility may have conducted. Members of the public are also encouraged to request a copy of their drinking water Consumer Confidence Report. While there are currently no federal drinking water regulations for PFAS, this report provides useful information on other regulated contaminants found in local drinking water. If owners of drinking water wells not regulated by the SDWA (i.e., private potable wells) have reason to believe their well may contain PFAS (e.g., due to proximity to a known contamination site or probable source of PFAS), they could consider contacting their state or local health department for further guidance. Owners may also consider well testing to learn about PFAS that may be in their drinking water. For more information about well testing, please visit https://www.epa.gov/privatewells/protect-your-homes-water. The EPA recommends contacting your state for a list of laboratories that are certified to test for PFAS using EPA Method 537. If you find PFAS in your drinking water, certain PFAS can be reduced or removed through

the use of in-home point-of-use or point-of-entry water filters. It is important to keep in mind that any in-home treatment device should be certified by an independent party, currently available for PFAS (NSF 2018), and should be properly maintained to ensure that the treatment system remains effective over time.

For those concerned about food (plant or animal) collected from an environment that may contain PFAS, the EPA recommends contacting your local health department. All 50 states and some U.S. territories and tribes have fish consumption advisory programs to protect people from potential human health risks of eating contaminated fish caught in local waters. However, due to the limited sampling at this time, few locations have information specific to PFAS. In some states, pollutant levels in certain types of fish and shellfish collected from contaminated bodies of water have led to health-based consumption advisories for some PFAS, particularly PFOS (USEPA 2016d, State Impact Pennsylvania 2018, State of Michigan 2018). The EPA maintains a national database of fish and shellfish advisories issued by states where the public can find information on safe consumption guidelines (https://fishadvisoryonline.epa.gov/General.aspx) and for the most up to date information links to state and tribal fish consumption advisory websites (https://fishadvisoryonline.epa.gov/Contacts.aspx).

IX. Conclusion

In addition to the four priority actions the EPA announced at its May 2018 National Leadership Summit, this Action Plan highlights the many activities that the EPA plans to lead in collaboration with federal, state, tribal, and local partners to understand, communicate, and take steps to effectively manage potential concerns associated with the presence of PFAS in the environment. Where deemed appropriate and necessary, the EPA will prioritize preventing environmental contamination and identifying approaches that reduce the costs of PFAS management faced by local communities. Efforts discussed in this plan are also intended to encourage the use of safer PFAS formulations and/or PFAS alternatives and limit PFAS discharges, releases, and emissions. Where PFAS contamination in the environment has already occurred, the Agency will facilitate remediation efforts by providing groundwater cleanup recommendations and initiating the regulatory development process for listing certain PFAS as hazardous substances. For those cases where cleanup actions are necessary to prevent exposure to contaminated environmental media, the Agency is evaluating active management and treatment options and evaluating available treatment technologies. The EPA is also proposing a national drinking water regulatory determination for PFOA and PFOS in 2019 for public comment. The Agency will also gather and evaluate additional information that may inform the development of a national drinking water regulation for a broader class of PFAS in the future. The EPA is committed to working with other federal agencies, states, tribes, and local communities to coordinate and advance how we respond to PFAS concerns throughout the country.

The EPA is taking a leadership role to ensure that instances where PFAS pose risk to human health or the environment are identified and quickly addressed. The EPA plans to work in close coordination with multiple entities, including other federal agencies, states, tribes, local governments, water utilities, industry, and the public. This PFAS Action Plan highlights key EPA PFAS-related activities and reinforces the EPA's commitment to better understand potential impacts from a broad suite of PFAS, and, where necessary, take steps to reduce any risks they may pose to public health and the environment.

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Appendix A: EPA PFAS Activities

Appendix A contains a detailed list of completed and ongoing PFAS activities at the EPA. This list is not intended to be exhaustive of all the EPA's activities on PFAS.

Tool/Activity	Purpose	Timeframe
Preventing PFAS Exposures: What is EPA doing to reduce risks from PFAS?		
Significant New Use Rule; Final Rule and Supplemental Proposed Rule: Perfluoroalkyl Sulfonates (67 FR 11008)	The EPA published a SNUR to require notification to the EPA before any future manufacture (including import) of 13 PFAS chemicals specifically included in the voluntary phaseout of PFOS by 3M that took place between 2000 and 2002.	Completed March 2002
Significant New Use Rule: Perfluoroalkyl Sulfonates (67 FR 72854)	The EPA issued a SNUR for 75 PFAS, requiring manufacturers and importers to notify the EPA at least 90 days before starting the manufacture or importation of these chemical substances for the significant new uses described.	Completed December 2002
2010/2015 EPA PFOA Stewardship Program	The EPA launched 2010/2015 PFOA Stewardship Program with eight companies in 2006 to reduce PFAS emissions and product content by 95%; by 2015 reduce PFAS emissions and product content by 100%. All participating companies met the program goals.	Ongoing Started in 2006
Premanufacture Notification Exemption for Polymers; Amendment of Polymer Exemption Rule to Exclude Certain Perfluorinated Polymers (75 FR 4295)	The EPA published a final rule that amended the Polymer Exemption Rule to no longer exclude from eligibility polymers that include any one or more of the following: PFAS, PFAC, 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. Compliance date was January 27, 2012.	Completed May 2012
Significant New Use Rules: Perfluoroalkyl Sulfonates and Long-Chain Perfluoroalkyl Carboxylate Chemical Substances (78 FR 62443)	The EPA amended a SNUR to designate as a significant new use PFAS that have completed the new chemical review process under TSCA but have not yet commenced production or import and processing. The EPA also finalized a SNUR to designate as a significant new use LCPFAC chemical substances used in manufacturing (including importing) and processing of carpets or for treating carpet.	Completed October 2013
Significant New Use Rules: Long-Chain Perfluoroalkyl Carboxylate and Perfluoroalkyl Sulfonate Chemical Substances Proposed Rule (80 FR 2885)	The EPA proposed a SNUR for LCPFAC chemical substances that would require manufacturers (including importers) of PFOA and PFOA-related chemicals, including as part of articles, and processors of these chemicals to notify the EPA at least 90 days before starting or resuming new uses of these chemicals in any products. The EPA plans to follow up on the 2015 SNUR.	Completed January 2015

Tool/Activity	Purpose	Timeframe
New Chemicals Program Review of Alternatives for PFOA and Related Chemicals	The EPA has reviewed hundreds of new chemical substitutes for PFOA, PFOS, and other long-chain PFAS under the EPA's New Chemicals Program since 2000. The EPA reviews the new substances to identify whether the range of toxicity, fate, and bioaccumulation issues that have caused past concerns with perfluorinated substances may be present, as well as any issues that may arise by new chemistries, to ensure that the new chemical may not present an unreasonable risk to health or the environment. One outcome of the EPA's review of a PMN for a new chemical substance or review of a SNUN is the issuance of an order under section 5(e) of TSCA. Most TSCA section 5(e) Orders issued by the EPA are Consent Orders that are negotiated with the submitter of the notification.	Ongoing Started 2000
Understanding and Addressing PFAS Toxicity: What is the EPA doing to advance the science to support New Benchmarks?		
Lifetime Health Advisories for PFOA and PFOS	The EPA released lifetime health advisories (HAs) and health effects support documents for PFOA and PFOS. The EPA's HAs, which are not regulations, identify the concentration of PFOA and PFOS in drinking water at or below which adverse health effects are not anticipated to occur over a lifetime of exposure.	Completed May 2016
List of available scientific literature on toxicity for 31 PFAS of interest loaded to the HERO database	The EPA updated the Health and Environmental Research Online (HERO) database with available scientific literature (as of August 2017) on PFAS toxicity to detail which scientific studies the EPA has collected.	Completed April 2018
PFAS Chemical Library	Development of a chemical library of PFAS standards (pure samples of PFAS) to support consistent research and method development across the EPA.	Completed April 2018
Provide states access to GenX chemicals data	Provide states access to test data obtained under TSCA authority for information on GenX chemicals (acid and salt).	Completed March 2018
Information on Transcriptomic and *in vitro* assay toxicity testing (Tier 0 and Tier I)	Generate and publish first approximation toxicity and toxicokinetic data from the larger universe of PFAS compounds, in order to make inferences about which subcategories of PFAS might be of highest toxicological concern and thus prioritized for further near-term investigation. These data will also be useful for enabling read-across activities for PFAS with little to no available data. Tests will include a battery of transcriptomic *in vitro* assays (toxicity and kinetics) implemented by the EPA and the NTP.	Anticipated 2019
Tier II PFAS testing	Conduct Tier II *in vivo* toxicity testing for a subset of prioritized compounds based upon data provided from Tier I testing.	Anticipated 2019

Tool/Activity	Purpose	Timeframe
Tri-Services Ecological Risk Assessment Work Group	The EPA Ecological Risk Assessment Forum has a joint work group with the DoD Tri-Services Environmental Risk Assessment Work Group (TSERAWG) to develop ecological risk assessment screening values for PFAS. The DoD has an interagency agreement between the Air Force Civil Engineering Center and the Department of Energy (DOE) Argonne National Laboratory for the development of screening values for PFAS compounds. The PFAS screening values will be available for use at CERCLA sites and RCRA facilities.	Ongoing
Tools and data for evaluating ecotoxicity effects	Identify sensitive and susceptible taxa, synthesize information on bioaccumulation in organisms and food chains, and develop benchmarks and thresholds for ecological toxicity.	Anticipated 2022
Toxicity assessments for additional PFAS	Development of additional peer-reviewed PFAS toxicity assessments for PFBA, PFHxA, PFHxS, PFNA, and PFDA to support stakeholders.	Anticipated 2020
Toxicity assessments for GenX chemicals and PFBS	Provide toxicity assessments to stakeholders for GenX chemicals and an updated PFBS assessment. Both assessments underwent independent peer-review and review by federal partners prior to public comment.	Draft completed November 2018 Finalize 2019
Update Chemistry Dashboard with Information for Additional PFAS	The CompTox Chemicals Dashboard provides users with information on chemical structures, experimental and predicted physicochemical and toxicity data, and additional links to relevant websites and applications. The EPA updated the Dashboard with additional PFAS.	Completed March 2018
Water Contaminant Information Tool (WCIT) Profiles for PFOA and PFOS	Contaminant Profiles for two PFAS, PFOS and PFOA, to be added to the EPA's Water Contaminant Information Tool.	Completed December 2018
CWA Effluent Guidelines Planning PFAS Review	Through the Clean Water Act Effluent Guidelines Planning process, the EPA is examining readily-available information about PFAS surface water discharges to identify industrial sources that may warrant further study for potential regulation through Effluent Limitation Guidelines.	Ongoing
Interim Recommendations for Addressing Groundwater Contaminated with PFOA and PFOS	The EPA anticipates releasing interim cleanup recommendations to address groundwater contaminated with PFOA and/or PFOS to support stakeholders in their remediation efforts.	Anticipated 2019
Evaluation of CWA 304(a) Ambient water quality criteria for PFAS	The EPA is evaluating available data and research to support development of Clean Water Act Section 304(a) Ambient water quality criteria for PFAS.	Anticipated 2022

Tool/Activity	Purpose	Timeframe
Identifying and Addressing PFAS Exposures: What is the EPA doing to help identify communities with potential PFAS impacts, remediate PFAS exposures, and monitor compliance?		
Method Development	The EPA developed Method 537 for measuring PFOA, PFOS, and 12 other PFAS in drinking water to support the Unregulated Contaminant Monitoring Rule.	Completed 2009
Method Development	The EPA expanded Method 537 to measure four additional short-chain PFAS, including HFPO-DA (GenX chemicals) and ADONA. Method 537.1 is available on the EPA's website.	Completed November 2018
Method Development	Validated Direct Injection Method (SW-846) for quantifying 24 PFAS in surface, ground, and waste water matrices (non-drinking water) and solids (e.g., soil and sediment).	Anticipated 2019
Method Development	Validated Isotope Dilution Method (SW-846) for quantifying 24 PFAS in surface, ground, and waste water matrices (non-drinking water) and solids (e.g., soil and sediment).	Anticipated 2019
Method Development	New validated analytical method for PFAS in drinking water focusing on short-chain PFAS which cannot be measured by Method 537.1.	Anticipated 2019
Method Development	Method for sampling and analyzing PFAS in factory stack air emissions.	Anticipated 2020
Method Development	Testing and developing additional methods for possible refinement, including methods to quantify PFAS precursors; Total Organic Fluorine for a general PFAS detection method; and refinement of non-targeted high-resolution mass spectrometry approaches for suspect screening and novel PFAS discovery.	Ongoing
PFAS Geospatial Analytical Tool	Working with states and other federal partners, the EPA is evaluating how to best develop and maintain a GIS resource to consolidate and present PFAS data to inform analysis and understanding of PFAS sources and occurrence in the environment.	Anticipated 2019
Modeling atmospheric fate and transport of PFAS	Incorporate PFAS information into the EPA air models (e.g., the Community Multiscale Air Quality modeling system, AERMOD atmospheric dispersion model) to inform understanding of the potential and significance of atmospheric transport of PFAS.	Anticipated 2022
Unregulated Contaminant Monitoring Rule 3 for Public Water Systems	The third UCMR required monitoring for 30 contaminants (28 chemicals and two viruses) between 2013 and 2015 using analytical methods developed by the EPA, consensus organizations, or both. The purpose of UCMR3 was to collect occurrence data for contaminants suspected to be present in drinking water, but that do not have regulatory standards set under the SDWA. Six PFAS compounds were included in the UCMR3: PFOS, PFOA, PFNA, PFHxS, PFBS, and PFHpA. Of these six compounds, PFOA and PFOS were found in the greatest number of samples, and 1.3% of the public water systems sampled had results that exceeded the reference dose (lifetime HA limit of 70 ppt or 0.07µg/L).	Completed 2013-2015

Tool/Activity	Purpose	Timeframe
Unregulated Contaminant Monitoring Rule 5	The EPA intends to propose nationwide drinking water monitoring for PFAS under the next UCMR monitoring cycle utilizing newer methods available to detect more PFAS and at lower minimum reporting levels (MRLs) than previously possible in earlier monitoring.	Anticipated 2020-2025
Drinking Water Treatability Database-Update for Additional PFAS	Users can utilize the database to identify effective drinking water treatment processes for PFOA, PFOS, and additional PFAS chemicals. This database is continually updated as additional information becomes available.	Ongoing Updated September 2018
Research for Drinking Water Treatment	Conduct bench-, pilot-, and full-scale experiments to discern performance and cost of treatment (both capital and operations and maintenance), along with potential unintended consequences of employing specific technologies. Following a literature review for data gap identification, granular activated carbon and ion exchange treatment technologies will be tested under varying water qualities.	Anticipated Fall 2019
Treatability Cost Models	Updated drinking water PFAS treatability cost models.	Ongoing Updated September 2018
Evaluation of commercially Point-of-Use (POU) and Point-of-Entry (POE) home treatment systems	Investigate commercially available reverse osmosis and granular activated carbon units that can serve households in a point-of-use or point-of-entry applications for 6 PFAS included in UCMR3.	Completed 2018
Evaluation of treatment technologies for contaminated sites	A series of studies evaluating effectiveness and cost of different combinations of treatment train approaches for remediating contaminated sites.	2021
Fourth Contaminant Candidate List (CCL)	The EPA is required by the Safe Drinking Water Act to publish a list of contaminants known or anticipated to occur in public water systems which may require regulation under the Safe Drinking Water Act. The EPA included PFOA and PFOS on the fourth Contaminant Candidate List (the most recent CCL list).	2016
Fourth Regulatory Determination Process	The EPA is working on the Fourth Regulatory Determination process in which the EPA determines whether to regulate at least five contaminants on the CCL and issue final regulatory determinations after considering public input. The EPA is evaluating available information to determine if contaminants on the CCL, including PFOA and PFOS, meet the three criteria for regulation in accordance with the SDWA: (1) whether a contaminant may have adverse health effects; (2) whether a contaminant is found in public water systems with a frequency and at levels of concern; and (3) whether, in the sole judgment of the Administrator, there is a meaningful opportunity for health risk reduction through a national drinking water regulation.	Ongoing Anticipated 2019

Tool/Activity	Purpose	Timeframe
Collection of Great Lakes Environmental PFAS data	The EPA collects and analyzes environmental samples, including whole fish tissue, sediment, air, and water, to determine concentrations and trends of PFAS in the Great Lakes and occurrence in fish tissue.	Ongoing
Evaluate PFAS exposure through fish consumption	Evaluate temporal and demographic patterns of PFAS exposure and the relationship with fish consumption, in the U.S. general population.	Anticipated 2019
Fish Tissue Contamination Studies	To ensure that communities are aware of levels of PFAS in fish they may consume, continue to analyze PFAS in edible fish tissue as part of the National Rivers and Streams Assessment and the Great Lakes portion of the National Coastal Condition Assessment, and include PFAS in the revised list of target analytes that states may consider including in their fish and shellfish contaminant monitoring and advisory programs.	Ongoing
CERCLA Hazardous Substance Listing	The EPA has initiated the regulatory development process for listing PFOA/PFOA as CERCLA hazardous substances.	Ongoing
Scoping biosolids risk assessment for PFOA/PFOS	The EPA is in the early scoping stages of risk assessment for PFOA and PFOS in biosolids to better understand the implications of PFOA and PFOS in biosolids to determine if there are any potential risks.	Anticipated 2020
Identifying PFAS Risks from Chromic Acid Etch Facilities	The EPA's Office of Research and Development and Region 5 are collaborating on a study to characterize PFAS fume suppressants used at chromic acid etch facilities. Both Minnesota and Michigan have identified high levels of PFOS releases from these facilities, even after PFOS was phased out of the fume suppressant products in 2015. Region 5 is assessing if the current PFOS releases are the result of legacy use of PFOS fume suppressants or related to the replacement chemical formulations.	Ongoing
Identify PFAS sources, concentrations, uses, locations, and exposure routes most likely to pose threats to human health and the environment	Continue to make Toxic Substances Control Act (TSCA) data available where possible; identify sources, uses, and locations; develop information on potential high-impact locations; work with states to develop consistent sampling protocols.	Ongoing
Need to integrate data from multiple sources to better understand the presence of PFAS in the environment	Develop data sharing standards that allows states, tribes, communities, public water systems, and other organizations to contribute data about PFAS testing in a consistent manner.	Ongoing

Tool/Activity	Purpose	Timeframe
EPA TSCA section 5(e) order for GenX Chemicals	In 2009 the EPA entered into a Consent Order under TSCA section 5(e) with Dupont (now Chemours) that imposes requirements on the manufacture, processing, use, and disposal of GenX chemicals. Among other requirements, the Consent Order restricts the releases of the GenX chemicals by requiring the recapture of 99% of the chemicals. It also requires certain worker personal protective equipment as well as certain studies to be performed.	Ongoing
TRI listing for PFAS chemicals	Section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) created the TRI Program. The TRI Program is another tool EPA may use to understand the releases of PFAS by industrial and federal facilities. Currently, no PFAS chemicals are included on the list of chemicals required to report to TRI; however, the EPA is considering whether to add PFAS chemicals. In considering listing, the EPA must determine whether data and information are available to fulfill the listing criteria and the extent and utility of the data that would be gathered. In addition, in considering if TRI will provide useful information to stakeholders, the EPA also will consider if those PFAS are still active in commerce. The process for listing includes notice and comment rulemaking to list PFAS chemicals for reporting prior to adding these chemicals to the TRI for annual reporting.	Ongoing

Tool/Activity	Purpose	Timeframe
Regions 1 and 3: Safe Drinking Water Act Section 1431 Emergency Orders to Department of Defense	2014 order to Navy at Warminster (PA) NPL Site directing the Navy to address high levels of PFOS discovered in three drinking water supply wells at and off the Warminster Naval Warfare Center where the elevated levels were four times the provisional health advisory level (which was 200 ppt for PFOS and 400 ppt for PFOA) in one case: Where levels in finished drinking water are above the HA for PFOA or PFOS, the Order required the Navy to provide a permanent drinking water supply as soon as practicable, but in no event later than 6 months after execution of the order. 2015 order to Air Force and Air National Guard at Horsham Air Guard Station/Willow Grove (PA) NPL Site (2015): The order directs the Air Guard/Air Force to treat two onsite public water supply wells and supply treatment to any private well found to exceed the provisional health advisory for PFOS in drinking water. Sampling confirmed that the Guard portion of the facility is also (like the Navy portion from Willow Grove) a source of PFOS offsite migration. The order covers long term treatment for private homes and also for short- and long-term public water supply concerns. 2015 order to Air Force for Contamination at Pease Air Force Base (NH) NPL Site: The order directs the Air Force to address contamination from perfluorinated compounds in drinking water at Pease Air Force Base including a number of actions to address the partial loss of the city's water supply attributed to firefighting foams used at the Base. The PFAS contamination resulted in the shutdown of one public water supply well, and two others could have been impacted if action were not taken to control PFAS migration. Under the order, the Air Force will restore contaminated groundwater in the Pease aquifer.	Ongoing
Annex 3, Chemicals of Mutual Concern, of the Great Lakes Water Quality Agreement	The goal of Annex 3 under the Canada-United States Great Lakes Water Quality Agreement (GLWQA) is to reduce the anthropogenic release of chemicals of mutual concern into the waters of the Great Lakes. In 2016, PFOS, PFOA, and LC-PFCAs—or collectively, PFAS—were designated as chemicals of mutual concern. In designating PFAS as a chemical of mutual concern, Canada and the United States have agreed that they may pose a threat to the Great Lakes. An Annex 3 binational strategy for PFAS is under development.	Anticipated September 2019
Belmont and Rockford, Michigan	The EPA is coordinating with the State of Michigan by overseeing a federal CERCLA time-critical removal action focused on hazardous substances at the Wolverine World Wide (Wolverine) Tannery and House Street Disposal Site and providing technical assistance to MDEQ while it responds to PFAS contamination of residential wells from Wolverine's former Tannery, shoe factory, and disposal locations in the Rockford area.	Ongoing

Tool/Activity	Purpose	Timeframe
Regions 3 and 5: Amendment to 2009 Safe Drinking Water Act Section 1431 Emergency Order on Consent with DuPont and Chemours	In 2009, the EPA issued a 1431 order on consent to Chemours' Washington Works Facility that contaminated sources of drinking water in WV and OH primarily via air deposition from the Facility. That order was amended in 2017, incorporating the Lifetime Health Advisory and requiring DuPont and Chemours to offer treatment, connection to a PWS, or bottled water to people on public or private water systems with PFOA levels above 70 ppt. In 2018, at the EPA's request, Chemours has also voluntarily sampled numerous private and PWSs for GenX chemicals.	Ongoing
Region 4 coordination of assistance to North Carolina Department of Environmental Quality (NCDEQ) – Chemours Fayetteville Works Facility	Region 4 has provided ongoing support to the NCDEQ as it has responded to GenX chemicals in the Cape Fear River and Fayetteville area. • Analytical testing via ORD-RTP and Region 4 Science and Ecosystem Support Division labs (testing of raw & finished water in the Cape Fear, rainwater, and air emissions stack testing for GenX chemicals and 22 other PFAS compounds) • Technical input as the state established its interim health goal • Coordinated treatment technique assistance for water systems • Technical assistance with NPDES permitting related matters and air emissions control.	Ongoing Started June 2017
Grant Funding Opportunity: National Priorities: Per- and polyfluoroalkyl substances	The EPA solicited proposals for EPA-G2018-ORD-A1 that included the below desired research areas: • Short-chain PFAS (C4 to C7) • PFAS found as residuals from manufacturing processes • Alternatives for long-chain PFAS (≥ C8) such as per- and poly-fluoroethers • PFAS generated through environmental chemical transformation	Ongoing Completed June 2018
Technical Support	The EPA will continue to assist states and tribes in bringing on PFAS analytical capabilities.	Ongoing

Tool/Activity	Purpose	Timeframe
Risk Communication and Engagement: What is the EPA doing to provide consistent and accurate information and guidance to the public?		
Clearinghouse of PFAS information for states, tribes and local communities	The EPA compiled information from a wide range of sources on measurement, health impacts, and treatment and remediation technologies. The EPA continues to update this site as additional information becomes available.	Ongoing Started 2018
Engagement with states and stakeholders	Ongoing robust engagement effort with states, tribes, local communities, utilities, industry, and the public. Extensive outreach in 2018 included: • 5/22-5/23/2018: PFAS National Leadership Summit • 6/25-26/2018: Exeter, NH (Region 1 wide) Community Engagement • 7/25/2018: Horsham, PA Community Engagement • 8/7-8/2018: Colorado Springs, CO Community Engagement • 8/14/2018: Fayetteville, NC Community Engagement • 8/13/2018: Spokane WA, PFAS session at the Tribal Lands and Environment Forum meeting • 9/5/2018: Leavenworth, KS Community Engagement • 10/4-5/2018: Michigan site visits, Kalamazoo, MI Roundtable	Completed October 2018
EPA Region 7 participation in Kansas PFAS Monitoring Plan Advisory Committee	Region 7 to serve on Kansas Department of Health and Environment Per- and Polyfluoroalkyl Substance Monitoring Plan Advisory Workgroup for drinking water. The KDHE requested the EPA's participation to serve in an advisory capacity on a monitoring plan to be developed with the focus on drinking water.	Started Fall 2018
EPA Region 7 updates on PFAS for states and tribes	Activated the EPA Region 7 Science Council with state representation which will also include a PFAS update on a quarterly basis. The EPA Region 7's Regional POC for PFAS will also update our tribal representatives at the Regional Tribal Operation Committee meetings.	Started March 2018
Federal Remediation Technologies Roundtable Meeting	One-day interagency technical meeting meant to identify and discuss the emerging science behind PFAS characterization and remedial technologies. Technical presentations also remotely broadcasted. Primarily federal agency participation.	Completed November 7, 2018
Internal EPA regional coordination network	Activated internal EPA regional coordination network with representation from all regions and program offices to further support rapid dissemination of information in order to better support states, tribes, and local communities.	Started February 2018
Internal EPA regional coordination for cleanup programs	Created an internal EPA regional coordination group for cleanup programs with representation from all regions to further support rapid dissemination of information in order to better support states, tribes, and local communities.	Started Summer 2016

Tool/Activity	Purpose	Timeframe
Internal EPA Region 7 team	Activated internal EPA Region 7 network with representation from all programs further support rapid dissemination of information in order to better support states, tribes, and local communities.	Started February 2018
Quarterly Meetings with Region 10 Environmental and Health Departments	Region 10 quarterly conference calls with Region 10 PFAS contacts in state environmental and health departments to share information and discuss issues and topics of mutual interest.	Ongoing
Webinar on PFAS State case studies	Webinar showcasing PFAS risk communication activities by states; developed in coordination with ECOS and ASTHO.	Completed June 2018

Appendix B: Summary of PFAS National Leadership Summit and Community Engagements

In 2018, the EPA held a series of public community engagement events that brought together the EPA and state officials, federal partners, local speakers, community groups, and citizens to share perspectives and help inform future Agency actions for managing PFAS. Following the PFAS National Leadership Summit, these sessions continued EPA's commitment to foster an ongoing dialogue with stakeholders to address PFAS.

The National Leadership Summit included representatives from over 40 states, tribes, and territories; 13 federal agencies; congressional staff; associations; industry groups; and non-governmental organizations to engage in discussions about PFAS monitoring, risk characterization, near-term actions, and risk communications strategies. Key perspectives emphasized by participants during the summit included interest in:

1. An expansion of monitoring and sampling in the environment supported by sources of funding;
2. Continued advancement of the understanding of PFAS compounds, potential toxicity, and further development of analytical methods;
3. Increased understanding of exposures beyond drinking water;
4. Robust near-term action while long term actions are completed;
5. Identifying opportunities for collaboration and coordinated data sharing efforts among partners; and
6. Continued public engagement and development of risk communication resources.

The Community Engagements included panel discussions on the current state of science and potential risks posed by PFAS, as well as state and local actions towards 1) Identifying PFAS; 2) PFAS Risk Communications; and 3) Identifying Solutions for PFAS. Following the panel discussions, members of the public shared input and personal stories. During the community listening sessions, the EPA interacted with over 1,000 members of the public and heard from approximately 200 citizens in Exeter, New Hampshire; Horsham, Pennsylvania; Colorado Springs, Colorado; Fayetteville, North Carolina; and Leavenworth, Kansas.

The EPA developed summaries for the PFAS National Leadership Summit and each of the community engagements that can be found on EPA's PFAS webpages: https://www.epa.gov/pfas/pfas-national-leadership-summit-and-engagement and https://www.epa.gov/pfas/pfas-community-engagement.

Appendix C: Summary of Docket Comments

Background

Following the PFAS National Leadership Summit, the EPA requested input from the public on how the Agency can best help states, tribes, and communities facing PFAS challenges. The EPA has considered these comments in the development of this PFAS Action Plan and will continue to be informed by these comments as the Agency plans its next steps.

Docket Process and Summary of Submissions

The EPA opened the docket on PFAS, OW-2018-0270, from May 2, 2018 to September 28, 2018 and received approximately 120,000 comments via Regulations.gov. The docket comments are summarized below according to the themes requested by the EPA. The docket is available at: https://www.regulations.gov/docket?D=EPA-HQ-OW-2018-0270.

1. Obtaining information on ongoing efforts to characterize risks from PFAS and develop monitoring and treatment/cleanup techniques;
2. Informing specific near-term actions, beyond those already underway, that are needed to address challenges currently facing states and local communities;
3. Developing risk communication strategies to address public concerns with PFAS; and
4. General comments.

All comments were reviewed, categorized, and used to support the development of the PFAS Action Plan. The majority of comments received, approximately 97%, were from the public from across the United States representing rural and urban communities. Public citizen comments generally included a request for the EPA and the federal government to assist in managing PFAS in their community, concern for the health of their families and themselves, specific requests for action in managing and limiting PFAS in the environment, a desire to see PFAS removed at the source, a desire for responsible parties to pay for cleanup, and a universal expression for the right to have access to clean and healthy water.

Approximately 2.5% of comments were submitted by organizations, members of Congress, industry, water associations, governmental organizations at all levels, and not-for-profit organizations. The comments generally included support for the development of the PFAS Action Plan, an expression of the need for regulatory action, the need for science-based decisions, a desire for better communication regarding the Agency's planned activities, a request for the EPA to use regulatory authorities to manage PFAS, and a coordinated response from the federal government.

The following information is intended to provide an overview summary of the comments received in the public docket within each theme and is not meant to be comprehensive. Comments provided to the EPA are available in the docket at the link provided above.

Characterize Risks from PFAS and Develop Monitoring and Treatment/Cleanup Techniques

- Undue burden placed on communities and private well owners. Concerns on the costs to the taxpayer associated with treatment of PFAS in water, purchasing bottled water, point-of-use filters, and/or the cost associated with health care stemming from potential PFAS exposure.
- Desire for responsible parties to pay for the cost of cleanup/treatment and monitoring.
- Requests that the EPA consider the cost of treatment in the rulemaking process.
- Federal prioritization of PFAS compounds for additional study and effort.
- Concern on the movement of PFAS through groundwater and the potential for contamination to spread.
- Need for more science-based research and method development to monitor PFAS.

Near-term Actions Needed to Address Challenges Currently Facing States and Local Communities

- Desire for the EPA to use its regulatory authority to regulate PFAS and provide regulatory certainty.
- List PFAS as hazardous substances.
- Develop groundwater cleanup values in a way that encourages site-specific solutions and allow for use of available resources.
- Request for better risk communication and education from the public on health effects, more research on PFAS, identification of PFAS in media other than drinking water, and prevention of industrial releases of PFAS.
- Develop consistent and enforceable standards, including a maximum contaminant level for PFAS that is based on best-available and current science. Some members of the public expressed support for lowering EPA's Health Advisory Level.
- Follow up or expanded water testing and/or blood testing in local communities.
- Concern with the UCMR detection levels (too low and not representative of PFAS presence) and requests to expand the list of PFAS for future UCMR efforts.
- Need for funding for the federal, state, tribal, and local governments to adequately address PFAS.
- Regulate PFAS at the source; prevent PFAS from entering commerce and prevent releases into the environment.
- Concern that families and communities located near military installations are disproportionally affected by PFAS.
- Concern from site-specific contamination, including GenX chemicals.
- Make available technical assistance and funding to individual households and private well owners to address PFAS. Communities need assistance in determining the extent of their contamination.
- Need for new analytical methods to achieve lower detection limits, identify additional PFAS, and monitor in media other than water.

- Need any guidance developed by the EPA to be scalable, with special emphasis for small and tribal communities.

Risk Communication Strategies to Address Public Concern with PFAS

- Concern regarding the quality and accessibility of information from the EPA and other federal agencies. Desire to have information on: the proximity of a community to a PFAS source; the potential exposure of communities to individual and mixtures of PFAS; products that contain PFAS; guideline, standard, and method development process; and access to technical resources such as data, methods, and research.
- Need for a clear and concise communication plan from the EPA to inform the public and stakeholders regarding the risk of PFAS exposure and related the EPA activities (both ongoing and planned).
- Concern on the unknown human health effects from PFAS exposure, the cost of health insurance and mental health coverage from exposure and stress of exposure, and the possible health effects from PFAS exposures.
- Request for comprehensive testing of PFAS in drinking water and blood and communication of risk information in a clear and concise manner that is easy for the public to understand.
- Concern on the lack of risk communication for PFAS in food, such as fish and shellfish.
- Need for a comprehensive risk communication strategy that includes stakeholders and allows for the opportunity for the public to provide comments and questions.

General Comments

- Request the EPA exercise its regulatory authority to limit the use and manufacture of PFAS due to health concerns from exposure from air, water, and food.
- Commenters at community engagements provided both support and appreciation for the opportunity to participate, in addition to implying frustration at feeling excluded from presenting information to the panelists.
- Commenters provided personal accounts of PFAS exposure in their local community and the health and financial impacts of that exposure.
- Encourage the EPA to abide by its mission to protect human health and the environment by ensuring all citizens are provided healthy and clean drinking water and air.

Appendix D: Other Reference Materials

EPA Resources

- EPA's Webpage for Per- and Polyfluoroalkyl Substances (PFAS): https://www.epa.gov/pfas
- Information on the EPA Community Engagement Sessions on PFAS: https://www.epa.gov/pfas/pfas-community-engagement
- Information on the National Leadership Summit on Per- and Polyfluoroalkyl Substances (PFAS): https://www.epa.gov/pfas/pfas-national-leadership-summit-and-engagement
- PFAS National Leadership Summit and Engagement Federal Public Input Docket: https://www.regulations.gov/ – enter docket number: OW-2018-0270
- Drinking Water Health Advisories for PFOA and PFOS: https://www.epa.gov/ground-water-and-drinking-water/drinking-water-health-advisories-pfoa-and-pfos
- Third Unregulated Contaminant Monitoring Rule: https://www.epa.gov/dwucmr/third-unregulated-contaminant-monitoring-rule
- Contaminant Candidate List 4: https://www.epa.gov/ccl/contaminant-candidate-list-4-ccl-4-0
- EPA Drinking Water Laboratory Method 537 Q&A: https://www.epa.gov/pfas/epa-drinking-water-laboratory-method-537-qa
- Research on Per- and Polyfluoroalkyl Substances (PFAS): https://www.epa.gov/chemical-research/research-and-polyfluoroalkyl-substances-pfas
- EPA Actions to Address PFAS: https://www.epa.gov/pfas/epa-actions-address-pfas
- EPA 2010/2015 PFOA Stewardship Program: https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/fact-sheet-20102015-pfoa-stewardship-program
- Drinking Water Treatability Database: https://iaspub.epa.gov/tdb/pages/general/home.do
- Case Studies on State-Level Risk Communication of PFAS (EPA and ECOS collaboration): https://www.ecos.org/documents/state-level-risk-communication-of-pfas-and-habs/

Additional Resources (Non-EPA Materials)

- ATSDR Webpage Per- and Polyfluoroalkyl Substances (PFAS) and Your Health: https://www.atsdr.cdc.gov/pfas/
- ATSDR Overview of Perfluoroalkyl and Polyfluoroalkyl Substances and Interim Guidance for Clinicians Responding to Patient Exposure Concerns: https://www.atsdr.cdc.gov/pfc/docs/pfas_clinician_fact_sheet_508.pdf
- ToxFAQs™ for Perfluoroalkyls: https://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=1116&tid=237
- Toxicological Profile for Perfluoroalkyls: https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237
- CDC Per- and Polyfluorinated Substances (PFAS) Factsheet: https://www.cdc.gov/biomonitoring/PFAS_FactSheet.html

- CDC National Report on Human Exposure to Environmental Chemicals: https://www.cdc.gov/exposurereport/index.html
- Interstate Technology Regulatory Council (ITRC) PFAS website: https://pfas-1.itrcweb.org/
- ITRC PFAS fact sheets: https://pfas-1.itrcweb.org/fact-sheets
- Per- and Polyfluoroalkyl Substances (PFAS) Laboratory Testing Primer for State Drinking Water Programs and Public Water Systems: https://www.asdwa.org/wp-content/uploads/2018/10/ASDWA-PFAS-Lab-Testing-Primer-10-10-18-Final.pdf

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

FEB 15 2019

The Honorable Thomas R. Carper
United States Senate
Washington, D.C. 20510

OFFICE OF WATER

Dear Senator Carper:

Thank you for your letter of February 1, 2019, regarding Per- and Polyfluroalkyl Substances (PFAS). The U.S. Environmental Protection Agency (EPA) shares your concern for communities across the United States that continue to deal with these substances. The PFAS issue is a priority for the EPA and we are working cooperatively with our federal and state partners to address PFAS-related issues in order to protect human health and the environment.

On February 14, 2019, the EPA announced the first-ever PFAS Action Plan, available at: https://epa.gov/pfas. This historic plan responds to extensive public interest and input the EPA has received, including at the agency's May 2018 National Leadership Summit and subsequent visits to a number of states across the nation, at which the agency heard directly from the public about PFAS issues in their communities. The Action Plan represents the first time the EPA has built a national, multi-media, multi-program, research, management, and risk communication plan to address an emerging chemical of concern like PFAS. The Action Plan identifies both short-term solutions for addressing PFAS chemicals and long-term strategies that will help provide the tools and technologies states, tribes, and local communities need to clean up sites and provide clean and safe drinking water to their residents. Major actions described in the Action Plan are highlighted below.

Drinking Water: The EPA intends to establish a maximum contaminant level (MCL) for PFOA and PFOS—two of the most well-known and prevalent PFAS chemicals. To do so, the EPA is committed to following the MCL rulemaking process as established by the Safe Drinking Water Act (SDWA)—a process that is designed to ensure public participation, transparency, and the use of the best available science and other technical information. By the end of this year, the EPA will propose a regulatory determination, which is the next step in the Safe Drinking Water Act process for establishing an MCL. The EPA is also gathering and evaluating information to determine if a SDWA regulation is appropriate for a broader class of PFAS.

Cleanup: The EPA has already begun the regulatory development process for listing PFOA and PFOS as hazardous substances and will issue interim groundwater cleanup recommendations for sites contaminated with PFOA and PFOS. This important work will provide additional tools to help states and communities address existing contamination and enhance the ability to hold responsible parties accountable.

Enforcement: The EPA will continue its ongoing enforcement actions, create tools to address PFAS exposure in the environment, and assist states in enforcement activities. Where the EPA finds that there may be an imminent and substantial endangerment to public health related to PFAS contamination, the

agency will consider using its response authority under CERCLA section 104 or utilizing its enforcement authorities such as the SDWA section 1431 or RCRA section 7003.

Monitoring: The EPA will propose to include PFAS in the next round of nationwide drinking water monitoring under the Unregulated Contaminant Monitoring Program. This will improve the EPA's understanding of the frequency and concentration of PFAS occurrence in drinking water. This additional monitoring will utilize newer methods that will detect more PFAS chemicals and at lower levels. The EPA will also consider PFAS chemicals for listing in the Toxics Release Inventory to help the agency identify where these chemicals are being released.

Research: Through additional research, the EPA will rapidly expand the scientific foundation for understanding and managing risk from PFAS. The EPA will develop new analytical methods so that more PFAS chemicals can be detected in drinking water, in soil, and in groundwater. These efforts will improve our ability to monitor and assess potential risks. The EPA's research efforts also include developing new technologies and treatment options to remove PFAS from drinking water and at contaminated sites.

Risk Communications: The EPA will work across the agency—and the federal government—to develop a PFAS risk communication toolbox that includes materials that states, tribes, and local partners can use to effectively communicate with the public.

The PFAS Action Plan will help the EPA and its partners identify and better understand PFAS contaminants generally, clean up current PFAS contamination, prevent future contamination, and effectively communicate risk with the public. To implement the Action Plan, the EPA will continue to work in close coordination with multiple entities, including other federal agencies, states, tribes, local governments, water utilities, the regulated community, and the public.

Again, thank you for your letter and for your focused interest on PFAS. The EPA looks forward to working with you to address this challenge. If you have further questions, please contact me or your staff may contact Matt Klasen in the EPA's Office of Congressional and Intergovernmental Relations at klasen.matthew@epa.gov or (202) 566-0780.

Sincerely,

David P. Ross
Assistant Administrator