ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 723

[EPA–HQ–OPPT–2002–0051; FRL–7735–5]

RIN 2070–AD58

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

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

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

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

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

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

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

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

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

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

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

II. Background

A. What Action is the Agency Taking?

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

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

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

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

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

C. Why is the Agency Taking This Action?

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

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

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

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

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

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

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

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

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

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

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

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

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

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

III. Summary of This Proposed Rule

A. Polymers Containing PFAS or PFAC

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

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

B. Polymers Containing Fluorotelomers or Other Perfluoroalkyl Moieties

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

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

C. Proposed Implementation

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

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

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

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

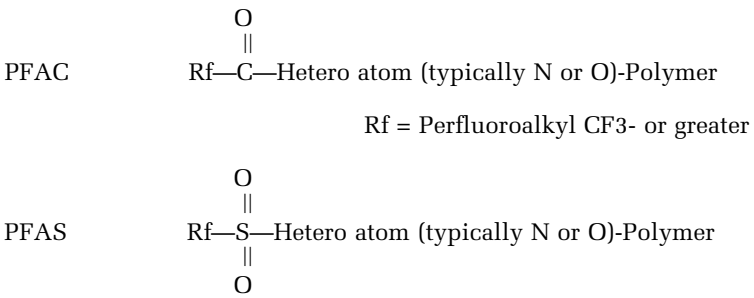
IV. Proposed Rule

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

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

B. Defining Polymers That Are Subject to This Proposed Rule

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



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

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

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

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

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

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

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

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

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

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

D. Summary of Data on PFAS and PFAC

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

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

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

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

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

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

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

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

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

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

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

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

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

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

For PFOS, these pathways may have included:

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

E. Summary of Data on Fluorotelomers and Other Perfluoroalkyl Moieties

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

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

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

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

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

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

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

V. Objectives and Rationale for This Proposed Rule

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

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

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

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

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

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

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

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

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

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

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

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

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

VI. Other Options Considered

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

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

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

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

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

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

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

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

D. Exclude From Exemption All Fluorine-containing Polymers

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

VII. Request for Comment on Specific Issues

EPA is requesting specific responses to the following:

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

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

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

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

VIII. Economic Considerations

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

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

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

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

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

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

annual cost of $316,500 in subsequent years.

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

IX. References

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

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8. (AR 226–1440) Hagen DF, Belisle J, Johnson JD, Venkateswarlu P., ''Characterization of fluorinated metabolites by a gas chromatographic-helium microwave plasma detector--the biotransformation of 1H, 1H, 2H, 2H-perfluorodecanol perfluorooctanoate.'' Analytical Biochemistry 118(2):336–343, 1981.

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18a. (AR 226–1094) The Society of the Plastics Industry, Inc., presentation to the EPA, Sanitized Copy. April 26, 2002.

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X. Statutory and Executive Order Reviews

A. Regulatory Planning and Review

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

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

B. Paperwork Reduction Act

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

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

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

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

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

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

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

C. Regulatory Flexibility Act

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

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

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

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

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

D. Unfunded Mandates Reform Act

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

E. Federalism

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

F. Consultation and Coordination With Indian Tribal Governments

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

G. Protection of Children From Environmental Health and Safety Risks

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

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

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

I. National Technology Transfer Advancement Act

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

J. Environmental Justice

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

List of Subjects in 40 CFR Part 723

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

Dated: February 8, 2006.

Susan B. Hazen,

Acting Assistant Administrator for Prevention, Pesticides and Toxics Substances.

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

PART 723—[AMENDED]

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

Authority: 15 U.S.C. 2604.

2. Section 723.250 is amended as follows:

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

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

§ 723.250 Polymers.

* * * * *

(b) * * *

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

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

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

* * * * *

(d) * * *

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

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

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

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