

carbon-hydrogen bonds on molecules of the organic feedstock with carbon-fluorine bonds. Perfluorination occurs when all the carbon-hydrogen bonds are replaced with carbon-fluorine ones. The ECF process yields between 30–45% straight chain (normal) perfluorooctanonyl fluoride (PFOF), along with a variable mixture of byproducts and impurities. The output of the ECF process consists of a complex combination of chemical substances with varying molecular weights, including higher and lower straight-chain homologues; branched-chain perfluoroalkyl fluorides of various chain lengths; straight-chain, branched, and cyclic perfluoroalkanes and ethers; and other byproducts. After disposal or recovery of some of the byproducts and impurities, the acid fluoride is base hydrolyzed in batch reactors to yield PFOA. The PFOA salts are synthesized by base neutralization of the acid to the salt in a separate reactor.

In the telomerization process, tetrafluoroethylene is reacted with other fluorine-bearing chemicals to yield fluorinated intermediates which are readily converted into PFOA. This process yields predominantly straight-chain acids with an even number of carbon atoms. Distillation can be used to obtain pure components. Commercial products manufactured through the telomerization process, sometimes known as telomers, are generally mixtures of perfluorinated compounds with even carbon numbers, although the process can also produce compounds with odd carbon numbers.

In addition to releases from the deliberate manufacture of PFOA through either the ECF or telomerization processes, and from the use of PFOA and its salts in the manufacture and processing of fluoropolymers and fluoroelastomers, PFOA may have entered the environment through other sources. 3M has indicated that PFOA may have been present as a trace contaminant in some of the fluorochemical products which it discontinued manufacturing between 2000 and 2002 (Ref. 7). Because these products are no longer being manufactured, they will likely not be a significant potential future source of PFOA.

EPA has also received data which indicate that the 8–2 telomer alcohol (1-Decanol, 3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heptadecafluoro- (CAS No. 678–39–7)) although not itself made with PFOA, can be metabolized by living organisms or biodegrade under environmental conditions to produce PFOA (Refs. 8 and 9). Other telomer chemicals have

not been tested to determine whether they may also metabolize or degrade to form PFOA. Telomers are used widely in a range of commercial products, including some that are directly released into the environment, such as fire fighting foams, as well as soil, stain, and grease resistant coatings on carpets, textiles, paper, and leather. The extent to which these telomer-containing products might degrade to release PFOA is unknown. However, anecdotal evidence of the atmospheric presence of telomer alcohols in a multi-city North American survey suggests that telomers may be one source of environmental PFOA (Ref. 10). Additional fate information is necessary to determine whether and the extent to which telomer product degradation may be a source of PFOA.

EPA is not currently aware of any other potential sources of PFOA in the environment. EPA specifically requests comment on this issue, and the submission of any data identifying or characterizing PFOA sources. EPA is especially interested in the thermal stability and oxidative degradation products of materials containing PFOA or telomer chemicals which are incinerated.

B. Hazard and Exposure

EPA has conducted a detailed review of all available hazard and exposure information on PFOA. This review is available in the Agency's *Revised Draft Hazard Assessment on PFOA and Its Salts* (Ref. 11). This draft hazard assessment has not been formally peer reviewed, but has been reviewed internally by the EPA Office of Research and Development (ORD).

PFOA is persistent in the environment. It does not hydrolyze, photolyze, or biodegrade under environmental conditions. Based on recent human biomonitoring data provided by industry, which found PFOA in the blood of workers and the general population in all geographic regions of the United States, exposure to PFOA is potentially nationwide, although the routes of exposure for the general population are unknown.

Several epidemiological studies on the effects of PFOA in humans have been conducted on workers. An 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 the levels of the hormone estradiol in workers with high serum PFOA levels (>30 parts per million (ppm)) was also reported, but none of

the other hormone levels analyzed indicated any adverse effects.

APFO is the most widely used salt of PFOA, and most animal toxicity studies have been conducted with APFO. An extensive array of animal toxicity studies have been conducted in rodents and monkeys. These studies have shown that APFO exposure can result in a variety of toxic effects in animals including liver toxicity, developmental toxicity, and immunotoxicity. In addition, rodent bioassays have shown that chronic APFO exposure is associated with a variety of tumor types. The mechanisms of APFO tumorigenesis are not clearly understood. At this time, EPA is evaluating the scientific evidence and has not reached any conclusions on the potential significance to humans of the rodent cancer data.

There are marked gender differences in the elimination of PFOA in rats. In addition, there are substantial differences in the half-life of PFOA in rats, monkeys, and humans. The gender and species differences are not completely understood and therefore the extent of potential risks to humans is uncertain.

C. Preliminary Risk Assessment

Because TSCA section 4(f) is focused narrowly on the specific toxicity endpoints of cancer, birth defects, and gene mutation, the preliminary risk assessment prepared as part of this priority review focused on the potential risks for developmental toxicity in humans. EPA did not include cancer risk in this preliminary assessment due to questions concerning the potential significance to humans of the rodent cancer data. Because data indicate that PFOA is not mutagenic, concern for gene mutation was not an issue for this preliminary assessment.

The preliminary risk assessment used a margin of exposure (MOE) approach (Ref. 1). For many risk assessments, the MOE is calculated as the ratio of the administered dose from the animal toxicology study to the estimated human exposure level. The human exposure is estimated from a variety of potential exposure scenarios, each of which requires a variety of assumptions.

A more accurate estimate of the MOE can be derived if measures of internal dose are available for humans and the animal model. In this preliminary risk assessment, serum levels of PFOA, which are a measure of internal dose, were available for some administered dose levels in the rat 2-generation reproductive toxicology study and from human biomonitoring studies. Thus, internal dose was used for the