TEFLON AND HUMAN HEALTH: 
DO THE CHARGES STICK? 

Assessing the Safety of 
the Chemical PFOA 

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I. Introduction and summary

Recently, the public has become concerned about the potential human health effects of PFOA (perfluorooctanoic acid or perfluorooctanoate), a chemical used to produce substances needed to manufacture Teflon and many other products. The Environmental Protection Agency (EPA) has been reviewing the scientific data on PFOA and at press time for this publication was working on its final report about the human health effects associated with PFOA (the draft assessment was released in January 2005). In 2004, concerns about PFOA were widely reported in the media because the EPA claimed that DuPont, the manufacturer of Teflon, had not adequately reported information about PFOA’s presence in water supplies and its ability to cross the placenta from mother to fetus. It is important to note that even while some media reports may have caused misunderstanding by calling PFOA a “Teflon chemical,” PFOA is not present in the final product of Teflon-coated cookware; it is only used in the manufacturing process of the product.

While concerns about PFOA’s effects on humans have arisen recently, data on PFOA’s presence in humans and its effects on both animals and humans has been collected for more than 20 years and can be used to evaluate the potential for harm from PFOA. Research has shown that very high doses of PFOA can cause harm in animals, but the amount of PFOA to which the general population is exposed is hundreds to thousands of times lower, and biological differences may make concerns about some of the observed effects irrelevant to humans. Additionally, studies of workers (who are exposed to much higher doses of PFOA than the general population) have not shown the same effects in humans that occur in animals.
II. What is PFOA?

PFOA* is mainly used to produce other chemicals, such as APFO (ammonium perfluorooctanoic acid - often referred to as C-8). (1) These chemicals are then used in the production of products such as Teflon coating on cookware. While the use of PFOA to make Teflon is most widely known, most of these chemical products of PFOA are used in other industries such as automotive, electronics, and defense. For example, they are used to produce insulation for wires, power steering and brake assemblies in cars, and gear lubrication.

III. How are humans exposed to PFOA?

While the presence of PFOA in the environment is at least partially due to the production, use, and disposal of PFOA itself, it may also result from a breakdown of other substances that are used in consumer products. (2) It is unclear, however, how much each source contributes to the chemical's presence in the environment. PFOA has been found in air, water, living organisms, and landfills, but there is only limited data on its levels. Mostly, PFOA levels have been measured in areas (particularly in water) near production plants that use PFOA. (1)

When PFOA is absorbed through inhalation or ingestion, some of it binds to proteins in the blood, and it can also accumulate in organs such as the liver and kidneys. (9) The length of time that PFOA remains in the body has been assessed only in one study, and that study included only nine workers. (10)

IV. How much PFOA are people exposed to?

A. Amount based on human blood levels of PFOA

A number of studies have measured the levels of PFOA in the blood of people around the world.

* In this document, references to PFOA also imply C-8 and APFO.
both in the general population and in workers who are much more highly exposed to PFOA. (3-7)
Because these studies have analyzed the blood of large numbers of people in widespread locales, we can be fairly confident that the studies represent the PFOA blood concentrations in the entire population. On average, the general population has about 5 parts per billion (ppb) of PFOA in their blood, with high values at about 20-30 ppb. (3-6) These concentrations do not seem to vary by age. (4, 5) Some workers in factories that use or manufacture PFOA have levels much higher than those found in the general population, averaging about 1-10 parts per million (ppm) (equivalent to 1,000 to 10,000 ppb). (7)

B. Amount based on water levels of PFOA

Drinking water levels of PFOA probably represent the largest source of human exposure to PFOA (1, 8) and can be used to estimate the amount of PFOA that people ingest (see section VI). Studies have measured PFOA levels in areas near plants that use or manufacture PFOA and sites contaminated with PFOA but have not reported country-wide, comprehensive levels of PFOA in air, soil, or water. (1, 8) While these studies cannot give us an idea of how much the average person is exposed to PFOA, they can be used to estimate the exposure to PFOA of people in the population expected to be most highly exposed. In areas surrounding plants in West Virginia and in Alabama, the levels of PFOA in drinking water have averaged about one part per billion (ppb), ranging up to about 10 parts per billion. (1,8) In a six-city survey, drinking water levels of PFOA were much lower, ranging from non-detectable to 0.029 ppb. (1)

V. What do we know about the health effects of PFOA?

A. From studies of animals

Various studies have shown that very high doses of PFOA have harmful effects on animals. In stud-
ies in which animals such as rats, rabbits, and monkeys ingested high doses of PFOA, the animals experienced adverse effects including liver changes, weight loss, and gastrointestinal irritation. (9) When exposed to high levels of PFOA in the air, these animals showed symptoms such as irregular breathing, changes in liver weight, weight loss, and eye corrosion. (9) High-dose studies of APFO have shown reproductive and developmental effects on rats; the offspring of these rats experienced increased mortality and weight loss. (11) One study found that rats exposed to high doses of PFOA had increased rates of liver, testicular, and pancreatic cancers. (9)

In applying these studies to predict risk for humans, it is important to note both that the animals were very highly exposed to PFOA (see section VI) and that a variety of studies suggest that the mechanisms by which PFOA causes cancer and other health effects in rats may not even be applicable to humans. (9) However, the exact biological mechanisms related to some of the health effects found in animals are not completely understood. (9)

B. From studies of humans

Some studies have been performed on worker populations in the U.S. and Europe who have been exposed to much higher doses of PFOA than the general population. These studies aimed at detecting in humans a variety of adverse effects that had been seen in laboratory animals but did not find these effects in workers. (12,13) A study of almost 4,000 workers did not find a relationship between PF OA exposure and all-cause mortality or cancer mortality. (9) Contrary to the results of an earlier study of workers (14), there was also no association between PF OA exposure (measured by length of employment) and prostate cancer. (9)

An unpublished report claimed that people who drank water contaminated with PFOA had higher
rates of various cancers. This report was made to support a lawsuit and was not reviewed by peers in the scientific community for its methodology and the accuracy of its claims (as studies published in peer-reviewed journals are). (15) Aside from this claim, the current data do not support a connection between PFOA and cancer, even in workers (who are most highly exposed to PFOA).

VI. Is the amount of PFOA that people are exposed to a cause for concern?

The data on effects of PFOA in humans do not give us a way to directly evaluate the potential human health risks of PFOA. However, regulatory agencies try to estimate the risk by either (A) comparing blood PFOA levels of animals experiencing adverse effects to the blood levels found in humans or (B) comparing the doses of PFOA associated with adverse effects in animals to the doses to which humans are exposed. These methods, however, are based on the assumption that the same biological mechanisms are present in humans, and that may not be true.

A. Risk analysis based on blood PFOA levels

Blood levels are not routinely measured in high-dose toxicology studies of animals, but one reproductive study recorded them for some animals. (16) The Environmental Protection Agency's preliminary risk assessment (17) was based on levels from that study, which indicated that the lowest parental PFOA blood levels associated with adverse effects in offspring were 0.37 parts per million for female rats and 51.1 parts per million in male rats. These levels are 100 to 10,000 times greater than the 5 parts per billion average level of PFOA found in blood of the general human population (in other words, there is a 100- to 10,000-fold margin of safety). (17)

Another risk assessment that used estimated blood PFOA levels based on ingested dose of PFOA concluded that the levels of PFOA in
humans were about 1,000 to 10,000* times lower than the levels that began to cause adverse effects in animals. (19)

B. Risk analysis based on administered doses of PFOA

One study indicated that the greatest amount of administered PFOA that does not cause adverse effects is 10 mg/kg/day in female rats and 3 mg/kg/day in male rats. (16) However, others suggest that this level is lower, because doses of 0.5 to 1 mg/kg/day (500 to 1000 micrograms/kg/day) of PFOA were associated with liver effects on laboratory animals. (18) As discussed earlier, the highest level of PFOA that has been measured in water is approximately 10 ppb (10 micrograms/liter). Therefore, in order to reach even the lower estimate of the amount of PFOA suspected to cause adverse effects (500 micrograms/kg/day), the average person (of 70 kg) would need to drink more than 3,500 liters of this most highly contaminated water daily.** Using water intake and weight guidelines for children, a similar calculation would also indicate a margin of safety that is somewhat smaller but still very large.

VII. Conclusions

While research has shown adverse effects of high doses of PFOA in animals, the existing studies of workers who are highly exposed do not indicate health effects of PFOA. Risk analysis based upon blood levels of PFOA and ingested amounts of PFOA by the general population show that laboratory animals experiencing adverse effects from PFOA are exposed to amounts that are hundreds to thousands of times higher than those to which the general human population is exposed.

There is doubt, however, as to whether at least some of the effects observed in animals are rele-

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* The variation depends on which effect is being examined.
**(70 kg X 500 micrograms/kg/day)/(10 micrograms/liter) = 3,500 liters/day.
vant to humans at all, since some biological mechanisms that produce these effects are not present in humans. Additionally, workers with blood levels of PFOA equal to or higher than those that have been found to cause adverse effects in animals have themselves not shown adverse effects. This suggests that the margins of safety for the general population may be even higher than the risk analyses predict.

While further research is needed in order to more fully understand how PFOA acts in the body, the current data indicate that we can expect no risk to human health associated with the levels of PFOA exposure found in the general population.

References


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