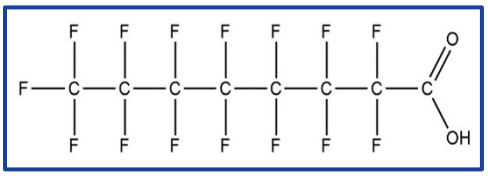
SUMMARY

Background: Perfluorooctanoic acid (PFOA) was widely used in the manufacturing of a variety of consumer products, including clothing and cookware. Due to concerns of potential toxicity, it was phased out of U.S. manufacturing production, which resulted in declining exposure. However, concentrations of PFOA can persist after exposure due to a slow elimination rate in humans and continued environmental exposure to PFOA, such as through drinking water. The current studies sought to identify potential toxicity or cancer-related outcomes during perinatal (effects in the womb and during nursing) and 2-year postnatal exposure to PFOA in male and female rat offspring. Because oral exposure to PFOA is common in humans, feed was selected as the route of exposure for these NTP studies.



Methods: Pregnant rats were fed diets containing 150 or 300 parts per million (ppm) PFOA during the perinatal period. Groups of 50 offspring male rats were fed diets containing 150 or 300 ppm PFOA, and groups of 50 offspring female rats were fed diets containing 300 or 1,000 ppm PFOA during the 2-year postweaning period. The higher feed exposure concentration was provided to female rats postweaning because PFOA is eliminated at a faster rate in females than in males. Control rats received feed without PFOA exposure. Comparisons were made between groups exposed during the perinatal and postweaning periods with groups exposed during the postweaning period only. At the end of the study, tissues from more than 40 sites from every animal were examined for signs of disease.

Results: Survival was unaffected by exposure to PFOA. However, exposure-related decreases in body weights were observed in all groups of male and female rats exposed to PFOA, compared to the control groups. Male rats had increased numbers of cancerous lesions in the liver and pancreas. Cancer-related responses to PFOA in female rats were generally less than in male rats. Cancerous lesions were marginally increased in the liver and uterus in female rats. Cancerous pancreatic lesions, which are uncommon in female rats, were observed. Very few significant cancer-related differences were observed between the groups of animals exposed to PFOA postweaning-only compared to groups with both perinatal and postweaning exposures, and most of these differences were not considered related to the timing of PFOA exposure. Noncancerous tissue abnormalities were observed in the liver and pancreas of male rats and in the liver, kidney, forestomach, and thyroid gland of female rats.

Conclusions: *The NTP four-point scale rates the level of evidence that a substance has the ability to cause cancer in laboratory animals. Under the conditions of these 2-year feed studies, there was clear evidence that exposure to PFOA has the ability to cause liver and pancreatic cancer in male rats. There was equivocal (uncertain) evidence that PFOA has the ability to cause pancreatic, liver, or uterine cancer in female rats. Exposure to PFOA increased noncancerous tissue abnormalities in the liver and pancreas of male rats and in the liver, kidney, forestomach, and thyroid gland of female rats. The additional effect of perinatal exposure combined with postweaning exposure was uncertain and limited to an increased number of liver lesions (carcinomas) in male rats.*