be absorbed dermally, work practices were changed and engineering controls were adopted that reduced dermal exposures.

PFOA 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. PFOA binds to macro-molecules in the tissues listed above. PFOA is not metabolized and there is evidence of enterohepatic circulation of the compound. The urine is the major route of excretion of PFOA in the female rat, while the urine and feces are both major routes of excretion in male rats. There are major gender differences in the elimination of PFOA in rats. In female rats, the half-life is 24 h in the serum and 60 h in the liver; in male rats, the half-life is 105 h in the serum and 210 h in the liver. The rapid excretion of PFOA by female rats is due to active renal tubular secretion (organic acid transport system); this renal tubular secretion is believed to be hormonally controlled, since castrated male rats treated with estradiol have excretion rates of PFOA similar to those of female rats. Hormonal changes during pregnancy do not appear to change the rate of elimination in rats. This gender difference has not been observed in primates and humans.

There are limited data on PFOA serum levels in workers and the general population. Occupational data from plants in the U.S. and Belgium that manufacture or use PFOA indicate that the most recent mean serum levels in workers range from 0.84 to 6.4 ppm. The highest level reported in a worker in 1997 was 81.3 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 U.S. (range, 1.9 – 56.1 ppb).

Epidemiological studies on the effects of PFOA in humans have been conducted on workers. Two mortality studies, a morbidity study, and studies examining effects on the liver, pancreas, endocrine system, and lipid metabolism, have been conducted to date. In addition, a cross-sectional as well as a longitudinal study of the worker surveillance data have recently been submitted. However, these latest 2 studies focus primarily on PFOS rather than PFOA, even though recent PFOA levels are similar to or higher than PFOS levels in workers at these plants. (It should be noted that PFOS levels in the sampled general population are much higher than PFOA levels).

A retrospective cohort mortality study demonstrated a weak, although statistically significant association between prostate cancer mortality and employment duration in the chemical facility of a plant that manufactures PFOA. However, in a recent update to this study in which more specific exposure measures were used, a significant association for prostate cancer was not observed. In a morbidity study, workers with the highest PFOA exposures for the longest durations sought care more often for prostate cancer treatment than workers with lower exposures.

Another study reported an increase in estradiol levels in workers with the highest PFOA serum levels; however, none of the other hormone levels analyzed indicated any adverse effects. Some