Distributions of Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) in Japan and Their Toxicities

Shoji Nakayama, Kouji Harada, Kayoko Inoue, Kazuaki Sasaki, Benjamin Seery, Norimitsu Saito and Akio Koizumi

Department of Health and Environmental Sciences, Kyoto University Graduate School of Medicine, Kyoto 606-8501, Japan

1Research Institute for Environmental Sciences and Public Health of Iwate Prefecture, Morioka 020-0852, Japan

(Received June 30, 2005; accepted November 11, 2005)

Key words: perfluorooctanoic acid, perfluorooctane sulfonate, distribution in Japan, toxicology, toxicokinetics

Perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) are end products of many fluorochemical compounds in the natural environment. The aim of this review is to summarize several studies in Japan and characterize the toxicities of these compounds. We also compared the levels of contamination with those reported from various countries to illustrate the unique situation of the toxicological issues within Japan.

PFOA and PFOS concentrations in surface water in Japan are in the ranges of 0.1–67,000 ng/L and 0.1–526 ng/L, respectively. While the origin of PFOS in surface water remains unknown, PFOA present in surface water is very likely to have been released from a few industries. The levels of PFOA and PFOS in the atmosphere are 71.8–919 pg/m³ and 2.3–21.8 pg/m³, respectively. The concentrations of PFOA and PFOS in Japanese serum range from an undetectable level to 52.2 ng/ml and from 0.2 to 57.7 ng/ml, respectively. The levels of PFOA and PFOS present in the serum of the inhabitants of Kyoto are higher than those of other cities. One epidemiological study conducted by 3M revealed an increase in prostate cancer mortality [3.3-fold increase (95% CI, 1.02–10.6)] among workers exposed to PFOA. Another study conducted by 3M revealed an increase in bladder cancer mortality (SMR 12.77, 95% CI 2.63–37.35) among workers exposed to PFOS.

PFOA and PFOS had a low order of toxicity in an acute toxicity study in rodents; however, they exhibited versatile toxicities in primates. Both chemicals are carcinogenic in rodents, causing reproductive toxicity, neurotoxicity, and hepatotoxicity. Additionally, peroxisome proliferation and calcium channel modulation are demonstrated effects. There are large interspecies differences in toxicokinetics.

*E-mail: koizumi@pbh.med.kyoto-u.ac.jp