Reference 4

Risk Report on Perfluorooctanesulfonate (PFOS) as a Component of Mist Suppressants in Chrome-Plating Tanks

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Naval Health Research Center Detachment (Toxicology)

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Andrew J. Bobb[1], Ph.D., USNR
Kenneth R. Still, Ph.D, MSC, USN

1. **Naval Health Research Center Detachment (Toxicology) – NHRC/TD**
   Bldg 433
   2612 5th St.
   Wright-Patterson AFB, OH 45433-7903

Correspondence to LT Andrew J. Bobb at NHRC/TD

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ABSTRACT

Perfluorooctanesulfonate (PFOS) is a synthetic perfluorinated surfactant recently discovered to be ubiquitous in the environment. Animal data suggest a high tolerance for PFOS, as does epidemiological analysis of workers in PFOS manufacturing plants. A suggested reference dose of 0.02 mg/kg/day is presented. This constitutes an unlikely exposure level in the application of interest, as a component of mist suppressant in chromium plating tanks.

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PFOS: A UBIQUITOUS FLUOROCARBON

Perfluorooctanesulfonate (PFOS) and Perfluorooctanoic acid (PFOA) are structurally and chemically related synthetic perfluorinated surfactants used in a number of industrial applications, including plasticizers, lubricants, wetting agents, etc. The current application of interest to the Navy is the use of PFOS in mist suppressants, preventing the release of aerosols from chromium plating tanks. PFOS/PFOA also appear to be the metabolic product of breakdown of other xenobiotic compounds (Olsen et al, 1999). Recent reports have suggested that PFOS is nearly ubiquitous in the environment (Giesy and Kannan, 2001; Kannan et al 2001a; Kannan et al 2001b), and that it may bioaccumulate at higher levels in the food chain (Giesy and Kannan, 2001). Commercially available human serum (presumably without occupational exposure to PFOS) contains an average PFOS concentration of 24 ppb (Hansen et al, 2001)

RODENT DATA

Toxicity data from rodents suggests a high potential for liver toxicity for both compounds, and some evidence for developmental toxicity. Inhalation of the ammonium salt of PFOA at 8 or 84 mg/m^3 results in liver-weight increases and microscopic liver necrosis in rats (Kennedy et al, 1986). No published data on inhalational PFOS exposure is available. Rats gavaged with up to 50 mg/kg/day ammonium PFOA had significant increases in estogen, and decreases in testosterone (Cook et al, 1992). Rats which were fed PFOA or PFOS exhibited reduced cholesterol synthesis and reduced serum triacylglcerides (Haughom and Spydevold, 1992). *In utero* exposure to PFOS at levels up to 1.0 mg/kg/day had no effect on rabbit pups up to the time of birth (Case et al, 2001a); but rat pups born to dams fed 1.6 mg/kg/day exhibited high infant mortality (Case et al 2001b).

HUMAN DATA

Humans have been regularly exposed to PFOA and PFOS in industrial synthesis plants. An epidemiological study of 2788 male and 749 female workers employed in a PFOA synthesis plant between 1947 and 1983 (Gilliland and Mandel, 1993) exhibited no significant deviations from unexposed individuals, except for a possible increase in prostrate cancer deaths (4 deaths in exposed workers, 2 in unexposed). Another study of 115 occupationally exposed workers found no changes in hepatic enzymes, lippoproteins and cholesterol (Gilliland and Mandel, 1996). Another study of a total of 191 occupationally-exposed workers (performed in two different years) found no significant effect of PFOA on human hormone levels (Olsen et al, 1998); a similar study with PFOS using 317 male workers found no effects on serum hepatic enzymes, cholesterol, or lipoproteins (Olsen et al 1999). The half-life of PFOA in human systems is estimated to be 18 to 24 months (Ubel et al, 1980) and the half-life of PFOS may be even longer (Olsen et al, 1999).

APPLICATION AND CONCLUSIONS

There exists significant contradiction between the rodent and human data for PFOS/PFOA exposure. A potent liver toxicant in rodents should produce some level of toxicity in humans, particularly over the long exposure times; therefore it may be that the toxicity seen in rodents is the result of a mechanism which is not active in humans. This is not unprecedented; saccharin causes bladder tumors in rats (Reuber 1978) yet epidemiological data demonstrate that it is clearly noncarcinogenic in humans (Elcock and Morgan, 1993). Specific mechanisms exist in some animals, particularly in response to high-dose exposure, that render extrapolation between species impossible, for a particular effect (Cohen, 1995; Whysner and Williams, 1996).

PFOS is a component of mist suppressants used in chrome plating tanks. The primary hazard in such applications is hexavalent chromium a known carcinogen. Analysis of plating tank contents (Naval Facilities Engineering, Personal Communication; testing done by Centre Analytical Laboratories, Inc., State College, PA) indicates a concentration of <37 mg/L. PFOS has a very low volatility (so much so that it has not been possible to obtain vapor inhalational toxicology data), therefore it is likely that the only airborne exposure will come from process-generated aerosols.

Given a lack of human exposure data (apart from cumulative serum levels) it is impossible to compare the animal and human data, or to derive a safe exposure level solely from the industrial exposure data. Both the liver toxicity and the potential reproductive toxicity (changes in hormone levels) exhibited in animal exposure data are specifically contradicted by human epidemiological data. There is, however, no evidence to suggest that the animal developmental toxicity data is inapplicable to humans. It seems therefore most conservative to base toxicity profiles on this data. The NOAEL is 1.0 mg/kg/day in rabbits (Case et al, 2001a). Multiplying by an interspecific uncertainty factor of 10 and an intraspecific uncertainty factor of 5 (reduced from 10 because the epidemiological data suggest similar response to this compound between males and females- Gilliland and Mandel, 1993), we would derive a maximum daily dose of 0.02 mg/kg/day. For a 70 kg individual, therefore, the recommended limit would equate to drinking ~35 mL of tank contents, an unlikely exposure level. Furthermore, personnel likely to be exposed to PFOS from tanks or process-generated aerosols will be co-exposed to hexavalent chromium at much higher concentrations, and with much more serious health consequences. Measures in place to monitor or control chromium exposure will be more than adequate to protect the health of workers from PFOS, and that PFOS in chrome plating tanks will not significantly increase the risk of heath consequences, barring any unforeseen complications of co-exposure.

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Andrew J. Bobb, Ph.D. Kenneth R. Still, Ph.D. MSC, USN

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Naval Health Research Center Detachment Toxicology
NHRC/TD
2612 Fifth Street, Building 433
Area B
Wright-Patterson AFB, OH 45433-7903

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Area B
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