Triclosan

Triclosan [2,4,4′- trichloro-2′-hydroxydiphenyl ether; 5-chloro-2-(2,4-dichlorophenoxy)phenol); Microban] is a polychlorinated diphenyl ether with a single hydroxyl group. It is a broad spectrum antimicrobial and antifungal agent used in personal care products (e.g., soaps, deodorants, toothpaste, shaving cream, mouth wash), household cleaning products, and other consumer products, such as toys, bedding, trash bags, kitchen utensils. Triclosan has been measured in human plasma and breast milk in studies conducted in Australia and Sweden (Allmyr *et al.*, 2008). It is present in wastewater effluent, and adsorbs to sewage sludge. The general population is exposed to Triclosan as a result of its use in a variety of consumer products.

Triclosan passed the animal data screen, underwent a preliminary toxicological evaluation, and is being brought to the Carcinogen Identification Committee for consultation. This is a compilation of the relevant studies identified during the preliminary toxicological evaluation.

Epidemiological data

No cancer epidemiology studies were identified.

Animal carcinogenicity studies

- Long-term diet studies in rats
  - Two-year studies in male and female Sprague-Dawley rats: as reviewed in U.S. EPA (2008, pp. 5-6, 10-13, 38)

- Long-term diet studies in hamsters
  - 95-week studies in male and female Alexander Syrian hamsters: as reviewed in U.S. EPA (2008, pp. 6, 14-16, 38)

- Long-term diet studies in mice
  - 18-month studies in male and female CD-1 mice: as reviewed in U.S. EPA (2008, pp. 6-7, 16-21, 38)

Other relevant data

- Genotoxicity
  - Review: U.S. EPA (2008, pp. 7, 22-24, 39)

- Metabolism
  - Review: U.S. EPA (2008, p. 21)

- Structure activity considerations
    - Structurally similar to polychlorinated biphenyls (PCBs), which are Proposition 65 carcinogens
    - Structurally similar to polybrominated diphenyl ethers (PBDE). DecaBDE (decabromodiphenyl oxide) induced liver tumors in mice: NTP (1986)
    - Review: U.S. EPA (2008, p. 24, 39)

- Mode of action considerations
    - Review: U.S EPA (2008, pp. 27-34, 39-40)
    - Mode of action studies on other peroxisome proliferators: Ito *et al.* (2007); Yang *et al*. (2007); Takashima *et al*. (2008);

Reviews

- U.S. EPA (2008)

References[1]

Allmyr M, Harden F, Toms LM, Mueller JF, McLachlan MS, Adolfsson-Erici M, Sandborgh-Englund G (2008). The influence of age and gender on triclosan concentrations in Australian human blood serum. *Sci Total Environ* **393**(1):162-167.

Ito Y, Yamanoshita O, Asaeda N, Tagawa Y, Lee, C-H, Aoyama T, Ichihara G, Furuhashi K, Kamijima M, Gonzalez FJ, Nakajima T (2007). Di(2-ethylhexyl)phthalate induces hepatic tumorigenesis through a peroxisome proliferatior-activated receptor α-independent pathway. *J Occup Health* **49**:172-182.

National Toxicology Program (NTP, 1986) *Technical Report on the Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No. 1163-19-5) in F344/N Rats and B6C3F$_1$ Mice (Feed Studies).* NTP TR 309. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health.

Takashima K, Ito Y, Gonzalez FJ, Nakajima T (2008). Different mechanisms of DEHP-induced hepatocellular adenoma tumorigenesis in wild-type and *Pparα*-null mice. *J Occup Health* **50**:169-80.

U.S. Environmental Protection Agency (U.S. EPA, 2008). Cancer Assessment Document. Evaluation of the carcinogenic potential of Triclosan. Cancer Assessment Review Committee. Health Effects Division. Office of Pesticide Programs. Final. January 4, 2008.

[1] Copies of these listed references, as either the abstract, the relevant sections of the publication, or the complete publication, have been provided to members of the Carcinogen Identification Committee. These references have been provided in the order in which they are discussed in this document.

Yang Q, Ito S, Gonzalez FJ (2007). Hepatocyte-restricted constitutive activation of PPARα induces hepatoproliferation but not hepatocarcinogenesis. *Carcinogenesis* **28**:1171-1177.