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NEW YORK STATE
- HUMAN HEALTH FACT SHEET -

Ambient Water Quality Value for Protection of Sources of Potable Water

SUBSTANCE: 2,3,6-Trichlorotoluene **CAS REGISTRY NUMBER:** 2077-46-5

AMBIENT WATER QUALITY VALUE: 0.34 ug/L

BASIS: Non-oncogenic, Chronic (6 NYCRR 702.5)

SUMMARY OF INFORMATION

Introduction:

2,3,6-Trichlorotoluene (TCT) is one isomeric form of trichlorotoluene ($C_7H_5Cl_3$), a substituted halobenzene, with a molecular weight of 195.47. No information on the physico-chemical properties of 2,3,6-TCT was found.[1,2]

Pharmacokinetics:

No data on the absorption or metabolism of 2,3,6-TCT in humans or animals were found. However, the observed systemic toxicity of 2,3,6-TCT following oral exposure indicates that the compound is absorbed from the gastrointestinal tract.

Acute Toxicity:

No information on the acute toxicity of 2,3,6-TCT in humans was found. A Soviet report indicates that LD_{50} values for 2,3,6-TCT in mice (oral route) and rats (route not specified) are 2,000 mg/kg and 4,800 mg/kg, respectively.[2]

Chronic Toxicity:

Data are limited to a study of rats fed 0, 0.5, 5.0, 50 or 500 ppm 2,3,6-TCT in the diet for 28 days.[3] Growth rate and food consumption were reportedly not affected by the treatment. 2,3,6-TCT ingestion in male rats was reported to cause a statistically significant increase in liver weight at 5 and 500 ppm and significantly increased serum sorbitol dehydrogenase (SDH) activity at 5 ppm. SDH is a specific indicator of hepatic injury and perturbation of organelle structure. SDH activity at 0.5 ppm 2,3,6-TCT was elevated but not significantly. The authors indicated that the liver, kidney and thyroid gland were target organs and that histological changes became progressively more severe and occurred more frequently as dose levels increased. Measured SDH levels at 50 and 500 ppm 2,3,6-TCT were slightly elevated but the assay results at these higher doses were unreliable due to methodological difficulties.[4] The data, which are limited by inadequate reporting of scant results and poorly-characterized histopathology, suggest a no observed effect level (NOEL) of 0.5 ppm (0.048 mg 2,3,6-TCT/kg/d) for the 28 day study.

Longer term studies to evaluate the chronic toxicity of 2,3,6-TCT were not found.

Developmental Effects:

Ruddick et al. (1982)[5] reported in abstract form that 2,3,6-TCT administered by gavage at doses of 0, 100, 200 or 400 mg/kg/day to pregnant rats on days 6-15 of gestation resulted in reduced fetal weight at the highest dose studied. Histological changes were observed in the thyroid, bone marrow, kidney and liver of dams, presumably at all doses. Pups had liver damage which was most severe at 400 mg/kg/day 2,3,6-TCT. It was inferred that pup liver damage occurred at all doses of 2,3,6-TCT.

Genotoxicity:

No data were found.

Oncogenicity:

No data were found.

Current Standards and Guidelines:

The USEPA[6] has proposed a subchronic oral risk reference dose (RfD) of 0.05 ug/kg/day based on the results of the 28-day Chu et al. study.

Under the State Sanitary Code (10 NYCRR Part 5, Public Water Supplies), the New York State Department of Health has established a maximum contaminant level of 5 ug/L for "Principal Organic Contaminants" such as 2,3,6-TCT in drinking water.[7]

DERIVATION OF VALUE

As required in 6 NYCRR 702.2(b), if a value derived from a no-observed-effect level in scientifically valid animal studies is most stringent, that value must be used. Following procedures in 6 NYCRR 702.5, the no effect level of 0.5 ppm (0.048 mg/kg/d) 2,3,6-TCT may be used with an uncertainty factor of 1,000 to derive an acceptable daily intake (ADI) of 0.048 ug 2,3,6-TCT/kg/day. Assuming an average adult body weight of 70 kg and a daily water consumption of two liters, a concentration of 0.34 ug 2,3,6-TCT/L in water would provide 20% of the ADI. Although the data are limited and the study is of relatively short duration, 0.34 ug/L is recommended as an ambient water quality value for 2,3,6-TCT until additional toxicological information becomes available.

REFERENCES

1. Toxic Substances Control Act (TSCA) Chemical Substance Inventory. 1986. Volume II. Washington, D.C: U.S. Environmental Protection Agency. P. 590.

2. Registry for Toxic Effects of Chemical Substances (RTECS). 1988. Trichlorotoluene. Updated 10/7/88. Cited from Gig. Sanit. 45(12): 64 (1980).

3. Chu, I., S.Y. Shen, D.C. Villeneuve, V.E. Secours and V.E. Valli. 1984. Toxicity of trichlorotoluene isomers: a 28-day feeding study in the rat. J. Environ. Sci. Health, Part B 19: 183-191.

4. Chu, I. May, 1991. Personal communication.

5. Ruddick J.A., D.C. Villeneuve, V. Secours and V.F. Valli. 1982. A transplacental and teratological evaluation of three trichlorotoluene congeners in the rat. Teratology. 25(2):72A-73A. (as cited in US Environmental Protection Agency, 1987.)

6. US Environmental Protection Agency (US EPA). 1987. Health and Environmental Effects Document for Chlorinated Toluenes, External Review Draft. Cincinnati, OH: Environmental Criteria and Assessment Office.

7. 10 NYCRR Part 5, Drinking Water Supplies (Statutory Authority: Public Health Law Section 225) Subpart 5-1. January, 1989. New York State Department of Health.

Search Strategy:

1. Index Medicus, 1981 - February 1991.

2. Chemical Abstracts, 1979-1988.

3. The following databases through 3/91: Toxline, Toxlit 65 and Toxlit, Chemline, and Hazardous Substances Data Bank (HSDB).

4. U.S. Environmental Protection Agency. 1991. Integrated Risk Information System (IRIS) Database. Washington, D.C: Office of Health and Environmental Assessment. (March 1, 1991).

5. National Research Council. Drinking Water and Health, Volumes 1-9. Safe Drinking Water Committee, Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences. Washington, D.C: National Academy Press.

6. International Agency for Research on Cancer (IARC). IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volumes 1-47. Lyon, France: IARC.

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