Fact Sheet Date: March 12, 1998

NEW YORK STATE
- HUMAN HEALTH FACT SHEET -

Ambient Water Quality Value for
Protection of Sources of Potable Water

SUBSTANCE: 1,2-Dichloropropane **CAS REGISTRY NUMBER:** 78-87-5

AMBIENT WATER QUALITY VALUE: 1 ug/L

BASIS: Oncogenic

SUMMARY OF INFORMATION

This fact sheet updates a fact sheet prepared in 1991 (NYS, 1991).

1,2-Dichloropropane is used as an intermediate in the synthesis of tetrachloroethylene and carbon tetrachloride (IARC, 1986). Toxicity to humans has been observed in case reports of ingestion and inhalation. Recent reviews of the toxicity of 1,2-dichloropropane have been conducted by the U.S. Environmental Protection Agency (Basu et al. 1990, 1987a, 1987b), University of California (Reed et al., 1988), the International Agency for Research on Cancer (IARC, 1986), and the Agency for Toxic Substances and Disease Registry (1989).

1. Acute/Chronic Toxicity

   The toxicity of 1,2-dichloropropane (1,2-DCP) has been observed in both humans and animals. Data on effects in humans as a result of exposure to 1,2-DCP are limited to case reports of acute effects (USEPA, 1987a; ATSDR, 1989), including bleeding from the uterus, central nervous system depression, respiratory irritation, hepatic failure and damage, renal failure after inhalation, and death after ingestion. Allergic dermatitis, haemolytic anemia, and intravascular coagulation have also been reported (IARC, 1986).

   In short-term oral or inhalation exposures of animals to 1,2-DCP significant mortality

was noted in several species (NTP, 1986; Heppel et al., 1946). The liver is the most severely affected organ followed by the kidneys, the adrenal glands, and central nervous system. Similar effects were noted in the livers of rats, mice, guinea pigs, and dogs that received daily acute inhalation exposures, including fatty degeneration, atrophy, necrosis, and lipid deposits. Kidneys and adrenals were affected in dogs, mice, rats, and guinea pigs. Mice, rats and dogs suffered incoordination and narcosis after acute inhalation exposure (Heppel et al., 1946, 1948). Liver glutathione (GSH) content decreased markedly after high-dose inhalation or intraperitoneal exposures in rats (DiNucci et al., 1990; Trevisan et al., 1989). Acute oral exposure caused GSH depletion in liver, kidney and blood and increases in biochemical indicators of liver and kidney damage and of hemolysis (Imberti et al., 1990).

After subchronic exposure to 1,2-DCP, increases in liver weight, centrilobular congestion, necrosis, increases in the size of hepatocytes and hemosiderin deposition in Kupffer cells and spleen were noted in rats (NTP, 1986; Heppel et al., 1946). Liver congestion, fatty degeneration, necrosis, clear cell changes, and liver enlargement were reported in mice, rats and dogs (Heppel et al., 1946, 1948). Other effects described include: hemolytic anemia; increases in serum cholesterol, beta-lipoproteins and gamma-globulins; and increases in non-protein sulfhydryl levels (GSH) and serum enzymes indicative of liver damage (Bruckner et al., 1989; Trevisan et al., 1989; Kurysheva and Ekshtat, 1975).

Renal damage in rats included congestion, fatty infiltration, atrophy and degeneration of the convoluted tubules. Guinea pigs developed renal fibrosis and fatty degeneration, tubular atrophy and dilation. Dogs developed granulomatous lesions. Respiratory effects of sub-chronic inhalation exposure to 1,2-DCP included pulmonary congestion and irregular respiration in rats and guinea pigs, and hyperplasia and degenerative changes in the nasal tissue of rats (Heppel et al., 1946, 1948).

After 103-week oral (gavage) exposure female rats and male mice exhibited liver necrosis. Mammary gland hyperplasia occurred in low-dose female rats. Body weight depression occurred in male and female rats (NTP, 1986).

2. Pharmacokinetics

1,2-Dichloropropane is absorbed rapidly and extensively in the gastrointestinal tract. Due to its lipophilic nature, it would be expected to distribute to tissues of high fat content. The metabolism of 1,2-DCP has not been well studied, but appears to occur mainly in the liver (Reed et al., 1988). From the metabolites formed, it is hypothesized that 1,2-DCP oxidation leads to formation of 1,2-epoxypropane and beta-chloroacetaldehyde. The epoxide conjugates with gluthathione to form N-acetyl-S-(2-hydroxypropyl)cysteine, the major urinary metabolite, or hydrolyzes to propane-1,2-diol. Oxidation of beta-chloroacetaldehyde produces beta-

chlorolactate and oxalate. Carbon dioxide is a major end product. The elimination of 1,2-DCP and its metabolites occurs through the lung, urine and feces, up to 90% of an oral dose in 24 hours (Hutson et al., 1971).

3. Developmental and Reproductive Effects

No dose-related effects on a number of reproductive parameters were found as a result of exposing pregnant rats to 1,2-DCP by gavage during gestational days 6-21 (Kirk et al., 1989). Testicular degeneration and reduced sperm production were found in rats treated with 500 mg/kg/day 1,2-DCP in corn oil (Bruckner et al., 1989). An increased incidence of delayed ossification of the skull was observed in fetuses of dams treated with 1,2-DCP during gestation (Kirk et al., 1989). No effects on fertility were found in a two-generation reproductive study in rats (Hanley et al. 1992).

4. Genotoxicity

1,2-Dichloropropane has shown genotoxic effects in several in vitro systems. Mutagenic activity in several strains of Salmonella typhimurium with or without the addition of metabolizing enzymes has been demonstrated by several investigators (Principe et al., 1981; Haworth et al., 1983; Carere et al., 1981; Tennant et al., 1987) and in Aspergillus (Principe et al., 1981; Carere et al., 1981) and Drosophila (Woodruff et al., 1985). It has shown evidence of activity in mammalian cells, inducing chromosomal aberrations and sister chromatid exchanges in Chinese hamster ovary cells (NTP, 1986; Von der Hude et al., 1987) and mutagenic activity in mouse lymphoma cells (Woodruff et al., 1985). Results of a dominant lethal study in male rats exposed to 1,2-DCP in drinking water for 10 weeks before breeding were negative (Hanley et al., 1989). The mutagenic activity in micro-organisms and chromosomal damage in mammalian cells are evidence of a potential for oncogenic activity due to 1,2-dichloropropane exposure (ATSDR, 1989).

5. Epidemiology

There are no epidemiologic studies available on the effects of 1,2-DCP (USEPA, 1990).

6. Carcinogenicity

The carcinogenicity of 1,2-DCP administered orally has been tested in two species in a 2-year bioassay in F344/N rats and B6C3F1 mice (NTP, 1986). Two dose levels of 1,2 -DCP in corn oil were administered by gavage to groups of 50 animals of each sex over a 103-week exposure period, with a concurrent group of 50 control animals.

In male mice, liver adenomas were significantly elevated as were non-neoplastic liver lesions (Table I). The incidence of liver adenoma was elevated, significantly, in female mice exposed to 1,2-DCP. The treated mice also had slightly higher incidences of liver carcinomas (male: control, 11/50; low-dose 16/50; high-dose 16/50; female: control 1/50; low-dose 3/50; high-dose 4/50), but the increases were not statistically significant (NTP, 1986). The combined incidence of adenoma and carcinoma was significant in both sexes compared to controls. The combined incidence of thyroid follicular cell adenomas and carcinomas was significantly elevated in high-dose female mice after adjustment for mortality. The uncombined incidence was not elevated, however. Squamous cell papillomas of the forestomach occurred in exposed male and female mice at levels above the incidence of historical controls. NTP concluded there was some evidence of carcinogenicity for male and female mice exposed to 1,2-dichloropropane (NTP, 1986).

In female rats fed 1,2-DCP, mammary gland adenocarcinomas increased with a significant positive trend (Table I). A positive trend in endometrial polyps, and non-neoplastic changes in the liver, but no liver tumors, were found. The marginal increases in mammary gland adenocarcinomas, relatively uncommon tumors, was found significant only in high-dose female rats after adjustment for mortality. NTP concluded there was equivocal evidence of carcinogenicity in female rats. Although an increased incidence of liver adenomas in male rats was found after 1,2-DCP exposure, NTP concluded the chemical was not a carcinogen in male rats (NTP, 1986; USEPA, 1987a).

On the basis of convincing mouse response and the marginal response in female rats, the Carcinogen Assessment Group in February 1987 classifed 1,2-dichloropropane as a B2 carcinogen, citing sufficient evidence in animals and other supporting evidence (USEPA, 1987b). There is evidence of mutagenic actvity in short-term tests. The metabolic intermediates, 1,2-epoxypropane and chloro-acetaldehyde are expected to have a carcinogenic potential. 1,2-dichloropropane is structurally related to compounds with known carcinogenic activity in animal test systems including 1,2-dichloroethane, 1,2-dibromoethane and 1,2-dibromo-3-chloropropane (USEPA, 1987a).

Table I

Rates of Tumor Incidence in Mice and Rats
in NTP (1986) 1,2-DCP Carcinogenesis Bioassay

Dose (mg/kg)	Tumor Type	Tumor Incidence	P

Male Mice			
0	Liver Adenoma	7/50 (14%)	
	Adenoma + Carcinoma	18/50 (36%)	0.002[C]
125	Liver Adenoma	10/50 (20%)	
	Adenoma + Carcinoma	26/50 (52%)	
250	Liver Adenoma	17/50 (34%)	0.017[F]
	Adenoma + Carcinoma	33/50 (66%)	0.002[F]
Female Mice			
0	Liver Adenoma	1/50 (2%)	
	Adenoma + Carcinoma	2/50 (4%)	0.025[C]
125	Liver Adenoma	5/50 (10%)	
	Adenoma + Carcinoma	8/50 (16%)	0.046[F]
250	Liver Adenoma	5/50 (10%)	
	Adenoma + Carcinoma	9/50 (18%)	0.026[F]
Female Rats			
0	Mammary Gland Adenocarcinoma	1/50 (2%)	0.06[C]
	Fibroadenoma	15/50 (30%)	
125	Mammary Gland Adenocarcinoma	2/50 (4%)	
	Fibroadenoma	20/50 (40%)	
250	Mammary Gland Adenocarcinoma	5/50 (10%)	0.01[L]
	Fibroadenoma	7/50 (14%)	

[C] Cochrane-Armitage Trend test
[F] Fisher Exact test
[L] Life Table test

DERIVATION OF VALUE

The significant oncogenic response after oral exposure to 1,2-dichloropropane in male and female mice in the liver in a well-conducted assay (NTP, 1986), the positive results in short-term tests that are indicative of potential oncogenic activity, and the marginal induction of uncommon tumors in the female rat satisfy the criteria set forth in the definition of an oncogenic chemical in section 700.1. Therefore, a value can be derived for 1,2-DCP using the section 702.4 procedure based on oncogenic effects. This value would be more stringent than any derived by other procedures based on the protection of human health.

1. Other Quantitative Risk Assessments

USEPA performed a quantitative risk assessment using the data in the NTP (1986) assay (USEPA, 1987a; 1990). "The data used for the calculation of potency (slope) values is shown in Table II. Based on these data and using a linearized multistage model a carcinogenic potency factor (q_1*) for humans of 6.7×10^{-2} $(mg/kg/day)^{-1}$ was calculated from the data for male mice and a q_1* of 2.2×10^{-2} $(mg/kg/day)^{-1}$ was calculated from the data for female mice. The higher of the two values is the appropriate basis for the estimation of cancer risk levels. The upper-limit risk estimates from the animal data are derived from a linearized multistage (LMS) nonthreshold extrapolation model which is currently programmed as GLOBAL 83" (USEPA 1987a).

Using a potency value of 6.7×10^{-2} $(mg/kg/day)^{-1}$, and a 70 kg adult consuming 2L of water per day, USEPA derived the concentration of 1,2-DCP in water of 0.52 ug/L, at an excess cancer risk of 10^{-6}.

The California Department of Health Services derived human potency factors for 1,2-DCP exposure based on the combined incidence of the adenoma/carcinomas in mice in the NTP (1986) bioassay of 6.41×10^{-2} $(mg/kg/day)^{-1}$ for male mice and 2.02×10^{-2} $(mg/kg/day)^{-1}$ for female mice, using the GLOBAL 83 model (Reed et al., 1988). They derived an ingestion-only criterion of 0.75 ug/L by averaging the male and female potencies and using slightly different water consumption rates than EPA.

2. Selection of Data

Although human data are preferred over animal data, there are no epidemiological studies on which to base a value. The NTP (1986) bioassay provides animal tumor incidence data after oral exposure that are appropriate for deriving a water quality standard. This study, which is the only carcinogenicity bioassay, reports data for

TABLE II
Data Used for the Derivation of q_1*[a]

(From USEPA, 1987a)

Compound: 1,2-Dichloropropane

Species, Strain, Sex: mice, B6C3F1, male and female

Body Weight: males 0.04 kg, females 0.038 kg (measured)

Length of Exposure (l_e) = 721 days

Length of Experiment (L_e) = 742 days

Lifespan of Animal (L) = 742 days

Tumor Site and Type: liver/adenoma or carcinoma

Route, Vehicle: by gavage/corn oil

Experimental Doses or Exposures (mg/kg/day, 5 days/week)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested (or Examined)[b]	
		males	females
0	0	18/50 (p=0.002)	2/50 (p=0.025)
125	86.8	26/50	8/50 (p=0.046)
250	173.5	33/50 (p=0.002)	9/50 (p=0.026)

Human q_1* = 6.7×10^{-2} $(mg/kg/day)^{-1}$ from the data for male mice[c]
Human q_1* = 2.2×10^{-2} $(mg/kg/day)^{-1}$ from the data for female mice

[a] Source: NTP, 1986

[b] Probability values for the Cochrane-Armitage test for linear trend are given after the incidence of tumors in controls and for the Fisher exact tests are given after the incidence of tumors in dosed groups, when $p<0.05$.

[c] human q_1* = $\left[\frac{\text{human body weight}}{\text{animal body weight}}\right]^{1/3}$ x animal potency $(mg/kg/day)^{-1}$

two sexes of two mammalian species exposed to two dose levels in a well-designed study. The study reported increases in hepatocellular adenoma and carcinoma in male and female mice, and mammary gland adenocarcinoma in the female rat. A summary of the data sets showing statistically and biologically significant increases in tumor response is presented in Table I. The liver has been shown to be the site of the principal toxic response to 1,2-DCP exposure (see Acute/Chronic Toxicity). The mouse liver data exhibits a significant dose - related response, whereas the female rat response is significant only at the highest dose. The high dose was toxic in the female rats, however. The dose-response data for hepatocellular adenoma and carcinoma in B6C3F1 mice (NTP, 1986), shown in Table II, are therefore considered the most appropriate for quantitative assessment of cancer risk from exposure to 1,2-dichloropropane.

3. Selection of Model/Output

For derivation of a water quality standard, Part 702 specifies use of a linear multi-stage (LMS) low-dose extrapolation model unless there is sufficient scientific evidence that supports use of another extrapolation procedure.

The GLOBAL82 LMS model (Crump, 1982) is chosen to estimate the dose from which the 1,2-DCP values are derived. Both the 95 percent lower confidence limit (LCL) and maximum likelihood estimate (MLE) are calculated for the animal dose associated with a 1×10^{-6} lifetime excess cancer risk. The confidence limit provides a value that is 95 percent certain not to exceed the value associated with the true risk. The maximum likelihood estimate should have the best association with true risk, which may be as low as zero. The animal doses associated with the LCL 10^{-6} excess cancer risk are 0.178 ug/kg/day for male mice and 0.559 ug/kg/day for female mice (Table III).

4. Conversions

The animal doses associated with a 1×10^{-6} excess cancer risk, are converted below to a human dose by the surface area conversion rule as specified in Part 702. Metabolism of 1,2-DCP in the liver lends support for the use of surface-area conversion, and there appears to be no compelling reason for using an alternative conversion procedure.

$$\text{Human dose} = \left(\frac{\text{animal body weight}}{\text{human body weight}} \right)^{0.33} \times \text{animal dose}$$

$$\text{Human dose} = \left(\frac{0.040\ \text{kg}}{70\ \text{kg}} \right)^{0.33} \times 0.178\ \text{ug/kg/day}$$

ug/kg/day
(Male Mice)

$$\text{Human dose} = \frac{0.038\ \text{kg}}{}^{0.33} \times 0.559\ \text{ug/kg/day}$$

(Female Mice) $\left(\frac{\text{70 kg}}{} \right)$

where 0.040 kg = weight of male mice

0.038 kg = weight of female mice

70 kg = weight of adult human

0.33 = surface area scaling factor

0.178 = male dose at 10^{-6} excess lifetime risk, (ug/kg/day)

0.559 = female dose at 10^{-6}

Human dose = 1.48 x 10^{-2} ug/kg/day from male mice (Male Mice)

Human dose = 4.56 x 10^{-2} ug/kg/day from female mice (Female Mice)

Human daily doses are converted to drinking water values that are based upon lifetime exposure of a 70-kg human consuming 2 liters of water per day (see Table III).

TABLE III. GLOBAL Output and Water Quality Value Derived Using NTP (1986) Mouse Liver Tumor Data

Data Set	Animal Dose (ug/kg/day)		Human Dose (ug/kg/day) x 10^{-2}	Water Value (ug/L)	Water Value ug/L
	MLE	95% LCL	95% LCL	95% LCL	MLE
Male Mice	.335	.178	1.48	0.52	0.98
Female Mice	.950	.559	4.56	1.6	2.72
Combined M/F	.522	.351	2.89	1.0	1.49

5. Values and Their Uncertainties

Results of the quantitative risk assessment based on male and female liver tumor data from the NTP (1986) bioassay are presented in Table III. Values based on both the 95 percent lower confidence limit and the MLE are provided, although only the former meet the Part 702 requirements as a basis for the value. Values based on the lower confidence limit of individual data sets range from 0.5 to 1.6 ug/L. Based on combined male and female data a water value is 1.0 ug/L. Values based on the MLE range from 0.98 ug/L to 2.72 ug/L. The small differences between the MLEs and LCLs for the individual or combined data suggest lower degree of uncertainty in predicting risk for the 10^{-6} level.

The value based on the male data in Table III is equal to that in USEPA (1990), because data sets, model and related assumptions are equivalent. The value based on combined data is slightly different than that in Reed et al. (1988), which is based on the average of potency values calculated from the data for male and female mice.

Some differences in the occurrence of tumors after oral exposure to 1,2-DCP were noted in males and females. Variabilities in responses may occur because of experimental conditions. Survival in female mice was reduced due to infections and significantly reduced in the high-dose group. This may have reduced the possibilities for tumors to develop resulting in lower overall tumor incidence in females.

6. Selection of the Value

A risk estimate based value can be selected as 0.5 ug/L based on the male mouse data or 1.0 ug/L based on the combined male and female data. Selection of 0.5 ug/L would be based on providing protection for the most sensitive sex and assumes that the different responses are from actual differences of sex. Selection of 1.0 ug/L would be based on the assumption that the different response rates resulted from inherent variability of animal bioassays for carcinogenesis and that the larger (combined) data set presents a better estimate of the true response. Because the response may be due to actual differences in sex and in order to provide the most protective value, a value of 0.5 ug/L is selected.

ADJUSTMENT TO DERIVATION OF VALUE

The above Derivation of Value was prepared in 1994 using an interspecies scaling of doses based on the 2/3 power of relative body weights. As proposed in Part 702, the Department is revising the interspecies scaling to be done on the basis of the 3/4 power of relative body weights. Accordingly, the ambient water quality value is recalculated from the animal dose of 0.178 ug/kg/day as follows:

Human dose = $\underline{0.040}^{0.25} \times 0.178 = 0.0275$ ug/kg/day

$$\left(70 \right)$$

Ambient Water Quality Value = $\frac{0.0275 \text{ ug/kg/day} \times 70 \text{ ug/L}}{2 \text{ L}}$ = 0.963 ug/L, rounded to 1 ug/L

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SEARCH STRATEGY

TOXLINE as of 5/94
NTIS as of 5/94
Integrated Risk Information System (IRIS) - Searched 4/94
RTECS, CCRIS - Searched 4/94

New York State Department of Environmental Conservation
Division of Water
AS
June, 1994
Revised SJS January 30, 1997