August, 1988

1,3-DICHLOROPROPENE

Health Advisory
Office of Drinking Water
U.S. Environmental Protection Agency

I. INTRODUCTION

The Health Advisory (HA) Program, sponsored by the Office of Drinking Water (ODW), provides information on the health effects, analytical methodology and treatment technology that would be useful in dealing with the contamination of drinking water. Health Advisories describe nonregulatory concentrations of drinking water contaminants at which adverse health effects would not be anticipated to occur over specific exposure durations. Health Advisories contain a margin of safety to protect sensitive members of the population.

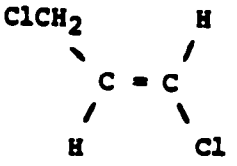
Health Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. The HAs are subject to change as new information becomes available.

Health Advisories are developed for one-day, ten-day, longer-term (approximately 7 years, or 10% of an individual's lifetime) and lifetime exposures based on data describing noncarcinogenic end points of toxicity. For those substances that are known or probable human carcinogens, according to the Agency classification scheme (Group A or B), Lifetime HAs are not recommended. The chemical concentration values for Group A or B carcinogens are correlated with carcinogenic risk estimates by employing a cancer potency (unit risk) value together with assumptions for lifetime exposure and the consumption of drinking water. The cancer unit risk is usually derived from the linear multistage model with 95% upper confidence limits. This provides a low-dose estimate of cancer risk to humans that is considered unlikely to pose a carcinogenic risk in excess of the stated values. Excess cancer risk estimates may also be calculated using the one-hit, Weibull, logit or probit models. There is no current understanding of the biological mechanisms involved in cancer to suggest that any one of these models is able to predict risk more accurately than another. Because each model is based on differing assumptions, the estimates that are derived can differ by several orders of magnitude.

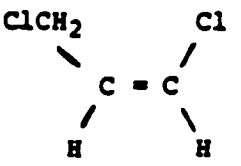
II. GENERAL INFORMATION AND PROPERTIES

CAS No. 542-75-6

Structural Formula



(trans)



(cis)

1,3-Dichloropropene
(approximately 46% trans/42% cis)

Synonyms

- Dichloro-1,3-propene; 1,3-dichloro-1-propene; cis/trans-1,3-dichloropropene; 1,3-D; DCP; D-D (approximately 28% cis/27% trans)

Uses

- DCP is the active ingredient in Telone®, a registered trademark of the Dow Chemical Company.
- The pesticide 1,3-dichloropropene (DCP) is a broad spectrum soil fumigant to control plant pests. Its major use is for nematode control on crops grown in sandy soils of the Eastern, Southern and Western U.S.
- The usage of DCP has increased due to cancellation of the once widely used product containing ethylene dibromide (EDB) and dibromochloropropane (DBCP) (U.S. EPA, 1986a).
- Estimated usage of DCP containing products in 1984 to 1985 ranged from about 34 to 40 million pounds (U.S. EPA, 1986a).

Properties (Dow Chemical USA, 1977, 1982; Clayton and Clayton, 1981)

Chemical Formula	$C_3H_4Cl_2$
Molecular Weight	110.98 (pure isomers)
Physical State (25°C)	Pale yellow to yellow liquid
Boiling Point	about 104°C (104.3°C, cis; 112°C, trans)
Density (25°C)	1.21 g/mL
Vapor Pressure (25°C)	27.3 mm·Hg
Specific Gravity	about 1.2 (20/20°C)
Water Solubility (25°C)	0.1 to about 0.25% (1 to 2.5 g/L) reported; miscible with most organic solvents
Log Octanol/Water Partition Coefficient	25
Conversion Factor (25°C)(air)	1 mg/L = 220 ppm; 1 ppm = 4.54 mg/m^3

Occurrence

- In California (Maddy et al., 1982), 54 wells were examined in areas where Telone or D-D were used for several years. The well water did not have measurable amounts of DCP (<0.1 ppb).

- Monitoring data from New York have shown positive results for DCP in ground water (U.S. EPA, 1986b).

- In deep well sampling in southern California (65 to 1,200 foot depths), no DCP was detected. In shallow wells (3 to 4 meters) around potato fields in Suffolk County, NY, DCP was detected up to 138 days after application (OPP, 1988).

- DCP has been found in 41 of 1,088 surface water samples analyzed and in 10 of 3,949 ground water samples (STORET, 1988). Samples were collected in 800 surface water locations and 2,506 ground water locations; DCP was found in 13 states. The range of concentrations found in ground water was 0.2 ug/L to 90 ug/L. The 85th percentile of all non-zero samples was 1.3 ug/L in surface water and 3.4 ug/L in ground water. This information is provided to give a general impression of the occurrence of this chemical in ground and surface waters as reported in the STORET database. The individual data points retrieved were used as they came from STORET and have not been confirmed as to their validity. STORET data is often not valid when individual numbers are used out of the context of the entire sampling regime, as they are here. Therefore, this information can only be used to form an impression of the intensity and location of sampling for a particular chemical.

Environmental Fate

- Available data indicate that DCP does leach to ground water. However, the relative hydrolytic instability of the parent compound would mitigate the potential for extensive contamination (U.S. EPA, 1986b; U.S. EPA, 1986c).

- The half-life of 1,3-DCP in soil was reported by Laskowski et al. (1982) to be approximately 10 days while Van Dijk (1974) reported 3 to 37 days depending on soil conditions and analytical methods.

- DCP hydrolyses as a function of temperature not as a function of pH. At 10°C, the half-life is 51 days while at 20°C it is 10 to 13 days. Chloroallyl alcohol is the main hydrolytic degradate. Some phololysis of DCP does occur (OPP, 1988).

- In laboratory aerobic soil metabolism studies, DCP degrades to chloroallyl alcohol in 20 to 30 days where soil pH is between 5.0 and 7.0, the temperature is between 15 and 20°C and the organic matter content is from 1.5 to 11.6 percent in sandy loam or clay soils. In anaerobic soil metabolism studies, DCP degrades to chloroallyl alcohol to less than 8 percent in 30 days. For anaerobic aquatic metabolism studies, the half-life was reported to be about 20 days at pHs of 6.9 to 7.5 (OPP, 1988).

- In a field dissipation study done in the Netherlands, DCP (220-250 lb/ injected into the soil at 9 to 19 cm depths was found to move rapidly downward over a 2 week period. In a similar study in Delano, CA, DCP was injected at 1,310 l/ha to 1,638 l/ha (=lb/A) to 81 cm. Samples at 14 days noted the presence of DCP (up to 0.5 ppm) at all depths to 8 feet (OPP, 1988).

III. PHARMACOKINETICS

Absorption

- Toxicity studies indicate that DCP is absorbed from skin, respiratory and gastrointestinal systems (Clayton and Clayton, 1981).

- Oral administration of DCP in rats resulted in approximately 90% absorption of the administered dose (Hutson et al., 1971).

Distribution

- Radiolabeled ^{14}C D-D (55% DCP) was administered orally in arachis oil in rats. After 4 days, most of the administered dose, based on measured radioactivity, was recovered primarily in urine and there were insignificant amounts (less than 5%) remaining in the gut, feces, skin and carcass (Hutson et al., 1971).

Metabolism

- cis-Dichloropropene in corn oil was given as a single oral dose (20 mg/kg bw) to two female Wistar rats. Urine and feces were collected separately. The main urinary metabolite (92%) was N-acetyl-S-[(cis)-3-chloroprop-2-enyl] cysteine. The cis-DCP has also been shown to react with glutathione in the presence of rat liver cystol to produce S[(cis)-3-chloroprop-2-enyl]glutathione. The cis-DCP is probably biotransformed to an intermediate glutathione conjugate and then follows the mercapturic acid pathway and is excreted in the urine as a cysteine derivitive (Climie and Morrison, 1978).

- In a study conducted by Dietz et al. (1984) rats and mice administered (via gavage) up to 50 and 100 mg DCP/kg bw, respectively, demonstrated no evidence of metabolic saturation.

Excretion

- In two studies (Hutson et al., 1971; Climie and Morrison, 1978) ^{14}C cis- and/or trans-DCP, administered orally in rats, were excreted primarily in the urine in 24 to 48 hours. When pulmonary excretion was evaluated (Hutson et al., 1971), the cis and trans isomers were 3.9% and 23.6% of the administered dose, respectively. Most of the cis-DCP was excreted in the urine.

IV. HEALTH EFFECTS

Humans

- The only known human fatality occurred a few hours after accidental ingestion of D-D mixture. The dosage was unknown. Symptoms were abdominal pain, vomiting, muscle twitching and pulmonary edema. Treatment by gastric lavage failed (Gosselin et al., 1976).

- Inhalation of DCP at high vapor concentrations resulted in gasping, refusal to breathe, coughing, substernal pain and extreme respiratory distress at vapor concentrations over 1,500 ppm (Gosselin et al., 1976).

- Venable et al. (1980) studied 64 male workers exposed to three carbon compounds including DCP to determine if fertility was adversely affected. The exposed study population was divided into $\leq$5 years exposure and >5 years exposure. Sperm counts and percent normal sperm forms were the major variables evaluated. Although the study participation rate for the exposed group was only 64%, no adverse effects on fertility were observed.

Animals

Short-term Exposure

- DCP is moderately toxic via single-dose oral administration. A technical product containing 92% cis-/trans-DCP was administered by gavage as a 10% solution in corn oil to rats. The oral LD_{50}s in male and female rats were 713 and 740 mg/kg, respectively (Torkelson and Oyen, 1977). In another study, the oral LD_{50} in the mouse for both males and females was 640 mg/kg (Toyoshima et al., 1978).

Dermal/Ocular Effects

- The percutaneous LD_{50}s for male and female mice dosed with DCP were greater than 1,211 mg/kg (Toyoshima et al., 1978).

- The percutaneous administration of DCP in rabbits (3 g/kg) resulted in mucous nasal discharge, depressed respiration and decreased body movements. The LD_{50} by this route was 2.1 g/kg (Torkelson and Oyen, 1977).

- Primary eye irritation and primary dermal irritation studies in rabbits indicated that DCP causes severe conjunctival irritation, moderate transient corneal injury and slight skin erythema/edema. Eye irritation was reversible 8 days post-instillation. The dermal LD_{50} in rabbits was 504 mg/kg (Dow, 1978).

Long-term Exposure

- Rats, guinea pigs, rabbits and dogs were exposed to 4.5 or 13.6 mg/m^3 DCP in air for 7 hours per day, and 5 days per week for 6 months.

The only effect noted was slight cloudy swelling of renal tubular epithelium in male rats exposed to the high dose (Torkelson and Oyen, 1977).

- Fischer 344 rats and CD-1 albino mice were exposed to Telone II (Production Grade) by inhalation exposure, 6 hours per day for 13 weeks at concentrations of 11.98, 32.14, or 93.02 ppm. Gross pathology revealed an increased incidence of kidney discoloration in the treated male rats relative to the control group. The significance of this lesion is unknown (Coate et al., 1979).

- Solutions of Telone (78.5% DCP) in propylene glycol were administered by gavage to 10 rats/sex/dose for six days per week for a period of 13 weeks. The dose levels were 1, 3, 10 or 30 mg/kg/day. The control groups were given propylene glycol. The daily administration of DCP to rats by stomach intubation up to a dosage of 30 mg/kg/day did not result in any major adverse effects. No significant effects on body weight, food consumption, hematology and histopathology were noted. However, at the 10 and 30 mg/kg/day doses, the relative weight of the kidney of males was higher than controls. The authors conclude that the no-toxic-effect level for DCP was between 3 and 10 mg/kg/day. The actual No-Observed-Adverse-Effect-Level (NOAEL) was 3 mg/kg/day (Til et al, 1973). This is the only study that can be used to develop a reference dose. However, because the design does not ideally address drinking water, a modifying factor will be used.

- The National Toxicology Program (NTP, 1985) evaluated the chronic toxicity and carcinogenicity of Telone II in rats and mice. These studies utilized Telone II fumigant containing approximately 89% cis- and trans-DCP. Groups of 52 male and female F344/N rats (doses 0, 25 or 50 mg/kg) and 50 male and female B6C3F$_1$ mice (doses 0, 50 or 100 mg/kg) were gavaged with Telone II in corn oil, 3 days per week up to 104 weeks. Ancillary studies were conducted in which dose groups containing five male and female rats were killed after receiving Telone II for 9, 16, 21, 24 or 27 months. Toxic effects (noncarcinogenic) included basal cell or epithelial hyperplasia of the forestomach of rats and mice at all treatment levels of DCP. Epithelial hyperplasia of the urinary bladder of mice occurred at both treatment levels in males and females. Kidney hydronephrosis also occurred in mice. The study in male mice was considered inadequate due to the deaths of vehicle control animals. Many chronic toxicity parameters (hematology/ clinical chemistry) were not determined. The DCP used in the NTP study had a different stabilizer from the current Telone II.

- Scott et al. (1987) exposed groups of male and female B6C3F1 mice (70 animals/sex/exposure concentration) to vapors of Telone II* soil fumigant for 6 hours/day, 5 days/week for up to 24 months at 0, 5, 20 or 60 ppm. Urinary bladder effects including hyperplasia of bladder epithelium were noted in both sexes at 20 and 60 ppm. Hypertrophy and hyperplasia of the nasal respiratory mucosa were observed in most 60 ppm exposed mice of both sexes and in 20 ppm exposed females. Hyperplasia of the epithelial lining of the nonglandular portion of the stomach was observed in 60 ppm exposed males.

- Lomax et al. (1987) exposed groups of 70 male and female Fischer 344 rats to vapors of Telone II* soil fumigant for 6 hours/day, 5 days/week for up to 24 months at targeted concentrations of 0, 5, 20 or 60 ppm. The NOAEL was 20 ppm. The highest dose caused histopathological changes in nasal tissue as well as a decrease in body weight gain during the first year of this study. Males and females exposed to 60 ppm showed decreased thickness and erosions of the nasal epithelium as well as minimal submucosa fibrosis.

Reproductive Effects

- Groups of male and female Wistar rats were exposed to technical D-D at 0, 64, 145 and 443 mg/m^3 (0, 14, 12 or 94 ppm) for 5 days per week over 10 weeks. Male mating indices, fertility indices and reproductive indices were not affected by D-D exposure. No gross morphological changes were seen in sperm. Female mating, fertility and other reproductive indices were normal. Litter sizes and weights were normal and pup survival over 4 days was not influenced by exposure (Clark et al., 1980).

- Breslin et al. (1987) exposed by inhalation groups (F_0) of 30 males and 40 females for 10 weeks, 6 hours/day, 5 days/week to Telone II* at concentrations of 0, 10, 30 and 90 ppm prior to breeding. Exposure was increased to 7 days/week during breeding at weeks 11 to 13. Exposure of F_1 male and female parents to Telone II* began after weaning (approximately week 32 of the study) and continued for 12 weeks (5 days/week and 6 hours/day). The NOAEL for reproductive effects in the study was ≥90 ppm, the highest dose tested. Conception indices of females were somewhat reduced in the F_1 and F_2 generations. At 90 ppm, both males and females developed hyperplasia of respiratory epithelium and focal degeneration of olefactory tissue. Decreased body weight was observed in males and females exposed to 90 ppm.

Developmental Effects

- Hanley et al. (1987) investigated the effects of inhalation exposure to DCP on fetal development in rats. Pregnant Fischer 344 rats were exposed to 0, 20, 60 and 120 ppm DCP for 6 hr/day during gestation days 6 to 15. Maternal body weight gain was depressed in all of the DCP-exposed rats in a dose-related manner. Therefore, the Lowest-Observed-Adverse-Effect Level (LOAEL) for this effect was 20 ppm DCP. There was also significant depression of feed consumption in all exposed rats, along with decreases in water consumption in rats exposed to 120 ppm DCP. At 120 ppm there were significant increases in relative kidney weights and decreases in absolute liver weights in all exposed rats. There was a statistical increase in the incidence of delayed ossification of the vertebral centra of rats exposed to 120 ppm DCP. This effect is of little toxicological significance due to maternal toxicity observed at 120 ppm DCP.

- Hanley et al. (1987) also studied the effects of inhalation exposure to DCP on fetal development in rabbits. Pregnant New Zealand White rabbits were exposed to 0, 20, 60 or 120 ppm DCP for 6 hr/day during

gestation days 6 through 18. In rabbits, evaluation of maternal weight gain over the entire exposure period indicated significant exposure-related decreases in both the 60- and 120-ppm groups. Therefore, the NOAEL was 20 ppm DCP. Statistically significant decreases in the incidence of delayed ossification of the hyoid and presence of cervical spurs among the exposed group were considered within normal variability in rabbits.

Mutagenicity

- Tests of commercial formulations containing DCP (DeLorenzo et al., 1975; Flessel, 1977; Neudecker et al., 1977; Brooks et al., 1978; Sudo et al., 1978; Stolzenberg and Hine, 1980), a mixture of pure cis-DCP and trans-DCP (DeLorenzo et al., 1975), and pure cis-DCP (Brooks et al, 1978) were positive in the Salmonella typhimurium strains TA1535 and TA100 with and without metabolic activation. These results indicate that DCP acts by base-pair substitution and is a direct acting mutagen.

- DCP may be a mutagen that acts via frame shift mutation since studies by DeLorenzo et al. (1975) reported positive results in TA1978 (with and without metabolic activation) for a commercial mixture of DCP and a mixture of pure cis- and trans-DCP.

- A commercial mixture of DCP and pure cis-DCP were also positive with and without metabolic activation in Salmonella typhimurium strain TA98 (Flessel, 1977; Sudo et al., 1978; Brooks et al., 1978).

- Sudo et al. (1978) tested DCP in a reverse mutation assay with E. coli B/r Wp2 with negative results.

- DCP was negative for reverse mutation in the mouse host-mediated test with S. typhimurium G46 in studies by Shirasu et al. (1976) and Sudo et al. (1978).

Carcinogenicity

- F344 rats of each sex were gavaged with Telone II in corn oil at doses of 0, 25 and 50 mg/kg/day for 3 days per week. A total of 77 rats/sex were used for each dose group (52 animals/sex/group were dosed for 104 weeks in the main oncogenicity study, and an ancillary study where 5 animals/sex/ group were sacrificed after 9, 16, 21, 24 and 27 months' exposure to DCP). No increased mortality occurred in treated animals. Neoplastic lesions associated with Telone II included squamous cell papillomas of the forestomach (male rats: 1/52; 1/52; 9/52; female rats: 0/52; 2/52; 3/52), squamous cell carcinomas of the forestomach (male rats: 0/52; 0/52; 4/52) and neoplastic nodules of the liver (male rats: 1/52; 6/52; 7/52). The increased incidence of forestomach tumors was accompanied by a positive trend for forestomach basal cell hyperplasia in male and female rats of both treated groups (25 and 50 mg/kg/day). The highest dose level tested in rats (50 mg/kg/day) approximated a maximum tolerated dose level (NTP, 1985).

- B6C3F$_1$ mice of each sex were gavaged with Telone II in corn oil at doses of 0, 50 and 100 mg/kg/day for 104 weeks. A total of 50 mice/sex were used for each dose group. Due to excessive mortality in control male mice from myocardial inflammation approximately 1 year after the initiation of the study, conclusions pertaining to oncogenicity were based on concurrent control data and NTP historical control data. Neoplastic lesions associated with the administration of Telone II included squamous cell papillomas of the forestomach (female mice: 0/50; 1/50; 2/50), squamous cell carcinomas of the forestomach (female mice: 0/50; 0/50; 2/50), transitional cell carcinomas of the urinary bladder (female mice: 0/50; 8/50; 21/48), and alveolar/bronchiolar adenomas (female mice: 0/50; 3/50; 8/50). The increased incidence of forestomach tumors was accompanied by an increased incidence of stomach epithelial cell hyperplasia in males and females at the highest dose level tested (100 mg/kg/day), and the increased incidence of urinary bladder transitional cell carcinoma was accompanied by a positive trend for bladder hyperplasia in male and female mice of both treated groups (50 and 100 mg/kg/day). Incidences of neoplasms were not significantly increased in male mice (NTP, 1985).

- Thirty female Ha:ICR Swiss mice received weekly subcutaneous injections of cis-DCP. The dose was 3 mg DCP/mouse in 0.05 mL trioctanoin delivered to the left flank. After 77 weeks, there was an increased incidence of fibrosarcomas at the site of injection. Six of the 30 exposed mice developed the tumors. There were no similar lesions in the controls (Van Duuren, 1979).

- Scott et al. (1987) exposed groups of male and female B6C3F1 mice (70 animals/sex/dose) to vapors of Telone II® for 6 hours/day, 5 days/week for up to 24 months at 0, 5, 20 or 60 ppm. The only tumorigenic effect was an increased incidence in benign lung tumors (bronchioloalveolar adenomas) in the 60 ppm exposed males. There were no tumorigenic effects in the lower-dose males or at any of the doses in females.

- Lomax et al. (1987) exposed Fisher 344 rats (70 rats/sex/dose) to vapors of Telone II® (0, 5, 20 and 60 ppm). The two year exposure by inhalation did not result in increases in tumor incidence.

V. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

Health Advisories (HAs) are generally determined for one-day, ten-day, longer-term (up to 7 years) and lifetime exposures if adequate data are available that identify a sensitive noncarcinogenic end point of toxicity. The HAs for noncarcinogenic toxicants are derived using the following formula:

$$HA = \frac{(NOAEL \text{ or } LOAEL) \times (BW)}{(UF) \times (\_\_\_ L/day)} = \_\_\_ mg/L \ (\_\_\_ ug/L)$$

where:

NOAEL or LOAEL = No- or Lowest-Observed-Adverse-Effect Level in mg/kg bw/day.

BW = assumed body weight of a child (10 kg) or an adult (70 kg).

UF = uncertainty factor (10, 100, 1,000 or 10,000), in accordance with EPA or NAS/ODW guidelines.

___ L/day = assumed daily water consumption of a child (1 L/day) or an adult (2 L/day).

One-day Health Advisory

There are not sufficient data to derive a One-day Health Advisory value for DCP. It is recommended that the Longer-term HA value for a 10-kg child (30 ug/L, calculated below) be used at this time as a conservative estimate of the One-day HA value.

Ten-day Health Advisory

There are not sufficient data to derive a Ten-day HA value for DCP. It is recommended that the Longer-term HA value for a 10-kg child (30 ug/L, calculated below) be used as a conservative estimate of the Ten-day HA value.

Longer-term Health Advisory

The Til et al. (1973) 13 weeks subchronic gavage study in rats has been selected to serve as the basis for calculating the Longer-term HA for DCP. This study resulted in a LOAEL of 10.0 mg/kg/day based on increased relative kidney weight in males. No adverse effects were noted at the next lowest dose (3.0 mg/kg/day). Therefore, the NOAEL is 3.0 mg/kg/day.

Based on the NOAEL of 3.0 mg/kg/day determined in this study, the Longer-term HAs are calculated as follows:

For a 10-kg child:

$$\text{Longer-term HA} = \frac{(3.0 \text{ mg/kg/day})\ (10 \text{ kg})}{(100)\ (10)\ (1 \text{ L/day})} = 0.03 \text{ mg/L } (30 \text{ ug/L})$$

where:

3.0 mg/kg/day = NOAEL based on the absence of increased relative kidney weights in rats.

10 kg = assumed body weight of a child.

100 = uncertainty factor, chosen in accordance with EPA or NAS/ODW guidelines for use with a NOAEL from an animal study.

10 = modifying factor, selected since this was the only useful gavage study available and classified as supplementary data. Also there were considerable toxicological data gar

1 L/day = assumed daily water consumption of a child.

For a 70-kg adult:

$$\text{Longer-term HA} = \frac{(3.0 \text{ mg/kg/day})\ (70 \text{ kg})}{(100)\ (10)\ (2 \text{ L/day})} = 0.105 \text{ mg/L } (100 \text{ ug/L})$$

where:

3.0 mg/kg/day = NOAEL based on the absence of increased relative kidney weights in rats.

70 kg = assumed body weight of an adult.

100 = uncertainty factor, chosen in accordance with EPA or NAS/ODW guidelines for use with a NOAEL from an animal study.

10 = modifying factor, selected since this was the only useful gavage study available and classified as supplementary data. Also there were considerable toxicological data gaps.

.2 L/day = assumed daily water consumption of an adult.

Lifetime Health Advisory

The Lifetime HA represents that portion of an individual's total exposure that is attributed to drinking water and is considered protective of noncarcinogenic adverse health effects over a lifetime exposure. The Lifetime HA is derived in a three-step process. Step 1 determines the Reference Dose (RfD), formerly called the Acceptable Daily Intake (ADI). The RfD is an estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime, and is derived from the NOAEL (or LOAEL), identified from a chronic (or subchronic) study, divided by an uncertainty factor(s). From the RfD, a Drinking Water Equivalent Level (DWEL) can be determined (Step 2). A DWEL is a medium-specific (i.e., drinking water) lifetime exposure level, assuming 100% exposure from that medium, at which adverse, noncarcinogenic health effects would not be expected to occur. The DWEL is derived from the multiplication of the RfD by the assumed body weight of an adult and divided by the assumed daily water consumption of an adult. The Lifetime HA is determined in Step 3 by factoring in other sources of exposure, the relative source contribution (RSC). The RSC from drinking water is based on actual exposure data or, if data are not available, a value of 20% is assumed. If the contaminant is classified as a Group A or B carcinogen, according to the Agency's classification scheme of carcinogenic potential, then caution should be exercised in assessing the risks associated with lifetime exposure to this chemical. For Group C carcinogens, an additional safety factor of 10 is added to the DWEL.

The Lifetime HA for a 70-kg adult has been determined on the basis of the study in rats by Til et al. (1973), as described above.

Using the NOAEL of 3.0 mg/kg/day, as determined in that study, the DWEL is calculated as follows:

Step 1: Determination of the Reference Dose (RfD)

$$RfD = \frac{(3.0 \text{ mg/kg/day})}{(1{,}000)\ (10)} = 0.0003 \text{ mg/kg/day}$$

where:

3.0 mg/kg/day = NOAEL based on the absence of increased relative kidney weights in rats.

1,000 = uncertainty factor, chosen in accordance with EPA or NAS/ODW guidelines for use with a NOAEL from an animal study of less-than-lifetime duration.

10 = modifying factor selected since this was the only useful gavage study available and classified as supplementary data. Also there were considerable toxicological data gaps.

Step 2: Determination of the Drinking Water Equivalent Level (DWEL)

$$DWEL = \frac{(0.0003 \text{ mg/kg/day})\ (70 \text{ kg})}{(2 \text{ L/day})} = .011 \text{ mg/L } (10 \text{ ug/L})$$

where:

0.0003 mg/kg/day = RfD.

70 kg = assumed body weight of an adult.

2 L/day = assumed daily water consumption of an adult.

Step 3: Determination of the Lifetime Health Advisory

Lifetime HAs are not recommended for Group A or B carcinogens. DCP is a Group B2, probable human carcinogen. The estimated cancer risk associated with lifetime exposure to drinking water containing DCP at 10 ug/L is approximately 5.0×10^{-5}. This estimate represents the upper 95% confidence limit using the linearized multistage model. The actual risk is unlikely to exceed this value.

Evaluation of Carcinogenic Potential

- DCP may be classified as a B2, probable human carcinogen based on sufficient evidence of tumor production in two rodent species and two routes of administration.

- Data on an increased incidence of squamous cell papilloma or carcinoma of the forestomach in rats exposed to DCP (NCI, 1985) were used for a quantitative assessment of cancer risk due to DCP. Based on the data from this study and using the linearized multistage model, a carcinogenic potency factor (q_1^*) for humans of 1.75×10^{-1} $(mg/kg/day)^{-1}$ was calculated.

- The drinking water concentrations corresponding to increased lifetime cancer risks of 10^{-4}, 10^{-5} and 10^{-6} (one excess cancer per one million population) for a 70-kg adult consuming 2 L/day are 20 ug/L, 2 ug/L and 0.2 ug/L, respectively.

- The forestomach tumor data in male rats used to calculate the q_1^* value (NCI, 1985) consisted of the 2-year study data excluding the ancillary studies data. The ancilliary studies involved serial sacrifice of animals (at 9, 16, 21, 24 and 27 months). It is not appropriate to include these data in the lifetime predictive model used (multistage).

- For comparison purposes, drinking water concentrations associated with an excess risk of 10^{-6} were 0.2 ug/L, 3.6 mg/L, 0.03 ug/L and 0.004 ug/L for the one-hit, Weibull, probit and logit models, respectively.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- The ACGIH recommended 1 ppm (5 mg/m^3) as a Threshold Limit Value for DCP (Clayton and Clayton, 1981).

VII. ANALYTICAL METHODS

- No specific methods have been published by U.S. EPA for analysis of DCP in water. However, EPA Method 524.2 (U.S. EPA, 1986d) and EPA Method 502.2 (USEPA, 1986e) both for volatile organic compounds in water should be suitable for analysis of DCP. Both are standard purge and trap capillary column gas chromatographic techniques. While an estimated detection limit has not been calculated for the two isomers of 1,3-dichloropropene, work done with 1,1-dichloropropene would indicate a range for 1,3-DCP of 0.02 to 0.05 ug/L.

VIII. TREATMENT TECHNOLOGIES

- There are no specific publications on treatment of 1,3-DCP. However, adequate treatment by granular activated carbon (GAC) should be possible. Freundlich carbon absorption isotherms for DCP indicate reasonably high adsorption capacity (U.S. EPA, 1980).

IX. REFERENCES

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