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DICHLOROMETHANE

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.

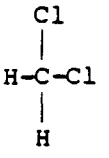
Health Advisories are developed for One-day, Ten-day, Longer-term (approximately 7 years, or 10% of an individual's lifetime) and Lifetime exposures based on data describing noncarcinogenic end points of toxicity. Health Advisories do not quantitatively incorporate any potential carcinogenic risk from such exposure. 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.

This Health Advisory is based upon information presented in the Office of Health and Environment Assessment Criteria Document (CD) for Dichloromethane (U.S. EPA, 1985a). The HA and CD formats are similar for easy reference. Individuals desiring further information on the toxicological data base or rationale for risk characterization should consult the CD. The CD is available for a fee from the National Technical Information Service, U.S. Department of Commerce, 5285 Port Royal Rd., Springfield, VA 22161, PB85 191559. The toll-free number is (800) 336-4700; in the Washington, D.C. area: (703) 487-4650.

II. GENERAL INFORMATION AND PROPERTIES

CAS No. 75-09-2

Structural Formula



Synonyms

- Methylene chloride, methylene dichloride, methylene bichloride, DCM

Uses

- Solvent for insecticides, paints, varnish and paint removers and in food processing; degreasing and cleaning fluids.

Properties (Verschueren, 1977; Windholtz, 1983)

Chemical Formula	CH_2Cl_2
Molecular Weight	84.94
Physical State	Colorless liquid
Boiling Point	40°C (760 mm Hg)
Melting Point	-95 to -97°C
Density	1.3255 (20/4°C)
Vapor Pressure	349 mm Hg (20°C)
Water Solubility	20 g/L (20°C)
Log Octanol/Water Partition Coefficient	--
Odor Threshold	--
Taste Threshold	--
Conversion Factor	--

Occurrence

- Dichloromethane (DCM) is a synthetic chemical with no known natural sources.

- Production of DCM was approximately 600 million lbs in 1983 (U.S. ITC, 1984).

- The major sources of DCM released to the environment are from its industrial uses where the majority of all DCM produced is released. Most of the releases occur to the atmosphere by evaporation. However, large amounts of DCM are disposed of by burial in landfills or dumping on the ground or into sewers. Because DCM is involved in industrial operations performed nationwide, releases occur in all urban areas. Releases of DCM during its production are relatively minor in comparison to releases during its use.

- Dichloromethane released to the air is degraded in a matter of a few days. Dichloromethane released to surface waters migrates to the atmosphere in a few days or weeks where it also degrades. Volatilization is the major transport process for its removal from aquatic systems (U.S. EPA, 1979). Dichloromethane which is released to the land does not sorb onto soil and migrates readily to ground water where it is expected to remain for months to years. Dichloromethane, unlike some other chlorinated compounds, does not bioaccumulate in individual animals or food chains.

- Because of the large and dispersed releases, DCM occurs widely in the environment. It is ubiquitous in the air with levels in the ppt range and is a common contaminant in ground and surface waters with higher levels found in ground water.

- Very limited information is available on the occurrence of dichloromethane in food. Dichloromethane has been reported to occur in fish. It is used as an extraction solvent for the decaffination of coffee and other food processing operations. Low levels of DCM have been reported to occur in some foods from these operations.

- The major sources of exposure to DCM are from contaminated water. Air and food are only a minor sources (U.S. EPA, 1980c).

III. PHARMACOKINETICS

Absorption

- Dichloromethane is expected to be absorbed completely when ingested. A single oral dose of 1 or 50 mg/kg ^{14}C-DCM administered to male rats (3/dose) was exhaled as unchanged DCM (12.3 or 72.1%, respectively) within 48 hours (McKenna and Zempel, 1981).

Distribution

- Tissue distribution after administration of 1 or 50 mg/kg of ^{14}C-DCM in water by gavage to male rats (3/dose) was measured by McKenna and Zempel (1981). The highest concentration of radioactivity was present in liver and the lowest in fat, 48 hours after either dose.

Metabolism

- The major metabolites of DCM are carbon monoxide and carbon dioxide. McKenna and Zempel (1981) studied the metabolism of ^{14}C-DCM after gavage administration to groups of three male Sprague-Dawley rats dosed at 1 or 50 mg/kg. They metabolized about 88 or 28% of the dose, respectively. The major metabolites exhaled after 48 hours were carbon monoxide (30.9 and 11.9% of the 1 or 50 mg/kg doses, respectively) and carbon dioxide (35.0 and 6.3% of the 1 or 50 mg/kg doses, respectively).

Excretion

- Metabolites of DCM are excreted in urine. McKenna and Zempel (1981) reported that, in rats given 1 or 50 mg/kg ^{14}C-DCM, 4.52 ±0.05% or 1.96 ±0.05% of the dose, respectively, was excreted in the urine within 48 hours. The fecal elimination of DCM after oral or intraperitoneal administration of DCM is low (<1.0%) (DiVincenzo and Hamilton, 1975; McKenna and Zempel, 1981).

IV. HEALTH EFFECTS

Humans

- Bonventre et al. (1977) described a fatal intoxication with DCM which was being used as a paint remover. Postmortem examination revealed the presence of DCM in the liver (14.4 mg/100 g tissue), blood (51 mg/dL or 510 mg/L) and brain (24.8 mg/100 g tissue). The carboxyhemoglobin content was 3% saturated.

Animals

Short-term Exposure

- Oral LD_{50}s for DCM were reported as 1,987 mg/kg for mice and 2,121 mg/kg for rats (Kimura et al. 1971; Aviado et al. 1977).

- Kimura et al. (1971) administered single oral doses of DCM to young adult Sprague-Dawley rats and determined that an approximate dose of 1.3 g/kg body weight was the lowest dose to induce the first observable signs of toxicity (dyspnea, ataxia, cyanosis and/or coma).

Long-term Exposure

- Bornmann and Loeser (1967) administered DCM in drinking water at 2.25 g/18L (or 125 mg/L) to 30 male and 30 female Wistar rats for 13 weeks. This is equivalent to a dose of about 15 mg/kg/day assuming that 10 mL of water is consumed daily. The animals were examined for changes in behavior, body weight, blood and urine chemistries, reproductive function, organ to body weight ratios and histology. No treatment-related effects were observed, even though some rats may have consumed as much as 250 mg DCM (36.6 mg/kg/day) during this

experiment. The urine albumin test was frequently positive; however, the authors did not attach any biological significance to this observation. From this study, a NOAEL of 125 mg/kg/day was identified.

- Hazelton Labs (1982) reported on the toxicity and carcinogenicity of DCM in a chronic two-year drinking water study in Fischer 344 rats. Two control groups (85 and 50 rats/sex/group) received deionized drinking water. Four groups of animals (85 rats/sex/group) were given DCM in drinking water at target doses of 5, 50, 125 and 250 mg/kg/day. A high-dose recovery group (25 rats/sex) was given DCM in drinking water at a target dose of 250 mg/kg/day for the initial 78 weeks and deionized drinking water subsequently for the remainder of the study. At 26, 52 and 78 weeks of treatment, there were incremental sacrifices of 5, 10 or 20 rats/sex/group, respectively. At 104 weeks of exposure, all survivors were sacrificed. Survival, body weight gains, total food consumption, water consumption, clinical observations, ophthalmoscopic findings, clinical pathology, absolute and relative organ weights and gross and microscopic pathology were examined to evaluate any compound-related effects. The dose of 5 mg/kg was identified as the no-effect level based on the absence of effects on body weight, hematological parameters and histopathological changes in the liver (incidence of foci/areas of cellular alteration and/or fatty changes).

Developmental Effects

- No positive conclusion can be drawn regarding the potential for developmental effects of DCM.

- Maternal exposure of rats and mice to DCM (4337 mg/m^3) on days 6 through 15 of gestation was associated with soft tissue abnormalities in the offspring of rats and skeletal changes in the offspring of both rats and mice (Schwetz et al., 1975).

- Other workers have found no increased incidence of gross external, skeletal or soft tissue anomalies in offspring after maternal exposure of rats to DCM at 15,615 mg/m^3 (6 hours/day, 7 days/wk) before and during gestation. (Hardin and Manson, 1980).

Mutagenicity

- DCM has been reported to be mutagenic in several bacterial and yeast test systems, as well as in mammalian test systems. DCM was also reported to be positive in a mammalian transformation test (U.S. EPA, 1985a).

Carcinogenicity

- In a pulmonary tumor response assay, DCM administered intraperitoneally did not produce an increased incidence of lung tumors in mice (Theiss et al. 1977).

- An inhalation bioassay conducted in male and female F344/N rats and B6C3F$_1$ mice indicated clear evidence of carcinogenicity in male and female mice as shown by increased incidences of lung (alveolar/bronchiolar adenoma and/or carcinoma) and liver (hepatocellular adenoma and carcinoma combined) tumors (NTP, 1985, as cited in U.S. EPA, 1985c). Some evidence of carcinogenicity in male rats and sufficient or clear evidence of carcinogenicity in female rats was indicated by an increased incidence of benign neoplasms of the mammary gland. These animals were exposed at concentrations of 0, 1,000, 2,000 and 4,000 ppm for rats and 0, 2,000 and 4,000 ppm for mice, 6 hours/day, 5 days/week for 102 weeks.

- Hazelton Laboratories (1982) studied the carcinogenicity of DCM in a chronic two-year drinking water study in Fischer 344 rats, using the protocol as described under longer-term exposure. Hepatic histological alteration detected in the 50 to 250 mg/kg/day dose groups (both sexes) included an increased incidence of foci/areas of cellular alteration. Fatty liver changes were detected in the 125 and 250 mg/kg/day groups after 78 and 104 weeks of treatment. The authors stated that DCM did not induce carcinogenicity under the conditions of the study.

- The U.S. EPA (1985b) performed an independent assessment of the data from the Hazelton Laboratories (1982) study and determined that incidences of hepatic neoplastic nodules and carcinomas (combined in females exposed to 50 mg/kg/day (4.8%), 250 mg/kg/day (7.1%) and 250 mg/kg/day, recovery group (8.0%) were significantly ($P<0.05$) higher than that in matched controls (0%). No significant increase in liver tumors was evident in any of the male dose groups. The U.S. EPA (1985b) considered data on historical control values and concluded that the 250 mg/kg/day dose was borderline for carcinogenicity in Fischer 344 rats.

V. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

Health Advisories (HAs) are generally determined for One-day, Ten-day, Longer-term (approximately 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:

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

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 or 1,000), in accordance with 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

The study by Kimura et al. (1971) has been selected to serve as the basis for the One-day HA for the 10 kg child because no other acute oral studies of appropriate duration or design were located in the literature. This study identified a LOAEL in young adult Sprague-Dawley rats on the basis of the first observable gross signs of toxicity (i.e., dyspnea, ataxia, cyanosis and/or coma) following administration of a single oral dose of DCM by gavage. The authors implied that multiple dose levels were administered to define dose-response, although details were not reported. The calculations for a One-day HA for a 10-kg child are given below:

$$\text{One-day HA} = \frac{1{,}326 \text{ mg/kg/day}) \text{ (10 kg)}}{(1{,}000) \text{ (1 L/day)}} = 13.3 \text{ mg/L (13,300 ug/L)}$$

where:

1,326 mg/kg/day = LOAEL, based on the first observable gross signs of toxicity in rats.

10 kg = assumed body weight of a child.

1,000 = uncertainty factor, chosen in accordance with ODW/NAS guidelines for use with a LOAEL from an animal study.

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

Ten-day Health Advisory

The study by Bornmann and Loeser (1967) in which DCM was administered in drinking water at 125 mg/L to Wistar rats for 13 weeks. has been selected to serve as the basis for the Ten-day HA for the 10-kg child because it was the most comprehensive short-term oral toxicity study located.

The Ten-day HA for a 10 kg child is calculated as follows:

$$\text{Ten-day HA} = \frac{(15 \text{ mg/kg/day})(10 \text{ kg})}{(100) \text{ (1 L/day)}} = 1.5 \text{ mg/L (1,500 ug/L)}$$

where:

15 mg/kg/day = NOAEL, based on absence of effects on body weight gain, blood and urine chemistries, reproductive function, organ/body weight ratios, or histopathological changes in Wistar rats.

10 kg = assumed body weight of a child.

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

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

Longer-term Exposure

There were no suitable data available from which to calculate Longer-Term Health Advisories.

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 for synthetic organic chemicals and a value of 10% is assumed for inorganic chemicals. If the contaminant is classified as a Group A or B carcinogen, according to the Agency's classification scheme of carcinogenic potential (U.S. EPA, 1986), then caution should be exercised in assessing the risks associated with lifetime exposure to this chemical.

Dichloromethane may be classified in Group B2: Probable Human Carcinogen, according to EPA's guidelines for assessment of carcinogenic risk (U.S. EPA, 1986). Because of this, caution must be exercised in making a decision on how to deal with possible lifetime exposure to this substance. The risk manager must balance this assessment of carcinogenic potential against the likelihood of occurrence of health effects related to non-carcinogenic end-points of toxicity. In order to assist the risk manager in this process, drinking water concentrations associated with estimated excess lifetime cancer risks over the range of one in ten thousand to one in a million for the 70-kg adult, drinking 2 liters of water per day, are provided in the following section. In addition, in this section, a Drinking Water Equivalent Level (DWEL) is derived. A DWEL is defined as the medium-specific (in this case, drinking water) exposure which is interpreted to be protective for non-carcinogenic end-points of toxicity over a lifetime of exposure. The DWEL is determined for the 70-kg adult, ingesting 2 liters of water per day. Also provided is an estimate of the excess cancer risk that would result if exposure were to occur at the DWEL over a lifetime.

Neither the risk estimates nor the DWEL take relative source contribution into account. The risk manager should do this on a case-by-case basis, considering the circumstances of the specific contamination incident that has occurred.

The study by Hazelton Laboratories (1982) is most appropriate from which to derive the DWEL because it is an oral chronic (two year) study that administered DCM in drinking water in multiple dose levels to rats. This is the most comprehensive chronic oral study available. There were sufficient numbers of animals in the dose groups and a dose-response was demonstrated. A NOAEL of 5 mg/kg/day was identified in this study.

The DWEL for a 70-kg adult is calculated as follows:

Step 1: Determination of the Reference Dose (RfD)

$$RfD = \frac{(5\ mg/kg/day)}{(100)} = 0.05\ mg/kg/day$$

where:

5 mg/kg/day = NOAEL based on the absence of liver and blood effects in rats.

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

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

$$DWEL = \frac{(0.05\ mg/kg/day)(70\ kg)}{(2\ L/day)} = 1.75\ mg/L\ (1,750\ ug/L)$$

where:

0.05 mg/kg/day = RfD.

70 kg = assumed body weight of an adult.

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

Step 3: Determination of the Lifetime Health Advisory

Dichloromethane is classified in Group B2: Probable Human Carcinogen. A Lifetime HA has not been calculated for DCM.

The estimated excess cancer risk associated with lifetime exposure to drinking water containing DCM at 1,750 ug/L is approximately 3.7×10^{-4}. This estimate represents the upper 95% confidence limit from extrapolations prepared by EPA's Carcinogen Assessment Group using the linearized, multistage model. The actual risk is unlikely to exceed this value, but there is considerable uncertainty as to the accuracy of risks calculated by this methodology.

Evaluation of Carcinogenic Potential

- IARC (1982) has classified DCM in group 3: Limited evidence of carcinogenicity in animals.

- Applying the criteria described in EPA's guidelines for assessment of carcinogenic risk (U.S. EPA, 1986), DCM may be classified in Group B2: Probable human carcinogen. This category is for agents for which there is inadequate evidence from human studies and sufficient evidence from animal studies.

- More recently, EPA's CAG (U.S. EPA, 1985c) estimated that the upper-bound incremental unit carcinogenic risk for drinking water containing 1 ug/L DCM for a lifetime was 2.1×10^{-7} $(ug/L)^{-1}$. This risk estimate was the mean of the derived carcinogenic risk estimates based on the finding of liver tumors (not based on lung tumors) in the NTP (1985) draft inhalation study in female mice and the suggestively positive finding of liver tumors in the Hazelton (1982) unpublished ingestion study in male mice. Since the extrapolation model is linear at low doses, additional lifetime cancer risk is directly proportional to the water concentration of DCM. Thus, levels of 10^{-4}, 10^{-5} and 10^{-6} are 0.48, 0.048 and 0.005 mg/L, respectively.

- The linear multistage model is only one method of estimating carcinogenic risk. Using the 10^{-6} risk level, the following comparisons in micrograms/L may be made: Multistage, 4.8; Probit, 74,000; Logit, 4,000; Weibull, 10. Each model is based on differing assumptions. No current understanding of the biological mechanism of carcinogenesis is able to predict which of these models is more accurate than another. While recognized as statistically alternative approaches, the range of risks described by using any of these modeling approaches has little biological significance unless data can be used to support the selection of one model over another. In the interest of consistency of approach and in providing an upper bound on the potential cancer risk, the Agency has recommended use of the linearized multistage approach.

VI. OTHER CRITERIA, GUIDANCE AND STANDARDS

- ACGIH (1984) has recommended a time-weighted average threshold limit value (TWA-TLV) of 100 ppm (360 mg/m^3) in the absence of occupational exposure to carbon monoxide and is based upon experimental data obtained from nonsmoking males at rest. A short-term exposure level (STEL) of 500 ppm is also recommended.

- The Occupational Health and Safety Administration (OSHA, 1979) has established occupational exposure standards as follows: an eight-hour time-weighted-average (TWA) of 1,737 mg/m^3; an acceptable ceiling concentration of 3,474 mg/m^3; and an acceptable maximum peak above the ceiling of 6,948 mg/m^3 (five minutes in any two hours).

- Due to the metabolic formation of carboxyhemoglobin and the additive toxicity with carbon monoxide, the National Institute of Occupational Safety and Health (NIOSH, 1976) has recommended a ten-hour TWA exposure limit of 261 mg/m^3 and a 1737 mg/m^3 peak (15 minute sampling), in the presence of carbon monoxide concentrations less than or equal to 9.9 mg/m^3 (TWA). Proportionately lower levels of DCM are required in the workplace when carbon monoxide concentrations greater than 9.9 mg/m^3 are present.

- Based on noncarcinogenic risks, a water quality criterion of 12.4 mg/L is the acceptable concentration of DCM in drinking water (U.S. EPA, 1980a). This calculation was performed by the U.S. EPA as part of the overall process for developing a U.S. EPA Water Quality Criteria for halomethanes as a group and uses a limit of 200 ppm (694 mg/m^3) for protection against excessive carboxy-hemaglobin formation. In that calculation, the EPA assumed that the average person consumes approximately two liters of water and eats 6.5 g of contaminants in fish and seafood per day, and that the estimated coefficient of absorption via inhalation versus ingestion is 0.5.

- The original U.S. EPA Suggested-No-Adverse-Response-Levels (SNARLs, now referred to as Health Advisories) for DCM were set at 13, 1.5 and 0.150 mg/L in drinking water for One-day, Ten-day and Longer-term exposures, respectively (U.S. EPA, 1980b). The U.S. EPA-SNARLs were established for a 10 kg body weight child and did not consider the possible carcinogenic risk that may result from exposure to a chemical.

- The NAS (1980) calculated one-day and seven-day NAS-SNARLs for DCM in drinking water based on the minimal-effect acute oral dose in rats reported by Kimura et al. (1971). The NAS concluded that data on the no-effect dose do not exist. Using the 1 ml/kg (1.3 g/kg) minimal-effect acute oral dose in the rat, assuming two liters/day of drinking water as the only source (consumed by a 70 kg adult) and employing a safety factor of 1,000, the NAS (1980) calculated the one-day SNARL. Since no appropriate data were available for the seven-day SNARL, the one-day SNARL was divided by a factor of seven (days). However, the NAS (1980) erroneously reported a value of 35 mg/L for the one-day and 5 mg/L for the seven-day calculation. Re-examination of calculations indicated that the one-day and seven-day adult NAS-SNARLs should be 45.5 mg/L and 6.5 mg/L, respectively.

VII. ANALYTICAL METHODS

- Analysis of DCM is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water (U.S. EPA, 1985d). This method calls for the bubbling of an inert gas through the sample and trapping DCM on an adsorbant material. The adsorbant material is heated to drive off the DCM onto a gas chromatographic column. This method is applicable to the measurement of DCM over a concentration range of less than 1 to 1500 ug/L; however, measurement of DCM at low concentrations is difficult due to problems with contamination. Dichloromethane vapors readily penetrate tubing

during the purge/trap procedure. Confirmatory analysis for DCM is by mass spectrometry. (U.S. EPA 1985e). The detection limit for confirmation by mass spectrometry is 0.3 ug/L.

VIII. TREATMENT TECHNOLOGIES

- Limited information is available concerning the removal of dichloromethane from drinking water. However, evaluation of physical and chemical properties and some experimental data suggest that adsorption by granular activated carbon (GAC) and aeration are feasible technologies to remove this contaminant in drinking water supplies.

- Dobbs and Cohen (1980) developed adsorption isotherms for several organic chemicals, including DCM. This study reported that Filtrasorb® 300 exhibited adsorptive capacities of 1.3 mg and 0.09 mg DCM per gm carbon at equilibrium concentrations of 1,000 mg/L and 100 mg/L, respectively.

- Another study reported activated carbon usage of 3.9 lb/1,000 gal of treated water to maintain an effluent DCM concentration below 1 ug/L from a raw water influent concentration above 20 mg/L (ESE, 1982). This particular treatment scheme employed two activated carbon columns operating in series with extremely long empty bed contact time (262 minutes).

- The calculated Henry's Law constant for DCM is 2.5×10^{-3} atm-m^3/mole (ESE, 1982). In a bench-scale study, distilled water which was spiked with 225 ug/L of DCM was passed through a diffused air aerator. The results showed 82 percent reduction in DCM at an air-to-water ratio of 15:1 (Love, 1983). Dichloromethane will, therefore, be amenable to air stripping treatment. Actual field performance data, however, have not been reported for this compound.

- Air stripping is an effective, simple and relatively inexpensive process for removing DCM and other volatile organics from water. However, use of this process then transfers the contaminant directly to the air stream. When considering use of air stripping as a treatment process, it is suggested that careful consideration be given to the overall environmental occurrence, fate, route of exposure and various hazards associated with the chemical.

- Treatment technologies for the removal of DCM from water have not been extensively evaluated except on an experimental level. Selection of individual or combinations of technologies to attempt DCM reduction must be based on a case-by-case technical evaluation, and an assessment of the economics involved.

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