October 1994
(edited October 1994)

1,1-Dichloroethylene

Guideline

The maximum acceptable concentration (MAC) for 1,1-dichloroethylene in drinking water is 0.014 mg/L (14 µg/L).

Identity, Use and Sources in the Environment

1,1-Dichloroethylene (1,1-dichloroethene, vinylidene chloride, 1,1-DCE) is a clear, colourless liquid with a characteristic sweet odour, a boiling point of approximately 32°C, a high vapour pressure of 65.8 kPa at 20°C and a Henry's law constant of 0.19 atm•m^3/mol.[1,2] It is soluble in most organic solvents, and its solubility in water is 2500 mg/L at 21°C. It has a log octanol–water partition coefficient in the range 1.66 to 2.13, indicating that it is unlikely to have significant potential for bioaccumulation.[1,2]

1,1-DCE is not produced in Canada.[3] Imports — primarily of its polymer polyvinylidene chloride (PVDC) as latex formulations, film or resin, from the United States — ranged from a low of 2.06 kt in 1990 to a high of 2.87 kt in 1988.[3] PVDC is used primarily in the food packaging industry as cast and extruded film (Saran® and other brands) and as a barrier coating for paper, cellulose, polypropylene and other plastics. Extruded filaments of PVDC are also used in the textile industry for furniture and automotive upholstery, drapery fabric and outdoor furniture.[3] Evaporative emissions and effluents of 1,1-DCE resulting from applications in polymers are expected to be very low. 1,1-DCE is also used in the production of 1,1,1-trichloroethane and copolymers (with vinyl chloride or acrylonitrile).

Exposure

1,1-DCE has been detected only infrequently in drinking water in Canada. In a 1979 national survey of 30 municipal drinking water supplies, 1,1-DCE was found in only one treated water supply at a mean concentration of <1 µg/L and a maximum concentration of 20 µg/L.[4] In a 1981 to 1982 survey of drinking water in the lower Great Lakes, 1,1-DCE was detected in one of the 10 municipal supplies, in treated water but not in the untreated supply, at a trace level (<0.1 µg/L).[5] In an Alberta survey conducted from 1978 to 1985, 1,1-DCE was detected in one of 29 municipal drinking water supplies, at a maximum concentration of 1.4 µg/L.[6] Between 1987 and 1994, none of 1900 samples from 300 municipal surface water and groundwater supplies within Alberta contained 1,1-DCE at a concentration above the detection limit of 1 µg/L.[7] 1,1-DCE was not found in surveys conducted in 1985 and 1986 on 40 municipal supplies in the Atlantic region[8] or of 18 supplies in Quebec.[9] In a 1987 survey of the Lemieux Island treatment plant in Ottawa, conducted by the Ontario Ministry of the Environment, 1,1-DCE was not detected in 36 samples taken at the treatment plant or at two distribution sites.[10] In a 1985 to 1988 survey of water supplies in the four Atlantic provinces, 1,1-DCE was not detected in raw or treated drinking water samples taken from 151 sampling stations; the minimum quantifiable limit ranged from 0.5 to 1.0 µg/L.[11–14] In a 1981 survey of surface water samples taken from Lake Ontario, 1,1-DCE was detected (detection limit 0.09 µg/L) in 11 of 82 samples taken; concentrations ranged from trace to 3.5 µg/L. Maximum concentrations were generally near 0.19 µg/L.[15] Because of its volatility, there is potential for exposure in the home to airborne 1,1-DCE released from tap water.

The leaching of solvents into groundwater is a potential source of 1,1-DCE contamination. The presence of 1,1-DCE in 43% of the groundwater samples at the Gloucester, Ontario, landfill site (concentrations ranged from 0.9 to 60 µg/L) sampled in 1988 is thought to have resulted from the degradation of tetrachloroethylene and 1,1,1-trichloroethane, as 1,1-DCE is a known degradation product of these two compounds and was not known to have been disposed of at the site.[16]

Ambient air sampling for 1,1-DCE was carried out by Environment Canada yearly between 1988 and 1990.[17] In 1988, 1,1-DCE was detected in only two of 21 samples (maximum concentration 0.1 µg/m^3) taken from five sites in two cities between October and December. In 1989 and 1990, sampling was carried out

at 17 sites in 10 cities. 1,1-DCE was detected in 17% (n = 503; maximum concentration 0.4 µg/m^3) and 3% (n = 750; maximum concentration 0.5 µg/m^3) of the samples in 1989 and 1990, respectively. 1,1-DCE was detected in a survey of ambient air in Windsor and at Walpole Island, Ontario.[18] The mean 1,1-DCE concentration was <0.1 µg/m^3 in samples taken between July 1987 and October 1990 in Windsor and in samples taken between January 1988 and October 1990 at Walpole Island; maximum concentrations were 0.3 µg/m^3 and 0.2 µg/m^3 for Windsor and Walpole Island, respectively. In a limited study, Chan *et al*.[19] found that concentrations of 1,1-DCE in ambient air in Canada were highest in November/December (3.2 µg/m^3).

Chan *et al*.[19] also detected 1,1-DCE in the indoor air of a small number of Canadian homes. Average indoor concentrations were 8.4 µg/m^3 and 8.8 µg/m^3 for sampling done in November/December and February/March, respectively; a maximum concentration of 77 µg/m^3 was detected. 1,1-DCE has been identified in indoor air samples (both residences and offices) as well as in commuting automobile air samples collected in metropolitan Toronto.[20] In a small number of home and office samples, the mean concentrations of 1,1-DCE were 5.4 µg/m^3 and 5.0 µg/m^3, respectively; maximum concentrations were 9.0 µg/m^3 and 20.2 µg/m^3, respectively. The average concentrations of 1,1-DCE in the air in five commuter vehicles (two private automobiles and three public transit vehicles, one to two hours each way) in the morning and evening were 4.3 µg/m^3 and 3.6 µg/m^3, respectively; mean concentrations in ambient air were 0.4 µg/m^3 at both times.

There are no Canadian or U.S. data on exposure to 1,1-DCE via food, and it is therefore not possible at this time to estimate average intake from food. However, residual 1,1-DCE monomer is known to be present in food packaging materials. Concentrations of 1,1-DCE ranging from <0.2 to 26.2 ppm (mg/kg) have been reported in various household and industrial films used for food packaging.[21,22] 1,1-DCE was not detected in snacks, cheese or baked goods, but it was present in the outer layers of cooked meat products at concentrations ranging from 5 to 10 ppb (µg/kg)[22] and in potato chips at 26 ppb and 34 ppb after 48 and 90 days, respectively.[23] 1,1-DCE has been detected in composite samples of clams from The Rigolets, a pass between the Mississippi River (U.S.A.) and the Gulf of Mexico, at a concentration of 4.4 ppb wet weight.[24] The factors that govern migration of 1,1-DCE into food include the type of food packaged, the original concentration of the monomer in the packaging, the storage temperature and the length of time of contact during storage.[23] This was demonstrated in a study in which potato chips were packaged and stored in film containing 1,1-DCE at 0.4, 0.8 or 1.2 mg/m^2 for 48 or 90 days.[25] The data indicated that the 1,1-DCE concentration was highest in the food product packaged in film with the highest original 1,1-DCE content and with the longest storage time.

It has been estimated in the United Kingdom that the maximum possible intake of 1,1-DCE from food as a result of the use of packaging materials containing the monomer is no more than 1 µg/d per person.[26] If a 1,1-DCE concentration of 0.5 µg/L in drinking water is assumed, the estimated intake would be less than 1 µg/d for an adult consuming 1.5 L of drinking water per day. The concentrations of 1,1-DCE found in both ambient and indoor air would result in a higher intake of 1,1-DCE from air than from either food or drinking water. It is therefore probable that less than 10% of total 1,1-DCE intake is normally ingested in drinking water.

Analytical Methods and Treatment Technology

1,1-DCE may be detected by purge and trap gas chromatography followed by flame ionization detection or mass spectroscopy.[4,5] Based on its similarity to seven other volatile organics, its minimum practical quantitation limit (PQL) is 5 µg/L.[27]

Available data indicate that concentrations of 1,1-DCE are not reduced significantly during conventional drinking water treatment processes.[4,5,27] Removal of 90 to 95% of 1,1-DCE, to levels below 1 µg/L, may be achieved using packed tower aeration or granular activated carbon.

Health Effects

Mammals readily absorb 1,1-DCE after either inhalation or ingestion; dermal absorption is also assumed to occur.[28,29] 1,1-DCE is rapidly distributed to the tissues following oral or inhalation exposure, with preferential accumulation in the liver, kidneys and lungs. Elimination occurs primarily via urine and exhaled air.[30]

Complete absorption was observed in fasted and fed male rats given a single oral 1,1-DCE dose of 200 mg/kg bw in mineral oil, corn oil or aqueous Tween-80.[31] The total amount of 1,1-DCE exhaled was not affected by the magnitude of the dose administered, nor was the initial rapid-phase exhalation half-time of 1,1-DCE. In contrast, however, the later slow phase of 1,1-DCE exhalation was predictably affected by the administrative vehicle, with $t_{½}$ values increasing in the following order: Tween-80 < corn oil < mineral oil.

1,1-DCE is metabolized to a reactive intermediate via the mixed-function oxidase (MFO) pathway, which is saturable at relatively low doses, irrespective of the route of administration.[32] Inhalation studies in rats exposed to 150 ppm 1,1-DCE for two hours demonstrated a metabolic threshold for the MFO

pathway.[29,33,34] Metabolic thresholds were also observed in small male rats orally dosed at 25 mg/kg bw and in female rats at 100 mg/kg bw.[32] By analogy with the proposed mechanism for other chlorinated ethylenes,[35] the metabolism of 1,1-DCE has been suggested to produce an epoxide, presumed to be a transient unstable intermediate. The epoxide would readily conjugate with glutathione by glutathione-S-transferase, be hydrolysed by epoxide hydratase or react with nucleophilic centres. Thus, depletion of glutathione, by pretreatment with inhibitors such as diethyl maleate or by fasting, would decrease the potential of the rat to detoxify the reactive intermediates.

In support of this hypothesis, hepatotoxicity has been found to increase in glutathione-depleted animals, which suggests that detoxification of the intermediate metabolite occurs mainly through glutathione conjugation.[32] As well, a reduced rate of 1,1-DCE metabolism and an increase in 1,1-DCE toxicity have been observed in fasted rats receiving oral doses of 1,1-DCE. The effects of an oral 1,1-DCE dose of 50 mg/kg bw on fed, fasted and hyperthyroid (T_4) male rats were reported by Kanz *et al.*[36] The differences observed in body temperature (hypothermia in fasted rats), serum glucose concentrations (hypoglycaemia in fed and T_4 rats; hyperglycaemia in fasted rats) and hepatic glutathione and glutathione transferase (decreased in both fasting and T_4 rats) were indicative of different pathways or mechanisms of toxicity in fasted and hyperthyroid rats. Liver injury was found to be minimal in fed rats, moderate in fasted rats and intermediate in T_4 rats. Chieco *et al.*[31] found that liver injury was only slight in fed animals regardless of vehicle of administration; however, liver injury in fasted rats was moderate (Tween-80) to massive (mineral oil and corn oil).

Oral LD_{50} values (olive or corn oil) of 1510 to 1800 mg/kg bw and 1500 mg/kg bw have been determined for male and female rats, respectively.[37,38] In mice, Jones and Hathway[39] reported LD_{50} values of 194 mg/kg bw in females and 217 mg/kg bw in males. Bronchial injury in mice and hepatic damage in rats have been reported following acute exposure to 1,1-DCE.[32,40]

In an abstract, Quast *et al.*[41] described a study in which Sprague-Dawley rats were exposed to 1,1-DCE in their drinking water at concentrations of 0, 60, 100 or 200 ppm for 90 days, equivalent to concentrations of 0, 6, 10 or 19 mg/kg bw per day in males and 0, 8, 13 or 26 mg/kg bw per day in females. The only effect observed was reversible minimal hepatocellular cytoplasmic vacuolation reported in several rats (sex not stated) in the 200 ppm dose group. In a 13-week study by the National Toxicology Program (NTP),[42] rats and mice (10 per sex per dose) were administered 1,1-DCE at 0, 5, 15, 40, 100 or 250 mg/kg bw per day by gavage in corn oil, five times a week. In both species, the liver was identified as the target organ, with fatty metamorphosis, congestion, centrilobular or cellular necrosis, hepatocytomegaly, fibrosis or focal areas of cellular alteration being observed. 1,1-DCE (99.5% purity) was administered in peanut oil incorporated into gelatin capsules to beagle dogs (four per sex per dose) at doses of 0, 6.25, 12.5 or 25 mg/kg bw per day for 97 days.[43] Necropsy and histopathological examination did not reveal any exposure-related changes in tissues.

Although a few epidemiological studies involving 1,1-DCE have been reported, interpretation of the data is confounded by concomitant exposure to vinyl chloride. In the only study reported where vinyl chloride was not used as a copolymer, Ott *et al.*[44] examined the mortality and health of 138 employees exposed to 1,1-DCE and reported no findings that were statistically related or individually attributable to 1,1-DCE exposure. The International Agency for Research on Cancer[45] concluded that this study was not adequate to permit an assessment of human carcinogenicity, noting that 27 workers were lost to follow-up but considered to be alive in the analyses, 55 individuals had less than 15 years since first exposure and only five deaths had been observed.

In the only study to date to report carcinogenicity, Maltoni *et al.*[23] exposed Swiss mice (either 30 or 60 per sex per dose; 100 per sex in the control group) to 1,1-DCE by inhalation at doses of 0, 10, 25, 50, 100 or 200 ppm, four hours per day, four to five days per week for 52 weeks. A second 25 ppm treatment group (120 per sex; 90 per sex for controls) was added to further assess the carcinogenicity at 25 ppm. In both studies, following the 52-week dosing, the mice were allowed to survive either until spontaneous death or until 126 weeks. Excesses in mortality were reported in mice exposed to 50, 100 and 200 ppm. An increase in kidney adenocarcinomas (significance not stated) in male mice, a rare tumour in mice, was observed in the 25 and 50 ppm dose groups. Incidences using corrected values (animals alive when first kidney adenocarcinoma was observed) were 0/54, 0/24, 3/21, 2/18, 0/13 and 0/1 for the 0, 10, 25, 50, 100 and 200 ppm dose groups and 0/66 and 25/98 for the second control and 25 ppm dose groups. A kidney adenocarcinoma was also reported in a female mouse in the larger 25 ppm group.

Several carcinogenicity studies in which 1,1-DCE was orally administered have yielded negative results. Groups of 50 male and 50 female rats were administered 1,1-DCE in olive oil by gavage for 52 weeks at doses of 0, 0.5, 5, 10 or 20 mg/kg bw per day, four to five days per week, and allowed to survive until spontaneous death. There was no evidence of treatment-related or dose-related effects from exposure to 1,1-DCE by

gavage.[23] The authors stated that the lack of neoplastic response in the study was difficult to assess and may have been due to the animal system, the route of administration or the level of the daily dose used in the experiment.

In the most adequate study to date to investigate carcinogenicity via the oral route using the most appropriate vehicle of administration, 1,1-DCE (99.5% purity) was incorporated into the drinking water of groups of male and female Sprague-Dawley rats (48 per sex per dose) for two years.[43] Time-weighted average daily dose levels were 0, 7, 10 or 20 mg/kg bw for males and 0, 9, 14 or 30 mg/kg bw for females. No treatment-related effects on mortality, body or organ weights or haematological, urinary or clinical chemistry end points were observed. Histopathological examination showed no exposure-related neoplastic changes in rats of any treatment group; however, a minimal amount of hepatocellular swelling with mid-zonal fatty change was observed in females at all dose levels and was significant in males in the 20 mg/kg bw per day dose group only. A dose-related trend for females was not stated; in males, however, a "trend towards increased hepatic changes" was observed in the 10 mg/kg bw per day dose group.

There was no evidence of carcinogenicity of 1,1-DCE in an investigation by the NTP.[42] Doses of 0, 1 or 5 mg/kg bw per day in corn oil were administered by gavage, five days per week for 104 weeks, to groups of 50 male and 50 female F344/N rats. Similarly, doses of 0, 2 or 10 mg/kg bw per day were administered to $B6C3F_1$/N mice. There was an increased incidence of liver necrosis (focal, multifocal or diffused) in high-dose male mice and low-dose female mice and chronic renal inflammation in high-dose rats of both sexes. Increased tumour incidences (lymphoma only; lymphoma or leukaemia) in low-dose female mice were not considered to be treatment-related, as these effects were not observed in high-dose female mice, in male mice or in rats. It should be noted that 12 control and 10 low-dose male rats were accidentally killed during week 82 of the study, which may have compromised the sensitivity of the male rat study. Under the conditions of the bioassay, 1,1-DCE administered by gavage was not carcinogenic for F344/N rats or $B6C3F_1$/N mice of either sex. The NTP noted, however, that the use of a maximum tolerated dose in the oral dose study had not been clearly demonstrated, as indicated by the absence of compound-related effects on survival or clinical signs of toxicity, and that carcinogenicity had been associated with inhalation exposure to 1,1-DCE.[23]

1,1-DCE was administered by stomach tube (150 mg/kg bw in olive oil) as a single dose to 24 female BD IV rats on day 17 of gestation, and weekly doses of 50 mg/kg bw were administered to the 89 male and 90 female progeny for 120 weeks.[37] Controls, receiving olive oil only, followed the same dosing schedule. Litter sizes, pre-weaning mortality and survival rates were similar in exposed and control groups. There was no significant difference between exposed and control rats in the total number of tumour-bearing animals, although there were increases in the incidence of some tumours at certain sites. Non-significant increases in tumours observed in treated males and females but not in vehicle controls included a squamous cell carcinoma of the stomach (male), liver cell carcinomas (one male; two females), a seminoma (male), a rectal adenomatous polyp (male), a liver cell adenoma (female) and a carcinoma and adenoma of the salivary gland (female). Hyperplastic liver nodules were significantly increased ($p = 0.04$) in both sexes of treated progeny (2/81 males; 6/80 females) and in 2/23 treated dams, but not in controls. The authors concluded that there was "limited evidence of carcinogenicity," although additional study was required. It was also stated that the maximum tolerated dose was not reached, as indicated by a lack of obvious toxic effects in the dosed animals.

1,1-DCE has been mutagenic in several reverse gene mutation assays using *Salmonella typhimurium*, *Escherichia coli* K12 and *Saccharomyces cerevisiae* D7, only in the presence of metabolic activation,[46–50] and in a mouse host-mediated microbial assay with *S. cerevisiae* D7 following gavage of 1,1-DCE in corn oil.[50] Negative results have been obtained in both rat and mouse dominant lethal studies.[51,52]

Several *in vivo* and *in vitro* mammalian cytogenetic studies have failed to produce any significant positive results. Drevon and Kuroki[53] reported no increases in 8-azaguanine-resistant and ouabain-resistant colonies in Chinese hamster V79 cells exposed for five hours to 1,1-DCE in desiccators. In a more recent *in vitro* study, Sawada *et al.*[54] reported a weak but significant increase in the incidence of sister chromatid exchanges and a dose-dependent induction of chromosomal aberrations observed in the Chinese hamster cell line (CHL), but only in the presence of S9 activation. In *in vivo* mammalian studies, Quast *et al.*[55] observed no adverse effects (chromatid or chromosomal aberrations) of femoral bone marrow cells of Sprague-Dawley rats exposed to 25 or 75 ppm 1,1-DCE via inhalation for six months. A micronucleus test in the liver and blood of fetuses of ddY mice treated with 1,1-DCE was also negative; there was also no increase in the frequency of micronucleated erythrocytes in bone marrow.[54] Positive findings of increases in chromosomal aberrations in bone marrow cells of Chinese hamsters have, however, been reported by Hofmann and Peh[56] and Zeller and Peh.[57]

Sawada *et al*.[54] also examined the role of cytochrome P-450 in the metabolic activation of 1,1-DCE. The results indicated that the induction of chromosomal aberrations was inhibited by the addition of metyrapone and glutathione; tests of two metabolites of 1,1-DCE, chloroacetyl chloride and chloroacetic acid, were also negative. The authors concluded that the findings were compatible with the hypothesis that 1,1-DCE oxide may be the active genotoxic metabolite, although mutagenicity testing of this compound has not been reported because stabilized 1,1-DCE oxide is difficult to obtain.

Developmental effects of 1,1-DCE have not been demonstrated. Murray *et al*.[58] exposed pregnant rats to 1,1-DCE in their drinking water at doses of 0 or 200 ppm (equivalent to 40 mg/kg bw per day) on days 6 to 15 of gestation. There was a significant increase in the mean fetal crown–rump length in the litters of exposed rats, but no evidence of toxicity to the dams or their offspring. The authors concluded that, under the conditions of the experiment, 1,1-DCE was not teratogenic to rats.

Nitschke *et al*.[59] investigated the fertility of male and female Sprague-Dawley rats (F_0: 10 males and 20 females) and neonatal toxicity in a three-generation, six-litter study in which animals were exposed to 0, 50, 100 or 200 ppm 1,1-DCE continuously in their drinking water. Calculated doses were equivalent to approximately 0, 7, 14 or 29 mg/kg bw per day in the F_0 adults.[60] Histopathological examination was conducted on tissues of rats that had been exposed *in utero*, during lactation and post-weaning. Dose-related mild hepatocellular fatty changes were observed, as well as an accentuated hepatic lobular pattern that was reversible in adult rats. There was no evidence that fertility was affected by exposure to 1,1-DCE, although low fertility rates were observed in both treatment and control groups. Observations of six sets of litters throughout the three generations suggested that the reproductive capacity had not been affected.

Classification and Assessment

1,1-DCE is mutagenic with metabolic activation in *in vitro* assays and weakly genotoxic in some *in vivo* studies. The mutagenicity of 1,1-DCE *in vitro* requires the use of S9 from metabolically induced mouse or rat liver. S9 from uninduced liver does not seem to be effective. These results are a clue to the rather specific conditions that are required to produce a positive result *in vivo*. Effects are seen only in hamsters that have a unique pattern of P-450 metabolism, whereas a number of other *in vivo* studies in mice are negative. The positive host-mediated result in mice may be due to detection of a weak effect by this rather specialized assay resulting from low levels of metabolites present in the mouse. The limited metabolic conditions under which mutagenic effects are seen support the finding of limited tissue-specific carcinogenicity.

Available epidemiological data are inadequate for assessment of the carcinogenicity of 1,1-DCE in humans. There has been some evidence, in one study, of increased kidney tumour (adenocarcinomas) incidence in male mice exposed to 1,1-DCE by inhalation.[23] The compound was not, however, carcinogenic in several (including one adequate) bioassays by the ingestion route. Limiting factors with the Maltoni study[23] include less-than-lifetime exposure, lack of a clear dose–response relationship and no indication of statistical significance for the observed tumours. Studies using orally administered 1,1-DCE have yielded negative results, although small increases in liver tumour incidence have also been observed in rats and offspring of exposed mothers in a limited bioassay by Ponomarkov and Tomatis.[37] The evidence for the carcinogenicity of 1,1-DCE is, therefore, considered to be limited, and the compound has been classified in Group IIIB (possibly carcinogenic to humans).

For compounds classified in Group IIIB, the acceptable daily intake (ADI) is usually derived on the basis of division of a no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL) by an uncertainty factor that takes into account the limited evidence of carcinogenicity. For 1,1-DCE, however, no adequate studies by the oral route that resulted in adverse effects were identified. Therefore, a LOEL (lowest-observed-effect level) of 9 mg/kg bw per day, based on mid-zonal fatty changes in the liver observed in female rats, and an additional uncertainty factor of 10 for use of a LOEL instead of a NOAEL were used to derive the ADI as follows:

$$\text{ADI} = \frac{9 \text{ mg/kg bw per day}}{3000} = 0.003 \text{ mg/kg bw per day}$$

where:

- 9 mg/kg bw per day is the LOEL for mid-zonal fatty changes in the liver of female rats, observed in the two-year study with the most appropriate route and vehicle of administration (i.e., drinking water)[43]
- 3000 is the uncertainty factor (×10 for interspecies variation; ×10 for intraspecies variation; ×10 for use of a LOEL; and ×3 for limited evidence of carcinogenicity, based on an inhalation study using a different species [mouse] and with a different target organ [kidney], in view of the fact that studies by the oral route are negative).

It should be noted that the effect level on which the ADI was derived is similar to that for the same effects observed in the reproductive study by Nitschke *et al*.[59]

Rationale

1,1-DCE is a known degradation product of tetrachloroethylene and 1,1,1-trichloroethane, which are common groundwater contaminants. Because 1,1-DCE is classified in Group IIIB, the maximum acceptable concentration (MAC) for 1,1-DCE in drinking water is derived from the ADI as follows:

$$\text{MAC} = \frac{0.003 \text{ mg/kg bw per day} \times 70 \text{ kg bw} \times 0.10}{1.5 \text{ L/d}} \approx 0.014 \text{ mg/L}$$

where:

- 0.003 mg/kg bw per day is the ADI, as derived above
- 70 kg bw is the average body weight of an adult
- 0.10 is the proportion of total 1,1-DCE intake considered to be ingested in drinking water (see "Exposure" section)
- 1.5 L/d is the average daily consumption of drinking water for an adult.

References

1. World Health Organization. Vinylidene chloride. Environmental Health Criteria 100. Geneva, Switzerland (1990).

2. Agency for Toxic Substances and Disease Registry. Draft update of the toxicological profile for 1,1-dichloroethene. U.S. Public Health Service, Atlanta, GA (1992).

3. Camford Information Services Inc. CPI product profiles. Polyvinylidene chloride (PVDC). Don Mills, Ontario (1992).

4. Otson, R., Williams, D.T. and Bothwell, P.D. Volatile organic compounds in water at thirty Canadian potable water treatment facilities. J. Assoc. Off. Anal. Chem., 65: 370 (1982).

5. Otson, R. Purgeable organics in Great Lakes raw and treated water. Int. J. Environ. Anal. Chem., 31: 41 (1987).

6. Alberta Ministry of Environmental Protection. Drinking water survey, 1978–1985. Unpublished report, Municipal Engineering Branch, Pollution Control Division, Edmonton (1985).

7. Milos, J.P. Personal communication. Municipal Water and Wastewater Branch, Air and Water Approvals Division, Alberta Ministry of Environmental Protection, Edmonton (1994).

8. Environment Canada. Data summary report, federal–provincial drinking water sources toxic chemical study, 1985–1986. Water Quality Branch, Inland Waters Directorate, Moncton (1987).

9. Ministère de l'Environnement, Gouvernement du Québec. Micropolluants organiques — Campagnes d'échantillonnage 1986. Direction des Eaux Souterraines et de Consommation, Sainte-Foy, Québec (1987).

10. Ontario Ministry of the Environment. Ottawa (Lemieux Island) water treatment plant — annual report. Drinking Water Surveillance Program (1987).

11. Environment Canada. Atlantic region federal–provincial toxic chemical survey of municipal drinking water sources. Data summary report, Province of Nova Scotia, 1985–1988. IWD-AR-WQB-89-154, Water Quality Branch, Inland Waters Directorate (1989).

12. Environment Canada. Atlantic region federal–provincial toxic chemical survey of municipal drinking water sources. Data summary report, Province of New Brunswick, 1985–1988. IWD-AR-WQB-89-155, Water Quality Branch, Inland Waters Directorate (1989).

13. Environment Canada. Atlantic region federal–provincial toxic chemical survey of municipal drinking water sources. Data summary report, Province of Prince Edward Island, 1985–1988. IWD-AR-WQB-89-156, Water Quality Branch, Inland Waters Directorate (1989).

14. Environment Canada. Atlantic region federal–provincial toxic chemical survey of municipal drinking water sources. Data summary report, Province of Newfoundland, 1985–1988. IWD-AR-WQB-89-157, Water Quality Branch, Inland Waters Directorate (1989).

15. Kaiser, K.L.E., Comba, M.E. and Huneault, H. Volatile halocarbon contaminants in the Niagara River and in Lake Ontario. J. Great Lakes Res., 9(2): 212 (1983).

16. Lesage, S., Jackson, R.E., Priddle, M.W. and Riemann, P.G. Occurrence and fate of organic solvent residues in anoxic groundwater at the Gloucester landfill, Canada. Environ. Sci. Technol., 24(4): 559 (1990).

17. Dann, T. Personal communication. Pollution Measurement Division, River Road Environmental Technology Centre, Environment Canada, Ottawa (1993).

18. Environment Canada. Detroit Incinerator Monitoring Program. Data Report No. 6. A) Summary of measurement data, July 1987 to October 1990, Windsor and Walpole Island monitoring sites. Appendices B to G. PMD-92-1, River Road Environmental Technology Centre, Ottawa (1992).

19. Chan, C.C., Valner, L., Martin, J.W. and Williams, D.T. Determination of organic contaminants in residential indoor air using an adsorption–thermal desorption technique. J. Air Waste Manage. Assoc., 40(1): 62 (1990).

20. Bell, R.W., Chapman, R.E., Kruschel, B.D., Spencer, M.J., Smith, K.V. and Lusis, M.A. The 1990 Toronto Personal Exposure Pilot (PEP) Study (draft report). ARB-207-90, Atmospheric Research and Special Programs Section, Air Resources Branch, Ontario Ministry of the Environment (1991).

21. Birkel, T.J., Roach, J.A.G. and Sphon, J.A. Determination of vinylidene chloride in Saran films by electron capture gas–solid chromatography and confirmation by mass spectrometry. J. Assoc. Off. Anal. Chem., 60(5): 1210 (1977).

22. Gilbert, J., Shepheard, M.J., Startin, J.R. and McWeeny, D.J. Gas chromatographic determination of vinylidene chloride monomer in packaging films and in foods. J. Chromatogr., 197: 71 (1980).

23. Maltoni, C., Lefemine, G., Cotti, G., Chieco, P. and Patella, V. Archives of research on industrial carcinogenesis. Vol. III. Experimental research on vinylidene chloride carcinogenesis. C. Maltoni and M.A. Mehlman (series eds.). Princeton Scientific Publishers, Princeton, NJ (1985).

24. Ferrario, J.B., Lawler, G.C., DeLeon, I.R. and Laseter, J.L. Volatile organic pollutants in biota and sediments of Lake Pontchartrain. Bull. Environ. Contam. Toxicol., 34: 246 (1985).

25. ICI, Food Science Division. Vinylidene chloride-containing food contact materials in the UK. Report from the United Kingdom, with limited circulation (1978), cited in reference 23.

26. Ministry of Agriculture, Fisheries and Food. Survey of vinylidene chloride levels in food contact materials and in foods. Third report of the Steering Group on Food Surveillance, Working Party on Vinylidene Chloride, London, UK. Food Surveillance Paper No. 3 (1980), cited in reference 1.

27. U.S. Environmental Protection Agency. National primary drinking water regulations. Fed. Regist., 50: 46907 (1985).

28. Jones, B.K. and Hathway, D.E. The biological fate of vinylidene chloride in rats. Chem.-Biol. Interact., 20: 27 (1978).

29. Dallas, C.E., Weir, F.W., Feldman, S., Putcha, L. and Bruckner, J.V. The uptake and disposition of 1,1-dichloroethylene in rats during inhalation exposure. Toxicol. Appl. Pharmacol., 68: 140 (1983).

30. McKenna, M.J., Zempel, J.A., Madrid, E.O. and Gehring, P.J. The pharmacokinetics of [^{14}C]vinylidene chloride in rats following inhalation exposure. Toxicol. Appl. Pharmacol., 45: 599 (1978).

31. Chieco, P., Molsen, M.T. and Reynolds, E.S. Effect of administrative vehicle on oral 1,1-dichloroethylene toxicity. Toxicol. Appl. Pharmacol., 57: 146 (1981).

32. Andersen, M.E. and Jenkins, L.J., Jr. Oral toxicity of 1,1-dichloroethylene in the rat: effects of sex, age, and fasting. Environ. Health Perspect., 21: 157 (1977).

33. Andersen, M.E., Gargas, M.L., Jones, R.A. and Jenkins, L.J., Jr. The use of inhalation techniques to assess the kinetic constants of 1,1-dichloroethylene metabolism. Toxicol. Appl. Pharmacol., 47: 395 (1979).

34. Filser, J.G. and Bolt, H.M. Pharmacokinetics of halogenated ethylenes in rats. Arch. Toxicol., 42: 123 (1979).

35. Daniel, J.W. The metabolism of ^{36}Cl-labelled trichloroethylene and tetrachloroethylene in the rat. Biochem. Pharmacol., 12: 795 (1963).

36. Kanz, M.F., Whitehead, R.F., Ferguson, A.E. and Moslen, M.T. Potentiation of 1,1-dichloroethylene hepatotoxicity: comparative effects of hyperthyroidism and fasting. Toxicol. Appl. Pharmacol., 95: 93 (1988).

37. Ponomarkov, V. and Tomatis, L. Long-term testing of vinylidene chloride and chloroprene for carcinogenicity in rats. Oncology, 37: 136 (1980).

38. Jenkins, L.J., Jr., Trabulus, M.J. and Murphy, S.D. Biochemical effects of 1,1-dichloroethylene in rats: comparison with carbon tetrachloride and 1,2-dichloroethylene. Toxicol. Appl. Pharmacol., 23: 501 (1972).

39. Jones, B.K. and Hathway, D.E. Differences in metabolism of vinylidene chloride between mice and rats. Br. J. Cancer, 37: 411 (1978).

40. Forkert, P.G., Geddes, B.A., Birch, D.W. and Massey, T.E. Morphologic changes and covalent binding of 1,1-dichloroethylene in clara and alveolar type II cells isolated from lungs of mice following in vivo administration. Drug Metab. Dispos., 18(4): 534 (1990).

41. Quast, J.F., Humiston, C.G., Schwetz, B.A., Balmer, M.F., Rampy, L.W., Norris, J.M. and Gehring, P.J. Results of 90-day toxicity studies in rats given vinylidene chloride in their drinking water or exposed to VDC vapors by inhalation [abstract]. Toxicol. Appl. Pharmacol., 41: 187 (1977).

42. National Toxicology Program. NTP technical report on the carcinogenesis bioassay of vinylidene chloride (CAS No. 75-35-4) in F344/N rats and B6C3F$_1$/N mice (gavage study). NTP TR 228, NIH Publication No. 82-1784, U.S. Department of Health and Human Services, Research Triangle Park, NC (1982).

43. Quast, J.F., Humiston, C.G., Wade, C.E., Ballard, J., Beyer, J.E., Schwetz, R.W. and Norris, J.M. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fundam. Appl. Toxicol., 3: 55 (1983).

44. Ott, M., Fishbeck, W., Townsend, J. and Schneider, E. A health study of employees exposed to vinylidene chloride. J. Occup. Med., 18(11): 735 (1976).

45. International Agency for Research on Cancer. Vinylidene chloride and vinylidene chloride–vinyl chloride copolymers. IARC Monogr. Eval. Carcinog. Risk Chem. Hum., 19: 439 (1979).

46. Bartsch, H., Malaveille, C., Montesano, R. and Tomatis, L. Tissue-mediated mutagenicity of vinylidene chloride and 2-chlorobutadiene in *Salmonella typhimurium*. Nature, 255: 641 (1975).

47. Bartsch, H., Malaveille, C., Barbin, A. and Planche, G. Mutagenic and alkylating metabolites of halo-ethylenes, chlorobutadienes and dichlorobutenes produced by rodent or human liver tissues. Evidence for oxirane formation by P450-linked microsomal mono-oxygenases. Arch. Toxicol., 41: 249 (1979).

48. Jones, B.K. and Hathway, D.E. Tissue-mediated mutagenicity of vinylidene chloride in *Salmonella typhimurium* TA1535. Cancer Lett., 5: 1 (1978).

49. Greim, H., Bonse, G., Radwan, Z., Reichert, D. and Henschler, D. Mutagenicity in vitro and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxirane formation. Biochem. Pharmacol., 24: 2013 (1975).

50. Bronzetti, G., Bauer, C., Corsi, C., Leporini, C., Nieri, R. and del Carratore, R. Genetic activity of vinylidene chloride in yeast. Mutat. Res., 89: 179 (1981).

51. Short, R.D., Minor, J.L., Winston, J.M. and Lee, C.-C. A dominant lethal study in male rats after repeated exposures to vinyl chloride or vinylidene chloride. J. Toxicol. Environ. Health, 3: 965 (1977).

52. Anderson, D., Hodge, M.C.E. and Purchase, I.F.H. Dominant lethal studies with the halogenated olefins vinyl chloride and vinylidene dichloride in male CD-1 mice. Environ. Health Perspect., 21: 71 (1977).

53. Drevon, C. and Kuroki, T. Mutagenicity of vinyl chloride, vinylidene chloride and chloroprene in V79 Chinese hamster cells. Mutat. Res., 76: 173 (1979).

54. Sawada, M., Sofuni, T. and Ishidate, M., Jr. Cytogenetic studies on 1,1-dichloroethylene and its two isomers in mammalian cells in vitro and in vivo. Mutat. Res., 187: 157 (1987).

55. Quast, J.F., McKenna, M.J., Rampy, L.W. and Norris, J.M. Chronic toxicity and oncogenicity on inhaled vinylidene chloride in rats. Fundam. Appl. Toxicol., 6: 105 (1986).

56. Hofmann, H.T. and Peh, J. Report on the test of vinylidene chloride for mutagenic effects in Chinese hamsters after subacute inhalation [in German]. Ludwigshafen, BASF Aktiengesellschaft. 22 pp. (1976), cited in reference 1.

57. Zeller, H. and Peh, J. Report on the tests of vinylidene chloride for mutagenic effects in Chinese hamsters after single oral application (chromosomal study) [in German]. Ludwigshafen, BASF Aktiengesellschaft. 12 pp. (1975), cited in reference 1.

58. Murray, F.J., Nitschke, K.D., Rampy, L.W. and Schwetz, B.A. Embryotoxicity and fetotoxicity of inhaled or ingested vinylidene chloride in rats and rabbits. Toxicol. Appl. Pharmacol., 49: 189 (1979).

59. Nitschke, K.D., Smith, F.A., Quast, J.F., Norris, J.M. and Schwetz, B.A. A three-generation rat reproductive toxicity study of vinylidene chloride in the drinking water. Fundam. Appl. Toxicol., 3: 75 (1983).

60. Health Canada. Human health risk assessment for priority substances. Ottawa (1994).