Canadian Environmental Protection Act

Priority Substances List
Assessment Report

1,2-Dichloroethane

Government of Canada / Gouvernement du Canada
Environment Canada / Environnement Canada
Health Canada / Santé Canada

Canada

CANADA'S GREEN PLAN

Canadian Environmental Protection Act

PRIORITY SUBSTANCES LIST ASSESSMENT REPORT

1,2-DICHLOROETHANE

Government of Canada
Environment Canada
Health Canada

Also available in French under the title:
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Synopsis

1,2-Dichloroethane is used in Canada primarily as an intermediate in the synthesis of vinyl chloride and, in small quantities, in the manufacture of motor antiknock fluids for export. Most of the 1,2-dichloroethane released to the Canadian environment enters the air, particularly during its production and during the production of vinyl chloride monomer. Other sources of entry include the discharge of effluents from industries that use or produce 1,2-dichloroethane, effluents from the treatment of contaminated groundwater, air emissions and leachates from waste disposal sites, and long-range atmospheric transport from other countries.

The highest concentration of 1,2-dichloroethane detected in Canadian ambient surface waters is eight times less than the estimated effects threshold which is based on the most sensitive aquatic organism. The highest concentration of 1,2-dichloroethane in ambient air measured in Canada is 1080 times less than the lowest reported effect level for a terrestrial plant.

Although 1,2-dichloroethane absorbs infrared radiation at wavelengths critical to global warming, the relatively low rate of release of 1,2-dichloroethane to the atmosphere and the low atmospheric concentration indicate that 1,2-dichloroethane will not contribute significantly to global warming. Furthermore, the calculated ozone-depleting potential of 1,2-dichloroethane is so low that it is not expected to contribute significantly to stratospheric ozone depletion.

Based on estimation of the total average daily intake of 1,2-dichloroethane by the general population in Canada, air appears to be the most significant source of exposure. Based on the weight of evidence of carcinogenicity in experimental animals, 1,2-dichloroethane is classified as "probably carcinogenic to humans", i.e., as a substance for which there is believed to be some chance of adverse health effects at any level of exposure. For such substances, where data permit, estimated exposure is compared to quantitative estimates of cancer potency to characterize risk and provide guidance for further action (i.e., analysis of options to reduce exposure). For 1,2-dichloroethane, such a comparison suggests that the priority for analysis of options to reduce exposure would be low to moderate.

Based on these considerations, it has been concluded that 1,2-dichloroethane is not entering the Canadian environment in a quantity or concentration or under conditions that constitute a danger to the environment or to the environment on which human life depends. However, it has been concluded that 1,2-dichloroethane occurs at concentrations that may constitute a danger in Canada to human life or health.

Canadian Environmental Protection Act
Priority Substances List Assessment Report

1,2 DICHLOROETHANE

NOTE TO READERS: Due to reanalysis of preliminary data obtained in a national pilot study, mean and maximum values for the concentrations of 1,2-dichloroethane in residential indoor air included in this report should be replaced by <0.1 and 1.7 µg/m^3, respectively. Estimated intakes for 1,2-dichloroethane in indoor air based on these revised concentrations range from <0.02 to <0.04 µg/m^3, while estimated total intake from all sources ranges from <0.03 to 0.07 µg/kg-bw/day. Revised exposure/potency indices range from 1.0×10^{-7} to 1.1×10^{-5}. The priority for analysis of options to reduce exposure remains low to moderate.

1.0 Introduction

The *Canadian Environmental Protection Act* (CEPA) requires the Minister of the Environment and the Minister of Health to prepare and publish a Priority Substances List that identifies substances, including chemicals, groups of chemicals, effluents, and wastes that may be harmful to the environment or constitute a danger to human health. The Act also requires both Ministers to assess these substances and determine whether they are "toxic" as defined under Section 11 of the Act which states:

> "..a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions
>
> a) having or that may have an immediate or long-term harmful effect on the environment;
>
> b) constituting or that may constitute a danger to the environment on which human life depends; or
>
> c) constituting or that may constitute a danger in Canada to human life or health."

Substances that are assessed as "toxic" as defined under Section 11 of the Act may be placed on the List of Toxic Substances in Schedule I of CEPA (Subsection 33(1)). Consideration can then be given to developing guidelines, codes of practice, or regulations necessary to control any aspect of these substances' life cycle, including manufacture, use, storage, transport, and ultimate disposal.

The assessment of whether 1,2-dichloroethane (often referred to as ethylene dichloride) is "toxic", as defined under CEPA, was based on the determination of whether it enters or is likely to **enter** the Canadian environment in a concentration or quantities or under conditions that could lead to **exposure** of humans or other biota at levels that could cause adverse **effects**.

Published data relevant to the assessment of whether 1,2-dichloroethane is "toxic" to the environment were identified through on-line searches conducted in June, 1992, of the following commercial databases: Aquatic Science and Fisheries Abstract (ASFA), BIOSIS, Chemical Abstracts, Enviroline, International Register of Potentially Toxic Chemicals (IRPTC), Science Citation Index (SCI), and TOXLINE. In addition, trade information was voluntarily supplied by the chlorinated solvents industry. Data on Canadian sources, use patterns, and levels of 1,2-dichloroethane were emphasized. Data relevant to the environmental assessment of 1,2-dichloroethane obtained after April 1993 were not considered for inclusion.

To identify data relevant to the assessment of exposure and health effects in humans, several reviews, including those prepared by the Agency for Toxic Substance and Disease Registry (1989; 1992), the United States Environmental Protection Agency (U.S. EPA, 1985a;b*)*, the World Health Organization (WHO, 1987), and a review of data on toxicokinetics and health effects prepared under contract by Global-Tox International

Corporation (1991) were consulted. In addition, a computerized literature search was conducted (May, 1991) on the following data bases: Chemid, Hazardous Substances Data Bank (HSDB), Registry of Toxic Effects of Chemical Substances (RTECS), and TOXLINE. The Medline subfile of TOXLINE was also searched in September, 1991. To identify data published after May, 1991, these data bases were searched in June, 1992 and again in May 1993. In addition, officials of Dow Chemical Canada were requested to provide any data relevant for consideration in this assessment. Only relevant data identified by May 1993 were considered for inclusion.

All original studies that form the basis for determining whether 1,2-dichloroethane is "toxic" under CEPA were critically evaluated by the following Environment Canada staff (entry, exposure, and effects on the environment) and Health Canada staff (human exposure and effects on human health):

Environment Canada	Health Canada
T. Dann	I. Caldwell
S. Jones	K. Hughes
K. Kaiser	B. Idris
S. Lesage	M.E. Meek
K. Lloyd	

Quantitative estimates of carcinogenic potency were provided by M. Walker and S. Bartlett of Health Canada.

In this report, a synopsis that will appear in the *Canada Gazette* is presented. Section 2.0 is an extended summary of the technical information that is critical to the assessment. This information is presented in greater detail in supporting documentation that is available upon request. The assessment of whether 1,2-dichloroethane is "toxic" under CEPA is presented in Section 3.0.

As part of the review and approvals process established by Environment Canada for its contribution, the environmental sections of the Assessment Report and supporting documentation were reviewed externally by Dr. D. Muir (Fisheries and Oceans), Dr. D. Singleton (National Research Council Canada), and Dr. K. Woodburn (Dow Chemical Canada Inc.). The sections related to the assessment of human health effects were approved by the Standards and Guidelines Rulings Committee of the Bureau of Chemical Hazards of Health Canada. The final Assessment Report was reviewed and approved by the Environment Canada/Health Canada CEPA Management Committee.

Copies of this Assessment Report and the unpublished supporting documentation are available upon request from:

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2.0 Summary of Information Critical to Assessment of "Toxic"

2.1 Identity, Properties, Production, and Uses

1,2-Dichloroethane [Chemical Abstract Service (CAS) Registry Number 107-06-2] is a colourless, flammable liquid at room temperature having a sweet, chloroform-like odour and a molecular formula of $C_2H_4Cl_2$ (Sax and Lewis, 1987). It is a highly volatile, synthetic chemical having a vapour pressure of 8.5 kPa (at 20^0C), water solubility of 8690 mg/L (at 20^0C), Henry's Law constant of 111.5 Pa·m^3/mol (at 25^0C), and log partition coefficients of less than 2 (log k_{ow} 1.76 and log k_{OC} = 1.28) (Archer, 1979; Konemann, 1981; Warner *et al,* 1987; Chiou *et al,* 1979). 1,2-Dichloroethane absorbs infrared light at several wavelengths (7, 12, and 13 µm) characteristic of trace gases associated with global warming. Substances that absorb strongly between 7 and 14 µm act to absorb thermal radiation from the Earth's surface that would otherwise escape into space (Ramanathan, 1985).

1,2-Dichloroethane is produced by either the catalytic vapour- or liquid-phase chlorination of ethylene or by oxychlorination of ethylene (Archer, 1979). Most commercial grade 1,2-dichloroethane is 97 to 99% pure (Drury and Hammons, 1979). 1,2-Dichloroethane is detected and quantified by gas chromatography with either electron capture or mass spectrometric detectors.

In Canada, 1,2-dichloroethane is produced by one company in Fort Saskatchewan, Alberta (SRI International, 1992); the second location, in Sarnia, Ontario, stopped production in May 1993. The total Canadian production of 1,2-dichloroethane in 1990 was estimated to be 922 kt, with the Fort Saskatchewan plant manufacturing approximately 80% of this total (CPI, 1991a). The total domestic demand and export of 1,2-dichloroethane in 1990 was 676 kt and 246 kt, respectively (CPI, 1991a). 1,2-Dichloroethane is not imported into Canada (CPI, 1991a).

The predominant use of 1,2-dichloroethane in Canada is as an intermediate in the synthesis of vinyl chloride (99% of the total domestic demand) and in the manufacture of antiknock fluids containing tetraethyl lead (TEL) for export (CPI, 1991a). Historically, 1,2-dichloroethane was also used in Canada in the synthesis of such chlorinated solvents as tri- and tetra-chloroethylene and methyl chloroform, and in fumigant formulations (Canada Gazette, 1992; CPI, 1990; Dow Canada, 1992; Agriculture Canada, 1992). In 1991, the total production of 1,2-dichloroethane in the United States was 6318 kt where it was used primarily for the synthesis of vinyl chloride monomer (approximately 88%), and in the synthesis of other chlorinated solvents and ethyleneamines (2%) (Chemical Marketing Reporter, 1992). In 1991, the United States imported and exported approximately 5 and 659 kt, of 1,2-dichloroethane, respectively.

2.2 Entry into the Environment

Most of the 1,2-dichloroethane released to the Canadian environment enters the air, particularly during its production and during the production of vinyl chloride monomer. Only one company manufactures 1,2-dichloroethane and vinyl chloride

monomer in Canada. Based on the 1990 Canadian production of 416.8 kt of vinyl chloride monomer and 922 kt of 1,2-dichloroethane (CPI, 1991a;b) and a waste-to-product ratio of 0.008 (Tsang and Bisson, 1992), the total waste generated would be 10.7 kt. 1,2-Dichloroethane is then recovered from waste streams in the vinyl chloride monomer/l,2-dichloroethane production in a two-stage distillation operation. An analysis by the producer of their combined vinyl chloride monomer plant and 1,2-dichloroethane plant, liquid waste streams under steady state operating conditions, determined the 1,2-dichloroethane content to range from 0.5 to 2.8% by weight, or 53.6 to 299.9 tonnes of 1,2-dichloroethane per year (Wright, 1992). The liquid waste stream is then incinerated (McPherson *et al.,* 1979). Assuming an incineration destruction efficiency of 99.99% (U.S. EPA, 1986), approximately 0.005 to 0.029 t (or 5 to 29 kg) of 1,2-dichloroethane would be emitted to the atmosphere each year from vinyl chloride monomer and 1,2-dichloroethane wastes.

Emissions of 1,2-dichloroethane to air were monitored in 1992 at the Fort Saskatchewan plant. Approximately 7.1 tonnes of 1,2-dichloroethane were released to the atmosphere: fugitive (accidental) emissions comprised 45% (3.2 t) of the total; secondary sources (including losses from process water) (1.8 t), storage and handling (1.7 t), process (including thermal oxidizer vents) (0.1 t), and other sources (0.3 t) (Tsang and Bisson, 1993).

1,2-Dichloroethane enters surface waters from the effluents of industries that manufacture or use this substance. In Ontario, the mean concentrations of 1,2-dichloroethane in undiluted effluents from the Sarnia plant ranged between 2.5 µg/L (0.5 kg/d) (1989/1990) and 25 µg/L (1992) (OME, 1992a; Street, 1992). It was not detected (detection limit = 1.0 µg/L) in the undiluted effluent of the plant manufacturing 1,2-dichloroethane in Alberta during 1991 and 1992 (AEC, 1992).

1,2-Dichloroethane is incorporated into antiknock gas formulations (tetraethyl lead) by one company in Ontario. It is possible, therefore, that tall pipe emissions could contain small quantities of 1,2-dichloroethane. In 1990, however, the Canadian government introduced Gasoline Regulations that prohibit the sale of leaded gasoline in Canada. The only exemptions to this ban include farm machinery, marine engines, airplanes, and heavy-duty trucks (Canada Gazette, 1990). Tetraethyl lead in gasoline is, therefore, no longer a significant source of release of 1,2-dichloroethane to ambient air in Canada.

1,2-Dichloroethane also enters the Canadian environment in air emissions and leachates from waste disposal sites (Lesage *et al.,* 1990; Ghassemi *et al.;* 1984; Harkov *et al.,* 1985; Pakdel *et al,* 1992). Two sites in Canada have been identified as having groundwater contaminated by 1,2-dichloroethane. Laboratory organic solvents were the primary wastes deposited in a Gloucester, Ontario landfill site and, although the site has been abandoned since 1980, concentrations of 1,2-dichloroethane in groundwater were reported to range between 3.9 and 58 µg/L in 1988 (Lesage *et al.,* 1990). At a hazardous waste site in Ville Mercier, Quebec, the maximum concentration of 1,2-dichloroethane in groundwater was 41.8 g/L in 1988,

although the site has not been used since 1972 (Pakdel *et al,* 1992). Remedial action was undertaken by the Quebec Ministry of the Environment and, between July 1989 and April 1993, the mean concentration of 1,2-dichloroethane in the effluent of the treatment system was 346 µg/L with a maximum single value of 840 µg/L (Fontaine, 1993).

Long-range transport of 1,2-dichloroethane through the atmosphere has been documented (Class and Ballschmiter, 1986; Singh *et al.,* 1983); 1,2-dichloroethane may enter Canada from the United States or other countries. It was reported in the Toxic Release Inventory that approximately 2000 t of 1,2-dichloroethane were released to the ambient air during 1989 in the United States from industrial producers and users (U.S. EPA, 1990). The contribution that this source made to the Canadian environment could not be estimated.

2.3 Exposure-related Information

2.3.1 Fate

The atmosphere is expected to be the predominant environmental sink for 1,2-dichloroethane because this substance has a high vapour pressure (Archer, 1979). The calculated reaction rates of 1,2-dichloroethane with hydroxyl (OH) radicals derived from the Structure-Activity Relations (SAR) model (Atkinson, 1987) [3.63 x 10^{-13} cm^3/mol-sec @ 298 K] and from the model of Nimitz and Skaggs (1992) [5.42x 10^{-13} cm^3/mol-sec @ 277 K] are similar to the experimental value determined by Qiu *et al.* (1992) [2.09 x 10^{-13} cm^3/mol-sec @ 292 K]. Based on these reaction rates and assuming an atmospheric OH concentration representative of a moderately polluted area (Finlayson-Pitts and Pitts, 1986), the estimated atmospheric lifetime of 1,2-dichloroethane is between $43_{calculated}$ to $111_{experimental}$ days. Due to the moderate persistence of 1,2-dichloroethane in the troposphere, long-range transport is possible. Further evidence of long-range transport is provided by two monitoring studies in which 1,2-dichloroethane was detected in the lower troposphere over the northern Atlantic Ocean and over the Pacific Ocean (Class and Ballschmiter, 1986; Singh *et al.,* 1983).

Once 1,2-dichloroethane enters the troposphere, it undergoes photo-oxidation to produce formyl chloride, chloroacetyl chloride, hydrochloric acid, carbon monoxide, and carbon dioxide (Spence and Hanst, 1978). Any 1,2-dichloroethane that reaches the stratosphere is photolyzed to produce chlorine radicals that may in turn react with ozone (Callahan *et al.,* 1979; Spence and Hanst, 1978). However, a simple method developed by Nimitz and Skaggs (1992) indicates that 1,2-dichloroethane is not expected to contribute significantly to the depletion of the stratospheric ozone layer. Based on either the experimental (Qiu *et al.,* 1992) or predicted (Atkinson, 1987; Nimitz and Skaggs, 1992) rates of reaction between OH and 1,2-dichloroethane, the ozone depletion potential (ODP) for 1,2-dichloroethane is very much less than 0.001 relative to the chlorofluorocarbon, CFC-11.

Volatilization is the major process for the removal of 1,2-dichloroethane from the aquatic environment (Dilling *et al, 1975).* The half-life in a stirred aqueous solution, at

varying depths and surface areas, ranges between 5 and 29 minutes (Chiou *et al.,* 1980; Dilling *et al., 1975).* The predicted half-live of 1,2-dichloroethane, based on fate modelling (EXAMS) was nine days in a eutrophic lake and one day in a 300-km reach of a river system when a loading rate of 0.1 kg of 1,2-dichloroethane per hour was assumed for both examples (U.S. EPA, 1982).

Hydrolysis of 1,2-dichloroethane can also occur in the aquatic environment; however, with an estimated half-life of 72 years at neutral pH and at 25^0C (Barbash and Reinhard, 1989), this process cannot be considered as a major removal pathway from surface waters. When examined under conditions common to groundwater (in the presence of sodium sulphide, a pH of 7, and a temperature of 15^0C, the estimated half-life of 1,2-dichloroethane was 23 years (Barbash and Reinhard, 1989). The primary products of hydrolysis for 1,2-dichloroethane are vinyl chloride and 2-chloroethano1 (Jeffers *et al,* 1989) which in turn can be further degraded to acetylene and acetaldehyde from vinyl chloride (Hill *et al.,* 1976) and to ethylene glycol from 2-chloroethanol (Ellington *et al,* 1988).

Microbial degradation of 1,2-dichloroethane in water has been observed but is a slow process. The short residence time of 1,2-dichloroethane in surface waters, because of its high volatility, may not provide sufficient time for microbial adaption to occur (U.S. EPA, 1982). In a static flask study with initial concentrations of 5 and 10 mg/L 1,2-dichloroethane, a loss from aerobic degradation of 20 to 63% in seven days following an acclimation period was reported. However, 5 to 27% of the total loss was attributed to volatilization (Tabak *et al.,* 1981). The methanotrophic bacterium *Methylosinus trichosporium* (Oldenhuis *et al,* 1989), methylotrophic bacterium *Ancylobacter aquaticus* (van den Wijngaard *et al,* 1992), and a nitrogen-fixing hydrogen bacterium *Xanthobacter autotrophicus* (Janssen *et al.,* 1985) have been identified as micro-organisms capable of biodegrading 1,2-dichloroethane under aerobic conditions. In a batch experiment under anaerobic conditions, Bouwer and McCarty (1983) reported a 63% reduction in 25 days, but were unable to induce transformation in a flow-through system when initial 1,2-dichloroethane concentrations were 174 and 22 µg/L, respectively.

No biodegradation was observed after 35 days of incubation in an anoxic sediment-water system in which the initial concentration of 1,2-dichloroethane was 1.0 mg/L (pH not reported) (Jafvert and Wolfe, 1987).

Given its low sorption coefficient, 1,2-dichloroethane is not expected to adsorb appreciably to soil, suspended solids, or sediments. In one study, 1,2-dichloroethane rapidly percolated through sandy soil with a low organic-matter content; no degradation was observed, and 72 to 74% was reported to have volatilized (Wilson *et al.,* 1981). Based on its solubility in water, low K_{oc} value, and high mobility in soil, 1,2-dichloroethane may leach to groundwater.

1,2-Dichloroethane has a low bioaccumulation potential based on experimental data and modelling predictions. A measured bioconcentration factor (BCF) of 2 and a clearance half-life in tissues of less than two days were observed in freshwater bluegill,

Lepomis macrochirus, exposed to 95.6 µg/L 1,2-dichloroethane for 14 days (Barrows *et al.*, 1980). The experimental BCF is identical to the estimated value reported by Isnar and Lambert (1988).

2.3.2 Concentrations

1,2-Dichloroethane was detected in Canadian ambient and indoor air, surface waters, groundwaters, and drinking water but was not detected in sediments or food. No studies were identified in which levels of 1,2-dichloroethane in human breast milk, cigarette smoke, precipitation, soil, and aquatic or terrestrial biota in Canada were monitored.

The mean concentration in indoor air in approximately 750 residences in 10 provinces in 1991 was 1.8 µg/m^3, with a maximum single value of 27 µg/m^3, based on preliminary results of a pilot study (Otson *et al.*, 1992).

Volatile organic chemicals have been monitored by Environment Canada in the ambient air of 12 Canadian cities in six provinces since 1988 and 1,2-dichloroethane has been detected at all sites with a detection frequency of greater than 60%. Between 1988 and 1990, concentrations of 1,2-dichloroethane in 1412 samples ranged from non-detectable (below 0.1 µg/m^3) to a maximum of 2.78 µg/m^3 in Edmonton, Alberta (Environment Canada, 1992). Mean concentrations at these sites ranged between 0.07 and 0.28 µg/m^3. In contrast, 1,2-dichloroethane was measured above the detection limit of 0.1 µg/m^3 in only 6% of 389 samples from several cities in Ontario between 1989 and 1991 (OME, 1992b;c;d).

1,2-Dichloroethane was detected in less than 1% of samples in drinking water in recent surveys in Canada. Mean concentrations ranged from non-detectable (i.e., <0.05 µg/L) to 0.139 µg/L in drinking water from 85 sites across Ontario surveyed between 1988 and 1991 (Lachmaniuk, 1991). It was not detected (detection limits ranged up to 0.2 µg/L) in smaller surveys in Ontario (OME, 1988; Otson, 1987) and New Brunswick Ecobichon and Allen, 1990).

1,2-Dichloroethane was detected in 2% of samples of Canadian surface waters in the early 1980s. For example, 1,2-dichloroethane was not detected above the detection limit of 0.08 µg/L in 351 samples from Lake St. Clair, Lake Ontario, and the St. Lawrence, Niagara, and Detroit rivers (Kaiser and Comba, 1986; Kaiser *et al.*, 1983; Lum and Kaiser, 1986; Comba and Kaiser, 1985). 1,2-Dichloroethane was detected 300 m downstream of the 1,2-dichloroethane manufacturing plant in Sarnia, Ontario in two of three surface water samples taken from the St. Clair River in 1985; the maximum concentration detected was 16 µg/L (COARGLWQ, 1986).

1,2-Dichloroethane is not frequently detected in groundwater (1%) and appears primarily in leachates from hazardous waste sites (Lesage *et al*, 1990; Pakdel *et al.*, 1992).

Sediment samples taken downstream of the only two manufacturing sites of 1,2-dichloroethane in Canada (from the St. Clair River, Ontario, and from the North Saskatchewan River, Alberta) did not contain measurable amounts of 1,2-dichloroethane (detection limit of 0.01 µg/kg) in 1985 (Oliver and Pugsley, 1986; AEC, 1989). No other information was identified on levels of 1,2-dichloroethane in Canadian sediments.

1,2-Dichloroethane was not detected in two surveys of 34 food groups in Calgary and Windsor in 1991 and 1992, respectively (Enviro-Test Laboratories, 1991; 1992). The detection limits for the survey having the more sensitive analytical methodology were 0.005 µg/g for solids and 1 µg/L for liquids (Enviro-Test Laboratories, 1992). 1,2-Dichloroethane has only been detected in a few food items in total diet or market basket surveys in the United States (Heikes, 1987a;b; 1990; Daft, 1988; Entz *et al.*, 1982). No information was identified on levels of 1,2-dichloroethane in the breast milk of women in the general population in Canada or elsewhere.

No information was identified on levels or use of 1,2-dichloroethane in consumer products in Canada. The U.S. EPA reported that 1,2-dichloroethane was emitted in environmental chambers from various household and consumer products, including cleaning agents, pesticides, and glued wallpaper and carpets (Wallace *et al.*, 1987), and it has been detected in personal hygiene products in Germany (Bauer, 1981). However, the use of 1,2-dichloroethane in many consumer products has largely been discontinued in the United States (Drury and Hammons, 1979).

2.4 Toxicokinetics

1,2-Dichloroethane is readily absorbed following inhalation, ingestion, or dermal exposure. Absorption by the gastrointestinal tract is greater when the compound is administered in water rather than in corn oil (Withey *et al.*, 1983). Relative distribution of radioactivity (presumably as metabolites) was similar in rats administered a single oral dose of 150 mg/kg body weight (b.w.) and those exposed via inhalation to 150 ppm (600 mg/m^3) for 6 hours (Reitz *et al.*, 1982). 1,2-Dichloroethane appears to be metabolized via two principal pathways. The first pathway involves a saturable microsomal oxidation mediated by cytochrome P450 to 2-chloroacetaldehyde and 2-chloroethanol followed by conjugation with glutathione. The second pathway entails direct conjugation with glutathione to form S(2-chloroethyl)-glutathione, which may be non-enzymatically converted to a glutathione episulphonium ion that can form adducts with protein, DNA, or RNA. Metabolic saturation (pathway not identified) occurred sooner in rats administered 1,2-dichloroethane by gavage than by inhalation, as peak levels in blood were greater in rats administered 150 mg/kg (b.w.) by gavage than in those exposed to 150 ppm (600 mg/m^3) for 6 hours, although administration by gavage resulted in the formation of about twice the amount of total metabolites than that resulting from exposure by inhalation (Reitz *et al.*, 1982). Similarly, on the basis of a physiologically-based pharmacokinetic model, D'Souza *et al*, (1987;1988) predicted that lesser amounts of putatively toxic glutathione-conjugated metabolites would be generated in the liver and lung following inhalation of 1,2-dichloroethane than following ingestion.

2.5 Effects-related Information

2.5.1 Experimental Animals and In Vitro

1,2-Dichloroethane is not highly acutely toxic in experimental animals. For example, LC_{50}s for rats exposed via inhalation for 6 or 7.25 hours ranged from 1000 ppm (4000 mg/m^3) (Spencer *et al*., 1951*)* to 1650 ppm (6600 mg/m^3) (Bonnet *et al.*, 1980), while oral LD_{50}s for rats, mice, dogs, and rabbits ranged from 413 to 2500 mg/kg (b.w.) (Larionov and Kokarovtseva, 1976; Smyth, 1969; McCollister *et al.*, 1956; WHO, 1987; NIOSH, 1977; Munson *et al.*, 1982; Barsoum and Saad, 1934).

The results of short-term and subchronic studies in several species of experimental animals indicate that the liver and kidneys are the target organs. Most of these studies, however, were inadequate to serve as a basis for establishment of reliable effect levels, generally because of inadequate documentation and the limited range of endpoints examined in small groups of animals. In early studies in which rats, mice, rabbits, guinea pigs, cats, dogs, and monkeys were exposed to 1,2-dichloroethane by inhalation, there were morphological changes in the liver and kidneys at concentrations as low as 200 ppm (800 mg/m^3). No effects were observed, however, in any of these species at 100 ppm (400 mg/m^3), based on examination of a limited range of endpoints (Heppel *et al.*, 1946; Spencer *et al.*, 1951; Hofmann *et al.*, 1971). Relative weights of the brain, liver, and kidneys were increased in rats administered oral doses of 90 mg/[kg (b.w.)·d] 1,2-dichloroethane for 90 days (van Esch *et al.*, 1977), while the fat content of the liver was slightly increased in rats consuming approximately 80 mg/[kg (b.w.)·d] for 7 weeks (Alumot *et al.*, 1976). Increases in organ weights, without accompanying histopathological changes were observed in three strains of rats and one strain of mice administered 1,2-dichloroethane in the drinking water for 13 weeks {approximately equivalent to doses of 49 to 82 mg/[kg (b.w.)·d] in rats and 244 to 249 mg/[kg (b.w.)·d] in mice}, while histopathological changes in the kidneys were observed at higher doses. Administration of 1,2-dichloroethane in corn oil by gavage to rats resulted in more severe toxic effects than similar doses administered in drinking water. These effects included hyperplasia and inflammation of the forestomach at doses as low as 240 mg/[kg (b.w.)·d] (National Toxicology Program, 1991).

The carcinogenicity of 1,2-dichloroethane has been investigated in a few limited bioassays in experimental animals (limitations include short duration of exposure, high mortality, and inadequate dose levels). Little information was presented on non-neoplastic effects in these studies. No significant increase in the incidence of any type of tumour was reported in groups of 90 male or female Sprague-Dawley rats exposed to 0, 5, 10, 50, or 250 ppm (reduced to 150 ppm after a few days due to severe toxic effects and death and presented hereafter as 250/150 ppm) (0, 20, 40, 202, or 1012/607 mg/m^3) for 78 weeks and observed until spontaneous death. However, when only those rats in which tumours were confirmed histopathologically were considered, the incidence of fibromas and fibroadenomas of the mammary gland (combined) in females was 47/90, 27/90, 56/90, 33/90, 49/90, and 47/90 in controls, chamber controls, 5, 10, 50, and 250/150 ppm, respectively (significantly different at 5, 50, and

250/150 ppm). The authors stated that the differences in incidences were due mainly to different survival rates within the groups (mortality was high in most groups, although it was not related to concentration) (Maltoni *et al.,* 1980). In a separate report of results in small groups of rats of the same strain exposed to these concentrations for up to 18 months, there were significant changes in parameters indicative of effects on liver and kidney function at 50 and 250/150 ppm (Spreafico *et al.,* 1980). There was a non-significant increase in the incidence of mammary gland adenomas (4 versus 2 in controls) and fibroadenomas (21/50 versus 15/50) in female Sprague-Dawley rats exposed to 50 ppm (200 mg/m^3) for 2 years; no compound-related effects on body weight gain or mortality were observed (Cheever *et al.,* 1990). No increase in the incidence of any type of tumour was observed in Swiss mice exposed to 5, 10, 50, or 250/150 ppm 1,2-dichloroethane for 78 weeks (Maltoni *et al.,* 1980).

There were significant increases in the incidence of tumours at several sites in Osborne-Mendel rats administered time-weighted average doses of 47 or 95 mg/[kg (b.w.)·d] in corn oil by gavage for 78 weeks, followed by 32 weeks of observation. The incidence of squamous cell carcinomas of the forestomach was significantly increased in both groups of exposed males (0/60, 0/20, 3/50, and 9/50 in pooled (from concurrent studies) vehicle controls, matched vehicle controls, low and high dose groups, respectively). There were also significant increases in the incidence of hemangiosarcomas in exposed males (1/60, 0/20, 9/50, and 7/50) and females (0/59, 0/20, 4/50, and 4/50*).* The incidence of fibromas of the subcutaneous tissue was significantly increased in exposed males (0/60, 0/20, 5/50, and 6/50*).* In females, there were significant increases in the incidences of adenocarcinomas and fibroadenomas of the mammary gland [1/59, 0/20, 1/50, and 18/50 (adenocarcinomas), 5/59, 0/20, 14/50, and 8/50 (fibroadenomas) and 6/59,0/20, 15/50, and 24/50 (adenocarcinomas and fibroadenomas combined)]. Mortality was significantly higher in both males and females in the high dose group (the data on tumour incidences did not take early mortality into account) and there was a greater frequency of clinical signs of toxicity in exposed rats compared to controls. Chronic murine pneumonia was present in 60 to 94% of rats in each group, although the incidence was not related to dose. The incidence of acanthosis and hyperkeratosis was greater in exposed females than in controls. It was concluded that 1,2-dichloroethane was carcinogenic in this strain of rat, under the conditions of this study (National Cancer Institute, 1978).

A similar bioassay was conducted by the National Cancer Institute (1978) in which $B6C3F_1$ mice were administered time-weighted average doses of 97 or 195 mg/[kg (b.w.)·d] (males) and 149 or 299 mg/[kg (b.w.)·d] (females) in corn oil by gavage for 78 weeks, followed by 13 weeks of observation. The incidence of hepatocellular carcinomas was significantly increased in exposed males (4/59, 1/19, 6/47, and 12/48 in pooled vehicle controls, matched vehicle controls, and low and high dose groups, respectively). The authors noted, however, that the increase in the incidence of this tumour could not be convincingly attributed to the test chemical, due to the high variability of hepatocellular neoplasms among historical controls. The incidence of alveolar/bronchiolar adenomas was significantly increased in males in the high dose group (0/59,0/19, 1/47, and 15/48), and in both groups of exposed females

(2/60, 1/20, 7/50, and 15/48); one female in the high dose group had an alveolar/bronchiolar carcinoma. The incidence of mammary gland adenocarcinomas was significantly increased in females at both doses (0/60, 0/20, 9/50, and 7/48). The incidence of endometrial stromal polyp or endometrial stromal sarcoma (combined) in females was significantly elevated at both doses (0/60, 0/20, 5/49, and 5/47). There was a dose-related increase in mortality in females, but not in males (the data on tumour incidences did not take early mortality into account); in addition, body weight was decreased in females receiving the higher dose. It was concluded that 1,2-dichloroethane was carcinogenic in this strain of mice, under the conditions of this study (National Cancer Institute, 1978).

Hooper *et al.* (1980) have determined that the discrepancy between the results of the inhalation (Maltoni *et al.*, 1980) and ingestion (National Cancer Institute, 1978) bioassays was unlikely to be due to differences in the purity of the test chemical, as both groups of investigators used 1,2-dichloroethane of equivalent high purity (99.8% and 99.9%, respectively). These authors also determined that, when the administered doses in each of the studies were calculated over the lifespan of the animals, the highest level of exposure in the inhalation study was similar to doses that induced increased incidences of tumours in the gavage study.

1,2-Dichloroethane was applied to groups of 30 female non-inbred Ha:ICR Swiss mice, 3 times/week to the shaved dorsal skin at concentrations of 0,42, and 126 mg per application per mouse, in acetone, for 440 to 594 days. Histopathological examination was limited to the skin, liver, kidney, and any "abnormal-appearing tissues". The incidence of lung tumours (benign lung papillomas) was significantly increased at the higher dose (26/30 compared to 11/30 in vehicle controls, and 30/100 in naive controls) (van Duuren *et al,* 1979). Repeated intraperitoneal injections of 20, 40, or 100 mg/[kg (b.w.)·d] (3 times per week for a total of 24 injections) resulted in dose-related increases in the number of pulmonary adenomas per mouse in a susceptible strain, although none of these increases was significant (Theiss *et al.*, 1977). Concomitant exposure to inhaled 1,2-dichloroethane (50 ppm or 200 mg/m^3) and Disulfiram in the diet resulted in an increased incidence of intrahepatic bile duct cholangiomas and cysts in rats, compared to rats administered either compound alone or untreated controls (Cheever *et al.,* 1990). No potential to initiate or promote tumour development was evident in four bioassays (van Duuren *et al,* 1979; Milman *et al*, 1988; Storey *et al.*, 1986; Klaunig *et al.*, 1986), although the extent of histopathological examination was limited in these studies.

1,2-Dichloroethane has been consistently genotoxic in *in vitro* assays in prokaryotic systems, fungi, and mammalian (including human) cells (see supporting documentation). Similarly, results were consistently positive for genotoxic activity [as well as binding to DNA in several organs (Prodi *et al.,* 1986; Hellman and Brandt, 1986; Banerjee, 1988; Inskeep *et al.,* 1986; Baertsch *et al.,* 1991; Cheever *et al.,* 1990)] in *in vivo* studies in rats, mice, and insects *(Drosophila).* The extent of DNA alkylation in rats was 3 to 5 times greater following administration by gavage

[150 mg/kg (b.w.)] than inhalation (150 ppm or 600 mg/m^3), although absolute levels were low compared to controls (Reitz *et al.,* 1982).

Based on the results of a limited number of studies, there is no evidence that 1,2-dichloroethane is teratogenic in experimental animals (Rao *et al.,* 1980; Vozovaya, 1977; Kavlock *et al.,* 1979). There is also little convincing evidence that it induces reproductive or developmental effects at doses below those which cause other organ-specific effects in subchronic or chronic bioassays (Rao *et al.,* 1980; Vozovaya, 1977; Alumot *et al.,* 1976; Kavlock *et al.,* 1979; Lane *et al.,* 1982).

Although immunological effects have been reported in mice and rabbits exposed via inhalation to concentrations as low as 5 ppm (20 mg/m^3 for 3 hours) and 100 mg/m^3 (for up to 8 months) of 1,2-dichloroethane, respectively (Sherwood *et al.,* 1987; Shmuter, 1977), and in mice ingesting 4.9 mg/[kg (b.w.)·d] (for 14 days) (Munson *et al.,* 1982), no effects were observed in one study in rats exposed to up to 200 ppm (800 mg/m^3) for 12 days. It is therefore difficult to draw meaningful conclusions regarding the potential immunotoxicity of 1,2-dichloroethane due to the lack of consistency in observed effects. In early studies, neurological effects have been reported in rats, guinea pigs, and rabbits exposed to high airborne concentrations [e.g., 3000 ppm (12000 mg/m^3) for 7 hours] (Spencer *et al,* 1951; Heppel *et al.,* 1945), whereas no clinical neurological effects were observed in dogs exposed to 400 ppm (1600 mg/m^3) for 8 months (Heppel *et al.,* 1946).

2.5.2 Humans

No adequate investigations of the potential carcinogenicity of 1,2-dichloroethane in exposed human populations have been identified. In a case-control study, the exposure of 21 male petrochemical plant workers who had died due to cancer of the brain was compared to that of controls. The proportion of cases who had been exposed to 1,2-dichloroethane did not differ from the proportion of controls who had been exposed to the substance (Austin and Schnatter, 1983a). Similarly, there was no increase in mortality due to brain cancer in an accompanying cohort study of 6588 workers at this plant, although exposure to 1,2-dichloroethane was not specifically considered in this study (Austin and Schnatter, 1983b).

In an ecological study, the average annual age-adjusted incidence (1969 to 1981) of colon and rectal cancer was statistically significantly greater in men aged $\geq$ 55 years whose drinking water contained $\geq$0.1 µg/L 1,2-dichloroethane than in those of similar socioeconomic status whose drinking water contained <0.1µg/L [222.8/100 000 versus 170.3/100 000 (193 and 633 cases) and 126.5/100 000 versus 92.9/100 000 (106 and 337 cases), respectively]. On the basis of these results, the authors suggested that cancer incidence may be elevated in populations consuming water from wells subject to anthropogenic contamination (with 1,2-dichloroethane and other substances) (Isacson *et al.,* 1985).

2.5.3 Ecotoxicology

Information has been identified on the acute and chronic toxicity of 1,2-dichloroethane for a number of trophic levels and taxa in the aquatic environment, from bacteria through to fish and amphibians. Available toxicological data for terrestrial organisms are limited to invertebrates, domestic birds, and plants.

Toxicity bioassays were conducted by Blum and Speece (1991) on three groups of aquatic bacteria: methanogens; aerobic heterotrophs; and *Nitrosomonas.* Inhibition of gas production (methanogens), oxygen uptake (aerobic heterotrophs), and ammonia consumption *(Nitrosomonas)* were the endpoints assessed in this study. *Nitrosomonas* and methanogens were more sensitive (IC_{50} 29 and 25 mg/L 1,2-dichloroethane, respectively) than aerobic heterotrophs (IC_{50} 470 mg/L) (Blum and Speece, 1991). For the bacteria, *Pseudomonas putida,* the nominal 16-h EC_{50} for the onset of cell multiplication inhibition was 135 mg/L at 25^0C (Bringmann and Kühn, 1980).

Freshwater algal species were exposed to 1,2-dichloroethane and the onset of cell multiplication inhibition was determined. The blue-green algae, *Microcystis aeruginosa,* was seven times more sensitive to 1,2-dichloroethane than the green algae, *Scenedesmus quadricauda,* with nominal 7-day EC_{50}s of 105 and 710 mg/L, respectively, at 27^0C (Bringmann and Kühn, 1978). Based on bioluminescence, the five-minute IC_{50} was 700 mg/L of 1,2-dichloroethane in a Microtox test with *Photobacterium phosphoreum* (Blum and Speece, 1991). No adequate toxicity studies involving marine algae were identified.

Of the acute and chronic toxicity studies identified pertaining to freshwater invertebrates, *Daphnia magna* were the most sensitive species. Under static test conditions, the measured 48-h LC_{50}s for fed and unfed first ins tar *Daphnia* were 320 and 270 mg/L, respectively; the 48-h EC_{50} values based on complete immobilization were 180 and 160 mg/L for fed and unfed organisms, respectively (Richter *et al,* 1983). Furthermore, Richter *et al.* (1983) examined the reproductive success and the length of first instar *Daphnia magna* during a 28-day flow-through test. For reproductive success, the measured lowest-observed-effect-level (LOEL) and no-observed-effect-level (NOEL) were 20.7 and 10.6 mg/L, respectively, and for growth, the measured LOEL and NOEL were 71.7 and 41.6 mg/L, respectively (Richter *et al.,* 1983).

Only two acute toxicity studies on marine invertebrates were identified and both were conducted under static test conditions. The nominal 24-h EC_{50} for immobilization of 30-h posthatch larvae of the brine shrimp, *Artemia salina,* was 93.6 mg/L (Foster and Tullis, 1984). For marine adult shrimp, *Crangon crangon,* the measured 24-h LC_{50} was 170 mg/L (Rosenberg *et al, 1975).*

The embryos and larvae of the northwestern salamander *(Ambystoma gracile)* and the leopard frog *(Rana pipiens)* were continuously exposed to 1,2-dichloroethane from within 30 minutes of fertilization (embryos) and maintained through four days posthatching (larvae) (Black *et al.,* 1982). The LC_{50}s for the salamander at the day of hatching (day 5) and 4-days posthatching (day 9) were 6.53 and 2.54 mg/L, respectively;

the measured LOEL for 23% reduction in egg hatchability was 0.99 mg/L. The measured 5-day and 9-day LC_{50}s for the frog were 4.52 and 4.40, respectively, while the 5-day posthatch LOEL was 1.07 mg/L (Black *et al,* 1982).

Acute toxicity studies have been conducted on several species of freshwater fish. The most sensitive freshwater fish were 2- to 3-month old guppies *(Poecilia reticulata)* with a nominal 7-day LC_{50} of 106 mg/L of 1,2-dichloroethane under static-renewal test conditions (Konemann, 1981). The acute response of 30-day old fathead minnows *(Pimephales promelas)* to 1,2-dichloroethane has been investigated in three studies under flow-through conditions and the resulting measured 96-h LC_{50}s were 118, 116, and 136 mg/L (Veith *et al.,* 1983; Walbridge *et al.,* 1983; Geiger *et al*, 1985). The only adequate acute toxicity study on marine fish involved tidewater silversides *(Menidia beryllina)* in which a nominal 96-h LC_{50} of 480 mg/L was reported under static test conditions (Dawson *et al.,* 1975/77).

In a long-term, flow-through study of the early life stages of fathead minnows *(Pimephales promelas),* there were no toxic effects on egg hatchability or larval survival and deformity at 29 mg/L NOEL); however, larval growth was significantly reduced by 62% at 59 mg/L (LOEL) (Benoit *et al.,* 1982). The embryos and larvae of rainbow trout *(Oncorhynchus mykiss)* were continuously exposed to 1,2-dichloroethane under flow-through conditions from within 30 minutes of fertilization (embryos) and maintained until four days posthatching. The resulting EC_{50} for hatchability and 27-day LC_{50} post-hatch survival were both 34 mg/L and the LOEL for a 24% reduction in egg hatchability was 3.49 mg/L (Black *et al.,* 1982). After 21 days of continuous exposure to 1,2-dichloroethane, 46% egg mortality of coho salmon *(Oncorhynchus kisutch)* occurred at 150 mg/L; 100% alevin mortality occurred 9 days after hatching at 320 mg/L (Reid *et al.,* 1982). In addition, at 56 mg/L of 1,2-dichloroethane, premature hatching was observed and, within one week of hatching, sublethal effects which included lethargy and loss of equilibrium were observed in alevins exposed to 56 mg/L; 100% mortality occurred 9 days after hatching.

1,2-Dichloroethane has been teratogenic to both freshwater and marine organisms. Teratogenic effects, expressed as surviving larvae with gross, debilitating abnormalities, were observed in the nauplii of the marine brine shrimp *(Artemia salina)* at concentrations between 0.25 and 25 mg/L (Kerster and Schaeffer, 1983); the northwestern salamander *(Ambystoma gracile)* and leopard frog *(Rana pipiens)* larvae at 21.4 and 21.9 mg/L, respectively (Black *et al,* 1982); and rainbow trout larvae *(Oncorhynchus mykiss)* at 34.4 mg/L (Black *et al.,* 1982).

Only one toxicity study was identified in which terrestrial plants were exposed to 1,2-dichloroethane. 1,2-Dichloroethane vapour was both lethal and mutagenic to barley kernels (two-rowed variety, *Bonus)* after exposure to 3 mg/m^3 for 24 hours (Ehrenberg *et al.,* 1974). In an acute contact test, the 48-h LC_{50} for earthworms *(Eisenia fetida)* exposed to 1,2-dichloroethane-treated filter paper was 60 $\mu g/m^2$ (Neuhauser *et al*., 1985).

For two years, male and female leghorn chickens were fed mash fumigated with two levels of 1,2-dichloroethane. The chickens were then examined for serum composition, growth, semen characteristics, and fertility. Only female chickens were affected by 1,2-dichloroethane, as the weight of eggs was significantly reduced at 250 mg/kg of 1,2-dichloroethane and both the number and weight of eggs were reduced at 500 mg/kg of 1,2-dichloroethane (Alumot *et al*, 1976).

3.0 Assessment of "Toxic" Under CEPA

3.1 CEPA 11(a) Environment

In Canada, 1,2-dichloroethane is used primarily as an intermediate in the synthesis of vinyl chloride monomer and, in small quantities, in the manufacture of motor antiknock fluids for export. 1,2-Dichloroethane enters the Canadian environment primarily through air emissions, predominantly from the production of 1,2-dichloroethane and vinyl chloride monomer. Other sources of entry to the Canadian environment include air emissions and leachates from waste disposal sites, the discharge of contaminated effluents from industries that use or produce 1,2-dichloroethane, effluents from contaminated groundwater treatment, and long-range atmospheric transport from other countries.

1,2-Dichloroethane is moderately persistent in air but it is not persistent in water, sediment, or soil because of its high volatility and its low sorption coefficient. 1,2-Dichloroethane is not expected to bioconcentrate in organisms or biomagnify within food chains because of its very low bioconcentration factor. Low concentrations of 1,2-dichloroethane have been detected in Canadian ambient air and water, but not sediments. 1,2-Dichloroethane has not been measured in aquatic or terrestrial biota in Canada.

Since most 1,2-dichloroethane is released to, and persists in, the atmosphere, terrestrial organisms will have the highest potential for exposure. Based on limited data, the most sensitive terrestrial organism was barley *(Bonus* variety) where, after 24 hours, 1,2-dichloroethane vapour was both lethal and mutagenic to kernels at 3 mg/m^3. The maximum ambient air concentration found in Canada was 2.78 µg/m^3, which is 1080 times less than the effects threshold of 3 mg/m^3. Therefore, it is unlikely that terrestrial organisms are at risk from exposure to 1,2-dichloroethane in ambient air.

The most sensitive freshwater organism to long-term exposure of 1,2-dichloroethane was the northwestern salamander *(Ambystoma gracile),* in which 9-day larval survival (4-days posthatch) was reduced at 2.54 mg/L. Using a factor of 20 to convert this value to a chronic NOEL for a non-persistent, non-bioaccumulative substance, yields an estimated effects threshold of 130 µg/L. The maximum concentration detected in Canadian ambient surface waters (16 µg/L), located downstream of a 1,2-dichloroethane/vinyl chloride monomer manufacturing plant, is eight times less than the estimated effects threshold.

Identified data on exposure and effects are insufficient to evaluate toxicity to terrestrial birds and mammals.

Therefore, on the basis of available data, 1,2-dichloroethane is not considered to be entering the environment in a quantity or concentration or under conditions that are having a harmful effect on the environment.

3.2 CEPA 11(b) Environment on Which Human Life Depends

1,2-Dichloroethane absorbs infrared light at wavelengths critical to global warming; however, because of the relatively low rate of release of 1,2-dichloroethane to the atmosphere and the low atmospheric concentration, it is unlikely that 1,2-dichloroethane will contribute significantly to global warming. Furthermore, as 1,2-dichloroethane has an atmospheric ozone-depleting potential of less than 0.001 relative to CFC-11, 1,2-dichloroethane is not expected to contribute significantly to stratospheric ozone depletion.

Therefore, on the basis of available data, 1,2-dichloroethane is not considered to be entering the environment in a quantity or concentration or under conditions that constitute a danger to the environment upon which human life depends.

3.3 CEPA 11 (c) Human Life or Health

3.3.1 Population Exposure

Estimated daily intake (on a body weight basis) of 1,2-dichloroethane for various age groups of the general population of Canada, and the assumptions upon which they are based, are presented in Table 1. Total daily intake of 1,2-dichloroethane from ambient and indoor air, drinking water, and food is estimated to range from 0.43 to 0.70 µg/[kg (b.w.)·d], with indoor air being the major source of exposure, and only minor amounts being contributed by ambient air and drinking water. It should be noted, however, that these estimates are based on the assumption that intake of 1,2-dichloroethane in food is negligible, as would be expected based on its low potential for bioaccumulation. If 1,2-dichloroethane was assumed to be present at concentrations up to the limits of detection in a recent survey of foodstuffs in Windsor in which the compound was not detected in any sample, the values for estimated total intake would not be substantially greater.

3.3.2 **Effects**

Due to concomitant exposure to other substances which may have contributed to the observed effects in the few limited identified studies in exposed human populations (Isacson *et al.*, 1985; Austin and Schnatter, 1983a), available epidemiological data are considered inadequate to assess the carcinogenicity of 1,2-dichloroethane. In addition, the few bioassays in which the carcinogenicity of 1,2-dichloroethane in experimental animals has been investigated have all been limited. Few increases in tumour incidence have been observed in these limited bioassays following inhalation of relatively low concentrations of 1,2-dichloroethane. There was no significant increase in the incidence of any specific type of tumour in Swiss mice or Sprague-Dawley rats exposed to up to 150 ppm (600 mg/m^3) of 1,2-dichloroethane by inhalation for 78 weeks and observed until spontaneous death. (Animals were initially exposed to 250 ppm (1000 mg/m^3), but

Table 1 Estimates of the Average Daily Intake of 1,2-Dichloroethane for the General Population in Canada

Medium*		Estimated Intake {µg/[kg (b.w.)·d]}				
		Age				
		0 to 6 mo[a]	7 mo to 4 yr[b]	5 to 11 yr[c]	12 to 19 yr[d]	20 to 70y[e]
Air	Ambient[f]	0.003 to 0.01	0.004 to 0.02	0.005 to 0.02	0.004 to 0.02	0.004 to 0.02
	Indoor[g]	0.43	0.58	0.67	0.55	0.49
Drinking Water[h]		<0.005 to 0.01	<0.003 to 0.009	<0.002 to 0.005	<0.001 to 0.003	<0.001 to 0.003
Total Intake		0.43 to 0.45	0.58 to 0.61	0.68 to 0.70	0.55 to 0.57	0.49 to 0.51

a Assumed to weigh 7 kg, breathe 2 m^3 of air, and drink 0.75 L of water per day (Environmental Health Directorate, 1992).

b Assumed to weigh 13 kg, breathe 5 m^3 of air, and drink 0.8 L of water per day (Environmental Health Directorate, 1992).

c Assumed to weigh 27 kg, breathe 12 m^3 of air, and drink 0.9 L of water per day (Environmental Health Directorate, 1992).

d Assumed to weigh 57 kg, breathe 21 m^3 of air, and drink 1.3 L of water per day (Environmental Health Directorate, 1992).

e Assumed to weigh 70 kg, breathe 23 m^3 of air and drink 1.5 L of water per day (Environmental Health Directorate, 1992).

f Based on a range of mean concentrations of 1,2-dichloroethane of 0.07 to 0.28 µg/m^3 in ambient air from 12 Canadian cities across 6 provinces (Environment Canada, 1992), assuming 4 of 24 hours are spent outdoors daily (Environmental Health Directorate, 1992).

g Based on a mean concentration of 1,2-dichloroethane in indoor air of 1.8 µg/m^3 in a national pilot study of approximately 750 residences in 10 provinces across Canada (Otson *et al*, 1992) assuming 20 of 24 hours are spent indoors daily (Environmental Health Directorate, 1992).

h Based on the range of mean concentrations of 1,2-dichloroethane [non-detectable (i.e., <0.05) to 0.139 µg(L] in the 1990 Drinking Water Surveillance Program in Ontario (Lachmaniuk, 1991). Similar results have been reported in recent surveys in Ontario and New Brunswick (Otson, 1987; OME, 1988; Ecobichon and Allen, 1990).

* Intake of 1,2-dichloroethane in food is assumed to be negligible, based on its low potential for bioaccumulation. If it was assumed to be present at concentrations up to the limits of detection in a recent survey of foodstuffs in Windsor in which the compound was not detected in any sample (Enviro-Test Laboratories, 1992), the values for estimated total intake would not be substantially greater. Available data were insufficient to estimate intake from soil.

the level of exposure was reduced after several days due to toxicity, including death). Although there were small increases in the incidence of fibromas and fibroadenomas of the mammary gland, the authors stated that the differences in the incidences were likely due to the variations in survival rates among the groups. Mortality was high in this study, although it was not related to concentration (Maltoni *et al,* 1980). No significant increases in the incidence of tumours were reported in rats exposed by inhalation to 50 ppm (200 mg/m^3) for 2 years, although there was a non-significant increase in the incidence of benign mammary gland tumours in exposed females (Cheever *et al,* 1990). However, the sensitivity of this investigation may have been compromised, based on the lack of convincing evidence of compound-related toxicity at the only concentration to which animals were exposed.

In contrast, there has been convincing evidence of increases in tumour incidence in two species (rats and mice) following ingestion (gavage in corn oil) and in mice following dermal application. Significant increases in the incidence of tumours at several sites [including squamous cell carcinomas of the stomach (males), hemangiosarcomas (males and females), fibromas of the subcutaneous tissue (males), and adenocarcinomas and fibroadenomas of the mammary gland (females)] were observed in Osborne-Mendel rats administered time-weighted average daily doses of 47 or 95 mg/[kg (b.w.)·d] by gavage for 78 weeks. The incidence of squamous cell carcinomas of the stomach and mammary gland tumours increased with dose (National Cancer Institute, 1978). Similar increases in the incidences of tumours at multiple sites [including alveolar/bronchiolar adenomas (males and females), mammary gland adenocarcinomas (females) and endometrial stromal polyp or endometrial stromal sarcoma combined (females), and hepatocellular carcinomas (males)] occurred in mice administered time-weighted average daily doses of 97 or 195 mg/[kg (b.w.)·d] (males) or 149 or 299 mg/[kg (b.w.)·d] (females) by gavage for 78 weeks. The incidences of alveolar/bronchiolar adenomas and hepatocellular carcinomas increased in a dose-related manner, although the authors noted that the increase in the incidence of hepatocellular carcinomas in males could not be convincingly attributed to 1,2-dichloroethane due to the high variability of the incidence of this tumour in historical controls (National Cancer Institute, 1978). Results of an additional study in which rats were fed diets containing 1,2-dichloroethane at concentrations approximately equivalent to daily doses of up to 17.5 mg/[kg (b.w.)·d] (Alumot *et al,* 1976) do not contribute meaningfully to assessment of the weight of evidence of carcinogenicity due to inadequacy of examination of relevant endpoints (i.e., histopathological examinations do not appear to have been conducted).

The incidence of benign lung papillomas was significantly increased in mice following dermal application of 1,2-dichloroethane for 440 to 594 days (van Duuren *et al,* 1979), even though the sensitivity of this study to detect an increase in the incidence of tumours was limited by the small group sizes (i.e., 30) and the limited histopathological examination. Non-significant increases in the number of pulmonary adenomas per animal were reported in a limited bioassay designed to investigate the potential of intraperitoneally injected 1,2-dichloroethane to induce this tumour in small groups of a susceptible strain of mice (Theiss *et al,* 1977). In addition, concomitant exposure of rats to inhaled 1,2-dichloroethane and Disulfiram in the diet resulted in an

increase in the incidence of intrahepatic bile duct cholangiomas and cysts compared to rats exposed to either compound alone (Cheever *et al*, 1990).

1,2-Dichloroethane was consistently genotoxic in numerous *in vitro* and *in vivo* assays. Covalent binding of 1,2-dichloroethane to DNA in the lung, liver, kidney, and stomach has been consistently reported in rats and mice exposed *in vivo* (Prodi *et al.*, 1986; Hellman and Brandt, 1986; Banerjee, 1988; Inskeep *et al.*, 1986; Baertsch *et al.*, 1991; Cheever *et al.*, 1990).

Thus, in view of the sufficient weight of evidence of carcinogenicity in two species of experimental animals and supporting data on genotoxicity, 1,2-dichloroethane is categorized in Group II ("Probably Carcinogenic to Humans") of the classification scheme developed for the determination of "toxic" under Paragraph 11(c) of CEPA (Environmental Health Directorate, 1992).

For such substances, where data permit, estimated total daily intake by the general population in Canada is compared to quantitative estimates of carcinogenic potency to characterize risk and provide guidance for further action under the Act (i.e., analysis of options to reduce exposure). The carcinogenic potency of 1,2-dichloroethane was derived based on the increased incidence of squamous cell carcinomas of the stomach, hemangiosarcomas, fibromas of the subcutaneous tissue, and adenocarcinomas and/or fibromas of the mammary gland in Osborne-Mendel rats exposed orally by gavage, as well as the increased incidence of alveolar/bronchiolar adenomas, hepatocellular carcinomas, mammary gland adenocarcinomas, and endometrial stromal polyp or sarcoma in similarly exposed $B6C3F_1$ mice; data from both the matched (same study) and pooled (concurrent studies) vehicle controls were incorporated (National Cancer Institute, 1978). It should be noted, however, that these animals were exposed for a fixed period of time (i.e., 78 weeks) and then observed for several additional weeks, in contrast to studies normally used in the quantitative estimation of carcinogenic potency in which animals are sacrificed immediately after exposure for a fixed duration (usually 104 weeks). In addition, mortality was higher at the high dose in female mice and rats of both sexes than in other dose groups in this study, and the data on tumour incidence do not take into account the increased early mortality (i.e., the incidences of several tumours may have been higher had all the animals survived long enough to develop cancer). Therefore, these high dose groups were not included in the derivation of quantitative estimates of carcinogenic potency, since, for most of the types of tumours, inclusion of these groups would result in an underestimation of the potency. *

Based on multistage modelling of these data, amortized for continuous exposure for a standard duration of 104 weeks and corrected for the expected rate of increase in tumour formation in rodents in a standard bioassay of 104 weeks, estimates of carcinogenic potency ($TD_{0.05}$) range from 6.2 to 297 mg/[kg (b.w.)·d]. Incorporation of

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* If data in female mice and male and female rats receiving the higher dose were included in the calculation of potency estimates (which may be appropriate for those tumours for which the incidence at the higher dose was substantially greater than the lower dose, even though early mortality was high), the values would fall within the range presented here.

a correction factor for the differences in body surface area between rodents and humans was not considered appropriate, as it is likely that the carcinogenicity of 1,2-dichloroethane is due to a metabolite, rather than to the parent compound. Calculated exposure/potency indices for the range of estimated total daily intakes by the general population of 0.43 to 0.70 µg/[kg b.w.)·d] are $1.5x\ 10^{-6}$ to $1.1\ x\ l0^{-4}$. The priority for further action (i.e., analysis of options to reduce exposure) is therefore considered to be low to moderate.

It should be noted, however, that these values are most likely overestimated, since the $TD_{0.05}$s were based on a study in which the experimental animals were administered bolus doses of l,2-dichloroethane by gavage, whereas exposure in the general population is likely to be mostly via inhalation. Based on available data, the potency of 1,2-dichloroethane to induce tumours appears to be less following inhalation than ingestion of bolus doses, due most likely to inter-route variations in toxicokinetics. *

Since 1,2-dichloroethane has been classified as being "Probably Carcinogenic to Humans", it has been concluded that this substance may enter the environment in quantities or concentrations or under conditions that may constitute a danger in Canada to human life or health.

This approach is consistent with the objective that exposure to non-threshold toxicants should be reduced wherever possible, and obviates the need to establish an arbitrary *"de minimis"* level of risk for the determination of "toxic" under CEPA.

3.4 Conclusion

Based on these considerations, it has been concluded that 1,2-dichloroethane is not entering the environment in a quantity or concentration or under conditions that are having a harmful effect on the environment or that constitute a danger to the environment upon which human life depends. However, it has been concluded that 1,2-dichloroethane occurs at concentrations that may constitute a danger in Canada to human life or health.

* Available data are consistent with the hypothesis that the detoxification pathway of metabolism (i.e., microsomal oxidation and glutathione conjugation) is saturated sooner in animals exposed by gavage, and, consequently, that more of the putatively toxic metabolites (i.e., glutathione episulphonium ions) are formed when 1,2-dichloroethane is administered by this route, compared to inhalation (Reitz *et al.*, 1982; D'Souza *et al*, 1987; 1988). Indeed, although absolute levels of DNA alkylation were low in rats exposed to 1 ,2-dichloroethane by gavage or inhalation compared to controls, the levels of DNA alkylation were three to five times greater following gavage than inhalation (Reitz *et al.*, 1982).

4.0 Recommendations for Research and Evaluation

1. Since the general population is exposed to 1,2-dichloroethane primarily in indoor air, it is recommended that the sources in this medium be characterized.

2. In order to derive estimates of the carcinogenic potency of inhaled 1,2-dichloroethane, it is recommended that additional carcinogenicity bioassays of improved sensitivity be conducted.

3. Additional studies on the effects of 1,2-dichloroethane on the immune system would be desirable.

4. To characterize the sources of release of 1,2-dichloroethane to the Canadian ambient air, continued air monitoring is desirable as well as an investigation into the importance of long-range atmospheric transport.

5. Information regarding the chronic effects of 1,2-dichloroethane on terrestrial plants would be desirable.

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