Screening Assessment Report

Ethene, 1,1-dichloro-
(1,1-Dichloroethene)

Chemical Abstracts Service Registry Number
75-35-4

Environment Canada
Health Canada

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Synopsis

Pursuant to paragraphs 68(*b*) and 68(*c*) of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), the Ministers of the Environment and of Health have conducted a screening assessment of Ethene, 1,1-dichloro- (1,1-dichloroethene), Chemical Abstracts Service Registry Number 75-35-4. 1,1-Dichloroethene was identified as a high priority for assessment of human health risk because it has been classified by other agencies on the basis of carcinogenicity.

1,1-Dichloroethene is a chlorinated organic compound that was used in solvents and as an intermediate in a variety of chemical processes. Based on a survey issued under section 71 of CEPA 1999, between 10 and 100 tonnes of 1,1-dichloroethene were manufactured and imported into Canada in 2000. However, it is no longer produced or imported into Canada. Small amounts of 1,1-dichloroethene are created unintentionally in several industrial processes; most of this material is reformed into other substances within the facilities.

Globally, 1,1-dichloroethene is used primarily as an intermediate in the manufacture of polyvinylidene chloride polymers and copolymers, which may in turn be used in a variety of end products such as food plastic wrap, carpet latex backing, fire- and ignition-resistant clothing, vapour barriers for insulation, paper and board coatings, and photographic film. 1,1-Dichloroethene may persist as an unintended manufacturing residue in some of these items that may be present in Canadian commerce. 1,1-Dichloroethene may also be used in the production of hydrochloro-fluorocarbons, chloroacetyl chloride, and latex and resins, as an aid in ore flotation, as a solvent in paint and varnish remover, and as a vapour degreaser and industrial cleaning agent.

1,1-Dichloroethene is a reportable substance to the National Pollutant Release Inventory (NPRI); reported releases have steadily declined from 87 kg in 2000 to 1 kg in 2003. Since 2003, no companies have reported releases of 1,1-dichloroethene to the NPRI.

1,1-Dichloroethene can be released during the breakdown of polyvinylidene chloride products and during the abiotic and biotic decomposition of the drycleaning and degreasing solvents 1,1,1-trichloroethane, 1,1,2,2-tetrachloroethene (tetrachloroethene or perchloroethylene), 1,1,2-trichloroethene and 1,2-dichloroethane. Many of these solvents are no longer used in Canada and disposal practices have improved, such that significant new sources of 1,1-dichloroethene in groundwater and soil are unlikely. Recent monitoring data show that 1,1-dichloroethene is present in urban air at very low concentrations, often just above analytical detection limits.

Based on experimental and modelled data for 1,1-dichloroethene, the substance is expected to degrade readily in air, soil and water. Based on its physical and chemical properties and predictions from bioaccumulation models, the substance is not expected to bioaccumulate in aquatic organisms. Therefore, 1,1-dichloroethene does not meet criteria for persistence and bioaccumulation potential as set out in the *Persistence and Bioaccumulation Regulations.* In addition, available empirical ecotoxicity data (for mammals, aquatic and terrestrial plants, invertebrates and vertebrates) indicate that 1,1-dichloroethene is not highly hazardous to non-human organisms.

Based on the low concentrations measured or expected in the Canadian environment and low hazards of the substance there is very low likelihood of ecological harm from 1,1-dichloroethene in the Canadian environment, especially given the low persistence and potential for bioaccumulation of 1,1-dichloroethene and decreasing uses and releases.

Based on the information available, it is concluded that 1,1-dichloroethene is not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

The general population exposure to 1,1-dichloroethene is mainly from indoor air and possibly food. A comparison of the lowest critical inhalation effect level for non-cancer effects with the highest median concentration of 1,1-dichloroethene in indoor air from recent studies in Canada, and a comparison of the critical oral effect level for non-cancer effects and the upper-bounding estimate of daily intake, result in margins of exposure which are considered adequate to address uncertainties in the health effects and exposure databases for chronic non-cancer effects. Additionally, for non-cancer effects based on a less than chronic period, a comparison of the lowest critical inhalation effect level for such periods with the highest 95th percentile concentration of 1,1-DCE in air from recent studies in Canada results in a margin of exposure which is considered adequate to address uncertainties in the health effects and exposure databases.

A critical effect for characterization of risk to 1,1-dichloroethene is carcinogenicity. Following lifetime inhalation of 1,1-dichloroethene at high concentrations, mice developed renal tumours. A comparison of the critical effect level for cancer and the upper-bounding estimate of daily intakes results in margins of exposure which are considered adequate to address uncertainties in the health effects and exposure databases for cancer effects. Additionally, available information suggests that the mode of tumour induction in experimental animals may not be relevant to humans.

Based on the information available, it is concluded that 1,1-dichloroethene is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

Based on available information for environmental and human health considerations, it is concluded that 1,1-dichloroethene does not meet the criteria set out in section 64 of the *Canadian Environmental Protection Act, 1999*.

Introduction

This screening assessment was conducted pursuant to section 68 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada 1999). This section of the Act authorizes the Ministers of the Environment and of Health to conduct assessments of substances to determine whether they meet or may meet the criteria set out in section 64 of the Act.

Screening assessments focus on information critical to determining whether a substance presents, or may present, a risk to the environment or to human health, according to the criteria set out in section 64 of CEPA 1999. Screening assessments examine scientific information and develop conclusions by incorporating a weight-of-evidence approach and precaution.[1] Ethene, 1,1-dichloro- (1,1-DCE) (Chemical Abstracts Service Registry Number 75-35-4) was identified as a priority for assessment of human health risk because it has been classified by other agencies on the basis of carcinogenicity.

The 2005 *State of the Science Report for a Screening Health Assessment* of 1,1-DCE was posted on the Health Canada website on November 3, 2005. The *State of the Science Report for a Screening Health Assessment* was externally reviewed by staff of Toxicology Advice and Consulting Limited and Toxicology Excellence in Risk Assessment, V.C. Armstrong (consultant) and P. Price (The Lifeline Group Inc.) for adequacy of data coverage and defensibility of the conclusions. The external comments were taken into consideration in drafting the *State of the Science Report*. The health screening assessment included here is an update of the *State of the Science Report* and, since limited new information was available, has not been subsequently peer reviewed.

This screening assessment includes consideration of information on chemical properties, hazards, uses and exposure. Data relevant to the screening assessment of this substance were identified in original literature, review and assessment documents, stakeholder research reports and from recent literature searches, up to December 2009 for ecological sections of the document and September 2009 for human health sections of the document. Also, data initially reported from draft reports of two Health Canada studies were updated in this assessment based on finalized reports published in 2010. In addition, an industry survey was conducted in 2000 through a *Canada Gazette* notice issued under authority of section 71 of CEPA 1999. This survey collected data on the Canadian manufacture and import of the DSL pilot project substances (Environment Canada 2001a). Key studies were critically evaluated; modelling results may have been used to reach conclusions.

[1] A determination of whether one or more of the criteria of section 64 are met is based upon an assessment of potential risks to the environment and/or to human health associated with exposures in the general environment. For humans, this includes, but is not limited to, exposures from ambient and indoor air, drinking water, foodstuffs, and the use of consumer products. A conclusion under CEPA 1999 on the substances in the Chemicals Management Plan (CMP) is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the *Controlled Products Regulations*, which is part of the regulatory framework for the Workplace Hazardous Materials Information System [WHMIS] for products intended for workplace use. Similarly, a conclusion based on the criteria contained in section 64 of CEPA 1999 does not preclude actions being taken under other sections of CEPA 1999 or other Acts.

Evaluation of risk to human health involves consideration of data relevant to estimation of exposure (non-occupational) of the general population, as well as information on health hazards. Decisions for human health are based on the nature of the critical effect and/or margins between conservative effect levels and estimates of exposure, taking into account confidence in the completeness of the identified databases on both exposure and effects, within a screening context. The screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents a summary of the critical information upon which the conclusion is based.

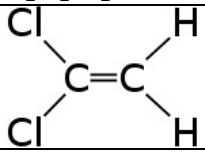
This final screening assessment was prepared by officials in the Existing Substances Programs at Health Canada and Environment Canada. As mentioned above, the *State of the Science Report for a Screening Health Assessment* was previously externally reviewed. The ecological component of this assessment has undergone external written scientific peer review/consultation and comments received were considered in the production of this report. Comments on the technical portions relevant to human health were received from BIBRA Toxicology Advice and Consulting, The LifeLine Group, and two independent consultants (Dr. Marla Shapiro and Dr. V.C. Armstrong). Additionally, the draft of this screening assessment was published on December 16, 2011 and subject to a 60-day public comment period. Although external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment Canada.

The critical information and considerations upon which this assessment is based are summarized below.

Substance Identity

Ethene, 1,1,-dichloro-, also known as 1,1-dichloroethene, will be referred to in this assessment by its acronym, 1,1-DCE. Information on its identity is presented in Table 1.

Table 1. Substance identity for 1,1-DCE

CAS RN	75-35-4
DSL name	Ethene, 1,1-dichloro-
NCI names[1]	Ethene, 1,1-dichloro- (TSCA, DSL, AICS, SWISS, PICCS, ASIA-PAC, NZIoC) 1,1-Dichloroethylene (EINECS) 1,1-Dichloroethene (ENCS, ECL) Vinylidene chloride (ENCS, PICCS)
Other names	Sconatex; Diofan A 565S; Ethene, 1,1-dichloro; Ethylene, 1,1-dichloro-; F 1130a; HCC 1130a; Iso-dichloroethylene; R 1130a; UN 1303; UN 1303 (DOT); VDC; Vinylidene dichloride
Chemical group (DSL Stream)	Discrete organics
Major chemical class or use	Alkenes
Major chemical sub-class	Halogenated alkene
Chemical formula	$C_2H_2Cl_2$
Chemical Structure	
SMILES[2]	C(=C)(Cl)Cl
Molecular mass	96.94 g/mol

[1] National Chemical Inventories (NCI). 2006: AICS (Australian Inventory of Chemical Substances); ASIA-PAC (Asia-Pacific Substances Lists); DSL (Domestic Substances List); ECL (Korean Existing Chemicals List); EINECS (European Inventory of Existing Commercial Chemical Substances); ENCS (Japanese Existing and New Chemical Substances); NZIoC (New Zealand Inventory of Chemicals); PICCS (Philippine Inventory of Chemicals and Chemical Substances); SWISS (Swiss Giftliste 1 and Inventory of Notified New Substances); and TSCA (Toxic Substances Control Act Chemical Substance Inventory).

[2] Simplified Molecular Input Line Entry Specification.

Physical and Chemical Properties

Physical and chemical properties of 1,1-DCE are summarized in Table 2 below.

1,1-DCE is a volatile substance that will exist as a liquid at most environmental temperatures. It will evaporate from most substrates including water, although it will also exist in a dissolved state in water. The relatively low K_{ow} and K_{oc} values indicate that it will not bind tightly to organic matter in the environment.

Table 2. Physical and chemical properties of 1,1-DCE

Property	Type	Value	Temperature	Reference
Melting point (°C)	Experimental	-123	–	PhysProp 2009
Boiling point (°C)	Experimental	31.6	–	PhysProp 2009
Vapour pressure (Pa)	Experimental	8×10^4	25°C	PhysProp 2009
Henry's Law constant ($Pa \cdot m^3/mol$)	Experimental	2644	24°C	PhysProp 2009
Octanol–water partition coefficient (log K_{OW}) (dimensionless)	Experimental	2.13	–	PhysProp 2009
Organic carbon-water partition coefficient (log K_{OC}) (dimensionless)	Estimated	1.85 (K_{OW} method)	–	KOCWIN 2008
Water solubility (mg/L)	Experimental	2420	25°C	PhysProp 2009
Rate constant for gas-phase reaction with hydroxyl radical (k_{OH}) cm^3/ molecule per second	Experimental	1.1×10^{-11}	25°C	PhysProp 2009

Sources

1,1-DCE is an anthropogenic substance that has not been identified to occur naturally (BUA 1998; WHO 2003a). Commercial production involves the dehydrochlorination of 1,1,2-trichloroethane in the presence of excess base or by thermal decomposition of methyl chloroform (1,1,1-trichloroethane) (Grosjean 1991; WHO 2003a). These production processes consist primarily of closed system operations in industrial settings (Williams et al. 2006). 1,1-DCE is also a byproduct in a process used to manufacture hydrogen chloride (Environment Canada 2001b).

An industry survey was conducted for the 2000 calendar year under section 71 of CEPA 1999 (Environment Canada 2001a). The *Notice with Respect to Certain Substances on the Domestic*

Substances List (DSL) applied to any person who, during the 2000 calendar year, manufactured or imported 1,1-DCE, whether alone or in a mixture or in a product, in a total quantity greater than 10 000 kg. Based on the survey, between 10 and 100 tonnes of 1,1-DCE were respectively reported to be manufactured and imported into Canada in 2000 (Environment Canada 2001b). During that year, 1,1-DCE was used in a sealer solvent and, as a by-product produced during the manufacture of 1,2-dichloroethane, to manufacture hydrochloric acid (Environment Canada 2001b). Follow-up with an importing company indicated that use of 1,1-DCE in a sealer solvent is no longer ongoing (2004 email from the importing company to Existing Substances Branch, Environment Canada; unreferenced). In addition, use of 1,1-DCE in the manufacture of hydrochloric acid, and its production as a by-product during the manufacture of 1,2-dichloroethane, is no longer ongoing (Dow 2006a). The total quantity in commerce in 2000 of 10 to 100 tonnes was similar to the total quantity in commerce during the period of DSL compilation (1984–1986) of 31 tonnes (Environment Canada 2000).

1,1-DCE is also a product of incomplete combustion of some chlorinated solvents, and thus incineration of hazardous waste presents a possible environmental source of 1,1-DCE (Fuerst et al. 1989). One study indicated that 1,1-DCE and methyl chloride were by far the principal incomplete combustion products for 1,1,1-trichloroethane, with a concentration of 1,1-DCE in incineration exhaust reported as > 200 ppb (Fuerst et al. 1989). However, 1,1,1-trichloroethane production was phased out by 2005 under the Montreal Protocol (Environment Canada 2003).

1,1-DCE was detected in sewage sludge in 3% of 436 samples from the US, at a concentration range of 1–14 000 µg/L in one study (Burns and Roe 1982). In another study, 1,1-DCE was detected at a mean concentration of 7.97 mg/kg dry weight (214 µg/L wet volume) in twelve digested sewage sludge samples (Wilson et al. 1994). Sewage sludge may represent a possible source of environmental 1,1-DCE in Canadian soil, though no recent US data or data on presence in sludge in Canada are available.

Uses

Based on responses to a survey issued under section 71 of CEPA 1999 (Environment Canada 2001a), in the 2000 calendar year over 10 tonnes were used in a sealer solvent and over 10 tonnes were used, as a by-product produced during the manufacture of 1,2-dichloroethane, to manufacture hydrochloric acid (Environment Canada 2001b). However, as of 2004, there is no record of 1,1-DCE being used in sealants in Canada (2004 email from the importing company to Existing Substances Branch, Environment Canada; unreferenced). In addition, the chlor-alkali and direct chlorination ethylene dichloride plants that manufactured 1,2-dichloroethane and hydrochloric acid in Fort Saskatchewan, Alberta, were shut down in October 2006 due to economic considerations (Dow 2006a).

1,1-DCE is not expected to be present in cosmetic products in Canada, as it is not listed as an ingredient in the Cosmetic Notification System database (CNS 2009). 1,1-DCE is currently regulated in cosmetic products in Canada as it is within the category of dichloroethylenes, or acetylene chlorides, as specified on the Health Canada cosmetic ingredient hotlist (Health

Canada 2009a). There are no registered pesticides that contain 1,1-DCE as an active ingredient or formulant in Canada (PMRA 2007), and 1,1-DCE is not a permitted food additive under Division 16 of the *Food and Drug Regulations* (Canada 1978).

1,1-DCE is not listed in the Drug Products Database, the Therapeutic Products Directorate's internal Non-Medicinal Ingredients Database, the Natural Health Products Ingredients Database or the Licensed Natural Health Products Database as a medicinal or non-medicinal ingredient in pharmaceutical drugs, natural health products or veterinary drugs (DPD 2010; NHPID 2010; LNHPD 2010; TPD NMID 2010). The International Conference on Harmonization Guideline Q3C(R4) (ICH 2009)—which is adopted by the Therapeutic Products Directorate (Health Canada 1999) and the Natural Health Products Directorate (Health Canada 2007)—and the International Cooperation on Harmonisation Guideline 18 (VICH 2000)—which is adopted by the Veterinary Drugs Directorate (Health Canada 2001)—list 1,1-DCE as a Class 1 residual solvent (i.e. solvent that should be avoided). As such, it should not be used in the manufacture of drug substances; however, if use is unavoidable in the manufacture of drug or veterinary medicinal products with significant therapeutic advances, the concentration limit for residual 1,1-DCE is 8 ppm.

Globally, 1,1-DCE is used primarily as an intermediate in the manufacture of polyvinylidene chloride (PVDC) polymers and copolymers. Consumer products where PVDC is used include flexible plastic films in the food industry, vapour barriers for insulation, carpets, awnings, piping, coatings for steel pipes, adhesives and photographic film (US EPA 2002a, 2003; WHO 2003a). PVDC and its copolymers are used as flame retardants in paper and board coatings (IPCS 1990). In these end products, 1,1-DCE is only present at low levels. For example, monomer residual levels are reported to be < 5 ppm in carpet latex, photographic film coating, flame retardant and ignition-resistant fibres for industrial clothing, food packaging, and as a component of a compound used in the production of automotive interior foam (US EPA 2002a). However, monomer levels are reported to be higher in an assessment by the World Health Organization released in 2003. 1,1-DCE was reported at a level of < 100 mg 1,1-DCE per kg article in photographic film coating, flame-retardant fibres for clothing and outdoor awnings, and PVDC-fluorinated copolymers for application on textiles (WHO 2003a). The lower levels of 1,1-DCE reported in the review published by the United States Environmental Protection Agency likely resulted from the effects of further processing of articles on reducing the concentration of monomer in the final consumer product (WHO 2003a). The degree to which these end products are present in the Canadian marketplace is unknown.

Other global uses of 1,1-DCE identified in literature searches were as a captive intermediate in the production of hydrochlorofluorocarbons (HCFC-141b and HCFC-142b) and hydrofluorocarbons (HFC 236fa), for use as refrigerants and in fire extinguishers; in chloroacetyl chloride and homo-, co- and terpolymers (latex and resin) (Connor et al. 1998; WHO 2003a), as an aid in ore flotation, as a solvent in paint and varnish remover, as a degreaser, and as an industrial cleaning agent (WHO 2003b). In addition, 1,1-DCE was formerly used as an anaesthetic (WHO 2003b).

Releases to the Environment

Based on responses to a survey issued under section 71 of CEPA 1999, total releases to the environment reported by one notifier were between 10 and 100 tonnes in the 2000 calendar year (Environment Canada 2001b). The section 71 survey did not require specification of the medium of release or of the form of substance released (e.g. pure substance, in a mixture or product). However, the notifier did indicate that the releases occurred as point sources at facilities where the 1,1-DCE was being used as a manufacturing intermediate or being processed for disposal, and that at the time of consumer use, releases to environmental media would no longer occur. As both notifiers that used 1,1-DCE in 2000 are not current users, point releases of 1,1-DCE are assumed to have substantially decreased.

Although 1,1-DCE is a reportable substance to the Canadian National Pollutant Release Inventory (NPRI), no releases were reported after the 2003 calendar year (NPRI 2009). According to the NPRI, releases in 2000 were 87 kg, in 2001 they were 25 kg, in 2002 they were 4 kg, and in 2003 they were 1 kg. Releases were typically from a single company, but usually not the same one.

Historically, there have been releases of 1,1-DCE from industrial sources to surface waters in Canada. The Ontario Ministry of the Environment (OME) reported 1,1-DCE emissions of 0.376 kg/day to the St. Clair River from industrial effluents in 1986–1987 (OME 1991a). The average concentration of 1,1-DCE in process effluents at seven Ontario petroleum refineries was 0.180 µg/L for 43 analyses (detection limit not stated) over a six-month period in 1989 (OME 1991b). In 1989–1990, Ontario chemical manufacturers discharged to Ontario rivers an average of 1.75 kg/day of 1,1-DCE (OME 1992). Over six months in 1990, 1,1-DCE was released at an average concentration of 4.47 µg/L (six analyses) at a company in Thorold, Ontario (OME 1991c). However, 1,1-DCE was not routinely monitored under Ontario's Municipal/Industrial Strategy for Abatement (MISA) program. The average total discharge of 1,1-DCE from 48 industrial sites along the St. Lawrence River in Quebec in 1992 was 0.136 kg/day (MENVIQ 1993).

In addition to being released into the environment during its manufacture and use, 1,1-DCE can also be released during the breakdown of PVDC products and during the abiotic and biotic decomposition of the drycleaning and vapour degreasing solvents 1,1,1-trichloroethane, tetrachloroethene (perchlorethylene), 1,1,2-trichloroethene and 1,2-dichloroethane due to poor disposal practices (OME 2001; Klier et al. 1999; IPCS 1990; ATSDR 1994; US EPA 1995). The formation of 1,1-DCE from these types of sources is highly variable, often depending on oxidative conditions in contaminated groundwater and landfills. Movement of these groundwater plumes below residences is a potential source of 1,1-DCE vapour intrusion to indoor air (Williams et al. 2006). For example, 1,1-DCE has been detected in groundwater in five studies (Appendix 1, Table A3). However, many of these solvents are no longer used in Canada and disposal practices have improved, such that significant new sources of 1,1-DCE in groundwater and soil are unlikely.

Environmental Fate

The results of Level III fugacity modelling (EQC 2003; Table 3) indicate that, if the chemical were released solely to air, the majority would remain in air. If released to water, the majority would remain in water with most of the rest partitioning to air. If released to soil, less than half would remain in soil while most of the rest would partition to air.

Table 3. Results of the Level III fugacity modelling (EQC 2003)

	Percentage of substance partitioning into each compartment (%)			
Substance released to:	**Air**	**Water**	**Soil**	**Sediment**
Air (100%)	99.9	0.1	0.03	0
Water (100%)	4.4	95.3	0	0.2
Soil (100%)	55.3	0.7	43.9	0

Due to the very high vapour pressure of 1,1-DCE (66 000 Pa at 20°C), a high proportion will ultimately partition to the atmosphere (WHO 2003a) despite its relatively high water solubility (2390 mg/L). The water-air partition coefficient of 0.16 reported by Pearson and McConnell (1975) also implies that most 1,1-DCE will partition to air and only a small amount will remain in water.

Persistence and Bioaccumulation Potential

Environmental Persistence

Empirical and modelled data for the degradation of 1,1-DCE in different media are presented in Tables 4a and 4b, respectively.

Gas-phase oxidation with photochemically produced hydroxyl radicals (OH•) is expected to be the most significant process for atmospheric removal, as this reaction is much faster than other atmospheric reactions (such as reactions with NO_3^-, ozone, and peroxy radicals), and the OH• is ubiquitous in air. Therefore, an atmospheric half-life was calculated for 1,1-DCE based on the following equation from Leifer (1993):

Half-life = 0.693/[(reaction rate)*(OH• concentration)*(43 200 seconds/12-hr day)]

Using a reaction rate constant of 1.09×10^{-11} (a unit-weighted average of the reaction rates reported by Atkinson [1989]) and a default OH• concentration of 1.5×10^6, this calculation produces an estimated tropospheric half-life of 0.98 days for 1,1-DCE. The primary reaction products for this reaction include formaldehyde (CH_2O), phosgene ($COCl_2$) and hydroxyacetyl chloride ($CH_2ClCOOH$) (Grosjean 1991). Other estimates of half-lives in air resulting from oxidation reactions with hydroxyl radicals were generally under 2 days (Table 4a).

1,1-DCE is a known precursor of formaldehyde in air following its degradation via oxidative reactions with hydroxyl radicals. Formaldehyde appears on the List of Toxic Substances in Schedule 1 of CEPA 1999.

Hydrolysis is not a significant degradation pathway for 1,1-DCE (Table 4a). Modelled half-lives for biodegradation reactions in water are estimated to be 28 to 180 days (Table 4b). The primary and ultimate biodegradation results are both ≤ 182 days in water. Due to its very high vapour pressure, the most important process for removal of 1,1-DCE from water is considered to be volatilization. Half-life values for volatilization from surface water bodies calculated by Mabey et al. (1981), and estimated in this assessment using the HENRYWIN (2008) model, ranged from 2.9 hours to 6 days. Measured biodegradation values in soil were 10 days (Ryan et al. 1988). Estimates of the half-life of 1,1-DCE in soil range from 28 days to 180 days and the estimated half-life in sediment is 150 days (BIOWIN 2009).

Table 4a. Empirical data for degradation of 1,1-DCE

Medium	Fate process	Degradation value	Degradation endpoint/units	Reference
Air	Photo-oxidation	0.46 days	Half-life	INERIS 2003
		0.67 days		Grosjean 1991
		0.98 day		Atkinson 1989
		2 days		Brown et al. 1975
	Reactions with NO_3^- at night	19 days		Grosjean 1990
	Photolysis	56 days		Pearson and McConnell 1975
	Ozone reaction	10 years		Grosjean 1990
	Reaction with peroxy radicals	22 years		Brown et al. 1975
Water	Hydrolysis	6–9 months	Half-life	Cline and Delfino 1987
	Hydrolysis (neutral to slightly basic pH)	1.2×10^8 years	Half-life	Jeffers et al. 1989
Soil	Biodegradation	< 10 days	Half-life	Ryan et al. 1988

Table 4b: Modelled data for degradation of 1,1-DCE

Media	Fate process	Model result and prediction	Extrapolated half-life (days)	Reference
Air	Photo-oxidation	4.7 days	≥ 2	AOPWIN 2008
		0.41–4.1 days		Howard et al. 1991
	Ozone reaction	219 days	≥ 2	US EPA 1985
		320 days	≥ 2	AOPWIN 2008
Water	Biodegradation MITI Linear	0.48 (not readily degradable)	≥ 182	BIOWIN 2009
	Primary biodegradation	Days–weeks	≤ 182	BIOWIN 2009
	Ultimate biodegradation	Weeks–months	≤ 182	BIOWIN 2009
	Biodegradation	28–180 days	≤ 182	Howard et al. 1991

Media	Fate process	Model result and prediction	Extrapolated half-life (days)	Reference
	Volatilization	0.12–6 days	≤ 182	Mabey et al. 1981; HENRYWIN 2008
Groundwater	Anaerobic biodegradation	56–132 days	≤ 182	Howard et al. 1991
	Anaerobic biodegradation	0.66 (degrades fast)	≤ 182	BIOWIN 2009
	Anaerobic biodegradation (simulated groundwater environment)	5–6 months	≤ 182	Barrio-Lage et al. 1986
Soil	Biodegradation half-life	37.5 days	≤ 182	BIOWIN 2009
	Biodegradation	28–180 days	≤ 182	Howard et al. 1991
Sediment	Biodegradation half-life	150 days	≤ 365	BIOWIN 2009

Fugacity modelling with the model TaPL3 (Beyer et al. 2000; TaPL3 2000) has been used to estimate a characteristic travel distance for 1,1-DCE of 524 km using the predicted half-life in air of 0.98 days. This is less than the 700 km criteria for long-range transport in air; 1,1-DCE is therefore considered to have a low potential for long-range transport.

Based on the information available, 1,1-DCE does not meet the criterion for persistence as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

Potential for Bioaccumulation

Bioaccumulation is expected to be low based on the octanol/water partition coefficient of 2.1 (Hansch et al. 1995) and water solubility of 1,1-DCE. A bioconcentration factor of 4 and a bioaccumulation factor of 6.9 were reported for fish (Atri 1985). A bioaccumulation factor of less than 13 was reported for common carp (*Cyprinus carpio*) (MITI 1992).

A bioaccumulation factor of 0.96 was estimated for 1,1-DCE with the BCFBAF (2008) model. This model includes the Arnot-Gobas bioaccumulation model for mid-trophic level bioaccumulation in fish with metabolism considered.

Based on information available, 1,1-DCE does not meet the bioaccumulation criteria (BAF or BCF greater than 5000) set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

Potential to Cause Ecological Harm

Ecological Exposure Assessment

Air

In a recent study of ambient air levels conducted as part of the ongoing National Air Pollution Surveillance (NAPS) network, 1,1-DCE was not detected in 1896 samples, at a detection limit of 0.026 μg/m^3, among 43 Canada-wide sites during the collection period of January to December 2008 (NAPS 2008). Another recent study, the Regina Indoor Air Quality Study (2007), detected 1,1-DCE at a maximum concentration of 0.014 μg/m^3 in outdoor air of residential backyards during the summer of 2007 in Regina, Saskatchewan, with a detection limit of 0.012 μg/m^3 (Health Canada 2010a). In a similar study conducted in Windsor, Ontario, the Windsor Exposure Assessment Study (2005-2006), 1,1-DCE was not detected in any sample of outdoor air above detection limits of 0.152 μg/m^3 and 0.046 μg/m^3 for 2005 and 2006 respectively (Health Canada 2010b).

Annual mean air concentrations of 1,1-DCE were obtained for 33 sites across Canada for 2004 as well (NAPS 2008). Montréal had the highest annual mean concentration, 0.016 μg/m^3, while Windsor had the lowest, 0.011 μg/m^3. The only rural air monitoring site was located at Simcoe, Ontario, which had an annual mean concentration of 0.012 μg/m^3, which was the same as the annual mean urban concentration. There were no statistically significant differences in the annual mean air concentrations between the various monitoring locations.

A separate four-week study was conducted in 2005 by the Air Quality Research Branch (AQRB) and the Canadian Meteorological Centre of Environment Canada in central Alberta. Data were collected from existing air quality stations and aircraft-mounted instruments. In the summer of 2005, there were only two samples with measured concentrations of 1,1-DCE above the detection limit of 0.011 μg/m^3, and both concentrations were below 0.05 μg/m^3 (2005 email from an Environment Canada Meteorological Service program manager to Ecological Assessment Division, Environment Canada; unreferenced).

In 2004 and 2005, Environment Canada and the Fort Air Partnership conducted an air monitoring program that included measuring 1,1-DCE near an industrial area in Fort Saskatchewan, Alberta, as well as at surrounding locations. A similar monitoring program was conducted by Environment Canada near an industrial area in North Vancouver, British Columbia. Air samples were taken from ten sites once every six days between September 2004 and July 2005. Mean monthly concentrations and an overall average concentration were calculated for each site. All monthly means in Fort Saskatchewan and North Vancouver were below the detection limit of 0.011 μg/m^3. The highest monthly mean concentration, 0.014 μg/m^3, was observed at a site 5 km north of Fort Saskatchewan (2005 email from an Environment Canada risk manager to Ecological Assessment Division, Environment Canada; unreferenced).

Monitoring by the Ontario Ministry of Environment and Energy in the late 1980s to early 1990s found the maximum concentration of 1,1-DCE measured over 30 minutes to be 0.81 μg/m^3 at a

hazardous waste facility, 0.25 to 0.65 μg/m^3 in industrial areas, and 0.68 to 5.7 μg/m^3 at landfills in the greater Toronto area (OME 1991d; OMEE 1997).

The atmospheric concentrations of 1,1-DCE in the vicinity of a hypothetical industrial facility were estimated using the air dispersion model SCREEN3 (US EPA 2006). It is a single-source Gaussian plume model that provides maximum 1-hour concentrations for point, area, flare and volume sources at a receptor height. Using a reasonable worst-case scenario for an industrial release of 10 tonnes over a year, the highest estimated 1-hour concentration of 1,1-DCE was 458.5 μg/m^3 at a distance of 52 m from the centre of the model facility. A concentration of 3.1 μg/m^3 of 1,1-DCE was estimated at a distance of 5 km from the facility.

The detection limit of 1,1-DCE in the recent NAPS survey, 0.026 μg/m^3, was used as the predicted environmental concentration (PEC) in deriving risk quotients for air. These are the most recent Canadian data, the concentration is the most conservative of the recent studies, the study is expansive across Canada and the sample size is large (n = 1896).

The highest atmospheric concentration of 458.5 μg/m^3 (0.459 mg/m^3) derived from the SCREEN3 model was selected as the PEC for use in deriving a risk quotient for a reasonable worst-case industrial scenario.

Groundwater

Concentrations of 1,1-DCE have been detected in groundwater samples associated with landfills. In the 1980s, levels ranging from 0.09 to 60 μg/L were detected in 43% of groundwater samples beneath the Gloucester landfill near Ottawa (Lesage et al. 1990). 1,1-DCE was not known to have been disposed of at the site but it is a known degradation product of tetrachloroethylene and 1,1,1-trichloroethane, which were disposed of at the landfill from 1969 to 1980 (Lesage et al. 1990). Carter et al. (2008) analyzed the results from U.S. groundwater quality surveys and found that 1,1-DCE was not a major contaminant, being detected in only 0.66% of sites (11 of 1686). Ellis and Rivett (2007) conducted analyses for volatile organic compounds (VOCs) in groundwater potentially entering the River Tame running through Birmingham, U.K. 1,1-DCE was found above detection limits in 20% of samples, with a maximum concentration of 20 μg/L. They estimated the mean daily flux from groundwater through the riverbed to be 0.1 mg/m^2/day or approximately 3 kg/year over 7 km of riverbed.

Given conditions favourable to oxidative growth (nutrients and an oxygen source), many groundwater bacteria are capable of degrading 1,1-DCE to vinyl chloride and ethene. Groundwater can be considered to be a pathway from contaminant sources to sediment and surface waters, if it can be shown that contaminated groundwater is recharging surface waters. This has not been the case with 1,1-DCE. An exposure scenario was not developed for groundwater.

Surface Water

There are few reports of surface water concentrations of 1,1-DCE in Canada above the detection limit of 0.08 μg/L, although 1,1-DCE has at times been detected in both raw and drinking water.

In the early 1980s, 1,1-DCE was detected at 12 of 95 water quality monitoring stations in Lake Ontario; the highest concentration was 3.5 µg/L near Scarborough, possibly close to a sewer discharge for the City of Toronto (Kaiser et al. 1983). Nine of 303 stations sampled in the St. Lawrence River in the mid-1980s had "trace" concentrations of 1,1-DCE just above the detection limit (Comba 1985; Comba et al. 1986), yet the maximum concentration reported was 100 µg/L at an industrial outfall near Prescott, Ontario (Comba et al. 1986). Studies across Canada on raw and potable drinking water supplies have rarely found any 1,1-DCE (Otson et al. 1982a; Otson et al. 1982b; Otson 1987; OME 1988; OME 1989; Toronto Water 2004; Health Canada 1994a), although there have been occasional reports of measurable concentrations. For example, a 1,1-DCE concentration of 20 µg/L was measured in one sample of treated drinking water in 1979 (Otson et al. 1982a), and a study of 29 Alberta municipal drinking water supplies by Health Canada between the years of 1978 and 1985 found a maximum concentration of 1.4 µg/L at one location (Health Canada 1994a).

The 1,1-DCE concentration of 100 µg/L (Comba et al. 1986) was selected as the PEC to be used for the risk quotient calculation for an aquatic scenario, as it was judged to represent a reasonable worst-case scenario for an historical industrial discharge.

Sediments and Soil

No data were found for measured concentrations of 1,1-DCE in sediments. Only one study was found, from Ontario in 1993, in which concentrations of 1,1-DCE in soil were measured. Three regions of rural and urban Ontario parkland were sampled, and the highest 98th percentile concentration of 1,1-DCE from all regions was 0.097 µg/kg (OMEE 1993). The maximum concentration was not reported. This result may not be representative of soil concentrations in areas where potential sources of contamination occur, such as industrial areas. As no data were found on 1,1-DCE in sediments, or on the potential toxicity of 1,1-DCE to soil or sediment organisms, scenarios could not be developed for soil or sediment exposures. However, as noted above, 1,1-DCE is not expected to partition to soil or sediment, and exposure could be expected to be negligible.

Ecological Effects Assessment

The toxicity of 1,1-DCE to aquatic organisms has been investigated in a number of studies (Table 5).

Dill et al. (1980) conducted a flow-through bioassay on the effects of 1,1-DCE to fathead minnows (*Pimephales promelas*), taking into account the volatilization of the chemical from the water. Little variation was found between the median lethal concentration (LC_{50}) values determined for tests of 48 to 96 hours duration. The 96-hr LC_{50} was determined to be 108 mg/L, and the 96-hr median effect concentration (EC_{50}) was determined to be 75 mg/L. The fish showed signs of distress (loss of equilibrium when swimming and disorientation) during the first 24 hours of exposure and did not recover.

The aquatic organism most sensitive to 1,1-DCE reported in the literature is the alga *Chlamydomonas reinhardtii*. Brack and Rottler (1994) reported a 72-hr EC_{10} for growth

inhibition of 3.94 mg/L in closed, measured conditions. This value was chosen as the critical toxicity value (CTV) for use in this assessment to predict risks to aquatic organisms.

Table 5. Empirical data for toxicity of 1,1-DCE to aquatic organisms

Test organism	Endpoint[2]	Value (mg/L)	Reference
Algae			
Pseudokirchneriella subcapitata Green alga	EC_{50} growth, 24 to 96-hr Freshwater	> 560	US EPA 1978
	NOEC, 96-hr Freshwater	<56	
Scenedesmus abundans Green alga	EC_{50} growth, 96-hr Freshwater	410	Geyer et al. 1985
Chlamydomonas reinhardtii Green alga[1]	72-hr EC_{10} growth 72-hr EC_{50} growth	3.94* 9.12	Brack and Rottler 1994
Skeletonema costatum Diatom	EC_{50} photosyn., 96-hr Saltwater	712	US EPA 1978
Aquatic invertebrates			
Daphnia magna Water flea	LC_{50} 24-hr, static	98	LeBlanc 1980
	LC_{50} 48-hr, static	76	
	NOEC 48-hr, static	< 2.4	
Daphnia magna Water flea	LC_{50} 24 to 48-hr, static	11.6	Dill et al. 1980
Americamysis bahia Oppossom shrimp	LC_{50} 96-hr	224	US EPA 1978
Daphnia magna	48-hr EC_{50} immobilization	16	CHRIP c2008
Vertebrates (fish)			
Cyprinodon variegates Sheepshead minnow	LC_{50} 24 to 96-hr static	250	Heitmuller et al. 1981
	NOEC 96-hr static	80	
Lepomis macrochirus Bluegill	LC_{50} 96-hr static	74	Buccafusco et al. 1981
Lepomis macrochirus Bluegill	LC_{50} 96-hr static	220	Dawson et al. 1977
Menidia beryllina Inland silverside	LC_{50} 96-hr static	250	Dawson et al. 1977
Pimephales promelas Fathead minnow	LC_{50} 24-hr static LC_{50} 24-hr flowthrough	175 116	Dill et al. 1980
	LC_{50} 48-hr static LC_{50} 48-hr flowthrough	169 108	
	LC_{50} 96-hr static LC_{50} 96-hr flowthrough	169 108	
	LC_{50} 10- to 13-day flowthrough	29	
	LC_{50} 5-day flowthrough LC_{50} 6-day flowthrough LC_{50} 7-day flowthrough LC_{50} 8-day flowthrough LC_{50} 9-day flowthrough	97 74 29 29 29	
Oryzias latipes	LC_{50} 96-hr	45	CHRIP c2008

Test organism	Endpoint[2]	Value (mg/L)	Reference
Rice fish			

[1] Brack and Rottler (1994) eliminated evaporative losses from the test system.

[2] EC_{50} – The concentration of a substance that is estimated to cause some effect on 50% of the test organisms.
LC_{50} – The concentration of a substance that is estimated to be lethal to 50% of the test organisms.
NOEC – The no-observed-effect concentration is the highest concentration in a toxicity test not causing a statistically significant effect in comparison to the controls.

* Critical toxicity value (CTV).

Terrestrial Compartment

Toxicity of 1,1-DCE to terrestrial organisms has been investigated in a number of studies, including in plants (Pestemer and Auspurg 1986), microorganisms (Greim et al. 1975; Bronzetti et al. 1983), invertebrates (Viswanathan 1984), and vertebrates (Prendergast et al. 1967; Murray et al. 1979; Quast et al. 1986; Van Duuren et al. 1979; Jones and Hathway 1978a).

No effects were noted in a 14-day test on the growth of wheat (*Triticum aestivum*), oats (*Avena sativa*), garden cress (*Lepidium sativum*), lettuce (*Lactuca sativa*), white mustard (*Sinapis alba*), Pak-choi (*Brassica chinensis*), colza (*Brassica napus*), turnips (*Brassica rapa*), perennial ryegrass (*Lolium perenne*), radish (*Raphanus sativus*), garden vetch (*Vicia sativa*), mung bean (*Vigna radiata*), red clover (*Trifolium pretense*), grain sorghum (*Sorghum bicolor*) at up to 1000 mg/kg in soil (Pestemer and Auspurg 1986). The only test found on soil organisms (earthworms) was found to be inadequate by the World Health Organization (WHO 2003a).

The mode of action of 1,1-DCE is non-polar narcosis (US EPA 1999). It is rapidly absorbed during oral and inhalation exposure, with most of the free 1,1-DCE and its metabolites found in the liver and kidney. The target organs during acute exposure by oral or inhalation routes are the liver, the kidney, and the Clara cells of the lungs. During chronic exposure the critical effect is a minor fatty change that occurs in the liver of many organisms. The metabolites of 1,1-DCE, including an epoxide, are responsible for the toxic effect within the target cells (US EPA 2002b).

Gallegos et al. (2007) evaluated data on inhalation toxicity to small mammals for many organic substances, including 1,1-DCE. They estimated a toxicity reference value for small mammals of 4.93 mg/kg-bw (kilograms of body weight) per day based on 17 data points ranging from 0.39 to 130 mg/kg-bw per day. All effect levels used were from chronic exposures resulting in no adverse effects; in essence they are no-observed-effect levels (NOELs).

A median lethal concentration (LC_{50}) in rats after 4 hours of inhalation was 6350 ppm (25 400 mg/m^3) (Kirk-Othmer 2007). In a study by Speerschneider and Dekant (1995), 1,1-DCE was shown to have a toxic effect in the kidneys of male mice via inhalation of 188 mg/m^3 (47 ppm) over 4 hours. This effect was species- and sex-specific due to the presence of cytochrome P450 2E1 found only in male mice.

A study by Prendergast et al. (1967) showed that continuous inhalation of a concentration of 1,1-DCE of 189 mg/m^3 (47.3 ppm) over a period of 90 days caused widespread morphological alterations in the livers and kidneys of rats, guinea pigs, dogs and monkeys. Continuous

inhalation of 101 mg/m^3 (25.3 ppm) had no effect on the test animals. Exposure for 8 hours/day 5 days/week had no effect at 395 mg/m^3.

The value by Prendergast et al. (1967) is a no-observed-adverse-effect level (NOAEL) for continuous inhalation effects to a variety of mammals at 101 mg/m^3 over 90 days. This value was selected for use as the CTV.

Characterization of Ecological Risk

The approach taken in the ecological risk characterization was to examine various supporting information and develop conclusions based on a weight-of-evidence approach and applying precaution as required under section 76.1 of CEPA 1999. Particular consideration has been given to risk quotient analyses, and to the environmental realism of the exposure scenarios used to derive predicted no-effect concentrations (PNECs) and occurrence in the environment. Endpoint organisms have been selected based on analysis of exposure pathways. For each endpoint organism, a conservative PEC and PNEC were determined. The PNEC is the lowest CTV for the organism of interest divided by an appropriate application factor. A risk quotient (PEC/PNEC) was calculated for each of the endpoint organisms in order to help determine whether there is a potential for ecological risk in Canada.

Application factors were derived using a multiplicative approach, in which 10-fold factors are used to account for various sources of uncertainty associated with making extrapolations and inferences related to intra- and interspecies variability, laboratory-to-field extrapolation, and acute-to-chronic toxicity values.

Selected PECs for this assessment, based on measured concentrations of 1,1-DCE in air (2.6×10^{-5} mg/m^3 at an urban site in Montréal and 5.7×10^{-3} mg/m^3 near a landfill site in Toronto), on modelled concentrations in air (0.46 mg/m^3 for an industrial release scenario), and measured in surface water (0.1 mg/L in Lake Ontario) have been previously discussed and are presented in Table 6.

The CTV of 101 mg/m^3 over 90 days to a variety of mammals was chosen to represent the concentration of 1,1-DCE resulting in no effects to small mammals continuously inhaling 1,1-DCE (Prendergast et al. 1967). The air concentration at a Toronto landfill (5.7×10^{-3} mg/m^3) was used as the PEC. An application factor of 10 was chosen to represent the dilution of 1,1-DCE from below ground to the air above a Toronto area landfill.

The aquatic species most sensitive to 1,1-DCE was the green alga *Chlamydomonas reinhardtii* (Brack and Rottler 1994). A CTV of 3.94 mg/L, the lowest concentration resulting in decreased growth (EC_{10}), was therefore chosen to represent the concentration of 1,1-DCE resulting in a non-significant level of effects in aquatic organisms. An application factor of 10 was applied to account for lab-to-field extrapolation, resulting in a PNEC for aquatic organisms of 0.394 mg/L.

Risk quotients for 1,1-DCE, obtained by dividing the PEC by the PNEC, are summarized in Table 6.

Table 6: Risk quotients calculated for 1,1-DCE

Medium	Organism	PEC	CTV	Application factor	PNEC	Risk quotient
Surface water (freshwater)	Green algae	0.10 mg/L	3.94 mg/L	10	0.394 mg/L	0.25
Soil at a landfill	Burrowing mammals	5.7×10^{-3} mg/m^3	101 mg/m^3	10	10.1 mg/m^3	6×10^{-4}
Urban air	Mammals	2.6×10^{-5} mg/m^3	101 mg/m^3	10	10.1 mg/m^3	2.5×10^{-6}
Air at an industrial site	Mammals	0.46 mg/m^3	101 mg/m^3	10	10.1 mg/m^3	0.05

The risk quotients calculated for water and air are significantly less than 1 (Table 6), indicating that there is very low likelihood of ecological harm from the concentrations of 1,1-DCE found in the Canadian environment.

1,1-DCE is a known precursor of formaldehyde in air following its degradation via oxidative reactions with hydroxyl radicals. Formaldehyde appears on the List of Toxic Substances in Schedule 1 of CEPA 1999.

All of the studies on the biotic effects of 1,1-DCE that were reviewed indicate that relatively high concentrations of 1,1-DCE are required to induce adverse effects, and these high concentrations are not found in the environment in Canada, either because sufficient volumes are not released, or due to environmental fate processes. Thus, based on consideration of 1,1-DCE's low persistence and low potential for accumulation in organisms, the lack of evidence for any recent, ongoing or anticipated increases in releases to the environment or in concentrations in the ambient environment, and based on indications that current concentrations are below levels that would be anticipated to cause ecological harm, it is proposed that 1,1-DCE is not causing ecological harm in Canada.

Uncertainty in the Evaluation of Ecological Risk

The major uncertainties in this assessment relate to exposure characterization. Monitoring studies during the 1980s and 1990s reported industrial releases of 1,1-DCE to water. However, it appears that there is presently low commercial usage of 1,1-DCE. The only industrial use of 1,1-DCE that produced significant releases of 1,1-DCE (as reported in the section 71 survey or to the NPRI) was in 2000 by one company which reported the use of a large volume of a commercial sealer-solvent, which may have resulted in releases to air. Other uses of 1,1-DCE

reportedly produced very little or no emissions, and consequently there may currently be very little exposure of 1,1-DCE to the environment from industrial releases. These uncertainties were dealt with by developing a series of exposure scenarios using ambient monitoring data for a variety of locations and by using a modelled approach for a hypothetical industrial release.

Potential to Cause Harm to Human Health

Exposure Assessment

Environmental Media and Diet

Empirical data were identified for 1,1-DCE environmental concentrations in ambient air, indoor air, raw and treated drinking water, soil and food and beverages in Canada. Empirical data were also identified for environmental media in other locations. All studies identified containing empirical data for each environmental medium are summarized in Appendix 1, Tables A1 to A5.

In a recent study of ambient air levels conducted as part of the ongoing NAPS network, 1,1-DCE was not detected in 1896 samples, at a detection limit of 0.026 μg/m^3, among 43 Canada-wide sites during the collection period of January to December 2008 (NAPS 2008). The substance was detected near the method detection limit (0.012 μg/m^3) in a limited number of samples of outdoor air of residential backyards in Regina in 2007; the median concentration was 0.006 μg/m^3 (Health Canada 2010a). Another study was unable to detect 1,1-DCE in outdoor air in Windsor, Ontario during either the summer or winter season above detection limits of 0.152 μg/m^3 and 0.046 μg/m^3 for 2005 and 2006, respectively (Health Canada 2010b). In a study of indoor and outdoor air of residences in Ottawa, Ontario, with sampling conducted during the winter of 2002-03, the median outdoor air concentration was 0.005 μg/m^3 (detection frequency: 18%) (Zhu et al. 2005). The highest median concentration of 1,1,-DCE in outdoor air in Windsor, Ontario for 2005 of 0.076 μg/m^3 was used in deriving the intake estimate (Appendix 2) as it represents the highest median concentration across recent Canadian outdoor air studies.

In an indoor air study of residences in Windsor, Ontario during 2005 and 2006, 1,1-DCE was detected near the method detection limits (0.152 μg/m^3 and 0.046 μg/m^3 for 2005 and 2006, respectively) in a limited number of samples (i.e., detection frequencies between 0.4 to 4.3%). For the summer of 2005, the median and 95th percentile concentrations were both 0.076 μg/m^3. Also, for the winter of 2005, the median and 95th percentile concentrations were both 0.076 μg/m^3. For the summer and winter of 2006, the median and 95th percentile concentrations were both 0.023 μg/m^3. (Health Canada 2010b). In a 2007 study in Regina, Saskatchewan, 1,1-DCE was detected near the method detection limit (0.012 μg/m^3) in a limited number of samples of indoor air (i.e., detection frequency of 5.9 – 10.1%). For the summer of 2007, the median and 95th percentile concentrations of the 5-day samples were 0.006 μg/m^3 and 0.023 μg/m^3, respectively. For the winter of 2007, the median and 95th percentile concentrations of the 5-day samples were 0.006 μg/m^3 and 0.027 μg/m^3, respectively (Health Canada 2010a). In a study of indoor and outdoor air of residences in Ottawa, Ontario, with sampling conducted during the winter of 2002-03, the median and 95th percentile indoor air concentrations were 0.005 μg/m^3 and 0.99 μg/m^3, respectively (detection frequency: 45%) (Zhu et al. 2005). The highest median

concentration of 1,1-DCE in indoor air from recent studies in Canada was 0.076 µg/m^3, based on Windsor, Ontario data collected during 2005; this value was used in deriving the environmental intake estimate (see Appendix 2). Also, the highest 95th percentile concentration in indoor air from recent studies in Canada was 0.99 µg/m^3, based on Ottawa, Ontario data collected during the winter of 2002-03.

Among fourteen recent Canadian surveys of drinking water among different cities of the country between 2003 and 2008, 1,1-DCE was not detected (CBWO 2008; City of Victoria 2008; City of Vancouver 2008; TDWS 2008; City of Niagara Falls 2008; CSWTP 2008; City of London 2008; OME 2008; Utilities Kingston 2008; BCOS 2008; EPCOR 2008; Ville de Montréal 2006; CCW 2003; COWQS 2003). A summary of drinking water data obtained from sites distributed across the United States conducted by the United States Geological Survey over a sampling period of 1985 to 2001 revealed median 1,1-DCE levels in samples where it was detected at 0.20 µg/L and 0.026 µg/L for public wells and domestic wells respectively (Zogorski et al. 2006). Zogorski et al. (2006) also determined the detection frequencies of DCE, by percent of total samples at an assessment level of 0.2 µg/L, in public wells and domestic wells of 1.3% (n = 1 096) and 0.21% (n = 2 400) respectively. The highest detection limit of the recent Canadian surveys, 0.52 µg/L for 35 samples in Ottawa, Ontario, in 2003 was used in deriving the intake estimate (see Appendix 2).

Studies analyzing for 1,1-DCE in food items available in Canada were conducted by Enviro-Test Laboratories in the early 1990s in Ville-Mercier, Quebec (ETL 1993), Windsor, Ontario (ETL 1992) and Cayley, Alberta (ETL 1991). 1,1-DCE was not detected in any of four samples of 34 food composites in these studies. The foods that comprised these composites are presented for Ville-Mercier, Quebec in Appendix 1, Table A4. The detection limits in the ETL (1991, 1992) studies were 50 µg/kg and 1.0 µg/L in solids and liquids, respectively, and in the ETL (1993) study the detection limits were 5.0 µg/kg and 1.0 µg/L for solids and liquids, respectively. Residuals of 1,1-DCE in food items are not currently monitored by the Canadian Food Inspection Agency (2009 email from the Canadian Food Inspection Agency to Risk Assessment Bureau, Health Canada; unreferenced).

In terms of food packaging, 1,1-DCE may exist as a manufacturing impurity in wrap that contains PVDC. Packaging of snack products may involve a PVDC coating on cellulose or polypropylene (Gilbert et al. 1980). A copolymer of PVDC and polyvinyl chloride (PVC) may be used to package patés, cooked sausages and processed cheeses as part of "chub" packs (Gilbert et al. 1980). One brand name of household plastic wrapping film used in a food context was a copolymer composed of PVC (15–20% by weight) and PVDC (80–85% by weight) (Birkel et al. 1977). While the formulation of this brand name of food plastic wrap in North America was changed in 2004 from PVDC to low-density polyethylene, this does not preclude other food plastic wraps containing PVDC being marketed under different brand names in Canada (Allen and Albala 2007; Dow 2006b). For example, PVDC, listed as an acceptable polymer for food packaging applications by Health Canada, was present in two new packaging applications registered in Canada as of 2007 (Health Canada 2009b). One of these packaging applications is considered suitable for meat, cheese, sausage packaging, and flame-retardant fibres and filaments (Solvay 2010).

In a 2005 Japanese study, 1,1-DCE was not detected by headspace gas chromatography analysis at a detection limit of 0.06 µg/g in PVDC home wrapping film and casing film for sausage, cheese and uiro [Japanese steamed cake], or in PVC food containers, water pipes, home wrapping film and toys (Ohno et al. 2005). In a 1976 Japanese study, no 1,1-DCE monomer was detected at a detection limit of 1 ppm in PVDC house hold wrap and casing film for fish sausage (Motegi et al. 1976). A 1977 American study of food plastic wrap determined mean 1,1-DCE monomer concentrations in household film and industrial-purpose films at 8.8 ppm (6.5–10.4 ppm) and 18.4 ppm (10.8–26.2 ppm) respectively (Birkel et al. 1977). A 1978 American study revealed a mean concentration of 1,1-DCE in food plastic wrap at 5.9 ppm (2.4–12.7 ppm) (Hollifield and McNeal 1978). This study also examined the mean concentration of 1,1-DCE determined in three food-simulating solvents resulting from contact with two different thicknesses of film (0.5 mm and 6.0 mm). This migration study was allowed to proceed for varying lengths of time (0.5 to 39 days) at 49°C until each sample had either experienced full migration of 1,1-DCE into the solvent or had reached an apparent equilibrium level (Hollifield and McNeal 1978). For the 0.5 mm-thick film, mean concentrations of 1,1-DCE in heptane, corn oil and water were 39 ppb (34–44 ppb), 34 ppb (18–41 ppb) and 25 ppb (24–27 ppb), respectively (Hollifield and McNeal 1978). For the 6 mm-thick film, mean concentrations of 1,1-DCE in heptane, corn oil and water were 320 ppb (66–579 ppb), 255 ppb (12–627 ppb) and 177 ppb (90–211 ppb), respectively (Hollifield and McNeal 1978). For exposure assessment purposes, the results of the 0.5 mm-thick film experiment may be more relevant for food exposure, as one brand name of household food plastic wrap was marketed at 0.5 mm thickness (Birkel et al. 1977).

A 1980 survey of foodstuffs packaged in films containing PVDC purchased in Great Britain revealed a mean 1,1-DCE monomer concentration of 0.019 ppm (0.010–0.025 ppm) in potato crisps after 30 days of storage at ambient temperature (Gilbert et al. 1980). In addition, of the other foods tested for migration of 1,1-DCE (biscuits, cakes, snack products, cooked meats and cheeses), only two food items had concentrations exceeding the detection limit of 0.005 ppm: black pudding and liver pate both had concentrations ranging from 0.005 to 0.01 ppm, with levels detected at the outer edges of the product (Gilbert et al. 1980). These food items had been stored for 60 days at ambient temperature (Gilbert et al. 1980). This study also determined mean concentrations of 1,1-DCE monomer in the actual PVDC-containing films used to package the foods: 0.49 ppm (< 0.06–1.26 ppm) for PVDC/polypropylene, < 0.04 ppm for PVDC/cellulose, 0.11 ppm (< 0.02–0.28 ppm) for PVDC-PVC and 0.15 ppm (0.12–0.16 ppm) for the potato crisp bags. In a related study, at a lower detection limit of 0.001 mg/kg, 1,1-DCE was detected in food in contact with PVDC film wrap (MAFF 1980). A Japanese study conducted in August 2004 of 13 samples of various foodstuffs (sausage, fish sausage, boiled fish paste and cheese) revealed a concentration range of 0.003–0.0095 µg/g (Ohno and Kawamura 2006). The food concentration data are presented in Appendix 1, Table A4.

The United States Food and Drug Administration lists 1,1-DCE as an indirect food additive, as some preservative polymer mixtures coated onto fruits and vegetables (especially citrus fruits) may contain 1,1-DCE (US FDA 2006). Food migration studies for these preservative mixtures have not been identified, though this does not preclude the possibility of citrus fruits imported from the United States being potential sources of 1,1-DCE exposure.

In terms of deriving an intake estimate, for each of the twelve food categories that comprise Canadian consumption as specified in the Health Canada (1998) report, detection limits were used for food items where no 1,1-DCE was detected, while mean concentrations were used for food items where 1,1-DCE was detected. These detection limits and mean concentrations were selected from all the available studies listed in Appendix 1, Table A4, regardless of location. This conservative approach of using detection limits for some food items may lead to an overestimate of actual exposure. The food intake estimates and studies selected are presented in Appendix 6. Scenarios involving 1,1-DCE monomer migration from food packaging to food items were not conducted, as empirical data exist for 1,1-DCE concentrations in PVDC-packaged foodstuffs (as provided in Appendix 4). However, a submission to the United States Environmental Protection Agency, as part of the Voluntary Children's Chemical Evaluation Program (VCCEP), provided a migration scenario for food wrap with typical and high-end oral intakes of 0.01 and 0.0375 µg/kg-bw per day respectively (US EPA 2002a; Williams et al. 2006). These intakes were substantially less than the estimated intakes from food and beverages presented in Appendix 2.

In terms of biomonitoring data, the National Health and Nutrition Examination Survey 2003–2004 conducted by the National Center for Health Statistics in the United States did not detect 1,1-DCE in any of 1367 samples of human blood from adults aged 20 to 59 years, detection limit of 0.009 ng/ml (NCHS 2009). 1,1-DCE was qualitatively detected in one of twelve breast milk samples derived from four cities in the United States, detection limit unspecified (Pellizzari et al. 1982). In a related study, 1,1-DCE was qualitatively detected in one of eight samples of breast milk derived from four cities in the United States, detection limit unspecified (Erickson et al. 1980).

In the most recent study identified for Canadian soil levels of 1,1-DCE, the maximum concentration detected in urban parkland and rural parkland soil of Ontario in the early 1990s was 0.12 ng/g solids and 0.098 ng/g solids, respectively (OMEE 1993). The weighted average of Ontario urban parkland, rural parkland (not including northwest region) and rural parkland (northwest region) soil of 0.046 µg/kg solids was used in deriving the intake estimate (see Appendix 2).

The maximum estimated intake for the general population was 1.34 µg/kg-bw per day for not formula-fed infants aged 0 to 6 months (Appendix 2). Indoor air and food and beverages are the main sources of estimated environmental exposure; however, the reliance on detection limits for several of the food items may indicate that the food and beverage intake estimate may exceed actual exposure. Food wrap containing PVDC is the expected major source of 1,1-DCE monomer in foodstuffs.

Consumer Products

Residual amounts of 1,1-DCE can be present in carpet latex backing, industrial insulation adhesives, photographic film coatings, flame-retardant clothing, and PVDC/fluorinated copolymer oil- and water-repellent coatings of textiles (US EPA 2002a), but were not detected in a recent Japanese study of PVC water pipes, home wrapping film and toys (e.g. ball, soft toy, food toy and face mask), detection limit of 0.06 µg/g (Ohno et al. 2005). 1,1-DCE was detected

in food plastic wrap at mean 1,1-DCE monomer concentrations in household film and industrial-purpose films at 8.8 ppm (6.5–10.4 ppm) and 18.4 ppm (10.8–26.2 ppm) respectively (Birkel et al. 1977).

As the 1,1-DCE is found bound within the polymer matrix, exposure resulting from use of these products is expected to be minimal (ATSDR 1994). However, as 1,1-DCE has a very high vapour pressure (600 mm Hg at 25°C), some of the residual monomer in consumer products may be released to indoor air. In an indoor air study of 75 residences in Ottawa, Ontario, conducted during the winter of 2002-2003, there was no correlation between the floor area carpeted (and backed with 1,1-DCE-containing latex) and measured values of 1,1-DCE in indoor air (Zhu et al. 2005). The arithmetic mean indoor air concentration was 0.27 μg/m^3, with a range of not detected to 4.05 μg/m^3, detection limit of 0.011 μg/m^3 (Zhu et al. 2005). The 75th percentile of the emission rate of 1,1-DCE in these 75 Ottawa homes was measured at 0.05 mg 1,1-DCE per hour (Zhu et al. 2005).

In a review of 50 indoor air studies of international locations conducted between 1978 and 1990, emission sources of 1,1-DCE that are unique to an indoor context (such as construction materials, including carpet latex backing) have resulted in concentrations of 1,1-DCE in indoor air that are elevated by an overall factor of approximately 13 compared to ambient air levels (Brown et al. 1994).

As the likely route of exposure to 1,1-DCE from consumer products is inhalation of indoor air containing 1,1-DCE emissions from construction materials, and as recent empirical data exist for indoor air of Canadian residences, use of the highest median concentration of 1,1-DCE across recent indoor air studies (i.e., 0.076 μg/m^3 in Windsor, Ontario during 2005) (Health Canada 2010b) in deriving the daily intake estimate for the indoor air component of environmental exposure is considered to account for any potential inhalation exposure to end products. In addition, use of the highest 95th percentile concentration from recent Canadian indoor air studies (i.e., 0.99 μg/m^3 in Ottawa, Ontario during the winter of 2002-03) (Zhu et al. 2005) would also account for any such inhalation exposure to end products when comparing to less than chronic non-cancer endpoints (see Characterization of Risk to Human Health section below).

Though end product exposure is considered accounted for in the overall intake estimate, a 1,1-DCE emission scenario from carpet latex backing to indoor air was presented in a report submitted by the Dow Chemical Company as part of the VCCEP program (US EPA 2002a). The submission estimated typical and high-end intakes of 0.023 and 0.027 μg/kg-bw per day for children, assuming that 80% of a 24-hour period is spent indoors (Williams et al. 2006). Newly constructed or renovated homes would presumably have the highest indoor air concentrations of 1,1-DCE from carpet latex backing, as the emission rate in the VCCEP scenario assumed first-order decay (Williams et al. 2006). These scenario-based inhalation intake estimates are roughly equivalent to the empirically based intake estimates presented in Appendix 2 (maximum inhalation intake estimate from indoor air of 0.04 μg/kg-bw per day) and indicate that use of recent empirical indoor air data identified in Windsor, Ontario in deriving the overall environmental exposure is protective of any potential exposures to end products through inhalation.

The VCCEP submission also concluded that dermal exposures to 1,1-DCE in textiles were insignificant or irrelevant due to the high-temperature processing of the fabrics that would drive off the minimal levels of 1,1-DCE present in coatings of textiles (< 5 ppm concentration in coating prior to processing) (US EPA 2002a). Therefore, dermal exposure to 1,1-DCE residuals in PVDC/fluorinated copolymer coatings of textiles in addition to flame-retardant and ignition-resistant clothing was not characterized on this basis. In addition, the VCCEP submission indicated that fire- and ignition-resistant clothing was typically used in industrial settings (US EPA 2002a). In addition, dermal exposures to 1,1-DCE in photographic paper and film are expected to be negligible, as the 1,1-DCE is contained in an internal latex layer and therefore essentially poses no potential for migration (US EPA 2002a). Dermal exposure to 1,1-DCE from its presence in textiles, flame retardants and ignition-resistant clothing, and photographic paper and film is considered to be negligible for the general population (i.e., primarily occupational). Therefore, the dermal route was not considered to be a significant route of consumer exposure relative to the inhalation route.

Confidence in Exposure Assessment

Confidence in the environmental exposure dataset is considered to be high. Empirical data specific to Canada were available for all environmental media and were recent for ambient air, indoor air and drinking water. The use of detection limits in deriving the overall intake estimate for drinking water and some food categories indicates that average concentrations in these media may have been substantially lower. As the main source of environmental releases of 1,1-DCE in the 2000 calendar year (point releases from facilities) is no longer ongoing, confidence that emissions to ambient air have decreased is high.

As for consumer product exposure, confidence is high that use of the highest median concentration for indoor air from Windsor, Ontario (2005) (0.076 $\mu g/m^3$) in deriving the indoor air component of the daily intake estimates for environmental exposures, and use of the highest 95^{th} percentile indoor air concentration (0.99 $\mu g/m^3$, Ottawa, Ontario, 2002-03) when comparing to less than chronic non-cancer endpoints (see Characterization of Risk to Human Health section below), would account for any potential inhalation exposures through releases of 1,1-DCE from PVDC-containing construction materials. In addition, as the environmental intake estimates for indoor air are similar to the intake estimates derived from the carpet latex backing release scenario in the VCCEP submission, confidence is high that any potential inhalation exposure through end products is addressed in the intake estimates.

Health Effects Assessment

An overview of key toxicological studies is presented in Appendix 3.

Carcinogenicity bioassays reviewed in several identified assessments (IARC 1986, 1999; IPCS 1990, 2003; US EPA 2002b) include those conducted by oral, inhalation and subcutaneous routes of exposure as well as a dermal tumour initiation study. Many of these studies are limited by study design or conduct, including exposure durations of 1 year or less or administration of less than the maximum tolerable dose.

An exposure-related, increased incidence of tumours (renal adenocarcinomas) was observed in male (but not female) Swiss mice exposed by inhalation for 1 year to 0, 10 or 25 ppm 1,1-DCE (equivalent to 0, 40 and 100 mg/m^3, respectively. This increase was only significant at the highest concentration (Maltoni et al. 1984, 1985; IPCS 1990) and it was the endpoint used by the U.S. EPA to develop an inhalation cancer potency factor (5×10^{-5} per μg/m^3) (Roberts et al. 2002).

Similarly, a cancer potency estimate was derived by Health Canada. A lowest tumorigenic concentration 05 (TC_{05}) of 4.2 mg/m^3 (equivalent to 5.6 mg/kg-bw per day, based on the incidence of pulmonary adenomas in both male and female mice (in the same inhalation carcinogenicity study used by the EPA) was calculated. The TC_{05} is defined as the concentration, generally in air, associated with a 5% increase in incidence or mortality due to tumours (Health Canada 1996).

The renal tumours seen were suggested to be related to the toxicity of a metabolite following metabolism via CYP2E1 in the kidney of the mouse. Some researchers have reported an absence of CYP2E1 in human kidney (Amet et al. 1997; Cummings et al. 2000), suggesting that these tumours may not be relevant to humans. The incidence of other tumours, namely mammary carcinomas in female Swiss mice and pulmonary adenomas in male and female Swiss mice, was significantly increased, but without a clear exposure–response relationship. 1,1-DCE was also active as an initiator of lung papillomas in female Swiss mice (Van Duuren et al. 1979). There was also no evidence of carcinogenicity in rat or hamster studies.

1,1-DCE appears to be genotoxic in micro-organisms in the presence of an exogenous metabolic activating system, while mixed results were obtained in the absence of such an activating system. Mixed results have also been produced in mammalian cells *in vitro*. It is generally non-genotoxic in *in vivo* assays (chromosomal aberration, rat; dominant lethal, mouse and rat; micronucleus, mouse), although chromosomal aberrations in the bone marrow of Chinese hamsters and minimal DNA binding in the liver and kidneys of mice and rats have been reported (IPCS 1990; US EPA 2002b).

No increase in tumour incidence was associated with 1,1-DCE-exposed workers in any of the three epidemiology studies in the literature (Ott et al. 1976; Thiess et al. 1979; Waxweiler et al. 1981), but their small cohort size, short observation period and potentially confounding variables preclude any evaluation of 1,1-DCE's carcinogenic potential in humans.

1,1-DCE has been classified by IARC (1999) as *not classifiable as to its carcinogenicity to humans* based on *inadequate evidence* in humans and *limited evidence* in experimental animals (Group 3); whereas the U.S. EPA (US EPA 2002b) concluded that there is *suggestive evidence* for the carcinogenicity of 1,1-DCE. In addition, it is classified in the European Union as Carcinogen Category 2, with the hazard statement "Suspected of causing cancer" (European Union 2008).

The target organs for non-cancer effects are the liver, the kidneys and the Clara cells of lungs. The lowest lowest-observed-adverse-effect concentration (LOAEC) identified was 10 ppm

(40 mg/m^3), based upon significant increases in kidney damage (regressive changes and/or abscesses and nephritis) in male Swiss mice exposed to 1,1-DCE for 52-weeks (Maltoni et al. 1984, 1985). The lowest oral lowest-observed-effect level (LOEL) was 5 mg/kg-bw per day, based upon chronic renal inflammation in male and female F344 rats in a 2-year gavage study (NTP 1982).

For non-cancer effects based on a less than chronic period, a lowest inhalation LOAEC of 60 mg/m^3 was determined in a mouse developmental toxicity study. An increase in the mean number of fetuses with skeletal effects was observed in this study (Short et al. 1977).

Characterization of Risk to Human Health

In the critical carcinogenicity bioassay (Maltoni et al. 1984, 1985), renal adenocarcinomas were observed at the highest concentration (100 mg/m^3) only. This concentration is approximately 1,300 000 times higher than the highest median concentration of 1,1-DCE in air in Canada (0.076 µg/m^3 in indoor air; Health Canada 2010b).

1,1-DCE is generally non-genotoxic in *in vivo* assays.

Using the cancer potency factor derived by Health Canada (i.e., lowest TC_{05} of 4.2 mg/m^3; equivalent to 5.6 mg/kg-bw per day) for the same inhalation carcinogenicity study in mice mentioned above, and using the upper-bounding estimate of inhalation intake for adults in the Canadian population (i.e., ≤ 0.017µg/kg-bw per day for adults aged 20–59 and 60+ years; approximates lifetime exposures), the margin between these estimates is in the order of magnitude of 3.3 x 10^5. This margin is considered adequate to address uncertainties in the health effects and exposure databases.

A comparison of the lowest critical inhalation effect level for chronic non-cancer effects (40 mg/m^3; Maltoni et al. 1984, 1985) with the highest median concentration of 1,1-DCE in air in Canada (0. 076 µg/m^3 in indoor air; Health Canada 2010b) results in a margin of exposure of approximately 530 000. This margin is considered adequate to address uncertainties in the health effects and exposure databases.

A comparison of the lowest critical inhalation effect level for non-cancer effects based on a less than chronic period (60 mg/m^3; Short et al. 1977), with the highest 95th percentile concentration of 1,1-DCE in air in Canada (0.99 µg/m^3) results in a margin of exposure of approximately 61 000. This margin is considered adequate to address uncertainties in the health effects and exposure databases.

The margin of exposure between the highest upper-bounding estimate of intake from all sources of exposure (1.34 µg/kg-bw per day for not formula-fed infants between 0 and 6 months of age; Appendix 2and the critical oral effect level for non-cancer effects (5 mg/kg-bw per day; NTP 1982) is approximately 3700. This margin is considered adequate to address uncertainties in the health effects and exposure databases.

The non-cancer critical effects of 1,1-DCE associated with cytotoxicity of the liver, kidney and Clara cells of the lung in rats and mice are considered to result from the damage caused through the covalent binding of CYP2E1 activated metabolic products of 1,1-DCE to cellular macromolecules (US EPA 2002b).

Uncertainties in Evaluation of Risk to Human Health

Several of the carcinogenicity bioassays reviewed in identified assessments had study design limitations, including exposure durations of one year or less or administration of less than the maximum tolerable dose. Uncertainties exist regarding inter- and intra-species variation, extrapolation of data from animals to humans and a lack of data in humans for several endpoints.

There is uncertainty in current levels of 1,1-DCE in foodstuffs consumed by Canadians due to minimal recent data identified and the unknown degree to which food package formulations containing PVDC may have changed in Canada or some imported products in recent times. Without conducting a survey on a typical market basket of foodstuffs consumed by Canadians, it is uncertain as to the degree to which the intake estimate presented in Appendix 2 is representative of current Canadian consumption patterns. However, the use of detection limits and mean concentrations for the food categories would likely prevent any underestimation of exposure to 1,1-DCE in food items. There is uncertainty in the current quantities of 1,1-DCE in commerce in Canada, as the reporting year for the survey issued under section 71 of CEPA 1999 was for the 2000 calendar year. There is also uncertainty in the market penetration of PVDC-containing end products for sale to consumers in Canada among total similar products available.

Conclusion

On the basis of the information presented, it is concluded that 1,1-dichloroethene is not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends. Additionally, 1,1-DCE does not meet the criteria for persistence or bioaccumulation potential as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

On the basis of the adequacy of the margins of exposure between estimated exposures to 1,1-dichloroethene and critical effect levels, it is concluded that 1,1-dichloroethene is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that 1,1-dichloroethene does not meet the criteria in section 64 of CEPA 1999.

References

Amet Y, Berthou F, Fournier G, Dreano Y, Bardou L, Cledes J, Menez JF. 1997. Cytochrome P450 4A and 2E1 expression in human kidney microsomes. Biochem Pharmacol 53:765–771 [cited in US EPA 2002a].

Allen G, Albala K. 2007. The Business of Food: Encyclopedia of the Food and Drink Industries. Santa Barbara (CA): Greenwood Press, Greenwood Publishing Group. p. 329.

Andersen ME, Jenkins LJ Jr. 1977. Oral toxicity of 1,1-dichloroethylene in the rat: effects of sex, age and fasting. Drug Chem Toxicol 1:63–74 [cited in IPCS 1990].

[AOPWIN] Atmospheric Oxidation Program for Microsoft Windows. Version 1.92a. 2008. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: http://www.epa.gov/oppt/exposure/pubs/episuite.htm

Atkinson R. 1989. Kinetics and mechanisms of the gas-phase reactions of the hydroxyl radical with organic compounds. J Phys Chem Ref Data. Monograph No. 1.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1994. Toxicological profile for 1,1-dichloroethylene (update). Atlanta (GA): US Department of Health and Human Services. 174 pp. (TP-93/07).

Atri FR. 1985. Chlorinated compounds in the environment. Schriftenreihe des Vereins fur Wasser-, Boden-, und Lufthygiene, Berlin-Dahlem, 60:309–317 [cited in WHO 2003a; IPCS 1990].

Baden JM, Brikenhoff M, Wharton RS, Hitt BA, Simmon FV, Mazze RI. 1976. Mutagenicity of volatile anesthetics. Anesthesiology 45:311–318 [cited in IPCS 1990].

Baden JM, Kelley M, Wharton RS, Hitt BA, Simmon VF, Mazze RI. 1977. Mutagenicity of halogenated ether anesthetics. Anesthesiology 46:346–350 [cited in US EPA 2002b].

Baden JM, Kelley M, Simmon VF, Rice SA, Mazze RI. 1978. Fluroxene mutagenicity. Mutat Res 58:183–191 [cited in IPCS 1990].

Baden JM, Kelley M, Mazze RI. 1982. Mutagenicity of experimental inhalational anesthetic agents: sevofluorane, synthane, dioxychlorane and dioxyfluorane. Anesthesiology 56:462–463 [cited in IPCS 1990].

Barrio-Lage G, Parsons FZ, Nassar RS, Lorenzo PA. 1986. Sequential dehalogenation of chlorinated ethenes. Environ Sci Tech 20:96–99 [cited in WHO 2003a; HSDB 2005].

Bartsch H, Malaveille C, Montesano R, Tomatis L. 1975. Tissue-mediated mutagenicity of vinylidene chloride and 2-chlorobutadiene in *Salmonella typhimurium.* Nature (London) 255:641–643 [cited in IPCS 1990].

Bartsch H, Malaveille C, Barbin A, Planche G. 1979. Mutagenic and alkylating metabolites of halo-ethylenes, chloro-butadienes and dichlorobutenes produced by rodent or human liver tissues. Evidence for

oxirane formation by P450-linked microsomal mono-oxygenases. Arch Toxicol 41:249–277 [cited in IPCS 1990].

[BCFBAF] Bioaccumulation Program for Windows [Estimation Model].2008. Version 3.00. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: http://www.epa.gov/oppt/exposure/pubs/episuite.htm

[BCOS] Beyond Compliance Operating System. 2008. Drinking-Water Systems Regulation O. Reg. 170/03 Section 11 Annual Report. Hamilton (ON): Beyond Compliance Operating System.

Beyer A, Mackay D, Matthies M, Wania F, Webster E. 2000. Assessing long-range transport potential of persistent organic pollutants. Environ Sci Technol 34:699–703.

[BIOWIN]. Biodegradation Probability Program for Windows [Estimation Model]. 2009. Version 4.10. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: http://www.epa.gov/oppt/exposure/pubs/episuite.htm

Birkel TJ, Roach JA, Sphon JA. 1977. Determination of vinylidene chloride in saran film by electron capture gas-solid chromatography and confirmation by mass spectrometry. J AOAC Int 60(5):1210–1213.

Brack W, Rottler H. 1994. Toxicity testing of highly volatile chemicals with green algae: a new assay. Environ Sci Pollut Res Int 1(4):223–228.

Bronzetti G, Bauer C, Corsi C, Leporini C, Nieri R, Del Carratore R. 1981. Genetic activity of vinylidene chloride in yeast. Mutat Res 89:179–185 [cited in IPCS 1990].

Bronzetti G, Bauer C, Corsi C, del Carratore R, Nieri R, Paolini M, Galli A, Giagoni P. 1983. Comparison of genetic and biochemical effects of halogenated olefins. Mutat Res 113:236–237 (Abstract No. 24) [cited in WHO 2003a].

Brown SK, Sim MR, Abramson MJ, Gray CN. 1994. Concentrations of volatile organic compounds in indoor air - a review. Indoor Air 4:123–134.

Brown SL, Chan FY, Jones JL, Liu DH, McCaleb KE, Mill T, Sapio KN, Schendel DE. 1975. Research program on hazard priority ranking of priority chemicals. Phase II: Final Report. Menlo Park (CA): Stanford Research Institute (NSF-RA-E-75-190A); NTIS PB-263-161 [cited in IPCS 1990].

[BUA] Beratergremium für Umweltrelevante Altstoffe. 1988. 1,1-Dichloroethene. Beratergremium für Umweltrelevante Altstoffe, Gesellschaft Deutscher Chemiker. BUA Report 33. 60 p.

Buccafusco RJ, Ellis SJ, Leblanc GA. 1981. Acute toxicity of priority pollutants to bluegill (*Lepomis macrochirus*). Bull Environ Contam Toxicol 26:446–452.

[Burns and Roe] Burns and Roe Industrial Services Corp. 1982. Fate of priority pollutants in publicly owned treatment works. EPA-440/1-82/303. Washington (DC): Effluent Guidelines Division, Office of Water Regulations and Standards, US Environmental Protection Agency.

Canada. 1978. *Food and Drug Regulations*, C.R.C., c. 870. Available from: http://laws.justice.gc.ca/eng/C.R.C.-c.870/index.html

Canada. 1999. *Canadian Environmental Protection Act, 1999*. S.C., 1999, c. 33. Canada Gazette, Part III, vol 22, no. 3. Available from: http://www.gazette.gc.ca/archives/p3/1999/g3-02203.pdf

Canada. 2000. *Canadian Environmental Protection Act, 1999: Persistence and Bioaccumulation Regulations*, P.C. 2000-348, 23 March, 2000, SOR/2000-107. Canada Gazette, Part II, vol. 134, no. 7, p. 607−612. Available from: http://canadagazette.gc.ca/archives/p2/2000/2000-03-29/pdf/g2-13407.pdf

[CARB] California Air Resources Board. 1992. Indoor Pollutant Concentrations and Exposures, Sacramento (CA): California Environmental Protection Agency. Final Report, Contract No. A833-156.

Carpenter CP, Smyth HF Jr, Pozzani UC. 1949. The assay of acute vapor toxicity and the grading and interpretation of results of 96 chemical compounds. J Ind Hyg Toxicol 31:343–346 [cited in IPCS 1990].

Carter J, Lapham W, Zogorski J. 2008. Occurrence of volatile organic compounds in aquifers of the United States. J Am Water Resour Assoc 44(2):399–416.

[CBWO] The City of Barrie Water Operations. 2008. City of Barrie Drinking Water System Annual Water Report for the Period of January 1 to December 31, 2008.

[CCW] The City of Calgary Waterworks. Drinking Water Quality Summary: Glenmore Water Treatment Plant, January 01 – December 31, 2003.

Cerna M, Kypenova H. 1977. Mutagenic activity of chloroethylenes analysed by screening system tests. Mutat Res 46:214–215 [cited in IPCS 1990].

Chan CC, Valner L, Martin JW, Williams JT. 1990. Determination of organic contaminants in residential indoor air using an adsorption-thermal desorption technique. J Air Waste Manage Assoc 40(1):62–67.

[CHRIP] Chemical Risk Information Platform [database on the Internet]. c2008. Tokyo (JP): National Institute of Technology and Evaluation, Chemical Management Centre (CMC). [cited 2011 March]. Available from: http://www.safe.nite.go.jp/english/db.html

City of London. 2008. City of London 2008 Annual Drinking Water Compliance Report. London (ON): Corporation of the City of London.

City of Niagara Falls. 2008. Ontario Drinking-Water Systems Regulation O. Reg. 170/03, Optional Annual Report Template. Niagara Falls (ON).

City of Toronto. 1990. The quality of drinking water in Toronto; A review of tap water, bottled water and water treated by a point-of-use device. Toronto (ON): Department of Public Health. 133 pp.

City of Vancouver. 2008. The Greater Vancouver Water District Quality Control Annual Report 2008. Vancouver (BC): Metro Vancouver.

City of Victoria. 2008. 2008 untreated (raw) water quality at Japan Gulch Plant. Victoria (BC): Capital Regional District Water Services.

Cline PV, Delfino JJ. 1987. American Chemical Society, Division of Environmental Chemistry Preprint New Orleans (LA). 27:577–579 [cited in HSDB 2005].

[CNS] Cosmetic Notification System [Proprietary Database]. 2009. Available from Health Canada, Cosmetics Division.

Comba ME. 1985. St. Lawrence River Trace Organic Contaminants Study (Part 1). NWRI Contribution No. 89-50. Burlington (ON): Environment Canada, National Water Research Institute.

Comba ME, Palabrica VS, Kaiser KL. 1986. St. Lawrence River Trace Organic Contaminants Study (Part 2). NWRI Contr. No. 89-51. Burlington (ON): Environment Canada, National Water Research Institute.

Connor BF, Rose DL, Noriega MC, Murtagh LK, Abney SR. 1998. Methods of analysis by the U.S. Geological Survey National Water Quality Laboratory – Determination of 86 volatile organic compounds in water by gas chromatography/mass spectrometry, including detections less than reporting limits [Internet]. Open-File Report 97-829. Denver (CO): US Geological Survey, US Department of the Interior. [cited 2010 June 1]. Available from: http://nwql.usgs.gov/Public/pubs/OFR97-829/OFR97-829.pdf

Costa AK, Ivanetich KM. 1982. Vinylidene chloride: its metabolism by hepatic microsomal cytochrome P-450 *in vitro*. Biochem Pharmacol 31:2083–2092 [cited in IPCS 1990].

Cotruvo JA. 1985. Organic micropollutants in drinking water: An overview. Sci Total Environ 47:7-26 [cited in ATSDR 1994].

Cotti G, Maltoni C, Lefemine G. 1988. Long-term carcinogenicity bioassay on vinylidene chloride administered by inhalation to Sprague-Dawley rats. New results. Ann N Y Acad Sci 534:160-168 [cited in IPCS 1990].

[COWQS] City of Ottawa Water Quality Section. 2003. 2003 Organics Summary. Ottawa (ON): City of Ottawa.

[CSWTP] City of Saskatchewan Water Treatment Plant. 2008. Drinking Water Quality and Compliance: City of Saskatoon – for Year 2008, Annual Notice to Consumers. Saskatoon (SK).

Cummings BS, Lasker JM, Lash LH. 2000. Expression of glutathione-dependent enzymes and cytochrome P450s in freshly isolated and primary cultures of proximal tubular cells from human kidney. J Pharmacol Exp Ther 293:677–685 [cited in US EPA 2002a].

Dawson BV, Johnson PD, Goldberg SJ, Ulreich JB. 1993. Cardiac teratogenesis of halogenated hydrocarbon contaminated drinking water. J Am Coll Cardiol 21:1466–1472 [cited in US EPA 2002a].

Dawson GW, Jennings AL, Drozdowski D, Rider E. 1977. The acute toxicity of 47 industrial chemicals to fresh and saltwater fishes. J Hazard Mater 1(4):303–318.

Dill DC, McCarty W, Alexander HC, Bartlett EA. 1980. Toxicity of 1,1-dichloroethylene (vinylidene chloride) to aquatic organisms. For U.S. EPA, report EPA-600/3-80-057. Duluth (MN): US Environmental Protection Agency.

Dow. 2006a. Dow announces plant closures to strengthen competitive position. [Internet]. Midland (MI): The Dow Chemical Company. [cited 2009 Aug 4]. Available from: http://news.dow.com/corporate/2006/20060831g.htm

Dow. 2006b. Product Safety Assessment: SARAN™ PVDC Resins and Films [Internet]. Midland (MI): The Dow Chemical Company. [cited 2010 Jun 3]. Available from: http://www.dow.com/productsafety/finder/saran.htm

[DPD] Drug Products Database [database on the Internet]. 2010. Ottawa (ON): Therapeutic Products Directorate, Health Canada. [cited 2010 Jun]. Available from: http://www.hc-sc.gc.ca/dhp-mps/prodnatur/applications/online-enligne/nhpid-bipsn-eng.php

Drevon C, Kuroki T. 1979. Mutagenicity of vinyl chloride, vinylidene chloride and chloroprene in V79 Chinese hamster cells. Mutat Res 67:173–182 [cited in IPCS 1990].

Ellis P, Rivett M. 2007. Assessing the impact of VOC-contaminated groundwater on surface water at the city scale. J Contam Hydrol 91:107–127.

Environment Canada. 1992. Detroit incinerator monitoring program, Data report #6. Ottawa (ON): Pollution Measurement Division.

Environment Canada. 1994. Volatile organic compound measurements in the greater Vancouver and regional district (GVRD) 1989-1992. Ottawa (ON): Environmental Technology Centre, Pollution Measurement Division. Report Series No. PMD 94-1.

Environment Canada. 1995. Volatile organic compounds in the ambient air of the province of Quebec (1989-1993). Ottawa (ON): Atmospheric Pollution and Toxic Substances Control Division and Pollution Measurement Division.

Environment Canada, 2000. Domestic Substances List: Use Pattern Summary. Internal database. Hull (QC): Commercial Chemicals Evaluation Branch.

Environment Canada. 2001a. *Canadian Environmental Protection Act, 1999. Notice with Respect to Certain Substances on the Domestic Substances List (DSL).* Canada Gazette, Part I, vol. 135, no. 46, p. 4194–4211. Available from: http://canadagazette.gc.ca/archives/p1/2001/2001-11-17/html/notice-avis-eng.html#i1

Environment Canada. 2001b. Data collected pursuant to section 71 (CEPA 1999) and in accordance with the published notice "*Notice with Respect to Certain Substances on the Domestic Substances List (DSL),* Canada Gazette, Vol. 135 no. 46".

Environment Canada. 2003. First Priority Substances List (PSL1): 1,1,1-Trichloroethane [updated: 2003 November 07]. Available from: http://www.ec.gc.ca/substances/ese/eng/psap/PSL1_1_1_1_trichloroethane.cfm

EPCOR. 2008. 2008 Edmonton Water Performance Report. Edmonton (AB): EPCOR.

[EQC]. Equilibrium Criterion Model. 2003. Version 2.02. Peterborough (ON): Trent University, Canadian Environmental Modelling Center. Available from: http://www.trentu.ca/cemc/models/EQC2.html

Erickson MD, Harris BSH, Pellizzari ED, Tomer KB, Waddell RD, Whitaker DA. 1980. Acquisition and chemical analysis of mother's milk for selected toxic substances. EPA-560/13-80-029. Washington (DC): US Environmental Protection Agency, Office of Pesticides and Toxic Substances.

[ETL] Enviro-Test Laboratories. 1991. Cayley background study: Analysis of food products for target organic and inorganic parameters. Series 91-E1208. Edmonton (AB). Under contract to Hazardous Waste Section, Environmental Health Directorate, Health and Welfare Canada, Ottawa (ON).

[ETL] Enviro-Test Laboratories. 1992. Windsor area background study: Analysis of food products for target organic and inorganic parameters. Series 92-E1052. Edmonton (AB). Under contract to Hazardous Waste Section, Environmental Health Directorate, Health and Welfare Canada, Ottawa (ON).

[ETL] Enviro-Test Laboratories. 1993. Ville-Mercier background study: Analysis of food products for target organic and inorganic parameters. Series E3-02-147.REP. Edmonton (AB). Under contract to Hazardous Waste Section, Health Protection Branch, Health and Welfare Canada, Ottawa (ON).

European Union. 2008. REGULATION (EC) No 1272/2008 OF THE EUROPEAN PARLIAMENT AND OF THE COUNCIL of 16 December 2008 on classification, labelling and packaging of substances and mixtures, amending and repealing Directives 67/548/EEC and 1999/45/EC, and amending Regulation (EC) No 1907/2006. Official Journal of the European Union L 353. 31 December 2008. Available at: http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2008:353:0001:1355:EN:PDF

Foureman P, Mason JM, Valencia R, Zimmering S. 1994. Chemical mutagenesis testing in *Drosophila*. IX. Results of 50 coded compounds tested for the National Toxicology Program. Environ Mol Mutagen 23(1):51–63.

Fuerst RG, Logan TJ, Midgett MR, Sykes AL, Buedel T, Bursey J, Homoloya JB. 1989. Sampling and analysis experiments for improved characterization of products of incomplete combustion. J Air Waste Manag Assoc 39(7):969–974.

Gage JC. 1970. The subacute inhalation toxicity of 109 industrial chemicals. Br J Ind Med 27: –18 [cited in IPCS 1990].

Gallegos P, Lutz J, Markweise J, Ryti R, Mirenda R. 2007. Wildlife ecological screening levels for inhalation of volatile organic chemicals. Environ Toxicol Chem 26(6):1299–1303.

Geyer H, Scheunert I, Korte F. 1985. The effects of organic environmental chemicals on the growth of the alga *Scenedesmus subspicatus*: a contribution to environmental biology. Chemosphere 14(9):1355–1369.

Gilbert J, Shepherd MJ, Startin JR, McWeeny DJ. 1980. Gas chromatographic determination of vinylidene chloride monomer in packaging films and in foods. J Chromatogr 197:71–78.

Greim H, Bonse G, Radwan Z, Reichert D, Henschler D. 1975. Mutagenicity *in vitro* and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxirane formation. Biochem Pharmacol 24:2013–2017 [cited in IPCS 1990].

Grosjean D. 1990. Atmospheric chemistry of toxic contaminants: 1. Reaction rates and atmospheric persistence. J Air Waste Manag Assoc 40(10):1397–1402 [cited in ATSDR 1994].

Grosjean D. 1991. Atmospheric chemistry of toxic contaminants. 5. Unsaturated halogenated aliphatics: allyl chloride, chloroprene, hexachloropentadiene, vinylidene chloride. J Air Waste Manag Assoc 41(2):182–189 [cited in WHO 2003a].

Hansch C, Leo A, Hoekman D. 1995. Exploring QSAR - Hydrophobic, Electronic, and Steric Constants. Washington (DC): American Chemical Society [cited in HSDB 2005].

Health Canada. 1994a. Guidelines for Canadian Drinking Water Quality, 1,1-Dichloroethylene. Prepared for the Federal-Provincial-Territorial Committee on Drinking Water. Environmental Health Centre, Ottawa (ON). 7 p.

Health Canada. 1996. Health-based tolerable daily intakes/concentrations and tumorigenic doses/concentrations for priority substances. Cat. H46-2/96-194E. Minister of Supply and Services Canada.

Health Canada. 1998. Exposure Factors for Assessing Total Daily Intake of Priority Substances by the General Population of Canada. Unpublished report. Ottawa (ON): Health Canada, Environmental Health Directorate. Available upon request.

Health Canada. 1999. Impurities: Guidelines for Residual Solvents [Internet]. International Conference on Harmonisation of Technical Requirements for the Registration of Pharmaceuticals for Human Use (ICH) / Therapeutic Products Programme guideline. Ottawa (ON): Health Canada, Therapeutic Products Programme. [cited 2009 Aug 6]. Available from: http://www.hc-sc.gc.ca/dhp-mps/prodpharma/applic-demande/guide-ld/ich/qual/q3c-eng.php

Health Canada. 2001. GL-18 – Impurities: Residual Solvents in New Veterinary Medicinal Products, Active Substances and Excipients [Internet]. Ottawa (ON): Veterinary Drugs Directorate, Health Canada. [cited 2010 Jun]. Available from: http://www.hc-sc.gc.ca/dhp-mps/vet/legislation/guide-ld/vich/guide-ligne-eng.php

Health Canada. 2007. Evidence for Quality of Finished Natural Health Products [Internet]. Ottawa (ON): Natural Health Products Directorate, Health Canada. [cited 2010 Jun]. Available from: http://www.hc-sc.gc.ca/dhp-mps/prodnatur/legislation/docs/eq-paq-eng.php#2432

Health Canada. 2009a. The cosmetic ingredient hotlist – September 2009 [Internet]. Ottawa (ON): Health Canada, Consumer Product Safety. [cited 2010 Jan 15]. Available from: http://www.hc-sc.gc.ca/cps-spc/person/cosmet/info-ind-prof/_hot-list-critique/hotlist-liste-eng.php

Health Canada. 2009b. Lists of acceptable polymers for use in food packaging applications [Internet]. Ottawa (ON): Food Directorate, Health Canada. [cited 2010 Jun 3]. Available from: http://www.hc-sc.gc.ca/fn-an/legislation/guide-ld/polymers_tc-polymere_tm-eng.php

Health Canada. 2010a. Regina indoor air quality study (2007): Data summary for Volatile Organic Compound Sampling. Water, Air and Climate Change Bureau, Healthy Environments and Consumer Safety Branch. Cat.: H128-1/10-617E-PDF.

Health Canada. 2010b. Windsor exposure assessment study (2005-2006): Data summary for Volatile Organic Compound Sampling. Water, Air and Climate Change Bureau, Health Environments and Consumer Safety Branch. Cat.: H128-1/10-618E-PDF.

Heitmuller PT, Hollister TA, Parrish PR. 1981. Acute toxicity of 54 industrial chemicals to sheepshead minnows (*Cyprinodon variegatus*). Bull Environ Contam Toxicology 27:596–604.

[HENRYWIN]. Henry's Law Constant for Windows [Estimation Model]. 2008. Version 3.20. Washington (DC): United States Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Hofmann HT, Peh J. 1976. [Report on the test of vinylidene chloride for mutagenic effects in Chinese hamsters after subacute inhalation.] BASF Aktiengesellschaft, Ludwigshafen. 22 p. [in German] [cited in IPCS 1990].

Hollifield HC, McNeal T. 1978. Gas-solid chromatographic determination of vinylidene chloride in saran film and three food-simulating solvents. J Assoc Off Anal Chem 61(3):537–544.

Hong CB, Winston, JM, Thornburg LP, Lee CC, Woods JS. 1981. Follow-up study on the carcinogenicity of vinyl chloride and vinylidene chloride in rats and mice: Tumor incidence and mortality subsequent to exposure. J Toxicol Environ Health 7:909–924 [cited in IARC 1986].

Howard PH, Boethling RS, Jarvis WF, Meylan WM, Michalenko EM. 1991. Handbook of Environmental Degradation Rates. New York (NY): Lewis Publishers. p. 439-440.

[HSDB] Hazardous Substances Data Bank [database on the Internet]. 2005. 1,1-Dichloroethylene. Bethesda (MD): US National Library of Medicine. Available from: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

[IARC] International Agency for Research on Cancer. 1986. Some chemicals used in plastics and elastomers. IARC Monogr Eval Carcinog Risk Hum 39:195–226.

[IARC] International Agency for Research on Cancer. 1999. Re-evaluation of some organic chemicals, hydrazine and hydrogen peroxide (part three). Summary of Data Reported and Evaluation. IARC Monogr Eval Carcinog Risk Hum 71:1163–1180. Available from: http://monographs.iarc.fr/ENG/Monographs/vol71/volume71.pdf

[ICH] International Conference on Harmonisation of Technical Requirements for Registration of Pharmaceuticals for Human Use. 2009. ICH Harmonised Tripartite Guideline: Impurities: Guidelines for Residual Solvents Q3C(R4). [cited 2010 Jun]. Available from: http://www.ich.org/LOB/media/MEDIA5254.pdf

[INERIS] Institut National de l'Environnment Industriel et des Risques. 2003. 1,1-Dichloroethylene. France. INIRIS-DRC-01-25590-ETSC-APi/SD No01DR022.doc.

[IPCS] International Programme on Chemical Safety. 1990. Environmental Health Criteria 100, Vinylidene Chloride. (ICSC 0083). Geneva (CH): World Health Organization. Available from: http://www.inchem.org/documents/ehc/ehc/ehc100.htm

[IPCS] International Programme on Chemical Safety. 2003. Concise International Chemical Assessment Document 51: 1,1-Dichloroethene (vinylidene chloride). Geneva (CH): World Health Organization. Available from: http://www.inchem.org/documents/cicads/cicads/cicad51.htm

Ishidate M, editor. 1983. The data book of chromosomal aberration tests *in vitro* on 587 chemical substances using a Chinese hamster fibroblast cell line (CHL cell). Tokyo (JP): Realize Inc. [cited in US EPA 2002a].

Jaeger RJ, Connolly RB, Murphy SD. 1973. Diurnal variation of hepatic glutathione concentration and its correlation with 1,1-dichloroethylene inhalation toxicity in rats. Res Commun Chem Pathol Pharmacol 6:465–471 [cited in IPCS 1990].

Jaeger RJ, Connolly RB, Murphy, SD. 1974. Effect of 18-hour fast and glutathione depletion on 1,1-dichloroethylene-induced hepatotoxicity and lethality in rats. Exp Mol Pathol 20:187–198 [cited in IPCS 1990].

Jeffers PM, Ward LM, Woytowitch LM, et al. 1989. Homogenous hydrolysis rate constants for selected chlorinated methanes, ethanes, ethenes, and propanes. Environ Sci Technol 23:965–969 [cited in ATSDR 1994].

Jenkins LJ Jr, Trabulus MJ, Murphy SD. 1972. Biochemical effects of 1,1-dichloroethylene in rats: comparison with carbon tetrachloride and 1,2-dichloroethylene. Toxicol Appl Pharmacol 23:501–510 [cited in IPCS 1990].

Jones BK, Hathway DE. 1978a. Differences in metabolism of vinylidene chloride between mice and rats. Br J Cancer 37:411–417 [cited in IPCS 1990].

Jones BK, Hathway DE. 1978b. Tissue-mediated mutagenicity of vinylidene chloride in *Salmonella typhimurium* TA 1535. Cancer Lett 5(1):1–6 [cited in IPCS 2003].

Kaiser KLE, Comba ME, Huneault H. 1983. Volatile halocarbon contaminants in the Niagara River and in Lake Ontario. J Great Lakes Res 9(2):212.

Kirk-Othmer. 2007. Kirk-Othmer Encyclopedia of Chemical Technology. (5th Edition) (2007), 25 691-745. Reported in, no further reference provided.

Klier NJ, West RJ, Donberg PA. 1999. Aerobic biodegradation of dichloroethylenes in surface and subsurface soils. Chemosphere. 38(5):1175–1188.

Klimisch JH, Freisberg KO. 1979a. [Report on the determination of acute toxicity (LC_{50}) by inhalation of vinylidene chloride in Chinese striped hamsters (fasting) during a 4-hour exposure period.] BASF Aktiengesellschaft, Ludwigshafen. 11 p. [in German] [cited in IPCS 1990].

Klimisch JH, Freisberg KO. 1979b. [Report on the determination of acute toxicity (LC_{50}) by inhalation of vinylidene chloride in Chinese striped hamsters (fed) during a 4-hour exposure period.] BASF Aktiengesellschaft, Ludwigshafen. 14 p. [in German] [cited in IPCS 1990].

Koch R, Schlegelmilch R, Wolf HU. 1988. Genetic effects of chlorinated ethylenes in the yeast *Saccharomyces cerevisiae*. Mutat Res 206:209–216 [cited in US EPA 2002a].

[KOCWIN] Soil Adsorption Coefficient Program for Windows [Estimation Model]. 2008. Version 2.00. Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. [cited 2009 Dec. 8]. Available from: http://www.epa.gov/oppt/exposure/pubs/episuite.htm

Lazarev NV, editor. 1960. [Vinylidene chloride.] In: [Harmful substances in industry.] Leningrad (RU): Chemia. p. 215–216 [in Russian] [cited in IPCS 1990].

LeBlanc GA. 1980. Acute toxicity of priority pollutants to water flea (*Daphnia magna*). Bull Environ Contam Toxicol 24(5):684–691.

Lee CC, Bhandari JC, Winston JM, House WB, Peters PJ, Dixon RL, Woods JS. 1977. Inhalation toxicity of vinyl chloride and vinylidene chloride. Environ Health Perspect 21:25–32 [cited in IPCS 1990].

Lee CC, Bhandari JC, Winston JM, House WB, Dixon RL, Woods JS. 1978. Carcinogenicity of vinyl chloride and vinylidene chloride. J Toxicol Environ Health 4:15–30 [cited in IARC 1986].

Leifer A. 1993. Determination of Rates of Reaction in the Gas-Phase in the Troposphere. Theory and Practice. 5. Rate of Indirect Photoreaction. EPA/744/R-93/001 (NTIS PB93-149334). Washington (DC): US Environmental Protection Agency, Office of Pollution Prevention and Toxics.

Lesage S, Jackson RE, Priddle MW, Riemann PG. 1990. Occurrence and fate of organic solvent residues in anoxic groundwater at the Gloucester landfill, Canada. Environ Sci Technol 24:559.

[LNHPD] Licensed Natural Health Products Database [database on the Internet]. 2010. Natural Health Products Directorate, Health Canada. [cited 2010 June]. Available from: http://www.hc-sc.gc.ca/dhp-mps/prodnatur/applications/licen-prod/lnhpd-bdpsnh-eng.php

Mabey WR, Smith JH, Pudoll RT, Johnson HL, Mill T, Chou TW, Gates J, Partridge IW, Vandenburg D. 1981. Aquatic fate process. Data for organic priority pollutants: Washington (DC): US Environmental Protection Agency. EPA-440/4-81-014 [cited in IPCS 1990].

Malaveille C, Plaanche G, Bartsch H. 1997. Factors for efficiency of the *Salmonella*/microsome mutagenicity assay. Chem-Biol Interact 17:129–136 [cited in US EPA 2002a].

Maltoni C, Patella V. 1983. Comparative acute toxicity of vinylidene chloride. The role of species, strain and sex. Acta Oncol 4:239–256.

Maltoni C, Cotti G, Chieco P. 1984. Chronic toxicity and carcinogenicity bioassays of vinylidene chloride. Acta Oncol 5:91–146 [cited in IPCS 1990].

Maltoni C, Lefemine G, Cotti G, Chieco P, Patella V. 1985. Experimental research on vinylidene chloride carcinogenesis. In Maltoni C, Mehlman MA, editors. Archives of research on industrial carcinogenesis. Vol. III. Princeton (NJ): Princeton Scientific Publishers [cited in IARC 1986, 1999; IPCS 1990; US EPA 2002a].

[MAFF] Ministry of Agriculture, Fisheries and Food. 1980. Survey of Vinylidene Chloride Levels in Food Contact Materials and in Foods. The Third Report of the Steering Group on Food Surveillance: The Working party on Vinylidene Chloride. London (UK): Ministry of Agriculture, Fisheries and Food (Food Surveillance Paper No. 3). 23 pp.

McCarroll NE, Cortina TA, Zito MJ, Farrow MG. 1983. Evaluation of methylene chloride and vinylidene chloride in mutational assays. Environ Mutagen 5:426–427 [cited in IPCS 1990].

McGregor D, Brown AG, Cattanach P, Edwards I, McBride D, Rianch C, Shepherd W, Caspary W. 1991. Responses of the L5178Y mouse lymphoma forward mutation assay; V. Gases and vapours. Environ Mol Mutagen 17:122–129 [cited in US EPA 2002b].

[MENVIQ] Ministry of the Environment of the Province of Quebec and Environment Canada. 1993. Plan d'action Saint-Laurent (PASL) volet protection. Resultats de la campagne de caractérisation. Equipe d'intervention Saint-Laurent.

[MITI] Japanese Ministry of International Trade and Industry. 1992. Biodegradation and bioaccumulation data of existing chemicals based on the CSCL Japan. Tokyo (JP): Ministry of International Trade and Industry [cited in WHO 2003a].

Mortelmans K, Haworth S, Lawlor T, Speck W, Tainer B, Zeiger E. 1986. *Salmonella* mutagenicity tests. II. Results from the testing of 270 chemicals. Environ Mutagen 8 (Suppl. 7):1–119 [cited in IPCS 1990].

Motegi S, UedaK, Tanaka H, Ohta M. 1976. Determination of residual vinylidene chloride monomer in polyvinylidene chloride films used for fish jelly products. Bull Jap Soc Sci Fisheries 42(12):1387-1394.

Murray FJ, Nitschke KD, Rampy LW, Schwetz BA. 1979. Embryotoxicity and fetotoxicity of inhaled or ingested vinylidene chloride in rats and rabbits. Toxicol Appl Pharmacol 49:189–202 [cited in IPCS 1990].

[NAPS] National Air Pollution Surveillance Program. 2004. Gatineau (QC): Environment Canada. Internal database cited in 2004.

[NAPS] National Air Pollution Surveillance Program. 2008. Gatineau (QC): Environment Canada. Internal database cited 2009 Aug. 4.

[NCHS] National Center for Health Statistics. 2009. National Health and Nutrition Examination Survey 2003-2004: Volatile Organic Compounds in Blood and Water. [publication date 2008 August]. [revision date 2009 Jun]. Hyattsville (MD): National Center for Health Statistics, Centers for Disease Control and Prevention, US Department of Health and Human Services. Available from: http://www.cdc.gov/nchs/nhanes/nhanes2003-2004/L04VOC_C.htm

[NCI] National Chemical Inventories [database on a CD-ROM]. 2006. Columbus (OH): American Chemical Society, Chemical Abstracts Service. Available from: http://www.cas.org/products/cd/nci/require.html

[NHPID]. Natural Health Products Ingredients Database [database on the Internet]. 2010. Ottawa (ON): Natural Health Products Directorate, Health Canada. [cited 2010 Jun]. Available from: http://www.hc-sc.gc.ca/dhp-mps/prodnatur/applications/online-enligne/nhpid-bipsn-eng.php=

[NHW] Department of National Health and Welfare. 1990. Present patterns and trends in infant feeding in Canada. Ottawa (ON): National Health and Welfare. Catalogue number H39-199/1990E. ISBN 0-662-18397-5. 9 p. [cited in Health Canada 1998].

[NPRI] National Pollutant Release Inventory [database on the Internet]. 2009. Database search 1994-2008; Data as of December 2009. Gatineau (QC): Environment Canada. Available from: http://www.ec.gc.ca/pdb/querysite/query_e.cfm

[NTP] National Toxicology Program. 1982. Carcinogenesis bioassay of vinylidene chloride (CAS No. 75-35-4) in F344 rats and B6C3F1 mice (gavage study). Research Triangle Park (NC): US National Toxicology Program. (Technical Report Series No. 228; PB 82-258393) [cited in US EPA 2002a].

Nitschke KD, Smith FA, Quast JF, Norris JM, Schwetz BA. 1983. A three-generation rat reproductive toxicity study of vinylidene chloride in the drinking water. Fundam Appl Toxicol 3:75–79 [cited in IPCS 1990].

Norris JM. 1977. Toxicological and pharmacokinetic studies on inhaled and ingested vinylidene chloride in laboratory animals. In: Proceedings of the Technical Association of the Pulp and Paper Industry (TAPPI) Paper Synthetics Conference, Chicago, Illinois, 1977. Atlanta, Georgia, Technical Association of the Pulp and Paper Industry. pp. 45–50 [cited in IPCS 1990].

Norris JM, Reitz RH. 1984. Interpretative review of the animal toxicological, pharmacokinetic/metabolism, biomolecular and *in vitro* mutagenicity studies on vinylidene chloride and the significance of the findings for man. Midland (MI): Dow Chemical Co. p. 24 [cited in IPCS 1990].

Oesch F, Protic-Sabljic M, Friedberg T, Klimisch HJ, Glatt HR. 1983. Vinylidene chloride: changes in drug metabolising enzymes, mutagenicity and relation to its targets for carcinogenesis. Carcinogenesis 4:1031–1038 [cited in IPCS 1990].

Ohno H, Mutsuga M, Kawamura Y, Suzuki M, Aoyama T. 2005. Headspace GC/MS analysis of residual vinyl chloride and vinylidene chloride in polyvinyl chloride and polyvinylidene chloride products. [Journal of the Food Hygienic Society of Japan] 46(1): 8–12 [in Japanese].

Ohno H, Kawamura Y. 2006. Analysis of vinylidene chloride and 1-chlorobutane in foods packaged with polyvinylidene chloride casing films by headspace gas chromatography/mass spectrometry (GC/MS). Food Addit Contam 23(8):839–844.

[OME] Ontario Ministry of the Environment. 1988. Ottawa (Lemieux Island) water treatment plant, Drinking water surveillance program, annual report 1987.

[OME] Ontario Ministry of the Environment. 1989. Drinking water surveillance program, overview annual report, 1987.

[OME] Ontario Ministry of the Environment. 1991a. St. Clair River MISA pilot site investigation, Vol II, Part II – Detailed Technical Findings. (MISA).

[OME] Ontario Ministry of the Environment. 1991b. Second report on the monitoring data for the petroleum refining sector.

[OME] Ontario Ministry of the Environment. 1991c. The preliminary report on the second six months of process effluent monitoring in the MISA pulp and paper sector (July 1, 1990 to Dec. 31, 1991).

[OME] Ontario Ministry of the Environment. 1991d. The 1990 Toronto personal exposure pilot (PEP) study. Atmospheric Research and Special Programs Section, Air Resources Branch. ISBN 0-7729-7962-6.

[OME] Ontario Ministry of the Environment. 1992. Six month monitoring data report organic manufacturing sector (October 1, 1989 to March 31, 1990). MISA.

[OME] Ontario Ministry of the Environment. 2001. Ontario Air Standards for Vinylidene Chloride. Standards Development Branch. EBR Registry No.:PA00E0019. Available from: http://www.ene.gov.on.ca/envision/env_reg/er/documents/2001/airstandards/PA00E0019.PDF

[OME] Ontario Ministry of the Environment. 2008. Drinking-Water Systems Regulation O. Reg. 170/03 Annual Report. Kitchener (ON): Ontario Ministry of the Environment.

[OMEE] Ontario Ministry of the Environment and Energy. 1993. Ontario typical range of chemical parameters in soil, vegetation, moss bags and snow. Phytotoxicology Section, Standards Development Branch. ISBN 0-7778-1979-1. [cited 2009 Dec. 9]. Available from: http://www.ene.gov.on.ca/envision/sudbury/ontario_typical_range/index.htm

[OMEE] Ontario Ministry of the Environment and Energy. 1997. Summary of TAGA monitoring results. Candidate list of 18 chemicals. Environmental Monitoring and Reporting Branch, Ministry of the Environment and Energy, Ontario [cited in OME 2001].

Otson R. 1987. Purgeable organics in Great Lakes raw and treated water. Int J Environ Anal Chem 31(1):41–53.

Otson R, Williams DT, Biggs DC. 1982a. Relationships between raw water quality, treatment and occurrence of organics in Canadian potable water. Bull Environ Contam Toxicol 28:396–403.

Otson R, Williams DT, Bothwell RD. 1982b. Volatile organic compounds in water at thirty Canadian potable water treatment facilities. J Assoc Off Anal Chem 65(6):1370–1374.

Ott MG, Fishbeck WA, Townsend JC, Schneider EJ. 1976. A health study of employees exposed to vinylidene chloride. J Occup Med 18:735–738 [cited in IARC 1986].

Pearson CR, McConnell G. 1975. Chlorinated C_1-undC_2-hydrocarbons in the marine environment. Proc R Soc Lond B Biol Sci 189:305–332.

Pellizzari E, Hartwell TD, Harris BSH III, Waddell RD, Whitaker DA, Erickson MD. 1982. Purgeable organic compounds in mother's milk. Bull Environ Contam Toxicol 28:322–328.

Pestemer W, Auspurg B. 1986. Eigung eines Testpflanzensortiments zur Risikoabschätzung von Stoffwirkungen auf höhere Pflanzen im Rahmen des Chemikaliengesetzes. Nachrichtenbl Deut Pflanzenschutzd 38:120–125 [data from US EPA ECOTOX database].

[PhysProp] Interactive PhysProp Database [database on the Internet]. 2009. Syracuse (NY): Syracuse Research Corporation. [cited 2009 Dec. 8]. Available from: http://esc.syrres.com/interkow/webprop.exe?CAS=106-93-4

Pleil J, Oliver K, McClenny W. 1985. Volatile organic compounds in indoor air: a survey of various structures. In Walkinshaw D, editor. Indoor Air Quality in Cold Climates: Hazards and Abatement Measures. Pittsburgh (PA): Air Pollution Control Association. p. 237–249.

Plummer JL, Hall PM, Ilsley AH, Jenner MA, Cousins MJ. 1990. Influence of enzyme induction and exposure profile on liver injury due to chlorinated hydrocarbon inhalation. Pharmacol Toxicol 67:329–335 [cited in IPCS 2003].

[PMRA] Pest Management Regulatory Agency. 2007. Regulatory Note REG 2007-04: PMRA list of formulants [Internet]. Ottawa (ON): Health Canada, Pest Management Regulatory Agency. [updated 2007 June; cited 2009 Aug. 6]. Available from: http://www.hc-sc.gc.ca/cps-spc/pubs/pest/_decisions/reg2007-04/index-eng.php

Ponomarkov V, Tomatis, L. 1980. Long-term testing of vinylidene chloride and chloroprene for carcinogenicity in rats. Oncology 37:136–141 [cited in IPCS 1990].

Prendergast JA, Jones RA, Jenkins LJ Jr, Siegel J. 1967. Effects on experimental animals of long-term inhalation of trichloroethane, carbon tetrachloride, 1,1,1-trichloroethylene, dichlorodifluoromethane and 1,1-dichloroethylene. Toxicol Appl Pharmacol 10:270–289 [cited in IPCS 1990].

Quast JF, Humiston CG, Schwetz BA, Balmer MF, Rampy LW, Norris JM, Gehring PJ. 1977. Results of 90-day toxicity study in rats given vinylidene chloride in their drinking water or exposed to VDC vapour by inhalation. Toxicol Appl Pharmacol 4:187 [cited in IPCS 1990].

Quast JF, Humiston CG, Wade CE, Ballard J, Beyer JE, Schwetz RW, Norris JM. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fundam Appl Toxicol 3(1):55–62 [cited in IPCS 1990].

Quast JF, McKenna MJ, Rampy LW, Norris JM. 1986. Chronic toxicity and oncogenicity study on inhaled vinylidene chloride in rats. Fundam Appl Toxicol 6:105–144.

Rajagopal R, Li P-C. 1991. Comparison of two screening methods for the detection of volatile organic compounds in ground water. J Chemometrics 5:3210-3331 [cited in ATSDR 1994].

Rampy LW, Quast JF, Humiston CG, Balmer MF, Schwetz BA. 1977. Interim results of two-year toxicological studies in rats of vinylidene chloride incorporated in the drinking water or administered by repeated inhalation. Environ Health Perspect 21:33–43 [cited in US EPA 2002a].

Rampy LW, Quast JF, Humiston CG, Balmer MF, Schwetz BA. 1978. Results of two-year toxicological studies in rats of vinylidene chloride incorporated in the drinking water or administered by repeated inhalation. Toxicol Appl Pharmacol 45:244–245 [cited in IPCS 1990].

Reitz RH, Watanabe PG, McKenna MJ, Quast JF, Gehring PJ. 1980. Effects of vinylidene chloride on DNA synthesis and DNA repair in the rat and mouse: a comparative study with dimethylnitrosamine. Toxicol Appl Pharmacol 52:357–370 [cited in IPCS 1990].

Roberts SM, Jordan KE, Warren DA, Britt JK, James RC. 2002. Evaluation of the carcinogenicity of 1,1-dichloroethylene (vinylidene chloride). Regul Toxicol Pharmacol 35(1):44-55.

Roldan-Arjona T, Garcia-Pedrajas MD, Luque-Romero FL, Hera C, Pueyo C. 1991. An association between the mutagenicity of the Ara test of *Salmonella typhimurium* and carcinogenicity in rodents for 16 halogenated aliphatic hydrocarbons. Mutagenesis 6(3):199–205 [cited in US EP, 2002a].

Ryan JA, Bell RM, Davidson JM, O'Connor GA. 1988. Plant uptake of non-ionic organic chemicals from soils. Chemosphere. 17:2299–2323.

Sasaki M, Sugimura K, Yoshida MA, Abe S. 1980. Cytogenetic effects of 60 chemicals on cultured human and Chinese hamster cells. Kromosomo II 20:574–584 [cited in IPCS 1990].

Sawada M, Sofuni T, Ishidate M Jr. 1987. Cytogenetic studies on 1,1-dichloroethylene and its two isomers in mammalian cells *in vitro* and *in vivo*. Mutat Res 187:157–163 [cited in IPCS 1990].

Shah JJ, Heyerdahl EK. 1988. National ambient volatile organic compounds (VOCs) database update. Report No. EPA 600/3-88-010(a). Washington (DC): US Environmental Protection Agency.

Short RD, Minor JL, Winston JM, Lee CC. 1977. A dominant lethal study in male rats after repeated exposures to vinyl chloride or vinylidene chloride. J Toxicol Environ Health 3:965–968 [cited in IPCS 1990].

Siegel J, Jones RA, Coon RA, Lyon JP. 1971. Effects on experimental animals of acute, repeated and continuous inhalation exposures to dichloroacetylene mixtures. Toxicol Appl Pharmacol 18:168–174 [cited in IPCS 1990].

Siegers C-P, Heidbuchel K, Younes M. 1983. Influence of alcohol, dithiocarb or (+)-catechin on the hepatotoxicity and metabolism of vinylidene chloride in rats. J Appl Toxicol 3:90–95 [cited in IPCS 1990].

Simmon VF, Tardiff RG. 1978. The mutagenicity activity of halogenated compounds found in chlorinated drinking water. In: Jolley RL, Gorchev H, Hamilton DH Jr, editors. Water chlorination: Environmental impact and health effects. Vol. 2. Ann Arbor (MI): Ann Arbor Science. p. 417–431 [cited in US EPA 2002a].

Solvay SA. 2010. PVDC [Internet]. Brussels (BG): Solvay S.A. [cited 2010 Jun 3]. Available from: http://www.solvayplastics.com/services/specialtypolymers/pvdc/0,,71968-2-0,00.htm

Speerschneider P, Dekant, W. 1995. Renal tumorigenicity of 1,1-dichloroethene in mice: the role of male specific expression of cytochrome P450 2E1 in the renal bioactivation of 1,1-dichloroethene. Toxicol Appl Pharmacol 130(1):48–56.

Strobel K, Grummt T. 1987. Aliphatic and aromatic hydrocarbons as potential mutagens in drinking water. III. Halogenated ethanes and ethenes. Toxicol Environ Chem 15:101–128 [cited in US EPA 2002a].

[TaPL3] Long Range Transport and Persistence Level III model [Internet]. 2000. Version 2.10. Peterborough (ON): Trent University, Canadian Environmental Modelling Centre. Available from: http://www.trentu.ca/academic/aminss/envmodel/models/TaPL3.html

[TDWS] Toronto Drinking Water Systems. 2008. Drinking Water Systems 2008 Annual Report. Toronto (ON).

Thiess AM, Frentzel-Beyme R, Penning E. 1979. Mortality study of vinylidene chloride exposed persons. In: Hien C, Kilian DJ, editors. Proceedings of the 5th Medichem Congress, San Francisco, CA, September, 1977. San Francisco (CA): University of California at San Francisco. p. 270–278 [cited in IARC 1986].

Toronto Water. 2004. Drinking water systems summary report for January 1, 2004 to December 31, 2004. Part III, Form 2, Section 11. Annual report. Available from: http://www.city.toronto.on.ca/water/system_quality/pdf/moe_annual_report_2004.pdf

[TPD NMID] Therapeutic Products Directorate's Non-Medicinal Ingredients Database [Proprietary Database]. 2010. [cited 2010 June]. Available from: Therapeutic Products Directorate, Health Canada.

[US EPA] United States Environmental Protection Agency. 1978. In-depth studies on health and environmental impacts of selected water pollutants. United States Environmental Protection Agency. Contract No. 68-01-4646.

[US EPA] United States Environmental Protection Agency. 1985. Health assessment documents for vinylidene chloride: Final report. Washington (DC): US Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/8-83-03 IF [cited in ATSDR 1994].

[US EPA] United States Environmental Protection Agency. 1995. Exposure profiles for HAPs– Group 1, Vinylidene Chloride. Available from: http://www.epa.gov/fedrgstr/EPA-TOX/1996/June/Day-26/pr-24153DIR/Support

[US EPA] United States Environmental Protection Agency. 1999. Assessment Tools for the Evaluation of Risk (ASTER) System. Duluth (MN): United States Environmental Protection Agency, Mid-Continent Ecology Division.

[US EPA] United States Environmental Protection Agency. 2002a. Voluntary Children's Chemical Evaluation Program (VCCEP). Peer consultation document submitted by The Dow Chemical Company, November 2002. [cited 2009 Dec. 9]. Available from: http://www.tera.org/peer/VCCEP/VDC/VDC%20DOW%20Submission.pdf

[US EPA] United States Environmental Protection Agency. 2002b. Toxicological review of 1,1-dichloroethylene (CAS No. 74-35-4). In support of summary information on the Integrated Risk Information System (IRIS). June 2002. Washington (DC): US Environmental Protection Agency. (EPA/635/R02/002). Available from: http://www.epa.gov/iris/toxreviews/0039-tr.pdf

[US EPA] United States Environmental Protection Agency. 2003. Technology transfer network air toxics website, US Environmental Protection Agency. Vinylidene chloride (1,1-dichloroethylene). February 12, 2003. 5 p. Available from: http://www.epa.gov/ttn/atw/hlthef/di-ethyl.html

[US EPA] United States Environmental Protection Agency. 2006. SCREEN3 air dispersion model. Support Center for Regulatory Air Models, February 2006. US Environmental Protection Agency [downloaded 2006 March]. Available from: http://www.epa.gov/scram001/dispersion_screening.htm#screen3

[US FDA] United States Food and Drug Administration. 2006. Office of food additive safety – Food additive status list [accessed 2009 Feb 21]. Available from: http://www.fda.gov/Food/FoodIngredientsPackaging/FoodAdditives/FoodAdditiveListings/ucm091048.htm

Utilities Kingston. 2008. Utilities Kingston 2008 Annual Report, January 1, 2008 – December 31, 2008. Kingston (ON): Utilities Kingston.

Van Duuren BL, Goldschmidt BM, Loewengart G, Smith AC, Melchionne S, Seldman I, Roth D. 1979. Carcinogenicity of halogenated olefinic and aliphatic hydrocarbons in mice. J Natl Cancer Inst 63:1433–1439 [cited in US EPA 2002a].

[VICH] International Cooperation on Harmonisation of Technical Requirements for Registration of Veterinary Medicinal Products. 2000. Impurities: Residual Solvents in New Veterinary Medicinal Products, Active Substances and Excipients. Brussels (BG): International Cooperation on Harmonisation of Technical Requirements for Registration of Veterinary Medicinal Products. [cited 2010 June]. Available from: http://www.vichsec.org/pdf/2000/Gl18_st7.pdf

Ville de Montréal. 2006. Municipal drinking water produced by Atwater and Charles-J Des Baillets drinking water plants. Montréal (QC): Division de l'expertise technique, Service des infrastructures et de l'environnement.

Ville de Québec. 2002. 2002 Quebec Water Testing. Quebec (QC).

Viola PL, Caputo A. 1977. Carcinogenicity studies on vinylidene chloride. Environ Health Perspect 21:45–47 [cited in US EPA 2002a].

Viswanathan R. 1984. Regenwurmtest. In Ballhorn L, Freitag D. editors. Überprüfung der Durchführbarkeit von Prüfungvorschriften und der aussagekraft der Stufe I and II des E.Chem. G. Neuherberg, Gesellschaft für Strahlen- und Umwelt-forschung München mbH. p. 124–131 [cited in WHO 2003a].

Waskell L. 1978. A study of the mutagenicity of anesthetics and their metabolism. Mutat Res 57:141–153 [cited in US EPA 2002a].

Waxweiler RJ, Smith AH, Falk H, Tyroler HA. 1981. Excess lung cancer risk in a synthetic chemicals plant. Environ Health Perspect 41:159-165 [cited in IARC 1986].

Westrick JJ, Mello JW, Thomas RF. 1984. The groundwater supply survey. J Am Waterworks Assoc 76(5):52-59 [cited in ATSDR 1994].

[WHO] World Health Organization. 2003a. Concise International Chemical Assessment Document 51, 1,1-dichloroethene (vinylidene chloride). Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organization, and the World Health Organization. ISSN 1020-61767. 36 pp.

[WHO] World Health Organization. 2003b. 1,1-Dichloroethane in drinking-water: Background document for development of WHO Guidelines for drinking-water quality. Geneva (CH): World Health Organization. WHO/SDE/WSH/03.04/19.

Williams PRD, Patterson J, Briggs DW. 2006. VCCEP Pilot: Progress on evaluating children's risks and data needs. Risk Analysis 26(3):781–801. [cited 2009 Dec. 9]. Available from: http://www.tera.org/Publications/williams%20et%20al%20accepted.pdf

Wilson SC, Burnett V, Waterhouse KS, Jones KC. 1994. Volatile organic compounds in digested United Kingdom sewage sludges. Environ Sci Technol 28:259–266.

Zeller H, Klimisch JH, Freisberg KO. 1979a. [Report on the determination of acute toxicity (LC_{50}) by inhalation of vinylidene chloride in NMRI mice (fed) during a 4-hour exposure.] BASF Aktiengesellschaft, Ludwigshafen. 12 p. [in German] [cited in IPCS 1990].

Zeller H, Klimisch JH, Freisberg KO. 1979b. [Report on the determination of acute toxicity (LC_{50}) by inhalation of vinylidene chloride in NMRI mice (fed) during a 4-hour exposure.] BASF Aktiengesellschaft, Ludwigshafen. 12 p. [in German] [cited in IPCS 1990].

Zeller H, Klimisch JH, Freisberg KO. 1979c. [Report on the determination of acute toxicity (LC_{50}) of vinylidene chloride in Sprague-Dawley rats (fed) during a 4-hour exposure.] BASF Aktiengesellschaft, Ludwigshafen. 14 p. [in German] [cited in IPCS 1990].

Zeller H, Klimisch JH, Freisberg KO. 1979d. [Report on the determination of acute toxicity (LC_{50}) by inhalation of vinylidene chloride in vapour form in Sprague-Dawley rats (fasting) during a 4-hour exposure period.] BASF Aktiengesellschaft, Ludwigshafen. 14 p. [in German] [cited in IPCS 1990].

Zhu J, Newhook R, Marro L, Chan CC. 2005. Selected volatile organic compounds in residential air in the City of Ottawa, Canada. Environ Sci Technol 39: 3964–3971.

Zogorski JS, Carter JM, Ivahnenko T, Lapham WW, Moran MJ, Rowe BL, Squillace PJ, Toccalino PL. 2006. Volatile organic compounds in the nation's ground water and drinking-water supply wells. [Internet]. US Geological Survey, US Department of the Interior. [cited 2009 Aug. 4]. Available from: http://pubs.usgs.gov/circ/circ1292/pdf/circ1292_front.pdf

Appendix 1. Concentrations of 1,1-DCE in different media

Table A1. Concentration of 1,1-DCE in ambient air

Location	Sampling period	Number of samples	Detection limit (µg/m^3)	Concentration[1] (µg/m^3)	Reference
Windsor, Ontario	January 23 to March 25, 2006	214	0.046	Arithmetic mean and median: 0.023[3] (all ND)	Health Canada 2010b
	July 3 to August 26, 2006	214		Arithmetic mean and median: 0.023[3] (all ND)	
Windsor, Ontario	January 24 to March 19, 2005	201	0.152	Arithmetic mean and median: 0.076[3] (all ND)	Health Canada 2010b
	July 4 to August 27, 2005	216		Arithmetic mean and median: 0.076[3] (all ND)	
Regina, Saskatchewan (full set)	January 8 to March 16, 2007	94 (winter; only 24-h canisters reported)	0.012	Arithmetic mean and median: 0.006[3] (all ND)	Health Canada 2010a
	June 20 to August 29, 2007	97 (summer; 5-day canisters)		Arithmetic mean and median: 0.006[3] (Range: 0.006 – 0.014)	
Canada-wide sites (43 locations)	January to December 2008	1896	0.026	0.013[3]	NAPS 2008
Ottawa, Ontario (residential areas)	Nov 20, 2002 to Mar 11, 2003	74	0.011	Arithmetic mean: 0.05 Median: 0.005 (Range: 0.005 – 0.83) (detected in 13 of 74 samples)	Zhu et al. 2005
Canada-wide sites	1989–1996	9128	ns	0.06 [ND–0.78]; 8% > detection limit)[4]	NAPS 2004
Montréal, Quebec (urban)	1993	160	0.2 (0.05 ppbv)[2]	0.03 [ND–0.30] (14% > detection limit)	Environment Canada 1995
Montréal, Quebec (suburban)	1993	24	0.2 (0.05 ppbv)[2]	0.00 [ND–0.04] (0% > detection limit)	Environment Canada 1995

Sainte-Françoise, Quebec (rural)	1993	34	0.2 (0.05 ppbv)[2]	0.02 [ND–0.12] (6% > detection limit)	Environment Canada 1995
Montréal, Quebec (urban)	1992	166	0.2 (0.05 ppbv)[2]	0.00 [ND–0.02] (0% > detection limit)	Environment Canada 1995
Montréal, Quebec (urban)	1991	91	0.2 (0.05 ppbv)[2]	0.01 [ND–0.22] (4% > detection limit)	Environment Canada 1995
Montréal, Quebec (urban)	1990	110	0.2 (0.05 ppbv)[2]	0.00 [ND–0.11] (2% > detection limit)	Environment Canada 1995
Montréal, Quebec (urban)	1989	76	0.2 (0.05 ppbv)[2]	0.03 [ND–0.44] (13% > detection limit)	Environment Canada 1995
Greater Vancouver Regional District	1989–1992	473	0.2 (0.05 ppbv)[2]	0.05 (4% > detection limit)[3]	Environment Canada 1994
Canada (sites unspecified)	1989–1990	1100	0.2 (0.05 ppbv)[2]	0.06 (9% > detection limit)[3]	Environment Canada 1994
Windsor, Ontario	July 1987 to October 1990	124	ns	ns [ND–0.3] (10 of 124 samples > detection limit)	Environment Canada 1992
Walpole Island, Ontario	January 1988 to October 1990	61	ns	ns [ND–0.2] (8 of 61 samples > detection limit)	Environment Canada 1992
Toronto, Ontario (downtown)	June–August 1990	16	0.4 (MQL = 2.1)	1.9	OME 1991d
Toronto, Ontario (residential)	June–August 1990	7	0.4 (MQL = 2.1)	0.4	OME 1991d
Canada (residential homes)	February–March 1987	6	6 ng/tube (collection vial)	0.3 [ND–1]	Chan et al. 1990
Canada (residential homes)	November–December 1986	12	6 ng/tube (collection vial)	3.2 [ND–7]	Chan et al. 1990

[1] Value in parentheses indicates range of concentrations when available.
[2] Value presented for the detection limit is the target or typical detection limit reported for volatile organic compounds.
[3] Mean calculated with values below detection set to 0.5*Method Detection Limit
[4] Values below detection limit set to 0.05 µg/m^3
MQL = method quantifiable limit
ns = not specified
ND = not detected

Table A2. Concentration of 1,1-DCE in indoor air

Location	Sampling period	Number of samples	Detection limit (µg/m^3)	Concentration[1] (µg/m^3)	Reference
Windsor, Ontario (personal breathing-zone air)	January 24 to March 19, 2005	225	0.152	Winter AM: 0.076 Median: 0.076 P-95: 0.076 R: All ND	Health Canada 2010b
	July 4 to August 27, 2005	207		Summer AM: 0.077 Median: 0.076 P-95: 0.076 R: 0.076 – 0.400	
Windsor, Ontario	January 23 to March 25, 2006	224	0.046	Winter AM: 0.025 Median: 0.023 P-95: 0.023 R: 0.023 – 0.463	Health Canada 2010b
	July 3 to August 26, 2006	211		Summer AM: 0.025 Median: 0.023 P-95: 0.023 R: 0.023 – 0.103	
Windsor, Ontario	January 24 to March 19, 2005	232	0.152	Winter AM: 0.076 Median: 0.076 P-95: 0.076 R: 0.076 – 0.185	Health Canada 2010b
	July 4 to August 27, 2005	217		Summer AM: 0.085 Median: 0.076 P-95: 0.076 R: 0.076 – 1.380	
Regina, Saskatchewan[2] (full set; 5-day data)	January 8 to March 16, 2007	89	0.012	Winter AM: 0.009 Median: 0.006 P-95: 0.027 R: 0.006 – 0.083	Health Canada 2010a
	June 20 to August 29, 2007	101		Summer AM: 0.007 Median: 0.006 P-95: 0.023 R: 0.006 – 0.033	
Ottawa,	Nov 20, 2002 to	75	0.011	AM: 0.27	Zhu et al.

Location	Sampling period	Number of samples	Detection limit (µg/m^3)	Concentration[1] (µg/m^3)	Reference
Ontario (detected in 34 of 75 homes)	Mar 11, 2003			Median: 0.005 P-95: 0.99 R: 0.005 – 4.05	2005
International locations (literature review of 50 studies)	1978–1990	n = 50 studies	ns	Mean: 1–< 5	Brown et al. 1994
Toronto, Ontario (office)	June–August 1990	8	0.4 (MQL = 2.1)	Mean: 5	OME 1991d
Toronto, Ontario (domestic)	June–August 1990	4	0.4 (MQL = 2.1)	Mean: 5.4	OME 1991d
Canada (residential homes)	November–December 1986	12	6 ng/tube (collection vial)	Mean: 8.4 Range: ND–77	Chan et al. 1990
Canada (residential homes)	February/March 1987	6	6 ng/tube (collection vial)	Mean: 3.8 Range: ND–13	Chan et al. 1990
Woodland, California (residential homes)	June 1990	128	0.78 (MQL)	not quantifiable in any sample	CARB 1992
North Carolina (Research Triangle Park area – residential homes)	Summer	15	ns	Detected in 4 of 15 homes, with a mean of 12.06 and a range of 0.46–23.9 µg/m^3	Pleil et al. 1985
North Carolina (Research Triangle Park area – residential homes)	Winter	16	ns	Detected in 4 of 16 homes, with a mean of 1.81 and a range of 1.3–2.5 µg/m^3	Pleil et al. 1985
United States (various sites)	1970–1987	2120	ns	Mean: 5.02 µg/m^3	Shah and Heyerdahl 1988

[1] Mean calculated with values below detection set to 0.5*Method Detection Limit
[2] 5-day canister data were selected as they represent time-weighted average over longer period than 24-h canisters.
R = range
P-95 = 95th percentile
AM = arithmetic mean
MQL = method quantifiable limit
ns = not specified
ND = not detected

Table A3. Concentration of 1,1-DCE in drinking water and groundwater

Location	Sampling period	Number of samples	Detection limit (µg/L)	Mean concentration[1] (µg/L)	Reference
DRINKING WATER					
Victoria, British Columbia	2008	2	0.1	ND	City of Victoria 2008
Vancouver, British Columbia	August 19, 2008	3	0.5	ND	City of Vancouver 2008
Toronto, Ontario	January–December 2008	ns	ns	ND	TDWS 2008
Niagara Falls, Ontario	November 6, 2008	1	0.41	ND	City of Niagara Falls 2008
Saskatoon, Saskatchewan	2008	1	0.2	ND	CSWTP 2008
London, Ontario	June 10, 2008	1	0.41	ND	City of London 2008
Kitchener, Ontario	January–November 2008	6	0.5	ND	OME 2008
Kingston, Ontario	2008	2	0.1	ND	Utilities Kingston 2008
Hamilton, Ontario	February–November 2008	ns	0.2	ND	BCOS 2008
Edmonton, Alberta	2008	ns	ns	ND	EPCOR 2008
Barrie, Ontario	2006	14	ns	ND –"< 0.41"	CBWO 2008
Montréal, Quebec	2006	ns	0.07	ND	Ville de Montreal 2006
Calgary, Alberta	2003	ns	0.5	ND	CCW 2003
Ottawa, Ontario	2003	35	0.52	ND	COWQS 2003
Québec, Quebec	February–November 2002	4	ns	< 0.2 (< 0.1 to < 0.4)	Ville de Québec 2002
United States	1985–2001	n = 1096 (public well samples)	MDL 0.047 (Connor et al. 1998)	< 0.16 (median of all samples) 0.20 (median of samples with detection)	Zogorski et al. 2006
United States	1985–2001	n = 2400 (domestic well samples)	MDL 0.047 (Connor et al. 1998)	<0.18 (median of all samples)	Zogorski et al. 2006

Location	Sampling period	Number of samples	Detection limit (µg/L)	Mean concentration[1] (µg/L)	Reference
				0.026 (median of samples with detection)	
Toronto, Ontario	1986 and 1987	2 (tap water) 7 (bottled water)	0.04	tap water - ND bottled water - ND	City of Toronto 1990
Ontario (water treatment plants, various locations)	1987	44 treatment plants	0.1	raw - ND treated - ND distribution water - ND	OME 1988, 1989
29 Alberta municipal drinking water supplies	1978–1985	ns	ns	ns (detected in one of 29 municipal supplies at a max concentration of 1.4 µg/L	Health Canada 1994a
10 Ontario water treatment plants (Great Lakes locations)	July–August 1982 January–February 1983 April–May 1983	42 raw 42 treated	[0.1–0.4][2]	raw - 0 [ND] treated- < 0.1 [ND–trace (1 sample at less than 0.1)]	Otson 1987
Canada-wide (29 municipalities, 30 water treatment plants)	August–September 1979	30 raw 30 treated	5.0 (MQL)	raw - 0 [ND] treated - < 1 [ND–~20]	Otson et al. 1982b
Canada-wide (29 municipalities, 30 water treatment plants)	November–December 1979	30 raw 30 treated	5.0 (MQL)	raw - 0 [ND] treated - 0 [ND]	Otson et al. 1982b
United States (EPA survey)	ns	ns	ns	detected in 3% of drinking water supplies; 0.3 µg/L (0.2–0.5 µg/L)	US EPA 1985 (cited in ATSDR 1994)

GROUNDWATER					
United States	1985–2001	3497	ns	< 0.20 (median for all samples) 0.068 (median for samples with detection)	Zogorski et al. 2006
Ottawa, Ontario	May 1988	37	ns	detected in 43% of samples [0.9–60]	Lesage et al. 1990
United States (community-based groundwater sources, nationwide survey)	ns	945	0.2 (MQL)	detected in 2.3% of samples (max. 6.3 µg/L, subset median values, 0.28–1.2 µg/L)	Rajagopal and Li 1991 [cited in ATSDR 1994] Westrick et al. 1984 [cited in ATSDR 1994]
United States, Ground Water Supply Survey	1982	466	ns	detected in 9 samples; 0.3 µg/L (median)	Cotruvo 1985 [cited in ATSDR 1994]

[1] Value in parentheses indicates range of concentrations when available.
[2] Value presented for the detection limit is the target or typical detection limit reported for volatile organic compounds.
MDL – method detection limit
MQL - method quantifiable limit
ns = not specified; ND = not detected

Table A4. Concentration of 1,1-DCE in foodstuffs

FOOD					
Item sampled	**Sampling period**	**No. of samples**	**Detection limit**	**Mean concentration (µg/kg)**	**Reference**
Ville-Mercier, Quebec Ice cream Cheese and butter Beef and veal Pork/cured pork Lamb chops Poultry Eggs Organ meats Luncheon meats Canned meats Marine fish Freshwater fish Canned fish Shellfish Canned meat soups Canned pea and tomato soups Dehydrated soups Bread Flour and cakes Cereals Pies Pasta Potatoes and vegetables Rice and vegetables Beets and tomatoes Fruits Juices and canned fruit Oils and fats Peanut butter	January 1993	4	5.0 µg/kg	ND	ETL 1993
Ville-Mercier, Quebec Dairy Coffee and tea Soft drinks Alcohols Water	January 1993	4	1.0 µg/L	ND	ETL 1993

FOOD					
Item sampled	**Sampling period**	**No. of samples**	**Detection limit**	**Mean concentration (µg/kg)**	**Reference**
United Kingdom Biscuits Marshmallow Swiss roll Snack biscuits Crisps and snack foods Whole turkey Black pudding Smoked cheese Liver pate Cooked sausage	ns	ns	1 µg/kg	 < 1 < 1 < 1 < 1 < 1 < 1 6 < 1 5 5	MAFF 1980
United States (food simulants) heptane (0.5-mm film) corn oil (0.5-mm film) water (0.5-mm film) Note: 0.5-mm film is equivalent thickness to plastic wrap used for food applications.	1977	 n = 4 n = 5 n = 4	5–10 ppb	 39 ppb (34–44 ppb) 34 ppb (18–41 ppb) 25 ppb (24–27 ppb)	Hollifield and McNeal 1978
Great Britain (potato crisps)	1979	n = 4	0.005 ppm	0.019 ppm (0.010–0.025 ppm)	Gilbert et al. 1980
Great Britain Biscuits Cakes Snack products Cheeses Cooked meats: Black pudding (n = 1) Liver pate (n = 1) Polony (n = 1) Bacon and liver pate (n = 1)	October 1978	 n = 7 n = 1 n = 3 n =1	0.005 ppm	 ND ND ND ND 0.005–0.01 ppm 0.005–0.01 ppm ND ns	Gilbert et al. 1980
Japan Sausage Fish sausage Boiled fish paste Cheese	August 2004	n = 13	0.001 µg/g	 0.008 µg/g 0.005 µg/g 0.003 µg/g 0.0095 µg/g	Ohno and Kawamura 2006

ND - not detected, below detection limit
ns - not specified

Table A5. Concentration of 1,1-DCE in soil

SOIL					
Location	**Sampling period**	**No. of samples**	**Detection limit (ng/g)[2]**	**Mean concentration[1] (ng/g)**	**Reference**
Ontario regions – urban parkland	ns (~1993)	59	MDL[4] = 2	0.074 [0.039–0.12][3]	OMEE 1993
Ontario regions – rural parkland (not including northwest region)	ns (~1993)	85	MDL[4] = 2	0.016 [0.010–0.024][3]	OMEE 1993
Ontario regions – rural parkland (northwest region)	ns (~1993)	17	MDL[4] = 2	0.097 [0.063–0.098][3]	OMEE 1993

[1] Value in parentheses indicates range of concentrations when available.
[2] The method detection limit is defined as three times the within-run analytical standard deviation and is considered only an estimate that may vary with time (OMEE 1993).
[3] The ranges are derived from the Ontario Typical Range Model released in 1993.
[4] Method detection limit (MDL).

Appendix 2. Upper-bounding Deterministic Estimate of 1,1-DCE Daily Intake (µg/kg-bw per day)

Route of Exposure	Estimated intake (µg/kg-bw per day) of 1,1-DCE by various age groups						
	0–6 months[1,2,3]		0.5–4 yr[4]	5–11 yr[5]	12–19 yr[6]	20–59 yr[7]	60+ yr[8]
	Formula fed	Not formula fed					
Ambient air[9]	2.66×10^{-3}		5.70×10^{-3}	4.44×10^{-3}	2.53×10^{-3}	2.17×10^{-3}	1.89×10^{-3}
Indoor air[10]	1.86×10^{-2}		3.99×10^{-2}	3.11×10^{-2}	1.77×10^{-2}	1.52×10^{-2}	1.32×10^{-2}
Drinking water[11]		1.39×10^{-2}	6.71×10^{-3}	6.71×10^{-3}	3.50×10^{-3}	2.93×10^{-3}	2.89×10^{-3}
Food and beverages[12]	5.55×10^{-2}	1.310	0.859	0.549	0.320	0.240	0.196
Soil[13]	<0.001		<0.001	<0.001	<0.001	<0.001	<0.001
Total intake	7.67×10^{-2}	1.340	0.911	0.591	0.344	0.260	0.214
Maximum total intake from all routes of exposure:							1.34

[1] No data were available for the presence of 1,1-DCE in breast milk, although it has been qualitatively detected in two related studies of breast milk from four cities across the United States, detection limits unspecified (Erickson et al. 1980; Pellizzari et al. 1982).

[2] Assumed to weigh 7.5 kg, to breathe 2.1 m^3 of air per day, drink 0.8 L of water per day (formula fed) or 0.3 L/day (not formula-fed) and ingest 30 mg of soil per day (Health Canada 1998).

[3] For exclusively formula-fed infants, intake from water is synonymous with intake from food. The concentration of 1,1-DCE in water used to reconstitute formula was the detection limit (0.52 µg/L) of a study of distribution water and raw and treated water located at two treatment plants in Ottawa, Ontario, in 2003 (COWQS 2003). No data on concentrations of 1,1-DCE in formula milk were identified for Canada. Approximately 50% of not-formula-fed infants are introduced to solid foods by 4 months of age and 90% by 6 months of age (NHW 1990 in Health Canada 1998).

[4] Assumed to weigh 15.5 kg, to breathe 9.3 m^3 of air per day, drink 0.7 L of water per day and ingest 100 mg of soil per day (Health Canada 1998).

[5] Assumed to weigh 31.0 kg, to breathe 14.5 m^3 of air per day, drink 1.1 L of water per day and ingest 65 mg of soil per day (Health Canada 1998).

[6] Assumed to weigh 59.4 kg, to breathe 15.8 m^3 of air per day, and to drink 1.2 L of water per day and ingest 30 mg of soil per day (Health Canada 1998).

[7] Assumed to weigh 70.9 kg, to breathe 16.2 m^3 of air per day, drink 1.5 L of water per day and ingest 30 mg of soil per day (Health Canada 1998).

[8] Assumed to weigh 72.0 kg, to breathe 14.3 m^3 of air per day, drink 1.6 L of water per day and ingest 30 mg of soil per day (Health Canada 1998).

[9] The median concentration of 1,1-DCE in outdoor air in Windsor, Ontario for 2005 of 0.076 µg/m^3 was used in deriving the intake estimate (Health Canada 2010b). This data point was selected as it represents the highest median concentration across recent Canadian outdoor air studies. Canadians are assumed to spend 3 h per day outdoors (Health Canada 1998). The critical data were identified from a dataset of studies of ambient air (Zhu et al. 2005; NAPS 2008; Health Canada 2010a, 2010b; Environment Canada 1992, 1994, 1995; OME 1991d; Chan et al. 1990).

[10] The median concentration of 1,1-DCE in indoor air in Windsor, Ontario during 2005 of 0.076 µg/m^3 was used in deriving the intake estimate (Health Canada 2010b). This data point was selected as it represents the highest median concentration across recent Canadian indoor air studies. Canadians are assumed to spend 21 h per day indoors (Health Canada 1998). The critical data were identified from a dataset of indoor air studies from Canada and international sites, primarily the United States (Health Canada 2010a, 2010b; Zhu et al. 2005; Brown et al. 1994; OME 1991d; CARB 1992; Chan et al. 1990; Pleil et al. 1985; Shah and Heyerdahl 1988).

[11] The detection limit (0.52 µg/L) of a study of distributed water and raw and treatment water located at two treatment plants in Ottawa, Ontario, in 2003 (n = 35 samples) was used as the most conservative estimate of exposure. Consumption estimates are for "total tap water" (Health Canada 1998). The critical data were identified from a dataset of drinking water studies from Canada and the United States (CBWO 2008; City of Victoria 2008; City of Vancouver 2008; TDWS 2008; City of Niagara Falls 2008; CSWTP 2008; City of London 2008; OME

2008; Utilities Kingston 2008; BCOS 2008; EPCOR 2008; Ville Montréal 2006; CCW 2003; COWQS 2003; Ville de Québec 2002; City of Toronto 1990; Zogorski et al. 2006; OME 1988, 1989; Health Canada 1994; Otson et al. 1982b; Otson 1987; US EPA 1985).

[12] In the absence of detected amounts in foods analyzed in Canada (ETL 1991, 1992, 1993), estimates of intakes for some food groups were based on studies conducted in Japan and the United Kingdom. The intake analysis is based on the following selected food groups (Health Canada 1998):

- Dairy products: 9.5 µg/kg; concentration measured in cheese in Japan (Ohno and Kawamura 2006)
- Fats: 34 µg/kg; mean concentration in corn oil (Hollifield and McNeal 1978)
- Fruits: 5.0 µg/kg; detection limit of fruit, canned fruit and juices in Ville-Mercier, Quebec (ETL 1993)
- Vegetables: 19 µg/kg; mean concentration measured in potato crisps in Great Britain (Gilbert et al. 1980)
- Cereal products: 5.0 µg/kg; detection limit in study in Ville-Mercier, Quebec (ETL 1993)
- Meat and poultry: 10.0 µg/kg; maximum concentration measured in black pudding and liver pate in Great Britain (Gilbert et al. 1980). Mean concentrations of 1,1-DCE in black pudding and liver pate were not provided in this study, only concentration ranges of 5.0–10.0 µg/kg (Gilbert et al. 1980). The detected levels of 1,1-DCE (above detection limit of 5.0 µg/kg) tended to be at the outer edges of these cooked meat products (Gilbert et al. 1980).
- Fish: 5.0 µg/kg; concentration measured in fish sausage in Japan (Ohno and Kawamura 2006)
- Eggs: 5.0 µg/kg; detection limit in study in Ville-Mercier, Quebec (ETL 1993)
- Foods, primarily sugar: 1 µg/kg; detection limit for marshmallow in the United Kingdom (MAFF 1980)
- Mixed dishes and soups: 5.0 µg/kg; detection limit in study in Ville-Mercier, Quebec (ETL 1993)
- Nuts and seeds: 5.0 µg/kg; detection limit for peanut butter in Ville-Mercier, Quebec (ETL 1993)
- Soft drinks and alcohol: 1.0 µg/L; detection limit for study in Ville-Mercier, Quebec (ETL 1993)

[13] The weighted average of Ontario urban parkland, rural parkland (not including northwest region) and rural parkland (northwest region) soil of 0.046 µg/kg solids of 161 samples was used in generating the intake estimate (OMEE 1993).

Appendix 3. Summary of health effects information for 1,1-dichloroethene

Endpoint	Lowest effect levels[1] / Results
Acute toxicity	**Lowest inhalation LC_{50}** (mouse) = 200 mg/m^3 (Zeller et al. 1979a, 1979b, 1979c, 1979d) [Additional studies: Carpenter et al. 1949; Siegel et al. 1971; Jaeger et al. 1973, 1974; Klimisch and Freisberg, 1979a, 1979b; Zeller et al. 1979a, 1979b, 1979c, 1979d] **Lowest oral LD_{50}** (mouse) = 194 mg/kg-bw (Jones and Hathway 1978a) [Additional studies: Jenkins et al. 1972; Andersen and Jenkins 1977; Ponomarkov and Tomatis 1980]
Short-term repeated-dose toxicity	**Lowest inhalation LOEC** (rat) = 200 mg/m^3: fatty changes and focal liver cell necrosis (4 weeks) (Plummer et al. 1990); changes to the liver and kidneys (7 days, with observation period to 28 days) (Maltoni and Patella 1983) [Additional studies: Gage 1970; Short et al. 1977; Oesch et al. 1983; Norris and Reitz 1984] **Lowest oral LOEL** (gavage) (rat) = 200 mg/kg-bw (2 times per week): increased serum sorbitol dehydrogenase and aminotransferases indicative of hepatotoxicity (4 weeks) (Siegers et al. 1983) [Additional studies: NTP 1982; Maltoni and Patella 1983]
Subchronic toxicity	**Lowest inhalation LOEC** (rat) = 100 mg/m^3: minimal, reversible liver cell cytoplasmic vacuolation (90 days) (Norris 1977; Quast et al. 1977) [Additional studies: Lazarev 1960; Prendergast et al. 1967] **Lowest oral LOEL** (rat) = 19 mg/kg-bw per day: minimal, recoverable liver cell cytoplasmic vacuolation (90 days) (Norris 1977; Quast et al. 1977) [Additional studies: NTP 1982; Quast et al. 1983]

Endpoint	Lowest effect levels[1] / Results
Chronic toxicity/ carcinogenicity	**Lowest inhalation LOAEC** (mice) = 40 mg/m^3: significant increases in kidney damage (regressive changes and/or abscesses and nephritis in males) (52 weeks) (Maltoni et al. 1984, 1985) [Additional studies: Lee et al. 1977; Rampy et al. 1977, 1978; Viola and Caputo 1977; Hong et al. 1981; Quast et al. 1986; Cotti et al. 1988] **Lowest oral LOEL** (rat) = 5 mg/kg-bw per day: increased incidence of chronic renal inflammation in male and female F344/N rats, 2-year gavage study (NTP 1982) [Additional studies: Ponomarkov and Tomatis 1980; Quast et al. 1983; Maltoni et al. 1984, 1985] **Inhalation study in Swiss mice:** 0, 10 or 25 ppm (0, 40 or 100 mg/m^3; conversion by IPCS 1990) for 52 weeks; significantly increased incidence of renal adenocarcinomas (0/126, 0/25 and 28/119 for the control, low and high concentrations, respectively) in males at 100 mg/m^3; mammary carcinomas (3/185, 6/30 and 16/148 for the control, low and high concentrations, respectively) in females and pulmonary adenomas (12/331, 14/58 and 41/288 for the control, low and high concentrations, respectively) in males and females were not clearly exposure-related (Maltoni et al. 1984, 1985) No significant increases in tumours considered to be related to exposure were observed in rats or hamsters in inhalation bioassays or in any species in studies by oral, dermal or subcutaneous routes of exposure (Lee et al. 1977, 1978; Rampy et al. 1977, 1978; Viola and Caputo 1977; Van Duuren et al. 1979; Hong et al. 1981; NTP 1982; Quast et al. 1983, 1986; Maltoni et al. 1984, 1985). **Dermal initiation–promotion study in female mice:** initiation with 1,1-DCE; promotion by phorbol myristate acetate for 428–576 days, beginning 14 days after exposure to 1,1-DCE; 8/30 treated mice with lung papillomas versus 9/120 controls (Van Duuren et al. 1979)
Developmental toxicity	**Lowest inhalation LOAEC** (mouse) = 60 mg/m^3: significant increase in the mean number of fetuses with an unossified incus and incompletely ossified sternebrae (gestation days 6–16); maternal LOEC = 119 mg/m^3, based upon decrease in weight gain (Short et al. 1977) [Additional studies: Murray et al. 1979] **Lowest oral LOEL** (maternal, rat) = 14 mg/kg-bw per day; *dams:* minimal hepatocellular fatty change; reversible, accentuated hepatic lobular pattern; *pups:* no effects were observed (three-generation study) (Nitschke et al. 1983) Note: Although 0.02 mg/kg-bw per day (rat) was the lowest identified oral LOAEL (Dawson et al. 1993), based on several factors, the US EPA (2002a) could not conclude that exposure to 1,1-DCE caused these effects. [Additional studies: Murray et al. 1979]

Endpoint	Lowest effect levels[1] / Results
Genotoxicity and related endpoints: *in vivo*	**CHROMOSOMAL ABERRATIONS** **Positive results:** Hamster, bone marrow (Hofmann and Peh 1976) [inhalation; 120 or 400 mg/m^3, 6 hours/day, 5 days/week, 6 weeks] **Negative results:** Rat, bone marrow (Rampy et al. 1977) [inhalation; 100 or 300 mg/m^3, 6 hours/day, 5 days/week, 6 months]; mouse, bone marrow (Cerna and Kypenova 1977) [intraperitoneal injection for 5 days] **DNA ADDUCT FORMATION** **Positive results:** CD1-mice [inhalation; 40 or 200 mg/m^3, 6 hours], Sprague-Dawley rats [inhalation, 40 mg/m^3, 6 hours], liver and kidney (Reitz et al. 1980) **DOMINANT LETHAL TEST** **Negative results:** Mouse (Andersen and Jenkins 1977) [inhalation; 50 ppm (198 mg/m^3), 6 hours/day, 5 days]; rat (Short et al. 1977) [inhalation; 55 ppm (218 mg/m^3), 6 hours/day, 5 days/week, 11 weeks] **MICRONUCLEI TEST** **Negative results:** Mouse, bone marrow [oral, 200 mg/kg-bw]; mouse, fetal erythrocytes [oral, 100 mg/kg-bw] (Sawada et al. 1987) **NON-MAMMALIAN SEX-LINKED RECESSIVE LETHAL ASSAY** **Negative results:** *Drosophila* (Foureman et al. 1994) [oral, 20 000 or 25 000 ppm, 72 hours; or injection, 5000 ppm, 24 hours] **UNSCHEDULED DNA SYNTHESIS** **Positive results:** CD-1 mice, liver and kidney (Reitz et al. 1980) [inhalation; 200 mg/m^3, 6 hours]
Genotoxicity and related endpoints: *in vitro*	**ANEUPLOIDY** **Positive results:** *Saccharomyces cerevisiae*, with and without activation (Koch et al. 1988) **CHROMOSOMAL ABERRATIONS** **Positive results:** Chinese hamster lung cells, with activation (Sawada et al. 1987) **Negative results:** Chinese hamster lung cells, without activation (Sawada et al. 1987); Chinese hamster fibroblast CHL cells (Ishidate 1983); Chinese hamster DON-6 cells (Sasaki et al. 1980) **GENE CONVERSION**

Endpoint	Lowest effect levels[1] / Results
	Positive results: *S. cerevisiae*, without activation (Koch et al. 1988); *S. cerevisiae*, with activation (Bronzetti et al. 1981) **Negative reults:** *S. cerevisiae*, without activation (Bronzetti et al. 1981); *S. cerevisiae*, with activation (Koch et al. 1988) **MUTAGENICITY** **Positive results:** *Salmonella typhimurium* BA13/BAL13, with activation (Roldan-Arjona et al. 1991) *S. typhimurium* TA100, with activation (Bartsch et al. 1975, 1979; Baden et al. 1976, 1978, 1982; Jones and Hathway 1978b; Simmon and Tardiff 1978; Waskell 1978; Oesch et al. 1983; Strobel and Grummt 1987; Malaveille et al. 1997) *S. typhimurium* TA100, without activation (Baden et al. 1976, 1978, 1982; Cerna and Kypenova 1977; Waskell 1978; Strobel and Grummt 1987) *S. typhimurium* TA1535, with activation (Baden et al. 1977; Jones and Hathway 1978b; Oesch et al. 1983) *S. typhimurium* TA1535, without activation (Cerna and Kypenova 1977) *S. typhimurium* TA1537, with activation (Oesch et al. 1983) *S. typhimurium* TA1538, without activation (Cerna and Kypenova 1977) *S. typhimurium* TA98, with activation (Oesch et al. 1983; Strobel and Grummt 1987) *S. typhimurium* TA98, without activation (Cerna and Kypenova 1977) *S. typhimurium* TA92, with activation (Oesch et al. 1983) *S. typhimurium* TA97, with activation (Strobel and Grummt 1987) *Escherichia coli* K12, with activation (Oesch et al. 1983) *E. coli* K12, without activation (Greim et al. 1975) *E. coli* WP2, with activation (Oesch et al. 1983) *S. cerevisiae*, with activation (Bronzetti et al. 1981; Koch et al. 1988); *S. cerevisiae*, without activation (Koch et al. 1988) Mouse lymphoma L5178Y T/K +/- cells, with activation (McGregor et al. 1991) **Negative results:** *S. typhimurium* BA13/BAL13, without activation (Roldan-Arjona et al. 1991) *S. typhimurium* TA100, with activation (Mortelmans et al. 1986) *S. typhimurium* TA100, without activation (Bartsch et al. 1975, 1979; Simmon and Tardiff 1978; Oesch et al. 1983; Mortelmans et al. 1986) *S. typhimurium* TA104, with and without activation (Strobel and Grummt 1987) *S. typhimurium* TA1535, with activation (Mortelmans et al. 1986) *S. typhimurium* TA1535, without activation (Baden et al. 1977; Oesch et al. 1983; Mortelmans et al. 1986) *S. typhimurium* TA1537, with activation (Mortelmans et al. 1986) *S. typhimurium* TA1537, without activation (Oesch et al. 1983; Mortelmans et al. 1986) *S. typhimurium* TA98, with activation (Mortelmans et al. 1986) *S. typhimurium* TA98, without activation (Oesch et al. 1983; Mortelmans et al.

Endpoint	Lowest effect levels[1] / Results
	1986; Strobel and Grummt 1987) *S. typhimurium* TA92, without activation (Oesch et al. 1983) *S. typhimurium* TA97, without activation (Strobel and Grummt 1987) *E. coli* K12, without activation (Oesch et al. 1983) *E. coli* WP2, without activation (Oesch et al. 1983) Chinese hamster lung V79 cells, *hprt* locus, with and without activation (Drevon and Kuroki 1979) Chinese hamster lung V79 cells, ouabain resistance, with and without activation (Drevon and Kuroki 1979) **SISTER CHROMATID EXCHANGE** **Positive results:** Chinese hamster lung cells, with activation (Sawada et al. 1987); Chinese hamster ovary cells (McCarroll et al. 1983) **Negative results:** Chinese hamster lung cells, without activation (Sawada et al. 1987) **UNSCHEDULED DNA SYNTHESIS** **Positive results:** Rat, hepatocytes (Costa and Ivanetich 1982)
Metabolism	1,1-DCE is rapidly absorbed following inhalation and oral exposures. The major route of excretion for unchanged 1,1-DCE is through the lung. Intraperitoneal (i.p.) administration of 125 mg/kg 14C-1,1-DCE to mice resulted in the highest concentrations of covalent binding (based on protein content) in the kidney, lung and liver. The covalent binding and cellular damage in kidney, lung and liver correlated with the high concentration of CYP2E1 Oxidation of 1,1-DCE by CYP2E1 should produce three metabolites: 1,1-DCE epoxide, 2-chloroacetyl chloride, and 2,2-dichloroacetaldehyde.The epoxide, and perhaps to a lesser extent the chloroacetaldehyde, are believed to be associated with the tissue reactivity and toxic effects in tissues that ensue after significant depletion of GSH. 1,1-DCE does not bioaccumulate in tissues to a significant extent. When the inhalation exposure was less than 100 ppm, the estimated amount of epoxide formed was fivefold lower in humans than in rats (US EPA 2002b).
Epidemiology	Cohort of 138 U.S. workers exposed to 1,1-DCE, where vinyl chloride was not used as a copolymer. Twenty-seven workers were lost to follow-up but considered to be alive in the analyses. Fifty-five people had less than 15 years since first exposure, and only five deaths were observed. The authors indicate no finding was statistically attributable to exposure to 1,1-DCE (Ott et al. 1976). Cohort of 629 males (447 German and 182 foreign workers) employed at two plants in the Federal Republic of Germany that had produced 1,1-DCE since 1955. Vital status was ascertained for 97% of the 447 German workers. Of the 182 foreign workers, 65 had worked for less than one year, and only 24% (44) were traced. Observed deaths were compared with local and regional rates, without making allowance for a latent period. Within the study period (approximately 20 years), 39 deaths were observed, where 57 [local] and 36 [regional] would have been expected. Five cases of lung carcinoma were observed, whereas 3.9 [local]

Endpoint	Lowest effect levels[1] / Results
	and 2.2 [regional] were expected; this result was not statistically significant. Workers in the factory were also potentially exposed to vinyl chloride and acrylonitrile (Thiess et al. 1979). The International Agency for Research on Cancer Working Group noted that both Ott et al. (1976) and Thiess et al. (1979) suffered from the limited size of cohorts, the short observation period and the small numbers of deaths from specific causes. The fact that no allowance was made for latent period may have resulted in an overestimation of the expected numbers and an underestimation of risk. In an attempt to identify the specific exposure associated with an excess lung cancer risk noted previously in a U.S. synthetic chemicals plant, Waxweiler et al. (1981) considered 19 chemicals, one of which was 1,1-DCE. Company personnel assigned a rank of exposure to 1,1-DCE (from 0 to 5) to each job in the plant for each year since its opening in 1942. These exposure data were then linked with detailed, individual work histories to obtain an individual estimate for each of the 4806 male workers employed at the plant. The doses calculated were the product of the exposure rank of the job and the number of days worked at that job. Cumulative doses for 45 workers who had died of lung cancer during the study period of 1942–1973 were then compared to expected doses based on the cumulative exposure of subcohorts of fellow workers matched individually to the cases by year of birth and age of hire into the plant. This comparison failed to suggest any specific association between exposure to 1,1-DCE in the plant and excess lung cancer risk.

[1.] LC_{50} = median lethal concentration; LD_{50} = median lethal dose; LOEC = lowest-observed-effect concentration; LOEL = lowest-observed-effect level; LOAEL = lowest-observed-adverse-effect level; LOAEC = lowest-observed-adverse-effect concentration

Appendix 4. Robust Study Summaries for Ecotoxicity Studies

Table A6. Robust Study Summary – Aquatic Toxicity – Alga

No.	Item	Weight	Yes/No	Specify
1	Reference: Brack W, Rottler H. 1994. Toxicity testing of highly volatile chemicals with green algae – a new assay. Environ Sci Pollut Res 1(4):223–228.			
2	Substance identity: 75-35-4	n/a[1]	Y	
3	Substance identity: 1,1-dichloroethene	n/a	Y	The test substance name
4	Chemical composition of the substance	2	Y	The test substance name
5	Chemical purity	1	Y	> 99%
6	Persistence/stability of test substance in aquatic solution reported?	1	Y	Data are available but not included in the study. See Table 4a
	Method			
7	Reference	1	N	New approach test
8	OECD, EU, national, or other standard method?	3	Y	Based on OECD tests
9	Justification of the method/protocol if a non-standard method was used	2	Y	
10	GLP (good laboratory practice)	3	N	n/a
	Test organism			
11	Organism identity: *Chlamydomonas reinhardtii*	n/a	Y	Green alga
12	Latin or both Latin and common names reported?	1	Y	Green alga
13	Life cycle age / stage of test organism	1		n/a
14	Length and/or weight	1		n/a
15	Sex	1		n/a
16	Number of organisms per replicate	1		n/a
17	Organism loading rate	1		NA[2]
18	Food type and feeding periods during the acclimation period	1	Y	Light, CO_2 source
	Test design / conditions			
19	Test type (acute or chronic)	n/a	Y	Acute
20	Experiment type (laboratory or field)	n/a	Y	Lab
21	Exposure pathways (food, water, both)	n/a	Y	Water
22	Exposure duration	n/a	Y	72 hrs
23	Negative or positive controls (specify)	1	Y	Negative
24	Number of replicates (including controls)	1	Y	
25	Nominal concentrations reported?	1	N	
26	Measured concentrations reported?	3	Y	
27	Food type and feeding periods during the long-term tests	1		n/a
28	Were concentrations measured periodically (especially in the chronic test)?	1	Y	
29	Were the exposure media conditions relevant to the particular chemical reported? (e.g. for the metal toxicity – pH, DOC/TOC, water hardness, temperature)	3	Y	
30	Photoperiod and light intensity	1	Y	
31	Stock and test solution preparation	1	Y	
32	Was solubilizer/emulsifier used if the chemical was poorly soluble or unstable?	1		n/a
33	If solubilizer/emulsifier was used, was its concentration reported?	1		n/a
34	If solubilizer/emulsifier was used, was its ecotoxicity reported?	1		n/a

35	Monitoring intervals (including observations and water quality parameters) reported?	1	Y	
36	Statistical methods used	1	Y	
Information relevant to the data quality				
37	Was the endpoint directly caused by the chemical's toxicity, not by the organism's health (e.g. when mortality in the control > 10%) or physical effects (e.g. "shading effect")?	n/a	Y	
38	Was the test organism relevant to the Canadian environment?	3	Y	
39	Were the test conditions (pH, temperature, DO, etc.) typical for the test organism?	1	Y	
40	Do system type and design (static, semi-static, flow-through; sealed or open; etc.) correspond to the substance's properties and organism's nature/habits?	2	Y	
41	Was pH of the test water within the range typical for the Canadian environment (6 to 9)?	1	Y	
42	Was temperature of the test water within the range typical for the Canadian environment (5 to 27°C)?	1	Y	
43	Was toxicity value below the chemical's water solubility?	3	Y	
Results				
44	Toxicity values (specify endpoint and value)	n/a	EC_{10}, EC_{50}	Growth
45	Other endpoints reported - e.g. BCF/BAF, LOEC/NOEC (specify)?	n/a	N	
46	Other adverse effects (e.g. carcinogenicity, mutagenicity) reported?	n/a	N	
47	***Score*: ... %**	***35/40 = 87.5***		
48	***EC reliability code*:**	***1***		
49	***Reliability category (high, satisfactory, low)*:**	***High Confidence***		
50	***Comments***			

[1] n/a – not applicable.
[2] NA – not available.

Table A7. Robust Study Summary – Terrestrial Toxicity – Mammals

No.	Item	Weight	Yes/No	Specify
1	Reference: Prendergast J, Jones R, Jenkins Jr L, Siegel J. 1967. Effects on experimental animals of long-term inhalation of trichloroethylene, carbon tetrachloride, 1,1,1-trichloroethane, dichlorofluoromethane and 1,1-dichloroethylene. Toxicol Appl Phamacol 10:270–289.			
2	Substance identity: CAS RN	n/a[1]	N	
3	Substance identity: 1,1-dichloroethene	n/a	Y	The test substance name
4	Chemical composition of the substance	2	Y	The test substance name
5	Chemical purity	1	Y	Reagent grade
6	Persistence/stability of test substance?	1	Y	Data are available but not included in the study. See Table 4a
	Method			
7	Reference	1	Y	
8	OECD, EU, national, or other standard method?	3	N	
9	Justification of the method/protocol if a non-standard method was used	2	Y	
10	GLP (good laboratory practice)	3		NA[2]
	Test organism			
11	Organism identity: rats (Sprague-Dawley or Long-Evans), guinea pigs (Hartley), squirrel monkeys, rabbits (New Zealand albino), beagle dogs	n/a	Y	
12	Latin or both Latin and common names reported?	1	N	
13	Life cycle age / stage of test organism	1	N	
14	Length and/or weight	1	Y	Trends noted
15	Sex	1		n/a to study
16	Number of organisms per replicate	1	Y	
17	Organism loading rate	1	Y	
18	Food type and feeding periods during the acclimation period	1	Y	
	Test design / conditions			
19	Test type (acute or chronic)	n/a	Y	90-day inhalation or "work week" inhalation
20	Experiment type (laboratory or field)	n/a	Y	Lab
21	Exposure pathways (food, water, both)	n/a	Y	Air
22	Exposure duration	n/a	Y	90-day or 5-day
23	Negative or positive controls (specify)	1	Y	Negative
24	Number of replicates (including controls)	1	Y	
25	Nominal concentrations reported?	1	N	
26	Measured concentrations reported?	3	Y	Continuous
27	Food type and feeding periods during the long-term tests	1	Y	
28	Were concentrations measured periodically (especially in the chronic test)?	1	Y	Continuous
29	Were the exposure media conditions relevant to the particular chemical reported? (e.g. for the metal toxicity – pH, DOC/TOC, water hardness, temperature)	3	Y	
30	Photoperiod and light intensity	1		n/a to study
31	Stock and test solution preparation	1	Y	
32	Was solubilizer/emulsifier used if the chemical was poorly soluble or unstable?	1		n/a

33	If solubilizer/emulsifier was used, was its concentration reported?	1		n/a
34	If solubilizer/emulsifier was used, was its ecotoxicity reported?	1		n/a
35	Monitoring intervals (including observations and water quality parameters) reported?	1	Y	
36	Statistical methods used	1	Y	
Information relevant to the data quality				
37	Was the endpoint directly caused by the chemical's toxicity, not by the organism's health (e.g. when mortality in the control > 10%) or physical effects (e.g. "shading effect")?	n/a	Y	
38	Was the test organism relevant to the Canadian environment?	3	Y	
39	Were the test conditions (pH, temperature, DO, etc.) typical for the test organism?	1	Y	
40	Do system type and design (static, semi-static, flow-through; sealed or open; etc.) correspond to the substance's properties and organism's nature/habits?	2	Y	
41	Was pH of the test water within the range typical for the Canadian environment (6 to 9)?	1		n/a in air
42	Was temperature of the test water within the range typical for the Canadian environment (5 to 27°C)?	1		n/a in air
43	Was toxicity value below the chemical's water solubility?	3		n/a in air
Results				
44	Toxicity values (specify endpoint and value)	n/a	Y	90-day LC_{50}
45	Other endpoints reported - e.g. BCF/BAF, LOEC/NOEC (specify)?	n/a	Y	NOEL = 101 mg/m^3; LOEL = 189 mg/m^3
46	Other adverse effects (e.g. carcinogenicity, mutagenicity) reported?	n/a	N	
47	***Score*: ... %**	***30/36 = 83.3***		
48	***EC reliability code*:**	***1***		
49	***Reliability category (high, satisfactory, low)*:**	***High Confidence***		
50	***Comments***			

[1] n/a – not applicable.
[2] NA – not available.