Government of Canada Gouvernement du Canada

Screening Assessment

Ethene (Ethylene)

Chemical Abstracts Service Registry Number (CAS RN)

74-85-1

Environment and Climate Change Canada

Health Canada

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Canada

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Synopsis

Pursuant to sections 68 of the *Canadian Environmental Protection Act, 1999* (CEPA1999), the Ministers of the Environment and Climate Change and of Health have conducted a screening assessment of ethene (commonly referred to as “ethylene”), Chemical Abstracts Service Registry Number (CAS RN) 74-85-1.

Ethene is a simple double-bonded hydrocarbon, and is a ubiquitous gas in the environment. It is introduced to the environment from both natural and anthropogenic sources, including emissions from vegetation of all types and microorganisms, as a product of incomplete combustion of organic material (such as wood and agricultural wastes) and of fossil fuels, and during its industrial production and use. Ethene is also produced endogenously by humans and mammals.

Ethene has been internationally identified as a high production volume (HPV) chemical. Worldwide, ethene is the petrochemical product manufactured in the largest quantity, with global production capacity in 2011 estimated to be 138 million tonnes per year. In 2011, Canada ranked sixth in worldwide ethene production capacity at nearly 5.5 million tonnes per year, representing 4.0 % of worldwide capacity. In 2000, ethene production in Canada was slightly lower, with a production of 4.3 million tonnes per year, based on results from a survey conducted under section 71 of CEPA. In this same survey, imported quantities of ethene were comparatively negligible.

In Canada and worldwide, ethene is predominantly used as a monomer for the manufacture of polyethylene plastics, as an intermediate for other organic chemicals, and as fuel gas in industrial facilities. Relatively small amounts are also used in commercial settings worldwide for the controlled growth or ripening of vegetation such as fruits, vegetables and flowers; in Canada, ethene is used for the postharvest ripening of bananas and other tropical fruits, and degreening of citrus.

In Canada, anthropogenic ethene concentrations in air are mostly attributed to the combustion of fossil fuels and to the use of ethene in various industrial processes. Canadian automotive releases to air were estimated at 3449 tonnes in 2005. The majority of this was estimated to be released from cars older than 1992. Vehicles newer than 1992 emit considerably less ethene due to advances in automotive engine technology and emissions controls and requirements for cleaner burning fuels in the United States (US) and Canada.

Ethene is included in the National Pollutant Release Inventory (NPRI), to which facilities manufacturing, processing, or otherwise using more than 10 tonnes per year of the substance must report their releases. In 2009, facilities across Canada reported to the NPRI on-site environmental releases totaling approximately 1320 tonnes. Industrial releases have dropped by over 50 % since 2000, due largely to the amount of ethene being recycled. The majority of reported ethene releases are to air.

Ethene has been measured in outdoor, indoor and personal air (i.e., air near a person’s breathing zone collected using mobile air sampling equipment carried by participants) in

Canada, as well as in vegetation, soil and surface seawater. As a combustion by-product, ethene has been measured in vehicular exhaust and in cigarette smoke. Ethene has not been reported in drinking water or consumer products in Canada.

Based on experimental and modelled data, ethene is neither persistent nor bioaccumulative in the environment.

Terrestrial plants are highly sensitive to ethene in air; critical toxicity values in air were determined for long-term and short-term exposures.

Air monitoring data were used to determine if ambient concentrations of ethene in urban and rural air or near industrial sites could be harmful to terrestrial plants. Based on comparison of levels expected to cause harm to organisms with estimated exposure levels and other information, ethene has a low risk of harm to terrestrial plants due to industrial emissions or ambient concentrations. The estimated frequency of occurrences of sufficient concentration from industrial emissions to be of concern is one occurrence per year.

Based on the information presented in this screening assessment, there is low risk of harm to organisms or the broader integrity of the environment from this substance. It is therefore concluded that ethene does not meet criteria under paragraphs 64(*a*) or (*b*) of CEPA as it is not entering the environment in a quantity or concentration or under conditions that have or may have immediate or long-term harmful effects on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

Based principally on weight of evidence-based assessments of international agencies, ethene was “not classifiable as to its carcinogenicity to humans (Group 3)” and the Organization for Economic Co-operation and Development (OECD) has concluded that relevant studies on ethene have indicated low toxicity.

The animal database for ethene, as well as the available epidemiology studies, did not demonstrate a cancer concern, and the overall genotoxicity test results were negative. The critical human health effect associated with exposure to ethene is nasal effects based on observations in experimental animals. This critical effect level was compared to the highest concentration of ethene measured in air in Canada and resulted in wide margins of exposure which were considered adequate to address uncertainties in the health effects and exposure databases. On the basis of the adequacy of the margins between upper-bounding estimates of exposure to ethene and critical effect levels, it is concluded that ethene does not meet the criteria under paragraph 64 (*c*) of CEPA as it 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.

Conclusion

We conclude that ethylene (CAS RN 74-85-1) does not meet any of the criteria set out in section 64 of CEPA.

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Introduction

Pursuant to sections 68 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999), the Minister of the Environment and Climate Change and the Minister of Health conduct screening assessments of substances to determine whether these substances present or may present a risk to the environment or to human health.

A screening assessment was undertaken on ethene (more commonly referred to as ethylene; Chemical Abstracts Service Registry Number 74-85-1), as it was identified as a priority for assessment on the basis of its greatest potential for human exposure prior to the completion of the Domestic Substances List (DSL) categorization. However, during the categorization of the DSL, ethene was not found to meet any of the categorization criteria. This screening assessment was therefore conducted pursuant to paragraphs 68(*b*) and (*c*) of CEPA.

Screening assessments focus on information critical to determining whether a substance meets the criteria as set out in section 64 of CEPA. Screening assessments examine scientific information and develop conclusions by incorporating a weight-of-evidence approach and precaution.[1]

This screening assessment includes consideration of information on chemical properties, hazards, uses, and exposure to ethene. Data relevant to the screening assessment of this substance were identified in original literature, review and assessment documents, and stakeholder research reports and from recent literature searches, up to May 2015 for ecological sections of the document and December 2011 for human health sections of the document. In addition, an industry survey was conducted in 2000 through a *Canada Gazette Notice* issued under the authority of section 71 of CEPA (Canada 2001). Key studies considered in this assessment were critically evaluated; modelling results may have been used to reach conclusions. The screening assessment does not present an exhaustive review of all available data. Instead, it presents the critical studies and lines of evidence supporting the conclusions.

Evaluation of risk to human health involves consideration of data relevant to estimation of exposure 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

[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 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 does not preclude actions being taken under other sections of CEPA or other Acts.

available data. Rather, it presents a summary of the critical information upon which the conclusion is based.

The approach taken in the ecological screening assessment is to examine various supporting information and develop conclusions based on a weight-of-evidence approach as required under section 76.1 of CEPA. The screening assessment does not present an exhaustive review of all available data. Instead, it presents the critical studies and lines of evidence supporting the conclusions.

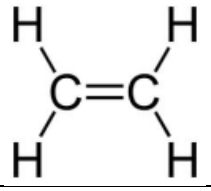
This screening assessment was prepared by staff in the Existing Substances programs at Health Canada and Environment and Climate Change Canada. This assessment has undergone external written peer review/consultation. Comments on the technical portions relevant to ecological exposure were received from scientific and industry experts, including Kent Woodburn and Gary Klecka (DOW Chemicals Inc.), Laura Blair (Government of Alberta), Tom Parkerton (ExxonMobile), and Grazyna Kalabis (Ontario Ministry of the Environment). Comments on the technical portions relevant to human health were received from scientific experts selected and directed by Gradient (an environmental and risk science consulting firm), including Cathy Petito Boyce, Leslie Beyer and Chris Long. Additionally, the draft of this screening assessment was subject to a 60-day public comment period. While external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment and Climate Change Canada.

The critical information and considerations upon which the assessment is based are summarized in the following sections.

Substance Identity

For the purposes of this document, the substance will be referred to as ethene —although commonly known as ethylene—as it is in accordance with the most recent IUPAC (International Union of Pure and Applied Chemistry) recommendations for this substance (IUPAC 1993). Information relevant to the identity of ethene is presented in Table 1.

Table 1. Substance identity for ethene

Chemical Abstracts Service Registry Number (CAS RN)	**74-85-1**
DSL name	**Ethene**
National Chemical Inventories (NCI) names[a]	Ethene (TSCA, AICS, ECL, SWISS, PICCS, ASIA-PAC, NZIoC) Ethylene (EINECS, ENCS, PICCS)
Other names	Ethylene, acetene, bicarburetted hydrogen, olefiant gas, and elayl
Chemical group (DSL Stream)	Discrete organics
Major chemical class or use	Organic
Chemical formula	C_2H_4
Chemical structure	
SMILES[b]	C=C
Molecular mass	28.05 g/mol

[a] National Chemical Inventories (NCI). 2007: AICS (Australian Inventory of Chemical Substances); ASIA-PAC (Asia–Pacific Substances Lists); 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 (Giftliste 1 and Inventory of Notified New Substances); and TSCA (Toxic Substances Control Act Chemical Substance Inventory).

[b] Simplified Molecular Input Line Entry System.

Physical and Chemical Properties

Ethene generally is present as a gas under normal environmental conditions as indicated by its vapour pressure, and partitions preferentially to the atmosphere from water bodies and soil surfaces (Mackay et al. 2003). Table 2 presents a range of physical-chemical properties identified for ethene that are relevant to its environmental fate.

Table 2. Physical and chemical properties of ethene

Property	Type	Value	Temperature (°C)	Reference
Melting Point (°C)	Experimental	-169.4	–	O'Neil et al. 2001
Boiling Point (°C)	Experimental	-102.4 to -103.7	–	O'Neil et al. 2001
Conversion Factor[a]	Calculated	1 mg/m^3 = 0.87 ppm 1 ppm = 1.15 mg/m^3	25	IARC 1994
Vapour Pressure (MPa)	Experimental	4.27	0	OECD 1998
Vapour Pressure (MPa)	Extrapolated	6.95	25	Daubert and Danner 1985
Relative Vapour Density (air =1)	-	0.9686	–	IARC 1994
Vapour Density (kg/m^3)	-	1.261	0	CGAI 1999
Specific Gravity	-	0.978	0	CGAI 1999
Solubility in Water (mg/l)	Experimental	280	0	IUCLID 1995
Solubility in Water (mg/l)	Experimental	131	25	McAuliffe 1966
Henry's Law Constant (Pa·m^3/mol)	Calculated from vapour pressure	2.17x10^4	25	SRC 2005
Octanol–water partition coefficient log K_{ow} (dimensionless; 3-solubility harmonized log K_{ow})	Extrapolated	1.85	–	Schenker et al. 2005
Reaction rate constants in air (cm^3/molecule/s)	Calculated — OH•	7.9 x 10^{-12}	25 (at 100 kPa)	Atkinson et al. 2006
Reaction rate constants in air (cm^3/molecule/s)	Calculated — NO_3	2.1 x 10^{-16}	25 (at 100 kPa)	Atkinson et al. 2006
Reaction rate constants in air (cm^3/molecule/s)	Calculated — O_3	1.6 x 10^{-18}	25 (at 100 kPa)	Atkinson et al. 2006

[a] Conversion factor represents the value in parts per million (ppm) converted to mg/m^3 and vice versa

Sources

Ethene is a ubiquitous gas in the environment. Approximately 74 % of global emissions are from natural sources and 26 % from anthropogenic sources. Most of the ethene produced naturally is released by plants and soil microorganisms in terrestrial ecosystems (Sawada and Totsuka 1986). Ethene is produced endogenously by fruits, flowers, leaves, roots and tubers as an important plant hormone which regulates various growing processes (Altshuller 1983), and is released during decomposition of litter in forest topsoil (Sawada and Totsuka 1986; Derendorp et al. 2011). Endogenous production of ethene also occurs in humans and other mammals (IARC 1994); ethene has been measured in exhaled breath (Filser et al. 1992) as well as in gas emanating from human skin (Nose et al. 2005). The burning of wood (Barrefors and Petersson 1995) during forest fires also constitutes a natural source of ethene emissions to the atmosphere. Other natural sources include volcanic emissions and natural gas leakage, but these sources are considered negligible (Sawada and Totsuka 1986).

It is estimated that 77 % of anthropogenic releases on a global scale are derived from biomass burning in terrestrial ecosystems to clear land for agriculture, focused primarily in tropical forests (Sawada and Totsuka 1986). The incomplete combustion of various fossil fuels accounts for 21 % of global anthropogenic releases (Sawada and Totsuka, 1986). Vehicle exhaust emissions were known to make an important contribution to urban air concentration of ethene (IARC 1994) although advances in automotive engine technology and fuels have greatly reduced these emissions. Other anthropogenic sources include incineration of refuse, burning of agricultural wastes (Sawada and Totsuka 1986) and industrial processes such as flaring at refineries and leakage from piping (IARC 1994).

Ethene has been internationally identified as a high production volume (HPV) chemical (US EPA 2009c; OECD 1998). According to IARC (1991), ethene is the petrochemical chemical manufactured in the largest volume worldwide (IARC 1994), although it is unclear whether this reflects more recent trends. As of January 1, 2011, the worldwide production capacity of ethene reached more than 138 million tonnes per year (tpy), which is up from roughly 119 million tpy in 2007 (True 2011). Canada possesses the 6th largest ethene production capacity at nearly 5.5 million tpy, which represents 4.0 % of worldwide capacity as of January 1, 2011 (True 2011). This reflects slight growth in Canada relative to the year 2000 when production was 4.3 million tpy as reported by Canadian manufacturers (Environment Canada 2003a) in a survey conducted under Section 71 of the *Canadian Environmental Protection Act, 1999* (Canada 2001). In this same survey, imported quantities of ethene were comparatively negligible; only 15 tonnes were imported by one company. Several changes in the Canadian manufacturing landscape for ethene have been reported in recent years. In 2008, a major Canadian petrochemicals producer closed its plants in Québec (Baumgarten 2008), while in March 2011, plans for the expansion of ethene production in the province of Alberta from the off-gas of the oil sands was announced (True 2011).

More than 95% of the annual commercial production of ethene is currently based on steam cracking of hydrocarbons, and by recovery from refinery cracked gas (Zimmerman and Walzl 2009). Ethene production and transfer to downstream uses occur in closed systems. Fugitive emissions of ethene from process equipment and piping in manufacturing plants do occur

although it is estimated that these losses account for a small percentage of production (ca. 0.03 %) (IARC 1994).

Ethene has been detected as a thermal degradation product of polyethylene and polypropylene in two laboratory studies (Hoff et al. 1982; Frostling et al. 1984), although this source has been considered to be negligible.

Uses

The worldwide increase in ethene production is largely a reflection of increased demand of downstream uses. Ethene is a major raw material in the synthetic organic chemicals industry (CGAI 1999). Ethene's largest use is in plastics, mostly for the production of high, low and linear low density polyethylene, a polymer consisting of ethene monomers (Zimmermann and Walzl 2009). The estimated growth in global demand for ethene accounts for the expected increase in plastic consumption per capita in growth markets such as India and China (ACML 2010). Ethene is also used for the manufacture of ethylene oxide, ethylene dichloride, ethylbenzene, alcohols, olefins, acetaldehyde and vinylacetate (Zimmermann and Walzl 2009), and for industrial fuel gas (Environment Canada 2003a). Small quantities of ethene are used as a refrigerant and as fuel for welding and cutting metals (CGAI 1999), and was used as an anaesthetic gas (Sneader 2005). Based on available information, uses in Canada are consistent with international use patterns.

Synthetic ethene is also used commercially to ripen fruit (CGAI 1999), although this use accounts for a small proportion of ethene production volumes (IARC 1994). As a plant growth regulator, ethene acts to stimulate or regulate the ripening of fruit, the inhibition of vegetative tissues, the opening of flowers, and the abscission (or shedding) of leaves. In Canada, ethene is used for postharvest ripening of bananas and other tropical fruits, and degreening of citrus (Lunau 2010; CGSB 2011). In the US, synthetic ethene can be applied in the field or after harvest for different crops, as a curing agent in tobacco, as a flower-producing agent in pineapples, and as a witchweed herbicide for corn, cotton, peanut and soybean crops (US EPA 1992a).

Ethene is not listed as an approved food additive in Canada under The Lists of Permitted Food Additives as incorporated by reference in Marketing Authorizations for Food Additives, issued under the authority of the *Food and Drugs Act* (email from Health Canada Food Directorate to Existing Substances Risk Assessment Bureau (ESRAB), unreferenced). Ethene is also not listed in the Drug Product Database (DPD 2012), the Therapeutic Products Directorate's internal Non-Medicinal Ingredient Database, the Natural Health Products Ingredients Database (NHPID 2012) or the Licensed Natural Health Products Database (LNHPD 2012) as a medicinal or a non-medicinal ingredient present in final pharmaceutical products, natural health products or veterinary drugs (October 2011 email from Therapeutic Products Directorate, Natural Health Products Directorate and Veterinary Drugs Directorate, Health Canada, to Risk Management Bureau, Health Canada; unreferenced).

Releases to the Environment

As has been stated previously, ethene is ubiquitous in the environment as it is naturally produced by plants, microbes and marine algae. In the soil, it is a by-product of microbial decomposition of organic material. Thus ethene is released to the environment through natural processes.

Anthropogenic ethene concentrations in Canadian air are largely attributed to vehicular emissions (Alberta Environment 2003); however, this source has decreased over the last 20 years due to advances in automotive engine technology. Ethene was one of the chemicals measured in vehicle emissions in 2003 by the AirCare program, a vehicle emissions testing and reduction program in the Lower Fraser Valley of British Columbia (Environment Canada 2003b). The vehicles tested ranged in model years from 1978 to 1998. Of these, 50 were light-duty passenger cars and 20 were light-duty trucks. The vehicles selected for testing were chosen to represent the top 70 % of the on-road vehicle fleet in British Columbia. The quantity of ethene emitted per km driven varied greatly. Vehicles older than 1992 often emitted considerably more ethene (96 mg/km driven) than did vehicles newer than 1992 (4.6 mg/km driven). This 20-fold reduction was largely the result of emissions controls and requirements for cleaner burning fuels in the United States and Canada (Environment Canada 2003b).

These data can be used to estimate tonnes of ethene potentially released in Canada, assuming that the vehicle distribution for Canada is similar to that of the Lower Fraser Valley. This estimate will likely be high, as the older vehicles (from 1991-1996) had ethene emissions that were high compared to more modern vehicles. Canadians in light-duty vehicles (under 4.5 tonnes) in 2005 drove an estimated 287.7 billion km in 17.9–18.2 million vehicles (Statistics Canada 2005; NRCan 2008). This calculation resulted in a total estimated release of ethene for Canada from light-duty vehicles at 3449 tonnes in 2005. The majority of this, 2396 tonnes (69% of the total), was from cars older than 1992 although they represented only 14% of the fleet (NRCan 2008). As the Canadian vehicle fleet ages the proportion of vehicles older than 1992 will drop resulting in substantial decreases in the amount of ethene released by Canadian vehicles.

Ethene is included in the National Pollutant Release Inventory (NPRI), to which facilities manufacturing, processing, or otherwise using more than 10 tonnes per year of the substance must report their releases. For the purpose of this assessment, the original site types were recategorized for clarity. Based on this information, the NPRI lists 1 facilities that reported releasing 1322 tonnes and recycling 723 tonnes (in Alberta) of ethene in 2009 (Environment Canada 2015; Table 3). Alberta had the highest industrial releases in Canada with 900 tonnes released (represents 68% of the total amount reported). Releases in Alberta were followed by Ontario with 357 tonnes (27% of total amount), while the remaining 65 tonnes (5% of total amount) were released in Quebec, Saskatchewan, Nova Scotia, British Columbia and Nunavut (Environment Canada 2015). Ethene is also a component of some Petroleum and Refinery Gases (PRGs) (Canada 2013a; Canada 2013b) and may be emitted to the environment along with the release of PRGs and is expected to be included in the Oil Production sector (5%) (Table 3; Environment Canada 2015). Releases of ethene in this report are considered from industrial operations generally, rather than from specific industries, unless otherwise noted.

The trend in actual releases (not counting tonnes sent for recycling or spillage) of ethene reported to the NPRI has been decreasing significantly so that releases to the environment in 2009 are less than half of that reported for 2000 (see Table 4) (Environment Canada 2015).

All releases in this report are expected to be to air since none have been identified to soil, water or sediment.

Table 3. NPRI reported releases of ethene based on industrial sector in 2009 (Environment Canada 2015)

Industrial Sector	Number of Facilities Reporting Releases	Release Mass (tonnes)	Percentage of Total (based on release mass)
Chemicals	20	1084	49
Oil Production (all types)	16	163	39
Power Production	2	46	5
Metals	3	29	7
TOTAL	**41**	**1322**	100%

Table 4. Ethene releases and recycled quantities (in tonnes) reported to NPRI between 2000 and 2009 (Environment Canada 2015).

Year	Releases	Recycled	Total
2000	2710.56	0.44	2711
2001	2472	0	2472
2002	2057	781	2838
2003	1921	422	2343
2004	1881	676	2557
2005	1840	1287	3127
2006	1463	1068	2531
2007	1427	894	2321
2008	1250	701	1951
2009	1320	723	2043

Environmental Fate

Ethene from anthropogenic sources is almost always released to air as it is a gas at environmental temperatures. Level III fugacity modelling (EQC Model; Mackay et al. 2003) estimates that almost all (>99.9%) ethene released to air will remain in air and that only negligible amounts will partition to soil, water and sediment. Ethene is not expected to be released to soil, sediment or water thus no exposure is expected and these routes were not

investigated. Thus, the most probable route of ethene exposure is through contact with air (e.g., by inhalation or respiration) and the organisms most likely to be exposed are terrestrial organisms such as plants, invertebrates, birds and mammals.

Ethene is considered to have a Photochemical Ozone Creation Potential (POCP) of 100 and is the standard chemical for comparison with other chemicals (Environment Canada 1996).

A common degradation product of ethene in air is formaldehyde, a substance already listed on Schedule 1 of CEPA (the *List of Toxic Substances*).

Physical removal of ethene from the atmosphere can occur through wet deposition; however, this process is negligible due to ethene's short half-life in the atmosphere and moderate water solubility. Certain evidence suggests that some ethene removal is facilitated by soil bacteria and fungi; however, as with wet deposition this process is not as efficient as the atmospheric chemical reactions which are 30 to 60 times more effective (Alberta Environment 2003).

Persistence and Bioaccumulation Potential

Experimental and modelled data concerning the biodegradation and persistence of ethene in different environmental media are presented in Table 6. Modelled biodegradation values for ethene indicate that the half-life is < 182 days in water and soil. Howard et al. (1991) provide an estimation of the aerobic half-life of 1 – 28 days, and an anaerobic half-life of 4 – 112 days. Ethene is readily oxidized in the atmosphere with a theoretical global residence time in the troposphere ranging from 2 to 4 days. There are, however, numerous chemical reactions associated with the breakdown of ethene which may decrease its half-life to just a few hours (Sawada and Totsuka 1986).

Ethene reacts in air primarily with hydroxyl radicals (OH•) but it can also react with nitrate ions (NO_3) and ozone (O_3). The oxidation of ethene can generate nitrogen dioxide (NO_2) which can later form ozone. In a report for the California Air Resources Board, Carter (2010) developed a complex model based on years of air chamber tests to determine the impacts of chemicals on O_3 and NO_3 formation. In their report are relative reactivities based on effects of chemicals on the maximum 8-hour average ozone concentration in 39 cities across the continental USA. Under these test conditions Carter calculated that ethene generated an average of 9 grams O_3 for each gram added to the atmosphere (Carter 2010). With an estimated release of 3449 tonnes of ethene from automobiles, this source would generate approximately 31 041 tonnes of O_3 across Canada (at 25 °C and 101.3 kPa).

Ethene is used as the criterion against which other chemicals are measured for tropospheric ozone formation. The rate constant for ethene reacting with OH• is 7.9×10^{-12} cm^3 $molecule^{-1}$ s^{-1} (at 298 K and 101 kPa); with NO_3 and O_3 the rate for ethene reacting are much slower (Atkinson et al. 2006). Calculations of a half-life in air are highly dependent on the concentration of OH• used. Atkinson (2000) found the peak day-time OH• concentration

close to ground level at two mid-latitude, northern hemisphere sites during August and September ranged from 2×10^6 to 10×10^6 molecule/cm^3. Prinn et al. (1995) calculated a diurnally, seasonally and annually-averaged 24-h OH• concentration of 1×10^6 molecule/cm^3. It appears that the value 2×10^6 molecule/cm^3 would be in the range of what should be expected in Canada.

An atmospheric half-life ($t_{½}$) was calculated for ethene based on the OH• rate constant, a 12-h day and default OH• concentration listed above and based on the equation presented in Leifer (1993). This gives an atmospheric half-life of 1.01 days.

Other estimates of the oxidative half-life in air were 1.255 days (using AOPWIN 2000) and 1.125 days (Alberta Environment 2003 using data from Prinn et al*.* 1992). Alberta Environment (2003) estimated that in more polluted urban areas where OH• concentrations can reach 10^7 molecules cm^{-3} (Eisele and Tanner 1991) the half-life is only about 2 hours.

Ethene has an expected reactive half-life in air of 1.01 days, and an estimated half-life in water of 1 – 28 days (Table 5). Using an extrapolation ratio of 1:1:4 for water:soil:sediment biodegradation half-lives (Boethling et al. 1995), the half-lives in soil and sediment can be extrapolated from the half-life estimations in water. Therefore, the half-life in soil is 1 – 28 days and in sediment is 4 – 112 days.

Table 5. Half-lives of ethene in environmental media

Medium	Half Life (days)	Reference
Water	1-28	Howard et al. 1991; OECD 2005
Air	1.01	Atkinson 2000; AOPWIN 2000
Soil	1-28	Howard et al. 1991
Sediment	4-112[a]	n/a

[a] As calculated using the 1:1:4 extrapolation ratio for degradation in water : soil : sediment from Boethling et al. 1995

The long-range transport potential of ethene in air was estimated using the TaPL3 model v3.00 (CEMC 2003). The characteristic travel distance (CTD) of the substance calculated by the model is 681 km. According to Beyer et al. (2000), ethene belongs to class 3 (short CTD < 700 km) and is considered to have a low potential for long-range transport in air.

There are no empirical data characterizing BCFs or BAFs for ethene; however, it is expected that bioaccumulation and bioconcentration of ethene in aquatic systems is limited due to its low log K_{ow} of 1.85 (Schenker et al. 2005). Since no experimental bioaccumulation or bioconcentration data for ethene were available, a predictive approach was applied using a model to calculate the BCF and BAF (BCFBAF 2008). The BAF model is usually preferred, as it represents uptake of chemicals from water and the diet however, no diet contribution is expected thus BAF was not considered. The result of the BCF model calculation was BCF = 1.774 L/kg wet-wt, which indicates a very low bioconcentration potential.

Based on the above information ethene is neither persistent nor bioaccumulative in the environment.

Potential to Cause Ecological Harm

Ecological Exposure Assessment

Experimental data on the distribution of ethene in environmental compartments is scarce so the EQC Level III fugacity model (Table 5; Mackay et al. 2003) was used. The only pathway of release known in Canada is through releases to air and in conjunction with the degradation information presented in Table 6, ethene is unlikely to be found in water, sediment or soil at concentrations greater than what is naturally present in the environment. Consequently, only air concentrations are reported in this section.

Although environmental ethene concentrations are dependent on both natural and anthropogenic sources, the consistently highest concentrations are measured in urban areas due to the combustion of fuel, coal and natural gas by vehicles and industrial facilities. Industrial facilities may also release ethene through production processes. Ambient remote and rural concentrations of ethene from across Canada were generally below 15 $\mu g/m^3$ (Alberta Environment 2003). The OECD Screening Information Data Set (OECD 1998) reported that ethene concentrations at rural sites ranged from < 1 – 5 $\mu g/m^3$.

During the period 1980-1993, ethene levels in Canadian urban environments ranged from 4 $\mu g/m^3$ to a high of 113 $\mu g/m^3$ under a winter inversion in Calgary (Alberta Environment 2003; Reid and Watson 1985). The Clean Air Strategic Alliance (CASA 2008) monitored air concentrations from 1995 to 2000 in Edmonton and Calgary. The range of concentrations in Edmonton was 0.29 to 71.5 $\mu g/m^3$ with an average of 7.3 $\mu g/m^3$ in the urban center, while Calgary had a range of 1.2 to 31.7 $\mu g/m^3$ with an average of 7.5 $\mu g/m^3$. In both cities, over 80% of the monitoring data was below 10 $\mu g/m^3$.

Air concentration data from forty-nine National Air Pollution Surveillance (NAPS) air monitoring network sites (Environment Canada 2011) for the years 2000 to 2009 were selected and analyzed. Data from some monitoring stations were not included in the ecological assessment as not all locations were considered relevant. Sampling periods were either 4 hours or 24 hours long depending on the site and other parameters (many sites were sampled for four hours). All sites were re-classified from their original designation as remote (background), rural, or urban based on their respective locations in Canada and proximity to cities and townships (urban), agricultural and semi-developed areas (rural) or areas that were considered minimally impacted (remote). Differences among these three types of sites were investigated using average values from each site in an Analysis of Variance test (ANOVA) followed by pair-wise comparisons using the Tukey test (Minitab 2005). There was a statistically significant difference among groups (P<0.001), with significant pair-wise differences between the urban and rural sites as well as the urban and background sites. Table 6 shows the air concentrations at the three different site types for two periods, 2000-2003 and 2005-2009.

The concentration of ethene in urban areas was considerably higher than that in background and rural areas. This was likely due to vehicle emissions and the presence of industrial sources of ethene. While two of the NAPS monitoring stations are near industrial sources of

pollution, the remainder are not, so a statistic analysis of the co-occurrence of ethene and industries was not possible. The few reported monitoring programs at petrochemical facilities reported highly variable concentrations of ethene. One facility in Alberta detected a range of 2.29 to 232.74 µg/m^3 from May to September of the same year, 2000, (the maximum 6-hr average for the month) with an average value of 48.6 µg/m^3 (Alberta Environment 2003). Areas close to such facilities will receive higher peak values of ethene.

The highest daily concentration (Table 6) from recent (2009) air monitoring in an urban setting was 32.9 µg/m^3 reported at a site in Sarnia, Ontario. This site has several ethene producing or using industries and has highly variable air concentrations of ethene. The average concentration of ethene in urban areas in Canada has decreased over a period of 10 years from 2000-2009. The average from 2000 to 2003 was 2.03 µg/m^3 while the average for 2005-2009 was 1.70 µg/m^3.

Between 2005 and 2009, the highest daily concentration at rural sites (Table 6) across Canada was 7.5 µg/m^3, which occurred in the town of Quesnel, British Columbia (Environment Canada 2011). The average concentration of ethene at rural monitoring locations has increased slightly from 0.34 µg/m^3 between 2000-2003 to 0.46 µg/m^3 between 2005 and 2009; it is not known if this is an actual trend or simply variation in the data.

The highest daily concentration at remote sites across Canada between 2005 and 2009 (Table 6) was 1.7 µg/m^3, nearly unchanged from the period 2000-2003. The average concentration of ethene was 0.23 µg/m^3 between 2000 and 2003 and 0.21 µg/m^3 between 2005 and 2009. It can be reasonably stated that ethene concentrations in remote regions of Canada are very low and have not changed significantly in the recent past nor is ethene being transported over long ranges from sites with higher concentrations.

Table 6. Daily air concentration data at different types of sites for the periods, 2000-2003 and 2005-2009 (Environment Canada 2011).

A) 2000-2003

Site Type	Min conc. (µg/m^3)	Max. conc. (µg/m^3)	Mean conc. (µg/m^3)	Data Points (n)
Remote Sites (n=5)	0.01	1.80	0.23	519
Rural Sites (n=9)	0.03	5.57	0.34	1653
Urban Sites (n=35)	0.05	38.43	2.03	3252

B) 2005-2009

Site Type	Min conc. (µg/m^3)	Max. conc. (µg/m^3)	Mean conc. (µg/m^3)	Data Points (n)
Remote Sites (n=5)	0.01	1.70	0.21	1841
Rural Sites (n=9)	0.05	7.5	0.46	7010
Urban Sites (n=35)	0.102	32.9	1.70	10 521

Canadian data on the release of ethene from industrial sites was provided by both industry and by regional environmental associations, representing monitoring at either only industrial fence lines (industry submissions) or both industrial fence lines and further afield (environmental association monitoring).

Considering only the industrial submissions, the range of 3-day maximum concentrations over the entire year reported by a variety of facilities at fence lines from 2007 – 2012 was 6 µg/m^3 to 78 µg/m^3, with an average of 27 µg/m^3 and a median of 22 µg/m^3. The majority of 3-day maximums were in the range of 10 – 35 µg/m^3. All measurements considered are offsite measurements located near ethene producing industries (NOVA Chemicals 2013; Dow Chemicals 2013).

The annual average concentration reported by industry ranges from 1.0 µg/m^3 to 5.6 µg/m^3 with an average at fence line of 3.5 µg/m^3 in Canada (NOVA Chemicals 2013; Dow Chemicals 2013).

Regional monitoring of ethene was conducted by an environmental association in the Sarnia-Lambton area of Ontario; this region contains several industrial sources of ethene as well as vehicle and urban emissions (including from a major provincial highway). Hourly data from January 2008 to December 2012 in this urbanized, industrial area were provided, and 3-day averages were calculated in order to compare against industry data. The maximum 3-day average ethene concentrations ranged from 4 – 140 µg/m^3 with an average maximum of 26 µg/m^3 and a median of 17 µg/m^3 (SLEA 2013). Data from five monitoring stations were provided. Three monitoring stations were at industrial fence lines while two were further afield. The two furthest stations were approximately 13 km apart.

The 2008 – 2012 environmental monitoring data in the Sarnia-Lambton area had an abnormally high 3-day average of 280 µg/m^3 in 2012. Upon further investigation, this value was caused by an event lasting only 6 hours in the early morning of August 3, 2012, when ethene concentrations spiked across all five monitoring stations in the region. The cause of this increase is not known but was atypical. Given that this event was isolated, was of very short duration and the ethene concentrations returned to their prior values, this data was removed from consideration in the risk assessment.

Ecological Effects Assessment

Terrestrial Plants

Ethene is produced and used as a hormone in higher plants, as such it has effects on many growth and developmental processes depending on concentration, growth stage during exposure, and length of exposure. Many growth effects are reversible if they do not continue for very long; developmental processes however, are often not reversible if the process has a short time-frame within which to occur, such as flower development. Ethene exposure promotes early leaf abscission (drop) and epinastic growth (leaf curling), it can stunt root growth, and it also affects developmental processes involved in reproduction such as flower bud formation and development, fruit ripening, and extent of flowering (Blankenship and

Kemble 1996; Alberta Environment 2003). The impact of effects on flowering is a reduction in the ability to reproduce; the effects on roots and leaves can lead to stunted growth. However, because ethene is a plant hormone not all effects are negative. An air concentration of approximately 12 µg/m^3 stimulates growth in many plants if exposure is at the right time, and can protect them against water loss (Reid and Watson 1985). The agriculture industry exploits these effects by using ethene to ripen green-picked fruit and to delay flower opening during transport to markets.

At air concentrations between 5.6 and 12 µg/m^3 both positive and negative effects start to appear in various plant species (see Tables 7a and 7b). Some cereals, such as barley and oats, appear to be highly sensitive to ethene at air concentrations as low as 34.4 µg/m^3, showing a 63% reduced seed production (Archambault and Li 2000). Tomatoes show slight curling of leaves at 11.45 µg/m^3, and peas show a reduction in the elongation of the epicotyl during germination at this concentration, while canola has increased seed production at 12 µg/m^3 (Blankenship and Kemble 1996; Goeschel and Kays 1975 ; Reid and Watson 1985). Conifers (spruce and pine) do not show effects at concentrations as high as 1,374 µg/m^3 over short exposure periods (1 hour) (Archambault and Li 2001). Goeschl and Kays (1975) note that the stage of development can influence the type of response to ethene, as can the length of exposure (Dueck et al. 2003) and whether plants have a recovery time (Tonneijck et al. 2000). Some authors suggest that the lack of a recovery period is why plants in experiments appear to be more sensitive to ethene than do plants exposed in the environment where exposure from industrial releases is not constant and is often of short duration (Tonneijck et al. 2003).

Potatoes (*Solanum tuberrosum*) exposed to 515 µg/m^3 of ethene for up to 12 h during daylight had increased stomatal closures, which reduced photosynthesis for up to three consecutive days. Similar exposure during dark hours did not affect photosynthesis. Potatoes that experienced reductions in photosynthesis were found to recover within 48 h (Dueck et al. 2003). When exposed to ethene at 515 µg/m^3 for longer than 12 h, irreversible damage of the photosynthetic apparatus followed (Dueck et al. 2003; Archambault and Li 2001; Table 7b).

It is difficult to predict the response of a particular species to exogenous ethene; closely related species can respond differently, and even agronomic cultivars can respond differently to ethene. In barley (*Hordeum vulgare*), the cultivar “Harrington” was very sensitive to ethene while the cultivar “AC Lacombe” was not (Archambault et al. 2006). Rajala et al. (2002) found that of the cereals, barley and oats are sensitive to ethene, but that wheat and rice are not. Fiorani et al. (2002) found that among four species of the grass genus *Poa*, two species responded positively to low concentrations of ethene and two responded negatively. This response was based more on growth habit than any other apparent attribute of the plants.

Table 7a. Effect of chronic (>14 days) ethene exposure and effects on Canadian crop species

Species	Ethene Air Conc. (µg/m^3)	Exposure Period	Effect	Reference
Barley (*Hordeum vulgare* cv. Harrington)	5.6	n/a	NOAEC/Threshold for 10% decrease in seed yield[1]	Archambault and Li 2001
Mixed species of plants	6.1	28 days	Threshold for epinastic effects	Tonneijck and van Dijk 2000
Oat (*Avena sativa* L. cv. Random)	8	100 days	Per plant floret number decreased by 22 %	Reid and Watson 1985
Tomato (*Lycopersicon esculentum*) “Red Robin”	11.45	56-77 days	No effect on fruit set	Blankenship and Kemble 1996
Tomato (*Lycopersicon esculentum*) “Red Robin”	11.45	50 days	38% incidence in mild epinastic growth of leaves	Blankenship and Kemble 1996
Canola (*Brassica campestris* L.)	12	87 days	Per plant seed yield increased by 188%	Reid and Watson 1985
Barley (*Hordeum vulgare* cv. Harrington)	34.4	14 days	63% seed yield reduction	Archambault and Li 2001
Canola (*Brassica campestris* L.)	41	87 days	No difference from control	Reid and Watson 1985
Oat (*Avena sativa* L. cv. Random)	41	100 days	Per plant floret number decreased by 42 %	Reid and Watson 1985
Canola (*Brassica campestris* L.)	57	31 days	20% seed yield reduction	Archambault and Li 2001
Wheat (*Triticum aestivum* L.)	57	20-25 days	37% seed yield reduction	Klassen and Bugbee 2002
Rice (*Oryza sativa* L.) “Super Dwarf”	57	49 days	50% seed yield reduction	Klassen and Bugbee 2002
Tomato (*Lycopersicon esculentum*) “Red Robin”	57.25	56-77 days	Fruit set reduced by up to 85%	Blankenship and Kemble 1996
Easter lily (*Lilium longiflorum*)	58	77 days	Flower stunting, bud abortion and distortion	Blankenship et al. 1993

Species	Ethene Air Conc. (µg/m^3)	Exposure Period	Effect	Reference
Barley (*Hordeum vulgare* cv. Harrington)	70.9	n/a	Threshold for 25% decrease in seed yield	Archambault and Li 2001
Tomato (*Lycopersicon esculentum*) “Red Robin”	114.5	56-77 days	Fruit number failed 100%	Blankenship and Kemble 1996
Tomato (*Lycopersicon esculentum*) “Red Robin”	114.5	50 days	No effect on height.	Blankenship and Kemble 1996
Canola (*Brassica campestris* L.)	175	87 days	Per plant seed yield reduced by 53%	Reid and Watson 1985

[1] This is based on a log-log relationship pooling all short and long-term data from Archambault and Li (2001). It represents the concentration at which a 10% decrease in seed yield may occur and is considered a NOAEC.

Table 7b. Effect of acute (<3 days) ethene exposure on Canadian crop species

Species	Ethene Air Conc. (µg/m^3)	Exposure Period	Effect	Reference
Pea (*Pisum sativum* L.)	11.5	2.5 days	8.5 ± 2.5% decrease in epicotyl elongation	Goeschl and Kays 1975
Morning glory (*Ipomoea tricolor* Cav.)	12	≥ 1 hour	Increased petal senescence	Hanson and Kende 1975
Rose (*Rosa* sp. cv. Lovely Girl)	23	2 days	Inhibition of flower opening	Reid et al. 1989
Barley (*Hordeum vulgare* cv. Harrington)	57	3 days	41% reduction in seed yield	Archambault and Li 2001
Carnation (*Dianthus caryophyllus*)	58	2 days	Increased flower senescence	Woltering and Harkema 1987
Tomato (*Lycopersicon esculentum* Mill)	58	1 day	Partial inhibition of root elongation	Konings and Jackson 1979
White mustard (*Sinapis alba*)	58	1 day	20% inhibition of root elongation	Konings and Jackson 1979
Pea (*Pisum sativum* L.)	124	2.5 days	50 % decrease in epicotyl elongation	Goeschl and Kays 1975

Species	Ethene Air Conc. (µg/m^3)	Exposure Period	Effect	Reference
Lodgepole pine (*Pinus contorta*)	1374	12 hours	No effect on germination, vigour or growth	Archambault and Li 2001
White spruce (*Picea glauca*)	1374	12 hours	No effect on germination, vigour or growth	Archambault and Li 2001

The critical toxicity value (CTV) for adverse effects was based on no observed adverse effect concentrations (NOAECs) derived from the following study. Archambault and Li (2001) developed a dose-response function to determine a threshold concentration (5.6 µg/m^3; Table 7a) that would approximate a 10% decrease in seed yield by pooling all of their short-term and long-term effects data for barley cv. Harrington, the most sensitive cultivar in the study. The 10% decrease in seed yield was determined to be the detection limit for a change in seed yield in their bioassays, so this would be a NOAEC within their study. However, the short-term effects data were likely of too short a duration to cause a permanent or non-reversible effect and thus the 10% decrease in seed yield is not considered significant. Archambault and Li (2001) further determined from the dose response curve that a concentration of 70.9 µg/m^3 would be required to cause a 25% decrease in seed yield. Thus 5.6 µg/m^3 was chosen as a conservative long-term CTV for sensitive plant species based on Archambault and Li's (2000) dose response function of barley.

The value of 57 µg/m^3 will be used as the short-term CTV as it reflects a 41% decrease in seed yield after 3 days of exposure to a sensitive cultivar of barley (Table 7b; Archambault and Li 2001). However, it should be noted that Archambault and Li (2001) tested two cultivars of barley and used the more sensitive cultivar (cv. *Harrington*) as it showed heightened sensitivity to ethene compared to the other cultivar (cv. *AC Lacombe*). Thus both CTVs reflect not only a sensitive species, but a sensitive cultivar within that species.

Hanson and Kende (1975) found that morning glory petals, when exposed to 12 µg/m^3 for less than an hour, had increased petal senescence; however, it should be noted that these were detached petals and not whole plants and therefore this study is not considered appropriate for extrapolation to whole plants. Additionally, studies that looked at the effects of epinasty were not considered relevant as epinasty is not considered a harmful effect, but merely an indicator of the presence of elevated levels of ethene.

Other Terrestrial Organisms

No effects data were found on invertebrates or birds, which are most likely to be exposed to ethene.

The concentrations of ethene tested within the following mammalian studies are considerably higher than concentrations expected in the Canadian environment. Exposure to

concentrations of ethene that are considered environmentally relevant did not lead to toxic effects after exposure to rats (see Appendix 2 for further details). Short term exposure of male Fischer 344 rats resulted in no toxic effects after 5 h at 11 500 mg/m^3 ethene (Guest et al., 1981). Subchronic toxicity (development of nasal legions) was observed following a 65-day exposure of male Wistar and male Fisher 344 rats to 11 472 mg/m^3 ethene (6 h/day, 5 days/week) (US EPA 2009a). One long term study found no significant carcinogenic effects after two-year exposures of male and female Fischer 344 rats to 3450 mg/m^3 ethene (6 h/day, 5 days/week) (CIIT 1979; Hamm et al., 1984).

The short-term LOAEC for subchronic effects on mammals is 11 500 mg/m^3, and the long-term NOAEC for reproduction and developmental toxicity is 5750 mg/m^3 with no adverse effects observed on reproductive performance, fertility or pregnancy (Aveyard et al. 1996 cited in OECD 1998).

Aquatic Organisms

Ethene is not expected to be released to water and thus no water exposure is expected. No adequate empirical toxicity studies on aquatic species were found.

Estimated aquatic 96-hour LC_{50} values derived from quantitative structure-activity relationship (QSAR) models range from 50 to 116 mg/L for different fish species and 48-hr LC_{50} values for *Daphnia spp.* from 53-153 mg/L (OECD 2005). A 16 day NOAEC for *Daphnia* was estimated to be 37 mg/L. A 28 day NOAEC for the fathead minnow was estimated to be 13 mg/L after a 28 day exposure (OECD 2005). Considering that ethene is not expected to be released to water these concentrations are highly unlikely over such periods of time such that the model results are not considered relevant to Canadian release scenarios of ethene.

Ecological Risk Characterization

The approach taken for this assessment was to examine the available scientific information and develop conclusions based on a weight-of-evidence approach and using precaution as required under CEPA. Lines of evidence considered include information on the environmental sources, fate, persistence, bioaccumulation potential, and ecotoxicity of the substance.

Risk quotient (RQ) analyses, which integrate known or potential exposures with known or potential adverse ecological effects, were also performed for ethene. Only air exposure scenarios were considered in this assessment due to the low exposure potential to ethene in water, soil and sediment. Terrestrial plants were selected as the most sensitive ecological receptors to ethene in this media.

Selection of exposure scenarios

To estimate the risk to Canadian ecosystems, four exposure scenarios were developed for short-term and long-term exposure regimes with their respective predicted environmental concentrations (PEC). Based on an initial analysis of air monitoring data from across

Canada, rural and urban locations were identified as being potentially at risk from ethene emissions, likely from automotive engine emissions. Air monitoring data were used to develop averaged short-term and long-term concentrations for rural and urban areas. Industrial emissions were also assessed using two scenarios under short-term and long-term exposure regimes. Industrial and regional monitoring data were used to develop scenarios for a realistic worst-case using the Sarnia-Lambton monitoring data which combines both industrial and ambient concentrations of ethene, and an average case using the annual average of the release data for all industrial facilities in Canada.

Urban/Rural Ambient Exposure Scenarios

Two exposure scenarios were developed to determine if the ambient ethene concentrations in rural and urban Canada pose a hazard to vegetation over short- and long-terms. Each scenario reflects ambient monitoring data from either rural or urban sites across Canada compared against short- and long-term thresholds for potential negative impacts to vegetation. These values reflect average annual ethene releases from 2005-2009 across Canada.

Based on air concentrations measured at sites across Canada from 2005 to 2010, PECs for long-term ambient concentration exposure scenarios were based on the mean daily concentrations from April – September of 0.3 µg/m^3 and 1.4 µg/m^3 for rural and urban areas, respectively (Table 8). For short-term ambient exposure scenarios, the maximum concentrations of 3.2 and 32.9 µg/m^3 were used as the PECs for rural and urban areas, respectively.

Industrial Site Exposure Scenarios

Two industrial exposure scenarios were developed based on industrial and/or regional air monitoring data for both an average and realistic worst-case scenario. Both the average and the worst-case scenarios were compared against short and long-term exposure thresholds to plants.

A short-term exposure PEC for the industrial average case scenario of 26 µg/m^3 was determined using the average 3-day maximum concentration reported for the Sarnia-Lambton area of Ontario between 2008-2012 (Table 8; SLEA 2013). A long-term exposure PEC of 3.5 µg/m^3 was chosen based on the annual average concentration reported by industry (Dow Chemicals 2013; NOVA Chemicals 2013) from sites near the fenceline of ethene-releasing industries from 2007-2012 (Table 8).

PECs for the industrial realistic worst case scenario were also determined based on regional monitoring data the Sarnia-Lambton area of Ontario. A short-term exposure PEC of 140 µg/m^3 was determined based on the highest measured 3-day maximum concentration reported by industry and the environmental association (Table 8; Dow Chemicals 2013; NOVA Chemicals 2013; SLEA 2013). A long-term exposure PEC of 5.6 µg/m^3 was determined based on the highest reported industry annual average concentration (Table 8; Dow Chemicals 2013; NOVA Chemicals 2013).

Table 8: Summary of predicted exposure concentrations (µg/m^3) for urban, rural and industrial exposure scenarios

Scenario	Short term	Long term
Urban ambient	32.9[a]	1.4[a]
Rural ambient	3.2[a]	0.3[a]
Industrial average	26[c]	3.5[b]
Industrial worst-case	140[c]	5.6[b]

[a] Environment Canada 2011: Short-term concentrations reflect daily maximums while long-term concentrations reflect the average of daily concentrations
[b] NOVA Chemicals 2013; Dow Chemicals 2013
[c] SLEA 2013

Selection of ecological receptors

Predicted no-effect concentrations (PNECs) are usually determined by dividing a critical toxicity value (CTV) by an assessment factor. An assessment factor of one was applied for terrestrial plants as the available data spanned a range of species, including sensitive species. Toxicity data included both laboratory and field studies; as laboratory studies are more sensitive to ethene than field exposures due to their continuous exposure, no assessment factor was considered necessary to take into account laboratory to field variability. CTVs typically represented the lowest ecotoxicity value from an available and acceptable data set. For this assessment, two CTVs were chosen to represent terrestrial plants in a short-term and a long-term exposure scenario.

A CTV of 5.6 µg/m^3 was selected to represent a concentration where no effects are seen over the long-term to a broad selection of plants (Archambault and Li 2001). For short-term exposures a CTV of 57 µg/m^3 was chosen to reflect a 41% decrease in the seed yield of a sensitive cultivar of barley (Archambault and Li 2001).

Discussion of results of risk quotient analysis

A summary of PECs, PNECs and RQ for the ambient urban and rural concentration scenarios is presented in Table 9. The RQs indicate that ambient concentrations of ethene do not pose short- or long-term risks to plants in urban areas or rural areas.

Table 9: Summary of risk quotient (RQ) analyses for ambient rural and urban concentrations of ethene, 2005 – 2009

Exposure Location	Exposure Duration	PEC µg/m^3	CTV µg/m^3	Assessment Factor	PNEC µg/m^3	RQ
Rural areas	Short term	1.4	57	1	57	**0.02**
Rural areas	Long term	0.3	5.6	1	5.6	**0.05**
Urban areas	Short term	32.9	57	1	57	**0.6**

Exposure Location	Exposure Duration	PEC µg/m^3	CTV µg/m^3	Assessment Factor	PNEC µg/m^3	RQ
Urban areas	Long term	1.4	5.6	1	5.6	**0.3**

Table 10 presents the summary of the risk quotient calculations for industrial releases. The realistic worst case for industrial releases generates an RQ of 2.4 for short term exposures and an RQ of 1 for long term exposures. The average case for industrial releases generates an RQ of 0.5 for short term exposures and an RQ of 0.6 for long term exposures. These risk quotient analyses indicate that ethene could pose short-term risks to local terrestrial vegetation from worst case industrial facility releases as indicated by a risk quotient of 1 or greater.

Table 10. Summary of risk quotient (RQ) analyses for industrial releases of ethene from 2007-2012

Exposure Scenario	Exposure Duration	PEC µg/m^3	CTV µg/m^3	Assessment Factor	PNEC µg/m^3	RQ
Realistic Worst Case	Short term	140	57	1	57	**2.4**
Realistic Worst Case	Long term[a]	5.6	5.6	1	5.6	**1**[b]
Average Case	Short term	26	57	1	57	**0.5**
Average Case	Long term[a]	3.5	5.6	1	5.6	**0.6**

[a] These reflect annual concentrations and as such are not restricted to April – September
[b] This reflects a NOAEC value.

Based on the PNEC of 57 µg/m^3 for an effect on seed yield of a sensitive crop plant (Archambault and Li, 2001), the number of days in 2008-2012 where the 3-day average exceeded this threshold were calculated. Only the data submitted by SLEA (2013) were suitable for this calculation. A total of 14 incidents in the entire dataset had 3-day averages above this threshold and of these days, only 7 exceedances occurred between April and September representing 0.002% of available days (SLEA, 2013). Exceedances occurred only at sites within close proximity to the source (i.e., at the fence line); none occurred further afield. This represents an approximate average of one short-term occurrence per year near ethene-releasing industrial sites of a sufficient concentration that may cause harm to plants.

For the realistic worst case industrial scenario, a risk quotient of 1 was obtained using a PNEC of 5.6 µg/m^3 for a predicted 10% decrease in seed yield in barley cv. Harrington, the most sensitive cultivar of a sensitive species tested (Archambault and Li, 2001). This value is considered to be a conservative NOAEC value and is, in fact, below the threshold concentration (12 µg/m^3) where no effects were expected by Archambault and Li (2001). At

this concentration, no effects on plants were found, regardless of exposure time, as Archambault and Li (2001) considered this value equivalent to the background, or control, concentration. Given this, the concentration of 5.6 µg/m^3 was considered a conservative value that would be protective of all impacts on plants.

In this scenario, it is assumed that plants will be exposed to a continuous concentration of ethene, which, based on monitoring data, is not the case. Ethene concentrations vary considerably both with time of day as well as with time of year. It is likely that concentrations of ethene would not be maintained at the highest levels for long periods of time, thus allowing most plants to recover. Because of these factors, the industrial worst-case scenario is not expected to cause long-term impacts on plants.

Atmospheric exposure of plants to ethene is heavily dependent on a number of external factors. Laboratory studies frequently expose plants to continuous sources of ethene while environmental exposure is far more variable, affected by such things as wind, weather and variability in stack releases over the course of a year, and often not sustained for long periods of time. Given that exposure will be discontinuous, plants may be able to recover from ethene exposure prior to being re-exposed. The propensity for recovery or reversibility of effects, however, is dependent upon concentration and duration of exposure, as well as the nature and the extent of the effects and on the species of plants, thus making the impact of environmental ethene difficult to predict.

Tonneijck et al. (2000) studied the effect of ethene exposure to potato, which is a species known to be sensitive to ethene, located near five ethene-emitting industrial plants over 10 years. Emission levels in this study were significantly higher than what would be expected from areas adjacent to ethene producing companies in Canada, with maximum hourly concentrations ranging from 307 to 7276 µg/m^3 at 1000m downwind from the emission source. Despite these elevated levels, plant yield in the surrounding area was unaffected by ethene emission. Tonneijck et al. (2003) also used this same study location and design on the more ethene sensitive petunia and marigold but found that the number of flowers was unchanged from controls at distances of 400 to 460 m downwind, and increased from control numbers at a distance of up to 1000m. Plants closer than 400 m to the source of emissions did show loss of flowers and decreases to the mean growth rate.

Dueck et al. (2003) used high concentrations of ethene (range: 230 to 920 µg/m^3) injected at ground level into open-top chambers on potatoes grown in the field. They found that potato leaves recovered fully after 12 hours of exposure and 3 days of recovery time. Overall, there was a decrease in the number of flower clusters; however, potato yield (including size or frequency of misformed potatoes) was unaffected regardless of concentration or frequency of treatment with ethene.

Other studies found that if exposure to ethene was terminated prior to irreversible effects, such as leaves falling off, plants were capable of recovering from exposure (Klassen and Bugbee 2002). Archambault and Li (2001) found that field pea were capable of recovering from long-term exposure of 115 µg/m^3 over 16 days, given sufficient time. Additionally, Archambault and Li (2001) found that exposure of barley plants to ethene concentrations

mimicking ethene monitoring data from the worst month at a nearby petrochemical facility (range ~11.5 to 300 µg/m^3) had no negative effects on the plants, likely caused by the intermittent nature of the exposure.

Given these kinds of emission data, it is very unlikely that plants are being exposed to ethene for sufficiently continuous lengths of time to cause long term impacts. While individual release events may cause very high concentrations of ethene in the atmosphere for a short period of time, these exposures are limited in time due to the rapid dispersal of ethene in the environment and the lack of long-term or prolonged exposure. Short-term exposure exceeds the PNEC approximately once per year and the long-term exposure values reflect the highest annual concentration in all years, including concentrations occurring over the winter months.

Some species of plants exposed to high concentrations of ethene can show an increase in epinasty, root length reduction, flower abscission, and ripening of the flower or fruit. However, these concentrations have also been found to promote fruit ripening and increase seed yield. Additionally, plants exposed in the environment tend to show a greater resistance to ethene exposure compared to plants studied in laboratory settings and, in both cases, when plants are given a recovery period, recovery can often be seen, especially in regards to leaf curling and growth inhibition.

Consideration of Lines of Evidence and Conclusion

Ethene is a naturally occurring substance and it is produced and used in large quantities in Canada. Anthropogenic releases are expected to be exclusively to air, mainly from combustion of fossil fuels (vehicle emissions) and industrial processes.

Ethene is not persistent in air and has low potential for long range transport. It is also not bioaccumulative.

Ethene is a precursor to ground level ozone. It is considered to have a Photochemical Ozone Creation Potential (POCP) of 100 and is the standard chemical for comparison with other chemicals. As well, a common degradation product of ethene in air is formaldehyde. Both ozone and formaldehyde are substances listed on Schedule 1 of CEPA.

Terrestrial plants are highly sensitive to ethene in air. However, risk quotients showed ambient concentrations were unlikely to have impacts in urban areas or in rural areas over either short- or long-term exposures. A risk quotient analysis using industrial ethene monitoring data for the years 2008 – 2012 indicated that there is, on average, one occurrence per year that has the potential to be harmful to terrestrial plants due to industrial emissions of ethene. A risk quotient analysis was not performed for terrestrial mammals as mammalian toxicity values were orders of magnitude greater than air concentrations expected to occur in Canada.

A major anthropogenic source of ethene is the internal combustion engine, which explains in part why ethene air concentrations in Canadian cities can be considerably higher than in rural and remote areas. Advances have been made in the last 20 years in reducing pollutants,

including ethene, from the exhaust of internal combustion engines. Canadian urban air ethene concentrations from the 1980s and 1990s ranged from 4 to 113 µg/m^3 (Alberta Environment 2003), while recent air monitoring (2005-2009) provides a range of 0.1 to 32.9 µg/m^3 (Environment Canada 2011). There appears to be a trend to lower air concentrations in Canadian cities, even over the last 10 years, which follows the reductions in other pollutants from automobile exhaust. Further reductions are expected as a result of more stringent requirements for NOx, SOx, and VOCs in automobile exhaust and with continual removal of older cars from use.

Facilities manufacturing, processing, or otherwise using more than 10 tonnes per year of the substance must report their releases to the National Pollutant Release Inventory (NPRI). In 2009, facilities across Canada reported to the NPRI on-site environmental releases totalling approximately 1320 tonnes. Industrial releases have dropped by over 50% since 2000 largely due to the amount of ethene being recycled.

Based on the information presented above, there is low risk of harm to organisms or the broader integrity of the environment from this substance. It is therefore concluded that ethene does not meet any of the criteria under paragraphs 64(*a*) or (*b*) of CEPA, as it 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.

Uncertainties in the Evaluation of Ecological Risk

The responses of specific plants to ethene are difficult to predict, as even closely related species can have variable responses when tested simultaneously. The duration and concentration of exposure to ethene are both important variables when considering the effects of ethene on plants; however, many studies only addressed one of these variables at a time. Additionally, there are few tests that specifically address sensitive life stages of plants as most studies focus on horticultural concerns such as flower appearance for the purpose of the transportation of cut flowers.

The most sensitive species available in addition to the most sensitive cultivar of that species was used to maintain a conservative approach to determining potential impacts on plants. The endpoint chosen for short-term exposure represented a significant effect and it is possible that lower concentrations may have still had a significant effect on barley. The long-term exposure scenario for ethene assumed that the modelled value of 5.6 µg/m^3 is a no effect concentration and it is considered a conservative value. However, the Archambault and Li study used 12 µg/m^3 as their baseline concentration as it aligned with monitoring data at the time and was considered a reasonable estimate of background concentrations. It is likely, based on monitoring data, that background concentrations are lower than those supposed in the Archambault and Li study.

There is also uncertainty in this assessment related to the exposure characterization of ethene, which is based on monitoring data that shows considerable variation between sites and considerable variation over time. Based on the available monitoring data, ethene

concentrations can vary on a daily basis or on an hourly basis due to the way it is released and the way ethene disperses in the environment. As opposed to the variable concentrations plants are exposed to in the environment, most studies used continuous exposure to ethene. This uncertainty was dealt with by developing conservative exposure scenarios using both the realistic worst case and the average case.

Potential to Cause Harm to Human Health

Exposure Assessment

Given ethene's physical-chemical properties, it is expected to volatilize to air and is not likely to occur in significant quantities in soil or drinking water. As such, inhalation is expected to be the predominant route of human exposure. As a common volatile organic compound (VOC), ethene has been well documented in air quality monitoring studies in Canada (e.g., Health Canada 2010a; Health Canada 2010b) and elsewhere (e.g., Altuzar et al. 2005; Badol et al. 2008; Olson et al. 2009; Lai and Peng 2011). Ethene has been investigated in traffic-related urban air studies (Cheng et al. 1994; Weichenthal et al. 2011; Environment Canada 2011), as well as in air monitoring surrounding industrial facilities in Canada (Cheng et al. 1997; Environment Canada 2011). Ethene is not added to foodstuffs or to beverages; however is it released naturally from fruits and other plant products. Ethene may be used as a welding gas, refrigerant and commercial ripening agent for produce. Ethene occurs as a product of incomplete combustion in vehicular exhaust (e.g., Tosaka et al. 1989; Taylor et al. 1994; Cheung et al. 2008) and in cigarette smoke (Löfroth et al. 1989; Barrefors and Petersson 1993; Baren et al. 2004).

Health Canada has recently measured personal exposure to ethene by monitoring outdoor, indoor and personal air concentrations in residential areas in three Canadian studies: Windsor Ontario Exposure Assessment Study (WOEAS) (Health Canada 2010 a), Regina Indoor Air Quality Study (RIAQS) (Health Canada 2010b), and Halifax Indoor Air Quality Study (HIAQS) (Health Canada 2011). As part of a residential air monitoring campaign, these studies serve to determine personal exposure to VOCs of the general population across Canada. Prior to this, available information on ethene with regards to indoor air and personal air in particular were limited. These studies, along with others deemed relevant for assessing ethene exposure for the general Canadian population, are discussed below and summarized in Table 11.

Outdoor Air

The National Air Pollution Surveillance (NAPS) program referred to in the Ecological Exposure Assessment Section represents the most exhaustive source of outdoor air monitoring data in Canada. A total of 64 monitoring sites across Canada gathered ethene concentrations between 2005 and 2009, the period selected and evaluated here. NAPS-designated monitoring site types for this 5-yr period were dominated by residential (28) and commercial (18) sites, followed by undeveloped rural (8), agricultural (6), forested rural (2), and industrial (2) sites. Ethene concentrations were measured either over a 24-hr or 4-hr period for a given site. Individual values measured across all sites ranged from 0.03 to 74.98 $\mu g/m^3$, while 95^{th} percentiles ranged from 0.39 to 14.2 $\mu g/m^3$. The lowest median concentration of ethene (0.03$\mu g/m^3$) was reported from the Alert site, an undeveloped rural site located in Alert, Nunavut. However, this site had been online during 2 years (i.e., 2005 and 2006) only. The Kemjimkujik National Park site located in Nova Scotia, which was online during the entirety of the 5 years, had the second lowest levels of ethene with a median value of 0.132 $\mu g/m^3$and a 95^{th} percentile 0.39 $\mu g/m^3$. The highest median concentration of ethene

(2.9 µg/m^3) was recorded at the Aamjiwnaang site, a residential site located in Sarnia, Ontario. Concentrations at this site ranged from 0.4 to 25.0 µg/m^{3}and corresponded to a 95th percentile of 11.0 µg/m^3. The highest 95th percentile of all sites, 14.2 µg/m^3, was reported at the Centennial Park monitoring site, a residential site also located in Sarnia, Ontario, (Environment Canada 2011).

As described in the Ecological Assessment section, regional monitoring of ethene in the Sarnia-Lambton area of Ontario was conducted from January 2008 to December 2012 by the Sarnia-Lambton Environmental Association (SLEA 2013). This region contains several industrial sources of ethene as well as vehicle and urban emissions (including from a major provincial highway). Hourly data for five monitoring stations were provided. Twenty-four-hour averages were computed for each year from 2008 to 2012 for the River Bend site, the site closest to a residential area. Overall values for this site ranged from detection limit to 30.0 ug/m^3 [26.1 ppb], and the highest 95th percentile of 1.8 ug/m^3 [5.4 ppb] was observed during 2009 (SLEA 2013).

Alberta is the largest fossil fuel producing province in Canada (Cheng et al. 1997). In Edmonton, besides vehicular emissions, VOCs are emitted from industrial facilities close to the city. In order to investigate emissions from industrial sources as well as from transportation, Cheng et al. (1997) measured VOC concentrations in air at two sites between 1991 and 1993: the downtown core of Edmonton, Alberta and an industrial complex in the outskirts of the city. Both sites were separated by roughly 9 km. The downtown sampling site was located on the roof of a one-storey building. The industrial complex, on the other hand, was sampled using a trailer placed in an open field with hydrocarbon storage tanks, oil refineries, smelter and chemical and shingle manufacturing plants within 200 m to 2.5 km. At the downtown site, ethene accounted for 3.9 % of total carbon measured, which corresponded to a median concentration of 4.99 µg/m^3. Levels at the industrial site were lower with a median concentration of 4.53 µg/m^3, accounting for 2.2 % of total carbon. Ethene was the 7th most abundant species at the downtown site while at the industrial site, it was ranked 10th out of the 20 most abundant species representing the majority (i.e., ~80%) of total VOC carbon. Vehicular emissions were expected to be the major sources for VOCs in the downtown area. The distinct concentration profile of VOCs at either location reflected the emissions limited to the surrounding area. As such, the authors noted that reducing emissions at one site would not significantly affect VOC concentrations at the other (Cheng et al. 1997).

In 2010 Health Canada published the Windsor Ontario Exposure Assessment Study (WOEAS) on the exposure of 188 VOCs in outdoor, indoor and personal air samples collected in the vicinity of Windsor, Ontario homes (Health Canada 2010a). One hundred participants were involved in this study. Five consecutive 24-hr samples were collected during the winter and summer periods of 2005 and 2006. Summary statistics were computed for both seasons during each year. Ethene concentrations in outdoor air ranged from 0.2 to 11.7 µg/m^3 across all measurements. The highest median concentration of ethene in outdoor air of 3.0 µg/m^3 was reported during the 2005 winter period, with a corresponding 95th percentile of 6.8 µg/m^3. Concentrations recorded during the following winter (2006) were lower with a median concentration of 1.6 µg/m^3, and a 95th percentile of 3.1 µg/m^3. Median

concentrations during the summer season for both 2005 and 2006 were 1.2 and 1.1 µg/m^3, respectively, and 95th percentile values were 3.6 and 2.4 µg/m^3.

In 2010 Health Canada also published the Regina Indoor Air Quality Study (RIAQS) on the exposure of 194 VOCs in outdoor and indoor air samples collected in residential areas located in Regina Saskatchewan to provide information on exposure (Health Canada 2010b). This city in particular was selected given the scarcity of air quality exposure data for the Prairie Provinces. Indoor and outdoor air samples were collected in one or both of two 10-week sampling sessions during the winter and summer of 2007 only from a total of 146 participating homes. Summary statistics for each VOC were calculated for each season. Concentrations of ethene in outdoor air throughout the entire study ranged from 0.2 to 8.1 µg/m^3. The highest ethene concentrations corresponded to the winter samples (24-hr only), with a median concentration of 1.0 µg/m^3 and a 95th percentile value of 4.0 µg/m^3. As for the summer season, the median concentration of ethene for the paired 24-hr samples was found to be 0.6 µg/m^3 with a 95th percentile of 1.4 µg/m^3. The 5-day samples that were also collected during the summer sampling period only were comparable to the summer 24-hr samples with a median concentration of 0.7 µg/m^3 and 95th percentile of 1.4 µg/m^3.

Health Canada's most recent study, the Halifax Indoor Air Quality Study (HIAQS), also measured 193 VOCs in outdoor and indoor air samples in residential areas of Halifax, Nova Scotia (Health Canada 2011). Samples were collected for 7 consecutive 24-hr periods over the 2009 winter and summer seasons from a total of 50 participating homes, and summary statistics were also computed per season. Ethene concentrations measured in outdoor air during the course of the study ranged from 0.1 to 5.9 µg/m^3. The highest median concentration of ethene (0.7 µg/m^3) was recorded during the winter period, with a 95th percentile concentration nearly three times higher (2.1 µg/m^3). The median concentration of ethene during the summer season was found to be 0.4 µg/m^3 with a 95th percentile concentration of 0.9 µg/m^3.

For all three HC studies, ethene outdoor air concentrations were higher (statistical significance unknown) in the winter than in the summer season. Several air monitoring studies in urban areas have also observed this same seasonal trend (Chang et al. 2005; Curren et al. 2006; Olson et al. 2009; Matsunaga et al. 2010; Lai and Peng 2011). An increase in VOC concentrations during the winter period can be due to the reduced height of the ground level mixing layer, thus limiting vertical transport of atmospheric pollutants and aggravating accumulation (Cheng et al. 1997; Badol et al. 2008). Also, higher concentrations of certain VOCs during the winter compared to the summer may be attributed to reduced atmospheric reactivity (Curren et al. 2006).

Although ethene is not a fuel component, it is present in motor vehicle exhaust as a result of incomplete combustion of fossil fuels (IARC 1994), which constitutes a major source of ethene exposure for commuters. Weichenthal et al. (2011) recently measured the concentrations of VOCs in outdoor samples in Ottawa, Ontario to examine the relationship between traffic pollution and impacts on heart rate variability and respiratory function for cyclists. Outdoor air samples were collected in the downtown core (high-traffic route) and along a bike path (low-traffic route). The median concentration of ethene along the high-

traffic route was 2.6 µg/m^3, which was expectedly higher than the median concentration of 0.8 µg/m^3 measured along the low-traffic route. A similar study was carried out among commuters in Dublin, Ireland in February of 2003 (O'Donoghue et al. 2007). Air samples were collected from the inside of a bus at respiratory level, and using an adapted carrier bag to house the sampling equipment on the bicycle. The bus and bicycle followed the same route. The mean air concentration of ethene for bus and bicycle passengers was 11.92 and 7.77 ppb (10.37 and 6.76 µg/m^3), respectively. Other international studies have reported similar ethene concentrations in outdoor air in high-traffic areas (Chang et al. 2005; Franco et al. 2010; Matsunaga et al. 2010).

A number of recent international studies have measured ethene in outdoor air also. For example, in the US, Olson et al. (2009) reported concentrations of 55 volatile organic compounds in samples collected near a highway in Raleigh, North Carolina. The highest mean concentration for ethene was 3.10 ppbv (2.70 µg/m^3). In Mexico, Altuzar et al. (2005) collected samples in March 1999 and November 2001. They reported the highest mean of 40.3 ppbv (35.06 µg/m^3) of ethene collected at their industrial sampling site in November 2001. In Europe, Badol et al. (2008) measured 53 VOCs for 1 year from September 2002 to August 2003 in an urban area of France. They reported an ethene concentration range of 0.02 to 231 ppb (mean of 3.06 ppb) (0.017 to 201, mean of 2.66 µg/m^3). In Asia, Lai and Peng (2011) measured 56 hydrocarbons in a vehicle tunnel for 12 days during 2007 and 2008. They reported a range of means of 15.2 to 118.3 ppb (13.2 to 103 µg/m^3). Matsunaga et al. (2010) reported a range of mean ethene concentrations of 1.1 to 14.4 ppbv (0.96 to 12.5 µg/m^3) measured at five sites in the Tokyo metropolitan area of Japan during the summer and winter of 2008. Finally, in the Arctic, Hopkins et al. (2002) reported the mean concentrations at three sampling sites to be 412.6, 21.7 and 17.1 pptv (0.3589, 0.0189 and 0.0149 µg/m^3) for samples collected during the summer of 1999.

Indoor Air

Much of the earlier ethene monitoring information is focused on the outdoor environment, particularly near high traffic areas. However, it is well known that indoor concentrations of VOCs are often more predictive of personal exposure given some important indoor sources of certain VOCs (Stocco et al. 2008). This is particularly relevant for the Canadian population given Canadians spend roughly 90% of their time indoors (Health Canada 1998).

In the Windsor Study, indoor air concentrations of ethene across all samples collected in 2005 and 2006 in Windsor, Ontario homes ranged from 0.3 to 133.7 µg/m^3 (Health Canada 2010a). The highest median concentration of ethene (3.5 µg/m^3) was captured during the 2005 winter period, and was associated to the highest 95th percentile of 23.9 µg/m^3. The median concentration of ethene measured indoors during the winter for the following year (2006) was found to be slightly lower 2.7 µg/m^3, with a 95th percentile concentration of 11.4 µg/m^3, and reasons and significance for this interannual difference were not discussed. As for the summer season, the median concentrations of ethene during 2005 and 2006 were 3.2 and 2.5, respectively, while 95th percentile values were considerably higher at 16.8 and 16.3 µg/m^3, respectively (Health Canada 2010a).

The Regina Study measured ethene concentrations in air samples collected in homes in Regina, Saskatchewan during the 2007 winter and summer seasons (Health Canada 2010b). Due to the different VOC signature produced by environmental tobacco smoke, the indoor VOC results were for homes without smokers were separated from those with smokers. Only homes without smokers are discussed in this section for comparability to the Windsor and Halifax studies, which did not include homes with smokers. Ethene concentrations in Regina homes (without smokers) ranged from 0.4 to 21.0 µg/m^3 across all samples. The 24-hr and 5-day samples were generally similar for a given season. The highest median concentration of ethene of 2.8 µg/m^3 was reported during the winter (24-hr and 5-day), although the highest 95th percentile (10.9 µg/m^3) for the 5-day samples was slightly higher than that of the 24-hr samples (10.5 µg/m^3). The summer indoor median values for this study were found to be 1.6 and 2.0 µg/m^3 for the 24-hr and 5-day samples, respectively, while the 95th percentiles were found to be 7.6 and 7.2 µg/m^3, respectively.

The Halifax Study reported ethene concentrations for indoor air samples collected in 2009 in homes located in Halifax, Nova Scotia (Health Canada 2011). Ethene concentrations in indoor air of all participating homes in Halifax ranged from 0.3 to 80.9 µg/m^3 across all samples. The median concentration of ethene in indoor air during the winter season was 1.7 µg/m^3 with a corresponding seasonal maximum 95th percentile of 7.4 µg/m^3. The summer season median concentration was 0.9 µg/m^3 with a corresponding 95th percentile of 3.5 µg/m^3.

In addition to the Health Canada air monitoring studies, Canadian indoor air concentrations of ethene in Ottawa, Ontario office building have recently been reported by Weichenthal et al. (2011). The aim of the study was to determine the exposure of cyclists to atmospheric pollutants as described earlier. The median concentration of ethene in the office building was 1.56 µg/m^3, which was lower than the outdoor high-traffic concentration (2.6 µg/m^3), but higher than the low-traffic concentration (0.8 µg/m^3) measured in this study.

The Health Canada studies (Health Canada 2010a; Health Canada 2010b; Health Canada 2011) as well as Weichenthal et al. (2011) show that indoor ethene concentrations can be higher than those outdoors. For instance, in the Windsor Study 2006 winter period, the 95th percentile concentration for indoor air (23.9 µg/m^3) was roughly 3.5-fold higher than the paired outdoor air 95th percentile concentration (6.8 µg/m^3). The presence of ethene in indoor environments may arise from both outdoor and indoor sources. As an incomplete combustion product, ethene and many VOCs in general may be present in homes as a result of cigarette smoking (Löfroth et al. 1989; Barrefors and Petersson 1993; Baren et al. 2004), vehicle exhaust infiltrating from attached garages (Stocco et al. 2008), wood burning (Barrefors and Petersson 1995), the presence of a gas stove (Stocco et al. 2008), and other combustion activities (e.g., cooking and candle burning).

Endogenous production of ethene by fruit and vegetables (see Environmental Assessment Section and Human Health Section–Food and Beverages) also constitutes a source of ethene in indoor air although the relative contribution is expected to be negligible in residential settings. Ethene has been measured in gas emanating from human skin, or skin gas, at an average amount of 20 ± 11 pg/cm^2 (mean ± SD) over the course of 30 minutes

(Nose et al. 2005); however, skin gas is also expected to be a negligible source of ethene in homes.

Personal Air

Health Canada's Windsor Exposure Assessment Study (WEAS) included personal air concentrations for the winter and summer of 2005 (Health Canada 2010a). Personal air reflects samples collected using an adapted carrier bag to house the sampling equipment which adult participants carried with them wherever they went throughout the day (i.e., mobile sampling). As opposed to stationary sampling, mobile sampling captures activity patterns (both indoors and outdoors) of participants, thus representing a refinement in exposure estimates. Research has shown that VOC concentrations are often higher in personal air than in corresponding outdoor air (Wallace et al 1985; Sexton et al 2007); personal concentrations of ethene in Health Canada's Windsor Study were no exception. The personal air concentrations of ethene for Windsor participants ranged from 1.1 to 120.5 µg/m^3. The median concentrations of personal air in the winter and summer were 3.8 and 3.4 µg/m^3, respectively, which were higher than the corresponding outdoor concentrations of 3.0 and 1.2 µg/m^3, respectively. However, median levels for personal air were slightly lower than paired indoor air levels of 3.5 and 3.2 µg/m^3 for winter and summer, respectively. Concentrations of ethene in personal air samples collected during the winter ranged from 1.1 to 66.1 µg/m^3 while concentrations of ethene during the summer season ranged from 1.3 to 120.5 µg/m^3. The highest 95th percentile for seasonal personal air concentrations was measured in the summer, at 16.9 µg/m^3

Table 11. Outdoor, indoor, and personal air concentrations of ethene in Canada.

Table 11-A. Outdoor Air

Reference	Location	Sampling Period and Duration	n	Mean (Range) (µg/m^3)	Median (µg/m^3)	P95 (µg/m^3)
Environment Canada 2011 (NAPS[i])	Residential Centennial Park–Sarnia, ON [ii]	2005-2009 24-hr	182	3.9 (0.2-32.9)	1.8	14.2
Environment Canada 2011 (NAPS)	Residential Aamjiwnaang – Sarnia, ON [iii]	2005-2009 24-hr	40	4.0 (0.4-25.0)	2.9	11.0
Environment Canada 2011 (NAPS)	Industrial Edmonton East – Edmonton, AB	2005-2009 24-hr	294	3.0 (0.2-31.6)	1.9	8.7
Environment Canada 2011 (NAPS)	Commercial Prg Plaza – Prince George, BC	2005-2009 24-hr	217	2.1 (0.2-14.2)	1.4	6.2

Reference	Location	Sampling Period and Duration	n	Mean (Range) (µg/m^3)	Median (µg/m^3)	P95 (µg/m^3)
Environment Canada 2011 (NAPS)	Agricultural Rural Rivière-des-prairies – Montreal, QC	2005-2009 24-hr	284	1.7 (0.2-39.4)	1.1	4.7
Environment Canada 2011 (NAPS)	Forested Rural Syncrude UE1– Fort Mackay, AB	2005-2009 24-hr	89	0.5 (0.1-2.3)	0.3	1.5
Environment Canada 2011 (NAPS)	Undeveloped rural Hope Airport – Metro Van-Hope, BC	2005-2009 24-hr	33	0.7 (0.2-1.7)	0.6	1.6
Environment Canada 2011 (NAPS)	Undeveloped rural Alert, NU[iv]	2005-2009 4-hr	98	0.1 (0.0-1.0)	0.0	0.4
Health Canada 2010a (WOAES)	Windsor, ON	2005 Winter 24-hr	126	3.2 (0.8-11.7)	3.0	6.8
Health Canada 2010a (WOAES)	Windsor, ON	2005 Summer 24-hr	216	1.5 (0.2-4.7)	1.2	3.6
Health Canada 2010a (WOAES)	Windsor, ON	2006 Winter 24-hr	215	1.7 (0.5-3.8)	1.6	3.1
Health Canada 2010a (WOAES)	Windsor, ON	2006 Summer 24-hr	214	1.2 (0.3-3.6)	1.1	2.4
Health Canada 2010b (RIAQS)	Regina, SK	2007 Winter 24-hr	94	1.5 (0.4-8.1)	1.0	4.0
Health Canada 2010b (RIAQS)	Regina, SK	2007 Summer 24-hr	108	0.7 (0.2-3.1)	0.6	1.4
Health Canada 2010b (RIAQS)	Regina, SK	2007 Summer 5-day	97	0.8 (0.4 - 2.2)	0.7	1.2

Reference	Location	Sampling Period and Duration	n	Mean (Range) (µg/m^3)	Median (µg/m^3)	P95 (µg/m^3)
Health Canada 2011 (HIAQS)	Halifax, NS	2009 Winter 24-hr	287	0.9 (0.2-6.0)	0.7	2.1
Health Canada 2011 (HIAQS)	Halifax, NS	2009 Summer 24-hr	324	0.5 (0.1-3.5)	0.4	0.9
Weichenthal et al. 2011	Ottawa, ON	2010 High-traffic 1-hr	39	2.8 (0.5-6.5)	2.6	5.7
Weichenthal et al. 2011	Ottawa, ON	2010 Low-traffic 1-hr	39	0.9 (0.4-2.8)	0.8	2.5
Cheng et al. 1997	Edmonton, AB	1991–1994 24-hr	212	4.5	5.0	
SLEA 2013	Sarnia-Lambton, ON	2008 24-hr	362	1.5 (0-13.9)	0.8	5.3
SLEA 2013	Sarnia-Lambton, ON	2009 24-hr	360	1.8 (0-30.0)	0.9	6.2
SLEA 2013	Sarnia-Lambton, ON	2010 24-hr	357	1.2 (0-18.4)	0.6	3.8
SLEA 2013	Sarnia-Lambton, ON	2011 24-hr	358	1.4 (0-28.8)	0.4	5.7
SLEA 2013	Sarnia-Lambton, ON	2012 24-hr	357	0.9 (0-11.5)	0.4	3.8

Abbreviations: P95, 95th percentile; NAPS, National Air Pollution Surveillance; WOAES, Windsor Ontario Exposure Assessment Study; RIAQS, Regina Indoor Air Quality Study; HIAQS, Halifax Indoor Air Quality Study

[i] Data for years 2005 – 2009; Sites provided represent those with the highest 95th percentile concentration for the site type (e.g., Residential), unless otherwise specified.
[ii] Highest 95th percentile of all sites
[iii] Highest median
[iv] Lowest 95th percentile of all sites

Table 11-B. Indoor Air

Reference	Location	Sampling Period and Duration	n	Mean (Range) (µg/m^3)	Median (µg/m^3)	P95 (µg/m^3)
Health Canada 2010a	Windsor, ON	2005 Winter 24-hr	91	6.0 (0.3-72.4)	3.5	23.9

Reference	Location	Sampling Period and Duration	n	Mean (Range) (µg/m^3)	Median (µg/m^3)	P95 (µg/m^3)
(WOAES)[v]						
Health Canada 2010a (WOAES)	Windsor, ON	2005 Summer 24-hr	217	5.8 (0.9-133.7)	3.2	16.8
Health Canada 2010a (WOAES)	Windsor, ON	2006 Winter 24-hr	224	3.6 (0.8-16.7)	2.7	11.4
Health Canada 2010a (WOAES)	Windsor, ON	2006 Summer 24-hr	205	4.5 (0.6-45.8)	2.5	16.3
Health Canada 2010b (RIAQS)[vi]	Regina, SK	2007 Winter 24-hr	83	3.8 (0.8-21.0)	2.8	10.5
Health Canada 2010b (RIAQS)	Regina, SK	2007 Winter 5-day	70	4.1 (1.1 - 20.6)	2.8	10.9
Health Canada 2010b (RIAQS)	Regina, SK	2007 Summer 24-hr	91	2.6 (0.4-12.4)	1.6	7.6
Health Canada 2010b (RIAQS)	Regina, SK	2007 Summer 5-day	88	2.8 (0.6 - 16.5)	2.0	7.2
Health Canada 2011 (HIAQS)[vii]	Halifax, NS	2009 Winter 24-hr	312	2.8 (0.4-60.7)	1.7	7.4
Health Canada 2011 (HIAQS)	Halifax, NS	2009 Summer 24-hr	331	1.5 (0.3-80.9)	0.9	3.5
Weichenthal et al. 2011[viii]	Ottawa, ON	2010 Summer	39	1.8 (0.7-3.1)	1.6	2.7

Abbreviations: P95, 95th percentile; WOAES, Windsor Ontario Exposure Assessment Study; RIAQS, Regina Indoor Air Quality Study; HIAQS, Halifax Indoor Air Quality Study

Notes:
[v] Home (without smokers only)
[vi] Home
[vii] Home
[viii] Office building

Table 11-C. Personal Air

Reference	Location	Sampling Period and Duration	n	Mean (Range) (µg/m^3)	Median (µg/m^3)	P95 (µg/m^3)
Health Canada 2010a (WOAES)	Windsor, ON	2005 Winter 24-hr	119	6.6 (1.1-66.1)	3.8	13.4
Health Canada 2010a (WOAES)	Windsor, ON	2005 Summer 24-hr	207	5.7 (1.3-120.5)	3.4	16.9

Abbreviations: P95, 95th percentile; WOAES, Windsor Ontario Exposure Assessment Study

Drinking Water

Based on its physical-chemical properties, ethene is not expected to occur in drinking water; hence, no data was found regarding concentrations of ethene in drinking water in Canada or elsewhere.

Food and Beverages

Ethene is not added to foodstuffs or beverages; however it is released naturally from fruits and vegetables. Endogenous production of ethene in plant tissue generally increases rapidly during ripening (IARC 1994). Although plants normally produce 0.6-6 µg/kg fresh weight per hour, production rates may increase to 120 µg/kg weight per hour during ripening (Dörffling 1982; Tille et al. 1985). Nonetheless, the potential exposure of ethene from the consumption of fruits and vegetables is expected to be negligible, and therefore has not been characterized here.

Furthermore, due to the substance's high volatility, ethene was exempted from the requirement of a tolerance for residues or an acceptable daily intake (ADI) from its use as a plant growth regulator on agricultural crop in the US and in Canada (US EPA 1992a; PMRA 2001). No dietary exposure of ethene is expected for Canadians from the use of ethene for the postharvest ripening of bananas and other tropical fruits, and for degreening of citrus (Lunau 2010; CGSB 2011).

Soil and Dust

The surface layer in soil comprised of litter emits considerable amounts of ethene, which is produced by plants and soil microorganisms, under aerobic conditions (Sawada and Totsuka 1986). Smith and Restall (1971) measured the production of ethene by soils under anaerobic conditions where levels were found to be more than 20 ppm (17.4 mg/m^3) in several soils after 10 days at 20°C. These concentrations exceeded those known to cause severe reductions in the growth of root axes of some plant species. The authors also showed that ethene was produced by enzyme activity and not by chemical action based on comparative experiments between sterilized and unsterilized soil (Smith and Restall 1971). While the surface layer of soil is expected to release ethene into the atmosphere, lower soil levels may actively degrade ethene, but only under aerobic conditions or when ethene levels are high (Sawada and Totsuka 1986).

Ethene is not expected to occur in dust based on its physical-chemical properties; hence no data was found regarding concentrations of ethene dust in Canada or elsewhere.

Consumer Products

Ethene has been identified in the international literature for its use as a welding gas and refrigerant (CGAI 1999). According to Dow (2007), no consumer uses of ethene are known. The survey conducted pursuant to section 71 of CEPA did not identify specific consumer products for ethene (Environment Canada 2003a). As such, exposure to ethene via consumer products is not expected.

Tobacco Smoke

There is strong evidence showing that cigarette smoke is a significant source of exposure to ethene.

In Health Canada's Regina Study, homes with smokers present corresponded to higher levels of ethene than did homes without smokers present (Health Canada 2010b). The median level of ethene in homes with smokers during the winter (6.6 µg/m^3, 5-day samples) was nearly double that found in homes without smokers (2.8 µg/m^3, 5-day samples). The corresponding 95th percentile in homes with smokers present (23.1 µg/m^3) was nearly twofold that found in homes without smokers (10.9 µg/m^3).

Barrefors and Petersson (1993) measured ethene levels in a small café (ca. 150 m^3) in Sweden which was occupied by 10 smoking and 10 non-smoking customers, where concentrations were found to be 56 µg/m^3. As a comparison, the authors later placed a burning cigarette in a flask then measured the ethene levels in the empty café, which were found to be 42 µg/m^3. Ethene was a major constituent of smoke in both cases: of all hydrocarbons measured, ethene ranked third highest (8.9 %) when customers were present, and second highest (11 %) when the cigarette was burning. The corresponding levels in a car during urban driving measured in this study were lower (18-30 µg/m^3) than the smoky café; however ethene levels measured in a nearby urban road tunnel (280 µg/m^3) were considerably higher.

The total mass of ethene produced per cigarette has also been investigated in several studies. Löfroth et al. (1989) determined that 1200□ μg of ethene were released per cigarette in sidestream smoke (smoke released from the burning cone during the puff). In an experimental study, Baren et al. (2004) measured ethene in mainstream smoke (generated during a puff when the air is drawn through the burning cone) as well as in sidestream smoke where both smoke types were sampled simultaneously, as opposed to separately. The total mass of ethene emitted per cigarette in mainstream smoke from three different cigarette types ranged from 140 to 190 μg, while for sidestream smoke ethene was below the limit of detection of 1600 μg per cigarette for the sidestream smoke method (Baren et al. 2004). Levels of ethene in mainstream and sidestream smoke collected separately were measured previously by Shi et al. (2003). Ethene yields for a single reference cigarette in this case were found to be 140 μg for mainstream smoke, and 700 μg for sidestream smoke (Shi et al. 2003).

Estimate of exposure

Given ethene's physical-chemical properties, air is considered to be the predominant source of exposure for the general public. The 95th percentiles for ambient air concentrations were, on average, lower than indoor air across all studies. From the available monitoring data, concentrations of ethene in indoor air were either higher or similar to personal air. Personal air data is considered to be more representative of air concentrations present in the breathing zone as it samples the air surrounding the individual, rather than in fixed indoor or outdoor locations. The highest 95th percentile concentrations for personal air and indoor air of 16.9 μg/m^3 and 23.9 μg/m^3, respectively, identified in the Windsor Study (Health Canada 2010a) are considered to be upper-bounding air concentrations to which the general population of Canada is exposed.

Confidence in Exposure Database

Confidence in the exposure data of ethene in environmental media is considered high. Ambient, indoor, and personal air monitoring data were available, and were recent and representative of Canadian houses. Despite the lack of data for some environmental media, such as drinking water, the physical chemical properties of ethene suggest that there would be minimal amounts present. Confidence in the ethene air concentrations is high given the conservative nature of the assessment.

Health Effects Assessment

A summary of the available health effects information for ethene is presented in Appendix A.

The International Agency for Research on Cancer (IARC) concluded that there is inadequate evidence in humans and experimental animals for the carcinogenicity of ethene. Therefore, ethene was evaluated as not classifiable as to its carcinogenicity to humans (Group 3

carcinogen) (IARC 1994). The OECD has also concluded: “Relevant studies on ethene have indicated a low toxicity and no risk to human health…” (OECD 1998). The above classification and conclusions were based principally on test results in experimental animals.

No tumors were observed in experimental animals exposed to ethene in a two-year carcinogenicity study. One hundred and twenty Fisher 344 rats per sex per dose were exposed to 0, 40, 1000 or 3000 ppm ethene via inhalation for up to 24 months. Randomly selected animals were necropsied and examined after 6, 12 and 18 months of exposure. All surviving rats were necropsied at 24 months. There were no statistically significant differences among any of the exposed groups with respect to any of the heamatology, blood chemistry or other parameters investigated. No gross or histopathology tissue changes attributable to the effects of the test material were observed in any of the exposed rats (CIIT 1979). Based on the observations from this study, Hamm et al. concluded that the results (of the study) provided “…no evidence that ethene at these concentrations causes chronic toxicity or is oncogenic in Fisher 344 rats” (Hamm et al. 1984).

Ethene can be metabolized to ethylene oxide *in vivo* in rodents and humans (Ehrenberg et al.1977; Törnqvisk et al. 1988; Törnqvisk et al. 1989; Walker et al. 1990). In contrast to ethene, ethylene oxide, is a potent alkylating agent and a genotoxic carcinogen both in experimental animals and humans (IARC 2008; Ehrenberg et al. 1977). Ethylene oxide has been shown to alkylate (2-hydroxyethylate) RNA, DNA and proteins, and the resulting genetic damage has been thought to play a critical role in the induction of mutations and cancers in rodents (IARC 2008). Identical products of alkylation have also been observed after exposure of rodents to ethene; this was attributed to the conversion of ethene to ethylene oxide (Ehrenberg et al. 1977; Sergerbäck 1983; Eide et al.1995).

Numerous studies on the rate at which ethene are metabolized to ethylene oxide have been conducted in experimental animals and in humans. Results of these studies have shown that when inhaled at low concentrations, 5- 10 % of ethene could be converted to ethylene oxide in exposed mice, rats and hamsters (Sergerbäck 1983; Törnqvisk 1988). The maximum conversion of ethene to ethylene oxide in humans was estimated to be 4 %, while about 1 % has been measured. In addition, it has been found that the level of endogenous ethene in humans is lower than that in rodents because there is a lower endogenous production rate in humans when normalized for body weight (Törnqvisk 1989; Törnqvisk 1994; Filser et al. 1992; Csanády et al., 2000; OECD 1998).

In order to more accurately assess potential for cancer risks from exposure to ethene, Walker et al. investigated potential use of N-(2-hydroxyethyl)-valine (HEV), N7-(2-hydroxyethyl)-guanine (N7-HEG) and hypoxanthine guanine phosphoribosyltranferase (Hprt) mutant frequency as quantitative indicators or biomarkers of *in vivo* conversion of ethene to ethylene oxide in experimental animals. In Walker’s study, groups of male F344 rats and male B6C3F1 mice were exposed to 0, 40, 1000 or 3000 ppm ethene, via inhalation, for 4 weeks. HEV, N7-HEG and Hprt mutant frequencies were assessed for determining the dose of ethylene oxide resulting from exogenous ethene exposures; these biomarker values were then compared with the same background biomarker values. The results of the study showed that the dose-response curves for N7-HEG and HEV were superlinear in exposed rats and

mice, indicating that metabolic activation of ethene was saturated at exposures >1000 ppm (Walker et al. 2000). This finding coincided with the results from an earlier study by Bolt and Filser in which a two-compartment pharmacokinetic model was used for the elimination of ethene in Sprague-Dawley rats. The results of Bolt and Filser's study indicated that above a concentration of 1000 ppm, ethene reached a maximal rate of metabolism (Vmax). Therefore, higher exposures to ethene would not yield greater conversion to ethylene oxide. Exposure of rats to concentrations of ethene >1000 ppm correspond to a theoretical exposure to about 6 ppm ethylene oxide (Bolt and Filser 1987).

The saturation of metabolism from ethene to ethylene oxide may explain the phenomenon that ethene failed to demonstrate the induction of mutations or cancers in experimental animals although it can be converted to ethylene oxide *in vivo*. In a cancer bioassay conducted by Snellings et al., exposure of F344 rats to ethylene oxide significantly increased incidences of mononuclear cell leukemia and brain tumours at > 10 and > 30 ppm exposure levels respectively (Snellings et al. 1984); yet, exposure of F344 rats to 3000 ppm ethene for 2 years failed to induce these tumours (Hamm et al. 1984). In addition, no significant mutagenic response was observed in the Hprt gene of ethene exposed animals, whereas exposure of rats and mice to 200 ppm ethylene oxide, as a positive control, led to a significant increase in Hprt mutant frequencies in splenic T cells. Walker et al., therefore, suggested that too little ethylene oxide can be produced from ethene exposure to cause mutagenic and carcinogenic responses in exposed experimental animals under current standard bioassay conditions (Walker et al. 2000). This finding is consistent with the OECD SIDS assessment: "In the case of ethylene (ethene), a possible mechanism for a toxic potential in humans has been identified, but few signs of toxicity have been observed. This is related to the fact that ethene gives rise only to minute doses of ethylene oxide" (OECD 1998).

Ethene has also been tested for its genotoxicity in *in vivo* and *in vitro* assays. An overview of the available genotoxicity studies is presented in Appendix A; these data are briefly summarized here.

The overall genotoxicity test results for ethene are negative. Ethene did not cause gene mutations in *Salmonella typhimurium* TA100 or in *Escherichia coli* with or without metabolic activation. It did not induce chromosome aberration in Chinese hamster ovary cells. No increase in Hprt frequency in splenic T cells in rats and mice exposed to ethene via inhalation for 4 weeks. In an *in vivo* micronucleus test, ethene did not significantly increase the frequency of micronucleated polychromatic erythrocytes in bone marrow of rats and mice exposed to ethene via inhalation for 4 weeks. However, it showed positive results with DNA alkylation in the exposed mice and rats, which was caused by its metabolite, ethylene oxide (IARC 1994). As reported earlier in this document, no tumours have been observed in the ethene exposed animals in a two year study, although positive results of alkylation have been found. This may be related to the fact that ethene gives rise only to minute doses of ethylene oxide.

Exposure to ethene has shown very limited non-cancer effects in experimental animals. No significant toxicity was observed in the male and female Fisher 344 rats exposed to ethene,

at concentrations of 0 to 3000 ppm, via inhalation, for up to 24 months (CIIT 1979). In a 13-week study, male and female Albino rats were exposed to 0 to 10,000 ppm ethene via inhalation. There were no differences between controls and exposed rats with respect to weekly mean weight change, total weight gain, food consumption, haematology, clinical chemistry, gross pathology or histopathology. Compared with the controls, the liver weights in several groups of exposed rats were significantly lower. There was, however, no dose-response relationship for this organ weight reduction and the cause was unknown (Rhudy et al. 1978; OECD 1998). In other two more recent 13 week studies, adverse effects were, however, observed in the exposed animals. In one 13-week study, male and female F344 rats were exposed to 0 to 10,000 ppm ethene via inhalation, very slight to moderate multifocal hyperplasia/hypertrophy of mucous secretory cells in nasal mucosa, accompanied by very slight to slight multifocal accumulations of eosinophils were observed (USEPA 2007). In another 13-week study, male Wistar and Fisher 344 rats were exposed to 0 or 10,000 ppm ethene via inhalation. Nasal lesions were observed in both strains of treated rats, but reduced severity of effects was seen in Wistar rats compared with the Fisher 344 rats (USEPA 2009a). Nasal lesions in treated rats were also reported in a 4-week study, in which male F344 rats were exposed to 0 or 10,000 ppm ethene via inhalation. Exposure related lesions both in the proximal and distal nasal airways were observed in the treated rats (US EPA 2009b). In another 4-week study, male Fisher 344 rats and male B6C3F1 mice were exposed to 0, 40, 1000 or 3000 ppm ethene via inhalation, formation of haemoglobin and DNA adducts were observed both in rats and mice at 40 ppm and higher concentrations (Walker et al. 2000). DNA alkylation was also observed in a 20-day study in male F344 rats exposed to 0 to 3000 ppm ethene via inhalation (Rusyn et al. 2005). Since no genotoxic or carcinogenic effects have been observed in ethene exposed animals (at much higher exposure concentrations), it would be more appropriate to consider the haemoglobin and DNA adducts as exposure biomarkers rather than toxicological effects.

The potential for reproductive effects on male and female rats and developmental effects on the offspring have been studied. Male and female rats were exposed to 0, 200, 1000 or 5000 ppm ethene via inhalation. Ethene was given to parent animals for 2 weeks prior to mating, during the mating period, until the day prior to necropsy for the males (minimum 28 days), and until day 20 of gestation for the females. Females and offspring were sacrificed on day 4 of postpartum. No effects on body weight gain, fertility or fecundity were observed. Litter size, sex ratio, mean pup weight and pup growth and clinical condition were not adversely affected by the treatment. Necropsy revealed no macroscopic finding and any suggestion of toxicity due to the treatment. No toxic effects on the testis were observed, and there were no deaths attributable to the exposure (OECD 1998).

Several epidemiological studies were identified. One study was conducted in workers at a petrochemical plant in the United States (US). An increased risk of developing brain cancer was found associated with exposure to (unspecified levels of) a number of chemicals including ethene. However, the investigators were unconvinced that the association reflected a causal relationship. In particular, ethene was considered to be unlikely to be associated with the effects because no tumours were found in rats in a two-year inhalation study and no mutagenic effects were observed in Ames tests (Leffingwell 1983). Another study was conducted among workers at a Swedish petrochemical plant using measurements of

haemoglobin adducts formed from ethylene oxide for monitoring of ethene exposure. The study was carried out in two parts, part one in 1989 and part two in 1993. In part one, eight workers exposed to high levels of ethene (4 mg/m^3) and three workers exposed to low levels (0.1 -0.3 mg/m^3) were compared to nine controls exposed to 0.01 mg/ m^3. All exposed workers showed elevated levels of haemoglobin adducts, and the adduct formation was dose-related. The results indicated that about 1 % of the inhaled ethene was metabolized to ethylene oxide. The second part of the study, which was made up of four workers, was designed to more accurately determine the exposure levels. The results of part two confirmed part one, showing that about 1 % of inhaled ethene was metabolized to ethylene oxide and the maximum amount to be converted was estimated to be 4 % (OECD 1998). No increase in cancer incidences was found in 31 workers exposed to ethene (at unspecified levels) in a case-control study of lung cancer among chemical workers at a petrochemical factory in the US (Bond et al.1986).

In summary, ethene did not induce tumours in rats in a two year inhalation study. It did not demonstrate genotoxic effects in *in vivo* or *in vitro* assays. Although ethene can be metabolized to ethylene oxide, pharmacokinetic studies have shown that saturation of metabolism from ethene to ethylene oxide exist both in experimental animals and in humans, and the quantity of ethylene oxide metabolized from ethene was insufficient to produce mutagenic or carcinogenic effects in experimental animals. Studies have further demonstrated that the rate in which ethene is metabolised to ethylene oxide is lower in humans than in experimental animals, thus, the possibility of inducing cancer in humans is even less than in rodents. In addition, no evidence of carcinogenicity has been found in epidemiology studies.

With respect to non-cancer effects, ethene caused slight nasal effects in exposed rats, via inhalation at concentration of 11,500 mg/m^3 (10,000 ppm), which is the lowest lowest-observed-adverse-effect concentration (LOAEC) for inhalation exposure. No reproductive and developmental effects were found in exposed male and female rats and in their offspring.

The confidence in the toxicity database for ethene is considered to be moderate to high as a comprehensive dataset including carcinogenicity, genotoxicity, reproductive/developmental toxicity and repeated-dose toxicity, as well as mode of action data were available to identify critical endpoints for risk characterization. However, only limited epidemiological studies targeting ethene were available.

Characterization of Risk to Human Health

Ethene was “not classifiable as to its carcinogenicity to humans (Group 3)” by IARC based upon “inadequate evidence in humans” and “inadequate evidence in experimental animals” for its carcinogenicity (IARC 1994). The OECD has also concluded: “Relevant studies on ethene have indicated a low toxicity …” (OECD 1998).

Ethene did not induce gene mutations in *in vivo* or *in vitro* assays. It was not carcinogenic in rats in a two-year inhalation study and in addition, limited epidemiology studies also did not show evidence of cancer in the exposed workers.With respect to non-cancer effects, the

lowest LOAEC for inhalation exposure (the principal route of exposure for the general population) was 11,500 mg/m^3 (10,000 ppm) based on slight nasal effects observed in rats in a 13-week inhalation study. Comparisons of this effect level with the highest 95th percentile concentrations for both indoor air and personal air measured for ethene in Canada (23.9 µg/m^3 or 16.9 µg/m^3) result in margins of exposure of approximately 481 200 or 680 500.

On the basis of the adequacy of the margins between upper-bounding estimates of exposure to ethene and critical effect levels, it is concluded that ethene does not meet the criteria under paragraph 64 (*c*) of CEPA as it 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.

Uncertainties

This final screening assessment does not include a full analysis of the mode of induction of effects associated with exposure to ethene, nor does it take into account possible differences between humans and experimental species with respect to effects induced by this substance. However, margins of exposure are considered sufficiently large to adequately account for any variations. The available human data were limited because of small sample sizes, exposure to a mixture of chemicals, lack of details on study protocols and exposure conditions, and because of confounding factors inherent in epidemiology studies.

Uncertainties surrounding exposure to ethene are minor given the high confidence in the ethene database based on the relevant, recent and well conducted Health Canada studies available with outdoor, indoor, and personal air—the most relevant sources of exposure via the environment for ethene.

Conclusion

Based on the information presented in this screening assessment on the concentration of ethene in the environment, there is low risk of harm to organisms or the broader integrity of the environment from this substance. It is concluded that ethene does not meet criteria under paragraphs 64(*a*) or (*b*) of CEPA as it is not entering the environment in a quantity or concentration or under conditions that have or may have immediate or long-term harmful effects on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

On the basis of the adequacy of the margins between conservative estimates of exposure to ethene and a critical effect level, it is concluded that ethene does not meet the criteria under paragraph 64(*c*) of CEPA as it 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 ethene does not meet any of the criteria set out in section 64 of CEPA.

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Appendix A. Summary of health effects information for ethene

Table A-1. Studies in experimental animals and cells

Endpoints	Lowest effect levels[1]/Results
Acute toxicity	**Inhalation LC_{50} (mice)** = 1,093,000 m g/m^3 (950,000 ppm) (OECD 1998). **Lowest inhalation LOEC (rats)** = 344 mg/m^3 (300 ppm) based on inhibition of enzymes (various monooxygenase and cytochrome P450) activity in male Fisher 344 rats exposed to ethylene via nose-only inhalation at 300, 600 and 1000 ppm for up to 6 hours (Fennell et al. 2004). **Other:** **LOEC (rats)** = 1147 mg/m^3 (1000 ppm) based on inhibition of cytochrome P450 2E1activity in the liver of rats exposed to ethylene via whole-body inhalation for 2 hours (Erbach et al. 2007). **NOAEC (rats)** = 10,000ppm based on no increase in serum enzyme activities and no necrotic tissue in Fisher rats exposed to ethene for 5 hours (Guest et al.1981). Ethene was in general use as an anaesthetic for many years. The initial symptoms of asphyxiant gas effects are rapid respiration and air hunger, diminished mental alertness, and impaired muscular coordination. Continuing lack of oxygen causes faulty judgement, depression of all sensations, rapid fatigue, emotional instability, nausea, vomiting, prostration, unconsciousness, and finally, convulsion, coma or death (Bibra 1993). No oral and dermal studies were identified.

Endpoints	**Lowest effect levels[1]/Results**
Short-term toxicity	**Lowest inhalation LOAEC(rats) =** 11,472 mg/m^3 based on exposure related lesions in both proximal and distal nasal airways in male F344 rats exposed to ethene at 0(filtered air) or 10,000 ppm (equivalent to 11,472 mg/m^3) via whole body inhalation, 6 hours per day, 5 days per week for 4 weeks (USEPA 2009b). **Other inhalation studies:** **LOEC(rats)** = 46 mg/m^3 based on formation of haemoglobin and DNA adducts observed in male F344 rats exposed to ethylene via whole body inhalation at 0, 40, 1000 or 3000 ppm (equivalent to 0, 46, 1147 or 3442 mg/m^3), 6 hours per day, 5 days per week for 4 weeks (Walker et al. 2000). **LOEC(mice)** = 46 mg/m^3 based on formation of haemoglobin and DNA adducts observed in male B6C3F1 mice exposed to ethylene via whole body inhalation at 0, 40, 1000 or 3000 ppm (equivalent to 0, 46, 1147 or 3442 mg/m^3), 6 hours per day, 5 days per week for 4 weeks (Walker et al. 2000). **LOEC(rats)** = 3442 mg/m^3 based on significant ($p<0.05$) increase in number of 7-HEV adducts observed in male F344 rats exposed to ethene via whole body inhalation at 0(clean air), 40 or 3000ppm (equivalent to 0, 46 or 3442 mg/m^3), 6 hours per day, 5 days per week for up to 20 days (Rusyn et al., 2005) No oral and dermal studies were identified.

Endpoints	Lowest effect levels[1]/Results
Sub-chronic toxicity	**Inhalation LOAEC (rats)** = 11,472 mg/m^3 based on nasal lesions observed in male Wistar and male Fisher 344 rats (10 per group) exposed to ethene via whole body inhalation at 0 or 10,000 ppm (equivalent to 0 or 11,472 mg/m^3), 6 hours per day, 5 days per week, for 13 weeks. Nasal lesions were observed in both strains of treated rats, but reduced severity of effects was seen in Wistar rats compared with the Fisher 344 rats (USEPA 2009a). **Other inhalation studies:** Albino rats (15 per group per sex) were exposed to ethene, via inhalation, at 0, 300, 1,000, 3,000 or 10,000 ppm (equivalent to 0, 345, 1150, 3450 or 11,500 mg/ m^3), 6 hours per day, 5 days per week, for 13 weeks. There were no differences between controls and treated rats with respect to total weights, weight change, food consumption, haematology, clinical chemistry, gross pathology or histopathology. Compared with the controls, the liver weights in several groups of exposed rats were significantly lower. There was, however, no dose response relationship for this weight reduction and the cause was unknown (CIIT 1977 cited in OECD 1998). Fisher 344 rats (10 per group per sex) were exposed to ethene via whole body inhalation at 0, 300, 1,000, 3,000 or 10,000 ppm (equivalent to 0, 345, 1150, 3450 or 11,500 mg/ m^3), 6 hours per day, 5 days per week, for 13 weeks. Very slight to moderate multifocal hyperplasia/hypertrophy of mucous secretory cells in nasal mucosa, accompanied by very slight to slight multifocal accumulations of eosinophils were observed and that appears to be treatment related, however, no dose-response relationship data were reported (USEPA 2007). No oral and dermal studies were identified.
Chronic toxicity/ carcinogenicity	**Inhalation carcinogenicity in rats:** Fisher 344 rats, 120 per group per sex were exposed to ethene via whole-body inhalation, 6 hours per day, and 5 days per week, at 0, 300, 1,000, or 3,000 ppm (equivalent to 0, 345, 1150 or 3450 mg /m^3) for up to 24 months. There were observations of hair loss, dry and dark deposits on and around the nose and eyes and gross eye abnormalities, but there were no obvious differences among the different treatment groups. There was an overall increase in the number of animals exhibiting gross tissue masses for the test groups as compared with the control group, but this trend was not statistically significant. The spontaneous mortality (15.7 %) was roughly equal in all treated groups. The final body weights and total weight changes for treated males were higher than those in the control groups, but no dose-related pattern was seen. There were no statistically significant

Endpoints	Lowest effect levels[1]/Results
	differences among any of the treatment groups on any of the haematology, blood chemistry or other parameters investigated. No gross or histopathologic tissue changes attributable to the effects of the test material were observed in any of the treated rats (CIIT 1979, Hamm et al. 1984). No oral and dermal studies were identified.
Reproductive/ developmental toxicity	**NOEC** = 5,750 mg/m^3 (5,000 ppm). Rats (10 per sex per group) were exposed to ethene via head only inhalation, 6 hours per day, at 0 (air only), 200, 1,000 or 5,000 ppm (correspond to 0, 230, 1,150 or 5,750 mg/m^3). Ethene was given to parent animals for 2 weeks prior to mating, during the mating period, until the day prior to necropsy for the males (minimum 28 days), and until day 20 of gestation for the females. The females were allowed to litter and rear their offspring to day 4 post-partum when they and their offspring were killed. There were no deaths attributable to the exposure. No effects on body weight gain, fertility or fecundity were observed. Litter size, sex ratio, mean pup weight and pup growth and clinical condition were not adversely affected by the treatment. Necropsy revealed no macroscopic finding, any suggestion of toxicity due to the treatment. No toxic effects on the testis were observed (Aveyard et al. 1996 cited in OECD 1998). No oral and dermal studies were identified.
Genotoxicity and related endpoints: *in vivo*	**Mutagenicity** **Negative:** Male Fisher 344 rats, 7 per group, were exposed to ethene via whole body inhalation at 0, 40, 1000 or 3000 ppm, 6 hour per day, 5 days per week, for 4 weeks. No increases in *Hprt* frequency in splenic T cells in exposed rats were observed (Walker et al. 2000). **Negative:** Male B6C3F1 mice, 7 per group, were exposed to ethene via whole body inhalation at 0, 40, 1000 or 3000 ppm, 6 hour per day, 5 days per week, for 4 weeks. No increases in *Hprt* frequency in splenic T cells in exposed rats were observed (Walker et al. 2000). **Micronucleus** **Negative:** Male Fischer 344 rats (10 per group) were exposed to ethene via inhalation, 6 hours per day, 5 days per week, at 0, 40, 1,000 or 3,000 ppm for 4 weeks. A positive control group was treated with ethylene oxide at 200 ppm under the same exposure conditions. Bone marrow was collected approximately 24 hours after the final exposure. Ethene did not produce statistically significant exposure related increase in the frequency of

Endpoints	Lowest effect levels[1]/Results
	micronucleated polychromatic erythrocytes in the bone marrow of rats while ethene oxide exposure resulted in significant increase in the frequencies in rats (Vergnes and Pritts 1994). **Negative:** Male B6C3F1 mice (10 per group) were exposed to ethene via inhalation, 6 hours per day, 5 days per week, at 0, 40, 1,000 or 3,000ppm for 4 weeks. A positive control group was treated with ethylene oxide at 200 ppm under the same exposure conditions. Bone marrow was collected approximately 24 hours after the final exposure. Ethene did not produce statistically significant exposure related increase in the frequency of micronucleated polychromatic erythrocytes in the bone marrow of mice while ethylene oxide exposure resulted in significant increase in the frequencies in mice (Vergnes and Pritts 1994). **DNA alkylation** **Positive:** Male Fisher 344 rats, 7 per group, were exposed to ethene via whole body inhalation at 0, 40, 1000 or 3000 ppm, 6 hour per day, 5 days per week, for 4 weeks. A significant increase ($p<0.05$) in N7-(2-hydroxyethyl) guanine (N7-HEG) (major DNA adduct of ethylene oxide) was observed in rats in all tissues evaluated (liver, spleen, brain, and lung). The greatest increase in N7-HEG occurred during the first week of exposure and accumulated more slowly and approached a steady state between one and four weeks of exposure with the greatest concentration increase occurring in the liver. (Walker et al. 2000). **Positive:** Male B6C3F1 mice, 7 per group, were exposed to ethene via whole body inhalation at 0, 40, 1000 or 3000 ppm, 6 hour per day, 5 days per week, for 4 weeks. A significant increase ($p<0.05$) in N7-(2-hydroxyethyl) guanine (N7-HEG) (major DNA adduct of ethylene oxide) was observed in mice in all tissues evaluated (liver, spleen, brain, and lung). The greatest increase in N7-HEG occurred during the first week of exposure and accumulated more slowly and approached a steady state between one and four weeks of exposure with the greatest concentration increase occurring in the liver. (Walker et al. 2000). **Positive:** Male F344 rats, 8 per group, were exposed via whole body inhalation to ethene at 40 or 3000 ppm (equivalent to 46 or 3442 mg/m^3), or to ethylene oxide at 100 ppm (equivalent to 115 mg/m^3), 6 hours per day, 5 days per week, for up to 20 days. Increases in N7-HEG adducts were observed in ethylene exposed rats, but much lower compared to exposure with ethylene oxide (Rusyn et al. 2005).

Endpoints	Lowest effect levels[1]/Results
Genotoxicity and related endpoints: *in vitro*	**Mutagenicity in bacteria** **Negative** in *Salmonella typhimurium* TA100 with or without metabolic activation (Victorin 1988). **Negative** in *Escherichia coli* (Landry and Fuerst 1968 cited in OECD 1998). **Chromosomal aberration** **Negative** in Chinese hamster ovary (CHO) cells with or without metabolic activation (Riley 1996 cited in OECD 1998).

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

Table B-2: Studies in humans

Endpoints	Results
Epidemiology studie	A study was carried out among workers at a Swedish petrochemical plant using measurements of haemoglobin adducts formed from ethylene oxide for monitoring of ethene exposure. The study was carried out in two parts, part one in 1989 and part two in 1993. Eight workers exposed to high levels of ethene (4 mg/m^3) and 3 workers exposed to low levels (0.1 -0.3 mg/m^3) were compared to nine controls exposed to 0.01 mg/m^3. All exposed workers showed elevated levels of haemoglobin adducts and adduct formation were dose-related. The results indicated that about 1 % of the inhaled ethene was metabolized to ethylene oxide. The second part of the study, which included four workers, was designed to more accurately determine the exposure levels, which turned out to have a mean of 4.5 mg/m^3. The results confirmed part one, showing that about 1 % of inhaled ethene was metabolized to ethylene oxide and the maximum fraction to be converted was estimated to be 4 % (Tornqvist 1994 cited in OECD 1998). A nested case-control study found no increase in lung cancer incidence in 31 workers exposed to ethene (at unspecified levels) at a US petrochemical factory (Bond et al.1986).