Environment Canada Environnement Canada

Screening Assessment for

1-Propene

Chemical Abstracts Service Registry Number 115-07-1

Environment Canada
Health Canada

September 2014

Canada

Cat. No.: En14-198/2014E-PDF
ISBN 978-1-100-24874-5

Information contained in this publication or product may be reproduced, in part or in whole, and by any means, for personal or public non-commercial purposes, without charge or further permission, unless otherwise specified.

You are asked to:

- Exercise due diligence in ensuring the accuracy of the materials reproduced;
- Indicate both the complete title of the materials reproduced, as well as the author organization; and
- Indicate that the reproduction is a copy of an official work that is published by the Government of Canada and that the reproduction has not been produced in affiliation with or with the endorsement of the Government of Canada.

Commercial reproduction and distribution is prohibited except with written permission from the author. For more information, please contact Environment Canada's Inquiry Centre at 1-800-668-6767 (in Canada only) or 819-997-2800 or email to enviroinfo@ec.gc.ca.

© Her Majesty the Queen in Right of Canada, represented by the Minister of the Environment, 2014.

Aussi disponible en français

Synopsis

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), the Ministers of the Environment and of Health have conducted a screening assessment of 1-propene, Chemical Abstracts Service Registry Number 115-07-1. 1-Propene (henceforth referred to as propene) was identified as a priority for screening assessment as it was considered to pose greatest potential for exposure of individuals in Canada and was considered to pose a moderate hazard to human health.

Propene is a naturally occurring gas that is emitted from many plants, and is a component in natural gas, volcanoes, and from incomplete biomass combustion. Propene is primarily used as a monomer for the production of polypropylene, a plastic. It can also serve as an intermediate to make many other plastics, as a fuel additive, as a fragrance or as a perfume ingredient. Based on submissions made under Section 71 of CEPA 1999, companies reported manufacturing a total of 930 000 tonnes of propene in Canada in 2000, mostly by the petrochemical industry. During the same year, over 10 000 tonnes of propene were reported as imported into Canada.

The National Pollutant Release Inventory reported that in 2009, a total of 404 tonnes of propene were released in Canada. There is an overall declining trend in reported releases from 1994 to 2009, due in part to closures of several chemical manufacturing facilities in 2008 and 2009.

Automobiles manufactured prior to 1992 are estimated to be a major source of propene in air. In 2005, these automobiles constituted 14% of all Canadian light-duty vehicles on the road but they contributed 76% of all propene releases from these vehicles. However, the amount of all volatile organic compounds, including propene, released by automobiles has been declining due to improved efficiency of automotive engines and the continual removal of older vehicles from usage.

Propene has been detected in outdoor, indoor and personal air. It has not been reported in surface water, drinking water, soil, sediment, consumer products or foodstuffs in Canada. Propene has been identified as a combustion by-product in cigarette smoke.

Based on its physical and chemical properties and modelled data, propene is not persistent or bioaccumulative. Propene does not appear to cause harmful effects to terrestrial plants or small mammals even when they are exposed to very high concentrations in air. No studies have been found on potential effects of propene on aquatic organisms.

Releases of propene to the environment occur almost exclusively to air. Based on a conservative risk quotient analysis, air concentrations of propene in Canada

are not expected to cause harmful effects to small mammals and terrestrial plants.

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 concluded that this substance does not meet the criteria under paragraph 64(*a*) or 64(*b*) of CEPA 1999 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.

Propene has been classified by the International Agency for Research on Cancer as “not classifiable as to its carcinogenicity to humans (Group 3)” on the basis of inadequate evidence of carcinogenicity (IARC 1994). The animal and human health effects database for propene did not demonstrate evidence of carcinogenicity and the available information on genotoxicity indicates that propene is not likely to be genotoxic. With respect to non-cancer effects, the lowest observed adverse effect concentration (LOAEC) for chronic exposures was 5000 ppm (8600 mg/m^3), based on significantly increased incidence of squamous metaplasia and inflammation in the nasal cavities of rats exposed for 2 years. Margins of exposure between effect levels and upper-bounding estimates of exposure are considered adequate to address uncertainties related to health effects and exposure.

On the basis of the adequacy of the margins between the upper-bounding estimates of exposure and the critical effect level for chronic exposure, it is concluded that propene does not meet the criteria under paragraph 64(*c*) of CEPA 1999 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

Based on available information for ecological and human health considerations, it is concluded that propene does not meet any of the criteria set out in section 64 of CEPA 1999.

Table of Contents

List of Tables

1 Introduction

This screening assessment report was conducted pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999). This section of the Act requires that the Ministers of the Environment and of Health conduct screening assessments of substances that have met the categorization criteria set out in the Act to determine whether these substances present or may present a risk to the environment or to human health.

A screening assessment was undertaken on 1-propene (Chemical Abstracts Service Registry Number 115-07-1, henceforth referred to as propene), a substance on the Domestic Substances List (DSL). Propene was identified as a priority for assessment during the categorization of the DSL, as it was determined to present the greatest potential for exposure of individuals in Canada, and was considered to present a moderate hazard to human health. Propene did not meet the categorization criteria for persistence, bioaccumulation or inherent toxicity to aquatic organisms.

Screening assessments focus on information critical to determining whether a substance meets the criteria as set out in section 64 of CEPA 1999 (Canada 1999). 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 propene. Data relevant to the screening assessment of propene were identified in original literature, review, and assessment documents, commercial and government databases and indices and from recent literature searches, up to April 2012 for both ecological and human health sections. Air monitoring data from the National Air Pollution Surveillance (NAPS) Network were also obtained (Environment Canada 2011). In addition, an industry survey on propene was conducted for the year 2000 through a *Canada Gazette* notice issued pursuant to Section 71 of CEPA 1999 (Canada 2001). This survey collected data on the Canadian manufacture, import, uses and releases of propene (Environment Canada 2003).

Original studies that form the basis for determining whether the substance meets the criteria set out in paragraphs 64(*a*) and 64(*b*) of CEPA 1999 have been

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

critically evaluated by staff of Environment Canada to ensure reliability of the data. The data from key toxicity studies were evaluated using Robust Study Summary forms similar to those recommended by the Organisation for Economic Co-operation and Development for the evaluation of studies for the Screening Information Data Set of high production volume substances (OECD 2003). Only studies with a score showing an appropriate degree of confidence were taken into account when selecting critical values for the assessment.

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

This screening assessment was prepared by staff in the Existing Substances Programs at Health Canada and Environment Canada and incorporates input from other programs within these departments. The environment and human health sections of this screening assessment have undergone external written peer review/consultation. 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 Canada.

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

2 Substance Identity

For the purposes of this document, the substance will be referred to as propene as it is in accordance with the most recent IUPAC (International Union of Pure and Applied Chemistry) recommendations for this substance. Information relevant to the identity of propene is presented in Table 2-1.

Table 2-1: Substance identity for propene

Chemical Abstracts Service Registry Number (CAS RN)	115-07-1
DSL name	**1-Propene**
National Chemical Inventories (NCI) names[a]	*Propene (TSCA, AICS, ECL, SWISS, PICCS, ASIA-PICS, NZIoC)* *Propylene (EINECS, ENCS, PICCS)*
Other names	1-propene, 1-propylene, methylethylene, methylethene
Chemical group **(DSL Stream)**	Discrete organics
Major chemical class or use	Organic
Chemical formula	C_3H_6
Chemical structure	**H_2C=CH-CH_3**
SMILES[b]	C(=C)C
Molecular mass	42.08 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.

3 Physical and Chemical Properties

At temperatures above -48°C, propene is a colourless gas, and is heavier than air (AIHA 1989). Table 3-1 presents a range of physical-chemical properties identified for propene that are relevant to its environmental fate. All of these properties were determined experimentally.

Table 3-1: Physical and Chemical Properties of Propene

Property	Type	Value	Temperature (°C)	Reference
Melting point (°C)	Experimental	-185	Not applicable	O'Neil et al.

				2001
Boiling point (°C)	Experimental	-48	Not applicable	O'Neil et al. 2001
Vapour density (kg/m^3)	Experimental	1.81	20	O'Neil et al. 2001
Vapour density (relative to air)	Experimental	1.49 (air=1)	20	O'Neil et al. 2001
Vapour pressure (kPa)	Experimental	779	10	Braker and Mossman 1980
Vapour pressure (kPa)	Experimental	1158	25	Inchem 2001
Vapour pressure (kPa)	Experimental	2531	50	Braker and Mossman 1980
Solubility in water (mg/L)	Experimental	200	25	McAuliffe 1966
Henry's Law constant	Experimental	1985 $Pa/m^3/mol$ (0.0196 atm m^3/mol)	25	Wasik and Tsang 1970
Octanol-water partition coefficient log K_{ow} (dimensionless)	Experimental	1.77	Not applicable	Inchem 2001
Rate constant for gas-phase reaction with hydroxyl radical (k_{OH}) (cm^3/molecule/ sec)	Experimental	2.60×10^{-11}	Not applicable	Atkinson et al. 1989

4 Sources

Propene is found to be a naturally occurring product in vegetation, especially bananas and apples and is released from many types of plants (Clayton and Clayton 1981; Isidorov et al. 1985). During the combustion of organic matter (i.e., from auto/aircraft exhaust, tobacco smoke or biomass burning) propene is released into the ambient air. In addition, propene may be released during the production and use of other products for which it is a chemical intermediate. Propene is most commonly obtained from petroleum refining through thermal cracking, where a mixture of hydrocarbons is heated and undergoes reactions with free radicals, which results in effluent separation. This produces gasoline and fuel oil in addition to the propene (Speight 2007; Mark et al. 1978).

Based on submissions made under Section 71 of CEPA 1999, 11 companies reported manufacturing propene in 2000, with a total production of 930 000 tonnes, with most manufacturing done by the petrochemical industry. Over 10 000 tonnes of propene were imported in 2000 (Environment Canada 2003).

More recent data from Statistics Canada indicate that Canadian production of propene has decreased when compared to the industry submissions from 2000.

In 2008, 770 672 tonnes of propene was produced, followed by 590 623 tonnes in 2009 and 660 474 tonnes in 2010. No recent information on the number of companies manufacturing propene was found (Statistics Canada 2011).

5 Uses

Propene is primarily used as a monomer for the production of polypropylene, a plastic. It can also serve as an intermediate to make acrylonitrile, propene oxide, isopropyl alcohol, cumene, butyraldehydes, propene oligomeres, acrolein, allyl chloride, allyl acetate, cresols, ethylene-propene rubbers, ethene and butanes (Speight 2007). Propene can be used as a fuel additive or fragrance, or a perfume deodorizer (HSDB 2003). If dimerized or alkylated, propene is used to produce polymer gasoline for gasoline blending (Mark et al. 1978; Marchionna et al. 2001).

Propene is not permitted for use as a food additive in Canada and is not found in the Lists of Permitted Food Additives and their associated Marketing Authorizations, issued under the authority of the *Food and Drugs Act* (April 2014 email from HPFB, Health Canada to Risk Management Bureau, Health Canada, unreferenced). Propene is not listed in the Drug Product Database (DPD), the Therapeutic Products Directorate's internal Non-Medicinal Ingredient Database, the Natural Health Products Ingredients Database or the Licensed Natural Health Products Database as a medicinal or a non-medicinal ingredient present in final pharmaceutical products, natural health products or veterinary drugs (DPD 2012; NHPID 2012; LNHPD 2012; February 2011 email from Therapeutic Products Directorate, Natural Health Products Directorate and Veterinary Drugs Directorate, Health Canada, to Risk Management Bureau, Health Canada; unreferenced).

In Canada, the majority of companies used propene in a destructive manner, typically as a feedstock to make other substances or as a fuel. Other reported uses were in the production of olefins and in other chemical manufacturing (Environment Canada 2003).

6 Releases to the Environment

Propene is a gas that is naturally emitted from garlic essential oil, European fir and Scots pine, and is released from germinating beans, corn, cotton and pea seeds (Isidorov et al. 1985). Natural gas, volcanoes, and incomplete biomass combustion release considerable quantities of propene into the atmosphere (HSDB 2003). Some soil microorganisms, such as the cyanobacteria *Oscillatoria* spp. and *Nostoc* spp. also release it (Hodges and Campbell 1998).

The National Pollutant Release Inventory (NPRI) (Environment Canada 2010) reported that a total of 404 tonnes of propene were released from 44 facilities in 2009. A generally declining trend in releases is observed when comparing

releases in recent years to totals from 1994-2009 Some of the recent reductions were due to closures of several chemical manufacturing facilities in 2008 and 2009.

The sectoral distribution of the releases for 2009 is shown in Table 6-1. Virtually all releases (>99%) are to air as propene is a gas under normal ambient temperatures and pressures. The amount of propene reportedly released by the oil and gas industry is likely underestimated; some gas processing facilities are not required to report to the NPRI as they do not meet NPRI reporting criteria.

Table 6-1: Quantities released as reported to the NPRI for 2009

Sector	Quantity Released (tonnes)
Oil sands and heavy oil	160
Petroleum extraction and refining	126
Chemical and chemical products	59
Primary metal manufacturing	32
Other manufacturing	21
Iron and steel	12
Oil and gas pipelines and storage	4
Upstream oil & gas	3.1
Mining	2
Pulp and paper	0.003
Transportation equipment manufacturing	0.002
Total	419.1

Propene 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 2003). 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.

In the AirCare program, the quantity of propene emitted per kilometre driven varied greatly; however, a general trend was apparent: vehicles older than 1992 often emitted considerably more propene than did vehicles newer than 1992. The average propene emission rate for pre-1992 vehicles was 50.19 mg/km driven, while the average for 1992 and later vehicles was 1.60 mg/km driven. This change was largely the result of emissions controls and requirements for cleaner-burning fuels in the United States and Canada.

These data can be used to estimate the amount of propene potentially released from motor vehicles in Canada, assuming that the vehicle distribution for Canada

is similar to that of the Lower Fraser Valley. In 2005, Canadians drove an estimated 287.7 billion km in 17.9–18.2 million in light-duty vehicles (weighing less than 4.5 tonnes) (Statistics Canada 2005; NRCan 2008). This calculation resulted in a total estimated release of propene for Canada from light-duty vehicles at 1774 tonnes in 2005. The majority of this, 1345 tonnes (76% of the total), was from cars older than 1992 although they represented only 14% of the fleet in 2005 (NRCan 2008). As of 2009 this age class of vehicle represented only 6.7% of the vehicle fleet (Statistics Canada 2010). As the Canadian vehicle fleet ages the proportion of vehicles older than 1992 will continue to drop resulting in substantial decreases in the amount of propene released by Canadian vehicles.

Environment Canada's national emission trends for criteria air contaminants indicates that the release of all volatile organic compounds (VOCs), including propene, from vehicles has dropped from 1 million tonnes in 1985 to 491 000 tonnes in 2010 despite an increase in the national light vehicle fleet (19.7 million) and total kilometres driven (303 billion km) (Environment Canada 2012; Statistics Canada 2010).

Current vehicle regulation such as the *On-Road Vehicle and Engine Emission Regulations* of CEPA 1999 (Canada 2003) contribute to reducing vehicular emissions.

7 Environmental Fate

As virtually all of the propene released in Canada is to air and propene is a gas at environmental temperatures, the main focus for this assessment will be on the air compartment. Results of Level III fugacity modelling with the Equilibrium Criterion (EQC) Model (Mackay et al. 2003), using average half-lives reported by Howard et al. (1991), indicate that when released to air, 99.99 percent of propene is expected to partition to air and that only negligible amounts will partition to soil, water, or sediment.

If released to surface water, propene will readily evaporate as it has a high Henry's Law constant of 1985 $Pa/m^3/mol$.

7.1 Persistence and Bioaccumulation Potential

Modelled data concerning the biodegradation and persistence of propene in different environmental media are presented in Table 7-1. Modelled biodegradation values for propene indicate that the half-life for propene is 17.5 days in water and soil (Howard et al. 1991).

Propene reacts in air primarily with hydroxyl radicals (OH•) but it can also react with nitrate ions (NO_3), ozone (O_3), and O(^{3}P) atoms—oxygen atoms in the "triplet P" state, an excited form of oxygen (Atkinson 1989). The hydroxyl radical

rate constant for propene is 2.60 x 10^{-11} cm^3/molecule/sec, indicating that propene reacts quickly in the atmosphere with OH• (Atkinson et al. 1997). Rate constants for reactions with NO_3, O_3 and $O(^3P)$ are 9.73 x 10^{-15}, 1.05 x 10^{-17} and 4.01 x 10^{-12} cm^3/molecule/sec, respectively (Atkinson et al. 1997). These rate constants indicate that propene will react much more slowly with NO_3, O_3 and $O(^3P)$ than with OH•.

Propene is a precursor of tropospheric ozone. Under non-polluted air conditions O_3 is formed photo-chemically from the photolysis of NO_2, resulting in a photo-equilibrium between NO, NO_2 and O_3, with no net formation or loss of O_3 (Atkinson 2000). When compounds such as propene are present and undergo degradation reactions, the intermediate organic peroxy radicals ($R\dot{O}_2$) and HO_2 radicals that are formed react with NO, converting it to NO_2 which then photolyzes to form O_3 resulting in the net formation of O_3.

Assuming an initial hydroxyl ion concentration of 1.5 x 10^6 molecules/cm^3, the tropospheric half-life of propene was calculated to be 4.9 hours in a normalized 12-hour day (AOPWIN 2008). The half-life of propene in air calculated by Howard et al. (1991) was between 1.7 and 13.7 hours, based upon a measured photo-oxidation rate of 2.60 x 10^{-11} cm^3/molecule/sec in air. Table 7-1 summarizes the half-life of propene in various media.

Table 7-1: Environmental half-lives and removal processes of propene in different media

Medium	Half-life (average)	Removal Process	References
Soil	17.5 days	Biotic degradation	Howard et al. 1991
Air	4.9–7.7 hours	OH• radical reactions	Howard et al. 1991
Air	4.9 hours	OH• radical reactions	EPISuite 2008
Surface water	17.5 days	Biotic degradation	Howard et al. 1991
Ground water	35 days	Biotic degradation	Howard et al. 1991

Propene has an expected reactive half-life of 5-8 hours in air and an estimated biodegradation half-life of 17.5 days in water and soil (Table 7-1). The half-lives in sediment can be extrapolated from the half-life estimations in water and soil using an extrapolation ratio of 1:1:4 for water:soil:sediment biodegradation half-lives (Boethling et al. 1995). Therefore, the half-life of propene in sediment is 70 days.

Bioaccumulation and bioconcentration factors were not found for propene; however it is expected that bioaccumulation and bioconcentration of propene in the aquatic system is limited due to its high volatility, short half-life, moderate water solubility and low density relative to water (HSDB 2003). When bioaccumulation and bioconcentration factors are lacking, the log K_{ow} of a substance may be used to determine its bioaccumulation potential. Propene has a log K_{ow} of 1.77 (Inchem 2001), indicating that is not likely to bioaccumulate.

8 Potential to Cause Ecological Harm

No monitoring data on propene in water or soil were found.

8.1 Ecological Exposure Assessment

Four- and 24-hour concentrations of propene were measured at 62 Canadian sites between January 2005 and December 2009 as part of the Environment Canada National Air Pollutant Surveillance (NAPS) air monitoring network (Environment Canada 2011). Edmonton, Montreal, Toronto and Vancouver each had several monitoring sites, while the other sites were located in smaller cities and towns, rural areas or parks. Sites were classified as urban, rural or remote, and minimum, maximum and average values were calculated for each of these groups (Table 8-1). The mean concentration of all 62 sites was 0.67µg/m^3 (Environment Canada 2011). The sites that had the highest concentrations of propene were Saint John, New Brunswick (maximum of 104.14µg/m^3 in 2006) and Oakville, Ontario (maximum of 104.20µg/m^3 in 2005).

Table 8-1: Summary of monitored monthly air concentrations of propene at NAPS sites from 2005-2009

Site Type	Number of Sites	Number of Samples	Minimum (µg/m^3)	Maximum (µg/m^3)	Mean (µg/m^3)
Remote sites	4	1198	0.01	0.66	0.07
Rural sites	13	4265	0.03	6.43	0.19
Urban sites	45	6827	0.05	104.20	0.90

There was a noticeable seasonal variation observed for atmospheric propene, with relatively low concentrations found during the summer months and higher concentrations during the winter months. The seasonal cycling was not always consistent and tended to be disrupted in areas with greater numbers of industrial propene point sources. This may be due to the more rapid destruction of propene in air through photochemical reactions due to longer day length in summer.

Environment Canada and the Fort Air Partnership conducted an air monitoring program that included propene near a chemical manufacturing area in Fort Saskatchewan, Alberta and surrounding locations. Air samples were taken over 24 hours from 10 sites once every 6 days between September 2004 and October 2006. Elk Island National Park, which was used as a background site for the Fort Saskatchewan sites, had the lowest minimum (0.04 µg/m^3), maximum (0.56 µg/m^3) and mean concentrations (0.17 µg/m^3). The concentrations of propene in the Fort Saskatchewan area ranged from 0.04 to 4.03 µg/m^3, and the highest mean concentration among these sites was 0.51 µg/m^3.

A similar monitoring program was undertaken near an industrial area in North Vancouver, British Columbia. Concentrations in North Vancouver ranged from 0.28 to 6.25 µg/m^3, with the highest mean concentration among the four sites being 1.75 µg/m^3 (Englot 2006). The North Vancouver site is located in the vicinity of chemical plants, concrete and mineral products industries, marine cargo, transportation and ship building/repair shops, and metal fabricating plants (Environment Canada 2010).

A separate four-week study was conducted by the Air Quality Research Branch and the Canadian Meteorological Centre of Environment Canada in central Alberta. Data was collected from existing air quality stations and with aircraft-mounted instruments. In the summer of 2005 propene concentrations were all below 0.2 µg/m^3 (Wiens 2005).

8.2 Ecological Effects Characterization

No studies were found on the potential effects of propene on aquatic organisms. Given that propene is unlikely to be found in any media other than air, only exposure to the air compartment is considered here.

No evidence of toxic effects was found in studies investigating the effect of propene on terrestrial plants. Propene promotes fruit ripening at concentrations of 172–344 mg/m^3 (Nanos et al. 2002), and accelerates fruit softening in apricots (Cardarelli et al. 2002) and bananas (Golding et al. 1999). Concentrations as high as 8600 mg/m^3 for up to 10 days did not appear to have any negative effects on strawberries (Perkins-Veazie et al. 1996). The concentration 8600 mg/m^3 is a no observed adverse effect concentration (NOAEC) and will be used as the critical toxicity value (CTV) for plants.

A concentration of 111 800 mg/m^3 caused no death or hepatotoxicity in Sprague-Dawley rats exposed to 0 or 111 800 mg/m^3 propene for 4 hours (Conolly and Osimitz 1981). The inhalation LC_{50} in rats was not reached at 111 800 mg/m^3 (6.5%) over 4 hours (Nova 2010). Short-term inhalation of 40 % propene led to slight anaesthesia but no toxic effects in rats (Browning 1987). A repeated dose of 17 200 mg/m^3 caused no mortality, morbidity, weight change, or other compound related effects in a 2-week mouse study and a 2-week rat study (NTP

1985). The value 111 800 mg/m^3 is a NOAEC and will be used as the CTV for mammals (Conolly and Osimitz 1981). A summary of further data on the effects of propene on mammals can be found in Appendix A.

8.3 Ecological Risk Characterization

The approach taken in this ecological screening assessment was to examine various supporting information and develop conclusions based on a weight-of-evidence approach as required under Section 76.1 of CEPA 1999. Particular consideration has been given to sources, releases, occurrence in the environment, persistence, bioaccumulation and risk quotient analyses.

Propene is not persistent or bioaccumulative. For the risk quotient analysis, an analysis of exposure pathways and subsequent identification of sensitive receptors is first done to select relevant ecological assessment endpoints. Canadian environmental concentrations are used preferentially as predicted environmental concentrations (PECs). PECs were selected to represent reasonable worse-case scenarios, as an indication of the potential for these substances to reach concentrations of concern and to identify areas where those concerns would be most likely. A predicted no-effect concentration (PNEC) was determined by dividing a CTV by an assessment factor. CTVs typically represented the lowest ecotoxicity value from an available and acceptable data set.

For propene, releases to the environment occur predominantly to air (>99%) and it is mainly found in air, so the risk quotient analysis focuses on air exposure scenarios. For these scenarios, the maximum monthly mean air concentration of propene recorded at the Oakville, Ontario, NAPS air monitoring site in 2005, 104.20 µg/m^3, was selected as a worst-case value for the PEC.

Empirical acute toxicity data (NOAECs) were used to select a CTV for mammalian and plant receptors. The PNEC for mammals was derived by dividing the CTV (the 4 hour NOAEC of 111 800 mg/m^3, the concentration of propene that produced no death or hepatotoxicity (Conolly and Osimitz 1981)) by an assessment factor of 100 to account for lab to field exposures, for extrapolation from rats to other species, and extrapolation from acute to chronic exposure. The PNEC for plants was derived by dividing the CTV (the NOAEC of 8600 mg/m^3; the lowest concentration where no effect was observed on strawberries after 10 days (Perkins-Veazie et al. 1996)) by an assessment factor of 100 to account for lab to field exposures, and the general lack of data.

A risk quotient (PEC/PNEC) was calculated for each of the endpoint organisms (mammals and plants) in order to determine likely current ecological risk in Canada. A summary of data used in the risk quotient analysis of propene is presented in Table 8-2. Risk quotients greatly less than 1 indicate that propene concentrations found in Canada are unlikely to pose a risk to various ecological

components. The exposure scenarios presented here are conservative and reflect worst case scenarios in terms of plant and animal sensitivity.

Table 8-2: Summary of data used in risk quotient analyses of propene

Medium	Organism	PEC (mg/m^3 or mg/L)	CTV (mg/m^3 or mg/L)	Assessment Factor	PNEC (mg/m^3 or mg/L)	Risk Quotient
Air	Plants	0.1042	8600	100	86	**0.001**
Air	Rats	0.1042	111800	100	1118	**9×10^{-5}**

Given that the primary sources of anthropogenic propene are vehicle and industrial emissions, it important to consider that both of these sources have decreasing emissions in Canada, thus reducing the potential for ecological effects. Propene emissions from vehicles have been declining due to improved efficiency of automotive engines, the continual removal of older vehicles from usage and current vehicle regulations (see releases section).

While it is noted that propene is a precursor of tropospheric ozone, the Government of Canada, through the *Air Quality Management System* (CCME 2012), is moving forward with the implementation of a number of measures for reducing emissions of nitrogen oxides (NOx) and VOCs (both ozone precursors) from key industrial sectors that are sources of propene emissions (including the Oil Sands, Petroleum Refining, Chemicals & Iron and Steel sectors).

Based on the information presented in this screening assessment, it is concluded that propene does not meet the criteria under paragraph 64(*a*) or 64(*b*) of CEPA 1999 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.

8.4 Uncertainties in the Evaluation of Ecological Risk

Confidence in the available data for propene exposure data is moderate to high. Air monitoring data from across Canada was available and this greatly reduces the uncertainty in the air exposure scenarios. These data provides both background concentrations from remote areas as well as concentrations in rural areas and in cities, thus allowing a distinction between natural and anthropogenic sources and releases. No data were available on the effects of propene in other compartments. However, releases and partitioning of propene to water, sediment and soil are expected to be negligible due to the physical and chemical properties of propene.

9 Potential to Cause Harm to Human Health

9.1 Exposure Assessment

9.1.1 Environmental Media

Propene is found naturally in vegetation, such as fruits, beans, corn and rice. Additionally it has been detected in the foliage of elm, ash, cypress and hackberry trees in the United States. Propene is also a product of incomplete combustion resulting from burning of fossil fuels. Given the high vapour pressure and low water solubility of propene, it is expected that the primary source of exposure would be from the air. In the literature, there are several propene monitoring studies from the United States and Europe, however, many are outdated. More recently, monitoring data from various provincial and national databases in Canada have measured the concentration of propene in ambient and indoor air which are listed in Tables 9-1, 9-2 and 9-3.

9.1.2 Outdoor Air

The Clean Air Strategic Alliance (CASA) in Alberta is a provincial air monitoring program which measures concentrations of various VOCs. Propene was passively monitored hourly from 4 sites between 2003 and 2007 giving a range of 0.02 – 13.8 µg/m^3. A maximum hourly concentration of 13.76 µg/m^3 was measured at a site in the Edmonton area, while the 99th percentile from all sites was 2.8 µg/m^3 (CASA 2010).

Propene is measured and reported as part of Environment Canada's National Air Pollution Surveillance (NAPS) program. Four and 24–hour propene concentrations were collected at 64 sites across Canada between January 2005 and December 2009. The monitoring sites ranged from rural to urban areas, dominated by residential (48) and commercial (12) sites, followed by undeveloped rural (3) and agriculture (1) sites. The lowest mean concentration of 0.03 ± 0.04 µg/m^3 was reported from an undeveloped rural site in Alert, Nunavut. The highest mean concentration of 5.7 ± 12.2 µg/m^3 was reported at the Forest Hills site, an urban residential area of Saint John, New Brunswick. Propene concentrations ranged from 0.013 to 104.2 µg/m^3 across all sites (Environment Canada 2011). While the highest concentration was reported from a residential area of Oakville, Ontario, the corresponding 95th percentile was reported at 13.7 µg/m^3. The highest 95th percentile of 23.8 µg/m^3 was measured instead at the Forest Hills site.

Propene was also measured in ambient and indoor air in three recent Canadian studies. Measurements were conducted between 2006 and 2011 and took place in Windsor, ON, Regina, SK, and Halifax, NS, as part of the Windsor Ontario Exposure Assessment Study (Health Canada 2010a), the Regina Indoor Air Quality Study (Health Canada 2010b) and the Halifax Indoor Air Quality Study (Health Canada 2011). The Windsor Ontario Exposure Assessment Study

(WOEAS), published in 2010, involved 45 to 48 homes and reported 188 VOCs in air samples (Health Canada 2010a). Twenty-four hour samples were collected during an 8 week period in the summer and winter seasons over a two year period from 2005 to 2006. Ambient air concentrations of propene in summer ranged from 0.2 – 2.2 $\mu g/m^3$. Concentrations in the winter were comparable ranging from 0.1 – 2.1 $\mu g/m^3$. The highest 95^{th} percentile of propene in ambient air was associated with the summer season with a value of 1.1 $\mu g/m^3$. However the winter 95^{th} percentile was 1.1 $\mu g/m^3$ suggesting that there is no strong seasonal trend.

The Regina Indoor Air Quality Study (RIAQS) also measured 194 VOCs in ambient air around residential areas in Regina, Saskatchewan (Health Canada 2010b). A total of 146 homes were sampled during 5 weeks in the summer and 10 weeks in the winter during 2007. Concentrations of propene ranged between 0.1 and 2.9 $\mu g/m^3$ over the year. Summer and winter air concentrations ranged from 0.1 – 0.8 $\mu g/m^3$ and 0.1 – 2.9 $\mu g/m^3$, respectively. Unlike the Windsor air study, the higher median (0.4 $\mu g/m^3$) and 95^{th} percentile of 1.8 $\mu g/m^3$ occurred in the winter.

More recently, Health Canada has published an additional air monitoring study conducted in Halifax, Nova Scotia. Data for the Halifax Indoor Air Quality Study were collected from 50 homes over the summer and winter of 2009. Propene concentrations in the summer ranged from 0.1 – 7.0 ug/m^3 and comparable values were observed in the winter, ranging from 0.045 – 6.50 $\mu g/m^3$. Similar to the Windsor study, the median concentration and 95^{th} percentile for the sites had minimal seasonal differences. For summer and winter samples, the 95^{th} percentiles were 0.8 $\mu g/m^3$ and 0.7 $\mu g/m^3$ respectively (Health Canada 2011).

In these exposure studies, there was no noticeable seasonal trend, with the exception of Regina, where propene was observed to have higher levels in the winter compared to the summer season. Several air monitoring studies in urban areas have observed higher concentration of VOCs in the winter versus the summer (Chang et al. 2005; Curren et al. 2006; Olsen et al. 2009; Matsunaga et al. 2010; Lai and Peng 2011), however, in the case of Windsor and Halifax, there is no seasonal trend and therefore is not substantiated by this urban evidence. The lack of obvious seasonality may be a result of the property of propene as a dense gas. There is no apparent increase or decrease in concentrations in the ground level mixing layer, as propene would be excluded from vertical transport, remaining close to the ground rather than in the upper atmosphere.

Propene was also studied as a component in motor vehicle exhaust and as a potential exposure for commuters, particularly cyclists (Weichenthal et al, 2011). The Weichenthal group recently measured a number of VOCs in outdoor samples in Ottawa, Ontario to determine the relationship between traffic pollution and acute changes in heart rate variability in cyclists. The study examined two outdoor scenarios, one in high traffic in the downtown core, and the other in low

traffic, along river valley bike routes. As expected the median of the high traffic propene concentrations (0.9 µg/m^3) was higher than the median concentration measured in the low traffic area (0.6 µg/m^3).

Table 9-1: Outdoor air concentrations of propene in Canada

Study	Area / Location	Sampling Period	Duration	n	Mean (Range) Conc. (µg/m^3)	Median (µg/m^3)	95th %
NAPS Study[a]	Residential / Forest Hills — Saint John, NB*	2005-2009	24-hr	209	5.7 (0.06-104.1)	1.4	23.8
NAPS Study[a]	Residential / Bronte Rd — Oakville, ON***	2005-2009	24-hr	93	3.4 (0.1-104.2)	0.5	13.7
NAPS Study[a]	Commercial / Prg Plaza — Prince George, BC	2005-2009	24-hr	166	0.9 (0.1-7.8)	0.6	2.8
NAPS Study[a]	Agricultural Rural / Experimental Farm - Simcoe, ON	2005-2009	24-hr	199	0.2 (0.04-0.9)	0.2	0.4
NAPS Study[a]	Undeveloped rural / Elk Island Nat'l Park- Elk Island, AB	2005-2009	24-hr	79	1.2 (0.04-1.1)	0.1	0.5
NAPS Study[a]	Undeveloped rural / Alert, NU**	2005-2009	24-hr	98	0.03 (0.0-0.3)	0.0	0.04
WOAES Study[b]	Windsor, ON	2005 Winter	N/A	201	0.5 (0.1-2.1)	0.5	1.1
WOAES Study[b]	Windsor, ON	2005 Summer	N/A	216	0.5 (0.2-2.2)	0.4	1.1
WOAES Study[b]	Windsor, ON	2006 Winter	N/A	213	0.4 (0.1-1.1)	0.4	0.8
WOAES Study[b]	Windsor, ON	2006 Summer	N/A	214	0.4 (0.2-1.3)	0.4	0.8
RIAQS Study[c]	Regina, SK	2007 Winter	24-hr	94	0.4 (0.1-2.9)	0.4	1.8
RIAQS Study[c]	Regina, SK	2007 Summer	24-hr	108	0.2 (0.1-0.8)	0.2	0.5
RIAQS Study[c]	Regina, SK	2007 Summer	5-day	97	0.3 (0.02-0.8)	0.3	0.5

Study	Area / Location	Sampling Period	Duration	n	Mean (Range) Conc. (µg/m^3)	Median (µg/m^3)	95th %
HIAQS Study[d]	Halifax, NS	2009 Winter	N/A	287	0.3 (MDL-6.5)	0.2	0.7
HIAQS Study[d]	Halifax, NS	2009 Summer	N/A	324	0.3 (0.1-7.0)	0.2	0.8
Other Studies[e]	Ottawa, ON	2010 High-traffic	1-hr	42	1.0 (0.2-3.0)	0.9	2.1
Other Studies[e]	Ottawa, ON	2010 Low-traffic	1-hr	42	0.3 (0.2-0.9)	0.6	0.8
Other Studies[f]	Edmonton and Calgary, AB	2003-2007	24-hr	N/A	0.02-13.8	N/A	2.8

N/A: Not Available
Acronyms : NAPS – National Air Pollution Surveillance 2005-2009, WOAES – Windsor Ontario Exposure Assessment Study, RIAQS – Regina Indoor Air Quality Study, HIAQS – Halifax Indoor Air Quality Study
[a] Environment Canada 2011b
[b] Health Canada 2010a
[c] Health Canada 2010b
[d] Health Canada 2011
[e] Weichenthal et al. 2011
[f] CASA 2010
* Highest 95th percentile
** Lowest 95th percentile
*** Highest maximum

9.1.3 Indoor Air

Recent exposure studies in Canada not only focussed on ambient air, but also measured indoor air from residential sites. A listing of indoor air concentrations can be found in Table 9-2.

Indoor air monitoring was conducted at homes in the same municipalities of Windsor, Regina and Halifax as those studies previously mentioned in the ambient air section. The air concentrations of the Windsor study ranged from 0.2 – 20.1 µg/m^3 overall (Health Canada 2010a). As seen with the ambient air data, there was minimal difference in the median indoor air concentrations between the seasons, 1.1 µg/m^3 and 1.0 µg/m^3 for summer and winter respectively. The highest 95th percentile of 3.9 µg/m^3 was measured during the summer; however with a 95th percentile of 3.2 µg/m^3 in the winter, there is a minimal seasonal association.

In the Regina study (Health Canada 2010b) indoor air concentrations ranged from 0.2 – 30.5 µg/m^3 over the course of the study. As seen in the exposure study, there was a seasonal difference in the median concentrations, 1.1 µg/m^3 in the winter and 0.6 µg/m^3 in the summer. The highest 95th percentile of 8.3 µg/m^3 was measured indoors during the winter season. Propene has been measured in smoke plumes from cigarettes in previous studies and this study conducts further investigations of the indoor air concentrations in smoking and non-smoking homes. The propene concentration range observed in non-smoking homes was

0.2 – 14.6 µg/m^3 across both seasons while higher concentrations were observed in homes with smokers, ranging between 0.4 – 30.5 µg/m^3.

The Halifax study (Health Canada 2011) also reported a large range in indoor air concentrations, however, this study showed no seasonal association. The concentration range in the summer was 0.1 – 135.8 µg/m^3 and 0.1 – 77.2 µg/m^3 in winter. Median concentrations between the two seasons are similar; however the highest 95th percentile of 18.3 µg/m^3 is in the winter. The 95th percentile measured in the summer months was 8.8 µg/m^3.

Weichenthal et al. (2011) also measured propene levels in indoor air as a part the study measuring indoor air pollution and heart rate variability in cyclists. Samples were collected from the indoor air to determine the exposure to propene of cyclists on stationary bikes. The median indoor concentration was 0.6 µg/m^3, which was between median concentrations from outside in high and low traffic. The 95th percentile during the indoor sampling period was 1.0 µg/m^3, expectedly lower than the outdoor high traffic scenario.

Table 9-2: Indoor air concentrations of propene in Canada

Study	Location	Sampling Period	Duration	n	Mean (Range) Conc. (µg/m^3)	Median (µg/m^3)	95th Percentile
WOAES Study[a]	Windsor, ON	2005 Winter	N/A	232	1.0 (0.2-20.1)	1.0	3.9
WOAES Study[a]	Windsor, ON	2005 Summer	N/A	217	1.2 (0.3-12.7)	1.1	3.2
WOAES Study[a]	Windsor, ON	2006 Winter	N/A	224	1.0 (0.2-7.0)	0.9	3.1
WOAES Study[a]	Windsor, ON	2006 Summer	N/A	211	1.2 (0.2-8.2)	1.1	4.0
RIAQS Study[b]	Regina, SK	2007 Winter	24-hr	105	1.4 (0.2-19.8)	1.1	8.3
RIAQS Study[b]	Regina, SK	2007 Winter	5-day	89	1.5 (0.3-17.9)	1.2	11.3
RIAQS Study[b]	Regina, SK	2007 Summer	24-hr	105	0.7 (0.2-30.5)	0.6	4.9
RIAQS Study[b]	Regina, SK	2007 Summer	5-day	101	0.8 (0.2-29.6)	0.7	6.4
HIAQS Study[c]	Halifax, NS	2009 Winter	N/A	312	1.0 (0.1-77.2)	0.7	18.3
HIAQS Study[c]	Halifax, NS	2009 Summer	N/A	331	0.6 (0.1-135.8)	0.5	8.8
Other Studies[d]	Ottawa, ON	2010 Summer	N/A	42	0.6 (0.3-1.0)	0.6	1.0

N/A: Not Available

Acronyms: WOAES – Windsor Ontario Exposure Assessment Study, RIAQS – Regina Indoor Air Quality Study, HIAQS – Halifax Indoor Air Quality Study

[a] Health Canada 2010a, Home

[b] Health Canada 2010b, Home with non-smokers only

[c] Health Canada 2011, Home

[d] Weichenthal et al. 2011, Office building

9.1.4 Personal Air

Personal air concentrations of propene were collected in the WOEAS (Health Canada 2010a) and are listed in Table 9-3. Selected participants wore backpacks equipped with sampling apparatus over 24-hour periods for five consecutive days to measure personal exposure to acetone in air. Participants were asked to wear the sampling equipment during the normal course of a day. The highest 95th percentile of 4.8 µg/m^3 was measured in the summer season (Health Canada 2010a). There was little difference found in the median concentrations of propene between summer and winter, reporting 1.10 µg/m^3 and 1.09 µg/m^3 respectively.

Table 9-3: Personal air concentrations of propene in Canada

Study	Location	Sampling Period	Duration	n	Mean (Range) Conc. (µg/m^3)	Median (µg/m^3)	95th Percentile
WOAES Study[a]	Windsor, ON	2005 Winter	N/A	225	1.1 (0.3-11.5)	1.1	2.8
WOAES Study[a]	Windsor, ON	2005 Summer	N/A	207	1.2 (0.5-9.8)	1.1	4.8

N/A: Not Available
Acronyms: WOAES – Windsor Ontario Exposure Assessment Study
[a] Health Canada 2010a

9.1.5 Water and Soil

Propene released to the environment is expected to remain in the air and not partition to soil or water. Empirical data on concentrations in water and soil in Canada were not identified.

9.1.6 Products

A survey conducted in 2000, pursuant to section 71 of CEPA 1999, reported the presence of propene as a component in fuels. Concentrations of propene were reported between 0 – 85% w/w in propene containing fuel products. The reported usage of fuels is intended for distributors or industry and not meant for use by the general population. No consumer uses were identified in the Household Product Database (HPD).

9.1.7 Tobacco Smoke

Propene is a component of cigarettes and tobacco smoke. In a study of organic compounds in cigarettes, the concentration of propene was found to be 0.18 mg/per smoke plume inhaled from an individual cigarette (Löfroth 1989). Median indoor propene concentrations in smoking and non-smoking households in the

Regina Indoor Air study were similar; however, smoking households had greater maximum concentrations in the summer and lower maximum concentrations in the winter (Health Canada 2010b).

9.1.8 Representative upper-bounding estimate of exposure

Given the high vapour pressure of propene, 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, indoor air concentrations were higher compared 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 percentiles concentrations for indoor and personal air of 18.3 and 4.8 µg/m^3 identified in the Halifax and Windsor studies (Health Canada 2010a, Health Canada 2011) are considered to be conservative upper-bounding air concentrations to which the general population of Canada is exposed.

9.1.9 Confidence in Exposure Assessment

Confidence in the exposure data of propene in environmental media is considered moderate to high. Ambient, indoor, and personal air monitoring data were available, and was both recent and representative of Canadian houses. Despite the lack of data for other environmental media, such as water and soil/sediment, the physical chemical properties of propene suggest that there would be minimal amounts present in other media. Confidence in the propene air concentrations is high given the conservative nature of the assessment.

9.2 Health Effects Assessment

Appendix A contains a summary of the available health effects information for propene.

The International Agency for Research on Cancer (IARC) has classified propene as a Group 3 carcinogen (not classifiable as to its carcinogenicity in humans). This classification was based on inadequate evidence of carcinogenicity in humans and experimental animals (IARC 1994).

Occupational cohort and case-control studies have been conducted in carpet factory workers and in polypropylene manufacturing workers to investigate the risk of developing colorectal cancer. The most relevant studies have been summarized in Appendix A. Although propene was likely to be handled in the different facilities, the risk cannot be related specifically to propene because no information was provided on workers' exposure. Multiple chemicals including propene were used in the facilities, and as such, specific chemical exposure could not be related specifically to colorectal cancer. However, a meta-analysis of

ten selected epidemiological studies found that the epidemiological evidence does not support a causal association between polypropylene production and colorectal cancer (summary risk ratio of 1.37 (95% Confidence Interval 0.83-2.11)). It was suggested that the positive associations reported in individual studies between polypropylene production and colorectal cancer (a common cancer) were largely driven by small clusters occurring in two plants. Excluding the original cluster, decreases the incidence to non–significant levels (Lagast et al. 1995).

Mammalian carcinogenicity studies with exposure to propene by inhalation have been conducted on both rats and mice. Although tumours were observed in some studies, the incidences were consistent with historical control data, or were not found to be statistically significant with respect to differences between exposed and control groups, thus limiting the significance of their occurrence. The studies are described below and are also presented in Appendix A.

In a chronic/carcinogenicity study in rats exposed by inhalation to 0, 5 000 and 10 000 ppm (0, 8 600 and 17 200 mg/m^3), C-cell neoplasms were observed in the thyroid in females. Negative trends were observed in adenomas and adenomas plus carcinomas, while positive trends were noted for hyperplasia. When all types of lesions (adenoma, carcinoma and hyperplasia) were combined, a positive trend was no longer apparent and the study authors concluded that these thyroid gland tumours were not due to propene exposure (NTP 1985; Quest et al. 1984). In a second chronic/carcinogenicity study, male and female rats were exposed to concentrations of 0, 200, 1 000 and 5 000 ppm (0, 344, 1 720, or 8 600 mg/m^3) propene for two years. No difference was reported in the incidences of tumours among the different groups, although the highest dose tested was lower than that employed in the NTP study (Ciliberti et al. 1988).

In a two-year study male and female mice were exposed to concentrations of 0, 5 000 or 10 000 ppm (0, 8 600 or 17 200 mg/m^3) propene. The appearance of tumours in the lungs of males, specifically the alveolar/bronchial region, was not likely to be exposure related. Exposed mice had lower incidence rates of alveolar/bronchial adenomas and carcinomas than control group animals. When the rates of adenoma and carcinoma were combined, the result was a negative trend in males. There was no significant difference in exposed and control females. The authors noted that the biological significance of this negative trend was hard to interpret, due to the incidence rates in the control and treatment groups, which fell into the historical control incidence range for this type of tumour. Observed decreased incidences of adenomas and carcinomas in the liver of mice were not considered to be propene related. Adenomas were observed in male mice only, while carcinomas were found in male and female animals. When the incidence rates for adenomas and carcinomas were combined for males, no significant difference was observed between the treatment groups and controls. Non-site specific hemangiosarcomas were observed in the subcutaneous tissue, spleen and uterus of females, while

hemangiomas were found in the liver. Combined hemangiosarcoma and hemangioma in females indicated a positive trend. However, incidences in the 10 000 ppm group were not significantly higher than in the control group. No significant difference between treated and control groups was noted for hemangiosarcomas observed in male mice. Due to the non-site specific nature of these neoplasms and the lack of higher occurrences of tumours in the 10 000 ppm group, the authors have suggested that these incidences were not related to propene exposure. Endometrial polyp incidence in the uterus produced a significant positive trend. The overall observed rate in the 10 000 ppm group was however not significantly different from the control group. Therefore, propene exposure was not associated with polyp formation (NTP 1985; Quest et al. 1984).

In a second chronic study, male and female mice were exposed to concentrations of 0, 200, 1 000, or 5 000 ppm (0, 344, 1 720, or 8 600 mg/m^3) propene, 7 hours per day, 5 days per week for 78 weeks. No difference was reported in the incidences of tumours among the different groups (Ciliberti et al. 1988).

In *in vitro* assays, propene was not mutagenic in bacterial mutation assays using *Salmonella typhimurium* strains TA97, TA98, TA100 and TA1537 *Escherichia coli* strains NP2uvrA/pKM101 and strain B and *Bacillus subtillis* strain Sd-4, with and without metabolic activation (Hugues et al. 1984; Inveresk Research 2003; Landry and Fuerst 1968; NTP 1989; Victorin and Stahlberg 1988). However, mutagenic activity was observed in *Salmonella. Typhimurium* strain TA1535 with or without activation (Inveresk Research 2003; NTP 1989). In a mouse lymphoma cell mutation assay, negative results were reported without metabolic activation and equivocal results were observed in presence of metabolic activation (McGregor et al. 1991).

Some *in vivo* studies where propene was administered via the inhalation route have been identified in the literature. Propene was not found to produce an increase in *hprt* mutant frequencies in splenic T-lymphocytes and an increase in micronucleated polychromatic erythrocytes in a short-term study in male rats (Dupont 2002a; Pottenger et al. 2007; Walker et al. 2004). Negative results were also observed in a sex linked recessive lethal mutation assay in *male Drosophila melanogaster* (Foureman et al. 1994). However, positive results for DNA adducts were reported in liver, spleen, lung and lymphocytes in rats (Eide et al. 1995; Pottenger et al. 2007). Similar results were observed in liver, kidney and spleen tissue in mice but it has been noted by the IARC working group that the findings of this study were based on low counts of radioactivity (IARC 1994; Svensson et al. 1991).

Based on the available information on genotoxicity, propene is not likely to be mutagenic in humans. Positive result were observed in DNA binding assays but the adducts observed are unlikely to result in genotoxic effects, as these adducts

were formed but no evidence of mutagenic or clastogenic effects were observed (*in vivo* and *in vitro*).

Oxidation of propene upon inhalation in both humans and animals can create propylene oxide. Propylene oxide has been classified by the International Agency for Research on Cancer (1994) as a Group 2B carcinogen (possibly carcinogenic to humans) based on tumours in rodents at high concentrations (Filser et al. 2008; OECD 2003). Due to the possibility of carcinogenesis as a result of propylene oxide exposure, the maximum body burden for propylene oxide has been calculated at 100 ppm by Golka et al. 1989, a concentration lower than the one at which neoplastics effects were observed. The body burden of propylene oxide was 124 nl gas/ml tissue at this exposure concentration. Due to saturation kinetics, the maximum body burden of propylene oxide cannot exceed a concentration of 71 nL propylene oxide gas/ml tissue if rats are exposed to propene even at very high concentrations. It is not likely that the body burden at which carcinogenesis would occur could be reached (Golka et al. 1989). Thus, this may provide a mechanistic basis that explains why no increase in cancer has been observed at 5000 and 10 000 ppm propene in chronic animal studies.

Chronic non-cancer effects have been identified in the NTP studies. In the rat study, increased incidence of squamous metaplasia was observed in the nasal cavities of females at both concentrations and in males at the lowest concentration only. Males experienced inflammatory changes at both testing concentrations, with a significant difference from the control group at 5000 ppm. Epithelial hyperplasia was observed in nasal cavities of female rats exposed at the highest concentration. Non–neoplastic lesions were not observed in the nasal cavities of mice. However, chronic kidney inflammation was observed in both male and female mice, with the highest incidences being in the 5 000 ppm group. The authors indicated that this result was due to propene exposure; however, the relationship was unclear (NTP 1985; Quest et al. 1984). A re-evaluation of archived specimens showed no evidence of renal tubule injury compared to the control animals. Chronic progressive nephropathy affected over 85% of mice in each group, but at low grade of severity. The severity of the lesion was minimal in each group, with no difference between control and exposed group (Hard 2001). It was proposed that the inflammation observed in the mouse kidney represents a spontaneous lesion without toxicological significance. A re-evaluation of the nasal cavity tissues by Harkema (2002) found that propene induces mild rhinitis (nasal inflammation) and associated epithelial alterations suggesting chronic, low grade irritation in both rats and mice. However, there was no obvious dose-response relationship for this effect in the two species. There was also a modest gender effect with female rodents (rats and mice) having a slightly higher incidence of propene-induced nasal lesions compared to similarly exposed males. In addition, rats had more exposure-related nasal epithelial alterations than did the similarly exposed mice.

In the second chronic study, no non-neoplastic effects were observed. A slightly increased mortality rate was observed in male rats being treated at 1 000 ppm and 5 000 ppm. The male mouse mortality rate was also slightly increased for mice in the 5 000 ppm group (Ciliberti et al., 1988). However, IARC (1994) notes incomplete reporting as the study does not provide information referring to performance of the study, such as the time of death of test or control animals, numbers of animals that died and toxic effects observed with respect to mortality rates. Also, nasal tissue histopathology was not evaluated in this study. Given the limitations of this study, the NTP study noted above was deemed to be more valid for risk characterization.

The lowest-observed-adverse-effect concentration (LOAEC) for non-neoplastic effect was 8600 mg/m^3 (5000 ppm), based on significantly increased incidence of squamous metaplasia (males and females) and inflammation in the nasal cavities (males only) of rats.

In a short-term toxicity study, the NTP studied the effects of propene inhalation (at concentrations of 0, 1 075, 2 151, 4 300, 8 600 or 17 200 mg/m^3) on rats and mice. No mortality, morbidity, weight change, or compound related effects were observed in both species following exposure to up to 17 200 mg/m^3 propene for 2 weeks (NTP 1985). In a 4-week study in rats, there were no treatment-related changes in body weight/body weight gain food consumption, mortality, and no treatment-related effects on cell proliferation in the liver or nasal respiratory epithelium up to doses of17 200 mg/m^3 (Dupont 2002b; Pottenger et al. 2007). In a 14 week-study in rat and mice, the same concentrations of exposure as the two week study were tested by the NTP. No compound related effects or pathologic changes (including reproductive organs, nasal cavity or kidney) were observed in rats or mice at any concentrations (NTP 1985, OECD 2003). The NOAEC for repeated-dose inhalation exposure was 17 200 mg/m^3 based on absence of treatment-related effects in rats and mice following exposure to propene for a period of up to 14 weeks.

No reproductive toxicity studies were identified for propene. However, developmental toxicity has been investigated in a study where pregnant Wistar rats were exposed by inhalation to 0, 344, 1720 or 17 200 mg propene/m^3, 6h/day on day 6 through 19 post coitum. No treatment-related effects on developmental parameters were observed in fetuses of pregnant rats. Likewise, no maternal toxicity was reported (BASF 2002). The NOAEC for developmental toxicity and maternal toxicity was 17 200 mg/m^3.

Similar to other hydrocarbons, inhalation of high doses of propene may cause central nervous system narcotic effects characterized by headache, dizziness, giddiness, nausea, vomiting, face reddening, coughing, and at high enough concentrations unconsciousness. High gas concentration may also cause irritation to mucous membranes as noted by tearing and reddening of the eyes, and increased sensitivity to light. Direct dermal or eye contact to this substance in

its liquid state may result in cryogenic burns (Information Handling Services, 1989; Clayton and Clayton, 1981; Sax and Lewis, 1987; Midwest Research Institute, 1978).

Recent studies in rats suggest that propene has a low order of acute toxicity by the inhalation route of exposure. In a study with exposure to propene for 4 hours, the NOAEC was 111 800 mg/m^3, the highest concentration tested, based on no deaths or hepatotoxicity (Conolly and Osimitz 1981). In a similar study, a NOAEC of 86 000 mg/m^3 was identified, based again on no deaths or hepatotoxicity (Osimitz and Conolly 1985). It is important to note however that in both study, the liver was the only organ examined.

Toxicokinetic studies on propene show that it is not readily absorbed from the lungs following inhalation exposure and is therefore not well distributed in the body. A recent report has stated that, at low concentrations, 7% of propene inhaled is metabolized in humans with the rest, 93%, exhaled immediately unchanged in humans (Golka et al. 1989; Filser et al., 2000). In rats, about 16% of the inhaled material is absorbed, of which almost one-half is exhaled, unchanged (for a total absorption of about 8%) (IARC 1994). Studies have shown that propene metabolites appear to be fairly evenly distributed throughout the body. However, higher concentrations will likely be found in fatty tissue based on its observed tissue: air partition coefficient. Propene which is absorbed is rapidly metabolized by oxidation to propylene oxide. Limited data is available concerning its elimination following metabolism. IARC (1994) reported 3 possible metabolic/elimination pathways for propylene oxide in humans. It is predominantly conjugated with glutathione, and eliminated rapidly. It may also be hydrolysed to lactic and pyruvic acids. Finally, propylene oxide may forms adducts with proteins, including haemoglobin, in man, dog, rat and mouse (IARC, 1994). Based on the low absorption of propene and its rapid elimination, it has been suggested that the concentration of bioavailable propene may not reach high enough levels in classical long-term inhalation studies to show serious chronic effects (Golka et al. 1989).

The confidence in the toxicity database for propene is considered to be moderate to high, as adequate information is available to identify critical endpoints based on repeated-dose inhalation exposure of acute to long-term duration, with the exception of reproductive toxicity studies for that route of exposure. No oral or dermal studies are available. However, propene is a gas at room temperature and thus exposition by other routes of exposure is not expected.

9.3 Characterization of Risk to Human Health

The International Agency for Research on Cancer has classified propene as a Group 3 carcinogen (not classifiable as to its carcinogenicity in humans). This classification was based on inadequate evidence of carcinogenicity in humans and experimental animals (IARC 1994). In humans, available epidemiological

studies do not demonstrate a causal association between polypropylene production (which likely involves propene handling) and colorectal cancer. In rats and mice, propene did not demonstrate carcinogenicity in two chronic/carcinogenicity inhalation studies. Consideration of the available information on genotoxicity indicates that propene is not likely to be genotoxic.

With respect to non-cancer effects, the lowest inhalation LOAEC for chronic exposures was 5000 ppm (8600 mg/m^3), based on significantly increased incidence of squamous metaplasia and inflammation in the nasal cavities of rats exposed for 2 years. This LOAEC has also been selected as a critical effect level by the US EPA (US EPA 1999). This effect level is more than five orders of magnitude higher than the highest 95th percentile concentrations for both indoor and personal air measured for propene in Canada (18.3 and 4.8 µg/m^3, respectively). The resulting margins of exposure are considered adequate to address uncertainties related to health effects and exposure. Therefore, based on the information available, it is concluded that propene does not meet the criteria under paragraph 64(*c*) of CEPA 1999 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.

9.4 Uncertainties in Evaluation of Risk to Human Health

The level of confidence in the environmental exposure data is moderate to high. Relevant and current ambient air concentrations of propene were available from Canadian monitoring sites. Potential exposure from other environmental media (water and soil) have a lower confidence due to estimations based on the known quantity in commerce for 2000 combined with estimated loss percentages from Environment Canada's Mass Flow tool. However, owing to the properties of propene, the monitoring data available is sufficient in conservatively estimating the exposure for the general population.

10 Conclusion

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 propene does not meet the criteria under paragraph 64(*a*) or 64(*b*) of CEPA 1999 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.

On the basis of the adequacy of the margins between upper-bounding estimates of exposure and the critical effect level for chronic exposure, it is concluded that propene does not meet the criteria under paragraph 64(*c*) of CEPA 1999 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 propene does not meet any of the criteria set out in section 64 of CEPA 1999.

References

Acquavella JF, Douglass TS, Philips SC. 1988. Evaluation of excess colorectal cancer incidence among workers in the manufacture of polypropylene. J Occup Med 30:438-442 [cited in IARC 1994].

Acquavella JF, Owen CV. 1990. Assessment of colorectal cancer incidence among polypropylene pilot plant employees. J Occup Med 32:438-442.

Allen JR, Ensign SA. 1998. Identification and characterization of epoxide carboxylase activity in cell extracts of *Nocardia corallina* B276. J. Bacteriol. 180: 2072–2078.

[AIHA] American Industrial Hygiene Association. 1989. Odor Thresholds for Chemicals with Established Occupational Health Standards. pp. 28, 74 [cited in CCOHS, 2004].

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

Atkinson, R. 1989. Kinetics and Mechanisms of the Gas-phase Reactions of the Hydroxyl Radical with Organic Compounds. J. Phys. Chem. Ref. Data. Monograph no. 1. American Chemical Society, Washington, D.C.; American Institute of Physics for the National Institute of Standards and Technology, New York, N.Y. 246 p.

Atkinson R. 2000. Atmospheric chemistry of VOCs and NOx. Atmos. Environ. 34: 2063–2101.

Atkinson R, Baulch DL, Cox RA, Hampson RF Jr, Kerr JA, Rossi MJ, Troe J. 1997. Evaluated kinetic, photochemical and heterogeneous data for atmospheric chemistry: Supplement V and VI, IUPAC Subcommittee on Gas Kinetic Data Evaluation for Atmospheric Chemistry. J. Phys. Chem. Ref. Data 26: 521–1011 (Supplement V) and 1329–1499 (Supplement VI).

BASF Aktiengesellschaft. 2002. Propylene - Prenatal developmental inhalation toxicity study in Wistar rats; vapor exposure. Experimental Toxicology and Ecology Laboratory, Rhein, Germany. Project #31R0416/01019 [cited in US HPV 2004].

Braker W, Mossman AL. 1980. Propylene. Matheson Gas Data Book. 6th ed. Matheson, New Jersey. pp. 624–631.

Browning E. 1987. Ethel Browning's Toxicity and Metabolism of Industrial Solvents. Vol. I: Hydrocarbons. 2nd ed. Elsevier, New York, N.Y. pp. 354-361 [cited in CCOHS, 2004].

Canada. 1999. *Canadian Environmental Protection Act, 1999*. S.C., 1999, c. 33. Available from: http://www.gazette.gc.ca/archives/p3/1999/g3-02203.pdf

Canada. 2000. *Persistence and Bioaccumulation Regulations = Règlement sur la persistence et la bioaccumulation.* Available at Canada Gazette (Part II) 134(7): 607–612 (March 29, 2000). http://laws-lois.justice.gc.ca/PDF/SOR-2000-107.pdf (accessed August 3, 2007).

Canada. 2003. *On-Road and Engine Emission Regulations.* Available at Canada Gazette (Part II) 137(1): 6–28 (January 1, 2003). Available from: http://ec.gc.ca/lcpe-cepa/eng/regulations/detailReg.cfm?intReg=65

Canada, Dept. of the Environment. 2001. *Canadian Environmental Protection Act, 1999*: *Notice with respect to certain substances on the Domestic Substances List (DSL).*Canada Gazette, Part

I, Vol. 135, no. 46, p. 4194–4210. Available from: http://www.gazette.gc.ca/archives/p1/2001/2001-11-17/pdf/g1-13546.pdf

Cardarelli M, Botondi R, Vizovitis K, Mencarelli F. 2002. Effects of exogenous propylene on softening, glycosidase, and pectinmethylesterase activity during postharvest ripening of apricots. J. Agric. Food Chem. 50: 1441–1446.

[CASA] Clean Air Strategic Alliance. 2010. Data Warehouse. Alberta Environment. Data available from: http://www.casadata.org/Reports/IDataReports/DataDownloadMain.aspx

[CCME] Canadian Council of Ministers of the Environment. 2012. Air Quality Management System. Available from: http://www.ccme.ca/ourwork/air.html?category_id=146

Chang C-C, Sree U, Lin Y-S, Lo J-G. 2005. An examination of 7:00-9:00 PM ambient air volatile organics in different seasons of Kaohsiung city, southern Taiwan. Atmos Environ 39: 867-884.

ChemCAN [Level III fugacity model of 24 regions of Canada]. 2003. Version 6.00. Peterborough (ON): Trent University, Canadian Centre for Environmental Modelling and Chemistry. [cited 2010 May]. Available from: http://www.trentu.ca/academic/aminss/envmodel/models/CC600.html.

Ciliberti A, Maltoni C, Perino G. 1988. Long-term carcinogenicity bioassays on propylene administered by inhalation to Sprague-Dawley rats and Swiss mice. Ann N.Y. Acad Sci 534: 235-245 [cited in US HPV, 2004].

Clayton GD, Clayton FE, eds. 1981. Patty's Industrial Hygiene and Toxicology. Vol. 2. Third Revised Edition. New York: John Wiley and Sons. Vol. IIB, pp. 3199-3201 [cited in NTP 1991]

Conolly R, Osimitz T. 1981. Mixed function oxidase system inducers and propylene hepatotoxicity. Toxicol 1: 112 [cited in US HPV 2004].

[CTUMS] Canadian Tobacco Use Monitoring Survey. 2008. Supplementary tables, CTUMS annual 2008 [Internet]. Ottawa (ON): Health Canada. Available from: http://www.hc-sc.gc.ca/hc-ps/tobac-tabac/research-recherche/stat/_ctums-esutc_2008/ann-table1-eng.php

Curren KC, Dann TG, Wang DK. 2006. Ambient air 1,3-butadiene concentrations in Canada (1995-2003): seasonal, day of week variations, trends and source influences. Atmos Environ 40: 5905-5912.

Curry S, Ciuffetti L, Hyman M. 1996. Inhibition of growth of a *Graphium* sp. on gaseous *n*-alkanes by gaseous *n*-alkynes and *n*-alkenes. Appl. Environ. Microbiol. 62(6): 2198–2200. Available at http://aem.asm.org/cgi/reprint/62/6/2198.pdf (accessed August 17, 2007).

DuPont. 2002a. Propylene Biomarker/Mutagenicity Dose-Response Study in Rats. Rat Bone Marrow Micronucleus Assay by Inhalation. DuPont Haskell Laboratory. Report No. DuPont-9106 [cited in US HPV 2004].

DuPont. 2002b. Propylene Biomarker/Mutagenicity Dose-Response Study in Rats. DuPont Haskell Laboratory. Draft Report No. DuPont8659 [cited in US HPV 2004].

Eide I, Hagemann R, Zahlsen K, Tareke E, Törnqvist M, Kumar R, Vodicka P, Hemminki K. 1995. Uptake, distribution, and formation of haemoglobin and DNA adducts after inhalation of C2-C8 1-alkenes (olefins) in the rat. Carcinogenesis 16(7): 1603-1609.

Englot C. 2006. Personal communication. Fort Saskatchewan VOC monitoring program, Environment Canada. Unpublished data.

Environment Canada. 2003. Data collected pursuant to section 71 (CEPA 1999) and in accordance with the published notice "*Notice with Respect to Certain Substances on the Domestic Substances List (DSL),* Canada Gazette, Vol. 135 no. 46". Data prepared by: Environment Canada, Health Canada, Existing Substances Program.

Environment Canada. 2003. Gaseous and particulate matter emissions from in-use light-duty gasoline motor vehicles. Environmental Technology Centre, Emissions Research and Measurement Division, Environment Canada, Ottawa, Canada.

Environment Canada. 2010. National Pollutant Release Inventory (NPRI). 2010. Data for reporting years 1994 to 2009. Environment Canada, Gatineau, Quebec. Available at www.ec.gc.ca/pdb/npri/npri_dat_rep_e.cfm (accessed August 17, 2010).

Environment Canada. 2011. NAPS Annual raw data [Internet]. Gatineau, QC: Environment Canada, Environmental Technology Centre [cited 2011 June]. Available from : http://www.etc-cte.ec.gc.ca/NapsAnnualRawData/Default.aspx?ReturnUrl=%2fNapsAnnualRawData%2fMain.aspx (protected)

Environment Canada. 2012. National Emission Trends for Key Air Pollutants. Criteria Air Contaminants (National: 1985-2010). Available from: http://www.ec.gc.ca/inrp-npri/default.asp?lang=en&n=0EC58C98-1%20

[EPISuite] Estimation Programs Interface Suite for Microsoft Windows [Estimation Model]. 2008. Version 3.4. Washington (DC): U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics; Syracuse (NY): Syracuse Research Corporation. Available from: www.epa.gov/oppt/exposure/pubs/episuite.htm

Filser JG, Schmidbauer R, Rampf F, Baur CM, Pütz C, Csanády GA. 2000. Toxicokinetics of inhaled propylene in mouse, rat, and human. Toxicol Appl Pharmacol 169(1):40-51.

Filser JG, Hutzler C, Rampf F, Kessler W, Faller TH, Leibold E, Pütz C, Halbach S, Csanády GA. 2008. Concentrations of the propylene metabolite propylene oxide in blood of propylene-exposed rats and humans – a basis for risk assessment. Toxicological Sciences 102(2):219-231.

Foureman P, Mason JM, Valencia R, Zimmering S. 1994. Chemical mutagenesis testing in *Drosophila*. X. Results of 70 coded chemicals tested for the National Toxicology Program. Environmental and Molecular Mutagenesis 23(3): 208-227.

Goldberg MS, Theriault G. 1994a. A retrospective cohort of workers of a synthetic textile plant in Quebec. I. General mortality. An J Ind Med 25:889-907.

Goldberg MS and Theriault G. 1994b. A retrospective cohort of workers of a synthetic textile plant in Quebec. II.Colorectal cancer mortality and incidence. An J Ind Med 25:909-22.

Golding JB, Shearer D, McGlasson WB, Wyllie SG. 1999. Relationships between respiration, ethylene, and aroma production in ripening banana. J. Agric. Food Chem. 47: 1646–1651.

Golka K, Peter H, Denk B, Filser JG. 1989. Pharmacokinetics of propylene and its reactive metabolite propylene oxide in Sprague-Dawley rats. Arch Toxicol Suppl 13:240-242 [cited in IARC 1994].

Guo H, Lee SC, Louie PKK, Ho KF. 2004. Characterization of hydrocarbons, halocarbons and carbonyls in the atmosphere of Hong Kong. Chemosphere 57: 1363–1372.

Hard G, 2001. Expert report on renal histopathological changes in mouse and rat inhalation studies with propylene. Prepared for: American Chemistry Council Olefins Panel, Arlington VA USA [cited in US HPV 2004].

Harkema J. 2002. Histopathology report: Evaluation of nasal cavity slides from NTP inhalation studies of propylene in rats and mice for the American Chemical Council [cited in US HPV 2004].

Health Canada. 1998. Exposure factors for assessing total daily intake of priority substances by the general population of Canada. Unpublished report. Ottawa (ON): Health Canada, Environmental Health Directorate. Available upon request.
Health Canada. 2010a. *Windsor Exposure Assessment Study (2005-2006)[WEAS]: Data Summary for Volatile Organic Compound Sampling.* Minister of Health, Health Canada, Ottawa, Ontario. pp

Health Canada. 2010b. *Regina Indoor Air Quality Study (2007) [RIAQS]: Data Summary for Volatile Organic Compound Sampling.* Minister of Health, Health Canada, Ottawa, Ontario. pp

Health Canada. 2011. *Halifax Indoor Air Quality Study (2009) [HIAQS]: Data Summary for Volatile Organic Compound Sampling* – Draft. Minister of Health, Health Canada, Ottawa, Ontario. pp

Health Canada. 2012. Food and Nutrition, Lists of Permitted Food Additives. [Internet]. Ottawa (ON): Health Canada [cited 2013 March 8]. Available from: http://www.hc-sc.gc.ca/fn-an/securit/addit/list/index-eng.php

Hodges CF, Campbell DA. 1998. Gaseous hydrocarbons associated with black layer induced by the interaction of cyanobacteria and *Desulfovibrio desulfuricans*. Plant and Soil 205: 77–83.

Howard PH, Boethling RS, Jarvis WF, Meylan WM, Michalenko EM. 1991. Handbook of Environmental Degradation Rates. Lewis Publishers, Chelsea, Michigan. pp. 439–440.

[HSDB] Hazardous Substances Data Bank. 2003.Propylene. Toxicology Data Network, National Library of Medicine, National Institutes of Health, U.S. Department of Health and Human Services, Bethesda, Maryland. Available at http://toxnet.nlm.nih.gov (searched July 2005).

Hughes TJ, Sparacino C, Frazier S. 1984. Validation of chemical and biological techniques for evaluation of vapors in ambient air/mutagenicity testing of twelve vapour-phase compounds. EPA Report No. EPA-600/1-84-005 [cited in US HPV 2004].

[IARC] International Agency for Research on Cancer. 1994. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 60 – Some Industrial Chemicals.

Inchem. 2001. Propylene. International Programme on Chemical Safety, Commission of the European Communities. Available at http://www.inchem.org/documents/icsc/icsc/eics0559.htm (searched July 2005).

Information Handling Services. 1989. Material Safety Data Sheets Service. Microfiche Ed. Bimonthly Updates. October/November #7030-227, C-03; #9910-032, B-05 [cited in NTP 1991]

Inveresk Research. 2003. Ames test draft report [cited in US HPV 2004].

Isidorov VA, Zenkevich IG, Ioffe BV. 1985. Volatile organic compounds in the atmosphere of forests. Atmos. Environ. 19: 1–8.

Kleindienst et al., 1992. Generation of mutagenic transformation products during the irradiation of simulated urban atmospheres Env Sci Technol 26: 320-329 [cited in US HPV 2004].

Lagast H, Tomenson J, Stringer DA. 1995. Polypropylene production and colorectal cancer: a review of the epidemiological evidence. Occup Med 45:69-74.
Lai C-H, Peng Y-P. 2011. Emissions of C_2-C_{12} hydrocarbons in the Hsuehshan tunnel, Taiwan. J Environ Sci 23: 941-948.
Landry M, Fuerst R. 1968. Dev Indust Microbiol 9: 370-380 [cited in US HPV 2004].

Lewis RJ, Schnatter AR, Lerman SE. 1994. Colorectal cancer incidence among polypropylene manufacturing workers: an update. J Occup Med 36:652-9.

Lewis RJ Sr. 2002. Hawley's Condensed Chemical Dictionary. 14th ed. Electronic version on CD. Wiley-Interscience, New York, N.Y.

Löfroth G. 1989. Environmental tobacco smoke: overview of chemical composition and genotoxic components. Mutation Research 222:73-80.

Mackay D, DiGuardo A, Paterson S, Cowan CE. 2003. EQC Model version 2.02. Released May 2003. Canadian Environmental Modelling Center, Trent University, Peterborough, Ontario, Canada. Available at http://www.trentu.ca/cemc/models/EQC2.html (downloaded April 2006).

Marchionna M, Girolamo MD, Patrini R. 2001. Light olefins dimerization to high quality gasoline components. Catalysis Today 65: 397-403.

Mark HF, Othmer DF, Overberger CG, Seaborg GT. 1978. Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed. Volume 19: Powder Coatings to Recycling. Wiley-Interscience Publications, New York, N.Y. pp. 228–246.

Matsunaga SN, Chatani S, Morikawa T, Nakatsuka S, Suthawaree J, Tajima Y, Kato S, Kajii Y, Minoura H. 2010. Evaluation of non-methane hydrocarbon (NMHC) emissions based on an ambient air measurement in Tokyo area, Japan. Atmos Environ 44: 4982-4993.

McAuliffe C. 1966. J Phys Chem 70: 1267-75. Via HSDB 2003

McGregor D, Brown AG, Cattanach P, Edwards D, McBride D, Riach C, Shepherd W, Caspary WJ. 1991. Responses of the L5178Y mouse lymphoma forward mutation assay: V. Gases and vapors. Environ Mol Mutag 17: 122-129 [cited in OECD 2003].

Midwest Research Institute. 1978. MRI Report for Propylene. Kansas City, MO [cited in NTP 1991].

Nanos GD, Agtsidou E, Sfakiotakis EM. 2002. Temperature and propylene effects on ripening of green and black 'Conservolea' olives. HortScience. 37: 1079–1081.

Nova, 2010. Nova Chemicals. Material Safety Data Sheet Propylene – Chemical Grade. January, 2010. MSDS ID NOVA-0016. Accessed November 2011. Available at: http://www.novachem.com/Product Documents/PropyleneChemicalGrade_MSDS_EN.pdf

[NRCAN] Natural Resources Canada.2008. Canadian Vehicle Survey Update Report – 2008. Accessed January, 2012. Available from:
http://oee.nrcan.gc.ca/publications/statistics/cvs08/pdf/cvs08.pdf

[NTP] National Toxicology Program. 1985. Toxicology and carcinogenesis studies of propylene (CAS No. 115-07-1) in F344/N rats and B6C3F_1 mice (inhalation studies). Report no. TR-272. 3 pp. [cited in IARC 1994].

[NTP] National Toxicology Program. 1989. Study ID 779144. Available on the Internet at: http://ntp-apps.niehs.nih.gov/ntp_tox/index.cfm?fuseaction=salmonella.salmonellaData&endpointlist=SA&study%5Fno=779144&cas%5Fno=115%2D07%2D1&activetab=detail.

[NTP] National Toxicology Program. 1991. NTP chemical repository: propylene. Available on the Internet at: http://157.98.10.135/NTP_Reports/NTP-Chem_H&S/NTP_Chem1/Radian115-07-1.txt Accessed February 18, 2002.

[OECD] Organisation for Economic Co-Operation and Development. 2003. SIDS Initial Assessment Profile – Propylene or propene. SIAM 16, 27-30 May 2003, UK/ICCA.

Olson DA, Hammond DM, Selia RL, Burke JM, Norris GA. 2009. Spatial gradients and source apportionment of volatile organic compounds near roadways. Atmospheric Environment 43: 5647-5643.

O'Neil MJ, Smith A, Heckelman PE, Obenchain Jr JR, Gallipeau JR, D'Arecca MA. 2001. The Merck Index: An Encyclopedia of Chemicals, Drugs and Biologicals. Thirteenth edition. Merck and Co., Inc., Whitehouse Station, New Jersey. pp. 1404–1405.

Osimitz TG, Conolly RB. 1985. Miked-function oxidase system induction and propylene hepatotoxicity. J Toxicol Environ Health 15: 39-49 [cited in IARC 1994].

Perkins-Veazie PM, Huber DJ Brecht JK. 1996. In vitro growth and ripening of strawberry fruit in the presence of ACC, STS or propylene. Ann. Appl. Biol. 128: 105–116.

Pottenger LH, Malley LA, Bogdanffy MS, Donner EM, Upton PB, Li Y, Walker VE, Harkema JR, Banton MI, Swenberg JA. 2007. Evaluation of effects from repeated inhalation exposure of F344 rats to high concentrations of propylene. Toxicological Sciences 97(2):336-347.

Quest J, Tomaszewski J, Haseman J, Boorman G, Douglas J, Clarke W. 1984. Two-year inhalation toxicity study of propylene in F344/N rats and B6C3F1 mice. Toxicol Appl Pharmacol 76: 288-295 [cited in US HPV 2004].

Reid RC, Prausnitz TM, Sherwood TK. 1977. The Properties of Gases and Liquids. 3rd ed. McGraw-Hill Book Co., New York.

Sax NI, Lewis Sr RJ, eds. 1987. Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold, pp. 972-973 [cited in NTP 1991].

Speight GJ. 2007. The Chemistry and Technology of Petroleum. Boca Raton (FL): CRC Press.

Statistics Canada. 2005. Canadian Vehicle Survey: Annual 2005 (revised). Catalogue no. 53-223-XIE. Statistics Canada, Ottawa, Ontario.

Statistics Canada. 2010. Canadian Vehicle Survey. 2009. Available from: http://publications.gc.ca/Collection/Statcan/53-223-X/53-223-XIE.html

Statistics Canada. 2011. Table 303-0053 - Production of industrial chemicals and synthetic resins, annual (tonnes). Accessed: October 11, 2011.

Svensson K, Olofsson K, Osterman-Golkar S. 1991. Alkylation of DNA and hemoglobin in the mouse following exposure to propene and propylene oxide. Chem Biol Interactions 78:55-66 [cited in IARC 1994].

Swinnerton J, Lamontagne R. 1974. Oceanic distribution of low-molecular-weight hydrocarbons. Envi. Sci. Tech. 8(7):657-663.

US EPA (United States Environmental Protection Agency). 1999. Propylene streams robust summary. Robust Summary no.: OP E423. Available at www.epa.gov/chemrtk/pubs/summaries/prplstrm/c13281rs.pdf#search=%22c13281rs.pdf%22 (accessed September 19, 2006).

[US HPV] US High Production Volume Chemical Program, 2004. Chemical Category Summary for Propylene Streams Prepared by: Olefins Panel of the American Chemistry Council (Document # 201-15584A & 201-15584B). Available at http://www.epa.gov/chemrtk/pubs/summaries/prplstrm/c13281rt.pdf (accessed September 25, 2006).

Victorin K, Stahlberg M. 1988. A method for studying the mutagenicity of some gaseous compounds in *Salmonella Typhimurium*. Environ Mol Mutag 11:65-77 [cited in OECD 2003].

Vobecky J, Devroede G, Lacaille J, Watier A. 1978. An occupational group with a high risk of large bowel cancer. Gastroenterology 75: 784-785. in: Lagast H, Tomenson J, Stringer DA. 1995. Polypropylene production and colorectal cancer: a review of the epidemiological evidence. Occup Med 45(2): 69-74.

Vobecky J, Caro J, Devroede G. 1983. A case-control study of risk factors for large bowel carcinoma. Cancer 51:1958-1963 in: Lagast H, Tomenson J, Stringer DA. 1995. Polypropylene production and colorectal cancer: a review of the epidemiological evidence. Occup Med 45(2): 69-74.

Vobecky J, Devroede G, Caro J. 1984. Risk of large bowel cancer in synthetic fibre manufacture. Cancer 54: 2537-2542 in: Lagast H, Tomenson J, Stringer DA. 1995. Polypropylene production and colorectal cancer: a review of the epidemiological evidence. Occup Med 45(2): 69-74.

Walker DM, Seilkop SK, Scott BR, Walker VE. 2004. Hprt mutant frequencies in splenic T-cells of male F344 rats exposed by inhalation to propylene. Environmental and Molecular Mutagenesis 43:265-272.

Wasik SP, Tsang W. 1970. J Phys Chem 74: 2970-6. Via HSDB 2003.

Weichenthal S, Kulka R, Dubeau A, Martin C, Wang D, Dales R. 2011. Traffic-related air pollution and acute changes in heart rate variability and respiratory function in urban cyclists. Environmental Health Perspectives 119(10). Available from: http://hero.epa.gov/index.cfm?action=reference.details&reference_id=786573

Wiens B. 2005. Personal communication. Meteorological Service of Canada, Environment Canada. Unpublished data.

Appendix A. Summary of health effects information for propene in mammals

Table A1. Health effects information for propene from animal studies

Endpoints	Lowest effect levels[a]/results
Acute toxicity	**Inhalation LC_{50}** (rat; 4 hours): > 65 000 ppm (111 800 mg/m^3) **Inhalation NOAEC (rat):** 111 800 mg/m^3 based on no death or hepatotoxicity in Sprague-Dawley rats exposed to 0 or 65 000 ppm (111 800 mg/m^3) propylene for 4 hour. However, the liver was the only organ examined (Conolly and Osimitz 1981). **Other inhalation NOAEC (rat):** 86 000 mg/m^3 based on no death or hepatotoxicity in Sprague-Dawley rats exposed to 0 or 50 000 ppm (86 000 mg/m^3) propylene for 4 hour. However, the liver was the only organ examined (Osimitz and Conolly 1985).
Short-term repeated-dose toxicity	**Inhalation NOAEC (mouse, rat):** 17 200 mg/m^3 based on no mortality, morbidity, weight change, or other compound related effects in a 2-week mouse study and a 2-week rat study (NTP 1985: male and female F344 rat and B6C3F1 mice (5 per dose) exposed to 0, 625, 1 250, 2 500, 5 000 or 10 000 ppm (0, 1 075, 2 151, 4 300, 8 600 or 17 200 mg/m^3), 6 h/day, 5 days/week, for 2 weeks). **Other inhalation NOAEC (rat):** 17 200 mg/m^3 based on no treatment-related statistically significant changes in body weight/body weight gain and food consumption, no mortality, no histopathological changes in nasal cavity tissues and no treatment related effects on cell proliferation in the liver or nasal respiratory epithelium in F344 rat (8 males and females per dose) exposed to 0, 200, 2 000 or 10 000 ppm (0, 344, 3442 or 17 200 mg/m^3), 6 h/day, 5 days/week, for up to 4 weeks (Dupont 2002b; Pottenger et al. 2007).

Endpoints	Lowest effect levels[a]/results
Subchronic toxicity	**Inhalation NOAEC (mouse):** 17 200 mg/m^3 based on no treatment-related mortality, morbidity, weight change or gross or microscopic pathology effects (including reproductive organs, nasal cavity and kidney) in B6C3F1 mice (9-11/sex/dose) exposed to 0, 625, 1 250, 2 500, 5 000 or 10 000 ppm (0, 1 075, 2 151, 4 300, 8 600 or 17 200 mg/m^3), 6 h/day, 5 days/week, for 14 weeks. Two mice exhibited morbidity and therefore were sacrificed; specifically a female on Day 35 exposed to 1250 ppm and a male on Day 67 exposed to 5000 ppm. Mean body weight were found to be decreased by 4 to 7% in exposed females in comparison to control weights. However, the weight changes and the morbidity observed were not a result of propylene exposure (NTP 1985; OECD 2003). **Other inhalation NOAEC (rat):** 17 200 mg/m^3 based on no treatment-related mortality, weight change or gross or microscopic pathology effects (including reproductive organs, nasal cavity and kidney) in F344 rat (9-11/sex/dose) exposed to 0, 625, 1 250, 2 500, 5 000 or 10 000 ppm (0, 1 075, 2 151, 4 300, 8 600 or 17 200 mg/m^3), 6 h/day, 5 days/week, for 14 weeks. The mean weight change observed in exposed male rats was 4-12% higher than the mean weights in the control group, while exposed and control females were found to be comparable with respect to weight change. The weight changes in males were judged to be not treatment-related (NTP 1985; OECD 2003).
Chronic toxicity/ carcinogenicity	**Inhalation study in rats:** Groups of 50 F344 rats per sex were exposed to propene by inhalation (whole-body) at 0, 5 000 or 10 000 ppm (0, 8 600 or 17 200 mg/m^3), 6 h/day, 5 days/week for 103 weeks. Negative trends were observed in adenomas and adenomas plus carcinomas, while positive trends were noted for hyperplasia (adenomas 5/39, 2/47 and 0/47 at 0, 8 600 and 17 200 mg/m^3, respectively; adenomas plus carcinomas 6/39, 2/47 and 2/47, respectively; hyperplasia 2/39,7/47 and 6/47, respectively). The combination of all types of lesions; adenoma, carcinoma and hyperplasia caused the trends to disappear. Based on this occurrence, the authors of the study concluded that these tumours of the thyroid gland were not due to propene exposure. **Non-neoplastic LOAEC:** 8600 mg/m^3 based on significantly increased incidence of squamous metaplasia in nasal cavities of both sexes and inflammation of the nasal cavities in males. The increased incidence of squamous metaplasia was observed in female rats at both concentrations and in males at the lowest concentration only. Males experienced inflammatory changes at both testing concentrations, with a significant difference from the control group at 5000 ppm. Other non-neoplastic effects included epithelial hyperplasia in nasal cavities of female rats exposed at the highest concentration (NTP, 1985; Quest et al., 1984). A reevaluation of the nasal cavity tissues by Harkema (2002) found that propene induces mild rhinitis (nasal inflammation) and associated epithelial alterations suggesting chronic, low grade irritation in rats. However, there was no obvious dose-response relationship for this effect. It was proposed that this may suggest a possible threshold effect at 5000 ppm for most of the observed nasal lesions. There was also a modest gender effect with female rats having a slightly higher incidence of propylene-induced nasal lesions compared to similarly exposed males.

Endpoints	Lowest effect levels[a]/results
	Other inhalation studies in rats: Groups of 100 or 120 F344 rats per sex were exposed to propylene by inhalation (whole-body) at 0, 200, 1000 or 5 000 ppm (0, 344, 1720 or 8 600 mg/m^3), 7 h/day, 5 days/week for 104 weeks. No increased incidence of tumours, differences in benign or malignant tumours or incidence of unexpected tumours were observed in exposed rats compared to controls. No non-neoplastics effects were reported. **Non-neoplastic NOAEC:** 17 200 mg/m^3, based on no treatment-related effects in both sexes. A slightly increased mortality rate was observed in male rats being treated at the intermediate and highest concentration. However, comments by the International Agency for Research on Cancer Working Group indicate incomplete recording of details such as time of death of mice, numbers of high-dose mice that died and toxic effects observed with respect to mortality rates. Also, nasal tissue histopathology was not evaluated in this study (Ciliberti et al. 1988; IARC 1994). **Inhalation study in mice:** Groups of 49-50 male and female B6C3F1 mice were exposed by inhalation (whole-body) to concentrations of 0, 5 000 or 10 000 ppm (0, 8 600 or 17 200 mg/m^3) propene for 6 hours per day, 5 days per week for 103 weeks. - In lungs of exposed mice, Alveolar/bronchial adenomas as well as carcinomas showed lower incidence rates when compared to the control group (adenomas 7/50, 3/49 and 3/50 at 0, 8 600 and 17 200 mg/m^3, respectively; carcinomas 9/50, 1/49 and 4/50, respectively). When the rates of adenoma and carcinoma were combined, the result was a negative trend in males (16/50, 4/49 and 7/50, respectively). There was no significant difference in exposed and control females (6/50, 4/49 and 7/50, respectively). The authors noted that the biological significance of this negative trend was hard to interpret, due to the incidence rates in the control and treatment groups, which fell into the historical control incidence range for this type of tumour (2-34%). - In the liver, decreased incidences of adenomas and carcinomas were observed but were not considered to be propene related. Adenomas were observed in male mice only, while carcinomas were found in male and female animals (males: adenomas 5/50, 0/49 and 3/50, respectively; carcinomas 9/50, 11/49 and 3/49, respectively; females: carcinomas 2/50, 3/49 and 5/49, respectively). When the incidence rates for occurrences of adenomas and carcinomas were combined for males, no significant difference was observed between the treatment groups and controls (14/50, 11/49 and 14/49, respectively). - Non-site specific hemangiosarcomas were observed in the subcutaneous tissue, spleen and uterus of affected females, while hemangiomas were found in the liver. Combined hemangiosarcoma and hemangioma in females indicated a positive trend (0/50, 1/49 and 4/50, respectively). However, incidences in the 10 000 ppm group were not significantly higher than in the control group. No significant difference between treated and control groups was noted for hemangiosarcomas observed in male mice. Due to the non-site specific nature of these neoplasms and the lack of higher occurrences of tumours in the 10 000 ppm group, the authors have suggested that these

Endpoints	Lowest effect levels[a]/results
	incidences were not related to propene exposure. - Endometrial polyp incidence in the uterus produced a significant positive trend (p=0.044; 0/47, 0/47 and 3/48, respectively). The overall observed rate in the 10 000 ppm group was 6%, and was not found to be significantly different from the control group. NTP historical incidence control data for this tumour type indicate a percentage occurrence of 0.9%, with an incidence range of 0% - 6%. Therefore, propene was not found to be connected with polyp formation. **Non-neoplastic NOAEC:** 17 200 mg/m^3, based on no treatment-related mortality, weight change or gross or microscopic lesions of the reproductive organs and nasal cavity (NTP 1985; Quest et al. 1984). Chronic kidney inflammation was observed in both male and female mice, with the highest incidences being in the 5 000 ppm group (male: 0/50, 17/49 and 9/49 at 0, 5000 ppm and 10000 ppm, respectively; female: 1/50, 7/49 and 6/49, respectively). The authors indicated that this result was due to propene exposure; however, the relationship was unclear (NTP 1985; Quest et al. 1984). A reevaluation of archived specimens showed no evidence of renal tubule injury compared to the control animals. Chronic progressive nephropathy affected over 85% of mice in each group, but at low grade of severity. The severity of the lesion was minimal in each group, with no difference between control and exposed group (Hard 2001). It was proposed that the inflammation observed in the mouse kidney represents a spontaneous lesion without toxicological significance. A reevaluation of the nasal cavity tissues by Harkema (2002) found a slightly higher incidence of rhinitis in exposed females and in males exposed at the lowest concentration. It was suggested that propylene induces mild rhinitis (nasal inflammation) and associated epithelial alterations suggesting chronic, low grade irritation in mice. However, there was no obvious dose-response relationship for this effect. **Other inhalation studies in mice:** Groups of 100 Swiss mice per sex were exposed to propylene by inhalation (whole-body) at 0, 200, 1000 or 5 000 ppm (0, 344, 1720 or 8 600 mg/m^3), 7 h/day, 5 days/week for 78 weeks. Mice were observed until spontaneous death and did not develop neoplastic effects. Propene exposure caused no increase in tumour incidence based on historical data, no difference in animals bearing tumours, and types of tumours. **Non-neoplastic NOAEC:** 17 200 mg/m^3, based on no treatment-related effects in both sexes. A slight increase in the mortality rate was noted in male mouse at the highest concentration. However, comments by the International Agency for Research on Cancer Working Group indicate incomplete recording of details such as time of death of mice, numbers of high-dose mice that died and toxic effects observed with respect to mortality rates. Also, nasal tissue histopathology was not evaluated in this study (Ciliberti et al. 1988; IARC 1994).

Endpoints	Lowest effect levels[a]/results
Reproductive toxicity	No reproductive toxicity study identified.
Developmental toxicity	**Inhalation NOAEC (rat):** 17 200 mg/m^3 based on no treatment-related effects on gestational and developmental parameters (conception rate, mean number of corpora lutea, total implantations, resorptions, live fetuses, fetal sex ratio, pre- and post-implantation losses, placental and fetal body weights, external and soft tissue changes, skeletal abnormalities) in pregnant Wistar rats (25 females per group) exposed by inhalation to 0, 200, 1000 or 10 000 ppm (0, 344, 1720 or 17 200 mg/m^3), 6h/day on day 6 through 19 post coitum. No maternal toxicity was reported (BASF 2002).
Genotoxicity and related endpoints: *in vivo*	**Micronuclei** **Negative:** polychromatic erythrocytes; male F344 rats; inhalation (0, 200, 2 000 or 10 000 ppm [0, 344, 3442 or 17 200 mg/m^3], 6 h/day, 5 days/week, for up to 4 weeks) (Dupont 2002a; Pottenger et al. 2007). **DNA binding** **Positive:** liver, spleen, lung tissues and lymphocytes; male and female F344 rats (16-24 males and 8 females/group); inhalation (0, 200, 2 000 or 10 000 ppm [0, 344, 3442 or 17 200 mg/m^3], 6 h/day, 5 days/week, for up to 4 weeks) (Pottenger et al. 2007). **Positive:** liver, kidney, spleen tissue; male CBA mice; inhalation (0 or 30 000 ppm [0 or 51 600 mg/m^3]) (Svensson et al. 1991). IARC Working Group notes that the results of this study were based on low counts of radioactivity (IARC 1994). **Positive:** liver and lymphocytes; male and female Sprague-Dawley rats; inhalation (0 or 300 ppm, 12 h/day for 3 consecutive days) (Eide et al. 1995). **Gene mutation at the HPRT locus** **Negative:** Splenic T-lymphocytes; male F344 rats (5-8 animals/group); inhalation (0, 200, 2 000 or 10 000 ppm [0, 344, 3442 or 17 200 mg/m^3], 6 h/day, 5 days/week, for 4 weeks) (Pottenger et al. 2007; Walker et al. 2004). **Sex linked recessive lethal mutation assay** **Negative:** Male *Drosophila melanogaster*; inhalation (0 or 720 000 ppm) (Foureman et al. 1994).
Genotoxicity and related endpoints: *in vitro*	**Mutagenicity in bacteria** **Positive:** *Salmonella typhimurium*, strain TA1535, with metabolic activation; negative without activation (Inveresk Research, 2003). **Positive:** *Salmonella typhimurium*, strain TA1535, with and without metabolic activation (NTP 1989). **Week positive:** *Salmonella typhimurium*, strain TA100, with metabolic activation; negative without activation (NTP 1989). **Negative:** *Salmonella typhimurium*, strains TA98, TA100, and TA1537, with and without activation (Inveresk Research, 2003). **Negative:** *S. typhimurium*, strains TA97 and TA98, with and without metabolic activation (NTP 1989).

Endpoints	Lowest effect levels[a]/results
	Negative: *S. typhimurium*, strains TA97 and TA98, with and without metabolic activation (Hughes et al. 1984). **Negative:** *S. typhimurium*, strain TA100, with and without metabolic activation (Victorin and Stahlberg 1988). **Negative:** *S. typhimurium*, strain TA100, with and without metabolic activation (Kleindienst et al. 1992). **Negative:** *Escherichia coli*, strain WP2uvrA/pKM101, with and without metabolic activation (Inveresk Research, 2003). **Negative:** *E. coli*, strain B, with and without activation (Landry and Fuerst 1968). **Negative:** *Bacillus Subtillis*, strain Sd-4, with and without activation (Landry and Fuerst 1968). **Mammalian cell mutation assay** **Negative:** Mouse lymphoma L5178Y TK+/- without metabolic activation (McGregor et al. 1991). **Equivocal:** Mouse lymphoma L5178Y TK+/- with metabolic activation (McGregor et al. 1991).
Sensitization	No sensitization study identified.
Irritation	No irritation study identified.

[a] Definitions; LC_{50}: median lethal8 concentration; LOEL/LOEC: lowest-observed-effect level/concentration; LOAEL/LOAEC: lowest-observed-adverse-effect level/concentration; NOAEL/NOAEC: no-observed-adverse-effect level/concentration.

Table A2. Health effects information for propene from human studies

Endpoints	Lowest effect levels[a]/results
Carcinogenicity	<u>Carpet manufacturing plant</u> In those investigations, no clear propylene exposure was identified. No information was provided on the nature and the importance of the workers' exposures. - In a case series study, Vobecky et al. (1978) first identified a cluster of 5 workers with colorectal cancer employed in a Canadian plant producing polypropylene-containing synthetic fibre unit (carpet production) in Québec, Canada. All cases were diagnosed within an 18 month period from 1974 to 1975. After the initial identification of the cluster, the authors extended their study to include all new cases of colorectal cancer for the years 1965 to 1975 in the Eastern Townships of Québec, where the plant is located. They interviewed the patients to obtain information on employment. A comparison was made of the proportion of male carpet factory workers with colorectal cancer to the proportion of men with this cancer from other occupations in the region. The proportion of male carpet factory workers with colorectal cancer was significantly greater than that of the other occupations for the period 1971 to 1975. - A case-control study of colorectal cancer was conducted in a 13-county region of Québec for the years 1965 to 1976. Two hundred seven patients (103 men and 104 women) with colorectal cancer were identified in the likely labour pool for a carpet factory. A similar number of controls was chosen from the same pool, matched on age, sex, and place of residence at time of

Endpoints	Lowest effect levels[a]/results
	diagnosis. The original cluster of 5 cases was included in the analysis (as described in Vobecky et al. 1978). Among men there was a significant excess of cases having been employed at the carpet factory (relative risk (RR) = 2.33; P<0.01). There was no excess of colorectal cancer among women employed in the factory (RR = 0.55) (Vobecky et al. 1983; Lagast et al. 1995). - A case-control study of colorectal cancer was conducted by choosing cases and controls from employees in a carpet plant (same as Vobecky et al. 1978; 1983). The time period for case ascertainment was expanded (1965 to 1979), during which 37 male and six female carpet manufacturing employees were diagnosed with colorectal cancer. Each case was matched to three controls according to sex, age, date of employment start, and duration of employment. Because of the small number of cases, women were excluded from the analysis. Company records were used for the occupational data. The original five cases that made up the initial observation were not excluded from the analysis. When combined, workers involved in the extrusion D department (solubilization of acetate), the extrusion TM department (extrusion of triacetate and polypropylene), and the textiles department (undefined) were at elevated risk (RR = 3.72; *P*<0.005). Taken separately, the RR for the extrusion D department was 1.75, that for the extrusion TM department was 2.74, and that for textiles was 2.95. Only the RR for work in textiles was statistically significant (P<0.03) (Vobecky et al. 1984; Lagast et al. 1995). - A retrospective cohort mortality study of the carpet manufacturing plant workers was conducted in 7487 men and 2624 women who had worked for at least one year at the plant from 1947 to 1977, with mortality follow-up through to 1986. The standardized mortality ratio (SMR) for colorectal cancer among male employees was significantly lower than expected (SMR = 0.69; 95% CI: 0.52-0.92), while mortality among women employees was close to expected (SMR= 1.02; 95% CI: 0.57-1.69) (Goldberg and Theriault 1994a; Lagast et al. 1995). In a further refinement, Goldberg and Theriault (1994b) performed a nested case-control study within the cohort of male carpet making workers, supplementing the deaths from colorectal cancer with incidence data from the Québec Tumour Registry for the years 1975 to 1987. An excess risk of colorectal cancer associated with employment in the combined polypropylene and cellulose triacetate extrusion unit was observed (odds ratio (OR) = 5.81; 95% CI: 0.98-34.46). However, of the four cases in this exposure category, three were from the original cluster identified previously by Vobecky et al. (1978). <u>Polypropylene manufacturing plant</u> In those studies, although propylene was handled in the facility, IARC (1994) states that the excess risk cannot be related specifically to propylene. Multiple chemicals including propylene were used in the facility, and as such, specific chemical exposure could not be related specifically to colorectal cancer. - An occupational study of workers in a polypropylene manufacturing facility in Texas, United States, was conducted on 355 male employees with more than

Endpoints	Lowest effect levels[a]/results
	6 months employment between 1960-1985. A significant increased incidence of colorectal cancer was noted; 7 observed and 1.3 expected (standardized incidence ratio (SIR) = 5.6, 95% CI: 2.2-11.5) (Acquavella et al. 1988). - The relationship between colorectal cancer and polypropylene manufacturing was assessed in a cohort study of workers employed in polypropylene pilot plants in Louisiana and Texas. The cohort consisted of 183 men who worked for six months or longer between 1956 and 1962 in the Louisiana plant or between 1959 and 1977 in the Texas plant. The workers were followed for colorectal cancer occurrence until 31 December 1985. No excess was observed. Three colorectal cancers were identified among workers compared with 3.3 expected (SIR = 0.9; 90% CI: 0.3-2.3). Further examination of cancer risk by type of job (mechanic, process and laboratory), duration of employment, and latency showed no association with risk (Acquavella and Owen 1990). - In a follow-up study, the original cohort of polypropylene production workers (Acquavella et al. 1988) was studied again but it included this time all employees who worked for 6 months or longer between 1960 and 1992 (for a total of 412 male workers). Nine colorectal cancers have been identified among the original cohort of polypropylene production workers compared with 3.8 expected based on Texas incidence rates and a ten-year latency period, giving an SIR of 2.4 (95% CI: 1.1-4.5). However, as a result of screening activities initiated among these workers, three adenocarcinomas *in situ* were also identified. The screening-adjusted SIR for colorectal cancer was 2.6 (95% CI: 1.4-4.6) based on 12 cancers. However, seven of the nine colorectal cancer cases were from the initial study. If these original cluster cases are eliminated, the excess risk is eliminated (SIR=1.10; 95% CI: 0.36-2.57) (Lewis et al. 1994).

See Table A1 for footnotes