Screening Assessment Report

Hexachloroethane

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67-72-1

Environment and Climate Change Canada

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Synopsis

Pursuant to section 74 of the *Canadian Environmental Protection Act*, *1999* (CEPA), the Ministers of the Environment and Climate Change and of Health have conducted a screening assessment of hexachloroethane, Chemical Abstracts Service Registry Number 67-72-1. Hexachloroethane was identified as priority for assessment as it met categorization criteria for persistence and greatest potential for human exposure under subsection 73(1) of CEPA. Hexachloroethane was also classified by other agencies as a possible carcinogen to humans according to the International Agency for Research on Cancer (Group 2B) and the United States Environmental Protection Agency (Group C).

Hexachloroethane is not known to occur naturally. Hexachloroethane was previously imported in Canada for use as a chemical intermediate, as a flux agent for grain refining and degassing of aluminum alloys, as a flame retardant in industrial laminating resins and as a reactant in military smoke ammunition. Recent information indicates that small quantities of hexachlorothane continue to be imported and used for degassing of aluminum alloys. In Canada, hexachloroethane is not intentionally manufactured for commercial distribution; rather, it is formed during other processes in the chlorinated chemical industry and can also be produced as a by-product of the chlorination of water and sewage and the incineration of chlorinated hydrocarbons. Based on the results of a survey conducted under section 71 of CEPA for the year 2000, approximately 150 tonnes of hexachloroethane were manufactured in Canada and between 10 and 100 tonnes were imported into Canada.

The use of hexachloroethane is slowly being phased out in Canada and internationally. Based on reported use patterns, hexachloroethane is expected to be released mainly to air, with smaller releases to water and soil.

Based on its physical and chemical properties, hexachloroethane is not expected to degrade quickly in the environment and is persistent in air, water and soil. Hexachloroethane has a moderate potential to accumulate in organisms; however, there is no evidence that the substance will biomagnify in trophic food chains. Empirical acute aquatic toxicity values indicate that the substance is highly hazardous to aquatic organisms.

For the ecological screening assessment, a number of models were run, using conservative scenarios and assumptions, to determine concentrations of hexachloroethane in air, water and sediment. The predicted environmental concentrations (PECs) in water and air do not exceed concentrations associated with effects. Although the available measured concentrations of hexachloroethane in sediments exceeded effect levels, the data are older and the concentrations were from an area where known releases have ceased and where remediation activities have taken place.

Considering all available lines of evidence presented in this Screening Assessment, there is low risk of harm to organisms and the broader integrity of the environment from hexachloroethane. It is conclude that hexacloroethane does not meet the criteria under paragraph 64(*a*) or (*b*) of CEPA as it is not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends.

The principal source of exposure of the general population to hexachloroethane is indoor air. Intakes from ambient air, drinking water and soil are expected to be negligible. Food was not considered to be a source of hexachloroethane exposure.

Based principally upon the weight of evidence evaluations of international agencies, a critical effect for the characterization of risk to human health is carcinogenicity. Following chronic oral exposure to hexachloroethane, significant increases in the incidence of liver tumours and kidney tumours were observed in mice and rats, respectively. Increased incidences of pheochromocytomas of the adrenal gland were also observed in hexachloroethane-exposed rats. However, results of assays for genotoxicity were generally negative. Other effects observed in experimental animals exposed to hexachloroethane include non-cancer effects in the kidney, as well as developmental toxicity at higher levels of exposure.

The margin between the estimates of intake of hexachloroethane by the general population and the critical non-cancer effect level for renal toxicity in experimental animals is considered adequate to account for uncertainty in the health effects and exposure databases.

On the basis of the adequacy of margins between exposure and critical effect levels, it is concluded that hexachloroethane does not meet the criteria set out in paragraph 64(c) of CEPA as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

Conclusion

Based on the information available, it is concluded that hexachloroethane does not meet any of the criteria set out in section 64 of CEPA.

Table of Contents

1. Introduction

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA) (Canada 1999),the Minister of the Environment and Climate Change and the Minister of Health conduct screening assessments of substances to determine whether these substances present or may present a risk to the environment or to human health.

A screening assessment was undertaken on hexachloroethane (Chemical Abstracts Service [CAS] Registry No. 67-72-1), a substance on the Domestic Substances List (DSL).Hexachloroethane was identified as priority for assessment during the categorization of the DSL as it met the criteria for persistence, bioaccumulation and inherent toxicity to aquatic life. Hexachloroethane was also classified by other agencies as a possible carcinogen to humans according to IARC (Group 2B) and the USEPA (Group C).

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

A draft State of the Science Report for a human health screening assessment and associated unpublished supporting working documentation were prepared in 2004 by Existing Substances Division of Health Canada. The draft report was externally reviewed for adequacy of data coverage and defensibility of the conclusions. The draft State of the Science Report was posted on the Health Canada website in January 2006. This screening assessment includes an update of the State of the Science Report with respect to human health aspects, along with consideration of ecological aspects.

This screening assessment includes consideration of information on chemical properties and uses to hexachloroethane and hazards associated with exposure to hexachloroethane. Data identified as of March 2013 and June 2014 was considered for inclusion in the human health and ecological sections of this report, respectively. In addition, an industry survey was conducted in 2000 through a *Canada Gazette* Notice issued under authority of section 71 of CEPA; this survey collected data on the

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

Canadian manufacture and import of hexachloroethane (Canada 2001). Key studies were critically evaluated and modelling results have been used to reach conclusions.

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). 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 and Climate Change Canada, and the content was reviewed by senior staff for adequacy of data and coverage and defensibility of the evaluation. The ecological portions of this report were also reviewed by Canadian experts selected from government and academia. Additionally, the draft of this screening assessment was subject to 60-day public comment period. Although external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment and Climate Change Canada.

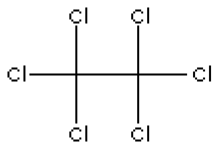
The critical information and considerations upon which this report is based are summarized below.

2. Substance Identity

Information relevant to the identity of hexachloroethane is presented in Table 1.

Table 1. Substance identity

CAS RN	67-72-1
DSL name	Hexachloroethane
NCI[1] names	Carbon hexachloride, ethane hexachloride, ethylene hexachloride, hexachlorethane, 1,1,1,2,2,2-hexachloroethane, hexachloroethylene, perchloroethane
Other names	Avlothane, Caswell No. 479, Distokal, Distopan, Distopin, Egitol, Falkitol, Fasciolin, Fron 110, Phenohep, Mottenhexe, NCI-C04604, NSC 9224
Chemical group (DSL stream)	Discrete organics
Major chemical class or use	Ethanes
Chemical formula	C_2Cl_6

Chemical structure	
SMILES	C(C(Cl)(Cl)Cl)(Cl)(Cl)Cl C(C(Cl)(Cl)Cl)(Cl)(Cl)Cl
Molecular mass	236.7 g/mol

Abbreviations: CAS RN, Chemical Abstracts Service Registry Number; DSL, Domestic Substances List; NCI, National Chemical Inventories; SMILES, simplified molecular input line entry system.

[1] 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; ELINCS, European List of Notified Chemical Substances; ENCS, Japanese Existing and New Chemical Substances; PICCS, Philippine Inventory of Chemicals and Chemical Substances; TSCA, *Toxic Substances Control Act* Chemical Substance Inventory.

Source: NCI (2006)

3. Physical and Chemical Properties

Table 2 summarizes data on the experimental and modelled physical and chemical properties of hexachloroethane that are relevant to its environmental fate.

Table 2. Experimental and modelled physical and chemical properties for hexachloroethane

Property	Type	Value	Temperature (°C)	Reference
Melting point (ºC)	Modelled	0.01		MPBPWIN 2008
Melting point (ºC)	Experimental[1]	185		Gerhartz et al. 1985; Lewis 1993
Boiling point (ºC)	Modelled	154.45		MPBPWIN 2008
Boiling point (ºC)	Experimental	185		Gerhartz et al. 1985; Lewis 1993
Vapour pressure (Pa)	Modelled	8.02	20	MPBPWIN 2008
Vapour pressure (Pa)	Experimental	28–29	20	Grayson and Eckroth 1979; Mackay and Shiu 1981; Verschueren 1983; Konietzko 1984; Gerhartz et al. 1985
Henry's Law constant ($Pa \cdot m^3/mol$)	Modelled	6.23×10^1	25	HENRYWIN 2008
Henry's Law constant ($Pa \cdot m^3/mol$)	Experimental	1.71×10^2		BUA 1989

Property	**Type**	**Value**	**Temperature (°C)**	**Reference**
Log K_{ow} (dimensionless)	Modelled	4.03		KOWWIN 2008
Log K_{ow} (dimensionless)	Experimental	3.34–5.31		Callahan et al. 1979; Könemann et al. 1979; McDuffe 1981; Veith et al. 1983; Curtis 1984; Konietzko 1984; Chiou 1985; Hansch and Leo 1985; Tu et al. 1985; Munz and Roberts 1987; Samiullah 1990; Ptacek and Gillham 1992; IPCS 2004
Log K_{oc} (dimensionless)	Modelled	3.59		PCKOCWIN 2008
Log K_{oc} (dimensionless)	Experimental	2.24–4.3		Lyman et al. 1982; Mabey et al. 1982; Sabljic 1984; Abdul et al. 1987; Oliver 1987
Water solubility (mg/L)	Modelled	1.47	25	WSKOWWIN 2008
Water solubility (mg/L)	Experimental	3.7	25	Grayson and Eckroth 1979

Abbreviations: K_{oc}, organic carbon–water partition coefficient; K_{ow}, octanol–water partition coefficient.
[1.] Sublimes without decomposition.

4. Sources

A survey conducted pursuant to section 71 of CEPA indicated that in 2000, approximately 150 tonnes of hexachloroethane were manufactured and 10–100 tonnes were imported (Environment Canada 2001). Although it is not manufactured for commercial distribution, hexachloroethane is formed during other processes in the chlorinated chemical industry; for example, this chemical is a by-product resulting from the 1, 2-dichloroethane manufacturing process (Environment Canada 2001). The hexachloroethane is then collected and thermally oxidized, and products of this destruction are used for other processes within the manufacturing company, such as producing aqueous hydrochloric acid solution, which is used internally. Some of the hexachloroethane produced can undergo sporadic and non-intentional mixing with inert solid materials (e.g., during maintenance activities); however, this contaminated material is sent off-site for disposal.

Hexachloroethane can also be produced as a by-product of the chlorination of water and sewage and the incineration of chlorinated hydrocarbons (Abrams et al. 1975; Class and Ballschmiter 1986; Howard 1989).

5. Uses

Hexachloroethane was reported to be used in Canada as a chemical intermediate, as a flux agent for grain refining and degassing of aluminum alloys, and as a flame retardant in industrial laminating resins. It was also reported to be used as a reactant in military smoke ammunition (Environment Canada 2001). Other uses of hexachloroethane noted in earlier scientific and technical literature were in military pyrotechnics, in the metallurgical industry, as a plasticizer, as an ignition suppressant, as a processing aid in various industrial processes, as a component of fungicidal and insecticidal formulations, and (formerly) as an anthelmintic in veterinary medicine (Kirk-Othmer 1993; ATSDR 1997; IARC 1999; DND 2001; HSDB 2003, US EPA 2011). The use of hexachloroethane in cosmetics appears on the List of Prohibited and Restricted Cosmetic Ingredients (more commonly referred to as the Cosmetic Ingredient Hotlist or simply the Hotlist), an administrative tool that Health Canada uses to communicate to manufacturers and others that certain substances, when present in a cosmetic, may contravene (a) the general prohibition found in section 16 of the *Food and Drugs Act* or (b) a provision of the *Cosmetic Regulations* (Health Canada 2011). Hexachloroethane is not used in pesticide formulations in Canada (2013 personal communication from Pest Management Regulatory Agency, Health Canada; unreferenced). Also, currently in Canada, hexachloroethane is not present in veterinary products (2013 personal communication from Veterinary Drugs Directorate, Health Canada; unreferenced), it is no longer used in military smoke ammunition, and no evidence has been found for its current use as a flame retardant (2013 personal communication from Risk Management Bureau Health Canada; unreferenced). Hexachloroethane is not an approved food additive in Canada and was not present in various regulatory food databases (Canada 2013, FDA 2013a, FDA 2013b, FAO 2013a, FAO 2013b). It does however continue to be imported into Canada for use as a degassing agent for oxides and hydrogen elimination from aluminum alloys during die casting at a quantity of less than 2000 kg per year (2013 personal communications from Risk Management Bureau, Health Canada; unreferenced).

The production and uses of hexachloroethane are being phased out internationally. The European Commission prohibits the use of hexachloroethane in the manufacturing or processing of non-ferrous metals (CEC 2001). In the United States, there has been a trend away from using hexachloroethane flux in the secondary aluminum industry (Strueter 1999). Similarly, representatives of the aluminum industry in the United States report that hexachloroethane is no longer used in most primary aluminum degassing (CGLI 1999). The Aluminum Association of Canada has also reported that its members do not use hexachloroethane in their activities (primary aluminum industry) (2005 personal communication from Environmental Protection Branch, Quebec Region, Environment Canada; unreferenced).

It was reported that hexachloroethane may be a constituent of lubricating greases and oils, non-structural caulking compounds and sealants, automotive chemicals, laundry and ironing aids and dry cleaning agents, but no quantitative data were provided (Scorecard 2005).

6. Releases to the Environment

Current Canadian sources of releases to the environment are minor but potentially numerous. They include possible releases from industrial facilities during manufacturing and processing, from the chlorination of water and sewage, from the incineration of chlorinated hydrocarbons, from municipal and industrial landfills by leaching and from the use of contaminated solvents (e.g., tetrachloroethylene).

Releases of hexachloroethane reported by Canadian industries to the National Pollutant Release Inventory (NPRI) are presented in Table 3 (NPRI 2003, 2013). There have been no releases, at reporting thresholds, since 2006, and, prior to that year, all releases occurred to air and off-site disposal. The NPRI database indicates that incineration and containment landfills are used for the disposal of hexachloroethane (NPRI 2013). From 1999 to 2005, on-site releases ranged from 0.001 to 0.012 tonne per year, and 0.004–19 tonnes per year were released to off-site disposal. Releases are much lower than total imported and manufactured amounts, as most of the quantity reported was associated with a destructive use. Hexachloroethane is expected to be released by users (rather than producers) mostly to air, with smaller releases to water and soil.

Releases of hexachloroethane associated with the die casting of aluminum products are expected to be minimal given the nominal amounts being used and the manner of processing (2013 personal communications from Risk Management Bureau, Health Canada; unreferenced).

Table 3. Releases of hexachloroethane relevant to Canada

Source and location	Period	On-site releases (kg)	Receiving medium	Transfer for disposal (kg)	Reference
Dow Chemical, Sarnia, Ontario[1]	1997	0	–	227 (incineration)	NPRI 2013
Dow Chemical, Sarnia, Ontario[1]	1999	0	–	19 455 (incineration)	NPRI 2013
Dow Chemical, Fort Saskatchewan, Alberta[1]	1999	12	Air	0 (incineration)	NPRI 2003
Dow Chemical, Fort Saskatchewan, Alberta[1]	2000	5	Air	7 096 (incineration)	NPRI 2003
Dow Chemical, Fort	2001	3	Air	4 (incineration)	NPRI 2003

Source and location	**Period**	**On-site releases (kg)**	**Receiving medium**	**Transfer for disposal (kg)**	**Reference**
Saskatchewan, Alberta[1]					
Dow Chemical, Fort Saskatchewan, Alberta[1]	2002	1	Air	3 918 (incineration)	NPRI 2003
Dow Chemical, Fort Saskatchewan, Alberta[1]	2003	8	Air	0	NPRI 2013
Dow Chemical, Fort Saskatchewan, Alberta[1]	2004	1	Air	0	NPRI 2013
Dow Chemical, Sarnia, Ontario	2004	1	Air	4928	NPRI 2013
Dow Chemical, Fort Saskatchewan, Alberta[1]	2005	1	Air	0	NPRI 2013
Dow Chemical, Fort Saskatchewan, Alberta[1]	2006–2011	0	Air	0	NPRI 2013
Nexen Chemicals Canada Limited Partnership, Nanaimo, B.C.[1]	2003	0	–	25 (landfill)	NPRI 2003
Aluminum foundries, Indiana, Ohio, Michigan (United States)	1999	18 366	Air	–	Great Lakes Commission 2002
Industrial (United States)	2003	474.9	Air emissions	0	TRI 2003
Industrial (United States)	2003	115.2	Underground injection	0	TRI 2003
Industrial (United States)	2003	3.6	Surface water discharge	0	TRI 2003
Industrial (United States)	2003	0	Off-site landfill	117.9	TRI 2003
Industrial (United States)	2003	0	Total Off-site management	121.1	TRI 2003
Industrial (United States)	2011	467.2	Air emissions	0	TRI 2013
Industrial (United States)	2011	75.7	Underground Injection	0	TRI 2013
Industrial (United States)	2011	142.4	On site and off site Landfills	22.2	TRI 2013
Industrial (United States)	2011	0	Other off site management	644.1	TRI 2013

[1] Facility is no longer operational (2013 personal communication from Risk Management Bureau, Health Canada; unreferenced).

In the 2002 Inventory of Toxic Air Emissions for the Great Lakes Region, 251 kg of hexachloroethane was reported to be released from a point source (Great Lakes Commission 2006). From 2011 to 2013, (the most recent years for which complete reviewed data are available), Dow Chemical Canada reported no releases or disposal of hexachloroethane (NPRI 2013). The US Toxics Release Inventory reported that 467.2 kg of hexachloroethane were emitted to the atmosphere, 75.7 kg injected to underground wells, 164.6 kg to on site and off site landfills and 644.1 kg were released to other off site management facilities in 2011 (TRI 2013).

7. Environmental Fate and Behaviour

7.1 Environmental Distribution

The results of Level III fugacity modelling (EQC 2003) are summarized in Table 4.

Table 4. Results of the Level III fugacity modelling of hexachloroethane (EQC 2003)

Substance released to:	**Fraction of substance partitioning to each medium (%)**			
	Air	**Water**	**Soil**	**Sediment**
Air (100%)	88.7	1.16	9.57	0.541
Water (100%)	13.1	58.4	1.41	27.1
Soil (100%)	0.965	0.08	98.9	0.038

Based on its experimental vapour pressure of 28–29 Pa and Henry's Law constant of 171 Pa·m^3/mol, hexachloroethane is volatile. Therefore, if released solely to air, it will tend to reside in this compartment, with smaller amounts partitioning to soil and negligible amounts to water and sediment (Table 4).

If released into water, hexachloroethane is expected to remain in this compartment based upon its moderate to high experimental log organic carbon–water partition coefficient (K_{oc}) of 2.24–4.3, although a large portion is also expected to adsorb to sediments and approximately 1% to soils. Volatilization from water surfaces is expected to be a significant fate process based upon this compound's experimental Henry's Law constant. Thus, if water is a receiving medium, hexachloroethane is expected to reside mainly in water and sediment and to some extent partition to air (Table 4).

If released to soil, hexachloroethane is expected to have moderate to high adsorptivity to soil (i.e., expected to be relatively immobile) based upon its experimental log K_{oc}. Volatilization from moist soil surfaces seems to be an unimportant fate process based upon its experimental Henry's Law constant. This chemical may slightly volatilize from dry soil surfaces based upon its vapour pressure. Therefore, if released to soil, hexachloroethane will reside mainly in this environmental compartment, which is illustrated by the results of the Level III fugacity modelling (Table 4).

These results represent the partitioning of the substance in a hypothetical evaluative environment resulting from intermedia partitioning and loss by both advective transport (out of the modelled region) and degradation/transformation processes. The partitioning values shown in Table 4 represent the net effect of these processes under conditions of continuous release when a non-equilibrium “steady state” has been achieved.

7.2 Environmental Persistence

Hexachloroethane is expected to persist in air, water and soil, based on mineralization half-lives presented in Table 5.

Table 5. Empirical and modelled data for persistence of hexachoroethane

Medium	Fate process	Degradation value	Endpoint (units)	References
Air	Mineralization	30[1]	Half-life (years)	Callahan et al. 1979
Air	Mineralization	>73[1]	Half-life (years)	Howard et al. 1991
Surface water	Volatilization	70[1]	Half-life (hours)	Spanggord et al. 1985
Surface water	Transformation	12[1]	Half-life (days)	Curtis and Reinhard 1994
Surface water	Mineralization	6–24[1]	Half-life (months)	Howard et al. 1991
Groundwater	Transformation	40	Half-life (days)	Criddle et al. 1986
Groundwater	Mineralization	12[1]	Half-life (months)	Howard et al. 1991
Groundwater	Mineralization	12	Half-life (months)	Kriegman-King and Reinhard 1991
Soil	Mineralization	6[1]	Half-life (months)	Howard et al. 1991
Sediment	Biodegradation	19.7[2]	Half-life (minutes)	Jafvert and Wolfe 1987

[1] Modelled/estimated values (as mentioned in the references).

[2] This very low value is surprising. It might be explained by experimental conditions; for example, the sediments contained high levels of acid-soluble iron (2–4 mg/g), which is thought to be involved with hexachloroethane degradation.

Among the environmental degradation products of hexachloroethane are tetrachloroethylene and trichloroethylene, two substances listed on Schedule 1 of CEPA (Canada 1993a,b).

In air, hexachloroethane is expected to persist, based on mineralization half-lives presented in Table 5 (Callahan et al. 1979; Howard et al. 1991). It is also a candidate for long-range transport, based on a very long characteristic travel distance of hexachloroethane estimated by TaPL3 fugacity modelling of 1.8×10^6 km (TaPL3 2000).

Hexachloroethane is expected to also persist in surface waters, with half-lives ranging from 70 hours as a result of volatilization (Spanggord et al. 1985) to 12 days for transformation (Curtis and Reinhard 1994) and up to 24 months for mineralization (Howard et al. 1991).

In soil, degradation of hexachloroethane is not expected to be significant. Howard et al. (1991) reported a half-life of 6 months, whereas Spanggord et al. (1985) reported a loss of 99% of hexachloroethane after 4 days of incubation anaerobically and after 4 weeks in aerobic soils.

Hexachloroethane was reported to be transformed in an unconfined sand aquifer, with a half-life of 40 days (Criddle et al. 1986). Laboratory studies showed evidence of microbial reduction of hexachloroethane to tetrachloroethylene under aerobic conditions in this aquifer.

7.3 Potential for Bioaccumulation

Although hexachloroethane is not considered to be a highly bioaccumulative substance, it does have the potential to bioaccumulate to some degree. A critical review of published bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) in the literature by Gobas and Arnot (2003) identified the highest validated BAF for aquatic organisms as being 1 513 for rainbow trout (*Oncorhynchus mykiss*) (Oliver and Niimi 1983). The other reported BAFs for fish varied between 661 and 1202 (Burkhard et al. 1997). BCFs in the range of 138–1 200 have also been reported for fish (Veith et al. 1980; Oliver and Niimi 1983; Smith et al. 1988; Wang et al. 2008). Liu et al. (2006) developed two quantitative structure–property relationship (QSPR) models that were used to predict the BCF for hexachloroethane. The experimental BCF of 832 obtained by Liu et al. (2006) is in agreement with the predicted values of 348 and 112. It has been stated that because of hexachloroethane's rapid metabolism and low incidence of water contamination, it is not likely to bioaccumulate in the food chain (ATSDR 1997).

Hexachloroethane is rarely detected in surface water or biota. However, concentrations in fish between 0.01 and 0.06 ng/g were measured in adult rainbow trout from Ganaraska River, a tributary of Lake Ontario, in the spring of 1981 (Oliver and Niimi 1983). No information was found on hexachloroethane concentrations in animals located higher in the food chain. The half-life for hexachloroethane in tissue of bluegill (*Lepomis macrochirus*) has been reported to be <1 day (Barrows et al. 1980). Tissue clearance data indicated that hexachloroethane was eliminated by the rat with a half-life of between 2 and 3 days (Gorzinski et al. 1985). However, other studies have shown slower metabolism of hexachloroethane in rabbits (Jondorf et al. 1957) and sheep (Fowler 1969). It has been stated that because of hexachloroethane's rapid metabolism and low incidence of water contamination, it is not likely to bioaccumulate in the food chain (ATSDR 1997).

8. Potential to Cause Ecological Harm

8.1 Ecological Exposure Assessment

Hexachloroethane is expected to be found throughout Canada, given its persistence in air, its potential for long-range transport and its numerous sources dispersed throughout the country. Hexachloroethane is not routinely monitored by Canadian provincial/territorial or federal regulatory agencies. No concentration data were found for hexachloroethane in Canadian air, and hexachloroethane was not detected in Canadian soil samples studied (Gizyn 1994; Webber 1994). Concentrations of hexachloroethane in Canadian water were measured in a few different water bodies and water treatment plants in Ontario between 1983 and 2002. Sediment concentrations were identified only for the St. Clair River in Ontario and date from 1985 to 2001. In order to address data gaps, environmental concentrations were predicted using models.

The ChemCAN model (ChemCAN 2003) was used to investigate regional concentrations. ChemCAN is a Level III fugacity model containing a database of environmental properties for 24 regions of Canada. It can estimate average steady-state concentrations of chemicals in air, fresh surface water, fish, sediments, soils, vegetation and marine nearshore waters based on input release data and physicochemical properties (ChemCAN 2003). To run the model, a reasonable worst-case scenario was developed based on known uses of hexachloroethane and releases given in Table 3. These included releases to air from non-ferrous metal facilities (secondary aluminum), other industrial sources, an incinerator, military smoke use, contaminated solvent use and long-range transport (total 273 220 kg/year). Releases to water originated from industrial activities and from water treatment plants (total 22 502 kg/year), and releases to soil considered releases from industrial activities (total 18 309 kg/year). Release quantities were estimated based on minimal requirements for NPRI reporting (threshold of 10 000 kg/year per facility), assuming that maximum amounts of substance used would be released, and the number of facilities contained in the industrialized zone of interest. When relevant, information from the United States was also included. Additionally, elevated background concentrations were used. Hexachloroethane was modelled as a type 2 chemical, using average physicochemical properties (from Table 2) and conservative half-lives (from Table 5), in Ontario Mixed Wood Plain and Northern Manitoba zones.

The possible concentration of hexachloroethane in air in the vicinity of dischargers was also investigated using the model SCREEN3 (2003), which was developed by the US Environmental Protection Agency and is a single-source Gaussian plume model that provides maximum 1-hour concentrations for point, area, flare and volume sources at receptor height. A general scenario was built using the volume source option (fugitive emissions) for a building 100 m × 100 m × 15 m. Releases of 73 900 kg/year were used to represent releases of 50% of total hexachloroethane produced by the single largest hexachloroethane-producing company in Canada. Releases were estimated to occur 18 hours/day, 350 days/year, giving a release rate of 3.26 g/s. The model was run for an urban setting with full meteorology and simple terrain. The receptor height was 0.1 m.

The possible concentration of hexachloroethane in water in the vicinity of dischargers was also investigated. Local aquatic dispersion scenarios were investigated using ChemSim, a geographic information system–based aquatic exposure estimation tool (CHC 2003). ChemSim combines estimated release quantities with information regarding the receiving watercourse to estimate aquatic exposure values. Again, the scenario considered the single largest producing facility releasing 1.35 kg/day in the North Saskatchewan River. These releases correspond to 1% of the total amount produced, released continuously over 350 days/year. These should account for releases from processes, handling and cleaning procedures. The retained scenario assumes releases to the watercourse through a sewage treatment facility, where a removal efficiency of 68% is anticipated (based on EPIWIN 2000). The concentration obtained at a distance of 250 m from the discharge point, using the 10th-percentile low-flow state, was selected.

Historical and predicted concentrations of hexachloroethane in the environment (relevant to Canada) are presented in Table 6.

Table 6. Environmental concentrations of hexachloroethane in locations relevant to Canada (values obtained through modelling are shaded)

Medium	Location	Sampling period	Mean concentration[1]	Reference
Air (troposphere)	Western Canada (rural areas)	April 2001 – December 2002	15.58 ng/m^3 (1480 ng/m^3 max.)	You et al. 2008
Air (troposphere)	Northern hemisphere	1982–1985	2.9 ng/m^3	Class and Ballschmiter 1987
Air (troposphere)	Ontario – Plain	–	8.13 × 10^{-3} ng/m^3	ChemCAN 2003
Air (troposphere)	Manitoba – Northern	–	1.45 × 10^{-2} ng/m^3	ChemCAN 2003
Air (troposphere)	Local air dispersion	–	1.215 × 10^6 ng/m^3 (max.)	SCREEN3 2003
Sediments	St. Clair River	1985	1.4–530 ng/g ss	Oliver and Kaiser 1986
Sediments	St. Clair River tributaries	1985	2.0–41 ng/g ss	Oliver and Kaiser 1986
Sediments	St. Clair River – Canada	1985	ND–2.9 × 10^5 ng/g	Oliver and Pugsley 1986
Sediments	St. Clair River – USA	1985	ND–0.8 ng/g	Oliver and Pugsley 1986
Sediments	St. Clair River – Canada	1994	<1–3100 ng/g	BII 1997

Medium	Location	Sampling period	Mean concentration[1]	Reference
Sediments	St. Clair River – Canada	2001	ND–660 ng/g	2001 personal communication from Ontario Ministry of the Environment; unreferenced
Sediments	Ontario – Plain	–	9.11 ng/g	ChemCAN 2003
Sediments	Manitoba – Northern	–	14.4 ng/g	ChemCAN 2003
Soil	Military (0–7 m from canister)	1985	6054 ng/g	Schaeffer et al. 1988
Soil	Ontario – Plain	–	20.9 ng/g	ChemCAN 2003
Soil	Manitoba – Northern	–	19.1 ng/g	ChemCAN 2003
Sludges from STPs	Canadian locations (Winnipeg, Hamilton, etc.)	1981–1985	Detected at a trace level (<1 µg/g) in 1 sample	Webber and Lesage 1989
Biosolids	Ontario	2002	3.0×10^5 ng/L (0.3 mg/L)	OME 2004
Water	St. Clair River	1985	0.94–1700 ng/L	Oliver and Kaiser 1986
Water	St. Clair River tributaries	1985	0.02–41 ng/L	Oliver and Kaiser 1986
Water	Lake Ontario	Before 1983	0.02 ng/L	Oliver and Niimi 1983
Water	Toronto waterfront	1987	0.009–0.030 ng/L	Halfon and Poulton 1992
Water	Toronto (tap water)	2003	<5 ng/L	City of Toronto, 2003a,b
Water	Ontario – raw water	1990–2002	0–6 ng/L	2003 personal communication from Government of Ontario; unpublished
Water	Ontario – treated water	1990–2002	0–46 ng/L	2003 personal communication from Government of Ontario; unpublished
Water	Ontario – raw sewage	1988	90 ng/L (max.)	Canviro Consultants 1988
Water	Ontario – Plain	–	145 ng/L	ChemCAN 2003
Water	Manitoba – Northern	–	229 ng/L	ChemCAN 2003
Water	North Saskatchewan River	–	741 ng/L	CHC 2003

Abbreviations: max., maximum; ND, not detected; ss, suspended sediments; STP, sewage treatment plant.

[1] Except where otherwise noted.

8.2 Ecological Effects Assessment

There is a satisfactory quantity of empirical toxicity data for hexachloroethane (Table 7). The majority of the ecotoxicity studies pertain to acute toxicity to aquatic organisms. Key studies have been critically reviewed and determined to have a satisfactory degree of reliability for the present risk assessment (Appendix 3). Even following short exposure times, hexachloroethane can be lethal to fish (median lethal concentrations [LC_{50}s] of 0.77–2.36 mg/L) (Call et al. 1983; Phipps and Holcombe 1985; Thurston et al. 1985; Russom et al. 1997) and aquatic invertebrates (LC_{50}s 1.36–2.70 mg/L) (Thurston et al. 1985). The toxicity to algae was reported (48-hour median effective concentration [EC_{50}] of 1.30–1.66 mg/L) (Hsieh et al. 2006). Katritzky et al. (2001) reported a predicted aquatic toxicity value of 1.53 mg/L. Acute lethality data for amphibians were also located in the literature (LC_{50} of 2.44 mg/L) (Thurston et al. 1985). No toxicity information was available for microbial organisms. Chronic toxicity data could be located only for fish. There is also a lack of data characterizing the effects of hexachloroethane on sediment-dwelling organisms and on terrestrial invertebrates and wildlife. Chronic exposure (32 days) of fathead minnow embryos/larvae to hexachloroethane at a concentration of 207 µg/L produced a decrease in weight gain (lowest-observed-effect concentration [LOEC] for growth). A concentration of 1604 µg/L produced 100% mortality (Ahmad et al. 1984).

Table 7. Empirical toxicity data for aquatic toxicity

Category	Organism	LC_{50} (95% CI) (mg/L)	Exposure duration	Reference
Aquatic invertebrate	*Daphnia magna* (<24 h)	1.36 (1.04–1.76)	48 h	Thurston et al. 1985
Aquatic invertebrate	*Tanytarsus dissimilis* (Chironomidae)	1.23 (1.07–1.42)	48 h	Thurston et al. 1985
Aquatic invertebrate	Crayfish (0.42 g)	2.70 (2.13–3.41)	96 h	Thurston et al. 1985
Fish	Rainbow trout (1.8 g)	0.77 (0.72–0.83)	192 h	Call et al. 1983
Fish	Rainbow trout (1.8 g)	0.97 (0.73–1.28)	96 h	Phipps and Holcombe 1985
Fish	Channel catfish (3.48 g)	2.36 (1.9–2.9)	96 h	Thurston et al. 1985
Fish	Fathead minnow (0.44 g)	1.24 (0.967–1.25)	96 h	Thurston et al. 1985
Fish	Fathead minnow (0.28 g)	1.42	96 h	Russom et al. 1997
Amphibian	Tadpole (4.21 g)	2.81 (1.47–4.06)	96 h	Thurston et al. 1985

Abbreviations: CI, confidence interval; LC_{50}, median lethal concentration.

Chronic sublethal toxicity experiments (inhalation) on small terrestrial mammals and birds have been used to estimate concentrations at which no effects are observed: 465 mg/m^3 and 2517 mg/m^3 for mammals and birds, respectively (Weeks et al. 1979). Oral toxicity studies on rodents (Weeks et al. 1979) have shown that a single dose of hexachloroethane can be lethal to rats (median lethal dose [LD$_{50}$] = 4460 mg/kg body weight [kg-bw]), but a dose of 100 mg/kg-bw per day provokes no effect in the rabbit exposed for 12 days. Doses of 15 mg/kg-bw per day and 62 mg/kg-bw per day provoked slight effects on body weight, liver and kidney in male and female rats, respectively, over a 16-week exposure (Gorzinski et al. 1985). Also, doses of 10 mg/kg-bw per day over a 2-year period caused changes in the tissues of the kidney in the male rat (NTP 1989). The latter concentrations are unlikely to be achieved in food sources of lower terrestrial mammals; therefore, the food pathway was not judged to be of concern. Also, acute sublethal toxicity studies on trees (Sadusky et al. 1993) allowed us to estimate a no-observed-effect concentration (NOEC): 1400 mg/m^3.

8.3 Characterization of Ecological Risk

The approach taken in this ecological screening assessment is to examine various supporting information and develop conclusions based on a weight of evidence approach as required under CEPA. Particular consideration has been given to risk quotient analyses, as well as persistence, bioaccumulation, and trends in ambient concentrations.

8.3.1 Risk Quotient Analysis

Critical exposure and effects results and risk quotients are summarized in Table 8 and described in more detail below.

Table 8. Calculation of risk quotients from measured and modelled exposure values for organisms in water, sediments, soil and air

Medium	**Organism**	**CTV**	**Reference**	**PNEC**	**$\text{PEC}_{\text{model}}$**	**$\text{PEC}_{\text{measured}}$**	**RQ_{model}**	**$\text{RQ}_{\text{measured}}$**
Water	*Daphnia magna*	1.36 mg/L	Thurston et al. 1985	1.36 µg/L	0.229 µg/L (Manitoba)	0.606 µg/L	0.17	0.45
Water	Rainbow trout	0.77 mg/L	Call et al. 1983	0.77 µg/L	0.229 µg/L (Manitoba)	0.606 µg/L	0.30	0.79
Water					0.741 µg/L[1] (local)	–	0.96	–
Sediment	Estimated value (equilibrium partitioning method)	–	–	91 µg/kg	14.4 µg/kg (Manitoba)	903 µg/kg	0.16	9.9
Air	Guinea	465[2]	Weeks et	465	0.000 015	1.48	3.2 ×	3.2 ×

Medium	**Organism**	**CTV**	**Reference**	**PNEC**	**PEC$_{model}$**	**PEC$_{measured}$**	**RQ$_{model}$**	**RQ$_{measured}$**
	pig, rat, Beagle dog	mg/m^3	al. 1979	µg/m^3	µg/m^3 (Manitoba)	µg/m^3	10^{-8}	10^{-3}
Air	Rat	2517[3] mg/m^3	Weeks et al. 1979	2517 µg/m^3	1215 µg/m^3 (local)	–	0.48	–
Air	Black locust and black cherry tree	1400 mg/m^3	Sadusky et al. 1993	1400 µg/m^3	1215 µg/m^3 (local)	1.48 µg/m^3	0.87	1.1×10^{-3}

Abbreviations: CTV, critical toxicity value; PEC, predicted exposure concentration; PNEC, predicted no-effect concentration; RQ, risk quotient.

[1] The local scenario is presented only for the most sensitive aquatic organism, for clarity reasons.

[2] Subchronic NOEC inhalation value of 48 ppm converted to mg/m^3 (1 ppm = 9.68 mg/m^3; from Verschueren 1983).

[3] Acute (8-hour) NOEC inhalation value of 260 ppm converted to mg/m^3 (1 ppm = 9.68 mg/m^3; from Verschueren 1983).

A risk quotient analysis, integrating known or potential exposures with known or potential adverse environmental effects, was performed for each relevant compartment. This consisted of first selecting a critical toxicity value (CTV) for different species in all compartments, from which a predicted no-effect concentration (PNEC) was derived by applying an application factor of 100 to account for interspecies and intraspecies variability, extrapolation of results from laboratory to field and extrapolation from acute to chronic toxicity. For sediments, the PNEC was estimated based on the most sensitive aquatic invertebrate and the equilibrium partitioning method. Conservative predicted exposure concentrations (PECs) were estimated for each medium according to values presented in Table 6: one based on modelled concentrations of hexachloroethane obtained using a reasonable worst-case scenario of current releases (PEC$_{model}$) and one based on measured concentrations from historical data (PEC$_{measured}$). A risk quotient value of greater than 1 suggests the possibility of adverse effects.

The PEC$_{model}$ corresponds to the highest concentration for each medium obtained in either the Ontario or Manitoba zone using the ChemCAN model (ChemCAN 2003). For air and water, an additional PEC$_{model}$ was obtained for local dispersion in the vicinity of a discharger. The PEC$_{measured}$ for air corresponds to the maximum concentration presented in Table 6. For water, an average concentration within about 1 km of the Dow Chemical outfall was calculated as a PEC$_{measured}$ (606 ng/L), based on 1985 results (Oliver and Kaiser 1986). This value is intended to represent the average concentration within a fish home range. Choosing the highest measured value in water given in Table 6 (1700 ng/L) would have been too unrealistic for current times, because this concentration was directly associated with effluents of Dow Chemical at the time. Since then, it has been stated that Dow Chemical has removed the plant from direct contact with the river (BPAC and RAP 1995), and more recent NPRI data show no release of hexachloroethane to water by Dow Chemical (NPRI 2003). Additionally, of the 21 measurements taken in the St. Clair River in 1985, only 3 had values greater than or equal to 110 ng/L (median = 14 ng/L, average = 127 ng/L).

For sediments, hexachloroethane concentrations from 2001 were deemed most representative of the current situation (2001 personal communication from Ontario Ministry of the Environment; unreferenced). Given the physical and chemical properties of hexachloroethane (log octanol–water partition coefficient [K_{ow}] of 3.34–5.31, log K_{oc} of 2.24–4.3 and water solubility of 3.7–99 mg/L), hexachloroethane would have a tendency to partition into the soil pore water and possibly present a risk to benthic organisms, although a portion may adsorb to soil particles. The $PEC_{measured}$ for sediments was determined based on sediment concentrations resulting from sampling in 1994, located closer to Dow Chemical's outfall (BII 1997). The average concentrations at sampling stations downstream of the discharge were 1232 ng/g (0 m), 710 ng/g (92 m), 478 ng/g (158 m) and 903 ng/g (260 m). Since it was judged that the concentration directly below the outfall would be too unrealistic, the next highest average of measurements (903 ng/g at a distance of 260 m) was selected as another $PEC_{measured}$ for sediments. Table 8 summarizes these values, as well as the risk quotients (PEC/PNEC) obtained.

8.3.2 Consideration of Lines of Evidence and Conclusion

Based on its physical and chemical properties, hexachloroethane is not expected to degrade quickly in the environment and is persistent in air, water and soil. BAFs for hexachloroethane indicate that it is moderately bioaccumulative, but available data do not indicate a high level of bioaccumulation or biomagnification in the environment. This substance is expected to partition to every medium and to undergo long-range transport. Empirical acute aquatic toxicity values also indicate that the substance is highly hazardous to aquatic organisms.

Although hexachloroethane is not used by many different types of industries, it may still be released by a number of point sources located throughout the country, but in small quantities. However, release data and information on commercial or industrial activities indicate a decrease in releases in the past decades.

Risk quotients (RQ_{model} for air, water, soil and sediment and $RQ_{measured}$ for air and water) indicate that hexachloroethane concentrations likely do not exceed concentrations associated with effects, even when using conservative scenarios and assumptions. Some of the risk quotients are close to unity as a result of those conservative scenarios and assumptions used to calculate them. The $RQ_{measured}$ obtained for sediment indicate that measured concentrations of hexachloroethane in sediments located in an industrialized zone exceeded the PNEC by a factor of up to 9.9. This is likely an overestimate of the actual risk because this area is known to have been highly contaminated and thereafter remediated (BPAC and RAP 1995; Dow Chemical Co. 2001, 2002a,b, 2003, 2004).

Based on the information available, there is low risk of harm to organisms or the broader integrity of the environment from this substance. It is therefore concluded that hexachloroethane does not meet the criteria set out in paragraph 64(a) or (b) of CEPA as it is not entering the environment in a quantity or concentration or under conditions that 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.

8.3.3 Uncertainties in Evaluation of Ecological Risk

Uncertainties associated with the ecological screening assessment of hexachloroethane are discussed below.

Physicochemical Properties

The selection of physicochemical properties for the assessment yields some uncertainty, especially because of the lack of actual validated experimental results to quantify most physicochemical properties of hexachloroethane. These are important to predict the fate of a chemical in the environment; when such properties cannot be properly quantified, the conclusions will reflect this lack of knowledge. In order to mitigate this effect, conservative values were selected for half-lives (i.e. high values). In this way, our predictions would not underestimate environmental persistence and resulting concentrations. In cases where selection of the most conservative value was not obvious because of varying effects in different media (e.g., log K_{ow}, log K_{oc}), an average of all values identified in the literature was used. This allowed us to have additional confidence in our results without having to select a single value. These approaches should decrease the impact of the lack of validated data on the outcome of the modelling exercise and subsequent conclusions.

Exposure Characterization

Because of a lack of current monitoring data, exposure estimates were based either on modelled or old monitoring data. The old monitoring data most likely are not representative of the current situation, as there are indications that the use of hexachloroethane has declined greatly over time. Also, some of the environmental concentrations were taken in the vicinity of highly contaminated areas, some of which have been subject to remediation.Because of the lack of current monitoring data, reasonable worst-case scenarios were developed for the risk analysis.

Effects Characterization

No literature concerning the toxicity of hexachloroethane to sediment-dwelling invertebrates could be located, and PNECs were calculated based on the most sensitive aquatic invertebrate and the equilibrium partitioning method. Similarly, almost no literature dealt with the effects of hexachloroethane on terrestrial organisms (plants, invertebrates, amphibians, birds, mammals). Although no literature exists for effects of hexachloroethane on terrestrial organisms, the rodent data used to estimate potential risk to the terrestrial environment indicated that little risk was likely. Effect values for sediment organisms had to be estimated based on aquatic invertebrate information. Although the sensitivities of benthic and aquatic organisms to hexachloroethane could be very different, this is unlikely. The use of the most sensitive invertebrate toxicity value to derive benthic toxicity and the use of high application factors when determining the PNEC for sediment are likely sufficient to yield conservative results.

Additionally, although no information was found on possible transfers within the food chain and on effects or concentrations in animals located higher in the food chain, this pathway was not judged to be of concern. Although hexachloroethane is moderately bioaccumulative, it is not believed to biomagnify, and oral toxicity effects are observed only when relatively high and unlikely concentrations in the food source are reached.

9. Potential to Cause Harm to Human Health

9.1 Exposure Assessment

9.1.1 Environmental Media and Food

Data pertaining to concentrations of hexachlorethane in drinking water, ambient water, indoor air, biota and soil was identified in Canada and other countries. The key studies used to quantify exposure to hexachloroethane from environmental media were used to develop exposure estimates and are outlined in Appendix 1.

Estimates of exposure to hexachloroethane for the general population range from 0.03 µg/kg-bw per day in the 0 – 0.5 years age group to 0.07 µg/kg-bw per day in the 0.5–4 years age group.

Ambient Air and Indoor Air

In ambient air, hexachloroethane has been detected in approximately 33% of 11 399 samples, with a mean concentration of 0.016 ug/m^3 (maximum = 1.5 ug/m^3) at sites associated with the primary oil and gas industry in Alberta, northeastern British Columbia and central and southern Saskatchewan between April 2001 to December 2002 (You et al 2008). The objective of the study was to evaluate the impact of emissions from facilities upon the surrounding farm industry. However it is important to note that monitoring sites were adjacent to pastures, and were not in the immediate vicinity of oil and gas vehicles and away from other emission sources such as farm vehicles. Hexachloroethane was also detected in ambient air (1 x 10^{-4} – 0.0035 ug/m^3) at various international sites (Class and Ballschmiter 1986, 1987) and in Oregon (0.0029 –0.0041 ug/m^3) during rain events (Ligocki et al 1985). The Canadian survey by You et al 2008 was used to quantify exposure from this medium.

A recent Canadian indoor air survey was conducted from 2009 to 2011 as part of the second cycle of the Canadian Health Measures Survey (CHMS), an ongoing national survey that collects important health information from individuals aged 3 to 79 years old living in private households (Statistics Canada 2012; Wheeler et al. 2013). Eighty-four volatile organic compounds, including hexachloroethane, were measured by survey participants who deployed the samplers in their homes for 7 consecutive days. A total of

3857 valid indoor air samplers, from various dwellings including houses, apartments, mobile homes, and hotels, and including both smoking and non-smoking occupants, were analysed from 18 sites across Canada. Hexachloroethane was detected in 0.78 % of households sampled with an arithmetic mean of 0.12 ug/m^3 and a 95th percentile of 0.13 ug/m^3 (2013 email from Environmental Health Sciences and Research Bureau, Health Canada to Existing Substances Risk Assessment Bureau, Health Canada). Hexachloroethane was also detected in the indoor air (0.1 – 0.5 ug/m^3) of 6 of 96 homes in Quebec City (Heroux et al. 2008) and in a survey 754 residential homes (max: 4.82 ug/m^3) across Canada (Fellin et al 1992). However two studies which surveyed indoor air from 75 homes in the Ottawa area and 15 garages and homes in Ann Arbor and Ypsilanti did not detect hexachloroethane (Zhu et al. 2005) (Batterman et al 2007). Finally, Otson et al. 1994 also did not detect hexachloroethane in 757 single family dwellings. The CHMS survey was used in this assessment to estimate exposure to hexachloroethane from indoor air due to the recent sampling period and large sample size.

Drinking Water, Soil, and Food

Hexachloroethane was detected in 7 out of 24 samples of tap water (max: 1.6 x 10^{-2} ug/L) in the Ottawa area (OME 1988). However it was not detected in 12 samples of tap water from Union, Ontario (OME 1989) and not detected in later surveys of Toronto drinking water (City of Toronto 2002abcd, 2003 abc). Hexachloroethane was also not detected in several surveys of soil

Hexachloroethane was detected in one northern pike sample taken from the Ashtabula River in Ohio (DeVaut 1985), however this data was not considered relevant for this exposure assessment. No other information as to its presence in food was identified.

Consumer Products

Hexachloroethane has not been identified, in consumer products, in Canada and exposure from this source is expected to be limited, as its use is being phased out or restricted in many countries. The presence of hexachloroethane in the headspace of both pure and diluted household bleach has been reported in Turkey (Odabasi 2008) however data from this study is inadequate to permit a quantitative estimate of potential exposure to hexachloroethane. However, indoor air monitoring data would take into account releases from consumer products used within the home environment.

Biomonitoring

Hexachloroethane was not detected in 1366 blood samples in the United States between the years of 2003 – 2004 (CDC 2009). Currently hexachloroethane has not been surveyed as part of the biomonitoring portion of the Canadian Health Measures Survey.

Confidence in exposure database

Overall confidence in the exposure database for determining hexachloroethane intake estimates from environmental media and food is considered moderate to high. Representative, high quality Canadian data were available for concentrations in indoor air resulting in high confidence in the intake estimates from this medium. Limited data was available quantifying hexachloroethane concentrations in ambient air, soil, and drinking water, however due to reduced use of hexachloroethane internationally and in Canada and conservative assumptions and parameters used (e.g.: maximum concentrations used for drinking water, use of data from surveys where significant number of samples show no detection) these media are considered to be minor contributors to total hexachloroethane intake. There is no data quantifying concentrations of hexachloroethane in breast milk; thus, contribution from breast milk was not captured in the dietary intake assessment for infants, which is an uncertainty. There is limited data quantifying concentrations of hexachloroethane in different food commodities; however its use pattern does not suggest that it would be used in the processing or packaging of food. For products the confidence in the exposure database is low since only one survey was identified as to its presence in products (detected in the headspace of a bleach product in Turkey), thus is an uncertainty. However it is important to note that hexachloroethane is no longer used in Canada in a majority of the uses reported (see Uses section). Additionally, no other information was indentified on theuse of heaxachloroethane in products.

Due to the fact that hexachloroethane use is being phased out internationally, its limited use in Canada, its non-detection in a majority of monitoring surveys, and conservative assumptions used in exposure scenarios (use of data from surveys where majority of samples show non-detection) it is likely that there is minimal exposure of hexachloroethane to the general population.

9.2 Health Effects Assessment

In an assessment prepared by the International Agency for Research on Cancer (IARC 1999), it was concluded that hexachloroethane was *possibly carcinogenic to humans* (Group 2B), based on *sufficient evidence* in experimental animals and *inadequate evidence* in humans. Similarly, the National Toxicology Program (NTP) of the United States has assessed hexachloroethane as “reasonably anticipated to be a human carcinogen” (NTP 2005), while the EPA (2011) similarly concluded that HCE was *likely to be carcinogenic to humans*. A statistically significant increase in the incidence of hepatocellular carcinomas was observed in male B6C3F1 mice exposed to a time-weighted average dose of 1179 mg/kg-bw per day by gavage for 78 weeks (Weisburger 1977; National Cancer Institute 1978). In females, an increase was only noted at the low dose of 590 mg/kg bw per day. There are uncertainties related to the relevance of these tumours, given the lack of a dose response in females.

There was a non-statistically significant increase in the incidence of kidney tubular cell adenomas in male Osborne-Mendel rats at a time-weighted average dose of 212 mg/kg-bw per day administered by gavage for 78 weeks (4/49 versus 0/20 in control group) (Weisburger 1977; National Cancer Institute 1978); however, there was no increase at 423 mg/kg-bw per day (0/29) in this study. High mortality[2] may have precluded the observation of late-developing tumours (National Cancer Institute 1978). In a later study with F344/N rats, there was a significantly increased incidence of renal tubular adenomas and carcinomas combined in male rats exposed by gavage to 20 mg/kg-bw per day for 2 years, with no significant differences in survival (NTP 1989). An increased incidence of pheochromocytomas of the adrenal gland was also observed in male rats, although the increase was significant only at the low dose (10 mg/kg-bw per day). There was no increase in tumour incidence in female rats exposed by gavage to up to 160 mg/kg-bw per day for 2 years compared with controls (NTP 1989). NTP (1989) concluded that there was “clear evidence” of carcinogenic activity of hexachloroethane in male rats based on the increased incidence of renal neoplasms and the possibly hexachloroethane-related marginally increased incidence of pheochromocytomas of the adrenal gland. Since the adrenal tumours did not follow a dose response trend Benchmark Dose calculations could not be performed. The lowest calculated $BMDL_{10}$ is 8.53 mg /kg-bw/day for renal adenoma or carcinoma (combined) in male F344 rats exposed to hexachloroethane by gavage. There was “no evidence” of carcinogenic activity in female rats.

In an initiation–promotion bioassay, there was no increase in preneoplastic lesions (i.e., gamma glutamyltranspeptidase-positive foci) observed in the liver of rats exposed to hexachloroethane at a dose of 500 mg/kg-bw per day by gavage, followed by 0.05% phenobarbital in the diet for 7 weeks. However, an increased incidence of preneoplastic lesions was observed in rats exposed intraperitoneally to diethylnitrosamine at 30 mg/kg-bw followed by hexachloroethane at 500 mg/kg-bw per day for 7 weeks (Story et al. 1986; Milman et al. 1988). In summary, results were negative in the initiation study and positive in the promotion study.

The pattern of renal histopathological effects (renal tubular hyperplasia, linear mineralization of the renal papillae and hyperplasia of the pelvic transitional epithelium) observed in male rats in the 2-year study, but not in female rats or mice of either sex, was consistent with those associated with alpha-2u-globulin nephropathy. In a 21-day study in male rats, hexachloroethane induced nephropathy, consisting of hyaline droplet accumulation and renal tubular regeneration, along with a dose-related increase in renal tubule cell labelling index (NTP 1996). However, the potential for reversible binding of hexachloroethane to a specifically identified protein was not investigated in either study. No studies have been identified that presented immunohistochemical evidence of the presence of alpha-2u-globulin in hyaline droplets in the kidneys of rats exposed to hexachloroethane. Thus, although evidence for a role of alpha-2u-globulin nephropathy in the induction of renal tumours in rats by hexachloroethane is suggestive, it is not conclusive. In addition, in a recent assessment, the US EPA (2011) examined the mode of action of the kidney tumours in male rats and similarly concluded that the evidence was insufficient to conclude that they were a result of alpha-2u-globulin accumulation.

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[2] 38% (19/50) of the high-dose male rats and 48% (24/50) of the low-dose male rats survived at least 90 weeks, compared with 70% (14/20) of the controls.

Furthermore, the potential mode of induction of the liver tumours in mice or adrenal tumours in rats by hexachloroethane have not been investigated. Since IRIS (2011) conducted an extensive mode-of-action and human relevance examination of the identified tumours, further examination of these issues is not included in this document and readers are directed to the IRIS document for further information.

The genotoxicity of hexachloroethane has been investigated in short-term screening assays addressing a range of endpoints (see Appendix 2). While identified *in vivo* studies are limited to a micronucleus test, an assay for deoxyribonucleic acid (DNA) damage in mice and DNA binding studies in rats and mice, *in vitro* data are more extensive. However, gene mutation tests in mammalian systems either *in vivo* or *in vitro* were not identified. With a few exceptions, all reported results were negative. The only positive results of potential significance were those in a single report in which DNA binding was reported *in vivo* and *in vitro*; however, there was no clear evidence of adduct formation (Lattanzi et al. 1988). Modelled predictions of the genotoxicity of hexachloroethane and related compounds were also generally negative (CASETOX 2003; DEREK 2003; TOPKAT 2004).

Although the mode of induction of tumours by hexachloroethane has not been well studied, the available data on genotoxicity is generally negative, suggesting that the mechanism of carcinogenicity in some target tissues may be non-genotoxic.

In the limited number of developmental studies identified, adverse fetal or maternal effects were reported, but at a higher dose level (Weeks et al. 1979) than that which resulted in an increased incidence of renal tubular adenomas and carcinomas combined (NTP 1989).

The lowest LOEC for non-neoplastic effects is 465 mg/m^3, based on the results of a developmental study with exposure of rats by inhalation during gestation days 6–16, in which a decrease in body weight gain was observed in dams at concentrations of 465 mg/m^3 and higher (Weeks et al. 1979).

The lowest lowest-observed-effect level (LOEL) for non-neoplastic effects following oral exposure is 10 mg/kg-bw per day, based on an increase in kidney histopathological lesions (including renal tubule hyperplasia, increased linear mineralization of the renal papillae and hyperplasia of the pelvic transitional epithelium) in male rats administered 10 mg/kg-bw per day or more by gavage for 2 years (NTP 1989). Although statistical significance was not indicated for the lowest dose level of 10 mg/kg-bw per day, the incidence data suggest a dose–response relationship. Based on this lesion, the lowest calculated BMDL$_{10}$ for non-cancer effects in the database is 0.728 mg /kg-bw/day for atrophy and degeneration of renal tubules in male F344 rats exposed to hexachloroethane.

Confidence in the toxicity database is considered to be moderate in a screening context, since, although it includes a considerable number of studies (including those for which animals were exposed for a significant proportion of their life span) addressing a wide range of endpoints, data on the toxicity of hexachloroethane for the principal route of exposure (inhalation) are limited (see Appendix 2).

9.3 Characterization of Risk to Human Health and Uncertainties

Based on consideration of the use patterns, physicochemical properties and the upper-bounding estimate of exposure, inhalation of both ambient air and indoor air is the likely predominant route of population exposure to hexachloroethane in Canada. However, exposures to hexachloroethane are decreasing due to a phase-out of this chemical and non-detection of the substance in the majority of recent monitoring surveys.

In studies with animals, increased incidences of tumours have been observed in multiple organs in both rats and mice. The majority of genotoxicity studies have had negative results, although binding to DNA was reported in one study both *in vitro* and *in vivo*. Although the mode of induction of tumours by hexachloroethane has not been elucidated the available information suggests a non-genotoxic mode of action and a margin of exposure approach is used to characterize risk.

The lowest oral LOEL identified was 10 mg/kg-bw per day (the lowest dose tested; gavage administration), which was associated with histopathological changes in the kidney in the male rat and an increased incidence of pheochromocytomas of the adrenal gland (NTP 1989). Since a NOAEL was not obtained, a BMDL was calculated for this endpoint. The calculated $BMDL_{10}$ was determined to be 0.728 mg /kg-bw per day. Comparison of the BMDL10 of 0.728 mg/kg-bw per day) with an upper-bounding estimate of exposure (0.07 µg/kg-bw per day, 0.5 – 4 year old group) results in a margin of 10400.
Although the predominant route of human exposure to hexachloroethane is likely to be inhalation, relevant toxicological data are more limited; the lowest inhalation effect level was 465 mg/m^3, based on a decrease in body weight gain in rat dams at this concentration and higher in a developmental study (Weeks et al. 1979). Following higher exposures by both gavage and inhalation, tremors have been observed in rats. Comparison of this effect level to the indoor air concentration used to estimate upper-bounding exposure estimates (0.12 ug/m^3) results in a margin of exposure approaching four million.

Given the conservative nature of generated exposure estimates, the continued phasing out of this chemical, and the uncertainty related to the relevance of the identified health endpoints, the margins of exposure are considered adequate to address uncertainty associated with the health effects and exposure databases.

Based on the adequacy of the margins of exposure between estimated exposures to hexachloroethane and critical effect levels, it is concluded that hexachloroethane does not meet the criteria set out in paragraph 64(c) of CEPA as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

9.4 Uncertainties in Evaluation of Risk to Human Health

Uncertainties relevant to interpretation of the adequacy of the margin include the limitations of the database regarding the potential modes of induction of the carcinogenic effects the limited characterization of dose–response in the critical study for non-cancer effects (a no-observed-effect level [NOEL] was not identified), and the limitations of the database on levels of hexachloroethane in media of importance to human exposure, such as the identity of consumer products that may contribute to exposure.

10. Conclusion

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

On the basis of the adequacy of the margins of exposure between estimated exposures to hexachloroethane and critical effect levels, it is concluded that hexachloroethane does not meet the criteria set out in paragraph 64(c) of CEPA as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that hexachloroethane does not meet any of the criteria set out in section 64 of CEPA.

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Appendix 1: Upper-bounding estimates of daily intake of hexachloroethane by the general population in Canada

Route of exposure	Estimated intake (µg/kg-bw per day) of hexachloroethane by various age groups							
	0–6 months[1,2,3]			0.5–4 years[4]	5–11 years[5]	12–19 years[6]	20–59 years[7]	60+ years[8]
	Breast Milk Fed	Formula fed	Not formula fed					
Ambient air[9]	6 x 10^{-4}			1x 10^{-3}	9 x 10^{-4}	5 x 10^{-4}	5 x 10^{-4}	4 x 10^{-4}
Indoor air[10]	0.03			0.06	0.05	0.03	0.02	0.02
Drinking water[11]	N/A	2 x 10^{-3}	6 × 10^{-4}	7 × 10^{-4}	6 × 10^{-4}	3 × 10^{-4}	3 × 10^{-4}	4 × 10^{-4}
Food and beverages[12]	N/A			N/A	N/A	N/A	N/A	N/A
Soil[13]	4 × 10^{-6}			7 × 10^{-6}	2 × 10^{-6}	5 × 10^{-7}	4 × 10^{-7}	4 × 10^{-7}
Total intake	0.03	0.03	0.03	0.07	0.05	0.03	0.03	0.02

NA, not applicable

[1] Data on concentrations of hexachloroethane in breast milk were not identified.

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

[3] For exclusively formula-fed infants, intake from water is synonymous with intake from food. The concentration of hexachloroethane in water used to reconstitute formula was based on a study of tap water from Union, Ontario, and Ottawa, Ontario (OME 1988, 1989). Data on concentrations of hexachloroethane in infant formula were not identified. Approximately 50% of non-formula-fed infants are introduced to solid foods by 4 months of age, and 90% by 6 months of age (NHW 1990).

[4] Assumed to weigh 15.5 kg, to breathe 9.3 m^3 of air per day, to drink 0.7 L of water per day, to ingest 54.7 g of fish per day and to ingest 100 mg of soil per day (Health Canada 1998).

[5] Assumed to weigh 31.0 kg, to breathe 14.5 m^3 of air per day, to drink 1.1 L of water per day, to ingest 89.8 g of fish per day and to ingest 65 mg of soil per day (Health Canada 1998).

[6] Assumed to weigh 59.4 kg, to breathe 15.8 m^3 of air per day, to drink 1.2 L of water per day, to ingest 97.3 g of fish per day and to ingest 30 mg of soil per day (Health Canada 1998).

[7] Assumed to weigh 70.9 kg, to breathe 16.2 m^3 of air per day, to drink 1.5 L of water per day, to ingest 111.7 g of fish per day and to ingest 30 mg of soil per day (Health Canada 1998).

[8] Assumed to weigh 72.0 kg, to breathe 14.3 m^3 of air per day, to drink 1.6 L of water per day, to ingest 72.9 g of fish per day and to ingest 30 mg of soil per day (Health Canada 1998).

[9] Hexachloroethane was detected in approximately 33% of 11 399 samples, with an arithmetic mean concentration of 0.016 ug/m^3 (maximum = 1.5 ug/m^3)(You et al 2008). The arithmetic mean concentration was used to calculate the estimates of intake. Canadians are assumed to spend 3 hours outdoors each day (Health Canada 1998).

[10] The arithmetic mean: 0.12 ug/m3 from the CHMS Cycle 2 Indoor air survey was used to estimate exposure from this source (2013 email from Environmental Health Sciences and Research Bureau, Health Canada to Existing Substances Risk Assessment Bureau, Health Canada). Canadians are assumed to spend 21 hours indoors each day (Health Canada 1998).

[11] The reported maximum hexachloroethane concentration of 1.6 × 10^{-2} µg/L measured in 24 samples of tap water from Ottawa, Ontario, in 1987 was used to calculate the upper-bounding

intake estimate (OME 1988)). Data considered in the selection of critical data also included Environment Canada (1989), Otson et al. (1982) and Clark et al. (1982).

[12] Hexachloroethane was detected in one fish sample (maximum concentration of 100 ug/kg) in the Ashtabula River in Ohio (DeVault 1985), however this data was not considered relevant for this exposure assessment. No investigations of the potential presence of hexachloroethane in other foodstuffs were identified.

[13] Hexachloroethane was not detected in the available surveys of soil. The highest detection limit (1 µg/kg) from a study that measured hexachloroethane in soil samples from urban and rural locations in Windsor, Ontario, was used to calculate the upper-bounding intake estimate (Gizyn 1994). Data considered in the selection of critical data also included Webber (1994), Oliver and Pugsley (1986), Oliver and Kaiser (1986) and Webber and Nichols (1995).

Appendix 2: Summary of health effects information for hexachloroethane

Endpoint	**Lowest effect levels[1]/Results**
Laboratory animals and *in vitro*	
Acute toxicity	Lowest **oral LD_{50}** (female rat) = 4460 mg/kg-bw (Weeks et al. 1979) [Additional studies: Exxon Chemical Americas 1962; Fowler 1969; Kinkead and Wolfe 1992] Lowest **dermal LD_{50}** = >3160 mg/kg-bw (Exxon Chemical Americas 1962) [No additional studies identified] Lowest **inhalation LC_{50}** = >8230 mg/m^3 (Exxon Chemical Americas 1962) [No additional studies identified]
Short-term repeated-dose toxicity	Lowest **oral (gavage) LOEL** (male rat) = 146.8 mg/kg-bw per day: increased absolute and relative kidney weights, hyaline droplet nephropathy and increased renal tubule cell labelling index (21-day study) (NTP 1996) [Additional studies: Dow Chemical Co. 1977; Weeks et al. 1979; NTP 1989] Lowest **inhalation LOEC** (male and female rats, male dogs and male guinea pigs) = 2517 mg/m^3: tremors and/or mortality; no gross changes observed at necropsy (6-week studies). There were minimal effects at 465 mg/m^3 and no detectable changes at 145 mg/m^3 (NOEC) (Weeks et al. 1979). [No additional studies identified]
Subchronic toxicity	Lowest **oral (diet) LOEL** (male and female rats) = 15 mg/kg-bw per day: swelling of hepatocytes and kidney tubular degeneration (16-week study) (NOEL = 1 mg/kg-bw per day) (Gorzinski et al. 1985) [Additional studies: NTP 1989]

Endpoint	**Lowest effect levels[1]/Results**
Chronic toxicity	Lowest **oral (gavage) LOEL (lowest dose in protocol)** (male F344/N rats) = 10 mg/kg-bw per day based on an increase in kidney histopathological lesions (increased linear mineralization of the renal papillae, hyperplasia of the pelvic transitional epithelium and renal tubule hyperplasia) of the kidney in male F344/N rats administered hexachloroethane by gavage 5 days per week for 2 years at doses of 0, 10, or 20 mg/kg-bw per day (NTP 1989). Although statistical significance was not indicated for the lowest dose level of 10 mg/kg-bw per day, the incidence data suggest a dose–response relationship. [Additional studies: Weisburger 1977; National Cancer Institute 1978]

Endpoint	**Lowest effect levels[1]/Results**
Carcinogenicity	An increased combined incidence of **renal adenomas or carcinomas** (1/50, 2/50 and 7/50 at 0, 10 and 20 mg/kg-bw per day, respectively) was observed in male F344/N rats administered hexachloroethane by gavage 5 days per week for 2 years at doses of 0, 10, or 20 mg/kg-bw per day. The increase was significant ($p < 0.01$) only at the high dose (NTP 1989). There was no significant difference in survival between any groups of either sex. An increased incidence of **pheochromocytomas** of the adrenal gland (15/50, 28/45 and 21/49 at 0, 10 and 20 mg/kg-bw per day, respectively) was observed in male rats administered hexachloroethane by gavage 5 days oer week for 2 years. The increase was significant ($p < 0.01$) only at the low dose (NTP 1989). There were no increases in the incidence of tumours at any site in female rats administered hexachloroethane at up to 160 mg/kg-bw per day by gavage for 2 years at doses of 0, 80, or 160 mg/kg-bw per day (NTP 1989). Since the adrenal tumours did not follow a dose response trend, Benchmark Dose (BMD) calculations could not be performed. For renal adenoma or carcinoma (combined) in male F344/N rats, the lowest calculated BMDL10 (the lower bound on the exposure associated with a 10% extra cancer risk) is 8.53 mg/kg-bw per day. An increased incidence of **kidney tubular cell adenomas** (0/20, 0/20, 4/49 and 0/50 at 0 [naive], 0 [vehicle], 212 and 423 mg/kg-bw per day, respectively) was observed in male Osborne-Mendel rats administered hexachloroethane by gavage 5 days per week for 22 weeks followed by a cyclic pattern of dosing for 56 weeks (1 dose free week followed by 4 weeks of dosing) at time-weighted average doses of 212 or 423 mg/kg-bw per day over the 78 week period, which was then followed by a dose-free observation period of 33 or 34 weeks. Fifty animals/sex/group were exposed by gavage to hexachloroethane. Twenty animals/sex/group were exposed by gavage to vehicle (corn oil) or placed on test as untreated controls without intubation. The increase was non-statistically significant only at the low dose (National Cancer Institute 1978). Survival of male rats at 90 weeks was 19/50 for the high dose, 24/50 for the low dose, 14/20 for controls and 11/20 for vehicle controls. High mortality may have precluded the observation of late-developing tumours. An increased incidence of **hepatocellular carcinomas** was observed in male (1/18, 3/20, 15/50 and 31/49 for 0 [naive], 0 [vehicle], 590 and 1179 mg/kg-bw per day, respectively) and female (0/18, 2/20, 20/50 and 15/49 at 0 [untreated], 0 [vehicle], 590 and 1179 mg/kg-bw per day, respectively) B6C3F1 mice exposed by gavage 5 days per week at time-weighted average doses of 590 or 1179 mg/kg-bw per day for 78 weeks, followed by a dose-free observation period of 12 or 13 weeks. Fifty animals/sex/group were exposed by gavage to hexachloroethane. Twenty animals/sex/group were exposed by gavage to vehicle (corn oil) or placed on test as untreated controls without intubation.. When compared to vehicle controls, the increase was statistically significant (*p* < 0.001) only at the high dose in males and the low dose in females (National Cancer Institute 1978).[2] To assess initiation potential, 10 male Osborne-Mendel rats received

Endpoint	**Lowest effect levels[1]/Results**
Genotoxicity and related endpoints: *in vivo*	**Gene mutation** **Equivocal results:** *Drosophila* [somatic cells] (Vogel and Nivard 1993) **DNA binding (covalent)** **Positive results:** Rat and mouse liver, kidney, lung, stomach cells but DNA adducts were not identified [i.p. administration] (Lattanzi et al. 1988) **Micronuclei induction** **Negative results:** Mouse bone marrow [i.p. administration] (Crebelli et al. 1999) **DNA unwinding** **Negative results:** Mouse (Taningher et al. 1991)

Endpoint	Lowest effect levels^1/Results
Genotoxicity and related endpoints: *in vitro*	**Gene mutation** **Positive results:** *Saccharomyces cerevisiae* (Bronzetti et al. 1989); without exogenous metabolic system (IARC 1999) **Negative results:** *Salmonella typhimurium* TA98 (with and without activation), TA100 (with and without activation), TA1535 (with and without activation), TA1537 (with and without activation), TA1538 (with and without activation), BA13 (with and without activation), BAL13 (Weeks et al. 1979; Kinae et al. 1981; Haworth et al. 1983; SRI International 1984; Milman et al. 1988; NTP 1989; Roldan-Arjona et al. 1991) *Saccharomyces cerevisiae* (Weeks et al. 1979; Bronzetti et al. 1989) **Sister chromatid exchange** **Positive results:** Chinese hamster ovary cells (Galloway et al. 1987). The authors noted that the sister chromatid exchange test was positive only with activation at doses that induced cell cycle delay. **Micronuclei induction** **Equivocal results:** Human blood cells (Tafazoli et al. 1998) **Negative results:** Human lymphoblastoid cells (Doherty et al. 1996; Parry et al. 1996) **DNA binding (covalent)** **Positive results:** Calf thymus DNA (Lattanzi et al. 1988); with exogenous metabolic system but DNA adducts not identified (IARC 1999) **DNA damage** **Negative results:** Cultured human lymphocytes with and without activation, but **Positive results:** Isolated human lymphocytes with and without activation (Tafazoli et al. 1998) **Negative results:** SOS induction and strand damage using *S. typhimurium* TA1535/pSK1002 (Nakamura et al. 1987) **Differential toxicity** **Negative results:** *Bacillus subtilis* (Kinae et al. 1981) **Aneuploidy** **Negative results:** *Aspergillus nidulans* (Crebelli et al. 1988) **Cell transformation** **Negative results:**

Endpoint	**Lowest effect levels[1]/Results**
	BALB/c-3T3 cells (Arthur D. Little Inc. 1983; Tu et al. 1985; Milman et al. 1988) **Chromosome aberrations** **Negative results:** Chinese hamster ovary cells (Galloway et al. 1987)
Developmental toxicity	Pregnant female rats were given oral doses of hexachloroethane (50, 100, or 500 mg/kg) from day 6-16 of gestation. Lowest **oral LOEL** (female rats) = 500 mg/kg-bw per day: lower gestation indices and number of live fetuses per dam and higher fetal resorption rates (gestation days 6–16) (Weeks et al. 1979) [No additional studies identified] Lowest **inhalation LOEC** (female rats) = 465 mg/m^3: decreased body weight gain of dams, increased mucopurulent nasal exudate (gestation days 6–16); no significant skeletal or soft tissue anomalies in fetuses (Weeks et al. 1979). Pregnant rats were exposed to 0, 145, 465 or 2517 mg/m^3 on days 6–16 of gestation. Dams were observed for toxicity, and fetuses were examined for soft tissue and skeletal abnormalities. [No additional studies identified]
Reproductive toxicity	No data identified
Behavioural toxicity/ neurotoxicity	In a 6-week inhalation study (Weeks et al. 1979): at 2517 mg/m^3, dogs developed tremors, were ataxic and showed severe head bobbing. Rats had tremors. At 145 and 465 mg/m^3, no adverse effects were reported in dogs or rats.
Humans	
Short-term repeated-dose toxicity	In an inhalation study of 11 munitions workers (5 men, 6 women) exposed to 10–20 mg/m^3 for 5 weeks, increases in serum creatinine, serum urate and serum bilirubin were observed; however, levels were within reference values. An increased prevalence of “dry skin/dry mucous membranes” was not statistically significant (Selden et al. 1994).
Carcinogenicity	In a cohort study (*n* = 1880) of male workers at aluminum foundries and aluminum smelters, no significant association was observed between exposure to hexachloroethane (levels were not quantified in secondary source) and incidences of anorectal, liver or lung cancer or malignant lymphoma (Selden et al. 1997).

[1] LC_{50}, median lethal concentration; LD_{50}, median lethal dose; LOEC, lowest-observed-effect concentration; LOEL, lowest-observed-effect level; NOEC, no-observed-effect concentration; NOEL, no-observed-effect level.

[2] Note that incidences for males were reported as 3/20, 15/50 and 29/49 for vehicle controls, low dose and high dose, respectively, in the publication of the study by Weisburger (1977).