Priority Substances List Assessment Report

1,2-Dichlorobenzene

Government of Canada Environment Canada Health Canada

Publié également en français sous le titre de : Loi canadienne sur la protection de l'environnement Liste des substances d'intérêt prioritaire Rapport d'évaluation : 1,2-Dichlorobenzène

CANADIAN CATALOGUING IN PUBLICATION DATA

Main entry under title:

1,2-Dichlorobenzene
(Priority substances list assessment report)
Issued also in French under title: 1,2-Dichlorobenzène.
At head of title: Canadian Environmental
Protection Act.
Includes bibliographical references.
ISBN 0-662-21072-7
Cat. No. En40-215/33E

Dichlorobenzene — Environmental aspects.
 Environmental monitoring — Canada.
 I. Canada. Environment Canada.
 II. Canada. Health Canada.
 III. Series

TD427.D52053 1993 363.73'84 C93-099694-1

© Minister of Supply and Services Canada 1993 Canada Communication Group — Publishing Ottawa, Canada K1A 0S9 Cat. No. En40-215/33E ISBN 0-662-21072-7



Table of Contents

1.0	Introduction								
2.0	Summary of Information Critical to Assessment of "Toxic"								
2.1	Identity, Properties, Production and Uses								
2.2	Entry into the Environment								
2.3	Exposure-related Information								
	2.3.1 Fate								
	2.3.2 Concentrations								
2.4	Effects-related Information								
	2.4.1 Experimental Animals and <i>In Vitro</i>								
	2.4.2 Humans								
	2.4.3 Ecotoxicology								
3.0	Assessment of "Toxic" Under CEPA								
3.1	CEPA 11(a): Environment								
3.2	CEPA 11(b): Environment on Which Human Health Depends								
3.3	CEPA 11(c): Human Life or Health								
3.4	Conclusion								
4.0	Recommendations								
5.0	References								

Synopsis

1,2-Dichlorobenzene is produced in Canada and is also imported from other countries. Approximately 350 tonnes per year of this substance are currently used in Canada, primarily as a solvent for carbon removal, degreasing of engines, and metal cleaning. 1,2-Dichlorobenzene is present at measurable concentrations in industrial and municipal effluents, surface water and sediments in Canada, while detection in other environmental media, such as ambient air, ground water and soil, is infrequent. 1,2-Dichlorobenzene is not persistent in air or surface water, but persists and accumulates in sediment under anaerobic conditions.

The maximum concentration of 1,2-dichlorobenzene measured in surface waters in Canada was approximately 9 000 times less than the effects threshold estimated for the most sensitive aquatic species identified. The estimated total daily intake of 1,2-dichlorobenzene was calculated to be approximately 12 000 times less than the effects threshold estimated for wild mammals. No data were identified on the toxicological effects of 1,2-dichlorobenzene to benthic organisms, and therefore it was not possible to conclude whether concentrations of this substance found in sediments could result in harmful effects to these biota.

1,2-Dichlorobenzene is present in low concentrations and has a short half-life in the atmosphere. As such, it is not expected to contribute significantly to the formation of ground-level ozone, global warming, or depletion of stratospheric ozone.

Based on data on concentrations of 1,2-dichlorobenzene in ambient air, drinking water and food, the total daily average intakes for various age groups in the general population have been estimated. These average daily intakes are considerably less (by approximately 500 to 14 000 times) than the tolerable daily intake derived on the basis of studies in laboratory animals. The tolerable daily intake is the intake to which it is believed that a person can be exposed over a lifetime without deleterious effect.

Based on these considerations, the Ministers of the Environment and of Health have determined that there is insufficient information upon which to conclude whether 1,2-dichlorobenzene constitutes a danger to the environment. It is concluded, however, that this substance does not constitute a danger to the environment upon which human life depends or to human health. Therefore, although it is not possible to conclude whether 1,2-dichlorobenzene is "toxic" as defined under paragraph 11(a), 1,2-dichlorobenzene is not considered to be "toxic" as defined under paragraphs 11(b) and 11(c) of the *Canadian Environmental Protection Act*.

1.0 Introduction

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

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

Substances assessed as "toxic" according to section 11 may be placed on Schedule I of the Act and considered for possible development of regulations, guidelines or codes of practice to control any aspect of their life cycle, from the research and development stage through manufacture, use, storage, transport and ultimate disposal.

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

The assessment of whether 1,2-dichlorobenzene is "toxic" to human health under CEPA is based principally on documentation prepared by staff of Health Canada (HC) for the World Health Organization (WHO)/International Programme on Chemical Safety (IPCS). Between 1984 and 1987, original data relevant to the assessment of risks to health associated with exposure to the chlorinated benzenes (excluding hexachlorobenzene) were reviewed by staff of HC in the preparation of a draft IPCS Environmental Health Criteria (EHC) document. The current assessment has been updated and expanded to emphasize data most relevant to the assessment of the risks associated with exposure of Canadians to 1,2-dichlorobenzene in the general environment.

During preparation of the WHO-IPCS document, a wide variety of scientific databases were searched in order to update information provided in earlier contractors' reports, including an annotated bibliography on the chlorobenzenes (excluding hexachlorobenzene) by Peter Strahlendorf (1978) and a criteria document on chlorobenzenes (including hexachlorobenzene) by Michael Holliday and Associates (1984a, 1984b). Additional information was identified during peer review of the draft Environmental Health Criteria Document by IPCS focal points and a Task Group of Experts, which met in June 1990. More recently, in February 1991, a search of Enviroline, Chemical Abstracts, Pollution Abstracts, Environmental Bibliography, IRIS, MEDLINE and BIOSIS databases to identify recent data relevant to assessment of the risks to Canadians, in particular, was conducted. Data relevant to assessment of whether 1,2-dichlorobenzene is "toxic" to human health obtained after completion of these sections of the report (i.e., in December 1991) were not considered for inclusion.

Data relevant to the assessment of whether 1,2-dichlorobenzene is "toxic" to the environment were identified from existing review documents, published reference texts and on-line searches completed in November 1990 of the following commercial databases: ASFA, BIOSIS, CAB Abstracts, Chemical Abstracts, Chemical Evaluation Search and Retrieval System (CESARS), Enviroline, Hazardous Substances Database, International Register of Potentially Toxic Chemicals (IRPTC), National Technical Information Service (NTIS), TOXLINE, and the Toxic Release Inventory Data Base. Studies published after November 1990 were identified by a review conducted until January 1992 of relevant journals as well as Current Contents (Agriculture, Biology and Environmental Sciences). Although much of the research on 1,2-dichlorobenzene has been conducted outside of Canada, Canadian data on sources, use patterns, fate and effects of 1,2-dichlorobenzene on the environment were emphasized, wherever possible. Data relevant to assessment of whether 1,2-dichlorobenzene is "toxic" to the environment obtained after October 1992 have not been incorporated.

Although review articles were consulted where considered appropriate, original studies that form the basis for the determination of "toxic" under CEPA were critically evaluated by staff of Health Canada and Environment Canada. The following officials contributed to preparation of the report:

B. Elliott (Environment Canada)

C. Fortin (Environment Canada)

M. Giddings (Health Canada)

R. Gomes (Health Canada)

R.G. Liteplo (Health Canada)

K. Lloyd (Environment Canada)

M.E. Meek (Health Canada)

In this report, a synopsis that will appear in the *Canada Gazette* is presented. A summary of the technical information that is critical to the assessment, and which is presented in greater detail in unpublished Supporting Documentation, is presented in Section 2. The assessment of whether 1,2-dichlorobenzene is "toxic" as defined under CEPA is presented in Section 3.

The environmental sections of this Assessment Report were reviewed by Dr. Brett Betts (Washington State Department of Ecology, Seattle, Washington), Dr. Peter Chapman (EVS Consultants, Vancouver, British Columbia), Dr. Arthur Niimi (Department of Fisheries and Oceans, Burlington, Ontario) and Dr. Barry Oliver (Zenon Environmental Laboratories, Burnaby, British Columbia). Sections related to the effects on human health were approved by the Standards and Guidelines Rulings Committee of the Bureau of Chemical Hazards of Health Canada. The entire Assessment Report was reviewed and approved by Environment Canada and Health Canada CEPA Management Committee.

Copies of this Assessment Report and the unpublished Supporting Documentation are available upon request from:

Environmental Health Centre Health Canada Room 104 Tunney's Pasture Ottawa, Ontario, Canada K1A 0L2

Branch
Environment Canada
14th Floor
Place Vincent Massey
351 Saint-Joseph Boulevard
Hull, Quebec, Canada
K1A 0H3

Commercial Chemicals

2.0 Summary of Information Critical to Assessment of "Toxic"

2.1 Identity, Properties, Production and Uses

- 1,2-Dichlorobenzene is a neutral, colourless, flammable liquid (U.S. EPA, 1986) with a molecular weight of 147.01 and the empirical formula, $C_6H_4Cl_2$. 1,2-Dichloro-benzene is also known as ortho-dichlorobenzene or o-dichlorobenzene. It has a moderate to high vapour pressure (196 Pa @ 25°C), a low water solubility (118 mg/L @ 25°C) and a moderate octanol/water partition coefficient (log $K_{ow} = 3.4$) [Mackay *et al.*, 1992]. Its organic carbon partition coefficient, calculated from its log K_{ow} value as suggested by Mackay *et al.* (1992), is 1 030. Sadtler Research Laboratories (1982) reported that 1,2-dichlorobenzene absorbs infrared radiation, including wavelengths in the 7 to 13 μ m region.
- 1,2-Dichlorobenzene is produced by chlorination of benzene in the liquid phase with a catalyst, and is available as technical grade (80% 1,2-dichlorobenzene; < 19% other isomers; < 1% trichlorobenzenes; and < 0.05% monochlorobenzene) and purified grade (98% 1,2-dichlorobenzene; < 0.2% 1,2,4-trichlorobenzene; and 0.05% monochlorobenzene) (Kao and Poffenberger, 1979). The analytical methods by which 1,2-dichlorobenzene is quantified in environmental media include gas chromatography, with flame ionization or electron capture detection, and gas chromatography/mass spectrometry (Oliver and Bothen, 1982; Oliver and Nicol, 1982a).
- 1,2-Dichlorobenzene is produced in Canada and is also imported from other countries (Camford Information Services, 1991). In the past few years, domestic production in Canada has declined, while the amount imported has increased. Overall, the Canadian demand for 1,2-dichlorobenzene is reported to have remained steady for the past 5 years, averaging approximately 350 tonnes per year. This trend is not expected to change during the next 5 years. By comparison, the demand for 1,2-dichlorobenzene in the United States in 1990 was reported to be 22.7 kilotonnes (Chemical Marketing Reporter, 1990).

In Canada, 1,2-dichlorobenzene is used primarily as a solvent for carbon removal and degreasing of engines and for metal cleaning in metal working shops (Camford Information Services, 1991). Quantitative data on use patterns for this substance were only identified for the period 1977–79, when 49% of the annual demand was used as a solvent for paint or carbon removal and degreasers, 31% for engine cleaning compounds, 19% for miscellaneous solvent use and 1% for photoresister solvents, dye carriers and fungicides (Environment Canada, 1983, unpublished). Although registration was discontinued in 1990, 1,2-dichlorobenzene is also being used, until

existing supplies run out, as the active ingredient in an industrial deodorizer and cleaner (Brien, 1992). Elsewhere, including the United States, 1,2-dichlorobenzene is currently used in a much wider variety of applications.

2.2 Entry into the Environment

There are no known natural sources of 1,2-dichlorobenzene, and information on releases of this substance to the Canadian environment from anthropogenic sources is limited. On the basis of its volatility and the dispersive nature of its uses, it is expected that the majority of the 350 tonnes of 1,2-dichlorobenzene used annually in Canada is released to the environment, primarily in liquid effluents and atmospheric emissions from production and other facilities. It has also been suggested that entry of 1,2-dichlorobenzene may result from the dehalogenation of more highly chlorinated chlorobenzenes (Bosma *et al.*, 1988) and in emissions from incineration of organic matter containing chlorine (Young and Voorhees, 1989).

Data concerning the release of 1,2-dichlorobenzene to the atmosphere from industrial and municipal facilities in Canada were not identified; however, releases of this substance in liquid effluents have been reported. Under the Ontario Municipal/ Industrial Strategy for Abatement (MISA) program, estimated loadings to the St. Clair, St. Lawrence and Welland rivers from organic and inorganic chemical manufacturing plants in Ontario were reported to be 0.772, 0.052 and 0.011 kg/day, respectively (OME, 1992, unpublished; 1992a, 1992b). It was also reported under MISA that concentrations of 1,2-dichlorobenzene ranged from 300 to 60 500 ng/L in the effluents discharged from organic chemical plants in the Sarnia, Maitland and Corunna areas (OME, 1992, unpublished). Effluents discharged from 4 of 27 pulp and paper mills in Ontario were reported to contain concentrations of 1,2-dichlorobenzene ranging from 420 to 15 600 ng/L (OME, 1991a, 1991b). 1,2-Dichlorobenzene was also measured in a survey of effluents of 10 Canadian textile mills conducted in 1985–86; concentrations were reported to range up to 95.5 μg/L (Environment Canada, 1989).

In 1987, 1,2-dichlorobenzene was measured in waste water treatment plant effluent that was being discharged into the Strait of Georgia near Vancouver, British Columbia. Concentrations ranged from less than the detection limit (10 ng/L) to 290 ng/L (Chapman *et al.*, 1987; Fanning *et al.*, 1989; Park *et al.*, 1990; Park, 1992). Webber and Lesage (1989) reported that samples of stabilized digested sludge from Winnipeg, Manitoba, contained 1,2-dichlorobenzene (maximum and median concentrations of 1 600 ng/g and 550 ng/g dry weight, respectively).

2.3 Exposure-related Information

2.3.1 Fate

A number of processes affect the distribution and transformation of 1,2-dichlorobenzene in the environment, including atmospheric photooxidation, volatilization, partitioning to soil, sediment and biota, and aerobic degradation (U.S. EPA, 1987; Park *et al.*, 1988; Callahan *et al.*, 1979; Ellington *et al.*, 1988; Weber *et al.*, 1987). 1,2-Dichlorobenzene that is not removed from the environment by degradative processes ultimately accumulates in anaerobic sediments and possibly ground water.

1,2-Dichlorobenzene absorbs radiation weakly at wavelengths greater than 300 nm, and therefore direct photolysis in the atmosphere is not likely (Bunce *et al.*, 1987). However, 1,2-dichlorobenzene will react with photochemically produced hydroxyl radicals in the atmosphere with an estimated half-life of 24 days (Howard, 1989). Mackay *et al.* (1992) estimated a mean half-life in the atmosphere of approximately 3 weeks for 1,2-dichlorobenzene on the basis of photooxidation and advection processes. The presence of 1,2-dichlorobenzene in rain water indicates that it persists long enough to be returned to the earth's surface by atmospheric wash out (Ligocki *et al.*, 1985).

Volatilization is reported to be a dominant mechanism for the removal of 1,2-dichlorobenzene from surface water or soil (Mackay *et al.*, 1992; Callahan *et al.*, 1979; U.S. EPA, 1987; Slimak *et al.*, 1980). Estimated half-lives in water range from 0.94 hours in a shallow river or stream to 60 days in a deep, slow-moving river (U.S. EPA, 1987), while rates of volatilization from soil were reported to be 10 to 100 times lower than in surface water (Park *et al.*, 1988).

- 1,2-Dichlorobenzene is slowly biodegraded in soil under aerobic conditions (Haider *et al.*, 1981; Schraa and van der Meer, 1987; van der Meer, 1987; Oldenhuis *et al.*, 1989). Mackay *et al.* (1992) selected a half-life of approximately 8 months for 1,2-dichlorobenzene in soil, while Howard (1991) reported the half-life to range from 4 weeks to 6 months. Based on its estimated organic carbon sorption coefficient (K_{oc}) of 1 030, 1,2-dichlorobenzene was classified as having a low potential for soil mobility according to the scale proposed by McCall *et al.* (1981).
- 1,2-Dichlorobenzene that partitions to sediment, particularly the organic fraction of bottom sediments, can persist for long periods of time with little likelihood of anaerobic degradation (Oliver and Nicol, 1982b). On the basis of an analysis of core sediment samples from Lake Ontario, it has been reported that 1,2-dichlorobenzene has been accumulating over a period of 60 years with the highest concentrations correlating with the 1960s, when North American production of chlorobenzenes peaked (Oliver and Nicol, 1984; Durham and Oliver, 1983). Based on review of the

literature, Mackay *et al.* (1992) selected a mean half-life of approximately 2 years for 1,2-dichlorobenzene in the first 1 cm of sediment. Within sediments, 1,2-dichloro-benzene is expected to equilibrate between the pore water and the organic phase (Di Toro *et al.*, 1991).

1,2-Dichlorobenzene was reported to be persistent and slightly mobile during field studies of ground water contaminated by sewage effluent and municipal and industrial wastes (Barber *et al.*, 1988; Barber, 1988; Roberts *et al.*, 1980; Roberts *et al.*, 1986; Reinhard *et al.*, 1984). Zoeteman *et al.* (1980) estimated that the half-life in ground water for 1,2-dichlorobenzene ranged between 30 and 300 days.

Oliver and Nimii (1983) reported bioconcentration factors ranging from 270 to 560 (whole fish) for the rainbow trout (*Oncorhynchus mykiss*) exposed under laboratory conditions. Biological half-lives of less than 1 day and less than 5 days were reported for bluegill sunfish (*Lepomis macrochirus*) [Barrows *et al.*, 1980] and oligochaete worms (*Tubifex tubifex* and *Limnodrilus hoffmeisteri*) [Oliver, 1987], respectively.

2.3.2 Concentrations

1,2-Dichlorobenzene has been measured in surface water, sediments, and biota in the Great Lakes Region. It has also been detected, albeit infrequently, in ambient air, ground water and soil in Canada.

The mean concentration of 1,2-dichlorobenzene measured between October 1988 and December 1990 during an extensive sampling program of ambient air at 22 sites across Canada was reported to be below the detection limit of 0.1 μ g/m³ (Environment Canada, 1991 unpublished). Daily maximum concentrations of 1,2-dichlorobenzene in ambient air ranged from 0.06 (Vancouver, British Columbia) to 0.61 μ g/m³ (Toronto, Ontario); 96% of the measurements were below the detection limit.

Identified information on concentrations of 1,2-dichlorobenzene in indoor air in Canada is restricted to a limited and probably unrepresentative number of homes in which maximum concentrations of 1,2-dichlorobenzene were 0.20 and 6.5 μ g/m ³, based on active and passive sampling techniques, respectively (Otson and Benoit, 1986). In general, based on surveys conducted in the United States, concentrations of 1,2-dichlorobenzene in indoor air are similar to those in ambient air. Mean or median concentrations have ranged from 0.03 to 0.12 μ g/m³ and maximum values have been less than 11 μ g/m³ (Pellizzari *et al.*, 1986).

Few studies have been identified concerning the presence of 1,2-dichlorobenzene in Canadian surface water. In 1988–89, mean annual concentrations of 1,2-dichlorobenzene in the Niagara River were reported to range from 0.27 ng/L at Fort Erie to 2.05 ng/L at Niagara-on-the-Lake, Ontario (NRDIG, 1990). Data for surface water

samples collected in the Great Lakes Basin during the early 1980s indicate that concentrations of 1,2-dichlorobenzene were typically much higher. Maximum concentrations of 1,2 dichlorobenzene were reported to range from 4 to 240 ng/L in samples of water collected during the period 1981–1983 from the Niagara River, at Niagara-on-the-Lake (Oliver and Nicol, 1984).

1,2-Dichlorobenzene was detected in ground water at a former disposal site for waste oils and solvents near Ville Mercier, Quebec, in 1988 (range = 3 300 to 46 000 ng/L) [Martel and Ayotte, 1989], and near a municipal landfill at North Bay, Ontario, in 1981 (range = 2 800 to 13 000 ng/L) [Reinhard *et al.*, 1984].

Information on concentrations of 1,2-dichlorobenzene in Canadian drinking-water supplies is limited. 1,2-Dichlorobenzene was not detected (detection limit = $1 \mu g/L$) in a survey of samples from 30 water-treatment facilities across Canada taken between August and December 1979 (Otson et al., 1982b). In the water supplies of three cities in the vicinity of Lake Ontario sampled between April and November 1980, levels of 1,2-dichlorobenzene ranged from not detected (detection limit = 1 ppt [ng/L]) to 7 ppt with a mean of 3 ppt (Oliver and Nicol, 1982b). Concentrations of 1,2-dichlorobenzene were below the detection limit (0.1 μg/L) in 144 of 145 samples of raw and treated water collected in Quebec in May 1985, and in February and July 1986. The measured concentration in the one sample in which 1,2-dichlorobenzene was detected was 3.4 µg/L (Vachon, 1986). Traces of 1,2-dichlorobenzene were detected (detection limit 1 µg/L) in 3 of 29 treated municipal-water supplies in Alberta from 1980–1985 (Alberta Environment, 1985). In 1 210 samples of drinking water from 139 locations in the four Atlantic provinces taken between 1985 and 1988, the dichlorobenzenes were not detected (detection limit = $0.02 \,\mu\text{g/L}$). It should be noted, however, that 23% and 77% of the total number of samples taken for analysis of the 1,2- and 1,4-dichlorobenzene isomers, respectively, were contaminated and, therefore, not included in the survey (Environment Canada, 1989a, 1989b, 1989c, 1989d).

Data on the levels of 1,2-dichlorobenzene in sediments in Canada are also limited. In a study of sediments near municipal sewage outfalls at Victoria, British Columbia, conducted in 1992, it was reported that concentrations of 1,2-dichlorobenzene were below the limit of detection of 1 μ g/kg (dry weight) [EVS Consultants, 1992]. Other identified data on the levels of 1,2-dichlorobenzene in Canadian sediment are restricted to concentrations determined in the late 1970s to the mid-1980s. In 1981, Durham and Oliver (1983) examined the vertical distribution of 1,2-dichlorobenzene in bottom sediments in Lake Ontario near the mouth of the Niagara River. The maximum concentration was reported to be 87 ng/g at a depth of 8–9 cm. The levels of total dichlorobenzene isomers have also been determined in sediment collected from the St. Clair River in 1984–85 near petrochemical plants near Sarnia (Oliver and Pugsley, 1986). The average concentration was reported to be 5 500 ng/g, while the maximum concentration was 34 000 ng/g (in 43 samples). Based on the ratio of

isomers provided by Oliver (1992), the corresponding average concentration of 1,2-dichlorobenzene in these sediments was approximately 1 700 ng/g. Sylvestre (1987) reported maximum (660 ng/g) and mean (403 ng/g) concentrations of 1,2-dichlorobenzene in suspended sediments near Wolfe Island, in the St. Lawrence River.

No data were identified on the concentrations of 1,2-dichlorobenzene in wild mammals or birds, and few data were found for other environmental biota in Canada. In a study conducted near a sewage treatment plant outfall at Clover Point, near Victoria, British Columbia, 1,2-dichlorobenzene was not detected in mussel (*Modiolus* sp.) tissue (detection limit = 5 μg/kg). Based on studies conducted in the Great Lakes in the early 1980s, the concentration of 1,2-dichlorobenzene in lake trout (*Salvelinus namaycush*) and rainbow trout (*Oncorhynchus mykiss*) ranged between 0.3 and 1 ng/g (wet weight) [Oliver and Nicol, 1982b; Oliver and Niimi, 1983; Fox *et al.*, 1983], respectively. Higher concentrations of 1,2-dichlorobenzene were reported in sediment-dwelling amphipods and oligochaetes (maxima of 18 and 100 ng/g dry weight, respectively) that were collected in Lake Ontario near the mouth of the Niagara River in 1981 (Fox *et al.*, 1983). MacLaren Marex Ltd. (1979) reported concentrations of 1,2-dichlorobenzene up to 40 ng/g (dry weight) in clams collected from sediment contaminated by discharge from a chemical manufacturing plant near Pictou, Nova Scotia.

Information on concentrations of 1,2-dichlorobenzene in Canadian food supplies is limited. In a limited study of fresh food composites from Ontario, the 1,2-isomer was detected only in the eggs/meat composite, at a concentration of 0.0018 μ g/g (Davies, 1988). Other composites included leafy vegetables, fruit, root vegetables (including potatoes) and 2% milk; the detection limit for all composites was 0.0001 μ g/g.

1,2-Dichlorobenzene has been detected in mothers' milk. The mean level of 1,2-dichlorobenzene in the breast milk of Canadian women (3 to 4 weeks after parturition) was 3 ng/g with a maximum of 29 ng/g; the sampling period and detection limits were not specified (Mes *et al.*, 1986). The isomer was detected in 69% of the 210 samples analyzed. In the breast milk of women of the Canadian indigenous population, 1,2-dichlorobenzene was detected in 50% of 18 samples analyzed; the mean level was 8.1 ng/g and the maximum concentration was not reported.

2.4 Effects-related Information

2.4.1 Experimental Animals and In Vitro

1,2-Dichlorobenzene has been acutely toxic following administration by all routes of exposure examined to date (i.e., inhalation, oral, intraperitoneal). Acute exposure to lethal concentrations results in CNS depression following inhalation, and respiratory paralysis following ingestion. In inhalation studies, reported LC₅₀s for rats were 9 192 mg/m³, 5 863 mg/m³ and 7 416 mg/m³ for female mice (Bonnet *et al.*, 1979, 1982; Hollingsworth *et al.*, 1958). No deaths were observed in guinea pigs following ingestion (gavage in olive oil) of 800 mg/kg bw, whereas 2 000 mg/kg bw was lethal to 100% of the animals (Hollingsworth *et al.*, 1958).

Effects similar to those observed in acute studies have been noted in rats and mice following short-term exposure to 1,2-dichlorobenzene, with deaths being observed following 14 days of ingestion by rats of 1 000 mg/kg bw per day (NTP, 1983). In a recently reported 10-day study, the no-observed-effect-level (NOEL) based on hepatotoxic and haematological effects in rats observed at higher concentrations was 75 mg/kg bw per day (Robinson *et al.*, 1991).

In the single identified subchronic study by the inhalation route, in which analyses of at least body weight gain, survival, clinical signs of toxicity, clinical chemistry, haematology and histopathology of major organs and tissues have been conducted, no treatment-related effects were observed at concentrations up to 560 mg/m³ (NOEL) in rats, guinea pigs, rabbits and monkeys (Hollingsworth et al., 1958). In two subchronic studies in which 1,2-dichlorobenzene has been administered by the oral route (gavage in corn oil) to both rats and mice, decreases in body weight gain and survival, renal tubular degeneration in male rats, increases in relative and absolute kidney weights in both sexes of rats, increases in urinary porphyrin, and some serum hepatic enzymes and slight haematological effects have been observed at high doses (400 to 500 mg/kg bw); at lower doses (> 100 mg/kg bw), centrilobular degeneration and necrosis of the liver and increases in relative and absolute liver weights have been observed. In mice, lymphoid depletion of the thymus and spleen and multifocal mineralization of myocardial fibres of the heart and skeletal muscle have been observed at high doses (> 500 mg/kg bw) (NTP, 1983; Robinson et al., 1991). NOELs in these studies were 25 to 125 mg/kg bw per day in rats and 125 mg/kg bw per day in mice.

Only one study in which the carcinogenicity of 1,2-dichlorobenzene was investigated has been identified. In this bioassay, conducted by the U.S. National Toxicology Program, groups of 50 each male and female rats and mice were administered 0,60 or 120 mg/kg bw 1,2-dichlorobenzene daily by gavage in corn oil, 5 days per week for 103 weeks (NTP, 1983). The only evidence of toxicity in the rats was a decrease in survival in males at the end of the study, particularly in the high-dose group (84%,

72% and 38% for the 0,60 and 120 mg/kg bw dose groups, respectively) and a dose-related trend in tubular regeneration of the kidney in male rats (17%, 24% and 35% for the 0,60 and 120 mg/kg bw dose groups, respectively). The decreased survival in the high-dose group has been attributed, at least in part, to gavage errors (NTP, 1983). Non-neoplastic effects in mice included a dose-related increase in the incidence of tubular regeneration of the kidney in males at 120 mg/kg bw. Treatment-related increases in neoplastic lesions were not observed, and it was concluded that under the conditions of these studies, there was no evidence for the carcinogenicity of 1,2-dichlorobenzene in male or female F344 rats or B6C3F₁ mice (NTP, 1983).

In the only identified developmental toxicity study conducted in two species, relatively minor embryotoxic and foetotoxic effects (delayed ossification) were observed in rats following exposure by inhalation, but only at concentrations (2 400 mg/m³) that were toxic to the mother (Hayes *et al.*, 1985). No embryotoxic, foetotoxic or teratogenic effects were observed in rabbits exposed to up to 2 400 mg/m³ 1,2-dichlorobenzene by inhalation (Hayes *et al.*, 1985). No studies on the reproductive effects of 1,2-dichlorobenzene were identified.

The weight of evidence indicates that 1,2-dichlorobenzene is not mutagenic in bacterial assays (see Supporting Documentation).

2.4.2 **Humans**

Available data on the effects of exposure to 1,2-dichlorobenzene in humans are restricted to case reports and two epidemiological studies; no clinical investigations on the effects of exposure in human volunteers were identified. Case reports of adverse effects associated with exposure to 1,2-dichlorobenzene or mixtures containing 1,2-dichlorobenzene are confined to haematological disorders, including anaemia and leukaemia (Girard *et al.*, 1969; Tolot *et al.*, 1969). In the only identified cross-sectional epidemiological study of workers exposed to 1,2-dichlorobenzene, there was no evidence of "organic injury or untoward haematological effects" in an unspecified number of workers exposed to mean levels of 15 ppm 1,2-dichlorobenzene (Hollingsworth *et al.*, 1958); however, little information on study design was presented in the published account of this investigation.

There was an increase in the total number of chromosomal aberrations (primarily single and double breaks) in the peripheral leucocytes of 26 laboratory workers exposed for 4 days, 8 hours per day to 1,2-dichlorobenzene vapour compared to a control group of 11 non-exposed laboratory personnel (Zapata-Gayon *et al.*, 1982). No quantitative information on exposure was provided in the account of this study.

2.4.3 Ecotoxicology

The information that was identified on the toxicity of 1,2-dichlorobenzene to aquatic biota includes acute and chronic data for bacteria and algae, invertebrates, fish and amphibians. Studies cited below were conducted under closed-, static- or continuous-flow conditions, and were considered reliable for a volatile compound like 1,2-dichlorobenzene. No suitable data were identified for sediment-dwelling biota or terrestrial biota, including wild mammals, birds and vascular plants. Similarly, no empirical data regarding adverse effects of 1,2-dichlorobenzene on wildlife due to decreased availability of prey or quality of prey were identified.

Impairment of reproduction by 1,2-dichlorobenzene was identified as the most sensitive toxicity end-point reported for aquatic organisms. Calamari *et al.* (1983) reported the 14-d EC_{50} (reduced fertility) in *Daphnia magna* to be 0.55 mg/L. A 24-h IC_{50} (immobilization) of 0.78 mg/L was also reported in this study for *Daphnia magna*. Ahmad *et al.* (1984) reported the 96-h LC_{50} in rainbow trout (*Oncorhynchus mykiss*) to be 1.61 mg/L.

Inhibition of cell multiplication was reported to occur at 15 mg/L in the bacterium *Pseudomonas putida* during a 16-h toxicity threshold study (Bringmann and Kuhn, 1980). Reduction of growth (96-h EC_{50}) and photosynthesis (3-h EC_{50}) were observed in the alga *Selenastrum capricornutum* at concentrations of 2.2 mg/L and 10.0 mg/L, respectively (Calamari *et al.*, 1983).

Black *et al.* (1982) studied the susceptibility of the embryo-larval stages of fish and amphibians to 1,2-dichlorobenzene. The organisms were exposed from 20 to 30 minutes following fertilization of the egg to 4 days after hatching of the larva. LC₅₀s were 3.01 mg/L for the rainbow trout (*Oncorhynchus mykiss*) and 5.56 mg/L for the leopard frog (*Rana pipiens*), following total exposure times of 27 and 9 days, respectively.

3.0 Assessment of "Toxic" under CEPA

3.1 CEPA 11(a): Environment

1,2-Dichlorobenzene is produced in, and imported into, Canada in quantities approaching 350 tonnes per year. Few data were identified concerning the amounts entering the environment and the sources. Due to its volatility and the dispersive nature of its uses, however, it is estimated that most of the 1,2-dichlorobenzene used in Canada is released to the environment. 1,2-Dichlorobenzene has been measured in industrial and municipal effluents, surface waters and sediments in Canada but has been detected infrequently in other media, such as ambient air. While 1,2-dichlorobenzene does not persist in ambient air and surface water, it can persist and accumulate in sediment under anaerobic conditions.

Exposure of benthic organisms to 1,2-dichlorobenzene is known to occur in sediments in Lake Ontario and the St. Clair River; however, no toxicological data were identified that would enable an assessment of effects resulting from this exposure.

Daphnia magna was identified to be the most sensitive aquatic organism to 1,2-dichlorobenzene. The 14-day EC_{50} for reduced fertility was reported to be 550 μ g/L. Dividing this value by a factor of 30 (10 to account for interspecies differences in sensitivity as well as extrapolation from laboratory to field conditions, and 3 to extrapolate from a chronic EC_{50} to a chronic NOEL) results in an estimated effects threshold of about 18 μ g/L. The highest concentration of 1,2-dichlorobenzene measured recently in Canadian surface waters was 2 μ g/L (in the Niagara River), which is 9 000 times less than the estimated effects threshold. Therefore, no adverse effects are expected to result from exposure of pelagic organisms to 1,2-dichlorobenzene in Canadian surface waters.

A worst-case exposure scenario was developed for a representative fish-eating mammal (mink, *Mustela vison*) in southern Ontario. Mink are opportunistic carnivores with aquatic organisms comprising up to 100% of their diet. The scenario presented in Table 1 indicates that the estimated total daily intake of 1,2-dichlorobenzene is 490 ng/kg bw with inhalation accounting for about two-thirds of the exposure and ingestion of contaminated food accounting for the other one-third. The only data identified on long-term toxicity following inhalation are the results of an early subchronic study in rats, guinea pigs, rabbits and monkeys, for which no effects were observed at any concentration (maximum concentration administered was 560 mg/m³) [Hollingsworth *et al.*, 1958]. In a long-term NTP bioassay by the oral route, however, a NOEL of 60 mg/kg bw/day was determined in rats and mice, based on tubular regeneration of the kidney at the next highest concentration. Using a factor of 10 to

account for variability in extrapolating from a laboratory to a field situation and interspecies sensitivity, the effects threshold is estimated to be 6 mg/kg bw/day, which is 12 000 times higher than the estimated total daily intake of mink in the wild.

Table 1
Estimated Total Daily Intake of a Piscivorous Mammal Exposed to 1,2-Dichlorobenzene under "Worst-case" Conditions

		Daily Rate of				
	Environmental		Consumption		Daily Intake	
Exposure Route	Le	Levelsa		(bw)b	(ng/kg bw/d)	
Air	0.61 μ	0.61 μg/m3		m ³ /d	335.5	
Surface-Water	2.0	ng/L	0.1	L/d	0.2	
Biota (fish)	1.0	ng/g	155.0	g/d	155.0	
Total		_		_	490.7	

- a. The level in air is the maximum level measured in Canada; the level in surface water is the mean level measured in the Niagara River at the Niagara-on-the-Lake station in 1989; based on a water concentration of 2 ng/L and a maximum measured bioconcentration of 560, the estimated concentration in fish is approximately 1 ng/g (this value is comparable to the maximum level reported in lake trout from the Great Lakes in the early 1980s).
- b. Inhalation rate from Stahl (1967); drinking rate from Calder and Braun (1983); and ingestion rate from Nagy (1987), assuming a diet of 75% fish.

Data were not identified on the toxicological effects of 1,2-dichlorobenzene on birds and terrestrial plants.

Although on the basis of available data, the levels of 1,2-dichlorobenzene present in air and surface water are not expected to cause adverse effects in aquatic biota or wildlife, there was an absence of data upon which to evaluate the significance of concentrations in sediment to benthic biota. Therefore, there is insufficient information to conclude whether 1,2-dichlorobenzene is "toxic" as defined under paragraph 11(a) of CEPA.

3.2 CEPA 11(*b*): Environment on Which Human Health Depends

Although 1,2-dichlorobenzene is volatile at tropospheric temperatures and absorbs infrared radiation in wavelengths ranging from 7 to 13 μ m, this substance is removed from the atmosphere by photooxidation (mean half-life of approximately 3 weeks), resulting in low steady-state concentrations in the atmosphere (< 0.1 μ g/m³). As such, 1,2-dichlorobenzene is not expected to contribute significantly to formation of ground-level ozone, global warming or depletion of stratospheric ozone.

On the basis of available data, 1,2-dichlorobenzene is not considered to be "toxic" as defined under paragraph 11(b) of CEPA.

3.3 CEPA 11(c): Human Life or Health

Population Exposure

Although available data are limited, it is possible to estimate the intake of 1,2-dichlorobenzene for different age groups in the general population in Canada from various sources (Table 2).

Table 2 Estimated Daily Intake ($\mu g/kg$) of 1,2-Dichlorobenzene by Canadians from Various Sources

	Estimated Intake μg/kg bw/day						
Medium ¹	0 - 6 mo ^a	7 mo -4 yr ^b	5 - 11 yr ^c	12 - 19 yr ^d	20 - 70 ^e		
Ambient Airf	< 0.03	< 0.04	< 0.04	< 0.03	< 0.03		
Drinking Water ^g	_	0.0002 - < 0.001	0.0007 - < 0.0007	0.00007 - < 0.0005	< 0.00007 - < 0.0004		
Foodh	0.30 - 0.9 ⁱ	0.01	0.006	0.004	0.004		
Total Intake	0.33 - 0.93	0.04	0.05	0.03	0.03		

mo = months

- a. Assumed to weigh 7 kg, breathe 2 m³ of air per day, drink 750 mL of breast milk (as food) per day (Environmental Health Directorate, 1991)
- b. Assumed to weigh 13 kg, breathe 5 m³ of air per day, drink 0.8 L of water per day and to consume 77.66 g per day of an egg/meat composite (Environmental Health Directorate, 1991)
- c. Assumed to weigh 27 kg, breathe 12 m³ of air per day, drink 0.9 L of water per day and to consume 96.50 g per day of an egg/meat composite (Environmental Health Directorate, 1991)
- d. Assumed to weigh 57 kg, breathe 19 m³ of air per day, drink 1.3 L of water per day and to consume 140.51 g per day of an egg/meat composite (Environmental Health Directorate, 1991)
- e. Assumed to weigh 69 kg, breathe 23 m³ of air per day, drink 1.5 L of water per day and to consume 155.71 g per day of an egg/meat composite (Environmental Health Directorate, 1991)
- f. Based on a mean concentration of 1,2-dichlorobenzene reported in a survey of ambient air from 22 sites across Canada ($< 0.10 \, \mu g/m^3$) [Environment Canada, 1991]; concentrations in indoor air are similar to those in ambient air.
- g. Based on a range of mean concentrations of 1,2-dichlorobenzene in Canadian drinking water of $0.003~\mu g/L$ (Oliver and Nicol, 1982) to $< 0.02~\mu g/L$ (Environment Canada, 1989)
- h. Based on a concentration of $0.0018 \, \mu g/g$ 1,2-dichlorobenzene detected in a egg/meat composite from Ontario (Davies, 1988)
- i. Based on a range of mean concentrations of 1,2-dichlorobenzene detected in breast milk (3 to 8.1 ng/g) from the Canadian National Survey and a survey of Canadian Indigenous Populations (Davies and Mes, 1987) and assuming the density of breast milk is equal to 1.0

^{1.} Data were insufficient to estimate intake from soil.

On the basis of the available data, it is likely that the general population (other than suckling infants) is exposed to 1,2-dichlorobenzene principally in air, although for some age groups, intake in food is within the range of that estimated for air. It should be noted, however, that the data available on intake in food are limited to a small study of food composites (Davies, 1988). The total daily intake of 1,2-dichlorobenzene for age groups older than 6 months is estimated to range from approximately 0.03 μ g/kg bw to 0.05 μ g/kg bw. Based on the concentrations determined in mothers' milk, total intake for suckling infants aged 0 to 6 months is estimated to be considerably greater, from 0.33 μ g/kg bw to 0.93 μ g/kg bw.

Effects

Available data are inadequate to assess the carcinogenicity of 1,2-dichlorobenzene in humans. Although available data on the genotoxicity of 1,2-dichlorobenzene are limited and inconclusive, the substance has not been shown to be carcinogenic following ingestion in rats and mice in a well-conducted bioassay (NTP, 1983b). It has been classified, therefore, in Group IV (probably not carcinogenic to man) of the classification scheme developed for use in the derivation of the "Guidelines for Canadian Drinking Water Quality" (Environmental Health Directorate, 1989).

For compounds classified in Group IVC, a Tolerable Daily Intake (TDI) is derived on the basis of a No- or Lowest-Observed-(Adverse)-Effect-Level [NO(A)EL or LO(A)EL] divided by an uncertainty factor. The only data identified on long-term toxicity following inhalation are the results of an early subchronic study, for which no effects were observed at any concentration (Hollingsworth et al., 1958). Available data are considered inadequate, therefore, to establish a TDI on the basis of the results of studies in which 1,2-dichlorobenzene has been administered by inhalation. Moreover, although the general population appears to be exposed to 1,2-dichlorobenzene principally in air, based on limited available data on concentrations in food, the estimated intake in food is within the range of that estimated for air for some age groups. In addition, the principal route of intake of the most exposed age group (i.e., suckling infants) is ingestion (of mothers' milk). Owing to the lack of adequate long-term toxicity studies by the inhalation route and the possible relatively important contribution that food makes to total exposure to 1,2-dichlorobenzene, a TDI has been derived on the basis of the long-term NTP bioassay conducted by the oral route, as follows:

TDI =
$$\frac{60 \text{ mg/kg bw per day} \times 5}{100 \times 7} \approx 0.43 \text{ mg/kg (430 µg/kg) bw per day}$$

where:

- 60 mg/kg bw per day is the lowest NOEL in a well-conducted and documented longest-term (chronic and carcinogenesis) study performed to date based on tubular regeneration of the kidney observed at higher doses (NTP, 1983); this value is similar to the lowest reported NOEL (25 mg/kg bw per day) [Robinson *et al.*, 1991] in subchronic studies reported to date;
- 5/7 is the conversion of 5 days per week of dosing to 7 days per week; and
- 100 is the uncertainty factor (× 10 for intraspecies variation; × 10 for interspecies variation).

Based on the limited available data, the total estimated average daily intakes of 1,2-dichlorobenzene for various age groups in the population range from approximately $0.03~\mu g/kg$ bw to $0.93~\mu g/kg$ bw. These estimated average daily intakes are less (by approximately 460 to 14 000 times) than the TDI derived above. It should be noted, however, that with the exception of breast-fed infants, whose intakes are elevated for only a short period of their lifespan, the estimated average daily intakes are 8 600 to 14 000 times less than the TDI.

On the basis of available data, therefore, 1,2-dichlorobenzene is not considered to be "toxic" as defined under paragraph 11(c) of CEPA.

3.4 Conclusion

Based on available data, there is insufficient information to conclude whether 1,2-dichlorobenzene is "toxic" as defined under paragraph 11(a) of CEPA. 1,2-Dichlorobenzene is not considered to be "toxic" as defined under paragraphs 11(b) and 11(c) of CEPA.

4.0 Recommendations

Several significant data gaps were identified that limited the assessment of environmental effects. It is thus recommended that the following studies be conducted on a high-priority basis:

- (i) determine the effects of sediment-bound 1,2-dichlorobenzene on freshwater and marine benthos;
- (ii) determine current concentrations of 1,2-dichlorobenzene in sediment near Canadian textile and chemical manufacturing plants; and
- (iii) determine current quantities and resulting concentrations of 1,2-dichlorobenzene released to the atmosphere from its use as a carbon remover and metal degreaser in Canada.

In addition, to permit a more comprehensive assessment of the exposure of Canadians to 1,2-dichlorobenzene, additional monitoring data are desirable, particularly for food and breast milk, although, based on the wide margin between estimated exposure and the TDI the priority for this research is currently considered to be low.

5.0 References

Ahmad, N., D. Benoit, L. Brooke, D. Call, A. Carlson, D. DeFoe, J. Huot, A. Moriarity, J. Richter, P. Shubat, G. Veith, and C. Wallbridge. 1984. Aquatic toxicity tests to characterize the hazard of volatile organic chemicals in water: a toxicity data summary. Parts I and II. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Duluth, Minnesota.

Alberta Environment. 1985. Drinking water survey. Municipal Engineering Branch, Pollution Control Division.

Azouz, W.M., D.V. Parke, and R.T. Williams. 1955. Studies in detoxication: The metabolism of halogenobenzenes. ortho- and para-dichlorobenzenes. Biochem. J. 59:3: 410–415.

Barber, L.B. 1988. Dichlorobenzene in ground water: evidence for long-term persistence. Ground Water 26(6): 696–702.

Barber, L.B., E.M. Thurman, M.P. Schroeder, and D.R. LeBlanc. 1988. Long-term fate of organic micropollutants in sewage-contaminated groundwater. Environ. Sci. Technol. 22(2): 205–211.

Barrows, M.E., S.R. Petrocelli, and K.J. Macek. 1980. Bioconcentration and elimination by bluegill sunfish (*Lepomis macrochirus*). In: R. Haque, ed., Dynamics, Exposure and Hazard Assessment of Toxic Chemicals. Ann Arbor Science, Michigan, pp. 379–392.

Black, J.A., W.J. Birge, W.E. McDonnell, A.G. Westerman, B.A. Ramey, and D.M. Bruser. 1982. The aquatic toxicity of organic compounds to embryo-larval stages of fish and amphibians. Research Report No. 133. Water Resources Research Institute, University of Kentucky, Lexington, Kentucky, p. 61.

Bonnet, P., Y. Morele, G. Raoult, D. Zissu, and D. Gradiski. 1982. Détermination de la concentration léthale 50 des principaux hydrocarbures aromatiques chez les rats. Arch. Mal. Prof. 43(4): 461–465.

Bonnet, P., G. Raoult, and D. Gradiski. 1979. Concentrations léthales 50 des principaux hydrocarbures aromatiques. Arch. Mal. Prof. 40(8-9): 805-810.

Bosma, T.N.P., J.R. van der Meer, G. Schraa, Marijke E. Tros, and A.J.B. Zehnder. 1988. Reductive dechlorination of all trichloro- and dichlorobenzene isomers. FEMS Microbiolog. Ecol. 53: 223–229.

Brien, E.B. 1992, personal communication. Use Patterns Division, Commercial Chemicals Branch, Environment Canada.

Bringmann, G., and R. Kuhn. 1980. Comparison of the toxicity thresholds of water pollutants to bacteria, algae, and protozoa in the cell multiplication inhibition test. Water Res. 14: 231–241.

Bunce, N.J., J.P. Landers, J. Langshaw, and J.S. Nakai. 1987. Laboratory experiments to assess the importance of photochemical transformation during the atmospheric transport of chlorinated aromatic pollutants. 80th Annual meeting of APCA, June 21–26, 1987, New York.

Calamari, D., S. Galassi, F. Setti, and M. Vighi. 1983. Toxicity of selected chlorobenzenes to aquatic organisms. Chemosphere 12(2): 253–262.

Calder, W.A., and E.J. Braun. 1983. Scaling of osmotic regulation in mammals and birds. Am. J. Physiol. 244: R601–R606.

Callahan, M., M. Slimak, N. Gabel, I. May, C. Fowler, R. Freed, P. Jennings, R. Durfee, F. Whitmore, B. Maestri, W. Mabey, B. Holt, and C. Gould. 1979. Water-related environmental fate of 129 priority pollutants, Volume II. Office of Water Planning and Standards/Office of Water and Waste Management, U.S. Environmental Protection Agency, Section V (EPA 440/4-79-029b).

Camford Information Services Inc. 1991. Chlorobenzene CPI Product Profile. Don Mills, Ontario. 4 pp.

Chapman, P.M., R.W. Deverall, and D.G. Mitchell. 1987. Environmental Monitoring 1986 Iona Deep Sea Outfall Project. EVS Consultants Ltd., North Vancouver, British Columbia.

Chemical Marketing Reporter. July 9, 1990. Chemical Profile: o-Dichlorobenzene.

Davies, K. 1988. Concentrations and dietary intake of selected organochlorines, including PCBs, PCDDs and PCDFs in fresh food composites grown in Ontario, Canada. Chemosphere 17(2): 263–276.

Di Toro, D.M., C.S. Zarba, D.J. Hansen, W.J. Berry, R.C. Swartz, C.E. Cowan, S.P. Pavlou, H.E. Allen, N.A. Thomas, and P.R. Paquin. 1991. Technical basis for establishing sediment quality criteria for nonionic organic chemicals using equilibrium partitioning. Environ. Toxicol. Chem. 10: 1541–1583.

Durham, R.W., and B.G. Oliver. 1983. History of Lake Ontario contamination from the Niagara River by sediment radiodating and chlorinated hydrocarbon analysis. J. Great Lakes Res. 9(2): 160–168.

Ellington, J.J., F.E. Stancil, W.D. Payne, and D.C. Trusty. 1988. Measurement of hydrolysis rate constants for evaluation of hazardous waste land disposal, Volume 3: Data on 70 chemicals. Environmental Research Laboratory, U.S. Environmental Protection Agency (EPA/600/3-88/-28).

Environment Canada. 1983. Use pattern of chlorobenzenes. Unpublished. Use Patterns Division. Commercial Chemicals Branch, Environment Canada, p. 5.

Environment Canada. 1989. Environmental assessment of the Canadian textile industry. Chemical Industries Division, Industrial Programs Branch, Environment Canada. EPS 5/TX/1. 104 pp.

Environment Canada. 1989a. Atlantic Region Federal-Provincial toxic chemical survey of municipal drinking water sources. Data Summary Report, Province of New Brunswick, 1985–1988. Inland Waters Directorate, Water Quality Branch. IWD-AR-WQB-89-155.

Environment Canada. 1989b. Atlantic Region Federal-Provincial toxic chemical survey of municipal drinking water sources. Data Summary Report, Province of Prince Edward Island, 1985–1988. Inland Waters Directorate, Water Quality Branch. IWD-AR-WQB-89-156.

Environment Canada. 1989c. Atlantic Region Federal-Provincial toxic chemical survey of municipal drinking water sources. Data Summary Report, Province of Newfoundland, 1985–1988. Inland Waters Directorate, Water Quality Branch. IWD-AR-WQB-89-157.

Environment Canada. 1989d. Atlantic Region Federal-Provincial toxic chemical survey of municipal drinking water sources. Data Summary Report, Province of Nova Scotia, 1985–1988. Inland Waters Directorate, Water Quality Branch. IWD-AR-WQB-89-154.

Environment Canada. 1991. Update and summary report. Measurement program for toxic contaminants in Canadian urban air. Unpublished. River Road Environmental Technology Centre. PMD 91–2.

Environmental Health Directorate. 1989. Derivation of maximum acceptable concentrations and aesthetic objectives for chemicals in drinking water. Guidelines for Canadian Drinking Water Quality – Supporting Documentation. Bureau of Chemical Hazards, Health Canada.

EVS Consultants. 1992. Sediment and related investigations off the MaCaulay and Clover Point sewage outfall. Final report to the Capital Regional District, Victoria, British Columbia. 193 pp.

Fanning, M.L., D.J. Jones, D.W. Larson, and R.G. Hunter. 1989. Environmental Monitoring 1987 Iona Deep Sea Outfall Project. Beak Associates Consulting (B.C.) Ltd., Vancouver, British Columbia.

Fox, M.E., J.H. Carey, and B.G. Oliver. 1983. Compartmental distribution of organochlorine contaminants in the Niagara River and the Western Basin of Lake Ontario. J. Great Lakes Res. 9(2): 287–294.

Girard, R., P. Martin, and J. Bourret. 1969. Hémopathies graves et exposition à des dérivés chlorés du Benzene (à propos de 7 cas). J. Med. Lyon. 50(1164): 771–773.

Haider, K., G. Jagnow, R. Kohnen, and S.U. Lim. 1981. Degradation of chlorinated benzenes, phenols and cyclohexane derivatives by benzene- and phenol-utilizing soil bacteria under aerobic conditions. In: M.R. Overcash, ed. Decomposition of Toxic and Nontoxic Organic Compounds in Soil. Ann Arbor Science, Ann Arbor, Michigan.

Hayes, W.C., T.R. Hanley, T.S. Gushow, K.A. Johnson, and J.A. John. 1985. Teratogenic potential of inhaled dichlorobenzenes in rats and rabbits. Fundam. Appl. Toxicol. 5(1): 190–202.

Holliday, M.G., and F.R. Engelhardt. 1984a. Chlorinated benzenes. A criteria review. Prepared for Monitoring and Criteria Division, Bureau of Chemical Hazards, Health Canada, Ottawa.

Holliday, M.G., F.R. Engelhardt, and I. MacLachlan. 1984b. Chlorobenzenes: an environmental health perspective. Prepared for Health Canada, Ottawa.

Hollingsworth, R.L., V.K. Rowe, F. Oyen, T.R. Torkelson, and E.M. Adam. 1958. Toxicity of o-dichlorobenzene; studies on animals and industrial experience. Arch. Ind. Health 17: 180–187.

Howard, P.H. 1989. Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Volume I: Large Production and Priority Pollutants. Lewis Publishers, Chelsea, Michigan. 574 pp.

Howard, P.H. 1991. Handbook of environmental degradation rates. Lewis Publishers, Inc., Chelsea, Michigan. 725 pp.

Jacobs, A., M. Blangetti, and E. Hellmund. 1974a. Accumulation of noxious chlorinated substances from Rhine River water in the fatty tissue of rats. Vom Wasser. 43: 259–274. (Cited in U.S. EPA, 1985).

Jacobs, A., M. Blangetti, and E. Hellmund, 1974b. Accumulation of organic compounds, identified as harmful substances in Rhine water in the fatty tissue of rats. Dernforschungszentrum Karlsruhe. KRK 2969, UF.P.1 (abstract). (Cited in U.S. EPA, 1985).

Kao, C.I., and N. Poffenberger. 1979. Chlorinated benzenes. In: M. Grayson and D. Eckroth, Kirk-Othmer Encyclopedia of Chemical Technology. 3rd Edition. Volume 5. John Wiley and Sons, Toronto, Ontario, 797–808.

Ligocki, M.P., C. Leuenberger, and J.F. Pankow. 1985. Trace organic compounds in rain-II. Gas scavenging of neutral organic compounds. Atmos. Environ. 19(10): 1609–1617.

Mackay, D., W.Y. Shiu, and K.C. Ma. 1992. The Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals, Volume I: Monoaromatic Hydrocarbons, Chlorobenzenes and PCBs. Lewis Publishers, Inc., Chelsea, Michigan. 697 pp.

MacLaren Marex Inc. 1979. Report on an Environmental Survey for Chlorobenzenes at Four Coastal Sites in Nova Scotia. Unpublished report prepared for Environment Canada, Environmental Protection Service, Halifax, Nova Scotia. 18 pp.

Martel, R., and P. Ayotte. 1989. État de la situation sur la contamination de la nappe souterraine dans la région de la ville de Mercier. Ministère de l'Environnement du Québec.

McCall, J.P., D.A. Laskowski, R.L. Swann, and H.J. Dishburger. 1981. Measurement of sorption coefficients of organic chemicals and their use in environmental fate analysis. In: Test protocols for environmental fate and movement of toxicants. Proceedings of a symposium, Association of Official Analytical Chemists, 94th Annual Meeting, October 21–22, 1980. Washington.

Mes, J., D.J. Davies, D. Turton, and W.F. Sun. 1986. Levels and trends of chlorinated hydrocarbon contaminants in the breast milk of Canadian women. Food Additives and Contaminants 3(4): 313–322.

Nagy, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Mono. 57: 111–128.

NRDIG (Niagara River Data Interpretation Group). 1990. Joint evaluation of upstream/downstream Niagara River monitoring data. 1988–1989. 72 pp.

NTP (National Toxicology Program). 1983. Technical report on the carcinogenesis studies of 1,2-dichlorobenzene (CAS No. 95-50-1) in F344/N rats and B6C3F₁ mice (gavage studies). NTP TR 255, U.S. Dept. of Health and Human Services, Research Triangle Park, North Carolina. p. 192.

Oldenhuis, R., L. Kuijk, A. Lammers, D.B. Janssen, and B. Witholt. 1989. Degradation of chlorinated and non-chlorinated aromatic solvents in soil suspensions by pure bacterial cultures. Appl. Microbiol. Biotechnol. 30: 211–217.

Oliver, B.G. 1987. Biouptake of chlorinated hydrocarbons from laboratory-spiked and field sediments by oligochaete worms. Environ. Sci. Technol. 21(8): 785–790.

Oliver, B.G. 1992, personal communication. Zenon Environmental Laboratories, Burnaby, British Columbia.

Oliver, B.G., and K.D. Bothen. 1982. Extraction and clean-up procedures for measuring chlorobenzenes in sediments and fish by capillary gas chromatography. Intern. J. Environ. Anal. Chem. 12: 131–139.

Oliver, B.G., and K.D. Nicol. 1982a. Gas chromatographic determination of chlorobenzenes and other chlorinated hydrocarbons in environmental samples using fused silica capillary columns. Chromatographia 16: 336–340.

Oliver, B.G., and K.D. Nicol. 1982b. Chlorobenzenes in sediments, water, and selected fish from lakes Superior, Huron, Erie, and Ontario. Environ. Sci. Technol. 16(8): 532–536.

Oliver B.G., and K.D. Nicol. 1984. Chlorinated contaminants in the Niagara River, 1981–1983. Sci. Total Environ. 39: 57–70.

Oliver, B.G., and A.J. Niimi. 1983. Bioconcentration of chlorobenzenes from water by rainbow trout: correlations with partition coefficients and environmental residues. Environ. Sci. Technol. 17: 287–291.

Oliver, B.G., and C.W. Pugsley. 1986. Chlorinated contaminants in St. Clair River sediments. Water Poll. Res. J. Canada 21(3): 368–379.

OME (Ontario Ministry of the Environment). 1991a. Preliminary report on the first six months of process effluent monitoring in the MISA pulp and paper sector (January 1, 1990 to June 30, 1990). Municipal/Industrial Strategy for Abatement (MISA), Water Resources Branch, Ontario Ministry of the Environment. 176 pp.

OME (Ontario Ministry of the Environment). 1991b. Preliminary report on the second six months of process effluent monitoring in the MISA pulp and paper sector (July 1, 1990 to December 31, 1990). Municipal/Industrial Strategy for Abatement (MISA), Water Resources Branch, Ontario Ministry of the Environment. 159 pp.

OME (Ontario Ministry of the Environment). 1992. Twelve month report. Unpublished. Organic chemical manufacturing sector (October 1, 1989 to September 30, 1990). Municipal Strategy for Abatement (MISA). 81 pp.

OME (Ontario Ministry of the Environment). 1992a. Six month monitoring data report. Organic manufacturing sector (October 1, 1989 to March 31, 1990). Municipal Strategy for Abatement (MISA). 123 pp.

OME (Ontario Ministry of the Environment). 1992b. Twelve month monitoring data report. Inorganic chemical sector. Periods covered: December 1, 1989 to November 30, 1990; February 1, 1990 to January 31, 1991. Municipal Strategy for Abatement (MISA). 81 pp.

Otson, R., and F.M. Benoit. 1986. In: D.S. Walkenshaw, ed. Surveys of selected organics in residential air. Transactions - Indoor air quality in cold climates – Hazards and abatement measures April 1985. Air Pollution Control Association, Pittsburgh, Pennsylvania, pp. 224–236.

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

Park, J.M. 1992, draft. Organic characterization of Iona Wastewater, dry-weather flow period sampling. Analytical Service Laboratories Ltd, Vancouver, British Columbia.

Park, J.M., J. Close, L. Rerup, and S. Hannam. 1990. Analysis of Iona effluent, Analytical Service Laboratories Ltd, Vancouver, British Columbia.

Park, K.S., D.L. Sorensen, J.L. Sims, and F.D. Adams. 1988. Volatilization of wastewater trace organics in slow rate land treatment systems. Hazard. Waste & Hazard. Mat. 5(3): 219–229.

Pellizzari, E.D., T.D. Hartwell, R.L. Perritt, C.M. Sparacino, L.S. Sheldon, H.S. Zelon, R.W. Whitmore, J.J. Breen, and L. Wallace. 1986. Comparison of indoor and outdoor residential levels of volatile organic chemicals in five U.S. geographical areas. Environ. Int. 12(6): 619–623.

Reid, W.D. 1973. Mechanism of renal necrosis induced by bromobenzene or chlorobenzene. Exp. Mol. Pathol. 19: 197–214.

Reid, W.D., and Krishna, G. 1973. Centrolobular hepatic necrosis related to covalent binding of metabolites of halogenated aromatic hydrocarbons. Exp. Mol. Pathol. 18: 80-99.

Reinhard, M., N.L. Goodman, and J.F. Barker. 1984. Occurrence and distribution of organic chemicals in two landfill leachate plumes. Environ. Sci. Technol. 18(12): 953–961.

Roberts, P.V., M.N. Goltz, and D.M. Mackay. 1986. A natural gradient experiment on solute transport in a sand aquifer. 3: Retardation estimates and mass balances for organic solutes. Water Res. Research 22(13): 2047–2058.

Roberts, P.V., P.L. McCarty, M. Reinhard, and J. Schreiner. 1980. Organic contaminant behaviour during groundwater recharge. Journal WPCF. 52(1): 161–172.

Robinson, M., J.P. Bercz, H.P. Ringhand, L.W. Condie, and Parnell, M.J. 1991. Ten- and ninety-day toxicity studies of 1,2-dichlorobenzene administered by oral gavage to Sprague-Dawley rats. Drug and Chemical Toxicology. 14(1 & 2): 83–112.

Sadtler Research Laboratories. 1982. The Sadtler Infrared Spectra Handbook of Priority Pollutants and Toxic Chemicals. Philadelphia, Pennsylvania.

Schraa, G., and J.R. van der Meer. 1987. Biotransformations of di- and trichlorinated benzenes. In: C.W. Moody, and P.B. Baker, eds. International Conference on Bioreactors and Biotransformations, Gleneagles, Scotland, pp. 253–262.

Slimak K., P. Johnston, and V. Hodge. 1980. Materials Balance for Chlorobenzenes. U.S. Environmental Protection Agency, Office of Toxic substances, Washington. January 1980: 3-1 to 3-19, EPA-560/13-80-001 (PB80-173651).

Stahl, W.R. 1967. Scaling of respiratory variables in mammals. J. Appl. Physiol. 22: 453–460.

Strahlendorf, P.W. 1978. Chlorinated benzenes as potential environmental health hazards: a review. Prepared for Monitoring and Criteria Section, Health Protection Branch, Health Canada, Ottawa.

Sylvestre, A. 1987. Organochlorines and polyaromatic hydrocarbons in the St. Lawrence River at Wolfe Island, 1982/84. Technical Bulletin No. 144, IWD Ontario Region Water Quality Branch. 11 pp.

Tolot, F., B. Soubrier, J.-R. Bresson, and P. Martin, 1969. Myélose proliférative d'évolution rapide. Rôle étiologique possible des dérivés chlorés du benzène. J. Med. Lyon 1164(3F): 761–768.

U.S. EPA. 1985. Health assessment document for chlorinated benzenes. Final Report. Office of Health and Environment Assessment, Washington, U.S. Environmental Protection Agency (EPA/600/8-84/015F).

U.S. EPA. 1986. Locating and estimating air emissions from sources of chlorobenzenes. U.S. Environmental Protection Agency, Office of Air Quality (EPA-450/4-84-007m). 135 pp.

U.S. EPA. 1987. Occurrence of synthetic organic chemicals in drinking water, food, and air. Revised Draft Report. U.S. Environmental Protection Agency, Office of Drinking Water (PB89-192520). 175 pp.

Vachon, J. 1986, personal communication. Direction de l'eau souterraine et potable, Ministère de l'environnement, Québec.

van der Meer, J.R., W. Roelofsen, G. Schraa, and A.J.B. Zehnder. 1987. Degradation of low concentrations of dichlorobenzenes and 1,2,4-trichlorobenzene by *Pseudomonas* sp. strain P51 in nonsterile soil columns. FEMS Microbiol. Ecology 45: 333–341.

Webber, M.D., and S. Lesage. 1989. Organic contaminants in Canadian municipal sludges. Waste Management & Research 7: 63–82.

Weber, W.J., B.E. Jones, and L.E. Katz. 1987. Fate of toxic organic compounds in activated sludge and integrated PAC systems. Wat. Sci. Tech. 19: 471–482.

Yang, K.H., R.E. Peterson, and J.M. Fujimoto. 1979. Increased bile duct-pancreatic fluid flow in benzene-treated rats. Toxicol. Appl. Pharmacol. 47(3): 505–514.

Young, C.M., and K.J. Voorhees. 1989. Thermal decomposition of 1,2-dichlorobenzene. American Chemical Society 3(1): 280–287.

Zapata-Gayon, C., N. Zapata-Gayon, and A. Gonzalez-Angulo. 1982. Clastogenic chromosomal aberrations in 26 individuals accidentally exposed to ortho dichlorobenzene vapours in the National Medical Centre in Mexico City. Arch. Environ. Health 37(4): 231–235.

Zoeteman, B.C.J., K. Harmsen, J.B.H.J. Linders, C.F.H. Morra, and W. Slooff. 1980. Persistent organic pollutants in river water and ground water of the Netherlands. Chemosphere 9: 231–249.