*Canadian Environmental Protection Act*

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Priority Substances List
Assessment Report

# 1,4-Dichlorobenzene

Government of Canada
Environment Canada
Health Canada

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# Synopsis

1,4-Dichlorobenzene is produced in and imported into Canada. Approximately 3 500 tonnes per year are currently used in Canada as an air freshener, as a deodorizer in urinals, and as a moth and bird repellent. In Canada 1,4-dichlorobenzene is present at measurable concentrations in municipal and industrial effluents, ambient and indoor air, ground and surface water, sediment, and biota. 1,4-Dichlorobenzene is not persistent in air or surface water but persists and accumulates in sediment under anaerobic conditions.

The maximum concentration of 1,4-dichlorobenzene in surface waters in Canada was approximately 300 times less than the effects threshold estimated for the most sensitive aquatic species identified. 1,4-Dichlorobenzene is volatile but the highest mean level of 1,4-dichlorobenzene in Canadian air was approximately 12 700 times less than the effects threshold estimated for wild mammals in studies in which inhalation was the principal route of exposure. No data on the toxicological effects of 1,4-dichlorobenzene to benthic organisms were identified. Therefore, it was not possible to determine whether 1,4-dichlorobenzene concentrations found in sediments could result in harmful effects to these biota.

1,4-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 limited available data on concentrations in ambient and indoor air, drinking water and food, the average total daily intakes of 1,4-dichlorobenzene for various age groups in the general population have been estimated. These average daily intakes are less (by approximately 40 to 780 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 harmful effect.

**Based on these considerations, it has been determined that there is insufficient information to conclude whether 1,4-dichlorobenzene is entering the environment in quantities or under conditions that may be harmful to the environment.**
**It has, however, been concluded that 1,4-dichlorobenzene is not entering the environment in quantities or under conditions that may constitute a danger to the environment on which human life depends, or to human life or health.**

# 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 that are 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 1,4-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 adverse **effects**.

The assessment of whether the 1,4-dichlorobenzene is "toxic" to human health under CEPA, is based principally on documentation prepared by staff of Health Canada for the 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 Health Canada staff in the preparation of a draft IPCS Environmental Health Criteria Document (EHC). The current assessment has been updated and expanded to emphasize data most relevant to the assessment of the risks associated with exposure to 1,4-dichlorobenzene in the general environment in Canada.

In preparation of the IPCS document, a wide variety of scientific databases were searched to update information provided in earlier contractors' reports. These earlier reports included 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 who met in June 1990. In February 1991, a search of ENVIROLINE, Chemical Abstracts, Pollution Abstracts, Environmental Bibliography, IRIS, MEDLINE, and BIOSIS databases was conducted to identify recent data relevant to assessment, in particular, of the risks to Canadians. Data relevant to assessment of whether 1,4-dichlorobenzene is "toxic" to human health obtained after completion of these sections of this report (i.e., May 1992), were not considered for inclusion.

Data relevant to the assessment of whether 1,4-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), CIS, 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 of relevant journals as well as *Current Contents* (agriculture, biology, and environmental sciences). Information received after December 1992 was not included in the environmental sections of this report. Although much of the research on 1,4-dichlorobenzene has been conducted outside of Canada, where possible, Canadian data on sources, use patterns, fate, and effects of this substance on the environment were emphasized.

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 (human exposure and effects on human health) and Environment Canada (entry and environmental exposure and effects). The following officials contributed to preparation of the report:

B. Elliott (Environment Canada)
C. Fortin (Environment Canada)
M. Giddings (Health Canada)
R.G. Liteplo (Health Canada)
K. Lloyd (Environment Canada)
M.E. Meek (Health Canada)

R. Gomes of Health Canada also contributed to the consolidation of the Assessment Report.

In this report, a synopsis is presented that will appear in the *Canada Gazette*. Section 2.0 presents a summary of the technical information critical to the assessment, which is presented in greater detail in unpublished supporting documentation. The assessment of whether 1,4-dichlorobenzene is “toxic”, as defined under CEPA, is presented in Section 3.0.

As part of the review and approvals process established by Environment Canada, 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 the CEPA Management Committee of Environment Canada and Health Canada.

Copies of this Assessment Report and the unpublished supporting documentation are available upon request from the following:

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# 2.0 Summary of Information Critical to Assessment of "Toxic"

## 2.1 Identity, Properties, Production and Uses

1,4-Dichlorobenzene, also known as para-dichlorobenzene or p-dichlorobenzene, is a neutral, colourless, flammable solid (U.S. EPA, 1986) with a molecular weight of 147.01 and the empirical formula $C_6H_4Cl_2$. 1,4-Dichlorobenzene has a moderate vapour pressure (90 Pa @ 25°C), a low water solubility (79 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 the log $K_{ow}$ value suggested by Mackay *et al.* (1992), is 1 030. 1,4-Dichlorobenzene absorbs infrared radiation, including wavelengths in the 7-13 μm region (Sadtler Research Laboratories, 1982).

1,4-Dichlorobenzene is produced by chlorination of benzene in the liquid phase with a catalyst. This substance typically contains less than 0.1% monochlorobenzene and trichlorobenzenes, and less than 0.5% each of 1,2- and 1,3-dichlorobenzene (Kao and Poffenberger, 1979). The analytical methods by which 1,4-dichlorobenzene is quantified in environmental media include gas chromatography with flame ionization or electron capture detection and gas chromatography/mass spectrometry (Oliver and Nicol, 1982; Oliver and Bothen, 1982).

1,4-Dichlorobenzene is produced in and imported into Canada (Camford Information Services, 1991). In the past few years, the amount imported into Canada has increased while domestic production has declined. Overall, the Canadian demand for 1,4-dichlorobenzene has remained steady at approximately 3 500 tonnes per year over the past 5 years. This number is not expected to change during the next 5 years (Camford Information Services, 1991). The amount of 1,4-dichlorobenzene produced in the United States in 1990 was 62.6 kilotonnes (Chemical Marketing Reporter, 1990).

In Canada, 1,4-dichlorobenzene is used as an air freshener, a urinal deodorizer, and in moth and bird repellents (Camford Information Services, 1991). Quantitative data on use patterns for this substance were only identified for the period 1977 to 1979, when 9.3% of the annual demand was used in the production of deodorant blocks, and 0.7% was used in the production of moth crystals (Environment Canada, 1983, unpublished). In the United States, 1,4-dichlorobenzene has a wider variety of applications, including use in the synthesis of polyphenylene sulfide (PPS) resin and 1,2,4-trichlorobenzene (Chemical Marketing Reporter, 1990).

## 2.2 Entry into the Environment

There are no known natural sources of 1,4-dichlorobenzene, and quantitative information on its release into 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 all of the 3 500 tonnes of 1,4-dichlorobenzene used annually in Canada are released into the environment. It has also been suggested that 1,4-dichlorobenzene may be produced from the dehalogenation of more highly chlorinated chlorobenzenes (Bosma *et al.*, 1988; Holliger *et al.*, 1992; Fathepure *et al.*, 1988).

Data concerning the release of 1,4-dichlorobenzene into the atmosphere from industrial and municipal facilities in Canada were not identified. However, releases of the substance in liquid effluents from these facilities have been reported. Under the Ontario Municipal/Industrial Strategy for Abatement (MISA) program, 1,4-dichlorobenzene was detected in effluents discharged from organic chemical manufacturing plants into the St. Clair River at Sarnia, and Corunna, Ontario, between October 1, 1989 and January 31, 1991. The amounts released were estimated to range between 0.002 and 0.365 kg/day. Also under the MISA program, loading values ranging between 0.001 and 0.055 kg/day were reported for the St. Lawrence, Welland, and Mary's Rivers (OME, 1992, unpublished; OME, 1992a, 1992b).

1,4-Dichlorobenzene was detected (mean mass loading value of 143 g/day) in waste water treatment plant effluents discharged into the Strait of Georgia in the Vancouver area between March and December 1987 (Fanning *et al.*, 1989). Based on the results of a study conducted between 1985 and 1986, 1,4-dichlorobenzene was also present in effluent from 5 of 10 textile mills located in Ontario and Quebec (Environment Canada, 1989a) where concentrations as high as 71.1 μg/L were reported. 1,4-Dichlorobenzene was detected at concentrations up to 9.6 μg/L in process effluents from pulp and paper mills in Ontario (OME, 1991a, 1991b). Mean concentrations ranging from 900 to 1 400 ng/L 1,4-dichlorobenzene were detected in effluents from water pollution control plants at Galt, Waterloo, and Welland, Ontario (Melcer *et al.*, 1988). 1,4-Dichlorobenzene was also detected in one third of the samples of digested sludge collected between 1980 and 1985 from 15 Canadian municipal treatment facilities where the maximum concentration was 1 500 ng/g (Webber and Lesage, 1989). The highest concentration of 1,4-dichlorobenzene (detection limit = 220 ng/g; 4% detection frequency), in treated sludge obtained from 2 of 37 municipal water pollution control plants in Ontario in 1987 was 2 644 ng/g (OME, 1988).

## 2.3 Exposure-related Information

### *2.3.1 Fate*

There are a number of processes that affect the distribution and transformation of 1,4-dichlorobenzene in the environment, including the following: atmospheric photo-oxidation; volatilization; partitioning to soil, sediment and biota; and aerobic degradation (Howard *et al.*, 1989; Howard, 1991; Mackay *et al.*, 1992; Garrison and Hill, 1972, Wakeham *et al.*, 1983; Callahan *et al.*, 1979; Ellington *et al.*, 1988). 1,4-Dichlorobenzene that is not removed from the environment by degradative processes, ultimately accumulates in anaerobic sediments and possibly groundwater.

1,4-Dichlorobenzene absorbs radiation weakly at wavelengths greater than 300 nm, and therefore, direct photolysis of this substance in the atmosphere is not likely (Bunce *et al.*, 1987). However, 1,4-dichlorobenzene will react with photochemically produced hydroxyl radicals in the atmosphere with an estimated half-life of 31 days (Howard, 1989). Mackay *et al.*, (1992) estimated a mean half-life in the atmosphere of approximately 3 weeks for 1,4-dichlorobenzene on the basis of photo-oxidation and advection processes. The presence of 1,4-dichlorobenzene in rainwater indicates that it persists long enough to be returned to the earth's surface by atmospheric wash out (Ligocki *et al.*, 1985).

Volatilization is the predominant mechanism for the removal of 1,4-dichlorobenzene from surface water and soil (Mackay *et al.*, 1992; Callahan *et al.*, 1979; Garrison and Hill, 1972; Wakeham *et al.*, 1983; Howard, 1991). Volatilization half-lives ranging between < 1 day and 31 days have been reported (Wakeham *et al.*, 1983; Howard *et al.*, 1989).

1,4-Dichlorobenzene is slowly biodegraded in soil under aerobic conditions (Bouwer *et al.*, 1981; Haider *et al.*, 1981; van der Meer *et al.*, 1987). Mackay *et al.* (1992) selected a half-life of approximately 8 months for 1,4-dichlorobenzene in soil, while Howard (1991) reported a half-life ranging from 4 weeks to 6 months. Based on its estimated organic carbon sorption coefficient ($K_{oc}$) of 1 030, the soil mobility potential of 1,4-dichlorobenzene was classified as low, according to the scale presented by McCall *et al.* (1981). However, evidence for leaching of 1,4-dichlorobenzene through soil has been reported by Elder *et al.*, (1981), Zoeteman *et al.*, (1980), and Wilson *et al.* (1981). The presence of 1,4-dichlorobenzene in a plume of contaminated groundwater a distance of 3 500 metres from sewage disposal beds confirms the persistence of this substance under anaerobic conditions (Barber, 1988).

1,4-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, 1982). On the basis of an analysis of core

sediment samples obtained from Lake Ontario, it has been reported that 1,4-dichlorobenzene has been accumulating over a period of 60 years, with the highest concentrations correlating with peak production of chlorobenzenes in North America during the 1960s (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 1,4-dichlorobenzene in the first 1 cm of sediment. Within sediments, 1,4-dichlorobenzene is expected to equilibrate between the pore water and the organic phase (Di Toro *et al.*, 1991).

Oliver and Nimii (1983) reported bioconcentration factors ranging from 370 to 720 (whole fish) for the rainbow trout (*Oncorhynchus mykiss*) exposed to 1,4-dichlorobenzene under laboratory conditions. Bioconcentration factors ranged from 100 to 450 and increased to 1 400 at day 23 of a 60-day early life stage (egg to 2-week alevin stage) study of the rainbow trout, in a continuous flow system (Calamari *et al.*, 1982). Biological half-lives of less than 1 day and less than 5 days were reported for the bluegill sunfish (*Lepomis macrochirus*) [Barrows *et al.*, 1980] and the oligochaete worms (*Tubifex tubifex* and *Limnodrilus hoffmeisteri*) [Oliver, 1987], respectively.

### *2.3.2 Concentrations*

1,4-Dichlorobenzene has been detected in air, surface water, groundwater, drinking water, sediments, biota, and food in Canada.

The mean concentration of 1,4-dichlorobenzene in ambient air sampled between October 1988 and December 1990 at 23 sites across Canada was 0.92 $\mu g/m^3$ (Environment Canada, 1991). 1,4-Dichlorobenzene was measured above the detection limit (0.1 $\mu g/m^3$) in 99% of the samples, and the mean levels of 1,4-dichlorobenzene ranged from 0.22 to 2.94 $\mu g/m^3$ (Environment Canada, 1991). The maximum (24-hour) concentrations were highest in industrial sections of Vancouver (4.82 $\mu g/m^3$), Toronto (15.7 $\mu g/m^3$), and Windsor (14.6 $\mu g/m^3$). The mean concentration of 1,4-dichlorobenzene in air sampled between February 1989 and November 1990 at a rural site (Walpole Island) in Ontario was 1.37 $\mu g/m^3$; the maximum level was 3.53 $\mu g/m^3$ (Environment Canada, 1991).

Concentrations of 1,4-dichlorobenzene in indoor air are often higher than those in ambient air because of its presence in consumer products (e.g., moth repellents and space deodorants). In a 1984 study of 188 homes in Los Angeles and Contra Costa, California, the ratios of median overnight concentrations of 1,4-dichlorobenzene in indoor/outdoor air in a sub-sample of 57 homes ranged from 1.4 to 1.8 (Wallace *et al.*, 1988). It is possible that ratios may be higher in colder climates, because of better insulation and less ventilation in winter, although reliable relevant data on ratios of concentrations in indoor/outdoor air in Canada were not identified.

Mean and median concentrations of 1,3- and 1,4-dichlorobenzene (combined) in indoor air in the U.S. Total Exposure Assessment Methodology (TEAM) study ranged from 0.44 to 71 μg/m$^3$ (Wallace *et al.*, 1985, 1986; Wallace, 1986; Pellizzari *et al.*, 1982). In the only report of the TEAM study in which 1,4-dichlorobenzene was measured separately, median overnight concentrations in indoor air in 57 homes in California ranged from 0.4 to 2.8 μg/m$^3$ (Wallace *et al.*, 1988). The range of individual measured concentrations is often very broad, with maximum values sometimes exceeding 1 000 μg/m$^3$ (1,3- and 1,4-dichlorobenzene combined) [Pellizari *et al.*, 1982]. Information on concentrations of 1,4-dichlorobenzene in indoor air in Canada, available at the time of completion of the health-related sections of this assessment, was restricted to a limited and probably unrepresentative number of homes (Chan *et al.*, 1990). The average concentration of 1,4-dichlorobenzene (detection limit = 6.0 ng/tube) in 12 homes sampled in November and December, 1986 was 15.0 μg/m$^3$ (range 1 to 107 μg/m$^3$) [Chan *et al.*, 1990]. The average concentration in ambient air outside these homes was 0.3 μg/m$^3$. In repeat sampling of 6 of these homes in February and March 1987, 1,4-dichlorobenzene was not detected in 3 (detection limit = 6 ng/tube), while the reported mean concentration in ambient air was 10.2 μg/m$^3$.

For samples of surface water collected between 1986 and 1989, the mean annual concentrations of 1,4-dichlorobenzene in the Niagara River were reported to range between 0.82 ng/L at Fort Erie and 2.47 ng/L at Niagara-on-the-Lake (NRDIG, 1988, 1990). Based on a monitoring program of surface and raw drinking water supplies in six provinces (Alberta, Quebec, New Brunswick, Nova Scotia, Newfoundland, and Prince Edward Island) conducted from February 1987 to May 1989, the levels of 1,4-dichlorobenzene in surface water ranged between 0.57 to 130 ng/L in the provinces where it was detected (Environment Canada, 1992).

1,4-Dichlorobenzene was detected in groundwater adjacent to landfill sites in North Bay, and Burlington, Ontario, and at an industrial waste site near Ville Mercier, Quebec. Concentrations ranged from 2 000 to 40 000 ng/L (Reinhard *et al.*, 1984; Pankow *et al.*, 1984 and 1985; Martel and Ayotte, 1989). A maximum concentration of 19 000 ng/L was reported for untreated leachate obtained from a chemical manufacturing plant landfill in Sarnia, Ontario in 1985 (King and Sherbin, 1986).

Information on concentrations of 1,4-dichlorobenzene in Canadian drinking water supplies is limited. 1,4-Dichlorobenzene was detected in 6 of 90 potable water samples taken at 30 water treatment facilities across Canada between August and December 1979 (Otson *et al.*, 1982a, 1982b). Mean and maximum concentrations were less than 1 μg/L (the limit of detection for 1,4-dichlorobenzene was not specified, however, for 1,2-dichlorobenzene and 1,2,4-trichlorobenzene, the quantitation limit was 1 μg/L). Based on analysis of dichlorobenzenes in the water supplies of three cities in Ontario, Oliver and Nicol (1982) concluded that most of the dichlorobenzene present in

drinking water occurs as the 1,4-isomer, due probably to its release into surface water from urinal deodorant blocks. Concentrations in this study ranged from 8 to 20 ppt with a mean of 13 ppt (0.013 μg/L) [detection limit ~ 1 ppt (0.001 μg/L)]. The 1,4-isomer of dichlorobenzene was detected in 4 of 143 samples of raw and treated water collected in Quebec in May 1985, and February and July 1986 at concentrations below 1 μg/L (detection limit = 0.1 μg/L) [Vachon, 1986]. Traces of 1,4-dichlorobenzene were detected (detection limit < 1 μg/L) in 3 of 29 treated municipal water supplies in Alberta during 1980 to 1985 (Alberta Environment, 1985). In a survey of drinking water from 139 locations in the 4 Atlantic provinces between 1985 and 1988, the dichlorobenzenes were not detected in 1 210 samples analyzed (detection limit = 0.02 μg/L). However, it should be noted that 23% and 77% of the samples taken for analysis of 1,2- and 1,4-dichlorobenzene isomers, respectively, were contaminated and therefore not analyzed or included (Environment Canada, 1989b, 1989c, 1989d, 1989e).

Recent data on the concentrations of 1,4-dichlorobenzene in sediments in Canada are limited to one investigation of the effects of municipal sewage outfalls from Victoria, British Columbia, on sea bottom sediments. 1,4-Dichlorobenzene was consistently detected in sediments at the Macaulay Point outfall at concentrations ranging from 1 to 1 710 ng/g (mean 141 ng/g) [dry weight] (EVS Consultants, 1992). All other identified data on the levels in Canadian sediment are from the late 1970s to early 1980s. Durham and Oliver (1983) examined the vertical distribution of 1,4-dichlorobenzene in bottom sediments in Lake Ontario near the mouth of the Niagara River in 1981, and reported that the maximum concentration of 1,4-dichlorobenzene was 1 100 ng/g (dry weight) at a depth from 8 to 9 cm. Concentrations of total dichlorobenzene isomers have also been measured in sediments collected during 1984 and 1985 from the St. Clair River near petrochemical plants near Sarnia (Oliver and Pugsley, 1986). The average concentration was 5 500 ng/g, while the maximum concentration was 34 000 ng/g (43 samples were analyzed). Based on data for the ratio of isomers (Oliver, 1992, personal communication), the corresponding average concentration of 1,4-dichlorobenzene in these sediments was approximately 524 ng/g.

No data on the concentrations of 1,4-dichlorobenzene in wild mammals or birds, and few data on levels in other environmental biota in Canada were identified. The level of 1,4-dichlorobenzene in tissue from mussels (*Modiolus sp.*), collected at the outfall of a municipal sewage effluent at Clover Point near Victoria, British Columbia, was below the detection limit (5 μg/kg) [EVS Consultants, 1992]. Levels of 1,4-dichlorobenzene in lake trout (*Salvelinus namaycush*), collected from Lake Ontario in 1980 and 1981, ranged up to 4 ng/g (wet weight) [Oliver and Nicol, 1982; Fox *et al.*, 1983]. Concentrations in this species were lower elsewhere in the Great Lakes (Oliver and Nicol, 1982). Concentrations of 1,4-dichlorobenzene in amphipods and oligochaetes collected in Lake Ontario in 1981 were reported to be 370 and 630 ng/g (dry weight), respectively (Fox *et al.*, 1983). MacLaren Marex Ltd. (1979) reported concentrations

up to 590 ng/g (dry weight) for total 1,3- and 1,4-dichlorobenzenes in clams (blue mussels) taken from estuarine sediment contaminated by industrial discharge from a chemical manufacturing plant in the East River of Pictou Harbour at Pictou, Nova Scotia.

Information on concentrations of 1,4-dichlorobenzene in Canadian food supplies is limited. In a limited study of fresh food composites from Ontario, 1,4-dichlorobenzene was detected only in the milk (2% fat content) composite at a concentration of 0.00055 μg/g (Davies, 1988). Other composites analyzed included the following: leafy vegetables; fruits; root vegetables, including potatoes; and eggs and meat. The composites were prepared from samples obtained in 4 retail grocery stores; the detection limit for all composite samples was 0.0001 μg/g.

1,4-Dichlorobenzene has been detected in breast milk. The mean concentration of the 1,3- and 1,4-isomers combined (calculated as 1,3-dichlorobenzene) was 6.1 ng/g with a maximum of 75 ng/g in breast milk of Canadian women taken 3 to 4 weeks after parturition (detected in 86% of the 210 samples, though the limit of detection was not specified) [Mes *et al.*, 1986].

## 2.4 Effects-related Information

### *2.4.1 Experimental Animals and* In Vitro

Following oral administration of 1,4-dichlorobenzene, Hollingsworth *et al.* (1956) reported that 2 800 mg/kg bw and 4 000 mg/kg bw were lethal to 100% of guinea pigs and rats, respectively. There were no deaths in guinea pigs and rats exposed to 1 600 mg/kg bw and 1 000 mg/kg bw, respectively.

The only sub-chronic study in which 1,4-dichlorobenzene has been administered by inhalation is that conducted in rats, guinea pigs, rabbits and monkeys by Hollingsworth *et al.* (1956). Following exposure to 950 mg/m$^3$ 7 hours per day, 5 days per week in this study, there was a decrease in body weight gain (> 10%) in guinea pigs and in rats, an increase in liver and kidney weights, and cloudy swelling and granular degeneration in the liver of rats. The no-observed-effect-level (NOEL) for rats and guinea pigs was 580 mg/m$^3$; for monkeys, mice and rabbits, it was 950 mg/m$^3$.

Increased incidence and severity of kidney cortical tubular degeneration in male Fischer 344/N rats (600 mg/kg bw/day) and mild to moderate centrilobular hepatocytomegaly (900 mg/kg bw/day) and minimal to mild hepatocytomegaly (675 mg/kg bw/day) in B6C3F$_1$ mice have been observed following oral administration of 1,4-dichlorobenzene for 13 weeks (NTP, 1987). The NOELs in these studies were 337.5 mg/kg bw in mice and 300 to 600 mg/kg bw in male and female rats, respectively (NTP, 1987).

The chronic toxicity and carcinogenicity of 1,4-dichlorobenzene have been examined in rats and mice following both inhalation (Loeser and Litchfield, 1983) and ingestion (NTP, 1987). Loeser and Litchfield (1983) reported increases in liver and kidney weights, urinary protein, and coproporphyrin in the high dose group of rats administered 0, 450 or 3 000 mg/$m^3$ 1,4-dichlorobenzene 5 hrs per day, 5 days per week for 76 weeks followed by 36 weeks without exposure. There was no evidence of toxicity in mice following exposure to the same concentrations for a shorter period, 57 weeks followed by 19 weeks without exposure. There were no treatment-related effects on the incidence of tumours observed in either species, though the relatively short exposure period (76 weeks in rats, 57 weeks in mice) and the high early mortality in mice may have decreased the sensitivity of this bioassay for carcinogenicity. The NOELs in rats and mice were 450 and 3000 mg/$m^3$, respectively.

In the U.S. NTP (1987) study, female F344 rats and male and female B6C3$F_1$ mice ingested 0, 300 or 600 mg/kg bw/day while male rats received 0, 150 or 300 mg/kg bw/day by gavage in corn oil 5 days per week for 103 weeks. In both sexes of mice, there was an increased incidence of non-neoplastic liver lesions, including cytomegaly and karyomegaly, hepatocellular degeneration, and individual cell necrosis at both doses. There was also an increased incidence of nephropathy in male mice and renal tubular regeneration in female mice at both doses. Positive trends in the incidence of hepatocellular adenomas, hepatocellular carcinomas, and hepatocellular adenomas and carcinomas (combined), were observed in both sexes of mice exposed to 1,4-dichlorobenzene with the incidences in the high dose groups all being significantly greater than those in vehicle controls. There was also a significantly increased incidence of hepatocellular adenomas and adenomas or carcinomas (combined) in low dose male mice. Rare tumours called hepatoblastomas were also observed in 4 male mice in the high dose group, each of which also had a hepatocellular carcinoma. Marginal increases in the incidences of pheochromocytomas (all types) of the adrenal gland in male mice (statistically significant only in the high dose group), and in the incidence of follicular cell adenomas of the thyroid gland in female mice were observed.

In male rats exposed to 1,4-dichlorobenzene in the NTP study, increased incidence of the following were observed: nephropathy; epithelial hyperplasia of the renal pelvis; mineralization of the collecting tubules in the renal medulla; and focal hyperplasia of the renal tubular epithelium. The incidence of nephropathy in low and high dose females was also increased in comparison with vehicle controls. Neoplastic lesions in rats in the U.S. NTP bioassay included a dose-related increase in the incidence of tubular cell adenocarcinomas in the kidney of male rats. One high dose male also had a tubular cell adenoma. These tumours were not observed in exposed or vehicle control female rats. A marginal increase in the incidence of mononuclear cell leukaemia in exposed male rats was observed when compared to controls.

On the basis of these results, it was concluded that there was clear evidence of carcinogenicity in male F344/N rats and both male and female $B6C3F_1$ mice. There was, however, no evidence of carcinogenicity in female F344/N rats (NTP, 1987).

The induction by 1,4-dichlorobenzene of kidney tumours in the male rat is believed to be associated with hyaline droplet formation resulting in a characteristic alpha-2-microglobulin nephropathy (hyaline droplet nephropathy). The mechanism of induction of such tumours in the male rat by several chemicals including 1,4-dichlorobenzene, is well established (Short *et al.*, 1987; Goldsworthy *et al.*, 1988; Murty *et al.*, 1988; Charbonneau *et al.*, 1989). It involves increased formation of protein droplets in kidney tubules associated with reabsorption of low molecular weight proteins (the P-2 segment). The reabsorbed protein is taken up by lysozymes and accumulates, due to a defect in protein metabolism by the renal tissue. The hyaline droplets characteristic of this effect have been shown to be composed of lysosomes overloaded with alpha-2-microglobulin. Excessive lysosomal activity is associated with increased cell proliferation leading ultimately, in a few cases, to renal tubular adenocarcinomas in the male rat. The protein involved, alpha-2-microglobulin, occurs in large amounts in the male Fischer 344 rat, but amounts are not significant in the female rat, mice or humans (Olson *et al.*, 1990).

For 1,4-dichlorobenzene, there is much evidence to support the involvement of hyaline droplet accumulation in the induction of the tubular cell adenocarcinomas in the male rat. Bomhard *et al.* (1988) demonstrated hyaline droplet accumulation in male rats following exposure to 1,4-dichlorobenzene with no indication of any such effects in female rats. Charbonneau *et al.* (1989) reported that 1,4-dichlorobenzene (single exposure in F344 rats to 300 or 500 mg/kg bw in corn oil) increased protein droplet formation and cell proliferation in male but not female rat kidneys, whereas equimolar doses of 1,2-dichlorobenzene had no effect. The maximum amount of radioactivity reversibly bound to alpha-2-microglobulin by 1,4-[$^{14}$C]dichlorobenzene was also 2 times that of an equimolar dose of 1,2-[$^{14}$C]dichlorobenzene.

The weight of evidence indicates that 1,4-dichlorobenzene is not genotoxic in either *in vivo* or *in vitro* assays (see Supporting Documentation). Negative results were obtained in an assay of DNA damage (unscheduled DNA synthesis) in the liver of mice following oral exposure to up to 1 000 mg/kg bw 1,4-dichlorobenzene (Steinmetz and Spanggord, 1987), though there was a small statistically significant increase in S-phase DNA replication (indicative of proliferation) seen in males only.

Available studies concerning the embryotoxicity, foetotoxicity, and teratogenicity of 1,4-dichlorobenzene include two studies following inhalation in rabbits (Hayes *et al.*, 1985) and rats (Loeser and Litchfield, 1983) and one study following ingestion in rats (Giavini *et al.*, 1986). No investigations on the reproductive effects of 1,4-dichlorobenzene were identified. In several of these studies, relatively minor embryotoxic and foetotoxic effects have been observed, but only at doses of 1,4-dichlorobenzene which were toxic to the mother.

### *2.4.2 Humans*

Available data on the effects of exposure to 1,4-dichlorobenzene in humans are restricted to one epidemiological study and to case reports concerning mainly accidental exposure to or misuse of moth crystals and space deodorants. No clinical investigations on the effects of exposure in human volunteers were identified. In the only identified cross-sectional epidemiological study of workers exposed to 1,4-dichlorobenzene, there was no evidence of "organic injury or untoward haematological effects" reported during periodic medical examinations in 58 workers exposed to various concentrations of 1,4-dichlorobenzene, although levels between 50 and 80 ppm were irritating to the eyes and nose; irritation was severe at 160 ppm (Hollingsworth *et al.*, 1956). However, little information on study design was presented in the published account of this investigation.

### *2.4.3 Ecotoxicology*

The information that was identified on the toxicity of 1,4-dichlorobenzene to aquatic biota includes data on acute and chronic effects in bacteria, algae, invertebrates, and fish. Studies that are cited below were conducted under closed, static or continuous flow conditions and were considered reliable for a volatile compound such as 1,4-dichlorobenzene. No suitable data on toxicity to sediment-dwelling biota or terrestrial biota including wild mammals, birds, and vascular plants were identified. Similarly, no empirical data regarding adverse effects of 1,4-dichlorobenzene on wildlife due to decreased availability or quality of prey were identified.

The lowest $LC_{50}$ (48 hour) reported for acute toxicity was 1.18 mg/L for rainbow trout (*Oncorhynchus mykiss*) [Calamari *et al.*, 1983]. For the water flea (*Daphnia magna*), Canton *et al.* (1985) reported a 48-h $LC_{50}$ of 2.2 mg/L, while Calamari *et al.* (1983) reported a 24-h $IC_{50}$ (for immobilization) of 1.6 mg/L. Inhibition of growth (96-h $EC_{50}$) and photosynthesis (3-h $EC_{50}$) in the freshwater algae (*Selenastrum capricornutum*) were reported at concentrations of 1.6 mg/L and 5.2 mg/L 1,4-dichlorobenzene, respectively (Calamari *et al.*, 1982, 1983).

Impairment of reproduction was the most sensitive endpoint identified, related to the toxicity of 1,4-dichlorobenzene to aquatic organisms. Calamari *et al.* (1982) reported a 28-d no-observed-effect-concentration (NOEC) and lowest-observed-effect-concentration (LOEC) [for effects on reproduction] of 0.22 and 0.40 mg/L, respectively, for *Daphnia magna*. In a similar study, the authors reported a 14-d $EC_{50}$ (for effects on reproduction) of 0.93 mg/L (Calamari *et al.*, 1983). For fish, Carlson and Kosian (1987) reported a NOEC of 0.57 mg/L and a LOEC (for effects on growth) of 1.0 mg/L, for the fathead minnow (*Pimephales promelas*), continuously exposed to 1,4-dichlorobenzene for 32 days during the embryo and early larval stages.

# 3.0 Assessment of "Toxic" under CEPA

## 3.1 CEPA 11(*a*): Environment

1,4-Dichlorobenzene is produced in and imported into Canada and is released into the Canadian environment. Few data were identified concerning the amounts entering the environment. Due to its volatility and the dispersive nature of its uses, most of the 3 500 tonnes of 1,4-dichlorobenzene used in Canada is estimated to be released into the environment. 1,4-Dichlorobenzene has been measured in municipal and industrial effluents, air, ground and surface waters, sediment, and biota in Canada. While 1,4-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 1,4-dichlorobenzene is known to occur in sediments in Lake Ontario, the St. Clair River, and Macaulay Point, Victoria, British Columbia. However, no toxicological data were identified that would enable a conclusion to be reached on the possible biological effects resulting from this exposure.

*Daphnia magna* was identified as the most sensitive aquatic organism to 1,4-dichlorobenzene. The 28-day LOEC for reduced fertility was 400 μg/L. Dividing this value by a factor of 10 (to account for inter-species differences in sensitivity as well as extrapolation from laboratory to field conditions) results in an estimated effects threshold of 40 μg/L. The highest concentration of 1,4-dichlorobenzene in Canadian surface waters (0.130 μg/L, measured in the early 1980s in the Niagara River) is approximately 300 times less than the estimated effects threshold. Therefore, no adverse effects are expected to result from exposure of pelagic organisms to 1,4-dichlorobenzene in Canadian surface waters.

A worst-case exposure scenario was developed for a representative fish-eating mammal, mink (*Mustela vison*) in southern Ontario to ascertain the most significant route of exposure. Mink are opportunistic carnivores with aquatic organisms comprising up to 100% of their diet. The total daily intake of 1,4-dichlorobenzene estimated for mink (Table 1) was 2 096 ng/kg bw, with inhalation being the major route of exposure.

**Table 1**
**Estimated Total Daily Intake of a Piscivorous Mammal Exposed to 1,4-Dichlorobenzene under "Worst-case" Conditions**

| Exposure Route | Environmental Levels[a] | Daily Rate of Consumption (per kg bw)[b] | Daily Intake (ng/kg bw) |
|---|---|---|---|
| air | 3.53 μg/m$^3$ | 0.55 m$^3$/d | 1 941 |
| surface water | 1.6 ng/L | 0.1 L/d | 0.16 |
| biota (fish) | 1.0 ng/g | 155 g/d | 155 |
| Total | – | – | 2 096 |

a. The level in air is the maximum level measured in a rural environment (Walpole Island, Ontario); the level in surface water is the mean level measured in Lake Ontario, and comparable to that in the St. Clair River in 1985 and Niagara-on-the-Lake in 1988; the level in biota is that measured in predatory fish in Lake Ontario in the early 1980s and is comparable to the maximum level predicted based on a BCF of 720.

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.

In the absence of studies on wildlife, the effects threshold for mink was estimated on the basis of results of a chronic inhalation study in rats. In this study, no treatment-related effects were observed at a concentration of 450 mg/m$^3$ (Loeser and Litchfield, 1983). Using a factor of 10 to account for variability in extrapolating from a laboratory to a field situation, the effects threshold was estimated to be 45 mg/m$^3$. The highest concentration of 1,4-dichlorobenzene reported for ambient rural air in Canada is approximately 12 700 times less than this effects threshold. Data on the toxicological effects of 1,4-dichlorobenzene on birds and terrestrial plants were not identified.

**Although, on the basis of available data, the levels of 1,4-dichlorobenzene present in air and surface water are not expected to cause adverse effects in aquatic biota or wildlife, there were no data upon which to evaluate the significance of concentrations in sediment to benthic biota. Therefore, the available information is insufficient to conclude whether 1,4-dichlorobenzene is entering the environment in quantities or under conditions that may be harmful to the environment.**

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

Although 1,4-dichlorobenzene is volatile at tropospheric temperatures and absorbs infrared radiation in wavelengths ranging from 7 to 13 μm, this substance is removed from the atmosphere by photo-oxidation (mean half-life of approximately 3 weeks) resulting in low, steady-state concentrations in the atmosphere (< 1 $\mu g/m^3$). As such, 1,4-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, it has been concluded that 1,4-dichlorobenzene is not entering the environment in quantities or under conditions that may constitute a danger to the environment on which human life depends.**

## 3.3 CEPA 11(*c*): Human Life or Health

***Population Exposure***

On the basis of the available data, it is likely that the general population is exposed to 1,4-dichlorobenzene principally in air, particularly indoor air (Table 2). However, it should be noted that reliable data on concentrations of 1,4-dichlorobenzene in indoor air in Canada were not identified and, as a result, it was necessary to calculate intake from this source on the basis of data collected in the United States. For suckling infants, breast milk is a more important source (estimated daily intake from this source, 0.7 μg/kg bw) than food for other age groups (estimated daily intake from this source, 0.0005 to 0.008 μg/kg bw) though it appears still to contribute less to total intake for this age group than indoor air (estimated daily intake for infants aged 0 to 6 months from indoor air, 0.1 to 1.3 μg/kg bw). The total daily intake of 1,4-dichlorobenzene for various age groups in the Canadian population is estimated to range from 0.1 to 2.1 μg/kg bw.

**Table 2**
**Estimated Daily Intake of 1,4-Dichlorobenzene from Various Sources by Canadians**

| | 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 yr[e] |
| Ambient Air[f] | 0.01–0.1 | 0.01–0.2 | 0.02–0.2 | 0.01–0.2 | 0.01–0.2 |
| Indoor Air[g] | 0.1–1.3 | 0.1–1.7 | 0.1–1.9 | 0.1–1.5 | 0.1–1.5 |
| Drinking Water[h] | – | 0.0008–<br>< 0.06 | 0.0004–<br>< 0.03 | 0.0003–<br>< 0.02 | 0.0003–<br>< 0.02 |
| Food[i] | 0.7[j] | 0.008 | 0.004 | 0.002 | 0.0005 |
| Total Intake | 0.8–2.1 | 0.1–2.0 | 0.1–2.1 | 0.1–1.7 | 0.1–1.7 |

mo = months

a. Assumed to weigh 7 kg, breathe 2 $m^3$ of air per day, and drink 750 ml of breast milk (as food) per day (Environmental Health Directorate, 1991).
b. Assumed to weigh 13 kg, breathe 5 $m^3$ of air per day, drink 0.8 L of water per day, and consume 194.5 g per day of 2% cow's milk (Environmental Health Directorate, 1991).
c. Assumed to weigh 27 kg, breathe 12 $m^3$ of air per day, drink 0.9 L of water per day, and consume 185.61 g per day of 2% cow's milk (Environmental Health Directorate, 1991).
d. Assumed to weigh 57 kg, breathe 19 $m^3$ of air per day, drink 1.3 L of water per day, and consume 196.2 g per day of 2% cow's milk (Environmental Health Directorate, 1991).
e. Assumed to weigh 69 kg, breathe 23 $m^3$ of air per day, drink 1.5 L of water per day, and consume 60.64 g per day of 2% cow's milk (Environmental Health Directorate, 1991).
f. Based on range of mean concentrations of 1,4-dichlorobenzene reported in a survey of ambient air from 23 sites across Canada (0.22 to 2.94 $\mu g/m^3$) [Environment Canada, 1991], assuming 4 of 24 hours are spent outdoors daily (Environmental Health Directorate, 1991).
g. Based on maximum ratio of median overnight concentrations in indoor versus outdoor air in 57 homes in Los Angeles and Contra Costa (1.8) [Wallace *et al.*, 1988] times mean concentrations reported in a survey of ambient air from 23 sites across Canada (0.22 to 2.94 $\mu g/m^3$), assuming 20 of 24 hours are spent indoors daily (Environmental Health Directorate, 1991).
h. Based on a range of mean concentrations of 1,4-dichlorobenzene in Canadian drinking water of 0.013 μg/L (Oliver and Nicol, 1982) to < 1 μg/L (Otson *et al.*, 1982a, 1982b).
i. Based on a concentration of 0.00055 μg/g 1,4-dichlorobenzene detected in a 2% cow's milk composite from Ontario (Davies, 1988).
j. Based on a mean concentration of 1,3-dichlorobenzene and 1,4-dichlorobenzene (combined) detected in breast milk (6.1 ng/g) from the Canadian National Survey (Mes *et al.*, 1986) and assuming the density of breast milk is equal to 1.0.

1. Data were insufficient to estimate intake from soil.

***Effects***

Available data are inadequate to assess the carcinogenicity of 1,4-dichlorobenzene in humans. An increased incidence of renal tubular cell adenocarcinomas in male rats and hepatocellular adenomas and carcinomas in male and female mice has been observed in an NTP carcinogenesis bioassay (NTP, 1987); only marginal increases of other tumours, restricted to single sexes, were observed. On the basis of these results, it was concluded that there was clear evidence of carcinogenicity in male F344/N rats and both male and female $B6C3F_1$ mice.

In general, a compound for which there is adequate evidence of carcinogenicity in two species would be classified in Group II (probably carcinogenic to humans) of the classification scheme developed for use in the derivation of the *Guidelines for Canadian Drinking Water Quality* (Environmental Health Directorate, 1989). However, available data indicate that the observed increase in renal tumours in F344 rats caused by 1,4-dichlorobenzene is a species and sex specific response which is unlikely to be relevant to humans exposed to this substance in the general environment (see Section 2.4.1). The weight of the available data indicate that 1,4-dichlorobenzene is not genotoxic in either *in vitro* or *in vivo* assays. Based on these results and the additional observation of an increase in DNA replication but no evidence of DNA damage in the liver of $B6C3F_1$ mice following exposure to 1,4-dichlorobenzene (Steinmetz and Spanggord, 1987), this substance may act as a non-genotoxic, hepatic carcinogen in this species. Therefore, 1,4-dichlorobenzene has been classified in Group III (possibly carcinogenic to humans) 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 III, 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) in a study conducted by the most relevant route of exposure, divided by an uncertainty factor, which when considered appropriate, takes into account the limited evidence of carcinogenicity.

The TDI has been derived, on the basis of the results of studies conducted by the route of exposure which is most important for the general population (i.e., inhalation). This value is also more conservative than a TDI which would be derived on the basis of results of studies in which 1,4-dichlorobenzene was administered orally.

The TDI has been derived on the basis of the results of the longest-term inhalation study, as follows:

$$\text{TDI} = \frac{450\ \text{mg/m}^3 \times (5/24) \times (5/7) \times 0.144}{500 \times 0.25}$$

$$= 0.078\ \text{mg/kg bw/day}\ (78\ \mu\text{g/kg bw/day})$$

where:

- 450 mg/m$^3$ is the NOEL based on increased liver and kidney weights, urinary protein, and coproporphyrin observed at higher concentrations in rats in the longest-term inhalation study (Loeser and Litchfield, 1983);
- 5/24 and 5/7 is the conversion of 5 hours per day, 5 days per week of administration to continuous exposure;
- 0.144 m$^3$ is the assumed inhaled air volume of rats (NIOSH, 1985);
- 0.25 kg is the assumed body weight of adult rats (NIOSH, 1985);
- 500 is the uncertainty factor (× 10 for inter-species variation; × 10 for intra-species variation; × 5 for evidence of carcinogenicity, though not observed in this study).

The total daily intake of 1,4-dichlorobenzene for various age groups in the Canadian population is estimated to range from 0.1 to 2.1 μg/kg bw. These estimated average daily intakes are less (by 37 to 780 times) than the TDI derived above.

**On the basis of available data, it has been concluded that 1,4-dichlorobenzene is not entering the environment in quantities or under conditions that may constitute a danger in Canada to human life or health.**

## 3.4 Conclusion

**Based upon available data, it has been determined that there is insufficient information to conclude whether 1,4-dichlorobenzene is entering the environment in quantities or under conditions that may be harmful to the environment. It has, however, been concluded that 1,4-dichlorobenzene is not entering the environment in quantities or under conditions that may constitute a danger to the environment on which human life depends, or to human life or health.**

# 4.0 Recommendations

(i) Since 1,4-dichlorobenzene is manufactured in Canada and no data concerning emissions to the atmosphere from point sources were identified, it is recommended that additional data on quantities released to the atmosphere and resulting atmospheric concentrations be acquired.

(ii) Reliable data on concentrations of 1,4-dichlorobenzene in indoor air of homes and buildings in Canada have not been identified. Consequently, it is recommended that such data be acquired.

(iii) Since virtually all available data concerning concentrations of 1,4-dichlorobenzene in sediment are outdated, it is recommended that concentrations of this substance in sediment near effluent outfalls from municipal waste treatment plants, textile plants and chemical manufacturing plants be determined.

(iv) It is recommended that the toxicity of 1,4-dichlorobenzene in benthic organisms present in the Canadian environment be determined.

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