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INTERIM CHEMICAL REVIEW COMMITTEE  
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Item 6 of the provisional agenda\*

CONSIDERATION OF DRAFT DECISION GUIDANCE DOCUMENTS REFERRED TO THE INTERIM  
CHEMICAL REVIEW COMMITTEE BY THE INTERGOVERNMENTAL NEGOTIATING COMMITTEE  
FOR THE FOLLOWING FOUR CHEMICALS: ETHYLENE DICHLORIDE, ETHYLENE OXIDE,  
MALEIC HYDRAZIDE AND BROMACIL

Note by the secretariat

Addendum

Annexed to the present addendum is the draft decision guidance document  
for the following chemical:

Chemical	CAS number	Category
Ethylene dichloride	107-06-2	Pesticide

\* UNEP/FAO/PIC/ICRC.1/1.

PIC - Decision guidance document for a banned or severely restricted chemical
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# Ethylene dichloride

Published:

<b>Common name</b>	Ethylene dichloride (ISO)
<b>Other names/ Synonyms</b>	1,2-Dichloroethane (IUPAC, CA); alpha,beta-dichloroethane; 1,2-bichloroethane; ethane dichloride; ethylene chloride; 1,2-ethylene dichloride; sym-(metric)-dichlorethane.
<b>CAS No.</b>	107-06-2
<b>Use category</b>	Pesticide
<b>Use</b>	<p>Ethylene dichloride is used both as a pesticide and as an industrial chemical.</p> <p>Pesticide uses: A small fraction of the total production (approximately 0.1% in the USA in 1977) was used for pesticide solvent and as an insecticidal fumigant used mainly in stored products. When used as a fumigant, ethylene dichloride is usually mixed with carbon tetrachloride to reduce the fire hazard, and small portions of other fumigants may be added (<i>WHO, 1987</i>).</p> <p>Industrial uses: The major industrial use of the compound is in the synthesis of vinyl chloride (approximately 90% of the total production in Japan and approximately 85% of total production in the USA). Other chemicals produced from ethylene dichloride are 1,1,1-trichloroethane, ethyleneamines, vinylidene chloride, trichloroethylene and tetrachloroethylene. In 1977, 2 - 4% of the total production of ethylene dichloride in the USA was used for the synthesis of each of these chemicals. Another 2% was used in the USA as a lead scavenger in gasoline. This application will decline in importance with the world-wide conversion to unleaded fuel (<i>WHO, 1987</i>).</p>
<b>Trade names</b>	Borer-Sol, Brocide, Destruxol Dichlor-emulsion, Dichlor-mulsion, Dutch Liquid, Dutch Oil, ENT 1656, Gaze Olefiant.
<b>Formulation types</b>	Liquid.
<b>Basic manufacturers</b>	Dow Chemicals USA; Vulcan Materials Company, USA.

Reasons for inclusion in the PIC procedure
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*Ethylene dichloride is included in the PIC procedure as a pesticide. Inclusion was recommended at the eighth meeting of the FAO/UNEP Joint Group of Experts on Prior Informed Consent following detailed discussions during the sixth and seventh meetings. It is included in the procedure on the basis of the control actions reported by a number of Governments.*

## Summary of control actions (see Annex 2 for details)

Control actions were reported by 6 countries and the European Union. In all 6 countries (Austria, Belize, Canada, Slovenia, Thailand and the United Kingdom) and in the European Union ethylene dichloride was reported as banned. No remaining uses were reported. Countries listed concerns about the carcinogenic properties of ethylene dichloride on human health as a primary reason for the control actions.

## Hazard classification by organization

<b>WHO</b>	Not classified under the WHO recommended classification of pesticides by hazard.
<b>EPA</b>	Group B2 (probable human carcinogen).
<b>EU</b>	F; R11 carc. Cat. 2; R45 Xn; R 22 Xi; R 36/37/38 (classification in accordance with Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances).
<b>IARC</b>	Group 2B (possibly carcinogenic to humans).

## Protective measures that have been applied concerning the chemical

### Measures to reduce exposure

For the health and welfare of workers and the general public, the handling and application of the substance should be entrusted only to competently supervised and well-trained applicators who must follow adequate safety measures and use the chemical according to good application practices. Regularly exposed workers should receive appropriate monitoring and health evaluations. Protective clothing as indicated in the *FAO Guidelines for Personal Protection when Working with Pesticides in Tropical Climates* (FAO, 1990) is required.

### Packaging and labelling

Follow the *FAO Revised Guidelines on Good Labelling Practice for Pesticides* and the *Guidelines for the Packaging and Storage of Pesticides* (FAO, 1985). Unbreakable packaging required; put breakable packaging into closed unbreakable container. Do not transport with food and feed stuff.

The United Nations Committee of Experts on the Transportation of Dangerous Goods classifies the chemical in:

**Hazard class:** 3

**Packing group:** II

### Alternatives

No alternatives were reported by notifying countries.

*It is essential that before a country considers substituting any of the reported alternatives, it ensures that the use is relevant to their national needs.*

## Waste Disposal

Waste should be disposed of in accordance with the provisions of the Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and Their Disposal and any guidelines thereunder (SBC, 1994).

See the *FAO Guidelines on Prevention of Accumulation of Obsolete Pesticide Stocks* and *The Pesticide Storage and Stock Control Manual* (FAO,1996).

Wear protective clothing and respiratory equipment suitable for toxic materials. Sweep, scoop or pick up spilled material. Vacuuming or wet sweeping may be used to avoid dust dispersal. Do not flush to surface water or sanitary sewer system. Dispose of empty containers in a sanitary landfill or by incineration.

Precautions for "carcinogens": There is no universal method for disposal that has been proved satisfactory for all carcinogenic compounds. Ethylene dichloride is a candidate for liquid injection incineration, with a temperature of 650 to 1600 °C and a residence time of 0.1 to 2 seconds. (USEPA, 1987).

*It should be noted that the methods recommended in the literature are often not suitable in a specific country. High temperature incinerators may not be available. Consideration should be given to the use of alternative destruction technologies.*

## Exposure limits

	Type of limit	Value
<b>Food</b>	MRL's (Maximum Residue Limits in mg/kg) in specified products (FAO/WHO, 1999).	No MRL allocated.
	JMPR ADI (Acceptable Daily Intake) in mg/kg diet (WHO, 1992).	No ADI allocated.
<b>Workplace</b>	USA (ACGIH) TLV-TWA (Threshold Limit Value, Time-Weighted Average in mg/m <sup>3</sup> ).	10 ppm; 40 mg/m <sup>3</sup> .

## First aid

Persons who have been poisoned (accidentally or otherwise) should be transported immediately to a hospital and put under surveillance of properly trained medical staff.

Eyes: Immediately flush eyes with plenty of water for at least 15 minutes, occasionally lifting the upper and lower lids. Seek medical attention immediately.

Skin: Flush skin with plenty of soap and water for at least 15 minutes before removing contaminated clothing and shoes.

Ingestion: Do not induce vomiting. Have the victim rinse his or her mouth and then drink 2-4 cupfuls of water, and seek medical advice.

Inhalation: Remove from exposure into fresh air immediately.

## Annexes

- Annex 1 **Further information on the substance**
- Annex 2 **Details on reported control actions**
- Annex 3 **List of designated national authorities**
- Annex 4 **References**

## Annex 1 - Further information on the substance

### 1 Chemical and physical properties

1.1	<b>Identity</b>	Clear colourless liquid; chloroform-like odour; sweet taste ( <i>Tomlin, 1994</i> ).
1.2	<b>Formula</b>	C <sub>2</sub> H <sub>4</sub> Cl <sub>2</sub>
	<b>Chemical name</b>	1,2-dichloroethane (CA).
1.3	<b>Solubility</b>	8.69 g/litre at 20 °C, miscible with alcohol, chloroform, ether ( <i>IARC, 1979</i> ).
	<b>logP<sub>ow</sub></b>	1.76
1.4	<b>Vapour pressure</b>	8.53 kPa (64 mmHg), 20 °C, highly volatile.
1.5	<b>Melting point</b>	-36 °C
1.6	<b>Reactivity</b>	This compound is incompatible with strong alkalis, strong caustics, oxidizing materials, active metals such as aluminium, magnesium, sodium or potassium. It reacts violently with nitrogen tetraoxide, dimethylaminopropylamine or liquid ammonia. A vigorous reaction also occurs when a mixture of this compound, propylene dichloride and o-dichlorobenzene comes into contact with aluminium. It can corrode iron, zinc and aluminium in the presence of moisture ( <i>Sax, 1986</i> ). Mixtures with HNO <sub>3</sub> easily deteriorate ( <i>Bretherick, 1986</i> ).

### 2 Toxicity

#### 2.1 General

2.1.1 **Mode of action** Although only limited quantitative data are available, inhaled ethylene dichloride is likely to be adsorbed by the lungs in humans and experimental animals, based on its high vapour pressure and serum/air partition coefficient (*WHO, 1994*).

2.1.2 **Uptake** Ethylene dichloride can be found in the blood of rodents, almost immediately after dermal, oral or inhalation exposure. Peak blood level in rat during dermal exposure for 24 hours is 135 mg/l (*Morton, 1991 in Richardson, 1993*).

2.1.3 **Metabolism** Ethylene dichloride is metabolised in rat and mouse by two competing pathways, both of which involve glutathione (GSH). Oxidation gives chloroacetaldehyde which is detoxified by GSH; it also reacts with GSH to form S-(2-chloroethyl)glutathione. These reactions are of the second order (*D'sruza, 1988 in Richardson, 1993*).

Following intraperitoneal injection of mouse, the alkyl purines 7-(2-oxoethyl)guanine and 7-[S-(2-cysteinyl)ethyl]guanine were found in DNA hydrolyzates and in the urine. Chloroacetaldehyde and S-(2-chloroethyl)glutathione were found in haemoglobin (*Svensson, 1986 in Richardson, 1993*).

Following intraperitoneal injection of 50-170 mg/kg <sup>14</sup>C-ethylene dichloride to mice, 10-42% was expired unchanged and 12-15% as carbon dioxide. Most of the remainder was excreted in the urine, primarily as chloroacetic acid (via chloroacetaldehyde), S-(carboxymethyl)cysteine and thiodiacetic acid (*Yllner, 1971 in Richardson, 1993*).

Little dechlorination of ethylene dichloride was found to occur in rat and rabbit

liver preparations in vitro (*Rannug, 1978 in Richardson, 1993*).

Metabolism of ethylene dichloride appears to have a significant role in the manifestation of the toxic, carcinogenic and mutagenic effects of this chemical.

## 2.2 Known effects on human health

### 2.2.1 Acute toxicity

Symptoms of poisoning Breathing ethylene dichloride can irritate the nose, throat and lungs causing coughing, shortness of breath and difficulty in breathing. Higher levels can cause a build-up of fluid in the lungs (pulmonary oedema). This can cause death. Exposure can cause nausea, vomiting, headaches, increasing drowsiness and then loss of consciousness. Over-exposure can also cause liver and kidney damage, and irritate the eyes. Contact can irritate the skin causing redness and a rash, and irritate the eyes (*USEPA, 1987*).

The lethal oral dose of ethylene dichloride in humans has been estimated to be between 20 and 50 ml (*WHO, 1994*).

### 2.2.2 Short and long term exposure

Cancer Hazard: Ethylene dichloride may be a carcinogen in humans since it has been shown to cause stomach, lung, breast and other types of cancer in animals.

Other long term effects: Ethylene dichloride can irritate the lungs. Repeated exposure may cause bronchitis to develop with cough, phlegm and/or shortness of breath. Repeated, prolonged contact can chronically irritate the skin causing dryness, redness and a rash. Repeated, prolonged exposure can cause loss of appetite, nausea and vomiting, trembling and low blood sugar (with weakness). It may damage the liver and kidneys (*USEPA, 1987*).

### 2.2.3 Epidemiological studies

Significant excess of deaths due to pancreatic cancer was found in a study of 278 men working in the chlorohydrin unit of a chemical production plant between 1941 and 1967 (*Benson & Teta 1993 in WHO, 1995*).

No significant difference was found compared with control in a case-control study on 21 employees at a petrochemical plant in USA (*WHO, 1994*).

In a cohort study of 6588 workers at the same plant no significant excess of malignant brain tumours was observed (*Austin & Schnatter, 1983 in WHO, 1995*).

No association between ethylene dichloride spill and leukemia in childhood was found in a small case-control study (*Deschamps & Band, 1993 in WHO, 1995*).

A statistically significant increase in colon and rectal cancer was observed in men aged  $\geq 55$  years and whose drinking water contained  $\geq 0.1$   $\mu\text{g/l}$  ethylene dichloride, even if the authors did not suggest an association between ethylene dichloride and cancer but underlined the higher rectal cancer incidence in populations consuming chlorinated water (*Isacson, 1985 in WHO, 1995*).

Higher prevalence of subjective symptoms was observed in 10 male workers in an oil refinery exposed to 250-800  $\text{mg/m}^3$  than in those exposed to lower concentrations. However there was a co-exposure to benzene (*Cetnarowicz, 1959 in WHO, 1995*).

An increased morbidity for all disease categories was observed in a 5-year period (1951-55) in a group of workers at an aircraft factory exposed for 25-

30% of the working time to 80-150 mg/m<sup>3</sup> and to ≤ 5 mg/m<sup>3</sup> for the remainder (Kozik, 1957 in WHO, 1995).

## 2.3 Toxicity studies with laboratory animals and *in vitro* systems

### 2.3.1 Acute toxicity

**oral** LD<sub>50</sub> for rats, mice, dogs and rabbits ranged from 413 to 2500 mg/kg bw (WHO, 1995).

**Dermal** LD<sub>50</sub> for rabbits ranged from 2800 to 4900 mg/kg bw (Torkelson & Rowe, 1981 in WHO, 1995).

**Inhalation** LC<sub>50</sub> for rats exposed for 6 or 7.25 hours ranged from 4000 mg/m<sup>3</sup> to 6600 mg/m<sup>3</sup> (WHO, 1995).

**Irritation** Application of ethylene dichloride to the skin of experimental animals has resulted in microscopic changes and moderate oedema (Duprat, 1976).

### 2.3.2 Short-term-exposure

Several short-term and subchronic studies in different experimental species indicate that liver and kidneys are the target organs. The documentation was considered inadequate to derivate NOELs or LOELs. Some studies show morphological changes in the liver in several species following subchronic exposure to airborne concentrations as low as 800 mg/m<sup>3</sup>. Liver weight increase was observed in rats with subchronic oral administration of 49 to 82 mg/kg bw. Changes in serum parameters that indicate liver and kidney toxicity were observed in rats exposed to airborne concentrations as low as 202 mg/m<sup>3</sup> for 12 months (WHO, 1995).

### 2.3.3 Long-term exposure

Studies on the chronic effects are related to the carcinogenicity of the substance and do not give sufficient information on non-neoplastic effects of the substance. Ethylene dichloride was carcinogenic in mice and rats when administered by gavage or dermal application, while no increase in the incidence of tumours was noted in inhalation or in initiation/promotion bioassays (WHO, 1994).

### 2.3.4 Effects on reproduction

There is no evidence from a limited number of studies that ethylene dichloride is teratogenic in experimental animals. There is also little convincing evidence that ethylene dichloride induces reproductive or developmental effects at doses below those which cause other systemic effects (WHO, 1995).

### 2.3.5 Mutagenicity

Ethylene dichloride has been consistently positive in *in vitro* mutagenic bioassays in *Salmonella typhimurium*. Response has been greater in the presence of an exogenous activation system (cytochrome system) than in its absence, and mutagenicity was more than doubled in *S. typhimurium* expressing the human GSTA-1 gene. In cultured mammalian cells, ethylene dichloride forms DNA adducts. It also induces unscheduled DNA synthesis in primary cultures of rodents and human cells and gene mutation in several cell lines. Mutation frequency in human cell lines has been correlated with differences in glutathione-S-transferase activity. In *in vivo* studies ethylene dichloride induced somatic cell and sex-linked recessive lethal mutations in

*Drosophila melanogaster* and the compound bound to DNA in all reported studies in rats and mice. Although primary DNA damage in liver and sister chromatid exchange has been observed in studies in mice, there has been no

evidence for micronucleus induction (*WHO, 1995*).

**2.3.6 Carcinogenicity** Carcinogenicity of ethylene dichloride was investigated in a few limited bioassays on experimental animals. Significant increases were not found for any type of tumour in Sprague-Dawley rats or Swiss mice exposed to up to 607 mg/m<sup>3</sup> for 78 weeks (a high mortality was observed in this study although it was not related to concentration). No significant increase in the incidence of mammary gland adenomas and fibroadenomas in Sprague-Dawley females exposed to 200 mg/m<sup>3</sup> for 2 years (*WHO, 1995*).

Significant increased incidence of tumours was observed in two species following ingestion; squamous cell carcinomas of the stomach in males, haemangiosarcomas in both sexes. Fibromas of the subcutaneous tissue in males, adenocarcinomas and fibroadenomas of the mammary gland in females were observed in Osborne-Mendel rats with Time-Weighted Average (TWA) daily doses of 45 to 95 mg/kg bw/day for 78 weeks. Similar increases in alveolar/bronchiolar adenomas in males and females, mammary gland adenocarcinomas in females and endometrial stromal polyp or endometrial stromal sarcoma combined in females and hepatocellular carcinomas in males were observed in B6C3F1 mice administered TWA of 97 or 195 mg/kg bw/day for males and 149 or 299 mg/kg bw/day for females by gavage for 78 weeks (*WHO, 1995*).

A significant increase of lung tumours (benign papillomas) was found in female mice following repeated ethylene dichloride application for 440 to 594 days. A dose-related increase in the incidence of pulmonary adenomas was found in mice following repeated intraperitoneal injection of ethylene dichloride but was not significant. Concomitant exposure to inhaled ethylene dichloride and disulfaram in the diet resulted in an increased incidence of intrahepatic bile duct cholangiomas and cysts, subcutaneous fibromas, hepatic neoplastic nodules, interstitial cell tumours in the testes and mammary adenocarcinomas in rats compared to rats administered either the compound alone or untreated controls. A further three bioassays did not show evident tumour development initiating or promoting properties (*WHO, 1995*).

### 3 Exposure

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**3.1 Food** Very little information is available on ethylene dichloride in food. Ethylene dichloride was found in Germany in milk products with added fruits. In Canada it was used as an extractant in samples of spice oleoresins. Residue studies show that ethylene dichloride can be found in fumigated grain (*WHO, 1987*).

**3.2 Occupational** Ethylene dichloride levels of up to 150 mg/m<sup>3</sup> and ranging from 40 to 800 mg/m<sup>3</sup> were detected in industrial plants using the chemical as a solvent (*WHO, 1987*).

Time-weighted averages of 0.1 and 1 mg/m<sup>3</sup>, respectively, have been reported for two different jobs in an anti-knock agent blending plant in the USA. The maximum exposure level measured was 8.9 mg/m<sup>3</sup> (*WHO, 1987*).

- 3.3 Environment** Owing to the limited releases of ethylene dichloride, it is a rare environmental contaminant. It has been detected in both surface and groundwaters, but unlike other volatile organic compounds (VOCs), higher levels were reported in surface waters. USEPA estimates that 0.3% of all groundwater supplies contain ethylene dichloride concentrations ranging from 0.5 to 5.0 g/l. Three percent of surface waters are estimated to have concentrations from 0.5 to 20 g/l (*Howard, 1990; USEPA, 1987*).
- Ethylene dichloride commonly occurs in the air of urban and suburban areas at concentrations less than 0.2 ppb. The greatest source of ethylene dichloride exposure is from the air. Drinking water is the greatest source for populations with drinking water levels above 6 g/l (*Howard, 1990; USEPA, 1987*).
- 3.4 Accidental poisoning** Acute incidental exposure to ethylene dichloride by inhalation or ingestion has resulted in a variety of effects in humans, including effects on the central nervous system, liver, kidney, lung and cardiovascular system.

## 4 Effects on the environment

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- 4.1 Fate** Ethylene dichloride released to the air slowly degrades over a period of a few months. Photo-oxidation with hydroxyl radicals, that results in the production of carbon dioxide and hydrochloric acid, is believed to be the predominant removal process. It is expected that ethylene dichloride is transported over long distances and washed out during rainfall. Direct photolysis is not expected to occur (*Howard, 1990; USEPA, 1997*).
- Ethylene dichloride released to surface waters will be removed primarily by evaporation within a few days or weeks. Adsorption to sediment and hydrolysis is not expected.
- Releases of ethylene dichloride on to soil will evaporate fairly rapidly. Rapid migration to groundwater is expected for sandy soils (*Howard, 1990; USEPA, 1997*).
- 4.1.1 Persistence** Biodegradation is not expected to occur under either aerobic or anaerobic conditions. The photo-oxidation of ethylene dichloride in air is expected to be a slow process. No significant bioaccumulation is expected to occur in aquatic organisms (*Howard, 1990; USEPA, 1997*).
- 4.1.2 Bioconcentration** Ethylene dichloride is not expected to bioconcentrate in fish due to its low Kow. The measured bioconcentration factor for bluegill sun fish is 0.30 (*Richardson, 1993*).
- 4.2 Ecotoxicity**
- 4.2.1 Fish** Acute toxicity studies have been conducted on several species of freshwater fish. The most sensitive species was two to three-month old guppies (*Poecilia reticulata*), with a nominal 7-day LC<sub>50</sub> of 106 mg/l ethylene dichloride under static renewal test conditions. In three studies in 30-day old fathead minnows (*Pimephales promelas*) over 96-hour LC<sub>50</sub>s ranged from 116 to 136 mg/l under flow-through conditions. The only adequate acute toxicity study in marine fish involved tidewater silversides (*Minidia beryllina*) in which a nominal 96-hour LC<sub>50</sub> of 480 mg/l was reported under static test conditions (*WHO, 1994*).

In a long-term flow-through study of the early life stages of fathead minnows (*Pimephales Promelas*) a NOEL of 29 mg/l and a LOEL of 59 mg/l (reduced larval growth) were identified (WHO, 1994). The EC<sub>50</sub> for hatchability and a 27-day LC<sub>50</sub> for post-hatch survival both of 34 mg/l, resulted from an ethylene dichloride flow-through assay on embryos and larvae of rainbow trout (*Onchorhynchus mykiss*) and the LOEL identified was 3.49 mg/l (24% reduction in egg hatchability) (WHO, 1994).

After 21 days of continuous exposure to 150 mg/l ethylene dichloride, mortality of coho salmon (*Onchorhynchus kisutch*) eggs was 46%, while in alevins, 100% mortality occurred 9 days after hatching at 320 mg/l (WHO, 1994).

Teratogenic effects were observed in rainbow trout (*Onchorhynchus mykiss*).

#### 4.2.2 Aquatic invertebrates

*Daphnia magna* appear to be the invertebrate species most sensitive to ethylene dichloride in chronic toxicity studies in freshwater. Under static conditions, the measured 48-hour LC<sub>50</sub>s for fed and unfed first instar *Daphnia* were 320 and 270 mg/l, respectively; the 48-hour LC<sub>50</sub> based on complete immobilization, were 180 and 160 mg/l for fed and unfed organisms, respectively (WHO, 1994).

In a 28-day flow-through study on *Daphnia magna* the LOEL and NOEL for reproductive success were respectively 20.7 and 10.6 mg/l, while the LOEL and NOEL for growth were 71.7 and 41.6 mg/l (WHO, 1994).

With regard to acute toxicity studies in marine invertebrates under static test conditions, the nominal 24-hour EC<sub>50</sub> for immobilization of 30-hour posthatch larvae of the brine shrimp, *Artemia salina*, was 93.6 mg/l (WHO, 1994). For marine adult shrimp, *Crangon crangon*, the measured 24-hour LC<sub>50</sub> was 170 mg/l, under static test conditions (WHO, 1994).

#### 4.2.3 Birds

Significant reduction of the egg weight at 250 mg/kg and reduction of both the number and weight of eggs at 500 mg/kg were observed in a study in which male and female leghorn chickens were fed mash which had been fumigated with ethylene dichloride (WHO, 1994).

#### 4.2.4 Bees

There are no adequate studies to permit an assessment of effects on bees.

#### 4.2.5 Other

##### Aquatic micro-organisms

The IC<sub>50</sub>s for *Nitrosomonas* and methanogens (29 and 25 mg/l, respectively) were considerably lower than for aerobic heterotrophs (470 mg/l). For the bacteria, *Pseudomonas putida*, the nominal 16-hour EC<sub>50</sub> for the onset of cell multiplication inhibition was 135 mg/l (WHO, 1994).

The freshwater blue-green algae, *Microcystis aeruginosa*, was seven times more sensitive to ethylene dichloride than green algae, *Scenedemus quadricauda*, with a nominal 7-day ED<sub>50</sub>s for inhibition of cell multiplication at 27 °C of 105 and 710 mg/l, respectively (WHO, 1994).

Based on bioluminescence, the 5-minute IC<sub>50</sub> was 700 mg/l in a Microtox test with *Photobacterium phosphoreum* (WHO, 1994).

##### Aquatic vertebrates

In a study in which embryos and larvae of the northwestern salamander (*Ambystoma gracile*) and the leopard frog (*Rana pipiens*) were continuously exposed to ethylene dichloride from 30 minutes of fertilization (embryos) and maintained through four days posthatching (larvae), the resulting LC<sub>50</sub>s for the salamander were 6.53 mg/l at the day of hatching (day 5) and 2.54 mg/l 4-day posthatching (day 9). LOEL was 0.99 mg/l for 23% reduction in egg

	<p>hatchability. The measured 5-day and 9-day LC<sub>50</sub>s for the frog were 4.52 and 4.40 mg/l respectively, while the 5-day posthatch LOEL was 1.07 mg/l (WHO, 1994).</p>
Terrestrial invertebrates	<p>In an acute contact test, a 48-hour LC<sub>50</sub> for earthworms (<i>Esinia fetida</i>) exposed to ethylene dichloride-treated filter paper was 60 µg/m<sup>2</sup> (WHO, 1994).</p>
Plants	<p>Ethylene dichloride vapour was both lethal and mutagenic to barley kernels (two-rowed variety, <i>Bonus</i>) following exposure to 3 mg/m<sup>3</sup> for 24 hours.</p>

## Annex 2 - Details on reported control actions

### AUSTRIA

<b>Effective:</b>	1992.
<b>Control action:</b>	All uses banned.
<b>Reasons:</b>	Carcinogenic and mutagenic properties. The substance has a potential for reproductive effects in males and central nervous system effects.
<b>Alternatives:</b>	Many alternatives for designated purposes.

### BELIZE

<b>Effective:</b>	1985.
<b>Control action:</b>	The substance is banned for use.
<b>Reasons:</b>	Mixed with CC14, a carcinogen.

### CANADA

<b>Effective:</b>	1984.
<b>Control action:</b>	Suspended/banned. No remaining uses allowed.

### EUROPEAN UNION

<b>Effective:</b>	1989.
<b>Control action:</b>	The placing on the market and the use of plant protection products containing 1,2-dichloroethane is prohibited. No remaining uses allowed.
<b>Reasons:</b>	The use of 1,2-dichloroethane as a plant protection product, in particular to fumigate plants and soil, is likely to give rise to harmful effects on human and animal health as well as unreasonable adverse influence on the environment. 1,2-dichloroethane has been classified by the European Community as a category 2 carcinogen (probably carcinogenic to humans).

(Member States of the European Union are: Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Luxembourg, Netherlands, Portugal, Spain, Sweden, United Kingdom.)

### SLOVENIA

<b>Effective:</b>	1997.
<b>Control action:</b>	Banned for use in agriculture.
<b>Reasons:</b>	This chemical was banned from the use in agriculture due to the effect of its toxic properties to human health and the environment according to the opinion given by the Commission on Poisons.

**THAILAND**

**Effective:** 1995.  
**Control action:** All use categories have been banned.  
**Reasons:** Possibly carcinogenic in test animals.

**UNITED KINGDOM**

**Effective:** 1989.  
**Control action:** All agricultural uses revoked under the control of pesticides regulations.  
**Reasons:** Evidence of carcinogenicity.

## Annex 3 – List of Designated National Authorities

### AUSTRIA

#### CP

Department II/3  
 Ministry of the Environment  
 Stubenbastei 5  
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### BELIZE

#### P

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#### C

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**EUROPEAN UNION****CP**

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## Annex 4 - References

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