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# The Installation Restoration Program Toxicology Guide

## Volume 1

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*Aerospace Medical Division*
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# THE INSTALLATION RESTORATION PROGRAM TOXICOLOGY GUIDE

## Volume 1

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# PREFACE

One of the objectives of the U.S. Air Force Installation Restoration Program (IRP) is to provide individuals responsible for the management and implementation of the IRP with information to evaluate the health hazards associated with actual or potential contamination of drinking water supplies. The Harry G. Armstrong Aerospace Medical Research Laboratory was requested by HQ USAF/SGPA to develop health and environmental information for each potential contaminant of drinking water supplies associated with USAF installations. This IRP Toxicology Guide consists of four volumes which were initially issued in 1985-1987. The original Toxicology Guide was produced under contract F33615-81-D-0508 by Arthur D. Little, Inc. for the Biochemical Toxicology Branch, Toxic Hazards Division, Harry G. Armstrong Aerospace Medical Research Laboratory (AAMRL), Wright-Patterson AFB, OH. The updated volumes of the Toxicology Guide include new regulatory requirements and recently published toxicology information. The updated Toxicology Guide was produced under an Interagency Agreement with the U.S. Department of Energy, Oak Ridge National Laboratory (87-TH-0002) for the Hazard Assessment Branch, Toxic Hazards Division, AAMRL, Wright-Patterson AFB, OH.

For each chemical in the IRP Toxicology Guide, the environmental fate, exposure pathways, toxicity, sampling and analysis methods and state and federal regulatory status are outlined. The material provided is intended as an overview of key topic areas; no attempt was made to provide a comprehensive review. Users are encouraged to read the Introduction to Volume 1 of the IRP Toxicology Guide before applying chemical-specific information.

Candidate chemicals for inclusion in subsequent Toxicology Guide updates should be forwarded through MAJCOM bioenvironmental engineers to HQ USAF/SGPA. Consultant service for current toxicological information should be obtained from the USAF OEHL/ECO, Brooks AFB, TX 78235-5000.

Substantial effort was made to assure that the information contained in the Toxicology Guide was current and reliable at the time of publication. Users are encouraged to report apparent discrepancies or errors to AAMRL/THA, Wright-Patterson AFB, OH 45433-6573. Copies of this document are available from: National Technical Information Services, 5285 Port Royal Road, Springfield, VA 22161. Federal Government agencies and their contractors registered with Defense Technical Information Center should direct requests for copies to: Defense Technical Information Center, Cameron Station, Alexandria, VA 22314.

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# LIST OF ABBREVIATIONS, ACRONYMS, TERMS AND SYMBOLS

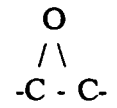
This list of abbreviations, acronyms, terms and symbols is selected from the pages of the Guide. Words and phrases defined here include those occurring in more than one chapter, those indispensable to understanding the material in a chapter and those that may help clarify some of the definitions themselves. Not listed are chemical synonyms which can be found in the chemical index and words adequately defined at the point of use.

| | |
|---|---|
| A | Acre |
| AA | Atomic absorption spectroscopy |
| ACGIH | American Conference of Governmental Industrial Hygienists |
| Active metals | This refers to metals such as aluminium, calcium, magnesium, potassium, sodium, tin, zinc, and their alloys. |
| ADI | Acceptable daily intake |
| ADL | Arthur D. Little, Inc. |
| Adenocarcinoma | A malignant tumor originating in glandular or ductal epithelium. |
| Adenoma | A benign growth of glandular tissue. |
| ae | Acid equivalent |
| Aerosol | A suspension or dispersion of small solid or liquid particles in air or gas. |
| AFOSH | Air Force Occupational Safety and Health Standard |
| Alkali metals | Metals (in Group 1A of the Periodic Table,) such as lithium, sodium, potassium, rubidium, cesium, and francium. The alkali metals react vigorously, at times violently, with water. These metals present a dangerous fire risk when in contact with moisture or oxidizing materials. |

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| Alkaline earth metals | Calcium, barium, strontium, and radium (Group IIA of Periodic Table). Alkaline earth metals are less reactive than sodium and potassium and have higher melting and boiling points. |
| Ambient water | Surface water |
| Ambient water criterion | That concentration of a pollutant in a navigable water that, based upon available data, will not result in adverse impact on important aquatic life, or on consumers of such aquatic life, after exposure of that aquatic life for periods of time exceeding 96 hours and continuing at least through one reproductive cycle; and will not result in a significant risk of adverse health effects in a large human population based on available information such as mammalian laboratory toxicity data, epidemiological studies of human occupational exposure data, or any other relevant data. |
| Amines | A class of organic compounds of nitrogen that may be considered as derived from ammonia ($NH_3$) by replacing one or more of the hydrogen atoms (H) with straight or branched hydrocarbon (alkyl) groups. All amines are basic in nature and usually combine readily with hydrochloric or other strong acids to form salts. |
| API | American Petroleum Institute |
| Aquifer | An underground, permeable saturated strata of rock, sand or gravel containing ground water. |
| Aromatic | A major group of hydrocarbons containing one or more rings like benzene, which has a six-carbon ring containing three double bonds. Most compounds in this group are derived from petroleum and coal tar and are reactive and chemically versatile. The name characterizes the strong and pleasant odor of most substances of this group. NOTE: The term "aromatic" is often used in perfume and fragrance industries to describe essential oils, which are not aromatic in the chemical sense. |
| atm | Atmosphere (760 Torr) |
| ATP | Adenosine triphosphate, a nucleotide cofactor important in many biological reactions where energy is transferred. |
| Autoignition temperature | The minimum temperature at which the material will ignite without a spark or flame being present. Along with the flash point, autoignition temperature gives an indication of relative flammability. |

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| BCF | Bioconcentration factor, a measure of the cumulative build-up of a specific compound sequentially through a food chain. |
| Benign | A term meaning noncancerous. |
| BOD | Biochemical oxygen demand |
| BUN | Blood urea nitrogen |
| bw | Body weight |
| C | Celsius (Centigrade) |
| CAA | Clean Air Act |
| CAG | Cancer Assessment Group of the U.S. Environmental Protection Agency |
| Calc | A number calculated by Arthur D. Little, Inc. |
| Carcinogen | Any cancer-producing substance. |
| Carcinoma | A malignant epithelial tumor. |
| CAS REG NO | Numeric designation assigned by the American Chemical Society's Chemical Abstract Service which uniquely identifies chemical compound. |
| cc | Cubic centimeter(s) |
| CERCLA | Comprehensive Environmental Response Compensation and Liability Act |
| CFR | Code of Federal Regulations |
| CL | Ceiling limit value |
| cm | Centimeter(s) (1E-02 meter) |
| Chemically active metals | This phrase generally refers to metals such as, calcium, magnesium, potassium, sodium, tin, zinc, and their alloys. |

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| CNS | Central nervous system which consists of the brain and spinal cord. The CNS controls mental activity plus voluntary muscular activity. It also coordinates the parasympathetic and sympathetic nervous systems, which command the body's involuntary functions. |
| CO | Carbon monoxide |
| $CO_2$ | Carbon dioxide |
| Cp | Centipoise |
| CPSA | Consumer Product Safety Act |
| C*t | Product of concentration multiplied by time of exposure |
| CWA | Clean Water Act |
| d | Density |
| da | Day(s) |
| ° | Degrees, as in 37°C |
| DNA | Deoxyribonucleic acid |
| DOT | U.S. Department of Transportation |
| Drinking Water | Water which meets the specifications of the water quality standards and is therefore suitable for human consumption and for all usual domestic purposes. |
| ECD | Electron capture detector |
| EEC | European Economic Community |
| EEG | Electroencephalogram, it detects abnormalities in the electrical waves emanating from different areas of the brain. |
| EKG | Electrocardiogram, a recording of the changes in electrical potential that occur during a cycle of heart muscle activity, producing a characteristic series of waves. |
| EPA | Environmental Protection Agency |
| Epithelium | The covering of internal and external surfaces of the body, including the lining of vessels and small cavities. |

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| Epoxide | An organic compound containing a reactive group resulting from the union of an oxygen atom with other atoms (usually carbon) that are joined as shown below:<br>  <br>This group, commonly called "epoxy", characterizes the epoxy resins. Epichlorohydrin and ethylene oxide are well-known epoxides. |
| estim | Estimated value |
| F | Fahrenheit |
| FDA | Food and Drug Administration (U.S.A.) |
| FDCA | Food, Drug and Cosmetic Act |
| FID | Flame ionization detector |
| FIFRA | Federal Insecticide, Fungicide and Rodenticide Act |
| Finished | Tap water, i.e., water that has undergone drinking water treatment |
| Flammable limits in air | The range of gas or vapor concentrations in air, generally expressed in units percent by volume, capable of supporting combustion when ignited. The lower end of the range is commonly referred to as the lower flammable limit (LFL) and sometimes as the lower explosive limit (LEL). The upper end of the range is called the upper flammable limit (UFL) or the upper explosive limit (UEL). |
| $f_{oc}$ | Fraction organic carbon in soil ($0 \leq f_{oc} \leq 1$) |
| FR | Federal Register |
| ft | Foot |
| g | Gram(s) |
| Gavage | Forced feeding through a tube passed into the stomach. |
| GC | Gas chromatography |

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| GI | Gastro-intestinal |
| Ground water | Subsurface water that occurs beneath the water table in soils and geologic forms that are fully saturated. |
| H | Henry's law constant (atm · $m^3$/mol) |
| $^3H$ | Chemical symbol for the radioactive isotope of hydrogen of atomic mass 3. |
| ha | Hectare, a unit of area equal to 10,000 square meters. |
| HA | EPA's Health Advisory (formerly termed SNARL), an estimate of the no adverse response level for short and long-term exposures to a chemical via drinking water. |
| Half-life | Time required for removal or degradation of one-half of the original quantity. |
| Halogen | One of the electronegative elements of Group VIIA of the Periodic Table: fluorine, chlorine, bromine, iodine, and astatine. Fluorine is the most active of all chemical elements. |
| Halogenated | Containing one or more atoms of halogens. |
| Hemangioma | A tumor composed of blood vessels. |
| Hemangiosarcoma | A malignant tumor composed of endothelial cells which line the heart and vessels of the circulatory system. |
| Hg | Mercury |
| HMTA | Hazardous Materials Transportation Act |
| HPLC | High-pressure liquid chromatography |
| hr | Hour(s) |
| HSDB | Hazardous Substances Data Bank |
| Hydrocarbon | An organic compound (as acetylene or benzene) consisting exclusively of the elements carbon and hydrogen and often occurring in petroleum, natural gas, coal, and bitumens. |

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| Hydrolysis | The addition of the hydrogen and hydroxyl ions of water to a molecule, with its consequent splitting into 2 or more simpler molecules. |
| IARC | International Agency for Research on Cancer |
| IDLH | Immediately dangerous to life or health concentration; represents the maximum level from which one could escape within 30 minutes without any escape-impairing symptoms or any irreversible health effects. |
| im | Intramuscular |
| in | Inch |
| intradermal | Situated or applied within the skin |
| in vitro | Describes biological experiments in laboratory apparatus rather than in a living organism. |
| in vivo | Describes process that occurs within a living organism. |
| ip | Intraperitoneal |
| IR | Infrared spectroscopy |
| IRP | Installation Restoration Program |
| IU | International units |
| iv | Intravenous |
| $K_d$ (or $K_p$) | Soil sorption coefficient |
| kg | kilogram(s) (1E+03 grams) |
| $K_{oc}$ | Soil absorption coefficient normalized to represent amount sorbed per unit weight of organic carbon in soil. |
| L | Liter(s) |
| lb | Pound(s) |
| $LC_{50}$ | The concentration required to kill 50% of test individuals. |
| $LC_{Lo}$ | Lowest reported lethal concentration. |

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|---|---|
| $LC^*t_{50}$ | Product of the concentration times time which causes lethality in 50% of the exposed population. |
| $LD_{50}$ | The dose required to kill 50% of test individuals. |
| $LD_{Lo}$ | Lowest reported lethal dose. |
| Lesion | An abnormal change in an organ because of injury or disease. |
| log $K_{ow}$ | Log of the octanol-water partition coefficient. |
| Lower flammable limit | The lowest concentration of the material in air which will support combustion. |
| m | Meter |
| $m^3$ | Cubic meter(s) |
| MAC | Maximum allowable concentration |
| Malignant | Pertaining to the growth and proliferation of certain tumors which terminate in death if not checked by treatment. |
| MCL | Maximum contaminant level |
| MDL | Minimum detection limit(s) |
| mEq | Milliequivalent (1/1000 of an equivalent) |
| mg | Milligram(s) (10E-3 gram) |
| mg% | The concentration of a solution expressed in milligrams per 100 mL. |
| min | Minute(s) |
| Mineral acids (non-oxiding) | Examples include boric, disulfuric, fluosilicic, hydriodic, hydrobromic, hydrochloric, hydrocyanic, hyfluoric, permonosulfuric, phosphoric, and selenous acids as well as chlorosulfonic acid and various fluorophosphoric acids. |
| Mineral acids (oxidizing) | Examples include bromic, chloric, chromic, acids hypochlorous, nitric, nitrohydrochloric, perbromic, perchloric, perchlorous, periodic, and sulfuric acids as well as oleum. |

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| mL | Milliliter (1E-03 liter) |
| MLD | Minimum lethal dose |
| mm | Millimeter(s) (1E-03 meter) |
| mM | Millimoles |
| mol | Gram mole |
| MPRSA | Marine Protection Research and Sanctuaries Act |
| MS | Mass spectrometry |
| Mutagen | A material that induces genetic damage. |
| MW | Molecular weight |
| n | Normal (isomer), as in n-butyl. |
| N | Normal (equivalents per liter, as applied to concentration); nitrogen (as in N-methylpyridine). |
| Narcosis | A state of stupor, unconsciousness or arrested activity. |
| NCI | National Cancer Institute |
| NEPA | National Environmental Policy Act |
| NFPA | National Fire Protection Association |
| NIOSH | The National Institute for Occupational Safety and Health |
| NIOSH No. | A unique, nine-position accession number assigned to each substance listed in the Registry of Toxic Effects of Chemical Substances published by NIOSH. |
| NIPDWR | National interim primary drinking water regulation |
| Nitride | Compounds of nitrogen with N≡ as the anion. These compounds may react with moisture to evolve flammable ammonia gas. |
| NOEL/NOAEL | No observed (adverse) effect level |
| NPL | National Priority List |
| NTP | National Toxicology Program |

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| ng | Nanogram(s) (1E-09 gram) |
| OHM/TADS | Oil and Hazardous Materials Technical Assistance Data System |
| OSHA | Occupational Safety and Health Act (or Administration) |
| Oxidation | Any process involving the addition of oxygen, loss of hydrogen, or loss of electrons from a compound. |
| Oxidizing materials | Any compound that spontaneously evolves oxygen either at room temperature or under slight heating. The term include such chemicals as peroxides, chlorates, perchlorates, nitrates, and permanganates. These can react vigorously at ambient temperatures when stored near or in contact with reducing materials such as cellulosic (i.e., cotton, paper, rayon) and other organic compounds. In general, storage areas for oxidizing materials should be well ventilated and kept as cool as possible. |
| PEL | Permissible exposure limit, as found in 29CFR 1910.1000. |
| Percutaneous | *Penetration of the skin* |
| pg | picogram(s) (1E-12 grams) |
| pH | A measure of acidity or alkalinity of a solution on a scale of 0-14; log of the reciprocal of the hydrogen ion concentration. |
| PID | Photo ionization detector |
| Pk | Peak concentration. |
| Plasma | The straw-colored, fluid portion of blood that remains when all cells are removed. |
| po | By mouth |
| Polymerizable material | A substance capable of self-polymerization under appropriate conditions. Polymerization reactions are often violent, exothermic, and capable of causing violent rupture of sealed containers. |

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| Polymerization | A chemical reaction, usually carried out with a catalyst, heat, or light, and often under high pressure. In this reaction, a large number of relatively simple molecules combine to form a chain-like macromolecule. This reaction can occur with the release of heat. In a container, the heat associated with polymerization may cause the substance to expand and/or release gas and cause the container to rupture, sometimes violently. The polymerization reaction occurs spontaneously in nature; industrially it is performed by subjecting unsaturated or otherwise reactive substances to conditions that will bring about the combination. |
| POTWs | Publicly owned treatment works |
| ppb | Part(s) per billion |
| ppm | Part(s) per million |
| ppt | Part(s) per thousand |
| PVA | Polyvinyl acetate |
| PVC | Polyvinyl chloride |
| Raw | Applied to water or waste water that has undergone no treatment. |
| RCRA | Resource Conservation and Recovery Act |
| Reactivity (chemical) | Relating to the potential for a substance to undergo chemical transformation or change in the presence of other materials. Such chemical reactions often (but not always) are hazardous and involve evolution of heat, toxic or flammable gases, fires, or expolsions. The products formed by the reaction may have properties or hazards different from those of the chemical reactants. |
| RBC | Red blood cells |

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| Reducing agents | These agents act to extract and liberate hydrogen from organic substances and may generate toxic and/or flammable gases and heat in contact with water. Many reducing agents may be pyrophoric and may ignite combustible materials in the presence of air. Contact with oxidizing materials may result in violent or explosive reactions. Examples of reducing agents include calcium, phosphorus, sodium, hydrazine, arsine, and metallic acetylides, aluminates, boranes, bromides, carbides, chlorides, hydrides, hydroborates, hyposulfites, iodides, phosphides, selenides, and silanes, as well as metal alkyls such as triethyl aluminum and diethyl zinc. |
| Reduction | Decreasing the oxygen content or increasing the proportion of hydrogen in a chemical compound or adding an electron to an atom or ion. |
| REL | Recommended exposure limit |
| Rf | Retardation factor, i.e., the ratio of the velocity of the interstitial water to the velocity of a pollutant in soil. |
| RfD | Reference dose |
| RMCL | Recommended maximum contaminant level |
| RNA | Ribonucleic acid |
| RQ | Reportable quantities |
| SAE | Society of Automotive Engineers |
| sc | Subcutaneous, beneath the skin |
| SD | Standard deviation, a measure of the spread of individual measurements of a normally distributed variable. |
| SDWA | Safe Drinking Water Act |
| sec | Second(s) |
| Serum | The clean amber fluid that remains after blood has clotted; plasma without any of the substances involved in clotting. |
| SGOT | Serum glutamic oxalacetic transaminase, an enzyme released into the serum as the result of tissue injury, especially injury to the heart and/or liver. |

| | |
|---|---|
| SGPT | Serum glutamic pyruvic transaminase, an enzyme released into the serum as a result of tissue injury, especially damage to liver cells. |
| SH | Sulfhydryl group |
| SNARL | Suggested no adverse response level |
| STEL | Short-term exposure limit |
| STP | Standard temperature and pressure |
| Subcutaneous | Beneath the skin |
| Surface water | That water contained on the exterior or upper portion of the earth's surface; it does not include ground water. |
| Sym | Symmetrical |
| $t_{1/2}$ | Half-life |
| $TD_{Lo}$ | Lowest reported toxic dose |
| Teratogen | A material that induces nontransmissible changes (birth defects) in the offspring. |
| TLV® | Threshold limit value; an ACGIH-recommended time-weighted average concentration of a substance to which most workers can be exposed without adverse effect. |
| TNT | Trinitrotoluene, an explosive used in the munitions industry. |
| Toxic metals and their compounds | These include antimony, arsenic, barium, beryllium, bismuth, cadmium, chromium, cobalt, copper, indium, lead, manganese, mercury, molybdenum, nickel, osmium, selenium, thallium, thorium, titanium, zinc, and zirconium; compounds containing these metals; and metallic compounds containing arsines, boron, calcium, cesium, magnesium, silver, strontium, tellurium, tin, tungsten, or vanadium, among others. |
| TSCA | Toxic Substances Control Act |
| TWA | Time-weighted-average |
| µg | Microgram(s) (1E-06 gram) |
| µL | Microliter(s) (1E-06 liter) |

| | |
|---|---|
| uns | Unsymmetrical |
| Upper flammable limit | The highest concentration of the material in air which will support combustion. |
| USAF | United States Air Force |
| USEPA | United States Environmental Protection Agency |
| Vol.% | The number of milliliters of a substance in 100 milliliters of the medium. |
| Water quality standard | Legally enforceable provisions of state or Federal law which consist of a designated use or uses for the waters of the United States and water quality criteria for such waters based upon such uses. |
| WHO | World Health Organization |
| wk | Week(s) |
| w/v | Weight per unit volume |
| w/w | Weight per unit weight |
| % | Percent |
| > | Greater than |
| ≥ | Greater than or equal to |
| < | Less than |
| ≤ | Less than or equal to |
| ~ | Approximately |
| -> | Yields or causes |
| + | Plus |

# THE INSTALLATION RESTORATION PROGRAM TOXICOLOGY GUIDE

# INTRODUCTION

## 1.1 INSTALLATION RESTORATION PROGRAM

### 1.1.1 Overview

Although the nation's ground-water resources were once considered to be virtually unlimited and pristine, contamination from improper disposal of hazardous waste is a growing threat. Between 40 and 50% of the U.S. population relies on ground water as its primary source of drinking water (530), thus ground water contamination has become a drinking water problem.

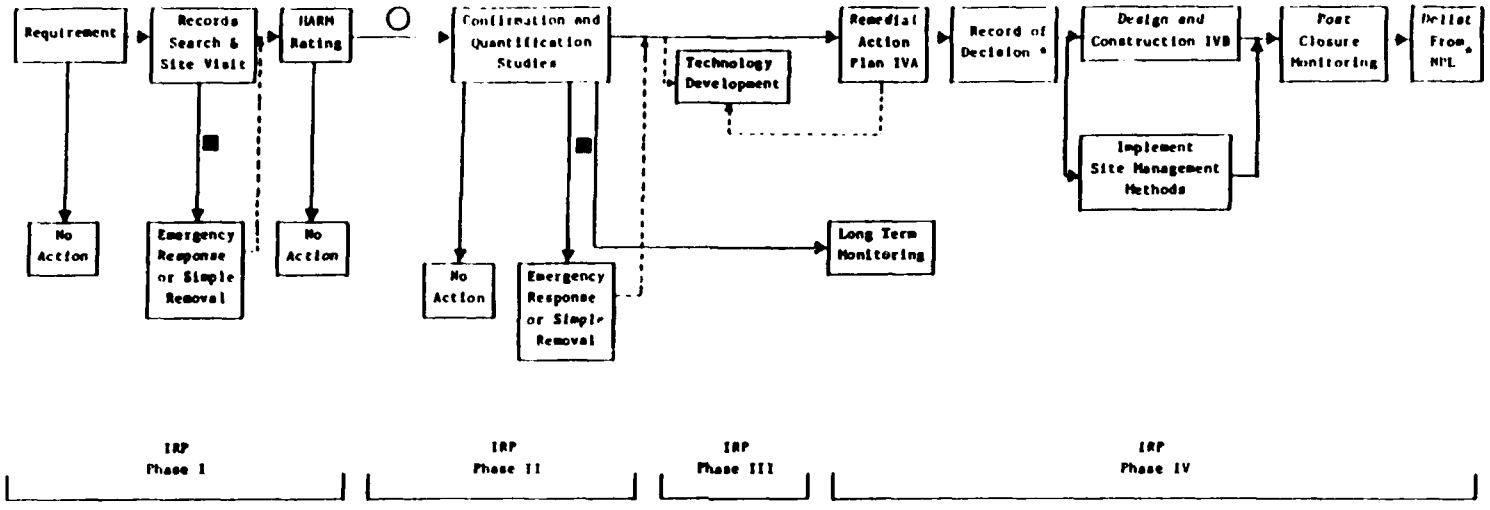
The Defense Environmental Quality Program Policy Memorandum (DEQPPM) 81-5 defines a comprehensive program to identify and evaluate past DOD hazardous material disposal sites on DOD installations and to control the migration of hazardous environmental contamination resulting from such sites into ground water. The Air Force Installation Restoration Program (IRP), initiated in December 1981, implements this DOD directive.

There are four phases to the IRP:

Phase I - Contaminant problem identification
Phase II - Verification/quantification via sampling and analysis
Phase III - Technology development
Phase IV - Remedial action

The four phases of the Air Force IRP are illustrated in Figure 1.

Experience gained through implementing Phases I and II of the IRP has shown a need for specific contaminant information for a vast array of chemicals commonly found in ground water. Specifically, the U.S. Air Force (USAF) requires contaminant information to evaluate health hazards associated with actual or potential contamination of drinking water supplies on and off USAF installations and to provide background and rationale for the development of remedial action efforts with federal, state and local regulatory agencies.




→ Optional Track

* For National Priority List sites

○ USAF Sites may proceed to Phase II whether included on the National Priority List or not.

■ Emergency Responses can be initiated in any Phase but are implemented as Phase IV actions.

Source: USAF (789)

FIGURE 1
AIR FORCE INSTALLATION RESTORATION PROGRAM

The Harry G. Armstrong Aerospace Medical Research Laboratory was requested by HQ USAF/SGPA to develop health and environmental information for each potential contaminant of drinking water supplies associated with USAF installations, i.e., a ground water contamination information data base. This Installation Restoration Program Toxicology Guide is a product of that effort.

It should be stressed that where legally promulgated standards exist, USAF installations must adhere to these standards. In cases where relevant standards do not exist, it is USAF policy to assure that past or present operations do not create unacceptable risks to the health of employees, the public or the environment. As the USAF does not set environmental standards, the ground-water contaminant information contained in this Guide should not be used as standards nor construed as standard setting. The objective of this Guide, therefore, is to (1) identify those contaminants for which criteria, standards or USEPA based guidelines are available, (2) provide a ground-water contaminant information data base for use at USAF installations where there are no relevant national, state, or local standards, and (3) provide guidelines to aid in the development of USAF policy and programs for IRP completion.

### 1.1.2 The Installation Restoration Program Toxicology Guide

#### 1.1.2.1 Purpose and Scope

The objective of the IRP Toxicology Guide is to serve as a basic day-to-day resource for USAF personnel who have responsibility for the management of hazardous waste, particularly in regard to contamination of ground-water supplies. The IRP Toxicology Guide was intended for and focused toward technical personnel with appropriate backgrounds to recognize the significance as well as the limitations of various test procedures and findings.

In scope, the IRP Toxicology Guide outlines the environmental fate and effects, exposure pathways, toxicity and sampling/analysis techniques for a selected list of chemicals, some of which, when accidently or unknowingly released to ground water, may present a hazard. The material provided is intended as an overview of key topic areas; no attempt was made to provide a comprehensive review.

Several different types of resources were used in compiling the data presented in the IRP Toxicology Guide. Much of the data was taken from pertinent criteria documents and review articles. A large group of texts and handbooks were reviewed as well as recent primary sources uncovered in computerized literature surveys conducted for each chemical included in the Guide. Computerized literature surveys were conducted from 1979 forward. All chemicals were identified in CHEMLINE, an on-line dictionary of chemicals available through the National Library of Medicine's MEDLARS data bases. Three other files, the Registry of Toxic Effects of Chemical Substances (RTECS), the Hazardous Substances Data Bank (HSDB) and TOXLINE, all available through MEDLARS, were included in the computerized

search. Additional searches were performed on the Chemical Abstract Service's CA file for data on properties and the environmental fate of the chemicals under evaluation. All citations and abstracts were reviewed and primary citations obtained in relevant areas as deemed appropriate to update secondary review sources and fill-in data gaps.

### 1.1.2.2 Selection of Chemicals

The chemicals included in the IRP Toxicology Guide resulted from the USAF Occupational and Environmental Health Laboratory (OEHL) review of all IRP Phase II reports. The selected chemicals were detected in ground water as part of the Phase II effort.

The Guide format was designed to permit inclusion of additional chemicals, as well as permitting periodic updates of information. Volume 4 includes additional industrial chemicals, pesticides, and POL (petroleum, oil and lubricant) products of interest to the USAF Medical Service. Candidate chemicals for inclusion in future Guide expansions should be forwarded through MAJCOM bioenvironmental engineers to HQ USAF/SGPA. Consultant service concerning the current status of the toxicological information contained in this Guide should be obtained from the USAF OEHL/ECO.

### 1.1.2.3 *Organization*

The IRP Toxicology Guide is organized by individual chapters for each of the chemicals selected. The major sections for each chapter are described below.

- Summary Chart which contains chemical and physical properties, reactivity, structural and molecular formulas, molecular weight, common synonyms, Chemical Abstract and NIOSH registry numbers, conversion values, and handling precautions. Summaries of persistence in soil-water systems, exposure pathways and health hazards are also provided.

- Environmental and Occupational Standards and Criteria lists existing environmental air and water standards and criteria and occupational exposure limits.

- Regulatory Status encompasses proposed and existing regulations in the U.S. at the federal and state level and in European Economic Community (EEC) countries as of March, 1989. Since this type of legislation is in a constant state of change, the reader is cautioned that future regulations may render some of this information obsolete.

- Major Uses section is meant to serve as a guide only, and is not comprehensive. It was usually obtained from published sources.

- Environmental Fate and Exposure Pathways deals with the transport, transformation and fate of the chemicals in soil/ground-water systems. Data on persistence and bioaccumulation are also included. Potential pathways of human exposure, particularly oral and dermal pathways from soil/ground-water systems, are discussed with respect to other sources of human exposure to the contaminant.

- Human Health Considerations summarizes acute and chronic effects noted in humans and experimental animals, including cancer, genetic and reproductive hazards and other chronic functional impairments. This section is not intended to cover all reported studies. In general, the doses and schedules are indicated as they appear in the original sources; sometimes units have been converted (see Appendix 3, Math, Conversions and Equivalents) for easier comparisons but are set off in parentheses. Also included are levels of concern (e.g., Reference Doses, Suggested No Adverse Response Levels) and an assessment of hazard, indicating areas of concern associated with exposure to a particular contaminant and the uncertainties involved in defining these concerns.

- Sampling and Analysis provides soil and water sampling analysis procedures required or recommended by regulatory agencies.

Each chemical has been assigned a chemical-specific number (i.e., 1 through 70), and the chapter pages for each chemical are numbered accordingly (e.g., 1-1, 70-3). Nonproprietary or trivial names have been utilized as chapter headings, with the Chemical Abstract Service name listed as the first synonym under common synonyms at the beginning of each chapter.

Four indices have been included as keys to chapters. These are based on:

- Chemical names, recognized common names and a few tradenames (Index 1); this list of synonyms is not comprehensive
- Molecular formula (Index 2)
- Chemical Abstracts Registry Numbers (Index 3)
- National Institute of Occupational Safety and Health Numbers (Index 4)

Unless otherwise specified, temperatures are given in degrees Celsius (centigrade). Data are generally reported in metric units.

A listing of handbooks, data books, response guides and USAF documents which may be useful to USAF personnel for IRP completion is given in Appendix 1. Air Force points of contact for the IRP are listed in Appendix 2. Appendix 3 contains mathematical formulas and conversion factors used for easier comparison of health

effect studies. Appendix 4 contains the addresses, telephone numbers and contacts for the State Water Agencies.

## 1.2 FACTORS THAT IMPACT WATER QUALITY AND WATER QUALITY REGULATIONS

The objectives of this section are two-fold: first, to provide the reader with some insights on issues that surround water quality and the development of water quality criteria and regulations; and second, to indicate the general approach and criteria adopted for this Guide in the analysis of environmental fate, exposure pathways, human health effects and hazard assessment, and sampling and analysis. The uncertainties that entered into the evaluations of each of these areas of analysis and the impact each of these items exerts on the development of criteria and regulations are also discussed.

### 1.2.1 Environmental Fate of Contaminants

The first "law" of environmental pollution states that: "Everything must go somewhere." An assessment of the ultimate fate of a ground-water pollutant should answer two questions following logically from this law: (1) "Where does it go?" and (2) "How fast does it get there?" Both of these questions may reasonably be asked with regard to three types of processes:

a) Partitioning of the chemical among the three phases (soil, water, air) of the soil

b) Transport of the chemical, either in the vapor phase to the atmosphere, or in solution or suspension with the ground water

c) Degradation of the chemical by such processes as hydrolysis, biodegradation and oxidation

Quantitative answers for the second type of process (transport) usually require modeling which may be beyond the resources and data availability associated with initial assessments. The importance of various transport pathways may be assessed qualitatively (or sometimes semiquantitatively) by considering how the chemical partitions itself into the mobile phases (air, water) of the soil/ground-water environment. This is discussed in more detail in the following subsection.

For partitioning, a preliminary assessment should determine (predict) how the chemical interacts between the soil, water and air phases of the ground-water system. This partially answers the question "Where does it go?" and provides important information on the mobility of the chemical. The question of "How fast?" is seldom important for partitioning since the time scales of ground water and soil-air movement are much longer than the time required for equilibrium partitioning to be achieved.

For degradation, the question of "How fast?" should come first. This refers to assessing the rates at which the chemical is transformed (degraded) from its original form to some other compound, or series of compounds, by the processes mentioned. The answers will be in the form of rate constants or half-lives and will be environment-specific (i.e.,will depend upon such properties as pH and temperature). The question of "Where does it go?" is translated in this case to "What are the products of degradation?" The answer to this question would include a list of "intermediate" and "final" (stable) chemicals which will also be environment-specific in many cases.

### 1.2.1.1 Equilibrium Partitioning Calculations

Where appropriate, the description of the environmental fate of each chemical starts with a presentation of the results of a model calculation that predicts the likely partitioning of the chemical between the soil, soil-water and soil-air portions of the soil/ground-water system. The general approach used is that of the Level I model of Mackay (34, 35, 36).

The key assumptions involved in the use of the model are:

- equilibrium partitioning
- no degradation of the chemical takes place
- all portions of the compartments (soil/water/air) are equally accessible
- the ground water is relatively clean, fresh water
- linear adsorption isotherm for soil sorption
- for topsoils, all sorption is due to physical interaction with the organic fraction of soil
- the chemical is present as a trace contaminant (below the solubility limit in water)

A model topsoil (unsaturated) was defined having the following characteristics:

Soil volume = 60%

Air volume = 30%
Water volume = 10%
} Total porosity = 40%

Organic carbon content of soil = 2% (by weight)

Bulk density of soil = 1.6 g/cc.

Temperature = 20°C or 25°C (with a few extra calculations at 10°C)

There are only two chemical-specific inputs required by the model:

(1) H: Henry's law constant (atm · $m^3$/mol)

(2) $K_{oc}$: Soil sorption constant per unit weight organic carbon in soil (no units)

At 20°C, the three model-derived equations for the fraction, F, of the chemical in each compartment are:

$$F\ (air) = 12.5/[12.5 + (0.1 + 0.0192\ K_{oc})/H]$$

$$F\ (water) = 0.1/H[12.5 + (0.1 + 0.0192\ K_{oc})/H]$$

$$F\ (soil) = 0.0192K/H\ [12.5 + (0.1 + 0.0192\ K_{oc})/H]$$

Note that F(air) + F(water) + F(soil) = 1. For a calculation at 10°C, change all "12.5" values to "12.9".

The calculated values of F(air), F(water) and F(soil) for each chemical are given in the individual chapters. Figure 2 provides a diagrammatic summary of the calculated values for 35 of the 70 chemicals evaluated in the IRP Toxicology Guide. Each chemical is represented in Figure 2 by a point (labeled with the chemical's corresponding chapter number) plotted with the values of F (air), F (water) and F (soil). As indicated in Figure 2, most of the chemicals considered are associated with the soil fraction of the selected model environment. By contrast, vinyl chloride (#13 in Figure 2) is partitioned primarily into the soil-air fraction of the model environment.

A model deep-soil (saturated) was defined having 30% (by volume) water and 70% soil with 0.1% by weight organic carbon. This small amount of organic carbon was assumed (unless otherwise noted) to be responsible for the extent of the chemical's sorption. The bulk density of the soil was taken as 1.8 g/cc. The equations for the fractions associated with the water and soil phases are then calculated from:

$$F(water) = 0.3/[0.3 + 1.26E\text{-}03\ K_{oc}]$$

$$F(soil) = 1.26E\text{-}03\ K_{oc}/[0.3 + 1.26E\text{-}03\ K_{oc}]$$

Note that F(water) + F(soil) = 1. The calculated values of F(water) and F(soil) are given in the individual chapters on each chemical. Figure 3 provides a diagrammatic

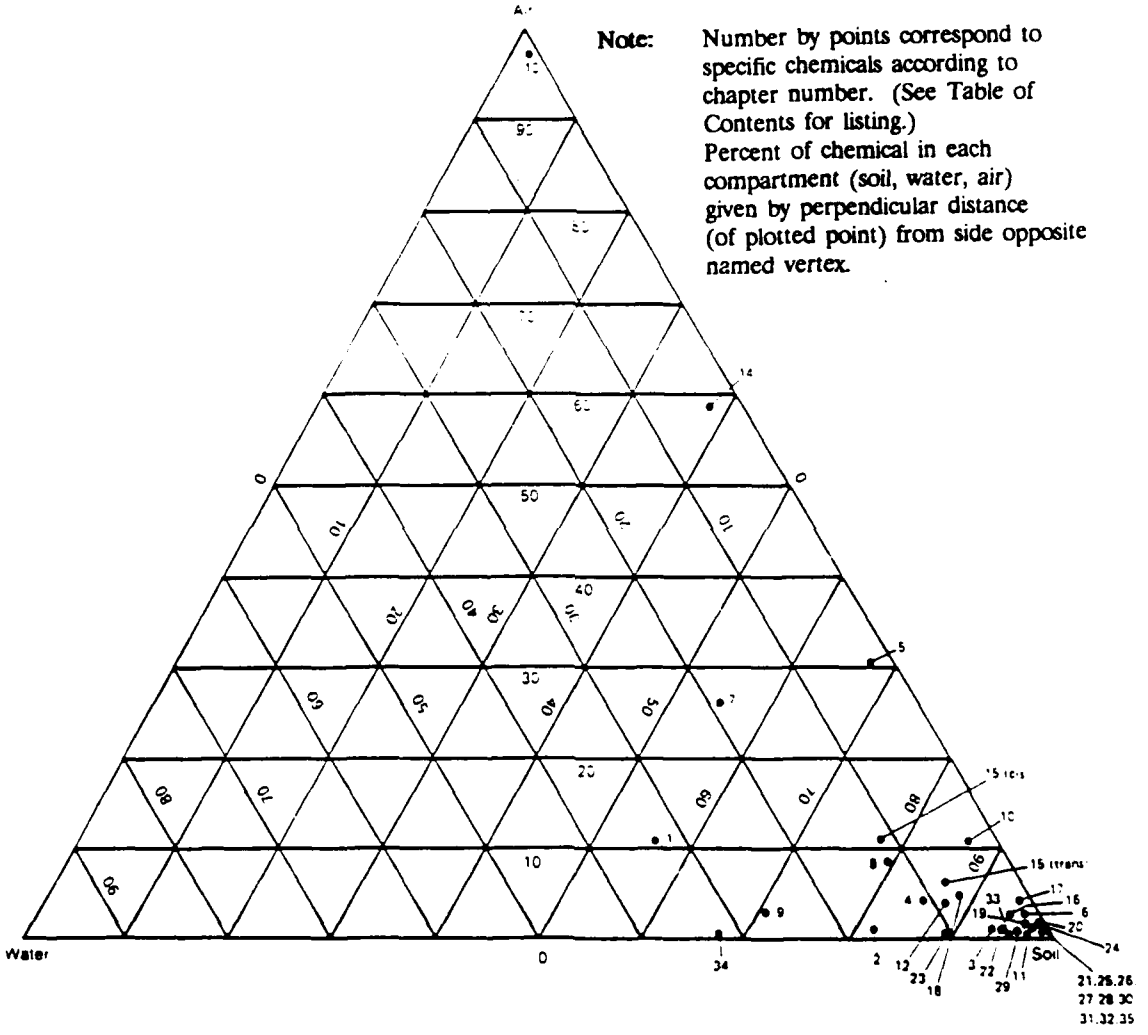



FIGURE 2

REPRESENTATION OF CHEMICAL PARTITIONING IN MODEL TOPSOIL BY IRP CHEMICALS

summary of the calculated values for 35 of the 70 chemicals evaluated in the IRP Toxicology Guide. In this figure, as in Figure 2, each chemical is represented by a point (labeled with the chemical's corresponding chapter number) reflecting the F(water) and F(soil) values. As depicted in Figure 3, chemicals like methylene chloride (#1) and vinyl chloride (#13) partition primarily into the soil-water compartment while chemicals like di-n-butyl phthalate (#30) and di(2-ethylhexl)- phthalate (#31) are primarily associated with the soil fraction.

In addition to uncertainties engendered by the above-mentioned model assumptions, other uncertainties may be associated with the values of H and $K_{oc}$ used. In many cases, the values of H and $K_{oc}$ actually used are estimates, although the estimates are based upon generally accepted estimation procedures. Because of these uncertainties, the model results ought only to be used in a general, semiquantitative way in any specific environmental assessment. Where appropriate, site-specific modeling should be undertaken to obtain a higher degree of reliability in the estimates.

#### 1.2.1.1.1 Fish Bioconcentration Factor

The summary table of physicochemical properties at the beginning of each chapter contains an estimated fish bioconcentration factor (BCF) based on the presumed equilibrium of the chemical between fish (whole body, wet weight) and the concentration in water:

$$BCF = \frac{\text{Concentration of chemical in fish } (\mu g/g)}{\text{Concentration of chemical in water } (\mu g/mL)}$$

In all cases, the estimates were derived from an equation derived by Mackay (37):

$$\text{Log BCF} = \text{Log } K_{ow} - 1.32$$

where $K_{ow}$ = octanol-water partition coefficient.

These estimated values of BCF should be presumed to have an uncertainty of between a factor of five and ten. Any measured values of BCF, if available, are also provided in the summary table of properties.

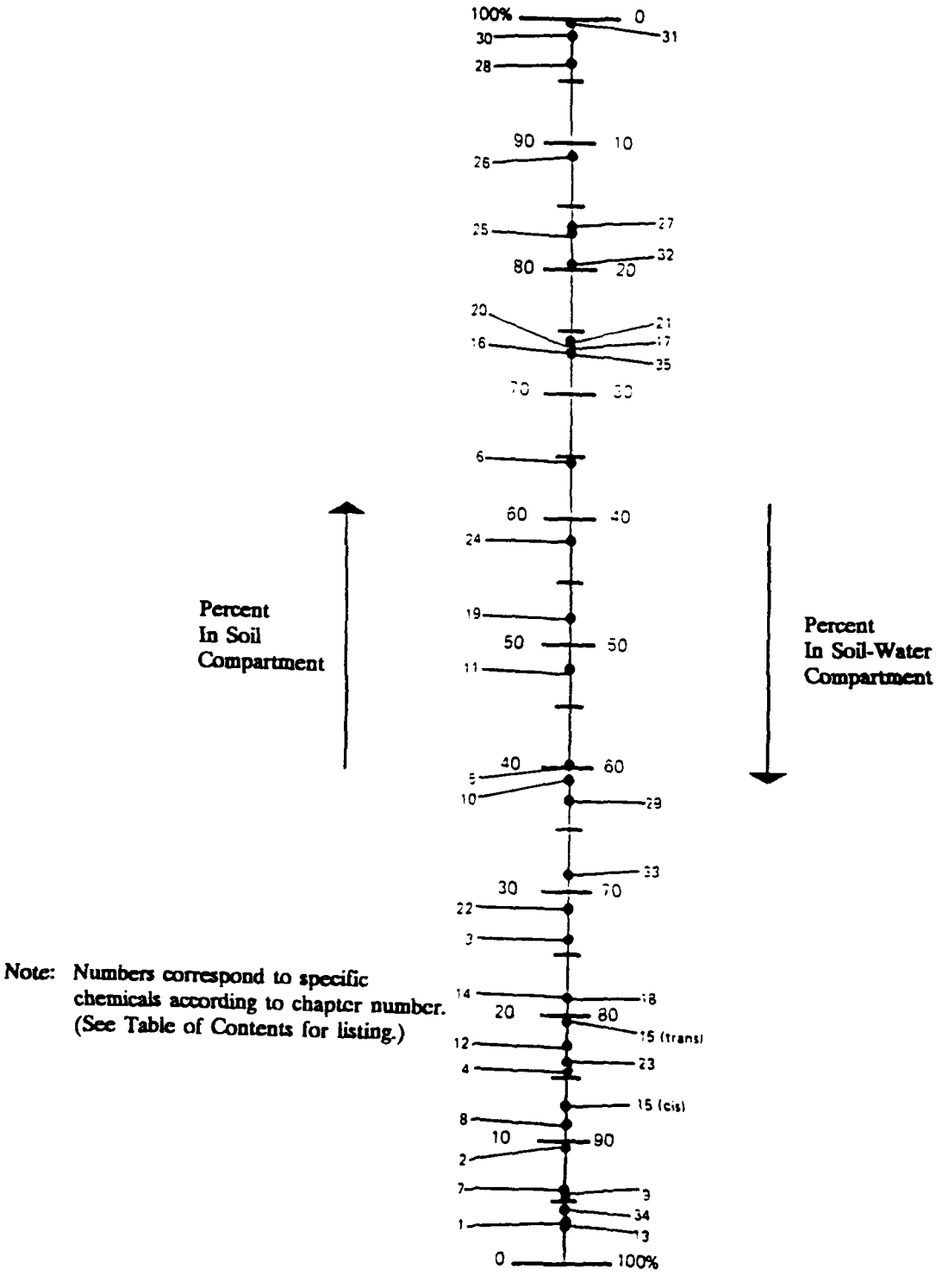



FIGURE 3

PARTITIONING OF SELECTED CHEMICALS IN MODELED DEEP SOIL SYSTEM

### 1.2.1.2 Transport Processes

#### 1.2.1.2.1 Soil Sorption

A brief description is given for each chemical of the extent of soil sorption to be expected. This is almost always based upon the value of $K_{oc}$ which is defined by:

$$K_{oc} = K_d/f_{oc}$$

where $K_d$ = measured soil sorption coefficient, and

$f_{oc}$ = fraction organic carbon in soil.

Most research studies have found that $K_{oc}$ for a chemical is reasonably constant from soil to soil (% coefficient of variation 10-140%) as long as $f_{oc}$ is above about 0.001 (i.e., the soil has over 0.1% by weight organic carbon). Values of $K_{oc}$ range over seven orders of magnitude, from 1 to 10,000,000. Values above 10,000 will be indicative of fairly strong sorption and corresponding low mobility in the ground-water phase.

In essentially all cases, a value of $K_{oc}$ has been estimated for each chemical using the equation of Means et al. *(611)*:

$$\text{Log } K_{oc} = \text{Log } K_{ow} - 0.317$$

where $K_{ow}$ = octanol-water partition coefficient.

The estimated values of $K_{oc}$, along with any experimental values, are provided in the table of properties at the front of each chapter. The uncertainty in the estimated values of $K_{oc}$ is about a factor of three to five.

It is possible, for neutral organic chemicals, to estimate the concentration of a pollutant in ground water if the concentration of the pollutant in the soil is known:

$$C \text{ (water)} = \frac{C \text{ (soil)}}{K_d} = \frac{C \text{ (soil)}}{K_{oc} \cdot f_{oc}}$$

where C (water) = pollutant concentration in water ($\mu$g/L)
C (soil) = pollutant concentration in soil ($\mu$g/kg)
$K_d$, $K_{oc}$, $f_{oc}$ are as defined above.

The use of this equation involves a number of subtle assumptions which can introduce errors into the estimated values of C (water), but the estimates should provide values that are within a factor of 5 to 10 of the actual values.

#### 1.2.1.2.2 Volatilization from Soil

A brief description is given for each chemical of the extent to which volatilization - transport throgh the air-filled pores up to the soil surface - is likely to be an important process. This is often based on the value of Henry's law constant, H, which can range over eight orders of magnitude, from 1E-07 to 10 atm · $m^3$/mol at ambient temperatures. If H is above 1E-03 atm · $m^3$/mol, the chemical may be considered fairly volatile.

There are cases where volatilization may be an important (if slow) transport pathway even when H is very small. This is when $K_{oc}$ is very large and the chemical is stable against degradation. Chemicals like DDT and dioxin fall into this category.

### 1.2.1.3 Degradation Processes

Only two types of degradation processes are routinely considered for each chemical, hydrolysis and biodegradation. If no data (or estimates) on these processes are available for the chemicals included in the Guide, qualitative estimates are given as to their importance based upon structural analogies with other chemicals. General rules for assessing the likely susceptibility to hydrolysis are given by Harris (529) while a number of (structural) rules-of-thumb for biodegradation are given by Scow (515).

Biodegradation will not be important if there are insufficient microbes present in the soil/ground-water system, or if the conditions for their growth are poor. Assessments of biodegradation potential are hampered by the fact that most of the available literature describes aerobic biodegradation while anaerobic conditions are more likely in soil/ground-water systems.

It is possible that other degradation reactions may act upon the chemicals in the soil/ground-water system. These would include oxidation/reduction reactions or reaction with other dissolved chemical species (e.g., by substitution), some of which may also be considered pollutants. A recent study by Schwarzenbach et al. (528) showed that halogenated alkenes and alkanes can react with sulfides (probably hydrogen sulfide) to form organic sulfides.

### 1.2.1.4 Overall Confidence in Environmental Fate Data

There are many data gaps in current knowledge of the environmental fate of the chemicals in this Guide. This has necessitated the use of many estimated physicochemical property values. There is a great need for additional basic data (i.e., fundamental partition coefficients and reaction rate constants) and field studies which provide not only "real-world" information, but test cases against which models can be

tested and calibrated. Given no evidence to the contrary, predictions of many environmental transport and fate variables (extent of sorption, rate of volatilization from soil, rates of degradation, time to travel given distances, etc.) ought to be considered to have an uncertainty of a factor of 5 to 10.

### 1.2.2 Exposure Pathways

There are two components to exposure pathways in the context of pollutants originating from disposal sites. The first component is the environmental pathways by which the pollutants migrate from the disposal site. These fate pathways include leaching, runoff, air entrainment and volatilization. Exposure may then occur if a receptor (human or other biota) comes into contact with the compound or compounds. Exposure pathways include ingestion of contaminants in food and drinking water, inhalation of contaminants in air (either as vapors or as particulates), or dermal contact with contaminants in soil, water or other media. Figure 4 illustrates the various environmental and exposure pathways from a disposal site which may result in human exposure. The nature of the site and the chemicals associated with it will determine which pathways are important in a particular situation. As discussed above, this Guide is limited to a consideration of environmental and exposure pathways related to soil/ground-water. Figure 4 may be used to consider potential pathways of exposure from a given site, or it may be used to evaluate the pathways which may have caused an observed exposure.

For each chemical considered in the Guide, two sections were prepared relevant to exposure, i.e., Primary Routes of Exposure from Soil/Ground-water and Other Sources of Human Exposure. Our approach to these two sections is discussed below.

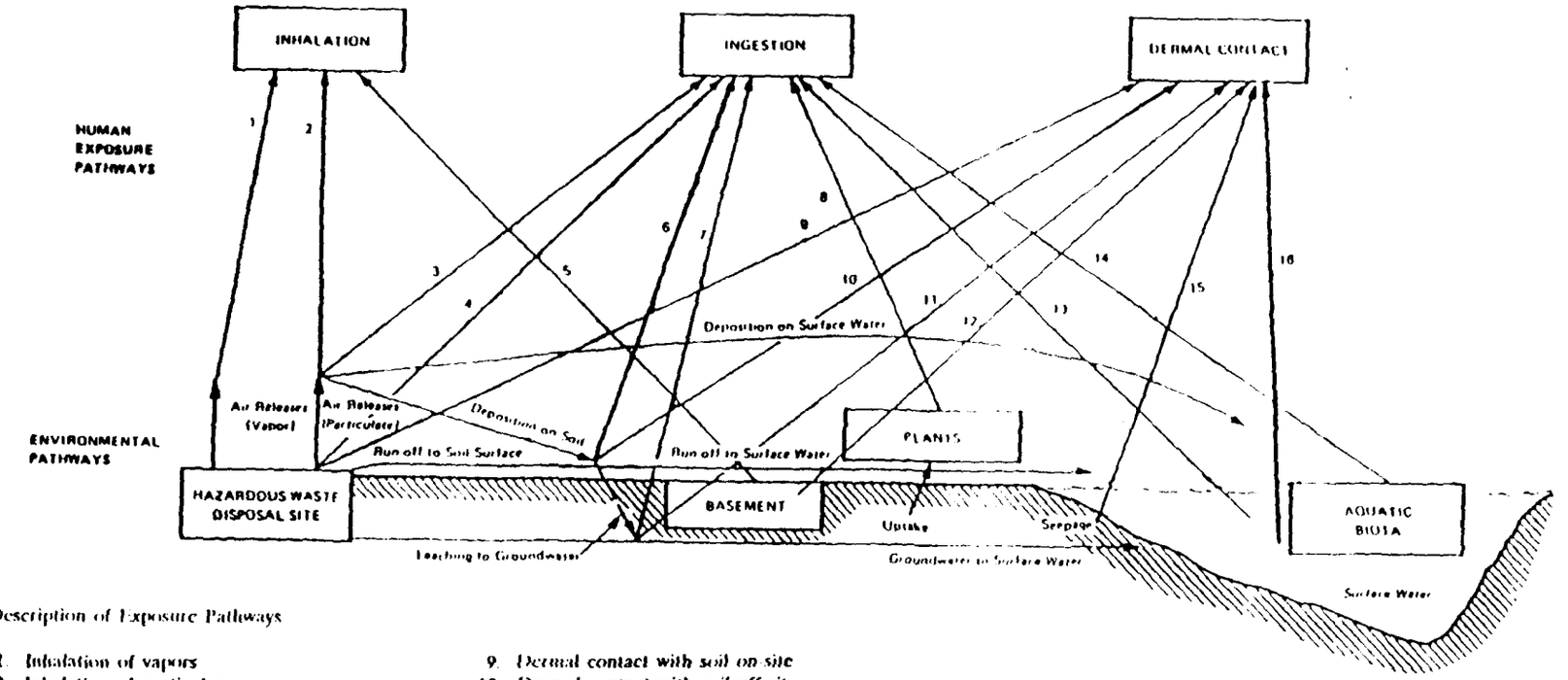
The Primary Routes of Exposure section is intended to provide an indication of the potential exposure pathways from soil/ground-water systems, given the nature of the chemical's fate properties. General conclusions about potential exposure pathways were drawn using these fate properties and information was provided, if available, which supports the importance of the exposure pathway.

In order to provide a consistent basis for exposure, chemicals were classified according to their physicochemical properties. Table 1 shows the fate property classifications that were used. The relative importance of exposure pathways was discussed qualitatively on the basis of these classifications and chemical-specific information presented in the fate section.

In addition to this qualitative discussion of exposure pathways, available information was included to document the importance of the exposure pathways. Several sources were used in this regard. First, a compilation of data prepared by Mitre Corporation (83) was included. This compilation includes data for 230 chemicals or

TABLE 1
CHEMICAL PROPERTY CLASSIFICATIONS AND
RELEVANT EXPOSURE PATHWAY

| Property | Range | Qualitative Description | Relevant Exposure Pathways |
|---|---|---|---|
| Henry's Law Constant<br>H(atm · $m^3$/mol) | <3E-07 | Nonvolatile | High volatility suggest inhalation pathway |
| | 3E-07 <H<1E-05 | Low volatility | |
| | 1E-05 <H<1E-03 | Moderate Volatility | |
| | H>1E-03 | High volatility | |
| Fish Bioconcentration Factor (BCF) | <5 | No significant potential for bioaccumulation | High BCF suggests ingestion food pathways |
| $\frac{\mu g/g}{\mu g/mL}$ | 5-50 | Low potential for bioaccumulation | |
| | 50-500 | Moderate potential for bioaccumulation | |
| | >500 | High potential for bioaccumulation | |
| Soil adsorption coefficient ($K_{oc}$) | <10 | Very weakly sorbed | Low $K_{oc}$ suggests movement with ground water and possible drinking water contamination |
| | 10-100 | Weakly sorbed | |
| | 100-1000 | Moderately sorbed | |
| | 1000-10,000 | Moderately to strongly sorbed | |
| | 10,000-100,000 | Strongly sorbed | |
| | >100,000 | Very strongly sorbed | |




Description of Exposure Pathways

1. Inhalation of vapors
2. Inhalation of particulates
3. Swallowing of larger particles
4. Ingestion of soil on-site (particularly children)
5. Inhalation of vapors form seepage in basements
6. *Ingestion of soil Off site (particularly children)*
7. Ingestion of groundwater
8. Ingestion of plants (home gardens)
9. *Dermal contact with soil on-site*
10. Dermal contact with soil off-site
11. *Dermal contact from household use of groundwater (bathing, etc.)*
12. Dermal contact w/seepage in basements
13. Ingestion of surface water
14. *Ingestion of aquatic biota*
15. Dermal contact with seepage
16. Dermal contact with surface water (swimming, etc.)

FIGURE 4

EXPOSURE PATHWAYS FROM A HAZARDOUS WASTE DISPOSAL SITE

groups of chemicals that have been reported in the vicinity of the 546 National Priority List (NPL) sites as designated under CERCLA (The Superfund Act). These authors report the number of sites where each compound was reported in ground water, surface water and air. The presence of a chemical in one of these media at a number of sites indicates that an environmental pathway exists, and an exposure pathway is certainly possible. A comparison of this information between chemicals or environmental media cannot be made since equivalent sampling and analysis was not performed at all sites for all media.

The potential for exposure through drinking water can be evaluated using various monitoring surveys. These data indicate whether contamination problems are prevalent and whether the contamination is related to ground water or surface water supplies. While the sources of contamination are not discussed in these surveys, frequent contamination, particularly of ground water, indicates that the movement of the particular chemical in ground water can occur, and that exposure has resulted.

The surveys available for drinking water include the United States Environmental Protection Agency (USEPA) (62, 64), and Westrick et al. (531); the USEPA surveys (62, 64) summarize state reports or local contamination problems. They also summarize data from the National Organics Monitoring Survey (NOMS), the National Screening Program, and the Community Water Supply Study. All of these surveys included both ground water and surface water supplies. The 1982 Groundwater Supply Survey (GWSS) is the most recent study (531). This survey sampled a total of almost 1000 drinking water systems using ground water, 466 selected at random, and about 500 selected by the states as potentially contaminated.

The section, Other Sources of Exposure, is intended to put the potential of exposure to the compound from a disposal site in perspective with typical exposures from other sources. Again, the routes of exposure included ingestion (food and drinking water), inhalation and dermal contact.

The prevalence of certain contaminants in drinking water was evaluated using the monitoring surveys indicated above.

Typical levels of various contaminants in air were summarized by Brodzinsky and Singh (84). In this study, ambient data for 151 chemicals were taken from 241 references from about 1970 to 1980. The data were compiled and percentile concentrations were obtained for rural and remote areas, urban and suburban areas, and source-dominated areas. There were common apparent conflicts in these data caused by inadequate or questionable data in one of the locations; however, they provide a good indication of the level of inhalation exposure for the chemicals included.

Other sources of exposure, such as food ingestion and product-use related exposures could only be evaluated on a case-by-case basis, as no good national data

base exists. The literature provided information for some chemicals but, in many cases, no information was available for these exposure routes.

### 1.2.3 Human Health Considerations

The best evidence that exposure to a particular chemical or product may pose a health hazard to humans is human data indicating a strong association between exposure and response. Generally, this information is not available, and by necessity, data from laboratory animal studies and various nonhuman test systems are used to assess potential adverse health effects in humans. This section presents the rationale for examining specific health effects, the limitations of various types of studies and a general approach to categorizing the likely impact exposure to a particular chemical or product will have on human beings. Various general aspects related to risk assessment/risk management will be discussed in a later section.

#### 1.2.3.1 Carcinogenicity

Carcinogenesis, the induction of cancer, is a biological process characterized by the unrestrained growth of the offspring of cells which have been modified by one or more events in which the genome (DNA) and/or other cellular regulatory mechanisms were altered. The outgrowth of a cancer in response to some triggering event most likely involves a sequence of events occurring over an extended period of time, and may, in fact, be manifested long after exposure stops. The processes of tumor formation and progression to malignancy subsequent to an initial carcinogenic event remain to be elucidated; host factors such as hormonal and immunological status, genetic background, and a variety of cultural and behavioral factors associated with life-style have been implicated as modifying factors (237, 238, 239).

Two major types of data are used to identify toxicants that may pose a carcinogenic risk to humans. They are epidemiological evidence derived from studies of exposed human populations and experimental data from long-term tests in laboratory animals. Human data provide the most reliable basis for estimating the carcinogenic effects of a toxicant. In practice, however, epidemiologic studies provide correlations or associations, but exposures are usually not well-defined enough to allow documentation of either the precise amount of exposure or the temporal sequence (246). A major source of this imprecision is the long and variable latency period (ranging from 2 to 40 years) between initial human exposure to a suspected carcinogen and clinical manifestations of cancer (240). Reports of occupational exposure may provide a somewhat more direct indication of causality but the dose-response relationships are often difficult to define and are frequently confounded by an occupational history of exposure to multiple toxic agents.

To permit a more rigorous analysis of carcinogenic risk, human data are therefore commonly coupled with experimental animal data. By convention, the National Cancer Institute/National Toxicology Program (NCI/NTP) studies are conducted with rats and mice (both sexes); the test substance is administered continually by the

selected route of administration from weaning (6-8 weeks of age) through the major portion of the animal's life (90-110 weeks). Following autopsy and microscopic examination of the tissues, the incidence and types of tumors at various sites are compared with those in control animals not exposed to the test substance.

Carcinogenic activity for each chemical evaluated in this Guide was characterized according to the National Toxicology Program (NTP) categories of interpretive conclusions, the International Agency for Research on Cancer (IARC) weight-of-evidence categories for potential human carcinogens, and the United States Environmental Protection Agency (USEPA) categorization based on animal and human data. The five categories of interpretive conclusions adopted by the NTP (241) to describe the findings of experiments designed to evaluate the carcinogenicity of chemicals in test animals are as follows:

- Clear Evidence of Carcinogenicity is demonstrated by studies that are interpreted as showing a chemically related increased incidence of malignant tumors, studies that exhibit a substantially increased incidence of benign tumors, or studies that exhibit an increased incidence of a combination of malignant and benign tumors where each increases with dose.

- Some Evidence of Carcinogenicity is demonstrated by studies that are interpreted as showing a chemically related increased incidence of benign tumors, studies that exhibit marginal increases in tumors of several organs/ tissues, or studies that exhibit a slight increase in uncommon malignant or benign tumors.

- Equivocal Evidence of Carcinogenicity is demonstrated by studies that are interpreted as showing a chemically related marginal increase of tumors.

- No Evidence of Carcinogenicity is demonstrated by studies that are interpreted as showing no chemically related increases in malignant or benign tumors.

- Inadequate Study of Carcinogenicity indicates that because of major qualitative or quantitative limitations, the studies cannot be interpreted as valid for showing either the presence or absence of a carcinogenic effect.

The weight-of-evidence scheme employed by IARC (804) to classify potential human carcinogens consists of three categories:

- Category 1 - Carcinogenic to Humans. This classification is used only when there is sufficient evidence in humans to support a causal association between exposure and human cancer. For a causal association to be inferred between exposure and human cancer, 3 criteria must be met: (1) no identified bias which could explain the association; (2) the possibility of

confounding has been considered and ruled out; and (3) the association is unlikely to be due to chance.

- Category 2 - Probably Carcinogenic to Humans. This category is divided into higher (2a) or lower (2b) degrees of evidence. Compounds included in category 2a are chemicals for which limited evidence exists of a possible carcinogenic effect in humans (i.e., the data are not sufficient to demonstrate a causal association) plus sufficient evidence in two or more animal species. Compounds included in category 2b are chemicals for which there are sufficient evidence of carcinogenicity from animal studies only (i.e., an increased incidence of malignant tumors in: multiple species or strains; or in multiple studies (preferably with different routes and dosages); or to an unusual degree with regard to incidence, type or site of tumor, or age at tumor onset.

- Category 3 - Cannot be Classified as to Its Carcinogenicity for Humans.

The categories established by the USEPA (3970) are as follows:

- Group A - Human Carcinogen. This group is used only when there is sufficient evidence from epidemiologic studies to support a causal association between the agents and cancer.

- Group B - Probable Human Carcinogen. This group includes agents for which the weight of evidence of human carcinogenicity based on epidemiologic studies is "limited" and also includes agents for which the weight of evidence of carcinogenicity based on animal studies is "sufficient". The group is divided into two subgroups. Usually, Group B1 is reserved for agents for which there is limited evidence of carcinogenicity from epidemiologic studies. It is reasonable, for practical purposes, to regard an agent for which there is "sufficient" evidence of carcinogenicity in animals as if it presented a carcinogenic risk to humans. Therefore, agents for which there is "sufficient" evidence from animal studies and for which there is "inadequate evidence" or "no data" from epidemiologic studies would usually be categorized under Group B2.

- Group C - Possible Human Carcinogen. This group is used for agents with limited evidence of carcinogenicity in animals in the absence of human data. It includes a wide variety of evidence, e.g.(a) a malignant tumor response in a single well-conducted experiment that does not meet conditions for sufficient evidence, (b) tumor responses of marginal statistical significance in studies having inadequate design or reporting, (c) benign but not malignant tumors with an agent showing no response in a variety of short-term tests for mutagenicity, and (d) responses of marginal statistical significance in a tissue known to have a high or variable background rate.

- Group D - Not Classifiable as to Human Carcinogenicity. This group is generally used for agents with inadequate human and animal evidence of carcinogenicity or for which no data are available.

- Group E - Evidence of Non-Carcinogenicity for Humans. This group is used for agents that show no evidence for carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies.

The designation of an agent as being in Group E is based on the available evidence and should not be interpreted as a definitive conclusion that the agent will not be a carcinogen under any circumstances.

### 1.2.3.2 Genotoxicity

A mutation can be defined as any stable and heritable change in the genetic material (DNA) of a cell or organism. The types of changes that occur in the genetic apparatus of a cell can range from modifications in the base sequence of DNA, resulting in point mutations in a single gene, through major chromosomal (structural or numerical) changes that may involve entire sets of genes or even entire sets of chromosomes. Some examples of these kinds of occurrences in humans are sickle cell anemia (an example of point mutation) and Trisomy 21 (Down's Syndrome, an example of a major chromosomal disorder).

In that many substances that are carcinogens also damage DNA, tests for mutagenesis and related genetic effects are commonly used as predictive tools to identify not only substances with possible mutagenic activity, but also those that may induce cancer.

Although mutations that are phenotypically expressed are generally recognized as being deleterious to human health, data on the impact of mutation on human populations are very poor. For the most part, the current state-of-the-art cannot define what a human mutant represents in terms of either the ultimate genetic response, or human disease burden (i.e., induced mutations do not necessarily translate into an increased incidence of human genetic disease).

In the absence of direct human data, test systems that measure specific heritable changes in experimental animals, in that they directly measure genetic damage induced in parental germ cells (egg or sperm) which is observed in future generations, provide the highest degree of confidence in assessing the risks associated with human exposure to chemical mutagens. Test systems providing such data are the heritable translocation test, spot and specific locus tests and the rodent dominant lethal test. Positive findings in these test systems are cause for concern and are generally given considerable weight in an evaluation of genetic hazard.

Positive findings in one or more of the remaining battery of somatic cell genetic tests, although indicative of the possibility of heritable genetic lesions, do not constitute definitive evidence that a compound poses a hazard to future generations of humans. The scientific consensus, at present, is that there are no reliable means to translate results from these largely cellular systems to a heritable mutagenic risk to humans (242, 243). Nevertheless, consistency of results obtained in different test systems is an important input in evaluating the mutagenic activity for a particular chemical.

The USEPA's weight-of-evidence approach (244) was utilized in classifying the genetic risk associated with human exposure to a particular chemical. Namely,

- Sufficient evidence of potential human germ-cell mutagenicity exists when:
  - positive responses are induced in an in vivo mammalian germ-cell test;
  - there is confirmed mutagenic activity in at least two point mutation tests (at least one in mammalian cells) coupled with sufficient evidence of germ-cell interaction;
  - positive responses are noted in at least two structural chromosome aberration tests (at least one in mammalian cells) and sufficient evidence for germ-cell interaction; or
  - positive responses are demonstrated in both a gene mutation test and a structural chromosome aberration test in mammalian cells coupled with sufficient evidence of chemical interaction with mammalian germ cells.
- Suggestive evidence encompasses a weight-of-evidence category in which there are positive data for both mutagenic activity and evidence for chemical interactions with germ cells, but the evidence is less than sufficient.
- Limited evidence denotes a situation in which evidence is available only for mutagenicity tests (other than mammalian germ cells) or only for chemical interactions with germ cells.

### 1.2.3.3 Teratogenicity, Embryotoxicity and Reproductive Effects

The interrelationships among lethal action upon the embryo, maternal toxicity, and teratogenic effect are complex and the distinction of one type of effect from another is not always clear. A major obstacle in resolving this problem is the serious lack of clear scientific knowledge about toxic agents that affect reproduction (245).

The reactions of an embryo to a particular chemical depend on a number of factors such as metabolic differences, excretion rates, placental variations, age of the mother and nutritional status (247). Moreover, the dose, route and time of gestation

at which a conceptus is exposed are critical in defining whether a particular toxicant is teratogenic (i.e., produces malformations) in a particular species. A teratogen, the agent exerting an adverse effect on the developing conceptus, exerts its effect in the time interval between conception and the termination of morphogenetic development in the post-partum animal. The picture is complicated somewhat in that certain morphogenic events are terminated at widely varying times in different species.

The test procedures presently available to evaluate the teratogenic potential of chemicals are empirical, largely because the detailed biological mechanisms of teratogenesis are not well understood (247). This lack of information on mechanisms is highlighted by the findings that show chemicals to which humans are widely exposed such as aspirin, vitamin A and hydrocortisone, to be teratogenic in certain experimental systems. Although there are no data to eliminate these chemicals from consideration as human teratogens, there is also no evidence that their consumption by pregnant females, or by males prior to mating, at doses normally employed, has resulted in malformation in their offspring.

One means of approaching a better understanding of the relationships between teratogenic effects of toxicants in humans and in experimental animals is to examine those instances for which a toxicant has been identified as a human teratogen and has been tested later in experimental animals. The most well known case of this type is the one concerning thalidomide, which is especially instructive, since it illustrates some of the problems in using experimental animal data in a prospective manner. Had thalidomide been tested for teratogenicity according to the usual protocols in rats or mice, there would have been little or no indication of any problem. In rabbits and subhuman primates, however, thalidomide was demonstrated to be a potent teratogen.

One of the primary difficulties in extrapolating experimental data from laboratory animals to man is the high probability of differences in metabolic fate of chemicals, especially their disposition in gonadal or placental tissues. Moreover, because of the complex behavioral and dietary practices of humans, in contrast to the controlled regimens of experimental animal test populations, there may be wide individual variation among humans with respect to the ultimate metabolic fate of chemicals taken into the body. Another source of variation in humans is the intrinsic genetic individuality of each person in comparison with the rather uniform genetic background of test animals. Since the susceptibility to teratogenic stimuli appears to have a genetic component in humans, the presence of genetic diversity is a further complicating issue in the use of experimental teratology data for estimation of such a risk in humans (248).

Many of the problems cited above are also relevant to the analysis of the fetotoxic and reproductive effects of compounds to man. Epidemiologic data in humans are generally unavailable since exposure is frequently unsuspected or difficult to quantitate. A notable exception is the fetal alcohol syndrome. Caution must therefore be exercised in attempting to relate teratogenic, fetotoxic and other

reproductive effects observed in laboratory animals to humans. However, positive findings in several laboratory species would suggest the possibility of similar effects in humans and given the present imprecision in estimating potential human developmental risk, it appears reasonable and prudent to limit exposure to the chemical in question.

#### 1.2.3.4 Other Toxicologic Effects

In the absence of carcinogenic, mutagenic and reproductive effects, frequently the only source of information available for assessing potential hazard to humans are indications of short-term and chronic toxicologic effects.

##### 1.2.3.4.1 Short-term Effects

Although cases of acute human effects resulting from exposure to environmental pollutants are not very prevalent, it is important to examine acute human toxicity data for several reasons:

(1) acute accidental or occupational exposure to high concentrations of pollutants may be the only human data available.

(2) acute effects may identify specific organ systems at risk in chronic exposure.

(3) the comparison of acute human effects with animal data, combined with metabolic and other data, can support the use of chronic animal data for extrapolation to humans.

Although human data are often limited, acute toxic symptoms and effects, and in some cases, minimal lethal values are generally available, as are data on the acute toxic effects of a chemical in laboratory animals, particularly rodents. The acute $LD_{50}$ (the dose found to be lethal to 50% of the exposed population) is the most readily available piece of toxicity information for any given chemical. A large value for the $LD_{50}$ indicates a substance of low acute toxicity, while a small $LD_{50}$ value indicates a potent compound. Although $LD_{50}$ values often vary with sex, strain, species, and experimental conditions, acute toxicity studies provide information on the relative effects of different exposure routes (inhalation, ingestion, skin contact), provide a measure of comparison among many substances whose mechanism and sites of action may be markedly different, and are roughly indicative of the effects of chronic exposure to small amounts of the chemical. Acute toxicity tests are also frequently conducted to determine local effects of chemicals when applied directly to the skin or eye. Thus, acute toxicity studies place the overall toxicity of different pollutants in perspective.

#### 1.2.3.4.2 Chronic Effects

Chronic effects are defined as changes resulting from intermittent or continual exposure to low levels of a pollutant that result in detectable detriments in functional capacity (pathological, physiological, biochemical, behavioral), the ability of the organism to maintain homeostasis, or to compensate for a treatment-induced enhanced susceptibility to the deleterious effects of other environmental insults. Although all significant toxic effects are of concern, a reversible functional effect, although undesirable, would be of vastly less consequence to man than the development of an irreversible functional effect. In addition, most human exposures to environmental pollutants are typically long-term exposures to low ambient concentrations, and, therefore, chronic functional effects may be the most widespread consequence of exposure to these compounds.

Two considerations necessary for evaluating the seriousness of an induced toxic response are the potential for tissue regeneration and the degree of redundancy. Many cells in the body are essentially in final form (i.e., differentiated cells that cannot divide and be replaced), and in limited supply. Chemical exposure that destroys these cells presents serious consequences. Perhaps the most well known example is the heart. In contrast, other types of cells undergo continual replacement or have a capacity to regenerate (e.g., skin, blood, liver), and have a chance of complete recovery. The second consideration that determines the seriousness of chemical-induced damage is the degree of redundancy in a particular organ. For example, the kidneys have sufficient functional reserve to provide entirely adequate function, even if 50% or more of renal function is lost (249).

The ideal data for assessing the significance of a chemical as a cause of chronic human disorders would be the results of chronic administration of measured amounts of pure chemical to human subjects by the appropriate route. Since these data are not likely to be available, one must consider whatever human data are available, data from laboratory animals, and, when there are no relevant data for the chemical of interest itself, data for similar chemicals.

Available human data generally consist of acute, accidental exposure data, which are not necessarily predictive of the potential for chronic functional disorders; epidemiologic studies which often serve to corroborate the findings of more specific animal or human work, but only infrequently define cause and effect relationships; and data from occupational exposures which often are chronic and the chemical agent may be well identified. These data need to be carefully scrutinized, however, since the occupational history of any individual may include many different exposures and other predisposing factors.

Thus, frequently the available data for humans for establishing the potential for a chemical to cause chronic effects are very much less useful than would be expected. They provide descriptive clues suggesting critical organ systems but are insufficient to characterize quantitatively the relationship between exposure and effect. Often,

animal data are the only source of information available for assessing potential for chronic human impairment. A number of problems are associated with the use of animal data to predict human health effects (see the following section) but results from chronic animal studies are the best tools currently available to assess the hazards associated with chronic exposure to various toxicants.

### 1.2.4 Sampling and Analysis

Evaluation of the extent of possible environmental contamination in the soil/ground-water system requires the collection of environmental samples and analysis for the presence of specific pollutants. A successful analytical program involves the following fundamental steps:

- collection of a representative sample
- preparation of the sample for analysis and sample introduction
- separation of the analytes from other interfering constituents
- identification and quantification of the analyte(s)
- calculation of the results, including an estimate of the precision, accuracy, and minimum detection limits (MDL) associated with the analysis

Several field samples may be collected and composited to produce a representative sample. Prior to removal of an aliquot of the sample for preparation and analysis, the field samples should be homogenized. Aqueous samples should be shaken and solid samples should be mixed.

Sample collection and preservation procedures designed to protect against loss of sample components are often required. For example, airtight sample containers to prevent volatilization losses should be used for volatile components; appropriate sample containers or chemical preservation techniques are also recommended in order to guard against adsorption or other reactions of the components in the sample containers.

Most methods for the analysis of complex environmental mixtures require that the analyte be extracted from the mixture and introduced into the appropriate separation scheme. For the analysis of highly volatile organic analytes, the sample may be purged with inert gas; the analyte is then trapped on a sorbent and ultimately desorbed. Other techniques include headspace analysis or direct injection. Compounds with lower volatility may be extracted from the environmental sample using an appropriate organic solvent. Inorganic analytes are often prepared for analysis by acid digestion or other similar techniques.

Separation and identification of specific components in complex environmental mixtures are often complicated procedures governed by the nature of the sample and the nature of the analyte, as well as the nature of the data required. The USEPA has developed several sets of methods specific to particular environmental matrices, or particular types of analytes. They describe in detail all the sampling and analysis steps. For example, USEPA publication, Test Methods for Evaluating Solid Waste (63) goes into detail in summarizing the methods appropriate for specific analyte classes.

One of the most widely used inorganic analytical procedures is atomic absorption (AA) spectroscopy. For organic species the most common analytical procedures involve separation by use of gas chromatography (GC) or high performance liquid chromatography (HPLC). Detection follows with a mass spectrometer (MS) or another detector appropriate to the specific class of compounds, such as electron capture detector (ECD), photo ionization detector (PID) or flame ionization detector (FID).

For any analytical technique selected or required, it is imperative to be able to estimate the precision, accuracy and method detection limits. A quality assurance plan that includes collection of duplicate samples, field blanks, matrix spikes and other performance evaluation samples should be followed. Established methods recommended by regulatory agencies generally provide guidance on the precision, accuracy and detection limits achievable for a given pollutant analyzed in accordance with the method. However, these are frequently highly dependent on the matrix type and may not apply to complex or intractable samples.

A brief sampling and analysis section was included for each of the chemicals in the Guide. Many of these chemicals have been recognized by the USEPA as chemicals of environmental interest for some time; soil and water sampling and analysis procedures are generally well documented. The primary objective of the sampling and analysis section was to identify the specific methods required or recommended by regulatory agencies so that the field engineer could then coordinate the regulatory requirements with USAF guidelines. Since sampling and analysis instructions are available to bioenvironmental engineering personnel (i.e., USAF OEHL Recommended Sampling Procedures) or are proscribed by the USAF OEHL to IRP Phase II contractors, the recommended methods were identified but were not described in detail.

Sampling considerations that are unique to a specific chemical were presented (e.g., collection of samples in airtight containers with no headspace for volatile compounds). The USEPA-approved procedures for analysis of the chemical in soil/waste samples and aqueous samples were reviewed. Generally, the sample preparation procedures for solid and aqueous samples were different while the recommended analytical separation technique (e.g., HPLC, GC) and the detector (e.g., MS, PID, ECD) were similar for both matrix types. Typical detection limits achievable with the recommended procedures were also presented when available.

## 1.3 ASPECTS OF RISK ASSESSMENT/RISK MANAGEMENT IMPLEMENTATION

Quantitative analysis of human health risks has become increasingly important as a means of supporting decisions that affect public health. Despite the many uncertainties that can arise, risk analysis is frequently used to estimate the impacts of chemicals or products on exposed populations, to guide policy-making and priority setting, and to support the development of government regulations. Although risk analysis is a complex endeavor, the following discussion attempts to describe the general framework for performing risk analysis, the important components of risk, some of the practical issues that arise in implementing a risk analysis and how the uncertainties and assumptions in risk assessment affect risk management.

Risk, in the context of assessing hazard from environmental pollutants, may be defined as the potential for negative consequences of release of a pollutant into and its subsequent traverse through the environment such that humans are impacted. The purpose of risk analysis is to go beyond a qualitative statement of potential risk by estimating or measuring this potential.

There are two necessary components of all risk statements, namely, a component expressing the best estimate of the actual risk (i.e., the probability or severity of a response at dose X) and a second component expressing the degree of confidence one can have in the best estimate. To illustrate, one can state the probabilities that certain fractions of the population will be adversely affected (e.g., 5% chance that 9/10 will be affected, 20% chance that 1/3 will be affected). This sort of quantitative estimate is usually difficult to achieve. Alternatively, one can state the expected number that may be affected, allowing a certain margin for error to reflect uncertainties in the underlying data (e.g., 200,000 +/- 50,000). Finally one can give an order-of-magnitude estimate that has no real measure of confidence attached to it (e.g., at most, 10% will be affected).

An evaluation of the risks of exposure to a chemical will usually consist of more than one result; it will describe the spectrum of risks identified in a variety of different cases characterized by features such as:

- Nature of the adverse effect
- Subpopulations affected
- Temporal aspects (e.g., frequency)

Often different receptor populations will be exposed in different ways over differing periods of time, and will experience different effects as a result. The spectrum of such risks must, therefore, be described to the extent permitted by the available data on exposure and effects. For some chemicals, these data may not be sufficient for quantitative estimates, and consequently the risk assessment may be only qualitative.

### 1.3.1 Specific Approaches to Risk Assessment

Two general types of risk statements are recognized. The first is based on the assumption of an underlying fixed relationship between dose and response which can be represented by one or more mathematical models. The second is not based on a presumed mathematical relationship but does imply the concept of thresholds of effects. Examples of the second type of risk assessment are the margin of safety calculation and use of safety factors.

The first type of statement can be utilized for acute toxicity data and often can be relatively accurate in predicting the levels of exposure which are acutely lethal, narcotic, or irritating to lungs, eyes or skin, for example. The more important use of mathematical models for predicting risk comes into play when dealing with carcinogenicity data.

As stated above, estimates of actual risk (i.e., the probability of severity of a response at dose "d") are based on the assumption of an underlying fixed relationship between dose and response. The shape ascribed to this dose-response curve is an issue of some controversy, particularly when extrapolating from high to low doses. Dose-response relationships can be represented by one or more mathematical models. In general, three models are most frequently used:

- the linear "one-hit" model
- the log-probit model, and
- the multistage model.

The "one-hit" model envisions a single molecule of pollutant invading a single cell as the sole precipitating event for tumor induction (500). In other words, there is no threshold; rather, there is some finite response at any dose or exposure level when dose>0. This reasoning leads to a linear dose-response curve over the entire dosage range of interest. The denial of thresholds tends to make the predictions obtained "conservative," meaning that they overstate the risk.

A rival hypothesis is offered by the log-probit model. This extrapolation assumes that human susceptibility to a pollutant is normally distributed with the logarithm of dose (501). This assumption has little theoretical basis, though some physiological

variables do seem to follow this log-normal distribution. This model usually yields much lower estimates of risk for typical human exposure levels because of the S-shape of the dose-response curve and the implied threshold effect of the shallow, concave-upward approach to the origin with decreasing dose.

The multistage model (502) is actually a generalization of the "one-hit" model, in which the hazard rate is taken to be a polynomial rather than a linear function of dose. The assumption of the multistage model is that a number of events may be needed to generate a critical mass of cells whose growth will outrun any homeostatic defense mechanisms and result in a tumor. The linearized multistage model proposed by Crump et al. (503) and used by many of the regulatory agencies, replaces the linear term of the polynomial function by its upper 95 percent confidence limit to reflect biological variability in the observed tumor frequencies. The dose-response predicted by this model is approximately linear at low doses, giving estimates of potential risk that are almost identical to those of the "one-hit" model. The multistage model generally gives dose-response estimates intermediate to the "one-hit" and log-probit models and appears to agree best with known biological phenomena.

Graphic representation of the extrapolated low-dose region of interest for these three models for two rat feeding studies are presented in Figure 5. The data represent the bladder tumor response in rats fed sodium saccharin in their diets for two years and the liver tumor response for another group of rats fed aflatoxin in their diets for two years. As seen in Figure 5, the one-hit model leads to the most conservative estimates of risk, followed by the multistage and log-probit models, respectively. One can clearly see that the estimates of added risk over background at low doses may be notably different, depending on the model selected for extrapolation.

There is presently no scientific consensus as to which model is most appropriate for specific health effects. However, the Interagency Regulatory Liaison Group of the Federal Government recommends the linearized multistage model for extrapolation of cancer risks, and regulatory decisions are based on the results of this model.

As briefly described above, a second type of risk statement as typified by the margin of safety estimate, generally presumes a threshold level for a particular effect. Thus, if the anticipated or reported exposures are well below this threshold, one can express the risk as the ratio of the exposure level to the threshold level. This ratio is called the margin of safety. The threshold is approximated by the highest reported No-Observed-Adverse-Effect Level (NOAEL) or the Lowest-Observed-Adverse-Effect Level (LOAEL), whichever is smaller. The reliability of the margin of safety really depends on the availability of a comprehensive toxicity spectrum so that one can at the very least discuss the ranges of nontoxic and toxic dose levels. The USEPA uses margins of safety to develop Health Advisories (HAs)(formerly termed Suggested--No-Adverse-Response Levels or SNARLs) and water quality criteria for chemicals that are not carcinogens. The margin of safety is not used if the chemical has been

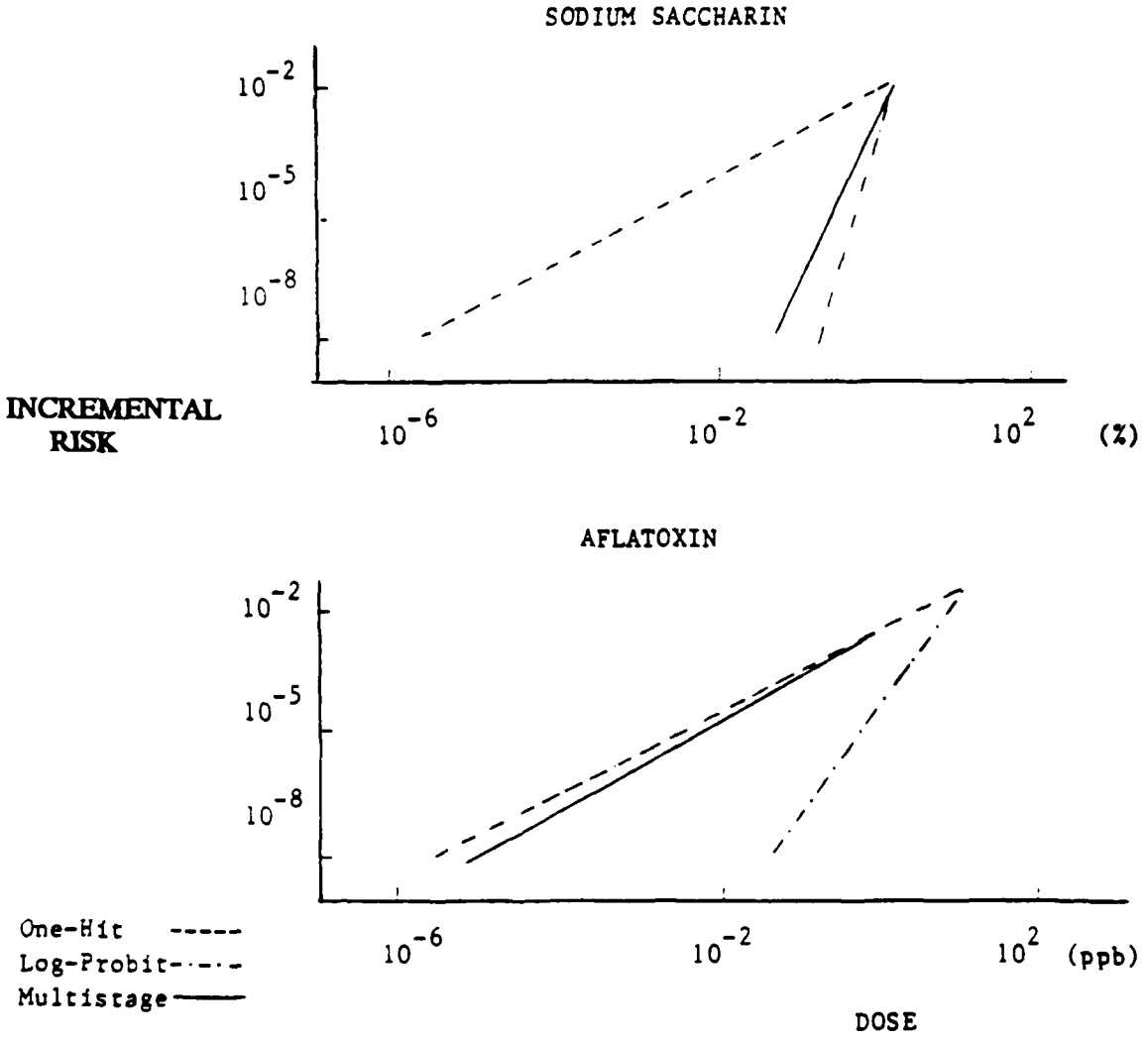



FIGURE 5

ESTIMATES OF INCREMENTAL RISK OVER BACKGROUND BASED ON THREE EXTRAPOLATION MODELS

determined to be a known carcinogen, since no threshold for carcinogenicity is presumed.

Another term frequently used to establish the magnitude of risk associated with ingestion or inhalation of an agent is the ADI (acceptable daily intake). The USEPA presently uses the term reference dose (RfD) and no longer uses ADI. The RfD is an empirically derived value that reflects a particular combination of knowledge and uncertainty concerning the relative safety of a chemical. The ADI concept is not applicable to heavy metals and lipophilic substances which tend to accumulate in body tissues. Uncertainty factors (alternatively known as safety factors) used to calculate RfD values (NOAEL/U.F. = RfD) represent the level of confidence that can be justified on the basis of the available toxicological data. The USEPA has identified five potential areas of uncertainty:

- A 10-fold factor is used to account for the variation in sensitivity among the members of the human population.
- A 10-fold factor is used when extrapolating from the results of experimental animals to humans.
- A 10-fold factor is generally used when extrapolating from the results of subchronic experiments in animals or humans to a chronic exposure situation.
- A 10-fold factor is generally used when deriving an RfD from a LOAEL, instead of a NOAEL.
- A 10-fold factor is generally used when extrapolating from valid results in experimental animals when the toxicity data base is "incomplete", i.e., to account for the inability of any single study to adequately address all possible adverse outcomes.

The USEPA has proposed guidelines for using the uncertainty factors such that a study where four of the five areas of uncertainty exist will not have a composite uncertainty factor greater than 3,000 and when all five areas of uncertainty exist, no greater than 10,000. A hypothetical example of a RFD calculation is as follows:

Three doses of Compound Y were administered to mice via the diet for 90 days. Analysis of the data indicated:

| Dose (ppm in diet) | Effect |
|---|---|
| 100 | Death |
| 10 | No effect |
| 1 | No effect |
| 0 | -- |

The highest NOAEL for this particular study was 10 ppm in the diet. For the purposes of this calculation, mice were assumed to ingest 3 g of feed per day and to weigh 0.025 kg. Therefore, at a dietary concentration of 10 ppm, a mouse would ingest 1.2 mg of Compound Y/kg bw/day (see Appendix 3 for calculation). The uncertainty factor would be 1000, i.e., 10 for sensitive humans, 10 for animals to humans, and 10 for subchronic to chronic exposure. It is assumed that the toxicity data base for compound Y is complete.

Thus,

$$\text{RFD for man} = \frac{1.2 \text{ mg/kg bw/day}}{1000 \text{ (uncertainty factor)}} = 0.0012 \text{ mg/kg/day}$$

### 1.3.2 Uncertainties and Assumptions of Risk Assessment Processes

In performing a risk analysis, a number of difficulties in terms of both data inadequacies and complexity of biological mechanisms come into play. While these difficulties can be overcome by using simplifying assumptions, subjective judgments and mathematical models, such techniques inevitably introduce uncertainties into the analysis.

Expressions of the quantitative relationships between human health effects and exposure to a particular pollutant can be derived from epidemiological studies of human populations or from controlled toxicological studies of animals. Animal experiments are hampered by uncertainty in species differences as well as the difficulty of extrapolating responses to genetically heterogeneous human populations. Epidemiological studies, while they provide a more reliable basis for determining the effects of a pollutant in humans, are limited by uncertainty in dose due to imperfect knowledge of food, air or water concentrations and intake rates. In addition, only small test populations are generally available for study. This means that a response can easily be missed if studies are conducted at low-dose levels, corresponding to typical ambient concentrations in the environment. Instead, a practice has evolved to perform such experiments with a moderate number of animals at high dose levels and then to extrapolate the results to the low exposure levels. Since ethical considera-

tions preclude controlled human experiments, society is usually faced with the need to extrapolate from animal tests and modify these with whatever human data are available.

The extrapolation procedures required to meet the objectives of risk analysis involve at least two difficult steps - (1) relating the risk in test animals at very high doses to doses very near zero, and (2) relating human risk to that in animals. The first step is overcome by selecting some mathematical method for extrapolating high-dose results with a few animals to low-dose expectations such as those discussed above. The other problem, that of animal-to-human extrapolation, is the subject of considerable debate.

One issue with species-to-species extrapolations involves the type of data that should be considered and the validity of the results. The derivation of a human dose-response curve (a relationship between the amount or rate of chemical administered and the probability of the subject experiencing an adverse effect at that dose) from animal data is predicated on the assumption that a pollutant with demonstrated toxicity in certain laboratory animals has a probable analogous effect upon man. However, the toxic effects of many substances appear to be species dependent, because of different metabolic patterns, and may vary even among strains of the same species, or for different sexes and ages. Ideally, the toxicity for man should be verified through epidemiologic studies in situations where the pollutant was known to be present. Even if the substance is indeed toxic to man, the issue remains of how to estimate the relative potency of the substance in man as compared with animals. The common practice is to use body weight or body surface area to normalize the dose levels between different species. The rationale for the first approach is that, since total body concentrations govern the toxic effects, fixing the dose of material per kilogram body weight establishes a constant level of risk. The second approach focuses on dynamic rather than static conditions. It is predicated on the principle that the rate of entry into the total body governs the intensity of the toxic effect, and that this influx rate is proportional to the (2/3)-power of weight or body surface area. Whether surface area or body weight is a more appropriate normalization factor is open to debate. The choice of method introduces an uncertainty of roughly an order of magnitude into the risk estimates.

Selecting the appropriate animal data, expressed in terms of a dose-response, for extrapolation is a complex judgmental process. Numerous factors must be considered, such as the adequacy and quality of the effects data available for extrapolation, the appropriateness of the species and strain, route, doses and rate of administration, study duration, sufficient number and adequate survival of test animals, the quality and extent of gross and microscopic pathology, and the presence of intervening diseases unrelated to treatment that might reduce survival or increase sensitivity. Other issues to be considered include similarity of metabolism, bioaccumulation, and excretion of the test material and its pharmacokinetics within the laboratory animal and man. One must also reconcile the life span of the animal and its stages of development with those of man.

Another point of controversy is the existence of a threshold for carcinogenic and mutagenic responses to a toxicant. Some argue that an organism is able to cope with low doses of a toxicant through metabolic processes or repair mechanisms, so that harmful effects do not appear until a certain minimum threshold, or "safe dose," has been surpassed. Evidence suggests that for many types of substances, metabolic pathways at high-dose levels differ from those at low-dose levels, and this raises questions about the validity of linear extrapolation models. Others contend that a toxic substance must be considered potentially harmful at any dose, and that a "zero tolerance" level should be assumed. This issue has often been circumvented by the approach of selecting an "acceptable" risk level and determining the corresponding "acceptable" dose.

### 1.3.3 Risk Management

In contrast to risk assessment which seeks to arrive at a probabilistic evaluation of some adverse outcome for an individual or a population, risk management is the process of weighing alternative actions where risk has been determined to exist as well as deciding about the degree of risk to be tolerated. The overall approach to health risk management is given in Figure 6.

Risk depends on the conditions under which one encounters a hazard. Due to the complexities and variability of circumstances, determining the extent of remedy or whether a certain level of chemical should give rise for concern has to be decided on a case-by-case, site-specific basis. Each site has unique characteristics that merit individual attention and incorporation within risk assessment processes. The site-specific topics that need to be addressed include:

- a description of the site and its location in relation to the surrounding population
- determination whether the contaminants are exposed or contained
- environmental concentrations and the dynamics of contaminants
- water and subsoil conditions at the site
- degree of access to the contaminated area
- the type and duration of activities conducted in the area
- exposed population (e.g., children, adults, animals used to produce food for humans)

- the probability of various types of injury likely (e.g., relatively brief exposure at the site could lead to chemical burns, lung or eye irritation vs. chronic health effects associated with exposure via drinking water)

Site-specific answers are needed to characterize actual risk and identify possible intervention options to reduce the risk. Risk management comes into play in deciding which intervention options are utilized at a particular site and what level of risk is acceptable. Clearly, there are many risks that each of us individually and in society as a whole, accept or consider to be acceptable (e.g., driving to work, smoking, generating electricity from coal). Though some of these risks are very small, not one of these risks is zero. For example, the risk associated with automobile accidents is about 50,000 accidental deaths per 230 millon people or an annual risk of about 2.2E-04 (or 1 in 4500). Risk management decisions regarding environmental health hazards from a particular site need to be viewed in this perspective of acceptability of risk as well.

In the practice of human health risk assessment, there are a number of expressions of risk which are frequently used and occasionally misunderstood. The most commonly used expression is the "unit risk" coefficient. If the unit risk of cancer is given as 0.0003 per mg/day and exposure is 0.2 mg/day, then an individual's excess lifetime risk above normal background levels of cancer can be expressed as exposure (0.2) times unit risk (0.0003), or 6E-05. If the size of the exposed population is known, cumulative risk may be calculated. For example, if the number of people exposed at the level noted above is two million, then the cumulative risk turns out to be 120. Put in another way, the estimated excess incidence of cancer in the exposed population is 120 cases out of two million people over their lifetime.

## 1.4 U.S. REGULATORY STATUS

Various federal statutes provide the framework for minimizing the amount of toxicants permitted in our food, drinking water, ambient air and in the workplace. Criteria and standards developed to protect human health and the environment are administered by several agencies, primarily the U.S. Environmental Protection Agency (USEPA), the Food and Drug Administration (FDA), the Occupational Safety and Health Administration (OSHA) and the Department of Transportation (DOT). The major statutes applicable to IRP chemicals are discussed below; statutes and permissible levels for particular IRP chemicals are itemized in the regulatory section of each chapter.

State water regulations for ground, surface, and drinking water were reviewed for all fifty states. State air and hazardous waste regulations were not reviewed. Most states have proposed or promulgated regulations establishing water quality criteria

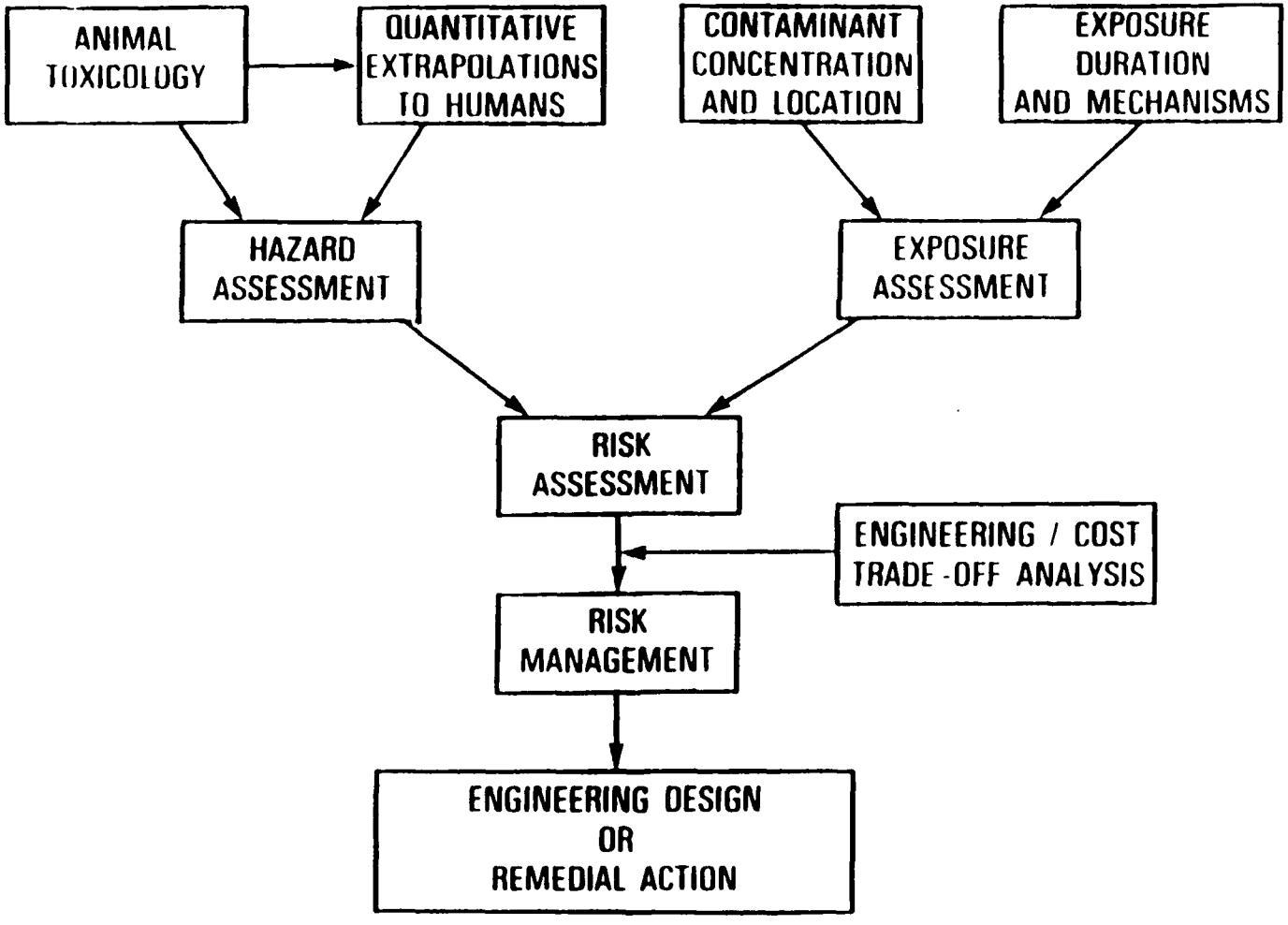



FIGURE 6

QUANTATITIVE HEALTH RISK MANAGEMENT

which are identical to the federal Ambient Water Quality Criteria and the National Primary Drinking Water Regulations. The federal numeric criteria, selected by the individual states as their state criteria, varies. For example, one state may set their groundwater quality standard to match the federal freshwater chronic toxicity standard for the protection of aquatic life while another state may set theirs to match the federal water and fish ingestion standard for the protection of human health. The state standards are itemized in the regulatory section for each chemical only when they are different from any federal criteria. If the standards are not itemized for a particular state, the assumption can be made that the standards for that state are identical to one of the federal criteria (3683, 3684).

Most states are in the process of proposing revisions to their regulations which will make them identical to USEPA's proposed regulations when they become final. These changes are projected for 1989-90. The state regulations listed in this guide are current as of March 1, 1989. Becuase the state regulations are changing rapidly, though, contact with state officers is advised to determine the current status of the standards. A current listing of state officers/contacts is included with this document (Appendix 4).

### 1.4.1 Clean Water Act (CWA)

The primary objective of the Clean Water Act of 1977 is the reduction and control of the discharge of pollutants into the nation's navigable waters. Under the terms of the Act, navigable waters are defined as "waters of the United States." While the major focus of the CWA has traditionally been on surface water, it does offer limited statutory authority to implement protection measures for ground water (350).

As part of the overall program to prevent and eliminate sources of water pollution, Section 104 of the Act requires USEPA to establish, equip and maintain a water quality surveillance system for the purpose of monitoring navigable waters and ground waters. At the present time, this system has been implemented only to a limited extent.

Section 208 deals with the development and implementation of area-wide plans for waste treatment management. It was enacted to eliminate water quality control problems in urban and industrial areas. Under this Section, the USEPA, after consultation with state and local authorities, is required to publish guidelines to identify areas with significant water quality problems. The state must then develop plans that identify the sources of pollution and how they will be controlled as well as the type of treatment facility needed. The programs proposed under this section have received lower priority and achieved more limited results.

Under Section 303, the states are required to establish water quality standards, the purpose of which are to protect public health and enhance water quality. A water quality standard consists of two parts: (1) a "designated use" for which the

water is to be protected, such as "recreation" or "agriculture" and (2) "criteria" which are numerical pollutant concentration limits or narrative statements which define how to preserve or achieve the designated use (355). State criteria are usually based on the ambient water quality criteria developed by USEPA as required under Section 304(a) for all pollutants designated as toxic (i.e., priority) under Section 307. These water quality criteria reflect the latest scientific knowledge on the relationship between pollutant concentrations and environmental and human health effects. They do not take into consideration the costs, treatment technology or other feasibility factors.

USEPA issues its ambient water quality criteria periodically as a guidance to the states. Two types of criteria are calculated: one to protect aquatic life and the other to protect human health. The water quality criteria have no direct regulatory impact. Moreover, in that human health criteria include provisions for consumption of both water and aquatic organisms, they are not directly applicable to ground water. However, the CWA contains no clear statement that the criteria developed under Section 304 (a) should only apply to surface waters. Indeed, Section 304 (a) states that USEPA should examine adverse effects "...from the presence of pollutants in any body of water, including ground water." Historically, implementation of the provisions of the CWA has been focused on surface waters. By tradition, however, EPA ambient water quality criteria have essentially become the "standards" by which the quality and safety of all sources of public drinking water supplies are judged.

The USEPA criteria may be adopted as standards by individual states. State water quality standards must be approved by USEPA before they can be implemented, but once implemented, they are legally enforceable. If USEPA disapproves of a state's standards, it may revise or promulgate new standards for that state in order to meet the requirements of the Act.

Other regulations established under the CWA are the toxic pollutant effluent standards and effluent limitations. These are established for pollutants designated as toxic (i.e., priority pollutants) under Section 307 of the Act. Toxic pollutant effluent standards apply to owners or operators of specified facilities discharging into navigable waters. Currently, there are standards for aldrin, dieldrin, DDT, PCBs, endrin, toxaphene and benzidine (805). Effluent limitations identify the degree of effluent reduction attainable through the application of varying levels of pollution control technology applicable to any point source. Effluent limitations have been set for a number of point source categories, including the following: electroplating; organic chemicals, plastics, and synthetic fibers; steam and electric power generating; metal finishing; iron and steel manufacturing; metal molding and casting; pesticide chemicals; non-ferrous metals manufacturing; petroleum refining; ferroalloy manufacturing; pulp, paper and paperboard; glass manufacturing; timber products processing; and textile mills.

Section 402 of the Act established the National Pollutant Discharge Elimination System (NPDES) which allows USEPA or the states to issue permits for the dis-

charge of any pollutant or combination of pollutants if certain specified conditions are met. These permits require compliance with certain standards in order to release certain types of industrial and municipal wastes into the nation's waters. NPDES is the principal mechanism for enforcing measures to reduce and control the discharge of pollutants into surface waters (349). Thirty-nine states currently have enforcement authority for their USEPA-approved NPDES programs.

Under Section 311, numerous compounds have been designated hazardous substances and reportable quantities (RQ's) have been assigned (347, 348). RQ levels are set to control short-term, non-routine discharges of hazardous substances. They range from 0.454 to 2270 kg. As of July 3, 1985, CWA RQs are to be the same as those issued under CERCLA (556). Any person who discharges a hazardous substance equal to or exceeding its RQ in any 24-hour period must report it to the appropriate government agency. Penalties will also be assessed. Discharges of mixtures and solutions are subject to the regulations only if a component hazardous substance is discharged in a quantity equal to or greater than its RQ. This regulation does not apply to discharges made in compliance with a NPDES permit.

In addition, the Act contains an imminent hazard provision authorizing USEPA to take any necessary action to restrain persons or activities allegedly causing the discharge of pollutants (346).

### 1.4.2 Safe Drinking Water Act (SDWA)

The Safe Drinking Water Act was enacted in 1974 to provide for sanitary drinking water supplies and to establish a program to control underground injection order to prevent endangerment of subsurface waters.

An important feature of the Act is the establishment of primary and secondary drinking water regulations which apply to public water systems in each state. The National Revised Primary Drinking Water Regulations specify the maximum permissible levels of contamination that may be present in drinking water and are based on consideration of health effects and economic feasibility. These maximum contaminant levels (MCLs) have been established for bacteriological, microbiological and radiological quality; and for specific organic and inorganic chemical contaminants. The regulations also specify monitoring frequencies and methods, and Maximum Contaminant Level Goals (MCLGs) which are non-enforceable health goals. All MCLs are legally enforcable standards. The National Secondary Drinking Water Regulations set a maximum level for contaminants that primarily affect the aesthetic qualities relating to public acceptance of drinking water. The secondary regulations are not federally enforceable but are intended as guidelines for the states. Primary and secondary drinking water standards have been set for some of the contaminants now found in surface waters and ground water. Standards for many of the remaining contaminants, including radionuclides, disinfectants, and about 70 organic and inorganic contaminants, are scheduled to be set by 1992.

In the absence of formal drinking water standards, the Office of Drinking Water within the USEPA has developed Health Advisories (HAs), formerly termed Suggested- No-Adverse-Response Levels (SNARLs) on various pollutants. These are not legally enforceable standards, although they may lead to the issuance of MCLs. Where data exist, HAs are issued for 1-day, 10-day and long-term exposure. The calculations do not take into account possible carcinogenic risk. In addition, they do not consider the health risks from possible synergistic effects of other chemicals in drinking water, food and air.

USEPA is authorized to give the states primary enforcement responsibility for assuring compliance with primary and secondary regulations. In order to obtain such authorization, the state must adopt drinking water standards in conformance with federal standards and adopt implementing procedures for their state-wide enforcement.

The Underground Injection Control (UIC) Program was established to prevent ground water contamination. Under this program, underground injection of hazardous wastes (329) is prohibited without an authorized state permit. To obtain a permit, the applicant must satisfy the state that the underground injection will not endanger drinking water sources. Inspection, monitoring, recordkeeping and reporting requirements are also imposed under the program. The act enables states to have primary enforcement responsibility for UIC programs. All states which are designated by USEPA to adopt such programs, must do so. These state programs must meet the requirements specified by USEPA. Should a state fail to ensure program enforcement, the USEPA is authorized to take over the program.

The USEPA is also authorized to take special measures to protect areas which have only one aquifer as their principal source of drinking water. No new underground injection wells can be drilled in these areas without a permit wherein the applicant must prove that underground injection will not contaminate the aquifer.

In order to protect drinking water supplies, the Act contains an imminent hazard provision which allows USEPA to institute a civil action when it receives information that a contaminant is present in the water supply which may endanger public health. This authority, however, may only be exercised if the state and local authorities have failed to act (293, 294).

### 1.4.3 Resource Conservation and Recovery Act (RCRA)

The Resource Conservation and Recovery Act of 1976 amends the Solid Waste Disposal Act as originally adopted in 1965. RCRA provides planning and management/guidelines for the treatment, storage and disposal of hazardous wastes (324).

The most relevant features are Subtitle C which governs hazardous waste management and Subtitle D which covers municipal solid-waste disposal. Under Section 1004, any material which is abandoned by being disposed of, burned, or

incinerated - or stored, treated or accumulated before, or in lieu of, these activities - is a solid waste. A recycled material may or may not be a hazardous waste depending on what the material is and how it is recycled (332). Among the wastes excluded are those contained in domestic sewage and irrigation return flows. A hazardous waste is defined as solid waste(s), which because of its quantity, concentration or characteristics may present risks or harm to human health or environment. The wastes specifically excluded include household wastes and wastes generated from the combustion of fossil fuels.

Under the solid waste disposal program of Subtitle D, states are required to develop plans for the management of solid wastes and the conservation of resources. The plans must be approved by USEPA and are subject to continuous review and revision. The key to the solid waste program is the classification of solid waste disposal facilities as either sanitary landfills or open dumps. USEPA has established eight criteria to be used by states to classify solid waste facilities. These criteria address environmental concerns. States use these guidelines to classify their solid waste disposal facilities and take action to close or upgrade any that are classified as open dumps.

Under the hazardous waste management program of Subtitle C, USEPA is required to establish standards for all hazardous waste management facilities to protect human health and the environment. The standards encompass generation, treatment, transportation, storage and disposal. Restrictions on land disposal of identified hazardous wastes are listed in 40CFR168. Absolute prohibition on land disposal of hazardous waste of any kind for which USEPA has not established a treatment standard takes effect on May 8, 1990. All hazardous wastes must be treated before disposal according to Best Demonstrated Available Technology (BDAT) treatment standards as specified by USEPA. Certain variances exist until May, 1990 for some hazardous wastes for which BDAT treatment standards have not been promulgated by USEPA. The treatment standards are the basis for issuing permits to new facilities. The regulations are applicable to containers, tanks, surface impoundments, waste piles, incinerators, land treatment facilities and landfills. Ongoing operations of other existing facilities will become subject to these standards only as USEPA and authorized state agencies process their permit applications and bring them under these controls. Pending final action on their permit applications, these facilities operate under interim state standards.

USEPA may authorize states to administer their own hazardous waste disposal programs. For authorization to be granted, the state program must be substantially equivalent to the federal program. Currently, almost half of the states have USEPA--authorized state programs.

The Act also contains an imminent hazard provision which allows USEPA to bring court action against any handler of solid or hazardous wastes who is presenting a danger to human health or the environment.

Numerous IRP chemicals have been designated hazardous wastes. For each of these IRP chemicals, there is an exclusion available for those who generate less than 1000 kg per month.

### 1.4.4 Toxic Substances Control Act (TSCA)

TSCA was enacted in 1976 to regulate the manufacture, use and disposal of chemical substances and mixtures that pose a significant risk of injury to health and the environment. It requires extensive testing, notification, labeling and recordkeeping. Under TSCA, USEPA has the authority to prohibit or limit the manufacturing, processing, distribution and use of chemicals. USEPA may also prohibit or regulate the manner or method of chemical disposal or the disposal of any chemical-containing article. This authority extends to any person who uses or disposes of a chemical substance for commercial purposes as well as the manufacturer and processor. Under Section 7, USEPA has powers to commence civil action to seize substances or mixtures which are imminently hazardous (i.e., pose immediate threat of serious or widespread injury to health or environment) (333).

Numerous IRP chemicals are subject to the reporting requirements in Section 8 of TSCA. This section requires those who manufacture, distribute or process a chemical substance or mixture regulated by TSCA and those who propose to do so to maintain and submit to USEPA records that include name, composition, amount to be processed or manufactured and its manner of disposal. In addition, these individuals are required to submit health and safety studies to USEPA. Others in possession of such studies must also submit them (334, 335).

Within the terms of TSCA, specific regulations exist for asbestos, fully halogenated chlorofluoroalkanes and polychlorinated biphenyls (333).

### 1.4.5 Comprehensive Environmental Response Compensation and Liability Act (CERCLA)

CERCLA was enacted in 1980 to allow the federal government to respond immediately to the release or threatened release of hazardous substances into the environment which may present an imminent and substantial danger to public health. The Act gives the federal government authority to remove the hazardous substance and provide remedial measures. Hazardous substances under the terms of CERCLA are those designated as such under the Clean Water Act as well as those identified as hazardous wastes (under RCRA), hazardous air pollutants (under CAA), toxic pollutants (under CWA) and hazardous chemical substances (under TSCA). USEPA has assigned reportable quantities (RQs) for these newly designated hazardous substances and has adjusted the RQs previously assigned under CWA (556). It has also assigned RQs for RCRA hazardous waste streams. In cases where the waste stream is composed of more than 1 chemical, the RQ for the waste stream depends on the concentrations of the chemicals which are present (556).

Environmental Protection Agency Program

The following information does not directly apply to DOD contaminated site remediation activities. It is included to contrast the USEPA program to the parallel DOD program. Furthermore, at third party sites, where a DOD agency is one of many parties responsible for site clean-up, that agency is often involved in the USEPA program procedures and associated litigation.

The removal and remedial activities authorized by the Act are carried out under the terms of a National Contingency Plan which is financed by a $1.6 billion Hazardous Substance Response Trust Fund commonly known as "Superfund." Federal agencies are not authorized to use this fund. For the DOD, resources are provided through a separate Defense Environmental Restoration Account (DERA).

The National Contingency Plan (NCP) ensures an effective response to CERCLA and CWA. The response plan for hazardous substances has been delineated into 7 phases:

I Discovery and Notification
II Preliminary Assessment
III Immediate Removal
IV Evaluation and Determination of Appropriate Response
V Planned Removal
VI Remedial Action
VII Documentation and Cost Recovery

Revisions have been proposed to streamline the National Contingency Plan, and are expected to be promulgated by January, 1990.

In cases where the responsibility for the wastes causing contamination can be traced to companies with financial resources, the government may call upon those companies to undertake the clean-up at their own cost. If such companies refuse, the government may carry out the remedial program using money from the fund and then bring suit against the companies for reimbursement.

Also under the NCP, the USEPA has developed a Hazard Ranking System (HRS). The HRS is the foundation for the National Priority Lists (NPL), a list of top priority response sites, which is revised and updated annually. The HRS attempts to take into account potential risk to the population and ecosystems as well as the potential for contamination of drinking water supplies. With these considerations in mind, USEPA arrived at a mathematical score for ranking of the priority list. A similar but separate ranking system known as the Hazard Assessment Rating Methodology (HARM) is used in the Air Force to prioritize its site investigations.

Section 106 of the Act gives USEPA a broad imminent hazard authority. These powers include securing judicial relief and the issuance of administrative orders to protect public health, welfare and the environment.

CERCLA also contains reporting requirements and stringent liability standards for the release or threatened release of hazardous substances (342).

## Comparison of Air Force IRP with the USEPA "Superfund" Program

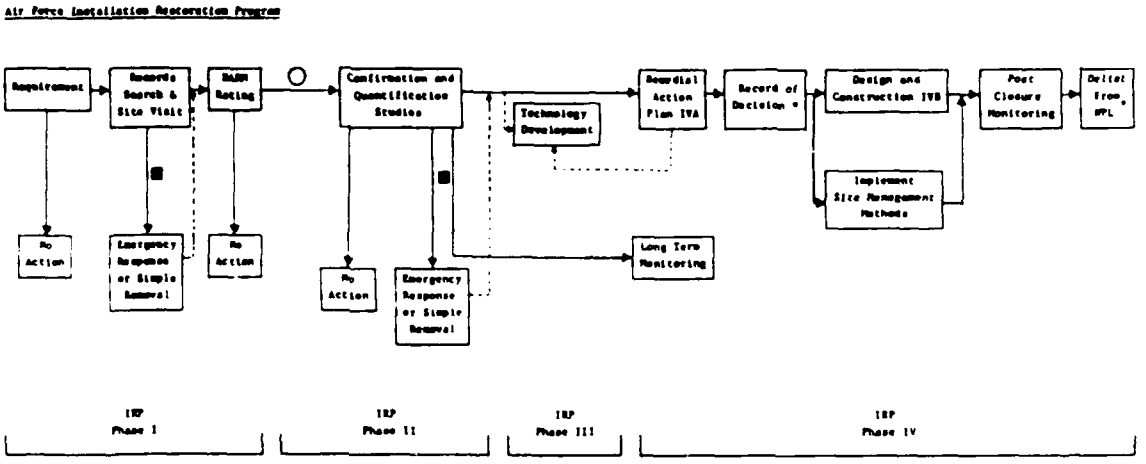
Executive Order 12316, August 14, 1981, delegates the responsibility for response action at DOD facilities to the Secretary of Defense. These actions must be consistent with the NCP described above; Figure 7 contrasts the NCP sequence of steps to the Air Force four phase sequence. Generally, the program sequences are conceptually the same although there are differences in terminology and grouping of steps. The following factors have caused many of these differences:

- Phase I of the IRP includes a site ranking step based upon available data obtained during the records search. The NCP does not call for ranking sites until initial field sampling efforts are conducted. The reason for early ranking in the IRP is that Phase I evaluates all sites on an Air Force installation, whereas the NCP is concerned with individual sites. The Air Force relies on its HARM as a resource management tool to screen out lower priority waste disposal sites and to prioritize waste disposal site investigations. In contrast, individual sites brought into the NCP evaluation already show some evidence of high hazards.

- The NCP provides for implementing "operable units" prior to the feasibility study. Operable units are control methods that will be consistent with the finally selected remedial actions and can be put in place without detailed planning. The Air Force IRP has an equivalent option that involves implementation of emergency responses or simple removals where judged necessary by Major Commands.

- Phase III of the Air Force IRP involves development of new technologies. The NCP has no equivalent requirement.

- The NCP does not spell out procedural steps for design, construction, and compliance review as does the IRP. There is no conflict, however, in the programs at this point.




→ Optional Track

* For National Priority List sites

○ USAF Sites may proceed to Phase II whether included on the National Priority List or not.

■ Emergency Responses can be initiated in any Phase but are implemented as Phase IV actions.

Source: USAF (789)

FIGURE 7

COMPARISON BETWEEN EPA SUPERFUND AND USAF INSTALLATION RESTORATION PROGRAM (IRP)

### 1.4.6 Clean Air Act (CAA)

The primary goal of the Clean Air Act is the prevention and control of air pollution (369,376).

Section 109 of the Act authorizes USEPA to set National Primary and Secondary Ambient Air Quality Standards (NAAQS). The primary standards define levels of air quality which are necessary to protect public health with an adequate margin of safety. The secondary standards are the levels of air quality necessary to protect the public from any known or anticipated adverse effects of a pollutant. To date, NAAQS have been issued for lead, ozone, sulfur dioxide, carbon monoxide, particulate matter and nitrogen dioxide (374, 375, 378, 379).

Under Section 111, the USEPA may issue New Source Performance Standards (NSPS) to regulate air pollutants from new stationary sources which endanger the public health or welfare. These standards regulate emissions from specific categories of pollution sources and in many cases are set by USEPA to facilitate the achievement of NAAQS. Emissions of volatile organic compounds, including many IRP chemicals, are regulated under this section (377).

Section 112 allows the USEPA to set National Emission Standards for Hazardous Air Pollutants (NESHAPs 373). Hazardous air pollutants are defined as those that cause an increase in mortality or an increase in serious irreversible or incapacitating reversible illness. NESHAPs may apply to one particular stationary source or to several categories of sources (380, 381).

The USEPA is in the process of reviewing the health effects, sources and emissions of various air pollutants in order to decide whether they should be controlled under the Clean Air Act.

### 1.4.7 National Environmental Policy Act (NEPA)

The National Environmental Policy Act of 1969 requires the agencies of the federal government to consider the environmental effects of any governmental legislation, regulation or program. Section 102 requires preparation of an environmental impact statement by any federal agency involved in a legislative proposal which would significantly affect environmental quality. The impact statement must assess the environmental effects of the proposed action, the unavoidable effects if the proposal were implemented, and alternatives to the proposal. Installation Restoration Program Phase IV remedial actions often require evaluation under NEPA.

Section 202 of the Act established the Council on Environmental Quality. The council analyzes and interprets environmental trends and information, evaluates government programs and activities and formulates national policies to promote the improvement of environmental quality (307).

### 1.4.8 Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)

The Federal Insecticide, Fungicide and Rodenticide Act of 1972 provides for the establishment of procedures for the registration, classification, sale, use, research, monitoring and disposal of pesticides ( 313, 322, 323). FIFRA gives the Environmental Protection Agency broad powers to regulate all pesticides. Under Section 3, pesticides must be registered before they can be bought, sold, distributed or otherwise handled. The stringency of the registration standards is commensurate with the anticipated pattern of use and the degree of potential exposure of man and the environment. As part of the registration process, USEPA classifies the pesticide for either general or restricted use. A general use pesticide is one which, usually, does not cause unreasonable adverse effects on the environment whereas restricted use pesticides are those which may cause adverse environmental effects without additional regulatory restrictions. Restricted use pesticides may only be used or applied by a certified applicator, a person authorized by USEPA to use or supervise the use of any pesticide classified for restricted use. Under Section 6, USEPA can change the classification of a pesticide or cancel its registration if it appears to cause unreasonable adverse effects on the environment. Hearings must be held before either of these actions can be taken; however, USEPA, if necessary, may suspend the registration of a pesticide to prevent an imminent hazard during the time required for cancellation or change in classification (313).

Some IRP chemicals are listed as pesticide ingredients and are subject to the Pesticide Chemical Amendments to the Food, Drug and Cosmetic Act. These amendments were adopted in 1954 and passed on to joint FDA-USEPA administration after the formation of USEPA in 1970. The FDA has responsibility to ensure that the chemical is cleared by USEPA for use as an agricultural pesticide. USEPA and FDA each participate in the development of a pesticide tolerance (i.e., maximum permissible levels) which will be permitted in FDA sampled raw agricultural commodities. The pesticide must be registered under FIFRA, however, before any tolerance can be set (314).

### 1.4.9 Marine Protection Research and Sanctuaries Act (MPRSA)

The purpose of the Marine Protection Research and Sanctuaries Act of 1972 is to regulate the dumping of all types of materials into ocean waters and to prevent or strictly limit the ocean dumping of any material which would adversely affect human health, the marine environment, ecological systems or economic potentialities (308). Under Section 101, a permit is required before any person can transport any material for the purpose of dumping it into ocean waters. Permit requests are evaluated by USEPA on the basis of the need for the proposed dumping and the effects it will have on human health, wildlife and marine ecosystems. The permits, when issued, indicate the type and amount of material to be dumped and where it is to occur.

Under Section 201, a comprehensive and continuing program of monitoring and research was instituted to evaluate the effects of ocean dumping, particularly over the

long term. Section 203 authorizes USEPA to conduct research for the purpose of determining means to minimize or end ocean dumping as soon as possible. Section 302 gives the Secretary of Commerce the right to designate areas of ocean or coastal waters as marine sanctuaries as a means of preserving or restoring them (308).

### 1.4.10 Occupational Safety and Health Act (OSHA)

The Occupational Safety and Health Act of 1970 provides for safe and healthful working conditions for working men and women. This is accomplished by setting occupational safety and health standards and by providing research, information and training in the field of occupational safety and health. Employers are required to keep records of company activities which relate to the Act. These include reports of work-related deaths, injuries and illnesses and employee exposure to potentially toxic materials or harmful physical agents. The Act is enforced by the use of inspections and investigations.

Under Section 6, the Occupational Safety and Health Administration has set exposure standards for certain airborne contaminants in the workplace. The standards are based upon health criteria and technical feasibility. They are designed to assure, to the extent feasible, that no employee suffers impairment of health or functional capacity even if he is regularly exposed to a toxic material for the period of his working life. The OSHA standard for each chemical is known as the permissible exposure limit (PEL). PELs are usually listed as 8-hour time-weighted- averages (TWA). The level may be exceeded, but the sum of the exposure levels averaged over 8 hours must not exceed the limit. In some cases, ceiling and peak levels are listed in place of or in addition to the 8-hour TWA. Ceiling values cannot be exceeded at any time except for a designated time period when it may reach a peak level (298).

The National Institute for Occupational Safety and Health (NIOSH) was created under Section 22 of the Act. NIOSH is authorized to develop and establish recommended occupational safety and health standards. The recommendations are based on health effects and carry no regulatory weight. OSHA evaluates these recommendations when developing regulatory standards. The "Immediately Dangerous to Life or Health" (IDLH) concentration represents a maximum level from which one could escape within 30 minutes without any escape-impairing symptoms or any irreversible health effects. This recommendation is used for the purpose of respirator selection (304).

The American Conference of Governmental Industrial Hygienists (ACGIH) also issues recommendations for airborne contaminants. They are known as threshold limit values TLV® and are based on information from industrial experience and experimental human and animal studies. The basis on which these values are established may differ from substance to substance. Wherever threshold limit values have been used or included by reference in Federal and/or state statutes and registers, they have the force and effect of law (3). The ACGIH TLV® recommendations have also been adopted by the Air Force as official Permissible Exposure Limits (PEL's) in Air

Force Occupational Safety and Health Standard (AFOSH) 161-8, "Permissible Exposure Limits for Chemical Substances", except where a more stringent standard has been set by OSHA (29 CFR 1910.1000, Table Z-1). In a few instances, a chemical-specific AFOSH standard is available (i.e., hydrazine, benzene, vinyl chloride, carcinogenic substances). These chemical- specific AFOSH Standards have precedence over AFOSH Standard 161-8. Three categories of TLV® are issued:

1. TLV®-TWA - the time-weighted-average concentration for a normal 8-hour work-day and a 40-hour work-week to which nearly all workers may be repeatedly exposed without adverse effect.

2. TLV®-ceiling (TLV®-C) - a concentration that should not be exceeded at any time except for a designated time period when it may reach a peak level.

3. TLV®-STEL (Short-Term-Exposure Limit) - a fifteen minute time-weighted- average exposure which should not be exceeded at any time during a work-day even if the 8-hour TWA is within the TLV®. Exposures at the STEL should not exceed 15 minutes and should not be repeated more than four times per day. There should be at least a 60-minute interval between successive exposures at the STEL. A STEL is recommended only in cases in which toxic effects have been reported from high short-term exposures in either humans or animals. It is not a separate independent exposure limit but rather a supplement to the TLV®-TWA.

A TLV® followed by the designation "skin" refers to the potential contribution to overall exposure by the cutaneous route (including eyes and mucous membranes) either by airborne or direct contact with the substance.

Substances used in industry that are established carcinogens in humans or have induced cancer in animals under appropriate experimental conditions are assigned to categories A1 or A2. Human carcinogens are classified as A1. Substances suspected of inducing cancer in man based on either limited epidemiological evidence or demonstrated carcinogenicity in one or more animal species are classified as A2.

It is important to understand that both PEL and TLV® values apply to healthy adult employees working 40-hour weeks, and do not apply to the general population - including children, the elderly and the sick - which may be subject to continuous environmental exposure.

### 1.4.11 Food, Drug and Cosmetic Act (FDCA)

The Food, Drug and Cosmetic Act gives the federal government authority to protect the public from adulterated or misbranded foods, drugs, devices or cosmetics. Misbranding, in general, refers to false or misleading labeling. Adulteration refers to the presence of any poisonous or deleterious substance in a food, device, drug or

cosmetic. In addition, a drug can be classified as adulterated if it falls below its purported strength and purity.

FDA may regulate poisonous and deleterious substances in food by issuing tolerance levels if the substance cannot be avoided by good manufacturing practice. The tolerance level must be sufficient to protect public health.

Numerous food additives are classified as "generally recognized as safe" (GRAS). In instances where the additives are not classified as GRAS, FDA must certify the additive and safe conditions and concentrations for use must be issued. Generally, the maximum permissible level of an additive must be 1% or less of the concentration found to produce no effect in experimental animals. Under the Delaney Amendment, no food additive can be approved or remain in approved status if it is found to be carcinogenic to man or animals. However, carcinogenic color additives may still be used in external drugs and cosmetics if their use in those products does not induce cancer.

Food additives can be classified as direct or indirect. Direct food additives are intentionally added to foods for a certain functional effect. Direct additives include such things as stabilizers, flavors and colors. Conversely, indirect additives are those which are not directly or intentionally added for a functional purpose. Such an additive might reasonably be expected to migrate or otherwise become a component of food because of some contact between the substance and the food. Examples of indirect additives are components of packaging materials.

Some IRP chemicals are approved for use as food additives (360).

The FDA has also set maximum contaminant levels (MCL's) for bottled drinking water. The limits are identical to those issued under the Safe Drinking Water Act (365).

### 1.4.12 Consumer Product Safety Act (CPSA)

Under CPSA, the Consumer Product Safety Commission (CPSC) regulates products which present an unreasonable risk of injury. The product may be banned or special labeling may be required. The standards are based on human health effects, the significance of exposure and the degree of risk involved. The economic effects of the rule as well as the public need for such products are also considered (310).

### 1.4.13 Hazardous Materials Transportation Act (HMTA)

The Department of Transportation, pursuant to the Hazardous Materials Transportation Act, has promulgated regulations governing the transportation and shipment of hazardous materials. These hazardous materials have been designated by the Department of Transportation and are subject to requirements for shipping papers, package marking, labeling and transport vehicle placarding. These regulations

apply to each person who offers a hazardous material for transportation and each carrier who transports a hazardous material by air, highway, rail or water (305). Many IRP chemicals have been designated as hazardous for the purpose of transportation.

## 1.5 EUROPEAN COMMUNITY DIRECTIVES

The documentaion for the European Community Directives reflects information available as of May, 1989. Information resource pamphlets from Barbara Sloan, Delegation of the Commission of the European Communities; 1987 Installation Restoration Program Toxicology Guide; Official Journal of the European Commission articles; and literature review of Chemical Abstracts serve as background material for compiling this information.

### 1.5.1 EEC Directives

Since 1972, the European Community, a confederation of independent countries alternatively called the European Economic Community, EEC or the Common Market, has adopted several "action plans" and more than 100 environmental legislative measures called directives which protect Europe's natural resources (3988). EEC directives limit air pollution by motor vehicles, the level of sulphur in heating oil and the lead content of gasoline. Others control the shipment of hazardous wastes across national borders, and establish safety standards designed to prevent industrial accidents. Quality standards have been set for bathing and drinking water, and strict rules apply to the release of certain chemicals into water.

Member EEC countries include Belgium, Denmark, F.R. Germany, Greece, Ireland, Italy, Luxembourg, Netherlands, Spain and the United Kingdom (3986).

Environmental laws discussed below are directives, (i.e., a directive is a official act which sets out objectives, standards and procedures. These directives must be implemented in each member country by national law and regulation within 18 months after its adoption (3985). The major directives applicable to IRP chemicals are presented below; permissible levels for specific IRP chemicals are itemized in the regulatory section of each chapter. Some 28 directives, 2 resolutions/proposals and 2 new directives will be highlighted throughout The Installation Restoration Program Toxicology Guide.

#### Directive on Drinking Water

The Directive on drinking water defines the quality requirements that surface fresh water must meet if used or intended for human consumption (533). Ground water, brackish water and water intended to replenish water-bearing beds are not subject to this Directive. For the purpose of the Directive, surface waters are divided into three categories, Al, A2 and A3, which correspond to treatment methods ranging from single to intensive. Water treated by these methods must conform to parameters which are specific for each category. The parameters include physical, chemical and

microbiological characteristics and values are listed for both guidelines and mandatory standards. Member countries designate sampling points and intervals so that waters may be tested for compliance. Member countries should apply this directive to national and waters crossing its frontiers and should also draw up a systematic plan of action including a timetable for improvement of surface water especially within category A3. (Amended October 29, 1979.)

### Directive Relating to the Quality of Water Intended for Human Consumption

The purpose of this Directive is to set standards for water intended for human consumption either in its original state or after treatment, regardless of its origin (540). Water used in food production is also subject to this Directive; however, natural mineral waters and medicinal waters are not. The organoleptic, physicochemical and toxic substances parameters which have been set include both guide levels (GL) and maximum allowable concentrations (MAC).

Member countries must ensure that these waters meet the requirements and that any substance used in water preparation not remain in quantities in excess of the MAC or pose a public health hazard. Measures for regular monitoring as well as reference methods of analysis are also defined. (Amended July 11, 1981.)

### Directive on the Protection of Ground Water

This Directive is aimed at protecting ground water from pollution by certain toxic, persistent and bioaccumulable substances (538). For the purposes of the Directive, substances are classified as either List I or List II substances. Direct discharge of List I substances into ground water without percolation through the ground or subsoil is prohibited. List I includes organohalogen and organonphosphorus compounds, hydrocarbons and substances which possess carcinogenic, mutagenic or teratogenic properties. Direct discharge of List II substances must be limited by member countries. List II includes metals, silicon, fluorides, biocides and substances affecting the taste or odor of ground water. (No Update.)

Member countries must investigate any indirect discharge (i.e., discharge into ground water after percolation through the ground or subsoil) of List I or List II substances. Authorizations for indirect discharge of List I substances may be granted only if precautions have been taken to ensure that the substance do not harm aquatic or other ecological systems. Authorization may also be granted if the ground water into which the substances is to be discharged is deemed to be permanently unsuitable for domestic or agricultural uses. Authorization for discharge of List II substances is granted when all technical precautions for preventing ground-water pollution by these substances are observed. Excluded from the scope of the Directive are domestic effluents from certain isolated dwellings, and discharges containing "very small" quantities of List I or II substances.

### Directive on Bathing Water Quality

This Directive concerns the quality of bathing water with the exception of water intended for therapeutic purposes and water used in swimming pools (534). The Directive defines bathing water to include all running or still fresh waters and sea water in which bathing is explicitly authorized by member countries or where bathing is not prohibited and is traditionally practiced by large numbers of people. Such waters are subject to physical, chemical and microbiological testing requirements. The directive lists both guideline and mandatory values for each testing parameter. Sampling points and intervals are also given so that waters may be tested for compliance. (Amended May 1976.)

### Directive on Fishing Water Quality

The purpose of this Directive is to protect or improve the quality of salmonid and cyprinid waters designated by member countries as needing protection or improvement in order to support fish life (536). Once such waters are designated, member countries establish programs to reduce pollution so that within 5 years the designated waters will conform to set physical and chemical parameters. The parameters include pH, suspended solids, dissolved oxygen, BOD, temperature, total phosphorus, nitrates, phenolics, petroleum hydrocarbons, non-ionized ammonia, total ammonium, total residual chlorine, total zinc and dissolved copper. Both mandatory and guideline values are set for each parameter. The designated waters are deemed to be in conformance with the Directive when all samples taken at the specified points and frequencies over a 12-month period conform to both values set for each parameter.

### Directive on the Quality Required of Shellfish Waters

This Directive applies to coastal and brackish waters designated by member countries as needing protection or improvement in order to support shellfish life and growth and thereby contribute to the high quality of shellfish products directly edible by man (537). The waters so designated must conform to physical, chemical and microbiological parameters. Both mandatory and guideline values are set for each parameter. The parameters include such things as metals, petroleum hydrocarbons, suspended solids, organohalogen compounds, pH, temperature and coliform count. Designated waters are deemed to be in conformance with the Directive when samples taken at the specified points and frequencies over a 12-month period conform to values set for each parameter.

### Directive on the Discharge of Dangerous Substances

The purpose of the Dangerous Substances Directive is the protection of the aquatic environment from pollution (535). The Directive authorizes member countries to issue permits and standards for the discharge of such substances into inland surface waters, territorial waters, internal coastal waters and ground water. The substances which are regulated are classified into two lists. List 1 substances are

selected on the basis of their toxicity, persistence and bioaccumulation. They include organohalogen and organophosphorus compounds, carcinogens, mercury and cadmium. List 2 substances are those which have a deleterious effect on the aquatic environment but depend on the characteristics and location of the water into which they are discharged. These substances must be able to be confined to a given area. This list includes metals, cyanides, fluorides, ammonia, nitrites, inorganic phosphorus and substances which will adversely affect the taste or odor of human food products derived from aquatic environments. This Directive does not apply to dredging discharges or to operational discharges or dumping from ships in territorial waters. (No Update.)

### Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents)

This Directive outlines the classification, packaging and labeling of solvents used either alone or in combination with other flammable liquids (544). The Directive does not apply to foodstuffs, medicines, pesticides, paints, adhesives, munitions, hazardous wastes, and compressed or liquified gases. Solvents which are regulated are listed as either Class I toxic substances or Class II harmful substances. Classes are broken down into subclasses. Each solvent is given a numerical classification index based upon its degree of toxicity or harmfulness. This index is used in calculations to determine the proper classification of a preparation containing mixtures of solvents.

The labeling requirements specify that the name and percentage of a toxic substance in a preparation (in excess of 0.2%) be indicated on the label. The name and percentage of any harmful substance present in excess of 3-20% (depending on subclass) must also be indicated. The label of all preparations must include safety advice, special risk involved in use of the preparation, symbols, indicating the relative danger of the products and the name and address of the package. Member countries must take all necessary measures to ensure that these products are not placed on the market unless they are in compliance with the Directive.

### Directive on Toxic and Dangerous Waste

Under the terms of this Directive, member counties must take measures to ensure that toxic and dangerous waste is disposed of without endangering human health or harming the environment (542). Establishments which carry out the storage, treatment or disposal of toxic or dangerous waste must obtain, from state authorities, permits which outline technical requirements, precautions and methods of disposal. The disposal cost, less any proceeds from waste treatment, must be defrayed in accordance with the "polluter pays" principle. Provision is made for a system of monitoring and supervision of all installations and for the keeping of proper records. Among the wastes regulated are organohalogen compounds, organic solvents, chlorinated solvents, phenols, ethers, biocides, metals and aromatic polycyclic compounds.

### Directive on Marketing and Use of Dangerous Substances

This Directive is concerned with restricting the marketing and use of dangerous substances and preparations in member countries (541). It specifies that designated dangerous substances and preparations may only be placed on the market or used subject to the conditions which have been set for each substance. Among the substances regulated are vinyl chloride monomer and benzene. The Directive does not apply to transport of the substances or to substances exported to other non-member countries.

The Directive does not apply to natural or artificial fish ponds used for intensive fish farming.

### Resolution on Fluorocarbons in the Environment

This resolution states that appropriate measures should be taken to insure that industries within the European Community do not increase their production of trichlorofluoromethane and dichlorodifluoromethane (539). Industries using these fluorocarbons should be encouraged to eliminate their discharge into the environment and to intensify their research into alternative products.

### Directive on Classification, Packaging and Labeling of Dangerous Substances

The purpose of this Directive is to approximate the laws, regulations and administrative provisions on the classification, labeling and packaging of substances dangerous to man and the environment (787). The Directive makes a distinction between new and existing substances. Existing substances are those which were placed on the Community market before September 18, 1981. Other substances are considered new. The Directive lists fourteen categories in which dangerous substances are classified which include: explosive, flammable, oxidizing, toxic, harmful, corrosive, irritating, carcinogenic, teratogenic, mutagenic or hazardous to the environment. The substances are placed into these categories on the basis of their physicochemical properties, human and environmental toxicity and the degree of risk and hazard involved in their use. These substances cannot be marketed unless they meet specific labeling and packaging requirements. Label size depends upon package capacity. The label must include the name and origin of the substance, danger symbol, standard phrases indicating safety advice and special risks, and the name and address of the manufacturer, distributor or importer. Effective as of June 26, 1986, each label must contain the correct CAS-number for each substance. A criteria for new safety advices and special risks are to be implemented (86/431/EEC). The packaging materials must not be susceptible to adverse attack by the contents and the package itself must be designed and constructed so that the contents cannot escape. Commission Directive 88/490/ EEC amends Directive 67/548, Annex I (list of dangerous substances) with: (a) A label should be provided in accordance with the rules for the labelling of dangerous preparations if the stablizer changes the dangerous properties of the substance, (b) The designation, CAS-number, classification and labelling of the substances in Annex 1 to this directive replace those in Annex I to directive

67/548/EEC with the same EEC-number, and (c) Substances listed in Annex II of this directive are added to Annex I of Directive 67/548/EEC.

### Directive on the Classification, Packaging and Labeling of Pesticides

This Directive sets forth regulations relating to the classification, labeling and packaging of pesticides, either alone or in combination with other materials (786). Pesticides are classified as very toxic, toxic or harmful by means of either acute oral, percutaneous or respiratory toxicity expressed in terms of $LD_{50}$ or $LC_{50}$ values for the rat. Pesticides cannot be marked unless their packaging is designed and constituted in a manner to ensure that the package can withstand normal handling without the escape of its contents. Labels size is dependent upon the capacity of the package. Label must show the trade name of the product, the name and amount of active and inactive ingredients, net quantity, batch number, name and address of the manufacturer, indications of danger and special risks involved in the use of the pesticide and a statement that the package is not to be re-used. If a member country establishes that a pesticide constitutes a danger to health or safety even though it satisfies the requirements of the Directive, it may impose special conditions or prohibit its sale. Manufacturers and importers of these substances are required to submit technical information about products they intend to market to member countries. The information must include an evaluation of risks to man and the environment, recommended precautions for safe use, and proposed classification and labeling. This information must be received by member countries at least 45 days before the substance is placed on the market. A decision is then made on whether the product can be marketed. Member countries may prohibit the sale of a substance or limit its sale if there is evidence that it is a hazard to man or the environment. Directive 83/291/EEC updates this directive.

### Directive on Paints, Varnishes, Printing Inks, Adhesives and Similar Products

This directive outlines the classification, packaging and labeling of paints, varnishes, printing inks, adhesives and similar products (1334). The Directive does not apply to the dangerous preparations shipped by railroad, inland waterway, sea or air, cosmetic preparations, additives for foodstuffs, animal feeds, fertilizers, pesticides, and substances in the form of waste which are covered by Directive 75/442/EEC. In preparations covered by this directive, concentrations of the dangerous substances listed in Annex I to Directive 67/548/EEC, whether present as impurities or additives, must be taken into account if they exceed limits classified by degrees of toxicity, harmfulness, corrosiveness, irritation, oxidization, and flammability. The label of all preparations should contain the trade name or designation of preparation, safety advice, special risk involved in use of products, chemical name, symbols indicating the relative danger of products and the name and address of the manufacturer or person placing the preparation on the market. Directive 83/265/EEC amends this directive to include corrosive and irritant solvents.

### Directive on Limit Value for Lead in the Air

This directive lays down a maximum limit value for lead concentrations in air of 2 micrograms per cubic meter, expressed as an annual average mean concentration. It does not apply to occupational exposure (1429). Member states may impose stricter limit values.

The member states must ensure that sampling stations are operated at places where individuals may be exposed continually for a long period and where the limit value is likely not to be observed. In places, where, after 4 years the limit value is exceeded, the member states must draw up plans for improvement of the air and send to the Commission. A final deadline for compliance is set for December 9, 1989. Member states must inform the Commission by July 1, annually, of places where the limit value is exceeded, by how much, and the measures to bring lead concentrations below the limit value.

### Directive on the Lead Content in Petroleum

This directive replaces 78/611/EEC which set limits on the lead content of petroleum (1430). The earlier directive had set limits of from 0.4 to 0.15 grams of lead per litre (g Pb/l) in petrol. This directive requires the member states to reduce the permitted lead content to 0.15 g pb/P as soon as they consider it appropriate, and also to ensure that availability and balanced distribution of unleaded petrol (having a content below 0.013 g Pb/l) from 1 October 1989, at the latest.

Member states may prohibit the marketing of leaded petroleum having the motor octane number lower than 85 at the pump and a research octane number lower than 95 at the pump for reasons of health and environmental protection and to promote availability and balanced distribution of unleaded petroleum within their territory, and must inform the Commission and the public 6 months in advance. The benzene content of both leaded and unleaded petroleum may not exceed 5.0% as of October 1, 1989.

### Directive on the Supervision and Control of Transfrontier Shipment of Hazardous Waste

When the holder of hazardous waste intends to ship it to another member state, authorities of the member states concerned must be provided with information on the source and composition of the waste, measures to be taken to ensure safe transport, insurance against damage and the existence of a contractual agreement with the consignee of the waste (1433). All transfrontier shipments must be properly packed and labeled and must be accompanied by instructions to be followed in the event of danger of accident. Commission Directive 87/1121 EEC amends this directive and updates Annex II, concerning wastes from non-ferrous metals for re-use, regeneration or recycling, and concerning the consignment note to be prepared by the holders of waste destined to be shipped to non-EEC countries for disposal.

**Council Directive on Major Accident Hazards at Certain Industrial Activities.**

On June 24, 1982 the EEC passed the Council Directive on major accident hazards of certain activities known as the Sevesco Directive (1794). The objectives of the Directive focus on the prevention of major-accidents and the limitations of their effects on man and the environment. The directive has two structural parts. The first gives the framework of general requirements. The directive has two structural parts. Under the first part, for general hazards, the manufacturer must be able to prove to the national competent authority at any time that it has identified existing major accident hazards, adopted the necessary safety measures, and provided the persons working on the site with information. Under the second part, certain industrial activities are subject to a notification procedure if the 180 chemicals listed in Annex III are or may be present in the designated quantities or if the chemicals listed in Anex II are stored in the designated quantities. The notification must contain detailed information about: substances and manufacturing process, hazards and risk, safety precautions and emergency procedures; the industrial plant, including siting, exposed groups and environment, sources of danger from location of the plant, preventive measures and technical controls; and possible major-accident situations, including emergency plans, safety equipment, and alarms and resources. In the case of an accident, the manufacturer must immediately inform the competent authority

**Council Resolution on Revised List of Second-Category Pollutants**

The Council of the European Communities has proposed a revised list of second-category pollutants to be studied as part of the programme of action of the European Communities in order to reduce pollution and nuisances in the air and water (545). Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied, will be assessed. The revised list includes vinyl chloride, fluorine and its compounds, cadmium, pesticides, chlorine and its compounds (including hydrochloric acid), nitrates and nitrites, organic solvents, phthlates, asbestos, ammonia, organic silicon compounds, and cationic, anionic and non ionic surfactants.

**Council of the European Communities Proposal for a Council Directive on Dumping of Waste at Sea COM(85) 373 Final**

EEC has proposed this directive to prevent and reduce marine pollution caused by dumping of waste or other materials at sea, including incineration, from ships and aircraft (1793). EEC has proposed that the dumping at sea of organohalogens, mercury and mercury compounds, cadmium and cadmium compounds, plastics, crude oil and hydrocarbons, substances which have a carcinogenic effect, acids and alkalis, materials produced by biological and chemical warfare and oil-based drilling muds be prohibited.

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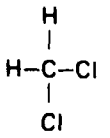
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| COMMON SYNONYMS: | CAS REG. NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25 °C (12) |
|---|---|---|
| DCM<br>Dichloromethane<br>Methane dichloride<br>Methylene bichloride<br>Methylene chloride<br>Methylene dichloride | 75-09-2 $CH_2Cl_2$<br>NIOSH NO.:<br>PA8050000<br>STRUCTURE:  | 3.48 mg/m³ ≈ 1 ppm;<br>0.288 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT:<br>84.94 |

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|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as methylene chloride with cyanides, mercaptans or other organic sulfides typically generate heat, while those with amines, azo compounds, hydrazines, caustics, or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505). |

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| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Ether-like | (23) |
| | • Odor Threshold: 214.000 ppm | (263) |
| | • Density: 1.3250 g/mL (at 20°C) | (12) |
| | • Freeze/Melt Point: -96.70°C | (21) |
| | • Boiling Point: 39.80°C | (21) |
| | • Flash Point: Nonflammable | (21) |
| | • Flammable Limits: 12.00 to 19.00% by volume | (504,506) |
| | • Autoignition Temp.: 556.0 to 640.0°C | (504,506) |
| | • Vapor Pressure: 3.50E+02 mm Hg (at 20°C) | (38) |
| | • Satd. Conc. in Air: 1.5490E+06 mg/m³ (at 20 °C) | (67) |
| | • Solubility in Water: 1.32E+04 mg/L (at 20 °C) | (21) |
| | • Viscosity: 0.430 cp (at 20 °C) | (21) |
| | • Surface Tension: 2.8000E-02 dyne/cm (at 20°C) | (21) |

| | |
|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Log (Octanol-Water Partition Coeff.): 1.25 (29)<br>• Soil Adsorp. Coeff.: 8.80E+00 (33)<br>• Henry's Law Const.: 2.57E-03 atm · $m^3$/mol (74) (at 25°C)<br>• Bioconc. Factor: 8.00E-01 (estim) (659) |
| PERSISTENCE IN THE SOIL-WATER SYSTEM | Methylene chloride is expected to be highly mobile in the soil/ground-water system; little or no retardation is expected in deep or sandy soils. Volatilization is an important removal process for methylene chloride near the surface or in the soil-air phase. Transformation processes such as hydrolysis and biodegradation are not expected to be important in natural soil systems. |
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of methylene chloride to groundwater drinking water supplies. Data from NPL sites show that such migration has commonly occurred in the past. Inhalation resulting from volatilization from surface soils may also be important. |
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (54)<br>Methylene chloride is a mild narcotic. Effects from intoxication include headache, giddiness, stupor, irritability, numbness and tingling of the limbs. Irritation to the eyes and upper respiratory passages occur with both liquid and vapor. If held in contact with the skin, the liquid may cause burns. Exposure may cause elevated carboxyhemoglobin levels. |

| | |
|---|---|
| HEALTH HAZARD DATA | Acute Toxicity Studies: (3504)<br><br>INHALATION:<br>$TC_{Lo}$ 1740 mg/m³ · 8 hr — Human<br>$LC_{50}$ 50112 mg/m³ · 7 hr — Mouse<br>$LC_{Lo}$ 43400 mg/m³ · 4.5 hr — Cat<br>$LC_{Lo}$ 17400 mg/m³ · 2 hr — Guinea Pig<br>$LC_{Lo}$ 34800 mg/m³ · 7 hr — Rabbit<br>$LC_{Lo}$ 49096 mg/m³ · 7 hr — Dog<br>$LC_{50}$ 88000 mg/m³ · 30 min — Rat<br><br>ORAL:<br>$LD_{Lo}$ 357 mg/kg — Human<br>$LD_{Lo}$ 1900 mg/kg — Rabbit<br>$LD_{50}$ 167 mg/kg — Rat<br>$LD_{50}$ 1600 mg/kg — Rat<br><br>Long-Term Effects: Possible liver damage, increased carboxyhemoglobin levels<br>Pregnancy/Neonate Data: Negative<br>Genotoxicity Data: Suggestive evidence of genotoxicity<br>Carcinogenicity Classification:<br>IARC - Group 2B (possibly carcinogenic to humans).<br>NTP - Clear evidence in mice & female rats, some evidence in male rats<br>EPA - Group B2 (sufficient evidence from animal studies, inadequate human evidence) |

| | |
|---|---|
| HANDLING PRECAUTIONS (38,52,54) | Handle chemical only with adequate ventilation. • Vapor concentrations of 500-5000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece. • Chemical goggles if there is probability of eye contact. • Natural rubber, neoprene, nitrile, PE, PVC or other protective clothing to prevent repeated or prolonged skin contact with the liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

### AIR EXPOSURE LIMITS:

Standards

- OSHA TWA (8-hr): 500 ppm; CL: 1000 ppm; Peak: 2000 ppm for 5 minutes in any 2 hours
- AFOSH PEL (8-hr TWA): 500 ppm; CL: 1000 ppm; Peak: 2000 ppm for 5 minutes in any 2 hours; STEL (15-min): 625 ppm

Criteria

- NIOSH IDLH (30 min): deleted, NIOSH has recommended that the substance be treated as a potential human carcinogen.
- ACGIH TLV (8-hr TWA): 50 ppm (A2, suspected human carcinogen)

### WATER EXPOSURE LIMITS:

Drinking Water Standards (3883)
MCLG: 0 µg/L (tentative)

EPA Health Advisories and Cancer Risk Levels (3977)
In the absence of formal drinking water standards, the EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- 1-day (child): 10 mg/L
- 10-day (child): 2 mg/L
- 1E-04 Cancer Risk Level: 500 µg/L

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria

- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms (1E-05, 1E-06, 1E-07 cancer risk), 1.9 μg/L, 0.19 μg/L, 0.019 μg/L halomethanes.
  - Based on ingestion of contaminated aquatic organisms only (1E-05, 1E-06, 1E-07 cancer risk), 157 μg/L, 15.7 μg/L, 1.57 μg/L halomethanes.
  - Based on ingestion of contaminated water only (1E-05, 1E-06, 1E-07 cancer risk), 1.9 μg/L, 0.19 μg/L, 0.019 μg/L halomethanes.

- Aquatic Life (355)
  - Freshwater species
    acute toxicity: no criterion, but lowest effect level occurs at 11,000 μg/L halomethanes.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity: no criterion, but lowest effect level occurs at 12,000 μg/L halomethanes.

    chronic toxicity: no criterion, but lowest effect level occurs at 6400 μg/L halomethanes.

REFERENCE DOSES:
ORAL: 6.000E+01 μg/kg/day (3744)

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Methylene chloride is listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and to effluent guidelines and standards (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), metal finishing (3768), and metal molding and casting (892). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

Methylene chloride is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). It is listed as an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3771). In states with an approved Underground Injection Control program, a permit is required for the injection of methylenechloride-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

Methylene chloride is identified as a toxic hazardous waste and listed as a hazardous waste constituent (3783, 3784). Non-specific sources of methylene chloride-containing waste are solvent use (or recovery) activities, chlorinated aliphatic hydrocarbon production, and spent solvent mixtures containing 10% or more methylene chloride (3765). Waste streams from the organic chemicals industry (acetaldehyde production) contain methylene chloride and are listed as specific sources of hazardous waste (3774, 3765). Methylene chloride is included on EPA's groundwater monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their groundwater for chemicals on this list when suspected contamination is first detected and annually thereafter (3775). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standard shave not been promulgated by EPA (3786).

Toxic Substances Control Act (TSCA)
Manufacturers, processors or distributors of methylene chloride must report production, usage and disposal information to EPA. They, as well as others who possess health and safety studies on methylene chloride, must submit them to EPA (334, 3789).

Comprehensive Environmental Response Compensation and Liability Act (CERCLA)
Methylene chloride is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing methylene chloride but these limits depend upon the concentration of the chemicals present in the wastestream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of methylene chloride must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
Methylene chloride is exempt from a tolerance requirement when used as a solvent or co-solvent in pesticide formulations applied to growing crops or to animals (315). Exemptions also apply when it is used as a fumigant after harvest for barley, corn, oats, popcorn, rice, rye, sorghum and wheat, and for citrus fruits (314).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to methylene chloride shall not exceed an 8-hour time-weighted average (TWA) of 500 ppm. A ceiling level of 1000 ppm shall not be exceeded at any time during an 8-hour work-shift except for a duration of 5 minutes in any 2 hours when it may reach a ceiling level of 2000 ppm (3539).

Clean Air Act (CAA)
EPA lists methylene chloride as a hazardous air pollutant for which it intends to establish emission standards under Section 112 of the Clean Air Act (3685).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated methylene chloride as a hazardous material with a reportable quantity of 454 kg, subject to requirements for packaging, labeling and transportation (3180).

Food,Drug and Cosmetic Act (FDCA)
Methylene chloride may be present as an extraction residue in the following foods (361):

- spice oleoresins, at a level not exceeding 30 ppm
- hops extract, at a level not exceeding 2.2%
- decaffeinated coffee, at a level not exceeding 10 ppm

Methylene chloride is approved for use as an indirect food additive as a component of adhesives (3209).

- State Water Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

CALIFORNIA
California has an action level of 40 μg/L for drinking water (3098).

CONNECTICUT
Connecticut has an action level of 25 μg/L for drinking water and a quantification limit of 2 μg/L (3138, 3137).

KANSAS
Kansas has an action level of 50 μg/L for groundwater (3213).

NEW HAMPSHIRE
New Hampshire has an enforceable one-day Toxic Contaminant Level (TCL) of 13 mg/L in drinking water (assuming a child weighing 10 kg who drinks one liter of water per day) (3710).

NEW JERSEY
New Jersey has set a maximum contaminant level of 2 μg/L (ppb) for methylene chloride in drinking water (3497).

NEW MEXICO
New Mexico has a human health criterion of 0.1 mg/L for groundwater (3499).

NEW YORK
New York has set a maximum contaminant level of 5 μg/L for drinking water, and has a nonenforceable guideline of 50 μg/L for ground and surface waters (3501).

OKLAHOMA
Oklahoma has a water quality criterion of 10 μg/L for groundwater (3534).

PENNSYLVANIA
Pennsylvania has a human health criterion (cancer risk level) of 5 μg/L for surface waters (3561).

RHODE ISLAND
Rhode Island has an acute freshwater quality guideline of 9650 μg/L and a chronic guideline of 214 μg/L for the protection of aquatic life in surface waters. These guidelines are enforceable under Rhode Island state law (3590).

SOUTH DAKOTA
South Dakota requires methylene chloride to be nondetectable, using designated test methods, in groundwater (3671).

VERMONT
Vermont has a preventive action limit of 2.5 μg/L and an enforcement standard of 5.0 μg/L for groundwater (3682).

WISCONSIN
Wisconsin has a preventive action limit of 15 μg/L and an enforcement standard of 150 μg/L for methylene chloride in groundwater (3840).

Proposed Regulations

- Federal Programs

Safe Drinking Water Act (SDWA)
EPA will propose MCLGs and MCLs for methylene chloride in March 1990, with final promulagation scheduled for March 1991 (3751).

Resource Conservation and Recovery Act (RCRA)
EPA has proposed that solid wastes be listed as hazardous in that they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is equal to or greater than 8.6 mg/L methylene chloride. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Water Programs

  MOST STATES

  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA

  Minnesota has proposed a Recommended Allowable Limit (RAL) of 48 μg/L for methylene chloride in drinking water (3451). Minnesota has also proposed a Sensitive Acute Limit (SAL) of 48250 μg/L for surface waters, chronic criteria ranging from 46 to 47 μg/L for designated surface waters, and a chronic criterion of 48 μg/L for designated groundwaters. These criteria are for the protection of human health (3451, 3452).

  NEW JERSEY

  New Jersey has proposed a surface water quality criterion of 2 μg/L for FW2 Waters (3496).

EEC Directives

Directive Relating to the Quality of Water for Human Consumption (540). There are no guidelines for maximum admissible concentrations of chlorides; however, effects may occur if concentrations exceed 200 mg/1.

Directive on Ground-Water (538)

Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
1,2-Dichlorobenzene is listed as a Class II/a harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into groundwater.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert poly-meric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto- pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,2-Dichlorobenzene is classified as a harmful substance and is subject to packaging and labeling regulation.

EEC Directive-Proposed Resolution
Resolution on a Revised List of Second-Category Pollutants (545)
1,2-dichlorobenzene is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 1.1 MAJOR USES

Methylene chloride is widely used in a variety of industrial and commercial applications including metal degreasing, paint stripping, and solvent extraction in the food processing industry. It replaced trichloroethylene as the caffeine extractant in the production of decaffeinated coffee. It is also used to extract spice oleoresins and the beer flavoring in hops (21).

It has additional uses as an aerosol propellent, an insecticidal fumigant and as a solvent in the manufacture of photographic film and synthetic fiber (3314).

## 1.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 1.2.1 Transport in Soil/Ground-water Systems

#### 1.2.1.1 Overview

Methylene chloride will be relatively mobile in the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical).

Transport pathways can be generally assessed by using an equilibrium partitioning model as shown in Table 1-1.

#### 1.2.1.2 Sorption on Soils

The mobility of methylene chloride in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of methylene chloride sorption on soil particles. Sorption of methylene chloride is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

The sorption of methylene chloride on soil particles is not well documented. Retardation rates ($R_f$), which represent the interstitial water velocity/pollutant velocity in the soil, were reported by Wilson et al. (82) to be a function of $K_{oc}$, the ratio of soil density (a) to soil water content (b), and the organic content (oc) of the soil according to the following equation:

$$R_f = 1 + (a/b)K_{oc}\,(oc)$$

Schwarzenbach et al. (77) report retardation factors for some organic compounds that are similar to methylene chloride but have higher $K_{oc}$ values; the data indicate some retardation in soils having 1-2% organic carbon and little or no retardation (i.e., absorption) in deep soils having less than 0.1% organic carbon. Assuming analogous soil conditions, adsorption of methylene chloride, particularly to deep soils, is not expected to be significant.

TABLE 1-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR METHYLENE CHLORIDE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 25°C | 56.1 | 33.2 | 10.7 |
| Saturated deep soil[d] | 3.6 | 96.4 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Used estimated soil sorption coefficient: $K_{oc}$ = 8.8 (33).
c) Henry's law constant taken as 2.57E-03 atm · $m^3$/mol at 25°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

The calculations shown in Table 1-1 predict the partitioning of low soil concentrations of methylene chloride among soil particles, soil water and soil air. The estimates for the unsaturated topsoil model show that significant amounts of methylene chloride are present in the soil-water (33%) and soil-air (11%) phases, and thus available to be transported through these phases by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. Diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, may also be a significant loss pathway. In saturated, deep soils (containing no soil air and negligible soil organic carbon), most of the methylene chloride is likely to be present in the soil-water phase (96%) and transported with flowing ground-water.

Ground water underlying methylene chloride-contaminated soils with low organic content is particularly vulnerable to contamination.

### 1.2.1.3 Volatilization from Soils

Transport of methylene chloride vapors through the air-filled pores of unsaturated soils is an important mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient and, to a lesser extent, the vapor phase diffusion coefficient (31).

Half-lives on the order of 20 to 90 minutes have been reported for the volatilization of methylene chloride, depending on the degree of agitation of the aqueous solutions (10). Compared to volatilization from well-stirred aqueous solutions, volatilization from near surface soils has been reported to be slower by approximately one order of magnitude for several chlorinated aliphatics (82).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. The temperature dependence of H for methylene chloride has been measured by Gossett and Lincoff (18), and is described by the following equation:

$$H\ (atm \cdot m^3/mol) = \exp[9.035\text{-}4472/T°K]$$

Gossett and Lincoff (18) have also examined the effect of other dissolved materials on the volatilization of methylene chloride. Moderate increases in H were observed with increasing salinity and the presence of other organic compounds. These results suggest that the presence of other chemicals in the soil/ground-water system may significantly affect the volatilization of methylene chloride from surface soils.

No information was available for the two other physicochemical properties influencing methylene chloride volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

## 1.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of methylene chloride in soil/ground-water systems is not well documented. In most cases, it should be assumed that methylene chloride will persist for months to years (or more). Methylene chloride that has been released into the air will eventually undergo photochemical oxidation; a tropospheric lifetime of 0.3 to 0.6 years has been reported for methylene chloride (6).

Methylene chloride does not undergo rapid hydrolysis under normal environmental conditions. The hydrolytic half-life of methylene chloride in water has been estimated in two separate studies at 18 months and 700 years (10). Since the experimental system used to obtain the 18 month value involved aerated water, some free radical oxidation could also have occurred. The lower temperatures and oxygen content in soils and ground waters compared to surface waters suggest that hydrolysis in moist soils and ground water may be slow.

No information pertaining specifically to the biodegradation of methylene chloride was found. The few available literature references report that low molecular weight chloroaliphatics are not metabolized (10). Thom and Agg (80) included methylene chloride on a list of organic chemicals considered to be potentially degradable by biological sewage treatment, provided suitable acclimatization can be achieved; they also noted that it is unlikely that microorganisms already possess the ability to degrade methylene chloride. Since the concentration of microorganisms capable of biodegradation is very low and drops off significantly with increasing depth, biodegradation of methylene chloride in the soil/ground-water system is assumed to be minimal except, perhaps, in landfills with active microbiological populations.

### 1.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The properties and the above discussion of fate pathways suggest that methylene chloride is highly volatile, is very weakly adsorbed to soil and has no significant potential for bioaccumulation. Methylene chloride on the soil surface is likely to volatilize, but that portion not removed by volatilization is likely to be mobile in ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of methylene chloride from a disposal site could result in inhalation exposures. In addition, the potential for ground-water contamination is high, particularly in sandy soils. Mitre (83) reported that methylene chloride has been found at 36 of the 546 National Priority List (NPL) sites. It was detected at 26 sites in ground water, 16 in surface water and 2 in air. These data indicate that both ground-water and surface-water pathways may be important. The lack of detection of methylene chloride in air may be somewhat misleading as air sampling at NPL sites is limited. In the National Organics Monitoring Survey (NOMS) conducted by the USEPA (90), methylene chloride was detected in 15 of 109 water supplies sampled including both ground and surface water supplies. The mean concentration of the positive results was 6.1 $\mu$g/L. This compound was not included in either the Community Water Supply Survey or the Ground-water Supply Survey of EPA (531).

The properties and the survey results discussed above suggest that methylene chloride in soil/ground-water transport systems can result in exposure through drinking water. The movement of methylene chloride in ground water may result in contamination of surface waters, and potential dermal and ingestion exposures. These exposure routes include the following:

- Surface waters may also be used as drinking water supplies resulting in direct ingestion exposure.

- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposure through bioaccumulation.

- Recreational use of these waters may result in dermal exposure.

- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the human consumption of meats and poultry could then result in ingestion exposure.

In general, exposures associated with surface water contamination can be expected to be lower than exposures from drinking contaminated ground water for two reasons. First, the Henry's law constant for methylene chloride suggests that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for methylene chloride is low and no significant bioaccumulation would be expected in fish or domestic animals.

### 1.2.4 Other Sources of Human Exposure

Methylene chloride has a variety of uses as a solvent. Several of these can result in direct human exposure. Page and Kennedy (88) reported methylene chloride in a variety of spices, the highest being 83 mg/kg in Cassia. Methylene chloride is used for extraction of some food material. In addition, methylene chloride may be found in paint removers and aerosol household products. The use of these products could result in dermal or inhalation exposures.

The production and use of methylene chloride has led to its presence in the atmosphere. Brodzinsky and Singh (84) summarized available air monitoring data for a variety of pollutants. For methylene chloride, they reported 850 data points. In rural and remote areas, the median concentration was 0.16 $\mu g/m^3$ and in urban and suburban areas, the median concentration was 2.2 $\mu g/m^3$; in source-dominated areas, it was 0.94 $\mu g/m^3$. These authors noted that methylene chloride was a ubiquitous air contaminant and that there was extreme variability in the data.

## 1.3 HUMAN HEALTH CONSIDERATIONS

### 1.3.1 Animal Studies

#### 1.3.1.1 Carcinogenicity

In a 2-year inhalation study, Burek et al. (85) exposed male and female Sprague-Dawley rats and Golden Syrian hamsters to technical grade methylene chloride (purity >99%) at vapor levels of 500, 1500 or 3500 ppm, 6 hours daily, 5 days per week. This exposure did not produce any compound-related lesions in the

hamsters or adversely affect their survival. In female rats, dose-related increases were observed in the total number of benign mammary gland tumors. However, the number of female rats with benign tumors was not increased above controls. Increased incidences of mammary tumors were also found in male rats exposed to 1500 or 3500 ppm but they were not as pronounced as those seen in the females. At these same exposure levels, male rats also had increased incidences of salivary gland sarcomas. This may have been related to a viral salivary gland infection in these animals, although these tumors were not detected among similarly infected females (85,635).

The National Toxicology Program recently completed a 2-year inhalation study in F344/N rats and B6C3F$_1$ mice which were exposed to methylene chloride (purity >99%) 6 hours daily, 5 days per week. Mice were exposed to 2000 or 4000 ppm while rats were exposed to 1000, 2000 or 4000 ppm. The survival of male rats, including controls, was low - 18 to 34% - with most of the deaths occurring after the 86th week. This decreased survival is believed to be related to a high incidence (52 to 70%) of leukemias in both treated and control male rats. Some evidence of carcinogenicity in male rats was shown by an increased dose-related incidence of neoplasms of the mammary gland ranging from 4 to 10%. There was clear evidence of carcinogenicity in female rats, i.e., a 26 to 46% incidence of mammary gland neoplasms. The survival of female rats was somewhat reduced and there was an increased incidence of mononuclear cell leukemia (46%) at the mid- and high-dose levels which was statistically significant by age-adjusted analyses (635).

In both male and female mice, there was clear evidence of carcinogenicity as shown by increased dose-related incidences of hepatocellular neoplasms and alveolar/bronchiolar neoplasms. The incidences of liver neoplasm were 33% and 49% in low-dose females and males, respectively, and 83% and 67% at the high-dose level. Control females and males had incidences of 6% and 44%, respectively. The incidences of lung adenomas/carcinomas, combined, were 54% and 63% for low-dose males and females, respectively, and 80% and 85% at the high dose, with a 6 to 10% incidence in controls (635).

IARC (3315) has listed methylene chloride in category 2B (inadequate evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals) in its weight-of-evidence ranking for potential carcinogens.

### 1.3.1.2 Genotoxicity

Methylene chloride was mutagenic in 2 strains of Salmonella typhimurium both in the presence and absence of a liver microsomal activating system (87, 95, 3653). It was also effective in inducing mitotic recombination and gene conversion in Saccharomyces cerevisiae, D7 (3099).

Methylene chloride also induced chromosomal aberrations in Chinese hamster ovary cells (86), but was negative in inducing point mutations at the HPRT locus in CHO and V79 Chinese hamster cells in culture (3344). Mixed results have been

obtained in the sex-linked recessive lethal assay in Drosophila and in cell transformation studies in vitro (635).

In vivo studies have been negative for gentoxicity: methylene chloride did not induce unscheduled DNA synthesis in the livers of mice or rats treated either by inhalation or by gavage (3727); there was no significant increase in micronuclei of the bone marrow cells of male and female mice treated by gavage with methylene chloride at the maximum tolerated dose or less (3638); and there was no increase in chromosomal aberrations in the bone marrow cells of male and female rats exposed via inhalation to methylene chloride concentrations of 500, 1500, or 3500 ppm for 6 months (85).

#### 1.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

One study demonstrated that methylene chloride crosses the placenta in experimental animals. Withey and Karpinski (3843) exposed rats to methylene chloride by inhalation for 5 hours on day 17 of gestation to seven concentrations ranging from 107 to 2,961 ppm. The agent crossed the placenta, but fetal levels were consistently less than half of maternal levels.

Schwetz et al. (115) exposed mice and rats to vapor levels of 1,250 ppm of methylene chloride for 7 hr/day on days 5 through 15 of gestation. Maternal and fetal toxicity were observed in both species, but there was no evidence of teratogenicity. In another study, Hardin and Manson (3270) found no teratological effects in rats that had been exposed to 4,500 ppm methylene chloride vapors for 6 hr/day for 3 weeks prior to pregnancy and through the first 17 days of gestation. However, symptoms of maternal toxicity and fetal weight reduction were evident. Exposed offspring that were retained for postnatal evaluation (3076) exhibited more rapid behavioral habituation, but postnatal growth, activity, and avoidance learning were not impaired.

Nishio et al. (3505) treated rats with dietary concentrations of 4.0, 0.4, or 0.04% of methylene chloride during their entire pregnancy. A significant reduction in maternal weight was observed in the 4.0% group. No differences were observed in numbers of implantations and resorptions, malformations, delayed ossification, or dilation of the renal pelvis at any level. Methylene chloride (125 ppm) added to the drinking water of male and female rats for 91 days caused no effects on the estrous cycle or on reproduction (3041).

#### 1.3.1.4 Other Toxicologic Effects

##### 1.3.1.4.1 Short-term Toxicologic Effects

Methylene chloride has a low to moderate acute oral toxicity in laboratory animals. The oral $LD_{50}$ for methylene chloride in rats is about 1600 mg/kg (3933). Another report noted an oral $LD_{50}$ value of 167 mg/kg for the rat (47). Mice given oral doses of 133, 333 or 665 mg/kg/day for 14 days exhibited histological liver

changes, predominantly cytoplasmic vacuolation, at the two upper dosage levels. No kidney changes were observed (636). The inhalation $LC_{50}$ for mice was approximately 15,000 ppm for an 8-hour exposure with all animals surviving at approximately 11,000 ppm (12). An $LC_{50}$ value of 88,000 mg/m$^3$ for 30 minutes was recorded for rats (3933). In cats exposed to 10,000 ppm, light narcosis occurred in 220 minutes and deep narcosis in 293 minutes. Both cats and rabbits tolerated exposure to 6000-7000 ppm, 8 to 9 hours daily for 4 weeks (12).

Cardiac arrhythmias attributed to sensitization of the myocardium have not been observed in dogs exposed to 10,000 or 20,000 ppm methylene chloride (38).

Methylene chloride is metabolized to carbon monoxide in both animals and man, leading to the formation of carboxyhemoglobin (COHb) and subsequent oxygen deprivation. The metabolism and pharmacokinetics of inhaled methylene chloride are dose-dependent and saturable but the relationship between its metabolism and toxicity remains unclear. In a study by McKenna et al., rats exposed to vapor levels of 50 ppm for 6 hours had COHb levels of 3%. COHb levels of 10 to 13% were seen after a 6-hour exposure to 500 ppm. However, no further increase in COHb above this level was observed even after exposure to 1500 ppm for 6 hours (637).

If allowed to evaporate, methylene chloride is mildly irritating to the skin of rabbits on repeated contact (12).

Instillation of 0.1 mL methylene chloride into the eyes of rabbits produced moderate inflammation of the eyelids and conjunctiva, excessive lacrimation and chemosis. Corneal thickness, an indicator of corneal injury, peaked at 6 hours and did not return to normal for 8 to 9 days. Similar, but less persistent effects were seen with 0.01 mL methylene chloride (640).

#### 1.3.1.4.2 Subchronic and Chronic Toxicity

Subchronic and chronic exposure of animals to methylene chloride results in liver and CNS toxicity.

Ninety-day drinking water studies were performed in Fischer 344 rats (20/sex/group) and B6C3F$_1$ mice (20/sex/group) (3360). The rats received methylene chloride doses of 166, 420, or 1200 mg/kg/day (males) and 209, 607, and 1469 mg/kg/day (females); the mice received methylene chloride doses of 226, 587, or 1911 mg/kg/day (males) and 231, 586, or 2030 mg/kg/day (females). The lowest toxic effect levels after 90 days of treatment were found to be approximately 166 and 586 mg/kg for rats and mice, respectively, with the liver as the target organ for both species. Histopathological effects included hepatocyte vacuolation, central lobular fatty change, necrosis with fatty change and pigment deposition.

In a chronic oral study, Fischer 344 rats administered 50 to 250 mg/kg/day DCM in drinking water for 2 years developed areas of hepatic cellular alteration and an increased incidence of foci (3489). In addition, fatty changes of the liver were

observed in rats treated with 125 and 250 mg of methylene chloride/kg/day for 78 and 104 weeks. No liver effects were observed at 5 mg/kg/day.

An inhalation study was conducted, in which Mongolian gerbils were exposed to methylene chloride vapors (210 ppm) continuously for 3 months (3347). Following a four-month postexposure solvent-free period, DNA content in the brain was determined as a measure of neurotoxicity. Although the total protein concentrations in different brain regions were not significantly altered in the exposed animals, the DNA concentrations in the hippocampal regions were significantly decreased ($p \leq 0.05$, Fisher's permutation test) when compared to air-exposed controls.

Haun et al. and MacEwen et al. (638, 639) exposed several species of animals to 1000 or 5000 ppm methylene chloride vapors continuously for periods up to 14 weeks. Results indicated severe weight loss in all species and signs of CNS depression in dogs and monkeys at the 1000 ppm exposure level and in dogs, monkeys, rats and mice at the 5000 ppm level. A significant number of dogs at 1000 ppm and mice at 5000 ppm died. Liver lesions associated with hepatic failure were noted in dogs, rats and mice. Haun et al. (639) also reported liver injury in monkeys exposed to 1000 ppm under the same conditions. Except for increased COHb levels, dogs and monkeys were unaffected by a continuous 100-day exposure to either 25 or 100 ppm. Mice exposed to 25 ppm were also without effect.

Methylene chloride-induced non-oncogenic effects were also observed in rats and hamsters, exposed to the chemical for two years (85). Both species, exposed to concentrations of 500, 1500, or 3500 ppm, exhibited increased carboxyhemoglobin values at all doses ($p < 0.05$, Dunnett's test); however, there was no dose response relationship. Mortality among female rats exposed to 3500 ppm was statistically significantly increased from the 18th to the 24th month. The liver was the major target organ for toxicity in the rats, with effects clearly occurring at all exposure levels at the 12-month interim kill and after; hepatic effects included elevated liver weights, increased hepatocellular vacuolization, multinucleated hepatocytes (females only), and increased hepatocellular necrosis. On the other hand, the hamsters lacked evidence of definite target organ toxicity, and both rats and hamsters exhibited statistically significant decreases in amyloidosis and other age-related pathologic alterations.

### 1.3.2 Human and Epidemiologic Studies

#### 1.3.2.1 Short-term Toxicologic Effects

In experiments with human volunteers, inhalation of 500 to 1000 ppm for 1 to 2 hours resulted in lightheadedness and a sustained elevation of COHb levels (38). Inhalation of 300-750 ppm for 3-4 hours was reported to result in decreased performance of psychomotor tasks (91).

No deleterious effects upon the health or performance of adults could be detected with repeated exposure to vapor levels of 250 ppm or less, 7.5 hours per day for 10 days (89).

Human exposure to vapor levels of 20,000 ppm for 30 minutes resulted in pulmonary edema and deep narcosis (56). Lower, but unknown concentrations, have caused symptoms of lightheadedness, weakness, nausea and drunken behavior (38).

Exposure to methylene chloride has resulted in deaths in industrial situations. Moskowitz and Shapiro (641) reported 4 cases of acute exposure with one fatality. The exposure times were known to range from less than one hour to three hours but the methylene chloride concentrations were unknown. The lung tissue of the dead worker was found to contain 0.1 mL of methylene chloride per 500 g tissue. The 3 survivors remained unconscious for 2.5 hours after they were removed from the work area. Two of the men were hospitalized for 4 days and the other, for 8 days. They exhibited signs of eye, lung or respiratory tract irritation. They had low hemoglobin levels and red blood cell counts. All other findings appeared normal.

Hughes reported that a worker exposed to unknown vapor levels of methylene chloride for 4 hours experienced eye irritation, fatigue, lightheadedness, shortness of breath, nausea, chest pain and pulmonary edema (642).

Acute renal tubular necrosis was reported in a man exposed to vapors of a tile remover containing methylene chloride, mineral spirits and methanol. Neither the percent composition of the product nor the vapor level of exposure were indicated. After 2 days of exposure, the man reported symptoms of intermittent abdominal pain associated with nausea, vomiting and loss of appetite. He further reported intermittent chills, mild diarrhea and mild headache but no fever. A kidney biopsy revealed widespread degeneration of the epithelial cells of both the proximal and distal tubules consistent with a diagnosis of acute tubular necrosis. Elevated serum enzyme levels (LDH, SGOT, SGPT) suggested hepatocellular injury as well. The victim recovered after 14 days (685).

Liquid methylene chloride is irritating to the skin on repeated contact. When splashed in the eye, it is painfully irritating but not likely to cause serious injury (38).

### 1.3.2.2 Chronic Toxicologic Effects

Weiss (643) reported that after 5 years of exposure to vapor concentrations ranging from 660-3600 ppm, a chemist developed forgetfulness, insomnia and auditory and visual hallucinations. Another report noted that in two workers exposed to unknown levels of methylene chloride intermittently for 13 to 20 years, one experienced irregular, severe leg and arm pain, dizziness, fatigue, loss of appetite and poor night vision. The other experienced drowsiness, headache and tingling of the hands and feet (644).

A retrospective mortality study conducted by Friedlander et al. (645) revealed no increased frequency of tumors or other causes of death in individuals occupationally exposed to methylene chloride. Over 30% of the employees had worked a minimum of 20 years and had occupational timed-weighted-average exposures ranging from 30-125 ppm.

Another recent retrospective cohort study found no significant increase in overall mortality or deaths due to ischemic heart disease in workers exposed to methylene chloride vapor levels of 140 to 475 ppm. The length of exposure was not indicated (646).

Methylene chloride crosses the placenta and has been found in breast milk of women working as gluing operatives (3473). Taskinen (3700) conducted a retrospective epidemiological study to assess the effect of working in the pharmaceutical industry on rate of spontaneous abortion. Exposure to chemicals was more common among those women experiencing abortion than among the controls. For methylene chloride, the increase in odds ratio was of borderline significance ($p=0.06$).

### 1.3.3 Levels of Concern

For the protection of human health, the U.S. Environmental Protection Agency has specified a water quality criterion of zero for halomethanes, including methylene chloride, based on the non-threshold assumption for these chemicals. Since the zero level may not be attainable, the concentration of halomethanes calculated to result in incremental lifetime cancer risks of 1.0E-05, 1.0E-06 and 1.0E-07 were estimated to be 1.9, 0.19 and 0.019 $\mu$g/L, respectively, based on ingestion of both water and contaminated aquatic organisms (355). Risk estimates are expressed as a probability of cancer after a lifetime daily consumption of two liters of water and 6.5 g of fish that have bioaccumulated methylene chloride. Thus a risk of 1.0E-05 implies that a lifetime daily consumption of two liters of drinking water and 6.5 g of fish that have bioaccumulated methylene chloride at the criterion level of 1.9 $\mu$g/L of methylene chloride would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 people exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

The NTP (635) classifies methylene chloride as presenting clear evidence of carcinogenic activity in animals. IARC (3315) lists this compound as a category 2B carcinogen (inadequate evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals).

As a result of recent NTP findings of carcinogenic activity for methylene chloride, in an advance notice of Proposed Rulemaking, EPA (788) announced that it intends to conduct a comprehensive and integrated regulatory review of methylene chloride to determine whether methylene chloride presents an unreasonable risk to

human health or the environment. Appropriate regulatory actions will be taken by OSHA, FDA and CPSC, as well as EPA, based on the outcome of this review.

As an estimate of a daily oral exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime, the EPA (3742) has calculated an RfD (reference dose) of 60 μg/kg/day.

OSHA (3539) has set an 8-hour time-weighted-average of 500 ppm with a ceiling limit of 1000 ppm and a peak of 2000 ppm for 5 minutes in any 2 hours. The ACGIH (3005) recommends a threshold limit value of 50 ppm (175 $mg/m^3$) to prevent excessive concentrations of carboxyhemoglobin. It is also a suspected human carcinogen (3005).

### 1.3.4 Hazard Assessment

Inhalation exposures to 1000 to 4000 ppm methylene chloride resulted in increased incidences of alveolar/bronchiolar neoplasms and hepatocellular carcinomas in $B6C3F_1$ mice and an increased incidence of benign neoplasms of the mammary gland in F344/N rats (635). There is also evidence that methylene chloride is weakly genotoxic. There is no evidence to suggest teratogenic activity, even at doses that produce slight maternal toxicity (12, 115).

Animal studies indicate that acute ingestion of high doses of methylene chloride (333 or 665 mg/kg/day) results in histological liver changes in mice (636). High level inhalation exposures result in CNS effects and increased levels of carboxyhemoglobin which may subsequently lead to oxygen deprivation. Continuous long-term inhalation up to 14 weeks at 1000-5000 ppm resulted in liver lesions associated with hepatic failure in 3 animals species (638, 639).

Human volunteers exposed to methylene chloride concentrations of 500 to 1000 ppm for one to two hours experienced lightheadedness and sustained, elevated carboxyhemoglobin levels (38) but no deleterious effects were noted upon repeated exposure to vapor levels of 250 ppm for 7.5 hours a day for 10 days (89). High level exposures have resulted in death in industrial situations. No ingestion data were found.

In general, the toxic effects associated with human exposure to methylene chloride indicate a low order of toxicity, with rapid recovery and no sequelae other than elevated carboxyhemoglobin levels. An epidemiologic mortality analysis of workers exposed to methylene chloride (30-125 ppm TWA) for a minimum of twenty years indicated no increased excess cause of death. However, because of the NTP data (635), and in the absence of adequate data in humans, it appears prudent to regard methylene chloride as if it presented a carcinogenic risk to humans.

## 1.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of methylene chloride concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of methylene chloride, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. Field samples can be contaminated by diffusion of volatile organics such as methylene chloride through the septum of the sample container during shipment and storage and as a result of its use as a common laboratory solvent. Therefore, field blanks and laboratory blanks are important to insure against false positive identifications of methylene chloride. In addition to the targeted field samples and blanks, duplicate samples and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of methylene chloride, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8240 and 8010 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the methylene chloride from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the methylene chloride and to transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; methylene chloride is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 8240, and 1624). For samples that contain high concentrations direct injection may also be used. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184).

The EPA procedures recommended for methylene chloride analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photoionization or electrolytic conductivity detection or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detection.

Typical methylene chloride detection limits that can be obtained in wastewaters and nonaqueous samples (wastes, soils, etc.) are shown below. Detection limits were not given for 8010. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Nonaqueous Detection Limit |
|---|---|
| 0.25 μg/L (Method 601) | 5 μg/kg (Method 8240) |
| 2.8 μg/L (Method 624) | |
| 10 μg/L (Method 1624) | |
| 5 μg/L (Method 8240) | |

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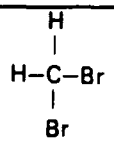
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| COMMON SYNONYMS: | CAS REG. NO.: 74-95-3 FORMULA: $CH_2Br_2$ | AIR W/V CONVERSION FACTOR at 25 °C |
|---|---|---|
| Dibromomethane<br>Methylene bromide<br>Methylene dibromide<br>RCRA waste number U068 | NIOSH NO: PA7350000<br>STRUCTURE:  | 7.11 mg/m³ ≈ 1 ppm;<br>0.1406 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 173.85 |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as dibromomethane with cyanides, mercaptans or other organic sulfides typically generate heat, while those with amines, azo compounds, hydrazines, caustics or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases and fires. Those with alkali or alkaline earth metals, certain other chemically active metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid, heavy (at 20 °C) | (12) |
| | • Color: Clear, colorless | (23) |
| | • Odor: No data | |
| | • Odor Threshold: No data | |
| | • Density: 2.4956 g/mL (at 20 °C) | (14,59) |
| | • Freeze/Melt Point: -52.70 °C | (14,21) |
| | • Boiling Point: 96.90 °C | (14,21) |
| | • Flash Point: Nonflammable | (59) |
| | • Flammable Limits: Nonflammable | (12) |
| | • Autoignition Temp.: Nonflammable | (12) |
| | • Vapor Pressure: 3.70E+01 mm Hg (at 20°C) | (1219) |
| | • Satd. Conc. in Air: 3.5265E+05 mg/m³ (at 20 °C) | (1219) |
| | • Solubility in Water: 1.18E+04 mg/L (at 20 °C) | (12) |
| | • Viscosity: 1.016 cp (at 20 °C) | (48) |
| | • Surface Tension: No data | |
| | • Log (Octanol-Water Partition Coeff.): 1.64 | (1219) |
| | • Soil Adsorp. Coeff.: 2.60E+01 | (1219) |
| | • Henry's Law Const.: 7.50E-04 atm · m³/mol (at 20°C) | (1219) |
| | • Bioconc. Factor: 2.10E+00 (estim) | (659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL-WATER SYSTEM | Dibromomethane is expected to be relatively mobile through surface soils of 1-2% organic carbon and highly mobile through deep soils or sandy soils. Removal by volatilization is expected to be important for material on the surface or in the soil-air. Biodegradation is not expected to be a significant transformation process. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of dibromomethane to ground-water drinking water supplies. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (12)<br>No data were found for human exposure. Animal experiments indicate slight irritation of eyes and skin with acute exposure and liver and kidney toxicity with repeated exposure.<br><br>Acute Toxicity Studies: (3504)<br><br>INHALATION:<br>$LC_{50}$ 40 $g/m^3 \cdot 2$ hr Rat<br><br>ORAL:<br>$LD_{50}$ 108 mg/kg Rat<br><br>Long-Term Effects: Liver and kidney injury<br>Pregnancy/Neonate Data: No data<br>Genotoxicity Data: Limited evidence is positive<br>Carcinogenicity Classification:<br>IARC - No data<br>NTP - No data<br>EPA - Group D (not classifiable as to human carcinogenicity) |

| | |
|---|---|
| HANDLING PRECAUTIONS | Handle chemical only with adequate ventilation. •There are no formal guidelines available for this chemical with respect to respirator use. Use a self-contained breathing apparatus with a full facepiece (or the equivalent) where there is any doubt as to the efficacy of gas masks or cartridge-type respirators. •Chemical goggles if there is a probability of eye contact. •Appropriate clothing to prevent repeated or prolonged skin contact. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

### AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): none established
- AFOSH PEL (8-hr TWA): none established

Criteria
- NIOSH IDLH (30 min): none established
- ACGIH TLV (8-hr TWA): none established
- ACGIH STEL (15-min): none established

### WATER EXPOSURE LIMITS:

Drinking Water Standards
None established

EPA Health Advisories and Cancer Risk Levels
None established

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria
- Human Health (355)
  - None established as dibromomethane is not a priority pollutant.
- Aquatic Life (355)
  - None established as dibromomethane is not a priority pollutant.

### REFERENCE DOSES:

No reference dose available.

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Safe Drinking Water Act (SDWA)

Dibromomethane was on the original list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986. In January, 1988, EPA removed it from this list and, as required under the SDWA, added it to the first drinking water priority list for which NPDWRs will be developed (3781). It is designated an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3771). In states with an approved Underground Injection Control program, a permit is required for the injection of dibromomethane-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

Dibromomethane is identified as a toxic hazardous waste (U068). and is listed as a hazardous waste constituent (3783, 3784). Dibromomethane is included on EPA's groundwater monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their groundwater for chemicals on this list when suspected contamination is first detected, and annually thereafter (3775). Effective July 8, 1987. the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786).

Toxic Substances Control Act (TSCA)

Manufacturers, processors, or importers who possess health and safety studies on dibromomethane must submit them to EPA (3789). Under TSCA Section 4, EPA requires that manufacturers and processors of dibromomethane perform human health effects and chemical fate testing in support of the RCRA program (3792).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
Dibromomethane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of dibromomethane must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated dibromomethane as a hazardous substance with a reportable quantity of 454 kg. subject to requirements for packaging. labeling. and transportation (3180).

- State Water Programs
  No state regulations

Proposed Regulations

- Federal Programs
  No proposed federal regulations are pending.

- State Water Programs
  MOST STATES
  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with state officers is advised. Changes are projected for 1989-90 (3683).

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentation of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
Dibromomethane is listed as a Class II/b harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Dibromomethane is classified as a harmful substance and is subject to packaging and labeling regulations.

## 2.1 MAJOR USES

Dibromomethane has limited use as a chemical intermediate, as a chemical solvent for fats, waxes and resins, and as an ingredient of fire-extinguishing fluids and gage fluids (21,59,12).

## 2.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 2.2.1 Transport in Soil/Ground-water Systems

#### 2.2.1.1 Overview

Dibromomethane will be relatively mobile in the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical).

Transport pathways of low soil concentrations can be generally assessed by estimating equilibrium partitioning as shown in Table 2-1. These calculations predict the partitioning of dibromomethane among soil particles, soil water and soil air. The estimates for the unsaturated topsoil model indicate that significant amounts of dibromomethane are expected to partition to the soil-water and soil-air phases, and can thus be transported through these phases by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion (e.g., through the soil-air pores up to the ground surface, and subsequent removal by wind). In saturated, deep soils (containing no soil air and negligible soil organic carbon), most of the dibromomethane (approximately 90%) is predicted to be present in the soil-water phase (Table 2-1) and available for transport with flowing ground water. Ground water underlying dibromomethane-contaminated soils with low organic content is particularly vulnerable to contamination.

#### 2.2.1.2 Sorption on Soils

The mobility of dibromomethane in the soil/ground-water system (and its eventual migration into aquifers) is affected by the extent of sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic content of the soil water.

There were no available data on the extent of sorption of dibromomethane on soils; data for other halomethanes indicate some sorption. Schwarzenbach et al. (77) determined retardation rates, which represent interstitial water velocity/pollutant velocity in the soil, for several chlorinated organics with higher $K_{oc}$'s than dibromomethane. The data indicate some retention in soils having 1-2% organic carbon content and little or no retention in soils having less than 0.1% organic carbon. Assuming analogous conditions, adsorption of dibromomethane, particularly to deep soils, is not expected to be significant.

TABLE 2-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR DIBROMOMETHANE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment[a] | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 25°C | 82.0 | 16.5 | 1.5 |
| Saturated deep soil[d] | 9.8 | 90.2 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 26 (Arthur D. Little, Inc. estimate).
c) Henry's law constant taken as 7.5E-04 atm · $m^3$/mol at 20°C (Arthur D. Little, Inc. estimate).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

### 2.2.1.3 Volatilization from Soils

Transport of dibromomethane vapors through the air-filled pores of unsaturated soils may be an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H were observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of dibromomethane from surface soils.

No information was available for the two other physicochemical properties influencing dibromomethane volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

No information regarding the rate of volatilization of dibromomethane from soils was available. Half-lives on the order of 20-90 minutes have been reported for dichloromethane in stirred aqueous solutions (10); half-lives for dibromochloromethane from rivers and streams have been reported to range from 43 minutes to 16.6 days depending on the re-aeration rate (72). Dibromomethane is expected to be less volatile than either of these compounds since it has a lower Henry's law constant. Compared to volatilization from well-stirred aqueous solutions, volatilization of some chlorinated organics from surface soils has been shown to be slower by approximately one order of magnitude (82).

### 2.2.2 Transformation Processes in Soil/Ground-water Systems

Data specific to the transformation of dibromomethane in soil/ground-water systems were not available. Photolysis, oxidation and hydrolysis are not expected to occur at significant rates.

Most references indicate that low molecular weight chloroaliphatics are not metabolized by microorganisms (10). However, Thom and Agg (80) have included several halomethanes in a list of organic chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as dibromomethane is very low and drops off sharply with increasing depth. Thus, biodegradation should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

### 2.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that dibromomethane in the environment is moderately volatile from aqueous solutions, is weakly sorbed to soil, and has no significant potential for bioaccumulation. Dibromomethane on the soil surface may volatilize, but that portion not removed by volatilization may eventually migrate to ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of dibromomethane from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. In addition, the potential for ground-water contamination should be high, as evidenced by the low $K_{oc}$, potentially resulting in ingestion exposures through drinking water. There is, however, no evidence of such exposure, either at Superfund sites (83) or from monitoring of ground-water supplies (531). These results may be due to the low production volume for this chemical.

Movement of dibromomethane in ground water may reach surface waters. In such a situation, several other exposure pathways are possible:

- Surface waters may be used as drinking water supplies and result in direct ingestion exposure;
- Aquatic organisms residing in these waters and bioaccumulating this chemical may be consumed, also resulting in ingestion exposures;
- Recreational use of these waters may result in dermal exposures; and
- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground water for two reasons. First, the Henry's law constant for dibromomethane suggests that it may volatilize upon reaching surface waters. Secondly, the bioconcentration factor for dibromomethane is very low, suggesting bioaccumulation in aquatic organisms or domestic animals will be insignificant.

### 2.2.5 Other Sources of Human Exposure

No evidence of exposure to dibromomethane was found in the literature as a result of air, drinking water or food contamination. This suggests that other sources of exposure to this compound are limited.

## 2.3 HUMAN HEALTH CONSIDERATIONS

### 2.3.1 Animal Studies

#### 2.3.1.1 Carcinogenicity

No data are available.

### 2.3.1.2 Genotoxicity

Dibromomethane was mutagenic in two strains of Salmonella typhimurium (266, 3653, 3541), induced reversions to tyrosine prototrophy in Escherichia coli (3541) and was weakly mutagenic in the yeast strain Saccharomyces cerevisiae D3 (12).

### 2.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No data are available.

### 2.3.1.4 Other Toxicologic Effects

#### 2.3.1.4.1 Short-term Toxicity

Dibromomethane has a relatively low acute oral toxicity; the oral $LD_{50}$ for rats is greater than 1000 mg/kg bw (12). An $LD_{50}$ value of 3738 mg/kg was found in mice given dibromomethane by subcutaneous injection and an $LD_{Lo}$ of 5000 mg/kg was reported for rabbits given the compound by rectal administration (47). Inhalation of dibromomethane vapors can induce anesthesia and even death (12).

Dibromomethane is slightly irritating to the eyes and skin of rabbits but does not appear to be significantly absorbed, even after repeated applications (12).

#### 2.3.1.4.2 Chronic Toxicologic Effects

Little information is available on the toxicity of dibromomethane. Reported to be more toxic than methylene chloride (12), it appears to be among the dihalomethanes known to be metabolized to carbon monoxide in rats, resulting in elevated blood carboxyhemoglobin levels (17).

Dibromomethane has the capacity to produce significant liver and kidney injury in animals on repeated exposure (12). In a limited study, a small group of rabbits (number undefined) were orally administered 300 mg/kg/day (60 doses in 92 days) of dibromomethane. No effect on body weight gain or liver histopathology was recorded. Similar treatment with 400 mg/kg or more produced marked anesthesia (526).

A series of limited inhalation studies was also conducted by this same group (526). In one experiment, one rabbit of each sex was exposed 54 times in 73 days to a nominal concentration of 1000 ppm dibromomethane. There were no observed adverse effects but liver and kidney degeneration were noted at autopsy, and blood bromide was elevated (526). In another test, ten rats of each sex received 30 to 40 seven-hour exposures to 1000 ppm. Incoordination and staggering were observed during exposure. Failure to gain weight, possible increased mortality and pathological changes in the lungs, liver and kidneys were noted (526). A third experiment involved exposure of rats and rabbits to 200 ppm for 79 exposures in 114 days.

Observed effects were reduced at this lower exposure level, but liver weight was increased in male rats and pathological changes were observed in the livers and kidneys of both rats and rabbits (526). The final test in this series involved 60 to 70 daily six-hour inhalation exposures to 25, 75 or 125 ppm dibromomethane over a 90-day period. No organ pathology was found in either rats or dogs. Blood bromide was markedly elevated as was carboxyhemoglobin (526).

### 2.3.2 Human and Epidemiologic Studies

No data with regard to human exposure to dibromomethane were located.

### 2.3.3 Levels of Concern

No criteria or standards have been established to date regarding this chemical. In view of the scarcity of data available on the adverse health effects and effect levels associated with exposure to dibromomethane, estimates of exposure levels of concern cannot be made with any confidence.

### 2.3.4 Hazard Assessment

The extent and quality of health effects data available for dibromomethane are inadequate. Limited unpublished findings suggest a no-observed-adverse-effect-level of 300 mg/kg/day for a small group of rabbits administered 60 oral doses over a 90-day period (526). Rats and dogs exposed by inhalation to 25-125 ppm dibromomethane exhibited elevated carboxyhemoglobin levels, and liver and kidney pathology was observed in both rats and rabbits exposed to dibromomethane concentrations of 200 ppm (526). The limited nature of these unpublished studies, linked with positive mutagenic findings in bacterial and yeast systems, do not allow reliable estimates of potential health hazards associated with human exposure to dibromomethane to be established.

## 2.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of dibromomethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of dibromomethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected within 14 days of sampling. Recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality assurance samples such as field blanks, duplicates, and spiked matrices should be included in the analytical program.

Dibromomethane is not included among the EPA-designated priority pollutants. However, EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63) would be appropriate methods of choice for the analysis of dibromomethane in aqueous samples. An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the dibromomethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the dibromomethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; dibromomethane is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). Direct injection may also be used for samples that contain high concentrations of chemical. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors. In addition to all these methods, dibromoethane has also been determined by concentrating it on an adsorption column from which it is eluted with a solvent, and analyzed by GC electron capture detection (3395).

The EPA procedures recommended for dibromomethane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Dibromomethane detection limits for the various methods were not determined but would be in the range of 1-10 $\mu$g/L for aqueous samples and 1-10 $\mu$g/kg for nonaqueous samples.

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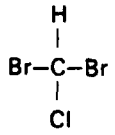
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| COMMON SYNONYMS: | CAS REG. NO.: 124-48-1 FORMULA: $CHBr_2Cl$<br>NIOSH NO: PA6360000<br>STRUCTURE:  | AIR W/V CONVERSION FACTOR at 25 °C<br>8.49 mg/m³ ≈ 1 ppm;<br>0.1177 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 208.29 |
|---|---|---|
| CDBM<br>Chlorodibromomethane<br>Dibromochloromethane<br>Dibromomonochloromethane<br>Monochlorodibromomethane | | |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as dibromochloromethane with cyanides, mercaptans or other organic sulfides typically generate heat, while those with amines, azo compounds, hydrazines, caustics or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases and fires. Those with alkali or alkaline earth elemental metals, certain other chemically active metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires. When heated to decomposition, emits toxic fumes of chloride and bromide (511, 505, 51). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid, heavy (at 20°C) | (54) |
| | • Color: Clear to pale yellow | (54) |
| | • Odor: No data | |
| | • Odor Threshold: No data | |
| | • Density: 2.3800 g/mL (at 20 °C) | (23) |
| | • Freeze/Melt Point: -22.00 °C | (1,14) |
| | • Boiling Point: 116.00 °C | (1) |
| | • Flash Point: None | (23) |
| | • Flammable Limits: Nonflammable | (23) |
| | • Autoignition Temp.: Nonflammable | (23) |
| | • Vapor Pressure: 1.80E+01 mm Hg (at 20 °C) | (1219) |
| | • Satd. Conc. in Air: 2.0600E+05 mg/m³ (at 20 °C) | (1219) |
| | • Solubility in Water: 3.08E+03 mg/L (at 20 °C) | (1219) |

| | |
|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Viscosity: No data<br>• Surface Tension: No data<br>• Log (Octanol-Water Partition Coeff.): 2.24 (33)<br>• Soil Adsorp. Coeff.: 8.40E+01 (33)<br>• Henry's Law Const.: 7.83E-04 atm · $m^3$/mol (81)<br>• Bioconc. Factor: 8.30E+00 (estim) (659) |
| PERSISTENCE IN THE SOIL-WATER SYSTEM | Dibromochloromethane is expected to be somewhat mobile in surface soils and highly mobile in deep soils or sandy soils. Removal by volatilization is important for material near the surface or in the soil-air. Biodegradation in natural soils is not expected to be significant. |
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of dibromochloromethane to groundwater drinking water supplies. Evidence of such migration is limited, probably because this compound is not generally a major constituent at hazardous waste sites. Nevertheless, this compound is frequently found in drinking water. Its prevalence in drinking water has been attributed to the production of trihalomethanes during chlorination procedures. Inhalation resulting from volatilization from surface soils may also be an important pathway. |
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (92, 93)<br>There are no data available on the effects of human exposure to dibromochloromethane. One study noted sedation in mice following ingestion of a nonlethal dose. A slight elevation of blood carbon monoxide is seen in rats |

| | |
|---|---|
| HEALTH HAZARD DATA (Cont.) | Acute Toxicity Studies: (3504)<br><br>ORAL: $LD_{50}$ 848 mg/kg Rat<br><br>Long-Term Effects: Liver and kidney injury<br>Pregnancy/Neonate Data: Negative<br>Genotoxicity Data: Suggestive evidence of genotoxic activity<br>Carcinogenicity Classification:<br>IARC - No data<br>NTP - Some evidence in female mice, equivocal evidence in male mice, no evidence in rats<br>EPA - No data |

| | |
|---|---|
| HANDLING PRECAUTIONS | Handle chemical only with adequate ventilation. •There are no formal guidelines available for this chemical with respect to respirator use. Use a self-contained breathing apparatus with a full facepiece (or the equivalent) where there is any doubt as to the efficacy of gas masks or cartridge-type respirators. •Chemical goggles if there is a probability of eye contact. •Appropriate clothing to prevent repeated or prolonged skin contact. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

### AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): none established
- AFOSH PEL (8-hr TWA): none established

Criteria
- NIOSH IDLH (30 min): none established
- ACGIH TLV (8-hr TWA): none established
- ACGIH STEL (15-min): none established

### WATER EXPOSURE LIMITS:

Drinking Water Standards (296)
Under the National Interim Primary Drinking Water Regulations, the maximum contaminant level (MCL) for total trihalomethanes is 0.10 mg/L. This includes dibromochloromethane in combination with three other trihalogenated methanes, bromoform, dichlorobromomethane and chloroform. This MCL applies to community water systems which serve a population of 10,000 people or more and which add a disinfectant as part of their treatment process.

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

EPA Health Advisories and Cancer Risk Levels
None established

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria

- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms (1E-05, 1E-06, 1E-07 cancer risk), 1.9, 0.19, 0.019 μg/L total halomethanes.
  - Based on ingestion of contaminated aquatic organisms only (1E-05, 1E-06, 1E-07 cancer risk), 157, 15.7, 1.57 μg/L total halomethanes.

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 11,000 μg/L total halomethanes.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 12,000 μg/L total halomethanes.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 6400 μg/L total halomethanes.

REFERENCE DOSES:
ORAL: 20 μg/kg/day (3744)

## REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Dibromochloromethane is listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources and to effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the electroplating and metal finishing point source categories (3767, 3768). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

Under the National Interim Primary Drinking Water Regulations, the maximum contaminant level (MCL) for total trihalomethanes is 0.10 mg/L. This includes dibromochloromethane in combination with 3 other trihalogenated methanes. This MCL applies to community water systems which serve a population of 10, 000 or more and which add a disinfectant as part of their treatment process (3801).
Dibromochloromethane is listed by EPA as an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3771).

Resource Conservation and Recovery Act (RCRA)

Dibromochloromethane is included on EPA's groundwater monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their groundwater for chemicals on this list when suspected contamination is first detected, and annually thereafter (3775). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786).

Toxic Substances Control Act (TSCA)

Manufacturers, processors, or importers who possess health and safety studies on dibromochloromethane must submit them to EPA (3789).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)

Dibromochloromethane is designated as a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 45.4 kg (3766).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated dibromochloromethane as a hazardous substance with a reportable quantity of 45.4 kg, subject to requirements for packaging, labeling, and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
The level for total trihalomethanes in bottled drinking water is 0.10 mg/L. This level is identical to the maximum contaminant level (MCL) given under the Safe Drinking Water Act (365).

- State Water Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

ALABAMA
Alabama requires that the sum of all trihalomethanes in drinking water exceed an annual average maximum contaminant level of 0.10 μg/L. This applies to community water systems serving 10,000 or more and which add a disinfectant as part of their treatment process, and to non-community non-transient watersystems which obtain water from a surface source (3015).

CONNECTICUT
Connecticut has a quantification limit of 2.0 μg/L in drinking water (3137).

NEW YORK
New York has a maximum contaminant level of 5 μg/L in drinking water, and a nonenforceable guideline of 50 μg/L for surface and groundwater (3501).

OKLAHOMA
Oklahoma has a water quality criterion of 0.4 μg/L for groundwater (3534).

PENNSYLVANIA
Pennsylvania has a human health criterion (cancer risk level) of 0.2 μg/L for surface waters (3561).

SOUTH DAKOTA
South Dakota requires that dibromochloromethane be nondetectable, using designated test methods, in groundwater (3671).

Proposed Regulations

- Federal Programs
  No proposed regulations are pending.

- State Programs
  MOST STATES
  Most States are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officers is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA
  Minnesota has proposed a Recommended Allowable Limit (RAL) of 140 μg/L for drinking water (3451).

EEC Directives

Directive on Ground-Water (538)
Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compound in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto- pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

EEC Directive-Proposed Resolution
Resolution on a Revised List of Second-Category Pollutants (545)
Dibromochloromethane is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 3.1 MAJOR USES

Dibromochloromethane is used as a chemical intermediate in the manufacture of fire-extinguishing agents, aerosol propellants, refrigerants and pesticides (54).

## 3.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 3.2.1 Transport in Soil/Ground-water Systems

#### 3.2.1.1 Overview

Dibromochloromethane may be relatively mobile in the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical).

Transport pathways for low soil concentrations can be generally assessed by estimating equilibrium partitioning as shown in Table 3-1. These calculations predict the partitioning of dibromochloromethane among soil particles, soil water and soil air. The estimates for the unsaturated topsoil model indicate that significant amounts of dibromochloromethane are expected to be present in the soil-water phase, and can thus be transported through these phases by bulk transport (e.g., the downward movement of infiltrating water) and dispersion and diffusion. Less than 1% is expected to partition to the soil-air phase; therefore, diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind is a less significant loss pathway. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the dibromochloromethane (75%) is likely to be present in the soil-water phase (Table 3-1) and available for transport with flowing ground water. Ground water underlying dibromochloromethane-contaminated soils with low organic content is particularly vulnerable to contamination.

#### 3.2.1.2 Sorption on Soils

The mobility of dibromochloromethane in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

TABLE 3-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR
DIBROMOCHLOROMETHANE MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 20°C | 93.6 | 5.8 | 0.6 |
| Saturated deep soil[d] | 26.1 | 73.9 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Use estimated soil sorption coefficient: $K_{oc}$ = 84 (33).
c) Henry's law constant taken as 7.83E-04 atm · $m^3$/mol at 20°C (81).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

No information specific to the sorption of dibromochloromethane was available. Retardation rates, which represent the interstitial water velocity/pollutant velocity in the soil, were reported by Wilson et al. (82) to be a function of $K_{oc}$, the ratio of soil density (a) to soil water content (b), and the organic content (oc) of the soil according to the following equation:

$$R_f = 1 + (a/b)(K_{oc})(oc).$$

Schwarzenbach et al. (77) report retardation factors for some organic compounds. The data reported for chloroform, which has a $K_{oc}$ somewhat lower than that of dibromochloromethane, indicate some sorption and decreased mobility of chloroform in sediments containing 1-2% organic carbon; the retardation factors for soils having less than 0.1% organic carbon suggest little or no adsorption. Sorption of dibromochloromethane should be equal to or greater than that of chloroform.

### 3.2.1.3 Volatilization from Soils

Transport of dibromochloromethane vapors through the air-filled pores of unsaturated soils may occur, particularly in near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important

physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

Half-lives for the volatilization of dibromochloromethane from stirred aqueous solutions were reported to be in the range of 20-26 minutes (10). Kaczmar et al. (72) report that the volatilization half-lives of dibromochloromethane in rivers and streams range from 43 minutes to 16.6 days, somewhat lower than the half-lives determined for chloroform in the same experiment. Compared to volatilization from well-stirred aqueous solutions, volatilization of other halogenated aliphatics from near-surface soil was slower by approximately one order of magnitude (82).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H were observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of dibromochloromethane from surface soils.

No information was available for the two other physicochemical properties influencing dibromochloromethane volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

### 3.2.2 Transformation Processes in Soil/Ground-water Systems

Data specific to the transformation of dibromochloromethane in soil/ground-water systems were not available. A maximum hydrolytic half-life of 274 years has been reported for dibromochloromethane at pH 7 and 25°C, suggesting more rapid hydrolysis than that reported for other chlorinated methanes (10). However, neither hydrolysis, photolysis nor oxidation are expected to occur in the environment at rates significant enough to compete with volatilization.

Most references indicate that low molecular weight chloroaliphatics are not metabolized by microorganisms (10). However, Thom and Agg (80) have included several halomethanes in a list of organic chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as dibromochloromethane is very low and drops off sharply with increasing depth. Thus, biodegradation should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

### 3.2.3 Primary Routes of Exposure from Soil/Ground-water

The properties and fate characteristics described above suggest that dibromochloromethane in the environment is moderately volatile in aqueous solutions, weakly absorbed to soil, and has a low potential for bioaccumulation. Dibromochloromethane on the soil surface may volatilize, but that portion not removed by volatilization may eventually migrate to ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of dibromochloromethane from a disposal site could result in inhalation exposures. The potential for ground water contamination is high, particularly in sandy soils. Mitre (83) reported that dibromochloromethane has been found at 3 of the 546 National Priority List (NPL) sites. It was detected at 2 sites in ground water and one site in surface water. These limited findings are probably due to the low production of this compound.

Dibromochloromethane was reported with a greater degree of frequency in the USEPA (531) Groundwater Supply Survey (GWSS). This survey examined 945 finished water supplies that use ground-water sources. The results for dibromochloromethane are shown below:

| Sample Type | Occurrences* No. | % | Median of Positives (µg/L) | Maximum (µg/L) |
|---|---|---|---|---|
| Random | | | | |
| Supplies serving <10,000 people (280 samples) | 87 | 31.1 | 2.1 | 52 |
| Supplies serving >10,000 people (186 samples) | 96 | 51.6 | 2.9 | 59 |
| Non-Random | | | | |
| Supplies serving <10,000 people (321 samples) | 135 | 42.1 | 3.5 | 63 |
| Supplies serving >10,000 people (158 samples) | 87 | 55.1 | 4.6 | 51 |

*Samples having levels over quantification limit of 0.5 µg/L.

The random results are intended to statistically represent the U.S. ground-water drinking water supplies. The non-random samples were chosen by the states as being potentially contaminated. The results of this survey are probably more a reflection of dibromochloromethane production during chlorination of drinking water supplies than of the movement of this compound in soil/ground-water systems.

This compound has the potential to be mobile in ground water. If it reaches surface water, several other exposure pathways are possible:

- Surface waters may be used as drinking water supplies and result in direct ingestion exposure;
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposures through bioaccumulation;
- Recreational use of these waters may result in dermal exposures;
- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground water for two reasons. First, the Henry's law constant for dibromochloromethane suggests that it may volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this compound is low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 3.2.4 Other Sources of Human Exposure

The data presented above on the Groundwater Supply Survey (531) suggest that dibromochloromethane is a common contaminant in finished drinking water from ground-water sources. This has also been shown in both ground and surface water supplies where 83 of 106 finished water supplies sampled contained dibromochloromethane at detectable concentrations (90). Again, the prevalence of this compound in drinking water has been attributed to trihalomethane production during chlorination.

Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organic compounds. For dibromochloromethane, they had data for 89 sampling locations. The median concentration in urban and suburban locations was 0.12 $\mu g/m^3$ and zero in source-dominated areas. Obviously, the data base was small. However, it does appear that humans may be exposed to this chemical in the atmosphere.

## 3.3 HUMAN HEALTH CONSIDERATIONS

### 3.3.1 Animal Studies

#### 3.3.1.1 Carcinogenicity

The carcinogenic activity of dibromochloromethane was examined in male and female F344/N rats and B6C3F$_1$ mice. Test animals each received dibromochloromethane (98% pure in corn oil) by gavage five times per week for 2 years at doses of 0, 40 or 80 kg/kg (rats) and 0, 40 or 100 mg/kg (mice). No evidence of carcinogenic effects was noted in rats. However, compound-related nephrosis (degenerative lesions of kidney tubules) was noted in female rats, and fatty infiltration and cytoplasmic changes of the liver were evident in both male and female treated rats. In mice, some evidence of carcinogenic activity - an increased incidence of hepatocellular adenomas and an increased combined incidence of hepatocellular adenomas or carcinomas - was observed in treated female mice. The evidence of carcinogenicity for male B6C3F$_1$ mice was equivocal - an increased incidence of hepatocellular carcinomas - but the combined incidence of hepatocellular adenomas or carcinomas was only marginally increased. Compound-related lesions were found in the kidneys of male mice and the livers of male and female mice (3189).

#### 3.3.1.2 Genotoxicity

Dibromochloromethane was not mutagenic in four strains of Salmonella typhimurium in the presence or absence of metabolic activation (3859) when tested in a 20-minute preincubation assay, but when these bacteria were exposed to this compound in a desiccator for about 8 hrs, a positive dose response was found even without metabolic activation (95, 3653).

Two strains of yeast were also exposed (in screw-top vials) to this compound. Dibromochloromethane was positive in inducing gene conversion at the tryptophan locus in strain D7, but was negative in inducing reversion at any of three markers in another strain (3974).

Morimoto and Koizumi (549) reported that dibromochloromethane induced sister chromatid exchanges both in human lymphocytes in vitro (4.0E-04 M) and mouse bone marrow cells in vivo (oral doses of 25, 50 or 100 mg/kg/day, for four days).

No dominant lethal effects were observed in offspring of male ICR Swiss mice ingesting 0.1, 1.0 or 4 mg/mL dibromochloromethane in their drinking water in a two-generation study (663).

#### 3.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No treatment-related malformations or fetotoxic responses were noted by Ruddick and coworkers (548) in SD rats following oral administration of dibromo-

chloromethane (98% pure in corn oil) at dosage levels of 0, 50, 100 or 200 mg/kg/day on days 6-15 of gestation. Maternal weight gain was depressed 25% at the highest dose but fetal parameters were not affected.

Impairment of reproduction was seen in ICR Swiss mice ingesting 4 mg/mL dibromochloromethane (99% pure) in their drinking water (nominal dose of 685 mg/kg/day) in a modified two-generation study. Significant decreases were observed in fertility, litter size and adult female survival during gestation but appear to be due to direct toxic effects on maternal mice. Only marginal toxicity was evident in another group of mice similarly exposed to 1 mg/mL, and exposure to 0.1 mg/mL was without effect on any reproductive or teratogenic parameters (663). No significant dominant lethal effects were noted in offspring of male mice in the 0.1, 1 or 4 mg/mL treatment groups in the above study (663).

No embryotoxicity was observed in a Russian study in which rats and mice were treated orally (3378).

### 3.3.1.4 Other Toxicologic Effects

#### 3.3.1.4.1 Short-term Toxicity

In rats, females appear to be more sensitive than males to the acute lethal effects of dibromochloromethane. Oral $LD_{50}$ values of 1186 mg/kg and 848 mg/kg have been reported for male and female Sprague-Dawley rats, respectively (546,664). Survivors showed reduced food intake, growth retardation and increased liver and kidney weights at 14 days post-treatment. Elevated serum cholesterol levels were noted in surviving male rats; hematological values were also affected.

In mice, males appear to be more sensitive to the acute lethal effects of dibromochloromethane, in contrast to the results noted above for rats. Bowman and coworkers (92) reported oral $LD_{50}$ values for dibromochloromethane of 800 mg/kg and 1200 mg/kg for male and female ICR Swiss mice, respectively. Deaths occurred from 1 to 5 days after exposure. At necropsy, livers appeared to have fatty infiltration, the kidneys were pale, and hemorrhaging was noted in the brain, lungs and adrenals. Bowman and coworkers (92) also observed that oral administration of a sublethal dose (500 mg/kg) of dibromochloromethane induced sedation and anesthesia in ICR mice within 30 minutes of administration which persisted for approximately 4 hours.

Toxic effects in the liver, spleen and immune system occurred in both sexes of CD-1 mice administered 0, 50, 125 or 250 mg/kg dibromochloromethane by gavage for 14 consecutive days. Liver weights were increased at the highest two treatment levels for both sexes, and spleen weights were decreased at the 250 mg/kg level (547). The spleen is intimately involved with immune system function.

No signs of toxicity or histopathological changes were seen in weanling Sprague-Dawley rats given 0, 5, 50 or 500 ppm dibromochloromethane in their drinking water for 28 days. Fluid intake ranged from 20 to 30 mL/rat/day, or approximately 0.13, 1.5 and 12 mg/rat/day for the three treatment groups (664).

Anders et al. (93) found a slight elevation of blood carbon monoxide levels in male rats given 200 mg/kg of dibromochloromethane by intraperitoneal injection. At 2 hours, blood levels of carbon monoxide were approximately 400 mmol CO/mL compared with a value of 50 mmol CO/mL for controls.

#### 3.3.1.4.2 Chronic Toxicity

Dibromochloromethane administered to both sexes of Sprague-Dawley rats in their drinking water at levels of 0, 5, 50, 500 or 2500 ppm for 90 days induced mild liver toxicity (fatty infiltration) which was reversible after a 90-day recovery period (665). Exposures were estimated to range from 0.13-0.14 mg/rat/day to 32-49 mg/rat/day for the low and high-dose groups, respectively, based on fluid intake measurements.

An increased incidence of fatty infiltration and other histopathological changes of the liver were observed in F344/N rats given 40 to 80 mg/kg of dibromochloromethane by gavage 5 days per week for two years (3189).

### 3.3.2 Human and Epidemiologic Studies

There are no data on the effects of dibromochloromethane in humans. Trace amounts of dibromochloromethane have been identified in pooled plasma samples that were obtained from eight individuals who consumed water taken from New Orleans' water supply; dibromochloromethane has been identified as a component of drinking water in New Orleans (43). It is believed to be formed by the haloform reaction that may occur during water chlorination (94).

### 3.3.3 Levels of Concern

For the maximum protection of human health from the potential carcinogenic effects of halomethanes through ingestion of contaminated water and contaminated aquatic organisms, the USEPA (355) calculated lifetime daily ingestion of 1.9, 0.19 or 0.019 $\mu$g/L trihalomethanes in water and aquatic organisms might result in an incremental increase of cancer risk over a lifetime of 1E-05, 1E-06 and 1E-07, respectively. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk, at best.

The NAS - NRC (43) calculated a 24-hour SNARL of 18 mg/L for dibromochloromethane in drinking water based on the sedation-inducing dose in mice (92), an uncertainty factor of 1000 and assumptions of a daily water consumption of two liters

per day and a 70-kg body weight. Data were inadequate to calculate SNARLs for exposures via drinking water which extended over a period of time longer than one day.

As an estimate of a daily oral exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime, the EPA has calculated an RfD (reference dose) of 20 μg/kg/day (3744).

A maximum contaminant level for total trihalomethanes (including dibromochloromethane) in drinking water has been set by the USEPA at 0.1 mg/L for community water systems serving 10,000 persons or more (296).

### 3.3.4 Hazard Assessment

The potential impact on human health resulting from long-term exposure to dibromochloromethane can not be established at this time. Some evidence of carcinogenic activity (increased incidence of liver adenomas) was demonstrated in female mice; the data were equivocal for male mice and negative for rats (3189). Negative responses were recorded for a teratology study in rats (548) and a dominant lethal test in two generations of mice ingesting the compound in their drinking water (663). Dibromochloromethane has also been reported to induce sister chromatid exchanges in mouse bone marrow cells in vivo (549).

## 3.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of dibromochloromethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of dibromochloromethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. Recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of dibromochloromethane, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the dibromochloromethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the dibromochloromethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; dibromochloromethane is then detected with a halide

specific detector (Methods 601 and 8010), a mass spectrometer (Methods 624, 1624, and 8240) or an electron capture detector (3036). Direct injection may also be used for samples that contain high concentrations of chemical. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for dibromochloromethane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (<1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical dibromochloromethane detection limits that can be obtained in waste-waters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.09 μg/L (Method 601) | 0.9 μg/kg (Method 8010) |
| 3.1 μg/L (Method 624) | 5 μg/kg (Method 8240) |
| 10 μg/L (Method 1624) | |
| 5 μg/L (Method 8240) | |
| 0.9 μg/L (Method 8010) | |

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Note: The nonsequential numbers ot the references reflect the order of references as they appear in the master bibliography.

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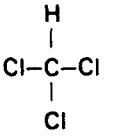
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| COMMON SYNONYMS: | CAS REG. NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25 °C (12) |
|---|---|---|
| Chloroform<br>Formyl trichloride<br>Methane trichloride<br>Methyl trichloride<br>Trichloromethane | 67-66-3 $CHCl_3$<br>NIOSH NO:<br>FS9100000<br><br>STRUCTURE:<br>  | 4.89 mg/m³ ≈ 1 ppm;<br>0.2045 ppm ≈ 1 mg/m³<br><br>MOLECULAR WEIGHT:<br>119.39 |

| | |
|---|---|
| REACTIVITY | Chloroform reacts slowly in air and light to form toxic phosgene and hydrogen chloride gases (38). Usually stored in absence of light using 0.5-1.0% ethanol as stabilizer (Merck Index). Reactions of halogenated organic materials such as chloroform with cyanides, mercaptans or other organic sulfides typically generate heat, while those with amines, azo compounds, hydrazines, caustics, or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases, and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (54, 511, 505). Explosions have also resulted from mixture of chloroform with acetone and a base, sodium methylate and methanol, and phosphorus pentoxide and perchloric acid (505). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Sweet, pleasant | (2) |
| | • Odor Threshold: 85.000 ppm | (384) |
| | • Density: 1.4850 g/mL (at 20°C) | (23) |
| | • Freeze/Melt Point: -63.50°C | (23) |
| | • Boiling Point: 61.20°C | (23) |
| | • Flash Point: Nonflammable | (23) |
| | • Flammable Limits: Will only burn under extreme fire conditions | (507)<br>(508) |
| | • Autoignition Temp.: Not pertinent | |
| | • Vapor Pressure: 1.60E+02 mm Hg (at 20°C) | (38) |
| | • Satd. Conc. in Air: 1.0270E+06 mg/m³ (at 20 °C) | (67) |
| | • Solubility in Water: 8.22E+03 mg/L (at 20°C) | (21) |

| | |
|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Viscosity: 0.563 cp (at 20°C) (21)<br>• Surface Tension: 2.7140E+01 dyne/cm (21)<br>• Log (Octanol-Water Partition Coeff.): 1.97 (29)<br>• Soil Adsorp. Coeff.: 4.40E+01 (33)<br>• Henry's Law Const.: 3.75E-03 atm · $m^3$/mol (at 20°C) (74)<br>• Bioconc. Factor: 4.5(estim), 6 (bluegill) (659) (719) |
| PERSISTENCE IN THE SOIL-WATER SYSTEM | Chloroform is expected to be highly mobile in soils and transport to ground water is likely. Volatilization of near surface material or material in the soil-air compartment may also be important. Transformation processes (e.g., hydrolysis, biodegradation) are not expected to be significant in natural soils. |
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of chloroform to groundwater drinking water supplies. The data from NPL sites show that such migration has commonly occurred in the past. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (12)<br>Chloroform vapor is a CNS depressant and may cause headache, drowsiness, vomiting, dizziness, unconsciousness, irregular heartbeat and death. Liver and kidney damage may also occur. Ingestion of chloroform is immediately followed by severe burning of the mouth and throat, vomiting, and chest and abdominal pain. When splashed in the eye, it causes burning pain and irritation; the corneal epithelium may be injured but regeneration is prompt. Prolonged or repeated contact with the skin may cause chronic skin irritation.<br><br>Acute Toxicity Studies: (3504)<br><br>INHALATION:<br>$LC_{Lo}$ 8000 ppm · 4 hr Rat<br>$LC_{50}$ 47702 $\mu g/m^3$ · 4 hr Rat<br><br>ORAL:<br>$LD_{50}$ 908 mg/kg Rat<br>$LD_{50}$ 36 mg/kg Mouse<br><br>SC:<br>$LD_{50}$ 704 mg/kg Mouse<br><br>Long-Term Effects: Liver necrosis and kidney damage<br>Pregnancy/Neonate Data: Embryolethality and fetotoxic effects at maternally toxic doses.<br>Genotoxicity Data: Conflicting results<br>Carcinogenicity Classification:<br>IARC - Group 2B (possibly carcinogenic to humans)<br>NTP - Positive evidence in mice and male rats<br>EPA - Group B2 (probable human carcinogen; sufficient evidence in animals and inadequate evidence in humans. |

| | |
|---|---|
| HANDLING PRECAUTIONS (38) | Handle only with adequate ventilation. • Vapor concentrations of 50-500 ppm: any supplied-air respirator or self-contained breathing apparatus. • 500-1000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece. • Chemical goggles if there is probability of eye contact with liquid. • Impervious clothing to prevent skin contact with the liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

### AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 2 ppm
- AFOSH PEL (8-hr TWA): 2 ppm; STEL (15-min): 4 ppm

Criteria
- NIOSH IDLH (30 min): deleted; NIOSH has recommended that the substance be treated as a potential human carcinogen
- NIOSH REL (10-hr TWA): 2 ppm (9.78 $mg/m^3$) ceiling (45 L, 60 min sample)
- ACGIH TLV (8-hr TWA): 10 ppm (A2, suspected human carcinogen)
- ACGIH STEL (15-min): deleted

### WATER EXPOSURE LIMITS:

Drinking Water Standards (296)
Under the National Interim Primary Drinking Water Regulations, the maximum contaminant level (MCL) for total trihalomethanes is 0.10 mg/L. This included chloroform in combination with three other trihalogenated methanes, bromoform, dichlorobromomethane and dibromochloromethane. This MCL applies to community water systems which serve a population of 10,000 people or more and which add a disinfectant as part of their treatment process.

EPA Health Advisories and Cancer Risk Levels (3742)
- 1E-04 cancer risk level: 600 $\mu g/L$

WHO Drinking Water Guideline (666)
A health-based guideline for drinking water of 30 $\mu g/L$ is recommended for chloroform. A daily per capita consumption of two liters of water was assumed.

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

EPA Ambient Water Quality Criteria

- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms, (1E-05, 1E-06, 1E-07 cancer risk), 1.9, 0.19, 0.019 μg/L.
  - Based on ingestion of contaminated aquatic organisms only (1E-05, 1E-06, 1E-07 cancer risk), 157, 15.7, 1.57 μg/L.
  - Based on ingestion of contaminated drinking water only (1E-05, 1E-06, 1E-07 cancer risk), 1.90 μg/L, 0.19 μg/L, 0.019 μg/L.

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 28,900 μg/L.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 1240 μg/L.

  - Saltwater species
    acute toxicity:
    no criterion established due to insufficient data.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:

ORAL: 1.000E+01 μg/kg/day (3744)

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Chloroform is designated a hazardous substance. It has a reportable quantity (RQ) limit of 2270 kg (347,3764). It is also listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and to effluent standards and guidelines (351,3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), metal finishing (3768), and metal molding and casting (892). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

Under the National Interim Primary Drinking Water Regulations, the maximum contaminant level (MCL) for total trihalomethanes is 0.10 mg/L. This includes chloroform in combination with 3 other trihalogenated methanes. This MCL applies to community water systems which serve a population of 10,000 or more and which add a disinfectant as part of their treatment process (3801). The specific chemical chloroform is listed as an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3771). In states with an approved Underground Injection Control program, a permit is required for the injection of chloroform-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

Chloroform is identified as a toxic hazardous waste (U044) and listed as a hazardous waste constituent (3783, 3784). Waste streams from the production of chlorinated aliphatic hydrocarbons are listed as non-specific sources of toxic hazardous waste (325, 3765). Waste streams from the following industries contain chloroform and are listed as specific sources of hazardous wastes: organic chemicals (production of acetaldehyde, vinyl chloride, fluromethanes, 1, 2-dichloroethane, 1, 1, 1-trichloroethane, and toluene diisocyanate) and inorganic chemicals (chlorine production) (3774, 3765). Chloroform is included on EPA's groundwater monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their groundwater for chemicals on this list when suspected contamination is first detected and annually thereafter (3775). Chloroform is subject to land disposal restrictions when its concentration as a hazardous constituent of certain wastewaters exceeds designated levels (3785). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to

1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786).

Toxic Substances Control Act (TSCA)
Manufacturers, processors, or importers who possess health and safety studies on chloroform must submit them to EPA (3789).

Comprehensive Environmental Response Compensation and Liability Act CERCLA)
Chloroform is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 2270 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing chloroform but these depend upon the concentration of the chemicals present in the waste stream (3766). Chloroform is designated an extremely hazardous substance under SARA Title III Section 302. Any facility at which chloroform is present in excess of its threshold planning quantity of 10,000 pounds must notify state and local emergency planning officials. If chloroform is released from a facility in excess of its reportable quantity (RQ), local emergency planning officials must be notified (3776). Under SARA Title III Section 313, manufacturers, processors, importers, and users of chloroform must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
Chloroform is exempt from a tolerance requirement when used as a solvent in pesticide formulations applied to growing crops. Exemptions also apply when it is used as a fumigant after harvest for barley, corn, oats, popcorn, rice, rye, sorghum and wheat (315).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to chloroform shall not exceed an 8-hour time-weighted average (TWA) of 2 ppm (3538).

Clean Air Act (CAA)
EPA intends to list chloroform as a hazardous air pollutant for which it will establish emission standards under Section 112 of the Clean Air Act (3685).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated chloroform as a hazardous material with a reportable quantity of 2270 kg, subject to requirements for packaging, labeling and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
Chloroform is approved for use as an indirect food additive as a component of adhesives (362). The level for total trihalomethanes in bottled drinking water is 0.10 mg/L. This level is identical to the maximum contaminant level (MCL) given under the Safe Drinking Water Act (365).

- State Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

ALABAMA
Alabama requires that the sum of all trihalomethanes in drinking water not exceed an annual average of 0.10 μg/L. This applies to community water systems serving 10,000 or more and which add a disinfectant as part of their treatment, and non-community non-transient water systems which obtain water from a surface source (3015).

CONNECTICUT
Connecticut has a quantification limit of 2.0 μ/L for drinking water (3137).

OKLAHOMA
Oklahoma has a water quality criterion of 10.0 μg/L for chloroform in groundwater (3534).

RHODE ISLAND
Rhode Island has an acute freshwater quality guideline of 1445 μg/L and a chronic guideline of 32 μg/L for the protection of aquatic life in surface waters. These guidelines are enforceable under Rhode Island state law (3590).

SOUTH DAKOTA
South Dakota requires that chloroform be nondetectable, using designated test methods, in groundwater (3671)

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed that solid wastes be listed as hazardous in that they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is equal to or greater than 0.07 mg/L chloroform. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Programs

  MOST STATES
  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officers is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA
  Minnesota has proposed a Recommended Allowable Limit (RAL) of 57 μg/L for chloroform in drinking water. Minnesota has also proposed a Sensitive Acute Limit (SAL) of 11560 μg/L for designated surface waters and 57 μg/L for designated groundwaters. These criteria are for the protection of human health (3451, 3452).

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by *member countries.*

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive Relating to the Classification, Packaging, and Labeling of Dangerous Preparations (Solvents) (544)
Chloroform is listed as a Class II/a harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substance into ground water.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Chloroform is classified as a harmful substance and is subject to packaging and labeling regulations. Chloroform may contain a stabilizer. If the stabilizer changes the properties of this substance, substance should be labeled in accordance to rules in Annex I and EEC/88/490, 22 July 1988.

EEC Directive-Proposed Resolution
Resolution on a Revised List of Second-Category Pollutants (545)
Chloroform is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 4.1 MAJOR USES

From 1847 until the 1940's, chloroform was primarily used as an anesthetic. Due to the resultant cardiac arrest and liver injury, this use is now obsolete. It was also utilized in many pharmaceutical preparations but due to its potential carcinogenicity in man, the FDA banned its use in drugs and cosmetics in July 1976 (12, 25).

Currently, its predominant use is as a feedstock for the manufacture of chlorodifluoromethane. It is also used as an extraction solvent in the pharmaceutical industry and as an industrial solvent in pesticides, textiles and dyes (94).

## 4.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 4.2.1 Transport in Soil/Ground-water Systems

#### 4.2.1.1 Overview

Chloroform may be relatively mobile in the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical).

Transport pathways can be generally assessed by using an equilibrium partitioning model as shown in Table 4-1. These calculations predict the partitioning of low soil concentrations of chloroform among soil particles, soil-water and soil-air. The estimates for the unsaturated topsoil model show that significant amounts of chloroform are present in the soil-water and soil-air phases, and can thus be transported through these phases by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. Diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, may be a significant loss pathway. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the chloroform is likely to be present in the soil-water phase (Table 4-1) and available to be transported with flowing ground-water. Ground-water underlying chloroform contaminated soils with low organic content are particularly vulnerable to pollution.

#### 4.2.1.2 Sorption on Soils

The mobility of chloroform in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of chloroform sorption on soil particles. Sorption of chloroform is expected to:

TABLE 4-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR CHLOROFORM IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 25 °C | 85.2 | 10.1 | 4.7 |
| Saturated deep soil[d] | 15.6 | 84.4 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Used estimated soil sorption coefficient: $K_{oc}$ = 44 (33).
c) Henry's law constant taken as 3.75 E-03 atm · $m^3$/mol at 25 °C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Schwarzenbach et al. (77) determined chloroform retardation rates, which represent interstitial water velocity/velocity of chloroform, for soils of varying organic carbon content. The data presented in Table 4-2 illustrate the increased sorption, and decreased mobility, of chloroform in sediments containing 1-2% organic carbon; the retardation factors for soils having less than 0.1% organic carbon suggest little or no adsorption.

Wilson et al. (82) investigated the transport and fate of chloroform in solutions applied to sandy soils. In a soil column receiving chloroform solutions of less than 1 mg/L, more than 50% was volatilized and 31-41% percolated through the soil column with minimal retardation.

### 4.2.1.3 Volatilization from Soils

Transport of chloroform vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

Half-lives for the volatilization of chloroform from stirred aqueous solutions were reported to be in the range of 20-26 minutes (10). Kaczmar et al. (72) report the volatilization half-lives of chloroform in rivers and streams to range from 29 minutes to 11.3 days. Smith et al. (78) report volatilization rates corresponding to half-lives of 1.2 days in a river, 13 days in a lake and 6.2 days in a pond. Compared to volatilization from well-stirred aqueous solutions, volatilization of chloroform from surface soil was reported to be slower by approximately one order of magnitude (82).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. The temperature dependence of H for chloroform has been measured by Gossett and Lincoff (18), and is described by the following equation:

$$H(atm \cdot m^3/mol) = \exp[8.956\text{-}4322/T^\circ K]$$

Gossett and Lincoff (18) have also examined the effect of other dissolved materials on the volatilization of chloroform. Moderate increases in H were observed with increasing salinity and the presence of other organic compounds. These results suggest that the presence of other materials may significantly affect the volatilization of chloroform from surface soils.

No information was available for the two other physicochemical properties influencing chloroform volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

### 4.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of chloroform in soil/ground-water systems is not well documented. In most cases, it should be assumed that chloroform will persist for months to years (or more).

Chloroform that has been released into the air will eventually undergo photochemical oxidation; a tropospheric lifetime of 0.2 to 0.3 years has been reported for chloroform (10). Chloroform under normal environmental conditions does not undergo rapid hydrolysis. Callahan et al. (10) report data from two conflicting studies estimating the hydrolysis half-life for chloroform at 15 months (minimum) and 3500 years.

Literature references to microbial degradation of compounds such as chloroform are very few. Most references indicate that low molecular weight chloroaliphatics are not metabolized (10). Schwarzenbach et al. (77) and Pearson and McConnell (75) observed no evidence of biological transformation of chloroform. However, Thom and Agg (80) included chloroform in a list of organic chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization could be achieved. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as chloroform is very low and drops off sharply with increasing depth. Thus, biodegradation should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

TABLE 4-2
RETARDATION FACTORS FOR CHLOROFORM IN RIVER SEDIMENTS AND SOILS

| Chloroform Retardation Factor | Matrix |
|---|---|
| 2.7 - 8 | River Sediment (1-2% organic carbon) |
| 1.2 - 4 | Aquifer Close to River Bed (0.1-1% organic carbon) |
| 1 - 1.2 | Aquifer Far from River Bed (0.1% organic carbon) |

Source: Schwarzenbach et al. (77).

### 4.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The properties and the above discussion of fate pathways suggest that chloroform in the environment is highly volatile, weakly adsorbed to soil and has no significant potential for bioaccumulation. Chloroform on the soil surface is likely to volatilize, but that portion not removed by volatilization may eventually migrate to ground-water. These fate characteristics suggest several potential exposure pathways.

Volatilization of chloroform from a disposal site could result in inhalation exposures. In addition, the potential for ground-water contamination is high, particularly in sandy soils. Mitre (83) reported that chloroform was found at 68 of the 546 National Priority List (NPL) sites. It was detected at 60 sites in ground-water, 22 sites in surface water and one site in air. The lack of detection of chloroform in air may be somewhat misleading as air sampling at NPL sites has been limited. Chloroform was detected with a greater degree of frequency in the

Groundwater Supply Survey (GWSS) conducted by EPA (531). This survey examined 945 finished water supplies that use ground-water sources. The results for chloroform are summarized below:

| Sample Type | Occurrences* No. | % | Median of Positives (μg/L) | Maximum (μg/L) |
|---|---|---|---|---|
| Random | | | | |
| Supplies serving <10,000 people (280 samples) | 104 | 37.1 | 1.4 | 140 |
| Supplies serving >10,000 people (186 samples) | 106 | 57.0 | 1.6 | 300 |
| Non-Random | | | | |
| Supplies serving <10,000 people (321 samples) | 155 | 48.3 | 1.6 | 100 |
| Supplies serving >10,000 people (158 samples) | 100 | 63.3 | 2.1 | 430 |

*Samples having levels over quantification limit of 0.5 μg/L.

The random samples represent a statistical sample for ground-water in the U.S. The non-random samples were chosen by the states as being potentially contaminated. The results of this survey are probably more a reflection of chloroform produced during chlorination of drinking water supplies than of the movement of chloroform in soil/ground-water systems. The potential for movement of chloroform in ground-water to surface water suggests several other exposure pathways:

- Surface waters may be used as drinking water supplies and result in direct ingestion exposure.
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposures through bioaccumulation.
- Recreational use of these waters may result in dermal exposures.
- Domestic animals may consume or be dermally exposed to contaminated ground- or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground-water for two reasons. First, the Henry's law constant for chloroform suggests that it will likely volatalize upon reaching surface waters. Secondly, the bioconcentration factor for chloroform is low, suggesting no significant bioaccumulation in aquatic organisms or domestic animals.

### 4.2.4. Other Sources of Human Exposure

The data presented above on the Ground-water Supply Survey suggest that chloroform is a common contaminant in drinking water. This has also been shown in previous surveys including both ground- and surface water supplies where 98 of 106 of the finished water supplies sampled contained chloroform at detectable concentrations (90). Again, these levels are largely due to trihalomethane production during chlorination.

Humans may also be exposed to chloroform in food. McConnell et al. (224) analyzed various foods in Great Britain and found chloroform levels ranging from 0-33 $\mu$g/kg. The higher levels were found in cheeses, olive oil, tea and potatoes. On the basis of these data, NAS (99) estimated an average intake for chloroform of 0.006 mg/day.

The volatile nature of chloroform suggests it will also be a common air contaminant. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For chloroform, they had data for 2577 sampling locations. The median concentration in rural and remote areas was 0.2 $\mu g/m^3$; in urban and suburban areas, 0.35 $\mu g/m^3$; and in source dominated areas, the median was 4.0 $\mu g/m^3$. The authors noted wide variability in the data.

It is evident that humans are commonly exposed to chloroform in treated drinking water and in the atmosphere. Food may also be a source of exposure, although data are limited.

## 4.3 HUMAN HEALTH CONSIDERATIONS

### 4.3.1 Animal Studies

#### 4.3.1.1 Carcinogenicity

A carcinogenesis bioassay of USP-grade chloroform was conducted by the National Cancer Institute (100) using Osborne-Mendel rats and $B6C3F_1$ mice. Both species received chloroform in corn oil by gavage 5 times per week for 78 weeks. Male rats received 90 or 180 mg/kg. Female rats were started at 125 or 250 mg/kg and reduced to 90 or 180 mg/kg after 22 weeks, with an average level of 100 or 200 mg/kg for the study. Male mice received initial doses of 100 or 200 mg/kg which was increased after 18 weeks to 150 or 300 mg/kg. Female mice initially received 200 or

400 mg/kg which was increased to 250 or 500 mg/kg after 18 weeks. Average doses were 138 or 277 mg/kg for male mice and 238 or 477 mg/kg for female mice. All controls received corn oil.

The rat study was terminated after 111 weeks. In male rats, there was a statistically significant incidence of kidney epithelial tumors - 24% in the high-dose group, 8% in the low-dose group and 0% in the controls. There was an increase in thyroid tumors in female rats but this was not considered biologically significant. A decrease in survival rate and weight gain was evident for all treated rat groups.

The mouse study was terminated after 92 to 93 weeks. There was a statistically significant increase in hepatocellular carcinoma in both sexes: 98% and 96% for high-dose males and females, respectively, and 36% and 80% for low-dose males and females. The incidence in controls was 6% for male mice and 0% for female mice. Nodular hyperplasia was observed in several low-dose male mice that had not developed hepatocellular carcinoma (100). This study was reviewed and reevaluated by Reuber (714) who identified additional sites of neoplasms. This suggests a tumor-initiating capability for chloroform. Reuber also noted an increase in testicular atrophy in male rats treated with chloroform.

Roe et al. (715) conducted a study in 4 strains of mice (male and female ICI/CFLP, male C57B6, CF/1 and CBA) which were administered chloroform in a toothpaste vehicle by gavage 6 days per week for 80 weeks, followed by a 16- to 24-week observation period. Tumors of the renal cortex were observed in 17% of male ICI/CFLP mice at the 60 mg/kg level compared with 2% in the vehicle controls. No overall increase in tumors was observed in males of this strain given a dose of 17 mg/kg/day nor in females of this strain or male C57BL, CBA or CF/1 strain mice given up to 60 mg/kg/day for 1.5 years. The kidney tumors observed in this case appear to be highly strain specific (150).

Sprague-Dawley rats intubated 6 days/week with 60 mg/kg chloroform in a toothpaste vehicle for 80 weeks, followed by 15 weeks of observation exhibited no clear histological evidence of toxic effects on liver or kidney in either sex. A wide spectrum of different types of neoplasms was seen but did not appear to be related to treatment.

Heywood et al. conducted a 7.5 year study in beagle dogs that were fed 15 or 30 mg/kg chloroform in a toothpaste base 6 days per week. The dogs were maintained for an additional 20 to 24 weeks without treatment. A small number of neoplasms were seen but were predominantly age-related. No neoplasms of the liver or kidney were found. "Fatty cysts", possibly chloroform-related, were seen in the livers of several dogs at post-mortem.

IARC (25) has listed chloroform in category 2B (sufficient evidence of animal carcinogenicity) in its weight of evidence ranking for potential carcinogens.

### 4.3.1.2 Genotoxicity

Bacterial studies with chloroform are uniformly negative. Simmon et al. (95, 3653) have tested this agent in the Salmonella/microsome assay in suspension and in desiccators and have been unable to demonstrate that this chemical is a mutagen in at least 5 strains. When two strains of Escherichia coli were treated with chloroform using two methods of treatment, plate incorporation and preincubation, with or without metabolic activation, no induction of revertants above control levels was observed (3359). A strain of Salmonella typhimurium designed to detect forward mutations to azaguanine resistance did not respond to chloroform treatment with or without activation (3661).

Callen et al. (649) reported that chloroform in suspension induced mitotic gene conversion and recombination in Saccharomyces cerevisiae D7.

Positive results have been obtained in vivo in a host-mediated assay conducted in mice with Salmonella typhimurium and in a mouse micronucleus test (698), suggesting in vivo activation may be necessary for mutagenic activity.

Chloroform was also tested in the mouse micronucleus test by Gocke et al. who used 3 different concentrations in both sexes. Although the number of bone marrow micronucleated cells was above the concurrent control, the increase was not significant (3246).

Positive results were obtained in a cell transformation assay using Syrian hamster embryo cells in a virus-mediated assay when the cells were exposed to chloroform for 3 hrs (3274).

There was no induction of DNA damage in liver DNA of rats given 200-400 mg/kg of chloroform orally (696), and there were no significant mutations at the 8-azaguanine locus in Chinese hamster lung fibroblast cells following 24-hour exposure to 3% chloroform in culture (697).

Nuclei of cultured rat hepatocytes from Holtzman males were treated with chloroform up to the toxic dose, and unscheduled DNA synthesis was measured. Results with chloroform were similar to the negative controls (3018).

In an in vivo study, Mirsalis et al. (3455) showed that the hepatocytes of F344 male rats gavaged with chloroform showed no increase over control levels in unscheduled DNA synthesis at 2 hrs after treatment in hepatocytes cultured from these animals.

It has been demonstrated that chloroform binds to mammalian hemoglobin. Rats were fed chloroform intragastrically and blood was withdrawn 24 hrs after treatment (3562).

Chinese hamster ovary cells treated with chloroform with or without metabolic activation for one hr or 24 hrs did not show an increase in SCEs above control values(3564, 3837).

When human whole blood was cultured in the dark with chloroform for 72 hrs, an increase in SCEs was found (3467). Conversely, when human blood was treated for 2 hrs in the presence of an exogenous activation system, no increase above controls was found for SCEs (3359). In an in vivo study, a dose response was found for the induction of SCEs in bone marrow cells of male mice given chloroform orally on four consecutive days (3467) and in female mice exposed to 300 ppm by inhalation for 3 or 6 hrs (3319). In the only report found to claim a positive effect on the induction of chromosomal breaks by chloroform, San Augustin and Lim-Sylianco (698) observed a three-fold increase in micronuclei in bone marrow cells of male mice treated with 700 mg/kg of chloroform. Chloroform has been shown to break chromosomes, in a dose-dependent manner in onion root tips (3142).

### 4.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Withey and Karpinski (3843) exposed rats to chloroform by inhalation for 5 hours on day 17 of gestation to 6 levels of chloroform ranging from 111 to 1984 ppm. Fetal tissue concentrations were linear functions of maternal dose, and the fetal/maternal blood concentration ratio averaged 0.316. Placental transfer also occurs in the mouse shortly after inhalation at all stages of gestation (3150).

Teratogenicity studies of chloroform have given mixed results. Schwetz et al. (699) exposed rats to vapor levels of 30, 100 or 300 ppm, 7 hours daily on days 6 to 15 of gestation. At the 300 ppm level, chloroform caused a decrease in conception rate and a high incidence of fetal resorption. This loss precluded significant data on malformations. At 100 ppm, there was a significant incidence of tail absence or shortening, imperforate anus, subcutaneous edema, missing ribs and delayed ossification of sternebrae. Decreased fetal body measurements were observed at both 30 and 300 ppm. Murray et al. (711) observed an increased incidence of cleft palate in mice exposed to 100 ppm on days 8 through 15 of gestation.

Exposure of male mice to 400 or 800 ppm chloroform vapors, 4 hr/day for 5 days caused significant increases in the percentage of abnormal sperm at both levels (Land et al. as reported in ATSDR Toxicology Profile for Chloroform).

No increase in abnormalities were observed by Thompson et al. (712), who administered chloroform by gavage to rats and rabbits on gestation days 6-15 and 6-18, respectively. Rats received 20, 50 or 126 mg/kg daily and rabbits received 20, 35, or 50 mg/kg daily. Maternal toxicity occurred at 50 mg/kg and higher in both species. Ruddick (713) also obtained negative results in rats given oral doses of 100, 200 or 400 mg/kg on days 6 through 15 of gestation. Balster and Borzelleca (3048) administered 31.1 mg/kg/day chloroform in drinking water to mice throughout gestation and lactation. No significant behavioral changes were observed in offspring. Palmer et al. (as reported in ATSDR Toxicology Profile for Chloroform) gavaged doses of 0,

15, 30, 150 or 410 mg/kg/day in toothpaste to male and female rats for 13 weeks and observed gonadal atrophy in the high-dose group.

### 4.3.1.4 Other Toxicologic Effects

#### 4.3.1.4.1 Short-term Toxicity

Available data indicate that the acute toxicity of chloroform in laboratory animals is dependent upon species, strain and sex variations (94). Oral $LD_{50}$ values ranged from 36 to 1400 mg/kg in the mouse to 900 to 2000 mg/kg in the rat (47,94). Oral $LD_{50}$ values in male mice varied from 120 mg/kg in DBA/2J mice to 490 mg/kg in C57BL/6J mice. The first generation offspring of these 2 strains exhibited a value midway between the parental strains (717). $LC_{Lo}$ values of 8000 ppm · 4 hr, 20,000 ppm · 2 hr and 35,000 ppm · 4 hr have been recorded for the rat, guinea pig and cat, respectively (47).

Males of several mouse strains are susceptible to renal tubular necrosis, whereas females are not similarly affected (25). Klaassen and Plaa (718) reported that male mice demonstrated renal dysfunction at 116 mg/kg bw, but females did not exhibit renal dysfunction at any time during or after exposure to even a lethal dose of chloroform.

Acute oral exposure to 250 mg/kg produced fatty infiltration and necrosis of the liver as well as kidney damage in rats (720). Similar findings were noted in mice given 350 mg/kg chloroform orally (721).

Moore et al. (3466) studied the effects of orally-administered chloroform in CFLP Swiss albino mice. (Groups of at least three animals were given single doses of the chemical by gastric intubation). A low dose of 17.3 mg/kg chloroform in corn oil had no toxic effects on the liver or kidneys. An intermediate dose of 65.6 mg/kg in corn oil was not very toxic to the liver, but there was necrosis of the kidney tubules. With an intermediate dose of 59.2 mg/kg chloroform in toothpaste there was little sign of either kidney or liver toxicity. In mice treated with the high chloroform dose in either vehicle (273 mg/kg in corn oil, 199 mg/kg in toothpaste), kidney necrosis and an increased uptake of [3H]thymidine was seen in all animals.

Condie et al. (3136) administered 37, 74, or 148 mg/kg chloroform in corn oil by gavage to male CD-1 mice for 14 consecutive days. There was a slight but significant body weight loss (9%) in the high dose treatment group. Microscopic examination revealed renal intratubular mineralization, epithelial hyperplasia, and cytomegaly, especially in the high dose group. The liver showed centrilobular cytoplasmic pallor, mitotic figures, and focal inflammation in the treated groups. Mitotic figures were particularly striking in the high dose group.

Kylin et al. (158) evaluated the acute effects of chloroform vapor on female albino mice which they exposed to 100, 200, 400 or 800 ppm for 4 hours and sacrificed 1 or 3 days after exposure. Mice exposed to 100 ppm showed a moderate

infiltration of fat in the liver. This effect was seen more frequently in mice killed after 1 day of exposure than in those sacrificed after 3 days. Exposure to 200, 400 or 800 ppm caused necrosis which increased in severity with increasing dose.

Inhalation studies were also done by Lundberg et al. (3410) who exposed female Sprague-Dawley rats for 4 hr to 1/2, 1/4, 1/8, 1/16, 1/32, 1/64, or 1/128 of the $LC_{50}$ value ($LC_{50}$ value, 24 hr after 4-hr exposure = 47.7 mg/m$^3$). Livers of the animals exposed to 1/2 of the $LC_{50}$ value were examined histologically and it was observed that the centrolobular hepatocytes were swollen, glycogen was depleted, and the nuclei were pycnotic. The $TC_{50}$ (toxic concentration 50%) value for chloroform was calculated as 585 mg/m$^3$.

One to two 24-hour applications of chloroform to rabbit skin produced slight hyperemia with moderate necrosis and scab formation. Healing of abraded skin appeared to be delayed by chloroform application. Single skin applications of 1000 mg/kg for 24 hours under an impermeable plastic cuff resulted in degenerative changes in the kidney tubules; however, doses as high as 3980 mg/kg were survived (720).

Chloroform dropped into the eyes of rabbits resulted in conjunctivitis and some corneal injury that required 1 week to heal (720).

#### 4.3.1.4.2 Chronic Toxicity

Torkelson et al. (720) exposed various species of laboratory animals to chloroform vapors 7 hr/day, 5 days/week for 6 months. Dogs exposed to 25 ppm and rabbits and guinea pigs exposed to 25 or 50 ppm showed inconsistent liver, kidney and lung changes. Male rats exposed to 25, 50 or 85 ppm all displayed an increased relative kidney weight, cloudy swelling of the renal tubular epithelium and centrilobular granular degeneration of the liver with focal areas of necrosis. Decreased body weight was observed in males at the 50 and 85 ppm levels, with pneumonia and an increase in the relative liver weight present in the 85 ppm male rats. In female rats at the 25 ppm level, only an increase in relative kidney weights was observed. At the 50 and 85 ppm levels, liver and kidney pathology was similar to that seen in males. No pathological effects were observed in male rats exposed to 25 ppm for 4, 2, or 1 hour daily for 6 months; also male rats exposed to 25 ppm for 7 hr/day for 6 months, and allowed to recover for 6 weeks after their last exposure, were normal in all respects.

Chu et al. (3126) exposed Sprague-Dawley rats (20/sex) to 5, 50, 500, or 2500 ppm chloroform in drinking water for 90 days. There was suppression of growth rate in rats of both sexes in the 2500 ppm group. Histological examination showed that the liver and the thyroid were the target organs. There was fatty infiltration of the liver and a reduction in follicular size and colloid density in the thyroid.

Munson et al. (3474) administered 50, 125, or 250 mg/kg/day chloroform by gavage to CD-1 mice of both sexes (7-12 animals/group) for 90 days. Liver weights were increased at all levels of treatment in both male and female mice. Treated mice

of both sexes showed slight histologic changes in the kidneys and livers. The kidneys exhibited small intertubular collections of chronic inflammatory cells, mostly lymphocytes. The liver showed generalized hydropic degeneration of hepatocytes and small focal collections of lymphocytes.

A 5-month ingestion study was carried out by Miklashevskii et al. (722) in male albino rats and male guinea pigs. Although the dosing schedule was not reported, daily administration was implied. No effects were noted in either species at the 0.4 mg/kg level. At 35 mg/kg, only two of six guinea pigs survived beyond 3 months. Those that died had structural lesions of the liver, heart muscle and stomach wall. Microscopic changes included fatty infiltration, necrosis and cirrhosis of the liver parenchyma, lipoid degeneration and proliferation of interstitial cells in the myocardium and acute edema of the submucosal and muscular layers of the stomach. Rats were not dosed at the 35 mg/kg level.

Dogs receiving 45 mg/kg/day of chloroform for 12 to 18 weeks developed liver discoloration and had increased liver weights. These effects were not seen at the 30 mg/kg/day level. At 60, 90 and 120 mg/kg/day, fat deposition within the hepatocytes was seen along with hepatocyte enlargement and vacuolation (716).

### 4.3.2 Human and Epidemiologic Studies

#### 4.3.2.1 Short-term Toxicologic Effects

Chloroform is a central nervous system depressant and is also toxic to the liver and kidneys (46). In experimental human exposures to chloroform vapors, approximately 14,000 to 16,000 ppm caused rapid loss of consciousness. Dizziness, intracranial pressure and nausea resulted after 7 minutes exposure to 1000 ppm. There were also after effects consisting of fatigue and headache. A 30 minute exposure to 390 ppm resulted in no adverse effects (12).

Individual toxicity by the oral route varies considerably. A dose of 5 mL has caused serious illness while a dose of 120 mL has been survived (101). The mean lethal dose probably lies near one fluid ounce (44g) (17).

In a case of acute ingestion of 120 mL, Schroeder (723) described a man who, when first observed, was cyanotic, had dilated pupils and was perspiring perfusely. An EKG obtained a few hours after hospital admission showed occasional cardiac arrhythmias which had ceased by the following day. He developed liver enlargement along with jaundice and vomiting. There were also initial rises in serum bilirubin, alkaline phosphatase and SGOT. Recovery began on the fifth day after ingestion.

NIOSH (158) described 6 cases of delayed chloroform poisoning in obstetric patients who had undergone chloroform anesthesia between 1924 and 1953. Patients developed symptoms of drowsiness, jaundice, vomiting, fever, liver enlargement, delirium and coma after a latency period ranging from a few hours to a day. Deaths occurred in 3 patients 5 to 8 days post-partum.

Dermal exposure to chloroform may cause irritation, erythema, hyperemia and destruction of the epithelium (158).

Splashing chloroform into the eye causes burning pain and conjunctival irritation. The corneal epithelium is sometimes injured, but usually will regenerate within one to three days (19).

#### 4.3.2.2 Teratogeniity, Embryotoxicity and Reproductive Effects

Chloroform was detected in fetal blood at levels equal to or greater than in maternal blood (3182). The Collaborative Study (3283) found no significant association between ingestion of chloroform as a food additive during the first four months of pregnancy and adverse reproductive outcome. Heath (3282) conducted a study at Love Canal in New York, an area contaminated with chloroform and other chemicals. No clear increased incidence of abortion, birth defects, or low birth weight was observed in women living next to the Canal. Tylleskar-Jensen (3053) reported that two women working in the same laboratory and exposed to a variety of organic chemicals, including chloroform at 300 to 1000 ppm, developed eclampsia of pregnancy.

#### 4.3.2.3 Chronic Toxicity

Chronic exposure to chloroform causes liver and kidney damage. Liver disturbances are more characteristic of exposure than central nervous system depression or renal injury (54). Ethyl alcohol is suspected of having a potentiating effect on the toxicity of chloroform vapor in occupational settings, but this has not been proven in industrial practice (38).

There are very few reports of occupational poisoning due in part to its limited industrial use (2). Bomski et al. reported that 17 of 68 workers exposed to chloroform vapors ranging from 2 to 205 ppm for 1 to 4 years had enlarged livers. Out of these 17 cases, 4 had toxic hepatitis and 13 had fatty degeneration of the liver (725). Challen et al. (726) reported no liver injury in workers exposed to vapor levels of 23 to 71 ppm, 4 hours daily for 10 to 24 months; however, symptoms such as lassitude and dryness of the mouth and throat were noted.

There was no indication of hepatotoxicity in chronic users of a dentifrice and mouthwash containing 3.4% and 0.43% chloroform, respectively. Ingestion was estimated to be 0.3 - 0.96 mg/kg/day over a 1- to 5-year period (727). Some reversible hepatotoxicity was seen in individuals who ingested a chloroform-containing cough suppressant over a 10-year period. Chloroform intake was estimated to be 23-37 mg/kg/day (728).

NIOSH (158) reported numerous cases of habitual inhalation of chloroform vapors for up to 15 years. Symptoms included hallucinations, loss of appetite,

incoordination, moodiness, mental and physical sluggishness, nausea, rheumatic pain and delirium.

A study by Hogan et al. (729) showed a positive correlation between rectal intestinal and bladder cancer mortality rates and chloroform levels in drinking water. These results, however, do not establish causality since they failed to take into account other carcinogenic risk factors such as cigarette smoking and diet. Conversely, a recent study found no relationship between chloroform intake from the water supply and deaths from colorectal cancer. In this study, the investigators compared a total of 395 colorectal cancer deaths among white women teachers in New York State to an equal number of deaths of teachers from non-cancerous causes. Cumulative chloroform exposure was estimated from operational records of water treatment facilities that served the home and work addresses of each study subject during the 20 years prior to death (730).

Sex-related differences associated with the effects of chloroform are also seen in humans where the percentage of chloroform metabolized to carbon dioxide is greater in females than males. This probably results from chloroform's affinity for adipose tissue, which is more prevalent in women and would result in less unchanged chloroform and more carbon dioxide exhaled. This raises questions as to whether women are at greater risk (96).

### 4.3.3 Levels of Concern

The USEPA (355) has specified an ambient water quality criterion of zero for chloroform, based on induction of liver carcinoma in female mice. In that attainment of a zero concentration level may be infeasible in some cases, the concentrations of chloroform in water calculated to result in incremental lifetime cancer risks of 1E-05, 1E-06 and 1E-07 from ingestion of both water and contaminated aquatic organisms were estimated to be 1.9, 0.19, and 0.019 $\mu$g/L, respectively. Risk estimates are expressed as a probability of cancer after a lifetime daily consumption of two liters of water and 6.5 g of fish that have bioaccumulated chloroform. Thus a risk of 1E-05 implies that a lifetime daily consumption of two liters of drinking water and 6.5 g of contaminated fish at the criterion level of 1.9 $\mu$g/L of chloroform would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 people exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

IARC (25) lists chloroform in category 2B (sufficient evidence of animal carcinogenicity) in its weight-of-evidence ranking for potential carcinogens.

A maximum contaminant level of 0.10 mg/L has been set for total trihalomethanes, including chloroform, in drinking water (296). The WHO (666) recommends a maximum level of 30 $\mu$g/L for chloroform in drinking water.

OSHA (3539) currently permits a ceiling level of 2 ppm (9.78 mg/m$^3$) for chloroform. The ACGIH (3005) has set 10 ppm as a TWA with the notation that chloroform is a suspected carcinogen.

### 4.3.4 Hazard Assessment

Chloroform has been tested for carcinogenic activity in mice, rats and dogs by oral administration. It produced hepatomas and hepatocellular carcinomas in mice, malignant kidney tumors in male rats, tumors of the thyroid in female rats, and no treatment-related tumors in dogs. There is sufficient evidence that chloroform is carcinogenic in rats and mice, and in the absence of adequate data in humans, it appears prudent to regard chloroform as if it presented a carcinogenic risk to humans.

In vitro studies of mutagenic activity are primarily negative for chloroform; two in vivo studies yielded positive results, suggesting in vivo activation may be necessary for mutagenic activity.

Chloroform, administered by gavage or by inhalation, results in embryolethality and fetotoxic effects at maternally toxic doses.

The liver and kidneys are the target tissues for chronic exposure to chloroform for both experimental animals and humans. High concentrations of chloroform result in narcosis and anesthesia. Acute inhalation exposures are associated with depression of the central nervous system and possible cardiac sensitization. Ingestion of 5 mL has caused serious illness but ingestion of 120 mL has been survived.

## 4.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of chloroform concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of chloroform, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of chloroform, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the chloroform from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the chloroform and transfer it onto a gas chromatographic

(GC) column. The GC column is programmed to separate the volatile organics; chloroform is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations, direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

Other methods that have been reported for the determination of chloroform in water include a spectrophotometric method (3548) and fluorimetry using a fiber optic probe (3448).

The EPA procedures recommended for chloroform analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical chloroform detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.05 μg/L (Method 601) | 0.5 μg/kg (Method 8010) |
| 1.6 μg/L (Method 624) | 5 μg/kg (Method 8240) |
| 10 μg/L (Method 1624) | |
| 0.5 μg/L (Method 8010) | |

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3766. U.S. Environmental Protection Agency 1986. Designation, reportable quantities, and notification of hazardous substances. Fed. Regist. 51:34534. 40 CFR302.4 (CERCLA).

3767. U.S. Environmental Protection Agency 1986. Electroplating point source category, pretreatment standards and monitoring requirements. Fed. Regist. 51:40421. 40 CFR413.

3768. U.S. Environmental Protection Agency 1986. Metal finishing point source category: pretreatment standards and monitoring requirements. Fed. Regist. 51:40421. 40 CFR433.

3771. U.S. Environmental Protection Agency 1987. NPDWR - Synthetic organic chemicals: Monitoring for unregulated contaminants. Fed. Regist. 52:25690. 40 CFR141.40.

3774. U.S. Environmental Protection Agency 1987. Identification and listing of hazardous wastes: hazardous wastes from specific sources. Fed. Regist. 52:28698. 40 CFR261.32.

3775. U.S. Environmental Protection Agency 1987. List of hazardous constituents for ground-water monitoring. Fed. Regist. 52:25942. 40 CFR 264 and 270 Appendix IX.

3776. U.S. Environmental Protection Agency. 1987. 40 CFR355 Appendix A and B (SARA Title III Section 302). Extremely hazardous substances list and threshold planning quantities. Final rule as published in Fed. Regist., 1987, 52:13378, and revised in 1987, 52:48072 and 1988, 53:5574.

3777. U.S. Environmental Protection Agency 1987. Organic chemicals, plastics, and synthetic fibers category: Effluent limitations guidelines, pretreatment standards, and new source performance standards. Fed. Regist. 52:42522. 40 CFR414.

3783. U.S. Environmental Protection Agency 1988. Identification and listing of hazardous wastes: Hazardous constituents. Fed. Regist. 53:1 3388. 40 CFR261 Appendix VIII.

3784. U.S. Environmental Protection Agency 1988. Identification and listing of hazardous wastes: discarded commercial chemical products and their hazardous waste numbers. Fed. Regist. 53:13384. 40 CFR261.33.

3785. U.S. Environmental Protection Agency 1988. Standards for the management of specific hazardous wastes and management facilities: Land disposal restrictions. Fed. Regist. 53:31138. 40 CFR268.

3786. U.S. Environmental Protection Agency 1988. Land disposal restrictions for first third scheduled wastes: Waste specific prohibitions-California list wastes. Fed. Regist. 53:31138-31222. 40 CFR268.32.

3787. U.S. Environmental Protection Agency 1988. Toxic chemical release reporting: Community right-to-know. Fed. Regist. 53:4500 and revised 1988, 53:23108. 40 CFR372 (SARA Title III Section 313).

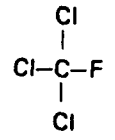
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| COMMON SYNONYMS: | CAS REG.NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25 °C |
|---|---|---|
| F11<br>Fluorocarbon 11<br>Fluorochloroform<br>Fluorotrichloro-methane<br>Freon 11<br>Propellant 11<br>Trichlorofluoro-methane | 75-69-4 $CCl_3F$<br>NIOSH NO.:<br>PB6125000<br>STRUCTURE:<br>  | 5.6 mg/m³ ≈ 1 ppm;<br>0.178 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT:<br>137.36 |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as trichlorofluoromethane with cyanides, mercaptans or other organic sulfides typically generate heat, while those with amines, azo compounds, hydrazines, caustics or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydro-peroxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505, 3429). Combustion products include hydrogen fluoride, hydrogen chloride, and possibly phosgene. |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid, volatile (at 20 deg C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Nearly odorless | (12) |
| | • Odor Threshold: 5.000 ppm | (67) |
| | • Density: 1.4940 g/mL (at 17 °C) | (14) |
| | • Freeze/Melt Point: -111.00 °C | (23) |
| | • Boiling Point: 23.82 °C | (21) |
| | • Flash Point: Noncombustable | (23) |
| | • Flammable Limits: Nonflammable | (60,509) |
| | • Autoignition Temp.: Nonflammable | (60,509) |
| | • Vapor Pressure: 6.90E+02 mm Hg (at 20°C) | (38) |

| | |
|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Solubility in Water: 1.10E+03 mg/L (at 20°C) (38)<br>• Viscosity: 0.430 cp (at 25°C) (21)<br>• Surface Tension: 1.8000E+01 dyne/cm (at 25°C) (21)<br>• Log (Octanol-Water Partition Coeff: 2.53 (29)<br>• Soil Adsorb. Coeff: 1.59E+02 (33)<br>• Henry's Law Constant: 1.10E-01 $atm \cdot m^3/mol$ (74)<br>• Bioconc. Factor: 1.60E+01 (estim.) (659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL WATER SYSTEM | Trichlorofluoromethane is expected to be highly mobile in the soil/ground-water system and has a strong potential for migration to groundwater. Volatilization of material near the surface or in the soil-air compartment is important. Transformation processes such as hydrolysis and biodegradation are not expected to be significant in natural soils. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of trichlorofluoromethane to groundwater drinking water supplies. It has been observed in ground water associated with some NPL sites. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (38, 34)<br>Inhalation of high concentrations of trichloro-fluoromethane may cause incoherence, tremors, cardiac arrhythmias and cardiac arrest. It is not known to be an eye irritant. Skin contact with the liquid may cause dermatitis and frostbite.<br><br>Acute Toxicity Studies: (3504)<br><br>INHALATION:<br>$LC_{50}$ 10 pph · 30 min Mouse<br><br>ORAL:<br>$TC_{Lo}$ 50000 ppm · 30 min Human<br><br>Long-Term Effects: No adverse effects reported in two 90-day studies<br>Pregnancy/Neonate Data: Negative<br>Genotoxicity Data: Limited data are negative<br>Carcinogenicity Classification:<br>IARC - None assigned<br>NTP - Inconclusive evidence in rats, negative in mice<br>EPA - No data |

| | |
|---|---|
| HANDLING PRECAUTIONS (3005,3306) | Handle chemicals only with adequate ventilation.<br>• Vapor concentrations of 1000-10,000 ppm; any supplied-air respirator or self-contained breathing apparatus with full facepiece. • Chemical goggles to prevent any possibility of eye contact with liquid.<br>• Protective clothing to prevent repeated or prolonged skin contact. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

### AIR EXPOSURE LIMITS:

Standards
- OSHA Ceiling Limit: 1000 ppm
- AFOSH Ceiling Limit: 1000 ppm

Criteria
- NIOSH IDLH (30 min): 10,000 ppm
- NIOSH REL: none established
- ACGIH CL: 1000 ppm
- ACGIH STEL (15-min): none established

### WATER EXPOSURE LIMITS:

Drinking Water Standards
None established

EPA Health Advisories and Cancer Risk Levels
None established

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria
- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms (1E-05, 1E-06, 1E-07 cancer risk), 1.9, 0.19, 0.019 μg/L total halomethanes.
  - Based on ingestion of contaminated aquatic organisms only, (1E-05, 1E-06, 1E-07 cancer risk), 157, 15.7, 1.57 μg/L total halomethanes.
  - Based on ingestion of contaminated drinking water only (1E-05, 1E-06, 1E-07 cancer risk), 1.9 ug/L, 0.19 μg/L, 0.019 μg/L for total halomethanes.

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 11,000 μg/L total halomethanes.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 12,000 μg/L total halomethanes.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 6,400 μg/L total halomethanes

REFERENCE DOSES:

No reference dose available.

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Trichlorofluoromethane was deleted from the toxic pollutant list in 1981 based on the conclusion of the EPA that no significant potential for exposure to this compound exists via drinking water (742).

Safe Drinking Water Act (SDWA)

Trichlorofluoromethane is listed by EPA as an unregulated contaminant with no EPA monitoring requirements. The states decide which systems require analysis for this contaminant (3771). In states with an approved Underground Injection Control program, a permit is required for the injection of trichlorofluoromethane-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

Trichlorofluoromethane is identified as a toxic hazardous waste (U121) and listed as a hazardous waste constituent (3783, 3784). Non-specific sources of trichlorofluoromethane-containing waste are solvent use and recovery activities, and spent solvent mixtures containing 10% or more trichlorofluoromethane (325, 3765). Trichlorofluoromethane is included on EPA's groundwater monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their groundwater for chemicals on this list when suspected contamination is first detected and annually thereafter (3775). Trichlorofluoromethane is subject to land disposal restrictions when its concentration as a hazardous constituent of certain wastewaters exceeds designated levels (3785). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786).

Toxic Substances Control Act (TSCA)

The manufacture, processing and distribution of fully halogenated chlorofluoroalkanes is prohibited. There are, however, numerous special and essential use exemptions (382).

Comprehensive Environmental Response Compensation and Liability Act (CERCLA)
Trichlorofluoromethane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 2270 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing trichlorofluoromethane but these limits depend upon the concentration of the chemicals present in the waste stream (3766).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
Trichlorofluoromethane is exempt from a tolerance requirement when used as a propellant in pesticide formulations applied to growing crops, raw agricultural commodities after harvest or to animals (315).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
This is a Transitional Limit, in effect until December 30, 1991. A ceiling level of 1000 ppm shall not be exceeded at any time during an 8-hour work-shift (3539).

Clean Air Act (CAA)
Trichlorofluoromethane (CFC-11) is listed as a controlled substance subject to production and consumption limits specified under the Montreal Protocol beginning January 1, 1989. It has an ozone depletion weight of 1.0 (3793).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated trichlorofluoromethane as a hazardous substance with are portable quantity of 2270 kg, subject to requirements for packaging, labeling, and transportation (3180).

Consumer Product Safety Act (CPSA)
Manufacturers of consumer products containing trichlorofluoromethane, a chlorofluorocarbon propellant, are required to label their products with a specific statement concerning the effect of the propellant on the upper atmosphere (311).

Food, Drug and Cosmetic Act (FDCA)
The use of chlorofluorocarbons as propellants in self-pressurized containers for human food is prohibited (363). However, they may be used for drugs under certain conditions (364).

- State Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

CALIFORNIA
California has an action level of 3,400 μg/L for trichlorofluoromethane in drinking water (3098).

KANSAS
Kansas has an action level of 8000 μg/L for groundwater (3213).

NEW YORK
New York has a maximum contaminant level of 5 μg/L for drinking water, and a nonenforceable guideline of 50 μg/L for surface and groundwater (3501).

OKLAHOMA
Oklahoma has a water quality criterion of 0.6 μg/L for groundwater (3213).

WISCONSIN
Wisconsin has a preventive action limit of 698 μg/L and an enforcement standard of 3490 μg/L for groundwater (3840).

Proposed Regulations

- Federal Programs
  None
  No proposed regulations are pending.

- State Programs
  MOST STATES
  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officers is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA
  Minnesota has proposed a Recommended Allowable Limit (RAL) of 2100 μg/L for drinking water (3451).

EEC Directives

Directive on Groundwater (538)
Direct discharge into groundwater (i.e., without percolation through the ground or subsoil) of organic-halogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into groundwater (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenolic compounds; organohalogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances and specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes o the high quality of the shellfish product.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

EEC Directives - Proposed

Council Resolution on Fluorocarbons in the Environment (539)
The European Economic Community recommends that immediate steps be taken to encourage all aerosol and plastic foam industries using fluorocarbons; Trichlorofluoromethane($CCl_3F$) and Dichlorodi-fluoromethane ($CCl_2F_2$) to intensify research into alternative products and to promote development of alternative methods of application. Immediate steps should be taken to encourage manufacturers and users containing chlorofluorcarbons to eliminate the discharge of these compounds. The EEC further recommends that states carry out national research on the problem of possible threat posed by fluorocarbons to the ozone layer.

Resolution on a Revised List of Second-Category Pollutants (545)
Trichlofluoromethane is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

Council Resolution for the Limitation of Use of Chlorofluorbons and Halons (3982)
In pursuant of EEC 322/88 and Council Resolutions on Fluorocarbons in the Environment, EEC proposes that urgent action be taken to limit the use of fluorocarbons and halons in products and equipment containing CFC 11 and CFC 12 and whenever feasible to substitute chlorofluorocarbons and halons in producers, such as aerosols in equipment or processes using them.

## 5.1 MAJOR USES

As recently as 1976, trichlorofluoromethane was the most widely used aerosol propellant. However, the Environmental Protection Agency prohibited the manufacture of fluorocarbons for aerosol propellant uses in 1978 due to theories which associated their use with depletion of the earth's ozone layer (747). Many products, such as medicated aerosols, are exempt from the ban (12). Trichlorofluoro- methane is also used as a plastic foaming agent, refrigerant and solvent (12, 21).

## 5.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 5.2.1 Transport in Soil/Ground-water Systems

#### 5.2.1.1 Overview

Trichlorofluoromethane will be relatively mobile in the soil/ ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways for low soil concentrations can be assessed by equilibrium partitioning, using a model as shown in Table 5-1. These calculations predict the partitioning of trichlorofluoromethane among soil particles, soil water and soil air. The estimates for the unsaturated topsoil model indicate that significant amounts of trichlorofluoromethane are expected to be present in the soil-water (2%) and soil-air phases (30%), and thus available to be transported through these phases by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. Diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, may be the most significant loss pathway, particularly for surface soils. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the trichlorofluoromethane is likely to be present in the soil-water phase (60%) and available for transport with flowing ground water.

#### 5.2.1.2 Sorption on Soils

The mobility of trichlorofluoromethane in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

No information specific to the sorption of trichlorofluoromethane was available. Retardation rates, which represent the interstitial water velocity/pollutant velocity in the soil, were reported by Wilson et al. (82) to be a function of $K_{oc}$, the ratio of soil density (a) to soil water content (b), and the organic content (oc) of the soil according to the following equation:

$$R_f = 1 + (a/b)(K_{oc})(oc).$$

TABLE 5-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR TRICHLOROFLUOROMETHANE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 20°C | 67.4 | 2.2 | 30.4 |
| Saturated deep soil[d] | 40.0 | 60.0 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 159 (33).
c) Henry's law constant taken as 0.11 atm · $m^3$/mol at 20°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

Schwarzenbach et al. (77) report retardation factors for some organic compounds. The data reported for trichloromethane, which has a $K_{oc}$ somewhat lower than that of trichlorofluoromethane, indicate some sorption and decreased mobility in sediments containing 1-2% organic carbon; the retardation factors for soils having less than 0.1% organic carbon suggest little or no sorption. Sorption of trichlorofluoromethane should be equal to or greater than that of trichloromethane.

### 5.2.1.3 Volatilization from Soils

Transport of trichlorofluoromethane vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure

changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

Though no specific data were available, Callahan et al. (10) report that volatilization is expected to be the major transport process for removal of trichlorofluoromethane from aqueous environmental systems. Compared to volatilization from well-stirred aqueous solutions, volatilization of near-surface volatile organics from soils was reported to be slower by approximately one order of magnitude (82).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H were observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect volatilization from surface soils.

No information was available for the two other physicochemical properties influencing trichlorofluoromethane volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

### 5.2.2 Transformation Processes in Soil/Ground-water Systems

Data specific to the transformation of trichlorofluoromethane in soil/ground-water systems were not available. Most references indicate that low molecular weight chloroaliphatics are not metabolized (10). However, Thom and Agg (80) have included several halomethanes in a list of organic chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as trichlorofluoromethane would be very low and would drop off sharply with increasing depth. Thus, biodegradation should be assumed to be of minimal importance and unlikely to compete with the rate of volatilization.

### 5.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggest that trichlorofluoromethane in the environment is highly volatile, moderately sorbed to soil, and has a low potential for bioaccumulation. Trichlorofluoromethane on the soil surface is likely to volatilize, but that portion not subject to volatilization is likely to be mobile in ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of trichlorofluoromethane from a disposal site could result in inhalation exposures. The potential for ground water contamination is high, particularly in sandy soils. Mitre (83) reported that trichlorofluoromethane has been found at seven of the 546 National Priority List (NPL) sites. It was detected at six sites in ground water, and one site in surface water. The infrequent detection is probably due to the limited production of this compound. The lack of detection of

trichlorofluoromethane in air may be somewhat misleading as air sampling at NPL sites has been limited.

The potential for movement of trichlorofluoromethane to surface water via movement in soil/ground-water system, suggests several other exposure pathways:

- Surface waters may be used as drinking water supplies and result in direct ingestion exposure.
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposures through bioaccumulation.
- Recreational use of these waters may result in dermal exposures.
- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground water for two reasons. First, the Henry's law constant for trichlorofluoromethane suggests that it will likely volatilize upon reaching surface waters. Secondly, the bioconcentration factor for trichlorofluoromethane is low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 5.2.4 Other Sources of Human Exposure

Little information is available on other sources of exposure to trichlorofluoromethane. It is used in a fire-extinguisher formulation (213). Direct exposure could occur if such a formulation is in actual use. It is used as a refrigerant and occupational exposure may result during manufacture of various products.

Trichlorofluoromethane was not reported in the summary of air monitoring data collected by Brodzinski and Singh (84). Exposure through drinking water or food has also not been reported extensively, although migration in ground water has occurred in a few cases. Other sources of exposure to trichlorofluoromethane are therefore likely to be limited.

## 5.3 HUMAN HEALTH CONSIDERATIONS

### 5.3.1 Animal Studies

#### 5.3.1.1 Carcinogenicity

Technical-grade trichlorofluoromethane was not carcinogenic to male or female B6C3F$_1$ mice when administered in corn oil by gavage, 5 days per week for 71 weeks. Time-weighted-average doses were 3925 and 1962 mg/kg/day for mice of both sexes. Mice were observed for an additional period of up to 13 weeks after the dosing period. No significant increases in tumor incidences or unusual tumors were observed among the treated animals. A similar study was conducted in Osborne-Mendel rats. Male rats were administered doses of 977 or 488 mg/kg/day and females received 1077 or 538 mg/kg/day for 5 days per week for 66 weeks. After the end of the dosing period, rats were observed for an additional period of up to 33 weeks. The results of the rat study were inconclusive because an inadequate number survived long enough to be at risk from late-developing tumors (747).

Negative results were also obtained by Epstein et al. (748) in a one-year bioassay of ICR/Ha Swiss mice in which 10% solutions of trichlorofluoromethane in tricaprylin were subcutaneously injected into the neck of neonatal mice (0.1 mL into 1- and 7-day-old mice, then 0.2 mL into 14- and 21-day-old mice).

#### 5.3.1.2 Genotoxicity

Genotoxicity data on trichlorofluoromethane are limited. No work on chromosomal effects has been reported. Negative results have been obtained in five strains (TA98, 100, 1535, 1537, 1538) of Salmonella with or without metabolic activation (3859, 3401). In experiments designed to assure testing of volatile liquids, trichlorofluoromethane was negative in inducing forward mutations in Chinese hamster ovary cells with or without exogenous metabolic activation (744).

#### 5.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

There are no published studies on the effects of trichlorofluoromethane on reproduction (12). Negative results were obtained in a test of teratogenicity conducted with rats and rabbits exposed to a 20% mixture of 10% trichloro- fluoromethane, and 90% dichlorodifluoromethane administered by inhalation 2 hours daily either from days 4-16 or 5-20 of gestation in rats and rabbits, respectively (745). Teratology studies carried out by I.C.I., Mond Division, U.K., were reviewed by Barlow and Sullivan (3053). Although the studies showed no teratogenic effects from trichlorofluoromethane, no study details were given in the review article. However, from the discussion of a companion study (3053), it can be implied that rat offspring were exposed at a level of 50,000 ppm (length of study and numbers tested were not given).

### 5.3.1.4 Other Toxicologic Effects

#### 5.3.1.4.1 Short-term Toxicity

Trichlorofluoromethane is a central nervous system depressant in animals (46). Rats are the most sensitive to its CNS effects with a 30 minute exposure to 9% of the compound causing complete unconsciousness; a 20 to 30 minute exposure to 10% trichlorofluoromethane was lethal (746). Guinea pigs are more resistant to its CNS depressant effects, requiring 1 hour exposure to 20% to produce lethality (749).

Exposure to a concentration of 10 parts per hundred of trichlorofluoromethane for 30 minutes was recorded as an $LC_{50}$ value for mice (47) and as an $LC_{Lo}$ value for rats (47).

Repeated inhalation exposure to trichlorofluoromethane does not appear to produce pathological lesions of the liver or other visceral organs (750). No such lesions were seen in guinea pigs, rats or cats after a 20-day exposure to 1.25 or 2.5% trichlorofluoromethane for 3.5 hours per day (750).

The most significant toxicological effects of trichlorofluoromethane are on the cardiovascular and bronchopulmonary systems. Dogs and monkeys exhibit arrhythmias upon exposure to trichlorofluoromethane at a lower concentration than that needed to induce the same response in rats and mice (751). The circulatory systems of the monkey and dog can be influenced by a concentration of 0.5% trichlorofluoromethane inhaled for about 5 minutes. This level caused cardiac arrhythmias in an unanesthetized dog and in anesthetized monkeys. The sensitivity of the monkey heart is increased by epinephrine infusion or by coronary arterial occlusion (12). Mice and rats, on the other hand, require a concentration of 2.5 to 5.0% trichlorofluoromethane in order to affect the circulatory system. When anesthetized, both species respond with bradycardia. In the unanesthetized rat, only tachycardia results (12). Acute inhalation of trichlorofluoromethane can also induce cardiac arrhythmias in these 4 species. Coronary ischemia, cardiac necrosis, epinephrine injection, experimental bronchitis and pulmonary thrombosis increase the sensitivity of the heart to arrhythmias by increasing cardiac irritability. On the other hand, general anesthesia and adrenalectomy decrease the sensitivity of the heart to the pro-arrhythmic action of trichlorofluoromethane (12).

The information on the bronchopulmonary effects of trichlorofluoromethane is not as extensive as that on the cardiovascular system. A decrease in respiratory volume was observed in dogs and mice exposed to vapor concentrations of 2.5% and in monkeys exposed to 5% trichlorofluoromethane (12). Airway resistance was decreased in anesthetized monkeys and dogs by concentrations of 5% and 2.5%, respectively. Conversely, anesthetized rats exhibited increases in airway resistance beginning at concentrations of 2.5% (12). Concentrations ranging from 1 to 5% did not affect the pulmonary compliance of monkeys or dogs but inhalation of 2.5% caused a decreased compliance in the rat (12).

Trichlorofluoromethane has no known systemic toxic effect on the eye (19). It is reported to have produced no harmful effects on rabbit eyes following 9 applications of 0.1 mL, made over an 11-day period (19). Applications of trichlorofluoromethane alone or in combination with other fluorocarbons produced irritation, inflammation and edema of the skin and oral mucosa of rats. The applications were made twice daily over several weeks. These effects were most marked after application of a trichlorofluoromethane/chlorodifluoromethane mixtures to older animals (755). The healing rate of burn lesions was retarded by trichlorofluoromethane application (755).

#### 5.3.1.4.2 Chronic Toxicity

In the few chronic studies of trichlorofluoromethane toxicity carried out in animals, no effects were observed in guinea pigs, rats and cats exposed to 0.1% trichlorofluoromethane continuously for 90 days (752) or in dogs or rats exposed to vapor levels of 5000 or 10,000 ppm, respectively, 6 hours daily for 90 days (753).

### 5.3.2 Human and Epidemiologic Studies

#### 5.3.2.1 Short-term Toxicologic Effects

Human experience with fluorocarbons has largely involved the intentional or unintentional misuse of fluorocarbon products, resulting in acute inhalation of high vapor concentrations (755). The primary human hazard from trichlorofluoromethane inhalation is the induction of cardiac arrhythmias (755). Hundreds of deaths have been attributed to the abuse of aerosols containing trichlorofluoromethane, but the vapor levels in these cases are difficult to quantify (12). Post-mortem tissue examinations reveal a distribution similar to that of chloroform, with a significant accumulation in the brain, liver and lungs. Death in these cases, was due to severe cardiac arrhythmias (12).

Experiments performed by Stewart et al. (756) on human volunteers exposed to concentrations of 0.1, 0.05 or 0.025% trichlorofluoromethane revealed no arrhythmogenic effects at these concentrations.

Trichlorofluoromethane is not a known eye irritant (19, 38). Chilling or freezing may be the principal hazard in acute dermal exposure (755).

#### 5.3.2.2 Chronic Toxicologic Effects

There are no reports of long-term human exposure to trichlorofluoromethane.

### 5.3.3 Levels of Concern

For the maximum protection of human health from the potential carcinogenic effects due to exposure to halomethanes (including trichlorofluoromethane) through the ingestion of contaminated water and aquatic organisms, the USEPA (355) has

established that the ambient water concentration of total trihalomethanes should preferably be zero. Additional lifetime cancer risks of 1E-05, 1E-06 and 1E-07 are estimated from the ingestion of both water and contaminated aquatic organisms at a criterion level of 1.9, 0.19 and 0.019 μg/L halomethanes, respectively. Risk estimates are expressed as a probability of cancer after a lifetime daily consumption of 2 liters of water and 6.5 g of fish that bioaccumulate trihalomethanes. Thus, a risk of 10E-5 implies that ingestion of 2 liters of water and 6.5 g contaminated fish daily at the criterion level of 1.9 μg/L would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 people exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

The OSHA (3539) standard is 1000 ppm (5600 $mg/m^3$) averaged over an 8-hour work-shift. The ACGIH (3005) recommends 1000 ppm as a ceiling value for trichlorofluoromethane.

### 5.3.4 Hazard Assessment

Trichlorofluoromethane appears to have little systemic toxicity in humans except at very high vapor concentrations which appear to sensitize the heart, resulting in severe cardiac arrhythmias, and often death. Experiments performed on human volunteers exposed to concentrations up to 0.1% trichlorofluoromethane indicated no arrhythmogenic effects (756).

Negative findings have been reported for tests of the carcinogenic, mutagenic and teratogenic potential of trichlorofluoromethane.

## 5.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of trichlorofluoromethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of trichlorofluoromethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. Since field samples can be contaminated by diffusion of volatile organics such as trichlorofluoromethane through the septum of the sample container during shipment and storage, a field blank sample must be included in the analytical program to insure against false positive identification of trichlorofluoromethane. Recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples and field blanks, duplicate samples and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of trichlorofluoromethane in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas

is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the trichlorofluoromethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the trichlorofluoromethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; trichlorofluoromethane is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). Direct injection may also be used for samples that contain high concentrations of chemical. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors, or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for chloroform analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical trichlorofluoromethane detection limits for the various methods were not determined but would be in the range of 1-10 $\mu$g/L for aqueous samples and 1-10 $\mu$g/kg for non-aqueous samples.

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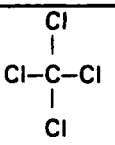
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| COMMON SYNONYMS: | CAS REG.NO.: 56-23-5 FORMULA: $CCl_4$<br>NIOSH NO: FG4900000<br>STRUCTURE:  | AIR W/V CONVERSION FACTOR at 25 °C (12)<br>6.29 mg/m³ ≈ 1 ppm;<br>0.159 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 153.84 |
|---|---|---|
| Carbon chloride<br>Carbon tetrachloride<br>Methane tetrachloride<br>Perchloromethane<br>Tetrachlorocarbon<br>Tetrachloromethane | | |

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| REACTIVITY | Reactions of halogenated organic materials such as carbon tetrachloride with cyanides, mercaptans or other organic sulfides typically generate heat, while those with mineral acids, amines, azo compounds, hydrazines, caustics, or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases, and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511). Known explosions have been caused by reactions with burning wax, calcium hypochlorite plutonium, uranium, borane, allyl alcohol, certain aluminum compounds, barium, silanes, silver perchlorate and other substances listed above (505). |

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| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Sweet, pungent | (263) |
| | • Odor Threshold: 21.000 to 100.000 ppm | (263) |
| | • Density: 1.5947 g/mL (at 20°C | (21) |
| | • Freeze/Melt Point: -23.00°C | (23) |
| | • Boiling Point: 76.74°C | (23) |
| | • Flash Point: Nonflammable | (23) |
| | • Flammable Limits: Nonflammable | (504,507) |
| | • Autoignition Temp.: Nonflammable | (504,507) |
| | • Vapor Pressure: 9.13E+01 mm Hg (at 20°C) | (23) |
| | • Satd. Conc. in Air: 7.5400E+05 mg/m³ (at 20°C) | (67) |

| | |
|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Solubility in Water: 8.00E+02 mg/L (at 20°C) (38)<br>• Viscosity: 0.965 cp (at 20°C) (21)<br>• Surface Tension: 2.6770E+01 dyne/cm (at 20°C) (21)<br>• Log (Octanol-Water Partition Coeff): 2.83 (29)<br>• Soil Adsorb. Coeff: 4.39E+02 (33)<br>• Henry's Law Const: 2.00E-02 $atm \cdot m^3/mol$ (74)<br>• Bioconc. Factor: 3.00E+01 (bluegill), 3.20E+01 (estim) (102,659) |

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| PERSISTENCE IN THE SOIL-WATER SYSTEM | Carbon tetrachloride is expected to be sorbed strongly onto surface soils, and less strongly in deep or sandy soils. Removal by volatilization may occur for near-surface carbon tetrachloride. Transformation processes such as hydrolysis and biodegradation are not expected to be significant in natural soils. |

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| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of carbon tetrachloride to groundwater drinking water supplies. There is extensive evidence that such migration has occurred in the past. Inhalation resulting from volatilization from surface soils may also be important. |

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| HEALTH HAZARD DATA (3933) | Signs and Symptoms of Short-term Human Exposure: (54)<br>Exposure may cause drowsiness, dizziness, incoordination and mental confusion; nausea, vomiting, diarrhea and abdominal pain are frequent. Delayed effects from short-term over-exposure include damage to the heart, liver and kidneys. Repeated or prolonged skin contact with the liquid may cause irritation. Eye contact with the liquid causes pain and minimal injury to the conjunctiva. |

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| HEALTH HAZARD DATA (Cont.) | Acute Toxicity Studies: (3504)<br><br>INHALATION:<br>$LC_{50}$ 59920 mg/m$^3$ · 8 hr — Mouse<br>$TC_{Lo}$ 20 ppm — Human<br><br>ORAL:<br>$LD_{50}$ 2800 mg/kg — Rat<br>$TD_{Lo}$ 1700 mg/kg — Woman<br>$TD_{Lo}$ 1800 mg/kg — Man<br><br>SKIN:<br>$LD_{50}$ 5070 mg/kg — Rat<br><br>Long-Term Effects: Liver and kidney injury<br>Pregnancy/Neonate Data: Fetotoxic<br>Genotoxicity Data: Conflicting evidence<br>Carcinogenicity Classification:<br>IARC - Group 2B (possibly carcinogenic to humans)<br>NTP - None assigned<br>EPA - Group B2 (probable human carcinogen; sufficient evidence in animals and inadequate evidence in humans) |

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| HANDLING PRECAUTIONS (38,52,134) | Handle chemical only with adequate ventilation.<br>• Vapor concentrations of 10-100 ppm: any supplied-air respirator or self-contained breathing apparatus.<br>• 100-300 ppm: any supplied air respirator or self-contained breathing apparatus with full facepiece.<br>• Above 300 ppm: self-contained breathing apparatus with full facepiece operated in positive pressure mode.<br>• Chemical goggles if there is probability of eye contact.<br>• Natural rubber, neoprene, nitrile, PE, PVA, PVC gloves/ apron/boots and protective clothing to prevent repeated or prolonged skin contact with the liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

### AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 2 ppm
- AFOSH PEL (8-hr TWA): 2 ppm; STEL (15-min): 4 ppm

Criteria
- NIOSH IDLH (30 min): deleted, NIOSH has recommended that the substance be treated as a potential human carcinogen.
- NIOSH REL: 2 ppm ceiling limit (45L, 60-min sample)
- ACGIH TLV® (8-hr TWA): 5 ppm (skin) (A2, suspected human carcinogen)

### WATER EXPOSURE LIMITS:

Drinking Water Standards (3742)
MCLG: 0 μg/L
MCL : 5 μg/L

EPA Health Advisories and Cancer Risk Levels (3977)
The EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- 1-day (child): 4000 μg/L
- 10-day (child): 200 μg/L
- longer-term (child): 70 μg/L
- longer-term (adult): 300 μg/L
- 1E-04 Cancer Risk Level: 30 μg/L

WHO Drinking Water Guideline (666)
A tentative, health-based guideline for drinking water of 3 μg/L has been proposed for carbon tetrachloride. A daily per capita consumption of two liters of water was assumed.

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

EPA Ambient Water Quality Criteria

- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms (1E-05, 1E-06, 1E-07 cancer risk), 4.0 µg/L, 0.40 µg/L, 0.04 µg/L.
  - Based on ingestion of contaminated aquatic organisms only, (1E-05, 1E-06, 1E-07 cancer risk), 69.4 µg/L, 6.94 µg/L, 0.694 µg/L.

  Drinking water only: not available.
- Aquatic Life (355)
  - Freshwater species

    acute toxicity:
    no criterion, but lowest effect level occurs at 35,200 µg/L.

    chronic toxicity:
    no criterion established due to insufficient data.
  - Saltwater species

    acute toxicity:
    no criterion, but lowest effect level occurs at 50,000 µg/L.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:

ORAL: 7.000E-01 µg/kg/day (3744)

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Carbon tetrachloride is designated a hazardous substance under CWA. It has a reportable quantity (RQ) limit of 2270 kg (347, 3764). It is also listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and to effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), and metal finishing (3768). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

Carbon tetrachloride is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). Under the National Interim Primary Drinking Water Regulations, the MCL for carbon tetrachloride in drinking water is set at 0.005 mg/L, the MCLG at zero (3773, 3772). In states with an approved Underground Injection Control program, a permit is required for the injection of carbon tetrachloride-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

Carbon tetrachloride is identified as a toxic hazardous waste (U211) and listed as a hazardous waste constituent (3783, 3784). Non-specific sources of carbon tetrachloride-containing waste are solvent use (or recovery) activities, chlorinated aliphatic hydrocarbon production, and spent solvent mixtures containing 10% or more carbon tetrachloride (325). Waste streams from the following industries contain carbon tetrachloride and are listed as specific sources of hazardous waste: organic chemicals (production of 1,2-dichloroethane, vinyl chloride, toluene diisocyanate, carbon tetrachloride and fluoromethanes) and inorganic chemicals (chlorine production) (326, 3774). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). Carbon tetrachloride is included on EPA's groundwater monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their groundwater for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Comprehensive Environmental Response Compensation and Liability Act (CERCLA)
Carbon tetrachloride is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 2270 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing carbon tetrachloride but these limits depend upon the concentration of the chemicals present in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of carbon tetrachloride must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
Carbon tetrachloride is exempt from a tolerance requirement when used as a fumigant after harvest for barley, corn, oats, popcorn, rice, rye, sorghum and wheat (315).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to carbon tetrachloride shall not exceed an 8-hour time-weighted average (TWA) of 2 ppm (3539).

Clean Air Act (CAA)
EPA intends to list carbon tetrachloride as a hazardous air pollutant for which it will establish emission standards under Section 112 of the Clean Air Act (3685).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated carbon tetrachloride as a hazardous material with a reportable quantity of 2270 kg, subject to requirements for packaging, labeling and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
Carbon tetrachloride is approved for use as an indirect food additive as a component of adhesives (3209).

- State Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. Other states have promulgated additional or more stringent criteria.

ALABAMA
Alabama requires that the annual average of carbon tetrachloride in drinking water not exceed a maximum contaminant level of 0.005 mg/L. This applies to all community water systems and non-community non-transient water systems (3015).

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. Other states have promulgated additional or more stringent criteria.

CALIFORNIA
California has an action level of 5 μg/L (ppb) for drinking water (3098).

CONNECTICUT
Connecticut has a quantification limit of 2.0 μg/L for drinking water (3137).

FLORIDA
Florida has a maximum contaminant level of 0.003 mg/L for carbon tetrachloride in drinking water (3219).

KANSAS
Kansas has a maximum contaminant level of 5 μg/L for groundwater (3213).

NEW JERSEY
New Jersey has set an MCL of 2 μg/L for drinking water (3497).

NEW YORK
New York has set a water quality standard of 5 μg/L for ground water used as a drinking water supply (3501).

OKLAHOMA
Oklahoma has a nonenforceable Toxic Substance Goal of 0.4 μg/L for surface waters for public and private water supply, and a water quality criterion of 0.4 μg/L for groundwater (3534).

PENNSYLVANIA
Pennsylvania has a human health criterion (cancer risk level) of 0.3 μg/L for surface waters (3561).

RHODE ISLAND
Rhode Island has an acute freshwater quality guideline of 1365 μg/L and a chronic guideline of 30 μg/L for the protection of aquatic life in surface waters. These guidelines are enforceable under Rhode Island state law (3590).

VERMONT
Vermont has a preventive action limit of 0.5 μg/L, and an enforcement standard of 5 μg/L for groundwater (3682).

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed that solid wastes be listed as hazardous in that they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is equal to or greater than 0.07 mg/L carbon tetrachloride. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Programs

  MOST STATES
  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officers is advised. Changes are projected for 1989-90 (3683).

  CALIFORNIA
  California has proposed a maximum contaminant level of 0.5 μg/L for carbon tetrachloride in drinking water (3096).

MINNESOTA
Minnesota has proposed a Recommended Allowable Limit (RAL) of 2.7 μg/L for drinking water. Minnesota has also proposed a Sensitive Acute Limit (SAL) of 8800 μg/L for designated surface waters, and chronic criteria of 1.9 μg/L for designated surface waters and 2.7 μg/L for designated groundwaters. These criteria are for the protection of human health (3451, 3452).

NEW JERSEY
New Jersey has proposed a surface water quality criterion of 2 μg/L for FW2 waters (3496).

WEST VIRGINIA
West Virginia has proposed a water quality criterion of 0.25 μg/L for Public A surface waters. Final action is expected in late spring 1989 (3835).

EEC Directives

Directive on Ground-Water (538)
Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
Carbon tetrachloride is listed as a Class I/a toxic substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used or disposing of such waste.

Directive on the Classification, Packaging and Labeling of Pesticides (786)
Carbon tetrachloride is listed as a class I/a substance and is subject to packaging and labeling regulations.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Carbon tetrachloride is classified as a toxic substance and is subject to packaging and labeling regulations.

Directive on Limit Values and Quality Objectives for Discharges of Certain Dangerous Substances (1792)
Pursuant to the Directive on the Discharge of Dangerous Substances, the quality objective for carbon tetrachloride is 12 $\mu$g/1 $CC1_4$. The emission standard of carbon tetrachloride production by perchlorination is 1.5 mg/L for s monthly average and 2 mg/L for a daily average. These regulations must be complied with as of January 1, 1988.

Directive on the Approximation of the Laws, Regulations and Administrative Provisions Relating to the Classification, Packaging and Labeling of Dangerous Preparations (3991)

The labels on packages containing preparations classified as very toxic, toxic or corrosive must bear the safety advise S1/S2 and S46 in addition to the specific safety advice. If it is physically impossible to give such information, the package must be accompanied by precise and easily understood instructions.

EEC Directives--Proposed Resolution

Resolution on the Revised List of Second-Category Pollutants (545) Carbon tetrachloride is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 6.1 MAJOR USES

Carbon tetrachloride is used primarily in the production of fluorocarbons such as dichlorofluoromethane and trichlorofluoromethane. Miscellaneous applications account for 9% of total usage. These include pesticide and grain fumigant use. Its use in fire extinguishers has essentially disappeared. It has also been used as a vermicidal agent in human medicine and as a dry-cleaning and fabric spotting fluid. Due to its toxicity, the FDA banned its use in consumer products in 1970 (12, 21, 25).

## 6.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 6.2.1 Transport in Soil/Ground-water Systems

#### 6.2.1.1 Overview

Carbon tetrachloride may be relatively mobile in the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). Transport pathways can be generally assessed by using an equilibrium partitioning model as shown in Table 6-1. These calculations predict the partitioning of low soil concentrations of carbon tetrachloride among soil particles, soil water and soil air. The estimates from the unsaturated topsoil model show that small percentages of carbon tetrachloride will partition to the soil-water and soil-air phases to be transported through these phases by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion (e.g., diffusion through the soil-air pores up to the ground surface for subsequent removal by wind). In saturated, deep soils (containing no soil air and negligible soil organic carbon), a higher percentage of carbon tetrachloride (35%) can be expected in the soil-water phase (Table 6-1) and may be transported with flowing ground-water. Sorption onto soil, however, still appears to be significant for carbon tetrachloride in deep soil.

#### 6.2.1.2 Sorption on Soils

The mobility of carbon tetrachloride in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of sorption on soil particles. Sorption of carbon tetrachloride is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water;
- decrease moderately with increasing dissolved organic matter content of the soil water.

There are no available data addressing the sorption of carbon tetrachloride on soil particles. Carbon tetrachloride has a higher $K_{oc}$ than the other chlorinated methanes; therefore, its sorption onto soils is also expected to be higher.

TABLE 6-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR CARBON TETRACHLORIDE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 25 °C | 96.0 | 1.1 | 2.9 |
| Saturated deep soil[d] | 64.8 | 35.2 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 439 (33).
c) Henry's law constant taken as 0.02 atm · $m^3$/mol at 25 °C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

Schwarzenbach et al. (77) and Wilson et al. (82) have reported retardation factors, which represent the interstitial water velocity/ pollutant velocity in the soil, for several chlorinated organics. Retardation in soils was reported to be a function of pollutant $K_{oc}$, the ratio of soil density (a) to soil water content (b), and the organic content (oc) of the soil according to the following equation:

$$R_f = 1 + (a/b)(K_{oc})(oc)$$

The data reported for organics with lower $K_{oc}$ values than that reported for carbon tetrachloride indicate some retention in soils having 1-2% organic carbon and little or no retention (i.e., adsorption) in deep soils having less than 0.1% organic carbon. Assuming analogous soil conditions, sorption of carbon tetrachloride onto soils is expected to be significant for surface soils with considerable organic content, and less important for deep soils.

### 6.2.1.3 Volatilization from Soils

Transport of carbon tetrachloride vapors through the air-filled pores of unsaturated soils may be an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

Half-lives on the order of 29 minutes have been reported for the volatilization of carbon tetrachloride from a constantly stirred 1 mg/L aqueous solution (10). Smith et al. (78) reported that the ratio of the rate of carbon tetrachloride volatilization to oxygen reaeration is constant. Using estimated oxygen reaeration rates for a river, pond and lake, they reported volatilization rates corresponding to half-lives of 1.2 days from the river, 5.8 days from the pond and 4.8 days from the lake. Compared to volatilization from well-stirred aqueous solutions, volatilization of several chlorinated organics from soil has been reported to be slower by approximately one order of magnitude (82).

The Henry's law constant, which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H are also expected with increasing salinity of the aqueous solution and the presence of other dissolved organic compounds (18). These results suggest that environmental soil conditions and the presence of other chemicals in the soil/ground-water system may significantly affect the volatilization of carbon tetrachloride from surface soils.

No information was available for the two other physicochemical properties influencing carbon tetrachloride volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

### 6.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of carbon tetrachloride in soil/ground-water systems is not well documented. In most cases, it should be assumed that carbon tetrachloride will persist for months to years (or more). Carbon tetrachloride that has been released into the air will eventually be transported from the troposphere to the stratosphere where it will undergo photochemical oxidation; a tropospheric lifetime of 330 years has been reported for carbon tetrachloride (10).

Carbon tetrachloride does not undergo rapid hydrolysis under normal environmental conditions. A maximum hydrolytic half-life of 7000 years was reported for 1 mg/L carbon tetrachloride at pH 7 and 25 °C; under the same conditions a half-life of 7 years was reported for 1000 mg/L carbon tetrachloride (10). Hydrolysis of carbon tetrachloride has been suggested to occur in the ocean (73).

No information pertaining specifically to the biodegradation of carbon tetrachloride was found. The few available literature references report that low molecular weight chloroaliphatics are not metabolized (10). Thom and Agg (80) have included carbon tetrachloride on a list of organic chemicals considered to be potentially degradable by biological sewage treatment, provided suitable acclimatization can be achieved; they also noted that it is unlikely that microorganisms already possess the ability to degrade carbon tetrachloride. Since the concentration of microorganisms capable of biodegradation is very low and drops off significantly with increasing depth, biodegradation of carbon tetrachloride in the soil/ground-water system is assumed to be minimal except, perhaps, in landfills with active microbiological populations.

### 6.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggest that carbon tetrachloride is highly volatile in aqueous solutions, and has a moderate potential for adsorption to soil, and a low potential for bioaccumulation. Carbon tetrachloride on the soil surface is likely to volatilize, but that portion not removed by volatilization is likely to be mobile in ground-water. These fate characteristics suggest several potential exposure pathways.

Volatilization of carbon tetrachloride from a disposal site could result in inhalation exposures. In addition, the potential for ground-water contamination is high, particularly in sandy soils. The potential for exposure through ground-water serving as drinking water supplies is confirmed by the pervasiveness of carbon tetrachloride in drinking water in the U.S. The USEPA (62,64) reported the following results from a variety of drinking water supplies.

| Survey | No. Sampled | No. Positive | Range of Positives ($\mu$g/L) |
|---|---|---|---|
| State Data | 2646 | 368 | Trace - 1300 |
| NOMS | 113 | 14 | 0.2 - 29 |
| NSP | 142 | 37 | Trace - 30 |
| CWSS | 452 | 9 | 0.5 - 2.0 |
| GWSS (random data) | 466 | 15 | 0.2 - 16 |

The state data include only ground-water sources and were compiled from various state reports on local contamination problems. The state data are not considered to be statistically representative of national occurrence. The National Organics Monitoring Survey (NOMS) included data from both ground- and surface supplies, as did the National Screening Program (NSP) and the Community Water Supply Study (CWSS). The 1982 Ground-water Supply Survey (GWSS) is the most recent study (531). This survey sampled a total of almost 1000 drinking water systems using ground-water; 466 selected at random, and about 500 selected by the state as potentially contaminated. The USEPA (64) estimates that 1.6% of the nation's ground-water supplies are contaminated with carbon tetrachloride ($\geq$0.5 $\mu$g/L).

In addition to this evidence of migration, Mitre (83) reported that carbon tetrachloride was reported at 22 of the 546 National Priority List (NPL) sites. It was detected at 18 sites in groundwater, 9 sites in surface water and 2 in air.

These data indicate that the movement of carbon tetrachloride in ground-water may be an important source of exposure. The subsequent contamination of surface water may result in a number of other exposure pathways:

- Surface water may also be used as drinking water supplies resulting in direct ingestion exposure.
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposure through bioaccumulation.
- Recreational use of these waters may result in dermal exposure.
- Domestic animals may consume or be dermally exposed to contaminated ground- or surface waters; the human consumption of meats and poultry could then result in ingestion exposure.

In general, exposures associated with surface water contamination can be expected to be lower than exposures from drinking contaminated ground-water for two reasons. First, the Henry's law constant for carbon tetrachloride suggests that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for carbon tetrachloride is low and concentrations would not be expected in fish or domestic animals that would result in exposures from those foods greater than drinking water.

### 6.2.4 Other Sources of Human Exposure

Carbon tetrachloride is used primarily as an intermediate in the production of fluorocarbons. However, its use in fire extinguishers, as a grain fumigant, and as a cleaning agent could result in direct human exposure.

The drinking-water survey data presented in the previous section show that carbon tetrachloride is a common contaminant in drinking water, both in surface and ground-water supplies.

The production and use of carbon tetrachloride has led to its presence in the atmosphere. Brodzinsky and Singh (84) summarized available air monitoring data for a variety of pollutants. For carbon tetrachloride, they reported 2760 data points. In rural and remote areas, the median concentration was 0.82 $\mu g/m^3$; in urban and suburban areas, the median concentration was 1.2 $\mu g/m^3$; and in source-dominated areas, it was 3.7 $\mu g/m^3$ These authors noted that carbon tetrachloride is a ubiquitous air contaminant, nearly uniformly distributed at background concentrations.

## 6.3 HUMAN HEALTH CONSIDERATIONS

### 6.3.1 Animal Studies

#### 6.3.1.1 Carcinogenicity

Carbon tetrachloride has produced liver tumors in mice, hamsters and rats by several routes of administration, providing sufficient evidence that carbon tetrachloride is carcinogenic in experimental animals, and a potential human carcinogen.

The National Cancer Institute (101) conducted a study with B6C3F$_1$ mice and Osborne-Mendel rats. Male and female mice received carbon tetrachloride in corn oil at doses of 1250 or 2500 mg/kg body weight by gavage 5 days per week for 78 weeks. Surviving mice were sacrificed 92 weeks from the start of the study. Liver carcinoma were found in 96 to 100% of the mice, including those dying before the end of the test. The incidence in the controls ranged from 1 to 11%.

In the rat study, males received doses of 47 or 94 mg/kg body weight and females received 80 or 160 mg/kg body weight, 5 times a week by gavage. The carbon tetrachloride was administered in corn oil. The surviving animals were killed at 110 weeks. Hepatocellular carcinoma and neoplastic nodules of the liver were found at both dosage levels in both sexes; however, their incidence was not statistically significant (100).

In a short-term study, ten 12-week-old hamsters of each sex received 30 weekly doses (by gavage) of 10-20 mg in a 5% corn oil solution. Among the 5 animals of each sex that were alive 10 or more weeks after termination of treatment, all had liver-cell carcinomas (647).

In a 68-week-study in which male rats were given subcutaneous doses of 1.3 mL/kg bw (2 g/kg bw) of a 50% carbon tetrachloride solution in corn oil twice weekly, 33% of the Wistar rats, 61.5% of the Osborne-Mendel rats and 80% of the Japanese rats that survived 68 or more weeks had hepatocellular carcinomas. No liver tumors were found in the controls (648). IARC classifies carbon tetrachloride as a group 2B carcinogen (possibly carcinogenic to humans).

#### 6.3.1.2 Genotoxicity

Carbon tetrachloride does not appear to be mutagenic in bacteria. It has been found to be negative in the Salmonella/microsome Ames test in all strains with or without activation (3860, 3081), and is negative in inducing SOS DNA repair functions in Escherichia coli (3081) and in Salmonella typhimurium (3479).

Callen et al. (649) reported that carbon tetrachloride induced recombination and mitotic gene conversion as well as point mutations when concentrations of 21, 28 and

34 mM were incubated with Saccharaomyces cerevisiae D7. No mammalian metabolic activation system was required.

Carbon tetrachloride also induced positive morphological transformations in Syrian hamster embryo cells (680). Pereira and Change (3562) demonstrated that carbon tetrachloride binds to mammalian hemoglobin. Rats fed carbon tetrachloride intragastrically showed a dose response for binding to hemoblobin 24 hrs after treatment. Mirsalis et al. (3455) showed that the hepatocytes of F344 male rats gavaged with carbon tetrachloride showed a 40-fold increase in DNA replication because carbon tetrachloride is a potent hepatotoxin, yet there was no increase above control levels in unscheduled DNA synthesis at 2, 12, 24, and 48 hrs after treatment in hepatocytes cultured from these animals. In a similar study with similar results, Doolittle et al. (3179) gavaged male mice with single and multiple doses of carbon tetrachloride and measured normal DNA synthesis as well as unscheduled DNA synthesis. These authors feel that induction of replicative DNA synthesis is related to hepatotoxicity, but no evidence for unscheduled DNA synthesis was found in these mice. Most published in vivo mammalian studies focus on DNA synthesis, its inhibition, and induction and repair of DNA breaks. An example is Storer et al (3687) who injected male mice with carbon tetrachloride as the negative control (the other negative control compound was corn oil) and subsequently looked for DNA breaks in liver cells of the treated mice. Carbon tetrachloride may affect the mitotic mechanism of cells. In an in vitro study with Chinese hamster V79 cells, carbon tetrachloride induced aneuploidy (3537). There are few reports on attempts to induce chromosome aberrations with carbon tetrachloride. When 50% of the growth inhibiting dose of carbon tetrachloride was added to rat liver cell cultures, chromosomal effects were within the control range (3164). Lil'p and coworkers in the USSR inject carbon tetrachloride intramuscularly to stimulate liver cells to divide so that they can study the effects of other agents (e.g., gamma rays, mitomycin C, thiophosphamide) on chromosomal aberrations (3377, 3396).

### 6.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Barlow and Sullivan (3053) published an excellent review of the effect of carbon tetrachloride on adult reproductive organs and functions, summarized as follows. A single ip injection of 1.5 mL/kg carbon tetrachloride to male rats caused a decrease in the weight of the testes and seminal vesicles and histological evidence of testicular atrophy and abnormal spermatogenesis. Similar results were obtained by other investigators. Chronic exposure of rats by inhalation caused no effect on the testes at 400 ppm for 1 hr/da, 5 da/wk for 6 wk. Exposure to 200 ppm or 400 ppm for 7 hr/da, 5 da/wk for 25 wk caused moderate to marked degeneration of the germinal elements of the testes but also severe general toxicity. In the female rat, intraperitoneal injections of 1.5 to 2 mL/kg arrests the estrous cycle and alters ovarian and uterine weights.

Schwetz et al. (126) exposed rats to vapor levels of 300 or 1000 ppm carbon tetrachloride for 7 hours per day on days 6 to 15 of gestation. Results indicated it was not highly embryotoxic but did cause some degree of retarded fetal development

such as delayed ossification of sternebrae. Considerable hepatotoxicity and weight reduction was observed in the dams at both concentrations.

Increased fetal mortality was observed in pregnant mice given single injections of 150 mg carbon tetrachloride during the last part of pregnancy. Cause of fetal death was suggested to be failure of peripheral circulation due to fetal liver damage. Circulatory disturbances and necrosis were found in the placentas, which probably also contributed to fetal death (618). Alumot et al. (3019) fumigated the feed of male and female rats with carbon tetrachloride to yield residue levels of 80 or 200 ppm, which had no adverse effects on the size, weight, or mortality of the offspring over the course of seven matings during two years. There was no effect on pregnancy rate, delivery rate, or litter size. At the highest dose, 200 ppm, the rats consumed 10-18 mg/kg/da. Several studies have shown that when pregnant rats are treated with carbon tetrachloride, biochemical and morphological changes occur in the liver of the offspring, but the damage is considerably less than that of the maternal liver (3053). Carbon tetrachloride crosses the human placenta, levels in cord blood being equal to or greater than levels in maternal blood (3186).

### 6.3.1.4 Other Toxicologic Effects

#### 6.3.1.4.1 Short-term Toxicity

*In animals, the primary* damage from carbon tetrachloride intoxication occurs in the liver (46). Single exposures to 50 ppm for 7 hours had no adverse effects upon male rats (682), but oral doses as low as 15.4 mg/kg bw altered liver biochemistry (683). High doses kill animals by CNS depression within hours. Smaller and subnarcotic doses produce death by liver damage after several days (25).

The maximum concentrations tolerated by rats for various times were as follows: 15 min - 12,000 ppm, 1.5 hr - 7300 ppm, 5 hr - 4600 ppm, 8 hr - 3000 ppm (682).

Oral $LD_{50}$ values of 2800 mg/kg and 12,800 mg/kg have been reported for the rat and mouse, respectively (47).

Absorption of carbon tetrachloride through the skin of monkeys was studied by McCollister et al. (684). They detected small amounts of carbon tetrachloride in the blood after skin exposure to vapor levels of 485 to 1150 ppm but concluded that absorption was insignificant by this route. The dermal $LD_{50}$ was 5070 mg/kg in rats (47).

#### 6.3.1.4.2 Chronic Toxicity

Adams et al. (682) conducted the most comprehensive long-term study of animal response to carbon tetrachloride. They studied rats, rabbits, guinea pigs and monkeys given repeated 7-hour exposures, 5 days per week. At a concentration of 400 ppm, rats and guinea pigs experienced severe intoxication. Less than half survived a 173-day exposure period. Animals demonstrated advanced liver and kidney changes

as early as 2 weeks into treatment. Histological examination revealed cirrhosis and fatty degeneration of the liver and slight degeneration of the tubular epithelium of the kidneys. At a level of 200 ppm, rats and guinea pigs still showed a high rate of mortality. The biochemical and histological effects were similar but less severe. At concentrations of 100 ppm, all species survived 146 to 163 exposures without evidence of adverse effects on growth, behavior or gross appearance but all showed histological changes. The changes in monkeys were equivocal at this treatment level. At a level of 50 ppm, rats, guinea pigs and rabbits tolerated up to 134 exposures in 187 days showing increased liver weight and moderate fatty degeneration. Monkeys tolerated 198 exposures without adverse effects. Exposure to 10 to 25 ppm resulted in a slight increase in liver weight and slight to moderate fatty degeneration in rats, guinea pigs and rabbits. At 5 ppm, rats and guinea pigs showed no effects.

### 6.3.2 Human and Epidemiologic Studies

#### 6.3.2.1 Short-term Toxicologic Effects

The toxic effects associated with carbon tetrachloride depend upon the exposure route. Although there are exceptions, nervous systems effects predominate after inhalation exposures whereas gastrointestinal and hepatorenal injuries are more prominent after ingestion. However, CNS depression and coma may occur without evidence of visceral injury, and renal failure may develop in cases without nervous system involvement (17).

With the exception of nausea and vomiting, symptoms develop more slowly after ingestion (24-36 hours) than after inhalation (few minutes). Primary toxic actions are central nervous system depression and cellular necrosis in the liver and/or kidneys. Death may be due to any of these lesions but occasional sudden deaths may be due to ventricular fibrillation and cardiac arrest (17).

Human studies indicate that single inhalation exposures to low concentrations result in variable symptoms. Acute toxicity is relatively low in contrast to that experienced with repeated exposures. Symptoms of mild exposure may be restricted to moderate eye irritation, moderate dizziness and headache which disappear upon cessation of the exposure (102).

Heimann and Ford (688) reported that 6 workers exposed to levels of 33-124 ppm became fatigued within a "few" hours. The sense of fatigue continued each workday, but disappeared on weekends. Five of the 6 men also complained of nausea and 4 experienced frequent vomiting.

Humans exhibit a wide variation in their sensitivity to ingested carbon tetrachloride. As little as 2 mL has been fatal, while doses of 4-12 ounces, although producing serious poisonings, have been survived. The probable oral human lethal dose ranges from 50 to 500 mg/kg (17).

Ruprah et al. (689) recently published a study of 19 victims of acute carbon tetrachloride poisoning. Fifteen of the poisonings occurred by ingestion of cleaning fluids which contained carbon tetrachloride. The amount ingested ranged from 4 to 400 mL. Nausea, vomiting and abdominal pain were common symptoms. All the victims except one who died after 15 days of hospitalization, recovered. The amount of carbon tetrachloride ingested in this case was unknown. Possible contributing factors in this victim's death may have been the additional presence of tetrachloroethylene in the blood (2.2 mg/L) and the 72-hour lag time before treatment was begun. At necropsy, there was evidence of massive hepatic and renal necrosis although the victim had shown signs of recovery for 4 days before her death. An additional 4 victims included in this study had inhalation exposures. Three became comatose after inhalation of fire-extinguisher fumes and one became drowsy after inhalation of cleaning fluid. All recovered within 2 to 4 days. Eight patients out of the total 19 victims showed signs of hepatic damage (increased serum enzyme levels) while 3 had hepatorenal failure and one, with a history of alcoholism, sustained massive hepatorenal damage.

There is a synergistic effect between excessive alcohol intake and exposure to carbon tetrachloride. Alcohol has been a contributing factor in many cases of human poisoning, especially in cases in which severe liver and kidney damage have occurred (46). Deng et al. (3167) reported three cases of acute hepatitis in three workers from a printing factory. These individuals had been passing tea-colored urine and were complaining of general malaise, nausea, vomiting, abdominal pain, diarrhea and dizziness for 2 days prior to seeking medical attention. All were mildly icteric and one exhibited acute renal failure and pulmonary edema. The adverse effects were attributed to inhalation exposure to carbon tetrachloride (300-500 ppm) and isopropyl alcohol (concentration not provided) while cleaning pump equipment.

Carbon tetrachloride vapor is slightly irritating to the eyes. Constriction of the visual field may be an early sign of poisoning. Four cases of definitely restricted visual field occurred in workmen for whom the estimated 8-hour TWA exposures ranged from 7 to 24 ppm with peaks of 22 to 232 ppm (687). From the available clinical evidence, carbon tetrachloride is strongly suspected of causing retrobulbar neuritis, optic neuritis and optic atrophy (19).

Direct application of carbon tetrachloride onto human skin causes a burning and stinging sensation within 5 minutes. The maximum pain is reached 6 minutes later and is associated with redness and wheal formation, followed by vesication (102). Stewart and Dodd (690) estimated that the amount of carbon tetrachloride absorbed during topical exposure of both hands for 30 minutes would be equivalent to a 10 ppm vapor exposure for 3 hours.

### 6.3.2.2 Chronic Toxicologic Effects

In cases of long-term chronic exposure to low concentrations, liver and kidney injuries predominate. The milder the exposure, the greater the tendency for the injury to be mainly in the liver (12). Dellian and Wittgens (686) reported that exposure to vapor levels ranging from 10-100 ppm, 3 days a week for 4 years resulted in nausea, stomach distress and enlargement and fatty degeneration of the liver. Even in the absence of symptoms, changes in the biochemical indicators of liver function are usually present (2). Kidney injury, if present, is characterized by lower back pain, painful and decreased urination, edema and weight gain (17).

Parkinsonism as a consequence of chronic carbon tetrachloride exposure has also been observed (692).

Three cases of liver cancer have been reported in humans several years after carbon tetrachloride poisoning. Simler et al. (693) reported that a fireman who was acutely intoxicated by carbon tetrachloride from a fire-extinguisher developed cirrhosis and "epithelioma" of the liver 4 years after exposure. Tracey and Sherlock (694) reported a hepatocellular carcinoma in a 59-year-old man, 7 years after acute poisoning with a cleaning compound containing carbon tetrachloride. A woman who developed nodular cirrhosis of the liver followed by liver cancer, died 3 years after being exposed to carbon tetrachloride for the first time. She had been exposed to vapors for 17 months (695).

### 6.3.3 Levels of Concern

Based on sufficient evidence to conclude that carbon tetrachloride is a carcinogen in experimental animals, the USEPA has specified an ambient water quality criterion for this compound of zero. In that attainment of a zero concentration level may be infeasible in some cases, the concentrations of carbon tetrachloride in water calculated to result in incremental lifetime cancer risks of 1E-05, 1E-06, and 1E-07 from ingestion of both water and contaminated aquatic organisms were estimated to be 4.0, 0.4 and 0.04 $\mu$g/L, respectively (355). Risk estimates are expressed as a probability of cancer after a lifetime consumption of two liters of water per day and consumption of 6.5 g per day of fish that have bioaccumulated the compound. Thus, a risk of 1E-05 implies that a lifetime daily consumption of two liters of drinking water and 6.5 g of fish at the criterion level of 4 $\mu$g/L carbon tetrachloride would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 people exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

For noncarcinogenic risks, the USEPA (383) has issued drinking water Health Advisories of 4 mg/L (1-day), 0.2 mg/L (10-day) and 0.07 mg/L (longer term) for children, and 0.3 mg/L (longer term) for adults. The WHO (666) recommends maximum levels of 3 $\mu$g/L carbon tetrachloride in drinking water.

OSHA (3539) currently permits exposure to 2 ppm (12.6 mg/m$^3$). The ACGIH (3005) has set a TWA of 5 ppm (30 mg/m$^3$).

### 6.3.4 Hazard Assessment

Carbon tetrachloride is an observed carcinogen in three animal species, indicating that it is probably carcinogenic to humans (25). Based on observed carcinogenicity in experimental animal systems, the USEPA (667) calculated an upper-limit incremental unit cancer risk of 0.13 (mg/kg/day)$^{-1}$ for oral exposures to carbon tetrachloride. IARC (25) has classified carbon tetrachloride in category 2B (sufficient evidence of animal carcinogenicity) in its weight-of-evidence ranking for potential carcinogens, and suggests that it appears reasonable to regard carbon tetrachloride as if it presented a carcinogenic risk to humans.

Carbon tetrachloride causes damage to the liver, kidneys and central nervous sytem in humans after high oral or inhalation exposures, and at low exposures may result in biochemical alterations, nausea and headaches.

There is little evidence for genotoxicity caused by carbon tetrachloride and further studies are required to clarify whether or not carbon tetrachloride is genotoxic. Teratogenic effects have not been noted but toxic effects have been demonstrated in mammalian fetuses.

## 6.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of carbon tetrachloride concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of carbon tetrachloride, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of carbon tetrachloride, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the carbon tetrachloride from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the carbon tetrachloride and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; carbon tetrachloride is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For

samples that contain high concentrations direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for carbon tetrachloride analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (<1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical carbon tetrachloride detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.12 μg/L (Method 601) | 1.2 μg/kg (Method 8010) |
| 2.8 μg/L (Method 624) | 5 μg/kg (Method 8240) |
| 10 μg/L (Method 1624) | |
| 5 μg/L (Method 8240) | |
| 1.2 μg/L (Method 8010) | |

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| COMMON SYNONYMS: | CAS REG.NO.: 75-00-3 NIOSH NO: KH7525000 FORMULA: $C_2H_5Cl$ | AIR W/V CONVERSION FACTOR at 25 °C (12) |
|---|---|---|
| Chloroethane<br>Ethyl chloride<br>Hydrochloric ether<br>Monochloroethane<br>Muriatic ether | STRUCTURE: $Cl-CH_2-CH_3$ | 2.64 mg/m³ ≈ 1 ppm;<br>0.379 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 64.52 |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as chloroethane with cyanides, amines, azo compounds, hydrazines, caustics, or nitrides, or with mercaptans or other organic sulfides, commonly evolve heat and toxic flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases, and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505, 3133). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Gas at room temperature; liquid when compressed at 20°C | (23,3615) |
| | • Color: Colorless | (23,3615) |
| | • Odor: Ether-like | (23,3615) |
| | • Odor Threshold: 4.200 ppm | (384) |
| | • Density: 0.9214 g/mL (at 0°C) | (59) |
| | • Freeze/Melt Point: -140.85°C | (23,3615) |
| | • Boiling Point: 12.50°C | (23,3615) |
| | • Flash Point: -50.00°C closed cup | (23,3615) |
| | • Flammable Limits: 3.60 to 15.40 % by volume | (51,60,504, 3615) |
| | • Autoignition Temp.: 519.0°C | (23,506,510) |
| | • Vapor Pressure: 1.00E+03 mm Hg (at 20°C) | (23) |
| | • Satd. Conc. in Air: 2.7600E+00 kg/m³ (at 20°C) | (3372 ) |
| | • Solubility in Water: 5.70E+03 mg/L (at 20°C) | (38,3446) |
| | • Viscosity: No data | |
| | • Surface Tension: No data | |
| | • Log (Octanol-Water Partition Coeff.): 1.43 | (29) |
| | • Soil Adsorp. Coeff.: 1.49E+01 | (33) |
| | • Henry's Law Const.: 1.10E-02 atm · m³/mol (at 20°C) | (74) |
| | • Bioconc. Factor: 1.30E+00 (estim) | (659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL-WATER SYSTEM | Chloroethane is expected to be highly mobile in soil/groundwater systems. Limited sorption onto soils, particularly soils of low organic content, is anticipated. Volatilization and migration with soil pore water are thought to be important transport processes. Degradation in natural soil/ground-water systems is not expected to be significant. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from soil-water systems is the migration of chloroethane in groundwater drinking water supplies. The high volatility of the chemical suggests that inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (54)<br>Chloroethane vapors are narcotic and produce depression of the central nervous system. Headache, dizziness, incoordination, stomach cramps and irritation of the respiratory tract have been noted. Deaths due to cardiac arrest have been recorded. Chloroethane is mildly irritating to the skin and eyes. In liquid form, frostbite can occur due to rapid evaporation.<br><br>Acute Toxicity Studies:<br><br>INHALATION:<br>$LC_{Lo}$ 10560 mg/m$^3$ · 45 min — Rat — (3504)<br>$LC_{50}$ 160 g/m$^3$ · 2 hr — Rat — (3504)<br>$LC_{Lo}$ 13000 ppm · 17 min — Human — (3152)<br><br>Long-Term Effects: Liver and kidney damage<br>Pregnancy/Neonate Data: no data<br>Genotoxicity Data: Negative data<br>Carcinogenicity Classification:<br>IARC - No data<br>NTP - Report scheduled for peer review<br>EPA - No data |

| | |
|---|---|
| HANDLING PRECAUTIONS (38) | Handle chemical only with adequate ventilation. • Vapor concentrations of 1000-10,000 ppm: any supplied air respirator or self-contained breathing apparatus. • 10,000-20,000 ppm: any supplied air respirator or self-contained breathing apparatus with a full facepiece. • Chemical goggles if there is probability of eye contact. • Impervious protective clothing and gloves should be used to prevent repeated or prolonged contact with liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 1000 ppm
- AFOSH PEL (8-hr TWA): 1000 ppm; STEL (15-min): 1250 ppm

Criteria
- NIOSH IDLH (30 min): 20,000 ppm
- ACGIH TLV® (8-hr TWA): 1000 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards
None established

EPA Health Advisories and Cancer Risk Levels
None established

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria
- Human Health (355)
  - No criterion established due to insufficient data.
- Aquatic Life (355)
  - No criterion established due to insufficient data.

REFERENCE DOSES:
No reference dose available.

## REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Chloroethane is listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), and steam electric power generating (3802). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

Chloroethane is listed on the first priority list of drinking water contaminants for which NPDWRs and MCLGs will be developed by January, 1991 (3781). EPA lists it as an unregulated contaminant requiring monitoring in all community water systems and non-transient non-community water systems (3771).

Resource Conservation and Recovery Act (RCRA)

Chloroethane is listed as a hazardous waste constituent (3783). Heavy ends from the fractionation column in ethyl chloride production contain chloroethane and are listed as specific sources of hazardous waste (3774, 3765). Chloroethane is subject to land disposal restrictions when its concentration as a hazardous constituent of certain wastewaters exceeds designated levels (3785). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). Chloroethane is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Toxic Substances Control Act (TSCA)

Effective April 13, 1989, manufacturers, processors, or importers of chloroethane must report production, usage, and exposure-related information to EPA (3797). They, as well as others who possess health and safety studies on chloroethane, must submit them to EPA (3789).

Comprehensive Environmental Response Compensation and Liability Act CERCLA)
Chloroethane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 45.4 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing chloroethane, but these depend upon the concentrations of the chemical in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of chloroethane must report annually, to EPA and state officials, their releases of this chemical to the environment (3787).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to chloroethane in any 8-hour work-shift of a 40 hour work-week shall not exceed an 8-hour time-weighted average (TWA) of 1000 ppm (3539).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated chloroethane as a hazardous material with a reportable quantity of 45.4 kg, subject to requirements for packaging, labeling and transportation (3180).

- State Programs

  ALL STATES
  All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. Other states have promulgated additional or more stringent criteria.

CONNECTICUT
Connecticut has a quantification limit of 2.0 μg/L for drinking water (3137).

DISTRICT OF COLUMBIA
The District of Columbia has a human health criterion of 1.0 μg/L for surface waters (3828).

KANSAS
Kansas has an action level of 37 μg/L for ground-water (3213).

NEW YORK
New York has set a maximum contaminant level of 5 μg/L for drinking water (3501).

OKLAHOMA
Oklahoma has a water quality criterion of 2.1 μg/L for groundwater (3534).

SOUTH DAKOTA
South Dakota requires that chloroethane be nondetectable, by designated test methods, in ground-water (3671).

Proposed Regulations

Federal Programs
NONE
No proposed regulations are pending.

- State Programs

MOST STATES
Most states are in the process of revising their water programs and proposing changes to their regulations which will follow EPA's changes when they become final. Contact with state officers is advised. Changes are projected for 1989-90 (3683).

NONE
No proposed regulations are pending.

EEC Directives

Directive on Ground-Water (538)
Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in the shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Chloroethane is classified as a flammable substance and is subject to packaging and labeling regulations.

EEC Directives--Proposed Resolution
Resolution on the Revised List of Second-Category Pollutants (545)
Chloroethane is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 7.1 MAJOR USES

Chloroethane is used as a chemical intermediate in the manufacture of tetraethyl lead, ethyl cellulose plastics, alkyl catalysts, dyes and pharmaceuticals, and as a solvent, aerosol propellant, local anesthetic, and refrigerant (3113, 3311, 3615). Production of tetraethyl lead has declined considerably in recent years due to the phaseout of leaded gasoline (3311).

## 7.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 7.2.1 Transport in Soil/Ground-water Systems

#### 7.2.1.1 Overview

Chloroethane may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). Transport pathways of low soil concentrations can be generally assessed by estimating equilibrium partitioning, as shown in Table 7-1.

These calculations estimate the partitioning of chloroethane among soil particles, soil water and soil air. The chloroethane associated with the air and water phases of the soil is expected to exhibit higher mobility than that adsorbed on soil.

Estimates for the unsaturated topsoil model indicate that a significant amount (approximately 19%) of the chloroethane is expected to be present in the soil-water phase and thus, be available to migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. A major portion of the chloroethane (26%) was estimated to partition to the gaseous phase of the soil; diffusion through the soil-air pores up to the ground surface, and subsequent removal by evaporation, may be a significant loss pathway.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), most of the chloroethane (approximately 94%) is estimated to be present in the soil-water phase (Table 7-1) and easily transported with flowing ground water. Ground water underlying chloroethane-contaminated soils with low organic content may be highly vulnerable to contamination.

TABLE 7-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR CHLOROETHANE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil at 20°C[b,c] | 54.6 | 19.1 | 26.3 |
| Saturated deep soil[d] | 5.9 | 94.1 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Use estimated soil sorption coefficient: $K_{oc}$ = 14.9 (33).
c) Henry's law constant taken as 1.1E-02 atm•m$^3$/mol at 20°C (74).
d) Used sorption coefficient $K_p$ calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

7.2.1.2 Sorption on Soils

The mobility of chloroethane in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Very little information specific to the sorption of chloroethane on soil particles was available. Soil adsorption coefficients ($K_{oc}$) of 14.9, 33, and 143 have been estimated for chloroethane (33, 3679, 31). These $K_{oc}$ values indicate high mobility in soils (3693). Retardation rates, which represent the interstitial water velocity/pollutant velocity in the soil, were reported by Wilson et al. (82) to be a function of $K_{oc}$, the ratio of soil density (a) to soil water content (b), and the organic content(oc) of the soil according to the following equation:

$$R_f = 1 + (a/b)K_{oc}(oc)$$

Schwarzenbach et al. (77) report retardation factors for some chlorinated organic compounds that have $K_{oc}$ values higher than that reported for chloroethane. The data indicate some retardation (i.e., adsorption) in soils having 1-2% organic carbon and little or no retardation in deep soils having less than 0.1% organic carbon. Wilson et al. (82) report that 1,2-dichloroethane moved rapidly through a sandy soil. Assuming analogous soil conditions, adsorption of chloroethane, particularly to deep soils, is not expected to be significant.

### 7.2.1.3 Volatilization from Soils

Transport of chloroethane vapors through the air-filled pores of unsaturated soils is expected to be an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H were also observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of chloroethane from surface soils.

No information was available for the two other physicochemical properties influencing chloroethane volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

The volatilization half-life for chloroethane in a stirred aqueous solution was reported to be 21-79 min (10, 3174, 3175). Based on a Henry's Law Constant of 0.00848 atm · $m^3$/mole at 25°C, the volatilization half-life from a model river was estimated to be about 2.5 hr. (31, 3679). Based on an estimated oxygen reaeration rate ratio of 0.645, the volatilization half-lives from an environmental pond, river, and lake were estimated to be 5.6 days, 1.1 days, and 4.5 days (3679, 3414).

Compared to volatilization from well-stirred aqueous solutions, volatilization from soil was shown to be slower by approximately one order of magnitude for other chlorinated aliphatics (82).

### 7.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of chloroethane in soil/ground-water systems is not well documented. In most cases, it should be assumed that chloroethane will persist for months to years (or more). Chloroethane that has volatilized will undergo photochemical oxidation. Because the compound does not absorb uv light above

290 nm, direct photolysis will not occur (3414, 3310). The half-life for its reaction with hydroxyl radicals in the atmosphere was reported to be 40 days (3039).

In aqueous environments, oxidation of chloroethane by singlet oxygen or peroxy radicals is not significant (3414). A maximum hydrolytic half-life for chloroethane was reported to be 40 days (10), implying that hydrolysis may be an important fate process in aqueous systems. However, the lower temperature and oxygen content in soils and ground water compared to surface waters suggest that hydrolysis in moist soils would be slower. Data reported for other chlorinated ethanes (10) indicate that hydrolysis would not be competitive with volatilization.

Literature references to microbial degradation of compounds such as chloroethane are very few. Most references indicate that low molecular weight chloroaliphatics are not rapidly metabolized in the environment (10). However, Tabak et al. (79, 3697) reported some degradation of chlorinated aliphatics with acclimated activated microbial populations (e.g., 53-91% degradation of dichloroethanes in 28 days). Haider (3259) reported that 50-70% of the organically bound chlorine was released from chlorinated ethanes and methanes when incubated under anerobic laboratory conditions; and Thom and Agg (80) included some chlorinated ethanes among those chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as chloroethane is very low and drops off sharply with increasing depth. Thus, biodegradation should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

Based on a log $K_{ow}$ of 1.43 and a water solubility of 5710 ppm at 20°C, the log bioconcentration factor (BCF) was estimated to be 0.86 and 0.67 (3679, 31). The potential for food chain bioaccumulation is considered to be zero (3259).

### 7.2.3 Primary Routes of Exposure From Soil/Ground-water Systems

The above discussion of fate pathways suggests that chloroethane is highly volatile from aqueous solutions, is weakly adsorbed, and has no significant potential for bioaccumulation. Chloroethane on soil surfaces is likely to volatilize, but that portion not subject to volatilization is likely to be mobile in ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of chloroethane from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. In addition, the potential for ground water contamination is high, particularly in sandy soils. Mitre (83) reported that chloroethane has been found in two of the 546 National Priority List (NPL) hazardous waste sites in ground water.

The properties of chloroethane suggest that it has the potential for movement in soil/ground-water systems. This compound may eventually reach surface waters by this mechanism, suggesting several other exposure pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion exposure.
- Aquatic organisms residing in these waters and bioaccumulating this chemical may be consumed, also resulting in ingestion exposures.
- Recreational use of these waters may result in dermal exposures.
- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower then exposure from drinking contaminated ground water for two reasons. First, the Henry's law constant for chloroethane suggests that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this compound is very low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 7.2.4 Other Sources of Exposure

The volatility of this compound suggests that it may be found in air. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For chloroethane, they had data for 348 locations. In urban and suburban areas, the median concentration was 0.16 $\mu g/m^3$. In source-dominated areas, the median concentration was 0.12 $\mu g/m^3$. These results suggest inhalation exposures to persons in these areas.

## 7.3 HUMAN HEALTH CONSIDERATIONS

### 7.3.1 Animal Studies

#### 7.3.1.1 Carcinogenicity

There are no published data available concerning the carcinogenicity of chloroethane. However, it is being tested in both rats and mice by inhalation (15,000 ppm) by the National Toxicology Program (594); chronic histopathology is now in progress.

### 7.3.1.2 Genotoxicity

Because of its extremely low boiling point, this compound is not suitable for testing in many genetic assays. Chloroethane was found to be positive, with or without metabolic activation, in at least one strain of Salmonella typhimurium when the bacteria were exposed for 8 hrs in a 9L desiccator (3591).

No activity was recorded for chloroethane in the BALB/c-3T3 cell transformation assay; tests were conducted in the absence of an exogenous metabolic activation system (3732).

### 7.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No data are available.

### 7.3.1.4 Other Toxicologic Effects

#### 7.3.1.4.1 Short-term Toxicity

Guinea pigs died from a 9-hour exposure to 40,000 ppm but survived a 4.5 hour exposure to this concentration (134). Pathological changes were observed in the lungs, liver and kidneys of the survivors (134). A concentration of 10,000 ppm for 810 min caused no pathological changes. There was a slight increase in the liver weight of male rats exposed to vapor concentrations of 4000 and 10,000 ppm six hours per day, 5 days per week for 2 weeks. Similarly exposed female rats and male dogs experienced no pathological or neurological effects at concentrations up to 10,000 ppm (132).

Liquid chloroethane has caused epithelial damage to rabbit corneas. The effect has been attributed to solvent action and not temperature-lowering (19).

#### 7.3.1.4.2 Chronic Toxicity

No chronic toxicity data are available; however, the National Toxicology Program study will provide chronic toxic effects data as part of its carcinogenesis bioassay. In a preliminary subchronic study, mice and rats exposed to a maximum chloroethane concentration of 19,000 ppm for 90 days exhibited no histopathological effects (3486).

### 7.3.2 Human and Epidemiologic Studies

#### 7.3.2.1 Short-term Toxicologic Effects

Chloroethane is considered one of the least toxic of the chlorinated ethanes (51). However, it is potentially damaging to the liver and is known to disturb cardiac rhythm (123). The most serious concern from acute high level exposures, other than

an anesthetic effect, is the possibility of potentiation of adrenalin and resultant cardiac arrhythmias (12).

Inhalation of 40,000 ppm resulted in dizziness, eye irritation and abdominal cramps after 2 inhalations. Weak analgesia was seen after 12 minutes at 19,000 ppm and at 13,000 ppm, only slight symptoms of inebriation were experienced (3152). Sudden fatalities from chloroethane anesthesia have been reported, probably due to cardiac or respiratory arrest (38).

Allergic eczematous eruption was reported in two subjects after chloroethane was sprayed on the skin in an allergy testing procedure. However, this type of reaction is believed to be rare. Also, if large amounts of liquid are spilled on the skin, the evaporation may cause rapid cooling and possibly frostbite (12).

#### 7.3.2.2 Chronic Toxicologic Effects

There is very little information on long-term human exposure to chloroethane. Reversible cerebellar dysfunction occurred in one individual who had used chloroethane as a narcotic for several months (3289). The neurological signs included ataxia, nystagmus and scanning dysarthria, dysdiadochokinesis of each arm and sluggish lower limb reflexes. A slight disturbance in liver function and hepatomegaly were also noted.

Troshina (3276) reported that some workers occupationally exposed to chloroethane exhibited some pathological changes in the sympathetic nervous system and decreased phagocytic activity of leukocytes.

### 7.3.3 Levels of Concern

In view of the paucity of data available on the adverse health effects and effect levels associated with exposure to chloroethane, estimates of exposure levels of concern cannot be made with any confidence. Due to lack of data, the USEPA has not set a water quality criterion for human health (355).

Both OSHA (3539) and the ACGIH (3005) have set an occupational exposure limit of 1000 ppm (2600 $mg/m^3$) for chloroethane, based on preventing narcosis.

### 7.3.5 Hazard Assessment

Although data are sparse, chloroethane is considered one of the least toxic of the chlorinated ethanes (51). No reports of human ingestion were located. Inhalation exposure to high concentrations of chloroethane produce depression of the central nervous system. Dizziness and abdominal cramps were seen in humans after four inhalations of 20,000 ppm; signs of inebriation were evident after 12 minutes exposure to 13,000 ppm (133). Cardiac arrhythmias and cardiac arrest have been linked to chloroethane exposure but very high levels are required (38). High level

exposures may also be damaging to the liver (123). Skin and eye irritation occur at lower levels and skin contact with the liquid may cause frostbite (12).

A negative response was recorded in a mammalian cell transformation assay (3732). However, the notable lack of data available concerning the carcinogenic and teratogenic potential of chloroethane as well as the absence of quantitative data on acute and long-term toxicity associated with chloroethane for either humans or laboratory animals provide a low degree of confidence in any assessment of hazard for chloroethane exposure, particularly with regard to long-term, low-level human exposures via drinking water.

## 7.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of chloroethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of chloroethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of chloroethane, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the chloroethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the chloroethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; chloroethane is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

In addition to all these methods, a rapid procedure based on GC with flame ionization detection has been reported (3131). In this method the sample is purged

with helium and the volatiles are trapped directly on the analytical column which is cryogenically cooled.

The EPA procedures recommended for chloroethane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical chloroethane detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.52 μg/L (Method 601) | 5.2 μg/kg (Method 8010) |
| 50 μg/L (Method 1624) | 10 μg/kg (Method 8240) |
| 10 μg/L (Method 8240) | |
| 5.2 μg/L (Method 8010) | |

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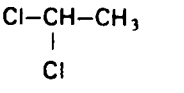
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3789. U.S. Environmental Protection Agency 1988. 40 CFR716. Health and safety data reporting. Fed. Regist. 53:38642.

3797. U.S. Environmental Protection Agency 1989. Addition of chemicals to information rules. Fed. Regist. 54:8484. 40 CFR712 and 716.

3802. U.S. Environmental Protection Agency 1982. Steam and electric power generating point source category: Pretreatment standards for new sources (PSNS), Table - 126 Priority Pollutants. 40 CFR423.17 Appendix A.

3828. District of Columbia Water Quality Standards 1985. Water Quality Standards of the District of Columbia, Final and Effective 12/27/85.

| COMMON SYNONYMS: | CAS REG.NO.: 75-34-3 FORMULA: $C_2H_4Cl_2$<br>NIOSH NO: KI0175000<br>STRUCTURE: | AIR W/V CONVERSION FACTOR at 25°C (12)<br>4.05 mg/m³ ≈ 1 ppm;<br>0.247 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 98.97 |
|---|---|---|
| 1,1-Dichloroethane<br>Chlorinated hydrochloric ether<br>Dichloromethyl-methane<br>Ethylidene chloride<br>Ethylidene dichloride |  | |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as 1,1-dichloroethane with cyanides, amines, azo compounds, hydrazines, caustics, or nitrides, or with mercaptans or other organic sulfides, commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases, and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Chloroform-like | (12) |
| | • Odor Threshold: 200.000 ppm | (38) |
| | • Density: 1.1750 g/mL (at 20°C) | (21) |
| | • Freeze/Melt Point: -96.70°C | (21) |
| | • Boiling Point: 57.30°C | (21) |
| | • Flash Point: -12.00°C closed cup | (21) |
| | • Flammable Limits: 5.60 to 11.40 % by volume | (12,60,506) |
| | • Autoignition Temp.: 458.0 to 493.0°C | (12,60) |
| | • Vapor Pressure: 1.82E+02 mm Hg (at 20°C) | (21) |
| | • Satd. Conc. in Air: 9.8750E+05 mg/m³ (at 20°C) | (1219) |
| | • Solubility in Water: 5.50E+03 mg/L (at 20°C) | (21) |
| | • Viscosity: 0.377 cp (at 20°C) | (21) |
| | • Surface Tension: No data | |
| | • Log (Octanol-Water Partition Coeff.): 1.79 | (29) |
| | • Soil Adsorp. Coeff.: 3.00E+01 | (33) |
| | • Henry's Law Const.: 5.7E-03 atm · m³/mol (at 25°C) | (33) |
| | • Bioconc. Factor: 2.90E+00 (estim) | (659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL-WATER SYSTEM | 1,1-Dichloroethane is expected to be highly mobile in the soil/ground-water system. Limited sorption on soils, particularily soils of low organic content is expected. Volatilization and migration with soil pore water are thought to be significant transport pathways. Degradation in natural soil/ground-water systems is not expected to be significant. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of 1,1-dichloroethane to groundwater drinking water supplies. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (54, 76)<br>Capable of causing anesthesia at high concentrations.<br><br>Acute Toxicity Studies:<br><br>INHALATION:<br>$TC_{Lo}$ 6000 ppm Rat (3504)<br><br>ORAL:<br>$LD_{50}$ 725 mg/kg Rat (47)<br><br>Long-Term Effects: Kidney toxicity in animals<br>Pregnancy/Neonate Data: Not embryotoxic at maternally toxic levels.<br>Genotoxicity Data: Conflicting results<br>Carcinogenicity Classification:<br>IARC - None assigned<br>NTP - None assigned<br>EPA - No data |

| | |
|---|---|
| HANDLING PRECAUTIONS (38) | Handle chemical only with adequate ventilation. • Vapor concentrations of 100-1000 ppm: any supplied-air respirator, self-contained breathing apparatus or chemical-cartridge respirator with an organic vapor cartridge. • 1000-4000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece; gas mask with organic vapor canister. • Chemical goggles if there is probability of eye contact. • Impervious clothing and gloves should be used to prevent repeated or prolonged skin contact with liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 100 ppm
- AFOSH PEL (8-hr TWA): 100 ppm; STEL (15-min): 150 ppm

Criteria
- ACGIH TLV® (8-hr TWA): 200 ppm
- ACGIH STEL (15 min): 250 ppm
- NIOSH IDLH (30 min): 4000 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards
None established

EPA Health Advisories and Cancer Risk Levels
None established

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria
- Human Health (355)
  - No criterion established due to insufficient data.

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion established due to insufficient data.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity:
    no criterion established due to insufficient data.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:

No reference dose available.

## REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

  Clean Water Act (CWA)
  1,1-Dichloroethane is listed as a toxic pollutant, subject to general pretreatment standards for new and existing sources, and to effluent standards and guidelines (351, 3763). Effluent limitations for 1,1-dichloroethane have been set in the following point source categories: electroplating (3767), organic chemicals, plastics and synthetic fibers (3777), steam electric power generating (3802), and metal finishing (3768). Limitations vary depending on the type of plant and industry.

  Safe Drinking Water Act (SDWA)
  1,1-Dichloroethane is listed as an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3771). It is also listed on the first priority list of drinking water contaminants for which NPDWRs will be developed (3781). In states with an approved Underground Injection Control program, a permit is required for the injection of 1,1-dichloroethane-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)
1,1-Dichloroethane is identified as a toxic hazardous waste (U076) and listed as a hazardous waste constituent (3783, 3784). A non-specific source of 1,1-dichloroethane- containing waste is the production of chlorinated aliphatic hydrocarbons (3765). 1,1-Dichloroethane is subject to land disposal restrictions when its concentration as a hazardous constituent of certain wastewaters exceeds designated levels (3785). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). 1,1-Dichloroethane is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Toxic Substances Control Act (TSCA)
Manufacturers, processors, or importers who possess health and safety studies on 1,1-dichloroethane must submit them to EPA (3789). Manufacturers and processors of 1,1-dichloroethane are also required to perform human health effects studies and chemical fate testing in support of the RCRA program (3792).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
1,1-Dichloroethane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing 1,1-dichloroethane but these depend upon the concentration of the chemicals in the waste stream (3766).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to 1,1-dichloroethane in any 8-hour work-shift of a 40-hour work-week shall not exceed an 8-hour time-weighted average (TWA) of 100 ppm (3539).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated 1,1-dichloroethane as a hazardous material with a reportable quantity of 454 kg, subject to requirements for packaging, labeling and transportation (3180).

- State Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. Other states have promulgated additional or more stringent criteria.

CALIFORNIA
California has an action level of 20 μg/L for 1,1-dichloroethane in drinking water (3098).

CONNECTICUT
Connecticut has a quantification limit of 2 μg/L for 1,1-dichloroethane in community drinking water systems (3137).

KANSAS
Kansas has an action level of 5 μg/L for 1,1-dichloroethane in groundwater (3213).

NEW MEXICO
New Mexico has a human health criterion of 0.025 mg/L for 1,1-dichloroethane in groundwater (3499).

NEW YORK
New York has an MCL of 5 μg/L for drinking water, and a nonenforceable water quality guideline of 50 μg/L for surface and groundwaters (3501).

OKLAHOMA
Oklahoma has a water quality criterion of 0.3 μg/L for groundwater (3534).

SOUTH DAKOTA

South Dakota requires that 1,1-dichloroethane be nondetectable, using designated test methods, in groundwater (3671).

WISCONSIN

Wisconsin has a preventive action limit of 85 μg/L and an enforcement standard of 850 μg/L for groundwater (3840).

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA) EPA has proposed listing wastestreams from the following industries as specific sources of 1,1-dichloroethane-containing wastes: organical chemicals (acetaldehyde production from ethylene, 1,1,1-trichloroethane production) (3795, 3774).

- State Water Programs

  MOST STATES

  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA

  Minnesota has proposed a Recommended Allowable Limit (RAL) of 810 μg/L for 1,1-dichloroethane in drinking water (3451).

EEC Directives

Directive on Ground-Water(538)
Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phytopharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
1,1-Dichloroethane is listed as a Class II/b harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,1-Dichloroethane is classified as a flammable, harmful substance and is subject to packaging and labeling regulations.

EEC Directives--Proposed Resolution
Resolution on the Revised List of Second-Category Pollutants (545)
1,1-Dichloroethane is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 8.1 MAJOR USES

The compound 1,1-dichloroethane is currently used commercially as an extractant for heat-sensitive substances, as a cleaning solvent and degreaser, and also as a fumigant. Its largest industrial use is as an intermediate in the manufacture of 1,1,1-trichloroethane. It was previously used as an anesthetic, but it is no longer used for this purpose (38, 21, 9).

## 8.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 8.2.1 Transport in Soil/Ground-water Systems

#### 8.2.1.1 Overview

The 1,1-isomer of dichloroethane may move through the soil/ ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). Transport pathways for low soil concentrations can be assessed by estimating equilibrium partitioning as shown in Table 8-1.

These calculations predict the distribution of 1,1-dichloroethane among soil particles, soil water, and soil air. The 1,1-dichloroethane associated with the air and water phases of the soil is more mobile than that adsorbed on soils.

The model for unsaturated topsoil indicates that approximately 12% of the 1,1-dichloroethane is expected to be present in the soil-water phase and can thus migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion, and diffusion. For the portion of 1,1-dichloroethane in the gaseous phase of the soil (approximately 8%), diffusion through the soil-air pores up to the ground surface, and subsequent removal by evaporation, may be a significant loss pathway.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the 1,1-dichloroethane (approximately 90%) is expected to be present in the soil-water phase (Table 8-1) and transported with flowing ground-water. Ground-water underlying 1,1-dichloroethane-contaminated soils with low organic content may therefore be highly vulnerable to contamination.

TABLE 8-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR
1,1-DICHLOROETHANE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil at 25°C[b,c] | 79.8 | 11.9 | 8.3 |
| Saturated deep soil[d] | 11.2 | 88.8 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Used estimated soil sorption coefficient: $K_{oc}$ = 30 (33).
c) Henry's law constant = 5.7E-03 atm · $m^3$/mol at 25°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

### 8.2.1.2 Sorption on Soils

The mobility of 1,1-dichloroethane in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. Soil sorption coefficients ($K_{oc}$) of 30, 43, and 58 have been estimated for 1,1-dichloroethane (33, 3679, 3740) indicating that the compound will not be strongly bound to soils (3740). In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Soil retardation rates, which represent the interstitial water velocity/ pollutant velocity in the soil, are a function of the soil adsorption coefficient $K_{oc}$, the ratio of soil

density (a) to soil water content (b), and the organic content (oc) of the soil according to the following equation (82):

$$R_f = 1 + (a/b)K_{oc}(oc)$$

Schwarzenbach et al. (77) reported retardation factors for some chlorinated organic compounds with $K_{oc}$ values higher than that reported for 1,1-dichloroethane. The data indicated some retardation (i.e., adsorption) in soils having 1-2% organic carbon and little or no retardation in deep soils having less than 0.1% organic carbon. Wilson et al. (82) reported that 1,2-dichloroethane moved rapidly through a sandy soil. Assuming analogous soil conditions, adsorption of 1,1-dichloroethane, particularly to deep soils, is not expected to be significant.

A soil retardation factor of 1.2 was calculated for 1,1-dichloroethane by Schwarzenbach and Westall (3629) using the soil adsorption coefficient ($K_{oc}$) and the octanol/water partition coefficient ($K_{ow}$).

### 8.2.1.3 Volatilization from Soils

Transport of 1,1-dichloroethane vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H were also observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of 1,1-dichloroethane from surface soils.

No information was available for the two other physicochemical properties influencing 1,1-dichloroethane volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

Smith et al. (78) report that the ratio of the 1,1-dichloroethane volatilization rate to the oxygen reaeration rate is a constant (0.71 ± 0.11). Using the reaeration rates reported for a river, lake and pond to determine 1,1-dichloroethane volatilization rates, the corresponding half-lives of 1.0 day in a river, 4.1 days in a lake and 5.1 days in a pond were calculated. Half-lives for the volatilization of 1,1-dichloroethane from stirred aqueous solutions in the laboratory were reported to be in the range of 20-90 minutes, depending on the degree of agitation (10). For other chlorinated ethanes,

volatilization from soil was about one order of magnitude slower than volatilization from well-stirred aqueous solution (82). A similar relationship would be expected for 1,1-dichloroethane.

### 8.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of 1,1-dichloroethane in soil/ground-water systems is not well documented. In most cases, it should be assumed that 1,1-dichloroethane will persist for months to years (or more). The 1,1-dichloroethane that has volatilized will eventually undergo photochemical oxidation. A tropospheric lifetime of 1.5 months has been estimated for 1,1-dichloroethane (10, 3300).

Under normal environmental conditions, 1,1-dichloroethane is not expected to undergo rapid hydrolysis in the soil/ground-water system. Data reported for other chlorinated ethanes (10) suggest that hydrolysis would not be competitive with volatilization.

Little specific information is available on the potential for microbial degradation of 1,1-dichloroethane. Generally, low molecular weight chloroaliphatics are not rapidly metabolized in the environment (10), although they can be degraded by acclimated microbial populations. Tabak et al. (79, 3697) reported some degradation of 1,1- dichloroethane by an acclimated activated sludge population; and Thom and Agg (80) included some chlorinated ethanes among those chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In-situ biodegradation by blended specialized bacterial seed cultures has been suggested as a decontamination procedure for soils contaminated with 1,2-dichloroethane (650). In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as 1,1-dichloroethane is very low and drops off sharply with increasing depth. Thus, biodegradation should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

In higher organisms, 1,1-dichlorethane is expected follow a metabolic pathway similar to that for 1,1,1-trichloroethane. As determined by whole body levels in bluegills, elimination half-lives of less than 2 days have been reported for choroethanes (3734). Bioconcentration factors of 1.3, 2.9, and 6.6 have been estimated for 1,1-dichloroethane (3679, 659, 3735). The concentration of 1,1-dichloroethane in oyster tissue was reported as 33 ppt (3212).

### 8.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The physicochemical properties of 1,1-dichloroethane and the above discussion of fate pathways suggest that 1,1-dichloroethane is highly volatile from aqueous solutions, weakly adsorbed by soil, and has a low potential for bioaccumulation. This compound

may volatilize from soil surfaces, but that portion not subject to volatilization is likely to be mobile in ground-water. These fate characteristics suggest several potential exposure pathways.

Volatilization of 1,1-dichloroethane from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. In addition, the potential for ground-water contamination is high, particularly in sandy soils. This compound is one of the most ubiquitous pollutants found in landfill leachates (3740). As evidence of its potential for migration, Mitre (83) reported that it has been found at 26 of the 546 National Priority List (NPL) sites. It was detected at 19 sites in ground-water and at 8 sites in surface water.

This compound was reported with a similar degree of frequency in the Ground-water Supply Survey (GWSS) conducted by USEPA (531). This Survey examined 945 finished water supplies that use ground-water sources. The results for 1,1-dichloroethane are summarized below.

| Sample Type | Occurrences* No. | % | Median of Positive (μg/L) | Maximum (μg/L) |
|---|---|---|---|---|
| Random Supplies: | | | | |
| serving <10,000 people (280 samples) | 10 | 3.6 | 0.51 | 3.2 |
| serving >10,000 people (186 samples) | 8 | 4.3 | 0.54 | 1.2 |
| Non-Random supplies: | | | | |
| serving <10,000 people (321 samples) | 6 | 1.9 | 0.62 | 1.2 |
| serving >10,000 people (158 samples) | 17 | 10.8 | 0.87 | 4.2 |

*Samples having levels over quantitation limit of 0.2 μg/L.

The random samples taken as part of the GWSS are intended to statistically represent the U.S. ground-water drinking water supplies. The non-random supplies were chosen by the states as being potentially contaminated.

These results indicate that this compound has the potential for movement in soil/ground-water systems and subsequent contamination of ground-water drinking water supplies. The movements of 1,1-dichloroethane in ground-water may eventually contaminate surface waters, suggesting several other exposure pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion exposure.
- Aquatic organisms residing in these waters and accumulating this chemical may be consumed, also resulting in ingestion exposures.
- Recreational use of these waters may result in dermal exposures.

- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

Exposures to 1,1-dichloroethane associated with surface water contamination can be expected generally to be lower than exposure from drinking contaminated ground-water for two reasons. First, the Henry's law constant for 1,1-dichloroethane suggests that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this compound is low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

Based on the maximum concentration of 1,1-dichloroethane reported for any U.S. water supply (4.2 μg/L), EPA estimated that the maximum intake of the compound from drinking water would be 8 μg/day (3740).

### 8.2.4 Other Sources of Exposure

The data presented above on the Ground-water Supply Survey (531) suggest that 1,1-dichloroethane is found in a limited number of ground-water supplies used as drinking water. Coniglio et al. (223) reported that 1,1-dichloroethane has occasionally been found in surface water supplies. In a summary of data available as of 1980, these authors reported that 2.9% of 103 finished surface water samples were contaminated, with a mean concentration of 0.2 μg/L.

The volatility of this compound suggests inhalation as an exposure route. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For 1,1-dichloroethane, they had data for 569 locations. In urban and suburban locations, the median concentration was 0.25 $\mu g/m^3$. In source-dominated areas, the median concentration was 0.045 $\mu g/m^3$. No explanation was given for the lower concentration in source-dominated areas but it is probably an artifact of the method used to categorize locations. In any case, the results suggest inhalation exposure at low levels to persons in urban and suburban as well as source-related areas.

## 8.3 HUMAN HEALTH CONSIDERATIONS

### 8.3.1 Animal Studies

#### 8.3.1.1 Carcinogenicity

A study investigating the carcinogenicity of 1,1-dichloroethane was conducted by the National Cancer Institute (125). Technical-grade 1,1-dichloroethane in corn oil was administered by gavage 5 days a week for 78 weeks to male and female Osborne-Mendel rats and $B6C3F_1$ mice. The high and low time-weighted average

dosages were 764 and 382 mg/kg/ day for male rats; 950 and 475 mg/kg/day for female rats; 2885 and 1442 mg/kg/day for male mice and 3331 and 1665 mg/kg/day for female mice. High early mortality occurred in both treated and control animals. Marginal dose-related increases in mammary adenocarcinomas and hemangiosarcomas among female rats as well as an increase in the incidence of endometrial stromal polyps (benign tumors) in female mice were considered to be indicative of a possible carcinogenic potential, but the evidence was not conclusive. The scientific review panel for the NCI study suggested that the compound would have to be retested before a determination could be made concerning its carcinogenicity.

The cancer initiating and promoting potential of 1,1-dichloroethane was evaluated by Story et al. (3688) using the rat liver foci assay. Negative results were obtained in the initiation tests in which phenobarbital was used as the promoting agent, and positive results were seen in the promotion tests in which diethylnitrosamine was used as the initiating agent. In an in vitro cell transformation assay using BALB/c-3T3 mouse cells and no exogenous metabolic activation system, 1,1-dichlorethane gave negative results (3732).

#### 8.3.1.2 Genotoxicity

The 1,1-isomer of dichloroethane was not mutagenic in the Ames Salmonella microsome assay (123, 3653, 3508), but it was reproducibly genotoxic when two strains were treated for 8 hrs in a desiccator (3450). Conflicting results were also found for cell transformation assays: 1,1-Dichloroethane was not positive in the BALB/ c-3T3 cell transformation assay (3732), but it did significantly enhance viral transformation of Syrian hamster embryo cells (3274).

#### 8.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No teratogenic effects were observed in rats exposed to vapor concentrations of 15,390 or 24,300 mg/m$^3$ (3800 or 6000 ppm) 1,1-dichloroethane for 7 hr/day on days 6 through 15 of gestation (126). Delayed ossification of the ribs was observed in the fetuses in the high-dose group but not in rats similarly exposed to 15,390 mg/m$^3$. 1,1-dichloroethane. Maternal food consumption and body weight were significantly reduced in both groups, but there were no effects on implants/dam, live fetuses/dam, resorptions/dam or fetal weight.

#### 8.3.1.4 Other Toxicologic Effects

##### 8.3.1.4.1 Short-term Toxicity

The limited data available indicate that 1,1-dichloroethane is low in acute toxicity, but is capable of causing narcosis at high concentrations. Rats survived an 8-hour vapor exposure to 4000 ppm dichlorethane but not 16,000 ppm (12). Those exposed to 32,000 ppm survived 30 minutes of exposure, but died after 2.5 hours (46). An acute oral $LD_{50}$ value of 725 mg/kg for rats has been reported (3504); however, in a

preliminary NCI study, a daily oral dose of 5,620 mg/kg given to rats 5 days/week for 6 weeks did not result in any lethal effects, and similar results were seen in mice dosed with 10,000 mg/kg/day (3735). Animal studies suggest that 1,1-dichloroethane has a relatively low potential for causing liver or kidney injury even on repeated exposure (12). In tests on mice, intraperitoneal doses of 1000 mg/kg resulted in some swelling of the renal tubules, but no necrosis (12).

Liquid 1,1-dichloroethane can be absorbed through skin, but not in amounts sufficient to produce systemic injury. In tests on rabbits, repeated skin application resulted in slight edema and very slight necrosis after 6 applications (38). When instilled in the eyes of rabbits, it caused immediate but moderate conjunctival irritation and swelling which subsided within one week (38).

#### 8.3.1.4.2 Chronic Toxicity

Few reports are available on the chronic toxicity of 1,1-dichloroethane. In a study conducted by Hoffmann et al. (124), rats, guinea pigs, rabbits, and cats exposed to a vapor concentration of 2025 mg/m$^3$, 6 hours daily, 5 days a week for 13 weeks exhibited no adverse effects. Rats, guinea pigs and rabbits tolerated an additional 13 weeks at 4050 mg/m$^3$ with no adverse effects. However, cats exposed to 4050 mg/m$^3$ suffered renal damage as evidence by elevated blood urea nitrogen levels and histopathological signs of renal tubular dilation and degeneration.

### 8.3.2 Human and Epidemiologic Studies

The available data indicate that 1,1-dichloroethane is capable of causing anesthesia at high concentrations (12). There is at present no clear evidence for toxic effects following chronic low level exposures.

### 8.3.3 Levels of Concern

Because of limitations in the available data, there are few estimates of exposure levels of concern for 1,1-dichloroethane. The USEPA has not set a water quality criterion for human health (355), nor has a carcinogenic potency value or reference dose been calculated. However, EPA (3735) has calculated acceptable subchronic (AIS) and chronic intake values (AIC) for 1,1-dichloroethane (3735). For oral exposures, the AIS was reported as 81 mg/day and the AIC as 8.1 mg/day. For inhalation exposures, the AIS was calculated as 97 mg/day and the AIC as 9.7 mg/day.

For occupational exposures to 1,1-dichloroethane, the current OSHA standard is 100 ppm (400 mg/m$^3$) averaged over an 8-hour workshift (3539). The current ACGIH recommendation is a threshold limit value of 200 ppm (800 mg/m$^3$) (3005).

### 8.3.4 Hazard Assessment

The data are sparse regarding the toxicity of 1,1-dichloroethane. Evidence for carcinogenicity is inconclusive. Small increases in some tumor types were observed in the NCI study (125); however, the overall evidence is inconclusive. Only additional study will clarify this issue. When tested in the rat liver foci assay, the compound gave negative results for initiation, but positive results for tumor promotion. Conflicting results were reported for the Ames mutagenicity assay and for a mammalian cell transformation assay. No teratogenic effects were observed in a study in which rats were exposed to 24,300 $mg/m^3$ of 1,1-dichloroethane during gestation.

Because of the paucity of data on the acute and chronic oral toxicity of 1,1-dichloroethane, a complete assessment of hazard associated with exposure to 1,1-dichloroethane cannot be made at this time.

## 8.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of 1,1-dichloroethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of 1,1-dichloroethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of 1,1-dichloroethane, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the 1,1-dichloroethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the 1,1-dichloroethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; 1,1-dichloroethane is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC

with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for 1,1-dichloroethane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical 1,1-dichloroethane detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.07 μg/L (Method 601) | 0.7 μg/kg (Method 8010) |
| 4.7 μg/L (Method 624) | 5 μg/kg (Method 8240) |
| 10 μg/L (Method 1624) | |
| 5 μg/L (Method 8240) | |
| 0.7 μg/L (Method 8010) | |

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| COMMON SYNONYMS: | CAS REG. NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25 °C (12) |
|---|---|---|
| 1,2-Dichloroethane<br>EDC<br>Ethane dichloride<br>Ethylene chloride<br>Ethylene dichloride<br>Glycol dichloride | 107-06-2 $C_2H_4Cl_2$<br>NIOSH Number:<br>KI0525000<br>STRUCTURE:<br>Cl–$CH_2$–$CH_2$–Cl | 4.05 mg/m³ ≈ 1 ppm;<br>0.247 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT:<br>98.96 |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as 1,2-dichloroethane with cyanides, amines, azo compounds, hydrazines, caustics, or nitrides, or with mercaptans or other organic sulfides, commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases, and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505, 3133). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Odor: Chloroform-like | (23) |
| | • Odor Threshold: 88.000 ppm | (384) |
| | • Density: 1.2530 g/mL (at 20°C) | (21) |
| | • Freeze/Melt Point: -35.50°C | (23) |
| | • Boiling Point: 83.50°C | (23) |
| | • Flash Point: 13.30°C closed cup | (23) |
| | • Flammable Limits: 6.20 to 16.00% by volume | (51,506) |
| | • Autoignition Temp.: 413.0°C | (51,506) |
| | • Vapor Pressure: 6.37E+01 mm Hg (at 20°C) | (21) |
| | • Satd. Conc. in Air: 3.5000E+05 mg/m³ (at 20°C) | (67) |
| | • Solubility in Water: 8.69E+03 mg/L (at 20°C) | (21) |

| | |
|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Viscosity: 0.840 cp (at 20°C) (21)<br>• Surface Tension: 3.1380E+01 dyne/cm (at 20°C) (21)<br>• Log (Octanol-Water Partition Coeff.): 1.48 (29)<br>• Soil Adsorp. Coeff.: 1.40E+01 (33)<br>• Henry's Law Const.: 1.10E-03 atm · $m^3$/mol (at 25°C) (74)<br>• Bioconc. Factor: 1.40 (estim), 2.00 (bluegills) (123,659) |
| PERSISTENCE IN THE SOIL-WATER SYSTEM | 1,2-Dichloroethane is expected to be highly mobile in the soil/ground-water system. Adsorption onto soil, particularly soil of <1% organic content, is low. Volatilization from surface soils and through soil air may be important transport processes. Microbial biodegradation in soil is not expected to be significant. |
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of 1,2-dichloroethane to groundwater drinking water supplies. Inhalation resulting from volatilization from surface soils may also be important. |
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (54)<br>Ingestion and inhalation may cause nausea, vomiting, mental confusion, dizziness and pulmonary edema. Liquid and vapor may cause eye and skin irritation. Acute exposures can lead to death from respiratory and circulatory failure.<br><br>Acute Toxicity Studies:<br><br>INHALATION:<br>$LC_{Lo}$ 1000 ppm · 4 hr Rat (59)<br>$LC_{50}$ 3000 ppm · 7 hr Monkey (3504)<br>$TC_{Lo}$ 4000 ppm · 1 hr Human (3038) |

| | |
|---|---|
| HEALTH HAZARD DATA (Cont.) | ORAL:<br>$LD_{50}$ 670 mg/kg — Rat (47)<br>$LD_{50}$ 5700 mg/kg — Dog (3504)<br>$TD_{Lo}$ 428 mg/kg — Human (3504)<br>$LD_{Lo}$ 286 mg/kg — Human (3504)<br><br>SKIN:<br>$LD_{50}$ 2800 mg/kg — Rabbit (12)<br><br>Long-Term Effects: Liver and kidney damage, neurologic changes<br>Pregnancy/Neonate Data: Conflicting results<br>Genotoxicity Data: Suggestive positive evidence of mutagenic potential<br>Carcinogenicity Classification:<br>IARC - Group 2B (possibly carcinogenic to humans)<br>NTP - Positive in rats and mice<br>EPA - Group B2 (probable human carcinogen; sufficient evidence in animals and inadequate evidence in humans) |
| HANDLING PRECAUTIONS (52,3306) | Handle chemical only with adequate ventilation.<br>• Vapor concentrations up to 50 ppm: any supplied-air respirator or self-contained breathing apparatus.<br>• 50-250 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece.<br>• Chemical goggles if there is probability of eye contact.<br>• Natural rubber, neoprene, PVC, PE, PVA gloves/ apron/boots and appropriate clothing to prevent prolonged or repeated skin contact with liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 1 ppm; STEL: 2 ppm
- AFOSH PEL (8-hr TWA): 1 ppm; STEL (15-min): 2 ppm

Criteria
- NIOSH REL (10-hr TWA): 1 ppm; ceiling limit (15-min): 2 ppm
- NIOSH IDLH (30-min): None, treat as a potential carcinogen
- ACGIH TLV® (8-hr TWA): 10 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards (3742)
MCLG: 0 μg/L
MCL : 5 μg/L

EPA Health Advisories and Cancer Risk Levels (3977)
The EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- 1-day (child): 700 μg/L
- 10-day (child): 700 μg/L
- longer-term (child): 700 μg/L
- longer-term (adult): 2600 μg/L
- 1E-04 cancer risk level: 40μg/L

WHO Drinking Water Guideline (666)
10 μg/L

EPA Ambient Water Quality Criteria (3734)
- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms, levels of 9.4 μg/L, 0.94 μ/L and 0.094 μ/L may result in estimated incremental lifetime cancer risks of 1E-05, 1E-06, and 1E-07, respectively.
  - Based on ingestion of contaminated aquatic organisms only, levels of 2430 μg/L, 243 μg/L, and 24.3 μg/L may result in estimated incremental lifetime cancer risks of 1E-05, 1E-06, and 1E-07, respectively.

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 118 mg/L.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 20 mg/L.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 113 mg/L.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:
No reference dose available.

## REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

1,2-Dichloroethane is designated a hazardous substance (347). It is also listed as a toxic pollutant, subject to pretreatment regulations for new and existing sources, and to effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), and metal finishing (3768). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

1,2-Dichloroethane is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). Under the National Primary Drinking Water Regulations, the MCL for 1,2-dichloroethane is 0.005 mg/L, and the MCLG is zero (3773, 3772). In states with an approved Underground Injection Control program, a permit is required for the injection of 1,2-dichloroethane-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

1,2-Dichloroethane is identified as a toxic hazardous waste (U077) and listed as a hazardous waste constituent (3783, 3784). A non-specific source of 1,2-dichloroethane-containing waste is the production of chlorinated aliphatic hydrocarbons (325, 3765). Waste streams from the following industries contain 1,2-dichloroethane and are listed as specific sources of hazardous wastes: organic chemicals (production of chloroethane, 1,2-dichloroethane, vinyl chloride, 1,1,1-trichloroethane, trichloroethylene and tetrachloroethylene) (3774, 3765).
1,2-Dichloroethane is subject to land disposal restrictions when its concentration as a hazardous constituent of certain wastewaters exceeds designated levels (3785). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited.

Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). 1,2-Dichloroethane is included on EPA's groundwater monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their groundwater for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Toxic Substances Control Act (TSCA)
Manufacturers, processors, or importers who possess health and safety studies on 1,2-dichloroethane must submit them to EPA (3789).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
1,2-Dichloroethane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 2270 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing 1,2-dichloroethane but these depend upon the concentration of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of 1,2-dichloroethane must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
1,2-Dichloroethane is exempt from a tolerance requirement when used as a solvent or cosolvent in pesticide formulations applied to growing crops or to animals (315). Exemptions also apply when it is used as a fumigant after harvest for barley, corn, oats, popcorn, rice, rye, sorghum and wheat (314).

Occupational Safety and Health Act (OSHA)
Employee exposure to 1,2-dichloroethane shall not exceed an 8-hour time-weighted average (TWA) of 1 ppm. Employee exposure shall not exceed 2 ppm for any 15 minute period during an 8-hour work-shift (3539).

Clean Air Act (CAA)
EPA intends to list 1,2-dichloroethane as a hazardous air pollutant for which it will establish emission standards under Section 112 of the Clean Air Act (3685).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated 1,2-dichloroethane as a hazardous material with a reportable quantity of 2270 kg, subject to requirements for packaging labeling and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
1,2-Dichloroethane may be present as an extraction residue in spice oleoresins at a level not exceeding 30 ppm (361). 1,2-Dichloroethane is approved for use as an indirect food additive as a component of adhesives (3209).

- State Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

ALABAMA
Alabama requires that the annual average maximum contaminant level of 1,2-dichloroethane in drinking water not exceed 0.005 mg/L in all community water systems, and non-community non-transient water systems (3015).

CALIFORNIA
California has an action level of 1 μg/L (1 ppb) for drinking water (3098).

CONNECTICUT
Connecticut has an action level and a quantification limit of 1 μg/L for drinking water (3138, 3137).

FLORIDA
Florida has set a maximum contaminant level of 3 μg/L for drinking water (3219).

NEW JERSEY
New Jersey has set a maximum contaminant level of 2 μg/L for drinking water, and a water quality criterion of 2 μg/L for surface waters (3497, 3496).

NEW YORK
New York has an ambient water quality standard of 0.8 μg/L for surface waters used for drinking water supply (3501).

PENNSYLVANIA
Pennsylvania has a human health criterion (cancer risk level) of 0.4 μg/L for surface waters (3561).

RHODE ISLAND
Rhode Island has an acute freshwater quality guideline of 5900 μg/L and a chronic guideline of 131 μg/L for surface waters for the protection of aquatic life. These guidelines are enforceable under Rhode Island state law (3590).

VERMONT
Vermont has a preventive action limit of 0.5 μg/L for groundwater, and an enforcement standard of 5.0 μg/L (3682).

WISCONSIN
Wisconsin has a preventive action limit of 0.05 μg/L and an enforcement standard of 0.5 μg/L for groundwater (3840).

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed that solid wastes be listed as hazardous because they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is equal to or greater than 0.40 mg/L 1,2-dichloroethane. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Programs

MOST STATES

Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3683).

CALIFORNIA

California has proposed a maximum contaminant level of 0.5 μg/L for 1,2-dichloroethane in drinking water (3096).

MINNESOTA

Minnesota has proposed a Recommended Allowable Limit (RAL) of 3.8 μg/L for drinking water (3451). Minnesota has also proposed a Sensitive Acute Limit (SAL) of 29500 μg/L for designated surface waters, and chronic criteria of 3.8 μg/L for designated groundwaters and 3.7 μg/L for designated surface waters for the protection of human health (3452).

WEST VIRGINIA

West Virginia has proposed a water quality criterion of 0.035 μg/L for Public A surface waters. Final action is expected in late spring 1989 (3835).

EEC Directives

Directive on Ground-Water (538)

Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)

The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)

1,1,2,2-Tetrachloroethane is listed as a Class I/a toxic substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto- pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the method and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,1,2,2-Tetrachloroethane is classified as a toxic substance and is subject to packaging and labeling regulations.

## 9.1 MAJOR USES

1,2-Dichloroethane is the largest volume chlorinated organic compound produced in the United States. Estimated U.S. production in 1985 was about 10 billion lb. It is used primarily as a starting material in the manufacture of vinyl chloride. It is also used in the production of tetrachloroethylene, trichloroethylene, 1,1,1-trichloroethane, vinylidene chloride, and ethyleneamines. Minor applications include use as a metal degreasing agent, solvent, fumigant for grain, upholstery and carpets, paint, varnish, and finish remover, wetting and penetrating agent, and as a lead-scavenging agent in gasoline. Because of its toxicity and flammability, its use as a solvent is decreasing as less hazardous replacements become available (25, 3353, 3615, 3114).

## 9.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 9.2.1 Transport in Soil/Ground-water Systems

#### 9.2.1.1 Overview

The 1,2-isomer of dichloroethane may move through the soil/ ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways for low soil concentrations can be assessed with an equilibrium partitioning model as shown in Table 9-1.

These calculations predict the distribution of 1,2-dichloroethane among soil particles, soil water and soil air. The 1,2-dichloroethane associated with the water and air phases of the soil is more mobile than that which is adsorbed on the soil.

The estimates derived from the model for unsaturated topsoil indicate that a substantial 26% of the 1,2-dichloroethane is expected to be present in the soil-water phase and can thus migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. For the portion of 1,2-dichloroethane in the gaseous phase of the soil (3.6%), diffusion through the soil-air pores up to the surface, and subsequent removal by evaporation, may be a significant loss pathway.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the 1,2-dichloroethane (94%) is expected to be in the soil-water phase (Table 9-1) and trans ported with flowing ground-water. Ground-water underlying 1,2-dichloroethane-contaminated soils with low organic content may therefore be highly vulnerable to contamination.

TABLE 9-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR
1,2-DICHLOROETHANE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil at 25°C[b,c] | 70.3 | 26.1 | 3.6 |
| Saturated deep soil[d] | 5.6 | 94.4 | - |

a) Calculations based on Mackay's equilibrium partitioning model(34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 14 (33).
c) Henry's law constant = 1.1E-03 atm · $m^3$/mol at 25°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

### 9.2.1.2 Sorption on Soils

The mobility of 1,2-dichloroethane in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. Soil sorption coefficients ($K_{oc}$) of 19-83 have been reported for 1,2-dichloroethane, indicating that the compound will not be strongly bound to soils (3740). In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Retardation rates, which represent the interstitial water velocity/pollutant velocity in the soil, were reported by Wilson et al. (82) to be a function of $K_{oc}$, the ratio of soil density (a) to soil water content (b) and the organic content (oc) of the soil according to the following equation:

$$R_f = 1 + (a/b)K_{oc}(oc)$$

Wilson et al. (82) reported a retardation factor of only 1.2 for 1,2-dichloroethane in sandy soil. Schwarzenbach et al. (77) reported retardation factors as a function of soil type for some chlorinated organic compounds that have $K_{oc}$ values higher than that reported for 1,2-dichloroethane. The data indicate some retardation (i.e., adsorption) in soils having 1-2% organic carbon and little or no retardation in deep soils having less than 0.1% organic carbon. Assuming analogous soil conditions, adsorption of 1,2-dichloroethane, particularly to deep soils, is not expected to be significant.

Wilson et al. (82) investigated the transport and fate of 1,2-dichloroethane in solutions applied to sandy soils. In a soil column receiving a 1,2-dichloroethane solution of less than 1 mg/L, approximately 50% was volatilized and 37-61% percolated through the soil column with minimal retardation.

### 9.2.1.3 Volatilization from Soils

Transport of 1,2-dichloroethane vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H were also observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of 1,2-dichloroethane from surface soils.

No information was available for the two other physicochemical properties influencing 1,2-dichloroethane volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

Volatilization of 1,2-dichloroethane from surface soils has been reported to be approximately one order of magnitude slower than volatilization from a well-stirred aqueous solution (82). Half-lives for volatilization from stirred aqueous solutions were reported to range from 29-90 minutes, depending on the degree of agitation (10). The evaporative half-life of 1,2-dichloroethane was estimated to be 4 hr for a water depth of 1 m, with a wind speed of 3 m/sec, and with a current speed of 1 m/sec (3740). Based on a reaeration rate ratio of 0.58, and oxygen reaeration rates of 0.19/day and 0.96/day, the evaporative half-life of the compound in a river and pond were estimated to be >1 day and >6 days, respectively (3740).

### 9.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of 1,2-dichloroethane in soil/ground-water systems is not well documented. In most cases, it should be assumed that 1,2-dichloroethane will persist for months to years (or more). The 1,2-dichloroethane released into the air will eventually undergo photochemical oxidation; a tropospheric lifetime of 1.7 months has been reported for 1,2-dichloroethane (10).

Under normal environmental conditions, 1,2-dichloroethane is not expected to undergo rapid hydrolysis. Callahan et al. (10) report data estimating the hydrolysis half-life for 1,2-dichloroethane at 50,000 years; hydrolysis is not expected to occur at a rate competitive with volatilization.

Literature references to microbial degradation of compounds such as 1,2-dichloroethane are very few. Most references indicate that low molecular weight chloroaliphatics are not rapidly metabolized in the environment (76), although biodegradation by acclimated populations may occur. Wilson et al. (82) and Pearson and McConnell (75) observed no evidence of microbial transformation of 1,2-dichloroethane. Biodegradation under anerobic conditions has also been reported to be very slow (3740).

Slow to moderate degradation of 1,2-dichloroethane was observed by Tabak et al. (79, 3697) with an acclimated, activated-sludge population; and Thom and Agg (80) included 1,2-dichloroethane in a list of organic chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization was achieved. In situ biodegradation by blended specialized bacterial seed cultures has been suggested as a decontamination procedure for soils contaminated with 1,2-dichloroethane (650).

In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as 1,2-dichloroethane is very low and drops off sharply with increasing depth. Thus, biodegradation in the soil/ground-water system would be assumed to be of minimal importance except, perhaps, in landfills with active microbio-logical populations.

Some evidence of the metabolism of 1,2-dichloroethane in fish and oysters has been presented (75).

### 9.2.3 Primary Routes of Exposure From Soil/Ground-water Systems

The above discussion of fate pathways suggests that 1,2-dichloroethane is highly volatile in aqueous solutions, weakly adsorbed by soil, and has no significant potential for bioaccumulation. This compound may volatilize from soil surfaces, but that portion not removed by volatilization may eventually migrate to ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of 1,2-dichloroethane from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. The potential for ground-water contamination is high, particularly in sandy soil. Mitre (83) reported that 1,2-dichloroethane has been found at 25 of the 546 National Priority List (NPL) sites. It was detected at 17 sites in ground water, 12 sites in surface water and 1 site in air. The potential for exposure through drinking water is confirmed by the presence of 1,2-dichloroethane in ground-water sources of drinking water in the United States. The USEPA (62, 64) reported the following results from a variety of surveys of drinking water supplies:

| Survey | No. Sampled | No. Positive | Range of Positives |
|---|---|---|---|
| State Data | 2628 | 117 | Trace - 2100 μg/L |
| NOMS | 113 | 2 | 0.1 - 1.8 μg/L |
| NSP | 142 | 2 | Trace - 4.8 μg/L |
| CWSS | 451 | 4 | 0.5 - 1.8 μg/L |
| GWSS (Random Data) | 466 | 3 | 0.5 - 0.95 μg/L |

The state data include only ground-water sources and were compiled from various state reports on local contamination problems. The state data are not considered to be statistically representative of national occurrence. The National Organics Monitoring Survey (NOMS) included data from both ground- and surface water supplies, as did the National Screening Program (NSP) and the Community Water Supply Study (CWSS). The USEPA (531) Ground-water Supply Survey (GWSS) is the most recent study. This survey sampled a total of almost 1000 drinking water systems using ground water; 466 selected at random and about 500 selected by the state as potentially contaminated. The survey results suggest that 1,2-dichloroethane is found in drinking water, particularly in ground water as evidenced by the state reports of contamination problems. The USEPA (64) estimates that 0.3% of the nation's ground-water supplies are contaminated with 1,2-dichloroethane (at concentrations >0.5 μg/L).

Based on the maximum reported concentration of 1,2-dichloroethane in a U.S. water supply (2.1 mg/L for a sample from Elkhart, IN)), EPA estimated that the maximum intake of the compound from drinking water would be 4.2 mg/day (3740).

The properties of 1,2-dichloroethane, as well as its occurrence in ground water at NPL sites indicate that 1,2-dichloroethane has the potential for movement in soil/ground-water systems. This compound may eventually reach surface waters by this mechanism, suggesting several other exposure pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion exposure or inhalation exposure following volatilization.
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposures.
- Recreational use of these waters may result in dermal exposures.

- Domestic animals may consume or be dermally exposed to contaminated ground- or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground water for two reasons. First, the Henry's law constant for 1,2-dichloroethane suggests that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this compound is low (2.0 determined experimentally for bluegill sunfish and <1 calculated from $K_{ow}$ values), suggesting no significant bioaccumulation in aquatic organisms (3740).

### 9.2.4 Other Sources of Exposure

The data presented above on the Ground-water Supply Survey (531) suggest that 1,2-dichloroethane is found only in a limited number of ground-water supplies used as drinking water. Coniglio et al. (223) reported that 1,2-dichloroethane was found in surface water supplies. In a summary of data available as of 1980, these authors reported that 4.5% of 133 finished surface water samples were contaminated. The average concentration was reported as 2.14 μg/L with a range of 0.8 to 4.8 μg/L.

The 1,2-isomer of dichloroethane is a large-volume chemical, used primarily in the production of vinyl chloride monomer and other chlorinated compounds. Large amounts of the compound are released to the atmosphere in the vicinity of production facilities (76). The volatility of this compound also suggests that it may be found in air. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For 1,2-dichloroethane, they had data for 1,675 locations. In urban and suburban locations, the median concentration was 0.49 $\mu g/m^3$ In source-dominated areas, the median concentration was 4.9 $\mu g/m^3$ These results suggest inhalation exposure to persons in these areas.

## 9.3 HUMAN HEALTH CONSIDERATIONS

### 9.3.1 Animal Studies

#### 9.3.1.1 Carcinogenicity

In long-term bioassays conducted on rodents, 1,2-dichloroethane has been found be tumorigenic when administered by gavage (138) or by repeated skin application (142), but nontumorigenic after chronic inhalation exposures (137). Positive findings were reported in a study conducted by the National Cancer Institute, in which male and female Osborne-Mendel rats and $B6C3F_1$ mice were administered technical-grade 1,2-dichloroethane in corn oil by gavage at 2 dosage levels, 5 days per week for 78 weeks. The time-weighted average doses for rats of both sexes were 95 and 47 mg/kg/day. For mice, dosages were 195 and 97 mg/kg/day (males) and 299 and 149

mg/kg/day (females). Mice were terminated at 90 weeks and rats at 106 weeks. Elevated incidences of squamous-cell carcinomas of the forestomach and hemangiosarcomas of the circulatory system were seen in male rats and mammary adenocarcinomas were seen in female rats. In mice, the incidence of alveolar/bronchiolar adenomas in both sexes was statistically significant. The incidence of mammary adenocarcinomas and endometrial tumors in female mice as well as the incidence of hepatocellular carcinomas in male mice were positively correlated with treatment (138).

Positive tumorigenicity of 1,2-dichloroethane following repeated skin applications was reported by Van Duuren et al. (142). Application of 125 mg of 1,2-dichloroethane in acetone to the dorsal skin of Swiss mice 3 times weekly for a lifetime resulted in the induction of benign lung tumors as well as a papilloma and two squamous-cell carcinomas of the forestomach.

The absence of a carcinogenic effect following chronic inhalation exposures were reported by Maltoni et al. (137). In this study Swiss mice and Sprague-Dawley rats of both sexes were exposed to 5, 10, 50 or 250 ppm 1,2-dichloroethane (99.8% pure) 7 hr/day, 5 days/week, for 78 weeks. The 250 ppm dose was dropped to 150 ppm after a few weeks because of severe toxic effects. All animals were allowed to live beyond treatment until spontaneous death occurred. No treatment-related increased incidence of tumors was seen in either mice or rats.

Cancer initiation/promotion assays have also been conducted on 1,2-dichloroethane. Story et al. (3688) tested the compound in the rat liver foci assay. Negative results were obtained in initiation tests in which phenobarbital was used as the promoting agent, but positive results were seen in the promotion tests in which dimethylnitrosamine (DENA) was used as the initiating agent. In contrast to the results reported by Story et al., Klaunig et al. (3364) found that 1,2-dichloroethane exhibited no liver or lung tumor promoting effects in B6C3F$_1$ mice following initiation with DENA. In an in vitro cell transformation assay using BALB/c-3T3 mouse cells (without an exogenous metabolic activation system), 1,2-dichlorethane gave negative results (3732).

Based on the available data, IARC (25) has listed 1,2-dichloroethane in category 2B (sufficient evidence of animal carcinogenicity, and inadequate evidence of carcinogenicity in humans) in its weight-of-evidence ranking for potential carcinogens.

### 9.3.1.2 Genotoxicity

1,2-Dichloroethane has been found to be genotoxic in 3 strains of Salmonella typhimurium without metabolic activation and in DNA-polymerase deficient E. coli. (135). 1,2-Dichloroethane also induced significant increases in sex-linked recessive lethal mutations in Drosophila melanogaster (135) but was inactive in a micronucleus test using bone marrow cells from mice injected intraperitoneally twice, 24 hours apart, with 396 mg/kg of 1,2-dichloroethane (668). Data from a mouse spot test, an

in utero whole mammal system which detects somatic gene mutations, showed a significant effect against pooled control values. However, the incidence of mutations was not statistically significant when compared to the vehicle controls for this compound. Based on these findings, the authors concluded that 1,2-dichloroethane was probably mutagenic in the mouse (669).

1,2-Dichloroethane administered in drinking water to male or female mice failed to produce dominant lethal mutations at 50 mg/kg (3388).

It was also found to be mutagenic in 2 human lymphoblastoid cell lines at concentrations ranging from 100 to 500 μg/ml (385).

No mutagenic activity was recorded for 1,2-dichloroethane in the BALB/c-3T3 cell transformation assay: tests were conducted in the absence of an exogenous metabolic activation system (708), but this agent did significantly enhance viral transformation of Syrian hamster embryo cells when the cells were treated in sealed chambers without activation (3274).

Storer et al. (3687) exposed male mice to 1,2-dichloroethane via three routes and measured DNA breaks in liver at 4 and 24 hrs after treatment. Significant breakage was found with all three routes 4 hrs after treatment. Treatment by gavage with 100 mg/kg body wt was the lowest demonstrably positive dose, 150 mg/kg was the lowest positive dose for intraperitoneal treatment, and the lowest effective dose via inhalation was 1072 ppm, a dose that killed 4 of the 5 males treated. At 200 mg/kg DCE given intraperitoneally, breaks were still detected at 24 hrs after treatment at levels that indicated little or no repair of DNA damage taking place between 4 and 24 hrs.

### 9.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Withey .nd Karpinski (3843) exposed rats to 1,2- dichloroethane by inhalation for 5 hours on day 17 of gestation to 6 levels ranging from 153 to 1999 ppm. Placental penetration occurred, with fetal levels averaging less than half of maternal levels. Inhalation was also the route of exposure chosen by Vozovaya (3821). Female rats breathing 14 ppm 1,2- dichloroethane 4 hr/day for 6 months and during gestation experienced some lengthening of the estrous cycle and a decrease in fertility. Litter size, birth weight and peri- and postnatal survival were significantly reduced. In another study, pregnant rats and rabbits were exposed to vapor levels of 100 or 300 ppm for seven hours daily on days 6-15 and 6-18 of gestation, respectively (3582). Severe maternal toxicity was observed in rats at 300 ppm (two-thirds of the dams died). Considerably less maternal toxicity occurred in rabbits at either exposure level. No embryotoxicity, fetotoxicity or malformations were observed in either species which could be attributed to 1,2- dichloroethane exposure. The same investigator, using rats, also studied the effects of parental exposure to 25, 75 or 150 ppm on fertility and reproduction in a single-generation study with two litters. No adverse effects on the reproductive capacity of the adults or on growth and survival of the offspring were noted (3582).

No dose-dependent effects on fertility, gestation or viability were seen in male and female mice that received 1,2- dichloroethane in drinking water at concentrations of 0, 0.03, 0.09 or 0.29 mg/mL/day for 25 weeks. These concentrations were designed to yield daily doses of 0, 5, 15, or 50 mg/kg. This treatment also failed to produce significant dominant lethal mutations or teratogenic effects (386). Alumot et al. (3019) administered 250 or 500 ppm in feed mash to male and female rats for two years. No significant decrease in fertility, litter size, fetal weight or offspring mortality before or after birth was observed.

### 9.3.1.4 Other Toxicologic Effects

#### 9.3.1.4.1 Short-term Toxicity

In inhalation studies on rats, Spencer et al. (141) found that the test animals survived a 12-min exposure to 12,000 ppm, a 1-hr exposure to 3000 ppm, and a 7-hr exposure to 300 ppm. Concentrations below 12,000 ppm produced various degrees of CNS depression but did not result in unconsciousness or death within the duration of the exposure. At concentrations of 3000 ppm or above, definite CNS depression was observed in the form of inactivity or stupor. Organic and histopathological changes were observed in the liver and kidneys. The responses of other species reported by other investigators fall within the same general range.

Oral $LD_{50}$ values of 670, 860, and 5700 mg/kg have been reported for rats, rabbits, and dogs, respectively (3504).

Vapors of 1,2-dichloroethane have been shown to cause reversible clouding of the corneas of dogs and foxes but not of other species (19). An $LC_{50}$ value of 3,000 ppm for a 7-hr exposure has been reported for monkeys (3045).

Storer et al. (3687) evaluated the acute hepatotoxicity of 1,2-dichloroethane in male $B6C3F_1$ mice following oral, intraperitoneal, and inhalation exposures. An oral dose of 100 mg/kg, or an i.p. dose of 150 mg/kg resulted in hepatic DNA damage, but a 4-hr exposure to 150 ppm (nonnecrogenic) or 500 ppm (necrogenic) resulted in no hepatic damage. Higher inhalation exposure levels caused liver damage, but acute necrogenic effects could not be ruled out.

Sherwood et al. (3641) reported on the effects of single or multiple inhalation exposures to 1,2-dichloroethane on the pulmonary defenses of mice and rats. In mice, inhalation exposure to 10 ppm resulted in reduced pulmonary bactericidal activity, and increased mortality from respiratory tract infections. The latter effect was also seen at 5 ppm, but not at 2.5 ppm, even after five consecutive daily exposures. In rats, no adverse effects on respiratory defense mechanisms were induced by single exposures as high as 200 ppm, or after 12 5-hr exposures to 10, 20, 50, or 200 ppm.

Although 1,2-dichloroethane is absorbed through the skin, it takes large doses to cause serious acute systemic poisoning (12). The dermal $LD_{50}$ in rabbits was calculated to be 2.8 g/kg body weight (12).

#### 9.3.1.4.2 Chronic Toxicity

Heppel et al. (136) studied the chronic toxicity of 1,2-dichloroethane by exposing animals 7 hours per day, 5 days a week to vapor concentrations ranging from 100 to 1000 ppm. At a concentration of 1000 ppm rats, rabbits and guinea pigs died after a few 7-hour exposures. Dogs, cats and monkeys proved to be more resistant but deaths eventually occurred. Pathological examination showed pulmonary congestion, renal tubular degeneration, fatty degeneration of the liver and less commonly, necrosis and hemorrhage of the adrenal cortex and fatty infiltration of the myocardium. Dogs appeared unaffected after 8 months exposure to 400 ppm. At autopsy, slight changes in the liver were detected. Deaths occurred among other animal species at this exposure level. Pathological examination revealed lesions similar to those seen at 1000 ppm. When the vapor concentration was lowered to 100 ppm, all animals survived exposures for 4 months with no demonstrable lesions.

A comparable study was carried out by Spencer et al. (141) who exposed animals by inhalation for 7 hours a day, 5 days per week. High mortality was again seen in rats and guinea pigs at 400 ppm after 14 to 56 days of exposure. Guinea pigs exhibited more liver and kidney changes than rats. At 100 ppm, rats, guinea pigs, rabbits and monkeys were exposed 120 times in 168 days without apparent effect.

### 9.3.2 Human and Epidemiologic Studies

#### 9.3.2.1 Short-term Toxicologic Effects

Short-term exposures to high vapor concentrations of 1,2-dichloroethane produce irritation of the eyes, nose and throat. Ingestion or inhalation of the compound causes dizziness, nausea, vomiting, increasing stupor, cyanosis, rapid pulse, loss of consciousness, and systemic injury to the liver, kidneys and lungs (3134). In oral exposures a latency period of about 1 hour often occurs before the onset of symptoms (139).

A significant number of human poisonings by ingestion of 1,2-dichloroethane have been reported in the literature. Most of these cases were fatal, with death attributed to respiratory and circulatory failure. A 14-year-old boy who ingested 15 mL developed symptoms of headache, lethargy, vomiting and decreased urination. He developed pulmonary edema and refractory hypotension. Death occurred on the sixth day after exposure. In another report, three men, 19-27 years of age, developed dizziness and weakness and began vomiting after ingesting 70-80 mL. They eventually lost consciousness and died 5-8 hours after ingestion (139). The acute oral $LD_{50}$ for 1,2-dichloroethane, was estimated to fall in the range of 0.2 to 1.0 g/kg (3134, 3153). Nonfatal cases of ingestion of 1,2-dichloroethane were reported by NIOSH (134).

The effects of acute exposure by inhalation are similar to those seen with ingestion exposure. The level at which symptoms appear is estimated to be 75-125 ppm (139). Two fatal cases of occupational poisoning with 1,2-dichloroethane occurred in chemical plant workers exposed to unknown vapor concentrations. Both workers initially lost consciousness but regained it when brought into fresh air. Both became lethargic and cyanotic. Circulatory weakness developed and death occurred about 30 hours after exposure. Autopsies revealed evidence of liver and kidney damage as well as pulmonary edema and hemorrhages throughout the body (139).

Prolonged skin exposure to the liquid produces severe irritation, moderate edema and necrosis (46). Skin absorption from liquid contact could be a significant route of entry into the body. The amount of 1,2-dichloroethane absorbed from immersion of both hands for 1 minute has been estimated to be 36.6 mg/minute. This is equivalent to an inhalation exposure concentration of 3615 ppm for 1 minute (135). When liquid 1,2-dichloroethane is splashed in the eyes, pain, irritation and lacrimation may occur. Prompt removal by washing should prevent significant injury (19). Among twenty cases of corneal burns resulting from splashes occurring in the eyes of workmen, all eventually recovered (19).

### 9.3.2.2 Chronic Toxicologic Effects

Chronic exposures to 1,2-dichloroethane in an occupational environment have been associated with loss of appetite, nausea, vomiting, epigastric pain, irritation of the mucous membranes, neurologic changes and liver and kidney impairment. Although fatal cases have been reported less frequently with chronic exposure than with acute exposure, chronic effects can progress unless the exposures are adequately reduced. In a study of agricultural workers using 1,2-dichloroethane as a fumigant, the most common symptoms of exposure were weakness, headache, conjunctival congestion and irritation, nausea, cough, liver pain and increased heart rate. Exposure levels averaged 15 ppm. Since the workers' skin came into contact with the 1,2-dichloroethane, absorption by this route probably was as significant a contribution to exposure as inhalation (139). A study of 100 factory workers exposed to 1,2-dichloroethane for 6 months to 5 years at concentrations not in excess of 25 ppm showed no changes in blood or internal organ functions. However, there were central nervous system disturbances of varying degrees. These included irritability, sleeplessness and decreased heart rate (139).

An epidemiological study conducted by Isacson et al. (3329) with data from the Iowa Cancer Registry evaluated the relationship between levels of volatile organic compounds in finished drinking water derived from a stable ground-water source with age-adjusted, sex-specific cancer rates. The results showed an association between 1,2-dichoroethane and cancers of the colon and rectum in men. The investigators, however, suggested that the data were not indicative of a causal relationship, but rather were indicative of the general state of anthropogenic contamination of the drinking water.

Austin and Schnatter (3043) conducted a case-control study of brain tumors in petrochemical workers, but found no evidence of an association between exposure to 1,2-dichloroethane and cancer.

### 9.3.3 Levels of Concern

The Environmental Protection Agency (355) has established an ambient water quality criterion of zero for the maximum protection of human health from the potential carcinogenic effects of 1,2-dichloroethane exposure through ingestion of water and contaminated aquatic organisms, based on the induction of hemangiosarcomas in male Osborne- Mendel rats. The concentrations of 1,2-dichloroethane in ambient waters resulting in incremental lifetime cancer risks of 1E-05, 1E-06, 1E-07 from ingestion of both water and contaminated aquatic organisms were estimated to be 9.4, 0.94 and 0.094 μg/L, respectively (3734). Risk estimates are expressed as a probability of cancer after a lifetime consumption of two liters of water per day and consumption of 6.5 g of fish per day containing a specified concentration of the contaminant. Thus, a risk of 1E-05 implies that at a 9.4 μg/L concentration of 1,2-dichloroethane in ambient waters, a lifetime daily ingestion of two liters of drinking water and consumption 6.5 g of fish would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 people exposed. For drinking water alone EPA calculated that the concentration of 1,2-dichloroethane corresponding to a 1E-04 risk would be 40 μg/L (3805).

EPA has set the maximum contaminant level goal (MCLG) for 1,2-dichloroethane in drinking water at 0 and the maximum contaminant level (MCL) at 5 μg/L (3742). Health Advisories (HA) for a 10-kg child have been set at 700 μg/L for a 1-day, 10-day, and longer term exposure. The longer term HA for a 70-kg adult has been set at 2,600 μg/L (3977).

A health-based guideline value of 10 μg/L of 1,2-dichloroethane is recommended by the WHO (666) for drinking water based on a daily per capita consumption of two liters of water.

The current OSHA occupational exposure standard for 1,2-dichloroethane is 1 ppm (4 mg/m$^3$) averaged over an 8-hour work-shift (3539). The OSHA short-term exposure limit is 2 ppm (8 mg/m$^3$) for a 15-min period (3539). These changes are in conformity with the recommendations made by NIOSH (3503). The current ACGIH recommended threshold limit value for 1,2-dichloroethane is 10 ppm (3005).

### 9.3.4 Hazard Assessment

In long-term animal studies, 1,2-dichloroethane was found to be tumorigenic when administered by gavage (138) or by repeated skin applications (142), but nontumorigenic after chronic inhalation exposures (137). Explanations for these differing results have included: 1) the possible present of impurities in the test chemicals; 2) strain differences in test animals; 3) contaminants in the diet and promoter effect of corn oil in the gavage exposures; 4) contaminants in the air from other chemicals being tested nearby; and 5) pharmacokinetic differences between oral and inhalation routes of exposure (135).

IARC (25) lists 1,2-dichloroethane as a category 2B carcinogen (i.e., sufficient evidence of animal carcinogenicity, inadequate evidence for human carcinogenicity). In EPA's weight-of-evidence classification, 1,2-dichloroethane is placed in category B2, probable human carcinogen (3744). Based on the data derived from the NCI study, EPA calculated an upper limit incremental unit cancer risk of 0.091 $(mg/kg/day)^{-1}$ for 1,2-dichloroethane (3738).

Reproduction studies with 1,2-dichloroethane have produced conflicting results. The compound has been shown to be mutagenic in the mouse spot bioassay, in Drosophila and in several bacterial assays.

In humans, acute ingestion or inhalation exposure to 1,2-dichloroethane results in symptoms of CNS depression, gastrointestinal upset and systemic injury to the liver, kidneys and lungs. A number of fatal poisonings by ingestion of as little as 15 mL are documented for this compound.

Long-term occupational exposures have been associated with weakness, loss of appetite, nausea, vomiting, epigastric pain, irritability and liver and kidney impairment.

## 9.4 SAMPLING ANALYSIS AND CONSIDERATIONS

Determination of 1,2-dichloroethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of 1,2-dichloroethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of 1,2-dichloroethane, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the 1,2-dichloroethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the 1,2-dichloroethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; 1,2-dichloroethane is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photoionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for 1,2-dichloroethane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical 1,2-dichloroethane detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.03 µg/L (Method 601) | 0.3 µg/kg (Method 8010) |
| 2.8 µg/L (Method 624) | 5 µg/kg (Method 8240) |
| 10 µg/L (Method 1624) | |
| 5 µg/L (Method 8240) | |
| 0.3 µg/L (Method 8010) | |

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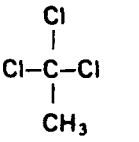
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| COMMON SYNONYMS: | CAS REG.NO.: 71-55-6 FORMULA: $C_2H_3Cl_3$<br>NIOSH Number: KJ2975000<br>STRUCTURE:  | AIR W/V CONVERSION FACTOR at 25 °C (12)<br>5.46 mg/m³ ≈ 1 ppm;<br>0.183 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 133.42 |
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1,1,1-Trichloroethane
Alpha-trichloroethane
Chloroethene
Methyl chloroform
Trichloroethane

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| REACTIVITY | Reactions of halogenated organic materials such as 1,1,1-trichloroethane with cyanides, amines, azo compounds, hydrazines, caustics, or nitrides, or with mercaptans or other organic sulfides, commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases, and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505, 3133). |

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| PHYSICO-CHEMICAL DATA | • Physical State: Liquid | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Ethereal | (21) |
| | • Odor Threshold: 120.000 ppm | (384) |
| | • Density: 1.3250 g/mL (at 20°C) | (21) |
| | • Freeze/Melt Point: -33.00°C | (21) |
| | • Boiling Point: 74.00°C | (21) |
| | • Flash Point: None | (23) |
| | • Flammable Limits*: 7.00 to 16.00% | (12,60,504) |
| | • Autoignition Temp.*: 500.0 to 537.0°C | (60,504) |
| | • Vapor Pressure: 1.00E+02 mm Hg (at 20°C) | (38) |
| | • Satd. Conc. in Air: 7.2600E+05 mg/m³ (at 20°C) | (67) |
| | • Solubility in Water: 9.50E+02 mg/L (at 20°C) | (21) |
| | • Viscosity: 0.858 cp (at 20°C) | (21) |
| | • Surface Tension: 2.5540E+01 dyne/cm (at 20°C) | (21) |
| | • Log (Octanol-Water Partition Coeff.): 2.49 | (29) |

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| PHYSICO-CHEMICAL DATA (Cont.) | • Soil Adsorp. Coeff.: 1.52E+02 (33)<br>• Henry's Law Const.: 2.76E-02 (74)<br>atm · $m^3$/mol (at 25°C)<br>• Bioconc. Factor: 5.60 (bluegills), (123,659,<br>8.9 (bluegills): 15 (estim) 3155)<br><br>• A considerable amount of energy is required for ignition. The substance will not sustain combustion. (12) |

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| PERSISTENCE IN THE SOIL-WATER SYSTEM | 1,1,1-Trichloroethane is expected to be fairly mobile in the soil/ground-water system, particularly in soils of low organic carbon where adsorption is low. Volatilization is an important removal process for near-surface 1,1,1-trichloroethane. Biodegradation in natural soils is not expected to be significant. |

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| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of 1,1,1-trichloroethane to groundwater drinking water supplies. This compound has been commonly detected in ground water at NPL sites as well as in drinking water surveys, illustrating the importance of this pathway. Inhalation from surface soils may also be important. |

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| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (38, 54)<br>1,1,1-Trichloroethane vapor is narcotic. Acute exposure leads to dizziness, drowsiness, lack of coordination, increased reaction time and irregular heart beat. Both liquid and vapor are irritating to the eyes. Skin contact may produce dermatitis.<br><br>Acute Toxicity Studies: (3504)<br><br>INHALATION:<br>$LC_{Lo}$ 11000 ppm · 2 hr Mouse<br>$TC_{Lo}$ 200 ppm · 4 hr Human<br>$TC_{Lo}$ 350 ppm Human |

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| HEALTH HAZARD DATA (Cont.) | ORAL:<br>$LD_{50}$ 10300 mg/kg Rat<br>$TD_{Lo}$ 670 mg/kg Human<br><br>SKIN:<br>$LD_{Lo}$ 1000 mg/kg Rabbit<br><br>Long-Term Effects: Liver toxicity<br>Pregnancy/Neonate Data: Negative<br>Genotoxicity Data: Conflicting data<br>Carcinogenicity Classification:<br>IARC - None assigned<br>NTP - Inconclusive<br>EPA - Group D (not classifiable as to human carcinogenicity) |

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| HANDLING PRECAUTIONS (3306) | Handle chemical only with adequate ventilation. • Vapor concentrations of 350-500 ppm: any supplied air respirator, self-contained breathing apparatus or chemical cartridge respirator with organic vapor cartridge. • 500- 1000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece. • Chemical goggles if there is probability of eye contact with liquid. • Impervious protective clothing and gloves (it attacks natural rubber) to prevent repeated or prolonged skin contact. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 350 ppm; STEL (15-min): 450 ppm
- AFOSH PEL (8-hr TWA): 350 ppm; STEL (15-min): 450 ppm

Criteria
- NIOSH CL (15 min): 350 ppm; Action level set at 200 ppm TWA
- NIOSH IDLH (30-min): 1000 ppm
- ACGIH TLV® (8-hr TWA): 350 ppm
- ACGIH STEL (15 min): 450 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards (3742)
MCLG: 200 µg/L
MCL : 200 µg/L

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

EPA Health Advisories and Cancer Risk Levels (3977)
The EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- 1-day (child): 100 mg/L
- 10-day (child): 40 mg/L
- longer-term (child): 40 mg/L
- longer-term (adult): 100 mg/L
- lifetime (adult): 0.2 mg/L

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria

- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms, 18.4 mg/L.
  - Based on ingestion of contaminated aquatic organisms only, 1.03 g/L.

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 18,000 µg/L trichloroethane.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 31,200 µg/L trichloroethane.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:
ORAL: 9.000E-02 mg/kg/day (3744)

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Program

Clean Water Act (CWA)

1,1,1-Trichloroethane is listed as a toxic pollutant, subject to pretreatment regulations for new and existing sources, and to effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), metal finishing (3768), and metal molding and casting (892). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

1,1,1-Trichloroethane is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). Under the National Primary Drinking Water Regulations, the MCL and MCLG for 1,1,1-trichloroethane is set at 0.20 mg/L for human health (3773, 3772). In states with an approved Underground Injection Control program, a permit is required for the injection of 1,1,1-trichloroethane containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

1,1,1-Trichloroethane is identified as a toxic hazardous waste (U226) and listed as a hazardous waste constituent (3783, 3789). Non-specific sources of 1,1,1-trichloroethane-containing waste are solvent use (or recovery) activities, chlorinated aliphatic hydrocarbon production, and spent solvents containing 10% or more 1,1,1-trichloroethane (325, 3765). Waste streams from the following industries contain 1,1,1-trichloroethane and are listed as specific sources of hazardous waste: organic chemicals (production of chloroethane, 1,2-dichloroethane, vinyl chloride and 1,1,1-trichloroethane), inorganic chemicals (chlorine production), and ink formulation (3774, 3765). 1,1,1-Trichloroethane is subject to land disposal restrictions when its concentration as a hazardous constituent of certain wastewaters exceeds designated levels (3785). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for

land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). 1,1,1-Trichloroethane is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Toxic Substances Control Act (TSCA)
Manufacturers, processors or distributors of 1,1,1-trichloroethane must report production, usage and disposal information to EPA. They, as well as others who possess health and safety studies on 1,1,1-trichloroethane, must submit them to EPA (334, 3789). Manufacturers and/or processors of 1,1,1-trichloroethane are required to sponsor tests for developmental toxicity, using the test protocol and schedule as submitted by an industry consortium and approved by EPA (3760).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
1,1,1-Trichloroethane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing 1,1,1-trichloroethane but these depend upon the concentrations of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers and users of 1,1,1-trichloroethane must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
1,1,1-Trichloroethane is exempt from a tolerance requirement when used as a solvent or cosolvent in pesticide formulations applied to growing crops or to raw agricultural commodities after harvest. It is also exempt when used as a solvent at a level no greater than 25% in pesticide formulation applied to animals (315) or when used in the post-harvest fumigation of citrus fruits (314).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to 1,1,1-trichloroethane in any 8-hour work-shift of a 40-hour work-week shall not exceed an 8-hour time-weighted average (TWA) of 350 ppm. An employee's 15-minute short-term exposure limit (STEL) of 450 ppm shall not be exceeded at any time during the work-day (3539).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated 1,1,1-trichloroethane as a hazardous material with a reportable quantity of 454 kg, subject to requirements for packaging, labeling and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
1,1,1-Trichloroethane is approved for use as an indirect food additive as a component of adhesives (3209). Any aerosol drug product containing 1,1,1-trichloroethane is a new drug and requires a new drug application (NDA) before it can be marketed (367).

- State Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. Other states have promulgated additional or more stringent criteria.

ALABAMA
Alabama requires that the annual average maximum contaminant level of 1,1,1-trichloroethane in drinking water not exceed 0.2 mg/L in all community water systems, and non-community non-transient water systems (3015).

CALIFORNIA
California has an MCL and an action level of 200 μg/L (ppb) for drinking water (3096, 3098).

CONNECTICUT
Connecticut has a quantification limit of 2.0 μg/L for drinking water (3137).

MISSOURI
Missouri has a water quality criterion of 200 μg/L for drinking water supply waters (3457).

NEW JERSEY
New Jersey has set a surface water quality criterion of 26 μg/L (ppb) for FW2 class surface waters, and an MCL of 26 μg/L (ppb) for drinking water (3496, 3497).

NEW MEXICO
New Mexico has set a human health standard of 0.06 mg/L for ground-water (3499).

NEW YORK
New York has set a maximum contaminant level of 5 μg/L for drinking water (3501).

OKLAHOMA
Oklahoma has a water quality criterion of 0.3 μg/L for ground-water (3534).

PENNSYLVANIA
Pennsylvania has set a human health criterion of 1 mg/L for surface waters (3561).

VERMONT
Vermont has a preventive action limit of 100 μg/L and an enforcement standard of 200 μg/L for ground-water (3682).

WISCONSIN
Wisconsin has a preventive action limit of 40 μg/L and an enforcement standard of 200 μg/L for ground-water (3840). Wisconsin has also set a human threshold criterion of 0.2 mg/L for surface waters for the public water supply (3842).

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed that solid wastes be listed as hazardous because they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is equal to or greater than 30 mg/L of 1,1,1-trichloroethane. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Programs

  MOST STATES

  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA

  Minnesota has proposed a Sensitive Acute Limit (SAL) of 6222 μg/L for designated surface waters, and chronic criteria of 138 μg/L for designated surface waters and 200 μg/L for designated ground-waters for the protection of human health (3452).

  WEST VIRGINIA

  West Virginia has proposed a water quality criterion of 1.2 mg/L for Public A surface waters. Final action is expected in late spring 1989 (3835).

EEC Directives

Directive on Ground-Water (538)

Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)

The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)

1,1,1-Trichloroethane is listed as a Class II/c harmful substance and is subject to packaging and labeling regulations.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, mature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,1,1-Trichloroethane is classified as a harmful substance and is subject to packaging and labeling regulations. 1,1,1-Trichloroethane may contain a stabilizer. If the stablizer changes the dangerous properties of this substance should be labeled in accordance to rules in Annex I and EEC/88/490, 22 July 1988.

EEC Directives--Proposed Resolution
Resolution on the Revised List of Second-Category Pollutants (545)
1,1,1-Trichloroethane is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 10.1 MAJOR USES

The 1,1,1-isomer of trichloroethane is widely used as a cleaning solvent because of its nonflammability and solvency properties. As of 1985, approximately 28% of the total production was used in vapor degreasing and 41% in cold cleaning (3844). Because of its reactivity with magnesium, aluminum and their alloys, inhibitors are usually added to increase its stability. Common solvent uses include cleaning of electrical equipment, motors, electronic components and instruments, missile hardware, photographic film, printed circuit boards, upholstery and various metal and plastic parts during manufacture. It is also used as a solvent for adhesives and coatings, photoresist polymers, textile dyes, as a coolant and lubricant in metal cutting oils, as a component in inks and drain cleaners, and as a chemical intermediate in the production of vinylidene chloride. It has a minor use in aerosols where it acts both as a vapor pressure depressant and as a solvent and carrier for the active ingredients (25, 21).

## 10.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 10.2.1 Transport in Soil/Ground-water Systems

#### 10.2.1.1 Overview

The 1,1,1-isomer of trichloroethane may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways for low soil concentrations can be assessed by estimating equilibrium partitioning, as shown in Table 10-1. These calculations predict the partitioning of 1,1,1-trichloroethane among soil particles, soil water and soil air. Portions of 1,1,1-trichloroethane associated with the water and air phases of the soil have higher mobility than the adsorbed portion.

For unsaturated topsoil, the model predicts that 3% percent of the 1,1,1-trichloroethane will be partitioned in the soil-water phase, and thus be available to migrate through this phase by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. A larger portion (10%) is expected to be in the gaseous phase of the soil. Diffusion through the soil-air pores up to the ground surface, and subsequent evaporation by wind, may be a significant loss pathway.

TABLE 10-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR
1,1,1-TRICHLOROETHANE IN MODEL ENVIRONMENTS[a]

| Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil at 25°C[b,c] | 86.7 | 3.0 | 10.3 |
| Saturated deep soil[d] | 39.0 | 61.0 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Use estimated soil sorption coefficient: $K_{oc}$ = 152 (33).
c) Henry's law constant = 2.76E-02 atm · $m^3$/mol at 25°C (74).
d) Used sorption coefficient (Kp) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the 1,1,1-trichloroethane (61%) is expected to be in the soil-water phase (Table 10-1) and transported with flowing ground-water. Sorption onto deep soils is lower than onto top soils, but may have some effect on mobility. Overall, ground-water underlying 1,1,1-trichloroethane-contaminated soils with low organic content are expected to be vulnerable to pollution.

Soil/water partition coefficients ($K_d$) for 1,1,1-trichloroethane were calculated by Sims et al. (3656). For a McLaurin soil the $K_d$ was 1.35 (log $K_d$ = 0.13), and for a Kidman soil the $K_d$ was 2.95 (log $K_d$ = 0.47).

### 10.2.1.2 Sorption on Soils

The mobility of 1,1,1-trichloroethane in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. The soil absorption coefficient ($K_{oc}$) for this compound was reported to be 152 (33). In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water;
- decrease moderately with increasing dissolved organic matter content of the soil water.

Schwarzenbach et al. (77) determined 1,1,1-trichloroethane retardation rates, which represent interstitial water velocity/velocity of 1,1,1-trichloroethane, for soils of varying organic carbon content (Table 10-2).

TABLE 10-2
RETARDATION FACTORS FOR 1,1,1-TRICHLOROETHANE
IN RIVER SEDIMENTS AND SOILS

| Matrix | Retardation Factor |
|---|---|
| River sediment (1-2% organic carbon) | 3.4 - 11 |
| Aquifer close to river bed (0.1-1% organic carbon) | 1.2 - 6 |
| Aquifer far from river bed (<0.1% organic carbon) | 1 - 1.2 |

SOURCE: Schwarzenbach et al. (77).

The retardation factors presented in Table 10-2 indicate that there is some sorption and a decrease in mobility of 1,1,1-trichloroethane in soils containing 1-2% organic carbon; sorption is lower in soils having 0.1-1% organic carbon. The retardation factors for soils having less than 0.1% organic carbon suggest little or no retention.

Comparative information on the soil absorption of 1,1,1-trichloroethane can be derived from studies on 1,1,2-trichloroethane. Wilson et al. (82) investigated the transport and fate of the latter isomer in a soil column containing sandy soil. When a 0.16 mg/L solution of 1,1,2-trichloroethane was applied to the column, approximately 65% percolated through the column with minimal retardation; with a 1 mg/L solution, about 61% percolated through the column. Sorption rates for 1,1,1-trichloromethane could be expected to be slightly higher than those for 1,1,2-trichloroethane due to higher values for $K_{oc}$ and Henry's law constant.

### 10.2.1.3 Volatilization from Soils

Transport of 1,1,1-trichloroethane vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. Sims et al. (3656) determined volatilization rates of 1,1,1-trichloroethane for McLaurin soil and found that 64.6% was volatilized over a 50-hr incubation period. The mean half-life was reported to be 0.15 days.

In general, important soil and environmental parameters influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31). No information was found concerning the vapor-soil sorption coefficient or the vapor phase diffusion coefficient for 1,1,1-trichloroethane. The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. The temperature dependence of H for 1,1,1-trichloroethane has been measured by Gossett and Lincoff (18), and is described by the following equation:

$$H(atm \cdot m^3/mol) = \exp[9.975\text{-}4186/T\ (°K)]$$

Gossett and Lincoff (18) have also examined the effect of other dissolved materials on the volatilization of 1,1,1-trichloroethane. Moderate increases in H were observed with increasing salinity and the presence of other organic compounds. These results suggest that the presence of other materials may significantly affect the volatilization of 1,1,1-trichloroethane from surface soils.

Comparative information on volatilization of 1,1,1-trichloroethane can be obtained from studies of the 1,1,2-trichloroethane isomer. Wilson et al. (82) investigated the transport and fate of the latter isomer in a soil column containing a sandy soil. When a solution containing 0.16 mg/L of 1,1,2-trichloroethane was applied to the column, approximately 27% was volatilized. A 1 mg/L solution resulted in about 47% being volatilized. Volatilization rates for 1,1,1-trichloroethane are probably slightly higher than those for 1,1,2-trichloroethane considering the higher $K_{oc}$ and Henry's law constant.

Soil volatilization rates of chlorinated ethanes have been reported to be about one order of magnitude slower than volatilization rates for well-stirred aqueous solutions (82). The half-life for the volatilization of 1,1,1-trichloroethane from a stirred aqueous solution has reported to be in the range of 20-90 minutes depending on the degree of agitation (10, 3175).

### 10.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of 1,1,1-trichloroethane in soil/ground-water systems is not well documented. In most cases, it should be assumed that 1,1,1-trichloroethane will persist for months to years (or more). The 1,1,1-trichloroethane isomer which has

been released into the air will eventually undergo photochemical oxidation; tropospheric lifetimes of 1.1 to 8 years have been calculated (10, 3300).

Under normal environmental conditions, 1,1,1-trichloroethane does not undergo hydrolysis at a sufficiently rapid rate to compete with volatilization. Callahan et al. (10) reported data estimating the hydrolysis half-life for 1,1,1-trichloroethane in water to be 6 months.

There is limited information concerning the biodegradation of 1,1,1-trichloroethane under natural conditions. Wilson et al. (82) and Pearson and McConnell (75) observed little evidence of biological transformation of 1,1,1-trichloroethane. However, Parsons et al. (3551) reported considerable degradation to vinylidene chloride within 2 weeks in a microcosm study using aquifer water and Everglades sediments. In addition, McCarty et al. (3436) obtained field evidence of biodegradation in an aquifer injected with a reclaimed ground water containing the compound. The half-life was estimated to be 231 days.

Evidence of the biodegradation of 1,1,1-trichloroethane was observed by Tabak et al. (79, 3697) with an acclimated microbial population. Thom and Agg (80) included 1,1,1-trichloroethane on a list of organic chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In most soil/ground-water systems, however, the concentration of microorganisms capable of biodegrading chemicals such as 1,1,1-trichloroethane is very low and drops off sharply with increasing depth. Thus, biodegradation in the soil/ground-water system should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

### 10.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The properties of 1,1,1-trichloroethane and the above discussion of fate pathways suggest that 1,1,1-trichloroethane is highly volatile in aqueous solutions, moderately adsorbed and has a low potential for bioaccumulation. This compound may volatilize from soil surfaces, but that portion not removed by volatilization will be somewhat mobile in ground-water. These fate characteristics suggest several potential exposure pathways.

Volatilization of 1,1,1-trichloroethane from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. In addition, the potential for ground-water contamination is high, particularly in sandy soils. Mitre (83) reported that 1,1,1-trichloroethane has been found at 54 of the 546 National Priority List (NPL) sites. It was detected in ground-water samples taken at 45 sites, in surface water samples at 19 sites and in air samples taken at 3 sites.

This compound has also been reported in various surveys of drinking water, some of which were ground-water supplies as shown below:

| Survey | No. Sampled | No. Positive | Range of Positives |
|---|---|---|---|
| State Data | 3,333 | 715 | Trace - 2,250 µg/L |
| NOMS | 113 | 19 | 0.2 - 1.3 µg/L |
| NSP | 142 | 32 | Trace - 21 µg/L |
| CWSS | 452 | 19 | 0.5 - 650 µg/L |
| GWSS (random data) | 466 | 27 | 0.2 - 18 µg/L |

The state data include only ground-water sources and were compiled from various state reports on local contamination problems. The state data are not considered to be statistically representative of national occurrence. The National Organics Monitoring Survey (NOMS) included data from both ground- and surface water supplies, as did the National Screening Program (NSP) and the Community Water Supply Study (CWSS). The 1982 Ground-water Supply Survey (GWSS) is the most recent study (731). This survey sampled a total of almost 1000 drinking water systems using ground-water; 466 selected at random, and about 500 selected by the state as potentially contaminated. The random results suggest that 1,1,1-trichloroethane is commonly found in drinking water, particularly in ground-water as evidenced by the state reports of contamination problems. The USEPA (64) estimates that 0.3% of the nation's ground-water supplies are contaminated with 1,1,1-trichloroethane (≥10 µg/L). In EPA's Total Exposure Assessment Methodology (TEAM) study, the mean concentration of 1,1,1-trichloroethane in drinking water was 0.2 to 0.6 ppb in samples taken in an urban industrial area, 0.03 ppb in an urban area without a petrochemical industry, and 0.04 ppb in an agricultural area (3826).

These survey data indicate that 1,1,1-trichloroethane is mobile in ground water and this mobility has resulted in exposure through drinking water. The subsequent contamination of surface waters may result in a number of exposure pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion exposure;
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposures through bioaccumulation.
- Recreational use of these waters may result in dermal exposures.
- Domestic animals may consume or be dermally exposed to contaminated ground- or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground water for two reasons. First, the Henry's law constant for 1,1,1-trichloroethane suggests that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this

compound is low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 10.2.4 Other Sources of Exposure

The data presented above from various drinking water surveys suggest that 1,1,1-trichloroethane is found in both ground water and surface waters used as drinking water supplies. The volatility of this compound suggests that it may be found in air as well. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For 1,1,1-trichloroethane, they had data for 2673 locations. In rural and remote areas, the median concentration was 0.6 $\mu g/m^3$; in urban and suburban, the median concentration was 2.8 $\mu g/m^3$; and in source-dominated areas, the median concentration was 6.5 $\mu g/m^3$. These data show that persons even in rural and remote areas may be exposed to this compound through inhalation. Wallace et al. (3826) reported median seasonal air concentrations of 1.4 to 5.1 $\mu g/m^3$ for an urban industrial area. The air concentration in a rural area was 0.05 $\mu g/m^3$.

1,1,1-Trichloroethane has been found in a variety of foods including oils and fats (5-10 $\mu g/kg$), fruits and vegetables (1-4 $\mu g/kg$), meats, tea, bread and other grain-based products (0.78-7 $\mu g/kg$), as well as in fish, shellfish, seabirds, and marine invertebrates (0-310 $\mu g/kg$) (224, 3212, 3559, 3172). These data suggest that people may be exposed to this compound in the diet.

## 10.3 HUMAN HEALTH CONSIDERATIONS

### 10.3.1 Animal Studies

#### 10.3.1.1 Carcinogenicity

The carcinogenicity of 1,1,1-trichloroethane has been evaluated in several animal studies (393, 660, 661, 662, 3578). In three of these studies deficiencies in the experimental protocol (e.g., inadequate exposure period) or excessive mortality of the test animals rendered the data of limited value in assessing human cancer risk (3737). A fourth study involved a retesting of the compound by the National Toxicology Program. The test chemical had a purity of 99.7% and contained 0.1% butylene oxide as a stabilizer. It was administered in corn oil by gavage to male and female F344/N rats at doses of 375 and 750 mg/kg. Male and female $B6C3F_1$ mice received 1500 or 3000 mg/kg. The animals were dosed 5 times per week for 103 weeks. No treatment-related tumors were observed in male rats. The study was inadequate for evaluation of female rats because the high dose was toxic and there was a large number of accidental deaths. In mice, the association between an increased incidence of hepatocellular carcinoma and exposure to 1,1,1-trichloroethane was considered equivocal in males. Although the compound caused an increased incidence of hepatocellular carcinoma in females, the significance of these results was unclear.

A more recent study evaluating the oncogenicity of 1,1,1-trichloroethane was conducted by Quast et al. (3578). In this study Fischer 344 rats and B6C3F$_1$ mice (80/sex/group) were exposed to 1,1,1-trichloroethane vapor concentrations of 0, 150, 500, or 1500 ppm for 6 hr/day, 5 days/week, for 2 years. There were no indications of any oncogenic effect in either species at any tested exposure level.

There is evidence that 1,1,1-trichloroethane can cause cell transformations in vitro. Price et al. (3572) tested the compound on Fischer rat embryo cell cultures, and observed transformed cells. These cells, when injected in newborn rats, induced the formation of fibrosarcomas. Tu et al. (3732) reported a clear positive transformation response when 1,1,1-trichloroethane was tested on BALB/c-3T3 mouse cells in the absence of any exogenous metabolic activation system.

### 10.3.1.2 Genotoxicity

Conflicting data concerning the genotoxity of the 1,1,1-isomer of trichloroethane were found in the literature. It is possible that the stabilizers or unknown impurities normally found with this compound were responsible for the reported genotoxic effects (3644). In one desiccator assay in which four strains of Salmonella typhimurium were tested in the presence or absence of an exogenous metabolic activation system, 1,1,1-trichloroethane gave negative results (3450). However, using the same type of assay, Gocke et al. (3246) reported positive responses, with or without metabolic activation, in two of five strains of Salmonella. Similarly, Shimada et al. (3644), using gas exposure chambers, found that 99.99% pure TCE induced reversions in two of the five standard strains, but at concentrations that were more than 95% toxic. Using the same lots of TCE, Shimada et al. found more revertants with exposure to commercial samples that contained "stabilizers" than with the pure samples. Shimada et al. also found no evidence of unscheduled DNA synthesis in rat hepatocytes treated in culture with TCE from the same lots as those used in the Ames assay. When Chinese hamster ovary cells were examined for SCEs and chromosome aberrations after treatment with 1,1,1-trichloroethane, a significant increase in chromosome aberrations was found without metabolic activation; with activation, results were negative. The number of sister chromatid exchanges was equivocal in tests with or without metabolic activation (3235).

1,1,1-Trichloroethane was also tested in the mouse micronucleus test by Gocke et al. (3246) who treated both sexes with 3 different concentrations. Although the number of bone marrow micronucleated cells was above the concurrent control, the increase was not significant. These same authors also found no genotoxic effects in the sex-linked recessive lethal test when Drosophila males were fed a dose close to the $LD_{50}$. When tested in a dominant lethal mouse assay, 1,1,1-trichloroethane gave a negative response at a daily oral dose as high as 1,000 mg/kg (3388).

### 10.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No embryo lethality, fetal toxicity or teratogenicity was found when rats and mice were exposed to 1,1,1-trichloroethane at vapor concentrations of 875 ppm seven hours daily on days 6 through 15 of gestation (115). Inhalation was also the route of exposure used by York (3857). Rats were exposed during gestation to 2,100 ppm 6 hr/day, resulting in some reduction in fetal weight and delay in osseous development but no terata. Postnatal studies of growth, survival, and neurobehavioral development revealed no differences from controls. Exposure of females to 1,1,1-trichloroethane only during the two weeks prior to mating had no significant effect on fertility, litter size, or fetal weight.

No dose-dependent effects on fertility, gestation or viability were observed in male and female mice that received 1,1,1-trichloroethane in drinking water at concentrations of 0, 0.58, 1.75, or 5.83 mg/mL/day for 25 weeks. These concentrations were designed to yield daily doses of 0, 100, 300 or 1000 mg/kg. Trichloroethane exposure also failed to produce significant dominant lethal mutations or teratogenic effects (386).

### 10.3.1.4 Other Toxicologic Effects

#### 10.3.1.4.1 Short-term Toxicity

Results of animal experimentation show that 1,1,1-trichloroethane is relatively nontoxic upon short-term exposure (43). In tests on dogs, Kobayashi et al. (3368) found that exposure to 13,200 ppm increased heart rate, but 27,900 ppm decreased the heart rate. Adams et al. (127) exposed several species of animals to 1,1,1-trichloroethane for 7 hours daily, 5 days per week, for 1 to 3 months. At 10,000 ppm, rats exhibited irregular respiration and became semiconsciousness after 3 hours. In those that died, death appeared to be due to cardiac or respiratory failure. At 5000 ppm, there was a mild narcotic effect within 1 hour. Rats survived for 31 exposures at this level without apparent injury. Rabbits at this level showed slight growth retardation. At 3000 ppm, rabbits and monkeys showed no response over a 2 month period. McNutt et al. (3442) exposed CF-1 mice to 1,1,1-trichloroethane concentrations of 250 and 1000 ppm continuously for up to 14 weeks. In the high dose animals cytoplasmic alterations were observed in the centrilobular hepatocytes upon electron microscopic evaluation. Necrosis of hepatocytes occurred in 40% of the high dose group after 12 weeks of exposure. Moderate liver triglyceride accumulation was evident in this group. "Mild to minimal" cytoplasmic alterations were also seen in the low dose group. Rats, dogs and monkeys were exposed to these same concentrations. Exposure-related effects were not demonstrated in these species.

#### 10.3.1.4.2 Chronic Toxicity

MacEwen and Vernot (395) reported no adverse effects in monkeys and dogs exposed to 250 or 1000 ppm continuously by inhalation for 90 or 100 days. Mice and rats exposed to the higher dosage level exhibited increased liver weights as well as cellular changes of the liver. No liver or kidney effects were observed in rats, rabbits, guinea pigs or monkeys exposed to 500 ppm, 7 hours per day, 5 days a week for 130 exposures (396).

Karlsson et al. (3347) reported that adult Mongolian gerbils exposed to 70 ppm 1,1,1-trichloroethane for three months had significantly reduced levels of DNA in three regions of the brain.

Quast et al. (3578) conducted a chronic inhalation toxicity study on Fischer 344 rats and B6C3F$_1$ mice in which the test animals (80/sex/group) were exposed to 0, 150, 500, or 1500 ppm 1,1,1-trichloroethane 6 hr/day, 5 days/week, for 2 years. No toxic effects were observed in the mice. At the exposure level of 1500 ppm, female rats exhibited a significant decrease in body weight and both male and female rats developed slight microscopic changes in the liver. No effects were seen at the lower exposure levels.

### 10.3.2 Human and Epidemiologic Studies

#### 10.3.2.1 Short-term Toxicologic Effects

Depression of the central nervous system is the primary toxic effect in humans who have been subjected to short-term, high-level inhalation exposure to trichloroethane. Exposure to vapor concentrations in excess of 1000 ppm for 15 minutes or 2000 ppm for 5 minutes causes disequilibrium in adults (38). Changes in reaction time, manual dexterity, and equilibrium occur following exposure to 350 ppm for 1-3 hr (129, 3741). Inhalation of 450 ppm for 8 hr caused eye, nose and throat irritation and impaired perceptive capabilities under stress conditions (3741). Deficits in psychomotor performance have occurred at exposures to 175 or 350 ppm; the effects, in some cases could be observed within 20 min from the start of the exposures (3417). Acute inhalation exposures can also adversely effect the cardiovascular system. Dornette and Jones (128) reported hypotension, premature ventricular contractions and, in one case, cardiac arrest in patients undergoing surgical anesthesia and exposed to 10,000 to 26,000 ppm 1.1.1-trichloroethane. No respiratory depression or hepatotoxicity was seen in any of the patients.

Numerous deaths have been attributed to deliberate or occupational inhalation exposure to trichloroethane. In the majority of human fatalities, death results from CNS depression, edema and pulmonary congestion. However, even in cases of acute, high-level inhalation exposures, 1,1,1-trichloroethane possesses a limited capacity to exert hepatic injury (43).

There is one account of accidental ingestion of 30 mL by a 47-year-old male. The initial symptoms were CNS depression and gastrointestinal upset. Clinical tests revealed only minimal evidence of hepatorenal injury. The patient recovered within 2 weeks (131).

NIOSH cites numerous instances of inhalation exposures to 1,1,1-trichloroethane. Several deaths have been reported; in most cases, the exposure levels have been estimated to be several thousand ppm (394). Trichloroethane is irritating to the skin. Various studies have shown the liquid to be absorbed through intact skin in moderate amounts. Vapors, however, are not absorbed by this route in toxicologically significant quantities (394).

Vapor concentrations of 500 to 1000 ppm cause minor, transient eye irritation in humans. The effect of splash contact is similar to that of chloroform. If the corneal epithelium is injured, it will regenerate within 3 days (19).

### 10.3.2.2 Chronic Toxicologic Effects

The most extensive study of industrially-exposed workers has been reported by Kramer and associates (130) who conducted an epidemiological study of 151 subjects and 151 matched controls with emphasis placed on cardiovascular and hepatic effects. Employees in the study population had solvent exposures ranging from several months up to 6 years. Vapor concentrations ranged from 1 to 250 ppm. Statistical analysis of the data did not reveal any clinically pertinent findings. In addition, Maroni et al. (398) reported no signs attributed to nervous system impairment in 22 factory workers exposed to vapor concentrations between 100 and 1000 ppm for up to 6 years.

During 1980 and 1981, an exceptionally high number of birth defects and miscarriages occurred in Los Paseos, California, an area serviced by a water well which had been contaminated by 1,1,1-trichloroethane and dichloroethylene (3606). The chemicals had leaked from storage tanks of a semiconductor manufacturer. Later, a 1,1,1-trichloroethane concentration of 8,800 ppb was measured in the well water. Because of too many uncertainties in the retrospective epidemiological studies, no clear association could be made between the unfavorable reproductive outcomes and the solvents in the drinking water.

### 10.3.3 Levels of Concern

EPA has established ambient water quality criteria for 1,1,1-trichloroethane (3744). For the protection of human health, the criterion is set at 18.4 mg/L for exposures through consumption of water and fish, and at 1.03 g/L for exposure through consumption of fish alone. Ambient water quality criteria for the protection of aquatic life have not been set by EPA; however, the lowest effect level for acute toxic effects in freshwater organisms has been reported to be 1.8E+04 $\mu$g/L, and that for marine organisms has been reported to be 3.12E+04 $\mu$g/L (355).

EPA has set the maximum contaminant level goal (MCLG) for 1,1,1-trichloroethane in drinking water at 200 μg/L and the maximum contaminant level (MCL) at 200 μg/L (3742). Health Advisories (HA) for a 10-kg child have been set at 100 mg/L for a 1-day exposure, 40 mg/L for a 10-day exposure, and 40 mg/L for longer term exposures. The longer term HA for a 70-kg adult has been set at 100 mg/L, the Drinking Water Equivalent Level (DWEL) at 1 mg/L, and the lifetime HA at 200 μg/L (3805). The DWEL value was derived from data for liver toxicity in mice exposed to 1,1,1-trichloroethane vapors.

EPA has calculated an oral Reference Dose (RfD) of 9E-02 mg/kg/day for 1,1,1-trichloroethane based on a 6-month inhalation study in guinea pigs (3977). EPA has also calculated Acceptable Intake values for subchronic (AIS) and chronic (AIC) exposures (3741). For oral exposures an AIC of 37.5 mg/day was calculated, and for inhalation exposures, an AIS of 756 mg/day and an AIC of 442 mg/day were calculated.

The OSHA permissible exposure limit for 1,1,1-trichloroethane is 350 ppm averaged over an 8-hour work-shift (3539). The OSHA short-term exposure limit (STEL) is 450 ppm (for 15-min exposures). The NIOSH recommended exposure limit, as of 1988, is 350 ppm for a Ceiling Limit. A TWA action level of 200 ppm is also recommended by NIOSH (3503). The ACGIH recommended threshold limit value, as of 1988, was 350 ppm for 8-hr TWA, and 450 ppm for a 15-min STEL (3005).

### 10.3.4 Hazard Assessment

Although inhalation exposure is most common, percutaneous absorption of both liquid and vapor 1,1,1-trichloroethane, as well as exposure via ingestion, have been demonstrated in humans. Accidental ingestion of 30 mL (approx 40 g) of 1,1,1-trichloroethane by an human adult was survived (131). Oral $LD_{50}$ values in experimental animals range from 500 to 12,000 mg/kg (21). The primary effects of low level inhalation exposures to 1,1,1-trichloroethane (≤1000 ppm ≤1 hr) are psychophysiological, including dose-related impairment of perception and coordination. The effects of chronic low-level exposures are not known.

Mixed carcinogenic results were observed in the recent NTP study (393) and negative results in a recent Dow Chemical study (3578). In the NTP study, the compound was found to be carcinogenic in female mice, causing an increased incidence of hepatocellular carcinomas. The evidence for carcinogenicity in male mice was equivocal. No increased incidence of tumors was observed in rats; however, the compound may not have been adequately studied in female rats due to the high mortality in this group.

Genotoxicity studies have revealed conflicting results. A weak mutagenic response was reported in several, but not all, strains of Salmonella tested in the Ames assay (3644, 3450, 3246). Chromosome aberrations, but no significant increases in SCEs, were seen in Chinese hamster cells exposed to 1,1,1-trichloroethane (3235).

The compound caused a slight increase in bone marrow micronucleated cells in the mouse micronucleus test, but the increase was not statistically significant (3246). In addition, negative responses have been observed in both a dominant-lethal mouse assay (3388) and in a sex-linked recessive lethal test with Drosophila (3246).

This compound induced a clear positive response in the BALB/c-3T3 cell transformation assay (708) and in a virus-mediated cell transformation assay (3274).

No reproductive abnormalities have been seen in laboratory animals exposed to 1,1,1-trichloroethane.

In EPA's weight-of-evidence classification, 1,1,1-trichloroethane is placed in category D, not classifiable as to human carcinogenicity (3744). This classification was based on the lack of human data and the inadequacy of the available animal data. Consequently, EPA has made no quantitative estimates of unit cancer risk for this compound.

## 10.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of 1,1,1-trichloroethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of 1,1,1-trichloroethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of 1,1,1-trichloroethane, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010, and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the 1,1,1-trichloroethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the 1,1,1-trichloroethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; 1,1,1-trichloroethane is then detected with a halide specified detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by

GC with photoionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for 1,1,1-trichloroethane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

The electron capture detector has also been used to determine 1,1,1-trichloroethane in water and soil samples using solvent (3352) and headspace extraction (3595, 3844). A comparison of the two extraction methods (3595) showed better reproducibility for solvent extraction but greater sensitivity for headspace analysis.

Typical 1,1,1-trichloroethane detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.03 µg/L (Method 601) | 0.3 µg/kg (Method 8010) |
| 3.8 µg/L (Method 624) | 5 µg/kg (Method 8240) |
| 10 µg/L (Method 1624) | |
| 5 µg/L (Method 8240) | |
| 0.3 µg/L (Method 8010) | |

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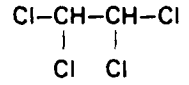
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| COMMON SYNONYMS: | CAS REG.NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25°C (12) |
|---|---|---|
| 1,1,2,2-Tetrachloroethane<br>Acetylene tetrachloride<br>Bonoform<br>Cellon<br>Dichloro-2,2-dichloroethane<br>S-Tetrachloroethane<br>Tetrachloroethane | 79-34-5 $C_2H_2Cl_4$<br>NIOSH NO:<br>KI8575000<br>STRUCTURE:<br>  | 6.86 mg/m³ ≈ 1 ppm;<br>0.1458 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT:<br>167.86 |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as 1,1,2,2-tetrachloroethane with cyanides, amines, azo compounds, hydrazines, caustics, or nitrides, or with mercaptans or other organic sulfides, commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases, and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505, 3133). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Chloroform-like | (23) |
| | • Odor Threshold: 1.500 ppm | (384) |
| | • Density: 1.6000 g/mL (at 20°C) | (14) |
| | • Freeze/Melt Point: -42.50°C | (21) |
| | • Boiling Point: 146.30°C | (21) |
| | • Flash Point: Nonflammable | (23) |
| | • Flammable Limits: Nonflammable | (60) |
| | • Autoignition Temp.: Nonflammable | (60) |
| | • Vapor Pressure: 4.90E+00 mm Hg (at 20°C) | (21)<br>(23) |
| | • Satd. Conc. in Air: 4.5000E+04 mg/m³ (at 20°C) | (1219) |
| | • Solubility in Water: 2.90E+03 mg/L (at 20°C) | (38) |
| | • Viscosity: 1.770 cp (at 20°C) | (21) |
| | • Surface Tension: 3.4720E+01 dyne/cm (at 20°C) | (21) |
| | • Log (Octanol-Water Partition Coeff.): 2.39 | (29) |
| | • Soil Adsorp. Coeff.: 2.20E+02 | (33) |
| | • Henry's Law Const.: 5.00E-04 atm · m³/mol (at 20°C) | (74) |
| | • Bioconc. Factor: 1.20E+01 (estim); 8 (bluegills) | (123,659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL-WATER SYSTEM | 1,1,2,2-Tetrachloroethane is expected to have limited mobility In surface soils with 1-2% organic carbon; mobility in deep soils is expected to be higher due to migration with the soil pore water. In the soil/groundwater system, 1,1,2,2-tetrachloroethane is expected to be persistent. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of 1,1,2,2-tetrachloroethane to groundwater drinking water supplies. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (54)<br>Ingestion and inhalation cause irritation of the nose and throat, fatigue, nausea, headache, tremors, loss of reflexes, insomnia and loss of appetite; liver dysfunction may occur with severe exposure. Prolonged skin contact with liquid may produce dermatitis due to its defatting action and may result in CNS effects. The liquid and vapor are irritating to the eyes, and may cause watering and burning.<br><br>Acute Toxicity Studies:<br><br>INHALATION:<br>$LC_{Lo}$ 1000 ppm · 4 hr — Rat (47)<br>$LC_{Lo}$ 19 gm/m$^3$ · 45 min — Cat (3504)<br>$TC_{Lo}$ 1000 mg/m$^3$ · 30 min — Human (3504)<br><br>ORAL:<br>$LD_{50}$ 250 mg/kg — Rat (25)<br>$LD_{Lo}$ 300 mg/kg — Dog (3504)<br>$TD_{Lo}$ 30 mg/kg — Human (3504)<br><br>SKIN:<br>$LD_{50}$ 6400 mg/kg — Rabbit (12) |

| | |
|---|---|
| HEALTH HAZARD DATA (Cont.) | Long-Term Effects: Liver and kidney damage<br>Pregnancy/Neonate Data: Positive teratogen<br>Genotoxicity Data: Conflicting data<br>Carcinogenicity Classification:<br>IARC - Group 3 (not classifiable as to its carcinogenicity to humans)<br>NTP - Positive evidence in mice, negative in female rats, equivocal in male rats<br>EPA - No data |

| | |
|---|---|
| HANDLING PRECAUTIONS (38,52) | Handle chemical only with adequate ventilation. • Vapor concentrations of 5-50 ppm: any supplied-air respirator, self-contained breathing apparatus or chemical cartridge respirator with an organic vapor cartridge. • 50-150 ppm: self-contained breathing apparatus or supplied-air respirator with full facepiece; gas mask with organic vapor canister. • Chemical goggles if there is probability of eye contact. • Natural rubber, neoprene, nitrile, PVC, PE gloves/apron/boots should be worn if direct contact is unavoidable. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 1 ppm (skin)
- AFOSH PEL (8-hr TWA): 1 ppm; STEL (15-min): 3 ppm (skin)

Criteria
- ACGIH TLV® (8-hr TWA): 1 ppm (skin)
- NIOSH IDLH (30-min): None - treat as a potential carcinogen
- NIOSH REL: Lowest detectable limit

WATER EXPOSURE LIMITS:

Drinking Water Standards
None established

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

EPA Health Advisories and Cancer Risk Levels
None established

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria

- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms, levels of 1.7 μg/L, 0.17 μg/L and 0.017 μg/L may result in estimated incremental lifetime cancer risks of 1E-05, 1E-06, and 1E-07, respectively.
  - Based on ingestion of contaminated aquatic organisms only, levels of 107 μg/L, 10.7 μg/L, 1.07 μg/L may result in estimated incremental lifetime cancer risks of 1E-05, 1E-06, 1E-07, respectively.

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 9320 μg/L tetrachloroethanes.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 2400 μg/L tetrachloroethanes.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 9020 μg/L tetrachloroethanes.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:
No reference dose available.

## REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

1,1,2,2-Tetrachloroethane is listed as a toxic pollutant, subject to general pretreatment standards for new and existing sources, and effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the electroplating, and steam electric power generating point source categories (3767, 3802). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

1,1,2,2-Tetrachloroethane is listed on the first priority list of drinking water contaminants for which NPDWRs will be developed, and is listed by EPA as an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3781, 3771). In states with an approved Underground Injection Control program, a permit is required for the injection of 1,1,2,2-tetrachloroethane-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

1,1,2,2-Tetrachloroethane is identified as a toxic hazardous waste (U209) and listed as a hazardous waste constituent (3783, 3784). A non-specific source of 1,1,2,2-tetrachloroethane-containing waste is the production of chlorinated aliphatic hydrocarbons (325, 3765). Waste streams from the following industries contain 1,1,2,2-tetrachloroethane and are listed as specific sources of hazardous waste: organic chemicals (production of 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, 1,2-dichloroethane and vinyl chloride) and inorganic chemicals (chlorine production) (3774, 3765). 1,1,2,2- Tetrachloroethane is subject to land disposal restrictions when its concentration as a hazardous constituent of certain wastewaters exceeds designated levels (3785). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786).

1,1,2,2-Tetrachloroethane is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
1,1,2,2-Tetrachloroethane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 0.454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing 1,1,2,2-tetrachloroethane but these depend upon the concentrations of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of 1,1,2,2-tetrachloroethane must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to 1,1,2,2-tetrachloroethane in any 8-hour workshift of a 40-hour work-week shall not exceed an 8-hour time-weighted average (TWA) of 1 ppm. Skin exposure shall be prevented/reduced through the use of protective clothing and procedures (3539).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated 1,1,2,2-tetrachloroethane as a hazardous material with a reportable quantity of 0.454 kg, subject to requirements for packaging, labeling and transportation (306).

- State Programs

ALL STATES

All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. Other states have promulgated additional or more stringent criteria.

CALIFORNIA

California has a maximum contaminant level of 1.0 μg/L for drinking water (3096).

CONNECTICUT

Connecticut has a quantification limit of 2.0 μg/L for drinking water (3137).

KANSAS

Kansas has set an action level of 1.7 μg/L for ground-water (3213).

NEW YORK

New York has set a maximum contaminant level of 5 μg/L for drinking water (3501).

OKLAHOMA

Oklahoma has a water quality criterion of 2.8 μg/L for ground-water (3534).

RHODE ISLAND

Rhode Island has an acute freshwater quality guideline of 466 μg/L and a chronic guideline of 10 μg/L for surface waters for the protection of aquatic life. These guidelines are enforceable under Rhode Island state law (3590).

SOUTH DAKOTA

South Dakota requires that 1,1,2,2-tetrachloroethane be nondetectable, using designated test methods, in ground-water (3671).

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA)

  EPA has proposed listing spent catalyst from the hydrochlorinator reactor in the production of 1,1,1-trichloroethane as a specific source of 1,1,2,2-tetrachloroethane-containing hazardous waste (3795). EPA has proposed that solid wastes be listed as hazardous because they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is equal to or greater than 1.3 mg/L of 1,1,2,2-tetrachloroethane. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Programs

  MOST STATES

  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA

  Minnesota has proposed a Recommended Allowable Limit (RAL) of 2.0 μg/L for drinking water (3451). Minnesota has also proposed a Sensitive Acute Limit (SAL) of 2330 μg/L for designated surface waters, and chronic citeria of 1.6 μg/L for designated surface waters and 2 μg/L for designated ground-waters for the protection of human health (3452).

EEC Directives

Directive on Ground Water (538)

Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
1,1,2,2-Tetrachloroethane is listed as a Class I/a toxic substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the method and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,1,2,2-Tetrachloroethane is classified as a toxic substance and is subject to packaging and labeling regulations.

## 11.1 MAJOR USES

The principal use for 1,1,2,2-tetrachloroethane is as an intermediate in the production of chloroethylenes, particularly trichloroethylene from acetylene. In 1967, an estimated 85% of the trichloroethylene produced in the U.S. was made in this manner; however, by 1974 the amount had decreased to 8%. Tetrachloroethane has been widely used as a solvent, principally for cleaning and extraction processes. It has also been used in paint removers, varnishes, lacquers, photographic film, resins and waxes, in extraction of fats and oils, and in insecticides, weed killers, and fumigants. Due to its toxicity and the availability of less toxic solvents, its use is now quite limited (3615).

## 11.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 11.2.1 Transport in Soil/Ground-water Systems

#### 11.2.1.1 Overview

1,1,2,2-Tetrachloroethane may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). Transport pathways of low soil concentrations can be generally assessed with the result of an equilibrium partitioning calculation, as shown in Table 11-1. These calculations estimate the partitioning of 1,1,2,2-tetrachloroethane among soil particles, soil water and soil air. The 1,1,2,2-tetrachloroethane associated with the air and water phases of the soil is expected to have higher mobility than the adsorbed compound.

The model for unsaturated topsoil indicates that approximately 2% of the 1,1,2,2-tetrachloroethane is expected to partition to the soil-water phase and be available to migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. For the small portion of 1,1,2,2-tetrachloroethane in the gaseous phase of the soil (approximately 0.1%), diffusion through the soil-air pores up to the ground surface, and subsequent removal by evaporation, may be a significant loss pathway.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the 1,1,2,2-tetrachloroethane (52%) is expected to be present in the soil-water phase (Table 11-1) and transported with flowing ground water. Ground water underlying tetrachloroethane-contaminated soils with low organic content is expected to be vulnerable to contamination.

TABLE 11-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR
1,1,2,2-TETRACHLOROETHANE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil at 25°C[b,c] | 97.6 | 2.3 | 0.1 |
| Saturated deep soil[d] | 48.0 | 52.0 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Used estimated soil sorption coefficient: $K_{oc}$ = 220 (33).
c) Henry's law constant taken as 5.0E-04 atm · $m^3$/mol at 25°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

Soil/water partition coefficients ($K_d$) for 1,1,2,2-tetrachloroethane were calculated by Sims et al. (3656). For a McLaurin soil the $K_d$ was 426.58 (log $K_d$ = 2.63), and for a Kidman soil the $K_d$ was 891.25 (log $K_d$ = 2.95).

### 11.2.1.2 Sorption on Soils

The mobility of 1,1,2,2-tetrachloroethane in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Relatively little information specific to the sorption of 1,1,2,2-tetrachloroethane on soil particles is available. A soil absorption coefficient of 220 (33) has been reported. A measure of soil absorption can be determined from retardation rates ($R_f$), which represent the ratio of interstitial water velocity to pollutant velocity within the soil. Retardation rates are a function of $K_{oc}$, the ratio of soil density (a) to soil

water content (b), and the organic content (oc) of the soil, as indicated in the following equation (82):

$$R_f = 1 + (a/b)K_{oc}(oc)$$

Schwarzenbach et al. (77) reported retardation factors for some chlorinated aliphatic compounds that have $K_{oc}$ values lower than that reported for 1,1,2,2-tetrachloroethane. The data indicate some retardation (i.e., adsorption) in soils having 1-2% organic carbon and little or no retardation in deep soils having less than 0.1% organic carbon. Wilson et al. (82) reported that several chlorinated aliphatics moved rapidly through a sandy soil. Assuming analogous soil conditions, and considering the higher $K_{oc}$ of 1,1,2,2-tetrachloroethane, some adsorption of 1,1,2,2-tetrachloroethane is expected to occur in surface soils; however, in deep soils, sorption is not expected to be particularly significant.

### 11.2.1.3 Volatilization from Soils

Transport of 1,1,2,2-tetrachloroethane vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. Sims et al. (3656) determined volatilization rates of 1,1,2,2-tetrachloroethane for McLaurin soil and found that 38.6% was volatilized over a 50-hr incubation period. The mean half-life was reported to be 0.38 days.

In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physico-chemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31). No information was found concerning the vapor-soil sorption coefficient or the vapor phase diffusion coefficient of 1,1,2,2-tetrachloroethane. The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. For other chlorinated aliphatic compounds, moderate increases in H have been seen with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of 1,1,2,2-tetrachloroethane from surface soils.

Soil volatilization rates of chlorinated ethanes from have been reported to be about one order of magnitude slower than volatilization rates for well-stirred aqueous solutions (82). The half-life for the volatilization of 1,1,2,2-tetrachloroethane from stirred aqueous solutions in the laboratory was reported to be in the range of 55-90 minutes, depending on the degree of agitation (10).

### 11.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of 1,1,2,2-tetrachloroethane in soil/ground-water systems is not well documented. In most cases, it should be assumed that 1,1,2,2-tetrachloroethane will persist for months to years (or more). The 1,1,2,2-tetrachloroethane that has volatilized will eventually undergo photochemical oxidation in air; a tropospheric lifetime on the order of a few years is expected for 1,1,2,2-tetrachloroethane (10).

No information on the hydrolysis of 1,1,2,2-tetrachloroethane was available; under normal environmental conditions, rapid hydrolysis is not expected to occur. Data reported for other chlorinated ethanes (10) suggest that hydrolysis would not be competitive with volatilization.

Literature references to microbial degradation of compounds such as 1,1,2,2-tetrachloroethane are very few. In general, most references indicate that low molecular weight chloroaliphatics are not rapidly metabolized in the environment (10). Tabak et al. (3697) reported only slight degradation of 1,1,2,2-tetrachloroethane with an acclimated microbial population; however, Mudder (3472) reported 41% degradation in 24 days in a modified shake flask test using an unacclimated inoculum, and 19% degradation in a river die-away test. Thom and Agg (80) included some chlorinated ethanes among those chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as 1,1,2,2-tetrachloroethane is very low and drops off sharply with increasing depth. Thus, biodegradation should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

### 11.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that 1,1,2,2-tetrachloroethane in the environment is moderately volatile from aqueous solutions, moderately adsorbed to soil, and has a low potential for bioaccumulation. This compound may volatilize from the soil surface, but that portion not subject to volatilization will be somewhat mobile in ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of 1,1,2,2-tetrachloroethane from a disposal site could result in inhalation exposures. In addition, there is some potential for ground-water contamination, particularly in sandy soils. As evidence of this potential, Mitre (83) reported that this compound has been found in 8 of the 546 National Priority List (NPL) sites. It was detected at all eight sites in ground water and one site in surface water. It was not detected in any of the 945 ground-water drinking water supplies sampled in the USEPA Groundwater Supply Survey (531) (detection limit of 0.5 μg/L). However, it was found in 64 of 1072 ground-water sources sampled in New

Jersey (3546). The maximum reported concentration was 2.7 ppb. It was also found in a municipal drinking water well in Tacoma, WA at a concentration of 17-300 ppb (3621).

While the potential for movement of 1,1,2,2-tetrachloroethane in ground water appears somewhat limited, under certain conditions discharge to surface water may occur. Concentrations up to 3 ppb have been measured in some U.S. surface waters (3546, 3183, 3371, 3532). In such situations, several other exposure pathways are possible:

- Surface waters may be used as drinking water supplies and result in direct oral exposure.
- Aquatic organisms residing in these waters may become contaminated, and consumption of these organisms would also result in oral exposures.
- Recreational use of these waters may result in dermal exposures.
- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in oral exposures.

Exposure to 1,1,2,2-tetrachloroethane associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground water for two reasons. First, the Henry's law constant for 1,1,2,2-tetrachloroethane suggests that it may volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this compound is low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 11.2.4 Other Sources of Exposure

The available data suggest that the general public's exposure to 1,1,2,2-tetrachloroethane might occur more commonly through air pollution than through contaminated water supplies. In a compilation of atmospheric monitoring data on volatile organics, Brodzinsky and Singh (84) obtained data for 1,1,2,2-tetrachloroethane from 915 sampling locations. In urban and suburban areas (853 sites), the median concentration was 0.037 $\mu g/m^3$ with a maximum reported value of 33 $\mu g/m^3$. For source-dominated areas (60 sites), the maximum reported value was 4.8 $\mu g/m^3$. Other studies have reported average atmospheric concentrations in U.S. cities ranging from trace amounts to 0.39 $mg/m^3$ (3504). Thus, persons in some locations are subject to inhalation exposure to 1,1,2,2-tetrachloroethane.

## 11.3 HUMAN HEALTH CONSIDERATIONS

### 11.3.1 Animal Studies

#### 11.3.1.1 Carcinogenicity

A study investigating the carcinogenicity of 1,1,2,2-tetrachloroethane was carried out by the National Cancer Institute (121). Technical-grade 1,1,2,2-tetrachloroethane (90% pure) was administered in corn oil by gavage to $B6C3F_1$ mice and Osborne-Mendel rats 5 days per week for 78 weeks. The time-weighted-average doses were 108 and 62 mg/kg/day for male rats, 76 and 43 mg/kg/day for female rats, and 282 and 142 mg/kg/day for all mice. The incidence of hepatocellular carcinoma in male and female mice was positively correlated with dosage level. Ninety percent of males and females at the high dosage level as well as 26% of the males and 63% of the females at the low dosage level developed liver carcinoma. The incidence of neoplasms in rats of either sex was not statistically significant. However, one neoplastic liver nodule and two hepatocellular carcinomas (rare tumors in male Osborne-Mendel rats) were seen in high-dose males.

The induction of pulmonary tumors by 1,1,2,2-tetrachloroethane in Strain A mice was assessed by Theiss et al. (3711). The test animals were injected intraperitoneally with doses of 80, 200, and 400 mg/kg over a two-week period (total doses 400, 3,600, and 6,400 mg/kg, respectively). There was no evidence of a positive carcinogenic response.

1,1,2,2-Tetrachloroethane was tested in the rat liver foci assay by Story et al. (3688) and gave a positive response for tumor promotion.

Based on available data, IARC (25) has listed 1,1,2,2-tetrachloroethane in category 3 (insufficient evidence) in its weight-of-evidence ranking for potential carcinogens.

#### 11.3.1.2 Genotoxicity

Studies have shown that 1,1,2,2-tetrachloroethane is mutagenic in some strains of Salmonella typhimurium (118), but negative in others (3690). It also inhibited the growth of DNA polymerase-deficient Escherichia coli (118).

Mixed results have also been found with Chinese hamster cells in culture. CHO cells showed no increase in chromosomal aberrations but did show an increase in sister chromatid exchanges with and without metabolic activation (3235). This compound was also shown to induce aneuploidy in Chinese hamster V79 cells but without a clear dose response (3537).

1,1,2,2-Tetrachloroethane did not induce sex-linked recessive lethals in meiotic and postmeiotic male germ cells of the fruit fly, Drosophila melanogaster, either via injection or by feeding (3845).

Tests with 1,1,2,2-tetrachloroethane in the BALB/c-3T3 cell transformation assay were negative; testing was conducted in the absence of an exogenous metabolic activation system (3732).

#### 11.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Intraperitoneal treatment of mice with 300-400 mg/kg/day of 1,1,2,2-tetrachloroethane during organogenesis produced embryotoxic (lethal) effects both pre- and postimplantationally. It also resulted in a low incidence of malformations including cleft palate, exencephaly (brain outside the skull) and fused ribs and vertebrae. The effects were related to the dose, period of treatment, and mouse strain used (3625).

In an inhalation reproductive toxicity study conducted by Schmidt et al. (3625), no grossly observable teratogenic or fetotoxic effects were observed in the offspring of male rats who were exposed for 9 months to 1,1,2,2-tetrachloroethane and then mated to unexposed females.

#### 11.3.1.4 *Other Toxicologic Effects*

##### 11.3.1.4.1 Short-term Toxicity

Animal studies have shown that short-term exposures to high concentrations of 1,1,2,2-tetrachloroethane can result in liver damage as indicated by changes in liver and serum enzyme levels and increases in liver lipid and triglyceride levels (388). Changes in liver enzymes have been observed in rats following a single gavage dose of 100 mg/kg (3624). Fatty degeneration of the liver was seen in mice exposed to 600 ppm 1,1,2,2-tetrachloroethane for 6 hours (3721), and in guinea pigs injected intravenously or intraperitoneally with 0.7 mL of the compound over 14 days (120). Elevated levels of serum SGOT (indicative of liver tissue injury) were observed in rats exposed to 10 or 100 ppm (70 or 700 $mg/m^3$) for 6 hours (3166). Exposure of rats to 13.3 $mg/m^3$ for 4 hr/day for 8-10 days reportedly caused no adverse liver effects (3623).

Several subchronic toxicity studies have been carried out on 1,1,2,2-tetrachloroethane. Golke et al. (3247) gave male albino rats the compound by gavage at doses of 0, 8, 20, or 50 mg/kg for up to 45 days. Statistically significant enzyme level changes and persistent degeneration of the kidneys, testicles, liver, and thyroid gland were observed in some animals at all dose levels. Specific dose-related details were not provided. A lowest observable adverse effect level of 8 mg/kg/day was reported for this study (3743). In an NCI study (121), rats and mice were dosed with 1,1,2,2-tetrachloroethane by gavage, 5 days/ week for 6 weeks. Retardation of body weight gain was reported for the rats dosed with 56, 100, and 178 mg/kg/day. No other

effects were reported. Rats exposed to 559 ppm several hours per day, 5 days/week, for 15 weeks, developed histological alterations in the liver and had a transitory increase in incorporation of thymidine into the liver (3728). Rats exposed to 516 ppm for 5 hr/day, 5 days/ week, for 13 weeks, developed small glomerular lesions, and had lower proteinuria and depressed body weights when compared to controls (3149).

#### 11.3.1.4.2 Chronic Toxicity

Animals studies have shown that long-term exposure to 1,1,2,2-tetrachloroethane can result in adverse effects on the liver and other organ systems. Rabbits exposed to 0.3, 1.5 and 14.6 ppm for 3-4 hours daily for 7-11 months exhibited liver and kidney changes as well as suppression of the immune system at the two highest exposure levels. No effects were seen at 0.3 ppm (389). Rats exposed to 1.9 ppm 4 hr/day for up to 265 days developed fatty livers, an increased number of white blood cells and elevated pituitary hormone levels (3623). Rats dosed by gavage with 0, 3.2, or 8.0 mg/kg (82 doses over 120 days) exhibited degeneration of the liver, kidneys, testicles, and thyroid gland (3247). Rats dosed by gavage with 62 or 108 mg/kg/day (males), or 43 or 76 mg/kg/ day (females) for 77 or 78 weeks exhibited decreased body weight and overt signs of toxicity including respiratory difficulties. Non-neoplastic histopathological lesions were not observed (121). Mice dosed by gavage with 142 or 284 mg/kg/day for 78 weeks developed acute toxic tubular necrosis and hepatocellular carcinoma at the high dose. No other non-neoplastic histopathological effects were observed (121).

### 11.3.2 Human and Epidemiological Studies

#### 11.3.2.1 Short-term Toxicologic Effects

Tetrachloroethane is considered to be among the more toxic of the chlorinated hydrocarbons. In humans, ingestion or inhalation may cause nausea, vomiting, drowsiness, tremor, headache, abdominal pain and irritability. More severe exposure results in nephritis and jaundice (46).

Human subjects exposed to vapor concentrations of 335 ppm for 10 minutes or 186 ppm for 30 minutes experienced respiratory irritation and central nervous system effects (46). Among a group of workers exposed to 20-65 ppm for an unspecified period of time, nausea, vomiting, abdominal pain and tremors of the hands were observed (119).

Eight adults who ingested 3 mL of tetrachloroethane (isomer unspecified) were comatose within 2 hours. Reflexes were absent, the pulse was barely perceptible and respiration was shallow and rapid. Eventually all recovered and showed no after effects (390).

NIOSH (388) reports numerous deaths resulting from tetrachloroethane ingestion. However, the ingested quantities were not indicated. In all cases, death occurred within 24 hours.

Tetrachloroethane is absorbed through intact skin. There is some evidence that if absorbed in this manner, it will effect only the central nervous system (54). One human fatality has been attributed primarily to skin absorption (391).

If tetrachloroethane is splashed in the eye, it will cause lacrimation and irritation. It may cause transient epithelial injury similar to that caused by chloroform (19).

### 11.3.2.2 Chronic Toxicologic Effects

Jeney et al. (392) conducted a 3-year study of workers exposed to tetrachloroethane concentrations up to 247 ppm. During the first year, 31% of the workers reported symptoms such as epigastric pain, headaches and loss of appetite. Their livers were found to be enlarged. Tests for liver dysfunction showed positive results. During the next two years, vapor levels dropped to 15 to 36 ppm. This led to a drop in the number of employees reporting symptoms - from 13% the second year to 2% the third year. In those reporting symptoms, however, the liver dysfunction was still present.

Norman et al. (3510) analyzed the mortality records of workers exposed to 1,1,2,2-tetrachloroethane in a chemical processing plant. Slight increases in relative risk of death due to genital and lymphatic cancers and leukemias were seen in 1,099 exposed workers when compared with 1,319 unexposed workers. The increases were not statistically significant.

### 11.3.3 Levels of Concern

USEPA has calculated that 1,1,2,2-tetrachloroethane concentrations of 1.7, 0.17 and 0.017 $\mu$g/L in ambient waters would correspond to incremental lifetime cancer risks of 1E-05, 1E-06, and 1E-07, respectively, as a result of ingestion of water and contaminated aquatic organisms (355). Risk estimates are expressed as a probability of cancer after a lifetime consumption of two liters of water per day and consumption of 6.5 g per day of fish that have bioaccumulated the compound. Thus, a risk of 1E-05 implies that a lifetime daily consumption of two liters of drinking water and 6.5 g of contaminated fish at a criterion of 1.7 $\mu$g 1,1,2,2-tetrachloroethane per liter would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 people exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

Insufficient data were available to derive a water criterion for the protection of human health from toxic effects of 1,1,2,2-tetrachloroethane other than cancer (355).

The OSHA permissible exposure limit for 1,1,2,2-tetrachloroethane is 1 ppm (with a skin notation) averaged over an 8-hour workshift (3539). NIOSH recommends that exposures be reduced to the lowest feasible level (3503). The ACGIH recommended threshold limit value, as of 1988, was 1 ppm for 8-hr TWA (with a skin notation) (3005).

### 11.3.4 Hazard Assessment

Oral administration of 1,1,2,2-tetrachloroethane induced liver cancer in $B6C3F_1$ mice (121). In another study conducted with Osborne-Mendel rats that received the material orally, the incidence of tumors was not statistically significant. However, two hepatocellular carcinomas and one liver nodule were observed in high-dose males (rare tumors in male Osborne-Mendel rats) (121). Historically, this strain of rat has shown a lack of sensitivity to induction of liver carcinomas by oral administration of chlorinated organic compounds. Based on these data, the USEPA (667) calculated an upper-limit incremental unit cancer risk of $0.2(mg/kg/day)^{-1}$ for 1,1,2,2-tetrachloroethane.

Mutagenic effects of 1,1,2,2-tetrachloroethane have been documented in some bacterial assays (118, 3690), but not in others. The compound gave a negative response in a mammalian cell transformation study (3732), did not induce sex-linked recessive lethal mutations in Drosophila (3732), and did not increase the incidence of chromosomal aberrations in Chinese hamster ovary cells; however, it did cause increases in sister chromatid exchanges in CHO cells (3235).

A study conducted with mice indicates that high concentrations of 1,1,2,2-tetrachloroethane (300-400 mg/kg) are embryotoxic and capable of inducing a low incidence of malformations (3625).

Among the simple chlorinated hydrocarbons, 1,1,2,2-tetrachloroethane is considered to be among the most toxic (12). Numerous deaths have been recorded from inhalation and ingestion (25); one fatality has been attributed to cutaneous absorption (391). Target organs include the liver, central nervous system, and kidney (38).

The notable lack of quantitative data available for either humans or experimental animals makes estimates of dose-response relationships uncertain, particularly with regard to long-term, low-level oral exposures.

## 11.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of 1,1,2,2-tetrachloroethane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of 1,1,2,2-tetrachloroethane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil

handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of 1,1,2,2-tetrachloroethane, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the 1,1,2,2-tetrachloroethane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the 1,1,2,2-tetrachloroethane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; 1,1,2,2-tetrachloroethane is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for 1,1,2,2-tetrachloroethane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical 1,1,2,2-tetrachloroethane detection limits that can be obtained in waste-waters and non-aqueous samples (wastes, soils, etc.) are shown below. Detection limits were not given for Method 8010. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.03 μg/L (Method 601) | 5 μg/kg (Method 8240) |
| 6.9 μg/L (Method 624) | |
| 10 μg/L (Method 1624) | |
| 5 μg/L (Method 8240) | |

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Note: The nonsequential numbers of the references reflect the order of references as they ppear in the master bibliography.

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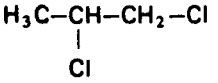
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| COMMON SYNONYMS: | CAS REG.NO.: 78-87-5 FORMULA: $C_3H_6Cl_2$<br>NIOSH Number: TX9625000<br>STRUCTURE:  | AIR W/V CONVERSION FACTOR at 25°C<br>4.62 mg/m³ ≈ 1 ppm<br>0.2165 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 112.99 |
|---|---|---|
| 1,2-Dichloropropane<br>Alpha, beta-dichloropropane<br>Propylene chloride<br>Propylene dichloride | | |

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|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as 1,2-dichloropropane with cyanides, mercaptans or other organic sulfides typically generate heat, while those with amines, azo compounds, hydrazines, caustics or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505). |

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| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Chloroform-like | (23) |
| | • Odor Threshold: 50 ppm | (38) |
| | • Density: 1.1580 g/mL (at 20°C) | (23) |
| | • Freeze/Melt Point: -80.00 to -100.00°C | (23,38) |
| | • Boiling Point: 96.30°C | (23) |
| | • Flash Point: 16.1°C (closed cup), 21°C (open cup) | (23,3302) |
| | • Flammable Limits: 3.40 to 14.50% by volume | (51,60,506) |
| | • Autoignition Temp.: 557°C | (51,60,506) |
| | • Vapor Pressure: 3.95E+01 mm Hg (at 20°C) | (38) |
| | • Satd. Conc. in Air: 2.5800E+05 mg/m³ (at 20°C) | (67) |
| | • Solubility in Water: 2.70E+03 mg/L (at 20°C) | (12) |

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|---|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Viscosity: 0.840 cp (at 20°C) | (48) |
| | • Surface Tension: 2.9000E+01 dyne/cm (at 20°C) | (3302) |
| | • Log (Octanol-Water Partition Coeff.): 2.28 | (3302) |
| | • Soil Adsorp. Coeff.: 5.10E+01 | (33) |
| | • Henry's Law Const.: 3.60E-03 atm · $m^3$/mol (at 20°C) | (74) |
| | • Bioconc. Factor: 4.1 (estim), 5.0 (estim), ~10 (carp) | (147,659,3804) |

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| PERSISTENCE IN THE SOIL-WATER SYSTEM | 1,2-Dichloropropane is expected to be somewhat mobile in surface soils and highly mobile in deep or sandy soils. Volatilization may be important for the near surface material or the small portion expected to be in the soil-air phase. Transformation processes (hydrolysis, biodegradation) are not expected to be significant in natural soils. |

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| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of 1,2-dichloropropane to groundwater drinking water supplies. Inhalation resulting from volatilization from surface soils may also be important in some situations. |

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| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (46, 54)<br>Prolonged contact with liquid 1,2-dichloropropane may cause dermatitis. Undiluted it is moderately irritating to the eyes, but does not cause permanent injury. Based on animal experiments, inhalation or ingestion of high levels of 1,2-dichloropropane can be expected to produce CNS depression.<br><br>Acute Toxicity Studies:<br><br>INHALATION:<br>$LC_{Lo}$ 1600 ppm · 7 hr Rat (3302) |

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|---|---|
| HEALTH HAZARD DATA (Cont.) | ORAL:<br>$LD_{50}$ 2196 mg/kg Rat (3302)<br>$LD_{50}$ 860 mg/kg Mouse (3614)<br><br>SKIN:<br>$LD_{50}$ 8750 mg/kg Rabbit (3302)<br><br>Long-Term Effects: Liver and kidney toxicity<br>Pregnancy/Neonate Data: no data<br>Genotoxicity Data: Conflicting data<br>Carcinogenicity Classification:<br>IARC - Group 3 (not classifiable as to its carcinogenicity to humans)<br>NTP - Some evidence in mice, equivocal in female rats, no evidence in male rats<br>EPA - Group B2 (probable human carcinogen; sufficient evidence in animals and inadequate evidence in humans) |

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| HANDLING PRECAUTIONS (38) | Handle chemical only with adequate ventilation.<br>• Vapor concentrations of 75-400 ppm: any supplied-air respirator, self-contained breathing apparatus or chemical cartridge respirator with an organic vapor cartridge.<br>• 400-2000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece; any gas mask with an organic vapor canister. • Chemical goggles if there is probability of eye contact.<br>• Impervious protective clothing and gloves to prevent repeated or prolonged skin contact with the liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 75 ppm; STEL: 110 ppm
- AFOSH PEL (8-hr TWA): 75 ppm; STEL (15 min): 110 ppm

Criteria
- NIOSH IDLH (30-min): 2000 ppm
- ACGIH TLV® (8-hr TWA): 75 ppm
- ACGIH STEL (15-min): 110 ppm

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

WATER EXPOSURE LIMITS:

Drinking Water Standards (3742)
MCLG: 0 μg/L (proposed)
MCL: 5 μg/L (proposed)

EPA Health Advisories and Cancer Risk Levels (3742)

In the absence of formal drinking water standards, the EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- 10-day 10 kg child: 90 μg/L
- 1E-04 cancer risk: 60 μg/L

WHO Drinking Water Guideline
No information available.

EPA Ambient Water Quality Criteria
- Human Health (355)
  - None, due to insufficient data.

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest level occurs at 23,000 μg/L dichloropropanes.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 5700 μg/L dichloropropanes.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 10,000 μg/L dichloropropanes.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 3040 μg/L dichloropropanes.

REFERENCE DOSES:
No reference dose available.

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

1,2-Dichloropropane is designated a hazardous substance. It has a reportable quantity (RQ) limit of 454 kg. (347, 3764). It is also listed as a toxic pollutant, subject to general pretreatment standards for new and existing sources, and to effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), and metal finishing (3768). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

1,2-Dichloropropane is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). EPA presently lists it as an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3771). In states with an approved Underground Injection Control program, a permit is required for the injection of 1,2-dichloropropane-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

1,2-Dichloropropane is identified as a toxic hazardous waste (U083) and listed as a hazardous waste constituent (3783, 3784). A non-specific source of 1,2-dichloropropane-containing waste is the production of chlorinated aliphatic hydrocarbons (325, 3765).

Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786).

1,2-Dichloropropane is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Toxic Substances Control Act (TSCA)
Manufacturers, processors or distributors of 1,2-dichloropropane must report production, usage and disposal information to EPA. They, as well as others who possess health and safety studies on 1,2-dichloropropane, must submit them to EPA (334, 3789). EPA requires that manufacturers and processors of 1,2-dichloropropane conduct health and environmental tests including neurotoxicity, mutagenicity, teratogenicity, developmental toxicity, and acute and chronic toxicity tests for aquatic invertebrates (3779).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
1,2-Dichloropropane is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing 1,2-dichloropropane but these depend upon the concentration of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of 1,2-dichloropropane must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
1,2-Dichloropropane is exempt from a tolerance requirement when used as a solvent for pesticide formulations applied before crops emerge from soil (315).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to 1,2-dichloropropane in any 8-hour work-shift of a 40-hour work-week shall not exceed an 8-hour time-weighted average (TWA) of 75 ppm. An employee's 15-minute short-term exposure limit (STEL) of 110 ppm shall not be exceeded at any time during a work day (3539).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated 1,2-dichloropropane as a hazardous material with a reportable quantity of 454 kg, subject to requirements for packaging, labeling and transportation (3180).

- State Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. Other states have promulgated additional or more stringent criteria.

CALIFORNIA
California has an action level of 10 μg/L (ppb) for drinking water (3098).

CONNECTICUT
Connecticut has a quantification limit of 2 μg/L for drinking water (3137).

KANSAS
Kansas has set an action level of 6 μg/L for ground-water contamination by 1,2-dichloropropane (3213).

NEW YORK
New York has an MCL of 5 μg/L for all dichloropropanes in drinking water (3501).

RHODE ISLAND
Rhode Island has an acute freshwater quality guideline of 2625 μg/L and a chronic guideline of 58 μg/L for the protection of aquatic life in surface waters. These guidelines are enforceable under Rhode Island state law (3590).

SOUTH DAKOTA
South Dakota requires that 1,2-dichloropropane be nondetectable, using designated test methods, in ground-water (3671).

VERMONT
Vermont has a preventive action limit of 0.056 μg/L and an enforcement standard of 0.56 μg/L for ground-water (3682).

Proposed Regulations

- Federal Programs

Safe Drinking Water Act (SDWA)
EPA will propose an MCLG of zero and an MCL of 5 μg/L for 1,2-dichloropropane in drinking water in May, 1989, with final action scheduled for May, 1990 (3759).

- State Programs

  MOST STATES

  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA

  Minnesota has proposed a Recommended Allowable Limit (RAL) of 5.6 μg/L for 1,2-dichloropropane in drinking water (3451). Minnesota has also proposed a Sensitive Acute Limit (SAL) of 13125 μg/L for designated surface waters, and chronic criteria of 5.6 μg/L for designated ground-waters, 5 μg/L for designated surface waters, and 4.6 μg/L for cold surface waters. These criteria are for the protection of human health (3452).

EEC Directives

Directive on Ground-Water (538)

Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)

The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
Dichloropropane is listed as a Class II/b harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,2-Dichloropropane is classified as a flammable, harmful substance and is subject to packaging and labeling regulations.

EEC Directives--Proposed Resolution
Resolution on the Revised List of Second-Category Pollutants (545)
1,2-Dichloropropane is one of the second-category pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 12.1 MAJOR USES

1,2-Dichloropropane is used as an intermediate in chemical synthesis, as a lead scavenger for gasoline and as a solvent for fats, oils, waxes, gums and resins. Mixtures of dichloropropane and dichloropropenes are used as soil fumigants (54).

## 12.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 12.2.1 Transport in Soil/Ground-water Systems

#### 12.2.1.1 Overview

1,2-Dichloropropane may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways of low soil concentrations can be assessed using an equilibrium partitioning model as shown in Table 12-1. These calculations predict the partitioning of 1,2-dichloropropane among soil particles, soil water and soil air. The 1,2-dichloropropane associated with the water and air phases of the soil is more mobile than that which is adsorbed.

The estimates for the unsaturated topsoil model indicate that approximately 9% of the 1,2-dichloropropane can be expected to partition to the soil-water phase and thus be available to migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. For the portion of 1,2-dichloropropane in the gaseous phase of the soil (4%), diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, may be a significant loss pathway.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the 1,2-dichloropropane (82%) is expected to be present in the soil-water phase (Table 12-1) and transported with flowing ground-water. Ground-water underlying 1,2-dichloropropane-contaminated soils with low organic content is highly vulnerable to pollution.

#### 12.2.1.2 Sorption on Soils

The mobility of 1,2-dichloropropane in the soil/ground-water system (and its eventual migration into aquifers) will be strongly affected by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;

- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

TABLE 12-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR 1,2-DICHLOROPROPANE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated[b,c] topsoil at 25°C | 87.1 | 8.9 | 4.0 |
| Saturated deep soil[d] | 17.6 | 82.4 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34-36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 51 (33).
c) Henry's law constant taken as 3.6E-03 atm · $m^3$/mol at 25°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

The sorption of 1,2-dichloropropane on soil particles is not well-documented. Retardation rates, which represent the interstitial water velocity/pollutant velocity in the soil, were reported by Wilson et al. (82) to be a function of $K_{oc}$ ratio of soil density (a) to soil water content (b), and the organic content (oc) of the soil according to the following equation:

$$R_f = 1 + (a/b)K_{oc}\,(oc)$$

Wilson et al. (82) report a retardation factor of only 1.2 for 1,2-dichloroethane in sandy soil; since 1,2-dichloropropane has a somewhat higher $K_{oc}$, sorption may be slightly greater. Schwarzenbach et al. (77) report retardation factors as a function of soil type for some chlorinated aliphatic compounds that have $K_{oc}$ values higher than that reported for 1,2-dichloropropane. The data indicate some adsorption (i.e., retardation factors ranging from 3 to 31) in soils having 1-2% organic carbon; in soils having 0.1-1% organic carbon, adsorption is lower. The retardation factors in deep soils having less than 0.1% organic carbon suggest little or no retention of the

chlorinated aliphatics. Assuming analogous soil conditions, adsorption of 1,2-dichloropropane, particularly to deep soils, is not expected to be very strong.

#### 12.2.1.3 Volatilization from Soils

Transport of 1,2-dichloropropane vapors through the air-filled pores of unsaturated soils may be an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

Volatilization rates from aqueous environments were not available for 1,2-dichloropropane. Callahan et al. (10) report that volatilization half-lives for 1,2-dichloropropane from stirred solutions in the laboratory would be expected to be on the order of 50 minutes; the volatilization half-lives are expected to vary considerably with the extent of agitation of the solution. Compared to their volatilization from well-stirred aqueous solutions, volatilization of other chlorinated ethanes from surface soils has been reported to be slower by approximately one order of magnitude (82).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H were also observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of 1,2-dichloropropane from surface soils.

No information was available for the two other physicochemical properties influencing 1,2-dichloropropane volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

### 12.2.2 Transformation Processes in Soil/Ground-water Systems

Volatilization appears to be the major mechanism in determining the fate of 1,2-dichloropropane in water and soil. Bioaccumulation in aquatic organisms and adsorption in sediments are not significant. 1,2-dichloropropane undergoes photochemical oxidation by OH radical. The half-life of the compound in air, soil, and water has been reported in the Physico-Chemical Data Table.

Under normal environmental conditions, 1,2-dichloropropane is not expected to undergo rapid hydrolysis. Callahan et al. (10) estimate the uncatalyzed hydrolysis half-life for 1,2-dichloropropane to be on the order of 6 months to several years. Base-catalyzed hydrolysis of 1,2-dichloropropane adsorbed on soil may also occur. However, hydrolysis is not expected to occur at a rate competitive with volatilization.

Literature references to microbial degradation of compounds such as 1,2-dichloropropane are very few. In general, most references indicate that low molecular weight chloroaliphatics are not rapidly metabolized in the environment (76); however, some degradation has been observed. Altman and Lawlor (568) report that 1,2-dichloropropane at concentrations up to 1000 mg/L can be utilized by soil bacteria. Thom and Agg (80) included several chlorinated aliphatics on a list of organic chemicals amenable to degradation by biological sewage treatment, providing suitable acclimatization can be achieved. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as 1,2-dichloropropane is very low and drops off sharply with increasing depth. Thus, biodegradation in the soil/ground-water system should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations. In surface soils, biodegradation is expected to be very slow in comparison to volatilization.

### 12.2.3 Primary Routes of Exposure From Soil/Ground-water Systems

The above discussion of fate pathways suggests that 1,2-dichloropropane is highly volatile, weakly adsorbed by soil, and has no significant potential for bioaccumulation. This compound may volatilize from soil surfaces, but that portion not removed by volatilization is likely to become mobile in ground-water once it reaches the saturated zone. These fate characteristics suggest several potential exposure pathways.

Volatilization of 1,2-dichloropropane from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. In addition, the potential for ground-water contamination is high, particularly in sandy soils. This compound was reported in the USEPA (531) Ground-water Supply Survey (GWSS). This survey examined 945 finished water supplies that use ground-water sources. The results for 1,2-dichloropropane are summarized below:

| | Occurrences* | | Median of Positive | Maximum |
|---|---|---|---|---|
| | No. | % | (μg/L) | (μg/L) |
| Random | | | | |
| Supplies serving <10,000 people (280 samples) | 1 | 0.4 | 0.75 | 0.75 |
| Supplies serving >10,000 people (186 samples) | 5 | 2.7 | 0.96 | 21 |
| Non-Random | | | | |
| Supplies serving <10,000 people (321 samples) | 3 | 0.9 | 1.2 | 1.4 |
| Supplies serving >10,000 people (158 samples) | 4 | 2.5 | 0.7 | 18 |

*Samples having levels over quantification limit of 0.2 μg/L.

The random results are intended to statistically represent the U.S. ground-water drinking water supplies. The non-random samples were chosen by the states as being potentially contaminated. 1,2-Dichloropropane was also detected in the National Organic Monitoring Survey (NOMS) (90).

The properties of 1,2-dichloropropane and the survey results above indicate that this compound has the potential for movement in soil/ground-water systems. This compound may eventually reach surface waters by this mechanism, suggesting several other exposures pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion exposure.
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposure.
- Recreational use of these waters may result in dermal exposure.
- Domestic animals may consume or be dermally exposed to contaminated ground- or surface waters; the consumption of meats and poultry could then result in ingestion exposure.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground-water. The Henry's law constant for 1,2-dichloropropane suggests that it is likely to volatilize upon reaching surface waters. In addition, the BCF for this compound indicates no significant potential for bioaccumulation.

#### 12.2.4 Other Sources of Exposure

The volatility of this compound suggests that it may be found in air. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For 1,2-dichloropropane, they had data for 422 locations. In urban and suburban locations, the median concentration was 0.26 $\mu g/m^3$. In source-dominated areas, the median concentration was 0.55 $\mu g/m^3$. These results suggest possible inhalation exposure to 1,2-dichloropropane for individuals residing in these areas.

## 12.3 HUMAN HEALTH CONSIDERATIONS

### 12.3.1 Animal Studies

#### 12.3.1.1 Carcinogenicity

Carcinogenesis studies of 1,2-dichloropropane were conducted by administering the chemical in corn oil by gavage to female F344/N rats and male and female

B6C3F$_1$ mice at doses of 125 or 250 mg/kg and to male F344/N rats at doses of 62 or 125 mg/kg. Doses were administered 5 times per week for 103 weeks. Survival was reduced for high-dose female rats and high-dose female mice. Survival in other groups was comparable to that of the controls. Some evidence of carcinogenic activity was indicated by dose-related increases in adenomas of the liver observed in both male and female mice. In female rats, there was equivocal evidence of carcinogenic activity as indicated by a marginally increased incidence of adenocarcinomas of the mammary gland; these borderline malignant lesions occurred concurrent with decreased survival and body weight gain, indicating that the 250 mg/kg dose was toxic. There was no evidence of carcinogenicity for male rats (146).

### 12.3.1.2 Genotoxicity

1,2-Dichloropropane was reported to be mutagenic in 2 strains of Salmonella typhimurium with and without metabolic activation and negative in three others (147, 3161, 3574). The compound was also reported to induce chromosomal aberrations in rat bone marrow cells (147). It also induced chromosomal aberrations as well as sister chromatid exchanges, with or without metabolic activation, in Chinese hamster ovary cells in culture (147, 3235), and it induced sister chromatid exchanges in Chinese hamster V79 lung cells in culture. No increase in sex-linked recessive lethals over controls was found when male Drosophila were exposed to 1,2-dichloropropane via feeding or inhalation (3845). No effect on cytotoxicity or replicative DNA synthesis was found when human lymphocytes were cultured in vitro for 4 hours in the presence of 1,2-dichloropropane with or without metabolic activation (3563).

### 12.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

The only study on the developmental toxicity of 1,2-dichloropropane found in the literature concerned an "early life stage toxicity test" using embryonic and larval fish (3060). Fathead minnow embryos before the eyed stage of development were placed in water containing five levels of 1,2-dichloropropane. Hatchability was not affected at any level. At the highest level, 110 mg/L, none of the larvae that hatched were normal. At 51 mg/L, a significant number of larvae (33%) were not normal while concentrations of 25 mg/L or less produced 100% normal larvae. The weights of 28 day old fish were significantly decreased by levels as low as 11 mg/L.

### 12.3.1.4 Other Toxicologic Effects

#### 12.3.1.4.1 Short-term Toxicity

The earliest reference to the acute oral toxicity of 1,2-dichloropropane in mammals was a 1932 study by Wright and Schaffer (148). Dogs orally administered 5700 mg/kg 1,2-dichloropropane exhibited loss of coordination within 15 minutes. Their condition deteriorated rapidly until death occurred 3.5 hours later. An oral dose of 3500 mg/kg caused staggering, partial narcosis and death within 24 hours. Autopsies revealed congestion of the lungs, liver, kidneys and bladder as well as

hemorrhages in the stomach and respiratory tract. Oral doses as low as 350 mg/kg caused moderately severe lesions in the liver, gastrointestinal tract and kidneys.

Inhalation studies were conducted by Heppel and coworkers in 1946 (144). These were done with rats, mice, guinea pigs, dogs and rabbits utilizing daily 7-hour exposures and a concentration range of 1000-2000 ppm. A concentration of 2200 ppm was lethal to over 50% of the animals of all 4 species after up to 8 exposures. Mice were the most sensitive with 90% dying after one exposure period. Pathological effects observed included fatty degeneration of the heart and liver, renal tubular necrosis and pulmonary congestion. Gross effects included weight loss, CNS depression, bronchitis and pneumonia. Repeated inhalation of 1000 ppm caused some deaths among dogs after 24 exposures and among rats after 7 exposures. There was early onset of eye irritation and incoordination. Liver damage was the main finding in the animals that died.

#### 12.3.1.4.2 Chronic Toxicity

Fatty changes and centrilobular congestion of the liver were observed in female rats given 1000 mg/kg of 1,2-dichloropropane by gavage for 13 weeks. No treatment-related lesions were seen in male rats or in mice of either sex given 500 mg/kg by gavage for the same duration (146).

In a long-term inhalation study, guinea pigs, dogs and rats were exposed to 400 ppm five days per week for 128 to 140 seven hour periods. The only effect observed was a decreased weight gain by rats (147).

A chronic toxicity test was conducted under the auspices of Dow Chemical Company (3181) using the mysid, Mysidopsis bahia. Less than 24 hours old mysids were exposed for 28 days in natural filtered seawater at 18-25 o/oo salinity and 22.9-28.0°C, to 0.41-4.09 mg/L 1,2-dichloropropane. The No-Observed-Effect-Concentration (NOEC) and the Maximum-Acceptable-Toxicant-Concentration (MATC) were found to be 4.09 mg/L and >4.09 mg/L, respectively.

### 12.3.2 Human and Epidemiologic Studies

#### 12.3.2.1 Short-term Toxicologic Effects

The literature contains one case report of a 46-year-old man who ingested 50 mL of a cleaning solution containing an unknown amount of 1,2-dichloropropane. Within 2 hours, he went into a deep coma. After 24 hours he regained consciousness. Treatment consisted of artificial ventilation and osmotic diuresis. After 36 hours he went into irreversible shock and died of cardiac failure. Autopsy showed acute hepatic necrosis (145). Another case report involves a 71-year-old man who attempted suicide by ingestion of about 180 mL of a dry-cleaning solvent containing 90% 1,2-dichloropropane and 10% 1,1,1-trichloroethylene. One hour after, he was hospitalized unconscious and treated by gastric lavage followed by activated charcoal and saline cathartic administration. The patient was always comatose, developed

progressively acute kidney and liver failure, severe blood coagulation disorders, metabolic acidosis, disseminated intravascular coagulation, shock, and myocardial failure. Death occurred 48 h after admission (3527).

Dichloropropane causes only mild irritation to the skin. Single short contact will probably be without any effects, but the intensity of the response is increased by occlusion (12).

It will cause pain and irritation when splashed into the eye but would not be expected to cause serious injury (12). A workman sprayed on the side of the face reported smarting of the eye on that side which persisted for several hours; the corneal epithelium was damaged in several small areas but recovery was prompt with no special treatment (19).

#### 12.3.2.2 Chronic Toxicologic Effects

There are no reports of chronic systemic exposures to 1,2-dichloropropane. Two cases of dermatitis resulting from occupational exposure to 1,2-dichloropropane have been documented (143).

### 12.3.3 Levels of Concern

In view of the limited data available on the adverse health effects and effect levels associated with exposure to 1,2-dichloropropane, estimates of exposure levels of concern cannot be made with any confidence. The USEPA (355) has not established an ambient water quality criterion for the protection of human health for 1,2-dichloropropane due to the insufficiency of available data. Indications of some carcinogenic activity for 1,2-dichloropropane were reported by the NTP (146) after the USEPA issued their water quality criteria. IARC (1357) lists 1,2-dichloropropane in Category 3 (no adequate data for carcinogenicity in humans; limited evidence for carcinogenicity in animals) in its weight-of-evidence ranking of potential carcinogens. EPA lists this chemical in Group B2 (sufficient evidence in animals and inadequate or no evidence in humans) (3328). Both OSHA (3539) and the ACGIH (291) have set an occupational exposure limit of 75 ppm (350 mg/m$^3$) for 1,2-dichloropropane, based on preventing systemic effects, with a short-term exposure limit (15 minutes) of 110 ppm (3539).

### 12.3.4 Hazard Assessment

1,2-Dichloropropane has not been adequately tested for chronic and subchronic toxicity, making estimation of the lowest adverse effect level difficult. Ingestion of 1000 mg/kg of 1,2-dichloropropane for 13 weeks caused injury to the liver in female rats but not males; no effects were seen in mice similarly treated at a level of 500 mg/kg (146). Ingestion of 50 mL of a cleaning solution containing unknown amounts of 1,2-dichloropropane was fatal.

Limited data indicate a capability for mutagenic activity for 1,2-dichloropropane. There are no data available on reproductive toxicity. Some evidence of carcinogenic activity for 1,2-dichloropropane was seen in $B6C3F_1$ mice, as indicated by an increased incidence of hepatocellular adenomas. A marginally increased incidence of adenocarcinomas of the mammary gland was seen in female rats; these borderline malignant lesions occurred concurrent with decreased survival and body weight gain indicating the administered dose (250 mg/kg) was toxic. There was no evidence of carcinogenicity in male rats (146).

The scarcity of data available on the adverse health effects associated with exposure to 1,2-dichloropropane linked with some indications of carcinogenic activity do not reasonably permit establishment of an acceptable daily intake for this compound.

## 12.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of 1,2-dichloropropane concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of 1,2-dichloropropane, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of 1,2-dichloropropane, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the 1,2-dichloropropane from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the 1,2-dichloropropane and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; 1,2-dichloropropane is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations, direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened, prior to the purge and trap step, to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for 1,2-dichloropropane analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Typical 1,2-dichloropropane detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.04 μg/L (Method 601) | 0.4 μg/kg (Method 8010) |
| 6.0 μg/L (Method 624) | 5.0 μg/kg (Method 8240) |
| 10.0 μg/L (Method 1624) | |
| 5.0 μg/L (Method 8240) | |
| 0.4 μg/L (Method 8010) | |

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Note: The nonsequential numbers of the references reflect the order of references as they appear in the master bibliography.

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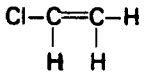
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| COMMON SYNONYMS: | CAS REG.NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25°C |
|---|---|---|
| Chloroethene<br>Chloroethylene<br>VC<br>VCM<br>Vinyl chloride | 75-01-4 $C_2H_3Cl$<br>NIOSH N0:<br>KU9625000<br>STRUCTURE:<br>  | 2.56 mg/m$^3$ ≈ 1 ppm;<br>0.391 ppm ≈ 1 mg/m$^3$<br>MOLECULAR WEIGHT:<br>62.50 |

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| REACTIVITY | Vinyl chloride reacts very slowly with atmospheric oxygen in the presence of various contaminants to produce an unstable polyperoxide that could attain hazardous levels with time (505). For compatibility classification purposes, vinyl chloride is considered to be a halogenated organic compound and a polymerizable compound. Such substances generally evolve heat, polymerize violently, evolve toxic or flammable gases, and occasionally catch fire or explode, in reactions with acids, azo or diazo compounds, hydrazines, caustics, cyanides, alkali or alkaline earth metals, certain other elemental metals and alloys, toxic metals and their compounds, nitrides, inorganic sulfides, organic peroxides or hydroperoxides, phenols or cresols. In addition, they commonly produce heat with mercaptans or other organic sulfides, heat and toxic gases with amines or strong oxidizing agents and heat and explosions with explosive materials or strong reducing agents (511). |

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|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Gas; easily liquified (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Faintly sweet | (51) |
| | • Odor Threshold: 4000.000 ppm | (67) |
| | • Density: 0.9121 specific gravity (at 20°C) | (23) |
| | • Freeze/Melt Point: -153.80°C | (68) |
| | • Boiling Point: -13.40°C | (68) |
| | • Flash Point: -77.75°C open cup | (21) |
| | • Flammable Limits: 3.60 to 33.00% by volume | (51,60,506) |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA (Cont.) | • Autoignition Temp.: 472.0°C | (51,60,506) |
| | • Vapor Pressure: 2300.00 mm Hg (at 20°C) | (23) |
| | • Satd. Conc. in Air: 7.8800E+06 mg/m$^3$ (at 20°C) | (1219) |
| | • Solubility in Water: 1100.00 mg/L (at 20°C) | (171) |
| | • Viscosity: 0.011 to 0.280 cp; minimum is for gas, 20°C; maximum is for liquid, -20°C | (3301) |
| | • Surface Tension: 23.1000 dyne/cm (at 20°C) | (3301) |
| | • Log (Octanol-Water Partition Coeff.): 1.23 (estimate) | (33) |
| | • Soil Adsorp. Coeff.: 8.20 | (652) |
| | • Henry's Law Const.: 0.69 atm · m$^3$/mol (at 20°C) | (74) |
| | • Bioconc. Factor: 2.97 (estim) 0.8 (estim) | (189,659) |

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| 'ERSISTENCE IN THE SOIL-WATER SYSTEM | Vinyl chloride is expected to be highly mobile in soil/groundwater systems. In surface soils, most of the vinyl chloride will be in the soil-air phase and removal by volatilization will be important. In deep soils, transport with soil-water is important. Transformation processes such as hydrolysis and biodegradation are not expected to be significant in natural soils. |

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| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of vinyl chloride to groundwater drinking water supplies. Data from NPL sites show that such migration has commonly occurred in the past. Inhalation resulting from surface soils may also be important. |

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| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (12, 45, 59)<br>Inhalation of vinyl chloride causes headache, dizziness, abdominal pain, numbness and tingling of the extremities. The vapors cause eye irritation. Skin contact with the liquid causes irritation and frostbite due to evaporation; vapor may cause irritation.<br><br>Acute Toxicity Studies:<br><br>INHALATION:<br>$LC_{50}$ 113,000 ppm · 2 hr — Mouse (171)<br><br>ORAL:<br>$LD_{50}$ 500 mg/kg — Rat (47)<br><br>Long-Term Effects: Liver damage, liver cancer<br>Pregnancy/Neonate Data: Negative<br>Genotoxicity Data: Sufficient evidence of genotoxicity<br>Carcinogenicity Classification:<br>IARC - Group 1 (carcinogenic to humans)<br>NTP - No data<br>EPA - Group A (human carcinogen) |

| | |
|---|---|
| HANDLING PRECAUTIONS (300) | Handling of vinyl chloride is to be conducted as outlined in 29 CFR 1910.1017. • Under 10 ppm: supplied air-respirator, demand-type with half facepiece. • 10-25 ppm: gas mask with canister which provides a 4-hour service life. • 25-100 ppm: open circuit, self-contained breathing apparatus with full facepiece in demand mode. • For complete guidelines, see 29 CFR 1910.1017. • Chemical goggles if there is probability of eye contact. • Full body protective clothing and gloves should be used. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

### AIR EXPOSURE LIMITS:

Standards

- OSHA TWA (8-hr): 1 ppm; STEL (15-min): 5 ppm. Direct contact with the liquid is prohibited.
- AFOSH PEL (8-hr TWA): 1 ppm; STEL (15-min): 5 ppm

Criteria

- NIOSH IDLH (30 min): NIOSH has recommended that the substance be treated as a potential human carcinogen.
- NIOSH REL: Lowest detectable limit
- ACGIH TLV® (8-hr TWA): 5 ppm (A1 - human carcinogen)
- ACGIH STEL (15 min): None established

### WATER EXPOSURE LIMITS:

Drinking Water Standards (3977)
MCLG: 0 μg/L
MCL : 2 μg/L

EPA Health Advisories and Cancer Risk Levels (3742)
The EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- 1-day (child): 3 mg/L
- 10-day (child): 3 mg/L
- longer-term (child): 10 μg/L
- longer-term (adult): 50 μg/L

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

WHO Drinking Water Guideline
No information available

EPA Ambient Water Quality Criteria

- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms (1E-05, 1E-06, 1E-07 cancer risk), 20 μg/L, 2.0 μg/L, 0.2 μg/L.
  - Based Based on ingestion of contaminated aquatic organisms only, (1E-05, 1E-06, 10E-7 cancer risk), 5246 μg/L, 525 μg/L, 52.5 μg/L.
  - Based on ingestion of contaminated water only (10E-04 cancer risk), 1.5 μg/L (3742).

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion established due to insufficient data.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity:
    no criterion established due to insufficient data.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:
No reference dose available.

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Vinyl chloride is listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and effluent standards and guidelines (351, 3763). Effluent guidelines exist for vinyl chloride in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), and metal finishing (3768). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

Vinyl chloride is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). Under the National Primary Drinking Water Regulations, the maximum contaminant level (MCL) for vinyl chloride is 0.002 mg/L and the maximum contaminant level goal (MCLG) is set at zero (3773, 3772). In states with an approved Underground Injection Control program, a permit is required for the injection of vinyl chloride containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

Vinyl chloride is identified as a toxic hazardous waste (U043) and listed as a hazardous waste constituent (3783, 3784). Waste streams from the organic chemicals industry (production of 1,1,1-trichloroethane, 1,2-dichloroethane and vinyl chloride) contain vinyl chloride and are listed as specific sources of hazardous wastes (3774, 3765). A non-specific source of vinyl chloride-containing hazardous waste is spent filters, spent desiccants, light ends, and filter aids from chlorinated aliphatic hydrogen production (325). Vinyl chloride is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg will be prohibited. Certain variances exist until May, 1990, for land and injection well disposal of some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786).

EPA requires that non-liquid hazardous wastes containing halogenated organic compounds (HOCs) in total concentrations greater than or equal to 1000 mg/kg or liquid hazardous wastes containing HOCs in total concentrations greater than or equal to 1% HOCs must be incinerated in accordance with the requirements of 40 CFR 264.343 or 265.343 (3782).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
Vinyl chloride is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 0.454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing vinyl chloride but these depend upon the concentration of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of vinyl chloride must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
Polyvinyl chloride is exempt from a tolerance requirement when used as a diluent or carrier in pesticide formulations applied to animals (315). Vinyl chloride vinyl-acetate copolymers are exempt from the requirement of a tolerance when used as inert binding agents, in amounts not more than 2%, for pesticide formulations applied only to soil (315). Polyvinyl chloride is exempt from the requirement of a tolerance for residues in or on cottonseed or raw agricultural commodity artichokes when used as inert controlled-release dispensers for formulations used to disrupt mating of particular worms and moths (314).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to vinyl chloride in any 8-hour shift of a 40-hour work- week shall not exceed an 8-hour time-weighted average (TWA) of 1 ppm. No employee may be exposed to concentrations greater than 5 ppm averaged over any period not exceeding 15 minutes. Direct contact with liquid vinyl chloride is prohibited. Regulations also exist for areas where vinyl chloride or polyvinyl chloride are manufactured, reacted, packaged, stored, handled or used. These include reporting, training and medical surveillance requirements (300, 301).

Clean Air Act (CAA)
Vinyl chloride is a hazardous air pollutant and is subject to national emission standards (NESHAPs) (3803).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated vinyl chloride as a hazardous material with a reportable quantity of 0.454 kg, subject to requirements for packaging, labeling and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
Vinyl chloride polymers are approved for use as indirect food additives (362). Any aerosol drug product containing vinyl chloride is a new drug and requires a new drug application (NDA) before it can be marketed (366).

- State Water Programs

ALL STATES
All state have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

ALABAMA
Alabama requires that the annual average maximum contaminant level of vinyl chloride in drinking water not exceed 0.002 mg/L. This applies to all community water systems, and non-community non-transient water systems (3015).

CALIFORNIA
California has an action level of 2 μg/L for drinking water (3098).

FLORIDA
Florida has set an MCL of 1 μg/L for drinking water (2319).

NEW MEXICO
New Mexico has a human health criterion of 0.001 mg/L for vinyl chloride in ground-water (3840).

NEW YORK
New York has a water quality standard of 5 μg/L for ground-water designated for drinking water supplies (3501).

OKLAHOMA
Oklahoma has a water quality criterion of 1.9 μg/L for ground-water (3534).

PENNSYLVANIA
Pennsylvania has a human health criterion (cancer risk level) of 0.02 μg/L for surface waters (3561).

VERMONT
Vermont has a preventive action limit of 0.2 μg/L and an enforcement standard of 2.0 μg/L for ground-water (3682)

WISCONSIN
Wisconsin has a preventive action limit of 0.0015 μg/L and an enforcement standard of 0.015 μg/L for vinlyl chloride in ground-water (3840).

Proposed Regulations

- Federal Programs

  EPA has proposed that solid wastes be listed as hazardous because they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is equal to or greater than 0.05 mg/L vinyl chloride. Final promulgation of this Toxicity Characteristic Rule is eexpected in June, 1989 (1565).

- State Water Programs

  MOST STATES
  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3683).

  CALIFORNIA
  California has proposed a maximum contaminant level of 0.5 μg/L for drinking water (3096).

  MINNESOTA
  Minnesota has proposed a Recommended Allowable Limit (RAL) of 0.15 μg/L for drinking water (3451). Minnesota has also proposed chronic criteria of 0.15 μg/L for designated surface waters, 0.14 μg/L for cold surface waters, and 0.15 μg/L for designated ground-waters for the protection of human health (3452).

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances and metals state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on Marketing and Use of Dangerous Substances (541)
Chloro-1-ethylene (monomer vinyl chlorides) may not be used as aerosol propellents for any use whatsoever.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-harmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
Vinyl chloride is listed as a Class II harmful substance and is subject to packaging and labeling regulations.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Vinyl chloride is classified as a flammable, toxic substance and is subject to packaging and labeling regulations. Vinyl chloride may contain a stablizer. If the stablizer changes the dangerous properties, this substance should be labeled in accordance to rules in Annex I and EEC-88-490, 22 July 1988.

Directive on the Approximation of the Laws, Regulations and Administrative Provisions Relating to the Classification, Packaging and Labeling of Dangerous Preparations (3991).
The labels on packages containing preparations classified as very toxic, toxic or corrosive must bear the safety advice S1/S2 and S46 in addition to the specific safety advice. If it is physically impossible to give such information, the package must be accompanied by precise and easily understood instructions.

EEC Directive-Proposed Resolution
Resolution on a Revised List of Second-Category Pollutants (545)
Vinyl Chloride is one of the second-category Pollutants to be studied by the Commission in the programme of action of the European Communities on Environment in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 13.1 MAJOR USES

About 96% of the vinyl chloride produced in the U.S. is used in the manufacture of polyvinyl chloride (PVC) and other vinyl polymers. The remainder is used in the synthesis of 1,1,1-trichloromethane. The major uses of PVC have been in the building and construction industries, in consumer goods, packaging and electrical wire insulation. In building and construction, PVC resins are used in piping, flooring, siding and windows. They are used in such consumer products as upholstery, wall coverings, appliances, stationery, footwear, outerwear, toys and sporting goods. The major uses of PVC in packaging are in plasticized film, bottles and bottle-cap liners and gaskets. The use of PVC for packaging of alcoholic beverages has been banned in the U.S. because of migration of vinyl chloride monomer into the alcohol. The U.S. Food and Drug Administration permits the use of PVC as a component of the following products when they are intended for use in contact with food: 1) adhesives; 2) resinous and polymeric coatings; 3) paper and paperboard (in contact with dry food only); and 4) semi-rigid and rigid acrylic and modified acrylic plastics (171).

The scope of this chapter will encompass vinyl chloride and polyvinyl chloride only. It does not include a discussion of vinyl chloride in combination with vinyl acetate or vinylidene chloride.

## 13.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 13.2.1 Transport in Soil/Ground-water Systems

#### 13.2.1.1 Overview

Vinyl chloride may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways of low soil concentrations can be assessed by using an equilibrium partitioning model as shown in Table 13-1. These calculations estimate the partitioning of vinyl chloride among soil particles, soil water and soil air. The portions of vinyl chloride associated with the water and air phases of the soil are more mobile than the adsorbed portion.

The estimates for the unsaturated topsoil model indicate that nearly all the vinyl chloride (97%) is expected to be present in the gaseous phase; for this portion, diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, will be a significant loss pathway. Only about 1% of the chemical is present in the soil-water phase which would be carried by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. Only a very small amount of the chemical (2%) is sorbed to the soil. In saturated, deep soils (containing no soil

TABLE 13-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR VINYL CHLORIDE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 2°C | 1.8 | 1.1 | 97.1 |
| Saturated deep soil[d] | 3.3 | 96.7 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 8.2 (Estimated by Arthur D. Little, Inc.)
c) Henry's law constant taken as 0.695 atm · $m^3$/mole at 20°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of Koc assuming 0.1% organic carbon: $K_p$ = 0.001 x $K_{oc}$.

air and negligible soil organic carbon), nearly all of the vinyl chloride (97%) is likely to be present in the soil-water phase (Table 13-1) and transported with flowing ground-water.

### 13.2.1.2 Sorption on Soils

The mobility of vinyl chloride in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. In general, sorption on soil is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Based upon an estimated octanol-water partition coefficient of 17, the soil sorption coefficient ($K_{oc}$) of vinyl chloride is estimated to be about 8. This is a very low number, indicative of very weak sorption to soils.

#### 13.2.1.3 Volatilization from Soils

Transport of vinyl chloride vapors through the air-filled pores of unsaturated soils will be the most important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31). There are no data from laboratory or field tests showing actual volatilization rates; however, the rates should be significantly faster than those for trichloroethylene or tetrachloroethylene for which some data are available.

### 13.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of vinyl chloride in soil/ground-water systems has not been investigated. In most cases, it should be assumed that the chemical will persist for months to years (or more) once it enters the saturated soil zone.

Vinyl chloride that has been released into the air will eventually undergo photochemical oxidation (10). Vinyl chloride under normal environmental conditions is not expected to undergo rapid hydrolysis. Callahan et al. (10) cite data estimating a hydrolysis half-life of less than 10 years at 25°C. Mabey et al. (33) indicate that hydrolysis is not an environmentally significant degradation pathway for this chemical.

Literature references to microbial degradation of compounds such as vinyl chloride are few. Most references indicate that low molecular weight chloroaliphatics are not rapidly metabolized in the environment (10). Studies by Hill et al. (225) using an isolated culture containing two species of bacteria and three mixed fungal populations indicated that concentrations of 20-120 mg/L of the chemical did not biodegrade over a five-week period.

### 13.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The properties and the above discussion of fate pathways suggest that vinyl chloride is highly volatile, very weakly adsorbed by soil, and has no significant potential for bioaccumulation. This compound will volatilize from soil surfaces, but that portion not removed by volatilization is likely to be mobile in ground-water. These fate characteristics suggest several potential exposure pathways.

Volatilization of vinyl chloride from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. The potential for ground-water contamination is high, particularly in sandy soil. Mitre (83) reported that vinyl

chloride has been found at 34 of the 546 National Priority List (NPL) sites. It was detected at 26 sites in ground-water, 9 sites in surface water and 4 sites in air. The potential for exposure through drinking water is confirmed by the presence of vinyl chloride in ground-water sources of drinking water in the United States. The USEPA (62, 64) reported the following results from a variety of surveys of drinking water supplies:

| Survey | No. Sampled | No. Positive | Range of Positives |
|---|---|---|---|
| State Data | 1973 | 126 | Trace - 380 μg/L |
| NOMS | 113 | 2 | 0.1 - 0.18 μg/L |
| NSP | 142 | 7 | Trace - 76 μg/L |
| GWSS (Random Data) | 466 | 1 | 1.1 μg/L |

Quantitation limit of 0.1 μg/L.

The state data include only ground-water sources and were compiled from various state reports on local contamination problems. The state data are not considered to be statistically representative of national occurrence. The National Organics Monitoring Survey (NOMS) included data from both ground- and surface water supplies, as did the National Screening Program (NSP). The USEPA (531) Ground-water Supply Survey (GWSS) is the most recent study. This survey sampled a total of almost 1000 drinking water systems using ground-water, 466 selected at random, and about 500 selected by the state as potentially contaminated. The random results suggest that vinyl chloride is found infrequently in drinking water. However, it was commonly detected by the states in their reports of local contamination problems. The USEPA (64) estimates that 0.06% of the nation's ground-water supplies are contaminated with vinyl chloride (>1 μg/L).

These results indicate that vinyl chloride has the potential for movement in soil/ground-water systems. This compound may eventually reach surface waters by this mechanism, suggesting several other exposure pathways:

- Surfaces waters may be used as drinking water supplies, resulting in direct ingestion exposure.
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposure through bioaccumulation.
- Recreational use of these waters may result in dermal exposure.

- Domestic animals may consume or be dermally exposed to contaminated ground-or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground-water for two reasons. First, the Henry's law constant for vinyl chloride suggests that it will volatilize upon reaching surface waters. Secondly, the bioconcentration factor for this compound is low, suggesting no significant bioaccumulation in aquatic organisms or domestic animals.

### 13.2.4 Other Sources of Exposure

The data presented above suggest that vinyl chloride is found in ground-water supplies used as drinking water. Coniglio et al. (223) reported that vinyl chloride was found in surface water supplies. In a summary of data available as of 1980, these authors reported that 2.3% of 133 finished surface water samples were contaminated with a mean concentration of 3.43 $\mu$g/L (median of 0.4 $\mu$g/L) and a range of 0.1 - 9.8 $\mu$g/L of vinyl chloride.

## 13.3 HUMAN HEALTH CONSIDERATIONS

### 13.3.1 Animal Studies

#### 13.3.1.1 Carcinogenicity

A 1971 report by Viola et al. (190) is apparently the earliest publication in which carcinogenic activity was ascribed to vinyl chloride. These investigators observed lung, skin and bone tumors in rats which were exposed to 30,000 ppm, 4 hours daily, 5 days per week for 12 months. Although this study contained many deficiencies, such as an excessive exposure concentration and an impure sample, it gave cause for concern and resulted in additional animal and human studies.

Numerous studies have been conducted evaluating the carcinogenicity of vinyl chloride in animals. The majority of these have been carried out by Maltoni and associates (185) in Sprague-Dawley and Wistar rats, Swiss mice and Golden hamsters. Maltoni and coworkers exposed laboratory animals 4 hours/day, 5 days/week for one year to vapor concentrations ranging from 1 ppm to 30,000 ppm or to 0.03 to 50 mg/kg bw vinyl chloride in olive oil by ingestion, 5 days/week for one year. Test animals were maintained for their lifetimes following treatment and exhibited a wide range of tumors (185). The conclusions drawn from the Maltoni studies are as follows:

1) Vinyl chloride caused tumors in all animal systems tested.

2) There was a clear-cut dose-response relationship via both the ingestion and inhalation routes.

3) The duration and schedule of treatment, as well as the species, strain and sex of the animals, greatly affected the neoplastic response.

4) Liver angiosarcomas were observed in all the animals tested. Other tumors noted in rats: nephroblastomas, brain neuroblastomas, skin carcinomas and carcinomas of the Zymbal gland, a sebaceous gland of the exterior acoustic duct. Other tumors noted in mice: pulmonary adenomas, mammary carcinomas and skin carcinomas.

5) Vinyl chloride produced carcinogenic effects on the embryo via the placenta.

Table 13-2, taken from Maltoni's summary paper (185), describes those tumors that occurred at the lowest doses. None of these tumors was noted in animals receiving 5 ppm vinyl chloride by inhalation or 0.03 mg/kg by ingestion. Additional information about these studies is available in several reviews (171, 185, 183, 184).

Based on Maltoni's data and the absorbed doses of vinyl chloride, a q1* of 2.95E-01/(mg/kg/day) has been estimated by USEPA (3736). Assuming a breathing volume of 20 $m^3$/day, 50 percent absorption rate of inhaled vinyl chloride and a bodyweight of 70 kg, concentrations associated with cancer risks of 1E-04, 1E-05, 1E-06, and 1E-07 have been estimated to be 9E-04, 9E-05, 9E-06, and 9E-07 ppm, respectively.

IARC (171) lists vinyl chloride in category 1 (sufficient evidence of human carcinogenicity) in its weight-of-evidence ranking for potential carcinogens. The NTP has not evaluated vinyl chloride.

### 13.3.1.2 Genotoxicity

Vinyl chloride is genotoxic in a number of biological systems. It induced reverse mutations of the base-pair substitution type in metabolically activated Salmonella typhimurium systems. It has produced reverse mutations in E. coli K12 and forward mutations in S. pombe and S. cerevisiae with metabolic activation. It was also mutagenic in the recessive lethal test in Drosophila melanogaster but not in tests for translocations and sex-chromosome loss. In addition, vinyl chloride induced forward mutations in CHO and V79 Chinese hamster cells in the presence of activation (569, 570, 3380). Vinyl chloride induced a clear positive response in the BALB/c-3T3 cell transformation assay (708) and in the virus-mediated Syrian hamster embryo cell transformation assay (3274). Negative results were obtained in a dominant lethal assay in CD-1 mice (562, 3290) and in the mouse spot test using C57BL/6J females mated to T-stock males (3565). The frequency of micronuclei in bone marrow cells of male and female mice exposed to the vapor phase of vinyl chloride was 10 times higher than controls (3593).

TABLE 13-2
SUMMARY OF VINYL CHLORIDE-CORRELATED TUMORS IN SPRAGUE-DAWLEY RATS AT THE LOWEST EFFECTIVE DOSE

| Dose | Tumors |
|---|---|
| 25 ppm by inhalation 4 hr/day, 5 days/wk for 52 weeks | Over 120 animals, 5 liver angiosarcomas, 4 Zymbal-gland carcinomas and 1 nephroblastoma |
| 10 ppm by inhalation 4 hr/day, 5 days/wk for 52 weeks | Over 120 animals, 1 liver angiosarcoma, 2 extra-hepatic angiosarcomas, and 2 Zymbal-gland carcinomas |
| 1 mg/kg by ingestion 5 times/wk for 52 weeks | Over 150 animals, 3 liver angiosarcomas, 1 extra-hepatic angiosarcoma, 1 hepatoma, and 5 Zymbal-gland carcinomas |
| 0.3 mg/kg by ingestion 5 times/wk for 52 weeks | Over 150 animals, 1 liver angiosarcoma and 1 hepatoma |

Source: Maltoni et al. (185)

### 13.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Animal studies using mice, rats and rabbits indicate that inhalation of vinyl chloride does not induce gross teratogenic abnormalities in offspring of females exposed 7 hours daily to concentrations ranging from 50 to 2500 ppm (128 to 6400 $mg/m^3$) (179). However, there was a statistically significant excess of minor skeletal abnormalities at 500 ppm and higher levels. Mice and rats were exposed on days 6-15 and rabbits on days 6-18 of gestation. In mice, there was a significant decrease in mean fetal weight and a significant increase in resorption rate in the 500 ppm group.

Vinyl chloride was present in fetal and maternal blood and amniotic fluid after exposure of pregnant rats to vapors ranging from 2000-12,000 ppm for 2.5 hours on day 18 of pregnancy (574). In this study, there was no teratogenicity in rats, but

there was a significant increase in resorption rate in dams exposed on days 1-9 of pregnancy to 1500 ppm for 24 hr/da. Exposure of pregnant rats to 6000 ppm vinyl chloride (4 hr/da on days 12-18 of gestation) produced various tumors in 6 of 32 offspring but did not produce tumors in the dams assessed 143 weeks later (3053).

The results of the human studies are reported in 13.3.2.2.

#### 13.3.1.4 Other Toxicologic Effects

##### 13.3.1.4.1 Short-term Toxicity

Vinyl chloride has a very low acute inhalation toxicity (2). Mice, rats and guinea pigs succumbed in a 30-minute exposure to 300,000 ppm of vinyl chloride. Deaths were due to narcosis. The investigators noted pulmonary edema but liver and kidney injury were remarkably low (186). The 2-hour $LC_{50}$ for vinyl chloride for both mice and rabbits was 113,000 ppm; for rats, 150,000 ppm and for guinea pigs, 230,000 ppm (171).

##### 13.3.1.4.2 Chronic Toxicity

Torkelson et al. (188) conducted the first studies of long-term vinyl chloride toxicity. They found that repeated exposures to 200 to 500 ppm, 7 hours daily, for 4.5 to 6 months caused histological changes in the liver and kidneys of rats and rabbits but not those of guinea pigs and dogs. All species tolerated exposure to 50 ppm for 6 months with no adverse effects. Polyvinylchloride dust was found to cause extensive lung damage in rats and guinea pigs continuously exposed for periods of 2 to 7 months (573).

### 13.3.2 Human and Epidemiologic Effects

#### 13.3.2.1 Short-term Toxicologic Effects

Acute exposure to high concentrations of vinyl chloride causes central nervous system depression. In a 1963 study by Lester et al. (182), humans exposed to 20,000 ppm for 5 minutes experienced lightheadedness, dizziness, nausea and dulling of vision. No clinical changes or abnormal neurologic responses were found after a 7.5-hour exposure to 500 ppm (181).

Skin contact with the liquid can cause irritation and frostbite due to evaporation (54). Absorption through the skin is minor; it has been estimated that a 6-foot, 90-kg man exposed dermally to 7000 ppm for 2 hours would absorb the equivalent of a 0.2 ppm inhalation exposure (189).

Grant (19) reports one instance of a corneal injury due to vinyl chloride exposure which healed within 48 hours. Vinyl chloride vapors also cause eye irritation.

Polyvinyl chloride dust causes fibrotic lung changes and altered pulmonary function tests in exposed workers (180).

### 13.3.2.2 Chronic Toxicologic Effects

There are numerous clinical indications that chronic exposure to vinyl chloride is toxic to humans. Hepatitis-like liver changes in workers exposed to vinyl chloride and PVC were first reported in 1949 (171). Other long-term effects include a decreased number of platelets in the blood, an enlarged spleen, and decreased pulmonary function (171).

A condition known as acroosteolysis has been seen almost exclusively in workers involved in cleaning vinyl chloride polymerization vessels. It consists of club-like swelling and shortening of the fingers, nodular growths on the bottom surface of the hands and forearms and Raynaud's phenomenon, a tingling of the fingers which is aggravated by cold (45). These changes may disappear when contact with vinyl chloride is eliminated (171).

Chromosome aberrations have been found in workers occupationally exposed to vinyl chloride. In most cases, aberrations consisted of breaks, fragmentations and rearrangements. The most significant abnormalities occurred in workers exposed to the highest concentrations (571). Picciano et al. (572) suggest that exposures below 15 ppm will not result in aberrations.

An increased incidence of birth defects was described for children born to parents residing near vinyl chloride polymerization plants. The largest number of defects were malformations of the CNS and genital organs, club foot and cleft lip and palate. There was also a significant increase in fetal loss rate in women whose husbands were employed in the vinyl chloride polymerization plants (575). However, reanalysis of the data and subsequent studies have not indicated a causal relationship between vinyl chloride exposure and human fetal loss or teratogenesis (3130, 3713, 3192, 3147). In direct contrast, Szentesi et al. (3695) found a significant increase in fetal death and congenital malformations in offspring of 231 polyvinyl chloride workers. No details were given; their publication was an abstract.

In 1974, the first report appeared which linked vinyl chloride exposure to human cancer (171). Since that time, epidemiological studies have shown that vinyl chloride workers are at increased risk for developing cancer. Liver angiosarcomas, brain, skin and lung tumors and tumors of the lymphatic and blood-forming systems are some of the cancers seen in exposed workers (187).

In a proportional mortality analysis of 161 deceased workers in 2 U.S. plants producing and polymerizing vinyl chloride, a 50% excess of deaths due to all cancers was found. There was a 90% increase in cancers of the liver and biliary tract, a 320% excess in brain tumors and a 60% excess in lung cancers (576). A study of the

cancer mortality of 1294 individuals with 5 or more years ot employment in jobs directly involved with vinyl chloride exposure found an excess of cancer in 4 organ systems: brain and central nervous system, respiratory system, hepatic system and the lymphatic and blood-forming system. This excess of organ-specific cancer was restricted to workers with 15 or more years since initial vinyl chloride exposure (577). Numerous other epidemiology studies have been conducted with similar results (171). It is noteworthy that in all of the vinyl chloride-induced angiosarcoma cases reported to date, individuals were first exposed between 1941 and 1967 when typical industry concentrations were estimated to be 300 to 1000 ppm (780 to 2600 $mg/m^3$). A study of the relationship between exposure and latency has shown that those with higher total exposures have shorter latency periods. This relationship is strengthened when total exposure is weighted according to the years in which exposure occurred with exposures during the 1940's and 1950's weighing more heavily than those of the 1960's and 1970's. It appears that vinyl chloride-induced angiosarcomas are less likely to occur at levels below 50 ppm (128 $mg/m^3$) while levels greater than 200 ppm (512 $mg/m^3$) induce hepatic angiosarcoma in the shortest time period (187).

In addition to the large number of workers occupationally exposed to vinyl chloride, individuals residing near PVC processing plants may also be at risk. Five cases of angiosarcoma of the liver were diagnosed in persons living in the vicinity of vinyl chloride fabrication and polymerization plants for 8 to 62 years prior to the diagnosis of the disease (189).

### 13.3.3 Levels of Concern

Based on the evidence of liver tumors induced in rats inhaling vinyl chloride, the USEPA has specified an ambient water quality criterion for this compound of zero. In that attainment of a zero concentration level may be infeasible in some cases, the concentrations of vinyl chloride in water calculated to result in incremental lifetime cancer risks of 1E-05, 1E-06 and 1E-07 from ingestion of both water and contaminated aquatic organisms were estimated to be 20, 2.0 and 0.2 $\mu g/L$, respectively (355). Risk estimates are expressed as a probability of cancer after a lifetime consumption of two liters of water per day and consumption of 6.5 g of contaminated fish per day. Thus a risk of 1E-05 implies that a lifetime daily consumption of two liters of drinking water and 6.5 g of contaminated fish at the criterion level of 20 $\mu g/L$ vinyl chloride would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 people exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

IARC (171) lists vinyl chloride in category 1 (sufficient evidence of human and animal carcinogenicity) in its weight-of-evidence ranking of potential carcinogens. The EPA lists the chemical in Group A (sufficient evidence of human and animal carcinogenicity).

The OSHA (3539) standard for vinyl chloride is 1 ppm over an 8-hour period and a ceiling of 5 ppm averaged over a period not exceeding 15 minutes. Direct contact with the liquid is prohibited. The ACGIH (3005) lists vinyl chloride as a human carcinogen and suggests a TLV® of 5 ppm (TWA).

### 13.3.4 Hazard Assessment

Vinyl chloride is a human carcinogen. The target organs include the liver, brain and lungs, and probably the lymphohematopoietic system. Cases of liver angiosarcoma have been reported among individuals employed in vinyl chloride polymerization facilities. Adenomas and adenocarcinomas of the lung, angiosarcomas of the liver, lymphomas and neuroblastomas of the brain have been induced in laboratory animals exposed to vinyl chloride by inhalation. The USEPA (667) has calculated an upper-limit incremental unit cancer risk of 1.75E-02 $(mg/kg/day)^{-1}$ for vinyl chloride.

Numerous reports attest to the genotoxic capability of vinyl chloride in bacteria, yeast and mammalian cells. Animals studies provide no indications of teratogenic effects; however, a report of increased birth defects in children born to parents residing near vinyl chloride plants suggests the need for further investigation.

Acroosterolysis, dissolution of the bones of the fingers, is also seen in vinyl chloride and polyvinyl chloride production workers. Chronic exposure may also cause hepatic damage, deceased pulmonary function and an enlarged spleen.

## 13.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of vinyl chloride concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of vinyl chloride, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of vinyl chloride, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the vinyl chloride from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the vinyl chloride and transfer it onto a gas

chromatographic (GC) column. The GC column is programmed to separate the volatile organics; vinyl chloride is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations, direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

In addition to all these methods, a rapid procedure based on GC with flame ionization detection has been reported (3131). In this method the sample is purged with helium and the volatiles are trapped directly on the analytical column which is cooled with liquid nitrogen.

The EPA procedures recommended for vinyl chloride analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Some typical vinyl chloride detection limits that can be obtained in aqueous samples (including wastewaters without interferences) and non-aqueous samples (wastes. soils, etc.) are shown below. The Method 624 detection limit was not determined for vinyl chloride in aqueous samples. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.18 μg/L (Method 601) | 1.8 μg/kg (Method 8010) |
| 10.0 μg/L (Method 1624) | 10.0 μg/kg (Method 8240) |
| 10.0 μg/L (Method 8240) | |
| 1.8 μg/L (Method 8010) | |

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Note: The nonsequential numbers of the references reflect the order of references as they appear in the master bibliography.


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| COMMON SYNONYMS: | CAS REG. NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25°C (12) |
|---|---|---|
| 1,1-DCE<br>1,1-Dichloroethene<br>1,1-Dichloroethylene<br>VDC<br>Vinylidene chloride<br>Vinylidene dichloride | 75-35-4 $C_2H_2Cl_2$<br>NIOSH NO.:<br>KV9275000<br>STRUCTURE:<br>$Cl-C(=CH_2)-Cl$ | 3.97 mg/m³ ≈ 1 ppm;<br>0.252 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT:<br>96.95 |

| | |
|---|---|
| REACTIVITY | Compounds such as 1,1-dichloroethylene are considered halogenated organics and polymerizable materials for purposes of compatibility classification. Such substances generally evolve heat, polymerize violently, evolve toxic or flammable gases, and occasionally catch fire or explode in reactions with acids, azo or diazo compounds, hydrazines, caustics, cyanides, alkali or alkaline earth metals, certain other elemental metals and alloys, toxic metals and their compounds, nitrides, inorganic sulfides, organic peroxides or hydroperoxides, phenols or cresols. In addition, they commonly produce heat with mercaptans or other organic sulfides, heat and toxic gases with amines or strong oxidizing agents, and heat and explosions with explosive materials or strong reducing agents (511). |

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|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Mild sweet | (54) |
| | • Odor Threshold: 500.000 to 1000.000 ppm | (12) |
| | • Density: 1.2140 g/mL (at 20°C) | (21) |
| | • Freeze/Melt Point: -122.60°C | (21) |
| | • Boiling Point: 31.60°C | (21) |
| | • Flash Point: -28.00°C closed cup | (21) |
| | • Flammable Limits: 5.60 to 16.00 % by volume | (51) |
| | • Autoignition Temp.: 458.0 to 570.0°C | (51,60,510) |
| | • Vapor Pressure: 500.00 mm Hg at 20°C | (67) |

| | |
|---|---|
| PHYSICO-CHEMICAL DATA | • Satd. Conc. in Air: 2.6400E+06 mg/m$^3$ (at 20°C) (67)<br>• Solubility in Water: 400.00 mg/L (at 20°C) (59)<br>• Viscosity: 0.330 cp (at 20°C) (21)<br>• Surface Tension: 24.0000 dyne/cm (59)<br>(at 15°C) (59)<br>• Log (Octanol-Water Partition Coeff.): 2.13 (29)<br>• Soil Adsorp. Coeff.: 65.00 (652)<br>• Henry's Law Const.: 0.154 atm · m$^3$/mol (at 20°C) (74)<br>• Bioconc. Factor: 6.40 (estim) (659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL-WATER SYSTEM | 1,1-Dichloroethylene is expected to be highly mobile in the soil/groundwater system; sorption of 1,1-dichloroethylene onto soils is weak. Volatilization of material near the surface or in the soil-air compartment is expected to be significant. Transformation processes are not expected to be significant in natural soils. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from soil-water systems is the migration of 1,1-dichloroethylene to groundwater sources of drinking water supplies. Data suggest that such migration has occurred in the past. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (12)<br>Exposure to high vapor concentrations results in CNS depression, which may progress to unconsciousness with prolonged exposure. The liquid is moderately irritating to the eyes, causing pain, conjunctival irritation and possible transient injury. The liquid is irritating to the skin after only a few minutes contact. |

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|---|---|
| HEALTH HAZARD DATA (Cont.) | Acute Toxicity Studies:<br><br>INHALATION:<br>$LC_{50}$ 500 ppm · 4 hr — Rat (564)<br><br>ORAL:<br>$LD_{50}$ 200 mg/kg — Rat (47)<br>$LD_{50}$ 194 mg/kg — Mouse (3603)<br><br>Long-Term Effects: Liver and kidney damage<br>Pregnancy/Neonate Data: Negative<br>Genotoxicity Data: Evidence of genotoxic activity.<br>Carcinogenicity Classification:<br>IARC - Group 3 (not classifiable as to its carcinogenicity to humans)<br>NTP - Negative evidence in rats and mice<br>EPA - Group C (possible human carcinogen) |

| | |
|---|---|
| HANDLING PRECAUTIONS (3306) | Handle chemical only with adequate ventilation. • A full face mask should be used in areas of excessive vapor concentrations. • Use chemical goggles if there is probability of eye contact with the liquid. • Protective clothing and gloves are indicated. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 1 ppm
- AFOSH PEL (8-hr TWA): 1 ppm; STEL (15-min): 3 ppm

Criteria
- NIOSH IDLH (30 min): None established
- NIOSH REL: None established
- ACGIH TLV® (8-hr TWA): 5 ppm
- ACGIH STEL (15 min): 20 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards (3977)
MCLG: 7 μg/L
MCL : 7 μg/L

ENVIRONMENTAL AND OCCUPAONAL STANDARDS AND CRITERIA (Cont.)

EPA Health Advisories and Cancer Risk Levels (3977)
The EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- 1-day (child): 2 mg/L
- 10-day (child): 1 mg/L
- longer-term (child): 1 mg/L
- longer-term (adult): 4 mg/L
- lifetime (adult): 7 μg/L

WHO Drinking Water Guideline (666)
A health-based guideline for drinking water of 0.3 μg/L is recommended for 1,1-dichloroethylene. A daily per capita consumption of two liters of water was assumed.

EPA Ambient Water Quality Criteria

- Human Health (3770)
  - Based on ingestion of contaminated water and aquatic organisms (1E-05, 1E-06, 1E-07 cancer risk), 0.33 μg/L, 0.033 μg/L, 0.0033 μg/L. Based on ingestion of contaminated aquatic organisms only, (1E-05, 1E-06, 1E-07 cancer risk), 18.5 μg/L, 1.85 μg/L, 0.185 μg/L.

- Aquatic Life (3770)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 11,600 μg/L dichloroethylenes.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 224,000 μg/L dichloroethylenes.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:
9.000E+00 μg/kg/day (3744)

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

1, 1-Dichloroethylene is designated a hazardous substance. It has a reportable quantity (RQ) limit of 2270 kg (347, 3764). It is also listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), and steam electric power generating (3802). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

1,1-Dichloroethylene is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). Under the National Primary Drinking Water Regulations, the maximum contaminant level (MCL) and the maximum contaminant level goal (MCL) for 1,1-dichloroethylene are both 7 μg/L (3773, 3772). In states with an approved Underground Injection Control program, a permit is required for the injection of 1,1-dichloroethylene-containing wastes designated as hazardous under RCRA (295)

Resource Conservation and Recovery Act (RCRA)

1, 1-Dichloroethylene is identified as a toxic hazardous waste (U078) and listed as a hazardous waste constituent (3783, 3784). A non-specific source of 1, 1-dichloroethylene-containing waste is the production of chlorinated aliphatic hydrocarbons (325). Waste streams from the following industries contain 1, 1-dichloroethylene and are listed as specific sources of hazardous wastes: organic chemicals (production of 1, 2-dichloroethane, vinyl chloride and 1, 1, 1-trichloroethane) and inorganic chemicals (chlorine production) (3774, 3765). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). 1,1-Dichloroethylene is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their gound-water for chemicals on this list when suspected contamination is first detected and annually thereafter.

Toxic Substances Control Act (TSCA)
EPA has proposed that manufacturers and processors of 1,1-dichloroethylene be required to conduct distribution excretion and metabolism (DEM) studies and a two-year inhalation oncogenicity bioassay in mice (3769).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
1, 1-Dichloroethylene is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 2270 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing 1, 1-dichloroethylene but these depend upon the concentration of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of 1, 1-dichloroethylene must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Occupational Safety and Health Act (OSHA)
Employee exposure to 1, 1-dichloroethylene in any 8-hour work shift of a 40-hour work week shall not exceed an 8-hour time-weighted average (TWA) of 1 ppm (3539).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated 1, 1-dichloroethylene as a hazardous material with a reportable quantity of 2270 kg, subject to requirements for packaging labeling and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
1, 1-Dichloroethylene polymers are approved for use as indirect food additives (3209).

- State Water Programs

ALL STATES

All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

ALABAMA

Alabama requires that the annual average maximum contaminant level of 1,1-dichloroethylene in drinking water not exceed 7 μg/L. This applies to all community water systems, and non-community non-transient water systems (3015).

CALIFORNIA

California has an MCL and an action level of 6 ug/L for 1,1-dichloroethylene in drinking water (3096, 3098).

CONNECTICUT

Connecticut has a quantification limit of 2 μg/L for drinking water (3137).

MISSOURI

Missouri has a water quality criterion of 7 μg/L for surface waters that supply drinking waters (3457).

NEW YORK

New York has an MCL of 5 μg/L for drinking water (3501).

PENNSYLVANIA

Pennsylvania has set a human health criterion (cancer risk level) of 0.06 μg/L for surface waters (3561).

RHODE ISLAND

Rhode Island has an acute freshwater quality guideline of 580 μg/L and a chronic guideline of 13 μg/L for the protection of aquatic life in surface waters. These guidelines are enforceable under Rhode Island state law (3590).

VERMONT

Vermont has a preventive action limit of 0.7 μg/L and an enforcement standard of 7.0 μg/L for 1,1-dichloroethylene in groundwater (3682).

WISCONSIN

Wisconsin has a preventive action limit of 0.024 μg/L and an enforcement standard of 0.24 μg/L for 1,1-dichloroethylene in groundwater (3840).

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA) EPA has proposed that solid wastes be listed as hazardous because they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is greater than or equal to 0.1 mg/L 1,1-dichloroethylene. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Water Programs

  MOST STATES

  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officer is advised. Changes are projected for 1989-90 (3452).

  MINNESOTA

  Minnesota has proposed a Sensitive Acute Limit (SAL) of 7568 μg/L for designated surface waters, and chronic criteria of 5.5 μg/L for designated surface waters and 7 ug/L for designated ground-waters. These criteria are for the protection of human health (3452).

  NEW JERSEY

  New Jersey has proposed an MCL of 2 μg/L for 1,1-dichloroethylene in drinking water, and a water quality criterion of 2 μg/L for class FW2 surface waters (3497, 3496).

EEC Directives

Directive on Ground-Water (538)

Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparation (Solvents) (544)
1, 1-Dichloroethylene is listed as a Class II/b harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,1-Dichloroethylene is classified as a flammable, harmful substance and as subject to packaging and labeling regulations.

EEC Directives-Proposed Resolution
Resolution on the Revised List of Second-Category Pollutants (545)
1,1-Dichloroethyle is one of the second-category pollutants to be studied by the Commissin in the programme of action of the European Communities on Environm,ent in order to reduce pollution and nuisances in the air and water. Risk to human health and the environment, limits of pollutant levels in the environment, and determination of quality standards to be applied will be assessed.

## 14.1 MAJOR USES

The primary use of the 1,1-dichloroethylene produced in the U.S. is in the manufacture of copolymers of high 1,1-dichloroethylene content; the other major monomer used is vinyl chloride. The Food and Drug Administration has approved the use of these copolymers in various products intended for use in contact with food, such as industrial and household food wraps and paper coatings for food packages. The copolymers are also used for coating the interiors of ship tanks and railroad tank cars as well as steel piles and structures.

1,1-Dichloroethylene is also used as an intermediate in the production of 1,1,1-trichloroethane and in the manufacture of modacrylic fibers where it is combined with acrylonitrile (171). Since 1,1-dichloroethylene polymerizes readily and can form explosive peroxides, 180 to 220 mg/kg of the monomethyl ether of hydroquinone (MEHQ) or 0.6-0.8% phenol (175) are generally added to commercial preparations of 1,1-dichloroethylene to inhibit these reactions.

## 14.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 14.2.1 Transport in Soil/Ground-water Systems

#### 14.2.1.1 Overview

The 1,1-isomer of dichloroethylene may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways for low soil concentrations can be assessed by an equilibrium partitioning model as shown in Table 14-1. These calculations predict the partitioning of 1,1-dichloroethylene among soil particles, soil water and soil air. The 1,1-dichloroethylene associated with the water and air phases of the soil are more mobile than the adsorbed portion.

The estimates for the unsaturated topsoil model indicate that a significant portion of 1,1-dichloroethylene is expected to be in the gaseous phase (59%); for this portion, diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind will be a significant loss pathway. For the minor amount (3%) of the chemical present in the soil-water phase, migration by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion will be possible.

In saturated, deep soil (containing no soil air and negligible soil organic carbon), a much higher fraction of the 1,1-dichloroethylene (78%) is likely to be present in the soil-water phase (Table 14-1) and transported with flowing ground-water.

TABLE 14-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR
1,1-DICHLOROETHYLENE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 20°C | 38.1 | 3.1 | 58.8 |
| Saturated deep soil[d] | 21.4 | 78.6 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 65 (Estimated by Arthur D. Little, Inc.).
c) Henry's law constant taken as 0.154 atm · $m^3$/mol at 200°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

### 14.2.1.2 Sorption on Soils

The mobility of 1,1-dichloroethylene in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water, and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Based upon its octanol-water partition coefficient of 135, the soil sorption coefficient ($K_{oc}$) is estimated to be 65. This is a relatively low number indicative of weak sorption to soils.

### 14.2.1.3 Volatilization from Soils

Transport of 1,1-dichloroethylene vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

There are no data from laboratory or field tests showing actual volatilization rates; however, the rates should be faster than those for trichloroethylene for which some data are available.

### 14.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of 1,1-dichloroethylene in soil/ground-water systems has not been investigated. In most cases, it should be assumed that the chemical will persist for months to years (or more). 1,1-Dichloroethylene that has been released into the air will undergo fairly rapid photochemical oxidation (10).

1,1-Dichloroethylene under normal environmental conditions is not expected to undergo rapid hydrolysis. Mabey et al. (33) indicate that hydrolysis is not an environmentally significant degradation pathway for this chemical.

Literature references to microbial degradation of compounds such as 1,1-dichloroethylene are very few. Most references indicate that low molecular weight chloroaliphatics are not rapidly metabolized in the environment (10). However, significant degradation may be achieved in biological waste water treatment plants where the microbes have become acclimated to the chemical. Tabak et al. (55), for example, found significant 1,1-dichloroethylene biodegradation with gradual adaptation at levels of 5 and 10 mg/L in a static culture flask-screening procedure. However, in most soil/ground-water systems, such aerobic degradation would be of minimal importance because of the low concentration of microorganisms (at depth) and the low dissolved oxygen (anaerobic) conditions. No studies have been done to investigate the possibility of anaerobic biodegradation.

### 14.2.3 Primary Routes of Exposure From Soil/Ground-water Systems

The above discussion of fate pathways suggests that 1,1-dichloroethylene is highly volatile, weakly adsorbed by soil and has a low potential for bioaccumulation. This compound may volatilize from soil surfaces, but that portion not subject to volatilization is likely to be mobile in ground-water. These fate characteristics suggest several potential exposure pathways.

Volatilization of 1,1-dichloroethylene from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. In addition, the

potential for ground-water contamination is high, particularly in sandy soil. Mitre (83) reported that 1,1-dichloroethylene has been found at 17 of the 546 National Priority List (NPL) sites. It was detected at 15 sites in ground-water and 3 sites in surface water. It was not detected in the air at the sites, although air sampling was generally limited.

This compound was reported with a similar degree of frequency in the USEPA (531) Ground-water Supply Survey (GWSS). This survey examined 945 finished water supplies that use ground-water sources. The results for 1,1-dichloroethylene are summarized below:

| Sample Type | Occurrences* No. | % | Median of Positives (μg/L) | Maximum (μg/L) |
|---|---|---|---|---|
| Random | | | | |
| Supplies serving <10,000 people (280 samples) | 4 | 1.4 | 1.2 | 6.3 |
| Supplies serving >10,000 people (186 samples) | 5 | 2.7 | 0.28 | 2.2 |
| Non-Random | | | | |
| Supplies serving <10,000 people (321 samples) | 5 | 1.6 | 0.35 | 3.0 |
| Supplies serving >10,000 people (158 samples) | 10 | 6.3 | 0.34 | 0.64 |

*Samples having levels over quantification limit of 0.2 μg/L.

The random results are intended to statistically represent the U.S. ground-water drinking water supplies. The non-random samples were chosen by the states as being potentially contaminated. 1,1-Dichloroethylene has also been detected in the National Organic Monitoring Survey (NOMS) (90).

The survey results indicate that 1,1-dichloroethylene has the potential for movement in soil/ground-water systems. This compound may eventually reach surface waters by this mechanism, suggesting several other exposure pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion as well as inhalation exposure.
- Aquatic organisms residing in these waters may bioaccumulate this chemical and be consumed, also resulting in ingestion exposures.
- Recreational use of these waters may result in dermal exposures.

- Domestic animals may consume or be dermally exposed to contaminated ground- or surface waters; the consumption of meats and poultry could then result in ingestion exposure.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated ground-water for two reasons. First, the Henry's law constant for 1,1-dichloroethylene suggests that it will volatilize, although not completely, upon reaching surface waters. Secondly, the bioconcentration factor for this compound is low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 14.2.4 Other Sources of Exposure

The data presented above on the Ground-water Supply Survey (531) suggest that 1,1-dichloroethylene is found in a limited number of ground-water supplies used for drinking water. Coniglio et al. (223) reported that 1,1-dichloroethylene was found in surface water supplies. In a summary of data available as of 1980, these authors reported that 1.9% of the 103 finished water supplies were contaminated with a mean concentration of 0.36 $\mu$g/L.

The volatility of this compound suggests it may be found in air. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For 1,1-dichloroethylene, they had data for 339 locations. In urban and suburban locations, the median concentration was 0.02 $\mu g/m^3$. In source-dominated areas, the median concentration was 14 $\mu g/m^3$. These results suggest inhalation exposure to persons, particularly in source-dominated areas.

## 14.3 HUMAN HEALTH CONSIDERATIONS

### 14.3.1 Animal Studies

#### 14.3.1.1 Carcinogenicity

The route of exposure may be one of the important factors influencing the carcinogenicity of 1,1-dichloroethylene in laboratory animals. It has been reported to be carcinogenic when administered by inhalation but not carcinogenic when administered orally. A 104-week chronic exposure study in F344/N rats and B6C3F$_1$/N mice was carried out by the National Toxicology Program in which the animals were given 1,1-dichloroethylene in corn oil by gavage at dosage levels of 1 or 5 mg/kg in rats and 2 or 10 mg/kg in mice. Controls received corn oil alone. The only observed increase in tumor incidence occurred in low-dose female mice where there was an increased number of lymphomas and leukemias; the increases were not considered to be related to 1,1-dichloroethylene administration (173).

In a 2-year drinking water study, no carcinogenic effects were observed in rats fed 50 to 200 ppm 1,1-dichloroethylene in drinking water. This is equivalent to 7-20 mg/kg/day in males and 9-30 mg/kg/day in females (561).

When given by inhalation to rats and mice, 1,1-dichloroethylene induced malignant tumors. Maltoni et al. noted renal adenocarcinomas in 16% of male and 0.6% of female Swiss mice exposed to 25 ppm 1,1-dichloroethylene 4 hours daily, 5 days per week for 52 weeks. No tumors were seen in mice similarly exposed to 10 ppm or in controls (560).

The same investigators also found an increased incidence of mammary fibroadenomas or carcinomas in female Sprague-Dawley rats exposed to 10-150 ppm, 4 hours per day, 5 days a week for 52 weeks. However, no dose-response relationship was found (560).

IARC (171) has listed 1,1-dichloroethylene in category 3 (not classifiable as to its carcinogenicity for humans) in its weight-of-evidence ranking for potential carcinogens.

### 14.3.1.2 Genotoxicity

When 1,1-dichloroethylene was studied in a dominant lethal test in CD-1 male mice exposed to 10-50 ppm, 6 hours per day for 5 days, no adverse effects were observed (562). There were no cytogenetic changes in the lymphocytes of rats exposed 6 hours per day for 6 months to 0-75 ppm 1,1-dichloroethylene (563). Sawada et al observed increases in chromosomal aberrations and sister chromatid exchanges in Chinese hamster lung cells treated with 1,1-dichloroethylene in vitro, but only with metabolic activation. They found no increase in micronuclei in the bone marrow of male mice gavaged with this compound, nor did they find an increase in micronuclei of fetal liver or fetal blood 24 hours after pregnant mice were injected with 1,1-dichloroethylene on the eighteenth day of gestation (3613). Conflicting results have been reported with Salmonella; it was found negative in preincubation assays in four strains with or without activation (3469) but was positive when strain TA100 was exposed to vapors of 1,1-dichloroethylene in a desiccator incubation assay with metabolic activation (3422). The chemical was also found to be mutagenic in a reverse mutation assay using E. coli K12 involving the arginine gene. The presence of microsomal enzymes from phenobarbital induced mouse liver and the NADPH generating system were necessary for the mutagenic effect.

### 14.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Murray et al. (172) evaluated the teratogenic potential of inhaled 1,1-dichloroethylene in rats and rabbits. Exposure was for 7 hr/day. Rats were exposed to 20, 80 or 160 ppm on days 6 through 15 of gestation. Rabbits were exposed to 80 or 160 ppm on days 6 through 18. In rats, inhalation of 80 or 160 ppm produced significant maternal effects such as decreased weight gain and food consumption. No teratogenic effects were seen although some evidence of fetotoxicity (delayed

ossification and wavy ribs) was observed. In rabbits, 160 ppm caused a significant increase in resorptions in the dams and minor skeletal variations in the offspring. Low-dose rats and rabbits showed little evidence of maternal or fetal toxicity. When Short et al. (3650) exposed rats (up to 460 ppm) and mice (15 ppm) to 1,1-dichloroethylene vapors for 23 hr/day from days 6-16 of gestation, no significant malformations occurred. However, no pregnant mice survived 130 or 300 ppm, and no litters were produced in mice at 30 or 55 ppm. Negative results were obtained in dominant lethal studies by Anderson et al. (3026) and Short et al. (3651) using male mice and rats, respectively. Mice were exposed 6 hr/day for 5 days at up to 50 ppm. Rats received 55 ppm 6 hr/da, 5 da/wk for 11 wk and experienced a reduced ratio of pregnant to mated females. Using the oral route, Murray et al. (172) exposed rats to drinking water containing 200 ppm 1,1-dichloroethylene on days 6 through 15 of gestation. There was no evidence of toxicity to the dams or their offspring.

Ingestion of drinking water containing up to 200 ppm did not affect the reproductive capacity of rats through three generations (770). Alumot et al. (3019), using rats, fumigated feed with 1,1-dichloroethylene up to 500 ppm. Approximately 60 to 70 percent of the substance was actually consumed. No changes in fetal mortality or weight were found over a two year period. During 1980 and 1981, an exceptionally high number of birth defects and miscarriages occurred in Los Paseos, California, an area serviced by a water well which had been contaminated by 1,1,1-trichloroethane and dichloroethylene (3606). The chemicals had leaked from storage tanks of a semiconductor manufacturer. Later, 57 ppb of 1,1-dichloroethylene was identified in the well water. There is no clear association between the unfavorable reproductive outcomes and the solvents in the drinking water because of too many uncertainties in the retrospective epidemiological studies. Heath (3282) conducted a study at Love Canal in New York, an area contaminated with dichloro- ethylene and other chemicals. No clear increased incidence of abortion, birth defects, or low infant birth weight was observed in women living next to the Canal.

### 14.3.1.4 Other Toxicologic Effects

#### 14.3.1.4.1 Short-term Toxicity

Results reported for acute toxicity studies in animals have been highly variable. Toxic concentrations are dependent on dietary parameters and hepatic glutathione content. Four-hour inhalation $LC_{50}$ values were 10,000-15,000 ppm in fed rats as compared to 500-2500 ppm in fasted rats (564). Fasted rats exposed to 200 ppm for 4 hours sustained liver and kidney damage whereas fed rats did not. This may be due to the fact that fasting prior to exposure reduced the number of detoxifying pathways and enhanced binding of metabolites to liver and kidney tissue (12). According to one of the reviewers, this may be related to the glutathione level in liver and kidney tissue under fasting conditions. Since the major route of detoxification for 1,1-dichloroethylene is by conjugation with liver glutathione, any interaction with this pathway which serves to deplete liver glutathione concentrations would increase hepatotoxicity.

In one short-term study, rabbits, monkeys, rats and guinea pigs were exposed to vapor concentrations of 100 ppm, 8 hours daily, 5 days per week, for 6 weeks. No visible signs of toxicity or histopathological changes were evident (565). However, twenty 6-hour exposures to 500 ppm caused liver cell degeneration, retarded weight gain and nasal irritation in rats (566).

The 1,1-isomer of dichloroethylene is irritating to the eyes and skin of rabbits after direct contact. The peroxide inhibitor added to commercial 1,1-dichloroethylene may be partly responsible for this irritation (12).

#### 14.3.1.4.2 Chronic Toxicity

Chronic exposure to 1,1-dichloroethylene results primarily in liver and kidney injury. Inhalation studies using rats, guinea pigs, dogs, rabbits and monkeys exposed to a mean level of 47 ppm continuously for 90 days showed significant mortality and liver damage (565). In another study, rats, rabbits, guinea pigs and dogs exposed 8 hours per day, 5 days per week for 6 months, were found to have injury of the liver and kidney at vapor concentrations of 50 and 100 ppm. There was minimal injury to these organs at a concentration of 25 ppm (12). Dogs fed doses ranging from 6.25 to 25 mg/kg/day exhibited no adverse effects after 97 days of treatment (561), and rats given 6-8 mg/kg bw/day of 1,1-dichloroethylene in their drinking water for 90 days showed no adverse effects other than minimal liver changes (171).

### 14.3.2 Human and Epidemiologic Studies

#### 14.3.2.1 Short-term Toxicologic Effects

Acute exposure to high concentrations of 1,1-dichloroethylene in air results in central nervous system depression and narcosis. In humans, a vapor concentration of 4000 ppm rapidly produces symptoms of dizziness and incoordination which may progress to unconsciousness (17). It has been suggested that human exposure to 1,1-dichloroethylene can result in lesions of the trigeminal nerve causing motor weakness of the jaw, eye and tongue muscles. Subsequent evaluation suggested that the toxic agent was either mono- or dichloroacetylene which was a contaminant of 1,1-dichloroethylene (175).

Skin contact with 1,1-dichloroethylene results in irritation which may be due in part to the addition of hydroquinone monomethyl ether to commercial formulations to inhibit peroxide formation. Where leaks occur, 1,1-dichloroethylene will evaporate leaving the inhibitor to accumulate until it reaches a concentration capable of causing burns. Caution should be used with regard to contaminated clothing which should be removed immediately (12).

If 1,1-dichloroethylene comes in contact with the eye, it will cause pain, conjunctival irritation and transient corneal injury. However, a high concentration of the

hydroquinone monomethyl ether inhibitor in the 1,1-dichloroethylene may cause eye injury (12).

### 14.3.2.2 Chronic Toxicologic Effects

No abnormal findings were related to 1,1-dichloroethylene exposure in a population of 138 workers exposed to concentrations ranging from 5 to 20 ppm time-weighted-averages. The length of exposure was not given (567).

### 14.3.3 Levels of Concern

For the maximum protection of human health from the potential carcinogenic effects due to exposure to 1,1-dichloroethylene through ingestion of contaminated water and aquatic organisms that have bioaccumulated this compound, the USEPA (355) has specified an ambient water quality criterion of zero for this compound. Since attainment of a zero (not measurable) level may be infeasible in some cases, the concentration of 1,1-dichloroethylene in water calculated to result in incremental lifetime cancer risks of 10E-05, 10E-06 and 10E-07 from ingestion of both water and contaminated aquatic organisms were estimated to be 0.33, 0.033 and 0.0033 μg/L, respectively. Risk estimates are expressed as a probability of cancer after a lifetime daily consumption of two liter of water and 6.5 g of fish that have bioaccumulated the compound. Thus, a risk of 10E-05 implies that lifetime daily consumption of two liters of drinking water and 6.5 g of contaminated fish at the criterion level of 0.33 μg/L of 1,1-dichloroethylene would be expected to produce one excess case of cancer above the normal background incidence for every 100,000 individuals exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

IARC (3317) lists 1,1-dichloroethylene in category 3 (not classifiable as to its carcinogenicity for humans) in its weight-of-evidence ranking for potential carcinogens. The USEPA lists it in Group C.

The USEPA (3739, 3806) has set a MCL (maximum contaminant level) of 7 μg/L as the formal drinking water standard for 1,1-dichloroethylene. The following health advisories have been developed by the EPA: 10-Kg child: one-day, 2 mg/L, ten-day, 1 mg/L, longer-term, 1 mg/L. 70-Kg adult: lifetime, 7 μg/L, longer term, 4 mg/L, DWEL (Drinking Water Equivalent Level) 400 μg/L. A lifetime exposure concentration protective of adverse, non-cancer health effects, that assumes all of the exposure to a contaminant is from a drinking water source.

The World Health Organization (666) has recommended a health-related guideline of 0.3 μg/L of 1,1-dichloroethylene for drinking water.

OSHA has established an 8-hr TWA of 1 ppm for 1,1-dichloroethylene (3539). The ACGIH (3005) recommends a threshold limit value of 5 ppm (20 $mg/m^3$) and a short-term exposure limit of 20 ppm (80 $mg/m^3$).

### 14.3.4 Hazard Assessment

Exposure to 25 ppm of 1,1-dichloroethylene by inhalation has been linked to the production of malignant renal tumors in mice (560); no effects were seen at 10 ppm. Carcinogenicity studies of this compound administered orally, either by gavage (10 mg/kg) or in the drinking water (200 ppm) have so far produced negative results (173, 561). The USEPA (667) noting the inadequate evidence, calculated an upper-limit incremental unit cancer risk of 0.147/(mg/kg/day) for 1,1-dichloroethylene.

The liver and kidney are the main target organs of 1,1-dichloroethylene toxicity, as indicated by subchronic studies in several species (565, 12, 561, 171). However, dogs fed up to 25 mg/kg for 3 months exhibited no significant effects (561). No effects on reproduction were seen in 3 generations of rats given up to 200 ppm 1,1-dichloroethylene in their drinking water (40 mg/kg/day) (770), and there are no indications that the compound is teratogenic, although it is fetotoxic at levels toxic to maternal animals (172). Mutagenic findings were weakly positive in bacteria but negative in a mouse dominant-lethal test (12, 562).

In humans, the predominant effect of acute exposure to high vapor concentrations (e.g., 4000 ppm) of 1,1-dichloroethylene is depression of the central nervous system, which may progress to unconsciousness if exposure is prolonged (17). A study of 138 industrial workers exposed to 1,1-dichloroethylene suggested no effects on mortality or health parameters. Genotoxic findings were conflicting in bacteria (12, 3422), positive in mammalian cells in culture (3613) but negative in a mouse dominant-lethal test and in a mouse micronucleus test (12, 562, 3613).

## 14.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of 1,1-dichloroethylene concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of 1,1-dichloroethylene, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of 1,1-dichloroethylene, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, 1624 (65), 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the 1,1-dichloroethylene from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the 1,1-dichloroethylene and transfer it onto a gas

chromatographic (GC) column. The GC column is programmed to separate the volatile organics; 1,1-dichloroethylene is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations, direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for 1,1-dichloroethylene analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (<1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3405, 3184, 3443).

Typical 1,1-dichloroethylene detection limits that can be obtained in aqueous samples (including wastewaters without interferences) and in non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.13 μg/L (Method 601) | 1.3 μg/kg (Method 8010) |
| 2.8 μg/L (Method 624) | 5.0 μg/kg (Method 8240) |
| 10.0 μg/L (Method 1624) | |
| 1.3 μg/L (Method 8010) | |
| 5.0 μg/L (Method 8240) | |

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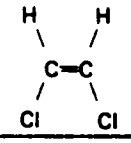
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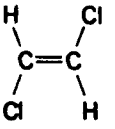
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| COMMON SYNONYMS: | CAS REG.NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25°C (12) |
|---|---|---|
| 1,2-Dichloro-(z)ethene<br>Cis-1,2-dichloroethene<br>Cis-1,2-dichloro-ethylene | CIS: 156-59-2 $C_2H_2Cl_2$<br>NIOSH NO: KV9420000<br>STRUCTURE:  | 3.97 mg/m$^3$ ≈ 1 ppm;<br>0.252 ppm ≈ 1 mg/m$^3$<br>MOLECULAR WEIGHT: 96.95 |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA (CIS) | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Slightly acrid | (54) |
| | • Odor Threshold: 0.085 ppm | (38) |
| | • Density: 1.2840 g/mL (at 20°C) | (68) |
| | • Freeze/Melt Point: -81.50°C | (21) |
| | • Boiling Point: 60.20°C | (21) |
| | • Flash Point: 6.00°C | (21) |
| | • Flammable Limits: 9.70 to 12.80 % by volume | (38,60,506) |
| | • Autoignition Temp.: 460.0°C (approximation) | (38,506) |
| | • Vapor Pressure: 20.3 mm Hg (at 20°C) | (21) |
| | • Satd. Conc. in Air: 1.4350E+04 mg/m$^3$ (at 20°C) | (1219) |
| | • Solubility in Water: 3500.00 mg/L (at 20°C) | (12) |
| | • Viscosity: 0.467 cp (at 20°C) | (21) |
| | • Surface Tension: 28.0000 dyne/cm (at 20°) | (21) |
| | • Log (Octanol-Water Partition Coeff.): 1.86 | (29) |
| | • Soil Adsorp. Coeff.: 35.00 | (652) |
| | • Henry's Law Const.: 0.01 atm · m$^3$/mol (at 25°C) | (74) |
| | • Bioconc. Factor: 4.50 (est-avg for mixture) | (659) |

| COMMON SYNONYMS: 1,2-Dichloroethylene | CAS REG.NO.: FORMULA:<br>MIX: $C_2H_2Cl_2$<br>540-59-0<br>NIOSH NO:<br>KV9360000<br><br>STRUCTURE:<br>Mixture | AIR W/V CONVERSION FACTOR at 25 °C (12)<br><br>3.97 mg/m³ ≈ 1 ppm;<br>0.252 ppm ≈ 1 mg/m³<br><br>MOLECULAR WEIGHT:<br>96.95 |
|---|---|---|

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA (MIX) | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Slightly acrid | (54) |
| | • Odor Threshold: 0.085 ppm | (38) |
| | • Density: No data | |
| | • Freeze/Melt Point: No data | |
| | • Boiling Point: No data | |
| | • Flash Point: No data | |
| | • Flammable Limits: 9.70 to 12.80 % by volume | (38,60,506) |
| | • Autoignition Temp.: 460.0°C (approx.) | (38,506) |
| | • Vapor Pressure: No data | |
| | • Satd. Conc. in Air: No data | |
| | • Solubility in Water: No data | |
| | • Viscosity: No data | |
| | • Surface Tension: No data | |
| | • Log (Octanol-Water Partition Coeff.): No data | |
| | • Soil Adsorp. Coeff.: No data | |
| | • Henry's Law Const.: No data | |
| | • Bioconc. Factor: 4.50 (estim-avg for mixture) | (659) |

| COMMON SYNONYMS: | CAS REG.NO.: FORMULA: | AIR W/V CONVERSION FACTOR at 25°C (12) |
|---|---|---|
| 1,2-Dichloro-(e)ethene<br>Trans-1,2-dichloro-ethene<br>Trans-1,2-dichloro-ethylene | TRANS: $C_2H_2Cl_2$<br>156-60-5<br>NIOSH NO:<br>KV9400000<br><br>STRUCTURE:  | 3.97 mg/m³ ≈ 1 ppm;<br>0.252 ppm ≈ 1 mg/m³<br><br>MOLECULAR WEIGHT:<br>96.95 |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA (TRANS) | • Physical State: Liquid (at 20°C) | (23) |
| | • Color: Colorless | (23) |
| | • Odor: Slightly acrid | (54) |
| | • Odor Threshold: 0.085 ppm | (38) |
| | • Density: 1.2570 g/mL (at 20°C) | (68) |
| | • Freeze/Melt Point: -49.40°C | (21) |
| | • Boiling Point: 47.70°C | (21) |
| | • Flash Point: 4.00°C | (21) |
| | • Flammable Limits: 9.70 to 12.80 % by volume | (38,60,506) |
| | • Autoignition Temp.: 460.0°C (approx.) | (38,506) |
| | • Vapor Pressure: 39.8 mm Hg (at 20°C) | (21) |
| | • Satd. Conc. in Air: 2.8200E+04 mg/m³ (at 20°C) | (1219) |
| | • Solubility in Water: 6300.00 mg/L (at 20°C) | (12) |
| | • Viscosity: 0.404 cp (at 20°C) | (21) |
| | • Surface Tension: 25.0000 dyne/cm (at 20°C) | (21) |
| | • Log (Octanol-Water Partition Coeff.): 2.09 | (29) |
| | • Soil Adsorp. Coeff.: 59.00 | (652) |
| | • Henry's Law Const.: 6.6E-03 atm · m³/mol (at 25°C) | (74) |
| | • Bioconc. Factor: 4.50 (estim avg for mixture) | (659) |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as 12-dichloroethylene with cyanides mercaptans or other organic sulfides typically generate heat while those with mineral acids amines azo compounds hydrazines caustics or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat toxic gases and fires. Those with alkali or alkaline earth metals certain other chemically active elemental metals like aluminum zinc or magnesium organic peroxides or hydroperoxides strong oxidizing agents or strong reducing agents typically result in heat generation and explosions and/or fires. Toxic combustion products phosgene and HCl may be formed (511, 505). |

| | |
|---|---|
| PERSISTENCE IN THE SOIL WATER SYSTEM | Cis- and trans-1,2-dichloroethylene are expected to be highly mobile in soil/ground-water systems, particularly in deep or sandy soils. Volatilization may be important for 1,2-dichloroethylene near the surface or in the soil-air compartment. Transformation processes such as hydrolysis or biodegradation are not expected to be significant in natural soils. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of cis- and trans-1,2-dichloroethylene to groundwater drinking water supplies. Such migration has commonly occurred in the past. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (45, 54)<br>Inhalation or ingestion will produce nausea, vomiting, dizziness and narcosis. The liquid can act as a primary irritant, producing dermatitis and irritation of mucous membranes.<br><br>CIS<br>Acute Toxicity Studies:<br>INHALATION:<br>$LC_{Lo}$ 65000 mg/m$^3$ · 2 hr Mouse (47)<br><br>MIXTURE:<br>Acute Toxicity Studies:<br>ORAL:<br>$LD_{50}$ 770 mg/kg Rat (770)<br><br>TRANS:<br>Acute Toxicity Studies:<br>INHALATION:<br>$LC_{Lo}$ 75000 mg/m$^3$ · 2 hr Mouse (47)<br>ORAL:<br>$LD_{50}$ 2221 mg/kg Mouse (624)<br><br>CIS:<br>Long-Term Effects: Possible liver and kidney damage<br>Pregnancy/Neonate Data: No data<br>Genotoxicity Data: Conflicting results<br>Carcinogenicity Classification:<br>IARC - No data<br>NTP - No data<br>EPA - Group D (not classifiable as to human carcinogenicity)<br><br>MIXTURE:<br>Long-Term Effects: Possible liver and kidney damage<br>Pregnancy/Neonate Data: No data<br><br>Genotoxicity Data: Limited evidence is negative<br>Carcinogenicity Classification:<br><br>IARC - No data<br>NTP - No data<br>EPA - No data<br><br>TRANS:<br>Long-Term Effects: Possible liver and kidney damage<br>Pregnancy/Neonate Data: No data<br>Genotoxicity Data: Limited evidence is negative<br>Carcinogenicity Classification:<br>IARC - No data<br>NTP - No data<br>EPA - Group D (not classifiable as to human carcinogenicity) |

| | |
|---|---|
| HANDLING PRECAUTIONS | Handle chemical only with adequate ventilation. • Vapor concentrations of 200-1000 ppm: chemical cartridge respirator with full facepiece and organic vapor cartridge. • 1000-4000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece; a gas mask with an organic vapor canister. • Chemical goggles if there is probability of eye contact with liquid. • Impervious clothing to prevent repeated or prolonged contact with liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 200 ppm (mixture)
- AFOSH PEL (8-hr TWA): 200 ppm; STEL (15-min): 250 ppm (mixture)

Criteria
- NIOSH IDLH (30 min): 4000 ppm (mixture)
- NIOSH REL: None established
- ACGIH TLV® (8-hr TWA): 200 ppm (mixture)
- ACGIH STEL (15 min): Deleted

WATER EXPOSURE LIMITS:

Drinking Water Standards (3883)

CIS:
MCLG: 70 μg/L (proposed)
MCL: 70 μg/L (proposed)

TRANS:
MCLG: 100 μg/L (proposed)
MCL: 100 μg/L (proposed)

EPA Health Advisories and Cancer Risk Levels (3770)

| CIS: | TRANS: |
|---|---|
| - 1 day (child): 4 mg/L | - 1 day (child): 20 mg/L |
| - 10 day (child): 1 mg/L | - 10 day (child): 2 mg/L |
| - longer term (child): 1 mg/L | - longer term (child): 2 mg/L |
| - longer term (adult): 1 mg/L | - longer term (adult): 6 mg/L |
| - lifetime (adult): 0.07 mg/L | - lifetime (adult): 0.1 mg/L |

WHO Drinking Water Guideline
No information available.

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA (Cont.)

EPA Ambient Water Quality Criteria

- Human Health (3770)
  - No criterion established due to insufficient data.

- Aquatic Life (3770)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 11,600 µg/L dichloroethylenes.

    chronic toxicity:
    no criterion established due to insufficient data.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 224,000 µg/L dichloroethylenes.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:

CIS: 1.000E-02 mg/kg/day (3744)
TRANS: 2.000E-02 mg/kg/day (3744)

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

1,2-Dichloroethylene is listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and effluent standards and guidelines (351, 3763). Effluent limitations specific to trans-1,2-dichloroethylene have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), and steam electric power generating (3802). Limitations vary depending on the type of plant and industry. Safe Drinking Water Act (SDWA) 1,2-Dichloroethylene is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). It is listed as an unregulated contaminant requiring monitoring in all community water systems and non-community non-transient water systems (3771). In states with an approved Underground Injection Control program, a permit is required for the injection of 1,2-dichloroethylene-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

1,2-Dichloroethylene is identified as a toxic hazardous waste (U079) and listed as a hazardous waste constituent (3783, 3784). A non-specific source of trans-1,2-dichloroethylene-containing waste is the production of chlorinated aliphatic hydrocarbons (325). Waste streams from the inorganic chemicals industry (chlorine production) contain 1,2-dichloroethylene and are listed as specific sources of hazardous wastes (3774, 3765). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in total concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). 1,2-Dichloroethylene is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)

Trans-1,2-dichloroethylene is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing trans-1,2-dichloroethylene but these depend upon the concentration of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of 1,2-dichloroethylene must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to 1,2-dichloroethylene in any 8-hour work-shift of a 40-hour work-week shall not exceed an 8-hour time-weighted average (TWA) of 200 ppm (3539).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated 1,2-dichloroethylene as a hazardous material with a reportable quantity of 454 kg, subject to requirements for packaging, labeling and transportation (3180).

Food, Drug and Cosmetic Act (FDCA)
1,2-Dichloroethylene (mixed isomers) is approved for use as an indirect food additive as a component of adhesives (3209).

- State Water Programs

ALL STATES
All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

CALIFORNIA
California has set an action level for drinking water of 16 μg/L for either cis-, trans-, or the sum of these two isomers of 1,2-dichloroethylene (3098).

CONNECTICUT
Connecticut has a quantification limit of 2.0 μg/L for 1,2-dichloroethylene in drinking water (3137).

NEW HAMPSHIRE
New Hampshire has set an enforceable Toxic Contaminant Level (TCL) for trans- 1,2-dichloroethylene in drinking water of 2.7 mg/L for a one-day exposure (assumes a child weighing 10 kg who drinks one liter of water per day), and a TCL of 4.0 mg/L for cis-1,2-dichloroethylene (3710).

NEW YORK
New York has an MCL of 5 μg/L for 1,2-dichloroethylene (both isomers) in drinking water, and a nonenforceable guideline of 50 μg/L for trans-1,2-dichloroethylene in surface and ground-waters (3501).

NORTH DAKOTA
North Dakota requires that 1,2-dichloroethylene be nondetectable, using designated test methods, in ground-water (3671).

PENNSYLVANIA
Pennsylvania has set a human health criterion of 350 μg/L for trans-1,2-dichloro- ethylene in surface waters (3561).

VERMONT
Vermont has a preventive action limit of 35 μg/L and an enforcement standard of 70 μg/L for the cis- or trans- isomers of 1,2-dichloroethylene in ground-water (3682).

WISCONSIN
Wisconsin has set a human threshold criterion of 270 μg/L for cis- and trans-isomers of 1,2-dichloroethylene for surface waters used for public water supply (3842). Wisconsin has also set a preventive action limit of 20 μg/L and an enforcement standard of 100 μg/L for trans-1,2-dichloroethylene in ground-water, and a preventive action limit of 10 μg/L and enforcement standard of 100 μg/L for the cis-isomer of 1,2-dichloroethylene (3840).

Proposed Regulations

- Federal Programs

  Safe Drinking Water Act (SDWA)
  EPA plans to propose a maximum contaminant level (MCL) of 70 μg/L and repropose a maximum contaminant level goal (MCLG) of 70 μg/L for 1,2-dichloroethylene in May, 1989, with final action expected in December, 1989 (3751).

- State Water Programs

  MOST STATES
  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with the state officers is advised. Changes are planned for 1989-90 (3683).

KANSAS

Kansas has proposed a water quality criterion of 70 μg/L for groundwater (3213).

MINNESOTA

Minnesota has proposed a Recommended Allowable Limit (RAL) of 70 μg/L for 1,2-dichloroethylene in drinking water (3451). Minnesota has also proposed chronic criteria of 70 μg/L for designated groundwaters, 67 μg/L for designated surface waters, and 65 μg/L for cold surface waters. These criteria are for the protection of human health (3452).

NEW JERSEY

New Jersey has proposed an MCL of 10 μg/L for 1,2-dichloroethylene in drinking water, and a water quality criterion of 10 μg/L for class FW2 surface waters (3496).

EEC Directives

Directive on Ground-Water (538)
Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Requir[illegible] of Shellfish Waters (537)
The mandatory specifications [illegible]organohalogenated substances specify that the concentra[illegible] of [illegible]bstance in the shellfish water or in shellfish flesh m[illegible] [illegible]xceed a level which has harmful effects on the [illegible]ae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
1,2-Dichloroethylene is listed as a Class II/b harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Class[illegible]ation, Packaging and Labeling of Dangerous Substances (787)
1,2-Dichloroethylene is classified as a flammable, harmful substance and is subject to packaging and labeling regulations.

## 15.1 MAJOR USES

Neither the cis nor the trans isomer of 1,2-dichloroethylene has developed wide industrial usage in the United States partly due to their flammability. The trans isomer is more widely used in industry than either the cis isomer or the 60:40 cis/trans mixture. It is primarily used as either a low-temperature extraction solvent or as a direct solvent in materials such as dyes, perfume oils, waxes, resins and thermoplastics. It is also used as a chemical intermediate in the synthesis of polymers. Miscellaneous applications include usage in food packaging adhesives and germicidal fumigants (17, 38).

## 15.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 15.2.1 Transport in Soil/Groundwater-Systems

#### 15.2.1.1 Overview

The two isomers of 1,2-dichloroethylene (cis and trans) have very similar physicochemical properties and thus their environmental fate and transport will be similar. Except where noted, the discussion below refers to both isomers.

1,2-Dichloroethylene may move through the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways can be assessed by using an equilibrium partitioning model as shown in Table 15-1. These calculations predict the partitioning of low soil concentrations of 1,2-dichloroethylene among soil particles, soil water and soil air. The portions of 1,2-dichloroethyene associated with the water and air phases of the soil are more mobile than the adsorbed portion.

The estimates for the unsaturated topsoil model show that a moderate amount (10% ±) of the chemical will be present in the soil-water phase and can thus migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. For the portion of 1,2-dichloroethylene in the gaseous phase (10% ±), diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, may be a significant loss pathway.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the 1,2-dichloroethylene (80-90%) is likely to be present in the soil-water phase (Table 15-1) and transported with flowing groundwater.

TABLE 15-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR CIS AND TRANS 1,2-DICHLOROETHYLENE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment[a] | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil[b,c] at 25°C | | | |
| - cis | 77.6 | 11.6 | 10.8 |
| - trans | 86.1 | 7.6 | 6.3 |
| Saturated deep soil[d]: | | | |
| - cis | 12.8 | 87.2 | - |
| - trans | 19.9 | 80.1 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 35 (cis) and 59 (trans). (Estimated by Arthur D. Little, Inc.)
c) Henry's law constant taken as 7.5E-03 atm · $m^3$/mol for cis and 6.60E-03 atm · $m^3$/mol for trans at 25°C (74).
d) Used sorption coefficient ($K_p$) calculated as a function of $K_{oc}$ assuming 0.1% organic carbon: $K_p = 0.001 \times K_{oc}$.

15.2.1.2 Sorption on Soils

The mobility of 1,2-dichloroethlyene in the soil/ground-water system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter content of the soil water.

Based upon their octanol-water partition coefficient of 72 (cis) and 120 (trans), the soil adsorption coefficients ($K_{oc}$) of the two isomers are estimated to be 35 and 59, respectively. These are relatively low numbers and indicative of weak sorption to soils.

### 15.2.1.3 Volatilization from Soils

Transport of 1,2-dichloroethylene vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

There are no data from laboratory or field tests showing actual volatilization rates; however, the rates should be similar to those for trichloroethylene for which some data are available.

## 15.2.2 Transformation Processes in Soil/Ground-water Systems

The persistence of 1,2-dichloroethylene in soil/ground-water systems has not been investigated. In most cases, it should be assumed that the chemical will persist for months to years (or more). 1,2-Dichloroethylene that has been released into the air will eventually undergo fairly rapid photochemical oxidation (10).

1,2-Dichloroethylene under normal environmental conditions is not expected to undergo rapid hydrolysis. Mabey et al. (33) indicate that hydrolysis is not an environmentally significant degradation pathway for trans-1,2-dichloroethylene.

Literature references to microbial degradation of compounds such as 1,2-dichloroethylene are very few. Most references indicate that low molecular weight chloroaliphatics are not rapidly metabolized in the environment (10). Slow to moderate degradation of cis and trans 1,2-dichloroethylene was observed by Tabak et al. (79) with acclimated activated sludge populations in a shake-flask screening procedure. Significant losses due to volatilization made it difficult to clearly identify the extent of losses due to biodegradation. However, in most soil/ground-water systems such aerobic degradation would be of minimal importance because of the low concentration of microorganisms (at depth) and the low dissolved oxygen (anaerobic) conditions. No studies have been done to investigate the possibility of anaerobic biodegradation.

### 15.2.3 Primary Routes of Exposure From Soil/Ground-water Systems

The above discussion of fate pathways suggests that cis- and trans-1,2-dichloroethylene are both highly volatile, weakly adsorbed by soil and have no significant potential for bioaccumulation. These compounds may volatilize from soil surfaces, but that portion not subject to volatilization is likely to be mobile in groundwater. These fate characteristics suggest several potential exposure pathways.

Volatilization of cis- and trans-1,2-dichloroethylene from a disposal site, particularly during drilling or restoration activities, could result in inhalation exposures. The potential for ground-water contamination is high, particularly in sandy soils. Mitre (83) reported that cis-1,2-dichloroethylene has been found at 5 of the 546 National Priority List (NPL) sites. It was detected at 4 sites in groundwater and 2 sites in surface water. Trans-1,2-dichloroethylene has been found at 46 of the 546 NPL sites, with 37 sites in groundwater, 13 sites in surface water and 1 site in air.

The potential for exposure through drinking water is confirmed by the presence of these compounds in groundwater sources of drinking water in the United States. The USEPA (64) reported from state data that cis- and/or trans-1,2-dichloroethylene had been found at a range of concentrations from trace to 860 μg/L in 197 samples out of 1249 samples. These compounds were also reported in the USEPA (531) Groundwater Supply Survey (GWSS) as shown below:

| Sample Type | Occurrences* No. | % | Median of Positives (μg/L) | Maximum (μg/L) |
|---|---|---|---|---|
| Random | | | | |
| Supplies serving <10,000 people (280 samples) | 3 | 1.1 | 0.23 | 1.7 |
| Supplies serving >10,000 people (186 samples) | 13 | 7.0 | 1.1 | 2.0 |
| Non-Random | | | | |
| Supplies serving <10,000 people (321 samples) | 11 | 3.4 | 1.3 | 17.0 |
| Supplies serving >10,000 people (158 samples) | 27 | 17.1 | 2.7 | 120.0 |

*Samples having levels over quantification limit of 0.2 μg/L.

The random results are intended to statistically represent the U.S. drinking water supplies from ground-water sources. The non-random samples were chosen by the states as being potentially contaminated. Cis- and trans-1,2-dichloroethylene have also been detected in the National Organic Monitoring Survey (NOMS) (90).

These results indicate that these 2 isomers have the potential for movement in soil/groundwater systems. They may eventually reach surface waters by this mechanism, suggesting several other exposure pathways:

- Surface waters may be used as drinking water supplies, resulting in direct ingestion exposure and potential inhalation exposure.
- Aquatic organisms residing in these waters may be consumed, also resulting in ingestion exposures through bioaccumulation.
- Recreational use of these waters may result in dermal exposures.
- Domestic animals may consume or be dermally exposed to contaminated ground or surface waters; the consumption of meats and poultry could then result in ingestion exposures.

In general, exposures associated with surface water contamination can be expected to be lower than exposure from drinking contaminated groundwater for two reasons. First, the Henry's law constants for cis- and trans-1,2-dichloroethylene suggest that they will volatilize upon reaching surface waters. Secondly, the bioconcentration factors for these compounds are low, suggesting limited bioaccumulation in aquatic organisms or domestic animals.

### 15.2.4 Other Sources of Exposure

The data presented above on the Ground Water Supply Survey (531) suggest that cis- and trans-1,2-dichloroethylene are found in a limited number of groundwater supplies used as drinking water. Coniglio et al. (223) reported that cis- and trans-1,2-dichloroethylene were found in raw surface water supplies. In finished water supplies, however, only the cis isomer was present, probably because of the loss of the trans isomer due to its higher volatility (almost twice as much compared with the cis isomer). In a summary of data available as of 1980, these authors reported that 4.9% of the 103 finished surface water samples were contaminated with cis-1,2-dichloroethylene, at a mean concentration of 0.66 $\mu$g/L.

The volatility of both isomers suggests that they may be found in air as well. Brodzinsky and Singh (84) compiled all available atmospheric monitoring data for a number of volatile organics. For cis-1,2-dichloroethylene, they had data for 772 locations. This compound was not found in rural and remote areas. In urban and suburban locations, the median concentration was 0.27 $\mu g/m^3$ In source-dominated areas, the median concentration was 1.2 $\mu g/m^3$ There were only 4 locations for trans-1,2-dichloroethylene, all in source-dominated areas. The median found for trans-1,2-dichloroethylene in source-dominated areas was 3.7 $\mu g/m^3$ These results suggest inhalation exposures to persons in the areas where the compounds have been found in air.

## 15.3 HUMAN HEALTH CONSIDERATIONS

### 15.3.1 Animal Studies

#### 15.3.1.1 Carcinogenicity

No carcinogenicity data are available for either the cis- or trans-1,2-dichloroethylene isomers. Neither IARC nor the NTP have evaluated 1,2-dichloroethylene.

#### 15.3.1.2 Genotoxicity

The trans isomer of 1,2-dichloroethylene and the cis/trans mixture were tested for the National Toxicology Program in four standard strains (TA98, TA100, TA1535 and TA1537) of Salmonella typhimurium, and both compounds were negative with or without rat or hamster liver metabolic activation systems (3469). The cis isomer was also tested in the strains listed above as well as in TA97 under the same test conditions; it was negative (3860). Nohmi et al tested the cis isomer the same assay but with strains TA98, TA100, TA97 and TA102 with a mouse liver metabolic activation system, and it also was negative (3508). In all studies, the compounds were tested to toxic levels. Negative results were also obtained in E. coli K12 reversion tests in the presence of mouse liver enzymes (156). The cis and trans isomers were tested in the yeast S. cerevisiae in suspension and in a host-mediated assay. Both isomers were able to induce convertants in the suspension assay but only with metabolic activation. In the host-mediated assay, only the cis isomer was capable of inducing revertants and convertants (3083). The trans isomer is claimed to induce aneuploidy without activation in V79 Chinese hamster cells (3537), but when cultured Chinese hamster lung cells were treated with the cis or trans isomer, no increase was seen in chromosomal aberrations or sister chromatid exchanges, with or without metabolic activation (3613).

#### 15.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No data on reproductive toxicity are available on the 1,2-dichloroethylene isomers.

#### 15.3.1.4 Other Toxicologic Effects

##### 15.3.1.4.1 Short-term Toxicity

Since 1,2-dichloroethylene is not widely used, toxicologic data are limited. The data that are available are old and must be considered suspect since the purity of the samples and the ratio of the isomers were not always indicated.

According to Smyth (178), the cis isomer did not kill or anesthetize rats in 4 hours at 8000 ppm. At 16,000 ppm, rats became anesthetized in 8 minutes and died

in 4 hours. Smyth also stated that he found the trans isomer to be twice as toxic as the cis isomer. However, this data is old and not corroborated in setting higher limits for exposure to the trans relative to the cis form in drinking water (see Section 15.3.3).

Oral $LD_{50}$ (2122 mg/kg in males and 2391 mg/kg in females) has been reported for CD-1 mice exposed to trans 1,2-dichloroethylene by Barnes et al. (3316). Mice, exposed to the agent at 0.1, 1.0, or 2.0 mg/mL in drinking water for 90 days, showed minimal toxic effects. However, at the 2.0 mg/mL level, hepatic glutathione level was significantly lowered in male mice and hepatic aniline hydroxylase activity level was lowered in female mice at all three dose levels.

In another study by Hayes et al. (3214), CD rats were exposed for 90 days to the trans isomer in drinking water at doses of 402, 1311, or 3114 mg/kg/day (males) and 353, 1257, or 2809 mg/kg/day (females). No treatment related effects on fluid consumption, body weights, hematology, serum chemistry, or urinalysis, were noted; however, a significant dose-dependent decrease in kidney weight was observed in female rats at the 1257 and 2809 mg/kg/day level.

A 6-hour $LC_{50}$ value of 22,000 ppm was reported for mice exposed to the trans isomer (558). In another study, adverse lung effects were reported in rats receiving a single 8-hour exposure to 200 ppm of the trans isomer (177).

Dogs repeatedly exposed to dichloroethylene vapor (produced by the evaporation of 10 to 15 mL in a vapor chamber of 0.115 $m^3$ volume), developed superficial corneal clouding which was reversible within 24 to 48 hours (19).

#### 15.3.1.4.2 Chronic Toxicity

Conflicting data exist regarding the chronic toxicity of 1,2-dichloroethylene. Torkelson reported no adverse effects in rats, rabbits, guinea pigs and dogs exposed to either 500 or 1000 ppm of 1,2-dichloroethylene 7 hours daily, 5 days per week for 6 months. The sample consisted of 60% cis- and 40% trans-1,2-dichloroethylene (2). Similarly no effects were seen in rats dosed subcutaneously, percutaneously or by ingestion (doses unspecified) (2).

In contrast, Fruendt et al. (177) reported marked effects in rats exposed 8 hours daily, 5 days per week for 16 weeks, to vapor levels of 200 ppm of the trans isomer. Liver and lungs were affected and leukocyte counts were decreased.

### 15.3.2 Human and Epidemiologic Studies

#### 15.3.2.1 Short-term Toxicologic Effects

1,2-Dichloroethylene was once used as a general anesthetic in humans (46). Exposure to the trans isomer at a level of 2000 ppm causes burning of the eyes, vertigo and nausea (46). In the small-scale industrial usage that exists, no toxic

effects in humans due to occupational exposure have been noted other than one old report of a fatality, attributable to very high vapor inhalation in a small enclosure (559).

No evidence of ocular or dermal toxicity has been found. However, irritation will occur if 1,2-dichloroethylene comes into contact with the eye or skin (19, 59).

#### 15.3.2.2 Chronic Toxicologic Effects

There are no reports of long-term human exposure to 1,2-dichloroethylene isomers.

### 15.3.3 Levels of Concern

In view of the paucity of data available on the adverse health effects and effect levels associated with exposure to the cis and trans isomers of 1,2-dichloroethylene, estimates of exposure levels of concern cannot be made with any confidence. Due to the lack of data, the USEPA has not set a water quality criterion for human health (355). Lifetime Health Advisories of 70 $\mu$g/L (cis) and 100 $\mu$g/L (trans) have been derived by the USEPA (3977).

Both OSHA (3539) and the ACGIH (3005) have set an occupational exposure limit of 200 ppm (790 mg/m$^3$) for the mixed isomers of 1,2-dichloroethylene, based on preventing narcosis.

### 15.3.4 Hazard Assessment

1,2-Dichloroethylene vapor is a narcotic and mucous membrane irritant. It was once used as a general anesthetic in humans (46). Limited data suggest the no-observed-effect-level in animals upon prolonged inhalation is at least 1000 ppm (3). No reports of human ingestion were found. Skin contact with liquid can induce a primary irritant response (54). Conflicting results have been reported for 1,2-dichloroethylene.

A limited number of tests suggest no mutagenic activity for 1,2-dichloroethylene. However, the notable lack of data available concerning the carcinogenic and teratogenic potential of 1,2-dichloroethylene as well as the absence of quantitative data on acute and long-term toxicity associated with this compound provide a low degree of confidence in any assessment of hazard for 1,2-dichloroethylene exposure, particularly with regard to long-term, low-level human exposure via drinking water.

## 15.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of concentrations of the cis or trans isomer of 1,3-dichloroethylene in soil and water requires collection of a representative field sample and subsequent laboratory analysis. Due to the volatility of 1,2-dichloroethylene, care is

required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of trans-1,2-dichloroethylene, one of the EPA priority pollutants, in aqueous samples include EPA Methods 601, 624, and 1624 (65), and Methods 8010 and 8240 (63). These procedures are valid for cis-1,2-dichloroethylene, as well. The sample introduction technique most useful for aqueous samples is the purge and trap method. An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the 1,2-dichloroethylene from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the 1,2-dichloroethylene and transfer it onto a gas chromatographic (GC) column. The GC column is programed to separate the volatile organics; 1,2-dichloroethylene is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624 and 8240). For samples that contain high concentrations, direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for trans-1,2-dichloroethylene analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the procedures for aqueous samples primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443). A method for the analysis of cis-1,2-dichloroethylene that has also been reported involves dispersing the sample in 2-methoxy-ethanol (3570). A portion of the liquid phase is then combined with water, purged and analyzed by electron capture detection.

Typical trans-1,2-dichloroethylene detection limits that can be obtained in aqueous samples (including wastewaters without interferences) and in non-aqueous samples (wastes, soils, etc.) are shown below. The actual detection limit achieved in a given analysis will vary with instrument sensitivity and matrix effects.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.10 μg/L (Method 601 or 8010) | 5.0 μg/kg (Method 8240) |
| 1.6 μg/L (Method 624 or 8240) | 1.0 μg/Kg (Method 8010) |
| 10.0 μg/L (Method 1624) | |
| 5.0 μg/L (Method 8240) | |
| 1.0 μg/L (Method 8010) | |

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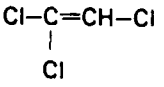
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| COMMON SYNONYMS: | CAS REG.NO.: 79-01-6 FORMULA: $C_2HCl_3$<br>NIOSH NO: KX4550000<br>STRUCTURE:  | AIR W/V CONVERSION FACTOR at 25°C (12)<br>5.38 mg/m³ ≈ 1 ppm;<br>0.1858 ppm ≈ 1 mg/m³<br>MOLECULAR WEIGHT: 131.39 |
|---|---|---|
| Acetylene trichloride<br>Ethinyl trichloride<br>Ethylene trichloride<br>TCE<br>TRI<br>Trichloroethene<br>Trichloroethylene | | |

| | |
|---|---|
| REACTIVITY | Reactions of halogenated organic materials such as trichloroethylene with cyanides, mercaptans or other organic sulfides typically generate heat, while those with mineral acids, amines, azo compounds, hydrazines, caustics or nitrides commonly evolve heat and toxic or flammable gases. Reactions with oxidizing mineral acids may generate heat, toxic gases and fires. Those with alkali or alkaline earth metals, certain other chemically active elemental metals like aluminum, zinc or magnesium, organic peroxides or hydroperoxides, strong oxidizing agents, or strong reducing agents typically result in heat generation and explosions and/or fires (511, 505). |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Physical State: Liquid (at 20°C) | (23)<br>(23) |
| | • Color: Colorless (often dyed pale blue) | (23,59)(23) |
| | • Odor: Sweet, chloroform-like | (23) |
| | • Odor Threshold: 100.000 ppm | (12) |
| | • Density: 1.4620 g/mL (at 20°C) | (48) |
| | • Freeze/Melt Point: -84.40/-73.00°C | (23,48) |
| | • Boiling Point: 87.20°C | (48) |
| | • Flash Point: Practically non-flammable | (12,23) |
| | • Flammable Limits: 8.0 to 10.5% by volume | (60,504,507) |
| | • Autoignition Temp.: 410.0 to 420.0°C | (60,504,506) |
| | • Vapor Pressure: 5.87E+01 mm Hg (at 20°) | (48) |
| | • Satd. Conc. in Air: 4.2200E+05 mg/m³ (at 20°C) | (1219) |
| | • Solubility in Water: 1.00E+03 mg/L (at 20°C) | (10) |
| | • Viscosity: 0.570 cp (at 20°C) | (48) |

| | | |
|---|---|---|
| PHYSICO-CHEMICAL DATA | • Surface Tension: 2.9300E+01 dyne/cm (at 20°C) | (60) |
| | • Log (Octanol-Water Partition Coeff.): 2.42 | (29) |
| | • Soil Adsorp. Coeff.: 1.27E+02 | (652) |
| | • Henry's Law Const.: 8.92E-03 atm · $m^3$/mol (at 20°C) | (74) |
| | • Bioconc. Factor: 13 (estim), 17 (bluegill sunfish), 39 (rainbow trout) | (31,61,659) |

| | |
|---|---|
| PERSISTENCE IN THE SOIL-WATER SYSTEM | Trichloroethylene is relatively mobile in the soil/groundwater system, particularly in soils with low organic content. Volatilization may be significant for trichloroethylene near the surface or in the soil-air phase. Transformation processes such as hydrolysis and biodegradation are not expected to be important in natural soils. |

| | |
|---|---|
| PATHWAYS OF EXPOSURE | The primary pathway of concern from a soil-water system is the migration of trichloroethylene to groundwater drinking water supplies. Drinking water surveys suggest that such migration has been common in the past. Inhalation resulting from volatilization from surface soils may also be important. |

| | |
|---|---|
| HEALTH HAZARD DATA | Signs and Symptoms of Short-term Human Exposure: (54)<br>Acute exposure to trichloroethylene depresses the central nervous system causing such symptoms as headache, dizziness, vertigo, tremors, irregular heartbeat, fatigue, nausea, vomiting, blurred vision and intoxication. The vapors may cause irritation of the eyes, nose and throat. The liquid may cause burning irritation and damage to the eye. Repeated or prolonged skin contact with the liquid may cause dermatitis. |

| | |
|---|---|
| HEALTH HAZARD DATA | Acute Toxicity Studies: (3504)<br><br>INHALATION:<br>$LC_{50}$ 26000 ppm · 1 hr Rat<br>$LC_{50}$ 5500 ppm · 10 hour Mouse<br><br>ORAL:<br>$LD_{50}$ 2402 mg/kg Mouse<br><br>Long-Term Effects: Liver and kidney damage<br>Pregnancy/Neonate Data: Negative<br>Genotoxicity Data: Conflicting results<br>Carcinogenicity Classification:<br>IARC - Group 3 (not classifiable as to its carcinogenicity to humans)<br>NTP - Inadequate study with rats<br>EPA - Group B2 (inadequate human evidence, sufficient animal evidence) |

| | |
|---|---|
| HANDLING PRECAUTIONS (709) | Handle chemical only with adequate ventilation. • Vapor concentrations of 100-1000 ppm: chemical cartridge respirator with organic vapor cartridge or type C demand-type supplied air respirator with half-mask facepiece. • Chemical goggles if there is probability of eye contact with liquid. • Impervious protective clothing to prevent contact with liquid. |

## ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA TWA (8-hr): 50 ppm; STEL: 200 ppm;
- AFOSH PEL (8-hr TWA): 50 ppm; STEL (15-min): 200 ppm

Criteria
- NIOSH IDLH (30-min): deleted; NIOSH has recommended that the substance be treated as a potential human carcinogen.
- NIOSH REL (10-hr TWA): 25 ppm
- ACGIH TLV® (8-hr TWA): 50 ppm
- ACGIH STEL (15-min): 200 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards (3742)
MCLG: 0 μg/L
MCL: 5 μg/L

EPA Health Advisories and Cancer Risk Levels (3977)
The EPA has developed the following Health Advisories which provide specific advice on the levels of contaminants in drinking water at which adverse health effects would not be anticipated.

- No health advisories
- 1E-04 cancer risk level: 300μg/L

WHO Drinking Water Guideline (666)
A tentative, health-based guideline for drinking water of 30 μg/L has been proposed for trichloroethylene. A daily per capita consumption of two liters of water was assumed.

EPA Ambient Water Quality Criteria
- Human Health (355)
  - Based on ingestion of contaminated water and aquatic organisms (1E-05, 1E-06, 1E-07 cancer risk), 27 μg/L, 2.7 μg/L, 0.27 μg/L.
    Based on ingestion of contaminated aquatic organisms only, (1E-05, 1E-06, 1E-07 cancer risk), 807 μg/L, 80.7 μg/L, 8.07 μg/L.

- Aquatic Life (355)
  - Freshwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 45,000 μg/L.

    chronic toxicity:
    no criterion, but lowest effect level occurs at 21,900 μg/L.

  - Saltwater species
    acute toxicity:
    no criterion, but lowest effect level occurs at 2000 μg/L.

    chronic toxicity:
    no criterion established due to insufficient data.

REFERENCE DOSES:
7.000E+00 μg/kg/day (3744)

REGULATORY STATUS (as of 01-MAR-89)

Promulgated Regulations

- Federal Programs

Clean Water Act (CWA)

Trichloroethylene is designated a hazardous substance. It has a reportable quantity (RQ) limit of 454 kg (347, 3764). It is also listed as a toxic pollutant, subject to general pretreatment regulations for new and existing sources, and effluent standards and guidelines (351, 3763). Effluent limitations specific to this chemical have been set in the following point source categories: electroplating (3767), organic chemicals, plastics, and synthetic fibers (3777), steam electric power generating (3802), and metal molding and casting (892). Limitations vary depending on the type of plant and industry.

Safe Drinking Water Act (SDWA)

Trichloroethylene is on the list of 83 contaminants required to be regulated under the SDWA of 1974 as amended in 1986 (3781). The maximum contaminant level (MCL) for trichloroethylene in drinking water is 5 μg/L, and the maximum contaminant level goal (MCLG) is zero (3773, 3772). In states with an approved Underground Injection Control program, a permit is required for the injection of trichloroethylene-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)

Trichloroethylene is identified as a toxic hazardous waste (U228) and listed as a hazardous waste constituent (3783, 3784). Non-specific sources of trichloroethylene-containing waste are solvent use (or recovery) activities, chlorinated aliphatic hydrocarbon production, and spent solvent mixtures containing 10% or more trichloroethylene (325). Waste streams from the organic chemicals industry (production of vinyl chloride, 1, 2-dichloroethane and chloroethane) contain trichloroethylene and are listed as specific sources of hazardous waste (3774, 3765). Effective July 8, 1987, the land disposal of hazardous wastes which contain halogenated organic compounds in concentrations greater than or equal to 1000 mg/kg is prohibited. Effective August 8, 1988, the underground injection into deep wells of these wastes is prohibited. Certain variances exist until May, 1990 for some wastewaters and nonwastewaters for which Best Demonstrated Available Technology (BDAT) treatment standards have not been promulgated by EPA (3786). Trichloroethylene is included on EPA's ground-water monitoring list. EPA requires that all hazardous waste treatment, storage, and disposal facilities monitor their ground-water for chemicals on this list when suspected contamination is first detected and annually thereafter (3775).

Comprehensive Environmental Response, Compensation and Liability Act (CERCLA)
Trichloroethylene is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 454 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing trichloroethylene but these depend upon the concentration of the chemicals in the waste stream (3766). Under SARA Title III Section 313, manufacturers, processors, importers, and users of trichloroethylene must report annually to EPA and state officials their releases of this chemical to the environment (3787).

Occupational Safety and Health Act (OSHA)
Employee exposure to trichloroethylene shall not exceed an 8-hour time-weighted average (TWA) of 50 ppm or a 15-minute short-term exposure limit (STEL) of 200 ppm (3539).

Clean Air Act (CAA)
EPA intends to list trichloroethylene as a hazardous air pollutant for which it will establish emission standards under Section 112 of the Clean Air Act (3685).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated trichloroethylene as a hazardous material with a reportable quantity of 454 kg, subject to requirements for packaging, labeling and transportation (3180).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Food, Drug and Cosmetic Act (FDCA)
Trichloroethylene may be present as an extraction residue in the following foods (361):

- spice oleoresins, at a level not exceeding 30 ppm;
- decaffeinated ground coffee, at a level not exceeding 25 ppm;
- decaffeinated instant coffee, at a level not exceeding 10 ppm.

Trichloroethylene is approved for use as an indirect food additive as a component of adhesives (3209).

- State Water Programs

  ALL STATES

  All states have adopted EPA Ambient Water Quality Criteria and NPDWRs (see Water Exposure Limits section) as their promulgated state regulations, either by narrative reference or by relisting the specific numeric criteria. These states have promulgated additional or more stringent criteria:

  ALABAMA

  Alabama requires that the annual average MCL of trichloroethylene in drinking water not exceed 5 μg/L. This applies to all community water systems, and non-community non-transient water systems (3015).

  CALIFORNIA

  California has an MCL and an action level of 5 μg/L for drinking water (3096, 3098).

  CONNECTICUT

  Connecticut has a quantification limit of 2 μg/L for drinking water (3137).

  FLORIDA

  Florida has set an MCL of 3 μg/L for drinking water (3219).

  NEW JERSEY

  New Jersey has set an MCL of 1 μg/L (ppb) for trichloroethylene in drinking waters (3497).

  NEW YORK

  New York has an MCL of 5 μg/L for drinking water, a water quality standard of 10 μg/L for groundwater classed for drinking water supply, and a nonenforceable guideline of 3 μg/L for surface waters (3501).

  OKLAHOMA

  Oklahoma has a water quality criterion of 0.3 μg/L for groundwater (3534).

  RHODE ISLAND

  Rhode Island has an acute freshwater quality guideline of 1950 μg/L and a chronic guideline of 43 μg/L for the protection of aquatic life in surface waters. These guidelines are enforceable under Rhode Island state law (3590).

VERMONT
Vermont has a preventive action limit of 0.5 μg/L and an enforcement standard of 5 μg/L for trichloroethylene in ground-water (3682).

WISCONSIN
Wisconsin has a preventive action limit of 0.18 μg/L and an enforcement standard of 1.8 μg/L for trichloroethylene in ground-water (3840).

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed that solid wastes be listed as hazardous because they exhibit the characteristic defined as EP toxicity when the TCLP extract concentration is greater than or equal to 70 μg/L trichloroethylene. Final promulgation of this Toxicity Characteristic Rule is expected in June, 1989 (1565).

- State Water Programs

  MOST STATES
  Most states are in the process of revising their water programs and proposing changes in their regulations which will follow EPA's changes when they become final. Contact with state officers is advised. Changes are projected for 1989-90 (3683).

  MINNESOTA
  Minnesota has proposed a Sensitive Acute Limit (SAL) of 10175 μg/L for designated surface waters, and chronic criteria of 5 μg/L for designated ground-waters and 25 μg/L for designated surface waters. These criteria are for the protection of human health (3452).

  NEW JERSEY
  New Jersey has proposed a water quality standard of 1 μg/L for class FW2 surface waters (3496).

  WEST VIRGINIA
  West Virginia has proposed a water quality criterion of 3.1 μg/L for Public A surface waters. Final action is expected in late spring 1989 (3835).

EEC Directives

Directive on Ground-Water (538)
Direct discharge into ground-water (i.e., without percolation through the ground or subsoil) of organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment, and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground-water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for organohalogenated substances specify that the concentration of each substance in the shellfish water or in shellfish flesh must not reach or exceed a level which has harmful effects on the shellfish and larvae. The specifications for organohalogenated substances state that the concentration of each substance in shellfish flesh must be so limited that it contributes to the high quality of the shellfish product.

Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (Solvents) (544)
1,2-Dichloroethylene is listed as a Class II/b harmful substance and is subject to packaging and labeling regulations.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground-water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds, excluding inert polymeric materials and other substances referred to in this list or covered by other Directives concerning the disposal of toxic and dangerous waste; chlorinated solvents; organic solvents; biocides and phyto-pharmaceutical substances; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
1,2-Dichloroethylene is classified as a flammable, harmful substance and is subject to packaging and labeling regulations.

## 16.1 MAJOR USES

Trichloroethylene (TCE) is widely used as an industrial solvent, particularly in metal degreasing, which consumes about 90% of approximately 130,000 metric tons of trichloroethylene produced annually in the U.S. (21, 23). The remainder of U.S. production is divided equally between exports and a variety of miscellaneous applications such as in dry cleaning, as a low-temperature heat exchange fluid, as a fumigant, as a diluent in paints and adhesives, in aerospace operations (i.e., to flush liquid oxygen) and in textile processing (21, 23, 25). Due to recycling practices, total solvent usage of trichloroethylene is greater than production and consumption figures would indicate.

In the past, trichloroethylene was used as an extractant in food-processing (e.g., decaffeinating coffee, isolation of spice oleoresins). These applications were discontinued in 1975 based on possible carcinogenic activity (3). Earlier uses of pharmaceutical- grade trichloroethylene as an anesthetic in surgical, dental and obstetrical procedures have also been abandoned (25).

## 16.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

### 16.2.1 Transport in Soil/Ground-water Systems

#### 16.2.1.1 Overview

Trichloroethylene will be relatively mobile in the soil/ground-water system when present at low concentrations (dissolved in water and sorbed on soil) or as a separate organic phase, e.g., from a spill of significant quantities of the chemical.

Transport pathways can be generally assessed by using an equilibrium partitioning model as shown in Table 16-1. These calculations predict the partitioning of low soil concentrations of trichloroethylene among soil particles, soil water and soil air. The estimates for an unsaturated topsoil model indicate that a significant amount of the trichloroethylene is expected to be present in the soil-water and soil-air phases, and thus available to be transported through these phases by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. Diffusion through the soil-air pores up to the surface, and subsequent removal by wind, may be a significant loss pathway. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a much higher fraction of the trichloroethylene is likely to be present in the soil-water phase; this will enhance its transport with flowing ground-water.

TABLE 16-1
EQUILIBRIUM PARTITIONING CALCULATIONS FOR TRICHLOROETHYLENE IN MODEL ENVIRONMENTS[a]

| Soil Environment | Estimated Percent of Total Mass of Chemical in Each Compartment | | |
|---|---|---|---|
| | Soil | Soil-Water | Soil-Air |
| Unsaturated topsoil: [b,c] | | | |
| at 20°C | 94.3 | 3.1 | 2.6 |
| at 10°C | 95.3 | 3.1 | 1.6 |
| Saturated deep soil[d] | 73.5 | 26.5 | - |

a) Calculations based on Mackay's equilibrium partitioning model (34, 35, 36); see Introduction for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.
b) Utilized estimated soil sorption coefficient: $K_{oc}$ = 160.
c) Henry's law constant taken as 0.00674 atm · $m^3$/mol at 20°C and 0.00401 $m^3$/mol at 10°C based on work of Gossett and Lincoff (18).
d) Used sorption coefficient ($K_p$ = 0.086) based on data for montmorillonite and Wurtsmith AFB soils provided by Richter (114).

Based on laboratory studies of the transport and fate of trichloroethylene solutions applied to surface soils (82), the following conclusions can be drawn:

- most trichloroethylene applied to surface soils will volatilize;
- trichloroethylene percolating through the soil column is minimally retarded by sandy soils. Organic matter in the soil increases the retardation rate somewhat;
- volatilization from the soil column occurs at a rate about ten times lower than from a (well-mixed) water column of similar depth.

### 16.2.1.2 Sorption on Soils

The mobility of trichloroethylene in the soil/ground-water system is inversely related to the extent of trichloroethylene sorption on the soil phase. Based on general information on sorption of neutral organic chemicals to soils (31), one would expect the extent of sorption of trichloroethylene to soil to:

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water;
- decrease moderately with increasing dissolved organic matter content of the soil water.

According to Richter (114), trichloroethylene sorption will be minimal on sandy soils, somewhat greater on soils with high clay content (due to the higher surface area available), and greatest on soils with a significant organic carbon content (i.e., greater than 0.1% by wt.).

### 16.2.1.3 Volatilization from Soils

Transport of trichloroethylene vapors through the air-filled pores of unsaturated soils is an important transport mechanism for near-surface soils. Important soil and environmental properties include soil porosity, temperature, convection currents and barometric pressure changes; important chemical properties include the Henry's law constant (H), the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31). The value of H, which represents the tendency of trichloroethylene to volatilize from solution, will:

- increase significantly with increasing temperature;
- increase moderately with increasing salinity of the soil water;
- increase or decrease moderately with increasing dissolved organic matter content of the soil water.

The temperature dependence of H for trichloroethylene has been measured by Gossett and Lincoff (18) and by Leighton and Calo (28). Gossett and Lincoff (18) calculated values of H for trichloroethylene at 20°C and 10°C of 0.0067 and 0.0040 $m^3$/mol, respectively, using the following equation:

$$H(atm \cdot m^3/mol) = \exp [9.703 - 4308/T(°K)].$$

The influence of salinity and dissolved organic matter on the value of H for trichloroethylene has been reported by Gossett and Lincoff (18). A moderate increase in H was observed with increasing concentrations of dissolved organic matter; a slight (perhaps insignificant) decrease in H was noted for trichloroethylene present in a primary municipal effluent. In the latter case, sorption onto suspended solids may have been the cause of the lowering. Callaway et al. (107) found that H (for trichloroethylene) was reduced to 0.28 of the pure water value when measured in a 10% by wt. sodium humate solution. These results suggest that the presence of other materials in water may significantly affect the volatilization of trichloroethylene from surface soils.

No information was available for trichloroethylene with respect to the two other chemical properties affecting volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

### 16.2.2 Transformation Processes in Soil/Ground-water Systems

#### 16.2.2.1 Overview

The persistence of trichloroethylene in soil/ground-water systems is not known. In most cases, it should be assumed that trichloroethylene will persist for months to years (or more). The evidence of possible hydrolysis and biodegradation is contradictory. For trichloroethylene that has been released into the air, photochemical oxidation will result in degradation with a half-life on the order of hours to weeks (6).

Trichloroethylene under normal environmental conditions does not undergo rapid hydrolysis. However, oxygen can accelerate the decomposition rate. The rate of degradation is not significantly pH-dependent, but it could be increased in the presence of metallic iron. The half-life of trichloroethylene due to chemical degradation in water has been estimated - in two separate studies - to be 10.7 months and 30 months (6).

Literature references to microbial biodegradation of compounds such as trichloroethylene are few; the majority report that low molecular weight chloroaliphatics are not metabolized (10). However, significant degradation may be achieved in biological wastewater treatment plants where the microbes have become acclimated to trichloroethylene. Tabak et al. (55), for example, showed significant trichloroethylene biodegradation with gradual adaptation at levels of 5 and 10 mg/L in a static-culture flask-screening procedure. In most soil/ground-water systems, the concentration of microorganisms capable of biodegrading chemicals such as trichloroethylene is very low and drops off sharply with depth. Thus, biodegradation should be assumed to be of minimal importance except, perhaps, in landfills with active microbiological populations.

### 16.2.3 Primary Routes of Exposure From Soil/Ground-water Systems

The above discussion of fate pathways suggests that trichloroethylene is highly volatile in aqueous solutions, moderately soluble in water and not strongly sorbed or bioaccumulated. Trichloroethylene on the soil surface is likely to volatilize, but that portion not removed by volatilization is likely to be mobile in ground-water. These fate characteristics suggest several potential exposure pathways.

Volatilization of trichloroethylene from a disposal site could result in inhalation exposures. The potential for ground-water contamination is high, particularly in sandy soils. The potential for exposure through drinking water is confirmed by the pervasiveness of trichloroethylene in ground-water sources of drinking water in the

United States. The USEPA (62, 64) reported the following results from a variety of surveys of drinking water supplies:

| Survey | No. Sampled | No. Positive | Range of Positives |
|---|---|---|---|
| State Data | 4228 | 624 | Trace - 510,000 μg/L |
| NOMS | 113 | 28 | 0.2 - 49.0 μg/L |
| NSP | 142 | 36 | Trace - 53 μg/L |
| CWSS | 452 | 15 | 0.5 - 210 μg/L |
| GWSS (random data) | 466 | 30 | 0.2 - 78 μg/L |

The state data include only ground-water sources and were compiled from various state reports on local contamination problems. The state data are not considered to be statistically representative of national occurrence. The National Organics Monitoring Survey (NOMS) included data from both ground- and surface water suppliers, as did the National Screening Program (NSP) and the Community Water Supply Study (CWSS). The 1982 Ground-water Supply Survey (GWSS) is the most recent study (531). This survey sampled a total of almost 1000 drinking water systems using ground-water; 466 selected at random, and 500 selected by the state as potentially contaminated. The random results suggest that trichloroethylene is a common contaminant in drinking water, particularly in ground-water as evidenced by the state reports of contamination problems. The USEPA (64) estimates that 3.6% of the nation's ground-water supplies are contaminated with trichloroethylene (≥0.5 μg/L).

The relatively low $K_{oc}$ of trichloroethylene suggests other potential dermal and ingestion exposures resulting from surface water contamination:

- Surface waters may be used as drinking water supplies and result in direct ingestion exposures.
- Aquatic organisms residing in these waters may bioaccumulate this compound and be consumed, also resulting in ingestion exposures.
- Recreational use of these waters may result in dermal exposures.
- Domestic animals may consume or be dermally exposed to contaminated ground- or surface waters; the consumption of meats and poultry could then result in ingestion exposure.

In general, exposures associated with surface water contamination can be expected to be lower than exposures from drinking contaminated ground-water for two reasons: first, trichloroethylene is likely to volatilize upon reaching surface waters; secondly, bioconcentration factors for trichloroethylene are low.

### 16.2.4 Other Sources of Human Exposure

Trichloroethylene is a widely used organic solvent, predominantly in degreasing operations. As a result of fugitive emissions during production, use, and disposal, trichloroethylene has become pervasive in the environment. It is commonly found in all environmental media-air, water (including ground-water) and soil. Due to its volatile nature, trichloroethylene is a common air contaminant. Concentrations in remote areas range from 0-3 $\mu g/m^3$ and in urban areas from 0-47 $\mu g/m^3$. In the vicinity of industrial users, concentrations can be as high as 235 $\mu g/m^3$, and near producers, concentrations of up to 1400 $\mu g/m^3$ have been reported (117). These levels suggest that the general population may be exposed to trichloroethylene in the air from a variety of sources.

## 16.3 HUMAN HEALTH CONSIDERATIONS

### 16.3.1 Animal Studies

#### 16.3.1.1 Carcinogenicity

Hepatocellular carcinomas were induced in $B6C3F_1$ mice exposed to technical-grade trichloroethylene (1170 mg/kg by oral intubation) (111). The validity of this finding has been questioned (771, 772) primarily on the basis of high spontaneous liver cancer rates in the mouse strain and the addition of highly mutagenic epoxides to technical-grade trichloroethylene as stabilizers. The high dosage levels employed may also have saturated usual pathways of removal from the body, resulting in atypical products which might be responsible for the carcinogenic effects found in mice. Several studies conducted with purified trichloroethylene in rats and other strains of mice failed to show liver carcinogenicity (773, 774, 775, 776); however, females of one mouse strain showed an increase in lymph cancer, attributed to an immunity- suppressing effect at high concentrations; lymph cancer is peculiar to this strain of mice (776).

Recently released data from a bioassay conducted by the NTP with a purified (>99.9% pure), epoxide-free sample of TCE confirm the results of the earlier study (111), and thus excluded the added epoxide stabilizers as a necessary factor in the increased incidence of hepatocellular carcinoma noted in $B6C3F_1$ mice. Only one dose was tested by the NTP, 1000 mg/kg/day by gavage, 5 days/week for 103 weeks; this treatment resulted in a statistically significant increase of hepatocellular carcinoma (i.e., clear evidence) in both male and female $B6C3F_1$ mice (777).

A study run concurrently with F344 rats given 500 or 1000 mg/kg/ day purified TCE by gavage, 5 days/week for two years was considered inadequate for evaluation of carcinogenic risk due to poor survival (777). Renal tubular-cell tumors were seen in treated males but no carcinogenic response was evident in female rats. Nephropathy was seen in both sexes (98-100% incidence).

In summary, TCE has not been shown to be a potent carcinogen in rats but it appears to be a potent carcinogen in one specific strain of mice (i.e., $B6C3F_1$). IARC (3316) has listed TCE in category 3 (insufficient evidence) in its weight-of-evidence ranking for potential carcinogens. EPA has listed it in Group B2 (inadequate human evidence, sufficient animal evidence, a probable human carcinogen) (3805).

### 16.3.1.2 Genotoxicity

Positive to weakly positive responses have been noted in a mammalian cell transformation assay (735,708) and weak responses in bacterial and yeast test systems in the presence of metabolic activation (156,649, 3653). Positive responses were observed in Salmonella exposed in a desiccator (3644, 3450), but negative responses were noted in a 20-minute preincubation assay with or without hamster liver microsomes (3469). Positive results were also reported in an in vivo somatic mutation assay (i.e., spot test) in mice (59). Negative results were observed in a dominant lethal assay with rats and mice (736, 737), and in a sister chromatid exchange study using CHO cells treated for one hour in the presence of a metabolic activation system (3837). Galloway et al. observed a weakly positive response for sister chromatid exchanges in CHO cells treated for 25 hrs without activation, but no increase in chromosomal aberrations in these cells with or without activation (3235). Positive results were claimed for the induction of aneuploidy in Chinese hamster V79 cells treated in culture (3537). Male and female mice gavaged with trichloroethylene showed no increase above controls of unscheduled DNA synthesis in their liver cells (3456). Shimada et al. suggested that "stabilizers" found in samples of trichloroethylene may be responsible for the positive effects observed (3644). Trichloroethylene with stabilizer induced 4- to 37-fold increases in revertants in Salmonella strains TA100 and 1535 regardless of the presence of activation, whereas low stabilized trichloroethylene was toxic above 5%, and produced no reversions at 1, 1.5 or 5% with moderate toxic effects. In this same study, trichloroethylene was negative in the unscheduled DNA synthesis test when rat hepatocytes were treated in cell culture.

### 16.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Trichloroethylene was neither embryotoxic nor teratogenic (i.e., did not produce birth defects) in either mice or rats exposed for 7 hours to 300 ppm on days 6 to 15 of pregnancy (115). Two other studies conducted in rats exposed to trichloroethylene concentrations up to 1800 ppm confirm these results (2). In a subchronic oral exposure study, Manson et al. (110) gavaged rats with 10, 100, or 1000 mg/kg/day of trichloroethylene for 2 weeks prior to mating and throughout the 21 days of pregnancy. Fertility was not affected in any treatment group; however, in the 1000 mg/kg group, 5 out of 23 treated females died and maternal weight gains were depressed. In this high dose group, neonatal survival was significantly depressed. Several studies have been conducted in rats and mice to determine a possible effect of TCE on the central nervous system. Westergren et al. (3836) exposed male and female mice by inhalation to 150 ppm TEC for 30 days. The mice were allowed to mate and

exposure of females continued until the first litter was born. The specific gravity of the treated pup's brain was significantly lower than that of the controls on days 1, 10, and 20-22. Specific gravity of the brain is considered an indication of myelination and maturation. The difference was no longer observed at one month of age. Noland-Gerbec et al. (3509) observed a decrease in glucose uptake and/or metabolism in the brains of pups exposed to 312 mg/L TEC during gestation and lactation. These values returned to control levels by day 21. Taylor et al. (3702) exposed pregnant rats to 312, 625, or 1250 mg/L TCE during the same period and observed higher exploratory behavior in 60- and 90-day old male offspring which were exposed to any level of TCE. Locomotor activity was significantly higher in 60-day old males which were exposed to 1250 mg/L. These data suggest that TCE has long-term effects on behavior.

### 16.3.1.4 Other Toxicologic Effects

#### 16.3.1.4.1 Short-term Toxicity

In laboratory animals the acute toxicity of trichloroethylene is low. Oral $LD_{50}$ value of 2402 mg/kg in the mouse has been reported (3280). Adams et al. (739) showed that the highest levels of exposure with no effects on rats were: 18 minutes at 20,000 ppm; 36 minutes at 12,000 ppm; 1.4 hours at 4800 ppm and 5 hours at 3000 ppm. $LC_{Lo}$ values of 8000 ppm · 4 hr and 300 ppm · 2 hr have been reported for the rat and mouse, respectively (59).

Unlike other chlorinated hydrocarbons, studies have shown trichloroethylene to be a low inducer of liver and kidney toxicity (12). Kylin et al. (108) found no evidence of liver damage in rats exposed to vapor levels of 3180 ppm for 4 hours. Exposure to vapor levels of 21,600 ppm for 15-90 hours showed increased intracellular lipid levels but no liver necrosis (740).

#### 16.3.1.4.2 Chronic Toxicity

Unlike other chlorinated hydrocarbons, chronic exposure to trichloroethylene induces low to moderate liver and kidney toxicity. Early experiments with dogs exposed to either 750 ppm, 8 hours/day, 6 days/week for 3 weeks, or 579 ppm for 6 hours/day, 5 days/week for 8 weeks, produced degeneration of liver cells, anemia, weight loss, lethargy and diarrhea (116). Later studies using more purified trichloroethylene, however, indicate little or no toxic effects on the liver (108). In general, liver toxicity is evident only with exposures sufficient to induce anesthesia (12).

In animals, the maximum concentrations of trichloroethylene producing no toxic effects were: rats and rabbits, 200 ppm; guinea pigs, 100 ppm; monkeys, 400 ppm; animals were exposed 7 hours/day, 5 days/ week for 6 months (739). In another study, rats, rabbits, dogs, monkeys and guinea pigs exposed to 700 ppm, 8 hours/day, five days/week for 6 weeks, or continuously to 35 ppm for 90 days, also exhibited no visible signs of toxicity (741).

### 16.3.2 Human and Epidemiologic Studies

#### 16.3.2.1 Short-term Toxicologic Effects

Trichloroethylene can be absorbed by inhalation or ingestion and through skin contact. In humans, the predominant toxic manifestation of trichloroethylene exposure, regardless of route, is depression of the central nervous system which is demonstrated by dizziness, headache, visual disturbances, incoordination similar to that induced by alcohol, sleepiness, tremors, nausea and vomiting. These effects may occur at vapor exposures ranging from 83 minutes at 160 ppm to 25 minutes at 1000 ppm (39, 117, 47). Complete recovery usually occurs within 6 to 72 hours (45). Exposures to trichloroethylene vapor concentrations of 3000 ppm for less than 10 minutes have produced loss of consciousness and death (21, 47).

Cardiac arrhythmias and death due to ventricular fibrillation and cardiac arrest have also been reported, usually subsequent to acute exposure to trichloroethylene concentrations above 15,000 ppm (23, 54). Sensitization of the heart to adrenalin has been demonstrated with exposure to anesthetic levels of trichloroethylene (i.e., about 4700 ppm or above) (25, 46). In addition to the involvement of the nervous system and the cardiovascular system, exposure to trichloroethylene may also cause transient increases in serum transaminases (an indication of liver damage) in humans; however, these increases usually disappear after exposure is terminated (70).

The effects produced by ingestion of trichloroethylene are similar to those noted with inhalation exposure. Pelka and Zach (113) reported that accidental ingestion of approximately 150 mL trichloroethylene resulted in acute kidney failure, uremia, and liver and cardiovascular damage. The lowest reported oral lethal dose in man is 7g/kg body weight (47).

Numerous fatalities, including incidents of sudden death, probably due to ventricular fibrillation, have occurred following accidental exposure to high concentrations of trichloroethylene (12, 49, 25, 46). Adverse effects on visual perception and motor skills were observed in volunteers exposed for 2 hours to 1000 ppm trichloroethylene (2).

Cranial nerve toxicity, particularly impairment of the trigeminal and optic nerves, has been linked to trichloroethylene exposure (105, 106, 19, 109); however, this is probably caused by a breakdown product, dichloroacetylene (103).

Local exposure to trichloroethylene vapor (5 ppm) may cause irritation of the eyes, nose and throat (47). The liquid, if splashed into the eyes, may cause transient pain, burning and irritation, but if flushed with water quickly, would not be expected to cause permanent injury (12). Prolonged skin contact may cause local irritation and blister formation; paralysis of the fingers has been reported after repeated, intermittent immersion of the hands in liquid trichloroethylene (117).

Alcohol may make the signs and symptoms of trichloroethylene exposure worse (54), and even low level trichloroethylene exposure followed by ingestion of alcohol produces a dermal flushing known as "degreaser's flush" (45). This effect does not appear to be injurious.

### 16.3.2.2 Chronic Toxicologic Effects

Chronic exposure to trichloroethylene at levels in excess of those found or expected in ambient air result primarily in neurological and neuropsychiatric symptoms (738). In a detailed study of 104 persons exposed to trichloroethylene in the metal, rubber and dry-cleaning industries, two-thirds exhibited CNS symptoms. These included headache, dizziness, tremors, sleepiness, fatigue, lightheadedness, nausea and vomiting. Only 8 of the 104 workers who had been exposed to trichloroethylene for more than 3 years were without symptoms. Follow-up studies conducted 3 to 7 years after exposure ceased showed little residual evidence of trichloroethylene intoxication. The workers reported that symptoms subsided within 4 to 5 months after termination of exposure. The average exposure level during the study ranged from 200 to 400 ppm (754).

No consistent behavioral deficits have been reported for trichloroethylene below exposures of 300 ppm (91). Repeated exposure to 100 or 200 ppm trichloroethylene, 7.5 hours/day, 5 days/week for several days (exact number of exposures unspecified) was reported to induce no deleterious behavioral effects in test individuals (2). Biochemical changes relevant to liver function impairment have repeatedly been reported; however, serious liver disease attributable to chronic exposure to trichloroethylene has not been observed (70).

No epidemiologic evidence suggests that trichloroethylene exposure is associated with an increased risk of cancer in humans. Two cohort mortality studies in Sweden and Finland suggest no increased risk of cancer; however, data are presently insufficient to analyze a site specific cancer risk (104, 112).

### 16.3.4 Levels of Concern

Based on the results of the controversial NCI (111) study in mice, the USEPA (61) has established a zero ambient water concentration for the maximum protection of human health from potential carcinogenic effects of exposure to trichloroethylene through ingestion of water and contaminated aquatic organisms. In that attaining a zero concentration level may be infeasible in some cases, the concentrations of trichloroethylene in water calculated to result in incremental lifetime cancer risks of 1E-05, 1E-06, and 1E-07 from ingestion of both water and contaminated aquatic organisms were estimated to be 27 $\mu$g/L, 2.7 $\mu$g/L and 0.27 $\mu$g/L, respectively (355). Risk estimates are expressed as a probability of cancer after a lifetime consumption of 2 liters of water per day and 6.5 g of fish per day containing a specified concentration of the contaminant. Thus, a risk of 1E-05 implies a lifetime daily consumption of two liters of drinking water and 6.5 g of contaminated fish at the criterion level of 27 $\mu$g TCE/L would be expected to produce one excess case of cancer above the

normal background incidence for every 100,000 people exposed. It should be emphasized that these extrapolations are based on a number of assumptions and should be taken as crude estimates of human risk at best.

The IARC (3316) lists TCE in category 3 (insufficient evidence in humans; limited evidence for carcinogenicity in animals) in its weight-of-evidence ranking for potential carcinogens. The NTP (3517) noted that studies were inadequate to evaluate the carcinogenic activity of TCE in rats. EPA (3805) lists TCE in Group B2 (inadequate human evidence, sufficient animal evidence, a probable carcinogen). The MCLG and MCL for TCE are 0 and 5 $\mu$g/L, respectively (3742). A lifetime exposure concentration protective of adverse, non-cancer health effects, that assumes all of the exposure to a contaminant is from a drinking water source. The WHO (666) recommends a level of 30 $\mu$g/L for drinking water.

OSHA (3539) currently permits exposure to 50 ppm as an 8-hour TWA with a STEL of 200 ppm.

### 16.3.4 Hazard Assessment

Exposure to both technical-grade and epoxide-free trichloroethylene has been shown to result in an increase in liver cancer in both sexes of $B6C3F_1$ mice (111, 777). However, there is a substantial body of opinion in the scientific community to the effect that the mouse liver overreacts to chlorinated organic compounds (738).

Other studies conducted with purified trichloroethylene have produced negative carcinogenic findings in rats and other mouse strains (773, 774, 775, 776). Despite its use as an anesthetic for several years and its extensive industrial applications, there is no epidemiological evidence to suggest that trichloroethylene exposure is associated with an increased risk of cancer in humans. Other than the cancer risk, studies in animals indicate trichloroethylene to be of low toxicity, both acutely and chronically (59, 117, 739, 740); damage to renal tubules has been observed in experimental animals at exposures above 1000 mg/kg (777). No fetal toxicity or reproductive problems have been noted (115, 110).

Signs of toxicity, primarily related to the depressant effects of trichloroethylene on the nervous system, become evident in humans only after exposure to trichloroethylene concentrations of 800 $mg/m^3$ and above, and disappear soon after removal from the contaminated environment (117). Long-term exposure to trichloroethylene has produced some degree of liver enlargement and biochemical changes related to liver function impairment in workers exposed to a broad range of trichloroethylene concentrations during their employment; serious liver disease attributable to chronic trichloroethylene exposure has not been observed. As with acute exposure, the nervous system is the main target of long-term trichloroethylene exposure, with psychological symptoms predominating (2, 91, 70). Rare cases of sudden death have been reported with trichloroethylene and are suspected to be due to ventricular fibrillation (12, 49, 25, 46).

## 16.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of trichloroethylene concentrations in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of trichloroethylene, care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. However, recent studies (3430) show large losses of volatiles from soil handling. At the present, the best procedure is to collect the needed sample in an EPA VOA vial, seal with a foil-lined septum cap, and analyze the entire contents in the vial using a modified purge and trap apparatus. In addition to the targeted samples, quality control samples such as field blanks, duplicates, and spiked matrices may be specified in the recommended methods.

EPA-approved procedures for the analysis of trichloroethylene in aqueous samples include EPA Methods 601, 624, 1624 (65), and Methods 8010 and 8240 (63). An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the trichloroethylene from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the trichloroethylene and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; trichloroethylene is then detected with a halide specific detector (Methods 601 and 8010) or a mass spectrometer (Methods 624, 1624, and 8240). For samples that contain high concentrations, direct injection may also be used. The generalized procedure for sample preparation for the analysis of volatile organics by purge and trap (Method 5030) (63) also recommends that samples be screened prior to the purge and trap step to prevent contamination of the system. The recommended screening techniques involve the analysis of a headspace sample by GC with photo-ionization or electrolytic conductivity detectors or the analysis of a solvent extract by GC with flame ionization or electrolytic conductivity detectors.

The EPA procedures recommended for trichloroethylene analysis in soil and waste samples, Methods 8010 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method for low level samples (< 1 mg/kg) involves dispersing the soil or waste sample in water and purging in a heated purge and trap device. The trap is desorbed and analyzed as described above. Recently introduced wide bore capillary columns show promise for increasing the performance of the GC analysis (3402, 3184, 3443).

Other methods that have been used to quantitate trichloroethylene in soil and water include purge and trap with flame ionization detection (3263) and solvent extraction with electron capture detection (3352).

Trichloroethylene detection limits that can be obtained in wastewaters and non-aqueous samples (wastes, soils, etc.) are shown below.

| Aqueous Detection Limit | Non-Aqueous Detection Limit |
|---|---|
| 0.12 μg/L (Method 601) | 1.2 μg/kg (Method 8010) |
| 1.9 μg/L (Method 624) | 5.0 μg/kg (Method 8240) |
| 10.0 μg/L (method 1624) | |
| 1.2 μg/L (Method 8010) | |
| 5.0 μg/L (Method 8240) | |

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# APPENDIX 1

## USEFUL HANDBOOKS, DATABOOKS, RESPONSE GUIDES AND AIR FORCE DOCUMENTS

A listing of useful handbooks, databooks and response guides, all relating to the release of hazardous or toxic chemicals to the environment, the properties and hazards of the chemicals, initial responses to spills of such chemicals, or subsequent remedial action follow. The contents of each publication is briefly described. The following listing is not intended to be inclusive of all publications of this kind. However, it is felt that the acquisition and central location of these reports (at key Air Force offices) would provide a valuable resource.

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- A Method for Determining the Compatibility of Hazardous Wastes

  Authors: H. K. Hatayama et al. (April 1980)

  Available from: U.S. Environmental Protection Agency
  Municipal Environmental Research Laboratory
  Cincinnati, OH
  (EPA Report No. EPA-600/2-80-076)
  (NTIS Report No. PB80-221005)

  Contents: Provides method and chart for defining compatibility of various families of hazardous materials and wastes.

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- Accident Management Orientation Guide

  Authors: D. K. Shaver et al. (October 1983)

  Available from: Air Force Rocket Propulsion Laboratory
  Air Force Systems Command
  Edwards Air Force Base
  California 93523
  (Report No. AFRPL-TR-82-075)

  Contents: This document identifies guidelines for mitigating hazards associated with an in-service railroad derailment or a railroad yard accident involving hazardous materials of interest to the Air Force.

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- Carbon Adsorption Isotherms for Toxic Organics

  Authors: R. A. Dobbs and J. M. Cohen (April 1980)

  Available from: U.S. Environmental Protection Agency
  Office of Research and Development
  Cincinnati, OH
  (EPA Report No. EPA-600/8-80-023)

  Contents: Provides detailed data on the effectiveness of carbon for removal of organic substances from water.

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- Chemical Hazards of the Workplace

  Authors: N. H. Proctor and J. P. Hughes (1978)

  Available from: J. B. Lippincott Company
  Philadelphia, PA

  Contents: Provides data on the toxicological effects of chemicals and suggests medical treatment protocols in more detail than given elsewhere.

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- CHRIS Hazardous Chemical Data

  Author: U.S. Coast Guard (1985)

  Available from: Superintendent of Documents
  U.S. Government Printing Office
  Washington, D.C. 20402
  (Stock No. 050-012-00147-2)

  Contents: Provides a wide variety of data on more than 1000 hazardous materials when ordered with various addendums. A separate volume (Stock No. 050-012-00158-8) provides graphs of temperature dependent physical properties.

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- **Dangerous Properties of Industrial Materials, 7th edition**

  Author: N. I. Sax, ed. (1989)

  Available from: Van Nostrand Reinhold
  New York, NY

  Contents: A well-known handbook that provides a brief summary of the toxicology and properties of numerous hazardous substances.

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- **Dangerous Properties of Industrial Materials Report**

  Author: N. I. Sax, ed. (bimonthly publication)

  Available from: Van Nostrand Reinhold Company
  New York, NY

  Contents: Each bimonthly report provides detailed data on the hazards and environmental effects of several chemicals. Much of the data is from the EPA's Oil and Hazardous Materials-Technical Assistance Data System (OHM-TADS) and similar sources.

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- **Emergency Action Guides**

  Authors: P. C. Conlon and A. M. Mason, eds. (1984)

  Available from: Bureau of Explosives
  Association of American Railroads
  1920 L Street N.W.
  Washington, D.C. 20036

  Contents: Provides detailed data and spill response information on each of the 134 materials that comprise over 98 percent of the hazardous commodities transported by rail in the United States.

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- **Emergency Handling of Hazardous Materials in Surface Transportation**

  Author: P. J. Student, ed. (1981)

  Available from: Bureau of Explosives
  Association of American Railroads
  1920 L Street N.W.
  Washington, D.C. 20036

  Contents: Provides brief spill response recommendations for each hazardous material regulated by the U.S. Department of Transportation.

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- **Emergency Response Guidebook**

  Author: Materials Transportation Bureau (1987)

  Available from: U.S. Department of Transportation
  Materials Transportation Bureau
  Attention: DMT-11
  Washington, DC 20590
  (Publication DOT P5800.3)

  Contents: A guide for initial actions to be taken by emergency service personnel during hazardous material incidents.

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- **Fire Protection Guide on Hazardous Materials**

  Author: National Fire Protection Association (1986)

  Available from: National Fire Protection Association
  Batterymarch Park
  Quincy, MA 02269

  Contents: Flash Point Index of Trade Name Liquids Fire Hazard Properties of Flammable Liquids, Gases, and Volatile Solids (NFPA 325M) Hazardous Chemicals Data (NFPA 49) Manual of Hazardous Chemical Reactions (NFPA491M)

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- Groundwater Contamination Response Guide, Volume I: Methodology, Volume II: Desk Reference

  Authors: J. H. Guswa and W. J. Lyman (1983)

  Available from: National Technical Information Service
  Springfield, VA
  (as U.S. Air Force Report ESL-TR-82-39)
  or
  Noyes Publications
  Park Ridge, NJ
  (under the title "Groundwater Contamination and Emergency Response Guide" (1984))*

  Contents: Provides an overview of ground-water hydrology and a current technology review of equipment, methods, and techniques used to investigate incidents of ground water contamination by chemicals.

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*Noyes Publications also contain a reproduction of the report by A. S. Donnigian, Jr. et al.: Rapid Assessment of Potential Ground-Water Contamination Under Emergency Response Conditions, a 1983 report to the U.S. Environmental Protection Agency.

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- Ground-Water Hydrology Workbook

  Authors: E.W. Artiglia and G.R. New (1984)

  Available from: USAF Occupational and Environmental Health Laboratory
  Brooks AFB, TX 78235
  (Report No. 84-168EQ111DGB)

  Contents: Summarizes introductory articles in ground-water hydrology of importance to base bioenvironmental engineers involved with the IRP program.

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- Guidelines Establishing Test Procedures For The Analysis of Pollutants Under the Clean Water Act, Appendix A.

  Author: U.S. Environmental Protection Agency (1984)

  Available from: Federal Register
  Volume 49(209):43234
  October 26, 1984

  Contents: Methods for analysis of environmental samples.

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- Guidelines for the Selection of Chemical Protective Clothing

  Authors: A.D. Schwope et al. (1987)

  Available from: U.S. Environmental Protection Agency
  Washington, D.C.

  Contents: Denotes compatibility of rubber and plastic clothing materials with various chemicals; provides guidelines for clothing selection and use.

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- Guidelines for the Use of Chemicals in Removing Hazardous Substances Discharges

  Authors: C. K. Akers, R. J. Pilie and J. G. Michalovic (1981)

  Available from: U.S. Environmental Protection Agency
  Office of Research and Development
  Cincinnati, OH
  (EPA Report No. EPA-600/2-81-205)

  Contents: Report provides guidelines on the use of various chemical and biological agents to mitigate discharges of hazardous substances.

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- **Handbook for Evaluating Remedial Action Technology Plans**

  Authors: J. Ehrenfeld and J. Bass (1983)

  Available from: U.S. Environmental Protection Agency
  Office of Research and Development
  Cincinnati, OH
  (EPA Report No. EPA-600/1-83-076)

  Contents: Provides information on over 50 remedial action technologies for cleanup of chemically-contaminated sites.

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- **Handbook of Chemical Property Estimation Methods**
  (subtitle: Environmental Behavior of Organic Compounds)

  Authors: W. J. Lyman, W. F. Rechl, D. H. Rosenblatt, eds. (1982)

  Available from: McGraw-Hill Book Co.
  New York, NY

  Contents: Provides estimation methods for (and discussion of) 26 environmentally-important properties of organic chemicals.

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- **Handbook of Environmental Data on Organic Chemicals, 2nd edition**

  Author: K. Verschueren (1983)

  Available from: Van Nostrand Reinhold
  New York, NY

  Contents: Provides detailed property and environmental data on numerous organic substances.

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- **Handbook of Toxic and Hazardous Chemicals**

  Author: M. Sittig (1985)

  Available from: Noyes Publications
  Park Ridge, NJ

  Contents: Discusses a wide range of topics for numerous chemicals, with special emphasis on toxicology and protective measures.

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- **Hazardous Chemicals Data Book, 2nd edition**

  Author: G. Weiss, ed. (1986)

  Available from: Noyes Data Corporation
  Park Ridge, NJ

  Contents: Reproduction of data (physicochemical properties, hazards, toxicity, etc.) related to chemical spill response from (1) CHRIS Hazardous Chemical Data (1978) and (2) *Material Safety Data Sheets* prepared by Oak Ridge National Laboratory.

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- **Herbicide Handbook, 5th edition**

  Author: Weed Science Society of America (1983)

  Available from: Weed Science Society of America
  309 West Clark Street
  Champaign, IL 61820

  Contents: Provides basic information on physiococchemical properties, uses, environmental fate, physiological and biochemical behavior, and toxicological properties for most herbicides in use. (Previous editions may cover out-of-use herbicides.)

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- **Manual for the Control of Hazardous Material Spills - Vol. 1: Spill Assessment and Water Treatment Techniques**

  Authors: K. R. Huibregtse et al. (November 1977)

  Available from: U.S. Environmental Protection Agency
  Office of Research and Development
  Cincinnati, OH
  (EPA Report No. EPA-600/2-77-227)

  Contents: Provides both general and specific information on responding to spills of hazardous materials, particularly those into water.

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- **Methods to Treat, Control and Monitor Spilled Hazardous Materials**

  Authors: R. J. Pilie et al. (1975)

  Available from: U.S. Environmental Protection Agency
  Industrial Waste Treatment Research Laboratory
  Edison, NJ
  (EPA Report No. EPA-670/2-75-042)

  Contents: Special studies of selected chemical spill response measures plus matrix of possible spill response measures for 250 hazardous liquids.

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- **NIOSH Manual of Analytical Methods, 3rd edition**

  Author: Peter M. Eller, ed. (1984)

  Available from: Superintendent of Documents
  U.S. Government Printing Office
  Washington, D.C. 20402

  Contents: Contains sampling and analytical methods for use in industrial hygiene environmental monitoring.

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- **NIOSH/OSHA Occupational Health Guidelines for Chemical Hazards**

  Authors: F. W. Mackison et al., eds. (January 1981)

  Available from: Superintendent of Documents
  U.S. Government Printing Office
  Washington, D.C. 20402
  (DHHS (NIOSH) Publication No. 81-123)

  Contents: Provides information on toxicology, chemical properties, first aid, and personal protective clothing and equipment for many OSHA-regulated commodities.

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- **Patty's Industrial Hygiene and Toxicology - Vol. 2A,B,C: Toxicology**

  Authors: G.D. Clayton and F.E. Clayton, eds. (1981-1982)

  Available from: John Wiley & Sons
  New York, NY

  Contents: Provides extensive discussion of the properties and toxicology of numerous chemicals.

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- **Perry's Chemical Engineers Handbook**

  Authors: R. H. Perry and D. Green, eds. (1984)

  Available from: McGraw-Hill Book Company
  New York, NY

  Contents: Contains extensive data on the properties of chemicals and on their compatibility with various materials of construction (plus numerous other topics).

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- **Pesticide Manual, 7th edition**

  Author: C. R. Worthing, ed. (1983)

  Available from: British Crop Protection Council Publications
  Worcestershire WR13 15LP
  ENGLAND

  Contents: Provides a brief review of analysis, uses and toxicity of chemicals and microbial agents used as active components of pest-control products.

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- **Post Accident Procedures for Chemicals and Propellants**

  - Interim Report for the Period 8/11/80 to 3/31/81 (September 1982) (Report No. AFRPL-TR-82-031)
  - Interim Report for the Period 4/81 to 1/82 (September 1982) (Report No. AFRPL-TR-82-032)
  - Guidelines Manual (January 1983) (Report No. AFRPL-TR-82-077)

  Authors: D. K. Shaver et al.

  Available from: Air Force Rocket Propulsion Laboratory
  Air Force Systems Command
  Edwards Air Force Base
  California 93523

  Contents: This is a series of manuals providing information and data required to respond to spills of chemicals and propellants of special interest to the Air Force.

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- Quality Criteria for Water

  Author: U.S. Environmental Protection Agency (July 1976)

  Available from: Superintendent of Documents
  U.S. Government Printing Office
  Washington, D.C. 20402
  (Stock No. 055-001-01049-4)

  Contents: This is EPA's well-known guide to water quality criteria commonly referred to as the "redbook."

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- Registry of Toxic Effects of Chemical Substances

  Authors: R. L. Tatken and R. J. Lewis, Sr., eds. (June 1983)

  Available from: Superintendent of Documents
  U.S. Government Printing Office
  Washington, D.C. 20402
  (DHHS [NIOSH] Publication 83-107)

  Contents: Summarizes results of primarily short-term toxicological experiments for thousands of chemicals.

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- Standard Methods for the Examination of Water and Wastewater, 15th edition

  Authors: Arnold Greenberg et al., eds. (1985)

  Available from: American Public Health Association
  1015 18th Street
  Washington, D.C.

  Contents: Methods for analysis of environmental samples.

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- Supplement to Development Document, Hazardous Substances Regulations, FWPCA as Amended 1972

  Author: U.S. Environmental Protection Agency (November 1975)

  Available from: U.S. Environmental Protection Agency
  Office of Water Planning and Standards
  Washington, D.C. 20460

  Contents: Discusses the environmental effects of numerous water pollutants.

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- Test Methods for Evaluating Solid Waste-Physical Chemical Methods, 3rd edition

  Author: U.S. Environmental Protection Agency (1987)

  Available from: Superintendent of Documents
  U.S. Government Printing Office
  Washington, D.C. 20460
  (Report No. SW-846)

  Contents: Methods for analysis of environmental samples.

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- TLVs-Threshold Limit Values for Chemical Substances and Physical Agents in the Work Environment and Biological Exposure Indices with Intended Changes for 1987-1988

  Author: American Conference of Governmental Industrial Hygienists (1987)

  Available from: American Conference of Governmental Industrial Hygienists
  6500 Glenway Ave., Bldg. D-5
  Cincinnati, OH 45211

  Contents: This booklet (or the latest version of it) presents recommended exposure limits for airborne concentrations of toxic materials in the working environment.

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- **Toxicology of the Eye**

  Author: W. M. Grant (1986)

  Available from: Charles C. Thomas - Publisher
  Springfield, IL

  Contents: An excellent source of information on the effects of numerous chemicals and other substances on the eyes.

---

- **USAF OEHL Recommended Sampling Procedures**

  Author: USAF Occupational and Environmental Health Laboratory (January 1982)

  Available from: USAF Occupational and Environmental Health Laboratory
  Brooks AFB, TX 78235
  (Limited Distribution)

  Contents: Outlines standardized sampling procedures with appropriate collection and preservation techniques for submission of samples to USAF OEHL for analysis.

---

- **Water-Related Environmental Fate of 129 Priority Pollutants (2 volumes)**

  Authors: M. A. Callahan et al. (December 1979)

  Available from: U.S. Environmental Protection Agency
  Washington, D.C.
  (EPA Report No. EPA-440/4-79-029a and -029b)
  (NTIS No. PB80-204373 and PB80-204381)

  Contents: Individual chapters address the fate of priority pollutants in the environment.

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# PERTINENT AIR FORCE PUBLICATIONS FOR THE USAF INSTALLATION RESTORATION PROGRAM

| PUBLICATION | COMMENT |
|---|---|
| AFR 161-8 | Establishes USAF permissible exposure limits for chemical substances. |
| AFR 161-17 | Establishes USAF OEHL consultant services in Environmental Engineering, Industrial Hygiene, Occupational Health, Radiation Protection, and Analytical Chemistry. |
| AFR 161-44 | Establishes USAF drinking water standards for common contaminants. For the most part, these are the same as the National Primary and Secondary Drinking Water Standards. |
| AFR 19-1 | Establishes the USAF Environmental Protection Program. |
| AFR 19-7 | Establishes responsibilities for environmental monitoring for Air Force installations. This regulation defines the roles of the Civil Engineer, the Bioenvironmental Engineer, and others with respect to environmental pollution monitoring. |
| DEQPPM 80-8 | DoD implementation of RCRA. |
| DEQPPM 80-9 | DoD guidance on the proper handling, storage, and disposal of PCB and PCB items. |
| DEQPPM 81-5 | DoD guidance on the Installation Restoration Program to identify and evaluate past DoD hazardous material disposal sites on DoD installations and control migration from such sites. |
| EO 12088 | Requires federal compliance with applicable federal, state, and local pollution control standards (procedural and substantiative) the same as any other industry or private person. |
| GWMR | Quarterly publication on ground-water monitoring remedial actions. Presents technical articles on contaminant transport, analytical methods, sampling methodology, and data interpretation. |
| IRPMC | Establishes the management concept for the IRP Phase II program. |
| LEEV LTR | Policy letters formulated by USAF HQ/LEEV. |

| | |
|---|---|
| NCP | Establishes procedures for response to potential for confirmed contamination of our nation. |

## APPENDIX 2

## U.S. AIR FORCE POINTS OF CONTACT FOR THE INSTALLATION RESTORATION PROGRAM

- Mr. Gary D. Vest
  Maj. Patrick T. Fink
  SAF/MIQ
  Washington, D.C. 20330-5000
  AV 227-9297
  Commercial: (202) 696-9297

  Office of the Assistant Secretary of the Air Force
  Deputy for Environment and Safety

  Responsible for overall Air Force IRP guidance.

---

IRP GROUP

- Maj. Scott L. Smith, Branch Chief
  AV 297-0275
  Responsible for IRP engineering policy formulation.

- Maj. Roy K. Soloman
  AV 297-0275
  Responsible for Environmental Compliance Assessment and Management Program (ECAMP), Environmental Protection Committee, and IRP implementation.

- Col. Raymond A. Malinovsky
  Chief, Environmental Quality Division
  Director of Engineering and Services
  HQ USAF/LEEV
  Bolling Air Force Base
  Washington, DC 20332-5000

- Capt. Gerald L. Hromowyk
  AV 297-0275
  Responsible for spill policy and management information systems.

- Capt. Charles M. Groover
  AV 297-0275
  Responsible for underground storage tanks and training.

- Mr. Earl E. Kneeling
  AV 297-4174
  Responsible for Defense Environmental Restoration Program policy.

- Mr. Jeffery J. Short
  AV 297-0275
  Responsible for Third Party Sites.

- Col. Thayer J. Lewis, Chief
  Bioenvironmental Engineering
  HQ USAF/SGPA
  Bolling AFB, DC 20332-6188
  AV 297-1737
  Commercial: (202) 767-1737

- Lt. Col. Edward W. Artiglia
  AV 297-1738
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---

- Col. Frank P. Gallagher
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  Tyndall AFB, FL 32403-6001
  AV 970-2097/2098
  Commercial: (904) 283-2097/2098

  USAF Engineering and Services Center
  Engineering and Services Laboratory
  Environics Division

  Responsible for IRP engineering research and development.

---

- Mr. Emile Y. Baladi
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  Brooks AFB, TX 78235-5000
  AV 240-2158/2159
  Commercial: (512) 536-2158/2159

  USAF Occupational and Environmental Health Laboratory Technical Services Division

  Responsible for IRP Phase II technical program management.

---

- Dr. Jeffrey W. Fisher
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  Wright-Patterson AFB, OH 45433-6573
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  Commercial: (513) 255-2704

  Harry G. Armstrong Aerospace Medical Research Laboratory Toxic Hazards Division

  Responsible for IRP health effects research.

---

- Lt. Col. Stanley O. Hewins
  USAF OEHL/ECO
  Brooks AFB, TX 78235-5000
  AV 240-2063
  Commercial: (512) 536-2063

  USAF Occupational and Environmental Health Laboratory Consultant Services Division
  Environmental Health Branch

  Responsible for Toxicology Consultant Service.

---

- Major Air Command Bioenvironmental Engineers
  See latest edition of the "Worldwide Listing of Bioenvironmental Engineering and Environmental Health Personnel."

  Responsible for implementing IRP policy and management decisions and coordinating with state/local regulatory agencies.

# APPENDIX 3

## MATH, CONVERSIONS AND EQUIVALENTS

- **Calculation of Air W/V Conversion Factors**

One liter of air at 25 °C (298.16 °K) contains:

$$\frac{(1 \text{ atm})(1 \text{ liter})}{.0821 \text{ liter atm/mole})(298.16 \text{ °K})} = 0.040874 \text{ moles of gas.}$$

Hence, one liter of air contains:

MW x $10^{-6}$ x 0.040874 grams of a contaminant at 1 ppm.

This is the same as saying 1 $m^3$ of air contains:

MW x 0.040874 mg of a contaminant at 1 ppm.

For example, chloroform has a MW of 119.39. Thus,

1 ppm = 119.39 x 0.040874 ≈ 4.88 $mg/m^3$ at 25°C.

- **Conversion for Solutes in Water**

1 mg/L ≈ 1 ppm (by weight).

- **Conversion of Percent in Food, Water or Air to Parts Per Million**

X% = X parts per 100 parts

$$\frac{X}{100} (10^6) = \text{ ppm.}$$

- **Conversion of Parts Per Million in Food or Water to mg/kg bw/day**

Since both food intake and body weight vary with age (and some times, with treatment), there is no single factor that precisely converts parts per million (ppm) in food or water to mg/kg body weight/day. However, by assuming 100% absorption and adopting a set of standard values for each species for daily food, water and air intake

and average body weight, one can convert a ppm dosage level, within reasonable limits, to mg/kg bw/day for the sake of comparisons.

The following standard body weights and intake values were used to convert dietary or respiratory intakes to estimated daily dose rate:

| Species | Body Weight (kg) | Food Consumption (g/day) | Approximate Water Intake (mL/day) | Minute Volume ($m^3$/min) |
|---|---|---|---|---|
| Human | 70 | 700 | 2000 | $7.4 \times 10^{-3}$ |
| Mouse | 0.025 | 3 | 4.5 | $2.3 \times 10^{-5}$ |
| Rat | 0.3 | 15 | 20 | $1.0 \times 10^{-4}$ |
| Monkey | 5 | 250 | 500 | $8.6 \times 10^{-4}$ |
| Rabbit | 2 | 60 | 330 | $1.1 \times 10^{-3}$ |
| Dog | 10 | 250 | 500 | $5.2 \times 10^{-3}$ |
| Guinea pig | 0.5 | 30 | 85 | $1.6 \times 10^{-4}$ |

For example, at a dietary concentration of 1 ppm of Chemical X, an average adult mouse would consume 3 g of food per day or 0.12 mg of Chemical X/kg bw/day. This value was calculated as follows:

Intake (mg/kg bw/day) = food consumption (g/day) x dietary concentration (ppm) x $1g/10^6$ g diet x 1000 mg/g x 1/bw (kg).

- **Calculation of Respiratory Uptake**

Uptake (mg) = Concentration ($mg/m^3$) x minute volume ($m^3$/min) x retention factor (assume 1.0 unless value is known) x time (minutes).

- **Temperature Conversions**

The formulas given below were used to convert temperatures from one scale to another.

To convert temperatures given in Celsius to Fahrenheit:

$$°F = 9/5\ (°C) + 32$$

To convert temperatures given in Fahrenheit to Celsius:

$$°C = 5/9\ (\ °F - 32)$$

## APPENDIX 4

## STATE WATER QUALITY AGENCIES AND CONTACTS

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Robert Stites

# INDEX 1

## CUMULATIVE CROSS INDEX OF CHEMICAL, COMMON AND TRIVIAL NAMES

The order of chemical, common and trivial names included in this index is strictly alphabetical; numerical and alphabetical prefixes signifying positions in a chemical name or stereochemistry have been ignored.

Acetone
See Chapter 40.

Acetylene tetrachloride
See 1,1,2,2-Tetrachloroethane, Chapter 11.

Acetylene trichloride
See Trichloroethylene, Chapter 16.

Agrotect
See 2,4-D, Chapter 60.

Aroclor®
See Chapter 52.

Automotive gasoline
See Chapter 65.

BBP
See Butyl benzyl phthalate, Chapter 46.

Benzene
See Chapter 18.

Benzene chloride
See Chlorobenzene, Chapter 24.

1,2-Benzenedicarboxylic acid, bis (2-ethylhexyl) ester
See Di(2-ethylhexyl)phthalate, Chapter 31.

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Toluol
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unsym-Trichlorobenzene
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1,1,1-Trichloroethane
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## MOLECULAR FORMULA INDEX

The arrangement used in this index is based on the general molecular formula:

$C_xH_y$ Element$_z$

where the order of elements is alphabetical. Inorganics precede carbon-containing compounds. Organics lacking hydrogen are listed before any CH's. Compounds without known molecular formulas are listed at the end of the index.

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$CCl_3F$
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$CCl_4$
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$C_2H_4Br_2$
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$C_6H_6O$
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$C_7H_6N_2O_4$
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$C_7H_8$
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$C_8H_5Cl_3O_3$
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$C_8H_6Cl_2O_3$
2,4-D. See Chapter 60.

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2,3,7,8-Tetrachlorodibenzo-p-dioxin. See Chapter 63.

$C_{12}H_{10}N_2O$
N-Nitrosodiphenylamine. See Chapter 35.

$C_{12}H_{14}O_4$
Diethyl phthalate. See Chapter 29.

$C_{12}H_{21}N_2O_3PS$
Diazinon® See Chapter 51.

$C_{14}H_8Cl_4$
DDE. See Chapter 59.

$C_{14}H_9Cl_5$
DDT. See Chapter 57.

$C_{14}H_{10}Cl_4$
DDD. See Chapter 58.

$C_{16}H_{22}O_4$
Di-n-butyl phthalate. See Chapter 30.

$C_{19}H_{20}O_4$
Butyl benzyl phthalate. See Chapter 46.

$C_{21}H_{21}O_4P$
Tri-o-cresyl phosphate. See Chapter 49.

$C_{24}H_{38}O_4$
Di(2-ethylhexyl)phthalate. See Chapter 31.

Molecular Formula Unknown

Aroclor ® congeners
See Chapter 52.

Automotive gasoline
See Chapter 65.

Fuel oils
See Chapter 66.

Hydraulic fluid
See Chapter 68.

JP-4
See Chapter 64.

Mineral base crankcase oil
See Chapter 69.

Stoddard solvent
See Chapter 67.

Synthetic crankcase oil
See Chapter 70.

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| 57-12-5 | 56 | 106-42-3 | 21 |
| 57-74-9 | 48 | 106-46-7 | 27 |
| 58-89-9 | 47 | 106-93-4 | 45 |
| 62-75-9 | 34 | 107-06-2 | 9 |
| 67-64-1 | 40 | 107-21-1 | 43 |
| 67-66-3 | 4 | 108-38-3 | 21 |
| 71-43-2 | 18 | 108-88-3 | 19 |
| 71-55-6 | 10 | 108-90-7 | 24 |
| 72-54-8 | 58 | 108-95-2 | 36 |
| 72-55-9 | 59 | 109-86-4 | 42 |
| 74-90-8 | 56 | 111-44-4 | 33 |
| 74-95-3 | 2 | 117-81-7 | 31 |
| 74-97-5 | 44 | 120-82-1 | 28 |
| 75-00-3 | 7 | 121-75-5 | 50 |
| 75-01-4 | 13 | 124-48-1 | 3 |
| 75-09-2 | 1 | 127-18-4 | 17 |
| 75-34-3 | 8 | 143-33-9 | 56 |
| 75-35-4 | 14 | 151-50-8 | 56 |
| 75-69-4 | 5 | 156-59-2 | 15 |
| 78-00-2 | 54 | 156-60-5 | 15 |
| 78-30-8 | 49 | 302-01-2 | 55 |
| 78-87-5 | 12 | 333-41-5 | 51 |
| 78-93-3 | 41 | 540-59-0 | 15 |
| 79-01-6 | 16 | 541-73-1 | 26 |
| 79-34-5 | 11 | 606-20-2 | 23 |
| 84-66-2 | 29 | 1330-20-7 | 21 |
| 84-74-2 | 30 | 1746-01-6 | 63 |
| 85-68-7 | 46 | 7775-11-3 | 53 |
| 86-30-6 | 35 | 8006-61-9 | 65 |
| 87-65-0 | 38 | 8008-20-6 | 66 |
| 87-86-5 | 39 | 8052-41-3 | 67 |
| 91-20-3 | 32 | 11096-82-5 | 52 |
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| 95-57-8 | 37 | 68553-00-4 | 66 |

No CAS Number Assigned:


| | |
|---|---|
| Hydraulic fluid | See Chapter 68 |
| JP-4 | See Chapter 64 |
| Mineral base crankcase oil | See Chapter 69 |
| Synthetic crankcase oil | See Chapter 70 |


*Numeric designation assigned by the American Chemical Society's Chemical Abstracts Service which uniquely identifies a specific chemical compound.

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| AJ8400000 | 61 |
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| CY1400000 | 18 |
| CZ0175000 | 24 |
| CZ4499000 | 26 |
| CZ4500000 | 25 |
| CZ4550000 | 27 |
| DA0700000 | 20 |
| DC2100000 | 28 |
| EL6475000 | 41 |
| FG4900000 | 6 |
| FS9100000 | 4 |
| GB2955000 | 53 |
| GS7175000 | 56 |
| GV4900000 | 47 |
| HP3500000 | 63 |
| HZ1800000 | 66 |
| IQ0525000 | 34 |
| JJ9800000 | 35 |
| KH7525000 | 7 |
| KH9275000 | 45 |
| KI0175000 | 8 |
| KI0525000 | 9 |
| KI0700000 | 58 |
| KI8575000 | 11 |
| KJ2975000 | 10 |
| KJ3325000 | 57 |
| KL5775000 | 42 |
| KN0875000 | 33 |
| KU9625000 | 13 |
| KV9360000 | 15 |
| KV9400000 | 15 |
| KV9422000 | 15 |
| KV9450000 | 59 |
| KW2975000 | 43 |
| KX3850000 | 17 |
| KX4550000 | 16 |
| LS8950000 | 66 |
| LX3300000 | 65 |
| MU7155000 | 55 |

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|---|---|
| MW6825000 | 56 |
| OA5500000 | 66 |
| PA5250000 | 44 |
| PA6360000 | 3 |
| PA7350000 | 2 |
| PA8050000 | 1 |
| PB6125000 | 5 |
| PB9800000 | 48 |
| QJ0525000 | 32 |
| SJ3325000 | 36 |
| SK2625000 | 37 |
| SK8750000 | 38 |
| SM3000000 | 39 |
| TD0350000 | 49 |
| TF3325000 | 51 |
| TH9990000 | 46 |
| TI0875000 | 30 |
| TI1050000 | 29 |
| TI1925000 | 31 |
| TP4550000 | 54 |
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| TQ1356000 | 52 |
| TQ1360000 | 52 |
| TQ1362000 | 52 |
| TS8760000 | 56 |
| TX9625000 | 12 |
| UF8225000 | 62 |
| VZ7525000 | 56 |
| WJ8925000 | 67 |
| WM8400000 | 50 |
| XS5250000 | 19 |
| XT1925000 | 23 |
| YZ8061000 | 14 |
| ZE2100000 | 21 |
| ZE2190000 | 21 |
| ZE2275000 | 21 |
| ZE2450000 | 21 |
| ZE2625000 | 21 |
| ZE5600000 | 22 |

No NIOSH Number Assigned:


| | |
|---|---|
| Hydraulic fluid | See Chapter 68 |
| JP-4 | See Chapter 64 |
| Mineral base crankcase oil | See Chapter 69 |
| Synthetic crankcase oil | See Chapter 70 |


*A unique nine-position accession number (two letters and seven numerals) assigned alphabetically to each substance in the Registry of Toxic Effects of Chemical Substances published by the National Institute for Occupational Safety and Health (Reference 47).

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