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HEALTH AND ENVIRONMENTAL EFFECTS DOCUMENT FOR 2-CHLOROPROPANE

Prepared for

OFFICE OF SOLID WASTE AND
EMERGENCY RESPONSE

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PREFACE

Health and Environmental Effects Documents (HEEDs) are prepared for the Office of Solid Waste and Emergency Response (OSWER). This document series is intended to support listings under the Resource Conservation and Recovery Act (RCRA) as well as to provide health-related limits and goals for emergency and remedial actions under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency Program Office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched for in this document and the dates searched are included in "Appendix: Literature Searched." Literature search material is current up to 8 months previous to the final draft date listed on the front cover. Final draft document dates (front cover) reflect the date the document is sent to the Program Officer (OSWER).

Several quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic and subchronic exposures for both the inhalation and oral exposures. The subchronic or partial lifetime RfD, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval, for example, one that does not constitute a significant portion of the lifespan. This type of exposure estimate has not been extensively used, or rigorously defined as previous risk assessment efforts have focused primarily on lifetime exposure scenarios. Animal data used for subchronic estimates generally reflect exposure durations of 30-90 days. The general methodology for estimating subchronic RfDs is the same as traditionally employed for chronic estimates, except that subchronic data are utilized when available.

In the case of suspected carcinogens, RfDs are not estimated. A carcinogenic potency factor, or q_1^* (U.S. EPA, 1980), is provided instead. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under the CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity, and acute mammalian toxicity). Chemical-specific RQs reflect the lowest of these six primary criteria. The methodology for chronic toxicity and cancer-based RQs are defined in U.S. EPA, 1983 and 1986a, respectively.

EXECUTIVE SUMMARY

2-Chloropropane is commonly known as isopropyl chloride. It is a colorless, highly flammable liquid at room temperature (Hawley, 1981), which is miscible with ethanol and ethyl ether, but is almost insoluble in water (Windholz, 1983; Perry and Green, 1984). This compound is prepared by refluxing isopropyl alcohol with concentrated hydrochloric acid in the presence of a zinc chloride catalyst (Papa, 1982). The public portion of the U.S. EPA TSCA Production File (U.S. EPA, 1977) reported that there were four manufacturers and one importer of 2-chloropropane during 1977. The lack of available production data on 2-chloropropane suggests that this compound is imported and produced on a specialty chemical basis in the United States. 2-Chloropropane is used as a solvent and as an intermediate in the production of other chemicals (Hawley, 1981) and to some extent as an anesthetic (Torkelson and Rowe, 1981).

In the atmosphere, 2-chloropropane is expected to exist almost entirely in the vapor phase (Eisenreich et al., 1981). Reaction with photochemically generated hydroxyl radicals ($t_{1/2}$ ~8 days) appears to be the primary removal mechanism (U.S. EPA, 1987). Small amounts of this compound may be removed from the atmosphere in wet precipitation; however, most of the 2-chloropropane removed by wet deposition is likely to reenter the atmosphere by volatilization. Reaction with ozone, reaction with atomic oxygen and dry deposition are not expected to be environmentally relevant fate processes (U.S. EPA, 1987; Herron and Huie, 1973). If 2-chloropropane is released to water, volatilization is expected to be the dominant removal mechanism. The volatilization half-life of this compound in a 1 m deep waterway, flowing at 1 m/sec with a wind speed of 3 m/sec was estimated to

be 3 hours (see Section 2.2.5.). Chemical hydrolysis [$t_{1/2}$ ~38-40 days (Koskikallio, 1976; Mabey and Mill, 1978)]; reaction with alkylperoxy radicals [$t_{1/2}$ of ~10^4 years (Hendry et al., 1974)]; bioaccumulation in aquatic organisms; and adsorption to suspended solids and sediments are not predictably significant fate processes. In dry soil, 2-chloropropane is expected to undergo rapid volatilization. Volatilization from wet soils is expected to be significant. Available data on hydrolysis in water suggest that chemical hydrolysis may be significant in the removal of this compound from moist soil. Based on the estimated K_{oc}, residual 2-chloropropane in soil is expected to leach from soil into groundwater.

There is a potential for 2-chloropropane to be released to the environment from production and use facilities. It might also be released to the atmosphere during chlorine disinfection of some wastewaters (Gould et al., 1983). This compound was detected in the finished drinking water in one of five selected cities in the United States (Coleman et al., 1976). It was identified as a volatile flavor component of Idaho Russet Burbank baked potatoes (Coleman et al, 1981).

The only available information concerning toxicity of 2-chloropropane to aquatic organisms was provided by Shell Oil Co. (1982), who reported that 140-280 mg/ℓ was the range of concentrations that caused 0-100% mortalilty of five goldfish, Carassius auratus.

In a Dow Chemical Company study (Torkelson and Rowe, 1981; Betso, 1987), mice, rats, guinea pigs, rabbits and monkeys exposed by inhalation to 2-chloropropane at 1000 ppm, 7 hours/day, 5 days/week developed liver and kidney necrosis. Evidence of lung edema or pneumonitis was also observed in female rabbits and monkeys. In rats, rabbits, guinea pigs and dogs exposed to 2-chloropropane at 500 ppm, 7 hours/day, 5 days/week for 6 months, no adverse effects were noted (Torkelson and Rowe, 1981). Extensive

vacuolation and necrosis were observed in the livers of rats exposed to 2-chloropropane at 1000 ppm, 6 hours/day, 5 days/week for 4 weeks (Gage, 1970). No toxic signs or lesions were observed in rats exposed to 250 ppm.

Guinea pigs survived oral doses of 3 g/kg 2-chloropropane but not doses of 10 g/kg (Torkelson and Rowe, 1981). Effects observed when 2-chloropropane was tested for use as an anesthetic in dogs include changes in blood pressure and respiratory rate, damage to the heart muscle and decreased coronary blood flow (Enders and Koner, 1952). Effects on the electrocardiogram and ventricular extrasystoles and unspecified cardiac irregularities were observed in humans (Elam and Newhouse, 1951; Buhr, 1953).

Tham et al. (1984) found that infusion of 2-chloropropane into rats at a rate of 160 μmol/kg/minute resulted in a decreased vestibulo-oculomotor reflex. The threshold for the effect was a blood 2-chloropropane level of 1.9 mmol/ℓ.

2-Chloropropane was mutagenic in S. typhimurium strain TA100, both with and without S-9 metabolic activation when tested in a desiccator (Simmon et al., 1977).

Pertinent data regarding the carcinogenicity, teratogenicity and other reproductive effects of 2-chloropropane could not be located in the available literature as cited in Appendix A. Because of the lack of carcinogenicity human or animal data, 2-chloropropane was assigned to EPA Group D, not classifiable as to human carcinogenicity.

Based on a NOAEL of 250 ppm (803 mg/m^3) in the 4-week rat study (Gage, 1970), a subchronic inhalation RfD of 1 mg/m^3 or 29 mg/day and a chronic inhalation RfD of 0.1 mg/m^3 or 3 mg/day were derived. Data were insufficient for derivation of RfDs for oral exposure. An RQ of 1000 was calculated for systemic toxicity based on histopathologic lesions in rats at 1000 ppm (3212 mg/m^3) in the 4-week inhalation rat study (Gage, 1970).

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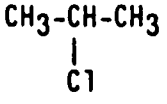
LIST OF ABBREVIATIONS

BCF	Bioconcentration factor
CAS	Chemical Abstract Service
CS	Composite score
K_{oc}	Soil sorption coefficient standardized with respect to organic carbon
K_{ow}	Octanol/water partition coefficient
K_w	Water-to-air ratio
MED	Minimum effective dose
NOAEL	No-observed-adverse-effect level
ppm	Parts per million
RfD	Reference dose
RQ	Reportable quantity
RV_d	Dose-rating value
RV_e	Effect-rating value

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

2-Chloropropane is also known as isopropyl chloride and 2-propyl chloride. The structure, molecular weight, empirical formula and CAS Registry number of this compound are as follows:



Molecular weight: 78.55

Empirical formula: C_3H_7Cl

CAS Registry number: 75-29-6

1.2. PHYSICAL AND CHEMICAL PROPERTIES

2-Chloropropane is a colorless, highly flammable liquid at room temperature (Hawley, 1981). It is expected to undergo reactions typical of alkyl halides. 2-Chloropropane is miscible with ethanol and ethyl ether (Windholz, 1983). Selected physical properties are listed below:

Melting point:	-118°C	Aldrich, 1984
Boiling point:	34-36°C	Aldrich, 1984
Vapor pressure at 20°C:	281 mm Hg	Boublik et al., 1984
Water solubility at 20°C:	3100 mg/ℓ	Perry and Green, 1984
Log K_{ow}:	1.90	Hansch and Leo, 1985
Specific gravity, 25/25°C:	0.858	Hawley, 1981
Refractive index, n_D^{20}:	1.3780	Aldrich, 1984
Flashpoint, closed cup:	-32°C	Windholz, 1983
Air conversion factor at 25°C:	1 ppm = 3.21 mg/m³	

1.3. PRODUCTION DATA

2-Chloropropane can be prepared by refluxing isopropyl alcohol with concentrated hydrochloric acid in the presence of a zinc chloride catalyst

(Papa, 1982). Production and import data on 2-chloropropane are presented in Table 1-1.

CMR (1986) lists Chemical Dynamics Corp., D&O Chemical, Filo Chemical and Jonas Chemical Corp. as suppliers of 2-chloropropane in the United States. Additional data regarding chemical production and sales could not be located in the available literature as cited in Appendix A. The lack of information concerning current domestic production of 2-chloropropane suggests that this compound is imported and produced on a specialty chemical basis in the United States.

1.4. USE DATA

2-Chloropropane is used as a solvent and intermediate for the production of other chemicals (Hawley, 1981).

1.5. SUMMARY

2-Chloropropane is commonly known as isopropyl chloride. It is a colorless, highly flammable liquid at room temperature (Hawley, 1981), which is miscible with ethanol and ethyl ether, but is almost insoluble in water (Windholz, 1983; Perry and Green, 1984). This compound is prepared by refluxing isopropyl alcohol with concentrated hydrochloric acid in the presence of a zinc chloride catalyst (Papa, 1982). The public portion of the U.S. EPA TSCA Production File (U.S. EPA, 1977) reported that there were four manufacturers and one importer of 2-chloropropane during 1977. The lack of available production data on 2-chloropropane suggests that this compound is imported and produced on a specialty chemical basis in the United States. 2-Chloropropane is used as a solvent and as an intermediate in the production of other chemicals (Hawley, 1981) and to some extent as an anesthetic (Torkelson and Rowe, 1981).

TABLE 1-1

Production and Import Volume Data for 2-Chloropropane in 1977[a]

Company	Location	Production and Import Volume (millions of pounds)
Eastman Kodak	Rochester, NY	<0.001
Dow Chemical	Freeport, TX	1-10
Filo Chemical	New York, NY[b]	small volume
Columbia Organics	Columbia, SC	0.001-0.010
Arapahoe Chemicals	Boulder, CO	0.001-0.010 (site-limited use)
Hooker Chemicals	Niagara Falls, NY	none[c]

[a]Source: U.S. EPA, 1977

[b]Importer

[c]This company has imported/produced 2-chloropropane in the past.

2. ENVIRONMENTAL FATE AND TRANSPORT

Limited data pertaining to the environmental fate and transport of 2-chloropropane were located in the available literature as cited in Appendix A. Information concerning fate and transport of this compound was derived from physical property data or the molecular structure of the compound.

2.1. AIR

Eisenreich et al. (1981) reported that organics with vapor pressures $\geq 10^{-4}$ mm Hg should exist almost entirely in the vapor phase in the atmosphere. Therefore, 2-chloropropane, with a vapor pressure of 281 mm Hg at 20°C (Boublik et al., 1984), is expected to exist primarily in the vapor phase in the atmosphere.

2.1.1. Reaction with Hydroxyl Radicals. The estimated half-life for 2-chloropropane vapor reacting with photochemically generated hydroxyl radicals is ~8 days. This estimation was made using an estimated reaction rate constant of 1.3×10^{-12} cm³/molecule-sec at 25°C and assuming an ambient hydroxyl radical concentration of 8.0×10^{5} molecules/cm³ in a typical atmosphere (U.S. EPA, 1987).

2.1.2. Reaction with Ozone. 2-Chloropropane is not susceptible to oxidation by ozone in the atmosphere (U.S. EPA, 1987).

2.1.3. Reaction with Oxygen Atoms. The reaction of 2-chloropropane with atomic oxygen (O^3P) in the atmosphere is not expected to be environmentally significant (Herron and Huie, 1973), and the half-life for this reaction is estimated to be >80 years. This estimation was made using a reaction rate constant of 10^{-15} cm³/molecule-sec at 25°C (Herron and Huie, 1973) and an ambient (O^3P) concentration of 2.5×10^{4} molecules/cm³ (Graedel, 1978).

2.1.4. Physical Removal Processes. Since the water solubility of 2-chloropropane is 3100 mg/ℓ at 20°C (Perry and Green, 1984), it seems likely that small amounts of the compound will be removed from the atmosphere in precipitation. Nevertheless, most of the 2-chloropropane removed from the atmosphere by wet deposition is likely to reenter the atmosphere by volatilization (Section 2.2.5.). Dry deposition is not expected to be a significant removal process for 2-chloropropane.

2.2. WATER

2.2.1. Hydrolysis. Based on measured first-order reaction rate constants of 2.30×10^{-7} and 2.12×10^{-7} sec^{-1}, the half-life for the hydrolysis of 2-chloropropane at neutral pH and 25°C is calculated to be 38 and 40 days, respectively (Koskikallio, 1967; Mabey and Mill, 1978).

2.2.2. Oxidation. Reaction of 2-chloropropane with alkylperoxy radicals in water is not expected to be environmentally relevant (Hendry et al., 1974). The half-life for the reaction of 2-chloropropane with tert-butylperoxy radicals is estimated to be ~10^4 years. This estimation was made using a reaction rate constant of 0.6×10^{-3} ℓ/mol-sec at 30°C (Hendry et al., 1974) and an ambient alkylperoxy radical concentration of 1×10^{-9} mol/ℓ (Mill et al., 1980).

2.2.3. Bioconcentration. Pertinent data regarding the bioconcentration of 2-chloropropane could not be located in the available literature as cited in Appendix A. Therefore, the BCFs of 7 or 16 were estimated using a log K_{ow} of 1.90, a water solubility of 3100 mg/ℓ at 20°C and the following recommended linear regression equations (Lyman et al., 1982):

$$\log BCF = 0.76 \log K_{ow} - 0.23 \qquad (2\text{-}1)$$

$$\log BCF = 2.791 - 0.564 \log S \qquad (2\text{-}2)$$

These values suggest that bioaccumulation of 2-chloropropane in aquatic organisms is not a significant fate process.

2.2.4. Adsorption. Based on K_{oc} values of 52-56 (Section 2.3.2.), physical adsorption of 2-chloropropane to sediments and suspended solids in water is not expected to be significant.

2.2.5. Volatilization. The Henry's Law constant for 2-chloropropane was estimated to be 1.6×10^{-2} atm-m³/mol at 25°C using a method of bond contributions to intrinsic hydrophilic character of the compound (Hine and Mookerjee, 1975). Based on this value, the volatilization half-life of this compound from water 1 m deep, flowing 1 m/sec with a wind speed of 3 m/sec was estimated to be ~3 hours, using the method of Lyman et al. (1982).

2.2.6. Biodegradation. Pertinent data regarding the biodegradation of 2-chloropropane in water could not be located in the available literature as cited in Appendix A.

2.3. SOIL

2.3.1. Hydrolysis. Available data on the hydrolysis of 2-chloropropane in water suggest that chemical hydrolysis may be significant in the removal of this compound from moist soil, particularly if the reaction is catalyzed by soil.

2.3.2. Leaching. A K_{oc} of 56 was estimated for 2-chloropropane, using the molecular topology and quantitative structure-activity analysis method of Sabljic (1984). A K_{oc} of 52 was estimated, using a water solubility of 3100 mg/ℓ at 20°C (Perry and Green, 1984) and the linear regression equation, $\log K_{oc} = -0.557 \log S + 4.277$ (Lyman et al., 1982), where S is in μmol/ℓ. These values suggest that 2-chloropropane is likely to be highly mobile in soil (Swann et al., 1983).

2.3.3. Volatilization. The relatively high vapor pressure of 2-chloropropane [281 mm Hg at 20°C (Boublik et al., 1984)] suggests that this compound will volatilize rapidly from dry soil surfaces. Evaporation from moist soils might also be significant, since this compound does not have a

tendency to adsorb significantly to soil and is expected to volatilize rapidly from water (see Sections 2.2.5. and 2.3.2.).

2.3.4. Biodegradation. Pertinent data regarding the biodegradation of 2-chloropropane in soil could not be located in the available literature as cited in Appendix A.

2.4. SUMMARY

In the atmosphere, 2-chloropropane is expected to exist almost entirely in the vapor phase (Eisenreich et al., 1981). Reaction with photochemically generated hydroxyl radicals ($t_{1/2}$ ~8 days) appears to be the primary removal mechanism (U.S. EPA, 1987). Small amounts of this compound may be removed from the atmosphere in wet precipitation; however, most of the 2-chloropropane removed by wet deposition is likely to reenter the atmosphere by volatilization. Reaction with ozone, reaction with atomic oxygen and dry deposition are not expected to be environmentally relevant fate processes (U.S. EPA, 1987; Herron and Huie, 1973). If 2-chloropropane is released to water, volatilization is expected to be the dominant removal mechanism. The volatilization half-life of this compound in a 1 m deep waterway, flowing at 1 m/sec with a wind speed of 3 m/sec was estimated to be 3 hours (see Section 2.2.5.). Chemical hydrolysis [$t_{1/2}$ ~38-40 days (Koskikallio, 1976; Mabey and Mill, 1978)]; reaction with alkylperoxy radicals [$t_{1/2}$ of ~10^4 years (Hendry et al., 1974)]; bioaccumulation in aquatic organisms and adsorption to suspended solids and sediments are not predictably significant fate processes. In dry soil, 2-chloropropane is expected to undergo rapid volatilization. Volatilization from wet soils is expected to be significant. Available data on hydrolysis in water suggest that chemical hydrolysis may be significant in the removal of this compound from moist soil. Based on the estimated K_{oc}, residual 2-chloropropane in soil is expected to leach from soil into groundwater.

3. EXPOSURE

Monitoring data pertaining to human exposure to 2-chloropropane by inhalation or dermal contact could not be located in the available literature as cited in Appendix A. Potentially, this compound might be released to the environment from production or use facilities. It might also be released to the atmosphere during chlorine disinfection of some wastewaters (Gould et al., 1983).

3.1. WATER

2-Chloropropane was qualitatively detected in the finished drinking water of at least one of five selected cities in the United States (Coleman et al., 1976).

3.2. FOOD

2-Chloropropane was identified as a volatile flavor component of Idaho Russet Burbank baked potatoes (Coleman et al., 1981).

3.3. SUMMARY

There is a potential for 2-chloropropane to be released to the environment from production and use facilities. It might also be released to the atmosphere during chlorine disinfection of some wastewaters (Gould et al., 1983). This compound was detected in the finished drinking water in one of five selected cities in the United States (Coleman et al., 1976). It was identified as a volatile flavor component of Idaho Russet Burbank baked potatoes (Coleman et al., 1981).

4. AQUATIC TOXICITY

4.1. ACUTE TOXICITY

The only available information concerning toxicity of 2-chloropropane to aquatic organisms was provided by Shell Oil Co. (1982), reporting that 140-280 mg/ℓ was the range of concentrations that caused 0-100% mortality of five goldfish, Carassius auratus.

4.2. CHRONIC EFFECTS

Pertinent data regarding chronic toxicity of 2-chloropropane to aquatic organisms could not be located in the available literature as cited in Appendix A.

4.3. PLANT EFFECTS

Pertinent data regarding effects of 2-chloropropane on aquatic plants could not be located in the available literature as cited in Appendix A.

4.4. SUMMARY

The only available information concerning toxicity of 2-chloropropane to aquatic organisms was provided by Shell Oil Co. (1982), reporting that 140-280 mg/ℓ was the range of concentrations that caused 0-100% mortality of five goldfish, Carassius auratus.

5. PHARMACOKINETICS

According to Van Dyke and Wineman (1971), ^{36}Cl-2-chloropropane is enzymatically dechlorinated by rat liver microsomes *in vitro*. The incubation was done in the presence of a NADPH-generating system and ^{36}Cl ion release was measured. Other pertinent data regarding the pharmacokinetics of 2-chloropropane could not be located in the available literature as cited in Appendix A.

6. EFFECTS

6.1. SYSTEMIC TOXICITY

6.1.1. Inhalation Exposures.

6.1.1.1. SUBCHRONIC -- Torkelson and Rowe (1981) reported a Dow Chemical Company study in which several species were exposed to 2-chloropropane at 1000 ppm (3212 mg/m^3), 7 hours/day, 5 days/week for 127 exposures over 181 days. Further information concerning this study, conducted in the 1950s, was obtained from Betso (1987). Groups of 10 female mice, 20 male and 20 female rats, 8 male and 8 female guinea pigs, 2 male and 2 female rabbits and 2 female monkeys were tested. Similar groups of animals, exposed to air, were maintained as controls. No adverse effects on behavior, appearance, growth, mortality, final average body weight, organ weights and hematological values occurred in any of the species examined. Histological examinations revealed necrosis of the parenchymal cells in the portal areas of the liver and degeneration of the tubular epithelium with some necrosis in the kidneys. These effects were noted in all groups except male rats. In addition, evidence of lung edema or pneumonitis was observed in female rabbits and monkeys.

Torkelson and Rowe (1981) also reported that Dow Chemical Company exposed rats, rabbits, guinea pigs and dogs to 2-chloropropane at 500 ppm, 7 hours/day, 5 days/week for 6 months. At this exposure regimen, no adverse effects were noted in appearance, growth, final organ and body weights, hematological and clinical studies or gross and histological examinations. Dow Chemical Company no longer has a record of this experiment (Betso, 1987).

In an inhalation study, Gage (1970) exposed groups of four male and four female Alderly Park SPF rats to 2-chloropropane at 250 or 1000 ppm (803 or 3212 mg/m^3) for 20 6-hour exposures (5 days/week). No toxic signs were

noted during the exposure. At necropsy, the livers of the 1000 ppm exposed rats showed extensive vacuolation and necrosis. The organs of the 250 ppm exposed rats appeared normal. Further details of this study were not available.

6.1.1.2. CHRONIC -- Pertinent data regarding the chronic inhalation toxicity of 2-chloropropane could not be located in the available literature as cited in Appendix A.

6.1.2. Oral Exposures. Pertinent data regarding the subchronic and chronic oral toxicity of 2-chloropropane could not be located in the available literature as cited in Appendix A.

6.1.3. Other Relevant Information. Torkelson and Rowe (1981) reviewed unpublished data by Dow Chemical Company that indicate that guinea pigs survived oral doses of 2-chloropropane at 3 g/kg, but died after doses of 10 g/kg.

2-Chloropropane has been tested for use as an anesthetic. In a study using dogs (Enders and Köner, 1952), 2-chloropropane at 0.8 mℓ/ℓ (694 mg/ℓ) was inhaled for 3-5 minutes. The effects noted were a decrease in arterial blood pressure, an increase in venous blood pressure, an increase in respiratory rate and a decrease in respiratory volume. In addition, electrocardiograms showed significant damage to the heart muscle, and coronary blood flow was decreased by 50%. In a human study (Elam and Newhouse, 1951), patients anesthetized with nitrous oxide and then with 2-chloropropane showed effects on the electrocardiogram and ventricular extrasystoles. In another study (Buhr, 1953), 12 patients were anesthetized with 2-chloropropane for ~12 minutes. Circulation was well maintained for up to 8 minutes, but it was depressed thereafter. Unspecified cardiac irregularities were observed in two of the patients.

Tham et al. (1984) studied the vestibulo-oculomotor reflex in female Sprague-Dawley rats dosed with 2-chloropropane by continuous intravenous infusion for 60 minutes. Throughout the dosing period, the vestibulo-oculomotor reflex and blood 2-chloropropane concentration were measured. Infusion of 2-chloropropane at 160 μmol/kg/minute resulted in a depression of the vestibulo-oculomotor reflex, with the threshold limit for the effect at a blood level of 1.9 mmol/ℓ.

6.2. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of 2-chloropropane could not be located in the available literature as cited in Appendix A. 2-Chloropropane has not been scheduled to be tested by the NTP (1987).

6.3. MUTAGENICITY

Simmon et al. (1977) reported that 2-chloropropane was mutagenic in Salmonella typhimurium strain TA100, both with and without S-9 metabolic activation when the bacteria were exposed to 2-chloropropane vapor for 7-10 hours in a desiccator.

6.4. TERATOGENICITY

Pertinent data regarding the teratogenicity of 2-chloropropane could not be located in the available literature as cited in Appendix A.

6.5. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of 2-chloropropane could not be located in the available literature as cited in Appendix A.

6.6. SUMMARY

In a Dow Chemical Company study (Torkelson and Rowe, 1981; Betso, 1987), mice, rats, guinea pigs, rabbits and monkeys exposed to 2-chloropropane at 1000 ppm, 7 hours/day, 5 days/week for 127 exposures over 181 days developed liver and kidney necrosis. Evidence of lung edema or pneumonitis was also

observed in female rabbits and monkeys. In rats, rabbits, guinea pigs and dogs exposed to 2-chloropropane at 500 ppm, 7 hours/day, 5 days/week for 6 months, no adverse effects were noted (Torkelson and Rowe, 1981). Extensive vacuolation and necrosis were observed in the livers of rats exposed to 2-chloropropane at 1000 ppm, 6 hours/day, 5 days/week for 4 weeks (Gage, 1970). No toxic signs or lesions were observed in rats exposed to 250 ppm.

Guinea pigs survived oral doses of 3 g/kg 2-chloropropane but not doses of 10 g/kg (Torkelson and Rowe, 1981). Effects observed when 2-chloropropane was tested for use as an anesthetic in dogs include changes in blood pressure and respiratory rate, damage to the heart muscle and decreased coronary blood flow (Enders and Kōner, 1952). Effects on the electrocardiogram, ventricular extrasystoles and unspecified cardiac irregularities were observed in humans (Elam and Newhouse, 1951; Buhr, 1953).

Tham et al. (1984) found that infusion of 2-chloropropane into rats at a rate of 160 μmol/kg/minute resulted in a decreased vestibulo-oculomotor reflex. The threshold for the effect was a blood 2-chloropropane level of 1.9 mmol/ℓ.

2-Chloropropane was mutagenic in S. typhimurium strain TA100, both with and without S-9 metabolic activation when tested in a desiccator (Simmon et al., 1977).

Pertinent data regarding the carcinogenicity, teratogenicity and other reproductive effects of 2-chloropropane could not be located in the available literature as cited in Appendix A.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Pertinent guidelines and standards, including EPA ambient water and air quality criteria, drinking water standards, FAO/WHO ADIs, EPA or FDA tolerances for raw agricultural commodities or foods, and ACGIH, NIOSH or OSHA occupational exposure limits could not be located in the available literature as cited in Appendix A.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the effects of 2-chloropropane could not be located in the available literature as cited in Appendix A.

8. RISK ASSESSMENT

8.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of 2-chloropropane could not be located in the available literature as cited in Appendix A.

8.1.1. Weight of Evidence. The carcinogenicity of 2-chloropropane has not been examined in humans or laboratory animals. Therefore, the compound can be placed in EPA Group D (U.S. EPA, 1986b), not classifiable as to human carcinogenicity.

8.1.2. Quantitative Risk Estimates. The lack of data regarding the carcinogenicity of 2-chloropropane precludes the derivation of risk assessment values based on carcinogenicity.

8.2. SYSTEMIC TOXICITY

8.2.1. Inhalation Exposure.

8.2.1.1. LESS THAN LIFETIME EXPOSURES (SUBCHRONIC) -- In a Dow Chemical Company study reported by Torkelson and Rowe (1981) and Betso (1987), necrosis in the liver and kidneys was observed in mice, rats, guinea pigs, rabbits and monkeys exposed to 2-chloropropane at 1000 ppm (3212 mg/m^3), 7 hours/day, 5 days/week for 127 exposures over 181 days. Evidence of lung edema or pneumonitis was also noted in rabbits and monkeys. No effects were noted in rats, rabbits, guinea pigs and dogs exposed to 2-chloropropane at 500 ppm (1606 mg/m^3), 7 hours/day, 5 days/week for 6 months (Torkelson and Rowe, 1981). Dow Chemical Company no longer has a record of the low dose experiment (Betso, 1987).

Extensive vacuolation and necrosis of the liver was observed in rats exposed to 2-chloropropane at 1000 ppm for 6 hours/day, 5 days/week for 4 weeks (Gage, 1970). No effects were observed in rats exposed to 250 ppm (803 mg/m^3).

Dow Chemical Company (Torkelson and Rowe, 1981) and Gage (1970) both observed histopathological lesions in laboratory animals exposed to 1000 ppm 2-chloropropane. In the 6-month study, Dow Chemical Company (Torkelson and Rowe, 1981) identified a NOAEL at 500 ppm, while in a 4-week study, Gage (1970) identified a NOAEL at 250 ppm. If additional data supporting the NOAEL identified at 500 ppm were available, the Dow Chemical Company study (Torkelson and Rowe, 1981) would be suitable for risk assessment. Because there are no data to support a NOAEL of 500 ppm, the more conservative NOAEL of 250 ppm identified in the 4-week study will be used for risk assessment. Expanding the 250 ppm (803 mg/m^3) dose level to continuous exposure by multiplying by 6 hours/24 hours and 5 days/7 days, and by dividing by an uncertainty factor of 100 (10 for interspecies extrapolation and 10 to protect sensitive individuals) a human subchronic inhalation RfD concentration of 1 mg/m^3 is derived. Multiplying the exposure concentration by the human breathing rate, 20 m^3/day, a subchronic inhalation RfD of 29 mg/day for a 70 kg human is derived.

Confidence in the subchronic RfD is low. The Gage (1970) study uses only eight rats per dose group, and the rats were exposed for only 4 weeks. The limited information that showed no effects in the 6-month Dow Chemical Company study at 500 ppm (Torkelson and Rowe, 1981) indicates that longer exposures at 250 ppm are likely to be safe. Confidence in the subchronic RfD is also low because 2-chloropropane has not been tested for carcinogenicity, teratogenicity or other reproductive effects.

8.2.1.2. **CHRONIC EXPOSURES** -- The toxicity of 2-chloropropane following chronic inhalation exposure has not been examined. A chronic inhalation

RfD of 0.1 mg/m³ or 3 mg/day for a 70 kg human is derived by dividing the subchronic RfD by an additional uncertainty factor of 10 to extrapolate from subchronic to chronic exposure.

Confidence in this RfD is low; the key study (Gage, 1970) was only 4 weeks long and only eight rats/dose group were examined. In addition, 2-chloropropane has not been examined in studies of carcinogenicity, teratogenicity or studies of other reproductive effects.

8.2.2. Oral Exposure. Data were not available from which to derive subchronic or chronic RfDs for oral exposure. NOAELs from available inhalation studies include 500 ppm in the 6-month Dow Chemical Company study (Torkelson and Rowe, 1981) and 250 ppm in the 4-week study by Gage (1970). Data from the Dow Chemical Company study (Torkelson and Rowe, 1981; Betso, 1987) were not available for evaluation, and the study is judged inadequate for use in quantitative risk assessment. The Gage (1970) study is too short for use in deriving an RfD for oral exposure, especially where the additional uncertainties of route-to-route extrapolation are involved. Thus, no subchronic or chronic RfD for oral exposure to 2-chloropropane can be derived.

9. REPORTABLE QUANTITIES

9.1. BASED ON SYSTEMIC TOXICITY

The toxicity of 2-chloropropane is discussed in Chapter 6. The toxicity data suitable for deriving an RQ are presented in Table 9-1. The Dow Chemical Company study (Torkelson and Rowe, 1981; Betso, 1987) that used monkeys was omitted from the table because the species of monkey used in the study was not provided, and therefore, a reasonable estimate of body weight cannot be made.

The derivation of RQ values is presented in Table 9-2. In the studies available for RQ derivation (Torkelson and Rowe, 1981; Betso, 1987; Gage, 1970), necrosis of the liver and the kidney was the only effect observed. This effect, corresponding to an RV_e of 6, was observed in rats exposed to 2-chloropropane at 1000 ppm, 6 hours/day, 5 days/week for 4 weeks (Gage, 1970) and in all species exposed at 1000 ppm, 7 hours/day, 5 days/week for 6 months (Torkelson and Rowe, 1981; Betso, 1987). The lowest MED values at which the effects were observed are from the Gage (1970) rat study, 438.9 mg/day, and the Dow Chemical Company mice study, 459.2 mg/day (Torkelson and Rowe, 1981; Betso, 1987). Both MEDs correspond to an RV_d of 1.5. Multiplying the RV_e by the RV_d, a CS of 9 is derived from both studies. This CS corresponds to an RQ of 1000. Because the duration of exposure was longer in the Dow Chemical Company study (Torkelson and Rowe, 1981; Betso, 1987) than in the study by Gage (1970), it was selected as the basis for the RQ (Table 9-3).

TABLE 9-1

Inhalation Toxicity Summary for 2-Chloropropane (Purity not reported)

Species/ Strain	Sex	No. at Start	Average Weight (kg)	Vehicle/ Physical State	Exposure	Transformed Animal Dose (mg/kg/day)	Equivalent Human Dose[a] (mg/kg/day)	Response	Reference
Mice/NR	F	10	0.03[b]	air	1000 ppm (3212 mg/m³), 7 hours/day, 5 days/week for 181 days	869.9[c]	65.6	Necrosis in the parenchymal cells in the portal areas of the liver; tubular degeneration of the epithelium with some necrosis in kidneys	Betso, 1987; Torkelson and Rowe, 1981
Rats/NR	F	20	0.35[b]	air	same as above	426.4[c]	72.9	Same as above	Betso, 1987; Torkelson and Rowe, 1981
Guinea pigs/NR	M,F	8/sex	0.84[b]	air	same as above	318.7[c]	73.0	Same as above	Betso, 1987; Torkelson and Rowe, 1981
Rabbits/ NR	M,F	2/sex	3.8[b]	air	same as above	352.2[c]	133.4	Necrosis in the parenchymal cells in the portal areas of the liver; tubular degeneration of the epithelium with some necrosis in kidneys; female rabbits showed evidence of lung edema or pneumonitis	Betso, 1987; Torkelson and Rowe, 1981
Rats/ Alderly-Park SPF	M,F	4/sex	0.2[d]	air	1000 ppm (3212 mg/m³), 6 hours/day, 5 days/week for 4 weeks	441.7[e]	62.7	Extensive vacuolation and necrosis of the liver	Gage, 1970

[a]Calculated by multiplying the animal transformed dose by the cube root of the ratio of the animal body weight to the human body weight (70 kg)

[b]Reference body weights (U.S. EPA, 1985)

[c]Calculated by multiplying the concentration by the number of hours/day exposed, number days/week, by the inhalation rates (0.039 m³/day mice; 0.223 m³/day rats; 0.40 m³/day guinea pigs; 2 m³/day rabbits) from U.S. EPA (1985) and by dividing by the animal body weight

[d]Data provided by investigators

[e]Calculated by multiplying the concentration by 6 hours/24 hours by 5 days/7 days, by the inhalation rate, 0.154 m³/day [calculated from $I = 0.105 (w/0.113)^{2/3}$ from U.S. EPA, 1985)] and dividing by the body weight.

TABLE 9-2

Inhalation Composite Scores for 2-Chloropropane

Species	Animal Dose (mg/kg/day)	Chronic Human MED* (mg/day)	RV_d	Effects	RV_e	CS	RQ	Reference
Rats	441.7	438.9	1.5	Extensive vacuolation and necrosis of the liver	6	9	1000	Gage, 1970
Mice	869.9	459.2	1.5	necrosis in the parenchymal cells in the portal areas of the liver; tubular degeneration of the epithelium with some necrosis in the kidneys	6	9	1000	Betso, 1987; Torkelson and Rowe, 1981
Rats	426.4	510.3	1.4	necrosis in the parenchymal cells in the portal areas of the liver; tubular degeneration of the epithelium with some necrosis in the kidneys	6	8.4	1000	Betso, 1987; Torkelson and Rowe, 1981
Guinea pigs	318.7	511	1.4	necrosis in the parenchymal cells in the portal areas of the liver; tubular degeneration of the epithelium with some necrosis in the kidneys	6	8.4	1000	Betso, 1987; Torkelson and Rowe, 1981
Rabbits	352.2	933.8	1	necrosis in the parenchymal cells in the portal areas of the liver; tubular degeneration of the epithelium with some necrosis in the kidneys; female rabbits showed evidence of lung edema or pneumonitis	6	6	1000	Betso, 1987; Torkelson and Rowe, 1981

*The dose was divided by an uncertainty factor of 10 to approximate chronic exposure.

TABLE 9-3

2-Chloropropane

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	inhalation
Dose*:	459.2 mg/day
Effect:	necrosis in the liver and kidneys
Reference:	Torkelson and Rowe, 1981; Betso, 1987
RV_d:	1.5
RV_e:	6
Composite Score:	9
RQ:	1000

*Equivalent human dose

9.2. BASED ON CARCINOGENICITY

Pertinent data regarding the carcinogenicity of 2-chloropropane in humans or animals could not be located in the available literature as cited in Appendix A. The compound is assigned an EPA classification of D -- cannot be classified as to carcinogenicity to humans, which precludes hazard ranking based on carcinogenicity.

10. REFERENCES

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APPENDIX A

LITERATURE SEARCHED

This HEED is based on data identified by computerized literature searches of the following:

TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in February, 1987. In addition, hand searches were made of Chemical Abstracts (Collective Indices 5-9), and the following secondary sources should be reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 1986-1987. TLVs: Threshold Limit Values for Chemical Substances in the Work Environment adopted by ACGIH with Intended Changes for 1986-1987. Cincinnati, OH. 111 p.

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Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

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APPENDIX B

Summary Table for 2-Chloropropane

	Species	Exposure	Effect	RfD	Reference
Inhalation Exposure					
Subchronic	rat	250 ppm (803 mg/m³), 6 hours/day, 5 days/week, for 4 weeks	NOAEL	1 mg/m³; 29 mg/day	Gage, 1970
Chronic	rat	250 ppm (803 mg/m³), 6 hours/day, 5 days/week, for 4 weeks	NOAEL	0.1 mg/m³; 3 mg/day	Gage, 1970
Carcinogenicity	ID	ID	ID	ID	ID
Oral Exposure					
Subchronic	ID	ID	ID	ID	ID
Chronic	ID	ID	ID	ID	ID
Carcinogenicity	ID	ID	ID	ID	ID
REPORTABLE QUANTITIES					
Based on Chronic Toxicity:		1000			Torkelson and Rowe, 1981; Betso, 1987
Based on Carcinogenicity:		ID			ID

ID = Insufficient data