

LITIGATION TECHNICAL SUPP

ROCKY MOUNTAIN ARSENAL

DRAFT FINAL REPORT
TOXICITY ASSESSMENT FOR
RMA TARGET CONTAMINANTS
VOLUME I

JUNE 1987
TASK ORDER 35

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ENDANGERMENT ASSESSMENT RMA
CONTRACT NO. DAAK11-84-D-0017

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<p>13. ABSTRACT (Maximum 200 words)</p> <p>THIS REPORT IS A DETAILED DISCUSSION OF THE EVALUATIONS PERFORMED TO DEVELOP THE TOXICITY ASSESSMENT FOR RMA CONTAMINANTS IN SOIL. THE OBJECTIVES OF THE TOXICITY ASSESSMENT ARE TO:</p> <ol style="list-style-type: none"> 1. DETERMINE THE NATURE AND EXTENT OF HEALTH AND ENVIRONMENTAL HAZARDS ASSOCIATED WITH EXPOSURE TO CONTAMINANTS PRESENT AT THE SITE 2. IDENTIFY A QUANTITATIVE INDEX OF TOXICITY FOR EACH TARGET CONTAMINANT, REFERRED TO IN THIS ASSESSMENT AS DT. <p>THE TOXICITY ASSESSMENT FOR THE RMA TARGET CONTAMINANTS HAS BEEN PERFORMED CONSISTENT WITH PUBLISHED EPA GUIDELINES AND ADDRESSES ONLY HUMAN HEALTH HAZARDS ASSOCIATED WITH CONTAMINANTS IN SOIL.</p> <p>EACH TOXICITY PROFILE IS COMPOSED OF SEVEN SECTIONS:</p> <ol style="list-style-type: none"> 1. SUMMARY 2. CHEMICAL AND PHYSICAL PROPERTIES 3. TRANSPORT AND FATE <p style="text-align: right;">DTIC QUALITY INSPECTED 2</p>				
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CONTRACT NO. DAAK11-84-D-0017

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Commerce City, Colorado

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ERRATA

<u>Volume Number</u>	<u>Chemical Profile</u>	<u>Ebasco Document Number</u>	<u>Page Number</u>	<u>Errata</u>
I	Appendix A (Arsenic)	5653a	A-1	Comment in Column 6 should read: DT: Superfund Public Health Evaluation Manual (1986).
I	Cadmium	3698a	3-5	The 3 pages which follow the profile for benzene are out of sequence and should be inserted into the cadmium profile.
I	Carbon tetrachloride	3708a	5	Line 1: <u>salmonella</u> should be capitalized.
I	1,1-Dichloro-ethylene	3808a	5	This page is missing and should be inserted. (Attachment 1)
I	p-Chlorophenyl methylsulfoxide	4279a	3	Paragraph 2, line 1: insert PCPMSO after the word "for."
II	Table of Contents	4452a	ii	Methylene chloride and methyl isobutyl ketone are listed twice under Appendix B erroneously.
II	Fluoride	4519a	3	This page should be replaced with the proper page 3 corresponding to fluoride. (Attachment 2)
II	Fluoroacetic acid	5371a	4-5	Incorrect print wheel resulted in transposition of all zeros to ones. A corrected page should be inserted. (Attachment 3)
II	Lead	3810a	8	Format error in DT calculation. Insert corrected page. (Attachment 4)
II	Zinc	3833a	7	The following reference is missing: USEPA 1986. Quality Criteria for Water 1986. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-86-001

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ALDRIN/DIELDRIN
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AZODRIN
BENZENE
BENZOTHIAZOLE
BICYCLOHEPTADIENE
CADMIUM
CARBON TETRACHLORIDE
CHLORDANE
CHLOROACETIC ACID
CHLOROBENZENE

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APPENDIX B: TOXICITY PROFILES (Continued)

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CHLOROPHENYL METHYL SULFONE
CHLOROPHENYL METHYL SULFOXIDE
CHROMIUM
COPPER
DDT/DDD/DDE
DIBROMOCHLOROPROPANE
1,1-DICHLOROETHANE
1,2-DICHLOROETHANE
1,1-DICHLOROETHYLENE
1,2-DICHLOROETHYLENE (CIS-,TRANS-)
DICYCLOPENTADIENE
DIISOPROPYL METHYLPHOSPHONATE
DIMETHYL ARSENIC ACID
DIMETHYL DISULFIDE
DIMETHYL METHYLPHOSPHONATE
DITHIANE
ENDRIN
ETHYLBENZENE

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GLOSSARY OF TERMS AND ACRONYMS

ACGIH	American Council of Governmental Industrial Hygienists
ADI	Acceptable Daily Intake
CAG	Cancer Assessment Group
BCF	Bioconcentration Factor
D_T	Allowable Dose
FDA	Food and Drug Administration
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System; an EPA database
K_H	Henry's Constant
K_{oc}	Soil/Water Partition Coefficient
LD ₅₀	Lethal dose 50; The dose that was lethal to 50 percent of the test organisms
LOAEL	Lowest-Observed-Adverse Effect Level
MCL	Maximum Contaminant Level in Drinking Water
NAS	National Academy of Science
NOAEL	No-Observed-Adverse-Effect-Level
NOEL	No-Observed-Effect Level
OERR	Office of Emergency and Remedial Response (OERR)
OWPE	Office of Waste Programs Enforcement
RfD	Risk Reference Dose Obtained from the EPA IRIS Database
RMA	Rocky Mountain Arsenal
RMCL	Recommended Maximum Contaminant Level developed by EPA for establishing Drinking Water Criteria
SEL	Short Term Effect Level
SF	Severity of Effects Factor
SPHEM	Superfund Public Health Evaluation Manual
TLV	Threshold Limit Value
TWA	Time Weighted Average
UF	Uncertainty Factor
USABRDL	U.S. Army Biomedical Research and Development Laboratory
WHO	World Health Organization

1.0 INTRODUCTION

1.1 SUMMARY OF THE ENDANGERMENT ASSESSMENT PROCESS FOR RMA

The goal of the Endangerment Assessment (EA) performed as part of the Remedial Investigation (RI)/Feasibility Study (FS) process is to determine the magnitude and probability of actual or potential harm to public health, welfare, or environment by the threatened or actual release of hazardous substances (EPA/Endangerment Assessment Handbook 1985).

To attain this goal the following evaluations must be made:

- o Identify the hazardous substances and/or wastes present in all relevant media
- o Characterize the fate and transport of site contaminants
- o Identify the exposure pathways
- o Characterize the populations at risk
- o Establish the toxicological properties of site contaminants, and
- o Characterize the risk posed to exposed populations.

The above evaluations are segregated under four separate components. These are:

- o Contaminant Identification
- o Exposure Assessment
- o Toxicity Assessment
- o Risk Characterization

For the Rocky Mountain Arsenal (RMA) environmental cleanup program, the Endangerment Assessment follows the above guidelines. This report is a detailed discussion of the evaluations performed to develop the toxicity assessment for RMA contaminants in soils.

1.2 TOXICITY ASSESSMENT AS PART OF THE ENDANGERMENT ASSESSMENT PROCESS

The objectives of the toxicity assessment (Endangerment Assessment Handbook, Section 3.3) are to determine the nature and extent of health and environmental hazards associated with exposure to contaminants present at the site and to identify a quantitative index of toxicity for each target contaminant, referred to in this assessment as D_T (see Section 3.0). The end-product of the toxicity assessment is a chemical-specific toxicity profile which addresses these elements. The toxicity assessment for the RMA target contaminants has been performed consistent with published EPA guidelines (Superfund Public Health Evaluation Manual USEPA 1986a; Endangerment Assessment Handbook USEPA 1985a; Superfund Exposure Assessment Manual USEPA 1986b) and addresses only human health hazards associated with contaminants in soils.

As part of the Endangerment Assessment performed for RMA, Preliminary Pollutant Limit Values (PPLVs) are being developed for each target chemical. These quantities represent in-place soil concentrations that will be protective of human health following cleanup. The PPLVs are computed for a number of exposure pathways appropriate to potential uses of the Arsenal property. Each exposure pathway specific PPLV is computed as a function of contaminant concentration in various pathway media (e.g., soils or foods), intake rates and inter-media partition coefficients. The transport of contaminants from soils to humans is modeled to relate the dose of a contaminant received by humans to the in-place soil concentration. A protective soil concentration is then computed by setting the dose received equal to a maximum allowable dose, D_T .

2.0 TOXICITY PROFILE DEVELOPMENT

Toxicity profiles for each RMA target contaminant were generated from current toxicological literature and include considerations of dose, routes of exposure, types of adverse effects manifested, transport and fate and a quantitative evaluation of an allowable dose (D_T). Each profile is composed of seven sections which address the following elements: Summary, Chemical and Physical Properties, Transport and Fate, Health Effects, Toxicity to Wildlife and Domestic Animals, Regulations and Standards, and D_T Value. The quantity D_T is defined as the contaminant intake rate which should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level. The target contaminants and types of information available for each are summarized in Table 1. There are a number of chemicals for which data were not identified for the parameters K_{oc} (soil/water partition coefficient), K_H (Henry's Law Constant), and BCF (bioconcentration factor). These limitations are discussed in Section 2.2.

2.1 INFORMATION SOURCES

The USEPA's Offices of Emergency and Remedial Response (OERR) and Waste Programs Enforcement (OWPE) have prepared toxicity profiles specifically for use in assessing toxicological risk at hazardous waste sites (USEPA 1985b). The chemical profiles prepared by OWPE briefly summarize the chemical and physical properties, fate and transport, health effects and environmental toxicity levels for chemicals found most often at hazardous waste sites. EPA chemical profiles were available for 31 of the RMA target chemicals. The U.S. Army Biomedical Research and Development Laboratory (USABRDL) has also developed data sheets for most of the target chemicals. The OWPE and USABRDL information provided the basis for most of the toxicity profiles developed for these contaminants (refer to Table 1). The OWPE profiles present information developed in a number of agency publications such as Health Effects Assessments and Water Quality Criteria documents.

TABLE 1

SUMMARY OF AVAILABLE INFORMATION FOR TARGET CONTAMINANTS AT RMA

Chemical	EPA/OWPE Toxicity Profiles	USABRDL Chemical Data Sheet	Literature Search With Information Identified	Literature Search With No Information Identified	Other
Aldrin	X	X			
Arsenic	X	X			
Atrazine		X			
Azodrin		X			
Benzene; Benzol	X	X			
Benzothiazole, BTA	X	X			
Bicycloheptadiene; BCH		X		X	
Bis(2-chlorovinyl) sulfone				X	
Bis(2-chlorovinyl) sulfoxide				X	x ^{1/}
Bromide					
Cadmium	X	X			
Carbon tetrachloride	X	X			
Chlordane	X	X			
Chloride					x ^{1/}
Chloroacetic acid			X		
Chlorobenzene	X	X			
Chloroform	X	X			
2-chlorovinyl arsonic acid				X	
2-chlorovinyl arsonous acid				X	
p-chlorophenyl methyl sulfide; CPMS				X	
p-chlorophenyl methyl sulfone; CPMSO				X	
p-chlorophenyl methyl sulfoxide; CPMSO ₂		X			
Chromium	X	X			
Copper	X	X			
DDE	X	X			

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TABLE 1 (Continued)
SUMMARY OF AVAILABLE INFORMATION FOR TARGET CONTAMINANTS AT RMA

Chemical	EPA/OWPE Toxicity Profiles	USABRD Chemical Data Sheet	Literature Search With Information Identified	Literature Search With No Information Identified	Other
DDT	X	X			
1,2-Dibromo-3-chloro-propane; DBCP; Nemagon	X	X			
1,1-Dichloroethane	X	X			
1,2-Dichloroethane	X	X			
1,1-Dichloroethylene	X	X			
1,2-Dichloroethylene	X	X			
Dicyclopentadiene; DCPD		X			
Dieldrin	X	X			
2-(Diisopropylamino)-n-ethyl sulfonate				X	
2-(Diisopropylamino)-n-ethanethiol				X	
Diisopropylmethylphosphonate; DIMP		X			
Dimethyl disulfide; DMDS		X			
Dimethyl methylphosphonate; DMMP		X			
Dimethyl arsenic acid			X		
Dimethyl mercury			X		
Dithiane		X			
Endrin	X	X			
Ethyl benzene	X	X			
Ethyl methyl phosphonate (EMP)					X
Ethyl methyl phosphonic acid (EMPA)					X
Fluoride		X			

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TABLE 1 (Continued)
SUMMARY OF AVAILABLE INFORMATION FOR TARGET CONTAMINANTS AT RMA

Chemical	EPA/OMPE Toxicity Profiles	USABRDL Chemical Data Sheet	Literature Search With Information Identified	Literature Search With No Information Identified	Other
Fluoroacetic acid			X		
GB; Sarin		X			
Hexachlorocyclopentadiene; HCCPD		X			
Isodrin		X			
Isopropyl methyl phosphonic acid				X	X ^{2/}
Isopropyl methyl phosphonate (IMP)				X	X ^{2/}
Lead	X				
Lewisite		X			
Lewisite oxide		X			
Malathion		X			
Mercury	X	X			
Methylene chloride	X	X			
Methyl arsonic acid			X		
Methyl isobutyl ketone					
Methyl mercury salts	X	X			
Methylphosphonic acid				X	
Mustard		X			
N-Nitrosodimethylamine		X			
Parathion		X			
Supona		X			
Tetrachloroethylene	X	X			
Thiodiglycol		X			
2,2'-Thiodiglycolic acid					X
Thioxane; 1,4-Oxathiane		X			

TABLE 1 (Continued)
SUMMARY OF AVAILABLE INFORMATION FOR TARGET CONTAMINANTS AT RMA

Chemical	EPA/OWPE Toxicity Profiles	USABRDL Chemical Data Sheet	Literature Search With Information Identified	Literature Search With No Information Identified	Other
Toluene	X	X			
1,1,1-Trichloroethane	X	X			
1,1,2-Trichloroethane	X	X			
Trichloroethylene; TCE	X	X			
Trimethyl phosphate			X		
Vapona		X			
o,m,p-Xylene	X	X			
Zinc	X	X			

1/ These chemicals were not evaluated in the Endangerment Assessment as they would not likely occur in the ionic form in environmental media present at RMA.

2/ Limited information available through USABRDL.

The original source of data in the OWPE profile is not always identified; therefore it was not possible to reference information obtained from these profiles to the original source.

As indicated in Table 1, OWPE and/or USABRDL profiles were not available for all of the 78 target contaminants; therefore information was sought from computerized literature searches of the Dialog and Chemical Information Systems for these chemicals. A search was also performed for a few of the chemicals for which available information appeared limited. Each of these systems offers a number of databases which contain references to published literature articles. Some of the databases in the Chemical Information System contain actual chemical specific data. The databases group information by specific topic areas. For example, the Medline database available through the Dialog Information System (DIS), is one of the major sources for biomedical literature. The databases Phytotox and Envirofate which are available on the Chemical Information System (CIS) contain information on the effects of chemicals on plants and chemical specific data on environmental fate, respectively. All CIS databases and 13 relevant DIS databases were searched using chemical names or Chemical Abstract Service (CAS) numbers as keywords.

As noted in Table 1, potentially useful information was identified for only seven of the chemicals for which a search was performed. As of the date of this report, information which would permit preparation of toxicity profiles for the following chemicals was not available:

- Biscarboxymethyl sulfone
- Biscarboxymethyl sulfoxide
- 2-Chlorovinyl arsonic acid
- 2-Chlorovinyl arsonous acid
- 2-(Diisopropylamino)-n-ethyl sulfonate
- 2-(Diisopropylamino)-n-ethanethiol
- Ethyl methyl phosphonate
- Ethyl methylphosphonic acid
- Methyl phosphonic acid
- 2,2-Thiodiglycolic acid

The search for additional information for these compounds is continuing. Profiles have not been developed for bromide and chloride since these chemicals are not expected to occur in the ionic form in soils at RMA.

2.2 PHYSICAL/CHEMICAL PARAMETERS WITH NO AVAILABLE DATA

For a number of the target chemicals, data were not identified for the parameters K_{OC} , K_H , and BCF. Where data were not available, predictive equations were used to estimate these parameters. For 13 of the target chemicals, however, the predictive equations were inappropriate therefore, the parameters K_{OC} , K_H , and BCF have been designated as "Not Applicable" in the toxicity profiles. The reasons for this designation and the impact it has on the PPLV computations are summarized below.

2.2.1 Soil/Water Partition Coefficient (K_{OC})

Estimation of K_{OC} values from K_{OC}/K_{OW} regression equations is valid only for chemicals similar to those upon which the equations are based and for K_{OW} s within the range of data used to develop the regressions. Available regressions have been developed for $\log K_{OW}$ values in the range 1-6 for a variety of organic compounds (Karickhoff 1983, Lyman and Loreti 1986); however, predictive regression equations have not been developed for organometal compounds. For 11 of the organic target contaminants, $\log K_{OW}$ values are less than 1 while an additional two contaminants are organometals. Consequently, use of the K_{OC}/K_{OW} regressions is inappropriate for these chemicals. These 13 chemicals include: Azodrin, Dimethyl methylphosphonate, Dithiane, Sarin, Isopropylmethyl Phosphonic Acid, 1,4-oxathiane N-nitrosodimethylamine, Thiodiglycol, Trimethyl phosphate, Chloroacetic acid, Fluoroacetic acid, Methylarsonic acid, and Methyl mercury.

Log K_{ow} values below one imply that the compound is non-hydrophobic as indicated by high water solubility and hence high mobility (low tendency for sorption). Compounds with log K_{ow} less than one would not exhibit a tendency to accumulate in plant or animal tissue. Therefore, human exposure via ingestion of contaminated foods would not occur.

2.2.2 Henry's Constant (K_H)

K_H is calculated from the vapor pressure of a pure chemical over a very dilute aqueous solution. K_H is negligible for contaminants with high solubilities due to high solute-solvent interactions. The 13 chemicals listed previously have high solubilities, therefore, volatilization will be negligible and exposure via the inhalation pathway does not endanger human health.

2.2.3 Bioconcentration Factor (BCF)

As discussed above for K_{oc} , the use of predictive regression equations is only appropriate for similar types of chemicals with K_{ow} 's within the range of data for which the equation was developed. The BCF equations reviewed were developed for K_{ow} values in the range 1 - 7 and are valid only for organic compounds (Lyman 1982, Davies and Dobbs 1984). Consequently, use of these equations to estimate BCF is also not appropriate for these 13 chemicals. Again, however, the properties of these chemicals (with the exception of methyl mercury) are such that human exposure as a result of bioconcentration is not expected.

3.0 DERIVATION OF ALLOWABLE DOSE VALUES (D_T)

The D_T (allowable dose) values for non-carcinogens have either been obtained from the EPA Integrated Risk Information System (IRIS) database or have been derived in a manner consistent with that employed by EPA in developing regulatory criteria and Risk Reference Doses (RfD). For carcinogens, the D_T values are based on the potency slopes computed by the EPA Cancer Assessment Group (CAG). The D_T values for the target contaminants are summarized in Appendix A. The basis for D_T development and the types of data available are described briefly below. The supporting data for each individual chemical D_T value is detailed in the profiles presented in Appendix B.

3.1 AVAILABLE DATA FOR D_T DEVELOPMENT

A variety of information sources were available for establishing the D_T values. The reliability and appropriateness of data sources were prioritized as follows.

For carcinogens: CAG potency slope

For noncarcinogens:

- o EPA Risk Reference Doses (RfDs) from the Integrated Risk Information System (IRIS) database
- o Data upon which EPA Drinking Water RMCLs are based (e.g., 50 Federal Register 46959-47008, Wednesday, Nov. 13, 1985)
- o Superfund Public Health Evaluation Manual (SPHEM) values (USEPA 1986a)^{1/}
- o Data upon which EPA Ambient Water Quality Criteria for protection of human health are based (e.g., EPA Ambient Water Quality Criteria Documents)

^{1/} The SPHEM data are consistent with the RfD, MCL and CAG data. This reference provides a convenient compilation of D_T data for a number of the indicator chemicals.

- o Data upon which EPA Drinking Water MCLs are based (e.g., Endrin: 50 Federal Register 47011, Wednesday, Nov. 13, 1985)
- o Data upon which EPA Drinking Water Health Advisory Values are based (e.g., Chlorobenzene: USEPA 1986c)
- o World Health Organization Guidelines (WHO 1984)
- o National Academy of Science (NAS) Drinking Water and Health Guidelines (NAS 1977)
- o Calculate Using NOEL or LOAEL data and appropriate uncertainty factors
- o Calculate from the LD₅₀ using the 10⁻⁵ application factor of Layton et al. 1986 (see Section 2.2.2).
- o Calculate from Threshold Limit Values (TLVs) (e.g., ACGIH 1980)
- o Calculate from Food and Drug Administration (FDA) guidelines

3.2 METHODOLOGY FOR D_T DERIVATION

For threshold-acting contaminants (those for which adverse effects are evident above some threshold concentration), available toxicity data were used to estimate a no-effect level. This information was then used to calculate an acceptable daily intake rate (D_T) which is the largest amount of toxicant not expected to result in any adverse effects to humans including sensitive subgroups, following chronic exposure. No such threshold mechanism has been universally accepted for carcinogens. The potential health risks associated with 54 carcinogens have been evaluated by the EPA CAG (USEPA 1985b). The results of the CAG evaluations are presented in terms of the likelihood that a given intake rate will result in an occurrence of cancer in the exposed individual. As discussed below, use of the CAG results requires determination of an acceptable risk which is then used to compute the contaminant intake rate associated with that risk level. The methods used to compute D_T values are detailed below. Due to the differences cited above, threshold-acting (non-carcinogenic) and carcinogenic contaminants are discussed separately.

3.2.1 Carcinogenic Contaminants

For carcinogens, the D_T value is based on the EPA CAG cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10^{-4} to 10^{-7} is considered for all carcinogens, therefore ranges of D_T values are presented. Derivation of the D_T values for carcinogens is as follows:

$$D_T = \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}}$$

Although not used directly in the computation of cancer potencies, a qualitative weight-of-evidence rating has been assigned by EPA for potential carcinogens (USEPA 1986a). The EPA approach for determining weight-of-evidence is similar to the International Agency for Research on Cancer (IARC) approach, differing primarily by having an additional category for "no evidence of carcinogenicity in humans" and revised criteria for defining evidence as "sufficient", "limited", or "inadequate" (USEPA 1986a). These criteria are presented in Table 2. The rating designations for specific chemicals are indicated in Appendix B.

3.2.2 Non-Carcinogenic Contaminants

The method employed by EPA for developing criteria or RfD values for non-carcinogens is based upon identification of a chronic No-Observed-Effect-Level (NOEL) and application of an Uncertainty factor (UF) (USEPA 1985c). Therefore D_T can be expressed as:

$$D_T = \frac{\text{NOEL}}{\text{UF}}$$

TABLE 2
 EPA WEIGHT-OF-EVIDENCE
 CATEGORIES FOR POTENTIAL CARCINOGENS^{1/}

EPA Category	Description of Group	Description of Evidence
Group A	Human Carcinogen	Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer
Group B1	Probable Human Carcinogen	Limited evidence of carcinogenicity in humans from epidemiologic studies
Group B2	Probable Human Carcinogen	Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans
Group C	Possible Human Carcinogen	Limited evidence of carcinogenicity in animals
Group D	Not Classified	Inadequate evidence of carcinogenicity in animals
Group E	No Evidence of Carcinogenicity in Humans	No evidence for carcinogenicity in at least two adequate animal tests or in both epidemiologic and animal studies

^{1/} From: USEPA 1986a

Where, the chronic NOEL is an intake rate that can be sustained over long periods of time without inducing any observable effect or change in the test species. Effects evaluated during testing include such factors as survival, bodyweight, histology, pathology, hematology and general behavior.

The appropriate UF depends upon the type of available data. Generally, the total UF is the product of a number of factors which are each based upon such considerations as test species, length of test and endpoint of test. For example, human data is preferred to animal test data, but human data are rarely available for most chemicals. Therefore, an Uncertainty Factor of 10 is assigned for interspecies extrapolation of animal data to humans. To account for intraspecies variability (sensitive humans), an additional Uncertainty Factor of 10 is appropriate. If testing has been performed for only a short period of time (i.e., less than chronic exposure), an additional Uncertainty Factor of 10 is employed to account for the necessary short-term to long-term extrapolation. In the above example a total UF of 1,000 would be used to derive D_T from a subchronic animal test with an identified NOEL. The EPA guidelines for selecting UFs are presented in Table 3 (USEPA 1985a).

Frequently the reported endpoint of toxicity testing is an effect level such as the Lowest-Observed-Effect-Level (LOEL). In this instance an additional uncertainty factor based upon the severity of the effect is required. Guidelines for selection of the additional Severity of effects Factor (SF) are presented in Table 4 (USEPA 1986a).

For many of the Rocky Mountain Arsenal target chemicals, the only available data are acute toxicity test results (LD_{50} s). For these compounds the D_T values have been obtained by multiplying the LD_{50} by an application factor equal to 10^{-5} (Layton et al. 1986). The application factor allows the derivation of an acceptable long-term intake rate (D_T) based on the results of acute tests (LD_{50}) in the absence of more suitable long-term studies (i.e., No-Observed-Effect-Level, NOEL, studies). The application factor corresponds to the

TABLE 3

EPA GUIDELINES FOR SELECTING UNCERTAINTY FACTORS^{a/b/c/}

1. Use a 10-fold factor when extrapolating from valid experimental results from studies on prolonged ingestion by man. This 10-fold factor protects the sensitive members of the human population estimated from data gathered on average healthy individuals.
2. Use a 100-fold factor when extrapolating from valid results of long-term feeding studies on experimental animals with results of studies of human ingestion not available or sparse (e.g., acute exposure only). This represents an additional 10-fold uncertainty factor in extrapolating data from the average animal to the average man.
3. Use a 1,000-fold factor when extrapolating from less than chronic results on experimental animals with no useful long-term or acute human data. This represents an additional 10-fold uncertainty factor in extrapolating from less-than-chronic to chronic exposures.
4. Use an additional factor of between 1 and 10 depending on the severity of the adverse effect when deriving an ADI from LOAEL. This uncertainty factor drops the LOAEL into the range of a NOAEL.

a/ In calculating an ADI when no indication of carcinogenicity of a chemical exists, these factors are to be applied to the highest valid NOAEL or NOEL that does not have a valid LOAEL equal to or below it. In some cases, an additional variable uncertainty factor should be applied. The EPA has recommended that this variable uncertainty factor reflect a scientific judgment of the difference between the observed LOAEL and the hypothesized NOAEL. This difference will not necessarily be the same from experiment to experiment. In lieu of specific data, the value of this additional uncertainty factor utilized by the EPA ranges from one through ten, based on the severity of the adverse effect at the LOAEL.

b/ Uncertainty factors one and two are supported by the FDA and the WHO/FAO; uncertainty factors one through three have been established by the NAS and are used in a similar form by the FDA; uncertainty factors one through four are recommended by the EPA.

c/ From: USEPA 1985a

TABLE 4
GUIDELINES FOR SELECTION OF THE SEVERITY OF EFFECTS FACTOR^{a/}

Effect	Severity Rating
Enzyme induction or other biochemical change with no pathologic changes and no change in organ weights.	1
Enzyme induction and subcellular proliferation or other changes in organelles but no other apparent effects.	2
Hyperplasia, hypertrophy or atrophy, but no change in organ weights.	3
Hyperplasia, hypertrophy or atrophy with changes in organ weights.	4
Reversible cellular changes: cloudy swelling, hydropic change, or fatty changes.	5
Necrosis, or metaplasia with no apparent decrement of organ function. Any neuropathy without apparent behavioral, sensory, or physiologic changes.	6
Necrosis, atrophy, hypertrophy, or metaplasia with a detectable decrement of organ functions. Any neuropathy with a measurable change in behavioral, sensory, or physiologic activity.	7
Necrosis, atrophy, hypertrophy, or metaplasia with definitive organ dysfunction. Any neuropathy with gross changes in behavior, sensory, or motor performance. Any decrease in reproductive capacity, any evidence of fetotoxicity.	8
Pronounced pathologic changes with severe organ dysfunction. Any neuropathy with loss of behavioral or motor control or loss of sensory ability. Reproductive dysfunction. Any teratogenic effect with maternal toxicity.	9
Death or pronounced life-shortening. Any teratogenic effect without signs of maternal toxicity.	10

^{a/} From: USEPA 1986a.

cumulative percentile on a lognormal distribution of NOEL/LD₅₀ ratios for various chemicals. The percentile was chosen to reduce the probability that the calculated dose rate would be above a toxic level; the 5th cumulative percentile was used by Layton et al. (1986) and was found to be equal to 10⁻³. The application factor also includes a safety factor of 100 to address interspecies and intraspecies variability; therefore, an interim estimate of D_T is obtained when the application factor is multiplied by the acute value. Calculation of these D_T values from LD₅₀ data is as follows:

$$D_T = \text{Acute oral LD}_{50} \times \text{Application Factor}$$

$$= \text{LD}_{50} \times 10^{-5}$$

4.0 SUMMARY

The toxicity profiles presented in Appendix B describe the toxicological properties and environmental behavior of the target contaminants. Sixty profiles are presented in Appendix B for sixty-five chemicals since some profiles address more than one chemical (e.g., Aldrin/Dieldrin, Lewisite/Lewisite Oxide, DDT/DDD/DDE, Methyl Mercury/Dimethyl Mercury, Isopropyl Methyl Phosphonate/Isopropyl Methyl Phosphonic Acid). As indicated in Table 1, data were not available for development of profiles for all 78 chemicals. The environmental transport data and D_T values presented in the profiles will be used to compute PPLVs for each target contaminant.

5.0 REFERENCES

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APPENDIX A
SUMMARY OF D_T VALUES

Chemical	EPA Carcinogenicity/Classification	Standards or Guidelines used as basis for D _T 2/3	D _T carcinogens (mg/kg/day) ^{2/3}	D _T noncarcinogens (mg/kg/day)	Comments
Aldrin		CAG Potency Slope	ARL(-4) 8.8 x 10 ⁻⁶ ARL(-5) 8.8 x 10 ⁻⁷ ARL(-6) 8.8 x 10 ⁻⁸ ARL(-7) 8.8 x 10 ⁻⁹	---	D _T : Superfund Public Health Evaluation Manual (1986)
Arsenic	Group A	CAG Potency Slope	ARL(-4) 6.7 x 10 ⁻⁶ ARL(-5) 6.7 x 10 ⁻⁷ ARL(-6) 6.7 x 10 ⁻⁸ ARL(-7) 6.7 x 10 ⁻⁹	---	D _T : Draft Report: Special Report on Ingested Inorganic Arsenic and Certain Human Health Effects (USEPA 1986)
Atrazine	Group D	EPA ADI	---	3.75 x 10 ⁻²	D _T : 46 Federal Register 63085, Wednesday December 30, 1981
Azodrin		EPA Risk Reference Dose	---	1.5 x 10 ⁻⁴	D _T : RfD for azodrin (lacks EPA verification) USEPA Integrated Risk Information System (IRIS)
Benzene	Group A	CAG Potency Slope	ARL(-4) 3.4 x 10 ⁻³ ARL(-5) 3.4 x 10 ⁻⁴ ARL(-6) 3.4 x 10 ⁻⁵ ARL(-7) 3.4 x 10 ⁻⁶		D _T : USEPA (1986) Cancer Assessment Group (Personal Communication, Bob McGaughy)
Benzothiazole (BTA)		Acute Oral LD ₅₀	---	4.7 x 10 ⁻³	D _T : Acute data from Mayhew and Muni (1986)
Bicycloheptadiene (BCH)		Acute Oral LD ₅₀	---	3.85 x 10 ⁻²	D _T : Acute data reported in Berkowitz et al. (1978)
Cadmium	Group B1	Drinking Water RMCL	---	5.0 x 10 ⁻⁴	D _T : 50 Federal Register 46964, Wednesday November 13, 1985
Carbon Tetrachloride	Group B2	CAG Potency Slope	ARL(-4) 7.7 x 10 ⁻⁴ 1.9 x 10 ⁻³ ARL(-5) 7.7 x 10 ⁻⁵ 1.9 x 10 ⁻⁴ ARL(-6) 7.7 x 10 ⁻⁶ 1.9 x 10 ⁻⁵ ARL(-7) 7.7 x 10 ⁻⁷ 1.9 x 10 ⁻⁶	---	D _T : Health Assessment Document for Carbon Tetrachloride (1984)

APPENDIX A (Continued)
SUMMARY OF DT VALUES

Chemical	EPA Carcinogenicity/Classification	Standards or Guidelines used as basis for DT	DT carcinogens (mg/kg/day) ^{3/}	DT noncarcinogens (mg/kg/day)	Comments
Chlordane	Group B2	CAG Potency Slope	ARL(-4) 6.2 x 10 ⁻⁵ ARL(-5) 6.2 x 10 ⁻⁶ ARL(-6) 6.2 x 10 ⁻⁷ ARL(-7) 6.2 x 10 ⁻⁸	---	DT: Superfund Public Health Evaluation Manual (1986)
Chloroacetic Acid		Acute Oral LD50	---	7.6 x 10 ⁻⁴	DT: Acute data is from Registry of Toxic Effects of Chemical Substances (1983)
Chlorobenzene	Group C	Drinking Water Health Advisory	---	4.3 x 10 ⁻²	DT: Office of Drinking Water Health Advisory for Chlorobenzene,
Chloroform	Group B2	CAG Potency Slope	ARL(-4) 1.2 x 10 ⁻³ ARL(-5) 1.2 x 10 ⁻⁴ ARL(-6) 1.2 x 10 ⁻⁵ ARL(-7) 1.2 x 10 ⁻⁶	---	DT: Health Assessment Document for Chloroform (1985)
p-Chlorophenyl methyl sulfide (CPMS)		LOEL basis	---	5.8 x 10 ⁻³	DT: Thake et al. (1979). Sub-chronic toxicity study using PCPMS.
p-Chlorophenyl methyl sulfone (CPMSO ₂)		LOEL basis	---	5.7 x 10 ⁻³	DT: Thake et al. (1979). Sub-chronic toxicity study using PCPMSO ₂ .
p-Chlorophenyl methyl sulfoxide (CPMSO)		LOEL basis	---	6.2 x 10 ⁻³	DT: Thake et al. (1979). Sub-chronic toxicity study using PCPMSO.
Chromium ⁺⁶	Group A	Drinking Water RMCL	---	4.82 x 10 ⁻³	DT: 50 Federal Register 46967, Wednesday November 13, 1985
Chromium ⁺³		EPA Risk Reference Dose	---	0.1 x 10 ¹	DT: USEPA Integrated Risk Information System (IRIS)
Copper	Group D	Drinking Water RMCL	---	3.8 x 10 ⁻²	DT: 50 Federal Register 46968 Wednesday November 13, 1985
DDE	Group B2	CAG Potency Slope	ARL(-4) 2.9 x 10 ⁻⁴ ARL(-5) 2.9 x 10 ⁻⁵ ARL(-6) 2.9 x 10 ⁻⁶ ARL(-7) 2.9 x 10 ⁻⁷	---	DT: The CAG potency slope for DDT is the basis for DT for this metabolite.

APPENDIX A (Continued)
SUMMARY OF DT VALUES

Chemical	EPA Carcinogenicity/Classification	Standards or Guidelines used as basis for DT	DT carcinogens (mg/kg/day) ³	DT noncarcinogens (mg/kg/day)	Comments
DDT	Group B2	CAG Potency Slope	ARL(-4) 2.9 x 10 ⁻⁴ ARL(-5) 2.9 x 10 ⁻⁵ ARL(-6) 2.9 x 10 ⁻⁶ ARL(-7) 2.9 x 10 ⁻⁷	---	DT: Superfund Public Health Evaluation Manual (1986)
Dibromochloropropane	Group B2	CAG Potency Slope	ARL(-4) 7.1 x 10 ⁻⁵ ARL(-5) 7.1 x 10 ⁻⁶ ARL(-6) 7.1 x 10 ⁻⁷ ARL(-7) 7.1 x 10 ⁻⁸	---	DT: USEPA Drinking Water Criteria Document (1985)
1,1-Dichloroethane	Group D	Acceptable Intake	---	1.15 x 10 ⁻¹	DT: The oral acceptable intake (chronic) is presented (Superfund Public Health Evaluation Manual 1986).
1,2-Dichloroethane	Group B2	CAG Potency Slope	ARL(-4) 1.1 x 10 ⁻³ ARL(-5) 1.1 x 10 ⁻⁴ ARL(-6) 1.1 x 10 ⁻⁵ ARL(-7) 1.1 x 10 ⁻⁶	---	DT: Superfund Public Health Evaluation Manual (1986)
1,1-Dichloroethylene	Group C	CAG Potency Slope Drinking Water RMCL	ARL(-4) 8.6 x 10 ⁻⁵ ARL(-5) 8.6 x 10 ⁻⁶ ARL(-6) 8.6 x 10 ⁻⁷ ARL(-7) 8.6 x 10 ⁻⁸	1.0 x 10 ⁻³	DT: 1,1-Dichloroethylene has not been adequately demonstrated to be carcinogenic by the oral route. Therefore an ADI based on the RMCL is used as DT for this exposure route.
1,2-Dichloroethylene	Group D	Drinking Water RMCL	---	1.0 x 10 ⁻²	DT: 50 Federal Register 46991, Wednesday November 13, 1985
Dicyclopentadiene (DCPD)		NOEL/NOAEL Basis	---	2.5 x 10 ⁻²	DT: Hart (1980). Subchronic toxicity study using DCPD.
Dieldrin	Group B2	CAG Potency Slope	ARL(-4) 3.3 x 10 ⁻⁶ ARL(-5) 3.3 x 10 ⁻⁷ ARL(-6) 3.3 x 10 ⁻⁸ ARL(-7) 3.3 x 10 ⁻⁹	---	DT: Superfund Public Health Evaluation Manual (1986)
Diisopropyl methyl phosphate (DIMP)		NOAEL Basis	---	7.5 x 10 ⁻¹	DT: National Research Council Committee on Toxicology
Dimethylarsonic Acid (DMAA)		NOAEL Basis	---	7.5 x 10 ⁻⁴	DT: Derse (1968). Subchronic toxicity study in Beagle dogs using DMAA (Cacodylic acid).

APPENDIX A (Continued)
SUMMARY OF DT VALUES

Chemical	EPA Carcinogenicity/Classification	Standards or Guidelines used as basis for DT $\frac{2}{3}$	DT carcinogens (mg/kg/day) $\frac{2}{3}$	DT noncarcinogens (mg/kg/day)	Comments
Dimethyl di-sulfide (DMDS)		Acute Oral LD50	---	8.13 x 10 ⁻³	DT: Acute data is from Atochem, Inc. 1986
Dimethyl Mercury		EPA Risk Reference Dose	---	3.0 x 10 ⁻⁴	DT: The RfD for methyl mercury is also used for dimethyl mercury.
Dimethyl methyl phosphonate (DMMP)		LOEL basis	---	3.12 x 10 ⁻²	DT: Dunnick et al. (1983). Reproductive toxicity study using DMMP.
Dithiane		LOEL basis	---	2.0 x 10 ⁻²	DT: Schieferstein (1986). Sub-chronic toxicity study using Dithiane.
Endrin	Group E	Interim Drinking Water MCL	---	4.5 x 10 ⁻⁵	DT: 50 Federal Register 47011 Wednesday November 13, 1985
Ethylbenzene	Group D	EPA Risk Reference Dose	---	1.0 x 10 ⁻¹	DT: USEPA Integrated Risk Information System (IRIS)
Fluoride		EPA Risk Reference Dose	---	6.0 x 10 ⁻²	DT: USEPA Integrated Risk Information System (IRIS)
Fluoroacetic Acid		Acute oral LD50	---	4.7 x 10 ⁻⁶	DT: Acute data is from Registry of Toxic Effects of Chemical Substances (1983)
Hexachlorocyclopentadiene (HCCPD)	Group D or E	EPA Risk Reference Dose	---	7.0 x 10 ⁻³	DT: USEPA Integrated Risk Information System (IRIS)
Isodrin		Acute Oral LD50	---	7.0 x 10 ⁻⁵	DT: Acute data is from Registry of Toxic Effects of Chemical Substances (1983).
Isopropyl methyl phosphonic acid (IMPA, IMP)		NOEL basis	---	3.0 x 10 ⁻¹	DT: For IMPA as computed by USAMBRDL in their derivation of Drinking Water Criteria.
Lead	Group B2, 4/	Drinking Water RMCL	---	1.88 x 10 ⁻³	DT: 50 Federal Register 46968 Wednesday November 13, 1985 (See also the Drinking Water Health Advisory for Lead)
Lewisite		Acute Oral LD50	---	2.0 x 10 ⁻⁵	DT: The DT value for Lewisite Oxide is also used for Lewisite

APPENDIX A (Continued)
SUMMARY OF D_T VALUES

Chemical	EPA Carcinogenicity/Classification	Standards or Guidelines used as basis for D _T	D _T carcinogens (mg/kg/day) ³	D _T noncarcinogens (mg/kg/day)	Comments
Lewisite Oxide		Acute Oral LD ₅₀	---	2.0 x 10 ⁻⁵	D _T : Acute data is from Registry of Toxic Effects of Chemical Substances (1983)
Malathion		EPA Risk Reference Dose	---	2.3 x 10 ⁻²	D _T : USEPA Integrated Risk Information System (IRIS)
Mercury (Inorganic) Group D		EPA Risk Reference Dose	---	2.0 x 10 ⁻³	D _T : Note: This is a verified Risk Reference Dose. It is not on IRIS because it is "at issue." Use of this value is recommended by Dr. M. Dourson, Chairman of the Reference Dose Committee.
Mercury (Methyl)		EPA Risk Reference Dose	---	3.0 x 10 ⁻⁴	D _T : USEPA Integrated Risk Information System (IRIS)
Methylarsonic Acid (MAA)		Acute Oral LD ₅₀	---	7.0 x 10 ⁻³	D _T : Acute data is from Registry of Toxic Effects of Chemical Substances (1983).
Methylene Chloride	Group B2	CAG Potency Slope	ARL(-4) 1.4 x 10 ⁻² 7.1 x 10 ⁻³ ARL(-5) 1.4 x 10 ⁻³ 7.1 x 10 ⁻⁴ ARL(-6) 1.4 x 10 ⁻⁴ 7.1 x 10 ⁻⁵ ARL(-7) 1.4 x 10 ⁻⁵ 7.1 x 10 ⁻⁶		D _T : Health Assessment Document for Methylene Chloride: Addendum (1985)
Methyl isobutyl ketone		EPA Risk Reference Dose	---	5.0 x 10 ⁻²	D _T : USEPA Integrated Risk Information System (IRIS)
Mustard	Group A	NOEL Basis	---	6.12 x 10 ⁻⁷	D _T : McNamara et al. (1975). Chronic toxicity data.
N-Nitrosodimethylamine	Group B1	CAG Potency Slope	ARL(-4) 3.9 x 10 ⁻⁶ ARL(-5) 3.9 x 10 ⁻⁷ ARL(-6) 3.9 x 10 ⁻⁸ ARL(-7) 3.9 x 10 ⁻⁹	---	D _T : Superfund Public Health Evaluation Manual (1986)
1,4-Oxathiane (Thioxane)		Acute Oral LD ₅₀	---	3.0 x 10 ⁻²	D _T : Mayhew and Munt (1986). American Biogenics Corporation.

APPENDIX A (Continued)
SUMMARY OF DT VALUES

Chemical	EPA Carcinogenicity/Classification	Standards or Guidelines used as basis for DT ²⁷	DT carcinogens (mg/kg/day) ^{3/}	DT noncarcinogens (mg/kg/day)	Comments
Parathion		NAS Guideline	---	4.3 x 10 ⁻⁴	DT: National Academy of Science (1977) Drinking Water and Health. Vol. I (With additional Safety Factor of 10)
Sarin (GB)		Control Level for General Public (CLGP)	---	8.6 x 10 ⁻⁷	DT: DA Pamphlet 40-8 (1982).
Supona		NOEL/NOAEL Basis.	---	1.5 x 10 ⁻⁴	DT: NOEL identified from Shell Chemical Company Proprietary Toxicity Report (1967)
Tetrachloro-ethylene	Group B	CAG Potency Slopes	ARL(-4) 1.96 x 10 ⁻³ 5.88 x 10 ⁻² ARL(-5) 1.96 x 10 ⁻⁴ 5.88 x 10 ⁻³ ARL(-6) 1.96 x 10 ⁻⁵ 5.88 x 10 ⁻⁴ ARL(-7) 1.96 x 10 ⁻⁶ 5.88 x 10 ⁻⁵	---	DT: Health Assessment Document for Tetrachloroethylene: Addendum (1986)
Thiodiglycol		Acute oral LD50	---	3.96 x 10 ⁻²	DT: Acute data is from Registry of Toxic Effects of Chemical Substances (1983)
Toluene	Group D	EPA Risk Reference Dose	---	3.0 x 10 ⁻¹	DT: USEPA Integrated Risk Information System (IRIS).
1,1,1-Trichloro-ethane	Group D	Drinking Water RMCL	---	3.5 x 10 ⁻²	DT: 50 Federal Register Wednesday November 13, 1985
1,1,2-Trichloro-ethane	Group C	CAG Potency Slope	ARL(-4) 1.7 x 10 ⁻³ ARL(-5) 1.7 x 10 ⁻⁴ ARL(-6) 1.7 x 10 ⁻⁵ ARL(-7) 1.7 x 10 ⁻⁶	---	DT: Health Assessment Document for Trichloroethylene Manual
Trichloro-ethylene	Group B2	CAG Potency Slopes	ARL(-4) 9.1 x 10 ⁻³ 2.2 x 10 ⁻² ARL(-5) 9.1 x 10 ⁻⁴ 2.2 x 10 ⁻³ ARL(-6) 9.1 x 10 ⁻⁵ 2.2 x 10 ⁻⁴ ARL(-7) 9.1 x 10 ⁻⁶ 2.2 x 10 ⁻⁵	---	DT: Health Assessment Document for Trichloroethylene

APPENDIX A (Continued)
SUMMARY OF DT VALUES

Chemical	EPA Carcinogenicity/ Classification	Standards or Guidelines used as basis for DT ^{2/}	DT carcinogens (mg/kg/day) ^{3/}	DT noncarcinogens (mg/kg/day)	Comments
Trimethylphosphate (TMP)		Acute Oral LD50	---	8.4 x 10 ⁻⁴	DT: Note TMP has been demonstrated to be carcinogenic in mice (NCI 1978). However, no cancer potency data are available; therefore, the acute oral LD50 value using the Layton Application factor has been used as a basis for the DT.
Vapona		NOEL/NOAEL basis.	---	8.0 x 10 ⁻⁵	DT: Chronic toxicity study using Dogs. Data from EPA Office of Pesticide Programs.
o,m,p-Xylene	Group D	Drinking Water RMCL	---	6.2 x 10 ⁻²	DT: 50 Federal Register Wednesday November 13, 1985
Zinc		Acceptable Intake Chronic	---	2.1 x 10 ⁻¹	DT: Superfund Public Health Evaluation Manual (1986)

1/ The EPA classification system for chemicals using the degree of evidence of carcinogenicity. See discussion of the classifications in Section 2.2.1 and Table 2.

2/ Where a CAG potency slope is used as a basis for the DT both oral and inhalation DT values are reported if available. Oral values are presented first. Where an RMCL is used as a basis for DT, the ADI upon which the criterion is based is reported. DT values based on acute oral LD50 values employ the Layton et al. (1986) application factor of 10⁻⁵.

3/ DT values for carcinogens are reported as a function of the Agency Risk Level (ARL). Chemicals are evaluated for the 10⁻⁴, 10⁻⁵, 10⁻⁶, and 10⁻⁷ risk levels.

4/ Certain lead salts have been documented to be carcinogenic in experimental animals. However, these salts are not the expected form of lead at RMA; therefore, the DT for lead is based on non-carcinogenic health effects.

APPENDIX B - TOXICITY PROFILES

ALDRIN/DIELDRIN^{1/}

Summary

In the environment aldrin degrades to its persistent epoxide derivative dieldrin. Both pesticides are carcinogenic in rats and mice and are teratogenic and reproductive toxicants. Aldrin and dieldrin cause liver toxicity and central nervous system abnormalities following chronic exposure. Both are acutely toxic, with oral LD₅₀ values ranging from 39-60 mg/kg in rats. Both pesticides are very toxic to aquatic organisms and have been associated with large-scale kills of terrestrial wildlife in treated areas.

CAS Number: Aldrin: 309-00-2
Dieldrin: 60-57-1

Chemical Formula: Aldrin: C₁₂H₈Cl₆
Dieldrin: C₁₂H₈Cl₆O

IUPAC Name: Aldrin: 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,
8a-hexahydro-1,4:5,8-exo-dimethanonaphthalene

Dieldrin: 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,
6,7,8,8a-octahydro-endo,exo-1,4:5,8-di methanonaphthalene

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical/Chemical and Toxicological Summaries for 62 Rocky Mountain Arsenal Contaminants. USAMBRDL. Fort Detrick, Frederick, MD.

Chemical and Physical Properties

Molecular Weights: Aldrin: 265
Dieldrin: 381

Melting Point: Aldrin: 104°C
Dieldrin: 176°C

Solubility in Water: Aldrin: 20 µg/liter at 25°C
Dieldrin: 200 µg/liter at 25°C

Solubility in Organics: Soluble in most organic solvents

Log Octanol/Water Partition Coefficient (K_{ow}):

Aldrin: 5.7 (Geyer, et al. 1984)
7.4 (Briggs 1981)
6.12 (Kadeg et al. 1986) (geo. mean of
3 literature values)
Dieldrin: 4.32 (Davies and Dobbs 1984)
6.2 (Briggs 1981)
3.69 (Rao and Davidson 1983)
5.48 (Kenaga 1980) Table III
4.95 (Kadeg et al. 1986)

Soil/Water Partition Coefficient (K_{oc}):

Aldrin:

410	Kenaga (1980) Table III (experimental)
74,000; 168,800; 1,680,000	Lyman and Loreti (1986) Eqn I ($\log K_{ow} =$ 5.66, 6.12, 7.4)
104,000; 251,800; 2,940,000	Lyman and Loreti (1986) Eqn II ($\log K_{ow} =$ 5.66, 6.12, 7.4)
98,100; 234,900; 2,660,000	Lyman and Loreti (1986) Eqn III ($\log K_{ow} =$ 5.66, 6.12, 7.4)
72,500; 154,900; 1,282,000	Kadeg et al. (1986) pesticide Eqn ($\log K_{ow}$ = 5.66, 6.12, 7.4)
47,000	Based on K_{om} . soil organic matter 60 percent organic carbon (USAMBRDL, Document B)

Aldrin (Cont):

13,200 - 17,500 Lyman et al. (1982) Eqn 4-5 (S = .002, .08)
28,200 Briggs (1981) Table III

Dieldrin:

22,600 Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 5$)
29,300 Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 5$)
28,100 Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 5$)
24,400 Kadeg et al. (1986) ($\log K_{ow} = 5$)
7,413 Briggs (1981) Table III (experimental)
35,600 Kenaga (1980) Table III (experimental)
6,600 Kadeg et al. (1986) (geo. mean 2 literature values)

Bioconcentration Factor:

Aldrin:

1,555 Davies and Dobbs (1984) Eqn C ($\log k_{ow} = 5.66$)
13,640 Davies and Dobbs (1984) Eqn C ($\log k_{ow} = 7.4$)
1,500 Lyman et al. (1982)
3,140 Kenaga (1980) Table 3 (experimental)
10,800 Kenaga (1980) Table 3 (experimental)
3,690 Davies and Dobbs (1984) Eqn B ($\log k_{ow} = 5.66$)
40,345 Davies and Dobbs (1984) Eqn C ($\log k_{ow} = 7.4$)
11,792 Lyman et al. (1982) Eqn 5-2 ($\log k_{ow} = 5.66$)
247,742 Lyman et al. (1982) Eqn 5-2 ($\log k_{ow} = 7.4$)
1,810 Davies and Dobbs (1984) Eqn C ($\log k_{ow} = 6.12$)
6,940 Davies and Dobbs (1984) Eqn B ($\log k_{ow} = 6.12$)
26,400 Lyman et al. (1982) Eqn 5-2 ($\log k_{ow} = 6.12$)

Dieldrin:

5,800; 4,420 Kenaga (1980) Table 3 (experimental)
1,489 Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 5.0$)
12,590 Davies and Dobbs (1984) Table 2 (experimental)
292 Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 4.32$)
1,130 Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 4.32$)
30,339 Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 6.2$)
1,350.7 Davies and Dobbs (1984) Eqn A (S = 0.25)
480 Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 5.0$)
3,700 Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 5.0$)

Vapor Pressure: Aldrin: 2.31×10^{-5} mm Hg at 20°C
Dieldrin: 2.8×10^{-6} mm Hg at 20°C

Henry's Law Constant: Aldrin: 2.4×10^{-5} atm-m³/mole (calculated)
 1.6×10^{-5} atm/mole (USEPA 1985)
Dieldrin: 1.4×10^{-5} atm-m³/mole (calculated)
 4.58×10^{-7} atm-m³/mole (USEPA 1985)

Transport and Fate

Aldrin evaporates rapidly from aquatic environments and soil. Photolysis occurs in aqueous solution or on plant surfaces, with conversion primarily to dieldrin, although a small fraction (generally less than 5 percent) is slowly converted to photodieldrin (Rosenblatt, et al. 1975). Hydrolysis of dieldrin is also quite slow with a half-life in excess of 4 years (USEPA 1979).

A range of experimental and estimated soil-water partition coefficients (K_{OC}) is reported above and indicates that substantial sorption of aldrin and dieldrin to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of nonpolar hydrophobic pesticides is very high; therefore little environmental mobility would be expected for these compounds.

In soil, aldrin is converted to its epoxide dieldrin, by oxidation. The conversion may be enhanced by microorganisms (Rosenblatt et al. 1975). The conversion appears to have a half-life on the order of 1 year. The persistence of dieldrin in soil is variable but may range upwards of seven years (Rosenblatt et al. 1975). Over 90 percent of applied dieldrin was still present in the top three inches of a loam soil after a period of 17 months (Rosenblatt et al. 1975). Microbial degradation does occur slowly with the main products being close derivatives (i.e., dihydroxydihydroaldrin, Rosenblatt et al. 1975).

Uptake of dieldrin in plants is variable. For example, potatoes grown in dieldrin treated soil had concentrations almost twice as high as soil levels (Telekar et al. 1983), while peeled beets had levels only one third the concentration in soil (Kohli et al. 1973). Concentrations in pasture crops appear to be less than the concentrations of the soil in which they were grown (Chawla et al. 1981).

A range of experimental and estimated bioconcentration factors (BCFs) for aldrin and dieldrin is also reported above. ASTM, (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration and biomagnification of aldrin/dieldrin residues will occur.

Health Effects

Both aldrin and dieldrin are carcinogens causing increases in a variety of tumors in rats at low but not at high doses. They also produce a higher incidence of liver tumors in mice. The reason for this reversed dose-response relationship is unclear. Neither appears to be mutagenic when tested in a number of systems. Aldrin and dieldrin are both toxic to the reproductive system and teratogenic. Reproductive effects include decreased fertility, increased fetal death, and effects on gestation. Teratogenic effects include cleft palate, webbed foot, and skeletal anomalies. Chronic effects attributed to aldrin and dieldrin include liver toxicity and central nervous system abnormalities. Both chemicals are acutely toxic. The oral LD_{50} for aldrin in rats is 39-60 mg/kg (Merck 1983). The oral LD_{50} for dieldrin in rats is 46 mg/kg (Merck 1983). The dermal LD_{50} for both aldrin and dieldrin is approximately 100 mg/kg.

Toxicity to Wildlife and Domestic Animals

Aldrin and dieldrin are both acutely toxic to freshwater species at low concentrations. Tests in fish showed that the two chemicals had similar toxicities, with LC_{50} values ranging from 1 to 46 $\mu\text{g/liter}$ for different species. Final acute values (i.e., the concentration of material protecting 95 percent of the organisms, USEPA 1980) for freshwater species were determined to be 2.5 $\mu\text{g/liter}$ for dieldrin and 3.0 $\mu\text{g/liter}$ for aldrin. Saltwater species were also quite sensitive to aldrin and dieldrin. The range of LC_{50} values was similar to that for freshwater species: 2 to 100 $\mu\text{g/liter}$ for aldrin and 1 to 34 $\mu\text{g/liter}$ for dieldrin. The saltwater Final Acute Values were 1.3 $\mu\text{g/liter}$ for aldrin and 0.71 $\mu\text{g/liter}$ for dieldrin.

Chronic studies have been conducted on the effects of dieldrin on freshwater and saltwater species. For freshwater organisms, chronic values as low as 0.2 $\mu\text{g/liter}$ were obtained. The Final Acute Chronic Ratio was determined to be 8.5, and the calculated Freshwater Final Chronic Value was 0.29 $\mu\text{g/liter}$. Only one chronic study was done on saltwater species. Therefore, the saltwater Final Chronic Value of 0.084 $\mu\text{g/liter}$ was determined by dividing the Final Acute Value by the Acute-Chronic Ratio. No chronic studies were identified for aldrin, but because its acute toxicity is comparable to that of dieldrin and because it is readily converted to dieldrin in animals and in the environment, it likely exhibits similar chronic toxicity.

Both pesticides, and especially dieldrin, have been associated with large-scale bird and mammal kills in treated areas. The acute oral LD_{50} of aldrin has been reported for a variety of avian species including mallard ducks, bobwhite quail, and pheasants. The oral LD_{50} values for aldrin in these birds are 520, 381 mg/kg and 6.59 mg/kg, respectively. The oral LD_{50} values for dieldrin in mallards and bobwhites are 16.8 and 79.0 mg/kg, respectively (Hudson et al. 1984)

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

Aquatic Life (Freshwater)

Acute toxicity: Aldrin 3.0 $\mu\text{g/liter}$
Dieldrin: 2.5 $\mu\text{g/liter}$

Chronic toxicity: Aldrin: No available data
Dieldrin: 0.0019 $\mu\text{g/liter}$

Aquatic Life (Saltwater)

Acute toxicity: Aldrin: 1.3 $\mu\text{g/liter}$
Dieldrin: 0.71 $\mu\text{g/liter}$

Chronic toxicity: Aldrin: No available data
Dieldrin: 0.0019 $\mu\text{g/liter}$

Human Health

Due to the carcinogenicity of both aldrin and dieldrin the ambient water criterion is set at zero. However, estimates of the carcinogenic risks due to ingestion of contaminated water and contaminated organisms are:

<u>Risk</u>	<u>Aldrin Concentration</u>	<u>Dieldrin Concentration</u>
10 ⁻⁵	0.74 ng/liter	0.71 ng/liter
10 ⁻⁶	0.074 ng/liter	0.071 ng/liter
10 ⁻⁷	0.0074 ng/liter	0.0071 ng/liter

CAG Potency Slope for oral exposure (USEPA 1985): Aldrin: 11.4 (mg/kg/day)⁻¹
Dieldrin: 30.4 (mg/kg/day)⁻¹

ACGIH Threshold Limit Value:* TWA^{1/} = 0.25 mg/m³
STEL^{2/} = 0.74mg/m³

OSHA Standard (air):* TWA = 250 µg/m³

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For carcinogens such as aldrin/dieldrin, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10⁻⁴ to 10⁻⁷ is considered for all carcinogens, therefore ranges of D_T values are presented. Derivation of the D_T values for aldrin/dieldrin are as follows:

$$D_T = \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}}$$
$$D_T = \frac{1 \times 10^{-4}}{11.4 \text{ (mg/kg/day)}^{-1}}$$

(aldrin)

$$= 8.8 \times 10^{-6} \text{ mg/kg/day}$$

* Applies to both aldrin and dieldrin
1/ Time Weighted Average
2/ Short Term Effect Level

The range of D_T values for aldrin/dieldrin are presented below:

<u>Risk Level</u>	<u>D_T Aldrin (mg/kg/day)</u>	<u>D_T Dieldrin (mg/kg/day)</u>
10 ⁻⁴	8.8 x 10 ⁻⁶	3.3 x 10 ⁻⁶
10 ⁻⁵	8.8 x 10 ⁻⁷	3.3 x 10 ⁻⁷
10 ⁻⁶	8.8 x 10 ⁻⁸	3.3 x 10 ⁻⁸
10 ⁻⁷	8.8 x 10 ⁻⁹	3.3 x 10 ⁻⁹

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ARSENIC^{1/}

Summary

Arsenic is a metal that is present in the environment as a constituent of organic and inorganic compounds; it also occurs in a number of valence states. Arsenic is generally rather mobile in the natural environment, with the degree of mobility dependent on its chemical form and the properties of the surrounding media. Arsenic is a human carcinogen; it causes skin tumors when it is ingested and lung tumors when it is inhaled. Arsenic compounds are teratogenic and cause adverse reproductive effects in animals. Chronic exposure to arsenic is associated with polyneuropathy (disorders of the nervous system) and skin lesions. It is acutely toxic to some early life stages of aquatic organisms at levels as low as 40 µg/liter.

Arsenic can be found in the environment in any of four valence states (-3, 0, +3,, and +5) depending on the pH, Eh, and other factors. It can exist as either inorganic or organic compounds and often will change forms as its moves through the various media. The chemical and physical properties depend on the state of the metalloid. Only the properties of metallic arsenic are presented below; properties of other arsenic compounds are often quite different.

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

CAS Number: 7440-38-2

Chemical Formula: As

IUPAC Name: Arsenic

Chemical and Physical Properties

Atomic Weight: 74.91

Boiling Point: 613°C

Melting Point: 817°C

Specific Gravity: 5.72 at 20°C

Solubility in Water: Insoluble; some salts are soluble

Transport and Fate

In the natural environment arsenic has four different oxidation states; chemical speciation is important in determining arsenic's distribution and mobility. Interconversions of the +3 and +5 states as well as organic complexation do occur and can be mediated by microorganisms. Arsenic is generally quite mobile in the environment and is mainly transported by water (WHO 1981). In oxygenated water, arsenic usually occurs as arsenate, but under reducing conditions, (i.e., deep well waters) arsenite predominates. In the aquatic environment, volatilization is important when biological activity or highly reducing conditions produce arsenine or methyl-arsenics. Sedimentation of arsenic in association with iron and aluminum does occur frequently (WHO 1981).

In oxygenated soil, inorganic arsenic is prevalent in the pentavalent (+5) form. Under reducing conditions, the trivalent form predominates (WHO 1981). Leaching of arsenates and arsenites occurs

slowly due to binding with hydrous oxides of iron and aluminum. Biomethylation in soil does occur and may be associated with the release of methylarsines into the air (WHO 1981). Plant uptake of arsenic from treated soils can occur, however, accumulation is not excessive.

Freshwater residue data for arsenic (organic and inorganic) indicate that arsenic is not bioconcentrated to a high degree but that lower forms of aquatic life may accumulate higher residues than fish (USEPA 1984a, 1986).

Health Effects

Arsenic has been implicated in the production of skin cancer in humans. There is also extensive evidence that inhalation of arsenic compounds causes lung cancer in occupationally exposed workers. Arsenic compounds also cause noncancerous (possibly precancerous) skin changes in exposed individuals. However, arsenic has not definitively been found to be a carcinogen in animal studies (USEPA 1984). EPA and the International Agency for Research on Cancer (IARC) have established that sufficient evidence exists to classify arsenic as a human carcinogen (USEPA 1984); it is therefore classified as a Group A carcinogen (i.e., human carcinogen) based upon evidence of human carcinogenicity through inhalation and ingestion exposure. Arsenic compounds have been observed to cause chromosome damage in animals. Humans exposed to arsenic compounds have been reported to have an elevated incidence of chromosome aberrations.

Arsenic compounds have been reported to be teratogenic, fetotoxic, and embryotoxic in several animal species, and an increased incidence of multiple malformations among children born to women occupationally exposed to arsenic has been reported. Several cases of progressive polyneuropathy involving motor and sensory nerves and particularly affecting the extremities and myelinated long-axon neurons have been reported in individuals occupationally exposed to inorganic arsenic. Polyneuropathies have also been reported following the ingestion of arsenic-contaminated foods.

Toxicity to Wildlife and Domestic Animals

Various inorganic forms of arsenic appear to have similar levels of toxicity. Inorganic arsenic appears to be more toxic than organic forms. Acute toxicity to adult freshwater animals occurs at levels of arsenic trioxide as low as 812 $\mu\text{g/liter}$ and at levels as low as 40 $\mu\text{g/liter}$ in early life stages of aquatic organisms. Acute toxicity to saltwater fish occurs at levels around 15 mg/liter , while some invertebrates are affected at much lower levels (508 $\mu\text{g/liter}$). Arsenic toxicity does not appear to increase greatly with chronic exposure, and it does not seem that arsenic is bioconcentrated to a great degree.

Arsenic poisoning is an uncommon but not a rare toxic syndrome among domestic animals. Arsenic causes hyperemia (site specific congestion) and edema (swelling) of the gastrointestinal tract, hemorrhage of the cardiac serosal surfaces and peritoneum, and pulmonary congestion and edema. It may also cause liver necrosis. Information on arsenic toxicity to terrestrial wildlife was not reported in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986a):

Aquatic Life (Freshwater)

Acute toxicity (As^{+3}): 360 $\mu\text{g/liter}$

Chronic toxicity (As^{+3}): 190 $\mu\text{g/liter}$

Aquatic Life (Saltwater)

Acute toxicity (As^{+3}): 69 $\mu\text{g/liter}$

Chronic toxicity (As^{+3}): 36 $\mu\text{g/liter}$

Human Health

Due to the carcinogenicity of arsenic the ambient water criterion is set at zero. However, estimates of the carcinogenic risks from the ingestion of contaminated water and contaminated aquatic organisms are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	22 ng/liter
10^{-6}	2.2 ng/liter
10^{-7}	0.22 ng/liter

CAG Potency Slope for Oral Exposure (USEPA 1986b): 15 (mg/kg/day)

CAG Potency Slope for Inhalation Exposure (USEPA 1984b): $50 \text{ (mg/kg/day)}^{-1}$

National Primary Drinking Water Standard (USEPA): 50 $\mu\text{g/liter}$
(Proposed RMCL; 50 Federal Register 46961, Wednesday November 13, 1985)

NIOSH Recommended Standard (air): Ceiling Level: 2 $\mu\text{g/m}^3$

OSHA Standard (air) TWA^{1/}: 500 $\mu\text{g/m}^3$ (organic arsenic compounds)

ACGIH Threshold Limit Value: 200 $\mu\text{g/m}^3$ (arsenic and soluble compounds)

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

^{1/} Time Weighted Average.

For carcinogens such as arsenic, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The potency slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for arsenic.

The D_T value for arsenic is based on the oral potency slope. The inhalation slope is not considered appropriate for assessing exposure to arsenic inhaled on suspended soil particulates because it has been developed from industrial emission data which are applicable to particulates smaller than 10 microns. Additionally, a substantial portion of large inhaled particulates are eventually swallowed as a result of mucocilliary transport processes ongoing in the respiratory tract.

Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10^{-4} to 10^{-7} is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values for arsenic is as follows:

$$\begin{aligned}
 D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\
 &= \frac{1 \times 10^{-4}}{15 \text{ (mg/kg/day)}^{-1}} \\
 &= 6.7 \times 10^{-6} \text{ mg/kg/day}
 \end{aligned}$$

The range of D_T values for arsenic is presented below:

<u>Risk Level</u>	<u>D_T (mg/kg/day)</u>
10^{-4}	6.7×10^{-6}
10^{-5}	6.7×10^{-7}
10^{-6}	6.7×10^{-8}
10^{-7}	6.7×10^{-9}

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ATRAZINE^{1/}

Summary

Atrazine is a triazine herbicide largely used as a preemergence pesticide for a number of fruit, vegetable, and other crops. Atrazine was fetotoxic to mice and rats following subcutaneous doses of 418 and 2,400 mg/kg body weight. Mutagenicity assays have yielded positive and negative results. Atrazine has a low chronic mammalian toxicity. Preliminary results of a two-year carcinogenicity study in rats have indicated an increased incidence of mammary adenocarcinoma in rats fed atrazine (IBT 1986, documentation pending).

CAS Number: 1912-24-9

Chemical Formula: $C_8H_{14}N_5Cl$

IUPAC Name: 2-chloro-4-ethylamino-6-isopropylamino-5-triazine

Important Synonyms and Trade Names: Aatrex; Atranex; Gesaprim;
Primatol A.

Chemical and Physical Properties

Molecular Weight: 216 (Merck 1983)

Melting Point: 173-175°C (Merck 1983)

Specific Gravity: 1.187 (TBD Peer Review Committee 1984)

Solubility in Water: 70 mg/liter at 25°C (Merck 1983)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Solubility in Organics: Soluble in ether, chloroform, and methanol

Log Octanol/Water Partition Coefficient (K_{ow}):

2.32	Rao and Davidson (1983) Table II
2.35	Rao and Davidson (1983) Table II ^{1/}
2.68	Lyman et al., (1982)
2.64	Geyer et al., (1984) Experimental ^{2/}
2.68	Geyer et al., (1984) Experimental ^{2/}
2.75	Geyer et al., (1984) Experimental ^{2/}

Soil/Water Partition Coefficient (K_{oc}):

163	Rao and Davidson (1983) Table 1 (experimental)
436 - 746	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 2.32 - 2.75$)
185; 256; 400	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 2.32, 2.5, 2.75$)

Bioconcentration Factor:

3	Davies and Dobbs (1984) Table 2 (experimental)
37.4	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.32$)
55	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.6$)
34.13 - 35.97	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.32 - 2.35$)
56	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.6$)
24 - 38	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 2.32 - 2.68$)

Vapor Pressure: 3×10^{-7} mm Hg at 20°C (TBD Peer Review Committee 1984)
 5.3×10^{-7} mm Hg at 25°C (estimated)

Henry's Law Constant: 1.2×10^{-9} atm-m³/mole (calculated based on 70 mg/l solubility)

^{1/} Cited From: Metcalf, R.L. and Lu, P.Y. 1978. Partition Coefficients as measures of bioaccumulation potential for organic compounds. EPA Report.

^{2/} Cited From: Kotzias, D. Unpublished results (1981). GSF, Institut fur Okologische Chemie, Schulstr. 10, 8050 Freising-Attaching, F.R.G.

Transport and Fate

Atrazine has a low vapor pressure and therefore vaporization is not expected to be a major transport process from surface waters or soils. Atrazine is stable in waters of neutral pH, however, in increasingly acidic or alkaline waters the stability decreases. Mabey and Mill (1978) estimate a half-life of 2.5 hours in water at a pH of 7.0 and a temperature of 25°C. Atrazine appears to be relatively persistent in soil with a half-life of 3-6 months in moist soil at 23°C.

A range of soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of atrazine to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of triazine pesticides such as atrazine will be low. The organic partitioning data presented above combined with the water solubility of atrazine suggest that this compound may exhibit some degree of environmental mobility.

Uptake of atrazine in plants can occur through both roots and foliage although quantities are usually small (Marochini 1984). Some plants (corn and several grasses) are able to hydrolyze or N-dealkylate (metabolize) atrazine (Menzie 1980). Plant metabolites include desethyl- and desisopropylatrazine and hydroxyatrazine.

A range of bioconcentration factors (BCFs) for atrazine is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of atrazine is not likely to occur.

Health Effects

Little or no data are available on the effects of human exposures to atrazine. Effects stemming from animal exposure indicate that atrazine has a low chronic toxicity. Rats fed 100 ppm of atrazine in their diets for two years produced no gross or microscopic signs of toxicity (WSSA 1974).

Atrazine has not been shown to be mutagenic in standard assays with microorganisms. Sex chromosome loss and nondisjunction were observed in mutagenicity tests with Drosophila melanogaster at oral administrations of 100 ppm atrazine (NIOSH 1983). A dominant lethal test with Drosophila m. yielded positive results following oral administration of 100 ppm atrazine (NIOSH 1983). An in vitro microsomal mutagenicity assay utilizing Aspergillus nidulans yielded positive results at a concentration of 6 mmole/liter; gene conversion and mitotic recombination were also observed at the same concentration (NIOSH 1983). Unscheduled DNA synthesis occurred in hamster fibroblasts exposed to concentrations of 3 mmole/liter atrazine. Positive results have also been obtained in a host-mediated assay with rats and mice at 100 mg/kg atrazine (NIOSH 1983). Carcinogenicity tests utilizing atrazine have yielded inconclusive results (USEPA 1985). However, preliminary results of a recently completed two-year oncogenicity study in male and female rats have indicated an increased incidence of mammary adenocarcinoma (females) and lactile engorgement (both sexes) (IBT 1986, documentation pending).

Reproductive effects have been observed in mice and rats dosed subcutaneously with atrazine. At a dose of 2,400 mg/kg during days 3-9 of gestation, rats experienced post-implantation mortality (NIOSH 1983). Rats receiving 6 g/kg atrazine subcutaneously on days 3-9 of gestation exhibited increased fetal mortality (NIOSH 1983). Mice receiving 418 mg/kg atrazine subcutaneously on days 6-14 of gestation experienced reduced litter size, fetotoxicity and fetal mortalities (NIOSH 1983).

The acute oral toxicities of atrazine in rats, mice, hamsters and rabbits are 1,750, 1,750, 1,000, and 750 mg/kg, respectively (NIOSH 1983).

Toxicity to Wildlife and Domestic Animals

Several studies have addressed the acute toxicity of atrazine in avian species. The oral LC₅₀ for young ring-necked pheasants and mallard ducks was greater than 5,000 ppm in the diet (Marochini 1984). The acute oral LD₅₀ in adult pheasants and mallards was greater than 2,000 mg/kg for both species (Hudson et al. 1984). No other data on the toxicity of atrazine to wild or domestic animals was located in available literature.

Regulations and Standards

ACGIH Threshold Limit Value: $TWA^{1/} = 10 \text{ mg/m}^3$

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For atrazine, the D_T value is derived from the same data used by EPA to compute an Acceptable Daily Intake (ADI) (46 Federal Register 63085, Wednesday December 30, 1981). The ADI is based on a chronic (2 year) oral toxicity study in dogs. Though no details on the parameters monitored or the range of dosages administered were disclosed by EPA (i.e., the study is proprietary), a No-Observed-Effect-Level of 3.75 mg/kg/day was identified. An Uncertainty Factor (UF) of 100 is included by EPA in their derivation to address

1/ Time Weighted Average.

extrapolation of the results to humans (10) and to account for intraspecies variability (sensitive subgroups) (10). Derivation of the D_T for atrazine is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOAEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{3.75}{100} \\ &= 0.0375 \text{ mg/kg/day} \end{aligned}$$

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AZODRIN^{1/}

Summary

Azodrin has a high acute toxicity and is a potent cholinesterase inhibitor in mammalian and avian systems. Rats chronically exposed to azodrin have experienced adverse reproductive effects. Standard tests for mutagenicity in bacterial systems were negative. Few data are available at present to assess the carcinogenicity of azodrin; however, in a two year study with rats, no increased incidence of tumors was observed in any dose groups.

CAS Number: 6923-22-4

Chemical Formula: $C_7H_{14}NO_5P$

IUPAC Name: Phosphoric acid dimethyl [1-methyl-3-(methylamino)-3-oxo-1-propenyl] ester

Important Synonyms and Trade Names: Monocrotophos

Chemical and Physical Properties

Molecular Weight: 223 (Merck 1983)

Melting Point: 54-55°C [pure] (Merck 1983)
25-30°C [technical] (TDB Peer Review Committee 1984)

Boiling Point: 125°C (TDB Peer Review Committee 1984)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Solubility in Water: Miscible (Merck 1983)
8100 mg/l (estimated; Lyman et al. 1982)

Solubility in Organics: Soluble in acetone, ethanol. Insoluble in diesel oils, kerosene.

Log Octanol/Water Partition Coefficient (K_{ow}): 0.30 (Lyman et al. 1982) From K_{sw} Eqn

Soil/Water Partition Coefficient (K_{oc}): Not Applicable

Bioconcentration Factor: Not Applicable

Log Benzene/Water Partition Coefficient: -0.21 (Hansch and Leo 1979)

Flash Point: >175°F

Vapor Pressure: 7×10^{-5} mm Hg at 20°C (TDB Peer Review Committee 1984)
 1.17×10^{-5} mm Hg at 25°C (estimated; Lyman et al. 1982)

Henry's Law Constant: Not Applicable

Transport and Fate

The low vapor pressure of azodrin indicates that volatilization to the atmosphere is not a major fate process for this chemical. Given the high water solubility of azodrin, sorption to soil is not expected to occur. Pavlou (1980) estimates that sorption of organophosphate pesticides ranges from low to moderate. The combined water solubility and low organic partitioning of azodrin suggest that the compound will exhibit some degree of environmental mobility.

The half-life of azodrin in water at 38°C and pH range 1-7 is estimated to be 22-23 days (TDB Peer Review Committee 1984). Uptake of azodrin in plants following foliar application leads to the formation of several by-products, however, it is unknown whether these are due to plant metabolism or hydrolysis (Menzie 1974).

Bioconcentration data for azodrin were not located in available literature. However, the high aqueous solubility combined with the low organic partitioning behavior suggest that appreciable bioconcentration is not likely to occur.

Health Effects

Principle effects in both humans and animals stemming from acute and chronic azodrin exposure is inhibition of the enzyme acetylcholinesterase in both whole blood and the brain. Chemical signs of organophosphorous pesticide poisoning in humans include tightness of the chest, difficulty in breathing, headache, dizziness, fatigue, muscle weakness and nausea. Complete recovery usually occurs within a few days, depending on the severity of exposure.

Reproductive effects in rats exposed chronically over successive generations included increased litter mortality, stunted growth or emaciation and reduced pup weights. The acute oral toxicity of azodrin is 6-23 mg/kg in rats and 11-19 mg/kg in mice. Tests for mutagenic activity of azodrin were negative in bacterial systems (Marochini 1984). A two-year feeding/oncogenicity study in rats (Shell Research LTD 1982) at doses of 0.01, 0.03, 0.10, 1.0, and 10 ppm did not result in an increased incidence of tumors at any dose level.

Toxicity to Wildlife and Domestic Animals

Azodrin has a high acute toxicity in mammals and birds. Acute oral LD₅₀ values have been reported for a number of avian species: Canada goose, 1.58 mg/kg; mallard duck, 4.76 mg/kg; bobwhite quail, 0.94 mg/kg; house sparrow, 1.48 mg/kg and house finch, 8.1 mg/kg (Hudson et al.

1984). The acute oral LD₅₀ value in female mule deer was 25-50 mg/kg and 20-50 mg/kg for female domestic goats (Hudson et al. 1984). Azodrin is less acutely toxic via the dermal exposure route. The dermal LD₅₀ is 30 mg/kg in the mallard duck.

Regulations and Standards

ACGIH Threshold Limit Value: $TWA^{1/} = 0.25 \text{ mg/m}^3$

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For azodrin, the D_T value is based on the same data used by EPA to compute a Risk Reference Dose (RfD) (USEPA 1986). The RfD is based on a chronic (2-year) feeding/oncogenicity test utilizing male and female rats (Shell Research Ltd., 1982). In this study, rats were administered doses of 0.01, 0.03, 0.10, 1.0 and 10 ppm in their diets. The principal effects at high dose levels were reduced survival of females, depressed body weights in males and significant inhibition of erythrocyte ChE in females and brain ChE in males, at 0.09 ppm. No increased incidence of tumors were observed. The No-Observed-Effect-Level (NOEL) (i.e., for ChE inhibition) identified from this study was 0.03 ppm (0.0015 mg/kg/day). An Uncertainty Factor (UF) of 10 is employed by EPA to address both cholinesterase inhibition and the extrapolation of results to humans. Derivation of the D_T value for azodrin is as follows:

1/ Time Weighted Average.

Derivation of the D_T value for azodrin is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{0.0015}{10} \\ &= 0.00015 \text{ mg/kg/day} \end{aligned}$$

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BENZENE^{1/}

Summary

Benzene is an important industrial solvent and chemical intermediate. As benzene is volatile, atmospheric photooxidation is probably an important chemical fate process. Benzene is a known human carcinogen, causing leukemia in exposed individuals. It also adversely affects the hematopoietic system. Benzene has been shown to cause fetotoxicity and embryolethality in exposed experimental animals. Exposure to high concentrations of benzene in the air causes central nervous system depression and cardiovascular effects. Dermal exposure at high concentrations may cause dermatitis.

CAS Number: 71-43-2

IUPAC Name: Benzene

Chemical Formula: C_6H_6

Chemical and Physical Properties

Molecular Weight: 78.12

Boiling Point: 80.1°C

Melting Point: 5.56°C

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical/chemical and toxicological summaries for 62 Rocky Mountain Arsenal Contaminants. USAMBRDL. Fort Detrick, Frederick, MD.

Specific Gravity: 0.88 at 20°C

Solubility in Water: 1,780 mg/liter at 25°C
1,750 mg/liter at 25°C (USEPA 1985)

Solubility in Organics: Miscible with ethanol, ether, acetic acid,
acetone, chloroform, carbon disulfide, and
carbon tetrachloride

Log Octanol/Water Partition Coefficient (K_{ow}):

2.01 (Valvani et al. 1980)
2.11 (Geyer et al. 1984)
2.12 (USEPA 1985)
2.13 (Moriguchi 1975)

Soil/Water Partition Coefficient (K_{oc}):

96	Rogers et al. (1980) Table V (experimental)
295; 343	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 2.01, 2.13$)
73	Lyman et al. (1982) Eqn 4-5 ($S = 1,700$)
106; 132	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 2.01, 2.13$)
94; 118	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 2.01, 2.13$)
95; 121	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 2.01, 2.13$)
175; 213	Kadeg et al. (1986) ($\log K_{ow} = 2.01, 2.13$)

Bioconcentration Factor:

5.2	USEPA (1985) (experimental)
24	USEPA (1980a) (experimental)
24	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2$)
19.8	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.01$)
23.6	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.11$)
24.5	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.13$)
18.5	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 2.11$)
9.3	Davies and Dobbs (1984) Eqn A ($S = 1,700$)
16.4	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 2.13$)
28.8	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.13$)

Vapor Pressure: -75 mm Hg at 20°C
95.2 mm Hg at 25°C (USEPA 1985)
100 mm Hg at 26°C (Perry and Chilton 1973)

Vapor Density: 2.77

Henry's Law Constant: 0.006 atm-m³/mole (calculated)
5.59 x 10⁻³ (USEPA 1985)

Flash Point: -11.1°C

Transport and Fate

Volatilization is the major transport process of benzene from surface waters to the ambient air and occurs readily (USEPA 1979). Atmospheric breakdown of benzene is the most likely chemical fate process following its release to air. Although direct oxidation of benzene in environmental waters is unlikely, cloud chamber data indicate that it may be photooxidized rapidly in the atmosphere. The half-life of benzene in air is approximately 6 days (USEPA 1985). In surface waters, the estimated half-life ranges from 1-6 days (USEPA 1985).

A range of experimental and estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of benzene to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and low organic partitioning of benzene suggests that this compound will exhibit some degree of environmental mobility.

A range of estimated bioconcentration factors (BCFs) for benzene is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors (BCFs) less than approximately 100 have low potential for causing harm to wildlife and human health via

biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of benzene residues is not likely to occur.

Health Effects

Benzene is a recognized human carcinogen (IARC 1982). Applying the criteria for weight of evidence proposed by the Carcinogen Assessment Group of the USEPA (50 Federal Register 46948 Wed. Nov. 13, 1985), benzene is most appropriately designated as a Group A (human) carcinogen. Several epidemiological studies provide sufficient evidence of a causal relationship between benzene exposure and leukemia in humans. Benzene is a known inducer of aplastic anemia in humans, with a latent period of up to 10 years. It produces leukopenia and thrombocytopenia, which may progress to pancytopenia. Similar adverse effects on the blood-cell-producing systems occur in animals exposed to benzene. In both humans and animals, benzene exposure is associated with chromosomal damage, although it is not mutagenic in microorganisms. Benzene was fetotoxic and caused embryoletality in experimental animals.

Exposure to very high concentrations of benzene [about 20,000 ppm (66,000 mg/m³) in air] can be fatal within minutes (IARC 1982). The prominent signs are central nervous system depression and convulsions with death usually following as a consequence of cardiovascular collapse. Milder exposure can produce vertigo, drowsiness, headache, nausea, and eventually unconsciousness if exposure continues. Deaths from cardiac sensitization and cardiac arrhythmias have also been reported after exposure to unknown concentrations. Although most benzene hazards are associated with inhalation exposure, dermal absorption of liquid benzene may occur, and prolonged or repeated skin contact may produce blistering, erythema, and a dry, scaly dermatitis. The acute oral LD₅₀ value of benzene in rats ranges from 3.4 g/kg (immature rats) to 5.6 g/kg (older adult rats, USEPA 1980b).

Toxicity to Wildlife and Domestic Animals

The EC₅₀ values for benzene in a variety of invertebrate and vertebrate freshwater aquatic species range from 5,300 µg/liter to 386,000 µg/liter (USEPA 1980b). However, only values for the rainbow trout (5,300 µg/liter) were obtained from a flow through test and were based on measured concentrations. Results based on unmeasured concentrations in static tests are likely to underestimate toxicity for relatively volatile compounds like benzene. A chronic toxicity test with Daphnia magna was incomplete, however, no adverse effects were observed at test concentrations as high as 98,000 µg/liter.

For saltwater species, acute values for one fish and five invertebrate species range from 10,900 µg/liter to 924,000 µg/liter (USEPA 1980b). Freshwater and saltwater plant species that have been studied exhibit toxic effects at benzene concentrations ranging from 20,000 µg/liter to 525,000 µg/liter (USEPA 1980b).

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

The available data are not adequate for establishing criteria. However, EPA does report the lowest concentrations of benzene known to cause toxic effects in aquatic organisms.

Aquatic Life (Freshwater)

Acute toxicity: 5,300 µg/liter
Chronic toxicity: No available data

Aquatic Life (Saltwater)

Acute toxicity: 5,100 µg/liter
Chronic toxicity: No available data

Human Health

Due to the carcinogenicity of benzene, the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from ingestion of contaminated water and contaminated aquatic organisms are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	6.6 ng/liter
10^{-6}	0.66 ng/liter
10^{-7}	0.066 ng/liter

CAG Potency Slope for Oral Exposure (USEPA 1985b): 2.9×10^{-2}
 $(\text{mg/kg/day})^{-1}$

CAG Potency Slope for Inhalation Exposure (USEPA 1985b): 2.9×10^{-2}
 $(\text{mg/kg/day})^{-1}$

ACGIH Threshold Limit Value: TWA^{1/} = 30 mg/m^3
STEL^{2/} = 75 mg/m^3

OSHA Standards: TWA = 30 mg/m^3
Ceiling Level = 75 mg/m^3
Peak Level = 150 mg/m^3 (10 min)

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

1/ Time Weighted Average

2/ Short Term Effect Level

For carcinogens such as benzene, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10^{-4} to 10^{-7} is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values for benzene is as follows:

$$\begin{aligned}
 D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\
 &= \frac{1 \times 10^{-4}}{2.9 \times 10^{-2} \text{ (mg/kg/day)}^{-1}} \\
 &= 3.4 \times 10^{-3} \text{ mg/kg/day}
 \end{aligned}$$

The range of D_T values for benzene is presented below:

<u>Risk Level</u>	<u>D_T (mg/kg/day)</u>
10 ⁻⁴	3.4 x 10 ⁻³
10 ⁻⁵	3.4 x 10 ⁻⁴
10 ⁻⁶	3.4 x 10 ⁻⁵
10 ⁻⁷	3.4 x 10 ⁻⁶

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BENZOTHIAZOLE^{1/}

Summary

No information on subchronic, chronic mutagenicity, carcinogenicity or reproductive toxicity was located in available literature. Benzothiazole has a moderate acute mammalian toxicity. The oral LD₅₀ is 900 mg/kg in the mouse and 493 and 466 mg/kg in male and female rats, respectively.

CAS Number: 95-16-9

Chemical Formula: C₆H₄SCHN

IUPAC Name: 2-Benzothiazole

Important Synonyms and Trade Names: Benzosulfonaxole, 0-2857,
1-Thia-3-azaindene, USAF EK-4812,
and 2-benzothiazole

Chemical and Physical Properties

Molecular Weight: 135.19

Boiling Point: 227°C (Merck 1983)

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Melting Point: 2°C (Weast 1977)

Specific Gravity: 1.246 at 20°C (Merck 1983)

Vapor Pressure: 0.01 - 0.05 mm Hg at 25°C (Estimated; Lyman et al. 1982)

Solubility in Water: 560 mg/liter (Estimated; Lyman et al. 1982)

Solubility in Organics: Freely soluble in alcohol and carbon disulfide

Henry's Law Constant: 4.4×10^{-6} atm - m³/mole (calculated)

Log Octanol/Water Partition Coefficient (K_{ow})^{1/}:

1.45 (Lyman et al. 1982) Fragment Method

1.67 (Lyman et al. 1982) Fragment Method

Soil/Water Partition Coefficient (K_{oc}):

168	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.56$)
47	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.56$)
40	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.56$)
41	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.56$)
83	Kadeg et al. (1986) ($\log K_{ow} = 1.56$)
129	Lyman et al. (1982) Eqn 4-5 ($S = 600$)

Bioconcentration Factor:

9.02	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.56$)
9.3	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.56$)
17.4; 16.3	Davies and Dobbs (1984) Eqn A ($S = 560; 632$)
13.2	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.56$)

^{1/} A $\log K_{ow}$ of 1.45 is derived for BTH by summing its component fragments using benzene as the basis. A $\log K_{ow}$ of 1.67 is determined by summing the component fragments using benzothiophene as the basis.

Transport and Fate

The estimated vapor pressure of benzothiazole suggests that some losses from environmental media as a result of volatilization may occur; however, no data are available on the subsequent chemical fate of volatilized benzothiazole.

A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of benzothiazole to soils/sediments and dissolved organic material will occur. The combined water solubility and low organic partitioning indicates that benzothiazole may exhibit some degree of environmental mobility.

A range of estimated bioconcentration factors (BCFs) for benzothiazole is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of benzothiazole residues is not likely to occur.

No information on the persistence of benzothiazole in air, soils, or water was located in available literature.

Health Effects

Very few data on the toxicity of benzothiazole are available. The oral LD_{50} in the mouse is 900 mg/kg, and the intravenous injection LD_{50} value is 95 mg/kg. The oral LD_{50} values of benzothiazole in male and female rats are 493 and 466 mg/kg, respectively, and 479 mg/kg for the combined sexes (Mayhew and Muni, 1986). Antemortem (before death) observations included lethargy, ataxia (uncontrolled movements), prostration, lacrimation (tearing), squinting, body cool to touch, loose stools, few or no stools, crusty eyes and muzzle and stained fur in the perianal region. Necropsy revealed pale discolored livers, pale or red discoloration of the lungs, distended urinary bladder and dark

contents of the stomachs and small intestines. No subchronic, chronic, mutagenicity, carcinogenicity or reproductive toxicity data (animal or human) are available for benzothiazole.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of benzothiazole to wildlife and domestic animals was found in the sources reviewed.

Regulations and Standards

None located.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For benzothiazole, the D_T value is derived from an acute oral toxicity value (LD₅₀) in female rats (Mayhew and Muni 1976). The D_T is computed as the product of the acute value and an application factor of 1×10^{-5} (Layton et al., 1986). The application factor allows the derivation of an interim acceptable longterm intake rate (D_T) based on the results of acute tests (LD₅₀) in the absence of more suitable longterm studies (i.e., No-Observed-Effect-Level, NOEL, studies). The application factor corresponds to the cumulative percentile on a lognormal distribution of NOEL/LD₅₀ ratios for various chemicals. The percentile was chosen to reduce the probability that the calculated dose rate would be above a toxic level; the 5th cumulative percentile was used by Layton et al. (1986) and was found to be equal to 10^{-3}). The application factor also includes a safety factor of 100 to address interspecies and intraspecies variability; therefore, an interim estimate of D_T is obtained when the application factor is multiplied by the acute value. Derivation of this D_T value is as follows:

$$\begin{aligned}
 D_T &= \text{Acute oral LD}_{50} \times \text{Application Factor} \\
 &= 466 \text{ mg/kg/day} \times 1 \times 10^{-5} \\
 &= 0.0047 \text{ mg/kg/day}
 \end{aligned}$$

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BICYCLOHEPTADIENE^{1/}

Summary

Little information is available on the environmental behavior of bicycloheptadiene. Because it is a volatile chemical, evaporation to the atmosphere will likely be a primary process in exposed soils. The estimated soil-water partition coefficient indicates that some adsorption to soils would be expected to occur. The acute oral LD₅₀ in mice is 3,850 mg/kg (Berkowitz et al. 1978.)

CAS Number: 121-46-0

Chemical Formula: C₇H₈

IUPAC Name: Bicycloheptadiene

Important Synonyms and Trade Names: Norbornadiene

Chemical and Physical Properties

Molecular Weight: 92

Boiling Point: 89°C (Marochini et al. 1984)

Specific Gravity: 0.91 (Marochini et al. 1984)

Solubility in Water: 785 mg/l (Estimated; Lyman et al. 1982)

Log Octanol/Water Partition Coefficient (K_{ow}): 1.98 (Lyman et al. 1982)
Fragment Method

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Soil/Water Partition Coefficient (K_{oc}):

285	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.98$)
101	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.98$)
88.8	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.98$)
91.1	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.98$)
167	Kadeg et al. (1986) ($\log K_{ow} = 1.98$)
225; 112	Lyman et al. (1982) Eqn 4-5 ($S = 219; 785$)

Bioconcentration Factor:

14.4; 29.6	Davies and Dobbs (1984) Eqn A ($S = 785; 219$)
13.7	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.98$)
23.4	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.98$)
18.8	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.98$)

Vapor Pressure: 51 mm Hg at 25°C (Cogley and Foy 1978)
59.7 mm Hg at 25°C (estimated from melting point;
Lyman et al. 1982)

Henry's Law Constant: 9.2×10^{-3} atm-m³/mole (calculated)

Transport and Fate

Bicycloheptadiene has a relatively high vapor pressure; therefore volatilization from environmental media to the atmosphere is expected to be a primary transport mechanism. The subsequent chemical fate of bicycloheptadiene in the atmosphere is not known.

A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of bicycloheptadiene to soils/sediments and dissolved organic material will occur. The combined water solubility and low organic partitioning suggests that bicycloheptadiene will exhibit some degree of environmental mobility. The half-life of bicycloheptadiene in soils ranges from six months to one year (Cogley and Foy 1978). Microbial degradation does occur (Cogley and Foy 1978), but the extent to which it is utilized and the subsequent degradation products were not reported. Data on the uptake of bicycloheptadiene in plants was not located in available literature.

A range of estimated bioconcentration factors (BCFs) for bicycloheptadiene is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of bicycloheptadiene residues is not likely to occur.

Health Effects

No data on the toxicity of bicycloheptadiene to humans was found in available literature. Limited data on the acute toxicity of bicycloheptadiene were located for mice and rats. The acute oral LD₅₀ in mice is 3,850 mg/kg (In Berkowitz et al. 1978). The intravenous (iv.) LD₅₀ in mice is 56 mg/kg (NIOSH 1983) and the intraperitoneal (ip.) LD₅₀ in rats is 890 mg/kg (In Berkowitz et al. 1978).

Toxicity to Wildlife and Domestic Animals

No data concerning the toxicity of bicycloheptadiene to wildlife or domestic animals was located in available literature.

Regulations and Standards

None located.

D_T Values

The D_T value is defined as that contaminant intake rate that should not induce an adverse effect or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For bicycloheptadiene, the D_T value is derived from an acute oral toxicity value (LD₅₀) in mice (Berkowitz et al. 1978). The D_T value is computed as the product of the acute value and an application factor of 1×10^{-5} (Layton et al. 1986). The application factor allows the

derivation of an interim acceptable longterm intake rate (D_T) based on the results of acute tests (LD_{50}) in the absence of more suitable longterm studies (i.e., No-Observed-Effect-Level Studies, NOELs). The application factor corresponds to the cumulative percentile on a lognormal distribution of $NOEL/LD_{50}$ ratios for various chemicals. The percentile was chosen to reduce the probability that the calculated dose rate would be above a toxic level; the 5th cumulative percentile was used by Layton et al (1986) and was found to be equal to 10^{-3} . The application factor also includes a safety factor of 100 to address interspecies and intraspecies variability; therefore, an interim estimate of D_T is obtained when the application factor is multiplied by the acute value. Derivation of this D_T value is as follows:

$$\begin{aligned} D_T &= \text{Acute } LD_{50} \times \text{Application Factor} \\ &= 3,850 \text{ mg/kg/day} \times 1 \times 10^{-5} \\ &= 0.0385 \text{ mg/kg/day} \end{aligned}$$

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CADMIUM^{1/}

Summary

Cadmium is a metal that is present in a variety of chemical forms in wastes or in the environment. Cadmium has a valence of +2 and has properties similar to those of zinc. Cadmium forms both organic and inorganic compounds. Cadmium sulfate is the most common salt. Some forms are insoluble in water, but cadmium is relatively mobile in the aquatic environment. Cadmium is carcinogenic in animals exposed by inhalation and may also be in humans. It is uncertain whether it is carcinogenic in animals or humans following oral exposure. Cadmium is a known animal teratogen and reproductive toxin. It has chronic effects on the kidney, and background levels of human exposure are thought to provide only a relatively small margin of safety for these effects.

CAS Number: 7440-43-9

Chemical Formula: Cd

IUPAC Name: Cadmium

1/ Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Chemical and Physical Properties

Atomic Weight: 112.41

Boiling Point: 765°C (Fleischer et al. 1974)

Melting Point: 321°C (Fleischer et al. 1974)

Specific Gravity: 8.642

Solubility in Water: Salts are water soluble; metal is insoluble

Solubility in Organics: Variable, based on compound

Vapor Pressure: 1 mm Hg at 394°C

Transport and Fate

Cadmium is relatively mobile in the aquatic environment compared to other heavy metals (USEPA 1979). It is removed from aqueous media by complexing with organic materials. The solubility of Cd in equilibrium with mineralized soil is primarily controlled by carbonate, and to a lesser extent by hydroxyl ion (USEPA 1979). Minimum levels of Cd solubility occur at pH levels of 9 or above. It appears that cadmium moves slowly through soil, but only limited information on soil mobility is available. Organic constituents of soil apparently can adsorb Cd from aqueous systems.

In general, Cd is readily concentrated into vegetable plant matter. However, concentration effects may be confounded by the onset of phytotoxic effects. Uptake in aquatic weeds is variable, with bioconcentration factors ranging between 600-1200 (USEPA 1980). Bioconcentration factors for cadmium in freshwater aquatic life range from 164 to 4,190 for invertebrates and from 3 to 2,213 for fishes (USEPA 1986). ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential

for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the reported concentration factors suggests that appreciable bioconcentration or biomagnification of cadmium may occur.

Health Effects

There is suggestive evidence linking cadmium with cancer of the prostate in humans (USEPA 1980). In animal studies, exposure to cadmium by inhalation caused lung tumors in rats, and exposure by injection produced injection-site sarcomas and/or Leydig-cell tumors (Takenaka 1983; USEPA 1981). An increased incidence of tumors has not been seen in animals exposed to cadmium orally, but four of the five available studies were inadequate by current standards (Clement 1983). Cadmium has been classified in EPA's Group B1 according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon evidence of carcinogenicity in humans through inhalation exposure.

The evidence from a large number of studies on the mutagenicity of cadmium is equivocal, and it has been hypothesized that cadmium is not directly mutagenic but may impede repair mechanisms (Clement 1983). Cadmium is a known animal teratogen and reproductive toxin. It has been shown to cause renal dysfunction in both humans and animals. Other toxic effects attributed to cadmium include immunosuppression (in animals), anemia (in humans), pulmonary disease (in humans), pulmonary edema and pneumonitis, possible effects on the endocrine system, defects in sensory function, and bone damage (Casarett and Doull 1980). The oral LD₅₀ in the rat is 225 mg/kg (NIOSH 1983).

Toxicity to Wildlife and Domestic Animals

Laboratory experiments suggest that cadmium may have adverse effects on reproduction in fish at levels present in lightly to moderately polluted waters. The acute LC₅₀ for freshwater fish and invertebrates generally ranged from 100 to 1,000 µg/liter. Saltwater species were in general 10-fold more tolerant to the acute effects of cadmium; however, salmonids appear to be much more sensitive to cadmium

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CARBON TETRACHLORIDE^{1/}

Summary

Carbon tetrachloride is used as an industrial solvent and chemical intermediate. It is an animal carcinogen, causing liver tumors in mice, rats, and hamsters. Carbon tetrachloride also causes liver and kidney damage in animals and humans.

CAS Number: 56-23-5

Chemical Formula: CCl_4

IUPAC Name: Tetrachloromethane

Important Synonyms and Trade Names: Tetrachloromethane,
perchloromethane

Chemical and Physical Properties

Molecular Weight: 153.8

Boiling Point: 76.7°C (Perry and Chilton 1973)

Melting Point: -22.9°C

1/ Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Specific Gravity: 1.59 at 20°C (liquid) (Perry and Chilton 1973)
5.3 vapor (gas)

Solubility in Water: 800 mg/liter (Rogers 1980)
780 mg/liter (Davies and Dobbs 1984)
930 mg/liter (Valvani et al. 1980)

Solubility in Organics: Miscible with alcohol, benzene, chloroform,
ether, and carbon disulfide

Log Octanol/Water Partition Coefficient (K_{ow}):
2.64 (Neely et al. 1974)
2.73 (Davies and Dobbs 1984; Rogers 1980)
2.78 (Geyer et al. 1984)
2.83 (Valvani et al. 1980)

Soil/Water Partition Coefficient (K_{oc}):

72	Sabljić (1984)
45	Rogers et al. (1980) Table V (experimental)
438	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 2.8$)
429	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 2.8$)
432	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 2.8$)
645	Kadeg et al. (1986) ($\log K_{ow} = 2.8$)
574 - 102	Lyman et al. (1982) Eqn 4-5 ($S = 40 - 930$)

Bioconcentration Factor:

17	Neely et al. 1974 (experimental)
72	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.8$)
69.95	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.73$)
76.3	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.78$)
83.3	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.83$)
13.7; 77	Davies and Dobbs (1984) Eqn 3 ($S = 855; 40$)
30	Davies and Dobbs (1984) Table 2 (experimental)
36	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 2.8$)
79	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.8$)
30	Barrows et al. (1980) (experimental)

Vapor Pressure: 90 mm Hg at 20°C
100 mm Hg at 23°C (Perry and Chilton 1973)
115.2 mm Hg at 25°C (Johns 1976)

Vapor Density: 5.32

Henry's Law Constant: 2.4×10^{-2} atm-m³/mole (USEPA 1985)

Transport and Fate

Carbon tetrachloride has a high vapor pressure and therefore volatilizes rapidly into the atmosphere from surface water and from surface soils. A range of experimental and estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of carbon tetrachloride to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and organic partitioning of carbon tetrachloride suggests that this compound will exhibit some degree of environmental mobility.

Because of its high specific gravity, it may move independently from the groundwater as a nonaqueous phase liquid. An estimated 7000 years are required for half of a one ppm quantity of CCl₄ to decompose in water at a pH of 7 and a temperature of 25°C (Mabey and Mill 1978). This decomposition rate is considerably accelerated in the presence of metals such as iron (USEPA 1984). Hydrolytic decomposition as a means of removal from water appears to be insignificant compared to evaporation, as the physical/chemical properties of carbon tetrachloride favor volatilization of the compound from water to air. Once in the troposphere, carbon tetrachloride remains stable. It exhibits a slow rate of reaction with hydroxyl radicals present in the troposphere. The overall atmospheric lifetime of carbon tetrachloride is estimated at 60-100 years (USEPA 1984).

A range of experimental and estimated bioconcentration factors (BCFs) for carbon tetrachloride is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and

human health via biomagnification. The magnitude of the concentration factors suggest that appreciable bioconcentration or biomagnification of carbon tetrachloride residues is not likely to occur.

Health Effects

Carbon tetrachloride was carcinogenic in mice, rats, and hamsters; inducing liver tumors in all of the species (IARC 1979; USEPA 1980). In addition, mice also displayed a high incidence of tumors of the adrenal gland (Weisburger 1977). The few case reports associated with carbon tetrachloride provide limited, but not sufficient, evidence to confirm human carcinogenicity. On the basis of the criteria proposed by the Carcinogen Assessment Group of the USEPA for evaluating the overall weight of evidence for carcinogenicity to humans, carbon tetrachloride is classified as a Group B2 carcinogen (probable human carcinogen). Carbon tetrachloride also causes liver and kidney damage in both animals and humans. Guinea pigs repeatedly exposed to carbon tetrachloride vapor for several months exhibited damage to the optic nerve and degeneration of the myelin sheath of the sciatic nerve (Smyth et al. 1936). Pretreatment or concomitant administration to rats of trichloroethylene or chloroform markedly potentiated the hepatotoxicity of carbon tetrachloride (National Toxicology Program 1986).

Rats exposed to carbon tetrachloride in utero exhibited hepatic abnormalities at birth (USEPA 1984). It has produced degenerative changes in testicular histology in rats following intraperitoneal injection at high doses which eventually resulted in aspermatogenesis and functional male infertility (USEPA 1984).

Six of seven point mutation studies utilizing bacterial test systems have yielded negative results (USEPA 1984). The remaining study was preliminary and suggestive of a weak mutagenic response. Problems associated with most of the studies result in insufficient evidence to establish genotoxicity as an effect following carbon tetrachloride exposure (USEPA 1984). The National Toxicology Program (NTP) reports in its 1986 annual plan (NTP 1986) that negative results

were obtained in mutagenicity tests with salmonella. Carbon tetrachloride has been nominated for carcinogenicity tests in both mice and rats by inhalation route (NTP 1986).

Toxicity to Wildlife and Domestic Animals

Carbon tetrachloride has been shown to be acutely toxic to aquatic species at concentrations as low as 35 mg/liter. No data on chronic toxicity to aquatic life were reported in the literature reviewed.

The low oral lethal dose (LD_{L0}) of carbon tetrachloride in dogs is 1,000 mg/kg (NOISH 1983). The low inhalation lethal concentration (LC_{L0}) in cats is 38,110 ppm/2 hr (NOISH 1983). In rabbits, the intravenous (i.v.) LD_{50} is 5,840 mg/kg (NIOSH 1983).

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

The available data are not adequate for establishing criteria. However, EPA does report the lowest values known to cause toxicity in aquatic organisms.

Aquatic Life (Freshwater)

Acute toxicity: 35,200 μ g/liter
Chronic toxicity: No available data

Aquatic Life (Saltwater)

Acute toxicity: 50,000 μ g/liter
Chronic toxicity: No available data

Human Health-

Due to the carcinogenicity of carbon tetrachloride, the ambient water criterion is set at zero. However, estimates of the carcinogenic risks from the ingestion of contaminated water and aquatic organisms are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	4.0 $\mu\text{g/liter}$
10^{-6}	0.4 $\mu\text{g/liter}$
10^{-7}	0.04 $\mu\text{g/liter}$

National Primary Drinking Water Standard (USEPA): 0.005 mg/liter
(Proposed MCL; 50 Federal Register 46904, Wednesday, November 13, 1985).

CAG Potency Slope for Oral Exposure (USEPA 1984):
 $1.3 \times 10^{-1} \text{ (mg/kg/day)}^{-1}$

OSHA Standards (air): TWA:^{1/} = 10 ppm
Ceiling Level = 25 ppm

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For carcinogens such as carbon tetrachloride, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for carbon tetrachloride. The slopes are intended to be a plausible upper bound of the potency of a carcinogen

^{1/} Time weighted average.

in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10^{-4} to 10^{-7} is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values for carbon tetrachloride is as follows:

$$\begin{aligned}
 D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\
 &= \frac{1 \times 10^{-4}}{1.3 \times 10^{-1} \text{ (mg/kg/day)}^{-1}} \\
 &= 7.7 \times 10^{-4} \text{ mg/kg/day}
 \end{aligned}$$

The range of D_T values for carbon tetrachloride is presented below:

<u>Risk Level</u>	<u>D_T Oral Exposure (mg/kg/day)</u>	<u>D_T Inhalation Exposure (mg/kg/day)</u>
10^{-4}	7.7×10^{-4}	1.9×10^{-3}
10^{-5}	7.7×10^{-5}	1.9×10^{-4}
10^{-6}	7.7×10^{-6}	1.9×10^{-5}
10^{-7}	7.7×10^{-7}	1.9×10^{-6}

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CHLORDANE^{1/}

Summary

Chlordane is an organochlorine pesticide that was formerly used on field crops and is presently used to control structural pests in homes. Technical chlordane is a complex mixture that includes two isomers of chlordane, heptachlor, and two isomers of nonachlor. It is very persistent in the environment and is readily bioaccumulated in fish and other aquatic organisms. Chlordane causes liver tumors in mice, and the results of a mutagenicity assay were positive. It causes adverse reproductive effects in mice, and chronic exposure causes liver changes and adversely affects the central nervous system. Chlordane is very toxic to aquatic organisms.

Technical chlordane is a complex mixture, however, the major components are cis-chlordane and trans-chlordane. The technical product also contains a variety of other chlorinated hydrocarbons, including heptachlor. It is a viscous amber-colored liquid. Much of the available literature does not distinguish between the chlordane isomers and appears to discuss mixtures of these compounds.

CAS Number: Chlordane (mixture): 57-74-9

cis-Chlordane: 5103-74-2

trans-Chlordane: 5103-71-9

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Chemical Formula: $C_{10}H_6Cl_8$

IUPAC Name: 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4,
7-methamoindene

Important Synonyms and Trade Names: cis-chlordane: alpha-chlordane
trans-chlordane: gamma-chlordane

Chemical and Physical Properties

Molecular Weight: 409.3

Boiling Point: 175°C at 2 mm Hg

Melting Point: cis-chlordane: 107-109°C, trans-chlordane: 103-105°C

Specific Gravity: 1.59-1.635 at 16°C (technical chlordane)

Solubility in Water: From 0.056 to 1.85 mg/liter at 25°C

Solubility in Organics: Miscible in aliphatic and aromatic solvents
(technical chlordane)

Log Octanol/Water Partition Coefficient (K_{ow}):

5.16	Kenaga (1980) Table III
3.3	Rao and Davidson (1983) Table II
3.86	Kadeg et al. (1986) (geo. mean of 3 literature values)

Soil-Water Partition Coefficient (K_{oc}):

37,500	Lyman et al. (1982) Eqn 4-5 ($S = 0.02$)
16,100; 1,490	Lyman et al (1982) Eqn 5-2 ($\log K_{ow} = 5.2, 3.3$)
1,070; 2,930; 32,400	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 3.3;$ 3.86; 5.2)
1,120; 3,280; 43,000	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 3.3;$ 3.86; 5.2)
1,115; 3,225; 41,000	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 3.3;$ 3.86; 5.2)
21,300	Kenaga (1980) Table III
1,470; 3,700; 33,900	Kadeg et al. (1986) ($\log K_{ow} = 3.3; 3.86; 5.2$)

Bioconcentration-Factor:

126	Davies and Dobbs Eqn C (1984) ($\log K_{OW} = 3.86$)
311	Davies and Dobbs Eqn B (1984) ($\log K_{OW} = 3.86$)
14,100	ECAO (1980)
505	Lyman et al. (1982) Eqn 5-2 ($\log K_{OW} = 3.86$)
3,020; 8,800	Davies and Dobbs (1984) Eqn A ($S = 0.06; 0.009$)

Vapor Pressure: 1×10^{-5} mm Hg at 20°C (USEPA 1985)

Flash Point: Minimum 81°C (technical chlordane)

Henry's Law Constant: 9.6×10^{-5} atm-m³/mole (calculated)
 9.63×10^{-6} atm-m³/mole (USEPA 1985)

Transport and Fate

Chlordane is very persistent in the environment, resisting chemical and biological degradation into less harmful substances. Chlordane is virtually insoluble in water. Chlordane present in clear water may be somewhat volatile, and this may be an important loss process. Less loss of chlordane from aquatic systems will occur when organics are present due to adsorption processes. Therefore, residue concentrations in sediment are often much higher than in water.

Chlordane binds tightly to soil particles and persists for years in soils after surface application. A range of experimental and estimated soil-water partition coefficients (K_{OC}) is reported above and indicates that sorption of chlordane to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of organochlorine pesticides is very high; therefore, little environmental mobility would be expected for this compound.

Chlordane applied as an emulsifiable concentrate is more readily volatilized than when applied as a granular formulation. Certain food and feed crops can accumulate residues by absorption from the soil. Chlordane has been found to accumulate in the peels of root vegetables studied (Rosenblatt et al. 1975). The persistence (half-life) of

chlordane in soil ranges from 2 to greater than 13 years (Rosenblatt et al. 1975). Atmospheric transport of vapors and contaminated dust particles from soil application sites can occur.

Chlordane exhibits strong tendencies for bioaccumulation in some aquatic and terrestrial organisms. It can concentrate at levels thousands of times greater than the surrounding water medium in a variety of aquatic organisms, including bacteria, algae, daphnids, and fish (USEPA 1980). A range of estimated bioconcentration factors (BCFs) for chlordane is presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that bioconcentration or biomagnification of chlordane residues will occur.

Health Effects

Mice fed diets containing analytical-grade chlordane for 80 weeks exhibited a highly significant dose-dependant incidence of liver tumors (males and females). Positive results have also been reported in carcinogenicity tests with female rats (50 Federal Register 46988, Wed. Nov. 13, 1985). Chlordane has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the positive results of these studies (50 Federal Register 46988, Wed. Nov. 13, 1985). Chlordane has induced mutagenic effects in at least one test system. Negative results were obtained in chromosome aberration tests utilizing Chinese hamster ovary cells (National Toxicology Program 1985); however, positive evidence of sister chromatid exchange was obtained in the same test medium.

Reproductive effects, including developmental defects and neonatal metabolic and biochemical disorders, are observed in the offspring of mice exposed to chlordane. Tests with laboratory animals, primarily rodents, have demonstrated acute and chronic toxic effects. Mixtures of the two isomers appear to exhibit similar toxicities to that of

single isomers. Chronic exposure to chlordane causes liver changes and induces or suppresses a variety of enzyme systems. In addition, chlordane may act as a cumulative neurotoxin.

Acute effects include anorexia, weight loss, tremors, convulsions, and death. The oral LD₅₀ in the rat is 283 mg/kg. Oxychlordane, an epoxide metabolite formed from either chlordane isomer, is more acutely toxic than chlordane. The oral LD₅₀ of oxychlordane administered to rats in corn oil is 19 mg/kg, and 43 mg/kg when administered in an aqueous suspension.

Clinical symptoms of acute oral or dermal exposure to chlordane in humans include vomiting, seizures, electroencephalographic dysrhythmia, convulsions, and possible death. Oxychlordane has been found in a high percentage of sampled human adipose tissues and also in milk samples.

Toxicity to Wildlife and Domestic Animals

Chlordane or oxychlordane residues have been found in a wide variety of wildlife and domestic animal species, but usually at relatively low levels. Studies indicate that chlordane may produce toxic effects in certain soil invertebrates after surface application. Although little information concerning bioaccumulation in these organisms is available, the potential bioaccumulation of chlordane or oxychlordane by terrestrial insectivores is of concern. Little information on the toxic effects of chlordane to mammalian wildlife and domestic animal species is available. Chlordane or oxychlordane residues have been found in crops, meat, fish and poultry, dairy products, and eggs.

Oral LD₅₀ values for chlordane ranging from 331 to 858 ppm in the diet (approximately 25-50 mg/kg) are reported for a variety of wild bird species. Oral LD₅₀ values ranging from 100 to 1,000 mg/kg are reported for a variety of animals, including rodents, goats, sheep, and chickens.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

Aquatic Life (Freshwater)

Acute toxicity: 2.4 $\mu\text{g/liter}$
Chronic toxicity: 0.0043 $\mu\text{g/liter}$

Aquatic Life (Saltwater)

Acute toxicity: 0.09 $\mu\text{g/liter}$
Chronic toxicity: 0.0040 $\mu\text{g/liter}$

Human Health

Due to the carcinogenicity of chlordane, the ambient water criterion is set at zero. However, estimates of the carcinogenic risks due to ingestion of contaminated water and contaminated aquatic organisms are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	4.6 ng/liter
10^{-6}	0.46 ng/liter
10^{-7}	0.046 ng/liter

National Primary Drinking Water Standard (USEPA): 0.005 mg/liter
(Proposed MCL; 50 Federal Register 46904, Wednesday November 13, 1985)

CAG Potency Slope for Oral Exposure (USEPA 1985): $1.61 (\text{mg/kg/day})^{-1}$

OSHA Standard (skin): $\text{TWA}^{1/} = 0.5 \mu\text{g/m}^3$

1/ Time Weighted Average

ACGIH Threshold Limit Values (skin): TWA = 0.5 mg/m³
STEL^{1/} = 2 mg/m³

Department of Transportation: Combustible liquid

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For carcinogens such as chlordane, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10⁻⁴ to 10⁻⁷ is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values for chlordane is as follows:

$$\begin{aligned} D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\ &= \frac{1 \times 10^{-4}}{1.61 \text{ (mg/kg/day)}^{-1}} \\ &= 6.2 \times 10^{-5} \text{ mg/kg/day} \end{aligned}$$

1/ Short Term Effect Level

The range of D_T values for chlordane is presented below:

<u>Risk Level</u>	<u>D_T (mg/kg/day)</u>
10^{-4}	6.2×10^{-5}
10^{-5}	6.2×10^{-6}
10^{-6}	6.2×10^{-7}
10^{-7}	6.2×10^{-8}

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CHLOROACETIC ACID^{1/}

Summary

Chloroacetic acid (sodium salt) is used most often as a pre-emergent herbicide and as a defoliant. Both the salt and the acid are irritating to the eyes and skin. It is highly toxic in laboratory rodents and more toxic than the di- and trichloroacetates. The biochemical mode of toxicity of chloroacetic acid is similar to that of its structural analog, fluoroacetic acid, being an uncompetitive inhibitor of acetate oxidation (tricarboxylic acid cycle).

CAS Number: 79-11-8

Chemical Formula: ClCH_2COOH

IUPAC Name: 1-Chloroacetic Acid

Important Synonyms and Trade Names: Chloroethanoic Acid; Monochloroacetic Acid; MCA.

Chemical and Physical Properties

Molecular Weight: 94.5 (Merck 1983)

Boiling Point: 189°C (Merck 1979)

Melting Point: 61-63°C (Merck 1983)

Specific Gravity: 1.580 (USEPA 1985)

1/ Compiled From: Various referenced sources.

Solubility in Water: Very soluble (sodium salt: 850 g/liter at 20°C)
(Merck 1983)

Log/Octanol/Water Partition Coefficient (K_{ow}): 0.22 (Hansch and
Leo 1981)

Soil/Water Partition Coefficient (K_{oc}): Not Applicable

Bioconcentration Factor: Not Applicable

Vapor Pressure: 0.791 at 25°C (Jordan 1954)
0.0651 at 20°C (Freiter 1978)

Henry's Law Constant: Not Applicable

Transport and Fate

Very little data were located on the transport and fate of chloroacetic acid in environmental media. The pKa for chloroacetic acid (2.87) is below the normal range of pH values typical of soil or water. Therefore, it is likely that the predominant form of this compound will be a salt with cations such as Na^+ and Ca^{++} rather than a free acid. The vapor pressure of chloroacetic acid indicates that volatilization can occur. Considering the high water solubility and low organic partitioning behavior of chloroacetic acid, little sorption to soils is expected to occur and therefore it will likely display a high degree of environmental mobility.

Some plants are able to metabolize the more highly chlorinated trichloroacetate (TCA). Chow (1970) reports that the roots of wheat plants grown in TCA-treated solutions absorbed almost twice the amount of chemical as did roots of similarly exposed oat plants. Overall, the wheat plants accumulated three times as much TCA in both the roots and shoots as did oat plants. Both plants were able to metabolize accumulated TCA. The half-life for TCE in wheat (shoots, roots) was 11.3 days while in oats the root and shoot half-lives were shorter, averaging 3.5 and 7.9 days, respectively (Chow 1970).

Bioconcentration data were not located for chloroacetic acid in available literature. However, given the high water solubility and low octanol/water partitioning coefficient for chloroacetic acid, the potential for bioconcentration and/or food chain transfer is expected to be negligible.

Health Effects

Chloroacetic acid is highly toxic to mammalian species. It is irritating to the skin, eyes, respiratory tract and other mucous membranes and causes burns at very high concentrations. Like its structural analog fluoroacetate, chloroacetic acid is an uncompetitive inhibitor of acetate oxidation (tricarboxylic acid cycle) (CTCP 1982). It also reduces the sulfhydryl content of liver and kidney by acetylating sulfhydryl residues. Signs and symptoms of poisoning include clonic and tonic convulsions, anuria, and respiratory depression (CTCP 1985). Ingestion causes tissue perforation (stomach) and peritonitis. Burns to the skin can result in marked fluid and electrolyte loss (CTCP 1982). Other health effects include central nervous system depression and respiratory depression (acute, chronic) (Rumack 1975).

Equivocal evidence of tumorigenicity is reported following subcutaneous administration of chloroacetic acid (1,300 mg/kg, 65 weeks) in mice (NIOSH 1983). Positive results were reported for chloroacetic acid in mutagenicity tests with mouse lymphoma cells (NTP 1985). Negative results were obtained in sex-linked recessive lethal tests in Drosophila (National Toxicology Program 1986). No data on teratogenicity or other reproductive effects were located in available literature. Monochloroacetic acid is currently in the chronic phase of toxicology and carcinogenesis testing (NTP 1986).

The acute oral LD₅₀ for chloroacetic acid in rats and mice are 76 mg/kg and 165 mg/kg, respectively (NIOSH 1983). The lethal concentration for rats via inhalation (LC₅₀) is 180 mg/m³ (NIOSH 1983).

Toxicity to Wildlife and Domestic Animals

No data were located in available literature.

Regulations and Standards

None located.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For chloroacetic acid, the D_T value is derived from an acute oral toxicity value (LD₅₀) in rats. The D_T is computed as the product of the acute value and an application factor of 1×10^{-5} (Layton et al., 1986). The application factor allows the derivation of an interim acceptable long-term intake rate (D_T) based on the results of acute tests (LD₅₀) in the absence of more suitable long-term studies (i.e., No-Observed-Effect-Level, NOEL, studies). The application factor corresponds to the cumulative percentile on a lognormal distribution of NOEL/LD₅₀ ratios for various chemicals. The percentile was chosen to reduce the probability that the calculated dose rate would be above a toxic level; the 5th cumulative percentile was used by Layton et al. (1986) and was found to be equal to 10^{-3} . The application factor also includes a safety factor of 100 to address interspecies and intraspecies variability; therefore, an interim estimate of D_T is obtained when the application factor is multiplied by the acute value. Derivation of this D_T value is as follows:

$$\begin{aligned} D_T &= \text{Acute oral LD}_{50} \times \text{Application Factor} \\ &= 76 \text{ mg/kg/day} \times 1 \times 10^{-5} \\ &= 7.6 \times 10^{-4} \text{ mg/kg/day} \end{aligned}$$

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CHLOROBENZENE^{1/}

Summary

Chlorobenzene (monochlorobenzene) is used as a solvent and as a raw material in chemical manufacturing. It is persistent in the environment and can be adsorbed to organic material in soil. Chlorobenzene may cause liver tumors in male rats. Animals exposed to chlorobenzene have exhibited liver and kidney damage.

Background Information

CAS Number: 108-90-7

Chemical Formula: C_6H_5Cl

IUPAC Name: Chlorobenzene

Important Synonyms and Trade Names: Monochlorobenzene, benzene chloride, phenyl chloride

Chemical and Physical Properties

Molecular Weight: 112.6

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Boiling Point: 131°C

Melting Point: -46°C

Specific Gravity: 1.11 at 20°C (liquid)

Solubility in Water: 500 mg/liter (Verschueren, 1977)
300 mg/liter (Tewari et al. 1982)
448 mg/liter (Mayo 1980)
625 mg/liter (Valvani et al. 1980)

Solubility in Organics: Soluble in alcohol, benzene, chloroform,
ether, and carbon tetrachloride

Log Octanol/Water Partition

Coefficient (K_{ow}): 2.83
2.84 (Valvani et al. 1980; Leo et al., 1971)
2.98 (Tewari et al. 1982)

Soil/Water Partition Coefficient (K_{oc}):

126	Sabljić (1984) (experimental)
901	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 2.9$)
189 - 127	Lyman et al. (1982) Eqn 4-5 ($S = 300 - 625$)
524	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 2.9$)
520	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 2.9$)
522	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 2.9$)
761	Kadeg et al. (1986) ($\log K_{ow} = 2.9$)

Bioconcentration Factor:

24.8 - 16.4	Davies and Dobbs (1984) Eqn 3 ($S = 300 - 625$)
10.3	ECAO (1980)
84.8; 108.3	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.84; 2.98$)
83	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.9$)
41	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 2.9$)
94	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.9$)

Vapor Pressure: 8.8 mm Hg at 20°C (TBD Peer Review Committee 1984)
10 mm Hg at 22.2°C (Perry and Chilton 1973)
20 mm Hg at 35.3°C (Perry and Chilton 1973)

Vapor Density: 3.88

Henry's Law Constant: 3.56×10^{-3} atm-m³/mole at 25°C (calculated)
2.6 atm-m³/mole (USEPA 1985)

Flash Point: 28°C

Transport and Fate

Chlorobenzene is removed from surface water primarily by volatilization. Following emission to the air, chlorobenzene is likely to degrade slowly through chemical and photolytic reactions. A range of experimental and estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of chlorobenzene to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and moderate organic partitioning suggest that chlorobenzene will exhibit some degree of environmental mobility.

A range of bioconcentration factors (BCFs) for chlorobenzene is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of chlorobenzene residues is not likely to occur.

Health Effects

A study of the carcinogenicity of chlorobenzene was recently completed by the National Toxicology Program (NTP 1985). Results show that chlorobenzene caused a statistically significant increase in neoplastic nodules in the livers of high dose male rats but was not carcinogenic in female rats or in mice of either sex. However, there were also hepatocellular carcinomas in two vehicle control male rats

and combining these with the neoplastic nodule data results in a borderline significance for high dose males in at least one statistical test (USEPA 1986a). Monochlorobenzene has been classified in EPA's Group C according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the preliminary data from the NTP study.

Increased mitotic crossovers (indicators of DNA damage) were observed in Saccharomyces cerevisiae exposed to monochlorobenzene (USEPA 1985). Additionally, monochlorobenzene induced reversions to vitamin B1 prototrophy in another study utilizing Streptomyces (USEPA 1985). Negative results were obtained in chromosome aberration tests utilizing Chinese hamster ovary cells; however, positive evidence of sister chromatid exchange was achieved in the same test medium (National Toxicology Program 1986).

Occupational studies suggest that chronic exposure to monochlorobenzene vapor may cause blood disorders, hyperlipidemia, and cardiac dysfunction in humans. Like many organic solvents, monochlorobenzene is a central nervous system depressant in overexposed humans, but no chronic neurotoxic effects have been reported (USEPA 1985). It is also irritating to the eyes and respiratory tract (USEPA 1986a). Animals exposed to chlorobenzene have exhibited liver and kidney damage. Dogs exposed to chlorobenzene vapors at doses of 2.0 mg/l 6 hours/day, 5 days/week exhibited bilateral atrophy of the epithelial tissue in the seminiferous tubules (USEPA 1985). No studies on the teratogenicity of chlorobenzene were located in available literature. The oral LD₅₀ value for chlorobenzene in rats was 2910 mg/kg (NIOSH 1983).

Toxicity to Wildlife and Domestic Animals

Chlorobenzene was acutely toxic to fish at levels greater than 25 mg/liter and to aquatic invertebrates at levels greater than 10 mg/liter (USEPA 1980a). No chronic studies on the toxicity of

chlorobenzene to aquatic life were found in the literature reviewed. No studies on chlorobenzene toxicity to terrestrial wildlife or domestic animals were reported in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986b):

The available data are not adequate for establishing freshwater or saltwater criteria. However, EPA does report the lowest values known to cause toxicity in aquatic organisms. ^{1/}

Aquatic Life (Freshwater)

Acute toxicity: 250 µg/liter

Chronic toxicity: 50 µg/liter

Aquatic Life (Saltwater)

Acute toxicity: 160 µg/liter

Chronic toxicity: 129 µg/liter

Human Health

Health criterion: 488 µg/liter

Organoleptic criterion: 20 µg/liter

National Primary Drinking Water Standard (USEPA): 0.06 mg/liter
(Proposed RMCL; 50 Federal Register 47001, Wednesday, November 13, 1985)

OSHA Standard (air): $TWA^{2/} = 350 \text{ mg/m}^3$

^{1/} Values reported are for "chlorinated benzenes."

^{2/} Time Weighted Average.

ACGIH Threshold Limit Value: TWA = 350 $\mu\text{g}/\text{m}^3$

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For chlorobenzene (monochlorobenzene), the D_T value is derived from the same data used by EPA to establish a Lifetime Health Advisory (HA) (USEPA 1987) and a soon-to-be-proposed Recommended Maximum Contaminant Level (RMCL). The reference dose (RfD) used to compute the HA is based on a subchronic toxicity study utilizing rats and mice dosed orally 5 days/week (0, 60, 125, 250, 500 or 750 mg/kg) (NTP 1985; Battelle 1978a,b). Deaths occurred in the three highest dose groups in mice and the two highest doses in rats. No changes in hematological parameters were measured in either species. Clinical chemistry parameters were unchanged in mice, however in rats, serum glutamate pyruvate transaminase and alkaline phosphatase levels were slightly elevated at 500 and 750 mg/kg. Dose-dependent polyuria with decreases in specific gravity were observed in the two high dose groups (rats). Urinary coproporphyrin excretion was also increased in rats at the two high dose levels and in female mice at 250 and 500 mg/kg. Liver and body weight ratios were increased significantly in female mice at 250 and 500 mg/kg and in male mice at 125 and 500 mg/kg. Female rats at 125 mg/kg, and male and female rats at 250 and 500 mg/kg also showed increases in liver to bodyweight ratios. Mice and rats at the three highest doses all exhibited hepatic necrosis, nephrosis, myeloid depletion, lymphoid depletion and lymphoid necrosis.

The identified No-Observed-Adverse-Effect-Level (NOAEL) from this study was 60 mg/kg/day. An Uncertainty Factor (UF) of 1,000 is employed to address extrapolation of the results to humans (10), intraspecies variability (sensitive subgroups) (10), and to adjust for

the use of a subchronic rather than a chronic (lifetime) exposure study (10). EPA has included an adjustment in the derivation to address extrapolation of the 5 day/week experimental exposure period to 7 days/week (continuous exposure). Derivation of the D_T for chlorobenzene is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOAEL (mg/kg/day)} \times (5 \text{ days})}{(\text{UF}) \times (7 \text{ days})} \\ &= \frac{60 \times 5 \text{ days}}{1,000 \times 7 \text{ days}} \\ &= 0.043 \text{ mg/kg/day} \end{aligned}$$

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CHLOROFORM^{1/}

Summary

Chloroform (trichloromethane) is often produced during the chlorination of drinking water and thus is a common drinking water contaminant. It is volatile in surface waters and is not likely to be persistent in the environment. Chloroform causes an increase in kidney epithelial tumors in rats and in hepatocellular carcinomas in mice. In addition, there is suggestive evidence from epidemiological studies that exposure to chloroform and other trihalomethanes is associated with an increased incidence of bladder tumors in humans. Other toxic effects of chloroform include central nervous system depression; eye, skin, and gastrointestinal irritation; and damage to the liver, heart, and kidney.

CAS Number: 67-66-3

Chemical Formula: CHCl_3

IUPAC Name: Trichloromethane

Chemical and Physical Properties

Molecular Weight: 119.38

Boiling Point: 61.7°C

1/ Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Melting Point: -63.5°C

Specific Gravity: 1.4832 at 20°C

Solubility in Water: 8,200 mg/liter at 20°C
7,500 mg/liter at 20°C (Valvani et al., 1980)
9,200 mg/liter at 25°C (Davies and Dobbs 1984)

Solubility in Organics: Soluble in acetone; miscible with alcohol,
ether, and benzene

Log Octanol/Water Partition

Coefficient (K_{OW}): 1.97
1.90 (Davies and Dobbs 1984)
1.96 (Valvani et al. 1980)

Soil-Water Partition Coefficient (K_{OC}):

45	Sabljic (1984)
257	Lyman et al. (1982) Eqn 5-2 ($\log K_{OW} = 1.9$)
87	Lyman and Loreti (1986) Eqn I ($\log K_{OW} = 1.9$)
76	Lyman and Loreti (1986) Eqn II ($\log K_{OW} = 1.9$)
78	Lyman and Loreti (1986) Eqn III ($\log K_{OW} = 1.9$)
146	Kadeg et al. (1986) ($\log K_{OW} = 1.9$)

Bioconcentration Factor (BCF):

16	Lyman et al. (1982) Eqn 5-2 ($\log K_{OW} = 1.9$)
18.18	Lyman et al. (1982) Eqn 5-2 ($\log K_{OW} = 1.96$)
3.59; 4.03	Davies and Dobbs (1984) Eqn A ($S = 7,500, 9,200$)
21	Davies and Dobbs (1984) Eqn B ($\log K_{OW} = 1.9$)
12	Davies and Dobbs (1984) Eqn C ($\log K_{OW} = 1.9$)

Vapor Pressure: 150.5 mm Hg at 20°C
200 mm Hg at 25.9°C (Perry and Chilton 1973)

Vapor Density: 4.12

Henry's Law Constant: 3.9×10^{-3} atm-m³/mole (calculated)
 2.87×10^{-3} atm-m³/mole (USEPA 1985a)

Transport and Fate

Due to its high vapor pressure, volatilization is the major transport process for removal of chloroform from aquatic systems (USEPA 1979). Once in the troposphere, chloroform is attacked by hydroxyl radicals with the subsequent formation of phosgene (COCl_2) and possibly chlorine oxide (ClO) radicals. Neither of these reaction products is likely to persist; phosgene is readily hydrolyzed to hydrochloric acid and carbon dioxide. Reaction with hydroxy radicals is thought to be the primary environmental fate of chloroform. However, chloroform that remains in the troposphere may return to earth in precipitation or adsorbed on particulates, and a small amount may diffuse upward to the stratosphere where it photodissociates via interaction with ultraviolet light (USEPA 1985b). Neither photolysis or hydrolysis, appear to be significant environmental fate processes for chloroform (USEPA 1985b).

A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of chloroform to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined high water solubility and low organic partitioning of chloroform suggest that this compound will exhibit a high degree of environmental mobility.

Studies with marine organisms provide evidence for only weak to moderate bioaccumulation of chloroform. A range of estimated bioconcentration factors (BCFs) for chloroform is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of chloroform residues is not likely to occur.

Health Effects

Humans may be exposed to chloroform by inhalation, ingestion, or skin contact. Epidemiological studies suggest that higher concentrations of chloroform and other trihalomethanes in water supplies may be associated with an increased frequency of bladder cancer in humans. Other toxic effects include local irritation of the skin or eyes, central nervous system depression, gastrointestinal irritation, liver and kidney damage, cardiac arrhythmia, ventricular tachycardia, and bradycardia. Death from chloroform overdosing can occur and is attributed to ventricular fibrillation. Chloroform anesthesia can produce delayed death as a result of liver necrosis.

In laboratory animals, exposure to chloroform by inhalation, intragastric administration, or intraperitoneal injection produces liver and kidney damage. Chronic administration of chloroform by gavage is reported to produce a dose-related increase in the incidence of kidney epithelial tumors in rats and a dose-related increase in the incidence of hepatocellular carcinomas in mice (IARC 1979, USEPA 1980). Based on EPA's Proposed Carcinogen Risk Assessment Guidelines, chloroform is classified in EPA's Group B2 (probable human carcinogen) based upon sufficient evidence of carcinogenicity in animals and inadequate epidemiologic evidence (USEPA 1985b).

An increased incidence of fetal abnormalities was reported in offspring of pregnant rats exposed to chloroform by inhalation. Oral doses of chloroform that caused maternal toxicity produced relatively mild fetal toxicity in the form of reduced birth weights. There are limited data suggesting that chloroform has mutagenic activity in some test systems. However, negative results have been reported for bacterial mutagenesis assays.

The oral LD_{50} and inhalation LC_{LO} values for chloroform in the rat are 908 mg/kg and 39,000 mg/m³ per 4 hours, respectively (ACGIH 1980).

Toxicity to Wildlife and Domestic Animals

Limited information is available concerning the toxicity of chloroform to organisms exposed at known concentrations (USEPA 1980). Median effect concentrations for two freshwater and one invertebrate species range from 28,900 to 115,000 $\mu\text{g/liter}$. Twenty-seven day LC_{50} values of 2,030 and 1,240 $\mu\text{g/liter}$ were reported for embryo-larval tests with rainbow trout in water at two levels of hardness. The only reliable result concerning the toxicity of chloroform to saltwater aquatic life is a 96-hour LC_{50} value of 81,500 $\mu\text{g/liter}$ for pink shrimp.

No data were located on the toxicity of chloroform to wild or domestic animals in the literature reviewed. Conceivably, acute effects on wildlife can occur in the vicinity of a major chloroform spill, however, chronic effects from long term exposure to low ambient levels is unlikely (USEPA 1985b).

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

The available data are not adequate for establishing criteria. However, EPA does report the lowest values known to be toxic in freshwater aquatic organisms.

Aquatic Life (Freshwater)

Acute Toxicity: 28,900 $\mu\text{g/liter}$

Chronic Toxicity: 1,240 $\mu\text{g/liter}$

Human Health

Due to the carcinogenicity of chloroform the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from the ingestion of contaminated water and contaminated aquatic organisms are:

<u>Risk</u>	<u>Concentration</u>
10 ⁻⁵	1.90 µg/liter
10 ⁻⁶	0.19 µg/liter
10 ⁻⁷	0.019 µg/liter

CAG Potency Slope for oral exposure (USEPA 1985b): 8.1×10^{-2}
(mg/kg/day)⁻¹

Primary Drinking Water Standard (MCL): 0.10 mg/liter (total trihalomethanes) (40 CFR 141.12)

NIOSH Recommended Standard: Ceiling = 9.8 mg/m³ (1 hr)

OSHA Standard: Ceiling = 244 mg/m³

ACGIH: STEL^{1/} = 50 mg/m³

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For carcinogens such as chloroform, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10⁻⁴ to 10⁻⁷ is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values for chloroform is as follows:

1/ Short Term Effect Level

$$\begin{aligned}
 D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\
 &= \frac{1 \times 10^{-4}}{8.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}} \\
 &= 1.2 \times 10^{-3} \text{ mg/kg/day}
 \end{aligned}$$

The range of D_T values for chloroform is presented below:

<u>Risk Level</u>	<u>D_T Value (mg/kg/day)</u>
10 ⁻⁴	1.2 x 10 ⁻³
10 ⁻⁵	1.2 x 10 ⁻⁴
10 ⁻⁶	1.2 x 10 ⁻⁵
10 ⁻⁷	1.2 x 10 ⁻⁶

REFERENCES

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p-CHLOROPHENYL METHYLSULFIDE^{1/}

Summary

No data regarding the toxicity of p-chlorophenyl methylsulfide (PCPMS) to humans was located in available literature. Rats exposed chronically to oral doses of PCPMS experienced elevated serum potassium and calcium levels, reduced serum glutamic oxalacetic transaminase (SGOT) levels and red blood cell counts. Rhesus monkeys subacutely exposed to oral doses of PCPMS experienced mortality at the highest dose. At lower doses increased blood urea nitrogen (BUN), liver and kidney weights, and decreased serum alkaline phosphatase were observed. Liver lesions were observed at all dose levels. PCPMS tested negative in the Ames mutagenicity assay.

CAS Number: 123-09-1

Chemical Formula: C_7H_7SCl

IUPAC Name: p-Chlorophenyl Methylsulfide

Important Synonyms and Trade Names: PCPMS, p-chlorothioanisole

Chemical and Physical Properties

Molecular Weight: 158.7

Melting Point: 17-19°C, (Miller et al. 1976)

Boiling Point: 220-224°C (Miller et al. 1976)

Solubility in Water: 12 mg/liter (estimated; Lyman et al. 1982)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Specific Gravity: 1.2 at 49°C (Miller et al. 1976)

Vapor Pressure: 20 mm Hg at 118°C (Miller et al. 1976)
0.11 mm Hg at 25°C (estimated; Lyman et al. 1982)

Henry's Law Constant: 1.9×10^{-3} atm-m³/mole (calculated)
 8.35×10^{-4} atm-m³/mole (calculated)

Log Octanol/Water Partition Coefficient (K_{ow}): 3.22 (Brueggemann 1982)
3.35 (Lyman et al. 1982) Fragment Method

Soil/Water Partition Coefficient (K_{oc}):

1,583	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 3.35$)
1,174	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 3.35$)
1,233	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 3.35$)
1,224	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 3.35$)
1,599	Kadeg et al. (1986) ($\log K_{ow} = 3.35$)

Bioconcentration Factor:

69.1	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 3.35$)
154	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 3.35$)
207	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 3.35$)

Transport and Fate

The low to moderate vapor pressure of PCPMS indicates that some volatilization (evaporation) from soil surfaces would be expected to occur. PCPMS is relatively insoluble in water (Cogley and Foy 1978). A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of PCPMS to soils/sediments and dissolved organic material will occur. The combined low water solubility and high organic partitioning indicates that little environmental mobility would be expected for this compound.

Microbial degradation reportedly occurs (Cogley and Foy 1978) however the extent of degradation and the resulting degradation intermediates were not reported. PCPMS can be chemically oxidized to

sulfoxide under relatively mild conditions. The persistence (half-life) of PCPMS in soil may vary from one to six months depending on ambient conditions (Cogley and Foy 1978). Guenzi et al. (1979) reported that 61 percent of soil applied PCPMS was retained for 160 days.

Data on the uptake of PCPMS in various plant parts has been reported (Guenzi et al. 1979). At a soil concentration of 0.35 $\mu\text{g/g}$ soil, concentration factors for tops and roots, respectively, of various plants ranged from 15 and 6 in corn to 64 and 6 in sugarbeets.

A range of estimated bioconcentration factors (BCFs) for PCPMS is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that bioconcentration or biomagnification of PCPMS residues may occur.

Health Effects

No data on the toxicity of PCPMS in humans was located in available literature. Rats exposed subchronically (91 days) to PCPMS at 750, 1,500, and 3,000 ppm sulfide experienced reduced red blood cell counts and males experienced reduced SGOT levels (Thake et al. 1979). Serum potassium and calcium levels were markedly elevated in both males and females, and increased liver and kidney weights were observed. Compound-related lesions were present in the livers of exposed rats. Post-mortem necropsies of sacrificed animals indicated incipient tumor formation. Mutagenicity tests utilizing the Ames (Salmonella) assay were negative (Thake et al. 1979).

Subacute oral toxicity studies (14 days) employing dosages of 5, 10, and 20 mg/kg in Rhesus monkeys resulted in mortalities at the highest dose (Thake et al. 1979). The clinical signs of toxicity included anorexia, hypothermia, depression, weakness, and diarrhea. Thrombocytopenia and neutrophilia were apparent at the two lower

dosages. Increased blood urea nitrogen (BUN), decreased serum alkaline phosphatase, and increased liver and kidney weights were observed at the 10 mg/kg dose. Liver lesions consisting of vacuolization of hepatocytes and necrosis were observed at low dosages. At higher dosages, vacuolization of proximal tubular epithelium was observed.

Acute oral toxicity of PCPMS in rats ranged from 479 (female) to 619 mg/kg (male). Acute oral toxicity in mice ranged from 672 (female) to 877 mg/kg. Clinical signs included dyspnea (labored breathing) and lacrimation (Thake et al. 1979). Dermal LD₅₀ values in rats ranged from 2,190 to 5,630 mg/kg.

Toxicity to Wildlife and Domestic Animals

No data on the toxicity of PCPMS to other wildlife or domestic animal species was located in the available literature.

Regulations and Standards

None located.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For p-chlorophenyl methylsulfide (PCPMS) the D_T value is derived from a Low-Observed-Effect-Level (LOEL) based on a subchronic toxicity study in mice and rats (Thake et al. 1979). Reduced red blood cell counts and increased liver and kidney weights were observed at all dose levels in the experiment. The LOEL identified in this study was 58 mg/kg/day. An Uncertainty Factor (UF) of 1,000 is employed in computing D_T to address the extrapolation of the results to humans (10), intraspecies variability (sensitive subgroups) (10) and conversion of the results to a chronic (lifetime) basis (10). An

additional Uncertainty Factor of 10 is also included to address 1) the use of a LOEL rather than a NOEL and 2) the potential carcinogenicity of PCPMS. Derivation of this D_T value is as follows:

$$\begin{aligned} D_T &= \frac{\text{LOEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{58}{10,000} \\ &= 0.0058 \text{ mg/kg/day} \end{aligned}$$

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p-CHLOROPHENYL METHYLSULFONE^{1/}

Summary

No data on the toxicity of p-chlorophenyl methylsulfone (PCPMSO₂) in humans was located in available literature. PCPMSO₂ was mildly irritating to the skin of rabbits following dermal application. Subchronic exposures to PCPMSO₂ in rats resulted in altered blood serum chemistry, liver lesions, and increased liver and kidney weights. In monkeys, subacute oral exposures to PCPMSO₂ resulted in mortality at the highest doses, and increased levels of blood urea nitrogen (BUN), serum enzymes, and sodium at all other dose levels. PCPMSO₂ was not mutagenic when evaluated using the Ames Salmonella assay.

CAS Number: 98-57-7

Chemical Formula: C₇H₇SO₂Cl

IUPAC Name: p-Chlorophenyl Methylsulfone

Important Synonyms and Trade Names: PCPMSO₂, 1-chloro,
4-methylsulfoxybenzene

Chemical and Physical Properties

Molecular Weight: 190.6

Melting Point: 92-99°C, (Miller et al. 1976)

Solubility in Water: 1,050 mg/liter (estimated; Lyman et al. 1982)
1,170 mg/liter (estimated; Lyman et al. 1982)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Vapor Pressure: 5×10^{-4} mm Hg at 25°C (estimated; Lyman et al. 1982)
2.5 mm Hg at 132°C (Miller et al. 1976)

Henry's Law Constant: 1.2×10^{-7} atm-m³/mole (calculated)
 4.29×10^{-6} atm-m³/mole (calculated)

Log Octanol/Water Partition Coefficient (K_{ow}): 1.20 (Brueggemann 1982)
1.21 (Lyman et al.
1982) Fragment Method

Soil/Water Partition Coefficient (K_{oc}):

108	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.21$)
25.3	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.21$)
20.2	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.21$)
21.1	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.21$)
46.7	Kadeg et al. (1986) ($\log K_{ow} = 1.21$)

Bioconcentration Factor:

5.54	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.21$)
8.14	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.21$)
4.89	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.21$)

Transport and Fate

The low vapor pressure of PCPMSO₂ indicates that volatilization (evaporation) from soil/water surfaces is not likely to be a major transport process for this chemical. PCPMSO₂ is slightly soluble in water (Cogley and Foy 1978). A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of PCPMSO₂ to soils/sediments and dissolved organic material may occur. The combined water solubility and low organic partitioning indicate that PCPMSO₂ will exhibit some degree of environmental mobility. Microbial degradation reportedly occurs (Cogley and Foy 1978) however, the extent to which PCPMSO₂ is utilized and the resulting degradation products were not reported.

The persistence (half-life) of PCPMSO₂ in soil may vary from six months to one year depending on ambient conditions (Cogley and Foy 1978). Guenzi et al. (1979) reported that 82.5 percent of applied sulfone in soil (4.77 µg/g) incubated at 30°C was retained following 160 days. Uptake of sulfone in selected plants was also reported by Guenzi et al. Concentration factors in the tops and roots ranged from 19 and 6, respectively, in corn to 72 and 5 in sugar beets.

A range of estimated bioconcentration factors (BCFs) for PCPMSO₂ is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of PCPMSO₂ residues is not likely to occur.

Health Effects

No data on the toxicity of PCPMSO₂ in humans was located in available literature. Sulfone was nonmutagenic utilizing the Ames assay (Thake et al. 1979). Data on the carcinogenicity of PCPMSO₂ was not located in available literature.

Topical application of PCPMSO₂ to the skin of rabbits produced mild irritation and no effect was noted following ocular treatment (Thake et al. 1979). Rats exposed subchronically (91 days) to PCPMSO₂ at doses of 750, 1,500 and 3,000 mg/kg/day in the diet experienced reduced red blood cell counts and males had elevated BUN levels (Thake et al. 1979). Males and females experienced elevated serum potassium and calcium and liver and kidney weights were markedly increased. Compound-related lesions were present in the livers of rats of both sexes. Post-mortem necropsies of sacrificed animals indicated incipient tumor formation.

Subacute oral toxicity studies (14 days) were conducted with dosages of 2.5, 5, 10, 15, 20, and 30 mg/kg PCPMSO₂ in Rhesus monkeys (Thake et al. 1979). Doses of 20 and 30 mg/kg were lethal. Clinical

signs included anorexia, emesis, and diarrhea. Increased BUN, serum glutamic oxalate transaminase (SGOT), and sodium values as well as decreased serum glucose and inorganic phosphorus were observed at 10 mg/kg and higher doses. Increased adrenal weights were also noted at this dose level. Lymphoid tissue hyperplasia was noted as were liver lesions.

Toxicity to Wildlife and Domestic Animals

No data on the toxicity of PCPMSO₂ to wildlife or domestic animals was located in the available literature.

Regulations and Standards

None located.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For p-chlorophenyl methylsulfone (PCPMSO₂) the D_T value is derived from a Low-Observed-Effect-Level (LOEL) based on an oral subchronic toxicity study in mice and rats (Thake et al. 1979). Reduced red blood cell counts and increased liver and kidney weights were observed at all dose levels in the experiment. The LOEL identified from this study was 57 mg/kg/day. An Uncertainty Factor (UF) of 1,000 is employed in computing D_T to address extrapolation of the results to humans (10), intraspecies variability (sensitive groups) (10), and conversion from a subchronic to a lifetime (chronic) exposure period (10). An additional Uncertainty Factor of 10 is used to address

1) the use of a LOEL rather than a NOEL and 2) the potential carcinogenicity of PCPMSO₂. Derivation of this D_T value is as follows:

$$\begin{aligned} D_T &= \frac{\text{LOEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{57}{10,000} \\ &= 0.0057 \text{ mg/kg/day} \end{aligned}$$

REFERENCES

- ASTM. 1985. Standard Practice for conducting bioconcentration tests with fishes and saltwater bivalve mollusca. Designation E 1022-84, pages 590-62. In: 1985 Annual Book of ASTM Standards Volume 11.04. American Society for Testing and Materials, Philadelphia, Pennsylvania.
- BRUEGGEMANN, E. 1982. Partition Coefficients. Log P Determination by HPLC. Laboratory Research Notebook No. 748. U.S. Army Medical Bioengineering Research and Development Laboratory. Fort Detrick, Frederick, Maryland.
- COGLEY, D.R. AND FOY, W. 1978. Readily Available Data on 169 Compounds Associated with Operations at Rocky Mountain and Pine Bluff Arsenals. United States Army Medical Research and Development Command, Ft. Detrick, Frederick, MD. December 1978. AD A074214.
- DAVIES, R.P. and A.J. DOBBS. 1984. The prediction of bioconcentration in fish. Water Res. 18(10):1253-1262.
- GUENZI, W.D., W.E. BEARD, R.A. BOWMAN, and S.R. OLSEN. 1979. Plant Toxicity and Soil Transformation of Soil Incorporated Sulfur Compounds. Final Report, USDA-SEA-Agricultural Research-Western Region, Colorado-Wyoming Area. Fort Collins, CO.

p-CHLOROPHENYL METHYLSULFOXIDE^{1/}

Summary

No data regarding the toxicity of p-chlorophenyl methylsulfoxide (PCPMSO) to humans was located in available literature. Rats chronically exposed to PCPMSO experienced reduced red blood cell counts, reduced levels of certain serum enzymes, emaciation, increased liver and kidney weights, liver lesions, and mortality. Subacute dosages of PCPMSO to Rhesus monkeys resulted in depression, anorexia, emesis, hypothermia, decreased red blood cell count, increased levels of serum enzymes, nitrogen wastes, and calcium levels, and increased kidney and liver weights. No data on the carcinogenicity of PCPMSO was located in the available literature. Mutagenicity assays using Salmonella were negative.

CAS Number: 934-73-6

Chemical Formula: C_7H_7SOCl

IUPAC Name: p-Chlorophenyl Methylsulfoxide

Important Synonyms and Trade Names: PCPMSO, 1-chloro,
4-methylsulfinylbenzene

Chemical and Physical Properties

Molecular Weight: 174.6

Melting Point: 37-47°C (Miller et al. 1976)

Solubility in Water: 1,200 mg/liter (estimated; Lyman et al. 1982)
1,050 mg/liter (estimated; Lyman et al. 1982)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Vapor Pressure: 8×10^{-4} mm Hg at 25°C (estimated; Lyman et al. 1982)
2.5 mm Hg at 131.5°C (Miller et al. 1976)

Henry's Law Constant: 1.5×10^{-7} atm-m³/mole (calculated)

Log Octanol/Water Partition Coefficient (K_{ow}): 1.33 (Brueggemann 1982)
1.26 (Lyman et al. 1982) Fragment Method

Soil/Water Partition Coefficient (K_{oc}):

115	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.26$)
27.6	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.26$)
22.3	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.26$)
23.2	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.26$)
50.7	Kadeg et al. (1986) ($\log K_{ow} = 1.26$)

Bioconcentration Factor:

5.88	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.26$)
8.71	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.26$)
5.34	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.26$)

Transport and Fate

The low vapor pressure of PCPMSO indicates that volatilization (evaporation) from soil/water surfaces is not likely to be significant for this chemical. PCPMSO is low to moderately soluble in water (Cogley and Foy 1978). A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of PCPMSO to soils/sediments and dissolved organic material may occur. The combined water solubility and low organic partitioning suggest that PCPMSO will exhibit some degree of environmental mobility.

Microbial degradation of PCPMSO reportedly occurs (Cogley and Foy 1978), however, the extent to which is utilized and the resulting degradation products were not reported. The persistence (half-life) of PCPMSO in soil may vary from six months to one year depending on ambient conditions (Cogley and Foy 1978). Guenzi et al. (1979)

reported that 84.5 percent of applied sulfoxide in soil (4.77 ug/g) incubated at 30°C was retained following 160 days. Uptake of sulfoxide in selected plants was also reported by Guenzi et al. Concentration factors in the tops and roots ranged from 16 and 5, respectively, in corn, to 66 and 6 in sugarbeets.

A range of estimated bioconcentration factors (BCFs) for is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of PCPMSO residues is not likely to occur.

Health Effects

No data on the toxicity of PCPMSO in humans was located in available literature. PCPMSO was nonmutagenic utilizing the Ames assay (Thake et al. 1979). Data on the carcinogenicity of PCPMSO was not available.

PCPMSO topical treatment produced mild skin irritation in rabbits and reversible lesions of the iris and conjunctival and corneal opacity when applied to the eye (Thake et al. 1979). Rats exposed subchronically (91 days) to PCPMSO in their food experienced reduced red blood cell counts and males experienced reduced SGOT levels (Thake et al. 1979). Rats at the highest treatment were emaciated and some mortalities occurred. Increased serum mineral levels and increased liver and kidney weights were also observed. Compound-related liver lesions were present in all dose groups. Post-mortem necropsies of sacrificed animals indicated incipient tumor formation.

Subacute oral toxicity studies (14 days) were conducted with dosages of 5, 10, and 20 mg/kg PCPMSO in Rhesus monkeys (Thake et al. 1979). The highest dose produced depression, anorexia, emesis, hypothermia, and weakness. Clinical signs were also observed at the

lowest dose. Decreased red blood cell count occurred at 20 mg/kg and increases in blood urea nitrogen (BUN), serum glutamate oxalate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and calcium occurred at the two higher dosages. Increased liver and kidney weights were also observed at these dosages. Lymphoid tissue hyperplasia was observed at all dosages.

Toxicity to Wildlife and Domestic Animals

No data on the toxicity of PCPMSO to wildlife or domestic animals was located in the available literature.

Regulations and Standards

None located.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For p-chlorophenyl methylsulfoxide (PCPMSO) the D_T value is derived from a Low-Observed-Effect-Level (LOEL) based on an oral subchronic toxicity study in mice and rats (Thake et al. 1979). Reduced red blood cell counts and increased liver and kidney weights were observed at all dose levels in the experiment. The LOEL identified from this study was 62 mg/kg/day. An Uncertainty Factor (UF) of 1,000 is employed in computing D_T to address extrapolation of the results to humans (10), intraspecies variability (sensitive subgroups) (10) and conversion from a subchronic to a lifetime (chronic) exposure period

(10). An additional Uncertainty Factor of 10 is included to address 1) the use of LOEL rather than a NOEL and 2) to address the potential carcinogenicity of PCPMSO. Derivation of this D_T value is as follows:

$$\begin{aligned} D_T &= \frac{\text{LOEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{62}{(1,000)(10)} \\ &= 0.0062 \text{ mg/kg/day} \end{aligned}$$

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CHROMIUM^{1/}

Summary

Chromium is a heavy metal that generally exists in either a trivalent or hexavalent oxidation state. Hexavalent chromium (Cr VI) is rather soluble and is quite mobile in groundwater and surface water. However, in the presence of reducing agents it is rapidly converted to trivalent chromium (Cr III), which is strongly adsorbed to soil components and consequently is much less mobile. A number of salts of hexavalent chromium are carcinogenic in rats. In addition, an increased incidence of lung cancer was seen in workers occupationally exposed to chromium VI. Hexavalent chromium also causes kidney damage in animals and humans. Trivalent chromium is less toxic than hexavalent chromium; its main effect is contact dermatitis in sensitive individuals.

CAS Number: 7440-47-3

Chemical Formula: Cr

IUPAC Name: Chromium

1/ Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Chemical and Physical Properties

Atomic Weight: 51.996

Boiling Point: 2672°C

Melting Point: 1857°C

Specific Gravity: 7.20 at 28°C

Solubility in Water: Insoluble; some compounds are soluble

Transport and Fate

Hexavalent Cr is quite soluble, existing in solution as a component of a complex anion. It is not sorbed to any significant degree by clays or hydrous metal oxides. The anionic form varies according to pH and may be a chromate, hydrochromate, or dichromate. Because all anionic forms are so soluble, they are quite mobile in the aquatic environment. Cr VI is efficiently removed by activated carbon and thus may have some affinity for organic materials in natural water or soil. Cr VI is a moderately strong oxidizing agent and reacts with reducing materials to form trivalent chromium. Most Cr III in the aquatic environment is hydrolyzed and precipitates as chromium hydroxide. Sorption to sediments will remove much of the remaining Cr III from solution. Cr III is adsorbed only weakly to inorganic materials. Cr III and Cr VI are readily interconvertible in nature depending on microenvironmental conditions such as pH, hardness, and the types of other compounds present. Soluble forms of chromium accumulate if ambient conditions favor Cr VI. Conditions favorable for conversion to Cr III lead to precipitation and adsorption of chromium in sediments.

In air, chromium is associated almost entirely with particulate matter. Sources of chromium in air include windblown soil and particulate emissions from industrial processes. Little information is

available concerning the relative amounts of Cr III and Cr VI in various aerosols. Relatively small particles can form stable aerosols and can be transported many miles before settling out.

Cr III tends to be adsorbed strongly onto clay particles and organic particulate matter, but can be mobilized if it is complexed with organic molecules. Cr III present in minerals is mobilized to different extents depending on the weatherability and solubility of the mineral in which it is contained. Hexavalent compounds are not strongly adsorbed by soil components and therefore are mobile in groundwater. Cr VI is quickly reduced to Cr III in poorly drained soils having a high content of organic matter. None of the plants normally used as food or animal feed are chromium accumulators. Chromium absorbed by plants tends to remain primarily in the roots and is poorly translocated to the leaves.

No data are available on the bioconcentration of Cr⁺³ by freshwater organisms (USEPA 1984a). In saltwater, bioconcentration factors in blue mussels (Mytilus edulis), soft-shell clams (Mya arenaria), and eastern oysters (Crassostrea virginica) were 86, 153, and 116, respectively (USEPA 1984a). In freshwater, rainbow trout (Salmo gairdneri) exhibited bioconcentration factors for Cr⁺⁶ ranging from one to three (USEPA 1984a). In saltwater bioconcentration factors for Cr⁺⁶ in the polychaete worm (Neanthes arenaceodentata), blue mussel, and eastern oyster were 236, 192, and 125, respectively (USEPA 1984a).

Health Effects

The hexavalent form of chromium is of major toxicological importance in higher organisms. A variety of chromate (Cr VI) salts are carcinogenic in rats following inhalation exposure. An excess of lung cancer has been observed among workers in the chromate-producing industry. Chromium has been classified in EPA's Group A, according to

EPA's Proposed Guidelines for Carinogen Risk Assessment, based upon positive inhalation data for Chromium VI in humans and animals (50 Federal Register 46967, Wed. Nov. 13, 1985).

Cr VI compounds can cause DNA and chromosome damage in animals and humans, and Cr (VI) trioxide is teratogenic in the hamster. Inhalation of hexavalent chromium salts causes irritation and inflammation of the nasal mucosa, and ulceration and perforation of the nasal septum. Cr VI also produces kidney damage in animals and humans. The liver is also sensitive to the toxic effects of hexavalent Cr, but apparently less so than the kidneys or respiratory system. Cr III is less toxic than Cr VI; its main effect in humans is a form of contact dermatitis in sensitive individuals (USEPA 1984b).

Toxicity to Wildlife and Domestic Animals

Chromium is an essential nutrient and is accumulated in a variety of aquatic and marine biota, especially benthic organisms, to levels much higher than in ambient water. Levels in biota, however, usually are lower than levels in the sediments. Passage of chromium through the food chain can be demonstrated. The food chain appears to be a more efficient pathway for chromium uptake than direct uptake from seawater. Water hardness, temperature, dissolved oxygen, species, and age of the test organism all modify the toxic effects of chromium on aquatic life. Cr III appears to be more acutely toxic to fish than CR VI; the reverse is true in long term chronic exposure studies. There is little tendency for chromium to accumulate along food chains in the trivalent inorganic form. Organic chromium compounds, about which little is known, can have significantly different bioaccumulation tendencies. Little information concerning the toxic effects of chromium on mammalian wildlife and domestic animal species is available.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986a):

CR VI:

Aquatic Life (Freshwater)

Acute toxicity: 16 $\mu\text{g/liter}$

Chronic toxicity: 11 $\mu\text{g/liter}$

Aquatic Life (Saltwater)

Acute toxicity: 1,100 $\mu\text{g/liter}$

Chronic toxicity: 50 $\mu\text{g/liter}$

Human Health

Criterion: 50 $\mu\text{g/liter}$

CR III:

Aquatic Life (Freshwater)

Acute toxicity: $e^{(0.819 [\ln(\text{hardness})] + 3.568)}$

For hardness levels of 50, 100 and 200 mg/l CaCO_3 , the criteria are 980, 1,700, and 3,100 $\mu\text{g/liter}$.

Chronic toxicity: $e^{(0.819 (\ln(\text{hardness})) + 0.537)}$

For hardness levels of 50, 100 or 200 mg/l CaCO_3 , the criteria are 120, 210 and 370 $\mu\text{g/liter}$.

Aquatic Life (Saltwater)

The available data are not adequate for establishing criteria.

Human Health

Criterion: 170 mg/liter

CAG Potency Slope for CR VI (inhalation) (USEPA 1984c): $41 \text{ (mg/kg/day)}^{-1}$

National Primary Drinking Water Standard (USEPA): 120 $\mu\text{g/liter}$

Proposed RMCL; 50 Federal Register 46967, Wednesday, November 13, 1985

NIOSH Recommended Standard (air): 1 $\mu\text{g/m}^3$ carcinogenic
TWA^{1/} = 25 $\mu\text{g/m}^3$ noncarcinogenic
OSHA Standard: TWA = 50 $\mu\text{g/m}^3$ (15 min)
Ceiling = 100 $\mu\text{g/m}^3$

ACGIH Threshold Limit Values: Several chromium compounds have TWAs ranging from 0.05 to 0.5 mg/m^3 . Chromite ore processing (chromate), certain water insoluble CR VI compounds, and chromates of lead and zinc are recognized or suspected human carcinogens and have 0.04 mg/m^3 TWAs.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

D_T values will be developed for the two valence states of chromium: trivalent (III) and hexavalent (VI). For chromium (III), the D_T value is based on the same data used by EPA to compute the current Risk Reference Dose (RfD) (USEPA 1986b). The RfD is based on a chronic oral toxicity study utilizing male and female rats fed chromic oxide (0, 1, 2 or 5 percent) 5 days/week for 840 days (Ivankovic and

^{1/} Time Weighted Average

Preussmann 1975).~ Body weight and food consumption were monitored and all major organs were examined histologically. No effects were noted at any dose levels. The identified No-Observed-Effect-Level (NOEL) in this study was 5 percent chromic oxide in the diet (1,468 mg/kg/day). An Uncertainty Factor (UF) of 100 is employed to address extrapolation of the results to humans (10) and intraspecies variability (sensitive subgroups) (10). A Modifying Factor (MF) of 10 is used to address the following: 1) the possibility that the NOEL may be a Low-Observed-Effect-Level (LOEL) as indicated by a subchronic study by the same author; 2) the low absorption of chromium and its dependence on various factors, and 3) histology was performed only after the animals died a natural death following termination of the experiment. Derivation of the D_T (RfD) is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOEL (mg/kg/day)}}{\text{UF} \times \text{MF}} \\ &= \frac{1,468}{100 \times 10} \\ &= 1.468 \quad [\text{Note: EPA has rounded this number in deriving the final RfD of 1 mg/kg/day}] \\ &= 1 \text{ mg/kg/day} \end{aligned}$$

Chromium VI has been shown to be carcinogenic following inhalation, but not oral exposures (50 Federal Register 46967, Wednesday, November 13, 1985). EPA has developed a cancer potency slope to address cancer risks from chromium VI inhalation exposure. However, use of the inhalation slope is not considered appropriate for assessing exposure to chromium VI inhaled on suspended soil particulates because it has been developed from industrial emission data which are applicable to particulates smaller than 10 microns. Additionally, a substantial portion of larger inhaled particulates (e.g., wind entrained soil particles) are eventually swallowed as a result of mucocilliary transport processes ongoing in the respiratory tract.

For chromium VI oral exposures, the D_T is derived from the same data used by EPA to develop a Recommended Maximum Contaminant Level (RMCL) for chromium (50 Federal Register 46966 Wednesday, November 13,

1985). For chromium VI, the RMCL is based on a chronic (1 year) study in which rats were supplied water containing up to 25 mg/liter chromium VI (MacKenzie 1958). A No-Observed-Adverse- Effect-Level (NOAEL) of 2.41 mg/liter was identified from this study. An Uncertainty Factor of 500 is included by EPA to address extrapolation of the results to humans (10), intraspecies variability (sensitive subgroups) (10) and an additional factor of 5 because the rats were exposed for only 40 percent of a normal life span. Derivation of the oral D_T for chromium VI is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOAEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{2.41}{500} \\ &= 0.00482 \text{ mg/kg/day} \end{aligned}$$

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COPPER^{1/}

Summary

Copper is a mobile metal in the environment. Copper exists in a valence state of +1 or +2 and is found in nature as sulfide, oxide, or carbonate ore. It is toxic to humans at high levels and causes irritation following acute exposure and anemia following chronic exposure. Copper does not have teratogenic, mutagenic or carcinogenic effects in animals or humans. Sheep are very susceptible to copper toxicosis through the administration of worming medications. Copper appears to be toxic to some aquatic organisms at low water concentrations.

CAS Number: 7440-50-8

Chemical Formula: Cu

IUPAC Name: Copper

Chemical and Physical Properties

Atomic Weight: 63.546

Boiling Point: 2,567°C

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Melting Point: ~ 1,083°C

Specific Gravity: 8.92

Solubility in Water: Most copper salts are insoluble, with the exception of CuSO_4 , $\text{Cu}(\text{NO}_3)_2$, and CuCl_2 (the more common copper salts). The metal is insoluble in water.

Vapor Pressure: 1 mm Hg at 1,628°C

Transport and Fate

Copper has two oxidation states, +1 (cuprous) and +2 (cupric). Cuprous copper is unstable in aerated water over the pH range of most natural waters (6 to 8) and oxidizes to the cupric state. Several processes determine the fate of copper in the aquatic environment: formation of complexes, especially with humic substances; sorption to hydrous metal oxides, clays, and organic materials; and bioaccumulation. In waters polluted with soluble organic material, complexation with organic ligands can occur, thus favoring the prolonged dispersion of copper in solution. The presence of organic acids also can lead to the mobilization of copper from the sediments to solution.

Copper has a strong affinity for hydrous iron and manganese oxides, clays, carbonate minerals, and organic matter. Sorption to these materials, both suspended in the water column and in the sediment, results in relative enrichment of the solid phase and reduction in dissolved level. Sorption processes are quite efficient in scavenging dissolved copper and in controlling its mobility in natural unpolluted streams. The amounts of the various copper compounds and complexes that actually exist in solution depend on the pH, temperature, alkalinity, and concentration of other chemical species. The levels of copper able to remain in solution are directly dependent on water chemistry. Generally, ionic copper is more soluble in low pH waters and less soluble in high pH waters.

As an essential nutrient, copper is accumulated by plants and animals, although it is not generally biomagnified. Because copper is strongly bioaccumulated and because biogenic ligands play an important role in complexing copper, biological activity is a major factor in determining the distribution and occurrence of copper in the ecosystem.

Bioconcentration factors in freshwater species range from zero for the bluegill to 2,000 for the alga Chlorella regularis. Among saltwater species, the highest bioaccumulation factors are those for the bivalve molluscs. Oysters can bioaccumulate copper up to 28,200 times without any significant mortality.

Because many copper compounds and complexes are readily soluble, copper is among the more mobile heavy metals in soil and other surface environments. The major process that limits the environmental mobility of copper is adsorption to organic matter, clays, and other materials. Atmospheric transport of copper compounds can also occur.

Health Effects

Copper appears to increase the mutagenic activity of triose reductone and ascorbic acid in bacterial test systems. However, copper itself does not appear to have mutagenic, teratogenic, or carcinogenic effects in animals or humans. Copper has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate evidence of carcinogenicity in both animals and humans (50 Federal Register 46968, Wed. Nov. 13, 1985).

Dietary levels of trace elements such as molybdenum, sulfur, zinc, and iron can affect the level of copper that produces certain deficiency or toxicity symptoms. In general, more attention is given to the problems associated with copper deficiency than to problems of excess copper in the environment. However, high levels of copper can be toxic to humans.

Exposure to metallic copper dust can cause a short-term illness similar to metal fume fever that is characterized by chills, fever, aching muscles, dryness of mouth and throat, and headache. Exposure to copper fumes can produce upper respiratory tract irritation, a metallic or sweet taste, nausea, metal fume fever, and sometimes discoloration of skin and hair. Individuals exposed to dusts and mists of copper salts may exhibit congestion of nasal mucous membranes, sometimes of the pharynx, and occasionally ulceration with perforation of the nasal septum.

If sufficient concentrations of copper salts reach the gastrointestinal tract, they act as irritants and can produce salivation, nausea, vomiting, gastritis, and diarrhea. Elimination of ingested ionic copper by vomiting and diarrhea generally protects the patient from more serious systemic toxic effects, which can include hemolysis, hepatic necrosis, gastrointestinal bleeding, oliguria, azotemia, hemoglobinuria, hematuria, proteinuria, hypotension, tachycardia, convulsions, and death. Chronic exposure may result in anemia.

Copper salts act as skin irritants producing an itching eczema. Conjunctivitis or even ulceration and turbidity of the cornea may result from direct contact of ionic copper with the eye.

Toxicity to Wildlife and Domestic Animals

Mean acute toxicity values for a large number of freshwater animals range from 7.2 $\mu\text{g/liter}$ for Daphnia pulicaria to 10,200 $\mu\text{g/liter}$ for the bluegill (USEPA 1980). Toxicity tends to decrease as hardness, alkalinity, and total organic carbon increase. Chronic values for a variety of freshwater species range from 3.9 $\mu\text{g/liter}$ for brook trout to 60.4 $\mu\text{g/liter}$ for northern pike (USEPA 1980). Hardness does not appear to affect chronic toxicity. The acute-chronic ratios for different species range from 3 to 156 (USEPA 1980). The more sensitive species tend to have lower ratios than the less sensitive species. In

addition, the ratio seems to increase with hardness. Acute toxicity values for saltwater organisms range from 17 $\mu\text{g/liter}$ for a calanoid copepod to 600 $\mu\text{g/liter}$ for the shore crab (USEPA 1980). A chronic value of 54 $\mu\text{g/liter}$ and an acute-chronic ratio of 3.4 is reported for the mysid shrimp (USEPA 1980). Long-term exposure to 5 $\mu\text{g/liter}$ is fatal to the bay scallop.

Sheep are very susceptible to acute or chronic copper toxicosis (Bostwick 1982). Acute poisoning is caused by direct action of copper salts on the gastrointestinal tract, resulting in gastroenteritis, shock, and death. The toxic dose is about 200 $\mu\text{g/kg}$ and is usually obtained through an accidental overdose of an antihelminthic. Chronic ingestion of copper over a long period of time results in absorption and accumulation of copper by the liver. This type of chronic cumulative poisoning may suddenly develop into an acute hemolytic crisis. Copper intake of 1.5 g/day for 30 days is known to be fatal for many breeds of sheep. Excessive copper may be stored in the liver as a result of excess copper ingestion, as a consequence of impaired liver function or in connection with a deficiency or excess of other trace elements. Sheep eliminate accumulated copper very slowly following cessation of exposure.

Swine may develop copper poisoning at levels of 250 mg/kg in the diet unless zinc and iron levels are increased. Toxicosis develops with hypochromic microcytic anemia, jaundice and marked increases in liver and serum copper levels as well as serum aspartate amino transferase levels. High copper levels may be found in swine because of the practice of feeding them high copper diets to increase daily weight gain. However, swine rapidly eliminate copper once it is removed from the diet. Cattle are more resistant to copper in the diet than sheep or swine. Copper toxicity in ruminants can be counteracted by including molybdenum and sulfate in the diet.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

Aquatic Life (Freshwater)

Acute toxicity: $e^{(0.905 [\ln(\text{hardness})] - 1.413)}$ $\mu\text{g/liter}$

At hardnesses of 50, 100 or 200 mg/l CaCO_3 the acute criteria are 9.2, 18 and 34 $\mu\text{g/liter}$.

Chronic toxicity: $e^{(0.905 [\ln(\text{hardness})] - 1.785)}$ $\mu\text{g/liter}$

At hardnesses of 50, 100 or 200 mg/l CaCO_3 the chronic criteria are 6.5, 12 and 21 $\mu\text{g/liter}$.

Aquatic Life (Saltwater)

Acute toxicity: 2.9 $\mu\text{g/liter}$

Chronic toxicity: 2.9 $\mu\text{g/liter}$

Human Health

Organoleptic criterion: 1 mg/liter

National Primary Drinking Water Standards (USEPA): 1.3 mg/liter

Proposed RMCL; 50 Federal Register 46968, Wednesday, November 13, 1985

OSHA Standards: $\text{TWA}^{\underline{1/}} = 1.0 \text{ mg/m}^3$ (dust and mist)

$\text{TWA} = 0.1 \text{ mg/m}^3$ (fume)

ACGIH Threshold Limit Value: $\text{TWA} = 1.0 \text{ mg/m}^3$ (dust and mists)

$\text{TWA} = 0.2 \text{ mg/m}^3$ (fume)

$\text{STEL}^{\underline{2/}} = 2.0 \text{ mg/m}^3$ (dusts and mists)

1/ Time Weighted Average.

2/ Short Term Effect Level.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For copper, the D_T value is derived from the data used to establish an EPA proposed Drinking Water Recommended Maximum Contaminant Level (RMCL). The RMCL is typically based on a No-Observed-Adverse-Effect-Level (NOAEL) or Lowest-Observed-Adverse-Effect Levels (LOAEL) derived from subchronic or chronic animal or human toxicity studies including an Uncertainty Factor (UF). For copper, the RMCL is based on acute human clinical case studies (Chuttani 1965) in which 5.3 mg copper was the lowest oral dose at which gastrointestinal effects were observed. In developing criteria levels, EPA uses an Uncertainty Factor (UF) of two because 1) effects were localized and not permanent, 2) 5.3 mg was the lowest identified dosage in numerous studies and is considered to be conservative, 3) copper is an essential element and therefore application of a large UF would bring the level below that considered necessary for human nutrition and 4) copper absorption is controlled by a homeostatic mechanism and the compound does not tend to accumulate in the body (50 Federal Register 46968, Wednesday November 13, 1985). Derivation of the D_T value for copper is as follows:

$$\begin{aligned} D_T &= \frac{\text{LOAEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{0.0757}{2} \\ &= 0.038 \text{ mg/kg/day} \end{aligned}$$

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Summary

DDT is an organochlorine pesticide, which together with its metabolites, is very persistent in the environment. DDT, DDE, and DDD have been shown to be carcinogenic in mice, causing liver tumors, but also increasing the incidence of lung tumors and lymphomas. Chronic exposure can damage the central nervous system and liver. In addition, DDT is a reproductive toxin. DDT and other organochlorine pesticides are highly toxic to aquatic organisms and readily bioconcentrate in their tissues. Bioaccumulation and subsequent biomagnification processes are responsible for the decreased reproductive success of many bird species.

Technical DDT is a mixture containing 65-80 percent p, p'-DDT, 15-20 percent o, p'-DDT, up to 4 percent p, p'-DDD, and traces of other materials. Metabolites of DDT include p, p'-DDE and o,p'-DDD. The DDT isomers and metabolites are usually found together and generally have similar properties; therefore, they are considered together. Where differences occur, the specific isomer is identified. DDT is used to refer to the combination of technical material and metabolites. Specific DDT isomers are identified as such.

1/ Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

CAS Number: p,p'-DDT: 50-29-3
o,p'-DDT: 789-02-6
p,p'-DDD: 72-54-8
o,p'-DDD: 53-19-0
p,p'-DDE: 72-55-9

Chemical Formula: p,p'- and o,p'-DDT: $C_{14}H_9Cl_5$
p,p'- and o,p'-DDD: $C_{13}H_{10}Cl_4$
p,p'- and o,p'-DDE: $C_{14}H_8Cl_4$

IUPAC Name: p,p'-DDT: 1,1,1-Trichloro-2,2-bis(4-chlorophenyl) ethane
o,p'-DDT: 1,1,1-Trichloro-2-(2-chlorophenyl)-2-(4-chlorophenyl)ethane
p,p'-DDD: 1,1-Dichloro-2,2-bis(4-chlorophenyl)ethane
o,p'-DDE: 1,1-Dichloro-2,2-bis(4-chlorophenyl)ethane

Important Synonyms and Trade Names:

DDT: Dichlorodiphenyltrichloroethane, dicophane,
chlorophenotane, Gesarol, Neocid
p,p'-DDD: TDE, Rothane

Chemical and Physical Properties

Molecular Weight: o,p'- and p,p'-DDT: 354.5
DDD: 320
DDE: 318

Boiling Point: DDT: 260°C

Melting Point: DDT: 109°C
DDD: 112°C
DDE: 90°C
88.4°C (Burrows 1979)

Solubility in Water: p,p'-DDT: 5.5 µg/liter
o,p'-DDT: 26 µg/liter
p,p'-DDD: 20 µg/liter
DDE: 14 µg/liter

Solubility in Organics: DDT: Soluble in acetone, benzene,
cyclohexanane, morpholine, pyridine, and
dioxane

Log Octanol/Water Partition Coefficient (K_{ow}):

DDT: 4.98
3.98-6.19 (Cited in Hansch and Leo 1979)
5.98 (Kenaga 1980)
6.19 (Rao and Davidson 1983)
6.36 (Davies and Dobbs 1984)
6.07 (Kadeg et al. 1986)
p,p'-DDT: 3.98
p,p'-DDD: 5.99
o,p'-DDD: 6.08
DDE: 5.69 (Rao and Davidson 1983)
5.60 (Kadeg et al. 1986)
7.07 (Lyman et al. 1982) Fragment Method

Soil/Water Partition coefficient (K_{oc}):

p,p'-DDE:

50,100	Sabljic (1984) (experimental)
3,750	Lyman et al. (1982) Eqn 4-5 ($S = .12$)
30,000 - 174,000	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 5.7 - 7.1$)
66,400; 279,000	Lyman and Loreti (1986) Eqn I ($\log K_{ow} 5.6, 6.4$)
92,800; 431,000	Lyman and Loreti (1986) Eqn II ($\log K_{ow} 5.6, 6.4$)
87,600; 400,000	Lyman and Loreti (1986) Eqn III ($\log K_{ow} 5.6, 6.4$)
65,600; 245,900	Kadeg et al. (1986) ($\log K_{ow} = 5.6, 6.4$)
147,900	Kadeg et al. (1986) literature value

p,p'-DDT:

33,200	Lyman et al. (1982) Eqn 4-5 (S = .025)
23,800	Kenaga (1980) Table III (experimental)
3,570 - 72,210	Lyman et al. (1982) Eqn 4-8 (log K _{OW} = 4.0 - 6.4)
140,000	Chiou et al. (1979) (experimental)
302,000	Kadeg et al. (1986) (geo. mean 17 literature values)
243,000	Rao and Davidson (1983) Table I
3,770; 154,400; 279,000	Lyman and Loreti (1986) Eqn I (log K _{OW} = 4.0, 6.07, 6.4)
4,300; 228,800; 431,100	Lyman and Loreti (1986) Eqn II (log K _{OW} = 4.0, 6.07, 6.4)
4,210; 213,600; 399,600	Lyman and Loreti (1986) Eqn II (log K _{OW} = 4.0, 6.07, 6.4)
4,680; 142,600; 245,900	Kadeg et al. (1986) (log K _{OW} = 4.0, 6.07, 6.4)

Bioconcentration Factor:

p,p'-DDE:

13,900	Lyman et al. (1982) Eqn 5-2 (log K _{OW} = 7.07)
12,430	Lyman et al. (1982) Eqn 5-2 (log K _{OW} = 5.69)
2,043	Davies and Dobbs (1984) Eqn A (S = 0.12)
25,362	Davies and Dobbs (1984) Eqn B (log K _{OW} = 7.07)
980	Davies and Dobbs (1984) Eqn C (log K _{OW} = 5.60)
3,400	Davies and Dobbs (1984) Eqn B (log K _{OW} = 5.60)
10,600	Lyman et al. (1982) Eqn 5-2 (log K _{OW} = 5.60)
100,000	Davies and Dobbs (1984) Table 2 (experimental)
366 - 9,659	Davies and Dobbs (1984) Eqn B (log K _{OW} = 3.98 - 6.36)

p,p'-DDT:

61,600; 84,500	Kenaga (1980) Table 3 (experimental)
623 - 29,800	Lyman et al. (1982) Eqn 5-2 (log K _{OW} = 3.98 - 6.19)
20,600	Lyman et al. (1982) Eqn 5-2 (log K _{OW} = 5.98)
40,100	Lyman et al. (1982) Eqn 5-2 (log K _{OW} = 6.36)
27,436 - 13,913	Davies and Dobbs (1984) Eqn A (S = 0.0012 - 0.004)
1,710	Davies and Dobbs (1984) Eqn C (log K _{OW} = 6.07)
6,483	Davies and Dobbs (1984) Eqn B (log K _{OW} = 6.07)
24,200	Lyman et al. (1982) Eqn 5-2 (log K _{OW} = 6.07)

Vapor Pressure:

p,p'-DDT:	1.9×10^{-7}	mm Hg at 25°C
p,p'-DDT:	7.3×10^{-7}	mm Hg at 30°C
o,p'-DDT:	5.5×10^{-6}	mm Hg at 30°C
p,p'-DDD:	1.0×10^{-6}	mm Hg at 30°C
o,p'-DDD:	1.9×10^{-6}	mm Hg at 30°C
DDE:	6.5×10^{-6}	mm Hg at 20°C (USEPA 1979)

Henry's Law Constant:	DDD:	7.96×10^{-6}	atm-m ³ /mole (USEPA 1985)
	DDE:	1.1×10^{-4}	atm-m ³ /mole (calculated)
		6.8×10^{-5}	atm-m ³ /mole (USEPA 1985)
	DDT:	9×10^{-5}	atm-m ³ /mole (calculated)
		5.13×10^{-4}	atm-m ³ /mole (USEPA 1985)

Transport and Fate

DDT and its metabolites are very persistent in the environment. Volatilization is not likely to be an important transport process from soil and water for DDT and its metabolites as evidenced by their low vapor pressures. The half-life of DDT in the atmosphere is not certain, however it is lost from the atmosphere by rain and photochemical degradation (USEPA 1984).

The range of the soil-water partition coefficients (K_{oc}) reported above indicates that sorption of DDT and its metabolites to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of chlorinated hydrocarbon pesticides is very high. The combined low water solubility and high organic partitioning suggest that DDT will exhibit little environmental mobility. The half-life of DDT in soil is estimated to range between 3 and 15 years (USEPA 1984).

Although it occurs slowly, p,p'-DDT, o,p'-DDT, and DDD are ultimately biotransformed in the environment (microorganisms) to form bis(2-chlorophenyl) methanone (DDCO). In aquatic environments,

indirect photolysis may also be important for p,p'-DDT and o,p'-DDT. For DDE, direct photolysis is a more important fate process in the environment, although biotransformation may also be important.

A range of experimental and estimated bioconcentration factors (BCFs) for DDT and its metabolites in fish is reported above. Biomagnification of DDT and its metabolites has been demonstrated in many species, most notably in raptors. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors indicates that significant bioconcentration and biomagnification of DDT residues can occur.

Health Effects

DDT, DDE, and DDD have been shown to be carcinogenic in mice, causing liver tumors, lung tumors and lymphomas. DDT and its isomers have been classified according to EPA's Proposed Guidelines for Carcinogen Risk Assessment in EPA's Group B2 (probable human carcinogen), based on inadequate evidence for carcinogenicity in humans and sufficient evidence in animals (USEPA 1984). DDT does not appear to be mutagenic, but it has caused chromosomal damage in some studies. The National Toxicology Program (1986) reports positive results for mutagenic activity with DDE utilizing mouse lymphoma cells in recently completed tests. There is no evidence that DDT is a teratogen. However, it is a reproductive toxin, causing reduced fertility, reduced growth of offspring, and fetal toxicity in rats (NIOSH 1983).

Chronic exposure to DDT leads to a number of adverse effects of the liver and central nervous system (CNS). DDT induces various microsomal enzymes and therefore may affect the metabolism of steroid hormones and exogenous chemicals. Other effects on the liver include hypertrophy of the parenchymal cells and increased fat deposition. In the CNS, exposure to DDT causes behavioral effects such as decreased aggression and decreased conditional reflexes. Acute exposure to large doses or

chronic exposure to lower doses causes seizures. The oral LD₅₀ is between 113 and 450 mg/kg for the rat and is generally higher for other animals.

Toxicity to Wildlife and Domestic Animals

DDT has been extensively studied in freshwater invertebrates and fishes and is quite toxic to most species. The range of toxicities to these organisms was 0.18 to 1,800 µg/liter and the freshwater final acute value for DDT and its isomers was determined by EPA to be 1.1 µg/liter (USEPA 1980). Saltwater species were somewhat more sensitive to DDT. The saltwater final acute value for the DDT isomers was 0.13 µg/liter (USEPA 1980). Only one chronic toxicity test on aquatic species was reported. This test indicated that the acute-chronic ratio for DDT may be high (65 in the reported study), but the data were insufficient to allow calculation of a final acute-chronic ratio.

DDT, DDD, DDE and other persistent organochlorine pesticides are primarily responsible for decreases in the reproductive capabilities and consequently in the populations of some fish-eating birds; particularly the bald eagle, brown pelican, and osprey. DDT has also been shown to significantly decrease populations of other species of waterbirds, raptors, and passerines (EOP 1971).

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

Aquatic Life (Freshwater)

DDT:

Acute toxicity: 1.1 µg/liter

Chronic toxicity: 0.001 µg/liter

Aquatic Life (Saltwater)

Acute toxicity: 0.13 $\mu\text{g/liter}$

Chronic toxicity: 0.001 $\mu\text{g/liter}$

DDD and DDE: The available data are not adequate for establishing Ambient Water Quality Criteria. However, EPA does report the lowest values known to be toxic in aquatic organisms.

Aquatic Life (Freshwater)

Acute toxicity: DDD: 0.06 $\mu\text{g/liter}$

DDE: 1050 $\mu\text{g/liter}$

Chronic toxicity: DDD and DDE: No available data

Aquatic Life (Saltwater)

Acute toxicity: DDD: 3.6 $\mu\text{g/liter}$

DDE: 14 $\mu\text{g/liter}$

Chronic toxicity: DDD and DDE: No available data

Human Health

Due to the carcinogenicity of DDT and its isomers the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from ingestion of contaminated water and contaminated aquatic organisms are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	0.24 ng/liter
10^{-6}	0.024 ng/liter
10^{-7}	0.0024 ng/liter

CAG Potency Slope for Oral Exposure (USEPA 1984): $0.34 (\text{mg/kg/day})^{-1}$

OSHA Standard (air): $TWA^{1/} = 1 \text{ mg/m}^3$

ACGIH Threshold Limit Value: $TWA = 1 \text{ mg/m}^3$

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For carcinogens such as DDT and its isomers, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10⁻⁴ to 10⁻⁷ is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values for DDT and its isomers is as follows:

$$\begin{aligned} D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\ &= \frac{1 \times 10^{-4}}{0.34 \text{ (mg/kg/day)}^{-1}} \\ &= 2.9 \times 10^{-4} \text{ mg/kg/day} \end{aligned}$$

The range of D_T values presented for DDT is also used for DDE and DDD as the potencies of these compounds are the same. The range is presented below:

1/ Time Weighted Average

<u>Risk Level</u>	<u>D_T (mg/kg/day)</u>
10 ⁻⁴	2.9 x 10 ⁻⁴
10 ⁻⁵	2.9 x 10 ⁻⁵
10 ⁻⁶	2.9 x 10 ⁻⁶
10 ⁻⁷	2.9 x 10 ⁻⁷

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DIBROMOCHLOROPROPANE^{1/}

Summary

Dibromochloropropane (DBCP) is a persistent and environmentally mobile pesticide. Formerly, DBCP was used as a soil fumigant and nematocide. It is carcinogenic in mice and rats and mutagenic in bacterial systems and mammalian cell cultures. It causes mammary tumors (female rats) and forestomach tumors when administered orally. When administered via inhalation, it causes nasal, tongue, and lung tumors. Men occupationally exposed to DBCP exhibit abnormally low sperm counts. Animal studies have shown that dibromochloropropane has adverse effects on the testes, liver, kidneys, respiratory tract, central nervous system, and blood cells.

CAS Number: 96-12-8

Chemical Formula: $C_3H_5Br_2Cl$

IUPAC Name: 1,2-Dibromo-3-chloropropane

Important Synonyms and Trade Names; DBCP, Fumazone, Nemagon

Chemical and Physical Properties

Molecular Weight: 236.36

1/ Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Boiling Point: 196°C (Berkowitz et al. 1978)

Melting Point: 6°C
5°C (Berkowitz et al. 1978)

Specific Gravity: 2.093 at 14°C

Solubility in Water: 1230 mg/liter (USEPA 1985a)

Solubility in Organics: Miscible with oils, dichloropropane, and
isopropyl alcohol

Log Octanol/Water Partition Coefficient (K_{ow}): 2.29 (Lyman et al. 1982)
Fragment Method
2.43 (USEPA 1985a)

Soil/Water Partition Coefficient (K_{oc}):

130	Sabljić (1984) Table I (experimental)
87	Lyman et al. (1982) Eqn 4-5 ($S = 1,230$)
420	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 2.29$)
500	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 2.43$)
175	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 2.29$)
161	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 2.29$)
164	Lyman and Loreti (1986) Eqn III ($\log K_{ow} =$ 2.29)
225	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 2.43$)
211	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 2.43$)
214	Lyman and Loreti (1986) Eqn III ($\log K_{ow} =$ 2.43)
278	Kadeg et al. (1986) ($\log K_{ow} = 2.29$)
350	Kadeg et al. (1986) ($\log K_{ow} = 2.43$)

Bioconcentration Factor:

41.4	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.43$)
67.5	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.71$)
11.2	Davies and Dobbs (1984) Eqn A ($S = 1,230$)
63	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.71$)
43.5	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.43$)
35.9	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 2.29$)
19.8	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 2.29$)
27.6	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 2.43$)
32.4	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 2.29$)

Vapor Pressure: 0.8 mm Hg at 21°C (USEPA 1985a)
1.1 mm Hg at 25°C (estimated; Lyman et al. 1982)

Henry's Law Constant: 3.5×10^{-4} atm-m³/mole at 20°C (Burlinson
et al. 1982)
 3.11×10^{-4} atm-m³/mole (USEPA 1985b)

Transport and Fate

Dibromochloropropane (DBCP) is a persistent pesticide. The major route of its removal from soil and aqueous systems is by volatilization. DBCP is decomposed slowly in soil both by microbial action and by hydrolysis (USEPA 1985). DBCP may be converted to n-propanol, bromide, and chloride by soil-water culture (Berkowitz et al. 1978). A range of estimated and experimental soil-water partition coefficients is reported above and indicates that sorption of DBCP to soils/sediments and dissolved organic material will occur. The combined water solubility and organic partitioning data for dibromochloropropane suggest that this compound will exhibit some degree of environmental mobility.

Plant uptake can occur with DBCP levels generally highest in the root portion. Bromide ion has also been shown to be present in increased levels in plants grown in DPCP-treated fields (Guinn and Potter 1962), and may be due to microbial or plant enzyme activity.

A range of estimated bioconcentration factors (BCFs) for DBCP is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of DBCP residues is not likely to occur.

cells were also noted in several studies. These effects include severe leukopenias and anemias in exposed monkeys and decreased activity of phagocytic cells in exposed rats (USEPA 1985c).

Toxicity to Wildlife and Domestic Animals

The acute oral LD₅₀ value of DBCP to female mallard ducks is 66.8 mg/kg and 156 mg/kg in female pheasants. Both avian LD₅₀ values are lower than the acute oral LD₅₀ value of the rat (400 mg/kg) and indicate an increased sensitivity of these animals. Exposure to a water concentration of 1 mg/liter DBCP for 24 hours produced a 90 percent mortality in clam larvae. At a use concentration of 20 gallons DBCP per acre, 100 percent of exposed earthworms died in 1 day. At a use rate of 5 pounds per acre, DBCP killed 87 percent of the Lumbricus and 28 percent of the Helodrilus sp. in 32 days.

Regulations and Standards

NIOSH Recommended Standard: 10 ppb (0.00 mg/m³)

National Primary Drinking Water Standard (USEPA): Zero
(Proposed RMCL; 50 Federal Register 46988, Wednesday, November 13, 1985).

OSHA Standard (air): TWA^{1/}: 1 ppb (9.6 µg/m³)

CAG Potency Slope for Oral Exposure (USEPA 1985c): 1.4 (mg/kg/day⁻¹)

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

^{1/} Time Weighted Average.

For carcinogens such as DBCP, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10^{-4} to 10^{-7} is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values is as follows:

$$\begin{aligned}
 D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\
 &= \frac{1 \times 10^{-4}}{1.4 \text{ (mg/kg/day)}^{-1}} \\
 &= 7.1 \times 10^{-5} \text{ (mg/kg/day)}
 \end{aligned}$$

The range of D_T values for DBCP is presented below:

<u>Risk Level</u>	<u>D_T (mg/kg/day)</u>
10^{-4}	7.1×10^{-5}
10^{-5}	7.1×10^{-6}
10^{-6}	7.1×10^{-7}
10^{-7}	7.1×10^{-8}

REFERENCES

ASTM. 1985. Standard Practice for conducting bioconcentration tests with fishes and saltwater bivalve mollusca. Designation E 1022-84, pages 590-62. In: 1985 Annual Book of ASTM Standards Volume 11.04. American Society for Testing and Materials, Philadelphia, Pennsylvania.

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 &= 7.1 \times 10^{-5} \text{ (mg/kg/day)}
 \end{aligned}$$

The range of D_T values for DBCP is presented below:

<u>Risk Level</u>	<u>D_T (mg/kg/day)</u>
10^{-4}	7.1×10^{-5}
10^{-5}	7.1×10^{-6}
10^{-6}	7.1×10^{-7}
10^{-7}	7.1×10^{-8}

REFERENCES

ASTM. 1985. Standard Practice for conducting bioconcentration tests with fishes and saltwater bivalve mollusca. Designation E 1022-84, pages 590-62. In: 1985 Annual Book of ASTM Standards Volume 11.04. American Society for Testing and Materials, Philadelphia, Pennsylvania.

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1,1-DICHLOROETHANE^{1/}

Summary

1,1-Dichloroethane is volatile and therefore not likely to be persistent in aquatic environments. Inhalation exposure to high doses causes central nervous system depression and cardiac arrhythmia in humans and may cause hepatotoxicity. In animals, high doses cause liver and kidney damage and retarded fetal development.

CAS Number: 75-34-3

Chemical Formula: CH_3CHCl_2

IUPAC Name: 1,1-Dichloroethane

Important Synonyms and Trade Names; Ethylidene chloride; Ethylidene dichloride

Chemical and Physical Properties

Molecular Weight: 98.96

Boiling Point: 57.3°C

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Melting Point: -97°C

Specific Gravity: 1.1776 at 20°C

Solubility in Water: 5.5 g/liter (USEPA 1985)
8.1 g/liter (ECAO 1980)
8.45 g/liter (Chiou 1979)

Solubility in Organics: Miscible in alcohol

Log Octanol/Water Partition Coefficient (K_{ow}):
1.79 (USEPA 1985)
1.92 (Lyman et al. 1982)
Fragment Method

Soil-Water Partition Coefficient (K_{oc}):

40	Lyman et al. (1982) Eqn 4-5 ($S = 5,000$)
227	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.8$)
73	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.8$)
63	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.8$)
65	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.8$)
124	Kadeg et al. (1986) ($\log K_{ow} = 1.8$)

Bioconcentration Factor (BCF):

16.95	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.92$)
5.1	Davies and Dobbs (1984) Eqn A ($S = 5,000$)
2.3	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.9$)
11	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.8$)
18	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.8$)
14	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.8$)

Vapor Pressure: 180 mm Hg at 20°C (Valvani et al. 1980)
182 mm Hg at 20°C (USEPA 1985)

Henry's Law Constant: 6×10^{-4} atm-m³/mole (calculated)
 4.31×10^{-3} atm-m³/mole (USEPA 1985)

Transport and Fate

1,1-Dichloroethane disperses from surface water primarily by volatilization into the troposphere, where it is subsequently broken down by hydroxylation. The half-life of 1,1-dichloroethane in air is 1.5 months and in water the half-life is estimated to be 1-5 days (USEPA 1984).

A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of 1,1-dichloroethane to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and organic partitioning of 1,1-dichloroethane suggest that this compound will exhibit some degree of environmental mobility.

A range of estimated bioconcentration factors (BCFs) for 1,1-dichloroethane is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of 1,1-dichloroethane residues is not likely to occur.

Health Effects

Limited toxicological testing of 1,1-dichloroethane has been undertaken. The literature indicates that 1,1-dichloroethane is one of the least toxic of the chlorinated ethanes. A National Cancer Institute bioassay on 1,1-dichloroethane was limited by poor survival of test animals, but some marginal tumorigenic effects were seen. Inhalation exposure to high doses of 1,1-dichloroethane (over 16,000 mg/m³) caused retarded fetal development in rats (Schwetz et al. 1974). 1,1-Dichloroethane was not found to be mutagenic using the Ames assay. 1,1-Dichloroethane causes central nervous system depression

when inhaled at high concentrations, and evidence suggests that the compound is hepatotoxic in humans. Kidney and liver damage are seen in animals exposed to acutely high levels of 1,1-dichloroethane. The oral LD₅₀ value in the rat is 724 mg/kg.

Toxicity to Wildlife and Domestic Animals

No information on the toxicity of 1,1-dichloroethane to aquatic species was reported in the literature reviewed. However, the available information on chloroethanes indicates that toxicity declines with decreases in chlorination (USEPA 1980). Therefore, the toxicity of 1,1-dichloroethane is probably similar to that of 1,2-dichloroethane, which is acutely toxic at levels ranging from 100-500 mg/liter (USEPA 1980). Chronic toxicity occurs at levels as low as 20 mg/liter (USEPA 1980).

No information on the toxicity of 1,1-dichloroethane to terrestrial wildlife or domestic animals was found in the sources reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986).

The available data are inadequate for establishing criteria.

OSHA Standard (air): TWA^{1/} = 400 mg/m³

ACGIH Threshold Limit Value: TWA = 810 mg/m³

STEL^{2/} = 1,010 mg/m³

1/ Time Weighted Average.
2/ Short-Term Effect Level.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

The D_T value for 1,1-dichloroethane is based on the same data used by EPA to generate the acceptable subchronic intake value for oral exposure (USEPA 1984). The data are from a subchronic study (Hofmann et al. 1971) in which rats, cats, rabbits, and guinea pigs were exposed via inhalation to 2,025 mg/m³ (500 ppm) 1,1-dichloroethane 6 hours/day, 5 days/week. No effects were reported in any of the animals tested. EPA (USEPA 1984) used this data to estimate the No-Observed-Effect-Level (NOEL) in mg/kg/day as follows:

$$\begin{aligned} \text{NOEL} &= \frac{(2025 \text{ mg/m}^3)(0.22 \text{ m}^3/\text{day})(0.5)(6 \text{ hr}/24 \text{ hr})(5 \text{ days}/7 \text{ days})}{0.35 \text{ kg}} \\ &= 115 \text{ mg/kg/day} \end{aligned}$$

The value of 0.22 m³/day represents the default 24-hour rat breathing volume, 0.5 represents the assumed absorption coefficient, and 0.35 kg the default rat body weight.

An Uncertainty Factor (UF) of 1,000 is employed to address the extrapolation of results to humans (10), intraspecies variability (sensitive subgroups) (10), and the use of a subchronic rather than a chronic (lifetime) experimental study (10). Derivation of this D_T for 1,1-dichloroethane is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{115}{1,000} \\ &= 0.115 \text{ mg/kg/day} \end{aligned}$$

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1,2-DICHLOROETHANE^{1/}

Summary

1,2-Dichloroethane (ethylene dichloride) is a volatile organic solvent. Volatilization and leaching into groundwater may be significant routes of transport. Skin contact with 1,2-dichloroethane can cause dermatitis. 1,2-Dichloroethane is carcinogenic in animals and it is a suspected human carcinogen. It has produced positive results for mutagenicity in bacterial test systems.

CAS Number: 107-06-2

Chemical Formula: $\text{CH}_2\text{ClCH}_2\text{Cl}$

IUPAC Name: 1,2-Dichloroethane

Important Synonyms and Trade Names; Ethylene dichloride, glycol dichloride

Chemical and Physical Properties

Molecular Weight: 98.96

Boiling Point: 83-84°C

Melting Point: -35.4°C

1/ Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Specific Gravity: 1.253 at 20°C

Solubility in Water: 8.82 g/liter (Valvani et al., 1980)

8.7 g/liter at 20°C (TDB Peer Review Committee)

Solubility in Organics: Miscible with alcohol, chloroform, and ether

Log Octanol/Water Partition Coefficient (K_{ow}):

1.48	Valvani et al., 1980
1.79	Hansch and Leo 1979
1.45	Davies and Dobbs 1984

Soil-Water Partition Coefficient (K_{oc}):

19	Chiou et al. (1979) Fig. 2 (experimental)
0.1	Lyman et al. (1982) Eqn 4-5 ($S = 8.4$)
177	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.6$)
51	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.6$)
43	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.6$)
44	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.6$)
89	Kadeg et al. (1986) ($\log K_{ow} = 1.6$)

Bioconcentration Factor (BCF):

13.9	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.6$)
7.8; 13.5	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.48; 1.79$)
7.45	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.45$)
3.8	Davies and Dobbs (1984) Eqn A ($S = 8,417$)
2	Davies and Dobbs (1984) Table 2 (experimental)
8.8	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.6$)
9.7	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.6$)

Vapor Pressure: 61 mm Hg at 20°C

85 mm Hg at 25°C (USEPA 1980)

Flash Point: 15°C (closed cup)

Henry's Law Constant: 1.3×10^{-3} atm-m³/mole (calculated)

9.4×10^{-4} atm-m³/mole (Mabey et al. 1981)

Transport and Fate

The primary method of dispersion from surface water for 1,2-dichloroethane is by volatilization. In the atmosphere, 1,2-dichloroethane is rapidly broken down by hydroxylation, although

some may be absorbed by atmospheric moisture and returned with precipitation. In water the estimated half-life is 4 hours, assuming a wind speed of 3 m/sec, a water current of 1 m/sec and a depth of 1 meter (USEPA 1984).

A range of experimental and estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of 1,2-dichloroethane to soils/sediments and dissolved organic material may occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and low organic partitioning of 1,2-dichloroethane suggests that this compound will exhibit some degree of environmental mobility.

A range of estimated and experimental bioconcentration factors (BCFs) for 1,2-dichloroethane is also presented above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of residues is not likely to occur.

Health Effects

1,2-Dichloroethane has been shown to be carcinogenic in rats and mice (USEPA 1985). When administered by gavage, it produced carcinomas of the forestomach, lung adenomas (mice) and hemangiosarcomas of the circulatory system in male rats (USEPA 1985). Following oral administration of 1,2-DCE to female mice and rats numerous tumorigenic effects were seen including: lung adenomas and endometrial tumors (mice) and adenocarcinomas of the mammary gland (rats) (USEPA 1985). It has yielded positive mutagenicity results in tests with Salmonella (National Toxicology Program, 1985). 1,2-dichloroethane has been classified according to EPA's Proposed Guidelines for Carcinogenic Risk Assessment in EPA's Group B2 (probable human carcinogen), based upon positive results in oral tests with mice and rats and an absence of epidemiologic data.

Human inhalation exposure to 1,2-dichloroethane causes headache, dizziness, nausea, vomiting, abdominal pain, irritation of the mucous membranes, and liver and kidney dysfunction (USEPA 1985). Dermatitis may result from skin contact. In severe cases of exposure, leukocytosis (an excess of white blood cells) has been observed. At high exposure concentrations, internal hemorrhaging and pulmonary edema leading to death can occur (USEPA 1985). Similar effects are produced following exposure of experimental animals.

Toxicity to Wildlife and Domestic Animals

1,2-Dichloroethane is one of the chlorinated ethanes least toxic to aquatic life. It is acutely toxic to both freshwater and saltwater species at concentrations greater than 118 mg/liter (USEPA 1980). Toxicity following chronic exposure has been observed at 20 mg/liter concentrations (USEPA 1980).

No information on the toxicity of 1,2-dichloroethane to domestic animals or terrestrial wildlife was available in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986).

Aquatic Life (Freshwater)

The available data are not adequate for establishing criteria. However, EPA does report the lowest values known to be toxic in aquatic organisms.

Acute toxicity: 118 mg/liter
Chronic toxicity: 20 mg/liter

Aquatic Life (Saltwater)

Acute toxicity: 113 mg/liter

Chronic toxicity: No available data

Human Health

Due to the carcinogenicity of 1,2-dichloroethane the ambient water criterion is set at zero. However, estimates of the carcinogenic risks associated with lifetime exposure from ingestion of contaminated water and contaminated aquatic organisms are:

<u>Risk</u>	<u>Concentration</u>
10^{-5}	9.4 $\mu\text{g/liter}$
10^{-6}	0.94 $\mu\text{g/liter}$
10^{-7}	0.094 $\mu\text{g/liter}$

National Primary Drinking Water Standard (USEPA): 0.005 mg/liter
(Proposed MCL; 50 Federal Register 46904, Wednesday, November 13, 1985)

CAG Potency Slope for Oral Exposure (USEPA 1985): $9.1 \times 10^{-2} (\text{mg/kg/day})^{-1}$

CAG Potency Slope for Inhalation Exposure (USEPA 1985): $9.1 \times 10^{-2} (\text{mg/kg/day})^{-1}$

OSHA Standards: $\text{TWA}^{1/} = 200 \text{ mg/m}^3$

Ceiling Level = 400 mg/m^3

Peak Concentration = 800 mg/m^3 for 5 min every 3 hr.

AGIH Threshold Limit Values: $\text{TWA}: 40 \text{ mg/m}^3$

$\text{STEL}^{2/} = 60 \text{ mg/m}^3$

1/ Time Weighted Average
2/ Short Term Effect Level

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For carcinogens such as 1,2-dichloroethane, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes have been estimated for oral exposure routes and for inhalation exposure for some chemicals. The slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10⁻⁴ to 10⁻⁷ is considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the D_T values for 1,2-dichloroethane is as follows:

$$\begin{aligned} D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\ &= \frac{1 \times 10^{-4}}{9.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}} \\ &= 1.1 \times 10^{-3} \text{ mg/kg/day} \end{aligned}$$

The range of D_T values for 1,2-dichloroethane is presented below:

<u>Risk Level</u>	<u>D_T (mg/kg/day)</u>
10 ⁻⁴	1.1 x 10 ⁻³
10 ⁻⁵	1.1 x 10 ⁻⁴
10 ⁻⁶	1.1 x 10 ⁻⁵
10 ⁻⁷	1.1 x 10 ⁻⁶

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1,1-DICHLOROETHYLENE^{1/}

Summary

1,1-Dichloroethylene caused kidney tumors (male mice only) and leukemia in one study of mice exposed by inhalation to 1,1-dichloroethylene. The results of other studies were equivocal or negative. 1,1-Dichloroethylene is mutagenic, and has caused adverse reproductive effects when administered to rats and rabbits by inhalation. Chronic exposure causes liver damage, and acute exposure to high doses produces nervous system damage.

CAS Number: 75-35-4

Chemical Formula: CH_2CCl_2

IUPAC Name: 1,1-Dichloroethene

Important Synonyms and Trade Names; Vinylidene chloride, VDC,
1,1-dichloroethene, 1,1-DCE

Chemical and Physical Properties

Molecular Weight: 96.94

Boiling Point: 37°C

Melting Point: -122.1°C

Specific Gravity: 1.218 at 20°C

Solubility in Water: 400 g/liter at 20°C

^{1/} U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Solubility in Organics: Sparingly soluble in alcohol, ether, acetone, benzene, and chloroform

Log Octanol/Water Partition Coefficient (K_{OW}): 1.48
1.84 (Mabey et al. 1981) Estimated

Soil/Water Partition Coefficient (K_{OC}):

366	Lyman et al. (1982) Eqn 4-8 (log k_{OW} = 2.18)
144	Lyman and Loreti (1986) Eqn I (log K_{OW} = 2.18)
130	Lyman and Loreti (1986) Eqn II (log K_{OW} = 2.18)
133	Lyman and Loreti (1986) Eqn III (log K_{OW} = 2.18)
232	Kadeg et al. (1986) (log K_{OW} = 2.18)

Bioconcentration Factor:

20.2	Davies and Dobbs (1984) Eqn C (log K_{OW} = 2.18)
26.7	Lyman et al. (1982) Eqn 5-2 (log K_{OW} = 2.18)
30.9	Davies and Dobbs (1984) Eqn B (log K_{OW} = 2.18)

Vapor Pressure: 500 mm Hg at 20°C
600 mm Hg at 25°C (USEPA 1985a)

Vapor Density: 3.25

Henry's Law Constant: 3.4×10^{-2} atm-m³/mole (USEPA 1985a)

Transport and Fate

Volatilization is likely to be the primary transport process for 1,1-dichloroethylene (VDC), and its subsequent photooxidation in the atmosphere by reaction with hydroxyl radicals is the predominant fate process. The half-life of 1,1-dichloroethylene in water is estimated to be between one and six days (USEPA 1984).

A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of 1,1-dichloroethylene to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and low organic partitioning of 1,1-dichloroethylene suggests that this compound will exhibit some degree of environmental mobility.

A range of estimated bioconcentration factor (BCFs) for 1,1-dichloroethylene is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of 1,1-dichloroethylene residues is not likely to occur.

Health Effects

1,1-Dichloroethylene caused kidney tumors in male mice and leukemia in both males and females when exposed by inhalation. Equivocal results were obtained in other inhalation studies. Negative results were obtained in cancer studies with rats and mice following oral exposure or in hamsters following inhalation exposure. 1,1-Dichloroethylene has been classified according to EPA's Proposed Guidelines for Carcinogen Risk Assessment in EPA's Group C (possible human carcinogen) based on limited evidence in animals and inadequate evidence in humans (USEPA 1985b).

1,1-Dichloroethylene was mutagenic in several bacterial assays. 1,1-Dichloroethylene did not appear to be teratogenic but did cause embryotoxicity and fetotoxicity when administered to pregnant rats and rabbits by inhalation. Chronic exposure to oral doses of 1,1-dichloroethylene as low as 5 mg/kg/day caused liver changes in rats. Acute exposure to high doses causes central nervous system depression.

Neurotoxicity has not been associated with low-level chronic exposure. The oral LC₅₀ value for rats and mice are 1,500 mg/kg and 200 mg/kg, respectively.

Toxicity to Wildlife and Domestic Animals

1,1-Dichloroethylene is not very toxic to freshwater or saltwater species, with acute LC₅₀ values generally ranging from 80 to 200 mg/liter (USEPA 1980). A chronic study in which no adverse effects were observed indicated that the acute-chronic ratio was less than 40. A 13-day study reporting an LC₅₀ of 29 mg/liter indicated that the acute-chronic ratio for 1,1-dichloroethylene is greater than 4.

No reports of the toxicity of 1,1-dichloroethylene to terrestrial wildlife or domestic animals were found in the literature reviewed.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986).

Aquatic Life (Freshwater)

The available data are not adequate for establishing criteria. However, EPA does report the lowest values known to cause toxicity in aquatic organisms.

Acute toxicity: 11,600 µg/liter

Chronic toxicity: No available data

Aquatic Life (Saltwater)

Acute toxicity: 224,000 µg/liter

Chronic toxicity: No available data

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address the use of a LOEL rather than a NOEL (10). An additional Modifying Factor (MF) of 10 is included by EPA to increase the margin of safety by addressing the possible carcinogenicity of 1,1-dichloroethylene by oral exposure. Derivation of the oral D_T value for 1,1-dichloroethylene is as follows:

$$\begin{aligned}
 D_T &= \frac{\text{LOAEL (mg/kg/day)}}{\text{UF} \times \text{MF}} \\
 &= \frac{10}{1,000 \times 10} \\
 &= 0.001 \text{ mg/kg/day}
 \end{aligned}$$

For inhalation exposure to 1,1-dichloroethylene, the D_T value is based on the USEPA Cancer Assessment Group's cancer potency slopes. The cancer potency slopes are intended to be a plausible upper bound of the potency of a carcinogen in inducing cancer at low doses. Calculation of a D_T using a cancer potency slope requires selection of an acceptable cancer risk level. A range of risk levels from 10^{-4} to 10^{-7} will be considered for all carcinogens, therefore a range of D_T values is presented. Derivation of the inhalation D_T value for 1,1-dichloroethylene is as follows:

$$\begin{aligned}
 D_T &= \frac{\text{Risk Level}}{\text{Potency Slope (mg/kg/day)}^{-1}} \\
 &= \frac{1 \times 10^{-4}}{1.16 \text{ (mg/kg/day)}^{-1}} \\
 &= 8.6 \times 10^{-5} \text{ mg/kg/day}
 \end{aligned}$$

The range of inhalation D_T values for 1,1-dichloroethylene is presented below.

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1,2-DICHLOROETHYLENE (cis-,trans-)^{1/}

Summary

Chronic inhalation exposure of rats to 1,2-trans-dichloroethylene (1,2-trans-DCE) caused liver degeneration. No data are currently available to evaluate the teratogenicity or carcinogenicity of the 1,2-dichloroethylene isomers in animals or humans. The cis- isomer has tested positive for mutagenicity in a host mediated assay using Salmonella.

CAS Number: 4540-59-0 (cis-,trans)

Chemical Formula: $C_2H_2Cl_2$

IUPAC Names: 1,2-cis-dichloroethylene; 1,2-trans-dichloroethylene

Important Synonyms and Trade Names; trans-acetylene dichloride,
cis-dichloroethylene, trans-
dichloroethylene

Chemical and Physical Properties^{2/}

Molecular Weight: 96.95 (USEPA 1984)

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

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^{2/} Unless otherwise specified, data presented will apply to both isomers.

Boiling Point: -47.5°C (trans-) (USEPA 1984)
60°C (cis-) (USEPA 1984)

Melting Point: -50°C

Specific Gravity: 1.2565 at 20°C

Solubility in Water: 6300 mg/liter (trans-) (USEPA 1984)
3500 mg/liter (cis-) (USEPA 1984)

Solubility in Organics: Miscible with alcohol, ether, and acetone;
very soluble in benzene and chloroform

Log Octanol/water Partition Coefficient (K_{ow}): 1.48
1.53 (Lyman et al.,
1982) Fragment Method

Soil/Water Partition Coefficient (K_{oc}):

36 - 49	Lyman et al. (1982) Eqn 4-5 (S = 3,500 - 6,300)
156	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.5$)
43	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.5$)
35	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.5$)
37	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.5$)
75	Kadeg et al. (1986) ($\log K_{ow} = 1.5$)

Bioconcentration Factor:

8.6	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.53$)
6.2 - 4.4	Davies and Dobbs (1984) Eqn A (S = 3,500 - 6,300)
12.6	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.53$)
4.0; 1.58	ECAO (1980) (experimental)
8.08	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.53$)

Vapor Pressure: 265 mm Hg at 20°C (trans-) (USEPA 1984)
180 mm Hg at 20°C (cis-) (USEPA 1984)

Flash Point: 3°C (undefined isomers)

Henry's Law Constant: 7.58×10^{-3} atm-m³/mole (cis-) (USEPA 1985)
 6.56×10^{-3} atm-m³/mole (trans-) (USEPA 1985)

Transport and Fate

Due to the relatively high vapor pressure of 1,2-dichloroethylene (1,2-DCE), volatilization from soils and aquatic systems is quite rapid and appears to be a primary transport process. Photooxidation in the troposphere appears to be the dominant chemical fate of 1,2-trans-DCE following its release to the air. Once in the troposphere, the compound is attacked at the double bond by hydroxyl radicals, resulting in the formation of formic acid, hydrochloric acid, carbon monoxide, and formaldehyde. The half-lives of 1,2-trans-DCE and 1,2-cis-DCE in the atmosphere are approximately 2.1 days and 1.3 days, respectively (USEPA 1985). The half-life of the 1,2-DCE isomers in surface waters is estimated to range between one and six days (USEPA 1985).

A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of 1,2-dichloroethylene to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of volatile organic compounds will range from low to moderate. The combined water solubility and low organic partitioning of 1,2-dichloroethylene suggests that this compound will exhibit some degree of environmental mobility. Although no information pertaining specifically to biodegradation of 1,2-DCE is available, results with similar compounds suggest that this process probably occurs but at a very slow rate.

A range of experimental and estimated bioconcentration factors (BCFs) for 1,2-dichloroethylene is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of 1,2-dichloroethylene residues is not likely to occur.

Health Effects

Very little information concerning exposure to the individual 1,2-DCE isomers is available. No long-term studies have been carried out on the carcinogenic potential of cis- and trans-1,2-dichloroethylene and therefore both isomers are classified in EPA's Group D (not classified due to inadequate evidence of animal carcinogenicity). Both cis- and trans-1,2-DCE were non-mutagenic when assayed with E. coli k12 (USEPA 1984a). The cis-isomer of 1,2-DCE was mutagenic in a host-mediated assay using Salmonella tester strains in mice (Cerna and Kyenova, 1977). The same authors observed that cis-1,2-DCE produced chromosomal aberrations in mouse bone marrow cells following intraperitoneal injection.

Like other members of the chlorinated ethylene series, 1,2-DCE has anesthetic properties. The toxicity of 1,2-DCE, as determined by changes in hepatic enzymes, indicated that cis-1,2-DCE was slightly more hepatotoxic than trans-1,2-DCE (USEPA 1984a). Repeated inhalation exposures of rats to 200 ppm of trans-1,2-DCE for 8 hr/day, 5 days/week for 16 weeks produced fatty degeneration of the liver lobules and kupffer cells (USEPA 1984a). Fibrous swelling of the heart, and hyperemia were noted following exposure to 3,000 ppm trans-1,2-DCE. Rats exposed to 16,000 ppm cis-1,2-DCE were anesthetized in 8 minutes and died after 4 hours of continuous exposure (USEPA 1984a).

Although nephrotoxic and cardiac sensitizing effects are associated with exposure to 1,1-dichloroethylene, the 1,2-DCE isomers have not been investigated with respect to these type of effects (USEPA 1984b). 1,2-trans-dichloroethylene can inhibit aminopyrine demethylation in rat liver microsomes in vitro, and it may thus interact with the hepatic drug-metabolizing monooxygenase system.

Toxicity to Wildlife and Domestic Animals

Virtually no information concerning the toxicity of 1,2-DCE to wildlife and domestic animals exists. The reported 96-hour LC₅₀ value under static conditions for 1,2-trans-DCE is 135,000 µg/liter for the blue gill (USEPA 1980). Under the same test conditions, the LC₅₀ value for 1,1-dichloroethylene is 73,900 µg/liter. Recommended criteria for protection of aquatic life are based primarily on data concerning 1,1-dichloroethylene.

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986).

Aquatic Life (Freshwater)

The available data are not adequate for establishing criteria. However, EPA does report the lowest values known to be toxic in aquatic organisms.

Acute toxicity: 11.6 mg/liter (dichloroethylenes)

Chronic toxicity: no data are available

Aquatic Life (Saltwater)

Acute toxicity: 224 mg/liter (dichloroethylenes)

Chronic toxicity: no data are available

Human Health

The available data are not adequate for establishing criteria.

OSHA Standard: $\text{TWA}^{1/} = 790 \text{ mg/m}^3$

ACGIH Threshold Limit Values: $\text{TWA}^{1/} = 790 \text{ mg/m}^3$
 $\text{STEL}^{2/} = 1,000 \text{ mg/m}^3$

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For 1,2-(cis-,trans-)dichloroethylene (1,2-DCE) the D_T value is derived from the data used to establish an EPA proposed Drinking Water Recommended Maximum Contaminant Level (RMCL). The RMCL is based on No-Observed-Effect-Levels (NOELs) or Lowest-Observed-Effect-Levels (LOELs) derived from subchronic or chronic animal toxicity studies including an Uncertainty Factor (UF). For 1,2-dichloroethylene the RMCL is derived from a chronic oral toxicity/oncogenicity study in male and female rats administered 1,1-dichloroethylene in drinking water (Quast et al. 1983). Data on 1,1-dichloroethylene was used because the best available data on the three isomers (cis-, trans-1,2-DCE and 1,1-DCE) suggests that the toxicity induced by the 1,2-isomers is not likely to be more severe than that of 1,1-dichloroethylene (USEPA; 50 Federal Register 46991, Wednesday, November 13, 1985). The two-year study identified a LOAEL of 10 mg/kg based on observed liver changes. A UF of 1,000 was employed to address extrapolation of the results from animals to humans (10), intraspecies variability (sensitive subgroups) (10), and to account for the use of a LOAEL rather than a NOAEL (10). The Modification Factor for potential carcinogenicity included in the derivation of the D_T for 1,1-dichloroethylene is not applied to

1/ Time Weighted Average.

2/ Short-Term Effect Level.

1,2-dichloroethylenes due to a lack of studies demonstrating its carcinogenicity. Derivation of the D_T for 1,2-(cis-,trans-)dichloroethylene is as follows:

$$\begin{aligned} D_T &= \frac{LOAEL}{UF} \\ &= \frac{10 \text{ mg/kg/day}}{1,000} \\ &= 0.01 \text{ mg/kg/day} \end{aligned}$$

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DICYCLOPENTADIENE^{1/}

Summary

No data on the toxicity of dicyclopentadiene (DCPD) to humans was located in available literature. DCPD was not mutagenic in standard microbial assays (both activated and inactivated). No evidence of toxicity was observed following subchronic dietary administration to rats, mice or dogs at levels ranging up to 750 ppm, 273 ppm or 1,000 ppm, respectively. No reproductive effects occurred following DCPD exposure in male and female rats, nor were doses of DCPD teratogenic when administered to pregnant rats during gestation days 6-15.

CAS Number: 77-73-6

Chemical Formula: $C_{10}H_{12}$

IUPAC Name: Dicyclopentadiene

Important Synonyms and Trade Names: DCPD

Chemical and Physical Properties

Molecular Weight: 132

Melting Point: 32.9°C, (Rosenblatt et al. 1975)

Boiling Point: 170°C (Cogley and Foy 1978)

Solubility in Water: 20 mg/l (estimated; Lyman et al. 1982)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Log Octanol/Water Partition Coefficient (K_{ow}): 3.14 (Lyman et al.
1982) Fragment Method

Soil/Water Partition Coefficient (K_{oc}):

1,200	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 3.14$)
840	Lyman et al. (1982) Eqn 4-5 ($S = 20$)
805	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 3.14$)
824	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 3.14$)

Bioconcentration Factor:

53	Bentley et al. (1976) (experimental)
114	Davies and Dobbs (1984) Eqn A ($S = 20$)
143	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 3.14$)
115	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 3.14$)
53.9	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 3.14$)

Specific Gravity: 0.98 (Rosenblatt et al. 1982)

Vapor Pressure: 2.2 mm Hg at 25°C (estimated; Rosenblatt et al. 1975)
1.4 mm Hg at 20°C (estimated; Rosenblatt et al. 1975)

Henry's Law Constant: 1.9×10^{-2} atm-m³/mole (calculated)
 1.2×10^{-2} atm-m³/mole (calculated)

Transport and Fate

The relatively high vapor pressure of DCPD indicates the importance of volatilization (evaporation) as a transport process from surface water to the atmosphere. The chemical fate of DCPD in the atmosphere is not definitively known, however, photodegradation may occur. DCPD is virtually insoluble in water (Cogley and Foy, 1978). A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of DCPD to soils/sediments and dissolved organic materials will occur. The combined low water solubility and high organic partitioning coefficients suggest that dicyclopentadiene will not be mobile in the environment. The half-life of DCPD in soil ranges from six months to one year depending on ambient conditions (Cogley and Foy 1978). Degradation to more stable forms (degradation forms were not reported) occurs and the reported half-lives of these

products are greatly increased; ranging from one to greater than five years (Cogley and Foy 1978). Spangord et al. (1979) reported an estimated half-life of 4-7 years for DCPD incubated (25°C) soil samples.

Biodegradation in aquatic systems is not likely to be extensive (Spangord et al. 1979). An estimated 76-day or greater half-life of DCPD in water samples was also reported by Spangord et al. (1979), based upon sunlight exposure (photolysis) tests. A 5.3 day half-life for DCPD in water samples (25°C, without recharge) was also observed. Uptake of less than 100 ppm DCPD was observed in plants which were grown in hydroponic solutions (1,000 ppm) (O'Donovan and Woodward 1977). Evidence of stunted growth was also seen in plants at this concentration.

A range of experimental and estimated bioconcentration factors (BCFs) for dicyclopentadiene is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of DCPD residues is not likely to occur.

Health Effects

No data on the toxicity of DCPD in humans was located in available literature. DCPD was not mutagenic in a variety of microbial assays both with and without metabolic activation (Hart 1980). No data on the carcinogenicity of DCPD was located. Dicyclopentadiene was minimally irritating to rabbit skin and did not produce evidence of systemic toxicity following application (Hart 1976). No evidence of toxicity followed its dietary administration for 90 days to rats at levels up to 750 ppm or to mice at levels up to 273 ppm (Hart 1976). Hart (1980) administered DCPD to beagle dogs in their diets for 13 weeks at concentrations of 100, 300 or 1,000 ppm. Clinical pathological evaluations, including analyses of clinical chemical constituents of serum, urine and hemograms were performed at monthly intervals. Tissues

from control and treated dogs were compared histopathologically. No significant toxicity was observed with the possible exception of minor indications of intestinal distress expressed as vomiting and soft stool among treated groups, especially the highest dose (Hart 1980). The No-Observed-Adverse-Effect-Level identified from this study was 1,000 ppm (25 mg/kg/day). No effects on fertility indices, live-to-total pup ratios, mean litter sizes, pup survival indices or mean body weights of pups post partum were observed in rats given 80 or 750 ppm DCPD in the diet prior to mating. Likewise, no dose-related teratogenic effects were observed in pregnant females administered 80, 250, or 750 ppm in the diet during days 6-15 of gestation (Hart 1980). DCPD had oral LD50's of 520 and 378 mg/kg in male and female rats and 190 and 250 mg/kg in male and female mice (Hart 1976).

Toxicity to Wildlife and Domestic Animals

Dicyclopentadiene was found to be relatively non-toxic to mallard ducks (Aulerich et al. 1979). An oral LD₅₀ could not be determined, even when levels administered were as high as 40,000 mg/kg. The oral LD₅₀ in Bobwhite quail was 1,010 mg/kg and greater than 1,000 mg/kg in mink. The biological half-life of DCPD residues in ducks and quail fed ¹⁴C-DCPD-treated diets averaged 12.7 hours and was not concentrated in adipose tissue of either species.

Regulations and Standards

ACGIH Threshold Limit Value: $TWA^{1/} = 30 \text{ mg/m}^3$

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

1/ Time Weighted Average.

For dicyclopentadiene (DCPD), the D_T value is based on a subchronic oral toxicity study utilizing dogs (Hart 1980). No toxicity was observed (histopathologically or otherwise) at any dose level, with the exception of some vomiting and soft stools. The identified No-Observed-Adverse-Effect-Level (NOAEL) from this study was 1,000 ppm (25 mg/kg/day). An Uncertainty Factor (UF) of 1,000 is employed to address the extrapolation of results to humans (10), intraspecies variability (sensitive subgroups) (10), and to address the use of a subchronic rather than a chronic study (10). Derivation of the D_T for dicyclopentadiene is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOEL}}{\text{UF}} \\ &= \frac{25 \text{ mg/kg/day}}{1,000} \\ &= 0.025 \text{ mg/kg/day} \end{aligned}$$

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DIISOPROPYL METHYLPHOSPHONATE^{1/}

Summary

No data on the toxicity of diisopropyl methylphosphonate (DIMP) in humans was located. DIMP was not mutagenic in microbial assays. DIMP has a low subchronic toxicity in rats, mice and beagle dogs, with levels up to 2,100 ppm (mice) and 3,000 ppm (rats and dogs) causing no observable toxicological effects over a 90 day period. In rats, DIMP has no effect on reproductive measures at 300 or 3,000 ppm in the diet. No teratogenic effects were observed in female rats following administration of up to 3,000 ppm DIMP during gestation days 6-15. DIMP is an eye and skin irritant in rabbits.

CAS Number: 1445-75-6

Chemical Formula: $((\text{CH}_3)_2\text{CHO})_2\text{P}(\text{O})\text{CH}_3$

IUPAC Name: Diisopropyl Methyl Phosphonate

Important Synonyms and Trade Names: DIMP

Chemical and Physical Properties

Molecular Weight: 193

Boiling Point: 174°C (Rosenblatt et al 1975a)

Specific Gravity: 0.98 (Rosenblatt et al., 1975a)

Solubility in Water: 1,500 mg/liter (Rosenblatt et al., 1975a)
32 g/liter (Bentley et al. 1976)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Log Octanol/Water Partition Coefficient: 1.79 (Brueggemann 1982)
1.0 (Lyman et al. 1982)
Fragment Method

Soil/Water Partition Coefficient (K_{oc}):

15 - 78	Lyman et al. (1982) Eqn 4-5 ($S = 1,500 - 32,000$)
83	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.0$)
17	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.0$)
13.5	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.0$)
14	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.0$)
33	Kadeg et al. (1986) ($\log K_{ow} = 1.0$)

Bioconcentration Factor:

0.6	Bentley et al. (1976) (experimental)
2.08	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 0.72$)
3.4 - 1.8	Davies and Dobbs (1984) Eqn A ($S = 10,000 - 32,000$)
6.1	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.0$)
4.3	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.0$)
3.4	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.0$)

Vapor Pressure: 0.28 mm Hg at 25°C (Rosenblatt et al. 1975a)

Henry's Law Constant: 4.7×10^{-5} atm-m³/mole (calculated)
 8.5×10^{-6} atm-m³/mole (calculated)

Transport and Fate

The low vapor pressure of DIMP indicates that limited volatilization from environmental media would be expected to occur for this compound. Soil incubation studies (Spangord et al. 1979) indicate a slow loss of DIMP at 25°C and virtually no loss at 10°C. A half-life of two years was estimated for DIMP from these data. DIMP is a fairly water soluble compound (Rosenblatt et al. 1975b). Spangord et al. (1979) observed little hydrolysis of DIMP in water samples based on tests at 80°C or higher. Based on these data a half-life for DIMP on the order of 530 years at 10°C was estimated. Essentially no photolytic degradation of DIMP occurred in aqueous solutions exposed for up to 232 hours to light of wavelengths in excess of 290 nm (Spangord et al. 1979).

Additionally, no aquatic biodegradation of DIMP in acclimated water cultures from Rocky Mountain Arsenal or water cultures from sludge aeration occurred.

A range of estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that some sorption of DIMP to soils/sediments and dissolved organic materials will likely occur. The combined water solubility and low organic partitioning suggest that DIMP will be an environmentally mobile contaminant. Microbial degradation reportedly does not occur (Rosenblatt et al., 1975b).

DIMP uptake into plant tissues does occur with residues generally accumulating as follows: leafy tissues > stems > roots (O'Donovan and Woodward, 1977). Radishes grown in hydroponic solutions of 10 ppm DIMP exhibited levels of 48 ppm in leaves and 7.3 ppm in the roots (wet basis, O'Donovan and Woodward 1977). Greenhouse studies have indicated that DIMP is phytotoxic (Rosenblatt et al. 1975b).

A range of experimental and estimated bioconcentration factors (BCFs) for DIMP is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of DIMP residues is not likely to occur.

Health Effects

No data on the toxicity of DIMP to humans was located in available literature. DIMP tested negative in both activated and nonactivated microbial mutagenicity tests utilizing Salmonella and Saccharomyces (Hart 1980). No data on the possible carcinogenicity of DIMP was located.

No evidence of toxicity resulted from dietary administration of DIMP to rats at levels of 300, 1,000 and 3,000 ppm for 90 days or to mice at levels of 210, 700 and 2,100 ppm for 90 days (Hart 1976). No toxicity was observed (clinical chemistry, hematology or histopathology) in male and female beagle dogs administered 150, 1,500 or 3,000 pm DIMP in the diet for 90 days (Hart 1980). DIMP administered in the diet of pregnant rats at doses of 100, 300, and 3,000 ppm resulted in no maternal or fetal adverse compound-related effects (Hart 1980). Dietary incorporation of DIMP at levels of 300 and 3,000 ppm yielded no dose-related reproductive response in rats for three successive generations (two matings per generation) (Hart 1980).

Subacute oral exposure (14 days) of male and female dogs to DIMP at 150, 500, and 1,500 ppm produced no evidence of toxicity (Hart 1976). DIMP in the diet of rats for 90 days at levels up to 3,000 ppm did not affect body weights, organ weights, or clinical chemistry (Hart 1976). Some evidence of enzyme induction (unspecified) in the liver was also reported (Hart 1976). The acute oral LD₅₀ values for DIMP were 1,125 and 826 mg/kg in male and female rats, respectively, and 1,041 and 1,363 mg/kg in male and female mice, respectively (Hart 1976). DIMP was irritating following its application to rabbit eyes; however, in all but one case, the irritation had cleared by the seventh day and the remaining case had cleared by day eight (Hart 1976). Minimal skin irritation occurred in rabbits directly exposed at doses of 0.2, 0.63 and 2.0 g/kg DIMP; however, death occurred in three of four animals at the highest dose and in one of four animals at the intermediate dose (Hart 1976).

Toxicity to Wildlife and Domestic Animals

Bentley et al. (1976) report that the 96-hour LC₅₀ for DIMP in fathead minnows ranges between 15.7 and 31.1 mg/liter. Seven day-old fry appeared to exhibit the highest degree of tolerance to DIMP exposure. They also reported an LC₅₀ in bluegills of 257 mg/l DIMP at 25°C.

Chronic feeding studies with DIMP (3,200 ppm) in the diets of mallard ducks resulted in decreased food consumption and levels of 10,000 ppm resulted in reduced egg production (Aulerich et al. 1979). No other consistent adverse effects on reproduction, behavior, feed consumption, growth, hematology or mortality were observed in DIMP-fed ducks or quail during the 24-week test. Chronic ingestion of DIMP had no adverse effects on growth or reproductive performance of mink, although slightly higher mortality occurred in DIMP-fed females. Mallard ducks and bobwhite quail fed diets with radioactive DIMP had ^{14}C tissue residues averaging less than 1 ppm. These values averaged 0.04 ppm in most tissues by the 3rd day of withdrawal. All tissues but skin (0.05 - 0.1 ppm) were clear of residue by day 4. The acute oral LD_{50} values for DIMP in mallards, bobwhite quail and mink were 1,490, 1,000 and 403 mg/kg, respectively.

Regulations and Standards

The National Research Council Committee on Toxicology has reviewed the available toxicological literature on DIMP and has recommended a drinking water limit of 26.3 mg/liter (Marzulli 1986).

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For diisopropyl methylphosphonate (DIMP), the D_T value is derived from a subchronic (90-day) dietary toxicity study in dogs (Hart 1980). The highest No-Observed-Adverse-Effect-Level (NOAEL) identified from this study was 3,000 ppm (75 mg/kg/day) DIMP. A 100-fold Uncertainty Factor (UF) was considered appropriate by the National Research Council Committee on Toxicology in view of the amount and quality of the data available for DIMP. The UF addresses the extrapolation of results to humans (10) and the potential for intraspecies variability (sensitive subgroups) (10).

Derivation of this D_T value is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOAEL}}{\text{UF}} \\ &= \frac{75 \text{ mg/kg/day}}{100} \\ &= 0.75 \text{ mg/kg/day} \end{aligned}$$

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CA.

DIMETHYLARSENIC ACID^{1/}

Summary

Dimethylarsenic acid (DMAA) and its sodium salts have been used extensively as post-emergence herbicides. Clinical symptoms and histological findings following poisoning appear to be identical to those induced by inorganic arsenic. In a subacute toxicity study in rats, atrophic changes were observed in the seminiferous tubules concomitant with reduced activity of spermatogonia cells. In a subchronic toxicity study, female beagle dogs exhibited a slower weight gain at the highest dose level tested. Some evidence of teratogenicity has been observed in rats and mice. Positive results have been attained in mutagenicity tests in yeast and mice. No evidence of carcinogenicity has been demonstrated following oral and subcutaneous exposures in laboratory animals.

CAS Number: 75-60-5

Chemical Formula: $(\text{CH}_3)_2\text{As}(\text{O})\text{OH}$

IUPAC Name: Hydroxydimethylarsine oxide

Important Synonyms and Trade Names: Cacodylic acid

Chemical and Physical Properties

Molecular Weight: 138

Melting Point: 195-196°C (Merck 1983)

^{1/} Compiled from: U.S. Environmental Protection Agency (USEPA). Initial Scientific Review of Cacodylic Acid. Substitute Chemical Program. Office of Pesticide Programs, Criteria and Evaluation Division. Washington, D.C.. EPA-540/1-75-021.
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in humans include nausea and vomiting, diarrhea, muscular cramps, cold extremities, vertigo, tenderness in muscles, skin pigmentation, circulatory collapse, convulsions, coma, and polyneuropathy (OHM/TADS 1985).

Data on the acute and chronic toxicity of DMAA in humans are not available but are presented for laboratory animals. In a subacute study (20 day), weanling rats were fed diets containing DMAA at concentrations of 10, 20, or 40 percent of the estimated LD₅₀ of 0.7 g/kg (USEPA 1975 cite Nees 1960). Histological examinations of a variety of tissues including the liver, heart, lungs, kidneys and testes were performed at termination of the experiment. At dietary concentrations of 10 and 20 percent, no histologically significant changes were observed in any tissues. However, there was evidence of reduced activity of spermatogonia cells concomitant with atrophic changes in the seminiferous tubules of animals on the 40 percent diet (USEPA 1975 cite Nees 1960). In a similar study (USEPA 1975 cite Nees 1968), male and female weanling rats were fed concentrations of DMAA (3, 15, 30 and 100 ppm) in the diet for a period of 90 days. Gross pathology, urinalyses and hematological parameters were assessed. No significant differences in body weight or food consumption between control and test animals was observed. Observed variations in the hematological parameters and results of the urinalysis (unspecified) were not attributed to DMAA treatment. In another study, young beagle dogs were given dietary concentrations of 3, 15, or 30 ppm DMAA (USEPA 1975 cite Derse 1968). Hematological tests and urinalyses were conducted following a 90 day feeding regime. No differences in body weight were observed at any dose; however, females receiving 30 ppm DMAA did exhibit a slower weight gain. No hematological differences were observed. Results of urinalyses were not specified in the summary report cited.

DMAA was teratogenic in at least one rat orally dosed with 400 mg/kg (TD_{L0}) on days 7-16 of gestation (NIOSH 1986). Abnormalities of the musculoskeletal system and fetotoxicity were observed in the resulting offspring of this animal. In the same study,

at least one mouse orally dosed with 4 g/kg DMAA (TD_{LO}) during days 7-16 of gestation produced offspring with developmental abnormalities of the nose, tongue and musculoskeletal system. Fetotoxicity (e.g., stunted growth) was also observed in these offspring (NIOSH 1986).

Mutagenicity tests with DMAA in Salmonella strains did not result in a significant number of histidine revertants (USEPA 1975 cite Andersen et al. 1972). Positive results were achieved with Saccharomyces cerevisiae (yeast) at levels of 2 and 5 pph DMAA (NIOSH 1986) and also with mice in the micronucleus test following intraperitoneal injection of 7900 mg/kg DMAA (NIOSH 1986). Carcinogenicity was not demonstrated in two different strains of mice (male and female) given daily doses of 46.4 mg/kg DMAA from age 7 days to 28 days, followed by 121 mg/kg DMAA, for a period of 18 months (IARC 1980 cite Innes et al. 1969; NTIS 1968). The same strains of mice (male and female) administered a single subcutaneous injection of DMAA in water (464 mg/kg bw) did not exhibit an increased incidence of tumors as compared with controls (IARC 1980 cite NTIS 1968).

The acute oral LD_{50} value for DMAA in rats is 700 mg/kg (NIOSH 1986). The inhalation LC_{50} value in female rats is 3900 mg/m³ (IARC 1980 cite Stevens et al. 1979).

Toxicity to Wildlife and Domestic Animals

Two cows were fed 24.5 mg/day DMAA in cotton seed meal for a period of 60 days (USEPA 1975 cite Peoples 1963). No mortalities occurred at this intake level. Tissue levels of the slaughtered cows were low with the highest concentrations occurring in the liver, spleen and pancreas. No arsenic residues were detected in milk. In another study, drenching of cattle with a concentration of 50 mg/kg DMAA resulted in obvious signs of arsenic poisoning with death occurring four days following the seventh dose (USDA 1972). Six holstein calves fed dietary concentrations of Amar 560 (22.73 percent sodium cacodylate and 3.88 percent cacodylic acid [DMAA]) at levels corresponding to 10, 20, and 60 percent of the estimated LD_{50} value showed decreased feed

consumption over the seven-day duration of the test (USEPA 1975). Animals on the 20 percent diet refused their feed following the fifth day while those on the 60 percent diet refused feed on the fourth day of the test. Of the two calves at the 60 percent dose, one had diarrhea and the other died.

No mortality or signs of poisoning were observed in sheep when dosed with 10 to 25 mg/kg DMAA whether by capsule or drench (USDA 1972). However, treatment orally by capsule at a dose of 50 mg/kg resulted in poisoning of one animal following three daily doses. The animal survived 10 doses but exhibited a 21 percent weight loss.

Chickens were administered a commercial herbicide containing 26.5 percent DMAA in doses ranging from 50 to 500 mg/kg bw for a period of ten days (USDA 1972). Controls exhibited a 53 percent weight gain during this period while groups receiving doses of 175 and 250 mg/kg gained only 33 and 36 percent, respectively. Chickens receiving 500 mg/kg gained only 13 percent of their body weight during this period. No mortalities occurred at any treatment level (USDA 1972). Bobwhite quail (Colinus virginianus) chicks were fed a diet containing 5,000 ppm of a material which contained 29 percent sodium cacodylate and 5 percent cacodylic acid (DMAA). The eight-day LD₅₀ value was calculated to be in excess of 5,000 ppm cacodylate. No abnormal behaviors were observed nor were signs of any adverse gross pathology following necropsy (USEPA 1975 cite Industrial Bio-Test Laboratories 1973).

The acute oral LD₅₀ value for Silvisar (54.3 percent DMAA) in female mallard ducks (Anas platyrhynchos) was >2,400 mg/kg (Hudson et al. 1984). In male and female chukars (Alectoris chukar) the oral LD₅₀ was >2,000 mg/kg (Hudson et al. 1984). The acute oral LD₅₀ for DMAA in a female mule deer was >320 mg/kg (Hudson et al. 1984). Signs of intoxication in the doe included slight ataxia, imbalance, slowness, soft feces and anorexia.

The 96 hour LC_{50} for DMAA to cutthroat trout (Salmo clarki) and lake trout (Salvelinus namaycush) was between 10,000 and 100,000 $\mu\text{g/liter}$ (USEPA 1975 cite Woodward 1974). Bluegills exposed to Phytar (99.3 percent DMAA) at 100 ppm active ingredient for 72 hours at 65°F survived; however, all bluegills exposed to 22.6 percent DMAA at 210 ppm active ingredient died within the test period (USEPA 1975 cite McCann 1969). The 96-hour LC_{50} (static) for mature Gammarus fasciatus is 135 mg/l (Mayer and Ellersieck 1986). The 96-hour LC_{50} (static) for bluegills is 17 mg/l (Mayer and Ellersieck 1986).

Regulations and Standards

OSHA Time Weighted Average: 0.5 mg/m^3 [as arsenic] (39 Federal Register 23540, 1974)

ACGIH Threshold Limit Value: $TWA^{1/} = 0.2 \text{ mg/m}^3$ [as arsenic]
(ACGIH 1980)

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

The EPA Reference Dose Committee is currently reviewing the available toxicity data on DMAA for use in developing a chemical specific Risk Reference Dose. However, the Reference Dose value is not yet available; therefore, the D_T value is derived from a subchronic (90-day) toxicity test in male and female beagle dogs administered concentrations of 3, 15, or 30 ppm DMAA (0.075, 0.375, 0.75 mg/kg/day) in their feed (Derse 1968). Kidney and liver function tests, body weights, and urinalyses were conducted. No differences in body weight were observed at any dose; however, females receiving 30 ppm DMAA did

1/ Time Weighted Average.

exhibit a slower weight gain. No hematological differences were detected between control and treated groups. Results of urinalyses were not specified in the summary report cited. Some lesions were noted in the brain, heart, liver, kidney, spleen, and intestines; however, the lesions were randomly scattered in both control and test animals and were not considered treatment related (USEPA 1975 cite Derse 1968). A No-Observed-Adverse-Effect-Level (NOAEL) of 30 ppm is selected from these data on the basis of reduced weight gain in high dose females. An Uncertainty Factor (UF) of 1,000 was employed to address extrapolation of the results from animals to humans (10), intraspecies variability (sensitive subgroups) (10) and to account for the use of a subchronic rather than a chronic toxicity study (10). An additional Modification Factor is included in the derivation of the D_T for DMAA to address the carcinogenicity associated with the arsenic moiety. Derivation of the D_T for DMAA is as follows:

$$D_T = \frac{\text{NOAEL (mg/kg/day)}}{\text{UF}}$$

$$= \frac{0.75}{1,000}$$

$$= 0.00075 \text{ mg/kg/day}$$

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DIMETHYL DISULFIDE^{1/}

Summary

No data on the toxicity of dimethyl disulfide (DMDS) to humans was located in the available literature. Data on the mutagenicity, carcinogenicity, acute, chronic, and reproductive toxicity of dimethyl disulfide are not available. In mice, the acute oral LD₅₀ is 813 mg/kg.

CAS Number: 624-92-0

Chemical Formula: CH₃S₂CH₃

IUPAC Name: Dimethyl Disulfide

Important Synonyms and Trade Names: DMDS

Chemical and Physical Properties

Molecular Weight: 94.2

Boiling Point: 115°C, (Marochini 1984)

Specific Gravity: 1.06 (Marochini 1984)

Solubility in Water: 350 mg/l (estimated; Lyman et al. 1982)

Log Octanol/Water Partition Coefficient (K_{ow}): 1.77 (Hansch and Leo 1979)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Soil/Water Partition Coefficient (K_{oc}):

174	Lyman et al. (1982) Eqn 4-5 (S = 350)
219	Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 1.77$)
69	Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 1.77$)
59	Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 1.77$)
61	Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 1.77$)
118	Kadeg et al. (1986) ($\log K_{ow} = 1.77$)

Bioconcentration Factor:

17.6	Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 1.77$)
22.7	Davies and Dobbs (1984) Eqn A (S = 350)
13.04	Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 1.77$)
10.7	Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 1.77$)

Vapor Pressure: 21 mm Hg at 25°C (estimated from boiling point;
Lyman et al. 1982)

Henry's Law Constant: 7.3×10^{-3} atm-m³/mole (calculated)
 5.1×10^{-3} atm-m³/mole (calculated)

Transport and Fate

The estimated vapor pressure of DMDS suggests that volatilization will be a major transport mechanism for this chemical. However, no data is available on the chemical fate of DMDS released to the atmosphere. A range of estimated soil/water partition coefficients (K_{oc}) is reported above and indicates that some sorption of dimethyl disulfide to soils/sediments and dissolved organic material will occur. The combined water solubility and organic partitioning suggest that dimethyl disulfide will display some degree of environmental mobility.

No data is available on the uptake of DMDS in plants. A range of estimated bioconcentration factors (BCFs) for dimethyl disulfide is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of DMDS residues is not likely to occur.

No data on the persistence of DMDSE in environmental media were located in available literature.

Health Effects

No data on the toxicity of dimethyl disulfide (DMDS) to humans was located in the available literature. Likewise, data on the mutagenicity, carcinogenicity, acute, chronic, and reproductive toxicity of dimethyl disulfide were not available. The acute inhalation LC₅₀ is 805 ppm in the rat (NIOSH, 1983). The acute oral LD₅₀ in rats is 813 mg/kg (Atochem, 1986).

Toxicity to Wildlife and Domestic Animals

No data on the toxicity of DMDS to other wildlife or domestic animal species was located in the available literature.

Regulations and Standards

None located.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For dimethyl disulfide, the D_T value is derived from an acute oral LD₅₀ value in mice (Atochem, 1986). The D_T value is computed as the product of the acute value and an application factor of 1×10^{-5} (Layton et al. 1986). The application factor allows the derivation of an interim acceptable long-term intake rate (D_T) based on the results of acute tests in the absence of more suitable long-term studies (i.e., chronic studies). The application factor corresponds to the cumulative percentile on a lognormal distribution of NOEL/LD₅₀ ratios for various

chemicals. The percentile was chosen to reduce the probability that the calculated dose rate would be above a toxic level; the 5th cumulative percentile was used by Layton et al. (1986) and was found to be equal to 10^{-3} . The application factor also includes a safety factor of 100 to address interspecies and intraspecies variability; therefore, an interim estimate of D_T is obtained when the application factor is multiplied by the acute value. Derivation of this D_T value is as follows:

$$\begin{aligned} D_T &= \text{Acute LD}_{50} \times \text{Application Factor} \\ &= 813 \text{ mg/kg/day} \times 1 \times 10^{-5} \\ &= 0.00813 \text{ mg/kg/day} \end{aligned}$$

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DIMETHYL METHYLPHOSPHONATE^{1/}

Summary

No data on the toxicity of dimethyl methylphosphonate (DMMP) to humans was located in the available literature. Both negative and positive results have been obtained in various mutagenicity assays. The National Toxicology Program (NTP) is currently evaluating the carcinogenicity of DMMP in a 2-year rat study. Dosages of 0, 250, 500, 1,000, and 4,000 mg/kg DMMP fed daily to rats for three months resulted in increased liver-to-body-weight ratios, nephropathy, lesions of the salivary glands and testes, and mortalities. Dosages of 4,000 and 8,000 mg/kg resulted in high mortalities in an oral subchronic toxicity test utilizing mice. Dose-related reproductive effects were observed in dosed male rats which were subsequently mated with untreated females. Effects were seen even at the lowest dose level.

CAS Number: 756-79-6

Chemical Formula: $(\text{CH}_3\text{O})_2\text{P}(\text{O})\text{CH}_3$

IUPAC Name: Dimethyl Methylphosphonate

Important Synonyms and Trade Names: DMMP, dimethyl ester of methylphosphonic acid

Chemical and Physical Properties

Molecular Weight: 124

Boiling Point: 181°C (Berkowitz et al. 1978)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Specific Gravity: 1.14 (Arthur D. Little, Inc. 1982)

Solubility in Water: 8×10^5 mg/liter (estimated; Lyman et al. 1982)

Log Octanol/Water Partition Coefficient (K_{ow}): -1.88 (Lyman et al. 1982) Fragment Method

Soil/Water Partition Coefficient (K_{oc}): Not Applicable

Bioconcentration Factor: Not Applicable

Vapor Pressure: 0.62 mm Hg at 25°C (Nowak 1983)
0.87 mm Hg at 25°C (Berkowitz et al. 1978)

Henry's Law Constant: Not Applicable

Transport and Fate

Little information is available concerning the interactions of DMMP in the environment. The low vapor pressure and high aqueous solubility of DMMP suggest that little volatilization from environmental media will occur. The atmospheric half-life for DMMP is estimated to be 1.6 months (Howard et al. 1986). Bel'skii et al. (1969) studied the hydrolysis of DMMP at high temperatures and neutral conditions. Extrapolation of their data to 15°C indicates a half-life on the order of 13 years.

Little sorption of DMMP to soil is expected to occur given its high water solubility. The combined low organic partitioning and high water solubility indicates that DMMP will exhibit considerable environmental mobility. In environmental chamber studies, DMMP at initial soil concentrations from 100 - 1000 µg/g had half-lives from 0.2 - 60 days with an average half-life of 12.4 days (Howard et al. 1986). Temperatures in the chamber ranged from 10° - 40°C. The processes responsible for the loss of DMMP were not determined. Hydrolysis will occur under acidic and less rapidly, under alkaline conditions (Howard et al. 1986). The primary product following continual hydrolysis are methyl alcohol and methyl phosphoric acid or its salts (Howard et al. 1986). No specific data on plant uptake or microbial degradation were located in the available literature.

Photolysis data are lacking for DMMP. However, for a similar compound, diisopropylmethyl phosphonate, no photolysis occurred in distilled water or Rocky Mountain Arsenal water when exposed to light of a wavelength >290 nm for approximately 10 days (Howard et al. 1986).

Bioconcentration data for azodrin were not located in available literature. However, given the high aqueous solubility and low organic partitioning behavior, bioconcentration would not be expected to occur.

Health Effects

No data on the toxicity of DMMP to humans was located in the available literature. Conflicting results on the mutagenicity of DMMP have been obtained. Positive results in the mouse lymphoma assay were reported by Mobil (1981). Sivak (1983) reported low clastogenic activity in the Chinese hamster ovary chromosome aberration assay and Stauffer (1983) obtained positive results in a mouse cell morphological transformation assay and in mouse lymphoma chromosome aberration and sister chromatid exchange assays. DMMP reportedly induced no mutagenic response in the Ames (Salmonella) test (Dynamac Corp. 1983). More recent tests (National Toxicology Program 1986) indicate DMMP exposure in Drosophila resulted in positive results for sex-linked recessive lethals. DMMP is currently in the chronic phase of toxicology and carcinogenesis studies (National Toxicology Program 1986).

Subchronic (3-month) oral toxicity tests in rats in doses of 250, 500, 1,000, and 4,000 mg/kg resulted in dose-dependent mortality (at two highest dosages), increased liver to body weight ratios, nephropathy and lesions of the salivary gland and testes (Litton Bionetics, 1981). Subchronic oral toxicity tests in mice were conducted at dosages of 250, 500, 1,000, 2,000, 4,000, and 8,000 mg/kg (Litton Bionetics 1979). No effects on relative weight gain or signs of toxicity were seen in mice which died or were moribund. Dosages of 2,000 mg/kg and below were tolerated by mice. No microscope changes were evident in the 2,000 mg/kg dosed animals. Mortality was observed at 4,000 and 8,000 mg/kg.

Dunnick et al. (1983) reported dose-related reproductive effects when DMMP was administered to male rats at dose levels of 0, 250, 500, 1,000, and 2,000 mg/kg/day for 84 days prior to mating with untreated females. Reproductive function was altered at all dosages and included dose-related increases in the number of embryo resorptions, and related decreases in sperm counts, sperm motility and female reproductive index. Histologic abnormalities of the testes which were observed only in the highest dose group, included lack of spermatogenesis, and/or degeneration, vacuolization and necrosis of spermatogenic cells.

Stauffer chemical (1983) reported DMMP to be a mild irritant in eye irritation tests. They also reported an acute dermal toxicity of greater than 2 g/kg in the rabbit. Hollingshaus et al. (1981) reported an acute oral LD₅₀ of greater than 150 mg/kg for DMMP in fasted female rats, however this does not correlate with the LD₅₀ values in rats of >6,810 mg/kg reported by Litton bionetics (1978).

Toxicity to Wildlife and Domestic Animals

DMMP has a moderate acute toxicity to fish in 96-hour static bioassays. The median tolerance limits (TL_M) of bluegills and fathead minnows to DMMP were reported to be 51 mg/liter and 63 mg/liter, respectively (Department of the Army, Dugway Proving Ground, 1976).

Regulations and Standards

None located.

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For DMMP, the D_T value is derived from an oral subchronic toxicity test measuring reproductive impairment in male rats (Dunnick et al. 1983). Reproductive effects were observed in all the dose levels tested including the lowest (250 mg/kg/day). Effects included increases in embryo resorptions, decreased sperm counts and mobility and decreased indices of male fertility. The Lowest-Observed-Effect-Level (LOEL) identified from this study was 250 mg/kg/day. An Uncertainty Factor (UF) of 1,000 is employed to address extrapolation of results to human (10), intraspecies variability (sensitive subgroups) (10), and lack of chronic (long-term) toxicity data (10). An additional severity of effects factor (SF) of 8 is used to address the observed decrease in reproductive capacity which includes fetotoxicity. Derivation of this D_T values is as follows:

$$\begin{aligned} D_T &= \frac{LOEL}{UF \times SF} \\ &= \frac{250 \text{ mg/kg/day}}{(1,000)(8)} \\ &= 0.0312 \text{ mg/kg/day} \end{aligned}$$

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DITHIANE^{1/}

Summary

No data on the toxicity of dithiane to humans was located in available literature. No data are currently available on the mutagenicity, carcinogenicity, chronic toxicity, or reproductive toxicity of dithiane. The acute oral LD₅₀ of dithiane in male and female rats is 3,680 mg/kg and 2,768 mg/kg, respectively.

CAS Number: 505-29-3

Chemical Formula: C₄H₈S₂

IUPAC Name: 1,4-Dithiane

Important Synonyms and Trade Names: tetrahydro-1,4-dithiin, p-dithiane, diethylene disulfide

Chemical and Physical Properties

Molecular Weight: 120

Melting Point: 108-113°C (Berkowitz et al. 1978)

Boiling Point: 199-200°C (Buckingham 1982)

Solubility in Water: 3,000 mg/liter at 25°C (Estimated;
Lyman et al. 1982)

^{1/} Compiled from: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL), 1985. Physical, Chemical, and Toxicological Data Summaries for 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Log Octanol/Water Partition Coefficient (K_{ow}): 0.77 (Lyman et al. 1982)
Fragment Method

Soil/Water Partition Coefficient (K_{oc}): Not Applicable

Bioconcentration Factor: Not Applicable

Vapor Pressure: 0.80 mm Hg at 25°C (Berkowitz et al. 1978)

Henry's Law Constant: Not Applicable

Transport and Fate

Given the vapor pressure of dithiane, volatilization to the atmosphere will likely occur. Atmospheric oxidants such as O_3 and NO_x may react with dithiane and thus provide a removal pathway (Berkowitz et al. 1978). Dithiane is reported to readily oxidize to sulfoxides and sulfones (Schroyer and Jackman 1947). Foote and Peters (1971) noted that dithiane is readily photooxidized to the sulfoxide in non-aqueous solvents. The presence of water may, however, facilitate photooxidation (Berkowitz et al. 1978). Though no supportive data are available, dithiane may be vulnerable to photooxidation and/or to oxidation by dissolved oxidants in the water column.

Little sorption of dithiane to soils is expected to occur given its high solubility in water. The combined low organic partitioning and high water solubility of dithiane suggests that this compound will exhibit a high degree of environmental mobility. Though no data on biodegradation were located, this may occur as dithiane contains the elements carbon and sulfur -- microbial nutrients. Data on the uptake of dithiane in plants was not located. It appears likely that some plant uptake could occur in light of dithiane's water solubility; however, definitive data were not located. Data on the persistence of dithiane in environmental media were not located in available literature.

Bioconcentration data for dithiane were not located in available literature. However, given the high aqueous solubility and low organic partitioning behavior, bioconcentration would not be expected to occur.

Health Effects

No data on the toxicity of dithiane to humans was located in available literature. Mutagenicity, carcinogenicity and chronic toxicity data are not available for dithiane. In a 14-day oral range-finding study in rats, doses of 0, 25, 50, 100, 210, and 420 mg/kg/day did not result in overt toxicity or mortality. A five percent decrease in overall weight gain occurred in males and an eight percent decrease in females at the high dose (Schieferstein 1986). A follow-up subchronic (90 day) toxicity study utilizing doses of 0, 105, 210, and 420 mg/kg/day revealed no overt signs of toxicity or treatment-related mortality. Preliminary results indicated no treatment-related clinical chemical, hematologic or ophthalmologic defects. Female high dose livers weighed more than control medium or low dose livers. Male right and left kidneys weighed more in treated groups than in controls. Deposition of unknown crystals in the turbinates of both sexes occurred in medium dose males and medium and low dose females. Furthermore, high dose males exhibited increased incidence of cytoplasmic eosinophilic granulization of cortical renal cells (Schieferstein 1986).

The acute oral LD₅₀ of dithiane in male and female rats was 3,680 mg/kg and 2,768 mg/kg, respectively (Mayhew and Muni, 1986). Antemortem observations included crusty muzzle, hyperactivity, muscle tremors, red stained fur around the eyes, emaciation, crusty eyes, lethargy, few or no stools, ataxia, squinting, prostration, lacrimation, irregular breathing, damp fur and yellow/brown stained fur in the perianal region. Necropsy revealed red or dark red lung discolorations; discoloration on parts or all of the small intestine; gastrointestinal contents were dark, thick, red, fluid and/or white; black stomach discoloration; pale or tan discoloration of the liver and a solitary red cyst occurred on the left ovary of one female in the high dose (3,981 mg/kg) group.

$$\begin{aligned}
 D_T &= \frac{\text{LOEL (mg/kg/day)}}{\text{UF} \times \text{SF}} \\
 &= \frac{105}{1,000 (5)} \\
 &= 0.02 \text{ mg/kg/day}
 \end{aligned}$$

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ENDRIN^{1/}

Summary

Endrin, an isomer of dieldrin, is an insecticide belonging to the chemical class of cyclodienes. It is retained in soils and sediments and is very persistent in the environment by virtue of its structure and physical/chemical properties. It is readily bioaccumulated by aquatic organisms. Endrin is acutely toxic to mammals, aquatic organisms, and terrestrial wildlife. It has not yet been shown to be carcinogenic or mutagenic, but is is a potent teratogen and reproductive toxin.

CAS Number: 72-20-8

Chemical Formula: $C_{12}H_8Cl_6O$

IUPAC Name: 1,2,3,4,10-Hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a octahydro-endo-1,4:5,8-dimethanonaphthalene

Important Synonyms and Trade Names: Endrin, hexadrin, mendrin

Chemical and Physical Properties

Molecular Weight: 380.9

Melting Point: Decomposes at 235°C

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Specific Gravity: 1.65 at 25°C

Solubility in Water: 0.25 mg/liter at 25°C

0.23 mg/liter at 25°C (Rosenblatt et al. 1975)

Solubility in Organics: Soluble in acetone, benzene, carbon tetrachloride, hexane, and xylene

Log Octanol/Water Partition Coefficient (K_{ow}):

5.6

5.34 (Kenaga 1980)

3.2 (Rao and Davidson 1983)

4.44 (Kadeg et al. 1986)

Soil/Water Partition Coefficients (K_{oc}):

1,310 - 18,200

Lyman et al. (1982) Eqn 4-8 ($\log K_{ow} = 3.2 - 5.3$)

37,500 - 3,400

Lyman et al. (1982) Eqn 4-5

($S = 0.02 - 0.2$)

34,000

Kenaga (1980) Table III

897; 8,290; 38,800

Lyman and Loreti (1986) Eqn I ($\log K_{ow} = 3.2,$

4.44, 5.3)

924; 10,000; 52,100

Lyman and Loreti (1986) Eqn II ($\log K_{ow} = 3.2,$

4.44, 5.3)

922; 9,700; 49,600

Lyman and Loreti (1986) Eqn III ($\log K_{ow} = 3.2,$

4.44, 5.3)

1,250; 9,700; 40,000

Kadeg et al. (1986) ($\log K_{ow} = 3.2, 4.44, 5.3$)

3,630

Kadeg et al. (1986), (geo. mean 2 literature values)

Bioconcentration Factor:

4,050

Kenaga (1980) Table 3 (experimental)

1,360

Kenaga (1980) Table 3 (experimental)

2,377

Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 5.34$)

1,415.7

Davies and Dobbs (1984) Eqn A ($S = 0.23$)

5,012

Davies and Dobbs Table 2 (experimental)

6,736

Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 5.34$)

1,043

Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 5.34$)

250

Davies and Dobbs (1984) Eqn C ($\log K_{ow} = 4.44$)

690

Davies and Dobbs (1984) Eqn B ($\log K_{ow} = 4.44$)

1,390

Lyman et al. (1982) Eqn 5-2 ($\log K_{ow} = 4.44$)

1,640

Argyle (1973) (experimental)

13,000

Hermanutz (1978) (experimental)

Vapor Pressure: - 2.7×10^{-7} mm Hg at 25°C (Rao and Davidson 1983)
 2.0×10^{-7} mm Hg at 25°C (Rosenblatt et al. 1975)

Henry's Law Constant: 4.4×10^{-7} atm-m³/mole (calculated)

Transport and Fate

Endrin is quite persistent in the environment. Volatilization from soil surfaces and from surface water is not likely to be an important transport process (Nash 1983) in light of its very low vapor pressure. For the small portion that may volatilize, photolysis to delta-keto endrin and endrin aldehyde are important chemical fate processes.

A range of estimated soil-water partition coefficients (K_{OC}) is reported above and indicates that sorption of endrin to soils/sediments and dissolved organic materials will occur. Pavlou (1980) estimates that sorption of organochlorine pesticides such as endrin is very high. The combined low water solubility and high organic partitioning of endrin indicates that little environmental mobility will occur. Rosenblatt et al. (1975) report less than 10 cm of movement in situ following 150 cm of rainfall. Microbial degradation by soil microorganisms occurs but appears to be limited (Rosenblatt et al. 1975). The extent of utilization and the decomposition products were not reported. Endrin has a relatively long half-life in soil which may span upwards of 10 years.

Uptake in plants varies with species. For example, root crops (potatoes) grown in treated soil exhibited levels about twice that of the soil in which they were grown (Telekar et al. 1983). Levels in pasture crops appear to be less than those in soil (Chawla et al. 1981).

A range of experimental and estimated bioconcentration factors (BCFs) for endrin is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via

biomagnification of residues up food chains. The magnitude of the concentration factors suggests that bioconcentration and potential biomagnification will occur.

Health Effects

Endrin has not been shown to be carcinogenic in several animal studies including the National Cancer Institute bioassay (50 Federal Register 47011, Wed. Nov. 13, 1985). Endrin has not been shown to be mutagenic in microbial systems with or without activation (50 Federal Register 47011). Endrin has been classified according to EPA's Carcinogenic Risk Assessment Guidelines in EPA's Group E (no evidence of carcinogenicity for humans) based on negative results in several animal studies.

Endrin is a potent reproductive toxin and teratogen in experimental animals. Reproductive effects include fetal mortality and growth retardation, while teratogenic effects included cleft palate, open eye, clubbed foot, meningoencephales and fused ribs. Chronic exposure to low levels of endrin results primarily in nervous system damage; however adverse effects to the heart, lungs, liver, and kidneys also occur. The acute toxicity of endrin is due to its effects on the central nervous system. The acute oral LD₅₀ is 3 mg/kg in the rat and 1.37 mg/kg in mice (Sax 1979).

Toxicity to Wildlife and Domestic Animals

Endrin is very toxic to aquatic organisms. Freshwater fish were generally more sensitive than invertebrates, with species mean acute values ranging from 0.15 to 2.1 µg/liter (USEPA 1980). LC₅₀ values for saltwater organisms ranged from 0.037 to 14.25 µg/liter. Final acute values for freshwater and saltwater species were 0.18 µg/liter and 0.037 µg/liter, respectively (USEPA 1980). An acute-chronic ratio of 4.0 was determined from chronic tests on freshwater and saltwater species. Therefore, the freshwater final chronic value was calculated to be 0.045 µg/liter and the saltwater final chronic value was determined to be 0.0093 µg/liter (USEPA 1980).

Endrin is acutely toxic to terrestrial wildlife and domestic animals and has been used as a rodenticide and an avicide. It can also cause central nervous system effects and reproductive disorders following chronic exposure. Other effects observed in animals exposed to endrin include abnormal behavior, increased postnatal mortality, and increased fetal death. The LD₅₀ values for a variety of birds are 5.64 mg/kg (mallard), 1.1 mg/kg (grouse), 1.2 mg/kg (quail) and 1.8 mg/kg (pheasant). In terrestrial mammals, such as the mule deer and the domestic goat, the range of acute oral LD₅₀s are 6.25 - 12.5 mg/kg and 25-50 mg/kg, respectively (Hudson et al., 1984).

Regulations and Standards

Ambient Water Quality Criteria (USEPA 1986):

Aquatic Life (Freshwater)

Acute toxicity: 0.18 µg/liter

Chronic toxicity: 0.0023 µg/liter

Aquatic Life (Saltwater)

Acute toxicity: 0.037 µg/liter

Chronic toxicity: 0.0023 µg/liter

Human Health

Criterion: 1.0 µg/liter (water and fish ingestion)

National Interim Primary Drinking Water Standard (USEPA):

0.0002 mg/liter (MCL; 40 CFR 141.12 Subpart B)

OSHA Standard: $TWA^{1/} = 100 \text{ mg/m}^3$

1/ Time Weighted Average

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For endrin, the D_T value is based on the same oral subchronic toxicity study (Treon and Cleveland, 1955) used to establish the current Interim Maximum Contaminant Level (MCL). The study assessed the toxicity of dietary endrin (1, 3 or 4 ppm) in dogs over a period of 18.7 months. Body weight gain was depressed in the 4 ppm but not in the 1 or 3 ppm groups. Kidney and heart weights were significantly greater in the 3 ppm but not the 1 ppm group. EPA recently modified the estimated food consumption basis for dogs on which the original MCL was based to reflect actual consumption data (50 Federal Register 47011 Wednesday November 13, 1985). Utilizing this measured food intake, the No-Observed-Adverse-Effect-Level (NOAEL) from the study is recomputed as 0.045 mg/kg/day. An Uncertainty Factor (UF) of 1,000 is employed to address extrapolation of the results to humans (10), intraspecies variability (sensitive subgroups) (10) and use of a subchronic rather than a chronic toxicity study (10). Derivation of the D_T for endrin is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOAEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{0.045}{1,000} \\ &= 0.000045 \text{ mg/kg/day} \end{aligned}$$

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ETHYLBENZENE^{1/}

Summary

There is some evidence suggesting that ethylbenzene causes adverse reproductive effects in animals. Oral and inhalation exposures have caused minor liver and kidney changes in rats. The central nervous system is also a target organ following exposures to ethylbenzene. Ethylbenzene is a skin and eye irritant.

CAS Number: 100-41-4

Chemical Formula: $C_6H_5C_2H_5$

IUPAC Name: Ethylbenzene

Important Synonyms and Trade Names; Phenylethane, EB, ethylbenzol

Chemical and Physical Properties

Molecular Weight: 106.2

Boiling Point: 136.2°C

Melting Point: -95°C

^{1/} Compiled from: U.S. Environmental Protection Agency, Office of Waste Program Enforcement. September 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. A Final Report Prepared by Clement Associates, Inc., Arlington, Virginia.

Also: United States Army Medical Bioengineering Research and Development Laboratory (USAMBRDL). 1985. Physical, Chemical, and Toxicological Data Summaries of 62 Compounds Present at Rocky Mountain Arsenal. USAMBRDL. Fort Detrick, Frederick, MD.

Specific Gravity: 0.867 at 20°C (liquid)

Solubility in Water: 161 mg/liter at 25°C

140 mg/liter at 15°C (TDB Peer Review Committee,
1984)

Solubility in Organics: Freely soluble in organic solvents

Log Octanol/Water Partition Coefficient (K_{ow}): 3.13 (Tewari, et al.,
1982)

3.15 (Hansch and Leo,
1979; Moriguchi 1975)

Soil/Water Partition Coefficient (K_{oc}):

95	Sabljić (1984) (experimental)
277	Lyman et al. (1982) Eqn 4-5 (S = 150)
1,230	Lyman et al. (1982) Eqn 4-8 (log K_{ow} = 3.15)
820	Lyman and Loreti (1986) Eqn I (log K_{ow} = 3.15)
840	Lyman and Loreti (1986) Eqn II (log K_{ow} = 3.15)
839	Lyman and Loreti (1986) Eqn III (log K_{ow} = 3.15)
1,150	Kadeg et al. (1986) (log K_{ow} = 3.15)

Bioconcentration Factor:

109	Davies and Dobbs (1984) Eqn B (log K_{ow} = 3.1)
37.5	ECAO (1980)
67	Davies and Dobbs (1984) Eqn C (log K_{ow} = 3.14)
141	Lyman et al. (1982) Eqn 5-2 (log K_{ow} = 3.13)
146	Lyman et al. (1982) Eqn 5-2 (log K_{ow} = 3.15)
36.6	Davies and Dobbs (1984) Eqn A (S = 150)
117	Davies and Dobbs (1984) Eqn B (log K_{ow} = 3.15)
54.6	Davies and Dobbs (1984) Eqn C (log K_{ow} = 3.15)

Vapor Pressure: 7 mm Hg at 20°C

10 mm Hg at 25.9°C (TDB Peer Review Committee, 1984)

Vapor Density: 3.66

Flash Point: 17.2°C

Henry's Law Constant: 6.44×10^{-3} atm-m³/mole (USEPA 1985)

Transport and Fate

Only limited data are available on the transport and fate of ethylbenzene. Volatilization is probably the major route of elimination from surface water and soil surfaces. Once in the atmosphere, the primary chemical process involved in the breakdown of ethylbenzene is photooxidation.

A range of experimental and estimated soil-water partition coefficients (K_{oc}) is reported above and indicates that sorption of ethylbenzene to soils/sediments and dissolved organic material will occur. Pavlou (1980) estimates that sorption of semipolar organic compounds will range from low to moderate. The combined water solubility and organic partitioning data suggest that ethylbenzene will exhibit some degree of environmental mobility. Some soil bacteria are capable of using ethylbenzene as a carbon source. Microbial degradation would therefore contribute to the loss of ethylbenzene from environmental media. Ethylbenzene is likely to be fully converted to CO_2 and water as a result of microbial degradation processes.

A range of estimated bioconcentration factors for ethylbenzene is also reported above. ASTM (1985) indicates that chemicals with bioconcentration factors less than approximately 100 have low potential for causing harm to wildlife and human health via biomagnification of residues up food chains. The magnitude of the concentration factors suggests that appreciable bioconcentration or biomagnification of ethylbenzene residues is not likely to occur.

Health Effects

In humans, exposure to ethylbenzene at concentrations of 269 mg/m^3 resulted in a decrease in performance on tests of light perception, reaction time and short-term memory (USEPA 1985). Workers exposed to ethylbenzene in addition to other chemicals for greater than 5 years exhibited elevated serum ornithine carbamoyltransferase

D_T Value

The D_T value is defined as that contaminant intake rate (mg/kg/day) that should not induce an adverse effect to human health or should not pose a risk of cancer occurrence greater than a predetermined risk level.

For ethylbenzene the D_T value is based on the same data used to compute the current Risk Reference Dose (RfD) (USEPA 1986b). The RfD is based on a subchronic (182 days) oral toxicity study in which female rats were dosed orally 5 days/week with 13.6, 136, 408 or 680 mg/kg/day ethylbenzene (Wolf et al. 1956). The criteria considered in assessing toxicity to test animals were effects on growth, mortality, appearance and behavior, hematological findings, terminal blood urea nitrogen (BUN), final average organ and body weights, histopathological findings and bone marrow counts. The Lowest-Observed-Adverse-Effect-Level (LOAEL) of 408 mg/kg/day was associated with histopathological changes in the liver and kidney. The identified No-Observed-Effect-Level (NOEL) from this study was 136 mg/kg/day. This value was adjusted for the 5 day per week exposure duration to yield an adjusted NOEL of 97.1 mg/kg/day (136 mg/kg/day x 5 days/7 days). An Uncertainty Factor (UF) of 1,000 is employed to address extrapolation of the results to humans (10), intra-species variability (sensitive subgroups) (10) and a factor of 10 to account for the less than lifetime (chronic) experimental exposure. Derivation of the D_T (RfD) is as follows:

$$\begin{aligned} D_T &= \frac{\text{NOEL (mg/kg/day)}}{\text{UF}} \\ &= \frac{97.1}{1,000} \\ &= 0.097 \\ &= 0.1 \text{ mg/kg/day} \end{aligned}$$

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