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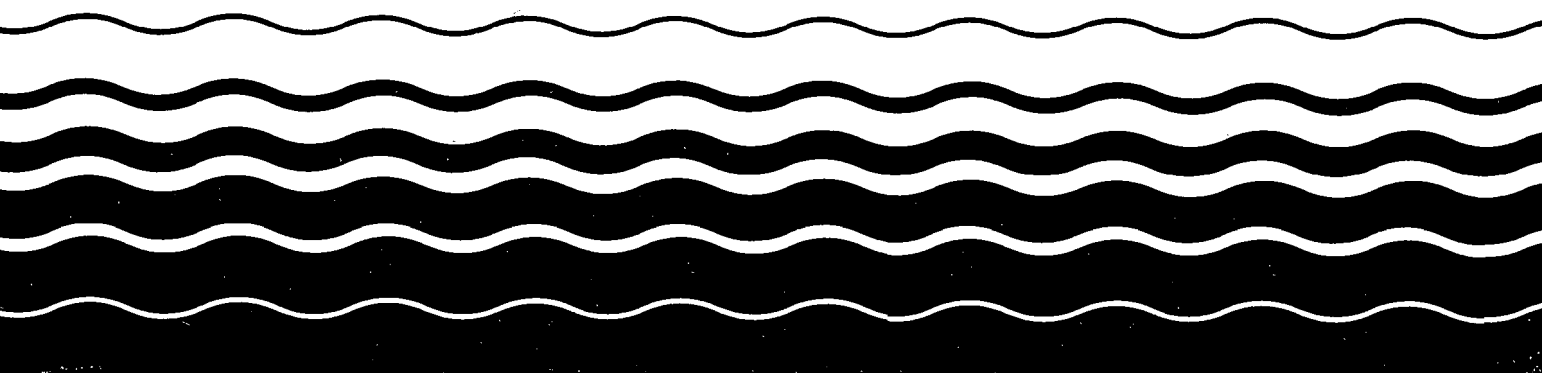
An Exposure and Risk Assessment for Trichloroethylene



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16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to trichloroethylene. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in March of 1981.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of trichloroethylene in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to trichloroethylene for various subpopulations.

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AN EXPOSURE AND RISK ASSESSMENT FOR TRICHLOROETHYLENE

by

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OFFICE OF WATER REGULATIONS AND STANDARDS
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FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

Michael W. Slimak, Chief
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EXECUTIVE CONCLUSIONS[1]

Trichloroethylene (TCE) is a synthetic chemical widely used as a solvent. It is now ubiquitous in the environment, particularly as an air pollutant. Concern about exposure to TCE stems from its toxicity and suspected carcinogenicity.

TCE is now produced from 1,2-dichloroethane at a rather steady level of about 130,000 metric tons per year, a volume perhaps one-half of the production 10 years ago. Ninety-two percent of the TCE production is consumed as a degreasing solvent, mostly for cleaning metal, secondarily for washing undyed fabrics. Recycle of TCE is practiced to some extent, resulting in total solvent use being perhaps 17% greater than solvent production and consumption. Four percent of production is consumed as a chemical feedstock, and four percent for other uses including fungicide. A variety of former uses have been discontinued, such as for coffee decaffeination and other food and cosmetic uses, and for anesthesia, dry cleaning, and paint formulation.

The entire TCE production is ultimately released to the environment, except for the 6% which is consumed as a feedstock or destroyed by incineration. During or following use or reuse, perhaps 79% of production is released to air, 14% to land, and 1% to ambient waters. Around 2% of this total release occurs through municipal conveyance or treatment systems; most of this volatilizes before being discharged to surface waters.

Environmental partitioning of TCE favors air rather than water; water concentrations in equilibrium with commonly occurring air concentrations would be in the undetectable ng/l range. Surface water concentrations above this level can be expected to volatilize, with half life often ranging from hours to weeks. In the atmosphere, TCE is destroyed by photo-oxidation, with a half life of about 1 day.

If TCE were completely stable chemically in water and soil, then virtually all of the quantity disposed of to land could ultimately be expected to migrate away from the disposal site, either by volatilizing into the atmosphere or by leaching into groundwater. However, since TCE dissolved in water may slowly hydrolyze, with a reported half life of about 1 year, a substantial amount of degradation might occur over the years. Whether current disposal technology is capable of containing TCE until chemical degradation has occurred is, however, not known. (With respect to kinetics in any medium, it should be noted that more than three half lives are required to achieve a 90% reduction and almost seven half lives for a 99% reduction.)

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[1]Prepared by EPA Technical Project Officer based in part on program considerations.

Ambient environmental data indicates that urban air tends to have elevated TCE levels. It is detected in surface and groundwater supplies with about equal frequency; however, the higher concentrations tend to be found in groundwater supplies. The effect these levels have on human health hinges partially on whether or not TCE is carcinogenic, a controversial question due to the existence of some positive indications on one hand, but lack of unambiguous and consistent results on the other.

Exposure to technical grade TCE has been shown to result in an apparent increase in liver cancer in one strain of mice. The validity of this finding has been questioned primarily on the basis of high spontaneous liver cancer rates indigenous to the mouse strain, and the presence of strongly mutagenic stabilizing chemicals (epoxides) added to technical grade TCE. Tests with pure TCE on three other species failed to show liver carcinogenicity; however, one sex of one species showed an increase in lymph cancer, attributed to an immunity suppressing effect at high concentrations, rather than a cancer initiating effect. Several other studies (including one of human epidemiology) failed to detect carcinogenesis in a variety of species.

TCE is toxic to the liver, kidneys, heart, and nervous system. However, a criterion derived to prevent such toxicity would be substantially higher than one intended to reduce cancer risks to negligible levels, assuming that TCE is as carcinogenic as some of the mouse studies suggest. For prevention of non-carcinogenic toxicity, the Water Quality Criteria document sets the threshhold at 800 µg/l, a level which would correspond to an estimated cancer risk of 3×10^{-4}. For prevention of more than a 10^{-5} excess cancer risk, the EPA water quality criterion is 27 µg/l. Thus, the severity of the environmental TCE problem is dependent on the carcinogenic potential of TCE, particularly pure grade TCE.

Environmental exposures to TCE, averaged nationwide, appear to result primarily from contamination of air, food, and water in descending order of importance. Considering all three routes, average exposure through water would be 2-10 percent of the total, depending on the estimate used for the mean air exposure.

If all carcinogenicity tests yielding negative results are ignored, and the mouse tests showing the most positive results are linearly extrapolated to low doses using the EPA Cancer Assessment Group model, then the magnitude of the nationwide risks would be roughly estimated as shown below. The assumptions incorporated into such extrapolations suggest that these predictions be regarded as the maximum risks for which a plausible argument could be made, rather than the risks which might actually seem most probable.

EPA ESTIMATES OF CANCER RISKS FROM ENVIRONMENTAL EXPOSURE BASED ON AVERAGE NATIONWIDE CONDITIONS[2]

Route	Average TCE Level	Average Excess Lifetime Risk	Nationwide Incidence (cancers/year)
Ambient Air	0.2 - 2 $\mu g/m^3$	10^{-6} - 10^{-5}	2.5 - 23
Drinking Water	0.5 µg/l	2×10^{-7}	0.6
Food	6 µg/day	10^{-6}	4
TOTAL	11 - 47 µg/day	10^{-6} - 10^{-5}	7 - 28

The ranges of individual risks from exposure via some routes may differ considerably from the population averages shown above. While the average drinking water risk is 2×10^{-7}, 0.5% of the EPA nationwide survey data [NOMS and NOSP (GC-MS) samples] exceed the 27 µg/l criterion suggested for protection from more than a 10^{-5} risk. Moreover, other information indicates that some groundwater supplies are contaminated with up to 3000 µg/l, representing a possible cancer risk of 10^{-3} for lifetime exposure. In no case does contaminated fish appear to be a significant route.

Waterborne routes of exposure are rarely expected to result in non-cancer related toxicity. Only 1% of all STORET ambient data, and none of the above mentioned nationwide survey data of drinking water exceed even one-tenth of the 800 µg/l non-cancer related safe dose ceiling. Similarly, current ambient levels appear to pose little hazard to aquatic life, since toxicity has not been reported at less than 2,000 and 22,000 µg/l for salt and freshwater organisms, respectively.

EPA's Office of Water Regulations and Standards has concluded that:

(1) The benefits of reducing TCE exposure are partially dependent on the carcinogenic potency attributed to TCE, and are thus currently not completely certain.

(2) On the average, exposure to TCE is primarily through air and food, with waterborne routes contributing possibly 2-10 percent. In some instances, however, TCE concentrations in groundwater may attain levels possibly associated with substantial health risks.

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[2]Table 7-1 in the following report depicts individual excess cancer risks for specific environmental situations, whereas the values here are intended to reflect excess cancer risks associated with average nationwide conditions. Hence, the two tables are complementary.

(3) Due to rapid volatilization, wastewater borne TCE may affect air as well as water media. Volatilization from water does not appear to be one of the major sources of atmospheric TCE, however.

(4) The fate of TCE disposed of on land is not well enough understood to know whether it may eventually enter the environment by volatilizing or leaching. Nevertheless, its gradual rate of degradation appears to place some limit on the distance it can travel in groundwater. The bulk of TCE disposed of by recycle or incineration appears not to enter the environment.

Notes on Derivation of TCE Risks:

Unit Risk (Dose-Response)

Lifetime risk is based on NCI mouse study (as reported by CAG and the Water Quality Criteria document):

1.26×10^{-5} lifetime cancer risk/(mg/kg-day) TCE intake.

For a 70 kg person:

55 µg/day TCE intake corresponds to 10^{-5} risk.

Nationwide incidence is based on 70 year average lifetime for 220 million persons in U.S.

Population Risk

Air: The inhaled volume is 20 m^3/day. Two estimates for the mean exposure are based on:

(1) EPA OAQPS modeling: 34.5 million person-µg/m^3 exposure for 159 million persons implies 0.22 µg/m^3 mean exposure (Anderson et al. 1980).

(2) Lillian et al. (1975) data for 8 cities: mean concentration 2 µg/m^3.

Drinking Water: The ingested volume is 2 l/day. The mean concentration from NOMS is approximately 0.5 µg/l regardless of whether undetected levels are assumed to be a minimum of zero or a maximum of just below the detection limit. NOSP (GC-MS) data support this estimate. It may be noted that heating is likely to drive off the volatile organics; 2 l/day may overestimate the unheated water intake.

Food: Data from McConnell et al. (1975), projected to various classes of food in a standard diet, as described in Chapter 5.

Uncertainties in the exposure estimates arise from several factors, including (a) very small sample sizes on which the air, water, and food projections are based, (b) possible sampling biases (for example, the air data is applicable to outdoor urban air rather than either rural or indoor air), (c) possible inaccuracies in laboratory methods, (d) uncertainties in the effects of food and beverage preparation on TCE content, and (e) on other minor biases such as ignoring small differences between the uptake efficiencies of inhalation and ingestion. In addition to the uncertainties relating to exposures, there is considerable uncertainty regarding the validity of extrapolating dose-response data from laboratory animals to humans.

(References listed in Chapters 4 and 5.)

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ACKNOWLEDGEMENTS

The Arthur D. Little, Inc. task manager for this study was Richard G. Thomas. Major contributors to this report were Melanie Byrne and Kathy Saterson (Biological Effects), Diane Gilbert (Human Exposure) and Muriel Goyer (Human Effects). In addition, Kate Scow did the EXAMS analysis and contributed to Biological Effects; John Ostlund, Alan Eschenroeder, and Joseph Fiksel estimated dose/response parameters, and Jane Metzger and Nina Green edited and documented the report.

The materials balance for trichloroethylene (Chapter 3.0) was provided by Acurex, Inc., produced under contract 68-01-6017 to the Monitoring and Data Support Division (MDSD), Office of Water Regulations and Standards (OWRS), U.S. EPA. Kenneth Moss was the task manager for Acurex, Inc. Patricia Leslie was responsible for report production.

Charles Delos, MDSD, was the project manager at EPA.

1.0 TECHNICAL SUMMARY

The Monitoring and Data Support Division, Office of Water Regulations and Standards, the U.S. Environmental Protection Agency, is conducting an ongoing program to assess the risk associated with exposure to 129 priority pollutants in the environment. This report assesses the exposure and risk associated with trichloroethylene (TCE).

1.1 MATERIALS BALANCE

Trichloroethylene is a high-vapor-pressure organic solvent, used almost exclusively (>90%) in degreasing operations. The chemical has become pervasive in the environment due to fugitive emissions during production, use, and disposal.

Production of trichloroethylene has declined sharply from 206,000 kkg in 1973 to 136,000 kkg in 1978, mostly in response to declining demand and improved solvent recovery. About 90% of the total used is estimated to be discharged to the environment, and degreasing operations account for almost all the releases of this amount. Environmental releases are primarily to air due to evaporation of the chemical. Discharges to water account for much less of the total and the amount disposed of on land is also relatively small. However, land discharges may pose significant problems for groundwater quality.

Only three sites in the U.S. manufacture the chemical, all located along the Gulf Coast. Releases from production are only a small part of the total released each year.

1.2 FATE AND DISTRIBUTION IN THE ENVIRONMENT

1.2.1 Distribution of Trichloroethylene in the Environment

Trichloroethylene appears in all environmental media - air, water (including groundwater), and soil. Concentrations detected in the atmosphere range from a background of 27-100 ng/m^3 to about 1.5 mg/m^3 in the vicinity of TCE production sites. Concentrations found in surface water range from none to 188 µg/l. When detected, however, concentrations are generally on the order of 1-10 µg/l. In groundwater, concentrations up to 35000 µg/l have been detected, but monitoring surveys indicate concentrations to be mostly in the <1-100 µg/l range. Data on only a few soil samples were found. These showed soil concentrations between none detected and <10 µg/kg (dry weight). Data concerning concentrations in sediments are too few to indicate specific sorption characteristics, although TCE does sorb and has been found in sediments.

1.2.2 Environmental Fate of Trichloroethylene

TCE is highly volatile. The half-life of trichloroethylene in surface waters is estimated to be on the order of a few hours to a few days, depending upon the characteristics of the water body. TCE will also volatilize from soil if exposed to the air, although estimates of the volatilization rate are imprecise. The process appears to occur up to ten times more slowly from soil than from water of a similar depth.

The ultimate disposition of TCE appears to be atmospheric photooxidation. The half-life in the atmosphere due to photooxidation is estimated to be a day or so, possibly one-half day to a couple of days. TCE does not undergo other chemical fate processes to any appreciable degree, although chemical degradation in groundwater may be significant, with a half-life on the order of a year. On the basis of this information, the fate of TCE is expected to be destruction by atmospheric photooxidation following direct emission or volatilization from water or soil.

Models of environmental fate indicate that changes in ambient concentrations are linearly dependent upon changes in environmental loadings. The EXAMS model indicates that most TCE is volatilized; transformation and bioaccumulation will not reduce water concentration significantly. These results are consistent with observations regarding fate.

1.3 RISKS TO HUMANS

1.3.1 Human Effects

Oral administration of TCE was carcinogenic in B6C3F1 mice including a 52% incidence of hepatocellular carcinoma in male mice at a level of 1170/mg/kg bw. Some controversy exists, however, regarding the design of this study. Repeated dermal application and initiation-promotion studies with Ha:ICR Swiss mice were negative and intubation of 1.5 mg TCE/mouse/week produced no tumors in liver, lungs or stomach in this strain. No carcinogenic activity was found in Osborne-Mendel rats given TCE by gavage; however, survival was poor, reducing the ability to detect a carcinogenic response in this study.

Negative results were reported in a dominant lethal assay with rats exposed by inhalation to 1614 mg/m^3 TCE for 9 months, and exposure of mice (1614 mg/m^3) and rats (9684 mg/m^3) during gestation gave no indication of fetotoxic or teratogenic effects. Positive mutagenic effects have been reported in a mammalian cell transformation assay and weak responses in bacterial and yeast test systems, but only if microsomal activation was provided.

The predominant toxic manifestation of TCE exposure in man is depression of the central nervous system. Transient increases in serum transaminases (an indication of liver toxicity) have also been

reported, but both these effects are reversible upon removal from the source. Intolerance to alcohol is a well-documented symptom of repeated TCE exposure. High acute doses have produced cardiac arrhythmias and death by ventricular fibrillation and cardiac arrest. The lowest reported oral lethal dose in man is 50 mg TCE/kg body weight.

1.3.2 Human Exposure to Trichloroethylene

Humans can be exposed to TCE by the presence of TCE in all environmental media - air, water, and soil - and through drinking water and foodstuffs. Exposures have been estimated based upon activity and location. Inhalation exposures range from 0.0006 to 14 mg/day for non-occupational activities and up to 5800 mg/day for occupational activities. Ingestion via foodstuffs and water has been estimated at $\leq$ 0.0006 to 6 mg/day. Thus ingestion of water (2 liters per day) contaminated at the maximum estimated concentration of TCE found in water (i.e., 3 mg/l) would contribute 6 mg/day to the total body burden of TCE, an amount less than one-half that possible from inhalation at the upper bound of non-occupational inhalation exposure (i.e., 14 mg/day), but could potentially exceed the lower bound of non-occupational inhalation exposure (0.0006 mg/day) by several thousand-fold. However, findings of the maximum estimated TCE concentration in water are an uncommon event, and the generally observed levels of TCE in drinking water would result in intakes (0.004 mg/day) substantially lower than those possible from non-occupational inhalation exposure, but roughly comparable to the estimated upper limit for intake from food (0.006 mg/day).

1.3.3 Risk Considerations for Humans

Exposure levels to individuals have been estimated for different exposure conditions. Dose/response extrapolations based on three models have been applied to these exposure levels using data from one positive study in B6C3F1 mice to estimate risk levels. Risk estimates of excess individual lifetime tumor incidence associated with TCE intakes due to inhalation range from 4×10^{-8} to 8×10^{-4}, corresponding to background TCE concentrations in air and high ambient concentrations near users, respectively. Estimated excess individual lifetime cancer risk due to continuous lifetime consumption of drinking water contaminated at the average observed TCE levels is in the $<10^{-7}$ to 10^{-6} range. At the highest TCE concentrations observed in drinking water, estimated excess individual lifetime cancer risk levels are $\approx 10^{-3}$.

Considerable controversy exists regarding the most appropriate method for extrapolating human equivalent doses from animal data. Due to this uncertainty, the range of risk estimated by the various extrapolation models may under- or overestimate the actual risk to man. Overestimation appears more likely due to the conservative assumptions utilized in the calculation of human equivalent doses.

Other than carcinogenic risks, acute human exposure to TCE is of concern only at high exposure concentrations. Inhalation of 800 mg/m^3 TCE for 2 hours can depress the central nervous system, while ingestion of 150 ml TCE can result in acute renal failure and cardiovascular damage. Data on chronic human exposure to TCE are not available. No-effect levels ranging from 540 to 2050 mg/m^3 have been established for laboratory animals exposed for periods up to 6 months.

1.4 RISKS TO AQUATIC BIOTA

1.4.1 Toxic Effects of Tricholoethylene to Aquatic Biota

Under laboratory conditions TCE has been shown to be lethal to fish and other aquatic organisms, and to affect such functions as equilibrium, respiration, and reproduction. Acute effects levels for fish are 40-79 mg/l (LC_{50}). One test for chronic effects showed a 48-hour LC_{50} for Daphnia of 85 mg/l and no effect during a chronic test at 10 mg/l. For 24 hr - 96 hr tests, the LC_{50} for fathead minnows was 22 - 23 mg/l and LC_{50}'s are in the same range: 16 - 100 mg/l. Algae showed toxic effects at 8 mg/l.

Results of the studies cited show most effects levels to be in the 10 - 100 mg/l range in most freshwater and marine fish.

1.4.2 Exposure of Aquatic Biota to Trichloroethylene

Ambient water concentrations of TCE seem to fall in the not detected to 10 µg/l range, although some concentrations in particular areas are higher. The highest reported was 5.3 mg/l in the vicinity of a TCE manufacturing plant. Significant exposures to TCE might, therefore, occur in extremely localized areas; however, most aquatic biota will experience little or no exposure. The high volatility of TCE also indicates that any high concentrations are not likely to persist for periods of longer than hours or days unless direct inputs at particular locations continue.

1.4.3 Risk Considerations for Aquatic Biota

Chronic and acute toxicity levels of tricholorethylene for aquatic biota are greater than 10 mg/l. The highest reported ambient concentration was 5.3 mg/l in the vicinity of a manufacturing plant. Other detected concentrations are much lower; consequently, biota will not be exposed to levels that overlap with those causing toxic effects.

On the basis of these considerations, the risk to aquatic biota due to trichloroethylene in ambient waters is determined to be negligible.

2.0 INTRODUCTION

The Office of Water Regulations and Standards, Monitoring and Data Support Division, the U.S. Environmental Protection Agency, is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated include potential harm to human beings and deleterious effects on fish and other biota. The goal of the task under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and estimate the risk based on receptor exposure to these substances. The results are intended to serve as a basis for developing suitable regulatory strategy for reducing the risk, if such action is indicated.

This report provides a brief, but comprehensive, summary of the production, use, distribution, fate, effects, exposure, and potential risks of trichloroethylene.

The report is organized as follows:

Chapter 3.0 presents a materials balance for trichloroethylene that considers quantities of the chemical consumed or produced in various processes, the form and amount of pollutant released to the environment, the environmental compartment initially receiving it, and, to the degree possible, the locations and timing of releases.

Chapter 4.0 describes the distribution of trichloroethylene in the environment by presenting available monitoring data for various media and by considering the physicochemical and biological fate processes that transform and transport the chemicals.

Chapter 5.0 describes the available data concerning the toxicity of trichloroethylene for humans and laboratory animals and quantifies the likely level of human exposure via major known exposure routes.

Chapter 6.0 considers toxicological effects on and exposure to biota, predominantly aquatic biota.

Chapter 7.0 compares exposure conditions for humans and other biota with the available data on effects levels from Chapters 5.0 and 6.0. The risks presented by various exposures to the trichloroethylene are estimated.

Appendices A-G present more detailed information supporting materials balance estimates in Chapter 3.0. Appendix H discusses the procedure for estimating the volatilization rates of trichloroethylene.

3.0 MATERIALS BALANCE

3.1 INTRODUCTION

This chapter presents an environmental materials balance for trichloroethylene.

As matter is neither created nor destroyed in chemical transformations, the total mass of all materials entering a system equals the total mass of all materials leaving that system, excluding those materials the system accumulates or retains. From the perspective of risk analysis, a materials balance may be performed around any individual operation that serves to identify a specific population at risk (e.g., process water discharges creating ground water contamination). An environmental materials balance, therefore, consists of a collection of materials balances, each of which is directed to a specific source and sink within the environment.

The scope of this report has been limited to a review of both published and unpublished data concerning the production, use, and disposal of trichloroethylene within the United States. Available literature has been critiqued and compiled in a single document to present an overview of major sources of environmental release of trichloroethylene and fully annotated tables to aid data evaluation.

3.2 SUMMARY

The flow of trichloroethylene through the environment is shown in Figure 3-1. Trichloroethylene is found in all media of the environment. Available literature indicates it is solely man-made. Like other low molecular-weight chlorinated hydrocarbons, trichloroethylene has a relatively high vapor pressure and low water solubility and thus tends to partition toward the atmosphere.

Historically, contamination of groundwater supplies has been tied to disposal practices. As discussed in Section 3-5, trichloroethylene pollution of groundwater and drinking water supplies appears largely attributable to migration and leaching of the chemical from unlined or improperly lined landfills, settling ponds, old dredge pits, open fields, and old industrial sludge pits. Documented incidents of environmental damage resulting from improper management of trichloroethylene-containing wastes are listed in Appendix G. In many cases, the specific source or generator of the waste is unknown or not easily identified. However, in the cases where a particular point source can be isolated, metal degreasing operations have been the primary contributors of the trichloroethylene wastes.

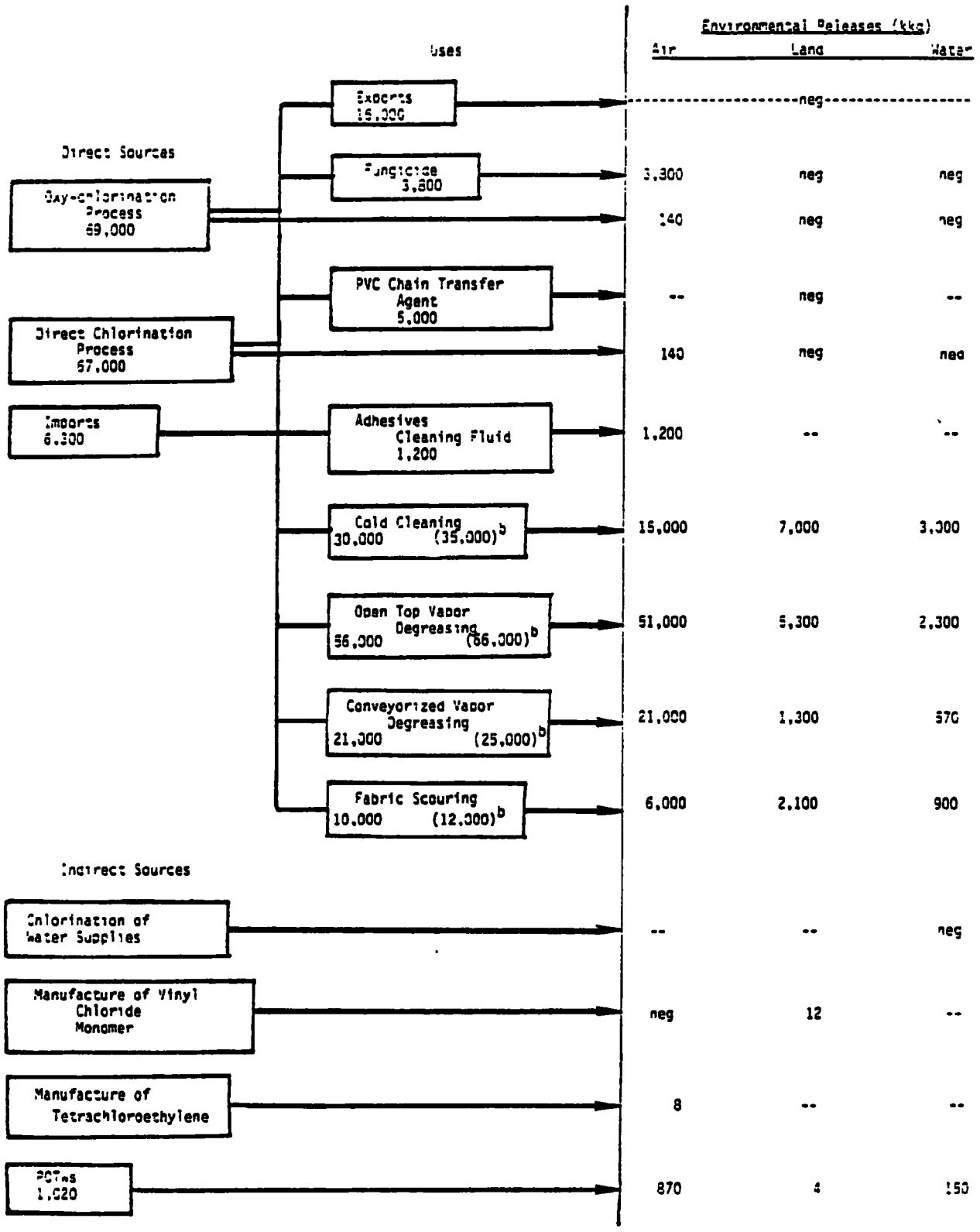

Figure 3-1. U.S. Trichloroethylene Materials Balance, 1978[a]

a) Totals reported to two significant figures and may not add due to rounding; uses not balanced by environmental releases are assumed contained in product or recycled.

b) Numbers in parentheses represent total solvent usage (virgin + recycle from previous use); environmental releases are calculated from these figures (See Table 3-5).

To assess and understand the impact of trichloroethylene in terms of demonstrated and potential ground water degradation, estimates of the cumulative amounts of trichloroethylene released to air, water, and, most importantly, land over the period of 1954-1978 are presented. Table 3-6 presents a summary of cumulative releases to land and water based on a simplified scenario in which only losses from degreasing operations are considered. As shown, an estimated 730,000 kkg of trichloroethylene were dumped on land and an additional 320,000 kkg released to water during the study period. This represents only the "worst case" accumulation over the past 25 years; indeed, an indeterminate yet significant amount will have volatilized from surface waters or open impoundments during this time. Atmospheric degradation is the primary sink for trichloroethylene. It is destroyed rapidly in the atmosphere. (See Chapter 4.0.)

Section 3.3 of this chapter investigates production of trichloroethylene, from either direct or inadvertent sources. Trichloroethylene is manufactured (as a coproduct with tetrachloroethylene) by either chlorination or oxy-chlorination of 1,2-dichloroethane or other C_2 chlorinated hydrocarbons. All of the 136,000 kkg of trichloroethylene produced in the United States during 1978 was from 1,2-dichloroethane based processes; production sites were limited to Dow Chemical Corp., Freeport, Texas; Ethyl Corp., Baton Rouge, Louisiana; and PPG Industries, Lake Charles, Louisiana. (See Table 3-1.) Prior to 1978, 8 percent of U.S. trichloroethylene production was via an acetylene-based process, which has been abandoned due to increasing feedstock costs.

Few data are available regarding emissions of trichloroethylene during production. Major point sources of atmospheric emissions are neutralization and drying vents and distillation vents; an estimated 280 kkg were dispersed to the air from trichloroethylene manufacture during 1978. Process discharges to land and water were negligible. (See Table 3-1.)

The second part of Section 3.3 is a discussion of inadvertent sources of trichloroethylene. Trichloroethylene emissions originate from the production of other chlorinated hydrocarbons--specifically during vinyl chloride monomer (VCM) and tetrachloroethylene production. However, since much of the waste tars are recyclable (or incinerated) and water emissions are negligible, trichloroethylene discharges from these sources are not significant. (See Table 3-2.)

Another possible inadvertent source of chlorinated hydrocarbons might be chlorination of drinking water and wastewater. Trichloroethylene has been identified in drinking water supplies in the ug/l range; however, the source of such chlorinated organic chemicals cannot be readily identified (e.g., whether they are in the raw water prior to treatment or introduced as a result of chlorination). For purposes of this materials balance, it was assumed that a negligible amount of trichloroethylene was created and discharged to the environment by water chlorination.

Trichloroethylene loading to POTWs varies widely in different municipal areas. Of the estimated 1,020 kkg trichloroethylene entering POTWs, calculations indicate that approximately 85 percent is lost to air and 15 percent is discharged to water. (See Section 3.3.3.)

Present use patterns, in particular trichloroethylene consumption and loss in degreasing operations, are presented in Section 3.4. In response to declining demand and improved solvent recovery, production of trichloroethylene dropped sharply from 206,000 kkg in 1973 to 136,000 kkg in 1978. Historically, however, over 90 percent of the trichloroethylene domestic supply has been consumed in degreasing operations. Minor uses of trichloroethylene, which include the production of fungicides, cleaning fluids and adhesives, represent only 4 percent of the total domestic consumption; the remainder (approximately 4 percent) is used as a chain terminator in polyvinyl chloride production.

Trichloroethylene is one of the most versatile solvents used for degreasing operations. Because of its low boiling point (relative to water), strong solvent properties, and, until recently, relatively low cost, trichloroethylene has been employed in an estimated 160,000 operations annually. This widespread use of trichloroethylene results in an estimated annual loss of 120,000 kkg from degreasing operations, either as process emissions or as waste solvent. As shown in Table 3-3, use of trichloroethylene in fungicides, adhesives, and cleaning fluids represent 5 percent of atmospheric emissions, whereas degreasing operations represent 95 percent of the total. Moreover, degreasing operations represent virtually 100 percent of all discharges to land and water disposal of trichloroethylene within the United States.

Available literature has been critiqued and compiled in this chapter to present an overview of major sources of environmental release of trichloroethylene and fully annotated tables to aid data evaluation. Data collection, sources of information, and problem areas are reviewed in Sections 3.3 through 3.5. The discussion of the fate and transport of trichloroethylene within air, soil, and water environmental compartments was limited to a review of damage incidents resulting from improper disposal of trichloroethylene-bearing wastes and an estimate of cummulative environmental releases of trichloroethylene from degreasing operations over the period 1954-1978. (See Section 3.5.)

3.3 PRODUCTION OF TRICHLOROETHYLENE

First synthesized by Fisher in 1864, trichloroethylene was not manufactured in the United States until 1925. Demand for trichloroethylene increased with improvements in metal degreasing techniques in the 1920's and with the growth of the dry-cleaning industry during the 1930's. Recently, however, trichloroethylene production has declined significantly as a result of its suspected carcinogenicity and contribution to air pollution.

3.3.1 Production of Trichloroethylene and Tetrachloroethylene

Trichloroethylene, along with coproduct tetrachloroethylene, is produced by either chlorination or oxy-chlorination of 1,2-dichloroethane or other C_2 chlorinated hydrocarbons (including waste streams). All of the 136,000 kkg of trichloroethylene produced in the United States during 1978 was from 1,2-dichloroethane-based processes. Prior to 1978, 8 percent of U.S. trichloroethylene production was via an acetylene-based process, which has been abandoned due to increasing feedstock costs (SRI 1979). Table 3-1 summarizes production and emission data for trichloroethylene manufacture.

The reaction for direct chlorination of 1,2-dichloroethene to tri- and tetrachloroethylene is:

$$2ClCH_2CH_2Cl + 5Cl \longrightarrow Cl_2C{=}CHC + Cl_2C{=}CCl_2 + 7HCl$$

This process is discussed in more detail in Appendix A. Figure 3-2 represents a simplified diagram of the emissions from production by chlorination.

Tri- and tetrachloroethylene may also be produced by oxy-chlorination of 1,2-dichloroethene:

$$5ClCH_2CH_2Cl + 3Cl_2 + 4O_2 \longrightarrow Cl_2C{=}CCl_2 + 4Cl_2C{=}CHCl + 8H_2O$$

This process is discussed in more detail in Appendix B. Figure 3-3 represents a simplified diagram of the environmental releases from production by oxy-chlorination.

3.3.2 Trichloroethylene Releases: Production, Storage and Fugitive Emissions

Few data are available regarding releases of trichloroethylene during production. Major point sources of atmospheric trichloroethylene emissions are neutralization and drying vents and distillation vents. It is estimated that 0.51 kg trichloroethylene/kkg trichloroethylene produced are discharged as process emissions, 0.81 kg/kkg emitted from storage facilities, and 0.75 kg/kkg as fugitive emissions (EPA 1980e). Based on the 136,000 kkg trichloroethylene produced in 1978 (USITC 1979), a total of 280 kkg are dispersed to the atmosphere during trichloroethylene manufacture from process, storage and fugitive sources. (See Table 3-1.)

Neutralization is the predominant method of treatment of trichloroethylene process wastes. All three of the producing plants discharge to surface waters; two report secondary treatment (e.g., aerated lagoons or activated sludge) and one reports primary treatment only. Assuming total wastewater production of 0.42 kkg H_2O/kkg product (Catalytic 1979), total trichloroethylene production of 136,000 kkg (USITC 1979), and an average trichloroethylene concentration of 6580 µg/l (Catalytic 1979),

Table 3-1. Trichloroethylene Environmental Releases: Production, 1978 (kkg/yr)

Company and Location	Process[a]	Capacity[b]	Production[c]	Releases		
				Air[d]	Land[e]	Water[f]
Dow Chemical Corp. Freeport, TX	DC	68,000	52,000	110	neg	neg
Ethyl Corp. Baton Rouge, LA	DC	20,000	15,000	30	neg	neg
PPG Industries Lake Charles, LA	OXY	91,000	68,000	140	neg	neg
TOTALS		179,000	136,000	280		

a) DC: Direct Chlorination; OXY: Oxy-chlorination.

b) SRI, 1979. Diamond Shamrock placed a 23,000 kkg/yr plant in Deer Park, Texas on standby in early 1978.

c) Based on individual plants operating at 76% capacity and total trichloroethylene production of 136,000 for 1978 (USITC, 1979).

d) Based on process emissions of 0.51 kg trichloroethylene/kkg trichloroethylene produced, storage emissions of 0.81 kg/kkg, and fugitive emissions of 0.75 kg/kkg (EPA, 1980e).

e) Solid wastes are assumed to be incinerated or recycled as chlorinolysis feedstock (e.g., carbon tetrachloride production).

f) Aqueous discharges based on 0.42 kkg H_2O produced/kkg product, 6,580 µg trichloroethylene/l wastewater (Catalytic, 1979) and total production of 136,000 kkg trichloroethylene. The resulting 380 kg, distributed proportionally between the plants, represent negligible emissions.

Table 3-2. Trichloroethylene Environmental Releases from Inadvertent Sources, 1978 (kkg/yr)

Source	Discharges		
	Air	Aqueous	Solid
Manufacture of 1,2-Dichloroethane by the Balanced Process	neg[a]		12[a]
Production of Tetrachloroethylene Diamond Shamrock Corp. Deer Park, TX	8[b]		
Chlorination of Drinking Water and Wastewater		neg[c]	

a) Based on 29 kg "heavy" chlorinated organic material generated/kkg EDC produced (Lunde, 1965), 5.1×10^6 kkg EDC produced by balanced process, approximately 4% non-recyclable tars in heavy material, and 0.2% trichloroethylene by weight in tars (EPA, 1975a). If incinerated, assume 99.9% combustion efficiency. The remainder of the heavy stream is presumed recycled. At 3.6% trichloroethylene (Lunde, 1965), ≈5200 kkg of trichloroethylene would be recycled, to carbon tetrachloride/tetrachloroethylene production.

b) Based on 0.11 kg trichloroethylene emitted from neutralization and drying vent/kkg tetrachloroethylene produced by direct chlorination of 1,2-dichloroethane, and 0.056 kg/kkg from distillation vent (EPA, 1977d). Capacity for this plant: 75×10^3 kkg tetrachloroethylene; production at 63% of capacity. (USITC, 1979; EPA, 1979d).

c) Based on National Organic Monitoring Survey data (EPA, 1977c). POTWs considered a throughput, rather than an indirect source of trichloroethylene; see Appendix C for POTW data.

Table 3-3. Trichloroethylene Materials Balance: Use, 1978 (kkg/yr)[a]

Use	Input	Contained/Recycle	Releases		
			Air	Land	Water
Degreasing[b]					
Cold Cleaning	30,000(35,000)	9,000	15,000	7,000	3,000
Open-top vapor	56,000(66,000)	6,000	51,000	5,300	2,300
Conveyorized vapor	21,000(25,000)	1,700	21,000	1,300	570
Fabric scouring	10,000(12,000)	2,700	6,000	2,100	900
Coffee Decaffeination Beer foam suppressant Dry cleaning Film cleaning Paint additive Grain drying Refrigerant Lumber drying Anaesthesia Cosmetics Oilfield wax removal Rubber products	Discontinued[c]				
Fungicides	3,800[d]		3,800[e]		
Adhesives Cleaning fluids	1,200[d]		1,200[e]		
Chain transfer agent in polyvinyl chloride production	5,000	5,000[f]			
Exports[g]	16,000	16,000	----------neg----------		

a) Numbers may not add due to rounding to two significant figures.
b) See Table 3-5 for derivation of degreasing figures; totals do not include ≈2% TCE destroyed by incineration. Numbers in parenthesis are total solvent (virgin + recycle from previous use).
c) See text for explanation of discontinued uses.
d) Munster, 1980; Bernard, 1980.
e) Assumed total input is released to the air.
f) Represents amount of trichloroethylene chemically incorporated into the polymer.
g) U.S. Department of Commerce, 1980.

approximately 380 kg of trichloroethylene were discharged to surface waters (Catalytic 1979). This value is calculated on the basis of raw wasteload and, therefore, represents a maximum value. Possible mechanisms for trichloroethylene removal from mixed liquor suspended solids in an activated sludge system include biological degradation and mechanical stripping as a result of aeration (Su and Garin 1972). Aerated lagoons lower aquatic discharges at the expense of air emissions. This total aqueous discharge was divided proportionally between the three plants producing trichloroethylene. (See Table 3-1.) Specific data regarding solid waste discharges are unavailable. Assuming that solid wastes are largely incinerated or recycled as chlorinolysis process feedstocks [the waste may be a suitable feedstock for tetrachloroethene/carbon tetrachloride via a chlorinolysis process (EPA 1976)], land discharges of trichloroethylene are presumed to be negligible.

3.3.3 Publicly-Owned Treatment Work (POTWs)

Trichloroethylene loading to POTWs is largely dependent upon variations in a particular municipal area. A framework for calculating the total trichloroethylene flow through the nation's POTWs is provided by data from a recent EPA study (EPA 1980b). A materials balance of trichloroethylene at the treatment plants can be constructed using a total POTW flow of approximately 10^{11} l/day (EPA 1978b) and median values of 28 µg trichloroethylene/l (influent) and 4 µg trichloroethylene/l (effluent). (See Appendix C.) A trichloroethylene "slug" discharged to a POTW would effectively settle to the bottom of a primary clarifier (Su and Garin 1972), since TCE is relatively more dense than water. It is assumed for purposes of these calculations that influent and effluent flow rates are equal, i.e., that water loss from sludge removal and evaporation are small compared to influent flows. The results of the calculations show an input to POTWs of 1,020 kkg trichloroethylene and an effluent of 150 kkg. (See Figure 3-1.)

Trichloroethylene discharged in sludge can be estimated from the trichloroethylene concentration in sludge and quantity of dry sludge produced annually, 6.0×10^6 kkg (EPA 1979f). Assuming the trichloroethylene concentration of POTW wet sludge to be 30 µg/l (see Appendix C) and that wet sludge is 95 percent water by weight, approximately 4 kkg of trichloroethylene are discharged as sludge. As ocean dumping of sludge is mandated to cease by 1981 and assuming that more stringent air quality standards curb incinerator use (EPA 1979g), the 4 kkg of trichloroethylene contained in sludge are assumed discharged to land. (See Figure 3-1.)

The trichloroethylene released to the atmosphere may be estimated by the difference from the above calculations given the following assumptions: (1) trichloroethylene recycled within the activated sludge process will eventually be "wasted"; (2) the trichloroethylene biologically degraded is negligible; and (3) trichloroethylene is lost to the atmosphere by mechanical stripping, or aeration. Thus, an estimated 870 kkg of trichloroethylene is released to the atmosphere from POTW's. (See Figure 3-1.)

3.3.4 Transportation-Related Accidents Involving Trichloroethylene

Transportation (loading and transfer) of trichloroethylene might result in accidental spills or leakage of vapor and/or liquid. Trichloroethylene is transported by rail (35.6 percent), truck (48.0 percent), and barge (16.2 percent) (EPA 1980c). Appendix D is a list, maintained by U.S. Department of Transportation's Material Transportation Bureau (DOT 1980), of transportation incidents involving the release of trichloroethylene to the environment from 1971 to the present. It is interesting to note that the number and severity of the accidents are low, with no deaths or injuries resulting. Further, there was a total of only $551 in damages reported. For 1979, 675 liters, approximately one kkg, were released to the atmosphere from such incidents. DOT now requires special modifications to tank cars shipping hazardous materials in commerce, including head shield, shelf couplers, and thermal protection (49 CFR 179.105) to further reduce the frequency and severity of future accidents.

3.3.5 Inadvertent Sources of Trichloroethylene Emissions

In general, any human activity that unintentionally disperses a chemical to the environment is an "inadvertent source" of that chemcial. Inadvertent sources are likely since chemical species do not react via a single reaction pathway. Depending on the nature of the reactive intermediate there are a variety of pathways that lead to a series of reaction products. Inadvertent sources of a chemical are not limited to those of chemical manufacturing processes, however. Disinfection of drinking water or wastewaters by chlorination, for example, is a potentially important inadvertent source of chlorinated organic hydrocarbons.

This section will examine possible inadvertant sources of trichloroethylene emissions grouped accordingly:

- Trichloroethylene emissions from production of other chlorinated hydrocarbons; and
- Chlorination of drinking water.

Table 3-2 is a summary chart for inadvertent sources.

3.3.5.1 Production of Other Chlorinated Hydrocarbons

Quantifiable discharges of trichloroethylene have been documented from the production of vinyl chloride monomer, and tetrachloroethene manufacture. Both will be discussed briefly here and put in the context of inadvertent sources of trichloroethylene releases, as illustrated in Table 3-2.

There are two sources of trichloroethylene emissions during vinyl chloride monomer (VCM) production: solid waste from the 1,2-dichloroethane purification column and reactor VCM tars. Solid wastes, containing tars,

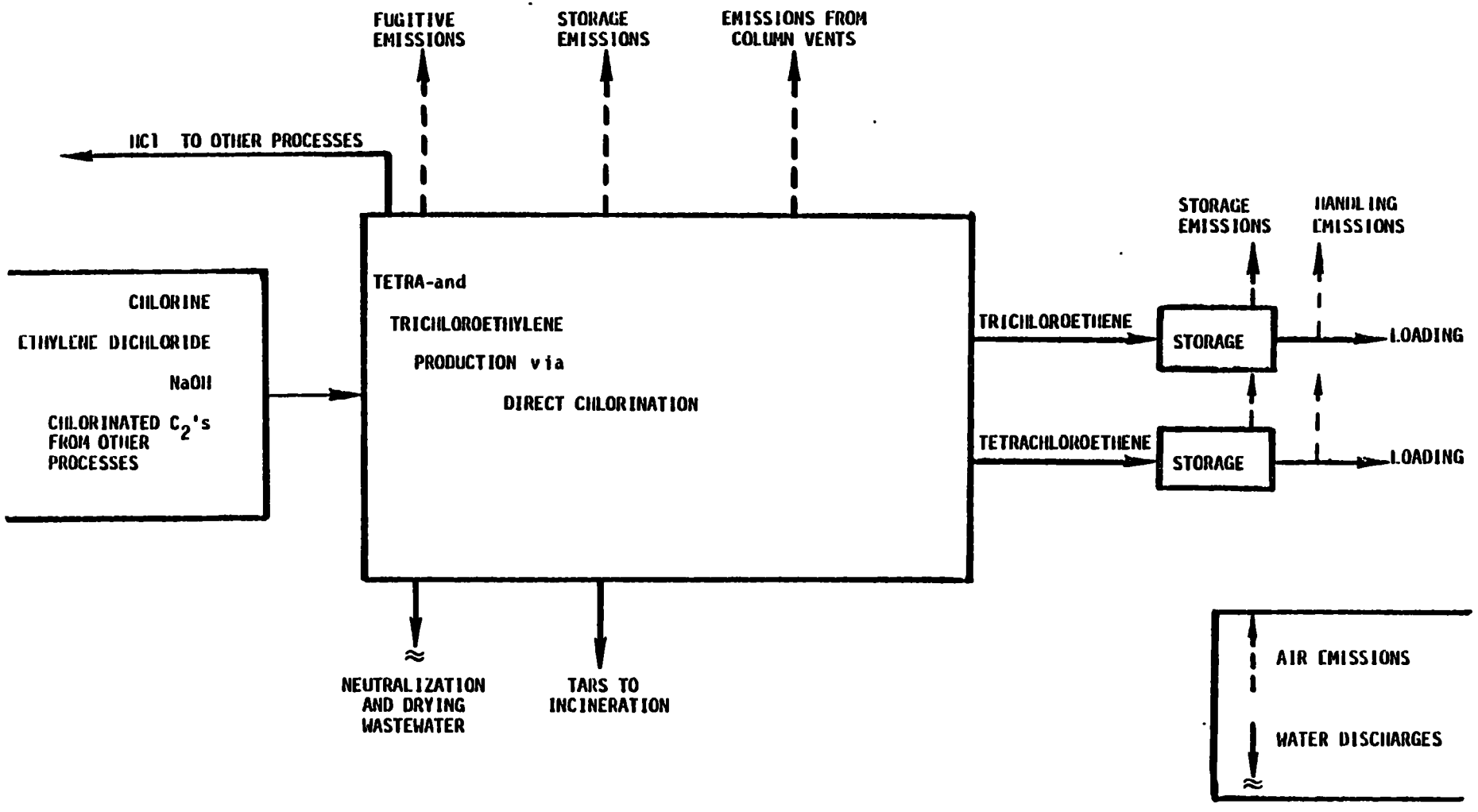

Figure 3-2. Environmental Releases from Trichloroethylene and Tetrachloroethylene Production by Chlorination

spent catalyst, and dessicants are usually treated to recover organic compounds present, then are either disposed in a landfill or incinerated, thereby recovering chlorine as hydrogen chloride (McPherson et al. 1979).

Process emissions are dependent upon plant specific operating parameters and configuration. Based on the process description of Lunde (1965) however, approximately 29 kg of "heavy" chlorinated organic material are generated per kkg of 1,2-dichloroethane produced. Based on a 1,2-dichloroethane production of 5.1×10^6 kkg, 1.5×10^5 kkg of "heavy" chlorinated organic material were generated from the balanced process. [This 1,2-dichloroethane production figure is at variance with data reported by the United States International Trade Commission (USITC), who exclude production data for intermediate products. The figure cited here is based upon end product production and reported yields.] Of this waste, approximately 4 percent may be regarded as nonrecyclable tars; the remainder is presumed to be recycled to carbon tetrachloride/tetrachloroethylene production. The trichloroethylene concentration of the recyclable stream is estimated to be 3.6 percent by weight (Lunde 1965) while that of tars is estimated to be 0.2 percent (EPA 1975a). Using these assumptions, approximately 5,200 kkg of trichloroethene are recycled. Assuming nonrecoverable tars to be the only source of trichloroethylene dispersion to the environment, and that these tars are incinerated with 99.9 percent efficiency, 12 kg of trichloroethylene are emitted from this source. If disposed as solid waste, 12 kkg are dispersed to land. (See Table 3-2.)

Production of tetrachloroethylene by direct chlorination at the Deer Park, Texas plant of Diamond Shamrock has provided an opportunity to examine trichloroethylene emissions from a facility that has put trichloroethylene production on standby (EPA 1979d, SRI 1979). Process discharges from a tetrachloroethylene plant have been estimated as 0.11 kg trichloroethylene/kkg product from the neutralization and drying vent and 0.056 kg/kkg from the distillation vent. Thus, from this 75×10^3 kkg capacity plant, and a production level 63 percent of capacity (USITC 1979), an estimated 8 kkg of trichloroethylene were emitted to the atmosphere. (See Table 3-2.) Aqueous discharges are calculated as 0.13 kkg based on emission factors described in Section 3.3.2. The wastewater production of 0.42 kkg H_2O/kkg product and average trichloroethylene concentration of 6,580 µg/l are assumed to be the same for both tetra- and trichloroethylene production; the two compounds commonly occur as coproducts in the same facility, and are subject to the same waste treatment processes. In comparison, total trichloroethylene production discharges are estimated as 280 kkg to the atmosphere and 380 kg to water. (See Section 3.3.2; Table 3-1.)

3.3.5.2 Chlorination of Drinking Water and Wastewater

The chlorination of drinking water and wastewater has come under close scrutiny recently, largely due to the discovery that chlorination of residual organic matter in such waters may lead to the formation of chlorinated degration products (Glaze and Henderson 1975, Rook 1977, Dowty et al. 1975). Trichloroethylene has been identified among thirteen halogenated hydrocarbons from New Orleans drinking water (Rook 1977). The EPA National Organic Monitoring Survey identified trichloroethylene at concentrations in the low µg/l range in samples from 113 U.S. community water supplies (EPA 1977c). As shown in Appendix E, however, the results of this broad sampling effort are ambiguous. That is, the source of such chlorinated organic chemicals cannot be readily identified (e.g., whether they are in the raw water prior to treatment or introduced as a result of chlorination). However, it is possible that chlorination of these waters could be a source of trichloroethylene, as the number of positive results is less in samples that have been quenched with a chlorine reducing agent than those that have not (i.e., terminal). In addition, trichloroethylene and other nonaromatic and aromatic compounds have also been found at µg/l levels in superchlorinated (2000-4000 µg/l) wastewater samples (Glaze and Henderson 1975, Dowty et al. 1975). Nonetheless, it is estimated for the purposes of this materials balance that a negligible amount of trichloroethylene is discharged to the environment by this inadvertent source.

3.4 USES OF TRICHLOROETHYLENE

In the past, trichloroethylene was used in a variety of applications, including medical anaesthesia, decaffeination of coffee, food and spice processing, dry cleaning and leather processing. As shown in Table 3-3, current uses of trichloroethylene have declined markedly.

Existing and anticipated regulations (reflecting concerns about trichloroethylene toxicity), increased cost of raw materials, and the availability of solvents with similar properties, such as tetrachloroethylene, carbon tetrachloride and 1,1,1-trichloroethane, have curtailed the trichloroethylene market. The remainder of this section consists of a discussion of the present use patterns of trichloroethylene. Specifically, trichloroethylene consumption and losses in degreasing operations and minor applications are presented. A brief discussion of discontinued miscellaneous uses is also included.

3.4.1 Present Use Patterns

In response to declining demand and improved solvent recovery, production of trichloroethylene dropped sharply from 206,000 kkg in 1973 to 136,000 kkg in 1978, see Figure 3-4, (USITC 1979). Moreover, an increasing proportion of U.S. trichloroethylene production, 11 percent in 1978 as compared to 25 percent in 1979, has been exported (Dept. of Commerce 1980).

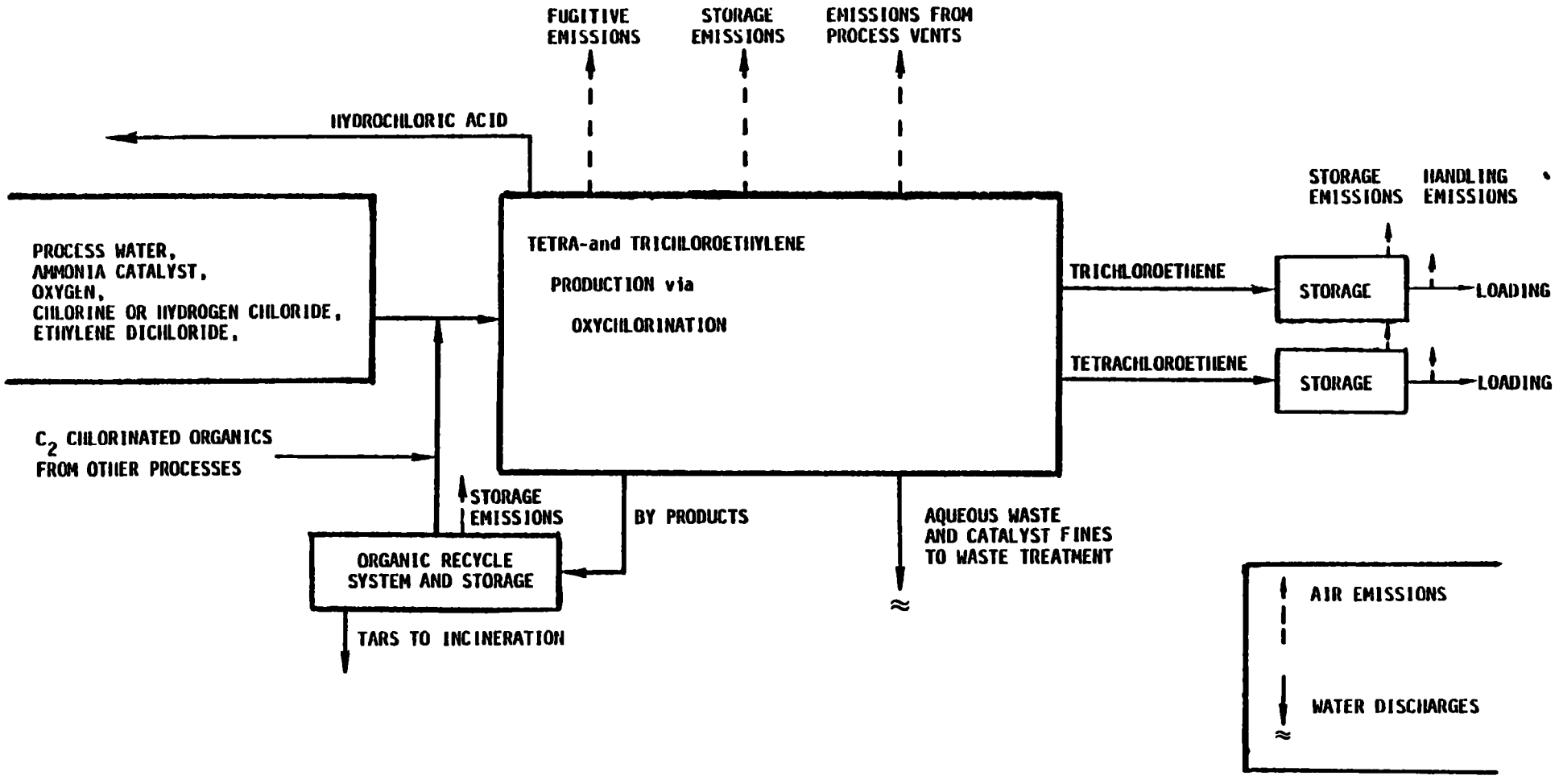

Figure 3-3. Environmental Releases from Tetra- and Trichloroethylene Production by Oxychlorination

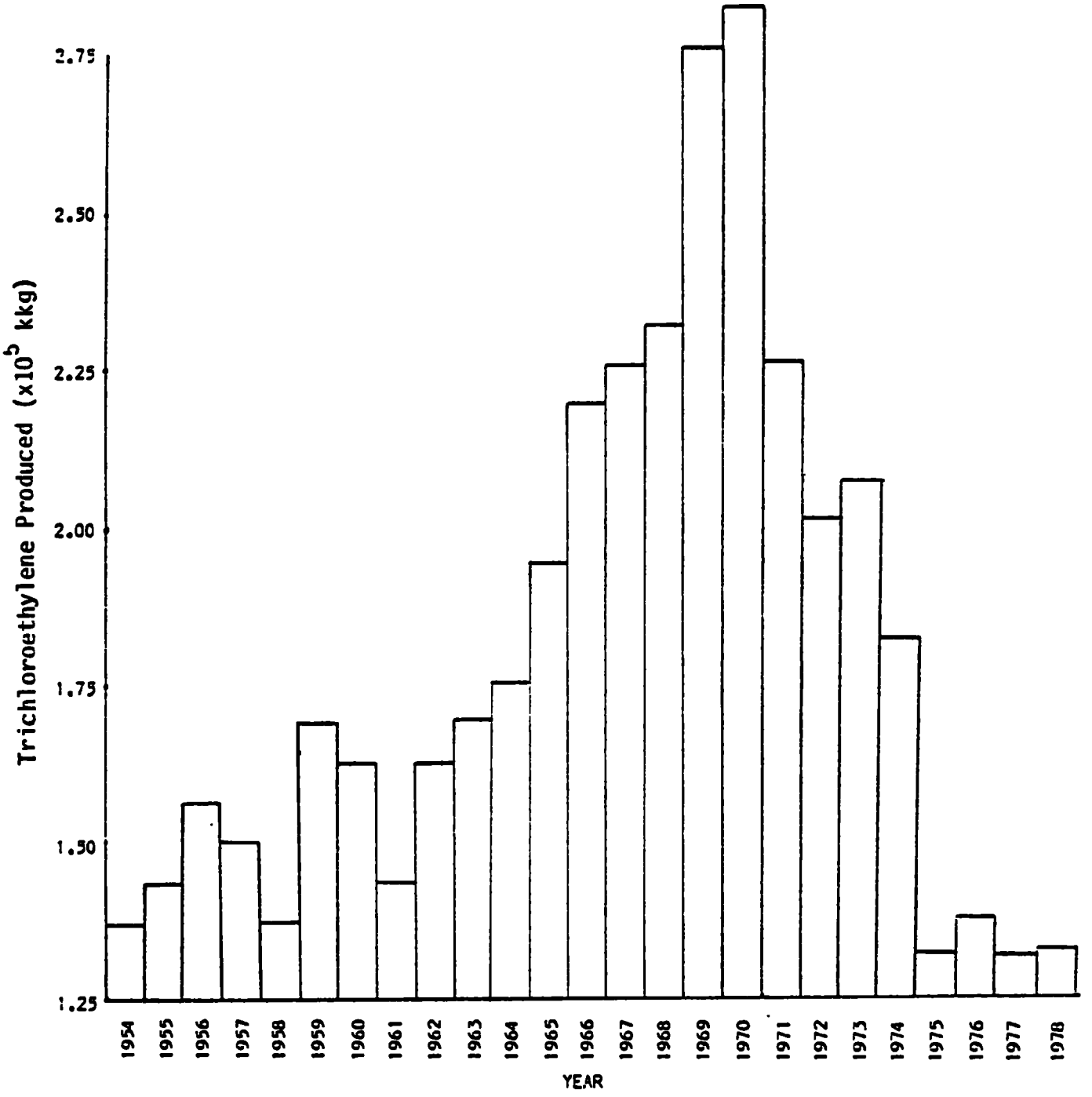

Figure 3-4. Domestic Production of Trichloroethylene, 1954-1978

Sources: Lowenheim and Moran, 1975; USTIC, 1975-1979.

Of the remaining domestic supply of trichloroethylene, over 90 percent has been consumed historically in degreasing operations (EPA 1979a). Minor uses of trichloroethylene represent only 4 percent of the total domestic consumption and include the production of fungicides, cleaning fluids and adhesives (Munster 1980, Bernard 1980); a list of representative products are presented in Table 3-4. The remaining trichloroethylene, or 4 percent of total production, is used as chain terminator in polyvinyl chloride production (Munster 1980, Bernard 1980).

3.4.2 Degreasing Operations

Solvent degreasing is the removal of oils, fats, waxes and grease from metal, plastic, glass and textiles by an organic solvent. This is a basic step in both large and small industries, such as machinery production prior to painting or electroplating, and small electronics workshops or auto service centers. (See Appendix F.)

Trichloroethylene is one of the most versatile solvents used for degreasing operations. Because of its low boiling point (relative to water), strong solvent properties, and, until recently, relatively low cost, trichloroethylene has been employed in an estimated 160,000 operations annually (EPA 1979b). Between 1956 and 1977, the price of trichloroethylene rose, with fluctuations, from $0.237/kg to $0.462/kg (USITC 1977).

Solvent operations are classified as either metal cleaning (cold cleaning, open-top vapor degreasing and conveyorized vapor degreasing) or fabric scouring. A flow diagram of a degreasing operation is presented in Figure 3-5. Approximately one-third of the total environmental releases from solvent degreasing occur as waste solvent (EPA 1977a). Waste solvent is the liquid containing solvent and impurities removed from degreased parts, and is distinct from vapor emissions (due to evaporation from the degreaser) or from carryout of solvent on treated parts. Estimates of waste solvent generation by degreasing operation are presented in Table F-2 (Appendix F). Information on waste solvent disposal practices is scant; however, approximately 45 percent of the waste solvent is assumed reclaimed by distillation. Distillation of solvent from oil-containing waste solvent is performed either by contractors or in-house by large users. Most degreasers have external stills, permitting uninterrupted operation. The bottoms from these stills, which contain the metals, oils and other impurities removed from the degreased parts, can be incinerated or landfilled. The latter is the less costly and more dominant practice (EPA 1977a). Emissions from each degreasing operation are discussed in the following sections and are summarized in Table 3-5.

Table 3-4. Selection of Commercial Products Containing Trichloroethylene

Adhes-Off	
(Harvey Labs Inc.)	
Trichloroethylene	
Petroleum base	
Balkamp Klean and Prime	
(Balkamp Inc., Mfr. Loctite Corp.)	
Trichloroethylene	
Bowes Buffing Solution	
(Bowes Corp.)	
Xylol	
Trichloroethylene	
Carboff	
(Holcomb Corp.)	
Cresol	>10%
Methylene chloride	>10%
Trichloroethylene	1-10%
Carbona Cleaning Fluid	
(Carbona Products Co.)	
Trichloroethylene	44%
Petroleum solvent	56%
Carbona No.10 Special Spot Remover	
(Carbona Products Co.)	
Trichloroethylene	40%
1,1,1-Trichloroethane	10%
Petroleum Solvent	50%
Lacco Chlorosan	
(Los Angeles Chemical Co.)	
Orthodichlorobenzene	59.5%
Trichlorobenzene	6.3%
Trichloroethylene	6.4%
Pine Oil	4.4%
Glamorene Dry Cleaner for Rugs	
(Glamorene Products Corp.)	
Chlorinated hydrocarbon (TCE)	
Petroleum distillate	
Wood flour	
Instant Chimney Sweep	
(Miracle Adhesives Corp.)	
Trichloroethylene	34%/wt.
Propellant freon	25%/wt.
Mole α Gopher Get	
(Mole α Gopher Get Mfg. Co.)	
Methylene Chloride	46.4 %
Naphthalene	1.24%
Paradichlorobenzene	1.24%
Trichloroethylene	51.1 %
PMD-77	
(Dixo Company, Inc.)	
Diethyl diphenyl dichloroethane and related compounds	10.5%
2,2'-Methylenebis (4-Chlorophenol)	0.2%
Tetrachloroethylene	45.8%
Trichloroethylene	42.6%
Sirotta's Sircofume Liquid Fumigating Gas	
(Sirotta, Bernard, Co., Inc.)	
Carbon tetrachloride	96.0%
Ethylene dichloride	1.0%
Tetrachloroethylene	1.0%
1,1,1-Trichloroethane	1.0%
Trichloroethylene	1.0%
Lethalaire B-5	
(Virginia Chem. Inc.)	
Trichloroethylene	31.0%

Source: EPA, 1979a

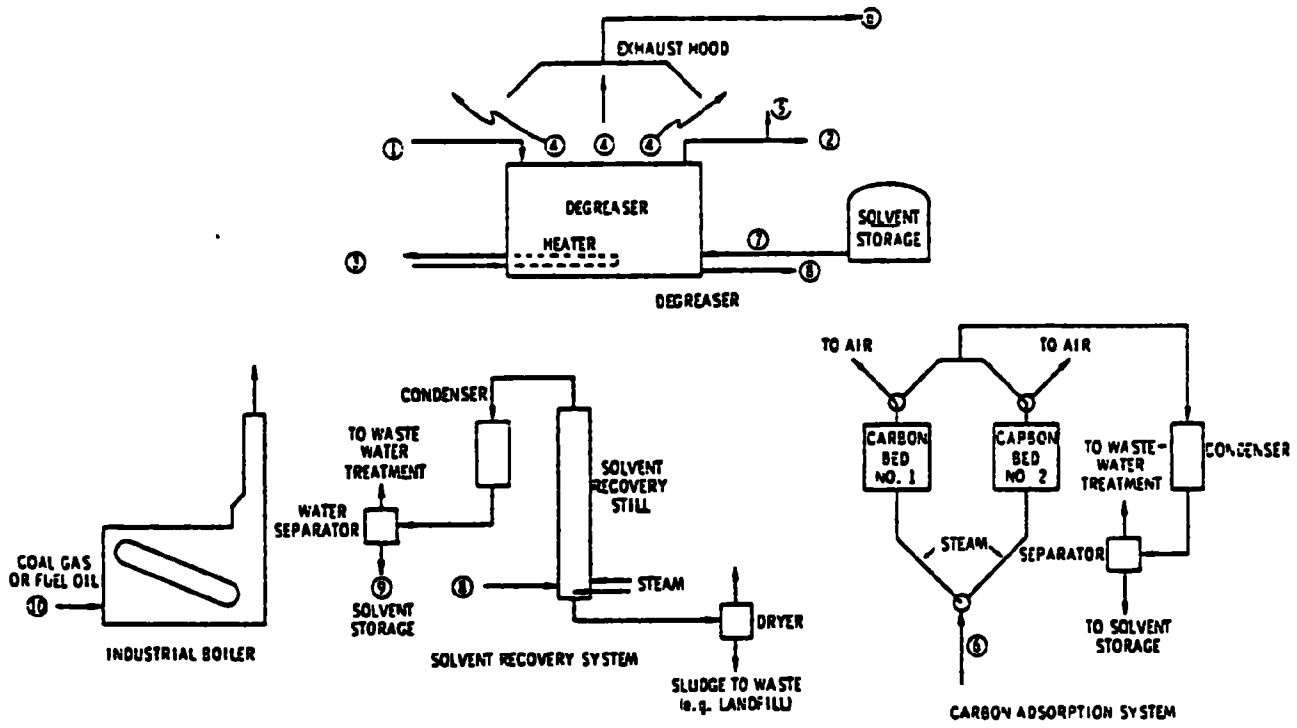

Figure 3-5 Degreaser Flow Diagram

1) Part to be cleaned is conveyed manually or automatically into the degreaser.
2) After degreasing, part manually or automatically withdrawn or sent to next operation.
3) Solvent heated by gas, electricity or steam.
4) Diffusing solvent can be collected by exhaust hood and vented to atmosphere or 6.
5) "Dragout" of solvent with the work.
6) Carbon adsorption system.
7) Solvent loss balanced by periodic addition of solvent from storage tanks or drums.
8) Solvent contaminated with grease or oil (waste solvent) sent to solvent recovery system.
9) Distillate is condensed, sent through water separator, and placed in storage.
10) Boiler provides steam if required.

Source: EPA. 1979b.

Table 3-5. Trichloroethylene Materials Balance: Degreasing Operations, 1978 (kkg/yr)[a]

Degreasing Operation	Total Use[b]	Virgin Solvent[b]	Recycle[c]	Incineration[d]	Total Waste Solvent Load[e]	Environmental Releases		
						Air[f,g] Process Emissions	Land[h]	Water[h]
Cold Cleaning	35,000	30,000	9,000	1,000	20,000[i,j]	15,000 (11,000-20,000)	7,000	3,000
Open-Top Vapor	66,000	56,000	6,800	750	15,000	51,000 (36,000-67,000)	5,300	2,300
Conveyorized Vapor	25,000	21,000	1,700	190	3,800	21,000 (15,000-28,000)	1,300	570
Fabric Scouring	12,000	10,000	2,700	300	6,000	6,000 (4,200-7,800)	2,100	900
TOTALS	140,000	120,000	20,000	2,200	45,000	93,000	16,000	6,800

a) All figures have been rounded to two significant figures.

b) Based on apparent 1978 consumption figures for trichloroethylene and distribution figures derived from EPA (1979b); total use = virgin solvent + recycle from previous use; see Figure F-1 (Appendix F) for calculations.

c) Assumes 45% of the waste solvent load is reclaimed through distillation and recycled (EPA, 1977a).

d) Approximately 5% of waste solvent is assumed to be disposed of by incineration (EPA, 1977a) resulting in negligible air emissions.

e) There are two categories of solvent losses recognized: evaporative and waste solvent loading. Waste solvent loading was calculated according to release factors presented in Table F-2 (Appendix F).

f) Insufficient data precluded the calculation of an emission factor on a solvent-by-solvent basis -- thus all solvents are assumed to have the same emission factor calculated as follows: cold cleaning: 430 g/kg ±30%; open-top vapor: 775 g/kg ±30%; conveyorized vapor; 850 g/kg ±30%; and fabric scouring: 500 g/kg ±30% (EPA, 1979a). The uncertainty range was based on calculations presented in EPA (1977a) and with personal communications of the authors with J. L. Shumaker of U.S. EPA. (EPA, 1979a).

g) The range presented represents the ±30% uncertainty factor on the calculated value.

h) Assumptions were made based on engineering judgements as follows: 35% of the total waste solvent load is assumed to be disposed of to land (i.e., dumped onto grounds surrounding the user facility or in landfills) and 15% to water (i.e., dumped in drains). These totals are undoubtedly high, as most of the solvent should volatilize to the atmosphere. Only 20% of the metal finishing plants are direct dischargers. (Note: the remaining 45% is recycled after distillation and 5% is assumed to be incinerated, see Footnotes c and d). (EPA, 1977a).

i) The waste solvent load from cold cleaners was calculated based on the factors for the maintenance and manufacturing subcategories presented in Table F-2. Thus, 7,700 kkg and 12,250 kkg of waste solvent were generated by manufacturing and maintenance facilities, respectively.

j) Approximately 33% of the waste solvent load from maintenance cold cleaners (i.e., automotive maintenance facilities) is assumed to be disposed with waste crankcase oil. It is further assumed that the crankcase oil is used for dust control on unpaved road (EPA, 1977a).

3.4.2.1 Cold Cleaning

There are two major applications for cold cleaners--maintenance and manufacture. Maintenance cleaners are used primarily in automotive and general plant cleaning, whereas the manufacture cleaners are integral to metal working production. Cold cleaning uses solvent in the liquid state (at room temperature or sometimes heated below the boiling point), and includes spraying, brushing, and soaking operations. Typically the solvent is agitated by a pump, compressed air, or by ultrasonics; in some cases, the parts to be treated are agitated within the bath. The design of a cleaner varies with the types of parts to be cleaned, required materials handling (i.e., manual and batch loaded conveyorized systems), the agitation technique, the size of tank required, and the frequency of cleaning. As shown in Figure F-1 (Appendix F), solvent is lost to the environment by: evaporation from the bath, spray and agitation operations, carry-out on treated parts; and waste solvent disposal (EPA 1977a).

Cold cleaning is the largest user of trichloroethylene in the degreasing industry with the compound utilized in approximately 150.000 operations annually (EPA 1979b). A geographic distribution of all cold cleaning operations is presented in Figure 3-6. As shown in Table 3-5, cold cleaning produced 45 percent (20,000 kkg) of the total waste solvent trichloroethylene load from degreasing operations. Total trichloroethylene releases from cold cleaning are estimated as follows: 15,000 kkg to air; 7,000 kkg to land; and 3,000 kkg to water. Approximately 9,000 kkg of trichloroethylene are assumed to be recycled (see Table 3-5; releases based on total solvent used, i.e., virgin solvent plus that solvent recycled).

3.4.2.2 Open-Top Vapor Degreasing

In open-top vapor degreasing, a vapor layer is created over a solvent bath by electric, steam or gas heat (Watson 1973). The metal parts to be cleaned are then dipped into the vapor. As the vapors condense on the metal, the impurities are washed off. In addition, a spray may be used to rinse the part (Spring 1967). Although cooling coils in the tank maintain a distinct boundary between the vapor layer and the surrounding air. vapor does escape as a result of evaporation, agitation of treated parts, and solvent carry-out on parts. (See Figure F-2, Appendix F). In practice, exhaust hoods are often used to vent escaping fumes to the air.

Open-top vapor degreasers are usually site-specific (i.e., located at the site of the material to be cleaned), operated manually, and used chiefly only during a small part of the work day. They are primarily used in metal working plants or for maintenance cleaning of intricately designed electrical parts requiring a high degree of cleanliness (EPA 1977a).

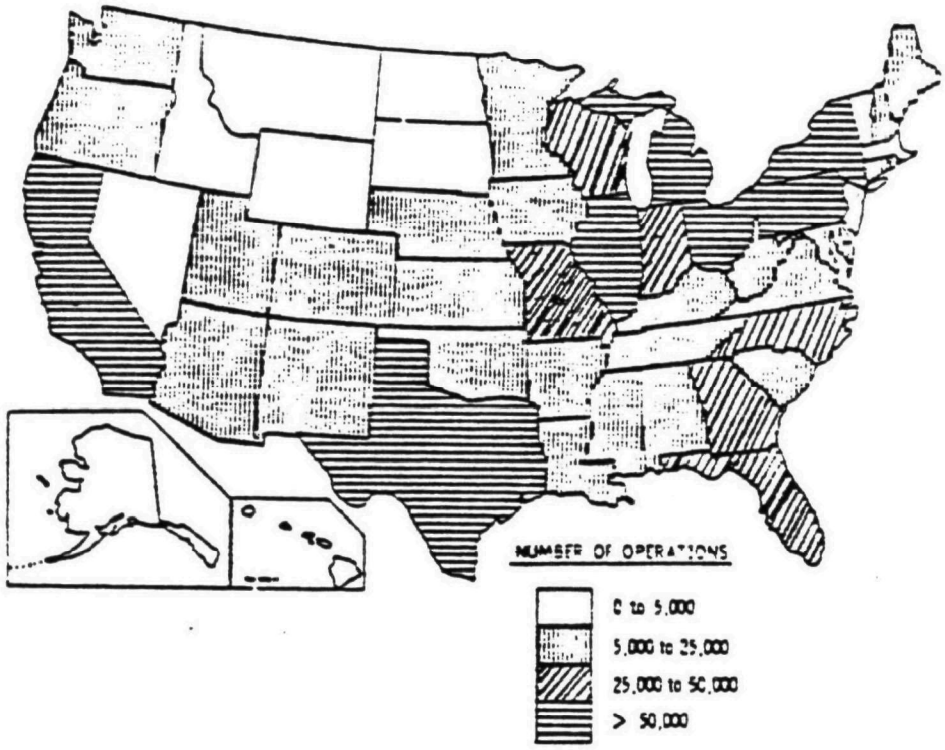

Figure 3-6. Geographic Distribution of Cold Cleaning Operations (EPA, 1979b)

Over 50 percent (or >11,000) of the estimated 21,000 degreasing operations use trichloroethylene as the solvent of choice (EPA 1979b). The geographic distribution of all open-top and conveyorized vapor degreasing operations is shown in Figure 3-7.

Emissions can be controlled with activated carbon systems, use of tank covers during idle time, adequate freeboard height, proper sizing of treated parts in relation to the tank, minimum agitation of the bath, and use of additional condensers to prevent evaporation and adequate freeboard height (Watson 1973, EPA 1979b). Freeboard is the distance from the top of the vapor zone to the top of the degreaser tank. This is usually established by the location of condenser coils, which protect the solvent vapor from outside air disturbance. For trichloroethylene, the freeboard is usually 50-60 percent of the degreaser width (EPA 1977a). In contrast to cold cleaners, most releases from open-top vapor degreasers are not from the waste solvent load but rather from vapor diffusing from the degreaser. Estimated total trichloroethylene releases in 1978 from this degreasing operation were 51,000 kkg to the atmosphere, 5,300 kkg to land, and 3,000 kkg to water; 6,800 kkg were assumed to be recycled. (See Table 3-5; based on total quantity of solvent used).

3.4.2.3 Conveyorized Vapor Degreasing

In this continuous version of the open-top method, manual handling has been eliminated and the operation is typically located at a central cleaning station within a plant requiring a steady flow of products to be degreased. Conveyorized degreasers continuously redistill solvent and return it to the bath; the conveyor speed can be controlled to minimize agitation of the bath (EPA 1977a). Sources of releases from conveyorized degreasers are depicted in Figure F-3 (Appendix F). Since the degreasers are normally enclosed and the workload large, solvent loss is greatest from carry-out with parts.

Trichloroethylene is used by 1,700 operations employing conveyorized vapor degreasing (EPA 1979b). The total geographic distribution of all vapor degreasing operations (i.e., open-top and conveyorized) is presented in Figure 3-7. Total air emissions from this degreasing operation in 1978 were calculated as 21,000 kkg. As shown in Table 3-5, waste solvent trichloroethylene disposed of on land and in water amounted to 1,300 kkg and 570 kkg, respectively; 1,700 kkg were assumed to be recycled.

3.4.2.4 Fabric Scouring

Fabric scouring, the only nonmetal cleaning degreasing operation discussed, is essentially a cold conveyorized degreaser. In this operation, "grey" goods (raw or untreated fabrics) are cleaned with solvent before finishing (dyeing and fabrication). There are three types of solvent scouring processes for fabrics; textile scouring, wool scouring, and multilayer treatment, in which textiles undergo

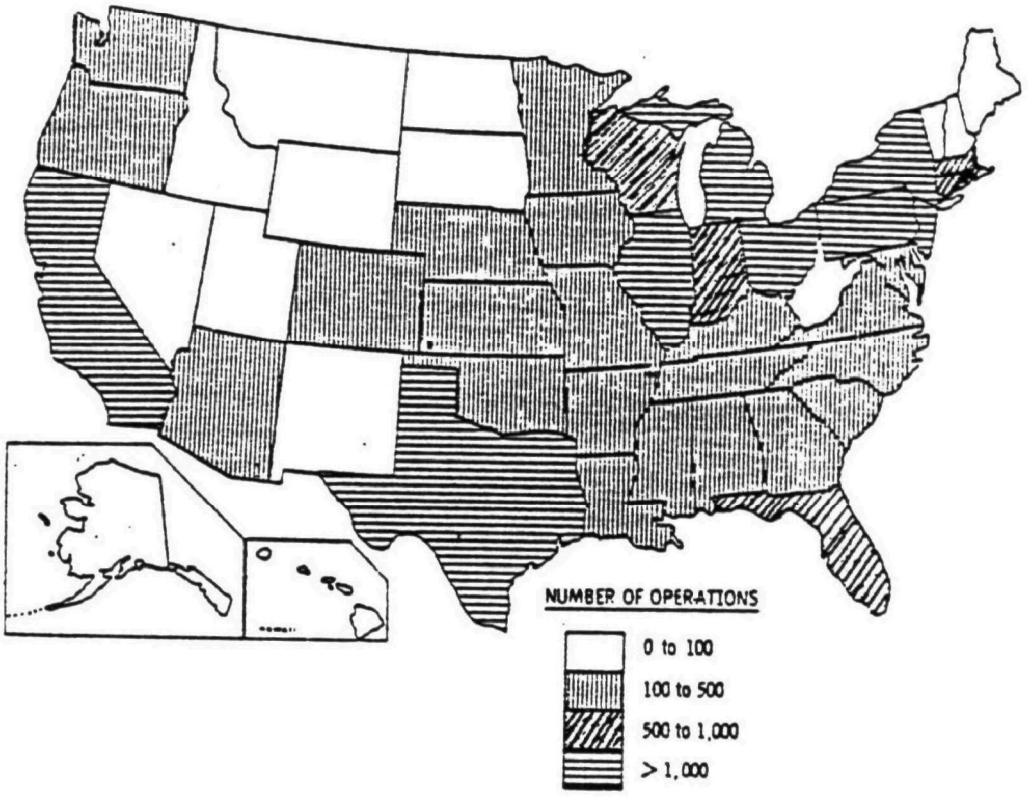

Figure 3-7. Geographic Distribution of Vapor Degreasing Operations

[a]Includes open-top and conveyorized degreasers.

Source: EPA, 1979b.

solvent scouring in several layers to increase throughput (EPA 1979b). The conveyorized fabric is sprayed, cooled and fed to the next finishing stage while the solvent and removed impurities are separated (EPA 1979b). Emissions occur through entrances to the scouring machine and are exhausted to the atmosphere (see Figure F-4, Appendix F). Although not in wide use, an activated-carbon system can recover up to 98 percent of the solvent from exhaust (Watson 1973).

Less than 700 (of the more than 9,000) fabric scouring operations are estimated to use trichloroethylene. The geographic distribution of all fabric scouring operations is shown in Figure 3-8. Total releases of trichloroethylene from this process in 1978 were estimated as 6,000 kkg to the air, 2,800 kkg to land, and 900 kkg to water. Approximately 2,700 kkg of trichloroethylene were assumed to be recycled. (See Tables F-2 and 3-5.)

3.4.3 Emissions from Minor Applications

Limited data are available concerning the miscellaneous minor uses of trichloroethylene. From the literature, it is estimated that 5,000 kkg of trichloroethylene are consumed on an annual basis in the production and use of fungicides, adhesives, and cleaning fluids. All of the solvent is assumed to be dispersed to the atmosphere (Bernard 1980, Munster 1980). Approximately 4 percent of trichloroethylene production (5,000 kkg) is used as a chain terminator in polyvinyl chloride (PVC) production (Bernard 1980, Munster 1980). Releases of trichloroethylene to the environment from PVC production are negligible,as trichloroethylene is introduced into the slurry in relatively small amounts and becomes chemically incorporated into the polymer. (See Table 3-3.)

3.4.4 Discontinued Miscellaneous Uses

Many of the minor applications for which trichloroethylene was once used or considered suitable are no longer practiced (ORNL 1976). Where published information was unavailable or in conflict, contacts were made with industry representatives to ascertain current trichloroethylene use practices. Although cited in the literature as a minor use (ORNL 1976, Joyce 1979), trichloroethylene is no longer used by the dry cleaning industry because of its aggressive solvent action on acetate dyes and textiles (Woolsey 1980). Because of its recognized toxicity, trichloroethylene is no longer used as an anaesthetic (Hattox 1980, Yamaner 1980). In a similar manner, coffee companies that had used trichloroethylene to decaffeinate coffee later abandoned its use in anticipation of its prohibition by the Food and Drug Administration (Gianetta 1980, Adinolfi 1980). Furthermore, trichloroethylene is regarded as too expensive for use in grain drying (Frederick 1980) or wood treatment (American Wood Preservers Association 1980) and unsuitable as a refrigerant (Evans 1980). An obscure application, wax removal from oil field pipelines, (ORNL 1976) is no longer practiced, as it

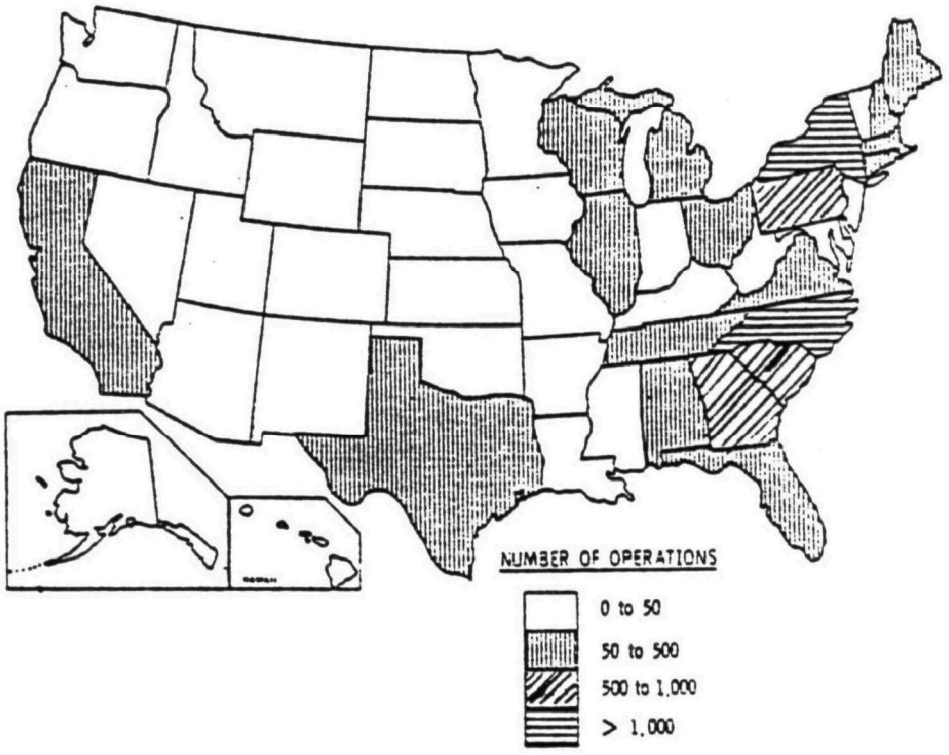

Figure 3-8. Geographic Distribution of Fabric Scouring Operations (EPA, 1979b)

results in unwanted carry-over of trichloroethylene into oil refineries (Smyth 1980, Grundman 1980). While research on the use of trichloroethylene as a solvent for surface coatings of paint has been reported in the literature, commercial development appears to have been unsuccessful (Leonard 1969).

3.5 MISMANAGEMENT OF TRICHLOROETHYLENE

Historically, contamination of ground water supplies has been tied to disposal practices. In terms of trichloroethylene, pollution of ground water and drinking water supplies appears largely attributable to migration and leaching of the chemical (either uncontained or stored in corroding 55-gallon drums) from unlined or improperly lined landfills (municipal and industrial), settling ponds, old dredge pits, open fields, and old industrial sludge pits. In addition, direct discharge of trichloroethylene into leaking sewer lines or from leaking storage tanks and faulty septic lines has been recorded (Massachusetts 1979, Fishburn 1980). Not surprisingly, EPA has documented several hundred cases of damage to human health and the environment resulting from improper management of hazardous waste. Those incidents specifically related to trichloroethylene use and disposal are listed in Appendix G. In many cases, the specific source or generator of the trichloroethylene-containing wastes is unknown or not easily identified. Nevertheless, a review of these damage incidents reveals present or imminent ground water contamination, resulting in partial or complete loss of private residential, industrial and/or municipal wells as drinking water sources.

3.5.1 Selected Damage Incidents

Reported damage incidents are not a recent development in the history of trichloroethylene use. Two cases of well contamination were described in 1949. In the first, trichloroethylene escaped from a burst tank during a factory fire, saturated the ground and contaminated a well located near the factory. As the well was situated in gravel only 20 ft from a river, the authors were surprised that trichloroethylene odors remained at the well after 4 years, stating that "one might have expected that the movement of water through the gravel would have removed the contaminant." In the second case, a trichloroethylene plume seeped through gravel from an open field dump site 150-200 yards to a well, registering 18,000 µg/l trichloroethylene,and causing stomach disorders and giddiness in consumers of the water. The authors' conclusions: "...contamination by compounds of this nature is likely to be very persistent, and there is some evidence of toxicity at very low concentrations." (Lyne and McLachlan 1949)

Recent analytical studies have revealed several cases of trichloroethylene contamination. In New Jersey, testing of eight public water supplies (rural, suburban and urban) showed levels of 170-400 µg/l trichloroethylene (Kasabach 1980). Near the Aerojet General rocket plant and its subsidiary Cordova Chemical Company (located east of

Sacramento, California), levels of 8,000 µg/l trichloroethylene have been found in ground water (Phillippe 1980). Extensive contamination of drinking water has been detected at various U.S. Air Force bases, prompting an Air Force-wide testing of the wells (Fishburn 1980). Presently the investigation is being conducted at Pease Air Force Base in New Hampshire, Wurtsmith Air Force Base in Michigan, Mather Air Force Base in California, and McClellan Air Force Base in California. Testing is in the preliminary stages, and information is incomplete. Thus far, however, levels of 6,700 µg/l trichloroethylene in a production well and 790 µg/l in a test well have been registered on-base at Wurtsmith Air Force Base (Grimes 1980), presumably the consequence of seepage from a corroded underground 500-gallon trichloroethylene storage tank. At McClellan, a base water supply well was similarly contaminated; recent analysis revealed trichloroethylene levels of 700 µg/l. Elsewhere on the McClellan property, an industrial waste sludge disposal pit is the suspected source of 48 µg/l trichloroethylene levels in an off-base private well. Although a centrifuge is normally used to dry the waste, with the dry sludge going to landfill, if the centrifuge fails, the solvent-containing wet sludge is deposited in the unlined disposal pits. A third potential on-base source of groundwater contamination is degreasing shops that dump small (≈100 liters) quantities of solvent directly into ditches behind the facilities (Phillippe 1980).

In the cases where a particular point source can be isolated, metal degreasing operations have been the primary contributors of trichloroethylene wastes. The industries involved include machine shops, electronics companies (Massachusetts 1979) and refrigerator manufacturers (Joyce 1980), in addition to the aforementioned cases. The Air Force used, and still uses, trichloroethylene (although in a limited capacity) primarily in the jet engine and aircraft shops for degreasing of parts. Aerojet General Rocket plant used trichloroethylene for degreasing during production of solid rocket propellant batch mixtures or to flush liquid propellant delivery pipes at test areas. Cordova Chemical Company, the Aerojet subsidiary sharing the common property, manufactured a variety of chemical products, including herbicides, pharmaceuticals and paint intermediates, and presumably used trichloroethylene as a process solvent, as well as for degreasing.

3.5.2 Cumulative Environmental Releases

To assess and understand the impact of trichloroethylene in terms of demonstrated and potential ground water degradation, estimates of the cumulative amounts of trichloroethylene released to air, water, and most importantly land over the period 1954-1978 are required. In the simplest case, ground water contamination results directly from improper disposal of trichloroethylene wastes; indeed, the many damage incidents involving trichloroethylene are suggested to have arisen in this way.

In this section, a summary table (see Table 3-6) has been developed to estimate cumulative releases of trichloroethylene to land and water for the period covering 1954-1978. To necessarily simplify the scenario, only releases from degreasing operations have been considered. Apparent consumption of trichloroethylene for degreasing operations has been assumed to be constant over the whole period at 90 percent of total U.S. production. Furthermore, for lack of better information, the distribution of trichloroethylene consumption among the different types of degreasing operations--cold cleaning, open-top vapor, conveyorized vapor, and fabric scouring--has been assumed to be constant. (See Tables 3-3 or 3-5.

Estimates of waste solvent loading, critical to any cumulative calculations of trichloroethylene dispersion to land and water, were based on release factors presented in Table F-2. From these estimates of total waste solvent loading, it is assumed that only 10 percent of the total waste solvent generated during the period of 1954 to 1972 was recovered (i.e., distillation), and that recovery practices increased (in a linear fashion) during the period between 1972 to 1978 to an estimated recovery/recycle level of 45 percent (EPA 1977a, see Table 3-6, Footnote G). In addition, it was assumed that throughout this period, approximately 5 percent of the total waste solvent load was destroyed by incineration (EPA 1977a), resulting in negligible air emissions. As shown in Figure 3-9, it is assumed, based on communications with users (Stranges 1980, Thorpe 1980) and EPA estimates (EPA 1977a), that the remaining waste solvent load is disposed of as follows: 70 percent on land and 30 percent to water. In a worst case scenario (in terms of ground water contamination), air emissions from open storage of waste solvent are considered insignificant (see EPA 1977a). From these calculations, an estimated 730,000 kkg of waste solvent trichloroethylene were dumped on land and 320,000 kkg discharged to water (i.e., dumped in drains) during the study period.

Table 3-6. Cumulative Releases of Trichloroethylene from Degreasing Operations, 1954 - 1978 (10^3 kkg)[a]

Year	Production[b]	Apparent U.S. Consumption Degreasing[c]	Process Waste Evaporative Emissions[d]	Process Waste Waste Solvent[e]	Waste Solvent Disposition Incineration	Recycle	Land Disposal	Discharge
1954	138	124	84	40	2.0	4.0	24	10
1955	145	131	89	42	2.1	4.2	25	11
1956	158	142	97	45	2.3	4.5	27	11
1957	150	135	92	43	2.2	4.3	26	11
1958	138	124	84	40	2.0	4.0	24	10
1959	165	149	101	48	2.4	4.8	29	12
1960	160	144	98	46	2.3	4.6	27	12
1961	145	131	89	42	2.1	4.2	25	11
1962	160	144	98	46	2.3	4.6	27	12
1963	165	149	101	48	2.4	4.8	29	12
1964	170	153	104	49	2.5	4.9	29	13
1965	190	171	116	55	2.8	5.5	33	14
1966	220	198	135	63	3.2	6.3	38	16
1967	225	203	138	65	3.3	6.5	39	17
1968	230	207	141	66	3.3	6.6	39	17
1969	275	248	169	79	4.0	7.9	47	20
1970	281	253	172	81	4.1	8.1	48	21
1971	225	203	138	65	3.3	6.5	38	16
1972	200	180	122	58	2.9	5.8	35	15
1973	206	185	126	59	3.0	8.9	33	14
1974	176	158	107	51	2.6	10.0	27	12
1975	132	119	81	38	1.9	9.9	18	8
1976	143	129	88	41	2.1	13.0	18	8
1977	135	122	83	39	2.0	15.0	15	7
1978	136	122[i]	83[i]	39[i]	2.0	18.0	13	6
TOTALS	4,500	4,000		1,300			730	320

FOONOTES next page

TABLE 3-6 (concluded)

a) Final totals do not add due to rounding.

b) Lowenheim and Moran, 1975; USITC; 1975-1979.

c) Apparent use of trichloroethylene for degreasing operations is reported in the literature as 90-99% of the total U.S. consumption of trichloroethylene (EPA, 1979a; EPA, 1979b). From these figures, the assumption was made that 90% of total U.S. production from 1954 to 1978 was consumed by degreasing operations.

d) Evaporative process emissions are calculated by determining the difference between the total amount of solvent utilized in the specific type of operations and the waste solvent load (EPA, 1979b).

e) Waste solvent load was derived from release factors presented in Table F-2 and a total degreasing trichloroethylene consumption of 120,000 kkg for 1978, resulting in a 32% value.

f) Approximately 5% of waste solvent is assumed to be disposed by incineration (EPA, 1977a) resulting in negligible air emissions.

g) It is assumed that only 10% of the total waste solvent generated was recovered from the period of 1954 to 1972, and that recovery practices increased (in a linear fashion) during the period of 1972 to 1978 to an estimated recovery/recycle level of 45% (EPA, 1977a). These assumptions were based on: personal communications with industry representatives (Stranges, 1980; Thorpe, 1980); the fact that the price of trichloroethylene more than doubled during the period of 1972 to 1978 (USITC, 1972-1979); and the promulgation of EPA regulations controlling solvent use and disposal.

h) The remaining waste solvent load is assumed, from communications with (Stranges, 1980; Thorpe, 1980) and EPA 1977a) to be disposed of as follows: 70% is disposed on land (i.e., dumped at a landfill site, on land at the user facility or mixed with crankcase oil and sprayed on land) and 30% to water (i.e., dumped in drains). In a worst case scenario (in terms of groundwater contamination), air emissions from open storage of waste solvent are considered insignificant (see EPA, 1977a).

i) These figures differ from those presented in lables 3-3, F-2 and 3-5 as exports and imports have not been adjusted for. Rather 90% of the total production for each year was calculated for the degreasing consumption figure.

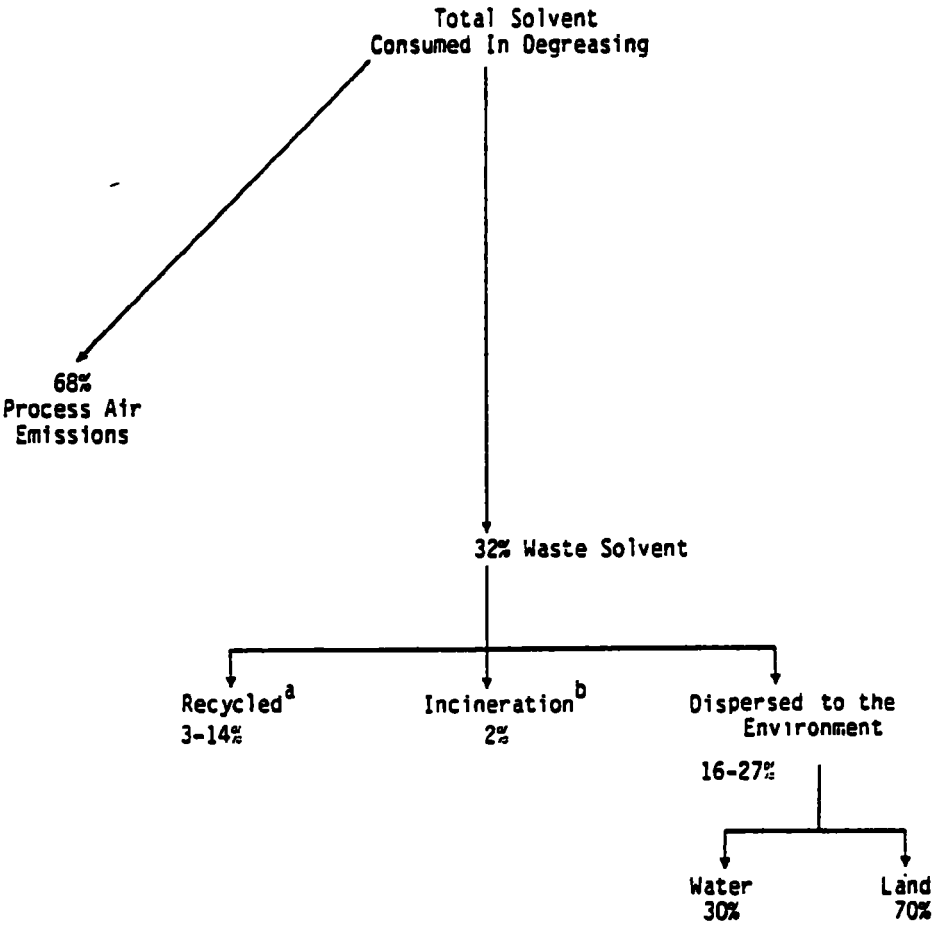

Figure 3-9. Disposition of Trichloroethylene Consumed in Solvent Degreasing for the Period 1954 - 1978

[a]It is assumed that only 10% of the total waste solvent generated was recovered from the period of 1954 to 1972, and that recovery practices increased (in a linear fashion) during the period of 1972 to 1978 to an estimated recovery/recycle level of 45% (EPA, 1977a). These assumptions were based on: personal communications with industry representatives (Stranges, 1980; Thorpe, 1980); the fact that the price of trichloroethene more than doubled during the period of 1972 to 1978 (USITC, 1972-1979); and the promulgation of EPA regulations controlling solvent use and disposal.

[b]Approximately 5% of waste solvent is assumed to be disposed of by incineration (EPA, 1977a), resulting in negligible air emissions.

[c]Percentage dispersed to the environment is dependent on recycle practices.

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4.0 DISTRIBUTION IN THE ENVIRONMENT

4.1 INTRODUCTION

Monitoring data are presented first in this chapter (Section 4.2). Available literature has been scanned to determine trichloroethylene concentration detected in various environmental media - air, water (including groundwater), soil, and biota. The second part of this chapter (Section 4.3) addresses the environmental fate of trichloroethylene.

4.2 TRICHLOROETHYLENE DETECTED IN THE ENVIRONMENT AND IN OTHER MEDIA

The U.S. EPA STORET files were searched for data concerning concentrations of TCE in ambient water and effluents. In six wells reported, TCE was found at 0.14 ug/l to 10 ug/l. Concentrations of 0.14 ug/l to 300 ug/l at about 180 ambient surface water monitoring stations were reported, but most had concentrations of 10 µg/l or less. At 17 stations, no TCE was found in the analysis. In 270 reports of concentrations in effluents, TCE was reported at concentrations of 0.01 to 1600 ug/l although all but a very few were less than about 10 µg/l. Comparatively few reports of TCE in sediments showed 0.07 to 580 µg/kg. Table 4-1 summarizes STORET ambient water data by region.

Table 4-2 summarizes typical TCE concentrations in different media. These readings are not specific to the U.S. In all media reported, typical TCE levels are 10^{-11} to 10^{-7} on a weight per weight basis. Most fall in the 10^{-9} to 10^{-8} w/w range.

Table 4-3 shows concentrations of TCE detected in human tissue. Most concentrations found in body fat, kidneys, livers, and brains were in the 1-10-µg TCE/kg wet tissue range. These measurements were taken in the U.K.

Table 4-4 shows TCE detected in foods from the UK. Concentrations in foods ranged from not detected to 60 µg/kg, with most less than 10 µg/kg. No US data were found.

Many reports of TCE concentrations found in air are available, both for the US and worldwide. These are given in Table 4-5. Background levels in areas away from centers of use (i.e. cities, etc.) over land range from <27 ng/m^3 to 4300 ng/m^3. On the basis of these measurements an estimate of the pervasive background level in air is 32-53 ng/m^3. Concentrations over the open ocean range from 1 ng/m^3 - 20 ng/m^3.

TCE has been found in wastewater, surface water, the ocean, drinking water, and groundwater. A summary of information found concerning these TCE levels is given in Table 4-6. TCE in wastewater was found at concentrations <10 ug/l to 8000 ug/l. TCE in surface water (rivers and lakes) was found up to 200 ug/l, but most levels were nearer

TABLE 4-1 TRICHLOROETHYLENE IN AMBIENT WATERS

River Basin/Region	Trichloroethylene (ug/l) Observations	% Observations in range <10	10.1-100	100.1-1000
Northeast	20	100		
North Atlantic	31	97	3	
Southeast	110	96	4	
Tennessee River	14	93		7
Ohio River	57	98	2	
Lake Erie	3	67		33
Upper Mississippi	19	74	21	5
Lake Michigan	94	87	13	
Missouri River	26	85	15	
Lower Mississippi	9	99	1	
Colorado River	3	100		
Western Gulf	18	100		
Pacific Northwest	78	99	1	
California	4	100		
Great Basin	1	100		
Unlabeled	1	100		
UNITED STATES	488	94	6	

Source: Combination of Remarked and Unremarked Data from U.S. EPA STORET Water Quality Information Systems, as of October 2, 1980.

Table 4-2 WEIGHT/WEIGHT COMPARISON OF TYPICAL TRICHLOROETHENE CONCENTRATIONS IN DIFFERENT MEDIA

DIFFERENT MEDIA

	Comparison of Typical TCE Concentrations (weight/weight)[a]	
Medium	Minimum	Maximum
Air	10^{-9}	10^{-8}
Rainwater	10^{-11}	10^{-9}
Surface Water	10^{-11}	10^{-9}
Potable Water	10^{-11}	10^{-9}
Sea Water	10^{-10}	10^{-9}
Marine Sediments	10^{-10}	10^{-9}
Marine Invertebrates	10^{-9}	10^{-8}
Fish	10^{-9}	10^{-8}
Waterbirds	10^{-9}	$>10^{-7}$
Marine Mammals	10^{-9}	$>10^{-8}$
Fatty Foods	10^{-9}	10^{-8}
Non-Fatty Foods	10^{-9}	10^{-9}
Human Organs	10^{-9}	10^{-9}
Human Body Fat	10^{-9}	10^{-8}

Source: McConnell et al. (1975)

[a] Concentrations have been expressed on a weight per weight basis in order to facilitate comparisons among different media. These values are not specific to the U.S.

TABLE 4-3 TRICHLOROETHYLENE CONCENTRATIONS IN HUMAN TISSUE[a]

Sample		Tissue	TCE Concentration (wet tissue) µg/kg
76	F	Body Fat	32
		Kidney	<1
		Liver	5
		Brain	1
76	F	Body Fat	2
		Kidney	3
		Liver	2
		Brain	<1
82	F	Body Fat	1.4
		Liver	3.2
48	M	Body Fat	6.4
		Liver	3.5
65	M	Body Fat	3.4
		Liver	5.2
75	M	Body Fat	14.1
		Liver	5.8
66	M	Body Fat	4.6
74	F	Body Fat	4.9

Source: McConnell *et al.* (1975)

[a]Data apparently from the UK.

TABLE 4-4 TRICHLOROETHYLENE DETECTED IN FOOD FROM THE UK

Food Item	TCE Concentration (µg/kg)
Dairy	
Fresh Milk	0.3
Cheshire Cheese	3
English Butter	10
Hens Eggs	0.6
Meat	
English Beef Steak	16
English Beef Fat	12
Pig Liver	22
Oils and Fats	
Margarine	6
Olive Oil (Spanish)	9
Cod Liver Oil	19
Vegetable Cooking Oil	7
Castor Oil	ND[1]
Beverages	
Canned Fruit Drink	5
Light Ale	0.7
Canned Orange Juice	ND
Instant Coffee	4
Tea (Packet)	60
Wine (Yugoslav)	0.02

TABLE 4-4 TRICHLOROETHYLENE DETECTED IN FOOD FROM THE UK (Continued)

Food Item	TCE Concentration (µg/kg)
Fruit and Vegetables	
Potatoes (S. Wales)	ND[a]
Potatoes (N.W. England)	3
Apples	5
Pears	4
Tomatoes (grown in reclaimed lagoon at a chemical plant)	1.7
Imported Black Grapes	2.9
Fresh Bread	7

[a] ND - Not Detected

Source: McConnell et al. (1975)

TABLE 4-5 SELECTED MEASUREMENTS OF TRICHLOROETHYLENE CONCENTRATIONS IN AIR

LOCATION	DATE	CONCENTRATION	COMMENT	REFERENCE
		min max mean $\mu g/m^3$		
East Coast Urban Areas	1974	<0.3 47 1-5		Lillian et al. (1975)
West Coast Urban Area	4/74 - 1/76	<3 - 34 $\mu g/m^3$	La Jolla, California	Su and Goldberg (1976)
Vicinities of TCE Manufacturing Sites	11-12-76	<5 - 1440 $\mu g/m^3$	Air was sampled in vicinity of four manufacturing plants - Dow Chemical, Freeport, TX - PPG Industries, Lake Charles, LA - Ethyl Corp., Baton Rouge, LA - Hooker Chemical, Taft, LA	Battelle Columbus Labs (1977)
Vicinity of TCE User	1/77	<1 - 235 $\mu g/m^3$	Air was sampled in the vicinity of a Boeing Company plant, Seattle, WA	
Industrialized Areas	pre 1979	Trace - 174 $\mu g/m^3$	New Jersey Houston, TX Niagara Falls/Buffalo, NY Rahway/Woodbridge, Boundbrook Passaic, NJ Baton Rouge, Geismar, Plaquemine, LA Houston, Deer Park, Pasadena, TX	Research Triangle Institute (1979)
Metalworking Shop in Sweden	pre 1963	Range Mean mg/m^3 0-2200 0-400	Degreasers in use	Ahlmark et al. (1963)

TABLE 4-5 SELECTED MEASUREMENTS OF TRICHLOROETHYLENE CONCENTRATIONS IN AIR (Continued)

LOCATION	DATE	CONCENTRATION	COMMENT	REFERENCE
Remote Areas - US	1975 - 1977	Range 0 - 3 $\mu g/m^3$	Most in 0 - 100 ng/m^3 range	
			Pacific N.W.	Cronn et al. (1977)
			Talladega National Forest	Holzer et al. (1977)
			California-Point Reyes, S.F. Bay	Singh et al. (1977)
			Pullman, Wash.	Grimsrud and Rasmussen(1975
Sea Stations	7-8/1972	Range 0 - 22 ng/m^3	Along a line from Lands End to Cap Blanc (Spanish Sahara)	Murray and Riley(1973)
		Mean 6 ng/m^3	North Atlantic	Su and Goldberg(1976)
	10/73	<65 ng/m^3		
Europe, Africa	1972, 1974	<100 ng/m^3		Murray and Riley 1973
				Cox et al. (1976)

TABLE 4-6 COMPILATION OF MEASUREMENTS OF TRICHLOROETHYLENE CONCENTRATIONS IN WATER

LOCATION	DATE	CONCENTRATION	COMMENT	REFERENCE
Surface Water - U.S.	1977	Range 0 - 5227 µg/l Mean 0 - 100 µg/l		Research Triangle Insitute (1979) Battelle Columbus Labs (1977)
Vicinities of TCE Manufacturing Plants	11-12/1976	0.1 - 5227 µg/l	Four manfacturing plants - Dow Chemical, Freeport, TX - PPG Industries, Lake Charles LA - Ethyl Corp., Baton Rouge, LA - Hooker Chemical, Taft, LA	
Marine-				
N.E. Atlantic Surface Water	pre 1973	0 - 10 ng/l		Murray and Riley (1973) Su and Goldberg (1976)
East Pacific Pacific Coast - US	1-7/75	0 - 15 ng/l		
Groundwater -				
Raw		Mean 29.72	Median 1.3	Range µg/l 0.2-125	13	38.5	Coniglio et al (1980)
Finished		6.76	0.31	0.21-53	25	36	
Groundwater - 8 states		35000 µg/l max			28% of 2894 wells were positive	
N.J. Groundwater		<1.0 µg/l	1-10	10-100	>100	73% of 397 wells positive
		377	41	15	4	
N.Y. Public Water System		Max 19 µg/l			18 of 39 positive	
Nassau Co., N.Y. Community Water Supply Wells	4/28/78	Max 300 µg/l			50 of 372 positive	
Massachusetts		0 - 1000 µg/l			13 communities	Special Legislative Commission on Water Supply (1979)

TABLE 4-6 COMPILATION OF MEASUREMENTS OF TRICHLOROETHYLENE CONCENTRATIONS IN WATER
(Continued)

LOCATION	DATE	CONCENTRATION	COMMENTS	REFERENCE
Other Surface/Drinking Water				
Finished Water Samples - US	May - July 1976	mean of positives 2.1 µg/l mean of all 0.5 µg/l <0.05 µg/l median	5 ng/l minimum quantifiable limit -28/113 positives in public water supplies in NOMS Phase II	Brass et al. (1977).
New Jersey Drinking Water		757 - 18017 ng/l	22/22 Samples	Research Triangle Institute (1979)
Jefferson Parish LA	2/7/77-8/5/77	Mean / Low / High 0.2 µg/l / 0.1 µg/l / 1.4 µg/l 0.087 µg/l / 0.2 µg/l / 0.5 µg/l	Mississippi River Water Water entering distribution system	
Jefferson Parish Tap Water		0.19 µg/l mean 1.6 µg/l high	found in 92/145 samples	
Surface Water		Mean / Median / Range µg/l	#cities sampled / %positive	Coniglio et al. (1980)
Raw		0.9 / 0.25 / 0.1-42	105 / 11.4	
Finished		0.47 / 0.26 / 0.06-3.2	133 / 32.3	
Water Supply Systems serving >75, 000 population		0.66 µg/l avg.	28/87 positive Phase I, II, III of NOMS	Pendeygraft et al. (1979)
Water Supply Systems serving <75,000 population		0.32 µg/l	9/26 positive 37/113 systems showed positive 7/87 systems serving >75,000 have >0.5 µg/l	
Wastewater -				
Semiconductor Raw Waste Water		min / max / mean 0.0066 / 3.5 / 0.25 µg/l	Flow proportioned average concentration 0.39 µg/l Flow rate 291.5 x 10^6 l/day	USEPA (1980a)

TABLE 4-6 COMPILATION OF MEASUREMENTS OF TRICHLOROETHYLENE CONCENTRATION IN WATER
(Continued)

LOCATION	DATE	CONCENTRATION	COMMENT	REFERENCES
Sewage Treatment Plant Effluents		max 10 µg/l	4/4 samples	EPA(1977b)
Finished Water		max 5 µg/l	8/18 samples	
Ambient		max 188/ µg/l	72/182 samples	
Rain				
La Jolla, Calif.		5 ± 2.6 (ng/l)		Su and Goldberg(1976)
Runcorn, U.K.		150		
Snow				
So. California		30		
Central California		<1.5		
Alaska		39 ± 8		
Ice		20	from a commercial machine	
Reservoir				
Lake		38 - 65		
Untreated Water		5.1 ± 4.6		

10 µg/l or less. This generally agrees with the STORET data shown in Table 4-1. In the vicinities of plants manufacturing TCE, concentrations as high as 5227 µg/l were found.

Drinking water was sampled in the National Organics Monitoring Survey. Where TCE was found in finished drinking water, concentrations were generally less than a few µg/l, with mean concentrations, if detected, less than 1 µg/l (see Table 4-6). With well water (groundwater) the situation is different. TCE has been found at concentrations of several hundred micrograms per liter and some have been in the high milligram per liter range. In finished groundwater used for drinking, concentrations up to 125 µg/l have been found. TCE was found in 28% of 2894 wells tested in eight states.

Soil samples were taken in the vicinities of TCE manufacturing and user sites (Battelle Columbus Labs 1977). Concentrations ranging from none detected to 5.6 µg/kg (dry weight) were found. Few other soil concentrations have been reported. Sediment samples at the same sites showed up to 300 µg/kg (dry weight) TCE.

Pearson and McConnell (1975) and Dickson and Riley (1976) report TCE concentrations found in marine fishes, birds, mammals, and algae in the UK. While these data are not specific to the U.S., they do indicate TCE levels that may be achieved. In marine fishes concentrations ranged from <0.1 µg/kg to 480 µg/kg. In marine birds and mammals TCE levels were between 2 µg/kg and 30 µg/kg. Marine invertebrates had levels ranging from not detected to 250 µg/kg. Marine algae showed up to 22 µg/kg. When expressed on a wet weight basis, the concentrations fall in the lower part of the concentration ranges reported and when expressed on a dry weight basis, the concentrations are in the upper part of the range.

4.3 ENVIRONMENTAL FATE

This section reviews the major environmental pathways and fate of trichloroethylene, as summarized in Figure 4-1. Atmospheric fate is discussed first, followed by discussions of fate in water and in soil.

Following discussions of environmental fate processes, information is presented concerning fate of TCE in solid waste materials and sewage treatment plants.

4.3.1 Atmospheric Fate

Trichloroethylene is not photolyzed in the atmosphere, but rather is photooxidized. TCE does not absorb light in the visible or near UV spectra of sunlight (Jaffe and Orchin 1962). Dahlberg (1969) determined the absorption spectra of TCE; as shown in Figure 4-2 the absorption spectra lie below the atmospheric cutoff limit of 290 nm.

Trichloroethylene forms many decomposition products when photooxidized in the atmosphere. The half-life for this process in nature has been estimated to be from a few hours to a few days. Decomposition

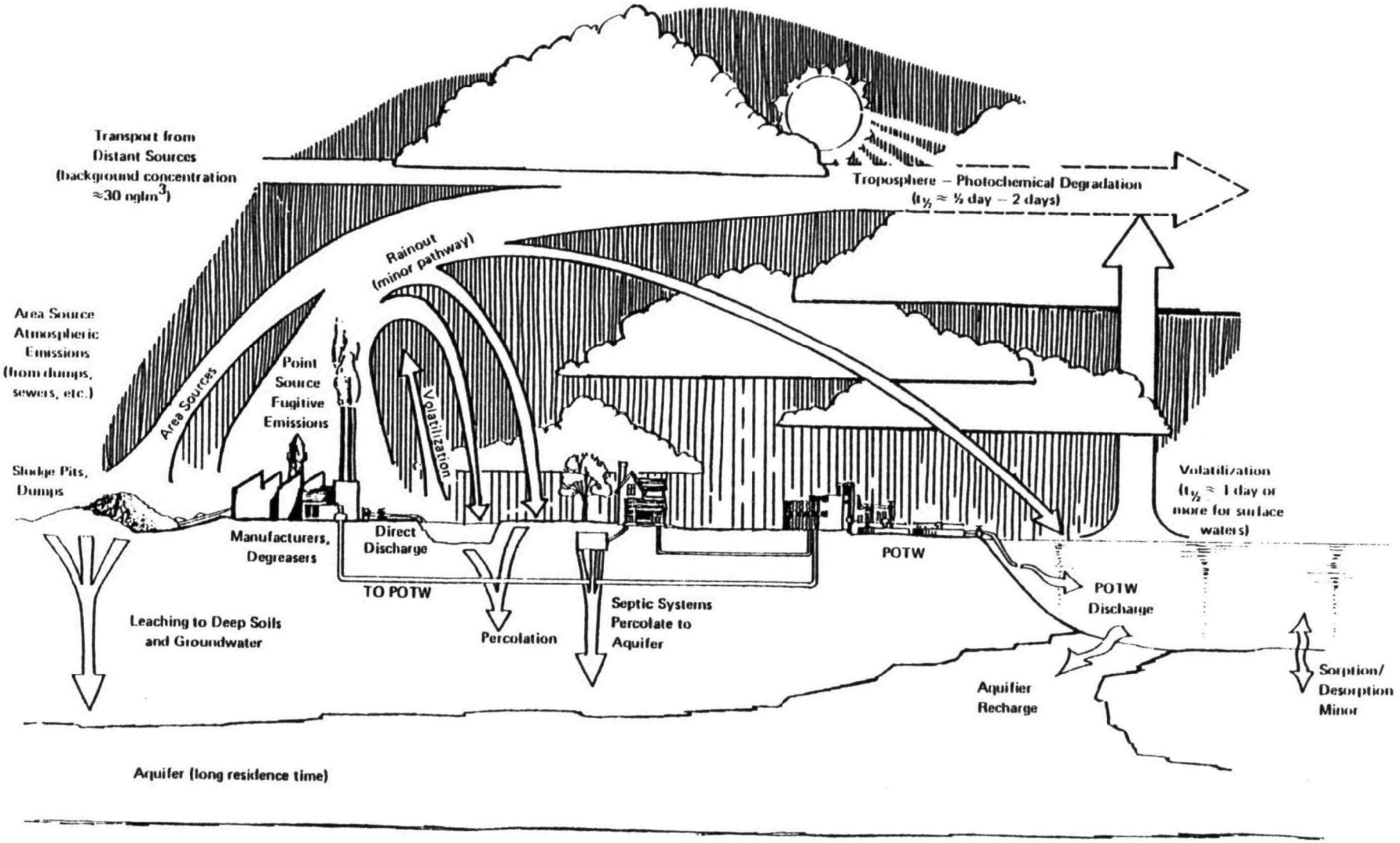
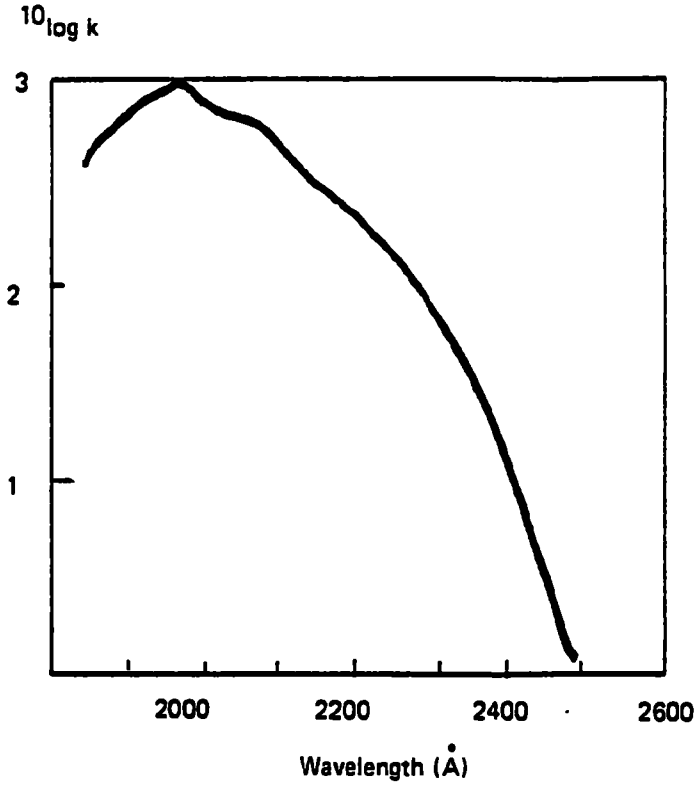


FIGURE 4–1 MAJOR PATHWAYS OF TRICHLOROETHYLENE IN THE ENVIRONMENT



Note: If the unit for the partial pressure of the absorbing molecule is at M, the absorption coefficient is defined by

$$k = \frac{T}{p \times 273 \times \ell} \cdot \ln \frac{I_o}{I} \ (cm^{-1})$$

Where T = Temperature, K
p = Partial Pressure of TCE
ℓ = Path Length of Light
I_o = Initial Intensity of Light
I = Measured Intensity of Light

Source: Dahlberg 1969.

FIGURE 4–2 ABSORPTION SPECTRA OF TRICHLOROETHYLENE

products include any or all of the following: phosgene, dichloroacetyl chloride, trichloroacetyl chloride, formyl chloride, hydrogen chloride, chlorine, carbon monoxide, chloral, ozone, formic acid, and nitric acid. These products further decompose at various rates.

Although no information was found concerning the propensity of TCE to sorb onto particulate, this is not expected to occur to a significant extent due to TCE's high vapor pressure.

4.3.2 Fate in Water

4.3.2.1 Volatilization

Several experimenters (Jensen and Rosenberg 1975, Dilling et al. 1975, Neely 1976, Dilling 1977) have determined in the laboratory the evaporation rate of trichloroethylene from water. (See Table 4-7) For most cases, a half-life of about 20 or 30 minutes was reported for studies in which evaporation from a stirred beaker was measured. The exception is the work of Jensen and Rosenberg (1975))in which TCE apparently evaporated from a partially covered aquarium containing seawater. The half-life in this case appeared to be 3-4 days.

Though these rates are indicative of the rapid volatilization of TCE from water, they are not directly applicable to environmental situations. Methods have been developed (Southworth 1979, Neely 1976, Liss and Slater 1974) whereby volatilization rates from water bodies can be estimated. (See Appendix G for a discussion of these methods.)

For representative environmental conditions (wind speed 3m/sec, current speed 1m/sec), the half-life in a 1-m deep stream is estimated to be about 3.5 hours. For lower wind speeds and stream depths up to 10 m, the estimated half-lives increase to about 11 days. These rates, on the order of hours or days, are indicative of TCE's propensity to volatilize rapidly from water bodies.

In a 1-m deep river flowing at 3.6 km/hr (1 m/sec) with a wind of 3 m/sec, 90% of the TCE is estimated to volatilize in about 11.7 hours, or 42 km downstream from the source. For the 10-m deep river, this time increases to 37 days, which would correspond to 3200 km downstream at a 1 m/sec flow rate. These estimates assume initially well-mixed conditions and constant environmental conditions. (The results of the EXAMS analysis discussed in Section 4.4.3 relate to this discussion.)

4.3.2.2 Hydrolysis

Trichloroethylene is reported to resist hydrolysis at 100°C, but oxygen accelerates the decomposition rate (Dilling et al. 1975). Products from dilute solution hydrolysis or oxidation have not been reported, but dichloroacetic acid and hydrogen chloride are likely products. In

TABLE 4-7 MEASURED EVAPORATION RATES FOR TRICHLOROETHYLENE

Experiment	Measured Half-life	Experimenter
250 m beaker 200 m solution, 6.5 cm deep Gently stirred c_o = 1 mg/	22 min	Neely (1976)
250 m beaker 200 m solution, 6.5 cm deep 200 rpm stirrer c_o = 1 mg/	19 min	Dilling et al. (1975)
No stirring	>∿90 min	
250 m beaker 200 m solution, 6.5 cm deep 200 rpm stirrer c_o = 1 mg/	16.9-24.5 min	Dilling (1977)

sealed tubes kept in the dark, the half-life of TCE was about 10.7 months at 25°C (Dilling et al. 1975). The reaction may have been oxidation.

TCE is, therefore, not expected to hydrolyze at an appreciable rate under environmental conditions, although hydrolysis may be significant in groundwater, where other fate mechanisms are not operative.

4.3.2.3 Photolysis

TCE-water solutions were sealed in tubes and placed in sunlight (Dilling et al. 1975). TCE loss was noted. Most of the activity "was probably due to oxidation and was probably free radical in character." Half-life in this experiment was about 6-8 months. This observation is in agreement with predictions based on vapor-phase photolysis studies, where it was shown that TCE disappeared when irradiated with long wavelength light in the presence of nitric oxide or nitrogen dioxide. Dichloroacetic acid and hydrogen chloride are likely products. Photolysis is, therefore, not expected to be an important loss mechanism for TCE from water under environmental conditions.

4.3.2.4 Biodegradation

TCE is metabolized by higher organisms (Versar 1979) and its metabolites (chloracetics) are readily degradable. (See Chapter 6.0). However, microbial degradation does not appear to play a significant role in the breakdown of trichloroethylene in the environment (McConnell 1975, Wilson et al. 1980).

4.3.2.5 Sorption/Desorption

Very little directly useful information is available concerning sorption of TCE. The effects of clay and peat moss in a solution on the evaporation of TCE from the solution were investigated (Dilling et al. 1975). Dry granular bentonite clay exhibited a sorption partition coefficient of ∿300-375. (This coefficient was not derived by the investigators but was derived from results presented in the paper.)

Other evidence (Versar 1979) shows no correlation between sorbed concentration and water concentration. Coarse gravels have little sorptive capacities. An octanol/water partition coefficient has been reported (SRI 1980) to be 69.2 and an organic carbon partition coefficient of 38 was derived from this value Versar (1979) reports a log octanol/water partition coefficient of 2.29. An organic carbon partition coefficient of 100 has been derived from a regression equation reported by Choiu et al. (1979).

Monitoring data from the vicinity of plants manufacturing TCE show concentrations in sediments to be similar to concentration in the water from which sediment samples were taken (USEPA 1977a). This indicates that little sorption occurs.

The section on waste treatment (4.3.4.1) discusses sorption of TCE onto sewage solids.

4.3.3 Fate in Soil

4.3.3.1 Transport and Volatilization

Wilson et al. (1980) performed studies to determine transport and fate of chemicals applied to soil; among these chemicals was trichloroethylene. Most of the TCE in a water solution applied to a column of sandy soil volatilized, while the rest percolated through a 140-cm column. Little, if any, was biodegraded. Table 4-8 shows the soil characteristics. Table 4-9 shows test results. Soil columns were not saturated.

In their comparison of volatilization from the soil column to volatilization from water, Wilson et al. found that volatilization from soil was inhibited by the soil by about a factor of ten. For an initial concentration of 0.90 mg/l, the measured hourly flux from soil was 0.34 $\mu g/cm^2$. Calculated flux from water was estimated to be 3.0 $\mu g/cm^3$. For a volatile chemical such as TCE, volatilization is probably limited by diffusion through air-filled pores.

Transport of TCE through the soil column, when defined as (interstitial water velocity/velocity of pollutant), was inhibited minimally by the soil. In a laboratory study using a sandy soil, the factor was at most 1.6 ± 0.2 and in field tests using a soil with a higher organic carbon content, the factor was at most 3.1 (Wilson et al. 1980). This increase in the retardation factor is probably due to sorption of TCE onto organic matter.

The following conclusions can be drawn from Wilson et al. (1980):

- Most TCE applied to soil will volatilize.
- TCE percolating through the soil column is minimally retarded by sandy soils. Organic matter increases the retardation rate somewhat.
- Volatilization from the soil column occurs at a rate about ten times lower than in a water column of similar depth.

4.3.3.2 Decomposition

Versar (1979) indicated that some organochlorine compounds degrade more rapidly in the presence of metallic iron than in an iron-free situation. This observation was mentioned in connection with lifetimes in water, but it may have some significance for TCE in soil as well. No rates or supporting data were included that would allow the importance of this observation to be assessed. No trichloroethylene degradation is assumed to occur in soil.

TABLE 4-8 COMPOSITION OF SOILS USED IN STUDIES OF TRICHLOROETHYLENE FATE IN SOIL

	Laboratory Column Tests		Field Tests	
	Average	% Range	Average	% Range
Sand	92	(95-89)	76	(91-64)
Silt	5.9	(8.0-4.0)	5.7	(9.4-3.0)
Clay	21	(3.5-1.5)	19	(33-2.6)
Organic Carbon	0.08	(0.22-0.02)	0.13	(0.25-0.05)

Source: Wilson et al.(1980)

TABLE 4-9 FATE OF TRICHLOROETHYLENE APPLIED TO A SOIL COLUMN IN THE LABORATORY

Concentration Applied, mg/l	% Volatilized	% in Column Effluent	% Degraded or not accounted for
0.90	59 ± 14	28 ± 1	14 ± 15
0.18	90 ± 18	21 ± 13	-10 ± 11

Source: Wilson et al. 1980

4.3.4 Other Fate Processes

Trichloroethylene is found in sewage and the effluent from sewage treatment plants. It is also found in landfills from waste deposited in the landfills. These are important routes for TCE input into the environment because of their potential effects on ground and surface waters. This section discusses TCE behavior in solid waste and in sewage treatment.

4.3.4.1 Waste Materials

Jones et al. (1977/78) performed experiments in which a TCE solution was mixed with fresh, untreated domestic refuse from a landfill. The refuse leachate was monitored over a period of a few months to determine TCE concentrations in the leachate.

A TCE preparation (Triklore) was mixed with equal volumes of used crankcase oil. The solution was mixed with the refuse to give 200 mg/kg and 500 mg/kg concentrations of TCE. Three canisters were prepared for each TCE concentration. One was left uncovered, one was closed, and one was fully saturated with water. All were left outdoors.

Only the leachate from the 500 mg/kg closed column and the 500 mg/kg fully saturated column contained >2 mg/kg concentration of TCE. Leachate from the closed column contained 4-7 mg/kg of TCE over a 4-month elution period, and 9-13 mg/kg was detected in leachates from the saturated column over a similar time period. None was detected in the open column or the 200-mg/kg columns.

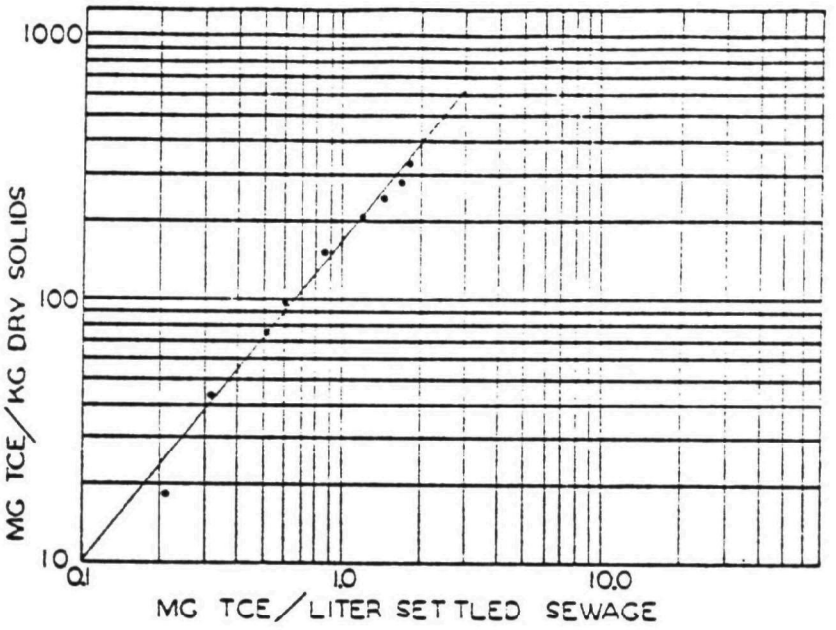
These experimental results indicate that evaporation and adsorption may result in leachate concentrations of TCE that are about two orders of magnitude lower than the initial concentration in the domestic waste being leached.

Sewage

Anaerobic digestion is inhibited by TCE at 200-1200 mg/kg dry solids (Camisa 1975). In an activated sludge system, 300 mg/l caused little repression of the glucose removal rate. More than 300 mg/l caused slight repression, and more than 500 mg/l caused significant repression of bacterial activities and partial inhibition of enzymes.

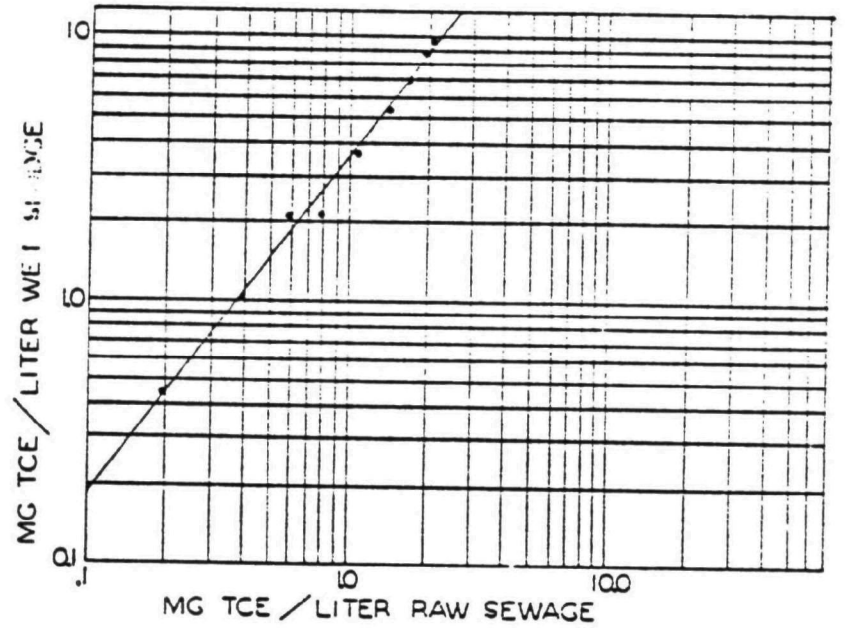
TCE partitions into the sludge. The maximum concentration expected to sorb onto sludge is 3030 mg/kg dry solids. This concentration has the potential to affect anaerobic digestion adversely.

Camisa (1975) determined the partitioning of TCE among several different components of sewage, wet sludge, settled sewage, raw sewage, and dry solids. The results are shown in Figures 4-3 to 4-5 and Table 4-10.



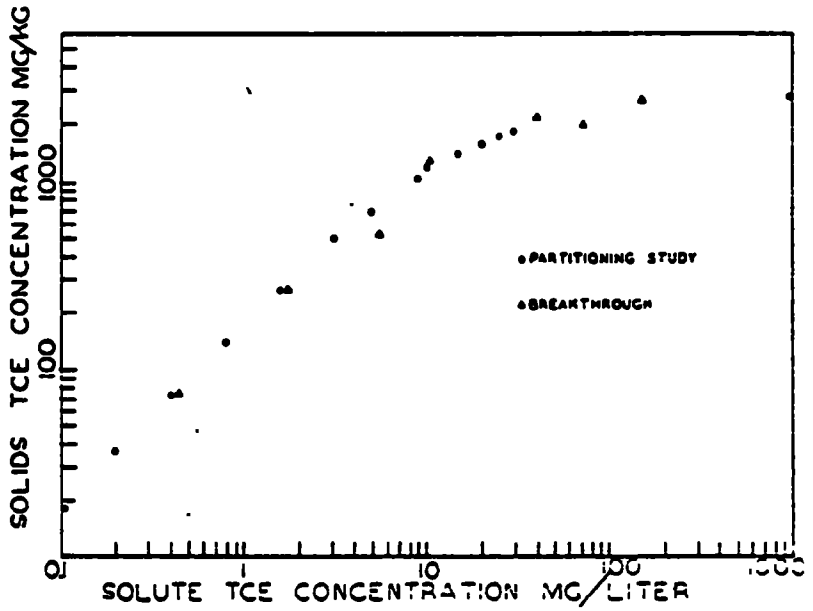
Source: Camisa 1975

FIGURE 4–3 CORRELATION BETWEEN TRICHLOROETHYLENE IN DRY SOLIDS AND THAT IN SETTLED WASTEWATER FROM SEWAGE TREATMENT



Source: Camisa 1975

FIGURE 4–4 CORRELATION BETWEEN TRICHLOROETHYLENE IN WET SLUDGE AND THAT IN UNTREATED SEWAGE



Source: Camisa 1975

FIGURE 4–5 PLOT FOR MAXIMUM TRICHLOROETHYLENE SORPTION ON DRIED SLUDGE SOLIDS

TABLE 4–10 TRICHLOROETHYLENE ADSORPTION ONTO SLUDGE–DATA SUMMARY

µg TCE added to 20 l	mg TCE·l raw wastewater	Settled sludge (%) solids	Volume of settled sludge (ml)	Weight of dried sludge (g)	µg of TCE recovered in sludge*	µg of TCE recovered in supernatant*	mg TCE kg dried sludge	mg TCE l supernatant
3,962	0.198	2.36	275	6.49	121	4,097	18.6	0.207
7,925	0.396	2.30	160	3.68	161	6,136	43.7	0.309
11,888	0.594	2.72	200	5.44	408	9,945	75.1	0.502
15,850	0.792	2.10	140	2.96	287	12,037	97.1	0.600
21,632	1.08	2.40	240	5.77	877	16,948	152.0	0.861
28,540	1.42	2.51	248	6.24	1,297	23,562	208.0	1.190
34,832	1.74	2.82	169	4.79	1,163	27,992	243.0	1.430
38,974	1.94	3.10	278	8.62	2,473	33,617	287.0	1.700
40,369	2.01	2.90	178	5.18	1,693	35,084	327.0	1.790

* Supernatant and sludge TCE totals have both been corrected to 100 percent recovery, based on an average distillation recovery of 87.6 percent.

Source: Camisa 1975

TABLE 4-11 TRICHLOROETHYLENE CONCENTRATIONS AT VARIOUS STAGES OF WASTEWATER TREATMENT

	TCE Concentration (µg/l)				TCE Concentration (µg/l)					
Plant	Influent	Effluent Pre-Cl.	Final Effluent	% Rem.	Primary Sludge	Combined Sludge	Gravity Thickener Overflow	Heat-Treated Sludge	Heat-Treated Decant	Digested Sludge
1	28	5	4	86	284	-				
2	2	0	0	100	-	<5				
3	2	1	<5	100	38	-	3			
4	497	-	37	93	-	467				120
5	49	-	14	71	163	-				2
6	487	-	64	87	30	-				4
7	17	4	-	76	-	2		7	<5	

Plant	Stage	Analyzed	Times Detected	% Detected	Average	Minimum	Maximum
8	Total Influent	4	4	100	30	11	78
8	Secondary Effluent (Chlorinated)	4	1	25	0	<2	1
9	Total Influent	6	6	100	33	3	55
9	Secondary Effluent (Chlorinated)	ND[a]	ND[a]	ND[a]	ND[a]	ND[a]	ND[a]

[a]Not Detected.

Source: Burns and Roe (1980)

Barrett (1972) investigated TCE in the gas from a sewage digester. TCE concentrations in the gas peaked immediately after the addition of TCE and then dropped steadily. Barrett (1972) attributed this behavior to the ability of the digester to digest the solvent, but it also seems plausible that the concentration in the gas was dropping due to depletion of TCE in the waste by volatilization. Barrett (1972) did note that some of the solvent was stripped out by the gas during digestion, somewhat confirming Camisa's (1975) results.

Barrett (1972) determined that in a digester with a 20-day retention period, the digester could survive shock doses of 1200 mg TCE/l sewage. This value is much higher than the 10-60 mg/l concentrations reported to cause inhibition of anaerobic digestion by Versar (1979) (4.3.2.4)

Data from U.S. EPA (1976) show TCE concentrations in water from sewage treatment plants in several cities to be 40.4 mg/l in influent before treatment, 8.6 mg/l in effluent before chlorination, and 9.8 mg/l in effluent after chlorination. Other data from a recent study (Burns and Roe 1980) give TCE concentrations for nine plants at various stages during the wastewater treatment process. As shown in Table 4-11, between 70% and 100% of the TCE appears to be removed between influent and effluent.

4.4 ENVIRONMENTAL FATE MODELLING

4.4.1 Overview

The environmental fate of trichloroethylene was analyzed through the use of mathematical models. These models are useful to indicate the behavior of chemicals in the absence of measured environmental processes or concentrations. The methods also are useful in estimating ambient concentrations under other circumstances, e.g., a reduction in environmental releases or different environmental conditions.

A fugacity model was used to estimate equilibrium concentrations in a river-basin-sized region. The EXAMS model was used to investigate TCE behavior in water.

4.4.2 Equilibrium Model

Concentrations in air, water, and soil were compiled using a Level II fugacity model (Mackay 1979). Fugacity can be regarded as the "escaping tendency" of a chemical substance from a compartment. When the escaping tendencies, the fugacities, from two or more compartments are equal they are in equilibrium and there is no net movement between compartments. The fugacities are proportional to concentrations; hence, chemical concentrations in each compartment can be estimated. Table 4-12 shows the input data used.

The fugacity coefficients, fugacity, masses in each subcompartment, and concentrations in each compartment were calculated. These are shown in Table 4-13.

TABLE 4-12 DATA USED IN LEVEL II FUGACITY CALCULATIONS[a]

Volume of the air compartment	$2.6 \times 10^{14} m^3$ (1 km depth)
Volume of the water compartment	$1.04 \times 10^{10} m^3$ (1 m depth)[b]
Volume of the soil compartment	$2.6 \times 10^{10} m^3$ (10 cm depth)
Density of soil	2×10^6 g/m^3
Organic carbon content of soil	15%
Temperature	20°C
Advection	None
Henry's Law Constant	0.0095 atm-m^3/mole
K_{oc}	100
Molecular Weight	131.4 g/mole
Photooxidation Rate Constant	$k_1 = 510$ yr^{-1}
Other rate constants (soil, water)	k_2, $k_3 = 0$
Input rate to compartment	$I = 3.12 \times 10^7$ moles/yr (4.1×10^6 kg/yr)

[a]Some input data are assumed values; some data are from the literature and are discussed in the appropriate sections.

[b]Water covers 4% of the region. Average water depth was arbitrarily assumed to be 1m since no data were found concerning stored water in the region.

TABLE 4-13 ESTIMATED TRICHLOROETHYLENE CONCENTRATION IN AIR, WATER AND SOIL DETERMINED BY FUGACITY CALCULATIONS

Sub-Compartment	Fugacity Coefficient	Mass In Subcompartment	Concentration In Subcompartment[a]
Air	41.6 $mol/m^3 atm$	8040 kg	30 ng/m^3
Water	105.3 "	0.8 kg	0.08 ng/l
Soil	3158 "	60 kg	1.2 ng/kg
		Σ = 8100 kg	

Rate of removal from air -- 4.1×10^6 kg/yr

Average Residence Time of TCE in the compartment -- 0.72 days

[a]Under equilibrium conditions the ratios between concentrations in the three media are independent of the volumes assumed for the media.

Reductions in emissions are reflected linearly in reductions in concentrations. Hence, a reduction by one third in emissions results in a similar reduction by one third in ambient equilibrium concentrations.

4.4.3 EXAMS

For the purpose of examining the probable fate of TCE in various aquatic environments under conditions of continuous discharge, the EXAMS (Exposure Assessment Modelling System) model AETOX 1 was implemented (USEPA 1980b). Rate constants and physical/chemical properties thought to influence the fate of TCE in the water environment are presented in Table 4-14 (SRI 1980). An arbitrary loading rate of 1.0 kg/hr was chosen for purposes of comparing different systems. Six prototype systems were simulated to provide a range of environmental conditions: pond, eutropic and oligotrophic lakes, and rivers (average, turbid and coastal plain).

As would be expected, in relatively static systems (ponds and lakes) in which physical transport processes did not dominate, volatilization was the most important removal mechanism, responsible for a 93-96% loss of the equilibrium TCE mass. Table 4-15 presents information on the distribution and transformation of TCE in the different systems. In all the river systems, (1 km segments) transport downstream alone accounted for at least 85% of the removal, and at a much faster rate than volatilization (on the order of hours rather than days). The overall time for self-purification (following cessation of discharge) was, thus, over 2 months for the lakes, approximately one month for the pond and less than 2 days for the rivers.

In order to provide a better understanding of the fate of trichloroethylene continually discharged into a river (flow = 2.4×10^7 m^3/day, depth = 4 m), the EXAMS river system was modified by extending its length by various increments from 2 km up to 1000 km from the point source. At approximately 50 km downstream, slightly more than 50% of the total mass had volatilized, a negligible amount had oxidized, and the remainder was transported further downstream. One hundred km downstream, 75% of the total amount had volatilized, and by 500 km downstream, about 98% was volatilized. Chemical oxidation had little effect on TCE concentration; even at 1000 km downstream; the fraction of the total mass lost via this process was <1%.

Table 4-16 presents the simulated TCE concentrations in different environmental compartments (water column, sediment, plankton and benthos) at steady-state conditions. Water concentrations were approximately 0.1-3 mg/l in the pond and lake systems and considerably lower (due to dilution and flow rates), 1-10 µg/l, in the river systems. Sediment concentrations were variable, ranging from ~0.3 µg/kg up to about 5000 µg/kg. The highest levels were in the pond where both volatilization and physical transport were not fast enough to remove TCE before a slight accumulation above water column concentrations could occur.

TABLE 4-14 PARAMETERS FOR TRICHLOROETHYLENE USED IN EXAMS ANALYSIS [a]

Property	Value	Units
Molecular Weight	131.4	g/mole
Solubility	1100	mg/l
Liquid Phase Transport Resistance	0.548	unitless ratio
Henry's Law Coefficient	9.1×10^{-3}	m^3/mole
Vapor Pressure	57.9	torr
Partition Coefficient:		
• Biomass/Water	11.4	$\frac{ug/g}{mg/l}$
• Sediment/Water	38.0	$\frac{mg/kg}{mg/l}$
• Octanol/Water	69.2	$\frac{mg/l}{mg/l}$
Chemical Oxidation Rate Constant		
• Water	6.0	mole/l/hr
• Sediment	1×10^{-3}	mole/l/hr

[a] All data from SRI (1980).

TABLE 4-15 THE FATE OF TRICHLOROETHYLENE IN VARIOUS GENERALIZED AQUATIC SYSTEMS[a]

System	% of TCE Residing in Water at Steady-State	% of TCE Residing in Sediment in Steady-State	% Transformed by Chemical Processes	% Transformed by Biological Processes	% Volatilized	% Lost by Other Processes[b]	Time for System Self-Purification[c]
Pond	93.3	6.7	0	0	93	7	620 hr.
Eutrophic Lake	>99.9	<0.1	0	0	96	4	63 days
Oligotrophic Lake	>99.9	<0.1	0	0	95	5	68 days
River	99.2	0.8	0	0	1.7	98.3	13 hr.
Turbid River	99.4	0.6	0	0	1.7	98.3	10 hr.
Coastal Plain	98.5	1.5	0	0	15	85	47 hr.

[a]All data simulated by the EXAMS model (see text for further information).

[b]Including loss through physical transport out of system.

[c]Estimate for removal of ca. 75% of the toxicant accumulated in system. Estimated from the results of the half-lives for the toxicant in bottom sediment and water columns, with overall cleansing time weighted according to the toxicant's initial distribution.

TABLE 4-16 ESTIMATED STEADY-STATE CONCENTRATIONS IN VARIOUS GENERALIZED AQUATIC SYSTEMS RESULTING FROM CONTINUOUS TRICHLOROETHYLENE DISCHARGE AT 1.0 kg/hr[a]

System	Loading	Water--Dissolved (mg/l)	Water--Total (mg/l)	Bottom Sediment (mg/l)	Maximum In Sediment Deposits (mg/g)	Plankton (µg/g)	Benthos (µg/g)	Total Steady State Accumulation (kg)	Total Daily Load (kg/day)
Pond	1.0 kg hr^{-1}	2.5	2.5	1.3	5.3	28	14	53	24
Eutrophic Lake		0.13	0.13	4.3×10^{-3}	7.8×10^{-3}	1.5	4.9×10^{-2}	310	24
Oligotrophic Lake		0.14	0.14	1.6×10^{-3}	3.0×10^{-3}	1.5	1.8×10^{-2}	350	24
River		9.9×10^{-4}	9.9×10^{-4}	3.3×10^{-4}	9.3×10^{-4}	1.1×10^{-2}	3.7×10^{-3}	0.89	24
Turbid River		9.9×10^{-4}	9.9×10^{-4}	6.5×10^{-4}	7.4×10^{-4}	1.1×10^{-2}	7.5×10^{-3}	0.89	24
Coastal Plain River		9.3×10^{-3}	9.3×10^{-3}	3.1×10^{-3}	1.3×10^{-2}	0.11	3.6×10^{-2}	8.0	24

[a]All data simulated by EXAMS model (see text for further information).

Concentrations in biota were generally one to two orders of magnitude above water levels which is compatible with observed bioaccumulation levels (see Section 6.2 for a discussion of biological fate).

Based on the results of the EXAMS run and dependent on the assumptions of the Model and the rate constants used as input, some conclusions can be drawn about TCE's potential behavior in water. In relatively static systems with slow flow rates (e.g. lakes) TCE's persistence is a function of volatilization. In more dynamic river systems, physical transport processes are much more competitive for TCE, transporting the chemical over a considerable distance before volatilization can remove a significant amount from a segment of the river. Transformation processes and bioaccumulation will not significantly reduce water concentrations. The sediment layer does not appear to absorb TCE at levels much above water concentrations; in fact, these levels are sometimes lower than water levels. Since volatilization is such an important fate mechanism, then conditions such as high temperature, high wind speeds, and high water turbulence would increase removal due to volatilization.

4.5 SUMMARY

Trichloroethylene appears in all environmental media - air, water (including groundwater), and soil. Concentrations detected in the atmosphere range from a background level of 27-100 ng/m^3 to ~1.5 mg/m^3 in the vicinity of TCE production sites. Concentrations detected in surface water range from none to 188 µg/l. When detected in surface water, however, TCE is generally found at a concentrations on the order of 1-10 µg/ℓ. In groundwater, concentrations up to 35 mg/l have been detected, but monitoring surveys indicate concentrations to be mostly in the <1-100 µg/ℓ range. Data on only a few soil samples were found. These showed soil concentrations between none detected and <10 µg/kg (dry weight). Data concerning concentrations in sediments are too few to indicate specific sorption characteristics, although TCE does sorb and has been found in sediments.

The environmental fate of trichloroethylene has been assessed. TCE is highly volatile. Its half-life in surface waters is estimated to be on the order of a few hours to a few days, depending upon the characteristics of the water body. TCE also volatilizes from soil if exposed to the air although estimates of the volatilization rate are imprecise. The process does appear to occur up to ten times more slowly than from water of a depth similar to that of the affected soil volume. The ultimate disposition of TCE appears to be atmospheric photooxidation. Half-life in the atmosphere due to photooxidation is estimated to be about 1 day, with a possible range from 12-48 hours. TCE does not undergo other chemical fate processes to any appreciable degree. Based on this information, the ultimate fate of TCE is expected to be destruction by atmospheric photooxidation following direct emission or volatilization from water or soil.

Fate processes have been modelled for an environmental situation. The results of the model agree fairly well with monitoring data. Background air concentration under current release conditions is about 30 ng/m^3, while in water and soil ambient concentrations are negligible, less than 1 µg/l and 1 µg/kg, respectively,

This positive evaluation indicates the usefulness of the model in estimating environmental concentrations. Ambient concentrations due to changes in emissions have been estimated using the model. The model used shows the emission reductions are reflected linearly in reduction of ambient equilibrium concentrations. Hence, a reduction in emissions by one third or one half results in a similar reduction in environmental concentrations.

The EXAMS model showed that volatilization was the most significant removal mechanism for TCE in water bodies. In the river system analyzed, half of the TCE had volatilized in 50 km downstream, and 98% was gone 500 km downstream. Other fate mechanisms contributed minimally to the loss of TCE from the water.

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5.0 EFFECTS AND EXPOSURE--HUMANS

5.1 HUMAN TOXICITY

5.1.1 Introduction

The human health effects of trichloroethylene have been investigated extensively, and several comprehensive reviews on the toxicity of TCE are available (MRI 1979; Waters et al. 1976; USEPA 1979; NIOSH 1973, 1978; NAS 1977; von Oettingen 1964; Browning 1965). Therefore, this chapter focuses on the data that provide the most useful assessment of acceptable limits of human exposure to TCE and the consequences of such exposure.

5.1.2 Metabolism and Bioaccumulation

5.1.2.1 Absorption and Distribution

Trichloroethylene can be absorbed by inhalation, ingestion, or cutaneous exposure. Inhalation is the most important route of entry. Persons inhaling TCE will retain between 50% and 76% and eliminate the remaining TCE unchanged (von Oettingen 1964, Bauer and Rabens 1977, Bartonicek 1962).

Numerous cases of human poisoning following ingestion of TCE attest to its absorption from the gastrointestinal tract (Waters et al. 1976, MRI 1979). Rats administered an oral dose of TCE expired 72-85% in air and 10-20% in urine. This indicates extensive absorption from the gastrointestinal tract (Daniel 1963).

Though TCE has been demonstrated to penetrate intact skin, it is considered unlikely that absorption of toxic quantities would occur by this route. Stewart and Dodd (1964) detected TCE in alveolar air following insertion of a person's thumb in TCE for 30 minutes. The mean peak of TCE in expired air was 2.69 mg/m^3.

Sato and Nakajima (1978) observed the effects of immersion in TCE of one hand by each of four healthy males wearing self-contained breathing apparatus. The end tidal air concentrations for the first 2 hours following a 30-minute immersion were approximately twice as high as the concentrations noted after inhalation of 538 mg/m^3 TCE for 4 hours. This occurred even though uptake through skin was approximately one-third of uptake by inhalation.

In mice, percutaneous absorption of TCE was found to increase linearly with time over a period of 5 - 15 minutes (Tsuruta 1978). An in vivo absorption rate of 7.82 $\mu g/min/cm^2$ was determined. The penetration rate may be somewhat higher since all TCE metabolites were not determined. The penetration rate with excised skin was calculated to be 12.1 $\mu g/min/cm^2$. If a similar absorption rate is assumed for man, immersion of both hands

($\sim$800 cm^2) into TCE for 1 minute would result in the absorption of approximately 6.3 - 9.7 mg TCE, or approximately one-third the uptake that would result by inhalation.

Following absorption, the blood transports TCE to body tissues. The mechanism of this transport is unknown. Because of its lipid solubility, TCE that is not immediately metabolized may be extracted from blood by fatty tissue (Waters et al. 1976). TCE metabolites are known to bind irreversibly to sulfhydryl groups in vivo and, to a lesser extent, to free amino groups of proteins, with the greatest protein binding seen in the liver (Bolt and Filser 1977). Van Duuren (1977) noted that the in vitro binding of TCE to liver microsomal proteins of male B6C3F1 mice was significantly higher than that seen in male Osborne-Mendel rats. Furthermore, the binding was higher in male mice than in female mice. The significance of these data will be discussed in the carcinogenicity section (See Section 5.1.3.1).

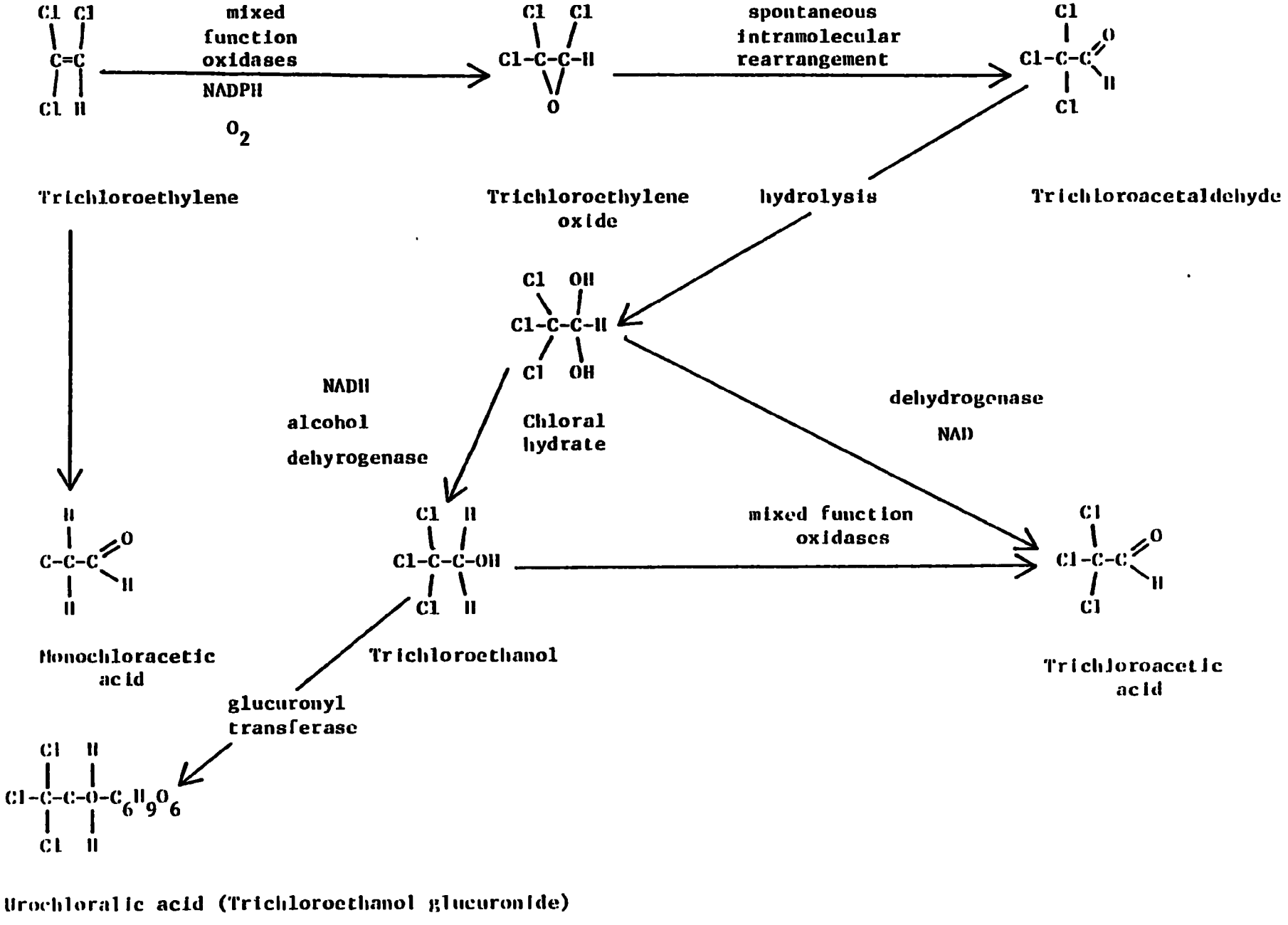
5.1.2.2 Biotransformation and Elimination

Several metabolic pathways have been suggested for TCE. The generally accepted pathway based on known metabolic products and possible intermediates is shown in Figure 5-1. Although the fraction of various eliminated metabolites differs, the overall pattern appears similar for the various mammalian species studied. The first step involves oxidation of TCE to chloral hydrate via an epoxide intermediate; the epoxide is believed to undergo spontaneous intramolecular rearrangement to form trichloroacetaldehyde, which is subsequently hydrolyzed to the sedative-hypnotic, chloral hydrate (Daniel 1963, Waters et al. 1976, Van Duuren 1977, Nomiyama and Nomiyama 1979b). Oxidation or reduction rapidly converts chloral hydrate to trichloroacetic acid (10-36%), or trichloroethanol (32-59%), respectively. Trichloroethanol is conjugated with a glucuronide and excreted in the urine in the same way as trichloroacetic acid and a minor metabolite, monochloroacetic acid (4%)(Leibman 1965, Soucek and Vlachova 1960, Bauer and Rabens 1977).

Parchman and Magee (1980) reported the production of $^{14}CO_2$, a previously unreported metabolite of TCE, in male Sprague-Dawley rats and male B6C3F1 mice injected intraperitoneally with low doses of ^{14}C-TCE (0.2-6 mg/kg). Approximately 70% of the label was excreted in urine within 6 hours; by 24 hours, 10% of the label was recovered as $^{14}CO_2$. Injection of higher doses (1.5-3 g/kg) produced "little CO_2," an observation suggesting dose-related differences in metabolite pathways.

The rate of conversion of TCE to chloral hydrate is relatively slow; once chloral hydrate is produced, however, the biotransformation of chloral hydrate to trichloroethanol and trichloroacetic acid proceeds so rapidly that chloral hydrate cannot be detected in plasma (Nomiyama and Nomiyama 1979b). For example, Marshall and Owens (1955) detected no chloral hydrate in human plasma 5-30 minutes after ingestion of 30 mg/kg chloral hydrate. Trichloroethanol has a biological half-life of $\sim$13 hours

Figure 5-1 Metabolism of Trichloroethylene in Animals and Man



in humans compared with a value of 50-90 hours for trichloroacetic acid. Thus, excretion of urochloralic acid is rapid compared to the slower rate of excretion for trichloroacetic acid, presumably as a result of high protein-binding affinity of the acid (Muller et al. 1974). Ikeda (1977) found a linear correlation between the concentration of TCE in the work environment and the level of total trichloro-compounds in urine. The trichloroethanol level was also linearly related to the TCE concentration, while trichloroacetic acid levels deviated from the linear relationship when the TCE level exceeded 268 mg/m^3.

Monster et al. (1976) exposed four male volunteers either at rest or rest combined with exercise to 376 or 753 mg/m^3 TCE for 4 hours at 3-week intervals. These experiments indicated that the concentration of TCE and trichloroethanol in blood and expired air were proportional to the dose. Workload increased the mean dose by approximately 40%; however, it did not influence distribution or metabolism. After 66 hours, 67% of the dose was recovered: 10% exhaled as TCE and 57% eliminated in urine as trichloroethanol (39%) and trichloroacetic acid (18%). Similar results were obtained by Astrand and Ovrum (1976) in a study with 15 healthy men exposed to 538-1076 mg/m^3 TCE during four 30-minute rest-exercise periods.

Nomiyama and Nomiyama (1979a) examined the metabolism of TCE in man, rabbits, and rats. Five male students were exposed to 1345-2044 (mean 1695) mg/m^3 TCE for 160 minutes (equivalent to 25 mg/kg intake of TCE); three male rabbits and five male rats were injected intraperitoneally with 200 and 50 mg/kg TCE, respectively. [Ikeda and Ohtsuji (1972) have shown that the route of administration does not modify the metabolism of TCE in the rat]. Urinary excretion of trichloroethanol and trichloroacetic acid decreased exponentially in the three species studied; however, the rates of decrease differed. These rates were considerably lower in man, with large amounts still detectable 6 days after exposure. In rats, the rate of decrease was rapid, with metabolites barely detectable at 3 days. The ratio of trichloroethanol to trichloroacetic acid also indicated differences in TCE metabolism among the three species: 207, 12.5 and 1.9 for rabbit, rat, and man, respectively. Trichloroethanol was the major metabolite in rabbits, while the reverse was true in man. Trichloroacetic acid has also been detected in the urine of dogs (von Oettingen 1964).

Nomiyama and Nomiyama (1971) reported that the pattern of TCE metabolism differed between men and women; i.e., within the first 24 hours following exposure to 1345- 2044 mg/m^3 TCE in air, women tended to excrete more trichloroacetic acid and less trichloroethanol than males. A similar study with rats showed no significant differences between sexes in TCE metabolism (Nomiyama and Nomiyama 1979a). Man also appears to exhibit an age-dependent pattern of TCE excretion. Grandjean et al. (1955) reported that younger workers excreted more inhaled TCE as urinary trichloroacetic acid than in expired breath (6:1 ratio) in contrast with the ratio in older workers (2:1), suggesting an age dependence for urinary:alveolar excretion of TCE.

Little information is available on the accumulation of TCE in human tissues. McConnell et al. (1975) reported that post-mortem samples of human tissues from eight individuals with unknown exposures to TCE contained less than 1-32 ug TCE/kg wet tissue, with no significant pattern of distribution or accumulation evident.

5.1.3 Human and Animal Studies

5.1.3.1 Carcinogenicity

Considerable controversy exists concerning whether TCE is carcinogenic. The available evidence presented in this section is insufficient to indict pure TCE as a carcinogen, but does suggest that technical grade TCE may be a potential, but apparently weak, carcinogen. Test data from studies under way are needed to clarify this issue.

No epidemiologic evidence suggests that TCE exposure is associated with an increased risk of cancer in humans. Preliminary analysis in two cohort mortality studies in Sweden and Finland suggests no increased risk of cancer; however, data are presently insufficient to analyze site-specifically for cancer risk (Axelson et al. 1978, Tola 1977).

The National Cancer Institute (1976) reported that the oral administration of time-weighted-average doses of 2339 and 1169 mg technical grade TCE/kg bodyweight (bw) in male and 1739 and 869 mg TCE/kg bw in female B6C3F1 mice, 5 times per week for 78 weeks, induced a significant increase in hepatocellular carcinoma:

Males	Females
Control 1/20 (5%)	0/20 (0%)
Low Dose 26/50 (52%) $p = 0.004$	4/5 (8%) $p = 0.090$
High Dose 31/48 (65%) $p < 0.001$	11/47 (23%) $p = 0.008$

Hepatocellular carcinoma was detected as early as 27 weeks in males at the high dose level, but developed later in low-dose males and all females.

No carcinogenic effect was observed, however, in a concurrent study with Osborne-Mendel rats exposed five times per week by gavage to time-weighted average doses of 1097 and 549 mg technical grade TCE/kg body weight (bw). Increased mortality necessitated a reduction in dosing to a 4-week treatment, with a 1-week no treatment regimen. The mortality rate reduced the ability to detect TCE-induced carcinogenicity in Osborne-Mendel rats. This strain also showed a poor response (5% hepatocellular carcinoma) to the positive control, carbon tetrachloride (NCI 1976).

Negative carcinogenic effects were observed in Sprague-Dawley rats administered 24 or 240 mg highly purified TCE/kg by gavage, 4-5 days/week for 52 weeks, then held up to 140 weeks (Maltoni 1979). Maltoni (1980)

reported a negative carcinogenic response in mice (strain unspecified) fed 500 mg highly purified TCE/kg bw 5 times/week for 52 weeks. No other details were available. In another study with mice, Rudali (1967) observed no liver lesions or hepatomas in 28 NLC mice (age unspecified) given 40 mg TCE in oil/mouse by gavage twice weekly for an unspecified period. The positive control, carbon tetrachloride,produced a good response in this strain.

Van Duuren and coworkers (1979) intubated Ha:ICR Swiss mice with a highly purified sample of 0.5 mg TCE/mouse, once a week for the duration of a 622-day study. No significant increase in forestomach tumors was found; only lung, liver and stomach were examined histologically. Repeated dermal application of highly purified TCE (1 mg in acetone/ mouse, 3 times/week for 581 days) to the dorsal skin of Ha:ICR Swiss mice produced no significant incidence of skin tumors (Van Duuren et al. 1979). A similar experiment involving an initiation-promotion sequence, i.e., a single application of TCE followed by repeated applications of the tumor promoter, phorbol myristate acetate, also produced negative results (Van Duuren et al. 1979).

The Manufacturing Chemists Association sponsored Industrial Bio-Test Laboratories, Inc. (IBT) to do a 2-year inhalation study with Charles River rats and B6C3F1 mice. These animals were exposed to 538, 1614, or 3228 mg technical grade TCE/m^3, 6 hours/day, 5 days/week for 24 months. The TCE used in this study was identical to the TCE sample used in the NCI (1976) study. Preliminary results of mice killed at 24 months and rats dying by 21 months indicated an apparent induction of liver cancer in mice but no evidence of tumors in rats (Clark 1977 a,b,c). This study has been criticized because exact concentrations of TCE administered were not known (analytical data indicate extreme deviations from nominal concentrations) and because the control animals were obtained from a different population (Infante and Marlow 1980).

No carcinogenic effects were reported for Han:Wist rats, Syrian hamsters or male Han:NMRI mice exposed to 0, 538, or 2690 mg purified TCE vapor/m^3, 6 hours/day, 5 days/week for 18 months. Surviving mice and hamsters were killed at 30 months and rats were killed at 36 months. An elevated incidence of malignant lymphoma was noted in treated female mice (17/30 low dose, 18/28 high dose vs. 9/29 controls). An immunosuppressive effect of TCE and/or its metabolites may be responsible for the observed increase in lymphomas (Henschler et al. 1980). The significance of this finding is open to question in view of the high incidence of lymphoma in controls (31%). Another lifetime inhalation study initiated in February 1979, with 3500 animals (Sprague-Dawley rats and Swiss mice), is currently in progress (Maltoni 1980). Test animals are being exposed 3228-18830 mg highly purified TCE/m^3. No carcinogenic effects were evident at 1 year. A second carcinogenicity study for TCE by the oral route is also underway in B6C3F1 mice and five strains of rats (Osborne-Mendel, Fisher 344, MA540, A28807, and ACI); completion is expected by 12/81 (NCI 1980).

Thus, other than the elevated incidence of lymphoma in female Han:NMRI mice exposed to purified TCE vapor for 18 months (Henschler et al. 1980), which is of debatable significance in view of the 30% incidence of lymphoma in controls, the sole carcinogenic response in which the dose of TCE is clearly established, is hepatocellular carcinoma noted in B6C3F1 mice given technical grade TCE by gavage for 78 weeks (NCI 1976). Several questions on a number of points have caused concern about the validity of these results (Henschler et al. 1977, Van Duuren 1978). Analysis (GC/MS) of the industrial grade TCE used in the NCI study indicated that it contained two epoxide stabilizers (0.22% epichlorohydrin and 0.19% 1,2-epoxbutane), both of which are highly mutagenic in the Ames assay (Henschler et al. 1977, Weisburger 1977).

Henschler et al. (1977) believe that the carcinogenic effects noted in the NCI study were predominantly, if not exclusively, due to the epoxides. Limited feeding experiments with epichlorohydrin resulted in negative carcinogenic effects that were ascribed to epichlorohydrin's rapid rate of hydrolysis at acidic pH encountered in the stomach (Van Duuren et al. 1966). A co-carcinogenic effect of TCE and impurities, however, cannot be ruled out.

Another issue concerning the NCI study is metabolic overload. The toxic doses utilized in the NCI study may have partially chemically hepatectomized the mice, with tumorigenesis occurring secondary to rapid cellular proliferation and liver regeneration; or, the high dose levels may have saturated the usual metabolic pathways, resulting in atypical metabolites and/or routes of metabolism. Indeed, recent findings of Parchman and Magee (1980) suggest dose-related differences in metabolic pathways for TCE; and Ikeda (1977) found that the linear correlation between TCE exposure level and trichloroacetic acid in human urine deviated from linearity when TCE exceeded 268 mg/m^3.

In addition, species differences are known to exist in hepatic epoxide hydrase activity, the enzyme that inactivates epoxides. Hydrase activity in humans is four times that of mice, two times that of rats (Oesch et al. 1974). The in vitro binding of TCE to liver microsomal proteins was found to be 37% higher in male B6C3F1 mice than in female mice and 46% higher in male mice than in male Osborne-Mendel rats. The data correlate with the carcinogenicity results of the NCI study (Van Duuren 1977). Parchman and Magee (1980) also reported small amounts of radioactivity in DNA extracted from livers of B6C3F1 mice 6 hours after administration of ^{14}C-TCE possibly suggesting that a TCE metabolite interacts with DNA.

The Carcinogen Assessment Group (1980) has taken the position that:

- the administration of technical grade TCE by gavage for 78 weeks induced a statistically significant incidence of hepatocellular carcinoma in male and female B6C3F1 mice (NCI 1976): and

- findings that inhalation of technical TCE vapor for 24 months showed a statistically significant increase in hepatocellular carcinoma in male B6C3F1 mice (Clark 1977)

are sufficient evidence that technical TCE is a carcinogen. Furthermore, CAG believes that a significant increase in malignant lymphoma noted in female Han:NMRI mice exposed to purified TCE vapor for 18 months (Henschler et al. 1980) is substantial evidence to indicate that purified TCE is a likely human carcinogen and grounds for disregarding all negative data. The CAG has discounted the absence of a dose-response, the absence of tumors in exposed rats, the effective partial chemical hepatectomy produced in mice with the high doses of TCE used in the NCI study and the very high incidence of lymphoma in female control NMRI mice. Resolution of these points must await studies currently in progress with purified TCE in various strains of rats and mice, scheduled for completion by the end of 1981 (i.e., via gavage by the National Toxicology Program and via inhalation by Maltoni and coworkers).

Estimates of the human risk associated with TCE exposure have been calculated based on the NCI (1976) mouse data of a 50% incidence of tumors in male mice given 1170 mg TCE/kg/day. Using a multi-stage model and the observed hepatocellular carcinoma incidence in male mice in the NCI (1976) study, the CAG (1980) estimated the dose-response slope and risk to an average 70 kg human from TCE exposure. The slope of the dose-response curve was 1.26×10^{-2} $(mg/kg/day)^{-1}$, with the additional risk for a lifetime continuous exposure to 1 $\mu g/m^3$ TCE of 3.6×10^{-6}.

5.1.3.2. Mutagenicity

The mutagenicity studies conducted with TCE have produced contradictory results. Price et al. (1978) noted transformation of Fischer rat embryo cells (F1706) to tumor-producing cells following exposure to TCE. Subcutaneous injection of transformed cells into newborn Fischer rats produced fibrosarcoma at the site of inoculation in all test animals within 55 days.

Ismailov and Ryskal (1976) reported that the addition of 0.01%, 0.03%, or 0.05% of trichloroethylene to the nutrient medium of Drosophila melanogaster caused sex-linked recessive lethal mutations in the chromosomes of 3.2%, 7.7%, and 8.2% of the offspring, respectively.

Negative findings were recorded, however, in a dominant lethal study with 15 male rats exposed by inhalation to 1614 mg TCE/m^3, 6 hours/day,

5 days/week for 9 months. Males were subsequently mated with two untreated females/week for 8 weeks. Reproductive performances in TCE-exposed rats were reported to be comparable with those of control rats (Bell 1977).

In bacterial mutagenicity assays, weakly positive results were found with Salmonella typhimurium TA100 exposed for 7 hours to 8070 mg TCE/m^3; however, this occurred only when exposure was coupled with liver microsomal activation (Simmon et al. 1977).

Similar results were noted by Bartsch et al. (1979). Weakly positive results were also reported in Escherichia coli K12 exposed to 434 mg TCE/l but only when activated with a mouse liver microsomal fraction (Greim et al. 1975).

Positive mutagenic activity has also been noted in the presence of microsomal activation with the yeast Saccharomyces cerevisiae in both gene conversion (frameshift and base-pair mutations) and mitotic recombination assays (Shahin and von Borstel 1977, Bronzetti et al. 1978, Callen et al. 1980). Bronzetti and coworkers (1978) also noted a positive host-mediated assay in S. cerevisiae with mice given a single gavage dose of TCE (400 mg/kg) or multiple doses over a 5-day period (total dose: 3700 mg TCE/kg).

In summary, positive mutagenic results have been noted in bacterial and yeast test systems. This occurred only if microsomal activation was provided in a mammalian cell transformation assay. On the other hand, negative results were reported in a dominant lethal assay with rats exposed to 1614 mg TCE/m^3 for 9 months.

5.1.3.3 Teratogenicity

Although TCE is not considered teratogenic, it may delay skeletal maturation in rats. Schwetz et al. (1975) reported no teratogenic effects or significant maternal or fetal toxicity in either Swiss Webster mice or Sprague-Dawley rats exposed to 1614 mg TCE/m^3 by inhalation, 7 hours/day on days 6 through 15 gestation.

Similar findings were noted in Charles River rats exposed by inhalation to 1614 mg TCE/m^3, 6 hours/day on days 6 through 15 of gestation. In addition, offspring of male rats similarly exposed for 9 months prior to mating exhibited no genetic changes in germinal cells (Bell 1977). In another study, female Long-Evans hooded rats were exposed by inhalation to 9684 $\pm$ 1076 mg TCE/m^3 according to one of four treatment regimens:

- TCE 2 weeks before mating and during first 20 days of pregnancy;
- TCE before mating with filtered air during pregnancy;

- filtered air before mating, TCE during pregnancy; and
- filtered air before mating and during pregnancy.

The group exposed only during gestation showed a significant elevation in skeletal anomalies (incomplete ossification of the sternum), which is indicative of developmental delay in maturation rather than teratogenesis. This treatment group also demonstrated an increased incidence of displaced right ovary(18.6% vs. 6.3% in controls). Dams exposed before mating and during pregnancy and those exposed before mating alone produced offspring that experienced a reduction in post-natal body weight from 20-100 days after birth. Behavioral tests revealed no statistically significant differences between treated and control animals as measured by general activity levels at 10 and 20 days of age. No indications of maternal toxicity or fetotoxicity were seen in any treatment group (Dorfmueller et al. 1979).

5.1.3.4 Other Toxic Effects

Exposure to TCE has been associated with toxic effects on the central nervous, cardiovascular, hepatic, and renal systems. The predominant toxic manifestation of acute TCE exposure in man is depression of the central nervous system. Reported symptoms include visual disturbances, mental confusion, fatigue, tremors, dizziness, nausea and vomiting following exposure to a concentration of 800 mg TCE/m^3 for 2 hours (Browning 1965, Lloyd et al. 1975, MRI 1979). High acute doses have produced cardiac arrhythmias, with deaths typically caused by ventricular fibrillation and cardiac arrest (Tomasini 1976, Waters et al. 1976). Pelka and Zach (1974) reported that accidental ingestion of ~150 ml TCE resulted in acute renal failure, anuria, uremia, and hepatic and cardiovascular damage. The lowest reported oral lethal dose in man is 50 mg TCE/kg (RTECS 1977). By inhalation, the lowest reported lethal concentration was 16,140 mg/m^3 for 10 minutes (MRI 1979). CNS effects have been reported due to 860 mg TCE/m^3 for 83 minutes (RTECS 1977). Prolonged skin contact may cause local irritation and blister formation; paralysis of the fingers has been reported after repeated, intermittent immersion of the hands in TCE (Lloyd et al. 1975).

The effects of chronic exposure to TCE in humans have not been extensively studied; therefore, they are not well characterized. Intolerance to alcohol, however, is a well-documented symptom of repeated TCE exposure (Waters et al. 1976, NIOSH 1978). The mixed function oxidases responsible for the metabolism of ethanol also metabolize TCE. A competitive inhibition between TCE and ethanol for the enzyme results in the depression of TCE metabolism and a subsequent build up of TCE in blood. Muller et al. (1975) demonstrated that concurrent administration of ethanol and TCE (538 mg/m^3 for 6 hours) resulted in a 2 1/2-fold increase in the concentration of TCE in the blood of human volunteers above that observed in the absence of ethanol and a 3-4-fold increase in the amount of TCE in expired air.

In laboratory animals, the acute oral toxicity of TCE is low. Oral LD_{50} values (lethal dose to 50% of the population) of 4920 mg/kg in the rat (RTECS 1977), 3200 mg/kg in the mouse (Klaassen and Plaa 1966), and 2800 mg/kg in the dog (Klaassen and Plaa 1967) have been reported.

Adams et al. (1951) reported that the maximum concentrations of TCE producing no toxic effects after exposure for 7 hours/day, 5 days/week for 6 months were: rats and rabbits, 1076 mg/m^3; guinea pigs, 538 mg/m^3; monkeys, 2052 mg/m^3. In another study, rats exposed to 3800 mg TCE/m^3, 8 hours/day, five days/week for 6 weeks exhibited no significant toxicity or evidence of histopathological changes. Rats exposed continuously to 188 mg/m^3 for 90 days also exhibited no visible signs of toxicity (Prendergest et al. 1967).

Unlike other chlorinated hydrocarbons, evidence of hepatoxic effects of TCE is largely inconclusive. Early experiments with dogs exposed to either 4035 mg TCE/m^3 for 8 hours/day, 6 days/week for 3 weeks or 3066 mg/m^3 for 6 hours/day, 5 days/week for 8 weeks produced degeneration of parenchymatous liver cells, anemia, weight loss, lethargy and diarrhea (Seifter 1944).

Later studies using more purified TCE, however, indicate little or no hepatoxic effects following TCE exposure. Rats exposed to 116,200 mg/m^3 TCE for 15-90 hours showed increase intracellular lipid levels but no liver necrosis (Verne et al. 1959). Kylin et al. (1963) found no evidence of histological damage in the liver of rats following a single 4-hour exposure to 17,210 mg/m^3.

In man, transient increases in serum transaminases (which indicates damage to liver parenchyma) have been observed, however, these increases usually disappear after exposure is terminated (MRI 1979).

5.1.4 Overview and Summary

5.1.4.1 Ambient Water Quality Criterion - Human Health

The U.S. Environmental Protection Agency (1980a) has established a zero ambient water concentration for the maximum protection of human health from potential carcinogenic effects of exposure to trichloroethylene through ingestion of water and contaminated aquatic organisms. The water quality criterion is based on the induction of hepatocellular carcinoma in male B6C3F1 mice given a time-weighted average dose of 1169 mg/kg/day for 78 weeks. The concentration of trichloroethylene in water calculated (via a linear non-threshold model) to keep any additional lifetime cancer risk below 10^{-5} is 27-28 µg/l (USEPA 1980a, USEPA 1980c).

5.1.4.2 Trichloroethylene Relation to Human Risk

The potential carcinogenic effect of TCE is controversial. Technical TCE (1170 mg/kg by gavage) was carcinogenic in B6C3F1 mice, inducing a

52% incidence of hepatocellular carcinoma in male mice compared with a 5% incidence in male control mice. No tumors were seen in liver, lung, or stomach of Ha:ICR Swiss mice intubated with a considerably lower dose (0.5 mg purified TCE/mouse/week) for a lifetime nor in mice fed 500 mg purified TCE/kg 5 times per week for 52 weeks. Repeated dermal application of TCE also produced no significant incidence of skin tumors in Ha:ICR mice and initiation-promotion studies were also negative. No carcinogenic activity was found in Sprague-Dawley rats fed 240 mg purified TCE for 52 weeks, then held up to 140 weeks; nor in Osborne-Mendel rats given technical TCE by gavage; however, high mortality reduced the ability to detect a carcinogenic response in the latter study. No carcinogenic effects were observed in Han:Wist rats, Syrian hamsters, or male Han:NMRI mice exposed to 2690 mg purified TCE/kg by inhalation for 18 months, although female mice exhibited an elevated incidence of malignant lymphoma. The significance of this finding, however, is questionable in view of the 30% incidence of lymphoma in controls. The malignant lymphomas in the exposed population may be related to an immunosuppressive effect caused by TCE.

Carcinogenic effects in B6C3F1 mice have been questioned because of epoxide stabilizers present in the TCE test sample and the high dosage levels employed. Metabolic overload is another unresolved issue. Limited data on epoxide stabilizers suggest that they are inactivated at the acidic pH in the stomach; therefore, it is unlikely that epoxide stabilizers produced the clear increase in hepatic tumors seen in TCE-fed B6C3F1 mice. In addition, the structural similarity of TCE to other known carcinogens, the greater in vitro binding of TCE to tissue macromolecules and DNA in mice compared with rats, and the lower levels of epoxide hydrase in mice compared with rats and humans are factors that may have contributed to the NCI findings.
(The CAG estimated the slope of the dose-response curve to be 1.26×10^{-2} $(mg/kg/day)^{-1}$. This value is based on a multi-stage extrapolation model and the observed incidence of hepatocellular carcinoma in male mice in the NCI study.)

Consumption of 2 liters drinking water/day containing 27-28 µg TCE/l has been estimated by the CAG to result in one additional cancer per 100,000 people exposed. At this time, no epidemiologic evidence suggests that TCE exposure is associated with an increased risk of cancer in humans. Epidemiologic studies, however, have only recently been initiated.

Positive mutagenic results have been noted in a mammalian cell transformation assay and weak responses in bacterial and yeast test systems. However, this occurred only if microsomal activation was provided. Negative results, however, were found in a dominant lethal assay with rats exposed to 1614 mg TCE/m^3 for 9 months; and inhalation exposure of mice (1614 mg/m^3) and rats (9684 mg/m^3) during gestation resulted in no indication of fetotoxic or teratogenic effects.

TCE is readily absorbed by ingestion or inhalation and is oxidized to chloral hydrate via an epoxide intermediate. Chloral hydrate is then either oxidized to trichloroacetic acid or reduced to trichloroethanol and eliminated in urine. A major toxic effect of TCE, depression of the central nervous system, is believed to be associated with the formation of chloral hydrate. The effects are reversible when removed from the source. Cardiac arrythmias have been noted with acute exposures to TCE; however, little liver toxicity has been observed in humans. The lowest oral lethal dose reported for humans is 50 mg TCE/kg bw. Intolerance to alcohol is a major interaction of TCE-ethanol exposure and appears related to a competitive inhibition between TCE and ethanol for microsomal mixed function oxidases, resulting in depression of TCE metabolism and its subsequent build up in the blood.

5.1.5 Estimation of Human Dose-Response Relationships for Cancer

5.1.5.1 Introduction

The potential carcinogenic effects of TCE upon humans can be quantitatively estimated through extrapolation of *in vivo* laboratory results. The available data concerning mammalian effects are summarized above. We have selected for extrapolation purposes the data which demonstrated increased hepatocellular carcinomas in mice. These data are listed in Table 5-1. It must be noted that interpretation of these results for human risk assessment is subject to a number of important qualifications and assumptions:

- Though positive carcinogenic findings exist, there have also been negative findings in tests with several species. In view of possible species differences in susceptibility, pharmaco-kinetics, and repair mechanisms, the carcinogenicity of TCE to humans is far from certain.

- Assuming that the positive findings indeed provide a basis for extrapolation to humans, the estimation of equivalent human doses involves considerable uncertainty. Scaling factors may be based on a number of variables, including relative body weights, body surface areas, and lifespans.

- The large difference between the typically high experimental doses and the actual exposure levels introduce uncertainty into the extrapolation from animals to humans. Due to inadequate understanding of the mechanisms of carcinogenesis, there is no scientific basis for selecting among several alternate dose-response models, which yield different results.

TABLE 5-1 INCIDENCE OF HEPATOCELLULAR CARCINOMA IN MICE (NCI 1976)

SPECIES	TCE DOSAGE[a] (mg/kg)	HUMAN EQUIVALENT (mg/day)	RESPONSE	EXCESS RESPONSE OVER CONTROLS
Male Mice	2339	7017	31/48 (65%)	60%
	1169	3507	26/50 (52%)	47%
	0	0	1/20 (5%)	-
Female Mice	1739	5217	11/47 (23%)	23%
	869	2607	4/50 (8%)	8%
	0	0	0/20	-

[a] 5 times/week orally for 78 weeks

5.1.5.2 Calculation of Human Equivalent Doses

The first step in extrapolating the carcinogenic effects of TCE to humans was to calculate the equivalent human dose rate corresponding to the experimental treatment. We have followed the approach recommended by the EPA (Federal Register 1979), which normalizes the dose rate according to body surface area. This approach is conservative, in that it results in a lower equivalent human dose than would be obtained from simple multiplication of animal dose rate (mg/kg/day) by human body weight. Whether surface area or body weight is a more appropriate normalization factor is still open to debate. The former method yields a dose rate about 14 times lower for mice. Thus, the choice of method introduces an uncertainty of roughly an order of magnitude into the risk estimates.

The actual calculation of equivalent human dose was performed as follows, assuming an average human weight of 70 kg:

$$\text{Human dose (mg/day)} = 70\ \text{kg} \times \text{animal dose (mg/kg/day)} \left(\frac{\text{animal weight}}{\text{human weight}}\right)^{1/3} \left(\frac{5}{7}\right) \times \left(\frac{\text{duration of exposure}}{\text{animal lifespan}}\right)$$

The correction factor for body surface area is the cube root of the ratio of animal to human weight, as shown in the Federal Register (1979). A correction factor of 5/7 was also included since the animals were treated only on five days per week. As a result, we conclude that one mg/kg/day to a mouse is equivalent to about 3 mg/day human intake.

5.1.5.3 Estimation of Human Risk Relationships

In order to indicate a range of possible carcinogenic risk to humans, three dose/response extrapolation models were applied to the human equivalent dose/response data. Results are shown in Table 5-2. These models were the "one-hit" model with a linear "hazard rate," (this model is also used by CAG) the log-probit model, and the one-hit model with a quadratic hazard rate (also known as a "multi-stage model). All of these models are well described in the literature, and theoretical discussions may be found in Arthur D. Little (1980) and the Federal Register (1979).

The one-hit models assume that the probability P(x) of carcinogenic response to dose x is described by

$$P(x) = 1 - e^{-h(x)},$$

where h(x) is the hazard rate function.

TABLE 5-2 ESTIMATED LIFETIME CARCINOGENIC RISK PER CAPITA DUE TO TCE INGESTION(a)

	EXPOSURE LEVEL OR DOSE (mg/day)				
	0.01	0.1	1	10	100
Linear Model[b] CAG Model[b]	1.8×10^{-6}	1.8×10^{-5}	1.8×10^{-4}	1.8×10^{-3}	1.8×10^{-2}
Log-Probit Model[b]	$< 10^{-6}$	2.0×10^{-6}	1.6×10^{-4}	4.7×10^{-3}	5.5×10^{-2}
Multi-Stage Model	6.5×10^{-7}	6.5×10^{-6}	6.5×10^{-5}	6.5×10^{-4}	6.5×10^{-3}

(a) These values are most likely conservative, i.e., the risk is overstated.

(b) Male mouse data only.

The log-probit model assumes that human susceptibility varies log-normally with dose.

Due to differing assumptions between the two dose-response models, they usually give widely differing results when effects data are extrapolated from high laboratory doses to the low doses typical of environmental exposure.

The one-hit model with linear hazard rate function (often simply referred to as the "linear one-hit model" since its behavior at low doses is linear) is the model used by the Carcinogen Assessment Group (CAG). According to this model, the probability $P_t(x)$ of carcinogenic response in test subjects treated with dose x is

$$P_t(x) = 1 - e^{-(Bx + C)},$$

where B and C are constants determined from the data. First solving for C, with $x = 0$, and rewriting, it is found that

$$P_t(x) = 1 - [1 - P_t(0)]\, e^{-Bx},$$

where $P_t(0)$ is the probability of response at dose zero, i.e., the proportional response of the control group. Thus,

$$B = \frac{1}{x} \ln \left(\frac{1 - P_t(0)}{1 - P_t(x)} \right).$$

The probability $P_a(x)$ of carcinogenic response attributable to dose x is then

$$P_a(x) = \frac{P_t(x) - P_t(0)}{1 - P_t(0)} = 1 - e^{-Bx} \qquad \text{(see footnote)}$$

It was found that the male mouse data indicated a higher rate of excess cancer incidence, yielding a conservative estimate of $B = 1.8 \times 10^{-4}$, roughly. The inferred human per capita risk at low dose levels may then be found simply by multiplying the coefficient B by the dose in mg/day. (Note that dose can also be normalized by body weight and can be expressed in mg/kg/day.)

$$P_a(x) \simeq Bx$$

Note the distinction between $P_a(x)$, the probability of response attributable to the carcinogen, and $P_t(x)$, the probability of response attributable to both the carcinogen and background effects. The probability $P_a(x)$ is referred to as the "excess cancer incidence."

The estimated incidence of cancer in a given population may then be found by multiplying the probability of response times the size of the population.

For the log-probit extrapolation, we solved for the "probit" intercept A in the following equation:

$$P(x) = \Phi \quad (A + \log_{10} (x))$$

where Φ is the cumulative normal distribution function.

This equation makes the usual assumption that the log-probit dose-response curve has unit slope with respect to the log-dose. Again, the more conservative results for male mice were utilized to estimate A, though in this case the difference was less important. Using tables of the standard normal distribution we find that A is approximately equal to -3.6. This value may then be used to find the probability of a response at various dose levels from the above equation.

The multi-stage model, using a quadratic hazard rate function,

$$h(x) = ax^2 + bx + c,$$

was fit to both the male and female mouse data combined. To estimate the parameters a, b, and c we used a maximum likelihood method, aided by a computer program which performed a heuristic search for the best fit. It was found that the parameter b dominated for small values of the dose x, so that the dose-response function was essentially linear in the low-dose region. The value of b was found to be $6.5 \times 10^{-5} (\text{mg/day})^{-1}$.

Table 5-2 summarizes for a range of exposure levels the risk estimates obtained from these three models. The estimated lifetime per capita risk for the exposed population is shown for daily exposures ranging from 1 ug/day to 100 mg/day.

The gap between the estimates is large in the low-dose region; thus, there is a substantial range of uncertainty concerning the actual carcinogenic effects of TCE. However, present scientific methods do not permit a more accurate or definitive assessment of human risk.

5.1.5.4 Other Considerations

Several approaches were taken to the estimation of human health risks from exposures to trichloroethylene by the Carcinogen Assessment Group (CAG 1980). These estimates were mainly directed toward the carcinogenic response to air pollution. They first used the data of Henschler et al. (1980), who observed the following incidence of malignant lymphomas in female mice under inhalation exposure: 9/29 (control); 17/30 (100 ppm); 18/28 (500 ppm). Formation of hepatocellular

carcinomas was not observed. We did not use these data in our risk extrapolation due to the high control incidence, the dubious pathological interpretation, and the negative results obtained with male mice, as well as rats and hamsters. In any case, the CAG's estimated risk of lymphomas did not differ greatly from the estimates based on the NCI study (Table 5-1), which offers a more solid basis for extrapolation.

The Carcinogen Assessment Group analysis also dealt with the results of the NCI study, where hepatocellular carcinomas were observed in male mice due to exposure to trichloroethylene by gavage. Although the Carcinogen Assessment Group stated that it used a multistage model, it, in fact, used a one-hit model by setting the second-order coefficient equal to zero. The potency or dose-response slope for humans derived from the one-hit model applied to this set of experiments was 1.26×10^{-2} per mg/kg/day (CAG 1980). This is equivalent to a risk of about 1.8×10^{-4} per mg/day, which is exactly the dose-response slope that was derived above.

5.2 HUMAN EXPOSURE

5.2.1 Introduction

For estimating human exposure to TCE, certain populations were assumed to be exposed to representative concentrations in the environment, foodstuffs, and drinking water. These concentrations were selected from monitoring data and estimates determined in the fate section. Estimates of exposure durations and maximum intake from exposure were used as an upper bound for the risk estimates in Chapter 7. These exposures are not definitive, but rather indicate the range of potential exposures.

The TCE environmental fate analysis has shown that measurable levels of TCE may occur in all environmental media -- air, water, soil, and sediment. Monitoring data support this analysis, demonstrating a wide range of TCE levels in the natural environment and in foodstuffs. Therefore, all three exposure routes--inhalation, ingestion, and dermal contact -- were considered.

Certain data are required in order to identify exposed populations and estimate the duration of exposure. Such data include the sources and amounts of TCE released to each medium, the persistence and concentration of the chemical, and the human activities occurring in proximity to each source type. Residence (urban, rural, or remote), occupation, and diet are the predominant factors influencing TCE exposure. Some exposure has occurred in the past during certain medical operations; however, it is reported that TCE is no longer used in these procedures. Therefore this exposure route will not be discussed.

Occupational exposure is the primary concern since TCE is used almost exclusively (90% of annual production) in the work environment. Other federal agencies have investigated these risks (Page and Arthur 1978, NIOSH 1978); therefore, they are not reviewed in detail in this report. For comparative purposes, however, the exposed population directly handling TCE will be reviewed.

In addition to the monitoring data and fate analysis, the work of Anderson et al. (1980) dealing with atmospheric exposure routes will be discussed in this section.

5.2.2 Exposure Scenarios

5.2.2.1 Populations Exposed Through Inhalation

The three plants that manufacture trichloroethylene and the 57,000 industrial and commercial degreasers identified by Anderson et al. (1980) account for most of the TCE release to air.

Populations exposed to TCE by inhalation are distinguished according to their general proximity to release of TCE into three groups: urban, rural, and remote. These groups have been arbitrarily assigned to coincide with selected ranges and levels of ambient atmospheric concentrations shown in Table 5-3. Specific subgroups of the three general groups include populations who live and/or work near user sites or near manufacturing sites.

Populations working in the vicinity of a source may be exposed 40 hours/week, while residents in the area of a source may only be exposed during the time a plume from the source is in the atmosphere around them. In this case, emissions may be reduced or eliminated at the end of the working day, and night-time exposure would drop to the local background level. Weather conditions will also alter the dispersion of the emissions, so that exposure may vary in any given location, even if the emission rate is absolutely constant over time. The duration of exposure to concentrations in the area of a source has been assumed to be 40 hrs/wk, corresponding with the length of the work week. An analysis of site-specific data would be required to refine this duration estimate.

Anderson et al. (1980) performed an exposure analysis based on plume dispersion and population distribution by Standard Metropolitan Statistical Areas (SMSA's). These authors estimate that about 4000 people in the US would be exposed to TCE levels >0.1 $\mu g/m^3$. This exposure assessment is not based on the Anderson et al. analysis for the following reasons: atmospheric TCE concentrations in remote areas have been found to be approximately 0.03 $\mu g/m^3$ and in many other places, particularly urban areas, concentrations >0.1 $\mu g/m^3$ have been detected. The Anderson et al. (1980) analysis also indicates that at 93% or more of the 57,000 degreasing sites, populations outside the plants will be exposed to no more than three times the ambient background levels. Higher concentrations have been measured in cities in which a substantial portion of the population may be exposed. Monitoring surveys have recorded up to 215 μg TCE/m^3 near user sites (see Chapter 4).

5.2.2.2 Populations Exposed through Ingestion of Contaminated Drinking Water and Foodstuffs

Human exposure to TCE may result from ingestion of contaminated drinking water, either surface or well water. The monitoring data

TABLE 5-3 ESTIMATED NONOCCUPATIONAL EXPOSURE TO TRICHLOROETHYLENE VIA INHALATION

Location[a]	Maximum Observed Concentration ($\mu g/m^3$)	Weekday Duration of Exposure[b] (hrs/day)	Estimated Total Intake[c] (mg/day)	
Near Manufacturing Site				
Urban - Day (near manufacturer)	1440	8	14	14.6
- Night (Bayonne, NJ)	47	16	0.6	
Rural - Day (near manufacturer)	1440	8	14	14.04
- Night (Talledega Nat. Forest)	3	16	0.04	
Near Degreasing Sites				
Urban - Day (Aircraft Factory)	235	8	2.3	2.9
- Night (Bayonne, NJ)	47	16	0.6	
Rural - Day (Aircraft Factory)	235	8	2.3	2.34
- Night (Talledega Nat. Forest)	3	16	0.04	
Low Ambient - Rural (Talledega Nat. Forest) or Urban (East Coast)	3	24	0.06	
Remote Locations (Ambient Background)	0.03	24	0.0006	

[a] Concentration estimates are taken from Table 4-5.

[b] Weekend exposures will be 24 hr/day at night time levels. Hence, these values provide an upperbound estimate daily on exposure levels.

[c] Values are rounded. Based on respiration of 1.2 m^3/hr (awake), 0.4 m^3/hr (sleeping), about 20 m^3/day (ICRP 1975).

indicate that some drinking water supplies, primarily wells, have been contaminated, some to high levels. Because the populations exposed to these sources are likely to be isolated and distinct with regard to the whole population, it is difficult to estimate a representative TCE exposure or the sizes of the affected populations. Because of the high degree of variability in the actual levels of TCE in drinking water (U.S. EPA 1980b), an average level of 2 µg TCE/l has been used to estimate the average exposure from all drinking water sources. This level is considered conservative in that it overstates generally observed concentrations, if detected at all, in the majority of surface and groundwater supplies sampled (see Table 4-6). It may, however, underestimate exposure for isolated cases in which high concentrations of TCE were detected. An exposure concentration of 3,000 µg/l (SLCWS 1975) was selected as the maximum potential exposure from drinking water. Although higher concentrations have been found, it was assumed that consumption of such water would be deterred by the characteristic odor of TCE.

Foodstuffs may be contaminated with TCE and human exposure may result from ingestion. Processing (i.e. caffeine and flavor extraction from coffee and spices) has been implicated in food contamination in the past, although the use of TCE in these applications has been generally discontinued. McConnell et al. (1975) have reported TCE concentrations in foods in the U.K. Although the relevancy of these concentrations to foods in general is unknown, in the absence of other information, it is assumed that they represent potential exposure levels. Information on food quantities consumed was taken from ICRP (1975).

5.2.2.3 Populations Exposed through Dermal Contact

Dermal exposure may occur through contact with washwater and during sports activities in freshwater. The data on the rate of absorption by the skin (See Section 5) show that dermal absorption is approximately one-third that noted for inhalation exposure (Sato and Nakajima 1978). Exposure durations are most likely very short. The potential environmental exposures via this route are assumed to be low relative to other routes. Therefore, no estimates were made concerning this route.

5.2.3 Exposure Estimates

5.2.3.1 Air

Estimates of exposure durations for various situations and typical ambient concentrations for each exposure group are presented in Table 5-3.

The product of the TCE concentration and duration of exposure for each specific human activity and appropriate respiratory rates were used to estimate total daily exposures (See Table 5-3). Exposures were calculated using the average active adult breathing rate of 1.2 m^3/hour (16 hours), which falls to 0.4 m^3/hour during sleep (8 hours) (ICRP 1975). The numbers presented in Table 5-3 represent possible intakes of TCE.

Occupational exposure estimates are shown in Table 5-4. The assumptions presented above also apply here. Concentrations were taken from various sources and compiled by Page and Arthur (1978).

The highest non-occupational inhalation exposures are near TCE manufacturing sites. Only three plants in the United States manufacture TCE. The exposed population is estimated to number fewer than 100,000 people based on census data on the populations outside the central cities of Lake Charles and Baton Rouge, and an additional 7,000 for Freeport, a highly industrialized area. Some 57,000 degreasing facilities are scattered throughout the United States and fugitive emissions from these facilities may affect some of the 120.7 million people living in urban areas (U.S. Bureau of Census 1979).

The exposure calculations in Table 5-3 and 5-4 show that non-occupational intakes may range from <0.01 mg/day to ∿15 mg/day, while those for occupational exposures are between 0.05 g/day and 5.8 g/day. The estimated non-occupational exposures that may occur in proximity to a source were from three to several hundred thousand times less than occupational exposures, while exposures in remote areas are up to nine million times less.

5.2.3.2 Water

Water consumption of 2 liters/day containing TCE at an average concentration of 2 µg/l or a maximum potential concentration of 3,000 µg/l would result in intakes of 0.004 mg and 6 mg TCE per day, respectively (Table 5-5). Therefore, contaminated water may contribute to ingestion of a substantial amount of TCE relative to inhalation if the water is highly contaminated, i.e., >300 µg/l (an uncommon event)(see Table 5-5 and Chapter 4). Generally observed levels result in intakes <0.1 mg/day. The size of the population exposed via this route is unknown.

5.2.3.3 Food

Ingestion of food contaminated at the assumed levels results in an estimated TCE intake of much less than 1 mg/day, a level similar to that estimated for drinking water (Table 5-5). As mentioned previously, this estimate may not represent actual situations because concentrations vary considerably and it is unknown how widespread food contamination may be.

5.2.4 Summary

The results of the exposure estimates are summarized in Table 5-6. Inhalation is the dominant exposure route. Depending on location and exposure duration, ingestion may be relatively more significant to total exposure. For these reasons, a 0.0006 mg/day (ambient atmospheric background inhalation exposure) minimum to approximately 15 mg/day (10 mg/day inhalation plus 5 mg/day ingestion) maximum potential exposure range is estimated.

TABLE 5-4 ESTIMATED OCCUPATIONAL EXPOSURE TO TRICHLOROETHYLENE VIA INHALATION

Facility or Operation	Exposure Concentration[a] (mg/m^3)	Exposure Duration (hours/day)	Daily Exposure[b] (mg/day)
DEGREASING TANK OPERATION[c]			
minimum	5.4	8	52
maximum	600	8	5800
median	270	8	2600
TANK CLEANING			
minimum	1240	0.5[d]	740[e]
maximum	6010	0.5[d]	3600[e]
ELECTRICAL COMPANY			
minimum	20	8	200
maximum	215	8	2100
EXPOSURE AT TLV (538 mg/m^3)	540	8	5200

[a] Data from Page and Arthur (1978).

[b] Estimates rounded. Respiration of 1.2 m^3/hr, 100% retention assumed.

[c] Three surveys of degreasers indicated that air concentrations less than 270 mg/m^3 occurred at 58%, 48% and 60% of the tanks sampled, while 81%, 93%, and 86% were less than 540 mg/m^3.

[d] Arthur D. Little, Inc. estimates.

[e] Tank cleaning will not necessarily occur weekly, but only when the grease sludge buildup is excessive, which will be dependent upon the size of the overall operation.

TABLE 5-3 ESTIMATED TRICHLOROETHYLENE EXPOSURE BY INGESTION: AMOUNTS, CONCENTRATIONS, AND EXPOSURE BY SOURCE

Source	Amount (kg/day)	Concentration (µg/kg)	Total TCE Exposure (mg/day)
DRINKING WATER			
Maximum Level[a]	2	3000	6
Typical Level[b]	2	2	0.004
Water Quality Criterion	2	2.	0.004
FOODSTUFF[c]			
Milk	0.5	0.3	1.5×10^{-4}
Cheese	0.016	3	5×10^{-5}
Eggs	0.02	0.6	1.2×10^{-5}
Meats and Products	0.2	16	3.2×10^{-3}
Oils and Fats	0.05	8	4×10^{-4}
Potatoes	0.10	3	3×10^{-4}
Fruits	0.2	4.5	9×10^{-4}
Grains	0.2	7	1.4×10^{-3}
		TOTAL FOOD	6.4×10^{-3}

[a] Although higher levels have been observed, it is assumed that consumption of water containing more than 3,000 µg/l would be deterred by the characteristic odor of TCE.

[b] Because of the high degree of variability in the actual levels of TCE in drinking water, an average level of 2 µg TCE/l was used to estimate the typical exposure from all drinking water sources. This level is considered conservative in that it overstates generally observed concentrations, if detected at all, in the majority of surface and groundwater supplies (see Table 4-6).

[c] Data from ICRP (1975).

TABLE 5-6 SUMMARY OF ESTIMATED EXPOSURE TO TRICHLOROETHYLENE[a]

Route	Estimated Total Exposure (mg/day)
Inhalation: Non-Occupational	
Activities near manufacturing sites	14
Activities near degreasing sites[b]	2.6
Night-time background: urban[c]	1.0
Night-time background: rural[d]	0.07
Remote Areas	0.0006
Inhalation: Occupational	52-5800
Ingestion:	
Food	0.006
Water	0 - 6

NOTE: [a]All populations may be exposed but not all will be exposed continuously. These estimates are, therefore, conservative and would overstate the possible exposure for the population in general.

[b]Average of rural and urban exposures, Table 5-3.

[c]Nighttime ambient concentration around Bayonne, N. J., (47 $\mu g/m^3$) taken as a representative ambient urban concentration.

[d]Nighttime ambient concentration at Talladega National Forest (3 $\mu g/m^3$) taken as representative of rural concentrations.

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6.0 EFFECTS AND EXPOSURE--BIOTA

6.1 EFFECTS ON BIOTA

6.1.1 Introduction

This section presents a discussion of the TCE levels that cause mortality or disrupt physiologic functions and processes in aquatic organisms. The effects of TCE in various organisms have been determined by laboratory studies under both static and flowthrough experimental conditions. Often, bioassay data from these two methods are inconsistent for the same species. The static bioassay test for aquatic organisms measures one initial concentration of a chemical to determine toxicity without compensation for loss or lack of availability. In flowthrough bioassay tests, a fresh solution containing the test substance is continuously or periodically supplied to the organisms throughout the test period. A problem with the static bioassay is that the test concentrations may change rapidly as a result of volatilization or degradation of certain test compounds (MRI 1979). In both kinds of bioassays, the concentrations are often determined nominally (i.e. by diluting a measured amount of the substance) rather than by direct periodic measurement during the bioassay. Nominal determination of concentrations does not account for toxicant evaporation, absorption onto particles or walls of a test tank, or absorption by test organisms; thus, lethal and sublethal levels may be overestimated. TCE is a highly volatile compound, with a half-life in water of only a few hours to a few days depending on the water body. (See Chapter 4)

TCE has been shown under laboratory conditions to be lethal to fish and other aquatic organisms and to affect such functions as equilibrium, respiration, and reproduction (Pearson and McConnell 1975, MRI 1979). Sensitivity to TCE differs among species and various life stages of single species. It is therefore difficult to define precise toxicity values for this compound.

6.1.2 Freshwater Organisms

6.1.2.1 Acute Effects

Acute toxicity is defined as toxicant-induced mortality over a short period, generally within 96 hours of exposure. Although aquatic organisms in natural waterways are more likely to be exposed to lower concentrations, which may result in chronic or sublethal effects, industrial discharges and accidental spills can temporarily result in levels high enough to cause mortality.

The acute effects of TCE to freshwater biota have been studied for three species, thus, information is limited. Fathead minnows, bluegill, and the cladoceran Daphnia magna, an invertebrate, were the organisms tested. The doses lethal to one-half the test population (LC_{50}'s) for these organisms are presented in Table 6-1. Both static and flowthrough tests were conducted for the finfish species; of these species, the bluegill was the most sensitive. The LC_{50} for Daphnia is 85 mg/l, about

TABLE 6-1. ACUTE TOXICITY OF TRICHLOROETHYLENE FOR FRESHWATER FISH AND INVERTEBRATES

Organism	Bioassay Method[a]	Test Conc.[b]	Time (hrs)	LC_{50} (mg/l)	Reference
Freshwater Fish:					
Fathead minnow (Pimephales promelas)	FT	M	96	40.7	Alexander et al. (1978)
Fathead minnow (Pimephales promelas)	S	U	96	66.8	Alexander et al. (1978)
Bluegill (Lepomis macrochirus)	S	U	96	44.7	U.S. EPA (1978)
Freshwater Invertebrates:					
Cladoceran (Daphnia magna)	S	U	48	85.2	U.S. EPA (1978)

[a] S - static, FT - flow-through

[b] U - unmeasured, M - measured

Note: The lowest value from a flowthrough test with measured concentrations = 40.7 mg/l.

2.5 times higher than the LC_{50} for fish. Bioconcentration by bluegill was studied by the U.S. EPA (1978) using radiolabeled TCE. After 14 days, the bioconcentration factor was 17.

6.1.2.2 Chronic Effects

Sublethal or chronic effects of a substance, indicated by the EC_{50} value (concentration causing the effect in 50% of test organisms), are generally determined by observation of effects such as loss of equilibrium, melanization, narcosis, swollen or hemorrhaging gills, and changes in reproductive habits of capabilities (MRI 1979). Data on chronic toxicity are very limited; only one test has been conducted on TCE and freshwater organisms. That study with Daphnia magna provided incomplete results because no adverse effects were detected at the maximum test concentration of 10 mg/l. Since the 48-hour EC_{50} value for Daphnia is 85.2 mg/l and there was no observed adverse effect during the chronic test at 10 mg/l, the difference between acute and chronic effects levels for this species is less than 8.5 times the chronic test level (U.S. EPA 1980).

A comparison of the effective concentration values (levels producing loss of equilibrium) and lethal concentrations of TCE in flowthrough tests on fathead minnows (Pimephales promelas) is presented in Table 6-2. Concentrations producing sub-lethal effects in 50% of the fish (EC_{50}) were 23.0 mg/l (MRI 1979).

6.1.3 Marine Organisms

6.1.3.1 Acute Effects

Information on acute toxic effects of TCE is limited; however, data are available concerning finfish, shrimp, polychaetes, barnacle larvae, and phytoplankton. Limited toxicity data indicate that marine mammals take up and accumulate TCE in the low µg/l range, 2.5-7.8 µg/kg wet weight (MRI 1979).

Data concerning TCE toxicity for saltwater fish are presented in Table 6-3. Investigations concerning acute and sublethal toxicity report median lethal concentration (LC_{50}). Non-lethal effects have also been noted. Fish appear to be sensitive to low concentrations (mg/l range).

The LC_{50} (48 hr) value for the barnacle nauplii was 20 mg/l. Another organism included in the study by Pearson and McConnell (1975) study was the unicellular algae Phaeodactylum tricornutum. The EC_{50} for this species was 5 mg/l. Marine algae take up and accumulate TCE in the low mg/l range and have an accumulation factor of less than 100 (MRI 1979).

Acute toxicity studies were conducted on the polychaete Ophryotrocha labronica using a static system. An initial momentary exposure to 400 mg TCE/l killed all test organisms within 24 hours. Approximately 40% of the test population was dead within 24 hours in 250 mg/l, however, the mortality rate decreased after the 24-hour period and was roughly constant for the remainder of the experiment. In 300 mg/l 75% of the

TABLE 6-2. RESULTS OF FLOWTHROUGH STUDIES OF CHRONIC AND ACUTE TOXICITY OF TRICHLOROETHYLENE FOR THE FATHEAD MINNOW

Effective concentration (EC) value[a,b] (mg/l)			
Hr	EC_{10}	EC_{50}	EC_{90}
24	15.2	23.0	36.2
48	16.9	22.7	30.6
72	15.5	22.2	31.8
96	13.7	21.9	34.9

Lethal concentration (LC) value[c] (mg/l)			
Hr	LC_{10}	LC_{50}	LC_{90}
24	34.7	52.3	79.1
48	27.7	53.3	102.6
72	20.9	39.0	72.6
96	17.4	40.7	95.0

[a] The effect noted was loss of equilibrium.

[b] MRI (1979).

[c] Alexander et al. (1975).

TABLE 6-3. TOXICITY OF TRICHLOROETHYLENE FOR SALTWATER FISH

Organism	Test Condition	Toxic Effect	Concentration (mg/l)	Reference
Dab (limnada limnada)	Flowthrough	LC_{50} (96/hr)	16	McConnell et al. (1975)
Pinpearch (Lagodon, rhomebodius)	Static	LC_{50} (24/hr)	75-100	Garrett (1957)
Grass Shrimp	96 hr	erratic swimming, uncontrolled movement, loss of equilibrium	2.0	Borthwick 1977
Sheepshead minnow	96 hr			

polychaetes were dead in 3 days following a shock test, whereas all polychaetes survived, with somewhat reduced activity, if exposure to 300 mg/l was made gradually. In 600 mg/l, the entire test population died within 96 hours; and in 400 mg/l, 25% of the polychaetes were alive after 120 hours and survived through the eighth day of experimentation (Rosenberg et al. 1975). The decrease in the mortality rate over time could result from volatilization and consequent decreased concentration of TCE (Rosenberg et al. 1975).

Grass shrimp and the sheepshead minnow demonstrated erratic swimming, uncontrolled movement, and loss of equilibrium after several minutes of exposure to 2 mg/l and 20 mg/l of TCE, respectively. (Borthwick 1977)

6.1.3.2 Chronic Effects

When reproductive female polychaetes were exposed to 200 mg/l TCE, fewer eggs and egg masses than normal were found. Far lower TCE concentrations affected the reproductivity of Ophryotrocha than those concentrations causing acute toxic effects. Eggs hatching decreased in 150-200 mg TCE/l; however, the LC_{50} value was 400 mg/l (Rosenberg et al. 1975).

6.1.4 Other Studies

Biggs et al. (1979) conducted experiments to determine if TCE had any effect on phytoplankton. Mixed laboratory cultures of known sensitive species, the estuarine centric diatom Thalassiosira pseudonana and a resistant green alga Dunaliella tertiolecta, were exposed. TCE concentrations of 50 µg/l and 100 µg/l caused no detectable effects on algae growth or size of progeny in both species. The authors predicted that TCE concentrations in this range (50-100 µg/l) and lower are unlikely to reduce algae growth or alter species succession of phytoplankton in natural systems (Biggs et al. 1979).

Limited data suggest that excessively high concentrations of TCE have a potential for disrupting activated sludge systems. Studies conducted on sewage treatment facilities in Michigan indicated that TCE concentrations >500 mg/l had a significant potential for completely inhibiting bacterial activity and partially inhibiting the synthesis of enzymes in these systems. It was concluded that at concentrations up to 300 mg/l, little, if any, effect would occur (MRI 1979). (Section 4.3.4.1 further discusses the waste treatment process.)

6.1.5 Summary

According to the literature, 2 mg/l was the lowest TCE concentration reported to affect aquatic organisms in the laboratory. Intoxication was noted in marine grass shrimp. The marine flatfish Limnada limnada was the most sensitive finfish species tested, with an LC_{50} of 16 mg/l. Barnacle nauplii were also sensitive to TCE concentrations in the low mg/l range, with a LC_{50} of 20 mg/l. The most resistant organism tested was the polychaete Ophryotracha labronica, with an LC_{50} of 400 mg/l.

General concentration ranges can be established for certain effects observed in the laboratory. These ranges are not rigidly defined, however,

and may overlap as a result of differences among species, life stages, or environmental variables. These ranges include:

- <10.0 mg/l represents the lowest range at which toxic effects were observed in any aquatic organism; toxic to unicellular marine algae.

- 10-20 mg/l represents the toxic concentration to marine flatfish and barnacle nauplii.

- 20-100 mg/l represents the acutely toxic range to several species of freshwater fish (40-70 mg/l), to the saltwater fish pinpearch (75-100 mg/l), and to Daphnia (85.2 mg/l). Chronic effects on fathead minnow were observed at 22.0 mg/l.

- 100-500 mg/l represents the acute and sub-lethal (reproductivity changes) effects to adult polychaetes of the species Ophryotracha labronica.

- >500 mg/l represents the significant potential believed to exist for major effects on waste treatment through inhibition of bacterial activity.

6.2 BIOLOGICAL FATE AND BIOACCUMULATION

The bioaccumulation of any chemical can be affected by those parameters of the soil, water, or air that affect the biological action of that compound. The behavior of a chemical in water is typically affected by temperature, oxygen concentration, water hardness, the presence or absence of other cations, and pH.

Accumulation has been defined as the increase in the level of a material in the issues of a test organism. Biomagnification is used herein to refer to increases in concentration up a food chain. (Accumulation and bioaccumulation are considered to be synonymous terms.) Little data are available on the variables that influence the bioaccumulation of TCE specifically.

6.2.1 Aquatic Biota

Aquatic organisms absorb TCE from the water by direct diffusion across wetted membranes and by absorption into the digestive tract (U.S. EPA 1977b). Based on a partition coefficient (K_{ow}) of 195 in an octanol/water system (log p = 2.29), TCE is expected to bioaccumulate slightly in fatty tissue. The 195 K_{ow} indicates a propensity to transfer from water to a lipid phase and bioaccumulate. In many cases lipophilicity is offset by susceptibility to metabolic degradation. Metabolism generally converts lipophilic compounds to polar metabolites, which are excreted by the kidneys (Radding et al. 1977). The biological half-life of TCE in freshwater fish tissue is reported to be less than one day (U.S. EPA 1979).

Aquatic plants absorb TCE directly from water and via root uptake from sediments.

6.2.1.1 Fish

Pearson and McConnell (1975) reported that the TCE concentrations in fish flesh from the U.K. range from 0.8 µg/kg to 11 µg/kg,while the concentration in fish liver ranged from 2 µg/kg to 56 µg/kg on a wet weight basis. Sampling of aquatic vertebrates from regions in England having major organochlorine plants showed a maximum of approximately 100-fold bioconcentration between seawater (0.5 µg/l) and the tissues such as ray liver (56 µg/lg) of animals higher in the food chain. (Pearson and McConnell 1975, U.S. EPA 1980.)

Dickson and Riley (1976) recorded TCE concentrations in various organs of five species of marine fish. Their results are in the same range as those of Pearson and McConnell (1975) if adjustments are made for the wet weight factors in the latter study. Dickson and Riley (1976) did not determine a TCE bioconcentration order for the various organs of each fish species. The highest concentration factor between the seawater and fish TCE concentrations was 171,which occurred in the dogfish liver.

Accumulation in marine species from the relatively uncontaminated Erin Sea show enrichment factors of 2-25 times (dry weight) (Dickson and Riley 1975).

Limited data are available on biocencentration of TCE in freshwater species. After a 14-day exposure, a BCF of 17 was obtained for bluegill exposed to radiolabelled TCE (U.S. EPA 1978).

6.2.1.2 Birds and Mammals

TCE has been observed to accumulate in the eggs and internal organs of aquatic birds and mammals. Freshwater and saltwater birds accumulated from 2.4 µg/l to 33 µg/l (wet weight) in eggs. A maximum of approximately a 66-fold increase in concentration was detected between seawater (0.5 µg/l) and kittiwake eggs (33 µg/kg).

Based on the limited data, aquatic mammals are able to take up and accumulate TCE in the low µg/l range. The grey seal had a TCE level of 2.5-7.2 µg/l in blubber and 3-6.2 ug/l in liver. (Pearson and McConnell 1975)

6.2.1.3 Invertebrates

Pearson and McConnell (1975) reported TCE levels in invertebrates ranging from none determined in whelk and ragworm to 16 µg/l, wet weight basis, in shrimp. Dickson and Riley (1976), however, detected a TCE level in the whelk (Baccinum undatum) digestive gland of 2 mg/l on a dry weight basis. They also found TCE in the Modiolus modiolus organs varying from 33 ug/l in muscle, and 56 ug/l in the digestive tissue, to 250 ug/l in the mantle.

The Pearson and McConnell (1975) study reported that the average concentration of TCE in the waters was 0.5 ug/l. This shows TCE uptake by marine invertebrates to levels that are roughly 1-50 times greater than that of the surrounding water. The U.S. EPA (1977) reported that the bioconcentration factor for TCE in marine invertebrates is roughly 10 to 100 times the level in the seawater.

6.2.1.4 Plants

Only one report was found in the literature (Pearson and McConnell 1975) containing data on the uptake and accumulation of TCE in aquatic plants. Marine algae containing 16 to 23 ug/l TCE were found in waters, with an average TCE level of 0.3 ug/l and a maximum level of 3.6 ug/l. This indicates a bioconcentration factor of roughly 4 to 70 times (U.S. EPA 1977).

6.2.2 Terrestrial Biota

Trichloroethylene,which has been used as a liquid anesthetic, biotransforms to three products: chloral hydrate, then trichloroacetic acid and trichloroethanol. In humans, both chloral hydrate and trichloroethanol possess hypnotic properties. Oxidation of TCE to chloral hydrate occurs in the microsomal fraction of liver cells, requiring the presence of NADPH and oxygen. Animal experiments indicate that TCE is a type I substrate for cytochrome P-450 of the microsomal mixed-function oxidase system, and that a pretreatment with phenobarbital may enhance metabolism. Metabolism of chloral hydrate, catalyzed by soluble enzymes, consists of either oxidation to trichloroacetic acid, or reduction to trichloroethanol. Trichloroacetic acid is excreted unchanged in the urine. Trichloroethanol is first conjugated with glucmonic acid, then excreted in urine (Kelly and Brown 1974).

Generally, animals inhaling TCE rapidly absorb it and readily metabolize it to chloral hydrate, trichloroethanol, and trichloroacetic acid; and then excrete it in the urine (U.S. EPA 1977).

6.2.2.1 Vertebrates

Pearson and McConnell (1975) have provided the only information on TCE levels in non-laboratory species. They reported the accumulation of 2.6-7.8 ug/kg (wet weight) of TCE in a common shrew (Sorex araneus) collected in a marsh.

The experimentally determined log P value for TCE is 2.9 (high), which indicates that the compound may be bioaccumulated. Cohen and coworkers (1958, cited in Walter et al., 1976) reported detectable levels of TCE in the blood, brain, adrenals, fat, heart, kidney, liver, lung, muscle, pancreas, spinal cord, cerebral spinal fluid, spleen, and thyroid of animals exposed to the compound for periods up to 219 hours. The relative amounts of TCE in these tissues were not reported in the secondary source because the data were not adequate for determining the relationship of the concentration in the tissues. Insufficient information on the distribution of TCE in different tissues did not allow verification

of the bioaccumulation potential suggested by the partition coefficient (U.S. EPA 1977).

Daniel (1963) reported that 72-85% and 10-20% of the total orally administered dose to rats could be accounted for in expired air and urine, respectively, with less than 0.5% appearing in the feces. This indicates that at least 80% (and probable more) of ingested TCE is systemically absorbed.

A study of TCE distribution in guinea pigs showed the highest concentrations in adrenals and fat and the lowest concentrations in liver and muscle (Fabre et al. 1952).

The biological half-life of TCE and its metabolites has been examined in humans and experimental animals. In the rat (male, SPE-Wistar II), concentrations of TCE in expired air were undetectable 8 hours after inhalation of TCE at concentrations of up to 330 ppm (Kimmerle and Eben 1973). After administration by gavage of ^{30}Cl-labelled TCE to Wistar rats, 72-85% of the radioactivity (presumably primarily TCE) was recovered in the expired air with a half-life of 5 hours (Daniel 1963).

When Nomiyama and Nomiyama (1979) injected rats and rabbits with TCE intraperitoneally, rats metabolized the substance more quickly. The biological half-time for urinary excretion of the total trichloro-compounds was 0.36 days for rabbits and 0.22 days for rats. The ratio of total trichloro-compounds in urine to the dose of TCE decreased with increases in dose level, and remained unchanged at exposure levels over 665 mg/m^3 TCE. Although the reasons for these results are not clearly understood, they may be due to increased respiratory excretion (Nomiyama and Nomiyama 1979).

6.2.2.2 Plants

Alumot and Bielorai (1969) fumigated cereals, for 48 hours, with a mixture that contained 37% TCE. The amount of TCE absorbed is summarized below:

Amounts(mg/kg) of TCE Initially Sorbed by Cereals at Two Temperatures (°C)

	Wheat		Barley		Corn		Sorghum	
Temperature:	17	30	17	30	17	30	17	30
mg/kg TCE:	119	136	105	97	184	187	129	120

In another study, the mean concentrations of TCE observed, after a 2-day extraction of wheat and corn, were 33 mg/kg and 106 mg/kg, respectively (Panel 1974).

Levels of TCE in fruit and vegetables are shown in Table 4-4. The levels range from no TCE detected in potatoes from South Wales to 5 ug/kg

detected in apples. No information was available on the TCE levels in food of plant origin in the United States (McConnell et al. 1975).

6.2.3 Biomagnification in the Food Chain

Pearson and McConnell (1975) determined the level of TCE and other chlorinated hydrocarbons in the tissues of a wide range of organisms. Species were chosen to represent significant trophic levels in the marine environment. The maximum overall increase in concentration between seawater and the tissues of animals at the top of food chains (such as fish liver, sea bird eggs, and seal blubber) was on the order of 100-fold for TCE (from 0.5 µg/l in water to 50 µg/l in tissues).

The concentrations of TCE in the marine organisms analyzed were in the range of 1-2 orders of magnitude greater than the level of TCE in the water. However, Pearson and McConnell (1975) found no evidence of biomagnification to any significant extent up aquatic food chains. They also found that birds and animals representing the higher trophic levels did not have significantly higher levels of TCE than the fish on which they fed. The grey seal, representing a higher trophic level, had a TCE level of 2.5-7.2 µg/l in blubber and 3-6.2 µg/l level in liver; thus, it differs slightly from the lower trophic level invertebrate mussel, (4-11.9 µg/l) and hermit crab (5-15 µg/l).

Pearson and McConnell (1975) concluded that significant biomagnification of TCE does not occur in aquatic food chains. Insufficient data are available to draw conclusions concerning biomagnification in terrestrial food chains.

6.2.4 Summary

Accumulation of TCE occurs in both aquatic and terrestrial plants and animals. The reported residues of TCE detected in aquatic organisms can be summarized as follows:

Aquatic Biota	Accumulated Concentration Range of TCE (µg/kg)
marine fish	<0.1-56 (wet weight)
	0-479 (dry weight)
birds	2.4-33 (wet weight)
mammals (grey seal)	2.5-7.2 (wet weight)
marine invertebrates	0-16 (wet weight)
	2-250 (dry weight)
marine algae	16-23 (wet weight)
plankton	0.05-0.9 (wet weight)

Terrestrial organisms have been observed to accumulate the following levels of TCE:

Terrestrial Biota	Accumulated Concentration Range of TCE (ug/kg)
domestic animals (beef and pig)	12-22
shrew	2.6-7.8 (wet weight)
guinea pigs (laboratory)	5,000-35,000
cereals (laboratory)	33,000-194,000
fruit and vegetables	0-5

The above results show the greater levels that can accumulate under laboratory conditions.

Animals tend to accumulate more TCE in fatty tissues such as liver. TCE can be metabolized to biotransformation products that are then excreted into urine. The biological half-life for TCE in freshwater tissue is reported to be less than one day; for laboratory animals, it is 5-8 hours.

Although it appears that the range of accumulated TCE is higher in organisms farther up the food chain, data are insufficient to conclude that TCE is biomagnified through the food chain.

6.3 EXPOSURE OF BIOTA TO TRICHLOROETHYLENE

6.3.1 Introduction

Trichloroethylene (TCE) is an organo-chlorine compound that does not occur naturally in the environment; rather,it arises from anthropic sources (MRI 1979). Its detection in rivers, municipal water supplies, groundwater, the ocean, and aquatic organisms indicates that TCE is widely distributed in the aquatic environment (Pearson and McConnell 1975). TCE is a highly volatile compound; however, with a half-life in water of from a few hours to a few days, depending on the waterbody. TCE has been found in both the influent and effluent water from sewage treatment plants, resulting in part from chlorination of waste effluents by both industries and municipalities (Jolley 1975). (See Monitoring Section)

6.3.2 Monitoring Data

Limited field monitoring data were available for TCE and included small-scale and localized sampling programs in the United States and England, and ambient and effluent concentrations of TCE from U.S. EPA's STORET files.

STORET data (Chapter 4.2) reveal that TCE is found in ambient water samples in the range of 0.14 ug/l-300 ug/l. Effluent concentrations ranged from 0.01 ug/l to 1600 ug/l, though most of the 270 samples were less

than 10 ug/l. Other studies showed TCE concentrations in surface waters up to approximately 200 ug/l, with most levels around 10 ug/l or less. Concentrations as high as 5227 ug/l were found in the vicinities of manufacturing plants in Louisiana and Texas.

Concentrations of TCE found in seawater in Liverpool Bay, England averaged 0.3 ug/l. Municipal waters in England, from upland surface sources, were found to contain up to 6×10^{-9} (by mass) TCE. Rainwater, also in England, contained a 1.5×10^{-10} (by mass) TCE. Marine sediments in Liverpool Bay were sampled and found to contain TCE in concentrations of 9.9×10^{-9} (Pearson and McConnell 1975). Sediment samples taken in the vicinities of TCE manufacturing and user sites (Battelle Columbus Laboratories 1977) showed up to 300 ug/l (dry weight) TCE.

6.3.3 Ingestion

Laboratory and field studies have attempted to determine the extent, if any, to which aquatic organisms take up and accumulate TCE. Bioaccumulation is considered the increase in the levels of a substance in the tissues of a test organism, whereas biomagnification refers to the increase in concentration of a material up the food chain (MRI 1979).

The transfer of compounds from ambient water to the tissues of organisms occur through two major pathways. The first route is by direct diffusion across wetted membranes, particularly those involved with respiration. The second, an indirect pathway is absorption into the digestive tract, either from particles that have adsorbed the substance, or directly from the tissues of their food (Pearson and McConnell 1975). No laboratory studies on toxicity of TCE to aquatic organisms as a result of ingestion were found. Field and laboratory data on the uptake and accumulation of TCE by fish are limited. However, these data indicate that fish do take up TCE from an aqueous environment. Field sampling showed an average concentration of TCE in fish (high ppb-low ppm range) to be up to 100 times the concentration found in ambient water. No evidence was presented in this study supporting accumulation through food chains (MRI 1979). Laboratory experiments showed that bioaccumulation does occur, but that is is not accompanied by any detected ill effects (Pearson and McConnell 1975). The lowest concentration observed to be toxic to aquatic organisms was 16 mg/l LC_{50} for the marine flatfish Limnada limnada (McConnell et al. 1975).

6.3.4 Fish Kills

No data were available concerning any fish kills related to TCE in aquatic environments.

6.3.5 Summary

Because of the lack of extensive monitoring data for TCE concentrations in aquatic environments and the high volatility of TCE from water, it is difficult to propose definitive conclusions regarding exposure levels,

on a national, regional, or local level. Based on the data available however, it would appear that where these compounds are detected, they are almost always found in low concentrations (≤10 µg/l), generally lower than those levels observed toxic to aquatic biota as discussed previously. Those few reports of higher concentrations that have occurred near TCE plant sites were also lower than the concentrations found to be toxic to the most sensitive aquatic species tested. No data were found on the length of time these concentrations existed, but given the relatively short half-life of TCE in water, these concentrations probably did not persist. Overall, the concentrations to which aquatic biota are exposed are in the low ug/l range, but exposures at this level appear to be localized and not pervasive on a nationwide basis.

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7.0 RISK CONSIDERATIONS

7.1 HUMAN RISK

7.1.1 The Carcinogenicity of Trichloroethylene

Considerable controversy exists concerning whether trichloroethylene (TCE) is carcinogenic (see Chapter 5.0). Laboratory studies indicate that technical TCE (1170 mg/kg body weight by gavage) is carcinogenic in B6C3F1 mice, inducing an elevated incidence of hepatocellular carcinoma. However, no tumors were seen in Ha:ICR Swiss mice intubated with a considerably lower dose (0.5 mg purified TCE/mouse/week) for a lifetime nor in mice fed 500 mg purified TCE/kg 5 times per week for 52 weeks. Repeated dermal application of TCE also produced no significant incidence of skin tumors in Ha:ICR mice and initiation-promotion studies were also negative. No carcinogenic activity was found in Sprague-Dawley rats fed 240 mg purified TCE for 52 weeks, then held up to 140 weeks; nor in Osborne-Mendel rats given technical TCE by gavage; however, high mortality reduced the ability to detect a carcinogenic response in the latter study. No carcinogenic effects were observed in Han:Wist rats, Syrian hamsters, or male Han:NMRI mice exposed to 2690 mg purified TCE/kg by inhalation for 18 months, although female mice exhibited an elevated incidence of malignant lymphoma. The significance of this finding, however, is questionable in view of the 30% incidence of lymphoma in controls. The malignant lymphomas in the exposed population may be related to an immunosuppressive effect caused by TCE.

The carcinogenic effects noted in B6C3F1 mice have thus been questioned. Among several issues raised are:

- The presence of carcinogenic epoxide stabilizers in the technical TCE sample administered to the B6C3F1 mice;
- The lower levels of epoxide hydrase in mice compared with rats and humans;
- The high dosage levels employed which may have saturated the usual metabolic pathways, resulting in atypical metabolites and/or routes of metabolism. Perhaps these high levels partially chemically hepatectomized the mice, with tumorigenesis occurring secondary to rapid cellular proliferation and liver regeneration.

There is no epidemiologic evidence to suggest that TCE exposure is associated with an increased risk of cancer in humans but cohort mortality studies have only recently been initiated. Thus, the available evidence presented in this report (see Chapter 5.0 for additional details) is insufficient to indict pure TCE as a carcinogen, but does suggest that technical grade TCE may be a potential, but apparently weak, carcinogen. Test studies currently underway will hopefully clarify this issue. However, until such time as this issue is resolved, a prudent course of action is to regard TCE as a suspected human carcinogen.

The potential carcinogenic effects of TCE on humans were quantitatively estimated through extrapolation of in vivo laboratory results (hepatocellular carcinoma in B6C3F1 mice), using three extrapolation models. A discussion of these models, as well as a number of important qualifications and assumptions utilized in the estimation of equivalent human doses and the extrapolation process, can be found in Section 5.1.5 of this report.

Exposure levels and doses to individuals have been estimated for many different exposure conditions. These conditions consider inhalation of TCE in air and ingestion of TCE in food and water. Risks associated with body intakes for these different conditions are summarized in Table 7-1. Non-occupational intakes due to inhalation range from 6×10^{-4} mg/day to 15 mg/day, with corresponding excess individual lifetime tumor incidence probabilities (estimated) of 4×10^{-8} to 3×10^{-3}. Estimated excess risks due to continuous lifetime consumption of water contaminated at average concentration levels are in the $<10^{-7}$ to 10^{-6} range. At the highest concentrations observed in drinking water, the estimated risk of excess individual lifetime cancer is on the order of 10^{-3}. These risk levels are based on the assumption that these exposures occur continuously over an individual's lifetime. Continuous lifetime exposures are most unlikely to occur over a large fraction of the population, except for background inhalation levels.

Occupational exposures might be much higher than 15 mg/day, with a corresponding increase in excess lifetime risk to populations exposed in this way.

No data were available nor were any estimates possible concerning the size of the population exposed to the chemical, except for the background inhalation value (0.6 µg/day). The total population potentially exposed to this level is 220×10^6. The background inhalation value (0.6 µg/day) combined with the risk models yields a range of 8.8 to 22 potential excess lifetime cancers for the 220 million people exposed, or less than one excess cancer/year, assuming a 70-year lifespan.

Table 7-1 ESTIMATED LEVELS OF HUMAN EXPOSURE AND EXCESS INDIVIDUAL LIFETIME PROBABILITY OF TUMOR INCIDENCE DUE TO EXPOSURE TO TRICHLOROETHYLENE

EXPOSURE SITUATION	LIFETIME EXPOSURE LEVEL OR DOSE[a] (mg/day)	LIFETIME EXPOSURE LEVEL OR DOSE[a] (mg/kg/day)	RISK ESTIMATION METHOD		
			LINEAR MODEL[b] CAG MODEL.[b]	LOG-PROBIT MODEL.[c]	MULTI-STAGE MODEL.[d]
Background Air Concentration - Inhalation	0.0006	8.6×10^{-6}	1×10^{-7}	$< 10^{-7}$	4×10^{-8}
Drinking Water - Average Observed Levels - Ingestion	0.004	5.7×10^{-5}	7×10^{-7}	$< 10^{-7}$	3×10^{-7}
Low Ambient Air Concentration (Urban or Rural)	0.06	8.5×10^{-4}	1×10^{-5}	8×10^{-7}	4×10^{-6}
High Ambient Air Concentration (Near Users) Inhalation	2.9	4.1×10^{-2}	5×10^{-4}	8×10^{-4}	2×10^{-4}
Drinking Water Maximum Concentration - Ingestion	6	8.6×10^{-2}	1×10^{-3}	3×10^{-3}	4×10^{-4}
Air Near Manufacturing Site - Inhalation	14.6	0.2	3×10^{-3}	7×10^{-3}	1×10^{-3}

Notes: (a) Dose = exposure (mg/day)/body weight (70 kg)

(b) Excess Lifetime Risk = $(1.26 \times 10^{-2})(\text{mg/kg/day})^{-1}$ * Dose (mg/kg/day) = 1.8×10^{-4} (mg/day) * Dose (mg/day)

(c) Excess Lifetime Risk = $\Phi\ [-3.6 + \log_{10}(\text{Dose, mg/day})]$

(d) Excess lifetime Risk = 6.5×10^{-5} $(\text{mg/day})^{-1}$ * Dose (mg/day)

7.1.2 Other Human Risks Associated with TCE Exposure

Other than carcinogenic risk, the risks associated with chronic exposure to TCE cannot be quantified. The effects of chronic exposure to TCE in humans have not been extensively studied; therefore, they are not well characterized. Intolerance to alcohol, however, is a well-documented symptom of repeated TCE exposure and appears to be related to a competitive inhibition between TCE and ethanol for microsomal mixed function oxidases, resulting in depression of TCE metabolism and its subsequent build up in the blood.

Tests with laboratory animals have established no-observed-effect levels of 540 to 2050 mg/m^3 over a 6-month exposure period. These levels are orders of magnitude above estimated human exposure levels. No indications of fetotoxic or teratogenic effects of TCE have been reported either.

Acute human exposure to TCE is of concern only at high exposure concentrations. Inhalation of 800 mg^3/m^3 TCE for 2 hours can depress the central nervous system. Reported symptoms include visual disturbances, mental confusion, fatigue, tremors, dizziness, nausea and vomiting. These effects are reversible when the exposed individual is removed from the source. High acute doses have produced cardiac arrhythmias, with deaths typically caused by ventricular fibrillation and cardiac arrest. Ingestion of 150 ml TCE can result in acute renal failure and cardiovascular damage; however, little liver toxicity has been observed in humans. The lowest oral lethal dose reported for humans is 50 mg TCE/kg bw. Prolonged skin contact may cause local irritation and blister formation; paralysis of the fingers has been reported after repeated, intermittent immersion of the hands in TCE.

7.2 RISK TO AQUATIC ORGANISMS

Ambient environmental levels of TCE appear to pose little risk to aquatic organisms. Water concentrations range from none detected to a high of 5300 µg/l in the vicinity of a TCE manufacturing facility. STORET retrievals indicate that most ambient concentrations seem to be in the 10 µg/l or less range. These concentrations, except for the high levels detected in the vicinity of the manufacturing plant, are generally lower than concentrations at which adverse effects have been noted.

The lowest LC_{50} to fish was found to be 16 mg/l and sublethal effects were found to occur at 2 mg/l. The lowest concentration for adverse effects in algae was 8 mg/l. These concentrations are approximately two to three factors of ten greater than the generally observed environmental concentrations. In the vicinity of manufacturing plants, however, maximum water concentrations are comparable to levels at which adverse effects have been noted. It is in these locations that TCE may pose a threat to aquatic organisms if discharges occur at levels that may cause acute or chronic effect levels to be reached.

APPENDICES A-G

These appendices contain supporting information for Chapter 3. The references for these appendices appear at the end of Chapter 3.

APPENDIX A

The reaction for direct chlorination of 1,2-dichloroethane to tri- and tetrachloroethylene is:

$$2\ ClCH_2CH_2Cl + 5Cl_2 \rightarrow Cl_2C{=}CHCl + Cl_2C{=}CCl_2 + 7HCl$$

The chlorination is carried out at temperatures between 400°C to 450°C, at approximately atmospheric pressure, and without the use of a catalyst. Other chlorinated C_2 hydrocarbons or recycled chlorinated hydrocarbon by-products may be used as feedstocks. By-product hydrogen chloride is typically used in other processes.

Figure A-1 represents a simplified process for manufacture of tri- and tetrachloroethylene via direct chlorination of 1,2-dichloroethane. 1,2-Dichloroethane and chlorine are first vaporized and fed to the reactor. Hydrogen chloride is separated from the reaction mixture and recovered as a by-product. The chlorinated hydrocarbon mixture is neutralized with sodium hydroxide solution.

The crude product is dried and separated by distillation into two crude streams. Crude trichloroethylene is distilled and the light ends taken overhead. The bottom stream which contains trichloroethylene and heavy chlorinated hydrocarbons is distilled in the finishing column. Trichloroethylene is taken overhead and sent to storage; the heavy by-products are combined with the light ends from the trichloroethylene column and recycled. The crude tetrachloroethylene is separated in the tetrachloroethylene column; purified tetrachloroethylene goes overhead to storage and the bottoms go to the heavy ends column. The heavy by-products are fractionated and recycled. The bottom product (largely tars) is assumed to be incinerated.

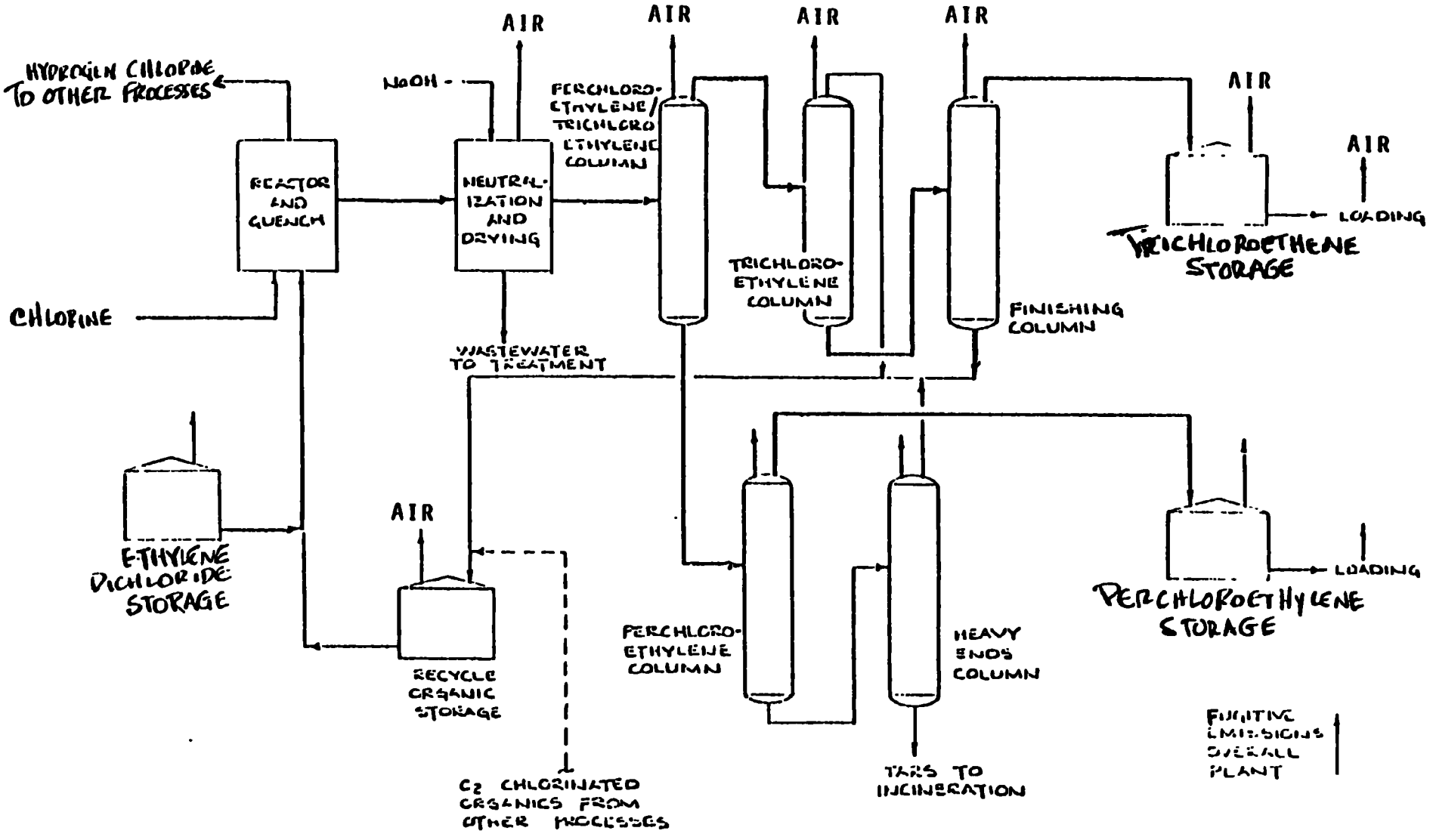

Figure A-1. Flow Diagram for Tetrachloroetylene by Chlorination (EPA, 1979d)

APPENDIX B

OXY-CHLORINATION

Tri- and tetrachloroethene may also be produced by oxy-chlorination of 1,2-dichloroethane:

$$5ClCH_2CH_2Cl + 3Cl_2 + 4O_2 \rightarrow Cl_2C{=}CCl_2 + 4Cl_2C{=}CHCl + 8H_2O$$

The reaction is carried out at an approximate temperature and pressure of 425°C and one atmosphere, respectively. Other chlorinated hydrocarbons may be used as feedstocks; indeed, most organic by-products may be recycled to the process. The process is relatively flexible and production of either tri- or tetrachloroethylene may be increased at the expense of the other.

Figure B-1 represents a simplified process for tri- and tetrachloroethylene manufacture via oxy-chlorination of 1,2-dichloroethane. Hydrogen chloride (or chlorine), oxygen, and 1,2-dichloroethane are vaporized and fed to a fluidized bed reactor. The crude product is cooled, separated from by-product water and noncondensed phases (e.g., carbon dioxide, hydrogen chloride nitrogen, and small amounts of uncondensed chlorinated hydrocarbons) and are scrubbed with process water to make by-product hydrochloric acid. The remaining inert gases are purged.

The crude product is dried by azeotropic distillation and separated into two product streams in the tetrachloroethene/ trichloroethylene column. Crude trichloroethylene is further fractionated and low boiling impurities (light ends) are recycled to the reactor. Trichloroethylene is neutralized with ammonia, dried, and sent to storage.

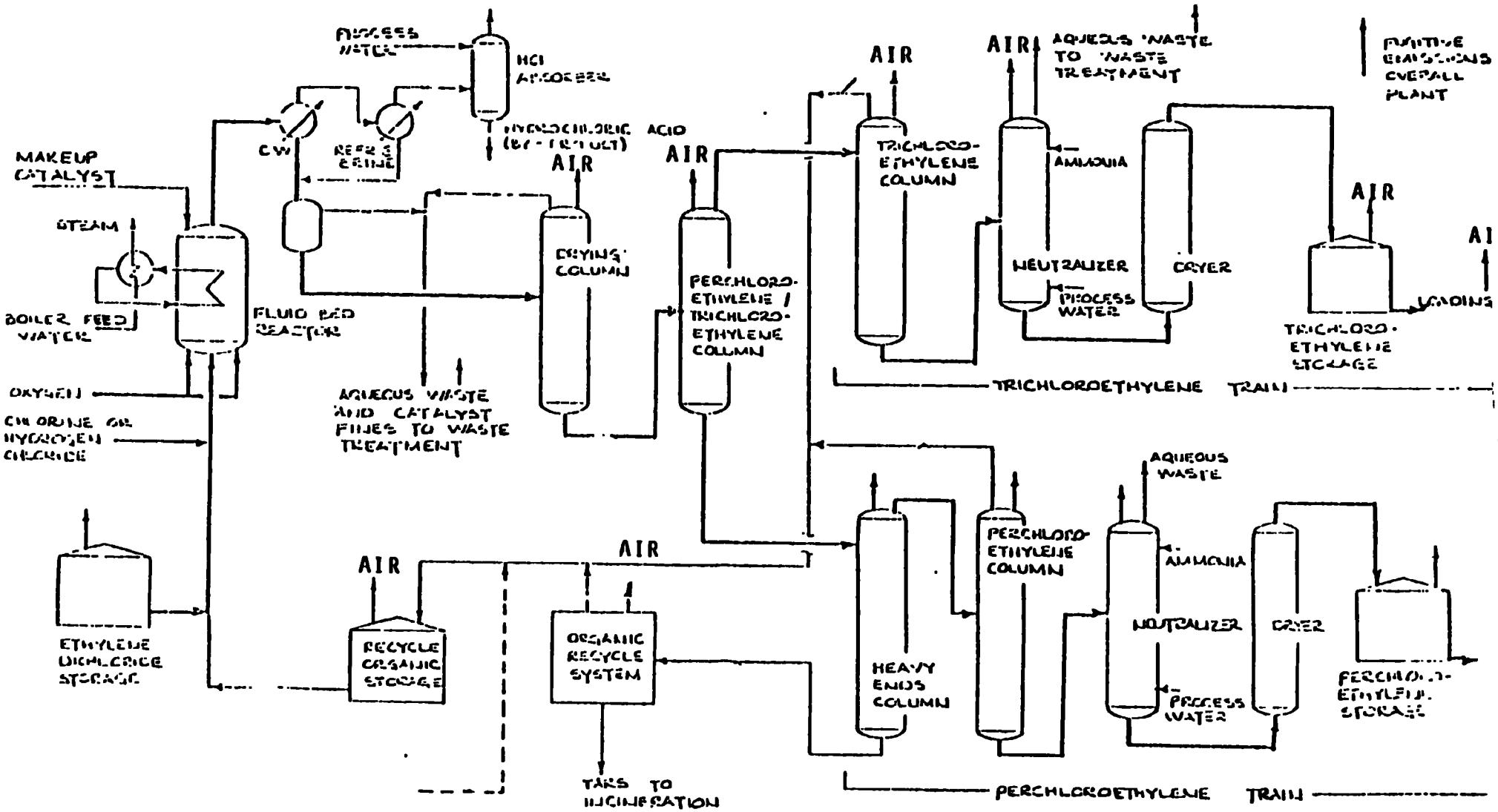

Figure B-1. Flow Diagram for Tetrachloroethylene and Trichloroethylene by Oxy-chlorination (EPA, 1979d)

Table C-1. TRICHLOROETHYLENE DISTRIBUTION IN POTWs, SLUDGE: Selected Urban Sites

PLANT	AVERAGE FLOW (10^6 l/day)	TRICHLOROETHYLENE CONCENTRATON (µg/l)		
		INFLUENT [a]	EFFLUENT [b]	COMBINED SLUDGE [c,d]
1	400	28	4	284
2	30	2	ND [e]	<5
3	38	2	1	ND
4	340	497	37	467
5	95	49	14	163
6	23	487	64	30
7	190	17	4	2

Source: EPA, 1980b

a) median value of influent is 28 µg/

b) median value of effluent is 4 µg/l

c) 1° and 2° sludge

d) median value of combined sludge is 30 µg/l.

e) ND = none detected

Table D-1. Incidents Involving Trichloroethylene (January 1971 through March 1980)

Carrier / Shipper	Mode / Date	Incident location / Shipment Origin		Container Types / —	Capacity(l) / Injury/Dead	Failures / Damages	Amt. Released(l) / Result
Johnson Motor Lines Inc. U.S. Government - DOD	Hwy 6/20/77	Atlanta Bellbluff	GA VA	Metal drum[a]	20 0 0	Bottom fail. $20	1 spill
Pacific Intermountain Express U.S. Government - DOD	Hwy 11/23/76	Peterson Fld. Lyoth	CO CA		--- 0 0	--- $0	20 spill
Emery Air Freight Corporation Burke Rubber Company	Air 11/1/78	San Francisco San Jose	CA CA	Metal can	4 0 0	Loose fittings $0	4 spill
Uintah Freightways U.S. Government - GSA	Hwy 2/1/79	Salt Lake Cy. Denver	UT CO	Metal drum[b]	210 0 0	External punct. $30	10 spill
Associated Truck Lines Inc.	Hwy	Louisville	KY	Metal drum[b]	210	Improper bracing/ damage by other freight	80
Mallinckrodt Chemical Works	3/9/79	St. Louis	MO		0 0	$100	spill
Wycoff Company Inc UWR Scientific Company	Hwy 3/14/79	Salt Lake Cy. Denver	UT CO	Glass bottle & fiberboard box or carton	4 0 0	Body/side fail. $0	4 spill
Cango Corporation Dow Chemical Company	Hwy 3/10/79	Iowa Freeport	IA TX	Tank truck	0 0 0	Vehicular accid. $0	4 spill
Ashland Chemical Company Ashland Chemical Company	Hwy 5/3/79	Columbus Columbus	OH OH	Cargo tank	15140 0 0	Hose burst $386	555 spill
Associated Truck Lines Inc. Bulk Terminals Company	Hwy 10/27/79	Alsip Chicago	IL IL	Metal drum[b]	210 0 0	External punct. $15	4 spill

Source: Department of Transportation, 1980

a) Removable head authorized
b) Removable head not authorized

Table E-1. National Organic Monitoring Survey, March 1976 through January 1977: Trichloroethylene

Sample	Phase[a]	Number of Positive Analyses per Number of Analyses I	II	III	Mean Concentration (µg/l) Positive Results Only I	II	III	Median Concentration (µg/l) All Results I	II	III
Quenched[b]		4/112[c]		10/106	11[c]		2.4	<1-5[d]		<0.2-0.3[d]
Terminal[e]			28/113	19/105		2.1	1.3		<0.03[d]	<0.2-0.3[d]

a) Dates of monitoring. Phase I: March - April, 1976; Phase II: May - July, 1976; Phase III: November, 1976 - January, 1977.

b) Samples preserved with Sodium Thiosulfate at time of sampling, shipped at ambient temperature, stored at 20°-25°C for 3-6 weeks prior to analyses.

c) Samples shipped iced (4°C), stored at 2°-8°C for 1-2 weeks prior to analyses.

d) Minimum quantifiable limits.

e) Samples shipped at ambient air temperature, stores ato 20°-25°C for 3-6 weeks prior to analyses.

Source: EPA, 1977c.

1. Recycle = (x)Waste Solvent, where x = 45%

2. Total Use = Virgin Solvent + Recycle
   = Virgin Solvent + (x) Waste Solvent

3. Waste Solvent = (y)Total Use = (y)(Virgin + (x)Waste)

y, the fraction wasted, is derived from Table F-2:
38,000 out of 117,000 kkg virgin solvent is wasted:
$y = 38{,}000/117{,}000 = 0.325$

Solving for Waste Solvent in Equation 3:
Waste = (y)Virgin Production + (x)(y)Waste
Waste (1-(x)(y)) = (y)Virgin

$$\text{Waste} = \frac{y}{1-(x)(y)} \text{ Virgin} = 0.38 \text{ Virgin}$$

From Equation 1:
Recycle = (x)waste
= (x)(0.38)Virgin = 0.171 Virgin

Figure F-1. Calculations: Total Solvent Usage in Degreasing Operations

Table F-1. Industrial Classes Utilizing Degreasing

Source Type	SIC
Industrial degreasing	
Metal furniture	25
Primary metals	33
Fabricated products	34
Nonelectric machinery	35
Electric equipment	36
Transportation equipment	37
Instruments and clocks	38
Miscellaneous	39
Automotive	[a]
Auto repair shops and garages	75
Automotive dealers	55
Gasoline stations	55
Maintenance shops	[a]
Textile plants (fabric scouring)	22

Source: EPA, 1979b.

[a]No applicable SIC for this category

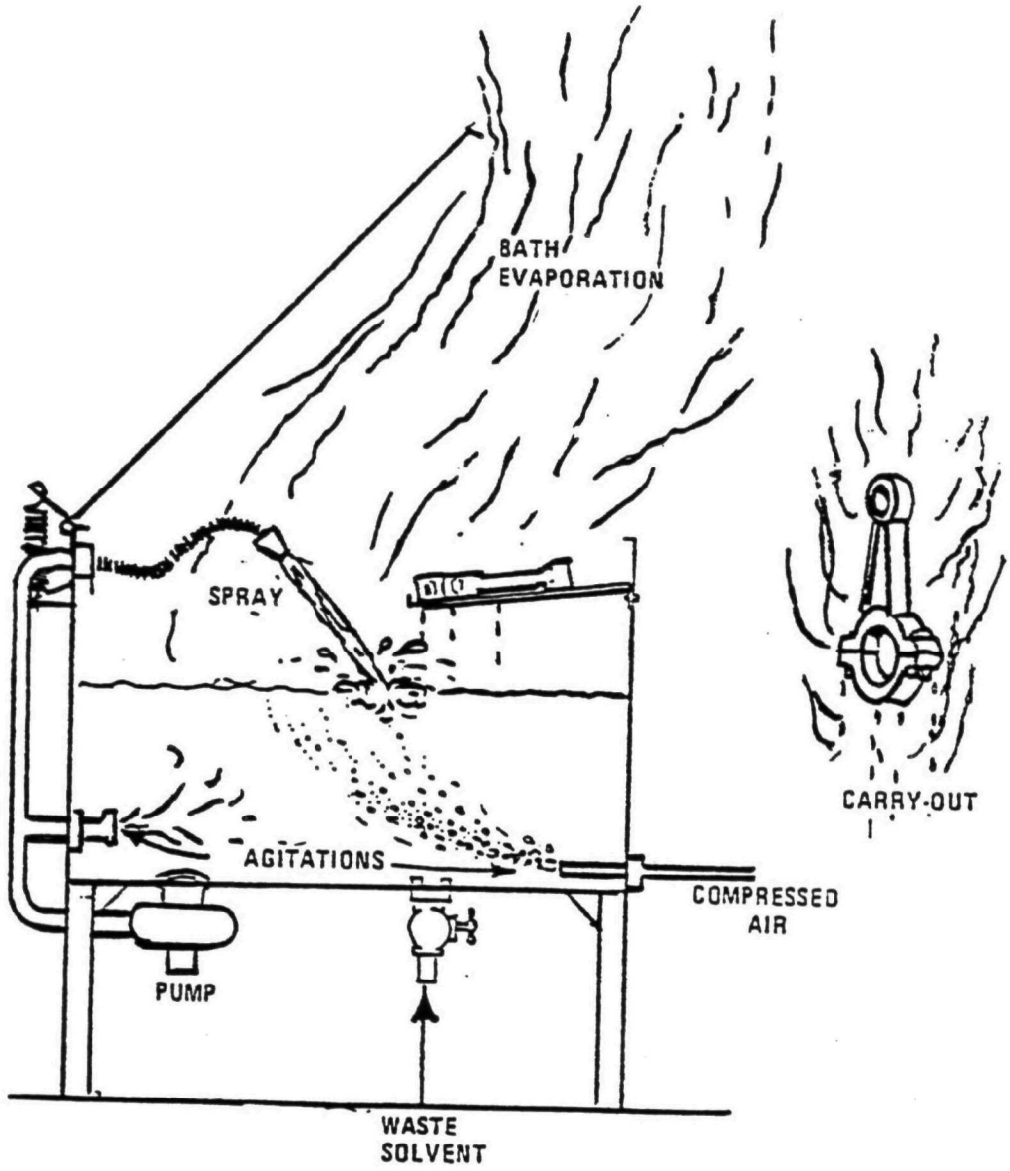

Figure F-2. Cold Cleaner (EPA, 1977a)

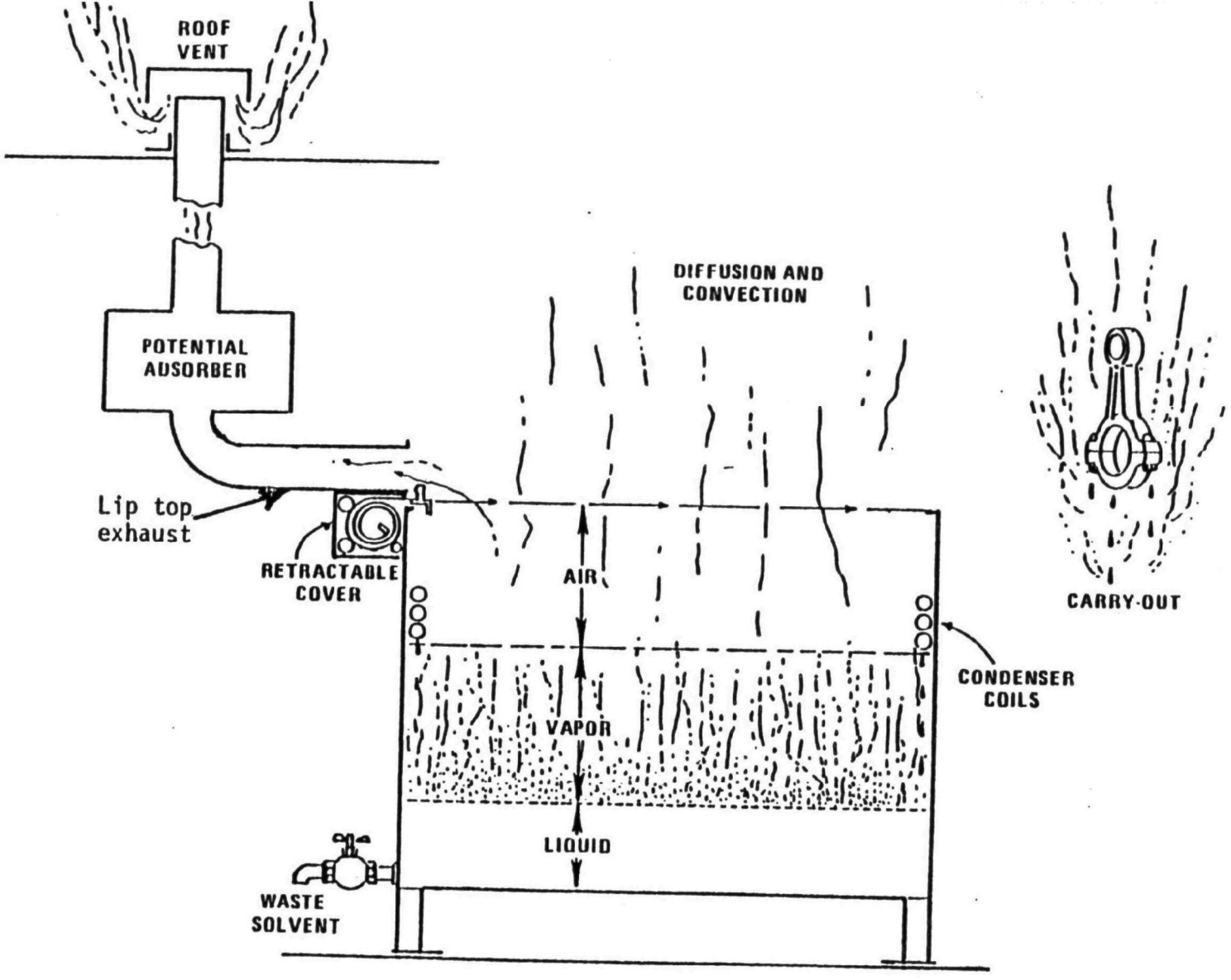

Figure F-3 Open-Top Degreasing Emission Points

Source: EPA, 1977a.

Figure F-3. Open-Top Degreasing Emission Points (EPA, 1977a)

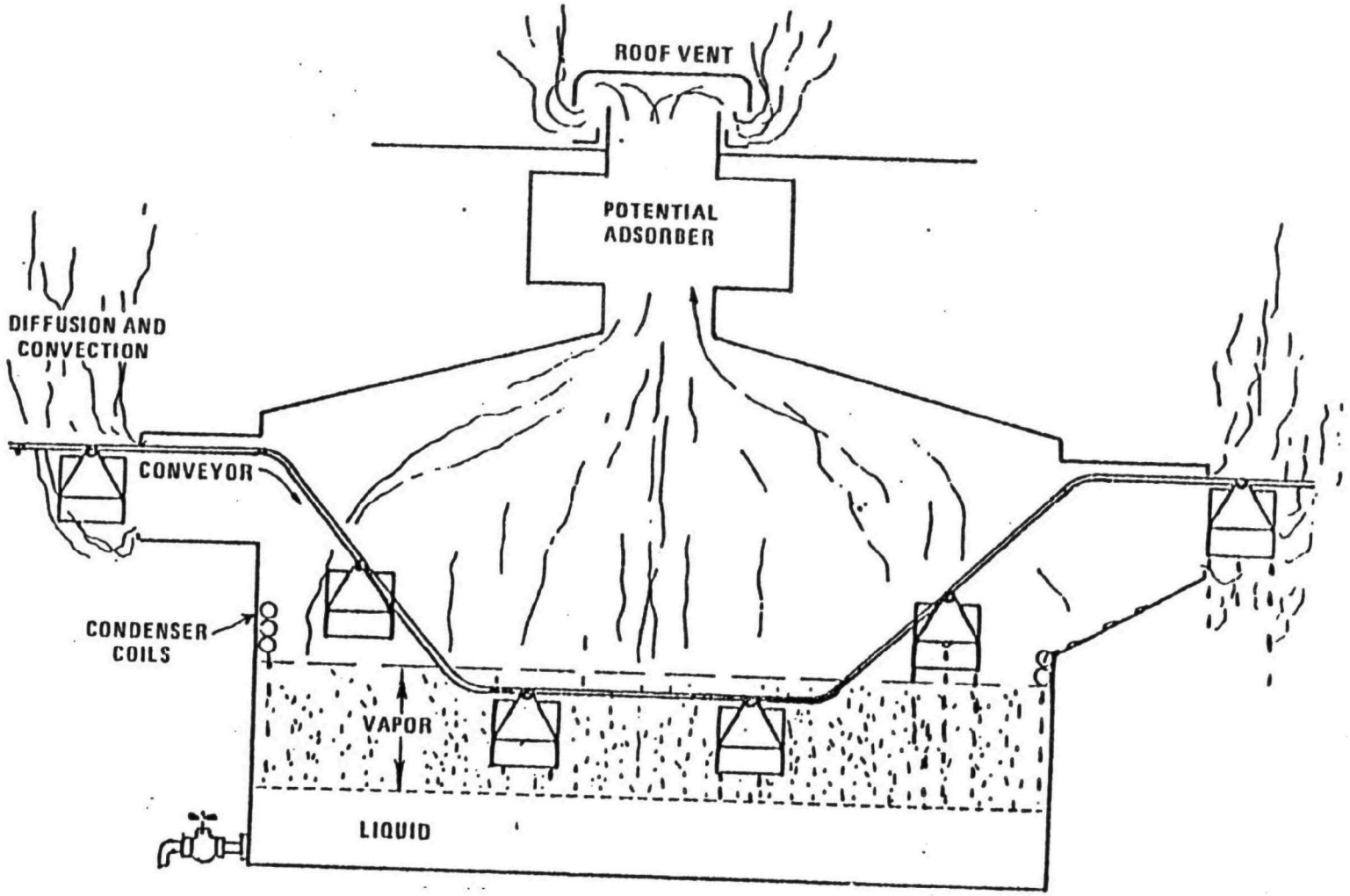

Figure F-4 Conveyorized Degreaser Emission Points

Source: EPA, 1977a.

Figure F-4. Conveyorized Degreaser Emission Points (EPA, 1977a)

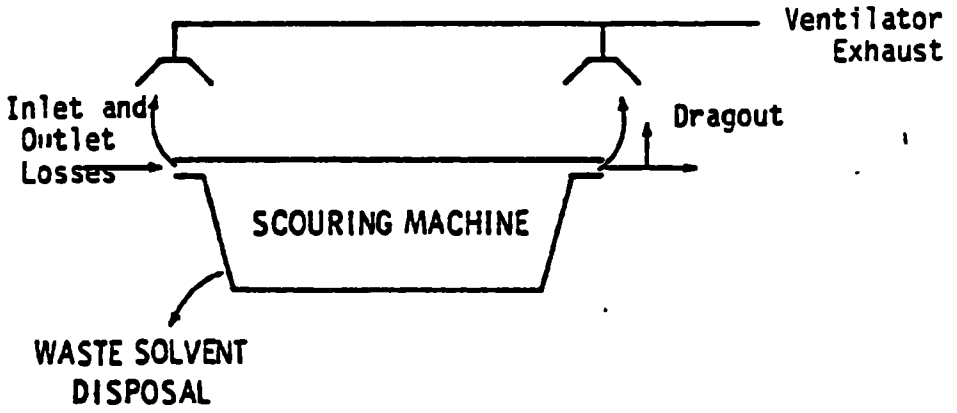

Figure F-5. Fabric Scourer (EPA, 1979b)

APPENDIX G

G.1 PHYSICAL PROPERTIES AND AMBIENT LEVELS

This section describes the physical properties of trichloroethylene and ambient levels in air, water and soil. Included is a list past damage incidents arising from improper disposal or storage of trichloroethylene-containing waste. Cumulative emissions from degreasing operations have been estimated for the past 20 years, with emphasis on waste solvent dispersion (see Section 3.5.2).

G.1.1 Physical Properties

Trichloroethylene ($CHCl=CCl_2$) is a colorless, sweet smelling, volatile liquid at normal temperatures. Its physical properties are listed in Table G-1. The fact that it is nonflammable under conditions of normal use and miscible with many organic liquids makes it a versatile solvent with many industrial applications, primarily in metal degreasing.

G.1.2 Ambient Levels of Trichloroethylene

Trichloroethylene is found in all media of the environment. It is, however, not ubiquitous and available literature indicates it is solely man-made (Derwent and Eggleton, 1977). Like other low molecular weight (C_1-C_3) chlorinated hydrocarbons, trichloroethylene has a relatively high vapor pressure and low water solubility and thus, tends to partition toward the atmosphere.

G.1.2.1 Atmosphere

In air, trichloroethylene is found in the part per trillion (ppt) range (see Table G.2), with concentrations highest near urban areas (Lillian, et al., 1975; Singh, et al., 1977a; Correia, et al., 1977; EPA, 1977b). The OSHA recommended occupational exposure standard is 100 ppm, averaged over an eight hour workday, and is based on the threshold limit value established by the American Conference of Governmental Industrial Hygienists (ACGIH, 1976). Trichloroethylene is thought to be rapidly oxidized in the atmosphere (NAS, 1975; Gay, et al., 1976; Dilling, et al., 1975), giving rise to toxic products such as phosgene (Lillian, et al., 1975) and chloroacetyl chlorides (Singh, et al., 1977a). A detailed discussion of the photochemical fate of trichloroethylene, however, is beyond the scope of this materials balance.

G.1.2.2 Water

Levels of trichloroethylene in water vary with the degree of pollutant contamination. Levels in surface waters of industrialized

Table G-1. Properties of Trichloroethylene

molecular weight		131.39	
melting point, °C		−87.1	
boiling point, °C		86.7	
specific gravity			
liquid			
20/4°C		1.465	
100/4°C		1.325	
vapor[a] at bp		4.54	
vapor density at bp, kg/m³		4.45	
n_D			
liquid, 20°C		1.4782	
vapor, 0°C		1.001784	
viscosity, mPa·s (= cP)			
liquid			
20°C		0.56	
60°C		0.42	
vapor at 60°		10.3[e]	
surface tension at 20°C, mN/m (= dyn/cm)		26.4	
heat capacity at 20°C, J/(kg·K)[c]			
liquid		941	
vapor		653	
critical temp, °C		271.0	
critical pressure, MPa[c]		5.02	
thermal conductivity, W/(m·K)			
liquid		136.5	
vapor, at bp		8.34	
coeff cubical expansion, liq, 0–40°C		0.00119	
dielectric constant, liquid, at 16°C		3.42	
dipole moment, C·m[d]		3.0×10^{-30}	
heat of combustion, MJ/kg[b]		7.325	
heat of formation, MJ/(kg-mol)[b]			
liquid		4.18	
vapor		−29.3	
latent heat of evaporation at bp, kJ/kg[b]		240	
explosive limits, vol % in air			
25°C		8.0–10.5	
100°C		8.0–52	
vapor pressure[e], kPa[f]			
Antoine constants	*A*	*B*	*C*
	5.94606	1187.51	214.474
solubility, g			
H_2O in 100 g trichloroethylene			
0°C		0.010	
20°C		0.0225	
60°C		0.080	
trichloroethylene in 100 g H_2O			
20°C		0.107	
60°C		0.124	

[a] Air = 1.
[b] To convert J to cal, divide by 4.184
[c] To convert MPa to atm, divide by 0.101.
[d] To convert C·m to debye, divide by 3.336×10^{-30}

[e] $\log_{10} P = A - \left(\frac{B}{T + C}\right)$

[f] To convert kPa to mm Hg, multiply by 7.5.

Source: McNeill, 1979.

Table G-2. Ambient Levels of Trichloroethylene in Air

Air Levels	Comments
0.05-8.8 ppb[a]	Measurable primarily near urban areas. Precursor of phosgene.
16 ± 8 ppt[b]	Northern hemisphere. Southern hemisphere background concentrations <3 ppt
20 ppt[c]	Averaged tropospheric background concentration.
5-15 ppt[d]	Lower troposphere
2.6-3.3 ppb[e]	Urban
14.5 ppt[f]	Significant urban-nonurban gradient
15.6 ppt[g]	N. hemisphere background concentration
16 ppt[h]	Ambient concentration in industrialized areas
1 ppt-100 ppb[i]	Remote areas Manufacturing areas (range)
2-16 ppt[j,k]	Ambient concentrations in industrialized areas in U.S.
0.1-3 ppb[k]	Western Europe concentration higher in urban areas than near manufacturers

a) Lillian, et al., 1975
b) Singh, et al., 1979
c) Cronn, et al., 1977
d) Derwent and Eggleton, 1978
e) Ohta, et al, 1977
f) Singh, et al., 1977a
g) Singh, et al., 1977b
h) EPA, 1976a
i) EPA, 1977
j) EPA, 1979b
k) Correia, et al., 1977

Table G-3. Ambient Levels of Trichloroethylene in Water

Water Levels	Comments
<5 ppb	Surface waters of industrialized river basins, average concentration
0.02-25 ppb	Rivers, canals, seawater in Europe
Trace amounts (≤1ppb)[c]	Drinking water
4.1-70 ppb	Municipal wells
Trace - 6700 ppb	Production wells near trichloroethene storage tanks on Air Force Base
1-21 ppb	Drinking water in various cities
0.1-32 ppb	Drinking water in various cities
< 10 ppm	Wells near lagoon receiving waste from tool manufacturer
< 260 ppm	Wells near sanitary landfill
0-188 ppb	Great Lakes area, water plus suspended sediment

a) EPA, 1977b
b) Correia, et al., 1977
c) Thomason, et al., 1978
d) Joyce, 1979; See Section 3.5.1
e) Grimes, 1980; See Section 3.5.1
f) EPA, 1978a
g) EPA, 1979c
h) EPA, 1979c
i) Council on Environmental Quality, 1978

areas are generally in the part per billion (ppb) range (see Table G-3). Levels in groundwater and municipal water supplies (e.g., wells, reservoirs) may be much higher, as a result of improper storage or disposal practices (e.g., leaching from landfills or leakage from storage facilities; see Table G-3 and Section 3.5.1 for documented levels of trichloroethylene contamination in well water of up to 260 ppm). Furthermore, chlorination of drinking water is a potential, yet apparently insignificant, inadvertent source of trichloroethylene (see Section 3.3.5.2). Contamination of a waterbody may result from ongoing discharges or from previous incidents in which contaminated water moved slowly and with little mixing through an aquifer (Walker, 1973). While trichloroethylene evaporates readily from agitated aqueous systems (NAS, 1975; Helz and Hsu, 1978; Dilling, et al., 1975), it may persist unchanged in undisturbed water bodies (i.e., plug flow). Evaporation of trichloroethylene from water is a more likely route of removal than by hydrolysis, biodegradation, or transfer to sediments (NAS, 1975; Helz and Hsu, 1978).

Data concerning ambient levels of trichloroethylene in soil and sediment are scarce, although levels of up to 7 ppm have been documented (EPA, 1979c). Major sources of trichloroethylene discharge to soil are leaching from landfills, illegal dumping, and subsurface waste injection. Trichloroethylene may also enter soil by injection of fungicides into crop land. From there it may evaporate (the most likely route), be leached away, or be taken up by plants. It is not known whether soil adsorption of trichloroethylene is a significant phenomenon.

G.2 ENVIRONMENTAL DAMAGE INCIDENTS

Table G-4 is a summary of documented U.S. environmental damage incidents involving trichloroethylene (see Section 3.5 for a discussion of waste mismanagement).

Table G-4. Recently Documented Damage Incidents as a Result of Improper Waste Management of Trichloroethylene

Location	Suspected/Confirmed Generator, Trichloroethene Use	Disposal Method/ Source of Pollution	Trichloroethene Contamination
Rancho Cordova, California	Aerojet General/ Cordova Chemical: solvent during production and delivery of rocket propellant	Open pit, old dredge pit, Unlined and defectively lined surface percolation ponds	Ground water to Dredge pit; 25-30/50 private wells sampled within 0.5 mile of Aerojet property
San Gabriel Valley, Los Angeles, California	Not specified	Not specified	56 municipal wells within 18 water supply systems
Mather AFB, California			16.5-30.2 ppb in drinking water wells
McClellan AFB, California	Degreasing, cleaning of machinery on base	Old industrial sludge pits, settling ponds, underground storage tanks	several wells, on and off base
Southington, Connecticut	Solvents recovery service: distillation, recovery and disposal of industrial solvents	Not specified	groundwater: three of six wells closed, two of these three contained hazardous levels of trichloroethene
Canton, Connecticut	John Swift Chemical Company	Not specified	11 Canton wells
East Gray, Maine	Waste disposal site accepting $1\text{-}2\times10^5$ gal/yr of waste oil and various liquid wastes	Tanks or 1/2-acre asphalt-lined lagoon	20 nearby residential wells
Gray, Maine	Solvent and oil waste processing facility	Not specified	Residential well

Table G-4. (Continued)

Location	Suspected/Confirmed Generator, Trichloroethene Use	Disposal Method/ Source of Pollution	Trichloroethene Contamination
Massachusetts: Acton, Bedford, Belchertown, Burlington, Canton, Danvers, Groveland, Lunenberg, North Reading, Norwood, Rehoboth, Rowley, Shrewsbury, Woburn, Wilmington, Yarmouth	Various: unidentified dumpers, industries using degreasers (eg. electronics, machine shops)	Illegal discharge into leaking sewer lines, illegal dumping of 55 gallon drums to unlined sites, discharge from septic leach lines	Private wells, municipal supplies: up to 100% contamination in some areas
Oscoda, Michigan	None identified	Open dumping of trichloroethene on site of nearby auto parts plant	Eight private wells and one industrial well
Camden, New Jersey	Harrison Avenue landfill - active until 1976	Abandoned gravel pit - hazardous materials dumped with municipal trash	Leachate over tidal mudflats to Delaware River - nearby residential population
Mt. Holly, New Jersey	Landfill and Development Company	20 acre landfill on banks of Rancocos Creek, adjacent to housing developments - no liner	Private water wells, groundwater
Niagara Falls, New York (Hyde Park site)	1953-1975 Hooker Chemical	15 acre landfill, two drainage ditches emptying into tributary of Niagara River	Migration from landfill, hazardous levels
Niagara Falls, New York (Love Canal site)	1942-1952 Hooker Chemical	Landfill - two 70 foot strips on either side of 60 foot canal	Migration from landfill at hazardous levels; 239 homes and grammar school built on land around canal; 3 storm sewers underlie the landfill, emptying into a tributary of Niagara River, which contains hazardous amounts in water and sediment
Islip (Suffolk County), New York	Hickey's Carting	Town dump	4×10^3 gallons of industrial cleaner trichloroethene dumped; numerous area wells showed trichloroethene

Table G-4. (Concluded)

Location	Suspected/Confirmed Generator, Trichloroethene Use	Disposal Method/ Source of Pollution	Trichloroethene Contamination
West Nyack, New York	None determined; various industries within one mile of site used trichloroethene	Not specified	16 private wells closed
Bucks and Montgomery Counties, Pennyslvania	None determined	12 active sites under investigation	Private wells, camp and municipal water supplies
Newbery Township, Pennsylvania	One company, not named	Not specified	Private wells
Hazelton, Pennsylvania	From New Jersey, midnight dumpers	Dumped into quarry	Leakage to aquifer, potential water supply contaminated
Lehigh County, Pennyslvania	None determined	Heleva landfill	Well supplying 50 homes
North Smithfield, Rhode Island	Chemical wastes and septage of undetermined origin	Western Sand and Gravel - trenches and unlined lagoons: sand and gravel	Leaching to brook and reservoir
Bristol, Rhode Island	None determined	>663 barrels in three illegal dump sites	Adjacent marshland, >11 wells

Source: EPA, 1980a, except Massachusetts incidents, which are from Massachusetts, 1979.