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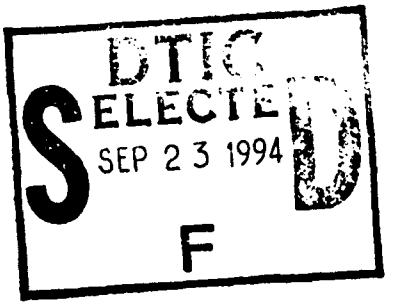

A RETROSPECTIVE QUANTITATIVE ASSESSMENT OF TRICHLOROETHYLENE EXPOSURE OF WORKERS AT AIRCRAFT MAINTENANCE FACILITIES AT HILL AIR FORCE BASE THROUGH THE USE OF MODELING

THESIS

Anthony O. Copeland, Captain, USAF

AFIT/GEE/ENV/94S-06

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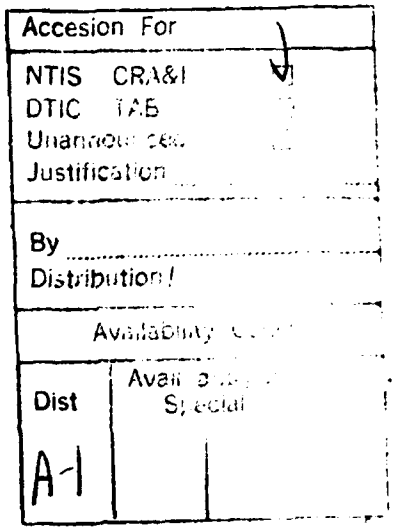

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THESIS

Presented to the Faculty of the School of Engineering

of the Air Force Institute of Technology

Air University

In Partial Fulfillment of the

Requirements for the Degree of

Master of Science in Engineering and Environmental Management

Anthony O. Copeland, B.S.

Captain, USAF

September 1994

Acknowledgments

Very special thanks goes to my lovely fiancée, Lisa. Without her support, encouragement, kind ear, and timely sense of humor, completing this project would have been nearly impossible. Thanks also goes to Fred Cade for providing comic relief when the going got rough - I' m going to miss those late night, stupid jokes and the Def Comedy Jam recordings. I'd also like to express my gratitude to Dan Marano, Lt Col Nu, Dr. Klinko, SSgt Fortunato, and the members of the Hill AFB Bio-Environmental Engineering Flight for the valuable assistance they provided during my site visit. Finally, I'd like to thank my advisor, Lt Col Dan Caldwell, for affording me the right mix of freedom and guidance, thus making the entire thesis writing process truly rewarding and memorable.

- Anthony O. Copeland.

Table of Contents

List of Figures

List of Tables

Abstract

Monte Carlo simulation and source-receptor modeling are used to estimate the TCE exposures encountered by select workers at aircraft maintenance facilities at Hill Air Force Base between 1955 and 1979. An epidemiological study of this group, a retrospective cohort study of 14,457 workers who were employed at the base for a minimum of one year between 1952 and 1956, was headed by Dr. R. Spirtas of the National Cancer Institute to evaluate mortality associated with occupational exposure. One of the major conclusions of the study was that TCE "probably does not pose a strong carcinogenic risk for man." In the Spirtas study, historic exposure levels were not quantitatively estimated. Instead, indices of exposure to TCE were assigned to reflect relative differences in exposure levels. It is the objective of this research effort to quantitatively estimate specific worker exposures, thus adding to the weight of evidence necessary to classify TCE as a human carcinogen or otherwise.

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I. *Introduction*

This chapter provides background information on the general issue of trichloroethylene (TCE) contamination of soil and groundwater found at military installations across the country. The importance from both a financial and human health standpoint are briefly discussed. Finally, discussed in this chapter are the specific problem, investigative questions, and the scope and limitations of this research.

General Issue

Trichloroethylene exists as a clear, colorless, nonflammable, volatile liquid and is primarily used as a dry-cleaning solvent or a degreasing agent (Manahan, 1990:76). The Department of Defense (DoD), in the past, used TCE primarily as an industrial solvent. Usage, storage, and disposal practices of the past have resurfaced as soil and groundwater contamination problems today. The majority of all DoD installations found on the National Priorities List (NPL) are in some manner contaminated with chlorinated organic liquids, with TCE being among the most common. In several cases the contamination has affected groundwater sources found on the installations.

Additionally, migration off-site has occurred and affects public and private water supplies in some areas. Incidents of solvent contamination of soils and groundwater are not confined to DoD installations. Private industry has experienced similar usage and disposal practices as military installations and subsequently is responsible for sites in which TCE and other halogenated hydrocarbons have contaminated soil and groundwater.

It has been suggested by John Cherry, a leading consultant on problems of groundwater contamination, that chlorinated organic solvents are by far the most prevalent sources of industrial groundwater contamination. He has also noted that we do not presently have the technological capability to remediate an aquifer to current drinking water standards if that aquifer is contaminated with a free or residual phase of an immiscible contaminant, such as chlorinated organic liquids (Bredehoett, 1992:834). The DoD and the United States Environmental Protection Agency (USEPA) have declared TCE a priority groundwater contaminant; the USAF has made TCE its number one chemical of concern (Fisher, 1993:2).

The magnitude of the TCE contamination problem, both in site size and numbers, coupled with the present maximum contaminant level (MCL) standard for TCE of 5 parts per billion (ppb) presents the DoD a situation in which cleanup costs associated with these sites are enormous. Add to this the fact that it is not always possible to remediate to this level, and the seriousness of TCE contaminated groundwater and its remediation become obvious. The current MCL standard used to determine the level of cleanup taking place on sites today was promulgated in 1987. The basis for the

standard was toxicological data that was current at that time (Fisher, 1993:3). Since the initial establishment of the MCL, the understanding of the mechanisms of action of TCE in rodents and the effects of human exposures have been furthered through additional toxicological and epidemiological data. However, the MCL has remained the same while the current policy guidelines on TCE are under review (Fisher, 1993:3).

Presently, there is an ongoing joint venture (Strategic Environmental Research and Development Program for the Scientific Advisory Board (SERDP SAB); Thrust: Installation Restoration (IR-27), Title: Toxicology and Human Health Risks) between the USEPA, US Army, US Air Force, and the US Navy. The goal of this project is to "conduct specific research studies and incorporate recent research findings on TCE and PCE [perchloroethylene] to support the development of biologically-based health risk standards for TCE and PCE in drinking water and air" (Fisher, 1993:4). Accomplishment of this objective would result in a current MCL for TCE which would be representative of the actual risks associated with exposure to TCE today. If the revised MCL turned out to be higher than present, this would translate into more cost-effective cleanups of TCE contaminated sites throughout the country for the US government and the private sector because of the less stringent remediation standard. The information necessary to make such a decision will be supported by both toxicological data resulting from the development of a TCE biologically-based model and epidemiological data from recent cohort studies. The purpose of this research is to contribute data to the body of evidence provided by epidemiological data and findings.

Specific Problem

The specific focus of this research will be to reconstruct, through source-receptor modeling, the TCE exposures encountered by selected workers at aircraft maintenance facilities at Hill Air Force Base between 1954 and 1978. The epidemiological study of this group, a retrospective cohort study of 14,457 workers who were employed at the base for a minimum of one year between 1952 and 1956 and herein referred to as the study, was undertaken by Spirtas et al. to evaluate mortality associated with occupational exposure. One of the major conclusions of the study was that TCE "probably does not pose a strong carcinogenic risk for man" (Spirtas, 1991:528). In the study, the development of historic exposure levels is based on information obtained from facility walk throughs, employee interviews, limited monitoring data, position descriptions, and work practices. It is the objective of this research effort to further refine the exposure estimates of Spirtas et al., thus adding to the weight of evidence necessary to classify TCE as not a human carcinogen.

Investigative Questions

The investigative questions of this research are as follows:

1. Does sufficient information exist to model historical exposures found in the Hill AFB epidemiology study performed by Spirtas et al?
2. What type of model will best estimate this exposure?
3. What quantitative exposure levels result from applying such a model?

Scope and Limitation

The study by Spirtas et al. encompassed 14,457 workers who worked under 43,000 unique job titles (Stewart, 1991:531). The difficulty in trying to assimilate occupational and exposure information on each of these jobs from over 40 years ago is the limiting factor with regard to the scope of this research. Evaluation of exposure will be limited to attempting to reconstruct or estimate exposures of workers for which the most information is available . The limitations of this research are determined by the available data describing the work activities found in the areas of interest and the fact that exposure levels will be assessed based on area of work, not specific individual exposures.

II. *Literature Review*

Introduction

The primary purpose of this literature review is two-fold. The first is to equip the reader with a fundamental understanding of the important characteristics of trichloroethylene and provide a perspective of the relative relationship between existing information regarding the health effects of TCE and the current regulatory requirements. A review of the history of TCE, its carcinogenicity, and exposure routes is presented. The secondary purpose is to familiarize the reader with the exposure estimation portion of the epidemiology study that this work is concerned with reconstructing. Additionally, methods for determining a historical occupational exposure history will be discussed.

Background of Trichloroethylene

A discussion of the history of TCE, its carcinogenicity, and potential exposure pathways is now presented.

History. TCE was first produced in 1864 by Fisher (Feldman, 1970:599). Following that time, it has been produced in Austria and the United Kingdom beginning in 1908, in Germany since 1910, in Japan since 1935, and in the United States since 1925 (Royal Society of Chemistry, 1986:102). TCE, an unsaturated chlorinated hydrocarbon, exists as a dense non-aqueous phase liquid at room temperature. In its liquid state, it is characterized by its clear, colorless appearance, sweet burning taste,

and a pleasant, sweetish odor very similar to that of chloroform (Royal Society of Chemistry, 1986:106). No sources of naturally occurring TCE of any form are known to exist (Royal Society of Chemistry, 1986:102). Primary uses of TCE have been in clinical medicine and industrial processes. The use of TCE for medicinal purposes gained recognition beginning in 1942 when Langston Hewer presented to the Royal Society of Medicine, a report detailing the particulars of the chemistry of TCE, its physical properties, and its potential as an anesthetic (Enderby, 1944:300). Prior to Hewer's presentation, others in the medical field were discovering the effects of TCE on various parts of the central nervous system. Oljenick, in 1928, described, in a report to the Boston Society of Psychiatry and Neurology, the "narcotic" effect of TCE. Three years later, Glaser revealed that TCE depressed the cortex affecting painful stimuli and therefore, could induce anesthesia (Feldman, 1979:458). Because of the known and reported trigeminal neuropathy associated with TCE intoxication and its associated analgesic properties, medical professionals sought its use as a therapy for the treatment of trigeminal neuralgia (tic douloureux) and as an inhalant-analgesic-anesthetic for brief operations and various dental and obstetrical procedures (Feldman et al., 1992:490). Presently, due to restrictions regarding its potential health effects, TCE is not used as a general anesthetic or as an analgesic. Additionally, these same restrictions have led to its discontinuance in its application in fumigant mixtures and as an extractant in the decaffeination of coffee (Royal Society of Chemistry, 1986:102). Today, the major use of TCE is in industrial metal cleaning applications. Other uses are found in the electronics industry, in dry-cleaning and textile treatment processes, and the

manufacture of paints, enamels, lacquers, and adhesives (Feldman et al., 1985:119). Additionally, TCE serves as a chain terminator in the production of polyvinyl chloride and is also used as a fire retardant and an extinguisher (Royal Society of Chemistry, 1986:102).

Carcinogenicity. In rodents, TCE and its metabolites have been found to be carcinogenic (Allen, 1993:71). As a result of this finding, the EPA currently classifies TCE as a B2 or probable human carcinogen (Cronin, 1993:2-4). The metabolites, trichloroacetic acid (TCA) and dichloroacetic acid (DCA), have been shown to be capable of producing hepatocarcinogenesis in B6C3F1 mice when administered through drinking water (Larson, 1992:278). This same result has been produced when the mice were exposed through inhalation and via gavage. Other rodents, specifically rats, exposed via gavage have developed renal tumors (Allen, 1993:72). These results and those of several other studies suggest that the mechanism of carcinogenicity of TCE is manifested in its metabolites TCA, DCA, and chloral hydrate (Steinberg, 1993:137). Selected steps in the process through which TCE is metabolized in rodents and humans is shown in Figure 1. Although TCE is primarily metabolized in the liver by cytochrome P450 in both rodents and humans, the extent to which the metabolites TCA and DCA are formed varies between the species. In both species the first metabolite formed is trichloroethylene epoxide which then quickly breaks down to form trichloroacetaldehyde and dichloroacetyl chloride. The trichloroacetaldehyde is then hydrated yielding chloral hydrate. The singular chlorine atom of the dichloroacetaldehyde is replaced with a hydroxyl radical, thus forming dichloroacetic

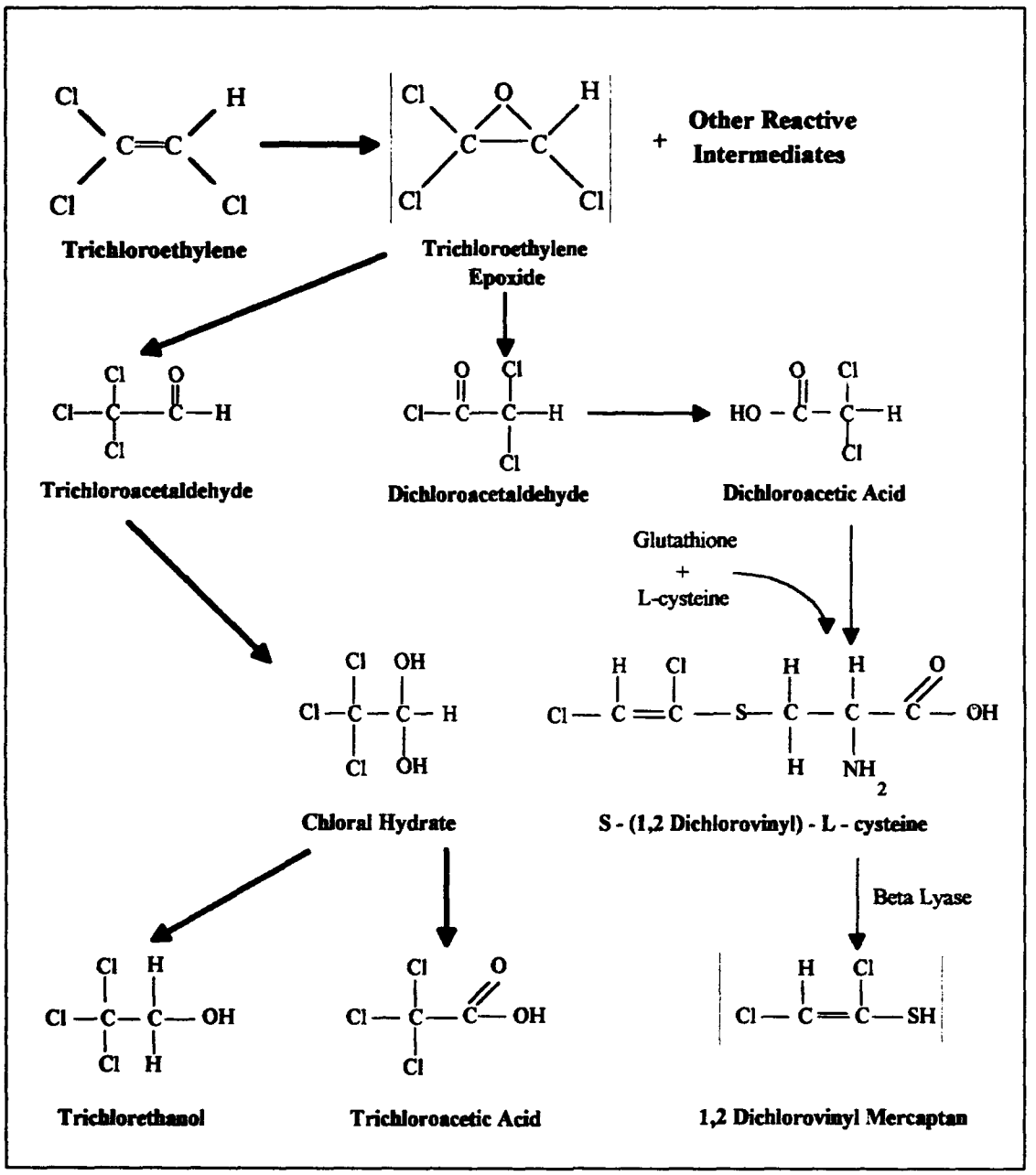


Figure 1. Metabolic Pathways of TCE in Humans and Rodents (Major metabolic pathways are shown using broad arrows, whereas minor pathways are shown with thin arrows. In rodents, 1,2 dichlorovinyl mercaptan is formed with a reactive thiol group.) (Steinberg, 1993:138).

acid. This reaction occurs more frequently in rodents than humans. The dichloroacetic acid is further broken down through enzymatic reactions which ultimately lead to

cellular necrosis. Whether chloral hydrate has entered the body as a distinct compound or whether it is formed as described above, once in the body it is quickly metabolized to trichloroethanol and TCA. In humans, TCA is the major toxic metabolite resulting from metabolism of chloral hydrate or TCE. Both trichloroethanol and TCA are further metabolized resulting in the formation of various acids and alcohols. Rodents, mice particularly, metabolize TCE to a greater extent than do humans (Steinberg, 1993:140).

Although no human epidemiological data exists showing increased occurrences of mortalities or malignancies resulting from chronic exposure to TCE, current regulatory guidance is based on a simple linear extrapolation from effects observed in rodents to expected risks in humans. However, the extrapolation is based on the assumption that the low dosages experienced by humans will have the same carcinogenic effect as the high doses which are given to rodents (Steinberg, 1993:137). The underlying assumption on which this linear extrapolation is based centers on TCE and its metabolites having a non-threshold effect - that is to say that any amount of TCE will result in some risk. The accuracy of this assumption is called into question by the fact that the mechanism of carcinogenesis for TCE and its metabolites in rodents is nonlinear. In the lab, large daily doses of TCE (on the order of 1000 mg/kg/day) are administered to rodents to produce cellular necrosis. Once necrosis occurs, a cyclic pattern of regeneration of the effected cells followed by additional necrosis eventually results in malignancy due to the onset of hyperplasia and then neoplasia (Steinberg, 1993:137). Steinberg and DeSesso also note that the aforementioned sequence of events occurs when high doses of chloral hydrate, the precursor to TCA, are

administered, thus further supporting the assumption that TCA and DCA mediate any carcinogenic effects. Hence, considering that TCE itself is not genotoxic and minimally mutagenic, the conclusion can be drawn that in rodents metabolism of TCE is required to produce carcinogenic effects (Steinberg, 1993:141). The relevance of this evidence is that chloral hydrate, the major metabolite of TCE, is also a widely used drug found in sleeping medications and hypnotic sedatives (Cronin, 1993:2-4). Furthermore, levels of TCA and DCA found in drinking water as by-products of the chlorination process are far in excess of those which would result from the metabolism of concentrations of TCE at the current regulatory maximum contaminant level for potable water of 5 ppb (Steinberg, 1993:137). Thus, the metabolites TCA, DCA, and chloral hydrate lead a dichotomous regulatory existence. When produced as a result of metabolism of TCE, they are considered toxic and the parent compound is regulated to the degree that its concentration in potable water shall not exceed 5 ppb. However, when used for medicinal purposes, chloral hydrate, which when metabolized yields TCA, is not subject to stringent regulatory stipulations as is TCE.

The chloracetic acids, TCA and DCA, have recommended safe levels of 175 ppb and 420 ppb respectively when occurring in drinking water as a result of the chlorination process (Steinberg, 1993:145). It is obvious that the likelihood of intaking TCA and DCA is greater if their origin is from the chlorination process, as opposed to the intake and subsequent metabolism of TCE contaminated water at concentrations in the range of 5 ppb. Because of this result and the lack of existence of any epidemiological data supporting TCE as a human carcinogen, Steinberg and DeSesso

suggest that it is possible to increase the allowable level of trichloroethylene in drinking water. They suggest that the current MCL of 5 ppb could be increased to 50 ppb without any increased health risks. Actions by Canadian health and welfare regulatory officials suggest they view the situation in a similar manner, as the recently promulgated TCE drinking water standard in Canada is 50 ppb (Steinberg, 1993:147).

Exposure Routes. Over the past several years, TCE has received much attention due to the fact that it was discovered in many drinking water supplies throughout the country (Wilkes, 1992:2227). The costs associated with remediating these many sites and the concerns of the potentially exposed public have made it a highly controversial contaminant. Within the Air Force, several installations (McClellan AFB, Wright-Patterson AFB, Hill AFB, Castle AFB, etc.) have had to deal with problems of TCE contaminated ground water affecting on-base and off-base water supplies. In cases where the incoming residential water supply was discovered to be contaminated with TCE at levels greater than the established health-based action level, bottled water was provided to the residents for drinking and cooking to minimize exposure through ingestion (Rao, 1993:37). However, because no such action levels exist for bathing, showering, laundry or dishwashing usage, the question arises whether it is safe to perform these activities using the contaminated water (Rao, 1993:37). Beginning in the early eighties and continuing until now, Andelman, McKone, Giardino, Hattis, and others have investigated the routes of exposure associated with residential usage of water contaminated with volatile organic chemicals (VOCs), particularly TCE. Because this route of exposure affects non-volunteer residential subjects and provides

useful information concerning bodily uptake of TCE, it along with occupational exposure routes will be discussed.

Trichloroethylene occurs throughout the environment. It has been found in surface waters, marine life, drinking water supplies, the atmosphere, food, plants, and animal tissues (Callahan et al., 1979:673). In the United States, it is estimated that 80-95% of all TCE produced eventually volatilizes into the atmosphere (Royal Society of Chemistry, 1986:96). Because of its high vapor pressure and Henry's Law Constant, most surface spills and accidental releases of TCE to soil evaporate rapidly. When TCE does leach into ground water, it does so rapidly because of its low adsorption coefficient in a variety of soils (Howard, 1990:468-469). In soil and sediments as well as ground water, it will slowly biodegrade. Ambient concentrations in industrial areas within the United States average 1.2 ppb (Howard, 1990:468-469). Because TCE is ubiquitous throughout the environment, Howard estimates the average daily intake through inhalation to be 0.011-0.033 mg and 0.002-0.020 mg as a result of uptake from ingestion of water.

Historically, quantifying exposure resulting from water ingestion has been viewed as the primary means of assessing exposure from contaminated water sources in residential settings. However, recently it has been found that inhalation of TCE as a result of volatilization from domestic water usage contributes substantially to the total amount of TCE intake. Though several factors such as activity patterns, water-use patterns, and location within the house affect the occupants' inhalation exposure, in cases where the occupant spends a majority of his time at home, inhalation exposure is

likely to equal or exceed exposure resulting from ingestion of the same water (Wilkes, 1992:2227). Experiments by McKone suggest that inhalation exposure derived from showering is equivalent to ingesting 1 to 4 liters of the water. Interestingly, for TCE and other volatile compounds, the transfer efficiency of the compound from the water to air is limited by liquid-phase mass transfer and not gas-phase mass transfer (McKone, 1991:282). This implies that water temperature has little effect on transfer efficiency. Several models including INDOOR, CONTAM, PAQM, and MAVRIQ are available for evaluating various aspects of indoor air quality. MAVRIQ combines various aspects of the aforementioned models to produce a model ideally suited for evaluating exposure resulting from volatilization from water-use activities. Occupant characteristics such as breathing rate, location within the house, activity patterns, water-use activity patterns, air flow patterns and rates, and chemical characteristics are input into the model which then calculates the integrated inhalation exposure of each specified occupant within the home (Wilkes, 1992:2228-2229). The model has been validated several times for theoretical and realistic accuracy. Most recently the model performed very well when applied to an actual case study of a community of several homes where the water supply was contaminated with TCE (Wilkes, 1992:2229).

Another route of exposure which also recently gained recognition as a viable and possible significant exposure pathway in residential exposures is dermal contact with contaminated water. Attempts to model the exposure resulting from dermal contact with contaminated water such as from bathing in contaminated water are presently not as developed and accurate as inhalation modeling. However, this route of exposure

could possibly be as significant as the oral ingestion and inhalation routes of exposure (Brown, 1989:848). The manner in which a chemical enters the body through the skin and the compartmental representation scheme used to model this phenomenon is described by Brown and Hattis below:

> We identify two relatively uniform, if not homogeneous, layers of skin: stratum corneum and "skin." The outermost stratum corneum, on the average is approximately 40 micrometers thick, is made up of densely packed keratinized cells and presents the greatest resistance to the movement of molecules across human skin. Organic molecules in dilute aqueous solutions diffuse across the stratum corneum by purely passive means, driven by the concentration gradient between the outer reservoir and the inner layer of skin. Because of the resistance of stratum corneum to the penetrant molecules, the passage across this barrier is the rate-determining step in dermal absorption of chemicals. The stratum corneum behaves as a solution phase of high viscosity and the penetrant must dissolve in it in its passage into the body; thus the rate of transport across stratum corneum depends on the penetrant's solubility in that membrane (stratum corneum/water partition coefficient). ...The compartment labeled skin consists of the viable epidermal layer, the papillary dermis, and possibly inner parts of dermis that may serve as a storage depot for lipophilic chemicals...Chemicals entering the "skin" layer are presumed to diffuse rapidly through the approximate distance of 200 [micrometers] (0.02 cm) between the stratum corneum and blood vessels, diffuse through the capillary walls, and be carried away from "skin" into the blood stream...No distinction is made between a chemical in the dermal blood vessels and a chemical in general circulation. The rate of transfer of a chemical from "skin" into blood is proportional to the instantaneous amount in the "skin," the perfusion rate of the "skin" with blood, and the relative solubilities of the agent in the two compartments (blood/skin partition coefficient). The net rate of change in the amount of chemical in the "skin" depends on the rate of entry from stratum corneum, the rate of removal into blood, and the rate of reentry from the blood (Hattis, 1989:840-841).

Estimating and predicting dermal absorption of a chemical using this approach should be done cautiously, for experimental data to further refine and validate the model is needed. Nonetheless, the model is physiologically representative of the actual process and has very useful applications when used to estimate relative amounts of chemical

absorbed by different individuals under several unique circumstances. Table 1 below shows the range of the predicted daily intake of TCE obtained from taking a 20 minute bath with 73% of the body immersed in water. These values are presented in conjunction with ingestion and inhalation exposures for comparison.

Table 1. Comparison of Adult Daily Doses of TCE in Tap Water by Dermal, Oral, and Respiratory Routes (mg/day) (Brown and Hattis,1989:848).

Compound	Concentration (mg/L)	Oral	Respiratory	Minimum Dermal	Maximum Dermal
Trichloro-ethylene	0.01 0.1	0.02 0.2	0.0287 0.287	0.0006 0.006	0.0096 0.096

NIOSH estimates that close to 400,000 workers in the United States are exposed to trichloroethylene. Concentrations range anywhere from ten to the hundred parts per million on a continuous basis (Howard, 1990:472). The primary exposure route is inhalation. Adsorption of TCE in its liquid phase through the skin occurs and can be a significant means of exposure if the duration and area of exposure is sufficient. Uptake as a result of adsorption of TCE vapor through the skin is negligible (Royal Society of Chemistry, 1986:114). Thus, the majority of occupational exposure results from inhalation of TCE vapors. Studies of workers who have been chronically exposed to TCE for several years show increased occurrences of various physical ailments (reddening of skin, fatigue, vertigo, headache, bradycardia, sensory desensitization) and effects on the central nervous system (NIOSH, 1973:37-39). However, epidemiological

studies on occupational exposure to TCE fail to show any link between TCE and the increased occurrences of any malignancies (Spirtas et al, 1991:528).

Exposure Assessment

A description of the epidemiological study performed at Hill Air Force Base as it pertains to this research, methods for estimating historical exposure, and relevant issues in modeling are presented.

Hill AFB Epidemiological Study. The Hill AFB epidemiology study was undertaken as result of actions by Hill AFB employees and their union, the American Federation of Government Employees (AFGE) Local 1592. On 2 March 1978, one present and two former Hill AFB employees filed a claim under the Federal Tort Claims Act alleging that the United States Air Force had "knowingly and intentionally furnished to claimant for use in the performance of his duties dangerous and poisonous substances and knowingly and intentionally failed to provide proper and adequate safety measures (Bayer et. al., 1978:79)." Immediately following this formal suit against the Air Force, it was made publicly known that there also existed a formal complaint filed with the AFGE Local 1592, which alleged that exposure to toxic chemicals had contributed to the death or illness of 44 workers. Two days following this public announcement, the President of the AFGE Local 1592, Mr. Breeden, in a letter to the OO-ALC/CC, stated that "he believed that toxic chemicals or other agents harmful to employee health had or was being utilized within portions of [the Maintenance Directorate], Building 100 (Bayer et. al., 1978:79)." The Air Force responded by

sending a staff assistance visit team composed of members from Hq USAF/SGPA, AFLC/SGB, AFLC/MAXF, the USAF Occupational and Environmental Health Laboratory, the National Institute for Occupational Safety and Health, and the Department of Health, Education and Welfare (Bayer et. al., 1978:79-80). Upon visiting the base, reviewing historical records, and interviewing employees, the Air Force/NIOSH Staff Assistance Team's primary recommendations were to establish and further improve management practices concerning worker training, exposure assessment, grievance procedures, and the worker medical program. It was also recommended that a statistical analysis of past and present cancer cases existing in workers be carried out. In January 1982, in response to the team's recommendations and mounting political and union pressure, the National Cancer Institute was contracted to perform a retrospective mortality study of 14,457 civilian workers who had been employed at Hill AFB for at least one year between 1952 and 1956. During that same year, the Rocky Mountain Center for Occupational and Environmental Health of the University of Utah School of Medicine was performing a study to determine the feasibility of morbidity research at the base. Their final report, "Report on the Feasibility of Morbidity Health Research at Hill Air Force Base, Utah", by the Rocky Mountain Center for Occupational and Environmental Health, Departments of Internal, and Family and Community Medicine, University of Utah School of Medicine, Salt Lake City, Utah 84112, NIOSH Health Hazard Evaluation Cooperative Agreement 1-UO1-OH-01249-01, January 1983, made several recommendations as to possible

morbidity research that could be performed on the base. However, no research into this area was performed.

The primary goal of NCI's retrospective mortality study was to determine if any relationship existed between exposure to TCE and specific causes of death (Spirtas et al., 1991:516). Resultant findings of the study are listed in Tables 1-4 of Appendix A. The task of determining the exposures was carried out by two industrial hygienists, J.S Lee and D.E. Marano. They performed shop inspections, personnel interviews and historical record and document reviews. Evaluation of this data led to the establishment of a job dictionary consisting of approximately 43,000 job and organization codes. From this job dictionary, qualitative and some quantitative estimates of exposures were made for combinations of specific jobs within an organization or shop. Jobs were classified according to whether exposure was intermittent or continuous, frequent or infrequent, and low level or peak (Spirtas et al., 1991:517).

For TCE and mixed solvents, exposure levels were estimated by first determining frequency and duration of use of the solvents based on information obtained from worker position descriptions, technical orders, and historical shop files. These results are shown in Table 2. Secondly, exposure indices were assigned which reflected the relative differences in TCE exposure levels which existed throughout time. Table 3 contains these results. Using this information, indexes of cumulative exposures were calculated by using the following relationship (Stewart et al., 1991:535):

$$I = (f)(d)(i)/480$$

f = frequency of use (Table 2)

d = duration of use (Table 2)
i = index of exposure (Table 3)
480 = number of minutes in eight hours

Specifically, cumulative exposure is the product of the index *I* and the number of years spent at each job. Thus, if an individual had worked from 1957-1958 as a pneudraulic systems mechanic for 300 days with frequent exposures to TCE, his index of exposure, *I*, for that period would equal 20.5 = 2 x 15 x 400 x 300/(480 x 365). Summation of all *I*s for all jobs held would yield the cumulative score. The cumulative score would then be divided by the total number of years the individual was exposed, thus yielding the average index which was used in the mortality analysis (Spirtas et al., 1990:5-6).

Table 2. Frequency and Duration of Use of TCE and Mixed Solvents (Stewart et al.,1991:535).

Types of Job	Frequency (f)	Duration (d), min.
Benchwork	Continuous, about 15/day	5
(low level exposures)	Intermittent, about 4/day	5
Vapor degreasing	Frequent, 2/day	15
(peak exposures)	Infrequent, 0.4/day	15

Table 3. Indices Used in Exposure Estimation (Stewart et al., 1991:535).

Period	Peak (TCE)	Low Level (TCE)	Mixed Solvents
1939-1954	600	10	10
1955-1967	400	10	10
1968-1978	200	0	10
1979-1983	0	0	10

Note: Use of TCE was eliminated from the base as a cold solvent in 1968 and as a degreasing solvent in 1978.

From their study, NCI concluded that "there were no significant or persuasive relationships between various measures of exposure to TCE and the risk of any specific malignancy" (Spirtas et al., 1990:30). Though limitations exist in this study, (i.e. quantification of exposure, low levels of exposure, and small numbers of deaths for some causes) it remains the largest study of industrial exposure to TCE with 7282 persons exposed (Stewart et al., 1991:536). Additionally, the conclusions reached by NCI are consistent with other epidemiology studies of persons exposed to TCE (Spirtas et al., 1991:528).

Estimating Historical Exposures. Accurately estimating historical exposure levels of potential hazardous agents is important because the "true test" of long term effects of human exposure to that agent are often provided by results of epidemiological studies (Nurtan, 1979:58). Specifically, for occupational exposure, the retrospective cohort study is the most widely used type of epidemiological study performed in the evaluation of potential hazardous agents found in industrial occupational settings (Monson, 1990:51). The process of determining historical exposures is usually carried out by industrial hygiene researchers and, more often than not, proves to be a difficult task at best (Smith et al., 1991:441).

Assessing exposures, regardless of whether they are present or historical, is accomplished by three general approaches or combinations thereof. The first is the predictive approach, which is characterized by estimating the distributions of chemicals encountered in the environment in conjunction with the activity patterns

of the exposed individual. The second approach entails inferring exposure levels based on observed levels of contaminants and metabolites observed in biological fluids and tissues of the exposed individual and is called the reconstructive approach. The final means, the direct approach, is the most straightforward and simply involves real-time measurement of exposure levels actually experienced by individuals (Whitmyre et al., 1992:297).

Within the boundaries described by these three approaches exist methods for assessing purely historical exposure levels. Two conceptual models which are identified as the source-receptor model and the task-TWA (time-weighted average) model of full-shift exposures provide the foundation for which most historical exposure assessments are based (Smith et al., 1991:441).

The source-receptor model involves the formal process by which the researcher, usually an industrial hygienist, exercises professional knowledge-based decision processes to analyze the situation and determine an appropriate exposure. Such a process typically includes record reviews to establish jobs held and areas worked by members of the cohort, tasks and resultant exposures associated with each job, reviews of historical exposure estimates and monitoring data, observations of present day activity patterns, physical layouts, work practices, etc., and finally, the synthesis of this and all other pertinent available information into an estimate of exposure (Stewart et al., 1986:34). Further classification of the source-receptor concept methodology leads to a categorization of the methods based on the general exposure experience of the cohort. Essentially, this means

that the estimate of a particular historical exposure is based on an analysis of a common parameter of exposure such as occupation or industry, exposure zone, task, or any combination of parameters (Stewart et al., 1986:34). Forms of this type of analysis were used by Stewart et al. in the Hill study.

The task-TWA model is used to develop historical exposure estimates based on full-shift exposures. This is done by developing a chronological matrix which details changes in practices, equipment, or anything that would result in a definable change in exposure. Coupled with limited monitoring data, this method yields very good results when used to determine TWA exposures from short term task data involving high exposure levels (Smith et al., 1991:441). Evidence of use of this type of approach is also found in the Hill study.

Exposure Modeling. Though methodologies and models are available which provide a means of retrospectively assessing occupational exposure, difficulty still exists largely due to lack of available historical exposure and industrial hygiene data existing prior to the mid-1970's (Stewart et al., 1986:34). For this reason, estimation of exposure by modeling of known physical phenomena is used as an aid to facilitate source-receptor conceptual modeling. The majority of these indoor air models are based on a basic mass continuity equation, which in its simplest form, equates the rate of increase of a contaminant in a defined volume to the difference between the rates which the contaminant enters and leaves the volume (Masters, 1991:238). The sophistication and complexity of each model is directly proportional to the degree to which the major factors affecting personal

exposure are addressed. The major factors which best describe personal exposure are individual activity patterns, characteristics of the model space (e.g. HVAC systems, filtration, infiltration, reactivity, cross drafts, room size and geometry, etc.), and source characteristics (e.g. emission rates, operating rates and methods, utilization rates, etc.) (Franke et al., 1990:765). Utilization of some of these factors can be seen in the mass balance equation used in the personal air quality model (PAQM) developed by Systems Applications, Inc. shown below (Hayes, 1989:1454):

$$V\frac{dC}{dt} = \underbrace{kQ_M(C_0 - C)}_{\text{Infiltration}} + \underbrace{kQ_F(E_F C_0 - C)}_{\text{Outdoor Makeup Air}} + \underbrace{kQ_R(E_R - 1)C}_{\text{Recirculation}} - \underbrace{kKAC}_{\substack{\text{Surface}\\ \text{Reactivity}\\ \text{Losses}}} + \underbrace{S(t)}_{\substack{\text{Indoor}\\ \text{Sources}}}$$

where:

C_0 = outdoor concentration

C = indoor concentration

V = building volume

Q_F = volumetric infiltration rate

Q_R = volumetric flow rate of recirculated air

Q_M = volumetric flow rate of makeup air

E_F = filter efficiency

E_R = efficiency of the recirculation

k = mixing factor

$S(t)$ = indoor source generation term

K = pollutant reactivity factor

A = interior surface area

Variations of this mass balance equation have been used in models developed by various other researchers such as Franke et al., McKone and Knezovich, Wadden et al., and Pedelty and Holcomb. For further reference, refer to the listing in the bibliography for each respective author.

Three of the more commonly used mass balance models are (1) the completely mixed reactor model, (2) the multi-point diffusion model, and (3) the multi-point diffusion model with advection (Franke et al., 1990:765). Model 1 assumes well-mixed conditions and is described by an equation of the form of that used in the PAQM model. Model 2 considers the interior space or volume to be a near infinite hemisphere or semi-hemisphere bounded by the dimensions of the space and centered at the emission source. Air movement is not affected by external influences, and is thus considered to be random. Under these circumstances, eddy diffusion is the primary dispersion mechanism which results in uniform decreasing of contaminant levels as the radial distance from the source is increased. Equation (1) describes this model and represents the solution to the mass balance on hemispherical space, ignoring surface deposition (Franke et al., 1990:766):

$$C = [S/(2\pi \times D \times r)]\text{erfc}[r/(4 \times D \times t)^{0.5}] \quad (1)$$

where:

C = concentration at any location (mass/volume)

r = radius of hemisphere at any time, t (length)

S = steady state emission rate of source (mass/time)

D = eddy diffusivity (area/time)

erfc = 1 - the error function

Given two simultaneously collected samples over the same averaging time, t_{av}, at two distances r_1 and r_2, the integral form of equation (1) yields two independent equations, (2) and (3), which can be solved simultaneously for D and S, thus allowing computation of the average area concentration (Wadden et al., 1989:4089):

$$C_{av,r1} = \int_0^{t_{av}} [S/(2\pi \times D \times r_1)]\text{erfc}[r_1/(4 \times D \times t)^{0.5}]dt/t_{av} \quad (2)$$

$$C_{av,r2} = \int_0^{t_{av}} [S/(2\pi \times D \times r_2)]\text{erfc}[r_2/(4 \times D \times t)^{0.5}]dt/t_{av} \quad (3)$$

The third model is most applicable when the interior air of the model volume is disturbed by external factors such as strong and sustained cross-drafts. When unidirectional advection disturbances of this sort are present, the equation describing model 3 exists in Gaussian plume form, which traditionally has been used in ambient air pollution modeling (Franke et al., 1990:766):

$$C = [S/(2\pi \times D \times x)][\exp(-u \times y^2/(4 \times D \times x))][\exp(-u \times z^2/(4 \times D \times x))] \quad (4)$$

where:

u = cross draft velocity in the x direction

Most situations can be described to some extent by one of the presented models above. In choosing a model, it is important the model closely describes the actual indoor environment being tested. Thus, it becomes just as important to

select exposure parameters which closely describe the indoor environment (Franke et al., 1990:767).

Traditionally, exposure parameters have been point estimators and represented "worst-case" or "typical" values which were one point on the distribution curve of that particular parameter (Whitmyre et al., 1992:298-301). However, the need to address the fact that workers and industrial environments are not constants and are more realistically described by stochastic means has resulted in the use of methods which address this variability. One of the more commonly used techniques is Monte Carlo simulation (Thompson et al., 1992:53-54). By using probability distribution functions instead of point estimators to describe the exposure model parameters, uncertainty and both individual and environmental variability are better addressed (Droz et al., 1989:447-448).

III. *Methodology*

Introduction

This section will briefly describe the manner in which the research objectives of this research were investigated. These objectives are defined within the investigative questions below:

1. Does sufficient information exist to model historical exposures found in the Hill AFB epidemiology study performed by Spirtas et al?
2. What type of model will best estimate this exposure?
3. What quantitative exposure levels result from applying such a model?

Objective 1. In order to determine whether sufficient information and data were available to model the historical exposures experienced by workers at Hill AFB, it was necessary to visit the base. During the seven day period I was at the base, I reviewed historical records of the Maintenance Directorate kept by the Base Historian, the Civil Engineering Flight, and the Bio-Environmental Engineering Flight, conducted a walk through of existing shops, and interviewed the industrial hygienist who performed the exposure assessments of the original study, Mr. D.E. Marano. Types of records included photographs, base year books, Bio-Environmental Engineering shop files, workers compensation claims, press reports, official correspondence, industrial hygiene surveys, and engineering drawings. Walk throughs of shops were conducted to get a visual understanding of the operations, processes, and work practices that took place within various shops. During these walk throughs I also talked to older employees to try to get

a firsthand account of how the working environment had changed during the time they had been employed there. Finally, the interview with Mr. Marano also included a review of his files which he had developed while performing his historical exposure assessment of the Hill AFB aircraft maintenance workers.

Objective 2. Using the historical monitoring data obtained, Monte Carlo simulation will be used to estimate TCE intake levels, TWA (time weighted average) exposure concentrations, cumulative exposure levels, and LCRs (lifetime cancer risks). This will be accomplished using a spreadsheet and @RISK, simulation and modeling software from Palisade Corporation.

Objective 3. Concentration levels in the immediate work area of vapor degreaser operators will be estimated using physical phenomena modeling. Because the exposure being modeled is based on a specific task performed by similar individuals, the source-receptor concept will be employed. Specifically, exposures zones will be determined and concentration levels within those zones will be modeled using the multi-point diffusion model described by equations (1), (2), and (3).

IV. *Analysis and Results*

Introduction

Presentation and discussion of the results of this research are presented in detail below.

Feasibility of Reconstructive Modeling

As noted by Stewart et al., I found there to be basically only two types of exposure to TCE which the aircraft maintenance workers were subjected to. The first occurred during the use of TCE as a cold state solvent where it was dispensed from containers or plastic bottles directly unto the surface of a part to be cleaned or unto a rag, which was then used to clean that part. Very little information and no exposure measurements were available for this type of exposure until the late 1970's. At this point however, the solvent used was 1,1,1 trichloroethane, as the use of TCE as a cold state solvent for most benchwork applications had ceased in 1968. The second type of exposure resulted from the use of vapor degreasers. Substantially more information was available relating to vapor degreaser operations. Monitoring data was available to various extents for each shop. Most reported levels were either instantaneous measurements taken with a Miran infrared analyzer or average concentration measurements collected over short time periods using pumps and collector tubes.

Because of the lack of information available regarding usage of TCE as a cold state solvent for benchwork combined with the finding that no trichloroethane exposure levels exceeding 60 ppm for benchwork were discovered, modeling of

this exposure route will not be pursued. However, sufficient information was available to attempt reconstruction of exposures resulting from usage of vapor degreasers. Specifically, information and monitoring pertaining to vapor degreasers found in the Wheels and Brakes Shop and Struts Shop was the most abundant. Information and monitoring data on other shops was available, however, it was very limited. Hence, the modeling of the exposure of vapor degreaser operators assigned to these shops is the only exposure which I believe can be modeled and substantiated.

Results. Very little monitoring data was found for shops other than the Struts and Wheels and Brakes shops. Prior to the mid to late 1970's, recorded monitoring data for individual shops seemed to be sporadic at best. The majority of the monitoring data obtained for the aforementioned shops was taken in 1978 and 1979 and was in response to the legal proceedings brought against the Air Force by Hill AFB employees and their union, the AFGE Local 1592. Though extensive monitoring was not performed prior to this time, an industrial hygiene program did exist. Annual surveys describing work practices and equipment conditions were undertaken with some regularity beginning in the early to mid sixties.

Shop Details. Both the Struts shop and the Wheels and Brakes shop were located in building 205 in 1954. The Wheels and Brakes shop remained there until 1980 when it was relocated to building 507, whereas the Struts shop was relocated to building 264 in 1964 and remained there through September 1979 when it was also moved to building 507. Neither building 264 nor 205 presently exists. Both buildings

were constructed of wood and masonry in the early 1940's. Similar in design (A-frame), each building resembled a small hangar. Descriptions from clinical records describe both buildings as having "a large open area with good natural ventilation." The description continues with "the doors and windows were kept closed in the winter months but vehicle entrances were opened periodically to allow vehicles to enter the building." The primary operation of the Struts shop was the disassembly, refurbishment, and rebuilding of aircraft struts, with the exception of C-5 struts, which were serviced by a separate C-5 only struts shop located in building 510. TCE vapor degreasers were used in two locations in the Struts shop - the magnaflux area and the parts buildup and testing area. The Wheels and Brakes shop had a similar mission as the Struts shop, only aircraft wheels and brakes as opposed to struts were cleaned, inspected, disassembled, and reassembled. TCE vapor degreasers were used specifically by workers in the magnaflux and brake and wheel disassembly sections of the shop. References to the duration and frequency of use of the tanks for both shops were found in clinical records and industrial hygiene surveys. These sources specified the degreasers were used for time periods ranging from twice daily for a total of thirty minutes to two hours per day.

Monitoring Data. All monitoring results found in the industrial hygiene files of both shops along with those of building 510 are listed in Appendix A. All breathing zone monitoring results for these shops are listed in Table 4. The remarks section of Table 4 consists of any information which was found with that specific monitoring result.

Table 4. TCE Breathing Zone Concentration Levels of Vapor Degreaser Workers.

Date	Building	Sample Location	Instrument Used	Conc. (ppm)	Remarks
09/22/65	264	NS	NS	100	
05/18/68	264	NS	NS	100	heated degreaser
05/18/68	264	NS	NS	250	
04/09/73	205	NS	NS	0.5	
09/25/75	264	60	Charcoal T.	145	
04/12/76	264	NS	NS	300	
04/27/76	205	NS	NS	5	
08/16/76	264	NS	NS	500	cross drafts, fans pulling vapors from tank
08/16/76	264	NS	NS	400	same as above, lid closed
12/21/77	205	NS	Draeger T.	500	door to E. of machine opened -cross drafts
12/21/77	205	NS	Draeger T.	350	
04/21/78	264	NS	NS	125	during spray lance use
04/26/78	264	NS	NS	450	during spray lance use
04/28/78	264	NS	NS	350	during spray lance use
05/18/78	264	NS	Draeger T.	75	
09/07/78	264	NS	NS	489	average while removing parts from VD
09/07/78	264	NS	NS	132	average while placing parts in VD
09/07/78	264	NS	NS	620	peak while placing parts in VD
09/07/78	264	NS	NS	0	low while placing parts in VD
09/07/78	264	NS	NS	1000	peak while removing parts from VD
09/07/78	264	NS	NS	25	low while removing parts from VD
09/07/78	264	NS	NS	9	from worker in magnaflux area
09/25/78	510	32	Charcoal T.	620	
09/25/78	510	30	Charcoal T.	470	
10/01/78	264	NS	Miran IR	100	removing parts from VD @ 11 fpm

Table 4 (continued). TCE Breathing Zone Concentration Levels of Vapor Degreaser Workers

Date	Building	Sample Location	Instrument Used	Conc. (ppm)	Remarks
10/01/78	264	NS	Miran IA	155	lowering parts into vapor layer of VD
10/01/78	264	NS	Miran IA	200	during spray lance use
11/06/78	510	255	Charcoal T.	29	
11/06/78	510	132	Charcoal T.	54	
01/05/79	510	NS	Miran-IA	300	inst. limit, during spray lance use
01/05/79	510	NS	Miran-IA	300	inst. limit, parts removed from tank
01/05/79	510	NS	Miran-IA	300	inst. limit, during spray lance use
01/05/79	510	NS	Miran-IA	180	Idle tank
01/05/79	510	NS	Miran-IA	180	idle tank
01/05/79	510	NS	Miran-IA	175	after 4 strut pistons lowered into tank
01/05/79	510	NS	Miran-IA	270	parts lowered into tank
03/08/79	510	25	NS	235	2' above tank while work performed
03/08/79	510	10	NS	200	2' above tank after work performed
03/08/79	510	20	NS	320	'2 above grated area on tank
03/29/79	264	120	NS	1	
03/29/79	205	NS	Miran-IA	80	5' above work platform
03/29/79	264	480	NS	13	
03/29/79	264	145	NS	2	
03/29/79	205	NS	Miran-IA	285	2' above tank opening
03/29/79	264	205	NS	15	
03/29/79	264	232	NS	33	
03/29/79	264	140	NS	45	
03/29/79	264	110	NS	37	
04/12/79	205	NS	NS	285	2' above tank
04/12/79	205	NS	NS	80	5' above tank
12/21/79	205	NS	NS	500	tank lid open
12/21/79	205	NS	NS	350	tank lid closed

The histogram shown in Figure 2 shows the relative frequencies of the breathing zone concentrations given in Table 4. Descriptive statistics are listed in Table 5.

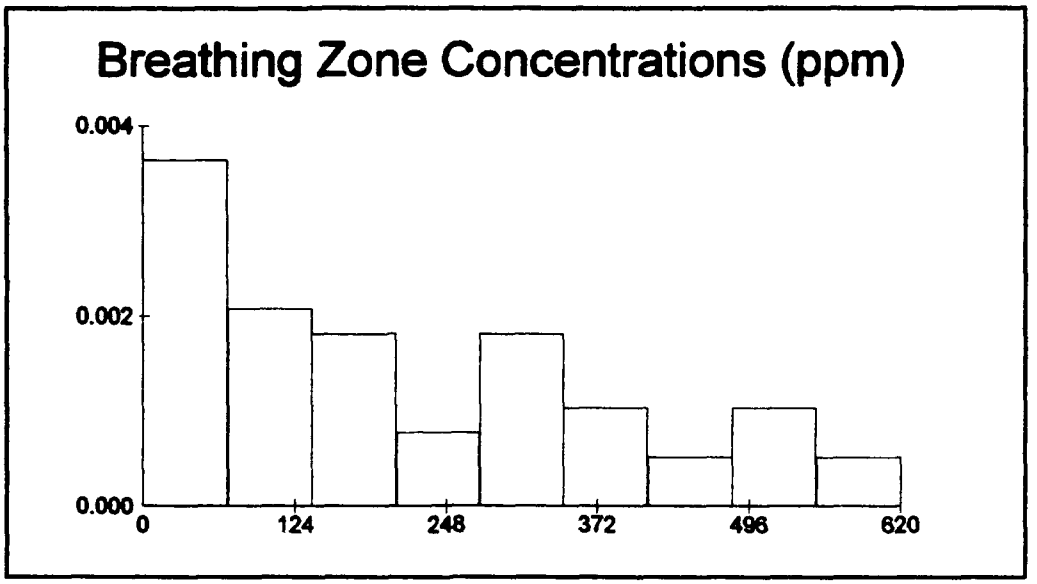


Figure 2. Distribution of Breathing Zone Concentration Data.

Table 5. Descriptive Statistics of Breathing Zone Concentration Data.

Statistic	Value
Mean	210.58
Std. Error of Mean	24.85
Standard Deviation	177.43
Median	180
Lower Limit of 95% Confidence Interval	160.67
Upper Limit of 95% Confidence Interval	260.48

The majority of the breathing zone samples were taken between 1978 and 1979. As a group, the samples represent all possible exposure scenarios (i.e. lowering parts into tank, removing parts from tank, idle tank, open tank, closed tank, spray lance use) associated with the use of the vapor degreasers. Only approximately 20 percent of the

samples were taken prior to 1978. However, during the time between 1954 and 1979, no major changes occurred in either shop's operations or the operation of other degreasers located on base which would suggest that this distribution would not be representative of exposure levels experienced throughout the entire period of concern. Reduction in exposure as a result of increased controls and equipment improvements did not become a major factor until both shop functions were relocated to new and more modern facilities beginning in late 1979. During this same time period, TCE was replaced by trichloroethane as the primary vapor degreaser solvent used (Stewart et al.,1991:534). Evidence supporting the premise that exposures over this 25 year time period were relatively the same manifests itself in work condition descriptions found in annual Bio-Environmental Engineering surveys and clinical records. References from these documents regarding ongoing ventilation and equipment problems (e.g. insufficient building makeup air, no local exhaust systems for degreasers, cross drafts removing vapors from degreasers) can be found dating well into the mid 1970's. More importantly, a pattern of worker behavior existing through the mid 1970's can be seen which indicates a lack of proper training in degreaser operations or simply an unwillingness to exercise proper procedures. The most common problems found in the majority of surveys were failure to close degreaser lids when not in use, failure to properly wear protective equipment, failure to use spray lance in full conjunction with the local exhaust system, and operation of basket hoists at excessive speeds. These problems were primarily supervisory and training related and existed until the mid to late 1970's when the industrial hygiene programs and recommendations of the

Bio-Environmental Shop began to be seriously implemented and followed by management and workers.

Summary. Workers performing vapor degreaser operations throughout the base performed the same type operations in similarly designed and equipped buildings. This suggests that mean exposures in such defined areas are dictated by similar exposure-modifying variables. When these variables are relatively constant, the mean of an exposure distribution would not be expected to change over time (Yu et al., 1990:194). Because evidence exists showing the major factors affecting exposure levels were existent in the latter half of the 25 years, it can be inferred that those same factors also existed during the first half of the 25 year period. As such, the distribution of breathing zone concentrations shown in Figure 2 is a valid representation of exposures experienced by personnel performing vapor degreaser operations and will be used as the input concentration distribution in the Monte Carlo simulation analysis.

Monte Carlo Simulation Analysis

Background. Estimating a historical exposure is difficult because of the lack of available information. Usually this forces us to make assumptions about the various parameters we are concerned with. When these assumptions are made using point estimators, the possibility exists that our assumptions may vary greatly from the parameter's true value. To compensate for this uncertainty, estimates are most often selected based on average, conservative, or worst-case values. This method has its limitations. Monte Carlo simulation is one means of avoiding the problems associated

with point estimators. Monte Carlo simply refers to the traditional method of sampling random variables in simulation modeling. The first step in the simulation is to choose probability distribution functions (PDFs) which describe the uncertain variables used in the simulation or model. These variables, thus, take on a range of values with known probability. Once all variables and constants to be used in the exposure model are defined, a computer program (@Risk by Palisade was used for all simulations in this research) is used to draw a random variable from each PDF used in the model. Modeled variables are then computed using the drawn random variables. This process is repeated a large number of times to produce complete distributions of the modeled variables.

Results. Three different probability distribution functions were fitted to breathing zone concentrations. The distributions used were chosen based on "goodness-of- fit." Specifically, the Weibull(0.96,275) distribution best approximated the breathing zone concentration data based on the chi-square test. The data was best fit by a normal(211,177) distribution when the measure of goodness-of-fit was the Anderson-Darling test. The triangle(0,0,620) distribution placed relatively high on both these tests. Graphs of these distributions as well as comparisons with the breathing zone concentration data are presented in Figures 3-8.

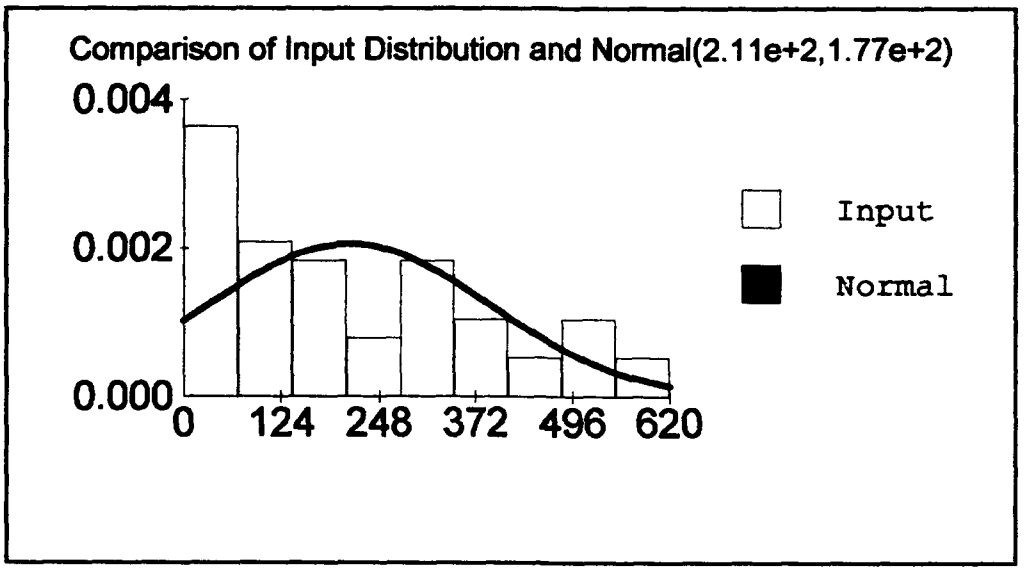


Figure 3. Normal Approximation of Breathing Zone Concentration Data.

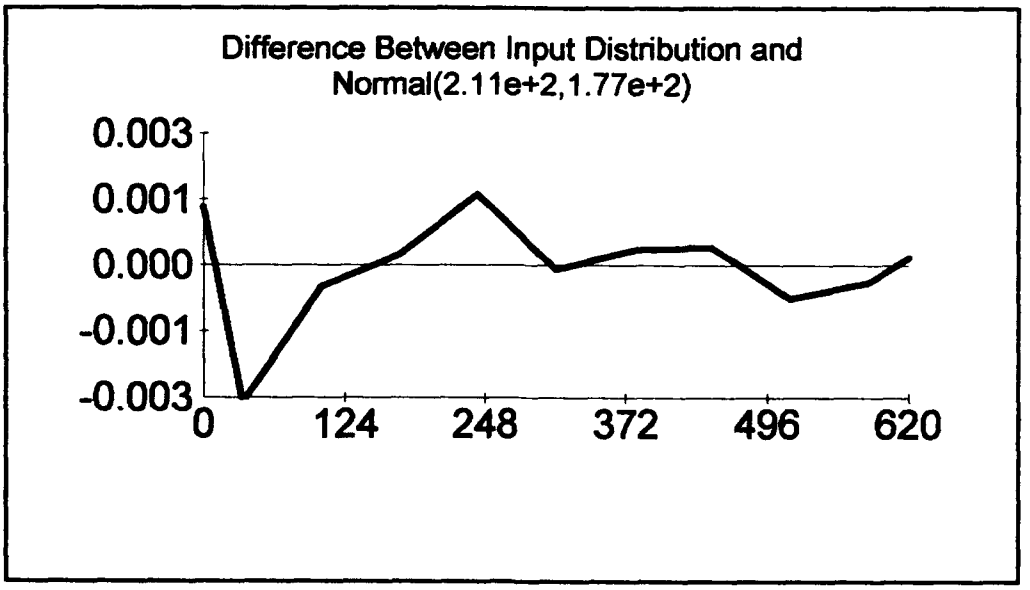


Figure 4. Difference in Normal Approximation and Breathing Zone Concentration Data

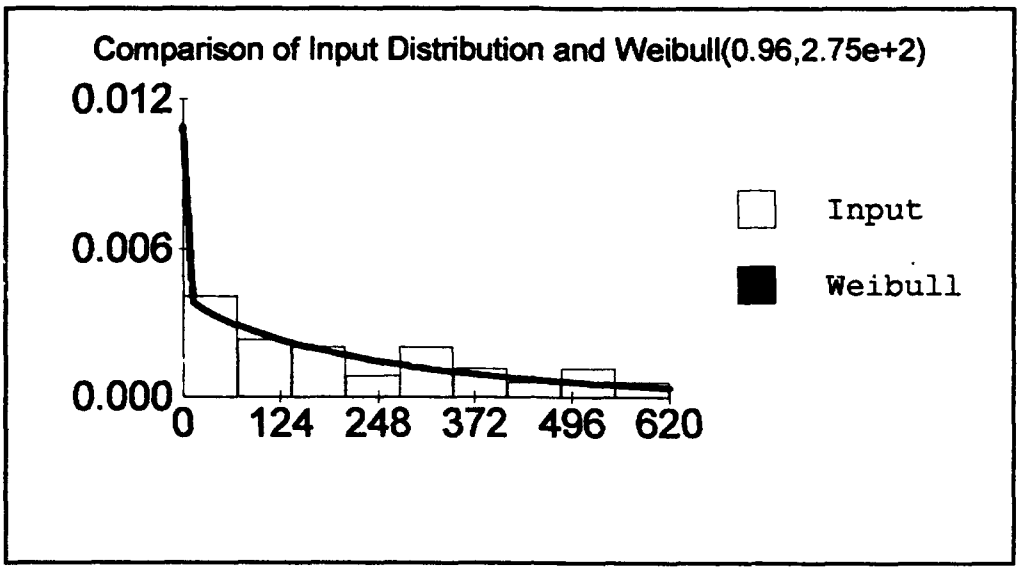


Figure 5. Weibull Approximation and Breathing Zone Concentration Data.

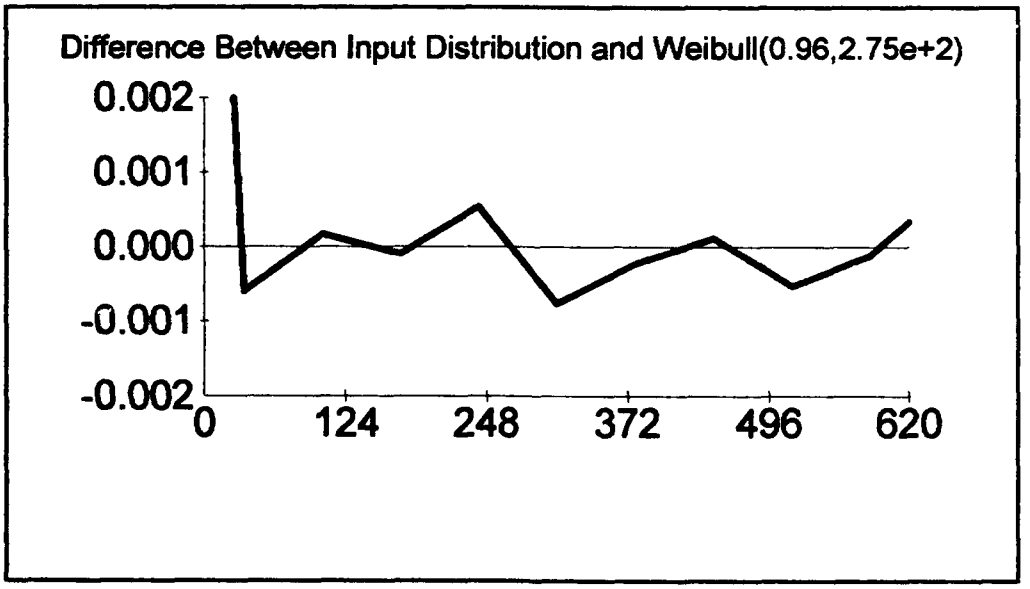


Figure 6. Difference in Weibull Approximation and Breathing Zone Concentration Data.

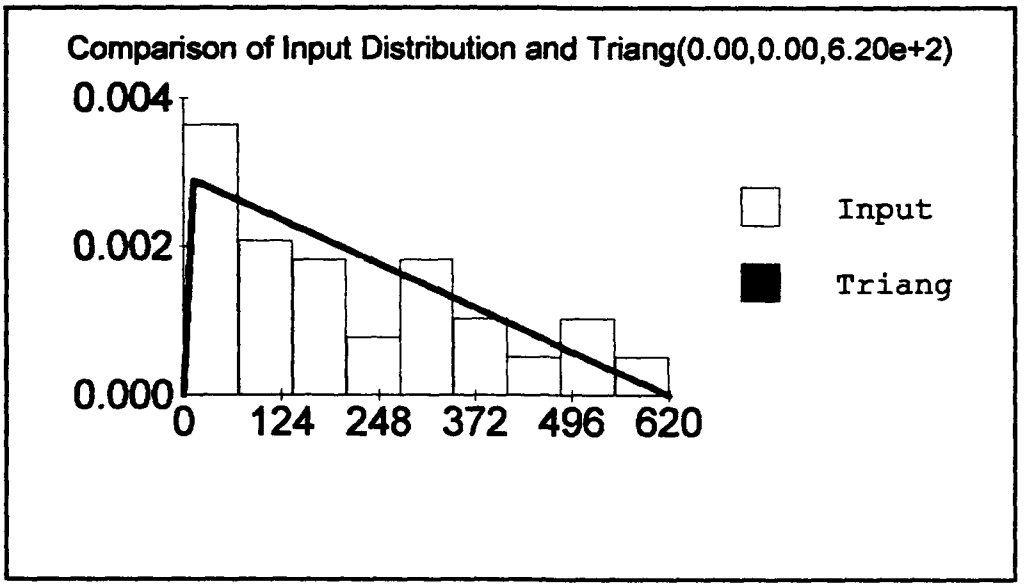


Figure 7. Difference in Triangle Approximation and Breathing Zone Concentration Data.

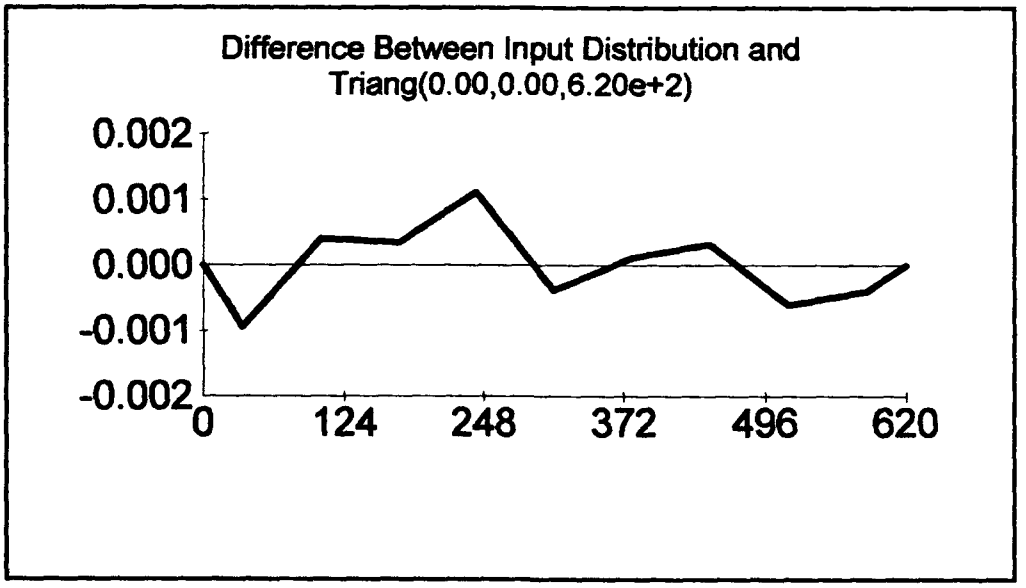


Figure 8. Difference in Triangle Approximation and Breathing Zone Concentration Data.

Neither the Weibull nor the triangle distribution approximated the breathing zone concentration data well at the tails of the distribution. As a result, neither was used as an estimation of the breathing zone concentration distribution. Instead, the breathing zone concentration was modeled using the normal distribution and the actual histogram of the breathing zone data. Table 6 contains distributions and parameters used in the Monte Carlo simulation of lifetime cancer risk (LCR), TWA, cumulative TWA, and TCE inhalation intake.

Table 6. Description of Model Parameters.

Variable	Distribution Description
Body Weight (BW) (lbs)	Lognormal2(5.13,0.17)[1]
Exposure Frequency (EF) (days/yr)	Triangle(240,250,260)
Exposure Time (ET) (min/day)	Uniform(30,120)
Exposure Duration (ED) (years)	Uniform(1,25)
Inhalation Rate (IR) (m^3/day)	20
Averaging Time (AT) (years)	70
TCE Cancer Slope Factor (SF) (mg/kg-day)$^{-1}$	1.17e-2

[1]Lognormal2 specifies a lognormal distribution where the entered mean and standard deviation equal the mean and standard deviation of the corresponding normal distribution. The arguments entered are the mean and standard deviation of the normal distribution for which an exponential of the values in the distribution was taken to generate the desired lognormal distribution.

Annual TWA concentration exposures were calculated on an 8 hour work day basis and are defined as the result of the exposure duration divided by 480 (number of

minutes in an 8 hour work day) times the exposure frequency. Figures 9 and 10 show the results of the simulation.

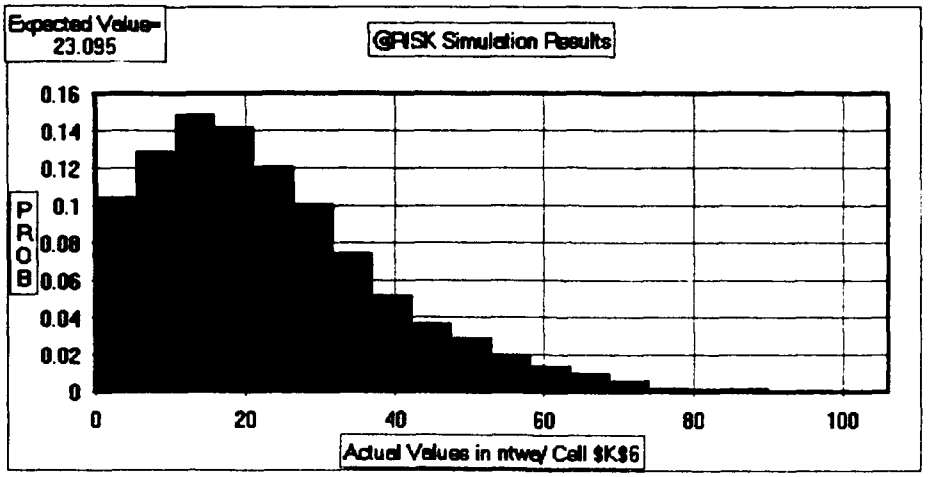


Figure 9. Annual TWA Concentration Exposure Calculated with Breathing Zone Concentrations Modeled Using the Normal Distribution.

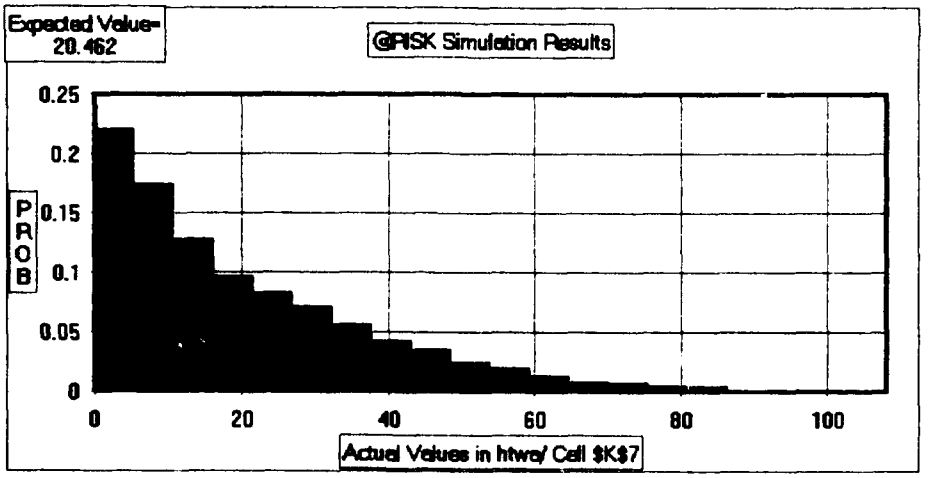


Figure 10. Annual TWA Concentration Exposure Calculated with Breathing Zone Concentrations Modeled Using the Histogram Generated by the Breathing Zone Concentration Data.

When the breathing zone concentration was modeled using the normal distribution, the mean annual TWA was 23.1 ppm. The 50th and 95th percentile values were 20.0 ppm and 54.8 ppm, respectively. When the distribution of the actual breathing zone concentration data was used, the mean annual TWA was 20.5 ppm. The 50th and 95th percentile values were 15.0 ppm and 57.4 ppm, respectively. Cumulative TWAs in which the cumulative integrated weighted exposure over the range of possible years a worker performing degreaser operations could have worked were calculated by summing the annual TWA exposures over the number of years worked. These results are shown in Figure 11.

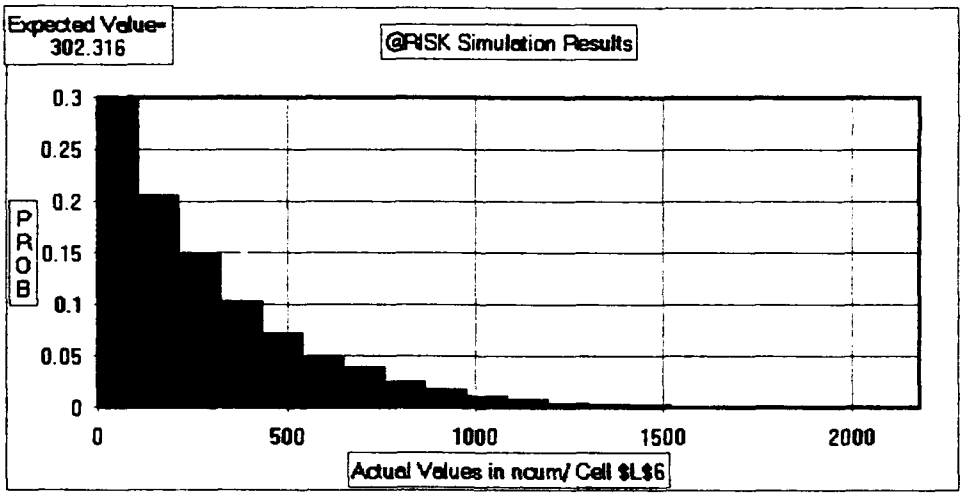


Figure 11. Cumulative TWA Concentration Exposure Calculated with Breathing Zone Concentrations Modeled Using the Normal Distribution.

The mean cumulative TWA exposure was 302 ppm. The 50th and 95th percentiles were 215 ppm and 892 ppm, respectively. Using the histogram of the actual breathing zone concentration data as the input to the model resulted in a mean cumulative TWA exposure of 265 ppm. The 50th and 95th percentile values were 158 ppm and 919 ppm,

respectively. The distribution of TWAs resulted from this simulation are shown in Figure 12. Higher values are representative of exposures experienced when a worker performed degreaser operations for the majority of the 25 years.

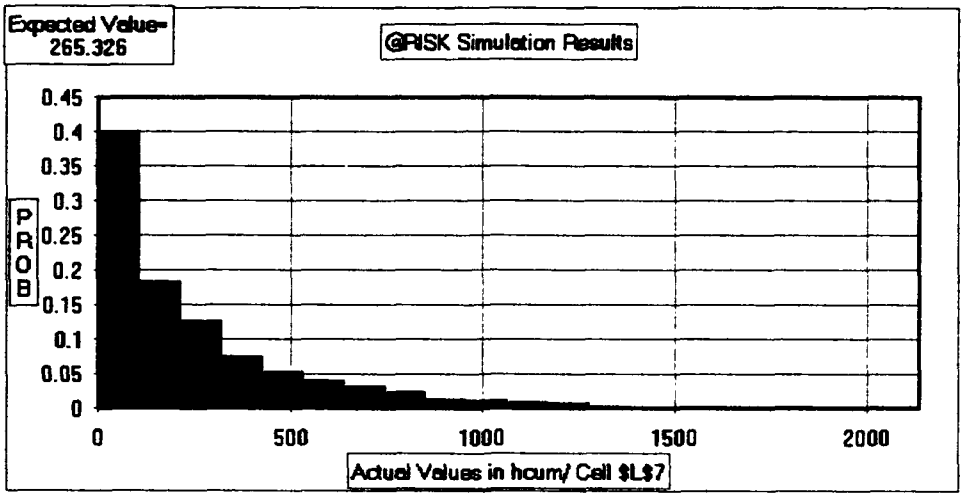


Figure 12. Cumulative TWA Concentration Exposure Calculated with Breathing Zone Concentrations Modeled Using the Histogram Generated by the Breathing Zone Concentration Data.

Daily inhalation intake amounts of TCE were calculated using equation (5).

$$Intake = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT} \qquad (5)$$

The distribution of the inhalation intake values are shown in Figures 13 and 14. Using the normal distribution as the input to the simulation, the mean value of TCE intake through inhalation was 6.7 mg/kg-day. The 50th and 95th percentile values were 4.7 mg/kg-day and 19.8 mg/kg-day, respectively. Using the histogram of the breathing zone

data as the concentration input resulted in a mean intake of 5.9 mg/kg-day. The 50th and 95th percentile values were 3.4 mg /kg-day and 20.4 mg/kg-day respectively.

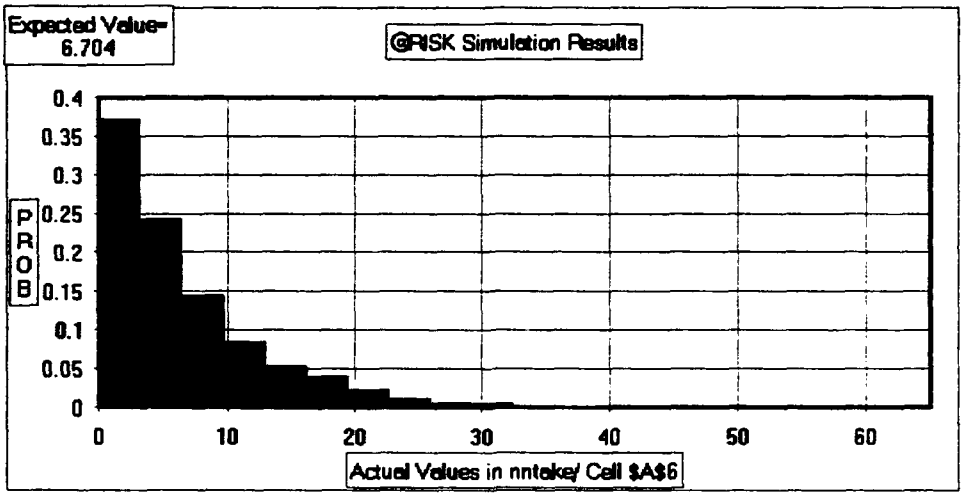


Figure 13. TCE Inhalation Intake (mg/kg-day) with Breathing Zone Concentrations Modeled Using the Normal Distribution.

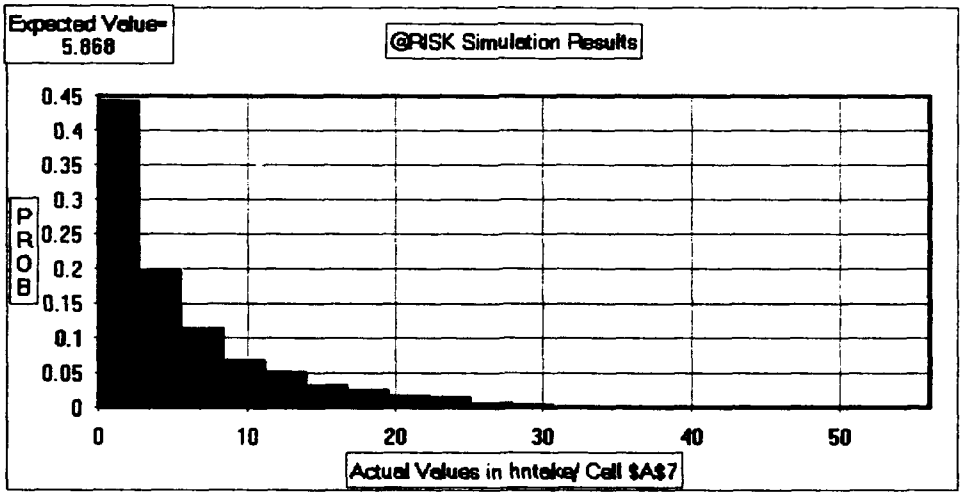


Figure 14. TCE Inhalation Intake (mg/kg-day) with Breathing Zone Concentrations Modeled Using the Histogram Generated from the Breathing Zone Concentration Data.

Multiplying the inhalation intake by the cancer slope factor for TCE produces a unitless probability which reflects the cancer risk associated with that dosage. Figures 15 and 16 show the risks and associated probabilities of that risk.

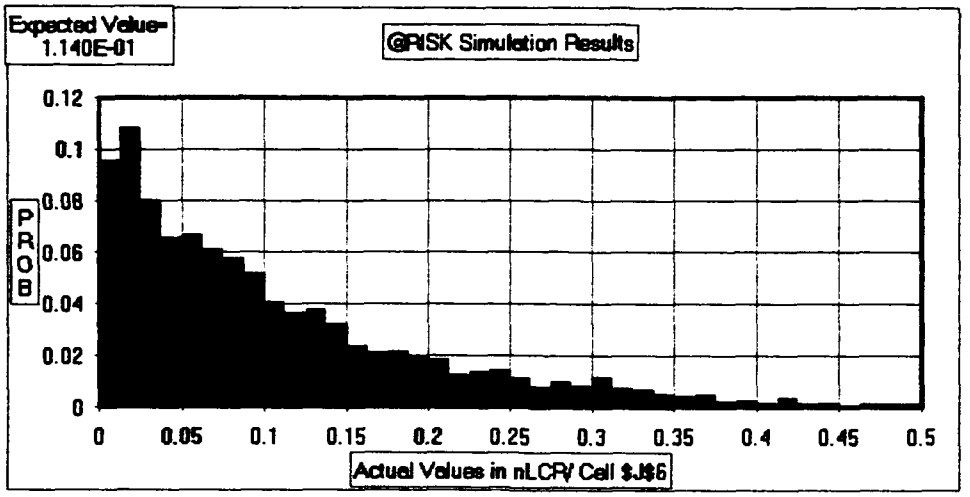


Figure 15. Cancer Risk with TCE Inhalation Intake (mg/kg-day) Calculated Using the Normal Distribution to Model Breathing Zone Concentrations.

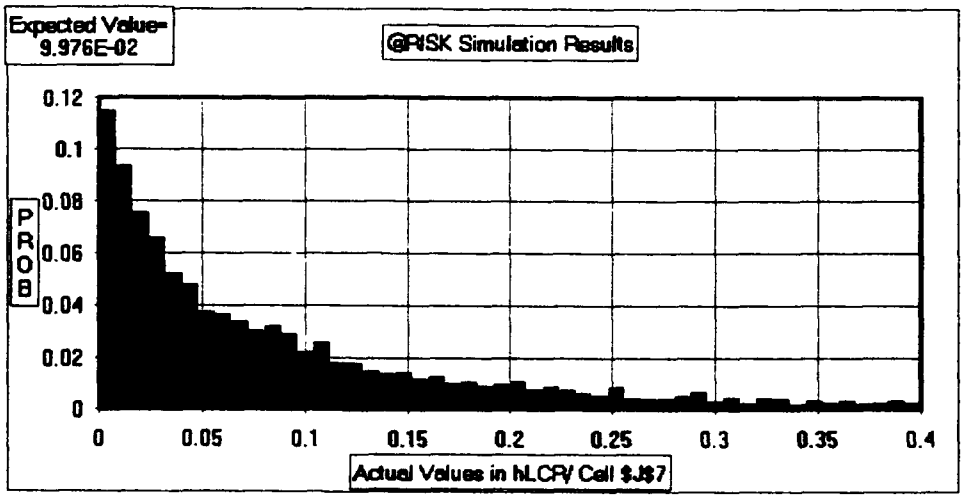


Figure 16. Cancer Risk with TCE Inhalation Intake (mg/kg-day) Calculated Using the Histogram of Breathing Zone Concentration Data.

The risks resulting from modeling the breathing zone concentration with a normal distribution fell between 3.6×10^{-5} and 1.0 with a mean value of 0.11. The 50th and 95th percentile values were 0.08 and 0.34, respectively. Using the histogram of the actual breathing zone concentration data resulted in a distribution with a maximum risk of 0.9 and a minimum risk of 2.7×10^{-6}. The 50th and 95th percentile values were 0.06 and 0.35, respectively. The LCRs produced are conditionally based on several variables and by no means imply that all possible uncertainties are accounted for. Uncertainties associated with the accuracy of the monitoring data, the population intake parameters, toxicity extrapolation techniques, and other factors prevent acceptance of the LCRs as being definitive. Instead, it is intended that the LCRs provide a reference from which to make comparisons and general inferences.

Summary statistics for all outputs discussed are presented in Table 7.

Table 7. Summary Statistics for Outputs of Monte Carlo Simulation of Exposure to Degreaser Breathing Zone Concentrations Only.

Parameter	Inhalation Intake		Lifetime Cancer Risk		Annual TWA Exposure		Cumulative TWA Exposure	
Distribution	Normal	Histogram	Normal	Histogram	Normal	Histogram	Normal	Histogram
Units	(m^3/day)	(m^3/day)			ppm	ppm	ppm	ppm
Minimum=	0	0	3.64E-05	2.66E-06	0.01	0	0.13	0.01
Maximum=	63.9	54.92	1.09	0.93	105.01	107.06	2175.45	2133.3
Mean=	6.7	5.87	0.11	0.1	23.1	20.46	302.32	265.33
Std Deviation=	6.64	6.75	0.11	0.11	16.09	18.27	288.83	297.84
Variance=	44.16	45.62	0.01	0.01	258.93	333.95	83420.42	88710.36
Skewness=	1.92	2.04	1.92	2.04	1.06	1.29	1.68	1.88
Kurtosis=	8.57	8.21	8.57	8.21	4.24	4.52	6.57	7.06
Percentile Values								
5Perc=	0.45	0.2	0.01	0	2.86	1.22	20	9.1

Table 7 (continued). Summary Statistics for Outputs of Monte Carlo Simulation of Exposure to Degreaser Breathing Zone Concentrations Only.

Parameter	Inhalation Intake		Lifetime Cancer Risk		Annual TWA Exposure		Cumulative TWA Exposure	
Distribution	Normal	Histogram	Normal	Histogram	Normal	Histogram	Normal	Histogram
Units	(m^3/day)	(m^3/day)			ppm	ppm	ppm	ppm
Percentile Values								
10Perc=	0.77	0.39	0.01	0.01	5.11	2.37	34.89	18.2
15Perc=	1.07	0.62	0.02	0.01	7.2	3.68	50.48	29.19
20Perc=	1.43	0.89	0.02	0.02	9.2	4.86	66.53	41.79
25Perc=	1.86	1.18	0.03	0.02	11.05	6.18	87.24	54.89
30Perc=	2.35	1.49	0.04	0.03	12.63	7.63	109.19	68.76
35Perc=	2.93	1.87	0.05	0.03	14.48	9.14	134.92	85.01
40Perc=	3.47	2.31	0.06	0.04	16.26	10.95	159.61	106
45Perc=	4.07	2.81	0.07	0.05	18.09	12.94	186.52	130.53
50Perc=	4.69	3.42	0.08	0.06	20.03	14.98	214.97	158.01
55Perc=	5.32	4.1	0.09	0.07	22.08	17.4	246.65	189.28
60Perc=	6.1	4.83	0.1	0.08	24.07	20.03	282.81	222.4
65Perc=	7.01	5.61	0.12	0.1	26.45	23.24	322.79	260.34
70Perc=	7.99	6.59	0.14	0.11	28.94	26.49	364.73	303.9
75Perc=	9.19	7.98	0.16	0.14	31.75	30.01	419.43	366.76
80Perc=	10.81	9.73	0.18	0.17	35.11	34.37	489.77	447.11
85Perc=	12.93	11.98	0.22	0.2	39.2	39.4	585.26	546.6
90Perc=	15.75	15.02	0.27	0.26	45.69	46.36	701.45	676.14
95Perc=	19.85	20.44	0.34	0.35	54.77	57.39	892.1	918.94

Multi-Point Diffusion Model Analysis.

Background. Cleaning of parts in vapor degreasers by workers was performed as a task in support of their primary job as a parts disassembler or assembler. From the review of the Hill AFB records, daily time spent degreasing parts varied from 30 minutes to 2 hours. The remainder of the day was spent away from the degreaser performing their primary job function. Drawings and descriptions show that the vapor

degreasers were generally located near the center of a work section. Once parts were degreased, they were taken to other area within the section where the overhauling process was continued. Shop drawings indicate the remainder of the work could have taken place up to 50 feet from the actual vapor degreaser.

Results. Applying the data found in Table 8 to equations (1), (2), and (3), emission factors were developed for two open top vapor degreasers. One was located in building 510, while the other was located in building 264.

Table 8. Two-Point Diffusion Model Parameters.

$C_{av,r1}$ (g/m^3)	$C_{av,r2}$ (g/m^3)	r_1 (meters	r_2 (meters	Bldg	Date	Description
1.03	0.15	0.43	1.3	510	01/05/79	after 4 strut pistons lowered into tank
1.76	0.16	0.43	1.3	510	01/05/79	during spray lance use
1.58	0.21	0.43	1.3	510	01/05/79	parts lowered into tank
1.76	0.21	0.43	1.3	510	01/05/79	during spray lance use
1.76	0.18	0.43	1.3	510	01/05/79	parts removed from tank
1.06	0.06	0.43	1.3	510	01/05/79	idle tank
1.38	0.15	0.61	1.83	510	03/08/79	heights above tank while work
1.17	0.06	0.61	4.57	510	03/08/79	while work performed
1.67	0.47	0.61	1.52	205	03/29/79	heights above tank opening
0.1	0	1.52	4.57	205	03/29/79	front and side of tank
0.44	0	0.43	3.69	264	04/28/78	NA

The averaging time used to calculate the emission factors was 0.25 minutes. Mathcad 4.0 for Windows was used to solve equations (2) and (3) simultaneously. The corresponding steady-state emission rate (S) and the eddy diffusivity (D) for each respective data pairing shown in Table 8 are tabulated in Table 9.

Table 9. Eddy Diffusivities and Steady-State Emissions Resulting from Application of the 2-Point Diffusion Model Using the Data in Table 8.

S (g TCE/min)	D (m^2/min)	Description
29.96	7.47	after 4 strut pistons lowered into tank
29.96	3.7	during spray lance use
41.21	6.47	parts lowered into tank
38.08	5.11	during spray lance use
32.1	4.07	parts removed from tank
12.42	2.13	idle tank
74.68	8.63	heights above tank while work performed
546.02	105.66	while work performed
307.66	38.03	heights above tank openings
54.72	1.66	front and side of tank
3.39	0.93	NA

Using the calculated eddy diffusivities and steady-state emission rates from Table 9, the predicted concentrations at distances ranging between 10 and 50 feet of the activity were calculated. The modeled concentrations are 30 minute average concentrations . Complete results are found in Table 10. Since the shops were designed such that the tear down and rebuilding of strut and brake parts occurred in work areas located away from the degreasing areas, predicted concentrations at these distances represent background levels a worker would experience in his work location resulting from another worker's performance of the activities described in Table 9. Using the diffusivity and emission rate values derived for the idle degreasing tank, daily ambient concentrations which would exist over an 8 hour workday were predicted. These results are also found in Table 10.

Table 10. Predicted Concentrations at Various Distances from Degreasing Activities Using the Calculated Eddy Diffusivities and Steady-State Emission Rates Presented in Table 9.

Description	Concentration (ppm) at Distance From Activity				
	10 ft	20 ft	30 ft	40 ft	50 ft
Averaging Time = 30 minutes					
after 4 strut pistons lowered into tank	28	11	6	3	2
during spray lance use	51	18	8	4	2
parts lowered into tank	44	17	9	5	3
during spray lance use	50	18	9	5	3
parts removed from tank	51	18	8	4	2
idle tank	33	10	4	2	1
heights above tank while work performed	62	25	13	8	5
while work performed	43	20	13	9	7
heights above tank openings	65	29	17	12	8
front and side of tank	175	50	18	7	3
NA	16	4	1	0	0
Averaging Time = 480 minutes					
idle tank	46	21	12	8	6

The steady-state emission rates derived from the monitoring data for various activities are not statistically significant because of the small sample size. They are, however, of the same magnitude as reported in the literature. Specifically, the emission rate for the idle degreaser described in Table 9 was calculated to be 12.42 g TCE/min. The open top area of the degreaser was 3.2 m^2. Dividing the emission rate by the open top area yields an emission factor of 3.9 g TCE/m^2-min. Wadden et al. reports the average emission factor for an open top degreaser to be 2.91 g TCE/m^2-min. This value represents 9.5% of the total degreaser emissions. The remainder of the emissions were

captured and removed by local exhaust systems. Thus, the total degreaser release was 30.94 g TCE/m^2-min. This value falls between 16.3 g TCE/m^2-min reported by Dow and 54.8 g TCE/m^2-min reported by the EPA for open top, uncontrolled, heated vapor degreasers. The agreement between the literature reported values and the calculated value as well as the fact that convergent solutions were found for equations (2) and (3) for all radius pairs suggests the model reflects the physical situation for the associated circumstances.

Using the background concentration modeling results in conjunction with the breathing zone concentration modeling results allows one to estimate the total exposure of a worker as a result of the combination of both exposures. In estimating the total exposure, the breathing zone concentrations were modeled using the normal distribution exactly as they were previously. The background concentrations which result from an idle tank, as given in Table 10, were used. The daily duration of exposure to the background concentration levels was determined by subtracting the degreaser breathing zone exposure duration time from eight hours (workday length). All other parameters were calculated in the same manner as they were previously and all variables defined in Table 6 remained the same.

The inhalation intake due solely to background concentration levels, as well as the total inhalation intake resulting from exposure to both the background and degreaser breathing zone concentration levels were modeled with the results presented in Tables 11 and 12. The annual TWA exposure and the LCR resulting from exposure to

both the background and degreaser breathing zone concentration levels were also modeled and are presented in Tables 13 and 14.

Table 11. Summary Statistics for Simulation of TCE Intake at Various Distances Resulting From Background Concentration Levels Only.

Parameter	Inhalation Intake Resulting From Background Concentrations				
Distribution	Normal				
Units	(m^3/day)				
Distance (feet)	10	20	30	40	50
Minimum=	0.31	0.09	0.04	0.02	0.01
Maximum=	16.46	4.99	1.99	1	0.5
Mean=	5.6	1.7	0.68	0.34	0.17
Std Deviation=	3.18	0.96	0.39	0.19	0.1
Variance=	10.14	0.93	0.15	0.04	0.01
Skewness=	0.34	0.34	0.34	0.34	0.34
Kurtosis=	2.38	2.38	2.38	2.38	2.38
Percentile Values					
5Perc=	0.94	0.28	0.11	0.06	0.03
10Perc=	1.42	0.43	0.17	0.09	0.04
15Perc=	1.91	0.58	0.23	0.12	0.06
20Perc=	2.46	0.74	0.3	0.15	0.07
25Perc=	3	0.91	0.36	0.18	0.09
30Perc=	3.5	1.06	0.42	0.21	0.11
35Perc=	4.01	1.21	0.49	0.24	0.12
40Perc=	4.5	1.36	0.54	0.27	0.14
45Perc=	4.92	1.49	0.6	0.3	0.15
50Perc=	5.39	1.63	0.65	0.33	0.16
55Perc=	5.91	1.79	0.72	0.36	0.18
60Perc=	6.32	1.92	0.77	0.38	0.19
65Perc=	6.82	2.07	0.83	0.41	0.21
70Perc=	7.39	2.24	0.9	0.45	0.22
75Perc=	7.91	2.4	0.96	0.48	0.24
80Perc=	8.46	2.56	1.03	0.51	0.26
85Perc=	9.13	2.77	1.11	0.55	0.28
90Perc=	10.08	3.06	1.22	0.61	0.31
95Perc=	11.1	3.36	1.35	0.67	0.34

Table 12. Summary Statistics for Simulation of Total TCE Intake at Various Distances Resulting From Both Background and Degreaser Breathing Zone Concentration Levels.

Parameter	Total Inhalation Intake Resulting From Exposure to Both Background and Degreaser Concentration Levels				
Distribution	Normal				
Units	(m^3/day)				
Distance (feet)	10	20	30	40	50
Minimum=	0.46	0.14	0.06	0.03	0.02
Maximum=	77.69	66.22	63.23	62.23	61.73
Mean=	12.41	8.51	7.49	7.15	6.98
Std Deviation=	8.96	7.36	6.99	6.88	6.82
Variance=	80.32	54.1	48.88	47.29	46.52
Skewness=	1.35	1.77	1.89	1.93	1.94
Kurtosis=	6.02	8.02	8.63	8.83	8.93
Percentile Values					
5Perc=	1.76	0.93	0.64	0.53	0.46
10Perc=	2.65	1.47	1.06	0.89	0.82
15Perc=	3.64	1.98	1.47	1.24	1.13
20Perc=	4.67	2.48	1.86	1.65	1.56
25Perc=	5.63	3.13	2.37	2.09	1.99
30Perc=	6.64	3.82	2.96	2.69	2.55
35Perc=	7.71	4.45	3.62	3.31	3.17
40Perc=	8.65	5.23	4.22	3.93	3.74
45Perc=	9.68	5.85	4.88	4.52	4.37
50Perc=	10.77	6.5	5.48	5.1	4.93
55Perc=	11.76	7.27	6.09	5.7	5.52
60Perc=	12.78	8.04	6.78	6.44	6.29
65Perc=	13.86	9.01	7.79	7.35	7.14
70Perc=	15.2	10.07	8.87	8.47	8.27
75Perc=	16.82	11.54	10.24	9.78	9.57
80Perc=	18.7	13.42	12.05	11.59	11.35
85Perc=	21.4	15.67	14.24	13.87	13.64
90Perc=	24.85	18.84	17.41	16.89	16.64
95Perc=	29.1	22.51	20.94	20.33	20.04

Table 13. Summary Statistics for Simulation of Annual TWA Exposure Due to Exposure to Both Background Concentrations at Various Distances and Degreaser Breathing Zone Concentrations.

Parameter	Annual TWA Exposure Resulting From Exposure to Both Background and Degreaser Concentration Levels				
Distribution	Normal				
Units	(ppm)				
Distance (feet)	10	20	30	40	50
Minimum=	20.31	6.67	2.87	1.51	0.77
Maximum=	183.36	168.14	164.16	162.84	162.18
Mean=	58.51	41.27	36.77	35.27	34.52
Std Deviation=	22.19	22.93	23.15	23.22	23.26
Variance=	492.31	525.96	535.78	539.15	540.85
Skewness=	0.96	0.98	0.98	0.98	0.98
Kurtosis=	4	3.98	3.96	3.96	3.96
Percentile Values					
5Perc=	29.93	11.86	7.08	5.48	4.69
10Perc=	33.4	15.39	10.72	9.15	8.34
15Perc=	36.39	18.35	13.58	12.02	11.21
20Perc=	39	21.24	16.52	14.96	14.15
25Perc=	41.91	23.86	19.14	17.53	16.73
30Perc=	44.29	26.04	21.33	19.75	18.94
35Perc=	46.78	28.83	24.1	22.5	21.7
40Perc=	49.08	31.57	26.99	25.46	24.65
45Perc=	51.83	34.24	29.63	28.13	27.36
50Perc=	54.36	37.05	32.52	30.93	30.21
55Perc=	57.38	40.31	35.79	34.23	33.47
60Perc=	60.52	43.38	38.71	37.25	36.51
65Perc=	63.51	46.41	42.1	40.59	39.83
70Perc=	66.85	49.75	45.48	44.11	43.38
75Perc=	71.16	54.15	49.85	48.34	47.58
80Perc=	75.7	58.99	54.78	53.31	52.59
85Perc=	81.58	64.61	60.32	58.96	58.2
90Perc=	89.97	73.3	68.98	67.47	66.73
95Perc=	101.22	85.23	81.27	79.99	79.36

Table 14. Summary Statistics for Simulation of Lifetime Cancer Risk Due to Exposure to Both Background Concentrations at Various Distances and Degreaser Breathing Zone Concentrations.

Parameter	Lifetime Cancer Risk Resulting From Exposure to Both Background and Degreaser Concentration Levels				
Distribution	Normal				
Units					
Distance (feet)	10	20	30	40	50
Minimum=	0.01	0	0	0	0
Maximum=	1.32	1.13	1.07	1.06	1.05
Mean=	0.21	0.14	0.13	0.12	0.12
Std Deviation=	0.15	0.13	0.12	0.12	0.12
Variance=	0.02	0.02	0.01	0.01	0.01
Skewness=	1.35	1.77	1.89	1.93	1.94
Kurtosis=	6.02	8.02	8.63	8.83	8.93
Percentile Values					
5Perc=	0.03	0.02	0.01	0.01	0.01
10Perc=	0.05	0.03	0.02	0.02	0.01
15Perc=	0.06	0.03	0.03	0.02	0.02
20Perc=	0.08	0.04	0.03	0.03	0.03
25Perc=	0.1	0.05	0.04	0.04	0.03
30Perc=	0.11	0.06	0.05	0.05	0.04
35Perc=	0.13	0.08	0.06	0.06	0.05
40Perc=	0.15	0.09	0.07	0.07	0.06
45Perc=	0.16	0.1	0.08	0.08	0.07
50Perc=	0.18	0.11	0.09	0.09	0.08
55Perc=	0.2	0.12	0.1	0.1	0.09
60Perc=	0.22	0.14	0.12	0.11	0.11
65Perc=	0.24	0.15	0.13	0.12	0.12
70Perc=	0.26	0.17	0.15	0.14	0.14
75Perc=	0.29	0.2	0.17	0.17	0.16
80Perc=	0.32	0.23	0.2	0.2	0.19
85Perc=	0.36	0.27	0.24	0.24	0.23
90Perc=	0.42	0.32	0.3	0.29	0.28
95Perc=	0.49	0.38	0.36	0.35	0.34

The distances at which workers performed their primary job of assembling, testing, or tearing-down aircraft parts ranged between approximately 20 and 60 feet. These are the distances at which they would have been exposed to the background concentrations caused by nearby degreasers. At these distances, the model results indicate that the exposure to background levels is relatively insignificant when compared with the exposure obtained from performing vapor degreasing operations. For comparison purposes, graphs of the LCR resulting from both exposures are presented in Figures 17 through 21.

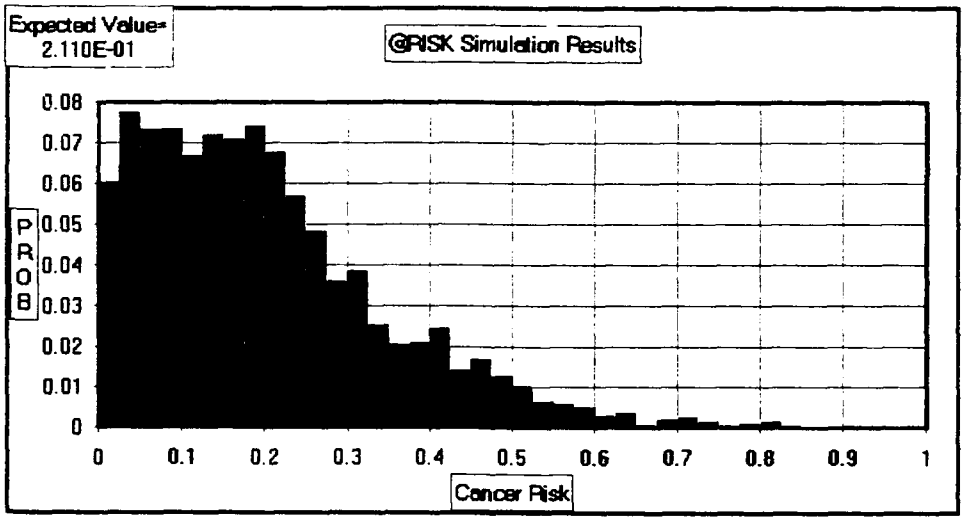


Figure 17. Lifetime Cancer Risk Due to Exposure to Both Background Concentrations at a Distance of 10 Feet From an Idle Tank and Degreaser Breathing Zone Concentrations.

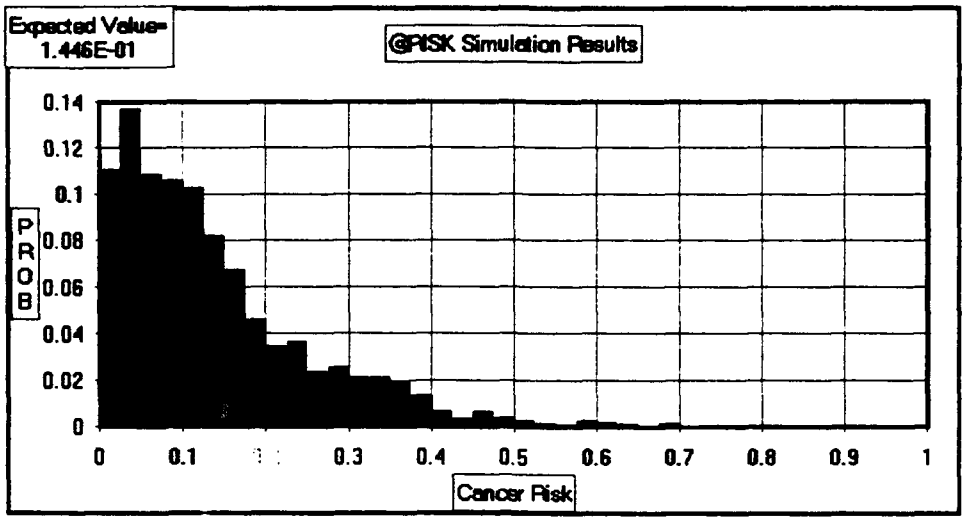


Figure 18. Lifetime Cancer Risk Due to Exposure to Both Background Concentrations at a Distance of 20 Feet From an Idle Tank and Degreaser Breathing Zone Concentrations.

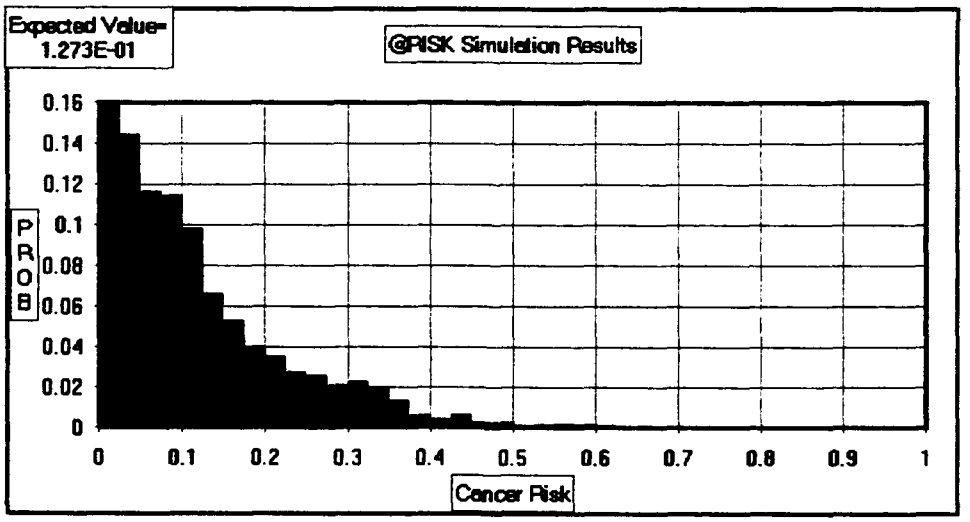


Figure 19. Lifetime Cancer Risk Due to Exposure to Both Background Concentrations at a Distance of 30 Feet From an Idle Tank and Degreaser Breathing Zone Concentrations.

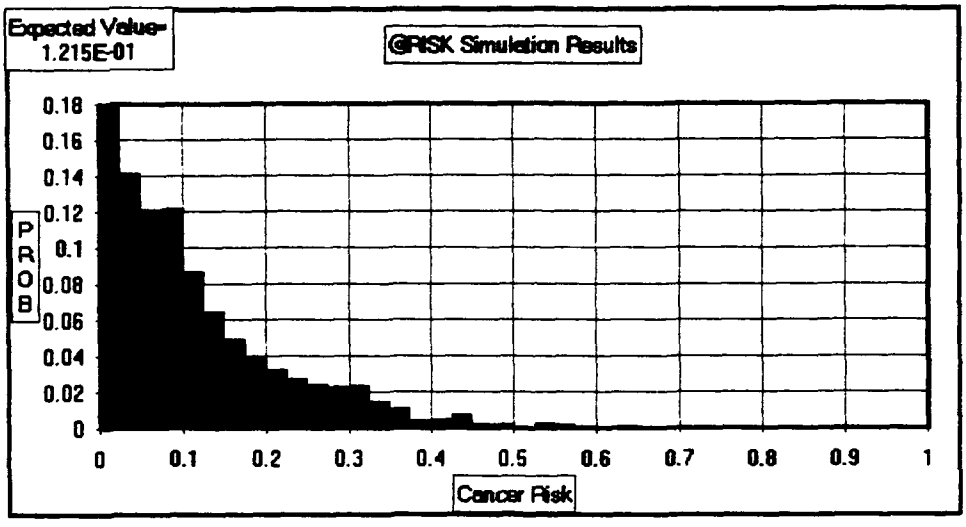


Figure 20. Lifetime Cancer Risk Due to Exposure to Both Background Concentrations at a Distance of 40 Feet From an Idle Tank and Degreaser Breathing Zone Concentrations.

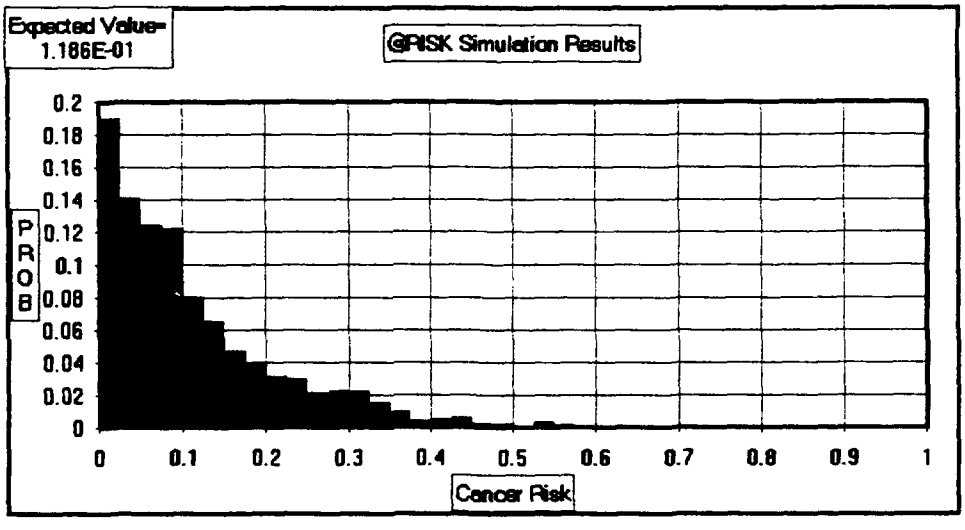


Figure 21. Lifetime Cancer Risk Due to Exposure to Both Background Concentrations at a Distance of 50 Feet From an Idle Tank and Degreaser Breathing Zone Concentrations.

V. Conclusion

The purpose of this research was to determine whether it was possible to quantitatively determine the exposure to trichloroethylene by aircraft workers employed at Hill Air Force Base, Ogden, Utah from 1955 through 1979. The interest in this particular cohort was due to an epidemiology study performed by Dr. R. Spirtas of the National Cancer Institute on the same cohort. One of the findings of the study was there existed no overall association between exposure to TCE and mortality. By better quantifying the exposures experienced by the cohort, an additional measure of validity could be applied to the study and its results. Consequently, there would be an addition to the weight of epidemiological evidence either supporting or refuting the current classification of TCE as a probable human carcinogen.

In order to determine the feasibility of modeling the exposure in question, it was necessary to visit Hill Air Force Base. Historical documents including photographs, industrial hygiene surveys, clinical records, shop drawings, and monitoring results were reviewed. Additionally, site surveys of existing facilities and conversations with current employees were made. Analysis of this information leads to the conclusion that the primary exposures to TCE at Hill AFB occurred as a result of vapor degreasing operations or its use as a cold state solvent for spot cleaning. Spot cleaning was performed primarily on benchtops on electrical, optical, and other components removed from the aircraft. It was also performed inside various sections of the aircraft such as the wings to clean fuel lines, electrical conductors, hydraulic lines, etc. Vapor degreasing was performed throughout the base in shops which overhauled major

components of the aircraft such as the struts, wheels, and brakes. The exposures experienced as a result of spot cleaning with cold state solvents were not monitored or recorded until the late 1970's. However, TCE had been replaced by trichloroethane as a cold state solvent in 1968. Conversely, results from monitoring of TCE vapor degreasers existed beginning in the mid to late 1960's. Until the late 1970's, monitoring efforts had been infrequent. The Struts shop and the Wheels and Brakes shops seemed to be the focus of vapor degreaser monitoring operations. As a result, data primarily pertaining to these shops was used to estimate the exposures which occurred over the study period.

Two methods were used to estimate the exposure of workers who performed vapor degreaser activities, Monte Carlo simulation and multi-point diffusion modeling. Monte Carlo simulation was used in an effort to account for some of the many uncertainties associated with estimating a historical exposure, especially when limited data is available. This method produces a more realistic representation of the actual ranges of exposure. The second method consisted of using monitoring data to estimate the steady-state emission rate of the degreasers. Using those calculated emission rates, concentrations at other locations within the work area were estimated.

The 50th and 95th percentile annual 8 hour TWA exposure levels for workers performing degreasing operations were 15 ppm and 57 ppm, respectively. Breathing zone concentrations while performing vapor degreasing operations ranged from 0 ppm to 620 ppm. The median value was 180 ppm and the mean was 210 ppm. Using the multi-point diffusion model, concentrations within 50 feet of the vapor degreaser were

estimated. The mean concentration at 10 feet was estimated at 56 ppm. At 20 feet the mean concentration was estimated to be 20 ppm. For a person working at a distance of 20 feet from an idle degreaser for six hours per day, with the remaining two hours spent cleaning parts at the vapor degreaser, the 8 hour TWA for this worker would be 41 ppm.

To put these estimates into perspective they should be compared with the regulatory guidelines. Through 1977 the threshold limit value (TLV) for TCE was 100 ppm. In 1982 it was lowered to 50 ppm. The OSHA permissible exposure levels (PEL) for TCE during this time were as follows:

100 ppm - 8 hour TWA

200 ppm - ceiling (5 minutes in any 2 hours)

300 ppm - peak

The 55th percentile of the vapor degreaser breathing zone data corresponds to the 200 ppm ceiling value. The 70th percentile value corresponds to the 300 ppm peak concentration level.

Model estimates indicate that the OSHA ceiling and peak limits were probably exceeded only slightly less often than they were adhered to. The 95th percentile of the annual 8 hour TWA is approximately half the OSHA 8 hour TWA. When the estimated background concentrations were factored into the TWA exposure, the majority of workers still did not exceed the OSHA limit. This occurred because the exposure due to background concentrations was relatively insignificant when compared to the exposure experienced during degreasing operations.

Lifetime cancer risks were calculated using the modeled exposures. The results indicate that the chances of a worker who performed vapor degreasing operations experiencing some form of cancer are one in ten. With a lifetime cancer risk of 0.1, one would have expected the incidences of cancer found by Spirtas et al. to have been much greater. The expected risks are factors of 3 to 5 times greater than the acceptable risks usually associated with occupational and residential exposure which are 1 in 10,000 and 1 in 1,000,000 respectively. Both occupational and residential exposure guidelines are established based on risks of this magnitude. The exposure of the workers were within the established regulatory guidelines which were designed to ensure those acceptable risk levels were not exceeded. The large difference between the expected cancer risk and the observed risk, coupled with the fact that the the MCL for TCE is approximately a factor of 2 smaller than the exposure levels experienced by the workers suggests that the MCL could be increased by at least a factor of 1 without exceeding already acceptable risk levels.

The major assumption of this research was that the monitoring data which was taken primarily from 1968-1979 was representative of the entire study period, 1955-1979. The assumption was made based on having analyzed the available data and concluding the major factors affecting concentration levels remained relatively consistent throughout the entire period. A study of 80 workers employed in 24 workshops belonging to10 different factories of the Swiss mechanical engineering industry was performed in 1954 to determine the effects of exposure to trichloroethylene. All factories used trichloroethylene in both open and closed tank

heated vapor degreasers. Factory average concentrations near the open tank degreasers ranged from 20 ppm to 325 ppm. The high end of this range was due to workers who ignored safety procedures and allowed parts to drip outside the tank. In the center of the workshop the resulting concentrations varied from 0 ppm to 225 ppm. This maximum was from the same factory and workshop as above. Concentrations near closed tanks varied from 11 ppm to 56 ppm. In the center of the workshop the resulting concentrations were all 0 ppm except for one factory where it was 35 ppm (Grandjean et al., 1955:131-133). The similarities of the concentrations measured in the Grandjean study with those found at Hill suggest that practices of that time were not that different than they were through the 1970's at Hill, thus supporting the major assumption of this research.

Appendix A: Tables

Table A.1 SMR's (Standardised Mortality Ratio's) for selected causes of death among white males by cumulative exposure to trichloroethylene (TCE) (Spirtas et al, 1990:46).

Cause of Death	< 5 years	5-25 years	>25 years	Total Exposure	Chi For Trend (1)
All Causes SMR	87**	88**	99	92*	2.48*
Observed/Expected	562/649.2	341/386.3	612/612.3	1508/1647.8	
All Cancer	94	87	94	92	0.12
	99/105.6	56/64.1	93/98.8	248/268.4	
Buccal/Pharynx	88	-	142	88	0.64
	0.87	0	1.43	0.88	
Billary Passages (2)	200	500*	106	236	-0.67
	2/1.0	5	1/1.0	2.4	
Primary Liver Cancer (2)	324	-	-	127	(3)
	3.33	0	0	1.25	
Pancreas	90	75	81	83	-0.18
	0.9	3/4.0	0.81	0.83	
Lung	96	88	107	98	0.42
	25/26.0	0.88	1.07	0.98	
Prostate	68	48	109	80	1.12
	0.68	0.48	12/11.0	0.8	
Testes	119	-	-	50	(3)
	1.25	0	0	0.5	
Kidney	191	-	124	120	-0.57
	1.92	0	1.25	1.19	
Bladder	142	177	107	136	-0.38
	1.43	1.76	1.07	1.37	
CNS	73	163	57	89	-0.19
	0.73	1.6	0.57	0.89	
All Lymph + Hemato.	73	61	119	87	1.26
	0.72	0.6	1.19	0.87	
Leukemia	58	-	124	68	1.34
	0.58	0	1.25	0.69	
Multiple Myeloma (2)	114	95	119	111	0.03
	1.11	0.91	1.18	1.11	

Cause of Death	< 5 years	5-25 years	>25 years	Total Exposure	Chi For Trend (1)
Non-Hodgkin's Lymph. (2)	128	129	57	102	-0.91
	1.28	1.3	0.57	1.02	
Ischemic HD	94	94	105	98	1.12
	0.94	0.94	1.05	0.98	
Emphysema	31**	90	131	83	2.82*
	0.31	0.88	1.32	0.83	
Asthma	129	423	250	244	0.33
	1.25	4	2.5	5/2.0	
Cirrhosis of Liver	47*	55	105	69	1.82
	0.47	0.55	1.06	0.69	
Nephritis	58	102	126	93	0.88
	0.59	2/2.0	1.25	0.93	

* Significant at 5% Level

(1) Derived from Chi-square test for linear trend (Breslow et al., 1983)
(2) Special categories created for this study
(3) Chi not computed if number of observed deaths < 5

Note:
1. Cumulative exposure categories were derived by cumulatively multiplying the exposure index assigned to each job by time exposed at that level. See Stewart et al. for a detailed explanation of the derivation.

Table A.2 SMR's for selected causes of death among white females by cumulative exposure to trichlorethylene (TCE) (Spirtas et al., 1990: 47).

Cause of Death	< 5 years	5-25 years	>25 years	Total Exposure	Chi For Trend (1)
All Causes SMR	78	60*	87	82*	0.9
Observed/Expected	35/44.7	18/29.9	133/152.5	186/227.2	
All Cancer	88	43	64	67*	-2.94*
	10/11.4	3/7.0	20/31.1	33/49.4	
Buccal/Pharynx	-	-	-	-	(3)
	0/0.1	0/0.1	0/0.4	0/0.6	
Billary Passages (2)	435	-	143	167	(3)
	1/0.2	0/0.2	1/0.7	2/1.2	
Primary Liver Cancer (2)	-	-	-	-	(3)
	0/0.0	0/0.0	0/0.1	0/0.2	
Pancreas	-	-	125	81	(3)
	0/0.5	0/0.3	2/1.6	2/2.5	
Lung	-	-	-	-	(3)
	0/0.7	0/0.4	0/1.7	0/2.8	
Breast	106	119	57	78	-0.89
	3/2.8	2/1.7	4/7.0	9/11.5	
Kidney	-	-	-	-	(3)
	0/0.2	0/0.1	0/0.5	0/0.8	
Bladder	-	-	265	183	(3)
	0/0.1	0/0.1	1/0.4	1/0.6	
CNS	-	-	-	-	(3)
	0/0.4	0/0.2	0/0.8	0/1.4	
All Lymph + Hemato.	277	-	125	143	-0.93
	3/1.1	0/0.7	4/3.1	7/4.8	
Leukemia	240	-	82	106	(3)
	1/0.4	0/0.3	1/1.2	2/1.9	
Multiple Myeloma (2)	602	-	-	130	(3)
	1/0.2	0/0.1	0/0.5	1/0.8	

Cause of Death	< 5 years	5-25 years	>25 years	Total Exposure	Chi For Trend (1)
Non-Hodgkin's Lymph. (2)	328	-	330	286	0.2
	1/0.3	0/0.2	3/0.9	4/1.4	
Ischemic HD	106	13*	99	90	0.43
	1/10.3	1/7.5	42/42.4	54/60.2	
Emphysema	-	-	314	198	(3)
	0/0.2	0/0.1	2/0.6	2/1.0	
Asthma	-	-	-	-	(3)
	0/0.1	0/0.1	0/0.3	0/0.5	
Cirrhosis of Liver	326	-	-	88	(3)
	3/0.9	0/0.5	0/2.0	3/3.4	
Nephritis	-	-	114	76	(3)
	0/0.3	0/0.2	1/0.9	1/1.3	

* Significant at 5% Level

(1) Derived from Chi-square test for linear trend (Breslow et al., 1983)
(2) Special categories created for this study
(3) Chi not computed if number of observed deaths < 5

Note:
1. Cumulative exposure categories were derived by cumulatively multiplying the exposure index assigned to each job by time exposed at that level. See Stewart et al. for a detailed explanation of the derivation.

Table A.3 Cause-Specific SMRs and confidence intervals adjusted for age and calender period of white males exposed to trichloroethylene (TCE) (Spirtas et al., 1990:44).

CAUSE OF DEATH	OBS	EXP	SMR (1)	95% CI Limits	
				Lower	Upper
All Causes of Death	1,508	1,647.8	92**	87	96
Tuberculosis	0	3.7	0*	0	99
All Malignant Neoplasms	248	268.5	92	81	105
Cancer of Buccal Cavity & Pharynx	5	5.7	88	29	206
Cancer of Digestive Organs & Peritoneum	74	74.7	99	78	124
Cancer of Esophagus	6	5.7	106	39	230
Cancer of Stomach	14	16	88	48	147
Cancer of Large Intestine	27	23.3	112	73	164
Cancer of Rectum	4	6.3	64	17	163
Cancer of Biliary Passages & Liver	8	4.1	196	85	386
Biliary Passages (2)	6	2.5	238	87	519
Liver, Primary (2)	2	1.6	---	---	---
Cancer of Pancreas	14	16.9	83	45	139
Cancer of All Other Digestive Organs	2	2.8	72	9	260
Cancer of Respiratory System	65	69.5	94	72	119
Cancer of Larynx	1	2.9	34	1	191
Cancer of Bronchus, Trachea, Lung	64	65.1	98	76	126
Cancer of All Other Respiratory	0	1.4	---	---	---
Cancer of Breast	0	0.4	---	---	---
All Uterine Cancers (Females Only)	---	---	---	---	---
Cancer of Cervix Uteri (Females Only)	---	---	---	---	---
Cancer of Other Female Genital Organs	---	---	---	---	---
Cancer of Prostate (Males Only)	22	27.6	80	50	121
Cancer of Testes and Other Male Genital	1	2	---	---	---
Cancer of Kidney & Other Urinary Organs	8	6.7	120	52	237
Cancer of Bladder	10	7.3	137	65	251
Malignant Melanoma of Skin	5	5.2	96	31	224
Cancer of Eye	0	0.5	---	---	---
Cancer of Central Nervous System	9	10.1	89	41	170
Cancer of Thyroid Gland & Other Endocrin	1	1.5	---	---	---
Cancer of Bone	3	1.1	263	54	767
Cancer of All Lymphatic & Haematopoietic	30	34.6	87	59	124

CAUSE OF DEATH	OBS	EXP	SMR (1)	95% CI Limits	
				Lower	Upper
Lymphosarcoma & Reticulosarcoma	9	8	112	51	213
Hodgkin's Disease	4	4.3	93	25	237
Leukemia & Aleukemia	9	13.1	69	31	130
Cancer of All Other Lymphopoietic Tissue	8	9.2	87	38	172
Multiple Myeloma (2)	5	4.5	111	36	259
Non-Hodgkin's Lymphoma (2)	10	9.8	103	49	189
All Other Malignant Neoplasms	15	21.2	71	40	117
Benign Neoplasms	5	4.3	117	38	273
Diabetes Mellitus	26	27.6	94	62	138
Cerebrovascular Disease	84	101	83	66	103
All Heart Disease	618	640.6	97	89	104
Rheumatic Heart Disease	34	33.4	102	71	142
Ischemic Heart Disease	551	561.7	98	90	107
Chronic Disease of Endocard.; Other Myocard	3	8	37	8	109
Hypertension with Heart Disease	5	6.8	73	24	171
All other Heart Disease	25	43	58**	38	86
Hypertension w/o Heart Disease	1	3.7	27	1	149
Non-malignant Respiratory Disease	106	118.3	88	72	107
Influenza & Pneumonia	28	39.5	68*	45	100
Bronchitis, Emphysema, Asthma	40	43.6	92	66	125
Bronchitis	7	8.4	84	34	172
Emphysema	28	33.6	83	55	121
Asthma	5	2.1	244	79	570
Other Non-malignant Respiratory Disease	37	36.9	100	71	138
Ulcer of Stomach & Duodenum	10	10.9	92	44	169
Cirrhosis of Liver	25	36.3	69	45	102
Nephritis & Nephrosis	8	8.6	93	40	184

CAUSE OF DEATH	OBS	EXP	SMR (1)	95% CI Limits	
				Lower	Upper
All External Causes of Death	128	205.6	62**	52	74
Accidents	79	141.1	56**	44	70
Motor Vehicle Accidents	33	61.2	54**	37	76
All Other Accidents	46	79.7	58**	42	77
Suicides	41	45.5	90	65	122
Homicides & Other External Causes	8	10	80	35	158
All Other Causes of Death	161	216.5	74**	63	87
Unknown Causes (999.9)	93				

* Significant at 5% Level

** Significant at 1% Level

(1) SMR's and CI's presented only if number of observed or expected deaths > 2

(2) Special categories created for this study

Table A.4 Cause-Specific SMRs and confidence intervals adjusted for age and calender period of white females exposed to trichloroethylene (TCE) (Spirtas et al., 1990:45).

CAUSE OF DEATH	OBS	EXP	SMR (1)	95% CI Limits	
				Lower	Upper
All Causes of Death	186	227.2	82**	71	95
Tuberculosis	0	0.2	---	---	---
All Malignant Neoplasms	33	49.4	67*	46	94
Cancer of Buccal Cavity & Pharynx	0	0.6	---	---	---
Cancer of Digestive Organs & Peritoneum	7	13	54	22	111
Cancer of Esophagus	0	0.2	---	---	---
Cancer of Stomach	0	2	---	---	---
Cancer of Large Intestine	2	5.5	37	4	132
Cancer of Rectum	1	1.1	---	---	---
Cancer of Biliary Passages & Liver	2	1.2	---	---	---
Biliary Passages (2)	2	1.1	---	---	---
Liver, Primary (2)	0	0.2	---	---	---
Cancer of Pancreas	2	2.5	81	10	291
Cancer of All Other Digestive Organs	0	0.5	---	---	---
Cancer of Respiratory System	0	3.1	0	0	121
Cancer of Larynx	0	0.1	---	---	---
Cancer of Bronchus, Trachea, Lung	0	2.8	0	0	131
Cancer of All Other Respiratory	0	0.2	---	---	---
Cancer of Breast	9	11.5	79	36	149
All Uterine Cancers (Females Only)	4	4.1	98	27	251
Cancer of Cervix Uteri (Females Only)	4	1.8	224	61	574
Cancer of Other Female Genital Organs	4	4	100	27	255
Cancer of Prostate (Males Only)	-	---	---	---	---
Cancer of Testes and Other Male Genital	-	---	---	---	---
Cancer of Kidney & Other Urinary Organs	0	0.8	---	---	---
Cancer of Bladder	1	0.6	---	---	---
Malignant Melanoma of Skin	1	0.6	---	---	---
Cancer of Eye	0	0.1	---	---	---
Cancer of Central Nervous System	0	1.4	---	---	---
Cancer of Thyroid Gland & Other Endocrin	0	0.4	---	---	---
Cancer of Bone	0	0.1	---	---	---
Cancer of All Lymphatic & Haematopoietic	7	4.9	143	58	295

CAUSE OF DEATH	OBS	EXP	SMR (1)	95% CI Limits	
				Lower	Upper
Lymphosarcoma & Reticulosarcoma	3	1.2	261	54	761
Hodgkin's Disease	0	0.3	---	---	---
Leukemia & Aleukemia	2	1.9	---	---	---
Cancer of All Other Lymphopoietic Tissue	2	1.5	---	---	---
Multiple Myeloma (2)	1	0.8	---	---	---
Non-Hodgkin's Lymphoma (2)	4	1.4	286	78	731
All Other Malignant Neoplasms	0	4.2	0*	0	86
Benign Neoplasms	2	0.8	---	---	---
Diabetes Mellitus	6	6.8	88	32	191
Cerebrovascular Disease	22	26.6	83	52	125
All Heart Disease	70	78.7	89	69	112
Rheumatic Heart Disease	8	8.6	93	40	184
Ischemic Heart Disease	54	60.2	90	67	117
Chronic Disease of Endocard.; Other Myocard	1	1.5	---	---	---
Hypertension with Heart Disease	1	2	---	---	---
All other Heart Disease	6	7.4	82	30	178
Hypertension w/o Heart Disease	0	0.7	---	---	---
Non-malignant Respiratory Disease	4	10.6	38*	10	97
Influenza & Pneumonia	0	6.3	0**	0	59
Bronchitis, Emphysema, Asthma	2	2.1	95	12	343
Bronchitis	0	0.5	---	---	---
Emphysema	2	1	---	---	---
Asthma	0	0.5	---	---	---
Other Non-malignant Respiratory Disease	2	2.5	80	10	287
Ulcer of Stomach & Duodenum	0	1.1	---	---	---
Cirrhosis of Liver	3	3.4	88	18	257
Nephritis & Nephrosis	1	1.3	---	---	---

CAUSE OF DEATH	OBS	EXP	SMR (1)	95% CI Limits	
				Lower	Upper
All External Causes of Death	9	13	69	32	131
Accidents	7	9.8	71	29	147
Motor Vehicle Accidents	5	4.7	106	34	247
All Other Accidents	2	5.2	39	5	140
Suicides	2	2	99	12	357
Homicides & Other External Causes	0	0.7	---	---	---
All Other Causes of Death	17	34.2	50**	29	80
Unknown Causes (999.9)	20				

* Significant at 5% Level

** Significant at 1% Level

(1) SMR's and CI's presented only if number of observed or expected deaths > 2

(2) Special categories created for this study

Table A.5 Recorded TCE Exposure Levels from Vapor Degreasing Activities

Date	Building	Sample Location	Sample Duration (min)	Instrument Used	Conc. (ppm)	Remarks
09/22/65	264	BZ	NS	NS	100	20
05/18/68	264	BZ	NS	NS	100	32
05/18/68	264	BZ	NS	NS	250	cold dip tank
04/09/73	205	BZ	NS	NS	0.5	
09/25/75	264	BZ	60	Charcoal T.	145	
11/03/75	510	NS	NS	Draeger T.	75	1' above edge of tank
11/03/75	510	NS	NS	Draeger T.	75	middle of tank
04/12/76	265	BZ	NS	NS	300	
04/27/76	205	BZ	NS	NS	5	
08/16/76	264	BZ	NS	NS	500	cross drafts, fans pulling vapors from tank
08/16/76	264	BZ	NS	NS	400	same as above, lid closed
06/14/77	264	NS	NS	Draeger T.	100	
12/21/77	205	BZ	NS	Draeger T.	500	door to E. of machine opened -cross drafts
12/21/77	205	BZ	NS	Draeger T.	350	
04/21/78	264	BZ	NS	NS	125	during spray lance use
04/21/78	264	BG,7.5	NS	NS	200	during spray lance use
04/26/78	264	BZ	NS	NS	450	during spray lance use
04/26/78	264	BG,20	NS	NS	45	during spray lance use
04/28/78	264	BZ	NS	NS	350	during spray lance use
04/28/78	264	BG, NS	NS	NS	0	during lance spray use
05/18/78	264	BG,10	NS	Draeger T.	0	
05/18/78	264	BZ	NS	Draeger T.	75	
09/07/78	264	BZ	NS	NS	0	low while placing parts in VD
09/07/78	264	BZ	NS	NS	25	low while removing parts from VD
09/07/78	264	Source	NS	NS	38	source sample at breathing level of VD
09/07/78	264	BZ	NS	NS	132	average while placing parts in VD
09/07/78	264	BZ	NS	NS	620	peak while placing parts in VD

Date	Building	Sample Location	Sample Duration (min)	Instrument Used	Conc. (ppm)	Remarks
09/07/78	264	BZ	NS	NS	1,000	peak while removing parts from VD
09/07/78	264	BZ	NS	NS	9	from worker in magnaflux area
09/07/78	264	BZ	NS	NS	489	average while removing parts from VD
09/25/78	510	BZ	32	Charcoal T.	620	
09/25/78	510	BZ	30	Charcoal T.	470	
10/01/78	264	BZ	NS	Miran IR	155	lowering parts into vapor layer of VD
10/01/78	264	BZ	NS	Miran IR	200	during spray lance use
10/01/78	264	BZ	NS	Miran IR	100	removing parts from VD @ 11 fpm
11/06/78	510	BZ	255	Charcoal T.	29	
11/06/78	510	GRA	130	Charcoal T.	25	
11/06/78	510	GRA	233	Charcoal T.	12	
11/06/78	510	GRA	356	Charcoal T.	7	
11/06/78	510	BZ	132	Charcoal T.	54	
01/05/79	510	BG,2	NS	Miran-1A	25	after 4 strut pistons lowered into tank
01/05/79	510	BZ	NS	Miran-1A	175	after 4 strut pistons lowered into tank
01/05/79	510	BG, 2	NS	Miran-1A	28	during spray lance use
01/05/79	510	BZ	NS	Miran-1A	300	inst. limit, during spray lance use
01/05/79	510	BZ	NS	Miran-1A	270	parts lowered into tank
01/05/79	510	BG,2	NS	Miran-1A	36	parts lowered into tank
01/05/79	510	BZ	NS	Miran-1A	300	inst. limit, during spray lance use
01/05/79	510	BG, 2	NS	Miran-1A	35	During spray lance use
01/05/79	510	BZ	NS	Miran-1A	300	inst. limit, parts removed from tank
01/05/79	510	BG, 2	NS	Miran-1A	30	parts removed from tank
01/05/79	510	BZ	NS	Miran-1A	180	Idle tank
01/05/79	510	BG, 2	NS	Miran-1A	10	idle tank
01/05/79	510	BG,2	NS	Miran-1A	10	inst. limit, idle tank, lid closed
01/05/79	510	BG,2	NS	Miran-1A	300	inst. limit, tank lid opened rapidly
03/08/79	510	BZ	25	NS	235	2' above tank while work performed

Date	Building	Sample Location	Sample Duration (min)	Instrument Used	Conc. (ppm)	Remarks
03/08/79	510	NS	12	NS	25	6' above work platform while work performed
03/08/79	510	BZ	10	NS	200	2' above tank after work performed
03/08/79	510	BG,15	15	NS	10	average less than 10 ppm
03/08/79	510	BZ	20	NS	320	'2 above grated area on tank
03/29/79	205	BG,5	NS	Miran-1A	35	north side of tank
03/29/79	264	GRA	155	NS	11	
03/29/79	205	BZ	NS	Miran-1A	285	2' above tank opening
03/29/79	205	BZ	NS	Miran-1A	80	5' above tank opening
03/29/79	205	BG,25	NS	Miran-1A	0	north side of tank
03/29/79	205	BG,15	NS	Miran-1A	0	south side of tank
03/29/79	205	BG,5	NS	Miran-1A	17	front of tank
03/29/79	264	BZ	232	NS	33	
03/29/79	264	BZ	480	NS	13	
03/29/79	205	BG,10	NS	Miran-1A	0	10' from front of tank
03/29/79	205	BG,5	NS	Miran-1A	0	south side of tank
03/29/79	264	BZ	110	NS	37	
03/29/79	264	BZ	140	NS	45	
03/29/79	264	BZ	205	NS	15	
03/29/79	264	BZ	145	NS	2	
03/29/79	264	BZ	120	NS	1	

Date	Building	Sample Location	Sample Duration (min)	Instrument Used	Conc. (ppm)	Remarks
04/12/79	205	BZ	NS	NS	285	2' above tank
04/12/79	205	GRA	NS	NS	0	
04/12/79	205	GRA	NS	NS	0	
04/12/79	205	GRA	NS	NS	0	
04/12/79	205	GRA	NS	NS	0	
04/12/79	205	GRA	NS	NS	17	
04/12/79	205	BZ	NS	NS	80	5' above tank
12/21/79	205	BZ	NS	NS	350	tank lid closed
12/21/79	205	BZ	NS	NS	500	tank lid open

Notes:
1. Draeger T. - Draeger Pump and Collector Tube
2. Charcoal T. - Dupont Pump and Charcoad Tube
3. GRA - General Room Air
4. BG,xx - Background sample taken at xx lateral feet from nearest side of tank
5. NS - Not Specified
6. A blank remarks sections indicates there was no additonal information available describing the sample.

Appendix B: Photographs

Photo 1: Hill Air Force Base, B264 (side), date not available.

Photo 2: Hill Air Force Base, B264 (side), date not available.

Photo 3: Hill Air Force Base, B264 (front), date not available.

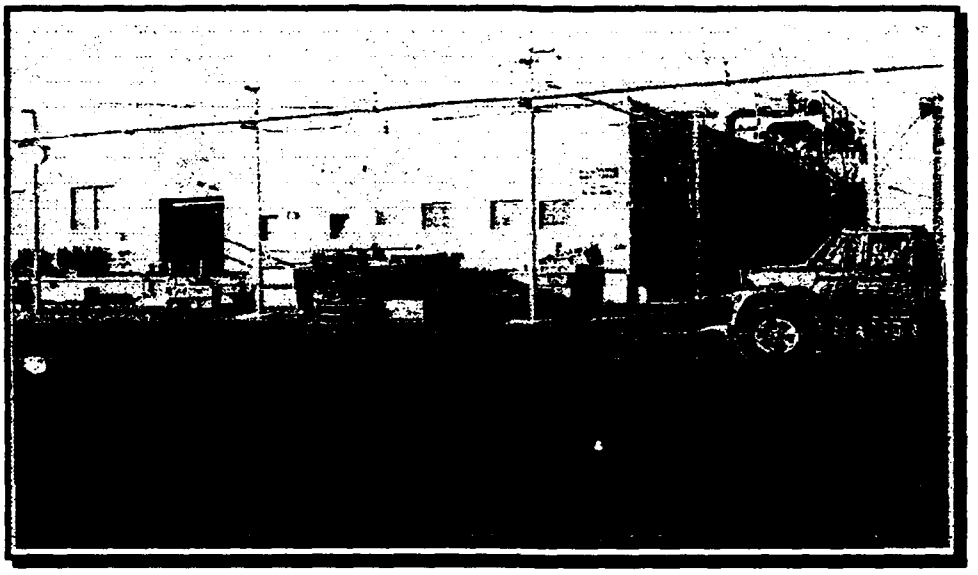


Photo 4: Hill Air Force Base, B264 (Aluminum parts cleaning line), date not available.

Photo 5: Hill Air Force Base, B205 (rear storage area), no date available.

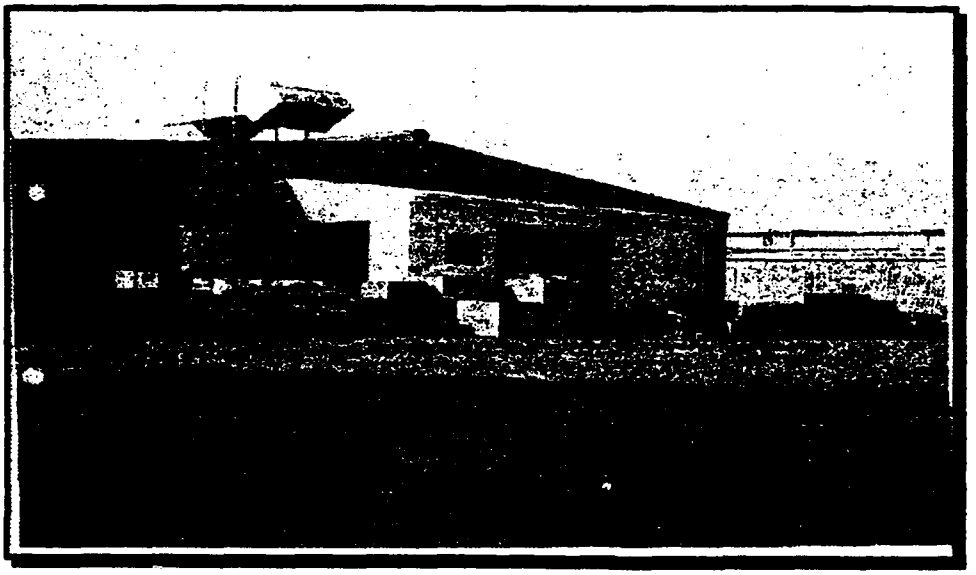


Photo 6: Hill Air Force Base, B205 (parts cleaning line), no date available.

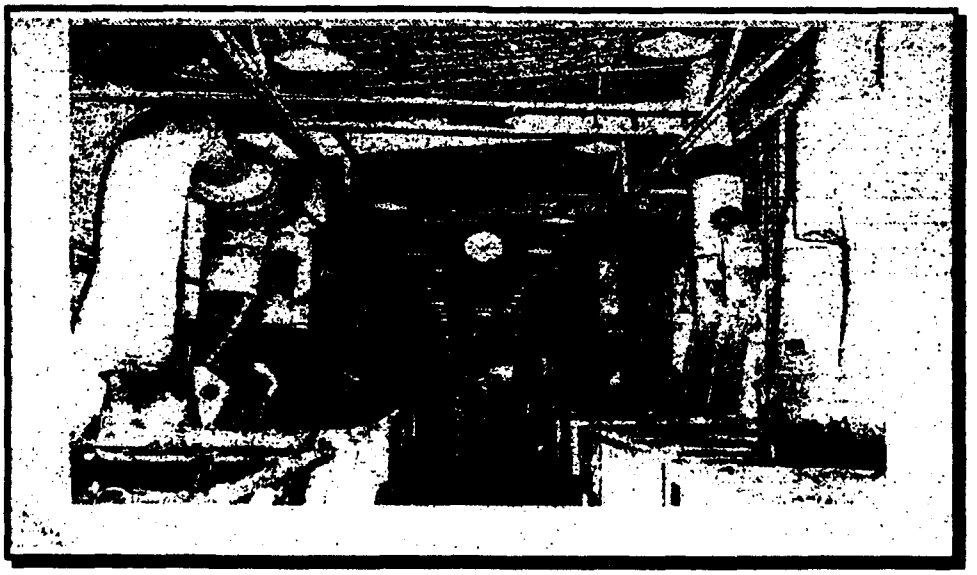
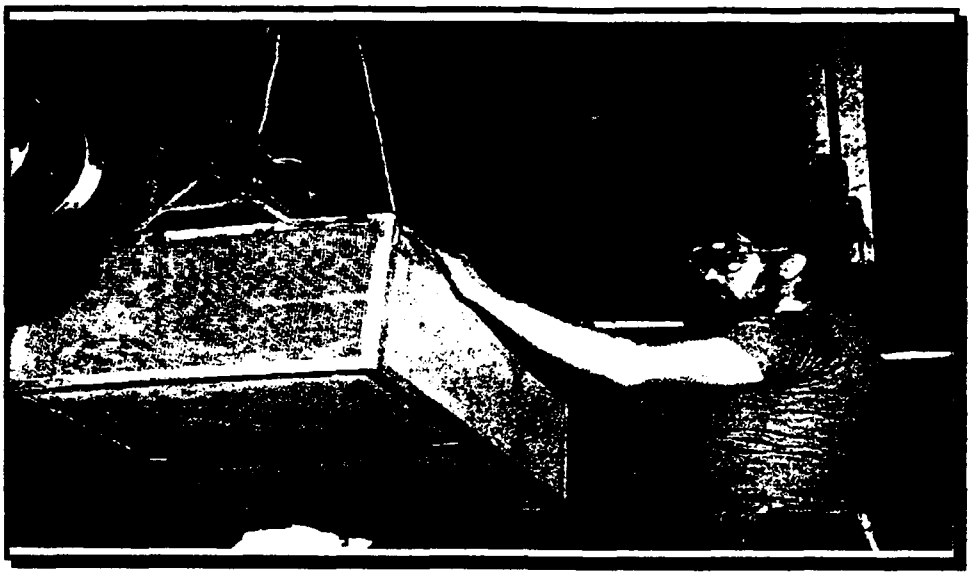


Photo 7: Hill Air Force Base, "Ron Baker dips a basket of parts in a degreaser.", 1977.



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Vita

Anthony O. Copeland was born 10 May 1967, at Duke University Medical Center in Durham, North Carolina. He spent the majority of his childhood in his grandmother's hometown of Warrenton, North Carolina. As a high school sophomore, he competed for and was selected to complete his junior and senior high school years at the North Carolina School of Science and Mathematics located also in Durham. Upon graduating, he attended North Carolina State University where he earned a Bachelor of Science Degree in Electrical Engineering. Commissioned as a Second Lieutenant in December 1989, he was assigned to the 14th Civil Engineering Squadron, Columbus Air Force Base, Mississippi. At Columbus AFB, he served as an electrical design and utility engineer from April 1990 to May 1992. His next assignment took him to Kunsan Air Base, Republic of Korea. While a member of the 8th Civil Engineering Squadron, he held the positions of Chief of Requirements and Chief of Maintenance Engineering. In June 1993, he entered the Graduate Engineering and Environmental Management Program at the Air Force Institute of Technology's School of Engineering.

REPORT DOCUMENTATION PAGE

Form Approved
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1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	September 1994	Master's Thesis

4. TITLE AND SUBTITLE
A RETROSPECTIVE QUANTITATIVE ASSESSMENT OF TRICHLOROETHYLENE EXPOSURE OF WORKERS AT AIRCRAFT MAINTENANCE FACILITIES AT HILL AIR FORCE BASE THROUGH THE USE OF MODELING

5. FUNDING NUMBERS

6. AUTHOR(S)
Anthony O. Copeland, Captain, USAF

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Air Force Institute of Technology, WPAFB OH. 45433-6583

8. PERFORMING ORGANIZATION REPORT NUMBER
AFIT/GEE/ENV/94S-06

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)
Armstrong Laboratory, Toxicology Division
Wright-Patterson Air Force Base, OH 45433
Strategic Environmental Research and Development Program (SERDP)
for the Scientific Advisory Board: Thrust: Installation Restoration (IR-27)
Title: Toxicology and Human Health Risks

10. SPONSORING / MONITORING AGENCY REPORT NUMBER

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12a. DISTRIBUTION / AVAILABILITY STATEMENT
Approved for public release; distribution unlimited

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Monte Carlo simulation and source-receptor modeling are used to estimate the TCE exposures encountered by select workers at aircraft maintenance facilities at Hill Air Force Base between 1955 and 1979. An epidemiological study of this group, a retrospective cohort study of 14,457 workers who were employed at the base for a minimum of one year between 1952 and 1956, was headed by Dr. R. Spirtas of the National Cancer Institute to evaluate mortality associated with occupational exposure. One of the major conclusions of the study was that TCE "probably does not pose a strong carcinogenic risk for man." In the Spirtas study, historic exposure levels were not quantitatively estimated. Instead, indices of exposure to TCE were assigned to reflect relative differences in exposure levels. It is the objective of this research effort to quantitatively estimate specific worker exposures, thus adding to the weight of evidence necessary to classify TCE as a human carcinogen or otherwise.

14. SUBJECT TERMS	15. NUMBER OF PAGES
Exposure Assessment, Monte Carlo Simulation, Hill Air Force Base Historical Exposure Estimation, Trichloroethylene, TCE, Computer Simulation Multi-Point Diffusion Analysis, Occupational Exposure, Vapor Degreasers	101
	16. PRICE CODE

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