Summary

Agent Orange, the United States military code name for a 50/50 mixture of the herbicides 2,4,5-T and 2,4-D, was used during the Vietnam War from 1965-1970 to defoliate jungle vegetation to protect U.S. servicemen from enemy ambush. Both of these herbicides had been used domestically without noteworthy adverse effect for about 20 years at the time they were selected by the military as the components of Agent Orange.

Present at trace levels in the 2,4,5-T component of Agent Orange is an unwanted contaminant, the dioxin compound 2,3,7,8-tetrachlorodibenzo-para-dioxin, commonly known as TCDD or dioxin. TCDD is extremely toxic and has been shown to cause a number of serious health conditions in laboratory animals, including birth defects, cancer, and death. It is not a product, and no one makes it on purpose. TCDD has also been shown to cause a serious skin disorder known as chloracne and reversible signs of toxicity in workers accidentally exposed to extremely high levels on the job.
In spite of the acknowledged toxicity of TCDD, the consensus of scientific opinion is that it has not been shown to cause harm in people at the trace levels at which it has been present in herbicides. Among the organizations which share this view are the American Medical Association; the United Kingdom's Ministry of Agriculture, Fisheries, and Foods; the World Health Organization of the United Nations and the Council for Agricultural Science and Technology.

During the Vietnam War, The Dow Chemical Company supplied about 32 percent of the Agent Orange applied. As a quality control measure, Dow analyzed the 2,4,5-T in all shipments of Agent Orange to the government to ensure the absence -- no detectable level -- of TCDD. Today, with better analytical techniques, we know that the level of TCDD in Dow-supplied Agent Orange was less than 0.5 parts per million.

In early 1979, the first of multiple lawsuits was filed against seven manufacturers of Agent Orange: Dow, the Monsanto Company, Diamond Shamrock Corporation, Uniroyal Inc., T.H. Agricultural and Nutrition Company, Thompson Chemical, and Hercules Inc. These claims have been consolidated into a class action suit in which thousands of Vietnam veterans and their family members will be represented by selected cases. These cases will be tried individually to determine whether Agent Orange has caused health problems to these veterans and their families. The trial is scheduled to begin in early May 1984 in the courtroom of Chief Judge Jack B. Weinstein of the Federal District Court for the Eastern District of New York (Brooklyn, NY).
We all appreciate that Vietnam veterans have been through tough, demanding duty, a difficult experience. Although preliminary findings, such as the Ranch Hand studies and the Veterans' Administration's Agent Orange Registry, have been reassuring, scientific research at this time is insufficient to determine definitively whether the health of the Vietnam veteran and his family is any different from the general U.S. population. We do know that overwhelming scientific evidence demonstrates that Agent Orange is neither a likely nor plausible cause of the health effects some Vietnam veterans and their families have been suffering. Studies which have examined this issue and found no link between Agent Orange and ill health include the Ranch Hand mortality and morbidity studies, the Australian birth defect study released in 1983, and the Veterans' Administration Agent Orange Registry.

There are presently about 95 studies ongoing to evaluate the potential health impacts of TCDD. Essentially, only one of these studies, presently being conducted by the U.S. Centers for Disease Control, considers health effects in the Vietnam veteran from any potential cause other than Agent Orange.
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Approximately 2.8 million servicemen served in Southeast Asia during the Vietnam War, according to the Veterans' Administration. Today, some of these veterans are experiencing a wide variety of health problems. The Centers for Disease Control (CDC) is attempting to determine whether the Vietnam experience is related to these health concerns. One aspect of the Vietnam experience being explored by the CDC, and the one which has received the greatest public and scientific attention, is the claim that the ill health of these veterans is related to Agent Orange exposure.

Agent Orange was a defoliant used by the U.S. military from 1965 to 1970 to eliminate dense jungle vegetation and protect allied troops from enemy ambush. This defoliant was a 50/50 mix of 2,4,5-T* and 2,4-D, two herbicides developed as a result of military research during World War II. Both of these herbicides had been used domestically, and without noteworthy adverse effect, for about 20 years at the time they were selected by the military as the components of Agent Orange. Code named by the orange identification band painted on the storage drum, Agent Orange was usually sprayed from fixed wing C-123 military aircraft. A small amount of the herbicide was also applied from ground sources, such as boats, trucks and backpack sprayers.

The source of today's public controversy is an unwanted trace contaminant, the dioxin compound 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD),

*Note: Underlined terms are defined in the glossary at the back of this paper.
present at trace levels in the 2,4,5-T component of Agent Orange. It should be noted that TCDD is not a commercial product, but rather an unavoidable manufacturing process contaminant. It is not shipped in drums or railcars across the United States.

Considerable controversy surrounds TCDD even today, primarily because of its high acute toxicity. TCDD has been shown to cause a number of serious conditions in laboratory animals, including birth defects, cancer and death. However, according to numerous independent scientific reviews, with the exception of the skin disorder chloracne and reversible signs of toxicity, none of these health conditions have been documented in people, even in cases of severe overexposure.

\[ \text{2,3,7,8-TCDD} \]

We must be concerned with establishing safe levels for human exposure to TCDD in our environment. However, there is extensive scientific knowledge about TCDD which does not suggest that the trace levels of this
contaminant found in herbicides or in the general environment pose a health threat to people.

We must also be concerned about the health problems of individual veterans and their families. These veterans have endured difficult, demanding duty in the service of their country. They deserve answers to their very real concerns. Although preliminary findings should be encouraging to veterans, scientific research is insufficient at this time to determine definitively whether the health of these veterans and that of their families is any different from the general population. However, a great deal of public anxiety and misunderstanding has developed about alleged links between health problems and Agent Orange. According to overwhelming scientific evidence, Agent Orange is not a plausible cause of the various illnesses experienced by some veterans.

The bulk of the scientific studies conducted in the United States, Europe, Australia, and New Zealand among industrial workers, herbicide sprayers, and military personnel demonstrates that the presence of TCDD at trace levels has not shown any long-term adverse health effects in people.
In 1981, the American Medical Association (AMA) published a technical report reviewing the medical evidence regarding the toxicity and long-term health effects of the TCDD contaminant in Agent Orange. As the report's preface states:

"In spite of the voluminous data . . . there is still very little substantive evidence for many of the alleged claims that have been made against these compounds (Agent Orange, 2,4,5-T, and TCDD). The most serious of these allegations assert that Agent Orange, or compounds of a like nature, have caused malignant tumors, spontaneous abortions, and birth defects. Although data from studies on experimental animals tend to support some of these claims, it is not certain that the animal data are extrapolatable to man. No laboratory animal can fully substitute for man; we must, therefore, depend on the results of ongoing epidemiological studies on persons who have been exposed."

Some of what we know of the potential health and environmental effects of TCDD exposure has been due to scientific studies conducted on laboratory animals. Yet as the AMA Advisory Panel on Toxic Substances which produced the report concluded, "there are significant differences between some of the toxic effects of TCDD in experimental animals and the human experience; thus the animal data cannot be translated directly to man."
In terms of acute toxicity, a wealth of epidemiological data support
the position that people seem less susceptible to TCDD than several of
the animal species which have been studied.

According to the AMA Panel, one of the more pronounced biological
effects of heavy exposure to TCDD, as well as a number of other
chlorinated compounds, is chloracne which occurs in humans and some
animals. This particular skin condition is regarded as the clinical
indicator of TCDD overexposure in people. The AMA Panel noted that
human systemic disorders from exposures to TCDD are unlikely to occur
in the absence of chloracne. Such findings as impaired liver and
kidney function, gastrointestinal irritation, muscle and nerve disorders,
depression and fatigue, and central nervous system effects have been
reported after exposure to relatively large amounts of TCDD. However,
these disorders "have not been progressive" and have diminished with
time after exposure ceased, the AMA report stated. In short, if there
is no chloracne, other persistent toxic effects from exposure to TCDD
are "unlikely".

The AMA report also stated that TCDD may act as a promoter of
carcinogenesis in some strains of rats and mice. For cancer to start,
it must go through several distinct steps. The first stage, called
initiation, is produced by a carcinogen (an initiator) and probably
involves irreversible mutational (genetic) changes. Later growth and
development of an actual cancer may require promotion, which could be
the result of additional applications of a carcinogen, or of other noncarcinogenic materials. Promotion may involve additional unknown processes which could aid the growth of the cancer or reduce resistance to the developing cancer.

By way of an analogy, the process of carcinogenesis could be compared to the events leading up to the rupture of a dam. Initiation could be compared to an event which produces a slight crack (genetic damage) in the dam, which in and of itself does not significantly jeopardize the dam's structure. Promotion could be compared to an unusually violent storm which raises the water level in the river, increasing the water pressure on the crack causing increased flow, and widening of the crack to the point at which the structure becomes undermined. Implicit in this analogy is the concept of a threshold in terms of promotion: that is, there is a water level below which the well-being of the structure is maintained, even though a small leak (initiation) was present.

TCDD is among the group of chemicals that may secondarily influence the formation of cancer in animals by promotion. The carcinogenicity associated with TCDD in laboratory animals is typically accompanied by other signs of systemic toxicity, in contrast to some other carcinogens for which cancer is the only observable toxic effect.

In terms of mutagenicity, TCDD has reportedly been studied in various tests using certain bacterial, viral, yeast and tissue cell cultures.
While these in vitro tests have given mixed results, tests using intact animals or human cells in culture indicate little potential for mutagenic effects from TCDD to occur in living higher animals.

The AMA Panel made clear in its summary that the extensive information collected for more than 30 years provides "no conclusive evidence" that either TCDD or 2,4,5-T are "mutagenic, carcinogenic, or teratogenic in man, nor that they have caused reproductive difficulties in the human."\(^3\)

An updated report from AMA is expected in late 1984.

**Other Independent Scientific Reviews**

Other independent scientific groups have reviewed the studies on TCDD.

**United Kingdom Pesticide Advisory Committee Report:** According to this 1980 report, updated in 1983, and commissioned by the Ministry of Agriculture, Fisheries, and Foods, "human health risks posed by dioxin contamination in 2,4,5-T formulations may hitherto have been overestimated." The report states that its authors "cast far and wide" in search of evidence that 2,4,5-T herbicide formulations, used properly, posed a threat to either humans or the environment; however, no such evidence was found. This report considered the presence of the TCDD contamination in those
herbicide formulations, recommended strict control over the contaminant, but found "no valid medical or scientific evidence that 2,4,5-T herbicides harm humans, animals, or the environment if they are used in the recommended way and for the recommended purposes."4

Queensland Cabinet Committee Report: "No evidence exists that the continuation of present and approved use of 2,4-D and 2,4,5-T will in any way harm the health and well being of any members of the general public," according to this 1981 Australian report. Regarding Agent Orange, the report also states that no evidence exists that use of the defoliant "caused any physical disability of the local population" and further adds that the military use of these two herbicides provided no indication "that their approved use in peacetime would cause any hazard to life."5

Council for Agricultural Science and Technology Report: On the presence of TCDD in Agent Orange, this report states that, "One is at first likely to be so overwhelmed by the enormity of the toxicity of the compound that he fails to comprehend the infinitesimal levels of exposure." This early, 1975 report did not examine the issue of carcinogenicity but found "no conclusive evidence of association between exposure to herbicides and birth defects in humans."6
INDUSTRIAL EXPOSURES TO TCDD

From the late 1940s to recent years, a number of chemical process upsets and industrial incidents have occurred around the globe unintentionally exposing people to high levels of TCDD. While these incidents are unfortunate, the knowledge gained by studying the exposed populations is important in evaluating the potential for TCDD to cause human harm. In addition, such knowledge should provide understanding needed to prevent future harm.

The Seveso Incident

The best known and most widely studied incident of community exposure to TCDD followed the July 10, 1976, process accident at the ICMESA trichlorophenol plant at Seveso, Italy. As a result of the accidental release, approximately seven square miles of the countryside were contaminated by chemicals, including TCDD, exposing as many as 30,000 people. The heaviest contamination involved an area of 180 acres (Zone A), occupied by 736 people. TCDD was reportedly detected on soil at levels in excess of 5.5 parts per million (ppm). This concentration is 5,000 times the level of concern later set for TCDD by the Centers for Disease Control (CDC) for evaluating the hazard of residential soil at Times Beach, Missouri. (See Appendix D for chart comparing various reported concentrations of TCDD.) It is also potentially tens of thousands of times greater than any TCDD exposure ground troops could have had to TCDD from the spraying of Agent Orange in Vietnam.
At Seveso minimal effect was noted on plant life as a result of this high exposure; but some wild animals especially herbivorous species did die, particularly in Zone A (nearest the chemical plant). About four percent of the domestic animals in contaminated zones died. ( Virtually all were small animals.) Some of these deaths may have been due to other chemicals released at the same time.

Chloracne, which has generally been regarded as the hallmark, or sentinel sign, of TCDD exposure, was observed in the Seveso population although at generally low rates and primarily in children. The highest incidence of reported chloracne was about 13 to 14 percent among elementary school children in Seveso, and this was generally mild and rapidly resolved in most affected people. Other than chloracne (of which there were ultimately 100 to 200 cases), transient nausea, vomiting, itching, and headache were the most commonly observed symptoms readily attributable to the exposure, which again may have involved chemicals other than TCDD. Headache, stomach and intestinal upset were more commonly noted in individuals with chloracne. In addition, very slight peripheral nerve impairment was reported, and there were also tentative signs of liver toxicity, both of which reversed over time. Immunoresponse was not diminished, nor was susceptibility to infectious diseases increased.

Two years after this incident, 12 researchers involved in the study of reproductive effects at Seveso concluded that no "major event" had
occurred with respect to birth defects, miscarriages (spontaneous abortions), births and deaths. However, changes in birth defect reporting procedures and in the reporting of spontaneous abortions may have obscured any small changes which might have occurred. Data on reproductive outcomes are limited, but one researcher reported that examination of fetuses in 34 cases of medically-induced abortion revealed no evidence of fetal injury attributable to TCDD. Pregnancy outcome in the years following 1976 appears to be comparable with the Western experience.

It is of particular interest that no birth defects were observed during 1977 among the 70 births in Zones A and B. Only those pregnancies coming to term after January 1977 would have been relevant, i.e., those in which July 1976 maternal exposure would have occurred during the critical period for the developing fetus. It should also be noted that many factors are known to cause human birth defects, which occur in three to six percent of live births: for example viral infections, genetic predisposition, smoking, alcohol, and even medications, including excess quantities of certain vitamins.

A long-term epidemiological survey of the 220,000 residents of the Seveso area is being conducted by the National Institute of Environmental Health Sciences (NIEHS) and the International Agency for Research on Cancer (IARC) working group.
Other Exposures From Industrial Incidents

While the Seveso incident provides considerable insight as to the reproductive effects and acute toxicity of TCDD, the latency period is insufficient to draw reliable conclusions about carcinogenicity. Studies of other industrial incidents are more revealing in terms of the potential of TCDD to cause cancer in people.

Nitro, West Virginia: In 1949, an industrial accident occurred at a Monsanto trichlorophenol plant resulting in 122 cases of chloracne among the workers. Of those exposed 121 were monitored for 30 years after their high peak exposure to TCDD. To date, there is no apparent excess in terms of total deaths, deaths from cancer or deaths from diseases of the circulatory system. Of the total cancers, there was one case of soft tissue sarcoma. A recently reported 1979 cross-sectional study of herbicide production workers at this plant concluded that it is "unlikely" that "permanent, severe, and debilitating" toxic effects are "inevitable" after exposure to TCDD sufficient to produce chloracne. The study also noted that individual susceptibility may make certain heavily exposed workers more vulnerable but found that "even severe acute toxicological effects of TCDD were reversible or markedly improved over time." 

Midland, Michigan: In 1964, a process change in a Dow trichlorophenol plant resulted in the unintentional exposure of 61 workers
to TCDD levels potentially as high as 6,000 to 10,000 parts per million. Of those exposed, 49 workers had some evidence of chloracne. All 61 of these workers have been monitored over the 20 years since their exposure. When last reported in 1980, no excess had occurred in terms of total deaths or deaths from diseases of the circulatory system. (There were, in fact, fewer deaths in both categories than statistically expected.) Cancer deaths have been slightly elevated (3 deaths v. 1.6 expected), but no single type of tumor was predominant. Of the three cancer deaths, there was one case of soft tissue sarcoma.

An additional study including both Dow and Monsanto occupationally exposed groups is being conducted by the National Institute of Occupational Health and Safety (NIOSH).

Other exposures to TCDD from industrial incidents have occurred outside the United States.

**Bolsover, United Kingdom:** As a result of a 1968 explosion in a Coalite and Chemical Products, Ltd. plant manufacturing 2,4,5-T from trichlorophenol, 79 workers developed chloracne, which was the only consistent finding. Initial findings on these men suggested some change in white blood cell counts, and in three
cases, an elevation of glucose in the urine, suggesting possible liver and kidney damage. But a repeat screening 10 days later did not confirm this pattern: all of the tests gave values within normal limits.\textsuperscript{21,22} It should be noted that the design of this study has received strong criticism, since less than half of the workers who developed chloracne were included.\textsuperscript{23} No findings on mortality are reported. While most cases of chloracne were resolved within a matter of months following the accident, new cases were noted in a report nine years later which found that one-half the workers studied who had once had chloracne were still suffering from "minor" cases of the skin disorder. Other than the chloracne, the study noted "no evidence" that the workers had been "adversely affected in any way."\textsuperscript{24}

\textbf{Ludwigshafen, Germany:} Due to a process accident in 1953 at a BASF Aktiengesellschaft trichlorophenol plant, 55 workers developed chloracne.\textsuperscript{25} Peripheral nerve damage and liver toxicity were reported.\textsuperscript{26} Due to insufficient decontamination, an additional case of chloracne developed five years later.\textsuperscript{27} Follow-up studies 27 years later reported no excess in total deaths, but did find an excess of total cancer deaths (7 v. 4.1 expected). Of these, three deaths were due to stomach cancer (v. 0.6 expected).\textsuperscript{27} However, the study's authors note that, "Many of the workers in the factory for some time had probably been exposed to other chemicals and the exposure to dioxin was an additional experience of temporary duration."\textsuperscript{27}
Amsterdam, The Netherlands: As a result of an explosion at an NV Philips herbicide factory in 1963, 145 people were exposed to TCDD, 69 of whom had definite signs of chloracne. Liver function tests did not indicate damage. Twenty years later, "no significant differences" were found between those workers and a matched control group. Data on cancers did not show any organ-related pattern and are not considered to represent any excess mortality.

Times Beach and Imperial, Missouri: Mismanagement of waste in 1971 by a now defunct company which produced hexachlorophene led to the spraying of TCDD-contaminated waste oils for dust control on southern Missouri roads and two horse arenas. Analysis 20 years later found that the waste oils may have contained as much as 350 parts per million of TCDD. In both of the arenas, a number of animals died; and in one of them, in which later analysis revealed about 33 parts per million of TCDD in the soil, reports indicate that two children developed lesions resembling chloracne. (See Appendix D for chart comparing various reported concentrations of TCDD.) Testing in Times Beach and Imperial in 1983 revealed TCDD in soil ranging from non-detectable levels to 300 parts per billion, a hundred times less than that found in the horse arena. Recent testing of 68 residents with presumed high exposure to TCDD found no cases of chloracne and no difference, in comparison with a control group, in birth defects, infertility, impotence, headaches, loss of memory or cancer rates. Slightly more urinary
product at this facility resulting in an internal standard developed in 1965 of less than one part per million, which was undetectable by analytical standards at the time.

It is important to note that most of these industrial incidents represent essentially worst-case scenarios involving exposures considerably higher than any that could be derived by ground troops, even if directly sprayed with Agent Orange: in some cases, the exposures were potentially from 350,000 to ten million times greater. The only lasting, consistent finding documented in these industrial incidents is chloracne.

**HYPOTHESES REGARDING THE HEALTH EFFECTS OF TCDD**

**Soft Tissue Sarcoma**

Perhaps the most publicized hypothesis about TCDD and human health involves the possible causal relationship between the compound and soft tissue sarcomas. In some studies based on detailed technical knowledge of the chemical process and clinical evidence of excessive exposure (chloracne) there is evidence that TCDD is at least one of a number of common exposures. In other reports the evidence is more presumptive, and in some cases it is totally lacking. To date, the bulk of the evidence does not demonstrate a causal link between TCDD and soft tissue sarcomas.
Soft tissue sarcoma is a generic term for a group of lesions that include more than 100 different types of rare cancers.\textsuperscript{36} Most experts believe that if TCDD were acting as a carcinogen it would cause an increase in one type of soft tissue sarcoma rather than causing an increased risk across the board. For example, one of the reasons vinyl chloride is recognized as a human carcinogen is the specific way it acts. At high doses it has been associated with a single type of soft tissue sarcoma: angiosarcoma of the liver.

The soft tissue sarcoma hypothesis derives from the epidemiological work reported in 1979 by Dr. Lennart Hardell and his associates in Sweden.\textsuperscript{37} No one has been able to replicate Dr. Hardell's findings, and his studies have been strongly criticized because of the potential for observer and recall bias in addition to other confounding elements.\textsuperscript{38,39}

Dr. Hardell first administered mail questionnaires in his studies then followed up with telephone interviews on a selected subset of subjects. While the questionnaire was designed with the idea of masking the intent of the investigation, the telephone inquiry specifically focused on the exposures of pertinent interest, which included 2,3,5-T and 2,4-D. Interviewers were given extensive information on the purpose of the study and could easily have known which subjects were soft tissue sarcoma cases. Interviewers probed respondents carefully only about occupations in which exposures to TCDD were likely.\textsuperscript{38}
Recall bias is also involved in that workers are unlikely to remember with accuracy the chemicals they encountered some years in the past, and it is also extremely difficult to estimate the extent or duration of the exposures. It should be noted that out of 15 chemicals evaluated by Dr. Hardell, only one, sodium chlorate, did not show an elevated risk. Nor is it known what elevations would have been shown for these other agents if they had been probed for in the targeted approach as for 2,4,5-T and 2,4-D.

Additional problems are posed by the rareness and diversity of these tumors. Few pathologists have been able to achieve a high degree of consistency in their diagnoses. Further, the identification and classification of soft tissue sarcomas have not been consistent over time.

The significance of this last point is illustrated by findings announced by Dr. Marilyn Fingerhut, an epidemiologist for the National Institute for Occupational Safety and Health (NIOSH), at an October 1983 dioxin conference at Rockefeller University. Prior to the conference, seven cases of soft tissue sarcoma had been reported in the literature among United States chemical plant employees with presumptive exposures to herbicides. These reports seemed to support the findings of Dr. Hardell. At the conference, however, a different picture emerged. NIOSH announced, after detailed evaluation of the employees' work histories and medical
records and microscopic examination of the tumors, that two of the seven had been misdiagnosed: the two deaths were due to carcinomas, not soft tissue sarcomas. In addition, of the remaining five, three were judged by NIOSH not to have had documented TCDD exposure. Further, neither of the remaining two cases had been exposed to 2,4,5-T but rather to the feedstock trichlorophenol. Furthermore, both of these cases had been exposed during upset operating conditions involving relatively high TCDD exposures sufficient to cause chloracne. Both of remaining cases were malignant fibrous histiocytomas.

At present, there is a growing body of literature which does not support Dr. Hardell's hypothesis.

**New Zealand:** No association between the use of these phenoxy herbicides and soft tissue sarcoma has been found in an ongoing case control study in New Zealand, where herbicide application is a registered profession. According to preliminary results published in 1982, not one of the soft tissue sarcoma patients had ever worked as a licensed applicator of these herbicides.41

**Washington State:** No consistent patterns of death due to soft tissue sarcomas were found among occupations related to the use of these herbicides. The two occupations found to be at highest risk in this 1982 review were marine engineers and bankers.42
Finland: No soft tissue sarcomas were found among 1,900 Finnish herbicide applicators in this 1982 review. Nor was the death rate for these applicators different from any natural cause in comparison with the Finnish total male population.43

Midland, Michigan: A statistically significant excess of connective soft tissue cancer has been found by the Environmental Protection Agency among white females in Midland County (where Dow has production facilities). The excess amounted to 13 deaths over a 20-year period. In some cases onset of the disease had begun before the woman moved to Midland. In its 1983 preliminary report, the Michigan Department of Public Health (MDPH) was unable to link the excess with any environmental factor, including TCDD. As part of this study the MDPH reviewed data for 28 other counties in which TCDD or other dioxins were likely to be produced as a chemical manufacturing contaminant. No increase in connective and soft tissue cancers was found in these counties versus those that did not have industrial manufacturing sources suspected of generating such dioxins.44 Research continues on the problem by the MDPH, funded by a $250,000 grant by Dow to the state of Michigan.

Contrary to expectations based on Dr. Hardell's findings, even in Sweden recent reports from Stockholm's Karolinska Institute indicate that Swedish farmers, about 15 percent of whom use the herbicides Hardell
probed for, have experienced slightly fewer cases of soft tissue sarcoma than expected, in comparison with the overall Swedish data.\textsuperscript{45} The only way that Dr. Hardell's findings and those of the Karolinska Institute could both be correct would be if those Swedish farmers not exposed to these herbicides developed soft tissue sarcomas at somewhere between one quarter and one half of the rest of the Swedish population. This seems unlikely.

In addition to the above, it should also be noted that no deaths from soft tissue sarcomas were noted in the U.S. Air Force Ranch Hand mortality study of servicemen who sprayed Agent Orange in Vietnam;\textsuperscript{46,47} nor were any cases of soft tissue sarcoma noted in the subsequent morbidity study. Further, examinations by the U.S. Veterans' Administration of 85,000 self-selected Vietnam veterans have found fewer cases of soft tissue sarcomas than would be expected from the national average.\textsuperscript{48} In the latter case, however, documented exposure to Agent Orange is uncertain; and the registry was not intended as an epidemiological study. Finally, the hypothesis that an association exists between TCDD exposure and soft tissue sarcomas is not supported by research on laboratory animals: animals fed TCDD during laboratory experiments did not develop soft tissue sarcomas.

Additional research is underway in the United States and elsewhere which should provide additional perspective on the soft tissue sarcoma hypothesis.
Reproductive Effects

Laboratory tests have shown that certain levels of TCDD can cause a variety of reproductive disorders in pregnant animals. In pregnant mice certain levels of TCDD can cause birth defects. In pregnant rats exposure to certain levels can be toxic to the fetus causing conditions such as low birth weight. In pregnant women, however, studies to date have found none of these effects from TCDD. In addition, there is no medical or scientific support for claims that toxic effects in a male from exposure to TCDD could impact a developing fetus in the female.

The most widely publicized claims that the spraying of herbicides contaminated with TCDD could cause reproductive disorders stems from the 1978 Alsea II study by the Environmental Protection Agency (EPA) which reportedly found a link between herbicide spraying and spontaneous abortions in Alsea, Oregon. But this study has been severely and almost universally criticized in the scientific community. At least 18 separate critiques of the study have found that its conclusions are not supported by its data. While some of these are brief assessments, the longest is a 100-page report by an interdisciplinary task force at Oregon State University, which concluded that, "If there is a relationship between herbicide use and miscarriages in the 'Alsea Basin'... it is not apparent and cannot be tested using the data from the Alsea II study." More recently, EPA's handling of the incident also came under criticism by the Agency's inspector general. Among the limitations
of the Alsea II study was the lack of evidence that the affected women ever came in contact with the herbicide. In addition, there was no dose/response effect: miscarriages did not increase substantially when herbicide application was doubled.

The EPA's 1979 Alsea II study findings resulted in the suspension of the major uses of the herbicide 2,4,5-T. Dow no longer manufactures 2,4,5-T in the United States. (A partially owned Dow subsidiary manufactures 2,4,5-T in New Zealand.) The following studies were conducted to determine whether TCDD causes reproductive disorders in humans. None of the studies support the conclusions of Alsea II.

**Yarram District, Australia:** This 1978 investigation by the Australian Commission of Public Health failed to find any link between the use of 2,4,5-T and 2,4-D and birth defects in people or domestic animals. 52

**Arkansas, 1948 to 1974:** This 1979 investigation found no association between 2,4,5-T and facial cleft defects in children. 53

**Long Island Railroad:** This study by the National Institute for Occupational Health and Safety (NIOSH), also conducted in 1979, found no definite excess of birth defects related to 2,4,5-T exposure in maintenance workers exposed to the herbicide, which was used for weed control along the railroad tracks. 54
Hungarian Agriculture and Forestry Workers: This 1980 study found that despite a large increase in the use of 2,4,5-T in that nation between 1969 to 1975, levels of various birth defects either remained stable or declined.\textsuperscript{55}

New Zealand 2,4,5-T Applicators: This 1982 study conducted by researchers at the Wellington Clinical School of Medicine found no statistically significant increase in relative risk among male professional 2,4,5-T sprayers in terms of birth defects or miscarriages. Wives involved with spraying activities or who washed contaminated clothes also had no detectable reproductive effects.\textsuperscript{56}

Midland, Michigan: This 1983 investigation by the Michigan Department of Public Health (MDPH), updating a previous report on an increase of birth defects in Midland County from 1971 to 1974, found that the increases "may not have been unusual" and speculated that the fluctuations may have been due to changes in classification and reporting of birth defects.\textsuperscript{57}

Further light is shed on the issue of reproductive disorders and the potential for toxic effects of TCDD to be transmitted from the male to the fetus by a study conducted by Dow Chemical on the wives of employees involved in herbicide production. In this 1982 study, the test group consisted of wives of employees with potential TCDD exposure, while the
control group consisted of wives of employees without such potential exposure.

One phase of analysis dealt with potential effects on fertility. The average number of pregnancies among wives of 370 employees with potential exposure to TCDD were compared to those among wives of 345 employees without such presumed potential exposure. Results showed no decrease in pregnancies among the test group: in fact, the test group had more pregnancies than the control.58

Another phase of analysis examined miscarriages, stillbirths, birth defects and infant deaths in terms of potential TCDD exposure. (In this phase, it was necessary to redefine the births in the test and control groups, because some pregnancies in the test group took place before the father's presumed dioxin exposure. This required the transfer of 766 births from the test group to the control.) Analysis found no statistically significant associations between TCDD exposure and pregnancy outcomes.59

Synergism and TCDD

Unsubstantiated claims have been made that TCDD exposure could account for any number of ill effects in humans, because of its ability to enhance the toxic effects of other compounds. A Dow Chemical mortality
report on 8,181 employees working in a large production complex involving as many as 500 distinct chemical processes found no observable adverse effects. This 24-year follow-up from 1954 to 1978 found overall mortality to be 19 percent less than that expected for the corresponding U.S. population. While the healthy-worker factor could have played a part here, if low-level TCDD exposure could cause a variety of increased illnesses, an increase in mortality should have been found, not a decrease.

AGENT ORANGE AND THE VETERAN

On the basis of the considerable knowledge of the human health impact of TCDD gained from instances of high-level industrial exposure and long-term occupational exposure, it might seem surprising that eight years after the herbicide program in Southeast Asia ceased health claims were filed by veterans alleging a link between their current health status and what they believe to have been exposure to Agent Orange. An excess in total cancers has not been seen in workers exposed to high levels of TCDD at Nitro, West Virginia; Amsterdam, the Netherlands; or Times Beach and Imperial, Missouri. Nor have studies found reproductive effects from low-level TCDD exposures as a result of herbicide spraying in Australia; Hungary; New Zealand; or Times Beach and Imperial, Missouri; nor from male railroad worker exposures on Long Island, or from the exposures of male herbicide production workers in Midland, Michigan.
Further, male mice fed simulated Agent Orange containing two parts per million TCDD (the average TCDD concentration in the Agent Orange used in Vietnam) did not show any reproductive disorders when mated with female mice after eight weeks of exposure.\textsuperscript{61} (See Appendix C.) In addition, in contrast to other documented cases of TCDD poisoning, in which chloracne serves as an indicator of overexposure, no cases of chloracne have been documented by government physicians in any of the Vietnam veterans examined.\textsuperscript{48}

On the basis of the available information, Agent Orange does not seem either a very likely or plausible explanation for the health problems experienced by some veterans and their families. Nor is there any scientific documentation to date that the health of Vietnam veterans as a group is any different from that of the general population. Nor until now has any concerted scientific effort been made to determine if the veterans of the Vietnam conflict or any other war have suffered any long-term health problems from any wartime experiences.

The plaintiffs in the present litigation represent less than one-tenth of one percent of the 2.8 million veterans who served in Vietnam. Many of these plaintiffs are very ill, from undetermined causes, and need help. While their problems are real and legitimate, their link to Agent Orange has yet to be established.
There are about 95 ongoing state and federal studies relating to TCDD and its potential health impact; however, few of these studies have relevance to the Vietnam veteran exposed to Agent Orange. Essentially, only one of these studies, which is being conducted by the Centers for Disease Control, examines in detail the potential for adverse health effects in Vietnam veterans from a variety of potential causes other than Agent Orange. The following studies have already been conducted to examine the potential for Agent Orange to cause harm, among Vietnam veterans allegedly exposed.

**Ranch Hand Studies**

In a 1983 study of more than 1,200 servicemen who applied Agent Orange in Vietnam and who were deemed by the U.S. military to have had the highest exposures to TCDD, there was no indication that herbicide exposure had any effect to date on their mortality. These servicemen are estimated by the military to have had exposures to Agent Orange lasting from ten to 12 hours a day, five to six days a week, for periods of at least a year. Each of these servicemen was matched in this study with five other comparisons based on similar job, race, age and month of birth, wherever possible. In addition, the Ranch Handers were compared with the 1978 Department of Defense Nondisability Retired Life Table and the mortality experience of the West Point class of 1956.
Results showed no adverse mortality trends among the Ranch Hand veterans. The Ranch Hands were found to have 40 percent fewer deaths than expected, compared to the statistics for U.S. white males.

While chloracne has been the most consistent finding among workers exposed to high levels of TCDD, a morbidity study recently completed on the Ranch Hands has found no chloracne in the group. While soft tissue sarcomas and porphyria cutanea tarda have been hypothesized as possible adverse outcomes of TCDD exposure, the recent study noted neither of these disorders among the Ranch Hands nor was there any excess in total cancers.

Some differences did exist between the Ranch Hands and the control, however. Among these findings were apparent increases in self-reported neonatal deaths and minor birth defects and an excess of nonmelanoma skin cancers. While these findings merit further investigation of the potential health impacts of the Vietnam experience, it should be noted that no exposure/response relationship with Agent Orange could be established. That is, those Ranch Hands with the highest presumed exposures to Agent Orange did not have a greater frequency of disease, neonatal deaths or birth defects when compared to less-exposed Ranch Hands.

Further, it should be noted that because the neonatal deaths and birth defects were self-reported, medical confirmation is needed before they
can be adequately interpreted. In addition, the skin cancers were of a type that is highly curable and which appears to be related to sunlight. That is, geographical areas with greater sunlight exposure have more cases of these skin cancers. The Air Force study notes that these apparent excesses of skin cancers among Ranch Hands have not been adjusted for sunlight in terms of geographical location of residence.47

**Australian Birth Defect Study**

According to this 1983 study, commissioned by the Australian Ministry for Veterans' Affairs, no evidence was found that army service in Vietnam has increased the risk of birth defects. The study was conducted by searching the records of 34 Australian hospitals and cytogenetics laboratories for birth defective children born to any of that country's veterans who served in Vietnam between 1962 and 1972. The study "gives persuasive evidence that Vietnam service has not been associated with any important increase in the risk of birth defects in children of veterans."63

A similar study of U.S. Vietnam veterans living near Atlanta, Georgia, is scheduled to be released in spring of 1984.
"No convincing evidence" was found in this 240-page report of the Australian Standing Committee on Science and the Environment in its investigation of the potential for Agent Orange to cause harm that the rates of birth defects or mortality were excessive among Vietnam veterans. Further, the Committee concluded that Agent Orange is an improbable cause of birth defects in children of Vietnam veterans. "The Committee believes," the report states, "that there is no biologically plausible mechanism whereby the father's exposure in Vietnam can lead, years later, to exposure of the fetus in the uterus, as would be required to produce teratogenic birth abnormalities." The report also noted the "striking similarities of the veterans' disorders in comparison with those found among veterans of World War I, World War II, the Korean War, and the Arab/Israeli Wars." No conclusion on carcinogenicity was reached by the report; the Committee believed there was insufficient evidence to make an assessment either way.

U.S. Agent Orange Registry Findings

No "unusual long-term morbidity or mortality associated with Vietnam service or Agent Orange exposure" was found in this recent Veterans'
Administration evaluation of 85,000 Vietnam servicemen who presented themselves to the agency for examination. While this assessment cannot be considered a full-fledged epidemiological study, it should be noted that "of the several thousand veterans complaining of dermatologic problems, only one may possibly turn out to be chloracne." While the report notes "a wide variety of health problems," they were "of the sort one sees in a population of males growing older."  

National Health Statistics and the Veteran

The studies just cited attempt to determine if the health effects of Vietnam veterans can be related to their potential exposure to Agent Orange. Results of these studies should be encouraging to the veteran, and as noted previously, further study continues. Statistics indicate, however, that serious medical conditions do occur in the normal course of events. Given a large enough population, these conditions occur with statistical regularity.

Presently there is no certainty that the 2.8 million Vietnam veterans as a group are, or are not sick, or dying from unusual causes or at unusual rates. There is some certainty, however, what the vital statistics of a group the same size, age, and sex as the veterans would be: that is, national health statistics suggest how many people in a similar population would die within a given time frame, and from what causes.
On the basis of previous research, over the ten year period 1970 to 1979, 48,592 veterans would have been expected to die. Accidents, homicides and suicides would be expected to account for the largest portion of these deaths: 30,972. An additional 17,620 deaths would occur from various physical disorders:

- Heart disease and stroke: 4973
- Cancer: 3819
- Disease of the digestive system: 2109
- Diseases of the respiratory system: 1193
- Mental disorders: 1010
- Diseases of the nervous system: 817
- Diseases of the endocrine system: 633
- Other physical disorders: 3066

It should be noted that these are statistics based on the mortality of U.S. white males. These are deaths that could be expected to occur in the "normal" course of events. In effect, what the study suggests is that many of the claims being made by the plaintiffs in the present litigation are no different from the types of ailments that would be expected in a similar group from the general population.
Recent Developments

In July 1983, TCDD was described as a probable human carcinogen by a panel called by the Environmental Protection Agency (EPA) to review the literature on the compound. A primary reason for this determination was the presence of seven cases of soft tissue sarcoma in a registry maintained by the National Institute for Occupational Safety and Health (NIOSH) of about 6,000 workers potentially exposed to TCDD. However, in October of the same year, NIOSH announced that three of this seven did not have significant dioxin exposure, and two of the remaining four had been misdiagnosed: they had died of carcinomas, not soft tissue sarcomas. Neither EPA's nor NIOSH's findings in this regard have been published yet in the scientific literature.

In September 1983, the Supreme Court of Nova Scotia ruled in favor of the continued use of 2,4,5-T in that province, denying plaintiffs an injunction to prevent the spraying. According to Justice D. Merlin Nunn, "This court is of the opinion that these spraying operations can be carried out in safety and without risk to the citizens of this province." In his decision Justice Nunn criticized witnesses for the plaintiffs for partisanship and refusing to accept criticism of studies supporting their points of view. By contrast, Justice Nunn stated that he did not detect partisanship on the part of the defendant, Nova Scotia Forest Industries, and accepted the evidence of the defendant's witnesses as representing the generally accepted view of responsible scientists.
In December 1983, a group of scientists attending an international conference on TCDD at Michigan State University concluded that humans appear to be less sensitive to TCDD than some laboratory animals, and that insufficient evidence exists to indicate TCDD is a human carcinogen. While the report noted the potential for TCDD to cause reproductive effects in pregnant women exposed to high levels, such as the most contaminated areas of Seveso, Italy, it is also noted that there is no evidence that TCDD can cause birth defects through paternal exposure.67

According to press accounts, researchers attending this conference stated that TCDD does not warrant "an exceptionally high public policy priority which diverts resources and public attention from other more widespread and dangerous compounds." However, the announcement noted that "hot spots" where major concentrations of TCDD exist should continue to be studied.68

Conclusion

Considerable research is being directed towards determining whether TCDD at trace levels poses a risk to human health. A considerable amount of research has already been conducted. This research suggests that:

1. TCDD causes chloracne, the most consistent indicator of a toxic exposure, at relatively high levels;
2. At higher levels of exposure, TCDD causes transient liver and nerve dysfunction;

3. TCDD does not adversely affect human reproduction from toxic exposures sustained by the father;

4. No deaths from TCDD exposure have been documented, nor has exposure to the compound been shown to increase the risk of dying at some later period, whether from all diseases in aggregate or total cancers; and

5. More research is needed to clarify the relationship, if any, between TCDD exposure and soft tissue sarcoma.

Based on the conclusions of this research, Agent Orange exposure as a result of military spraying to protect American servicemen in Vietnam does not seem a likely cause of the health effects presently suffered by some veterans and their families. Although preliminary findings to date should be encouraging to veterans, scientific research is insufficient to determine definitively whether the health of Vietnam veterans as a whole is any different from that of the general population. Further research is needed, and is now underway, on the potential health impact of the entire Vietnam experience on our veterans, who endured tough, demanding duty in the service of their country.
A BRIEF HISTORY OF THE AGENT ORANGE CONTROVERSY

Herbicide Use In Vietnam

Agent Orange, the United States military code name for a 50/50 mixture of herbicides 2,4,5-T and 2,4-D was used during the Vietnam War from 1965-1970 to defoliate jungle vegetation and to protect U.S. servicemen from enemy ambush.

During that time it is estimated that 10 percent of Vietnam was defoliated with herbicides and 60 percent of that area was sprayed with Agent Orange. (In terms of perspective, it might be noted that in 1982 the gypsy moth defoliated eight million acres in the Northeastern states according to the U.S. Forest Service -- twice as many acres as were defoliated during the entire Vietnam War.) Agent Orange was usually sprayed from C-123 (fixed-wing) military aircraft. A small amount of Agent Orange was applied from ground sources such as boats, trucks and backpack sprayers. The total amount of Agent Orange used during the herbicide program is estimated at between 10 and 12 million gallons.69

Studies indicate that only about six percent of the Agent Orange sprayed over Vietnam ever penetrated the dense foliar canopy to reach the jungle floor; and of the 94 percent caught in the dense foliage, direct exposure
to sunlight would have degraded most of the TCDD within 24 hours. The remaining TCDD which might have reached the jungle floor would have been tightly bound to the soil and therefore been less available for human contact. (Under these conditions, TCDD levels in Vietnamese soil from Dow-supplied Agent Orange would have been more than 100 times less than the level of concern recently set at Times Beach by the Centers for Disease Control for residential areas. See Appendix D.)

Development of Agent Orange

During World War II, the United States military began studying the use of 2,4,5-T and 2,4-D, during field trials to determine their capability in defoliating large areas of vegetation in tropical battlegrounds. Military experiments with these herbicides continued through the 1940s and 1950s. In 1961, a defoliant similar to Agent Orange was selected by the U.S. Secretary of Defense to be tested as the herbicide of choice for use in Vietnam.

In the early 1960s the Office of the Surgeon General, Department of the Army, conducted research on the trace contaminant TCDD. The published results acknowledged the work of German scientists Drs. Kimmig and Schulz who observed cases of chloracne among workers engaged in the production of 2,4,5-T from trichlorophenol in the late 1950s. Kimmig and Schulz had reported TCDD to be a contaminant of this manufacturing process.
In 1962, the U.S. Government published research sponsored by the Research and Development Division of the Office of the Surgeon General, Department of the Army and by the U.S. Public Health Service which reported on the process to determine the existence of TCDD in certain chemical products. This method, known as the rabbit ear test, was designed by Dow to measure skin response (chloracne) from TCDD exposure, as well as from other chemicals.

In 1965, the U.S. military decided upon a 50/50 mixture of 2,4,5-T and 2,4-D which later came to be known as Agent Orange. The military subsequently determined the rates and frequencies of Agent Orange application throughout the Vietnam herbicide program as well as the areas to be sprayed. As used in Vietnam Agent Orange has not been prescribed for any domestic weed control applications.

Dow supplied about 32 percent of the Agent Orange to the U.S. Government for the war effort. As a quality control measure Dow analyzed the 2,4,5-T in all shipments of the Agent Orange it supplied the government to insure the absence -- no detectable level -- of TCDD, which given the technology of the day meant less than 0.5 part per million in the Agent Orange.

**Agent Orange Lawsuits**

In early 1979, the first of multiple lawsuits was filed against seven manufacturers: Dow, the Monsanto Company, Diamond Shamrock
Corporation, Uniroyal Inc., T.H. Agriculture and Nutrition Company, Thompson Chemical, and Hercules Inc. alleging a connection between the spraying of Agent Orange in Vietnam and various maladies reported by some Vietnam veterans and their families.

These claims have been consolidated into a class action suit in which thousands of Vietnam veterans and their family members will be represented by selected cases. These model cases will be tried individually to determine whether Agent Orange has caused health problems to these veterans and their families. The trial is scheduled to begin in early May 1984 in the courtroom of Chief Judge Jack B. Weinstein of the Federal District Court for the Eastern District of New York.

In the meantime, more than 95 studies are underway to determine the validity of health claims involving dioxin and Agent Orange.

The results of these and other ongoing studies should help to address public anxiety over the alleged link between Agent Orange and human health problems.
CONSPIRACY OF SILENCE CONTENTION
IN THE AGENT ORANGE CONTROVERSY

Much of the present plaintiffs' case has centered around the so-called "conspiracy of silence" claim. In brief, this theory claims that Dow knew that Agent Orange was harmful to users and met with other chemical producers deliberately to conceal this information from the government. The theory does not hold up under scrutiny.

An examination of the chronology of events surrounding the selection of Agent Orange by the military for use in Vietnam shows 1) that the United States government had extensive knowledge of both the components of Agent Orange as well as the potential occupational health effects of the contaminant TCDD; 2) this knowledge on the part of the government was concurrent with that of Dow and the other co-producers; and 3) that Dow's concerns about TCDD centered around the potential of relatively high levels of the compound to cause harm in the workplace not on the end uses of the finished product which contained vastly lower levels of the contaminant.

Chronology

1949 The U.S. Public Health Service (USPH) investigates the chloracne incident at a trichlorophenol production plant at Nitro, West Virginia.
1957 Having determined TCDD to be the cause of chloracne in its trichlorophenol workers, the German firm Boehringer Sohn sends a letter to Dow Chemical regarding the availability of technology to address the problem. Dow has no chloracne problem at the time of receipt of the information, and the letter is filed for future reference.

1959-60 United States military personnel at Edgewood Arsenal, Fort Detrick, and the Army Corps Research and Development Laboratory become aware of the chloracne potential in trichlorophenol manufacture, through the research of two German scientists, Kimmig and Schulz. TCDD is considered briefly by the military as a weapon of war.

An article published by the USPHS indicates knowledge that chloracne was associated with 2,4,5-T production.

1962 Military personnel at Fort Detrick recommend a defoliant formulation, later to become known as Agent Orange, for use in Vietnam.

Research supported by the Research and Development Division of the Office of the Army Surgeon General indicates both knowledge of TCDD's acnegenic potential and that the contaminant could be produced at high temperatures from trichlorophenol.
1963 The USPHS completes investigation of a chloracne incident at a New Jersey 2,4,5-T production plant.

1964 A process change in Dow Chemical trichlorophenol production at Midland, Michigan, exposes 61 workers to high levels of TCDD. Of those exposed, 49 workers have some evidence of chloracne. The trichlorophenol plant is shut down for a period to resolve the problem. TCDD is first suspected, then confirmed, as the cause of the problem.

1965 Dow establishes an internal standard for detectable TCDD at an analytical limit of one part per million.

Dow calls a meeting with its competitors to discuss the presence of the acnegen in trichlorophenol.

Dow also informs the USPHS, the Michigan State Department of Public Health, the University of Michigan Institute for Industrial Health and other Michigan universities, the United States Army Biological Laboratories at Fort Detrick, the Ontario Department of Labor, and other government agencies of the Midland trichlorophenol chloracne incident. Also notified were a number of physicians and health officials from private industry, and from foreign countries including the Netherlands, England, South America and the Soviet Union.
Military personnel at Fort Detrick request toxicity data on TCDD from Edgewood Arsenal.

Late in the year, Dow supplies the military with its first shipment of Agent Orange.

1966

A new Dow trichlorophenol plant comes on line at Midland, Michigan, built with technology licensed from Boehringer Sohn in Europe.

The Army Surgeon General's Office requests toxicity information on 2,4,5-T from the National Academy of Sciences (NAS); the NAS response references porphyria and chloracne as effects of exposure.

Personnel at Eglin Air Force Base learn of the potential for TCDD to cause occupational harm as the military considers construction of its own 2,4,5-T plant, due to short supply of Agent Orange.

1967

With regard to the government's plan to build its own Agent Orange production facilities, Dow Chemical informs Edgewood Arsenal and the Department of Defense of the "serious potential health hazard" (chloracne) to 2,4,5-T industrial plant workers.